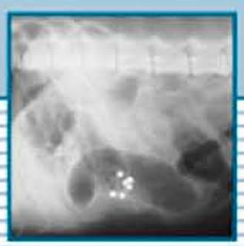
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MARJORIE **CHANDLER**

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SMALL ANIMAL GASTROENTEROLOGY

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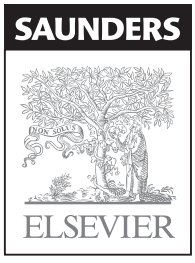
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SMALL ANIMAL GASTROENTEROLOGY

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Introduction

Saunders Solutions in Veterinary Practice series is a new range of veterinary text books covering all the main disciplines of companion animal practice.

Readers should realise that it is not the authors' intention to cover all that is known about each topic. As such the books in the *Solutions* series are not standard reference works. Instead they are intended to provide practical information on the more frequently encountered conditions in an easily accessible form based on real life case studies. They cover that range of cases that fall between the boringly routine and the referral. The books will help practitioners with a particular interest in a topic or those preparing for a specialist qualification. The cases are arranged by presenting sign rather than by the underlying pathology as this is how veterinary surgeons will see them in practice.

Each case also includes descriptions of underlying pathology and details of the nursing required both in the veterinary clinic and at home. It is hoped that the books will also, therefore, be of interest to veterinary students in the later parts of their course and to veterinary nurses.

Continuing professional development (CPD) is mandatory for many veterinarians and a recommended practice for others. The *Solutions* series will provide a CPD resource which can be accessed economically, shared with colleagues and used anywhere. They will also provide busy veterinary practitioners with quick access to authoritative information on the diagnosis and treatment of interesting and challenging cases. The robust cover has been made resistant to some of the more gruesome contaminants found in a veterinary clinic because this is where we hope these books will be used

Joyce Rodenhuis and Mary seager were the inspiration for the series and both the series editor and the individual authors are grateful for their foresight in commissioning the series and their unfailing support and guidance during their production.

GASTROENTEROLOGY

Gastroenterological disorders can be nearly as distressing for an owner as for the animal suffering the disease. Although straightforward cases may recover quickly with simple management changes, the range of GI cases includes patients whose signs may persist for months or even years and the cause of which may be devilishly hard to elucidate. That range also includes diseases which can be terminal and disease which can be fatal remarkably quickly. This book should help you navigate your way through the sometimes bewildering array of tests for gastrointestinal disorders, understand the sometimes complex aetiopathogenesis and earn the significant gratitude of those owners who no longer have to clear up diarrhoea or vomit from their living room carpet.

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Author's note

Gastrointestinal disorders are among the most common presenting complaints in small animal practice. The clinical signs may be due to disorders of the gastrointestinal tract itself such as inflammation, obstruction or neoplasia, or may be secondary to disorders of other organs, such as in cases of hypoadrenocorticism or animals with portosystemic shunts.

Because of the wide variety of potential aetiologies and the complexity of some cases, a problem-orientated approach is helpful and has been used in the cases presented in this book. In problem-orientated medicine, differential diagnoses are developed for each of the clinical problems. This approach aids the clinician in prioritizing diagnostic tests and decreases the likelihood of problems being missed.

The format of this book is primarily based on major presenting signs. Each section of cases is preceded by a short discussion of normal function and pathophysiology of the problem. The areas include swallowing disorders and regurgitation, vomiting, small intestinal diarrhoea, haematochezia and melaena and colonic disorders. Obviously there is overlap among the areas, as patients often present with more than one problem. In these, the cases are under the predominantly presenting complaint. The book does not include every possible type of gastrointestinal case, but attempts to present a range of common and uncommon, simple and complex cases.

For cases, there is also a discussion of the pathophysiology, treatment, outcome and prognosis. Additional information is provided in the appendices.

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SECTION 1

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1 Swallowing and regurgitation

THE OESOPHAGUS AND SWALLOWING

The oesophagus is divided into the cervical, thoracic and abdominal parts.

The cervical oesophagus runs ventral and to the left of the trachea, and thoracic oesophagus runs to the left of the trachea until the tracheal bifurcation, where it is dorsal to it, and then moves to the right of the aortic arch. There is a short abdominal oesophageal segment between the diaphragm and the stomach. The oesophageal muscularis in the dog is striated throughout the oesophagus; whereas in the cat the caudal oesophagus (thoracic and abdominal sections) contains an increasing amount of smooth muscle until the final 2 to 3 cm which is entirely smooth muscle.

The gastro-oesophageal sphincter (GES) is important in maintaining a high pressure zone between the oesophagus and the stomach so that gastric contents are less likely to be refluxed into the oesophagus. The type of food eaten affects the GES pressure. Protein meals increase the pressure, likely due to an increase in gastrin. Fat decreases GES pressure due to cholecystokinin stimulation and also inhibition of gastrin mediated pressure increases.

The GES relaxes transiently to allow for eructation of gas; during this relaxation fluid may also be refluxed into the oesophagus. This is a normal event, but in some dogs it may occur excessively or the rate of clearance of the acid from the oesophagus may be delayed, resulting in oesophagitis.

Swallowing involves three coordinated phases: oropharyngeal, oesophageal and gastro-oesophageal. Pharyngeal contact with food stimulates pharyngeal peristaltic contractions that push the food from the base of the tongue to the laryngopharynx and into the oesophagus. In the oesophagus, the bolus is initially carried by the primary peristaltic wave generated by the pharynx. If the primary peristaltic wave does not result in the bolus reaching the stomach, a secondary peristaltic wave is initiated by local oesophageal distension. The swallowing reflex in the dog requires sensation of the bolus within the oesophagus. Oesophageal peristaltic speed is faster in the dog than in the cat; in dogs, water moves at 80 to 100 cm/second compared to 1 to 2 cm/second in the cat.

DYSPHAGIA

Dysphagia means difficulty in swallowing and may be due to either functional disorders or physical obstruction disorders. The disorders may be oral, pharyngeal or oesophageal.

REGURGITATION

Regurgitation is expulsion of food or saliva from the pharynx or oesophagus and most often is caused by oesophageal disease.

Regurgitation must be differentiated from vomiting, although there are animals that present with both problems. Regurgitation is generally a passive action with no evidence of nausea. Some animals will retch due to the presence of food in the pharynx, which may be confused with an active action.

Regurgitated food is undigested and may be cylindrical in shape from having been formed in the oesophagus. The pH should not be acidic, although as gastric contents are not always acidic this may not be a useful way to distinguish between regurgitation and vomiting. Regurgitated material rarely contains bile, unless there has been gastro-oesophageal reflux prior to regurgitation. Regurgitation may occur immediately after eating or may occur several hours later, especially if the disorder is in the lower cervical or thoracic part of the oesophagus.

Dysphagia may occur in some animals with regurgitation and repeated swallowing attempts may be seen. Aspiration pneumonia is a serious risk in animals that are regurgitating and if present they may present with concurrent cough, respiratory distress or fever. Some animals

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with oesophageal disorders may also show nasal discharge due to ingesta entering through the nasopharyngeal opening.

In addition to aspiration pneumonia, common sequelae to chronic regurgitation are weight loss or poor growth as the animal is unable to assimilate sufficient nutrients.

DIAGNOSIS OF REGURGITATION

While oesophageal disorders may be suspected based on clinical signs, diagnosis usually requires radiological studies and endoscopy. Haematology and serum chemistry parameters are often not affected by regurgitation unless there is dehydration present due to insufficient fluid intake.

Radiography

The normal oesophagus is collapsed except during swallowing, so it not visualized on plain radiographs as it is of a similar density to the adjacent structures in the neck and mediastinum. The canine oesophagus has parallel longitudinal lines in the mucosa (Fig 1.1) whereas the caudal feline oesophagus has more oblique mucosal folds (Fig 1.2).

If air is seen in the oesophagus, it may be due to aerophagia, megaoesophagus, oesophagitis or an oesophageal obstruction. An increased radio-opacity in the oesophagus may be due to obstruction or food retention, or, less likely, oesophagitis.

Positive contrast studies utilizing fluoroscopy may be required to confirm oesophageal dysfunction and to evaluate oesophageal motility. Positive contrast studies are indicated when there is acute gagging, retching or acute or chronic regurgitation, dysphagia or known swallowing of a foreign body.

CLINICAL TIPS: SWALLOWING CONTRAST STUDIES

Ideally the patient should not be fed for 4 to 6 hours prior to the examination. If fluoroscopy is available, it should be performed prior to the administration of the contrast agent to ensure that the patient is in the correct position to clearly see the oesophagus. Plain radiographs are also usually taken at this time. It is recommended to use no sedation if possible, as sedation increases the risk of aspiration pneumonia and may affect oesophageal motility. A small dose of acetylpromazine (0.01 mg/kg slow iv or im) may be useful in some cases and should not affect motility. Liquid barium may be administered slowly orally or mixed with food to assess swallowing of solids. Barium injected into marshmallows has also been suggested to assess intramural oesophageal disease that cannot be detected with a liquid swallow. (This is unlikely to be useful in cats.) Iodine-containing contrast solutions are recommended over barium if a perforation is possible as they cause less tissue reaction.

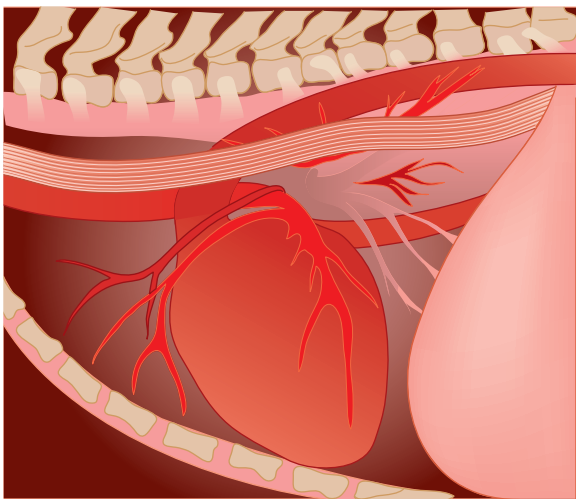


Fig 1.1 Normal canine oesophagus

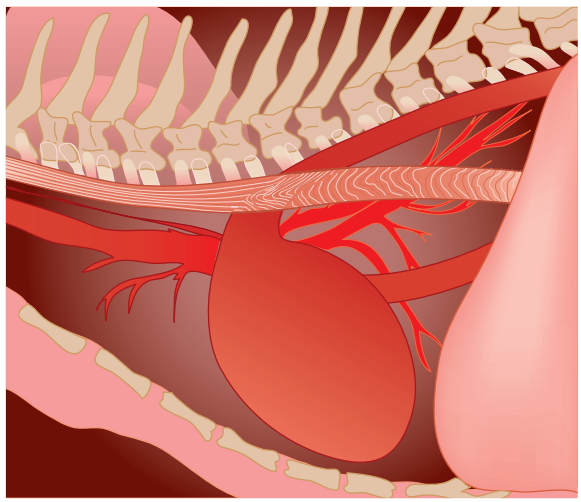


Fig 1.2 Normal feline oesophagus

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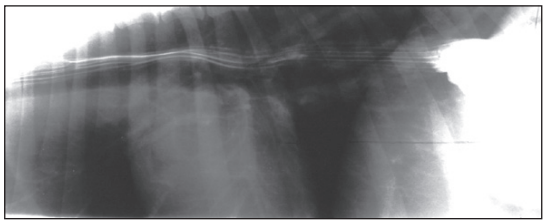


Fig 1.3 Normal oesophagram showing streaking of barium in the oesophagus (courtesy of Dr Tobias Schwarz)

Enlargement of the proximal oesophagus with normal function of the distal oesophagus suggests a vascular ring anomaly or oesophageal stricture. Other causes of megaoesophagus generally result in an enlargement of the entire oesophagus. Once a diagnosis of generalized megaoesophagus is confirmed, diagnostic testing should be performed to establish an underlying aetiology if possible.

A normal dog or cat restrained immediately in lateral recumbency after oral barium may retain some of it in the caudal cervical and cranial thoracic areas of the oesophagus (Fig 1.3), but reverse peristalsis should not occur (i.e. food moving backwards up the oesophagus or from the stomach back into the oesophagus). The first phase of swallowing usually can be seen to move the bolus and barium into the oesophagus, with a second phase sometimes needed to move it all the way into the stomach. Linear streaking of the oesophagus with barium after swallowing is normal.

Endoscopy of the oesophagus can be used to rule out underlying causes of megaoesophagus such as oesophagitis, neoplasia and radiolucent foreign bodies although it can be difficult to assess the size of the oesophagus in an animal under anaesthesia. The normal feline oesophagus contains rings due to transverse oesophageal folds (Fig 1.4).

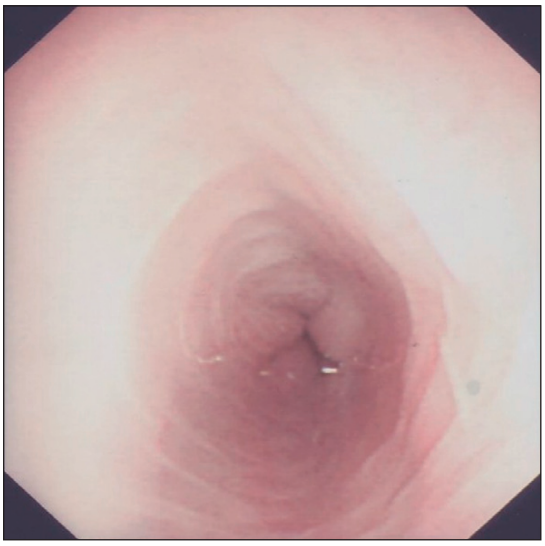


Fig 1.4 Endoscopic view of normal feline oesophagus (courtesy of Prof Danielle Gunn-Moore)

Other tests should be done to look for underlying causes if generalized megaoesophagus is diagnosed. These tests could include: a complete blood count, biochemical panel, urinalysis, blood lead level, creatine kinase concentration, acetylcholine receptor antibody test (evaluation for myasthenia gravis) and evaluation of adrenal and thyroid gland function. Additional diagnostic procedures that may be performed based on the animal's signalment, history and neurological examination, include an EMG study, nerve conduction velocities and muscle biopsies.

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2 Idiopathic megaoesophagus in a dog

INITIAL PRESENTATION

Vomiting and regurgitation every 1 to 3 days for at least 6 months; concurrent weight loss and recent (2 to 3 weeks) cough

Signalment: 2-year-old neutered male German shepherd dog, body weight 23.1 kg

CASE HISTORY

The dog had been obtained from a rescue centre 6 months earlier. The owner said that he was de-wormed regularly with a product from the grocery store and had been vaccinated at the time they obtained him.

He was usually bringing up food that appeared undigested and did not contain bile, although occasionally he also did bring up digested food and bile. There was no abdominal effort and he would bring up the food from minutes to about an hour after eating. It was determined that he was usually regurgitating, although sometimes there was an episode of true vomiting.

He was coughing occasionally during the previous 2 weeks and his exercise tolerance had decreased. The dog's stools had been normal and his water intake and urine output had not changed. The owner estimated that the dog had lost about 8 kg over the last couple of weeks.

His usual diet was a mixture of dry and canned dog food and he was normally fed twice a day. He was not fed table scraps and did not scavenge. His appetite had decreased over the past 2 to 3 weeks, but had been good prior to that time.

His prior treatment included oral cimetidine and metoclopramide, which the owner thought may have slightly helped decrease the frequency of the vomiting or regurgitation.

PHYSICAL EXAMINATION

The dog was bright and responsive. He had a body condition score (BCS) of 2/9. His mucous membranes were pink and his capillary refill time was less than 2 seconds. Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 90 beats per minute (bpm) and a respiratory rate of 36 breaths per minute. There was no evidence of pain on abdominal palpation. Rectal temperature was 38.8° C.

When the dog was observed eating, it was found that he could prehend the food, appeared to form a normal bolus and could initiate swallowing without a problem. This made a pharyngeal disorder less likely to be the cause of the regurgitation.

CLINICAL TIP

Differentiating vomiting from regurgitation can be a challenge and in some cases both are occurring. The time after eating does not always provide a clue, as some animals may vomit immediately after eating and others, especially those with megaoesophagus, may regurgitate hours after eating (Table 2.1).

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting
- Regurgitation
- Weight loss

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Table 2.1 Differentiating regurgitation from vomiting

Clinical sign	Regurgitation/oesophageal disorders	Vomiting
Abdominal effort	None	Present
Time of food ejection after eating	Immediate to delayed, possibly for hours	Delayed, possibly for hours, but may also happen immediately
Appearance of food	Undigested	Partly digested, may have bile present
pH of contents	Should not be acidic	Acid pH implies vomiting, but alkaline material may also be vomited

The dog's primary problem appeared to be regurgitation, although he also appeared to vomit occasionally as well. The weight loss was likely due to inadequate assimilation of food due to the regurgitating.

DIFFERENTIAL DIAGNOSIS

For the regurgitation

- Oesophagitis
- Megaoesophagus, which can be idiopathic or due to an underlying cause
- Oesophageal obstructive disorders, e.g. stricture, neoplasia, foreign body
- Hiatal hernia
- Vascular ring anomalies – unlikely due to the dog's age (usually present in puppies)
- Granulomas due to *Spirocerca lupi:* unlikely as this parasite is not endemic in the UK and he had not travelled out of the UK
- Oesophageal diverticula

For the vomiting

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilation-volvulus
  - neoplasia
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease
  - neoplasia
  - parasites
  - intussusception
- Disorders of the large intestine (unlikely as there were no signs consistent with colonic disorders)
  - colitis
  - obstipation
- Systemic disorders
  - Pancreatopathy
  - Hypoadrenocorticism
  - Diabetes mellitus
  - Liver disorders
  - Peritonitis
  - Renal disease/uraemia (unlikely)
- Dietary causes
  - Adverse reaction to food (allergy or intolerance)
  - Dietary indiscretion

CASE WORK-UP

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed. Haematology results showed a mild normocytic normochromic anaemia with a packed cell volume of 0.334 l/l (reference range 0.37 to 0.55 l/l) and an elevation in mature neutrophils to 18.5×10^9/l (reference range $3.6–12 \times 10^9$/l). Serum chemistry results were unremarkable other than a slightly low albumin of 22 g/l (reference range 26–35 g/l).

Urinalysis was unremarkable. The urine specific gravity was 1.038; chemical strip results were within normal limits and the sediment was not active.

Faecal analysis showed a positive egg count for ascarids, so a course of oral fenbendazole (50 mg/kg po q 24 hours for 3 days) was initiated.

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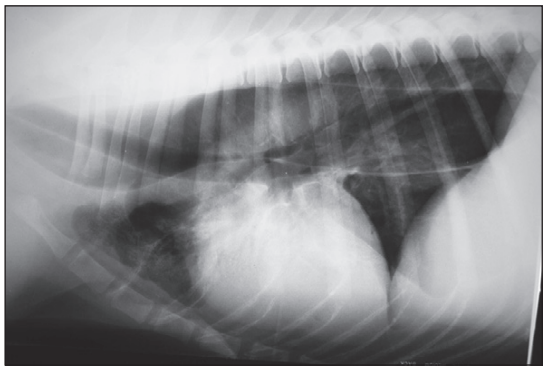


Fig 2.1 Radiograph of thorax showing dilated oesophagus (courtesy of Dr Tobias Schwarz).

Imaging

Thoracic radiography showed the presence of a marked megaoesophagus with ventral deviation of the trachea and a marked tracheal stripe sign (Fig 2.1). There was an increase in pulmonary opacity with an interstitial-alveolar pattern over the cardiac apex and cranial to the heart on the lateral radiograph, which was consistent with aspiration pneumonia.

DIAGNOSIS

Megaoesophagus with aspiration pneumonia. Megaoesophagus can have an underlying cause or may be idiopathic; differential diagnoses for the more common underlying causes include:

- Myasthenia gravis
- Polymyopathy
- Dysautonomia
- Oesophageal obstructive disorders (foreign body, stricture, neoplasia, extra-oesophageal compression)
- Lead toxicity
- Hypoadrenocorticism
- Hypothyroidism (possibly)

Further testing

This dog had no other signs consistent with a polymyopathy or dysautonomia. Further diagnostic tests included serum creatinine kinase and AST concentrations, which were within the reference range and also made a myopathy less likely.

Physical examination, Schirmer tear test and pilocarpine response tests were also not consistent with dysautonomia.

CLINICAL TIP: PILOCARPINE RESPONSE TEST

In an animal suspected to have dysautonomia, if the pupils are affected, one drop of dilute pilocarpine ophthalmic solution (0.05%) should be placed in one eye and the diameter of the pupil observed every 15 minutes for 1 hour. Most normal dogs will not usually respond to this concentration and those that do show only minimal response at 60 minutes. Miosis of the treated eye demonstrates denervation supersensitivity as expected with dysautonomia. However, not all affected dogs will respond to the dilute pilocarpine, which could be a function of the time required for denervation supersensitivity to develop. The pilocarpine test is useful to rule out anticholinergic toxicity, which could cause many of the signs of dysautonomia. An anticholinergic drug would block the response of the pupil to pilocarpine (see Chapter 8).

Blood lead concentration was within the normal reference range at 0.02 μmol/l. Values greater than 1.21 μmol/l indicate probable toxicity.

An adrenocorticotrophin hormone stimulation test showed pre-stimulation serum cortisol of 89 mmol/l and a post-stimulation test of 321 mmol/l; which rules out atypical hypoadrenocorticism (i.e. hypoadrenocorticism where only the corticosteroid concentrations are insufficient and mineralocorticoid concentrations are sufficient to maintain normal serum potassium and sodium concentrations).

The dog's serum acetylcholine receptor antibody titre was 0.02 nmol/l, which is considered normal. A value greater than 0.6 nmol/l is diagnostic of myasthenia gravis, although 15% of affected dogs may not show an increased titer.

The dog's total serum thyroxine concentration was just below the reference range at 12.9 nmol/l (reference range 13–52 nmol/l) and his TSH concentration was 0.13 ng/ml, with a reference range of less than 0.41 ng/ml. This most likely indicated a mild euthyroid sick condition in this dog rather than true hypothyroidism.

Endoscopy

At endoscopy, the oesophagus was found to be very large, flaccid and contained fermenting food and

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fluid within the folds. There was evidence of mild oesophagitis at the distal aspect of the oesophagus. There was also evidence of gastritis and inflammation of the duodenum. Histopathology of the gastric and duodenal mucosa showed mild lymphocytic plasmacytic inflammation.

DISCUSSION

The cause of this dog's vomiting was likely the mild inflammatory condition of his stomach and small intestine. This was treated successfully with metronidazole at 10 mg/kg twice a day for a month and a change to a novel protein, highly digestible diet.

The regurgitation was probably an unrelated condition. Reflux oesophagitis may cause regurgitation and even some dilation of the oesophagus, but not to the degree of dilation present in this dog. Underlying conditions were ruled out as far as possible, leaving the diagnosis as a probable idiopathic megaoesophagus.

Treatment for idiopathic megaoesophagus

Medical

Treatment for idiopathic megaoesophagus is supportive and includes providing food and water from elevated containers to allow the aid of gravity for swallowing (Fig 2.2) and feeding a high calorie food. Some dogs cope better with liquid or soft food and some do better with 'meatballs' of dog food, so the diet has to be tailored for the individual.



Fig 2.2 Example of feeding a dog from a height.

Many promotility drugs have been tried unsuccessfully in an attempt to improve oesophageal motility in dogs with idiopathic megaoesophagus. Drugs such as anticholinergics that decrease the gastro-oesophageal sphincter tone could theoretically be beneficial; however, these have not shown any benefit and could be detrimental in dogs with inhalation pneumonia or reflux oesophagitis. This dog appeared to have both those conditions.

Surgical

Myotomy has been attempted for megaoesophagus, similar to the surgical approach for achalasia; however, dogs which had surgery had a worse outcome than those without surgery, so this is not recommended.

Epidemiology

Oesophageal dilation is a common cause of regurgitation and congenital idiopathic megaoesophagus is the most common form in the dog. Idiopathic megaoesophagus is inherited in the wirehaired fox terrier by simple autosomal recessive gene and in miniature schnauzers by simple autosomal dominant of a 60% penetrance autosomal recessive pattern. A breed predisposition exists in the German shepherd dog, Great Dane, Irish Setter and Chinese Shar Pei and it likely has a hereditable basis in these dogs also.

Pathophysiology

Idiopathic megaoesophagus is likely due to a lesion in the afferent limb of the swallowing reflex resulting in a loss of peristaltic function in the oesophageal body. Sensory stimulus from the presence of food or liquid in the oesophagus is necessary to stimulate the oesophageal phase of swallowing and it is this sensory reinforcement of the swallowing reflex that appears to be affected.

Prognosis

The prognosis for patients with idiopathic megaoesophagus is guarded, although some young patients recover with time. Once chronic dilation is present there may be irreversible damage to the oesophagus. Many of the dogs will eventually die from aspiration pneumonia or undergo euthanasia due to debilitation from chronic malnutrition.

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3 Myasthenia gravis in a dog

INITIAL PRESENTATION

Regurgitation, cough and weakness/exercise intolerance
Signalment: 2-year-old neutered male collie crossbred dog, body weight 19.8 kg

CASE HISTORY

The dog had become progressively less exercise tolerant over the 2 months prior to presentation. He had also initially begun to regurgitate his food and then began to regurgitate water. The regurgitation usually occurred within a few minutes after eating, but occasionally he brought up foam without having eaten anything. The food that he regurgitated was formed and undigested. He was still interested in eating and the owners had started feeding him very small amounts, as any larger amounts were regurgitated.

He had become progressively less able to go for walks and would sit or lie down after several minutes of walking. He remained conscious during these times and he would usually be willing to walk again after a short rest. He had begun to cough during the previous week and his breathing was laboured. His faeces were formed and were passed normally and his urination was normal.

The dog had been in the owners' possession about a year and was current on vaccinations and de-worming.

PHYSICAL EXAMINATION

The dog was quiet but responsive and preferred to lie on the consulting room floor during the examination. He was reluctant to walk when taken outside and his gait was abnormal (Fig 3.1). His body condition score was 4/9. Mucus membranes were pink and capillary refill time was less than 2 seconds. Pulse quality was good.

Thoracic auscultation revealed normal heart sounds but crackles over the left lung fields. Heart rate was 140 beats per minute (bpm) and the respiratory rate was 60 breaths per minute with some inspiratory effort.

There were no abnormalities detected and no evidence of pain on abdominal palpation. His rectal temperature was 39.1° C.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Regurgitation
- Weakness and exercise intolerance
- Cough

Regurgitation in this dog may have been due to:

- Megaoesophagus
- Oesophageal stricture
- Oesophagitis
- Oesophageal dysmotility
- Hiatal hernia
- An oesophageal foreign body was less likely because of the duration of the problem.

Weakness and exercise intolerance can by caused by many systemic disorders, including (but not limited to) cardiopulmonary diseases, metabolic disorders such as hypothyroidism or hypoadrenocorticism, neurological disorders such as polyneuropathies or myasthenia gravis, myopathies or pain due to other disorders.

Coughing can be due to upper respiratory disorders, lower respiratory disorders or cardiac disorders. As this

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Fig 3.1 Dog with myasthenia gravis showing weakness and abnormal stance

dog had crackles on auscultation, a lower respiratory (lung) disorder was suspected. Crackles are discontinuous, non-musical sounds produced when airway pressures fluctuate or when air rushes through fluid- or mucus-filled alveoli, but can sometimes also be heard when there is fluid in the oesophagus.

CASE WORK-UP

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed. The haematology results showed an increase in neutrophils with a slight left shift, consistent with the presence of inflammation. The serum chemistry results were unremarkable, as was the urinalysis. Basal serum cortisol was 132 nmol/l (reference range 20–230 nmol/l).

CLINICAL TIP ON USE OF BASAL SERUM CORTISOL

A serum basal cortisol concentration of 70 nmol/l or greater effectively rules out hypoadrenocorticism. If the value is less than 70 nmol/l, an ACTH stimulation test should be performed to definitively rule out or diagnose this disorder.

Imaging

CLINICAL TIP ON IMAGING IN REGURGITATING CASES

Plain thoracic radiographs help rule out megaoesophagus and screen for cardiopulmonary disorders. A positive contrast study using barium mixed with dog food using fluoroscopy helps determine if there are oesophageal motility disorders or a hiatal hernia, but in cases like this dog, which have a cough, the risk of furthering an aspiration pneumonia is present.

Thoracic radiographs showed the presence of a large oesophagus and a bronchial alveolar pattern especially affecting the left cranioventral lung lobes, consistent with aspiration pneumonia (Figs 3.2 and 3.3).

Neurological examination

A neurological examination was largely unremarkable, although after walking for a couple of minutes the dog developed a stiff, choppy and hunched gait. He then lay down and developed tremors of the hind legs and was unable to rise for a short period. Once given time to recover, the dog could walk and then this pattern would reoccur. As this is typical of dogs with myasthenia gravis, an edrophonium response test was performed.

The response to this test was dramatic, in that the dog immediately stood up and was willing to trot down the corridor. This response is consistent with a presumptive diagnosis of myasthenia gravis.

CLINICAL TIP ON TENSILON TEST

The edrophonium chloride (Tensilon) response test is used to diagnose myasthenia gravis. The dog or cat is exercised (if necessary) until they sit or lie down or the gait becomes abnormal. A dose of 0.1 to 0.2 mg/kg of edrophonium chloride is administered intravenously. A positive test results in temporary improvement of locomotor signs within minutes. Oesophageal musculature does not improve with edrophonium. There are false positive and false negatives with this test, as well as equivocal results, so it should be used as an adjunct test and not for definitive diagnosis.

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The primary criterion for diagnosis of myasthenia gravis is the presence of serum antibodies that react with alpha-bungarotoxin extracted ACh receptors; this test is highly specific, although about 15% of suspected myasthenics are seronegative (see Appendix 6).

NURSING ASPECTS

As with other cases of megaoesophagus, this dog was fed frequent small meals from a height. Different patients do better on different types of food and this dog regurgitated less when fed meatballs of canned food. Water was also offered from a height.

For the aspiration pneumonia, in addition to antibiotic treatment (below), nebulization and coupage were carried out four times a day to help relieve pulmonary congestion.

Medical treatment

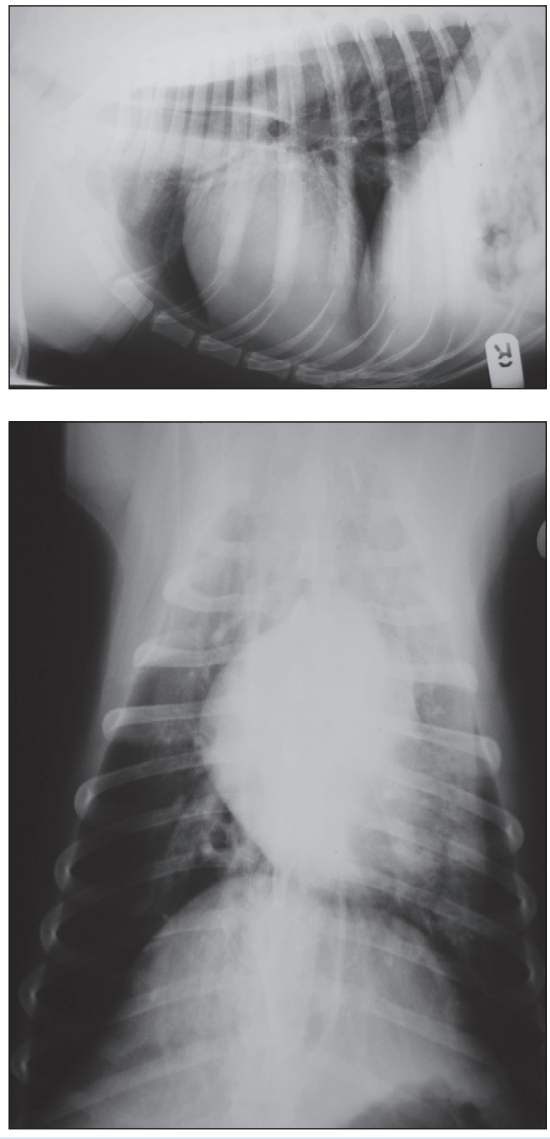
Pyridostigmine at 15 mg po q 8 hours was started. Some dogs with severe megaoesophagus are unable to take oral medications and treatment needs to initially be with injectable neostigmine (0.04 mg/kg im q 6 hours); however, this dog managed with the medication hidden in his dogfood meatballs.

Treatment for the aspiration pneumonia included antibiotic therapy with marbofloxacin (2 mg/kg po q 24 hours), clavulanate potentiated amoxicillin (15 mg/kg po q 12 hours) and clindamycin (7.5 mg/kg po q 12 hours). Antibiotic cover for aspiration pneumonia usually involves the use of multiple antibiotics to provide 'four quadrant' bacterial cover (i.e. Gram-negative and Gram-positive, aerobic and anaerobic bacteria).

Follow-up

A serum acetylcholine (ACh) receptor antibody test was submitted and these results came back with a high value of 2.8 nmol/l, confirming the diagnosis of myasthenia gravis. A value of greater than 0.6 nmol/l is consistent with myasthenia gravis.

The dog was seen 2 weeks later, when his gait was nearly normal and the cough was no longer present. He still occasionally had regurgitating episodes, but the frequency was decreased.



Figs 3.2 and 3.3 Lateral and ventrodorsal thoracic radiographs showing enlarged oesophagus (courtesy of Dr Tobias Schwarz)

DISCUSSION AND EPIDEMIOLOGY

Acquired canine myasthenia gravis is an autoimmune disease in which autoantibodies are directed against muscle postsynaptic nicotinic acetylcholine receptors (Fig 3.4). In some cases there is a concurrent thymoma which is the stimulus for the antibody production; this is more common in cats than in dogs.

Acquired myasthenia gravis is probably the most common neuromuscular disorder diagnosed in the dog.

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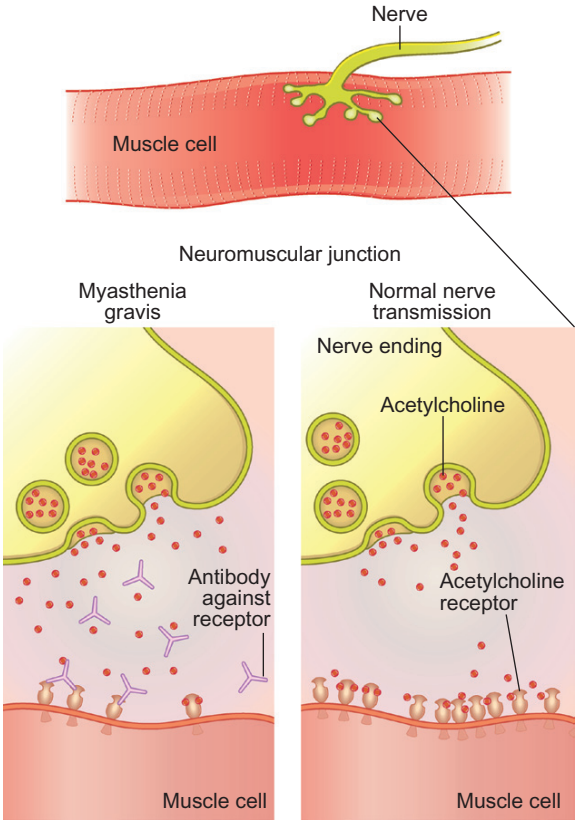


Fig 3.4 Anti-cholinesterase receptor and effects of antibodies against the receptor



Fig 3.5 Dog with myasthenia gravis after treatment showing improvement in strength

Studies have identified a high relative risk for acquired myasthenia gravis in the Akita, Scottish terrier, German shorthaired pointer and Chihuahua breeds of dogs and in Abyssinian and Somali cats. Familial myasthenia gravis has also been identified in Newfoundlands and Great Danes.

The typical signs include muscle weakness which can be generalized as in this case, focal, for example affecting only the oesophagus or the facial, pharyngeal or laryngeal areas, or can be acute, fulminant, and generalized which results in a very rapid onset of tetraparesis and dyspnoea. About 90% of the dogs with the generalized form have megaoesophagus. Acquired canine myasthenia gravis has a high mortality rate (approximately 50%) and according to one specialist in the field, this is due in part to a misdiagnosis of vomiting when actually the dog is regurgitating due to oesophageal dilatation. This delayed recognition of regurgitation usually involves inappropriate therapy with an increased risk of developing an aspiration pneumonia which in itself may be life threatening.

Treatment with anti-cholinesterase drugs like pyridostigmine prolongs the interaction of ACh with the receptors. The dose should begin low and be increased to effect. Oesophageal motility is minimally affected by this treatment. Some studies recommend using prednisolone, although this should not be initiated concurrently with anti-cholinesterase medications as it can exacerbate the weakness and should not be used in the presence of pneumonia. Low-dose alternate day oral prednisone therapy (0.5 mg/kg every other day) is suggested when anti-cholinesterase drugs are not effective.

Myasthenic female dogs and cats should be spayed as soon as possible after the signs of myasthenia gravis are under control as heat cycles and pregnancy can exacerbate the disorder. Vaccinations may exacerbate the clinical signs. It is generally best to avoid the use of drugs that may affect neuromuscular transmission such as ampicillin, aminoglycosides, anti-arrhythmic agents, phenothiazines, anaesthetics, narcotics and muscle relaxants. Also, organophosphate dips may result in a cholinergic crisis since they could be additive with pyridostigmine.

Prognosis

Many patients with myasthenia gravis have spontaneous remissions within a year of being diagnosed, although overall the prognosis is guarded as about 50% die or undergo euthanasia. Those that die usually have succumbed to aspiration pneumonia. In this case the initial response was good and the early prognosis was cautiously optimistic (Fig 3.5).

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4 Oesophageal stricture in a cat

INITIAL PRESENTATION

Regurgitation

Signalment: 9-year-old male neutered Siamese cat, body weight 3.5 kg

CASE HISTORY

The cat had been healthy until 3 weeks prior to admission, when he had been anaesthetized for a routine dental cleaning. Since then he had been regurgitating food and froth, but not bile. The regurgitated food appeared undigested. He was also retching.

He was de-wormed regularly and vaccinated yearly. He had arthritis and he was treated with meloxicam for this condition. He was an indoor and outdoor cat, but had no known exposure to toxins. He was fed canned cat food and chicken, but had a poor appetite during the 3 weeks prior to presentation. He was able to prehend and swallow his food and could drink water, although he may have been drinking less than usual.

His stools were normal in appearance and he was able to urinate and defecate normally. He was on no other medications or supplements.

PHYSICAL EXAMINATION

The cat was bright and responsive. His body condition score was 4/9 and he was estimated to be about 5% dehydrated. Mucus membrane colour was pink and capillary refill time was less than 2 seconds.

Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 190 beats per minute (bpm) and a respiratory rate of 30 breaths per minute. There was no evidence of abnormalities or pain on abdominal palpation. Rectal temperature was 38.0° C.

Problem list and discussion of problems

- Regurgitation

Regurgitation usually indicates an oesophageal disorder.

DIFFERENTIAL DIAGNOSIS

Oesophagitis

- Obstructive oesophageal disorders
  - foreign body
  - stricture
  - neoplasia
  - peri-oesophageal masses

Oesophageal neuromuscular disorders

- Megaoesophagus, including systemic disorders affecting swallowing, such as myasthenia gravis and dysautonomia
- Oesophageal motility abnormalities

CASE WORK-UP

The cat was admitted to the hospital for investigative procedures and rehydration with crystalloid fluids administered intravenously.

MINIMUM DATA BASE

Haematology and serum chemistry were performed and results were all within the reference ranges.

Imaging

Plain radiographs of the cat's thorax were unremarkable. While a barium swallowing study could have been performed to further evaluate the oesophageal function, this test carries the risk of aspiration of the barium and in this case it was elected to perform endoscopy rather than the swallowing study.

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CLINICAL TIP

While thoracic radiographs are useful to evaluate the lungs and many other oesophageal disorders, strictures may not be visible on a plain radiograph and even liquid barium may pass through a stricture rapidly enough that it is not detected. Barium mixed with food may allow visualization of a narrowed area of the oesophagus at the point of the stricture, but may or may not show multiple areas of strictures if the food is regurgitated when it reaches the first area of stricture. Barium swallow studies, whether liquid or liquid mixed with food, do carry a risk of aspiration pneumonia, especially if the patient must be put in lateral recumbency to obtain the radiograph.

Endoscopy

A severe concentric stricture extending throughout most of the thoracic oesophagus was found (Fig 4.1). The width of the narrowed oesophagus was about 3 to 4 mm. Gentle dilation with a balloon catheter was performed, stretching the affected part of the oesophagus to about 1 cm. After dilation with the balloon, a couple of tears were noted in the mucosa and a severe oesophagitis was noted to be present (Fig 4.2). A gastric ulcer was also found in the cardia of the stomach.

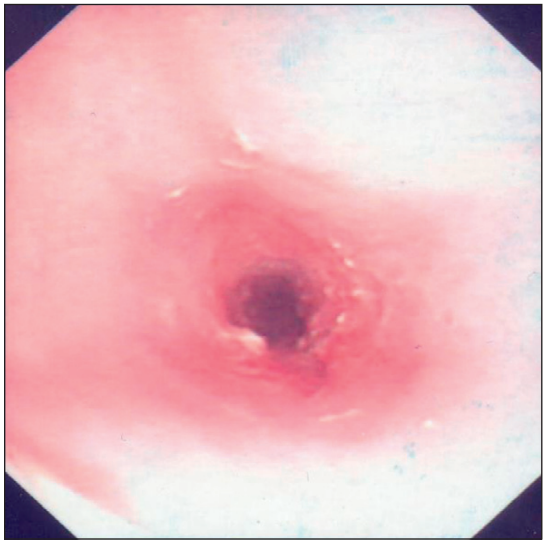


Fig 4.1 Endoscopic view of oesophageal stricture

NURSING TIPS

The main areas of nursing support for this case were analgesia and nutrition. Observation of difficulty or pain while swallowing would indicate that more attention be paid to analgesia. Generally nutritional support with liquid foods is initially indicated after balloon dilation or in other cases of severe oesophagitis. If the patient becomes able to eat food with a more solid or semi-solid consistency, they should be fed these foods. The presence of food in the oesophagus may decrease the risk of re-stricturing. If the animal is unable to take any nutrition orally a gastrostomy tube may need to be placed; however, fasting or 'oesophageal rest' is not indicated otherwise. There are already saliva and other secretions in the oesophagus so it does not 'rest'.

Follow-up

Probably due to the extent and severity of the oesophageal stricture in this cat, the strictures reformed and his oesophagus was repeatedly balloon dilated on about a weekly basis 11 more times (Fig 4.3). After the 12th balloon dilation, he was able to eat soft foods

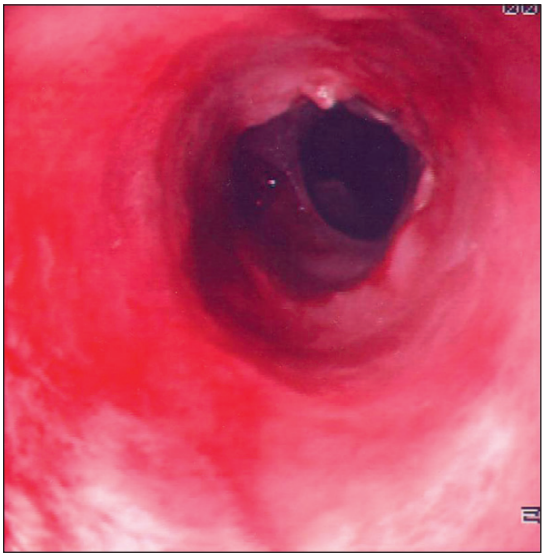


Fig 4.2 Endoscopic view of oesophageal stricture after dilation, showing mucosal tearing and oesophagitis

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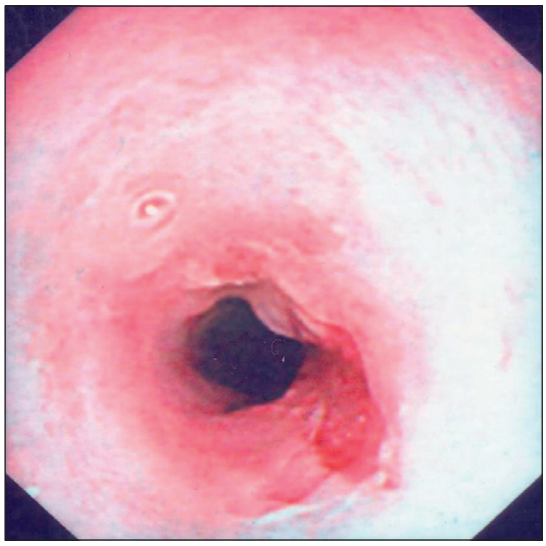


Fig 4.3 Endoscopic view of re-stricturing of oesophagus after dilation

successfully and has done well, although continues to need to eat soft food.

Medical therapy

Post-dilation treatment in this cat was mostly for the oesophagitis and the gastric ulcer.

The goals of therapy for oesophageal strictures were to decrease any ongoing reflux as the damage to the oesophageal mucosa can decrease the tone of the lower oesophageal sphincter, worsening the condition, to resolve the inflammation and to decrease the risk of repeat stricture formation.

Gastric acid inhibitors such as ranitidine were used as they decrease the output of gastric acid and pepsin. Human patients with oesophagitis report a decrease in symptoms when administered histamine H_2 receptor antagonists. These medications are adequate for mild to moderate oesophagitis, but may not be sufficiently effective for severe oesophagitis. Proton pump inhibitors such as omeprazole produce greater and more long lasting suppression of gastric acid than do the H_2 antagonists and are frequently used for human oesophagitis patients. Abrupt discontinuation of omeprazole is more likely to be associated with a relapse of the condition in humans; while this has not been studied in dogs or cats, a cautious approach to discontinuation of the drug may be indicated.

Sucralfate has a mucosal protective effect on the oesophageal mucosa. While it has been stated that it is only effective in an acid environment (i.e. during active reflux), others have indicated that it may be effective in a neutral pH environment as well as an acidic one.

Injection of corticosteroids into the dilated area via the endoscope is also recommended; however, it was not done in this cat as the necessary instrumentation was not available. Some clinicians use systemic corticosteroid administration after balloon dilation, although there is no evidence that this is effective.

Some clinicians also use antibiotic therapy in cases of oesophagitis; however, there are also no studies showing any added benefit for them. If aspiration pneumonia is present, appropriate antibiotics should be used and possibly if systemic corticosteroids are used prophylactic antibiotics should also be used.

Colchicine (0.03 mg/kg po q 48 hour) was used in this cat to attempt to decrease fibrosis and re-stricturing. The usefulness of this drug in these cases has not been studied. Colchicine inhibits collagen synthesis. Potential side effects of colchicine include vomiting, diarrhoea and abdominal pain.

Buprenorphine (0.01 mg/kg iv q 8 hour) was used for post-procedural analgesia in this cat. Initially liquid foods were fed and when these were tolerated, feeding with very soft food was started.

Surgical therapy

Surgical techniques for the treatment of fibrous oesophageal strictures have been described, but the outcome is likely to be less successful than balloon dilation in cases without neoplasia. While the prognosis remains guarded for animals with oesophageal strictures, many animals do well after balloon dilation; in one study 88% of patients had a successful outcome.

CASE DISCUSSION

Balloon dilation is the current treatment of choice for oesophageal strictures. Although many animals require repeated dilation before the oesophagus remains large enough for them to eat without regurgitating, repeating the procedure 12 times is exceptional (and also required a very dedicated owner and veterinary surgeon). Most veterinary patients require three dilations for adequate alleviations of an oesophageal stricture.

Pathogenesis

Severe oesophagitis and oesophageal strictures after anaesthesia are generally due to gastro-oesophageal

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reflux. Factors affecting the risk of oesophagitis include the tone of the lower oesophageal sphincter, the volume of gastric contents refluxed, the content of the refluxed material, how long it is present within the oesophagus and the healing ability of the oesophagus.

Some premedications such as atropine and xylazine used prior to general anaesthesia are associated with increased risk of gastro-oesophageal reflux. A pre-anaesthetic fast longer than 24 hours is also associated with an increased frequency of reflux, likely due to the more acidic nature of the contents. Alkaline reflux with a pH greater the 7.5 can also cause oesophagitis; however, it is thought to usually be less severe than that caused by acidic reflux.

When oesophageal injury due to reflux involves the deeper layers of the oesophagus, scarring and stricture are more likely. Strictures are most likely with circumferential injury and inflammation or that affecting opposing sides of the oesophagus. Inflammation stimulates lymphocytes, fibroblast and macrophage activity and formation of collagen. The initial response occurs immediately and collagen formation over several days. The signs of a stricture may be present within days to a couple of weeks after the injury.

Prognosis

Since the development of balloon dilation, the prognosis for patients with oesophageal strictures has improved. In one study a successful outcome occurred in 88% of patients treated with balloon dilation, with most animals able to eat canned, mashed or dry food without regurgitation.

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5 Phenobarbitone responsive retching in a dog

INITIAL PRESENTATION

Gagging, retching and vomiting for 10 days

Signalment: 6-year-old neutered male Jack Russell terrier x poodle, body weight 6.6 kg

CASE HISTORY

This dog had been referred for a suspected upper respiratory disorder or oesophageal foreign body based on his clinical signs and diet. He had been keen to eat, although according to the owner it appeared painful for him to eat and his food intake was decreasing. He was able to prehend and swallow food, but vomited 5 to 30 minutes after eating. Active abdominal contractions were present prior to and during the vomiting; the vomitus contained bile, digested and undigested food. The frequency of the episodes was increasing to several times per day.

His faeces were normal and he was defecating, drinking and urinating normally. He was fed dry food mixed with some canned food, plus table scraps including pork bones and chews. His vaccinations and de-worming programme were up to date.

He had lost weight and was becoming tired more quickly on his walks.

PHYSICAL EXAMINATION

The dog was dull but responsive and his body condition score was 4/9. Mucus membranes were pink but tacky and he was estimated to be about 6% dehydrated. His capillary refill time was less than 2 seconds. Oral examination revealed hypersalivation, halitosis and mild gingival tartar.

He was trembling and palpation of the ventral neck resulted in a marked pain reaction, with spasms of the neck, bruxism and vocalization. The submandibular salivary glands were mildly enlarged and very firm.

Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 80 beats per minute (bpm) and a respiratory rate of 24 breaths per minute. Rectal temperature was 36.7° C.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting with severe gagging and retching

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for the vomiting with gagging and retching may include some of the following categories of disorders.

- Oral/pharyngeal/oesophageal disorders are included in this case as the vomiting was accompanied by signs possibly relating to the upper alimentary tract. In some cases, patients with these disorders will also vomit.
  - salivary gland necrosis
  - oesophagitis
  - foreign body within the pharynx, oesophagus or trachea
  - gastro-oesophageal reflux
- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilation-volvulus
  - neoplasia
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease
  - neoplasia
  - intussusception (unlikely due to age and signs)
- Dietary causes
  - dietary sensitivity
  - dietary indiscretion

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CASE WORK-UP

The dog was admitted to the hospital and administered intravenous crystalloid fluids to correct the dehydration.

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed.

Haematology results showed a mature neutrophilia with a neutrophil count of 14.89×10^9/l (reference range 3.6–12.0 $\times 10^9$/l) and a monocytosis (2.1×10^9/l; reference range 0–1.5 $\times 10^9$/l), consistent with inflammation or infection.

Serum chemistry revealed a hyperalbuminaemia of 40.8 g/l (reference range 26–35 g/l), consistent with dehydration. Hypokalaemia with a potassium concentration of 3.1 mmol/l (reference range 3.6–5.6 mmol/l) was also noted and corrected with the addition of potassium chloride to the intravenous fluids.

Urinalysis showed a high specific gravity of 1.041, again consistent with dehydration and showing good renal concentrating ability; the rest of the urinalysis was unremarkable.

Imaging and histopathology of the biopsy

Thoracic and abdominal radiography showed no abnormalities. Abdominal ultrasound showed no abnormalities.

Upper gastrointestinal endoscopy revealed erosions and erythema of the distal oesophagus near the lower oesophageal sphincter (Fig 5.1). No foreign bodes or masses were observed. The gastric and duodenal mucosa appeared normal and histopathology of pinch biopsies taken from these areas were reported as normal tissue.

Surgical biopsies were taken of one of the enlarged salivary glands. Histopathology of this gland showed some evidence of necrosis.



Fig 5.1 Endoscopic view of distal oesophagus showing oesophagitis

DIAGNOSIS

The results for this case rule out many causes of gagging and vomiting and are most consistent with salivary gland necrosis, also termed phenobarbitone responsive hypersialism. In some cases the histopathology of the salivary gland will be normal. This dog also had evidence of oesophagitis.

Pathophysiology and epidemiology

This disorder is poorly understood but is thought to be a form of limbic epilepsy. While it has been reported in a wide variety of dogs, young- to middle-aged Jack Russell terriers and wirehaired fox terriers appear to be predisposed.

Medical treatment

Treatment with phenobarbitone at 2 mg/kg twice a day orally was initiated. The dog responded within 48 hours with a resolution of the bruxism, oesophageal spasms, hypersalivation and vomiting.

Treatment for the concurrent oesophagitis was also started using ranitidine (2 mg/kg po q 12 hours), sucralfate (1 ml po q 8 hours) and buprenorphine (20 μg/kg iv q 6 hours).

Follow-up

The dog was sent back to the referring veterinary surgery for assessment of clinical improvement and serum phenobarbitone concentration assay, which was 23 μg/ml. While this is on the low end of the therapeutic range for idiopathic epilepsy, it was adequate for controlling the signs in this dog. A commonly used reference range for serum phenobarbitone concentrations is 15–45 μg/ml; however, some neurologists feel that values below 20 μg/ml may not control clinical signs and with values over 40 μg/ml (or even over 35 μg/ml) there is a risk of hepatotoxicity. Because of this risk, monitoring of liver parameters was also recommended, as for any dog on phenobarbitone.

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CLINICAL TIP ON SERUM PHENOBARBITONE CONCENTRATIONS

The most useful single sample is the trough phenobarbitone concentration, i.e. immediately before a dose is due. If it is not practical to collect a sample at this time then try to take subsequent samples at the same time in the dosing cycle. Some authors advocate the use of peak phenobarbitone levels (2–4 hours post-pill) as well as trough levels (prior to dosing) to check the maximal serum concentration. A study has suggested that in most cases this is not necessary.

Prognosis

The prognosis in cases which respond to therapy is excellent. In most reported cases it has been possible to slowly wean the dogs off the phenobarbitone starting from 3 months post-diagnosis with cessation of medication within 6 months from diagnosis.

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6 Oesophageal foreign body in a dog

INITIAL PRESENTATION

Dysphagia, regurgitation

Signalment: 8-month-old entire male Labrador retriever, body weight 20 kg

CASE HISTORY

The dog had been regurgitating for about 2 days. He was still interested in eating, but appeared to be uncomfortable when trying to swallow. After swallowing, undigested food was frequently regurgitated, usually within 5 minutes. He was able to swallow water, although occasionally appeared to also regurgitate water. His faeces had been normal, although he had not passed any faeces for 1 day before admission.

Prior to this he had been a healthy puppy, had received his initial course of vaccinations and had been de-wormed twice with fenbendazole. His regular diet was a premium quality puppy food and he was given treats of human food. The owners were suspicious that the dog could have eaten a bone that he scavenged from the rubbish.

PHYSICAL EXAMINATION

At presentation, the dog was quiet but alert. He had a body condition score of 5/9 and was estimated to be about 6% dehydrated. His mucous membranes were pink and slightly tacky; his capillary refill time was about 3 seconds. Halitosis was noted during the oral examination and there was evidence of dried saliva around his mouth. A gag reflex was present, the dog could move his tongue normally and the oral cavity appeared normal.

Thoracic auscultation and abdominal palpation revealed no abnormalities and his lymph nodes were of normal size. His rectal temperature was 38° C, respiratory rate 40 breaths per minute and heart rate was 80 beats per minute.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The dog's problems were regurgitation and halitosis. The discomfort in trying to swallow appeared to be due to oesophageal dysphagia and was thought to be related to the cause of the regurgitation. There was also evidence of drooling, with the dried saliva present around his mouth. The dehydration was thought to be secondary to his swallowing problems.

DIFFERENTIAL DIAGNOSIS

Causes of regurgitation include:

- Pharyngeal disorders
  - pharyngeal obstructive disorders, such as foreign bodies, tonsillar neoplasia or retropharyngeal lymphadenopathy
  - pharyngeal neuromuscular disorders, such as myasthenia gravis, cranial nerve (IX, X) neuropathies, brain stem or cerebellar disorders, cricopharyngeal achalasia, botulism, rabies (although rabies is unlikely in the UK)
- Oesophageal disorders
  - oesophagitis
  - oesophageal obstructive disorders, such as foreign bodies, stricture, neoplasia, vascular ring anomalies, perioesophageal masses

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- oesophageal neuromuscular disorders, such as megaoesophagus or motility disorders
- oesophageal diverticula
- hiatal hernia

Differential diagnoses for halitosis include:

- Diet related cases, such as food remaining in the mouth, pharynx or oesophagus, and coprophagia
- Cheilitis
- Oral cavity or pharyngeal disorders such as foreign bodies or inflammatory lesions
- Nasal cavity or sinus disorders with inflammation or necrosis
- Dental disease
- Oesophageal diseases with food remaining in the oesophagus
- Malassimilation
- Systemic disorders such as uraemia or liver disease

Drooling can be due to excessive saliva production or failure to adequately swallow saliva. Some dogs also drool in anticipation of feeding and some cats while purring. With the concurrent history of regurgitation, the drooling in this dog was thought to be due to a swallowing problem or oesophageal discomfort.

CASE WORK-UP

Initial treatment included administration of intravenous crystalloid fluid therapy to correct the estimated 6% dehydration.

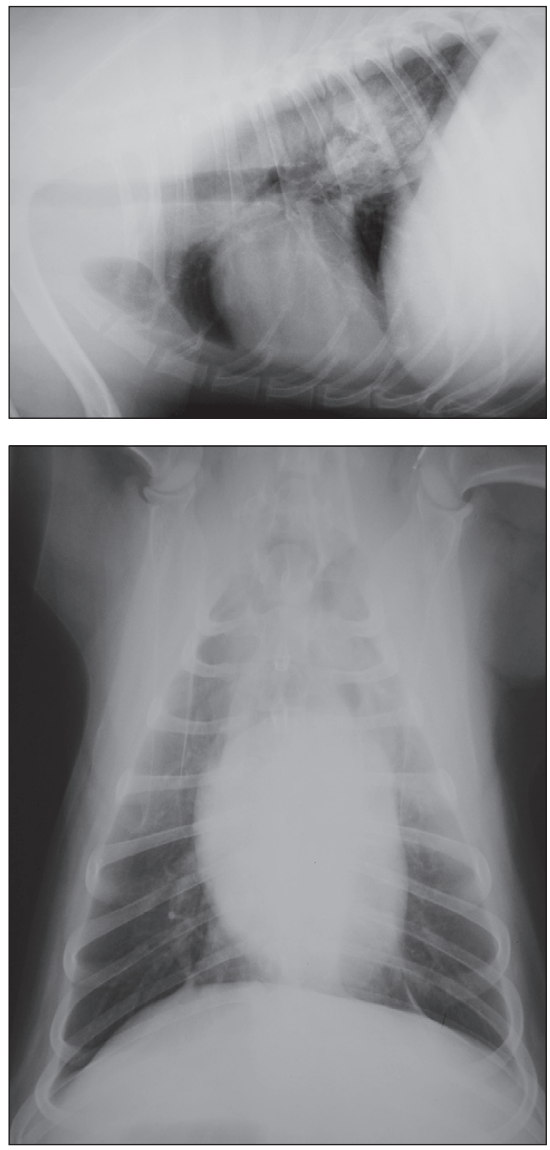
MINIMUM DATA BASE

Haematology showed a packed cell volume of 0.516 l/l (reference range 0.39–0.55 l/l). While this is within the reference range, it is higher than expected for a Labrador retriever and the increase was likely due to dehydration. Serum chemistry results, including electrolyte values, were within the reference ranges except for urea which was just above the upper end of the reference range at 8.1 mmol/l (reference range 1.7–7.4 mmol/l). The serum creatinine was within the reference range at 111 μmol/l (reference range 40–132 μmol/l). Elevations in urea can be due to haemoconcentration from dehydration, but can also occur due to renal causes or from bleeding into or from the gastrointestinal tract.

The urine specific gravity of this dog was 1.047, indicating good urine concentrating ability and consistent with dehydration, and as the serum creatinine was not elevated, renal or post-renal causes of the elevated urea were unlikely.

Imaging

Thoracic and abdominal radiographs showed a foreign body of mineral density in the caudal oesophagus at the level of the diaphragmatic hiatus (Figs 6.1 and 6.2) and



Figs 6.1 and 6.2 Lateral and ventrodorsal thoracic radiographs showing radiopaque foreign body in distal oesophagus (courtesy of Dr Tobias Schwarz)

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Fig 6.3 Endoscopic view of bone stuck in the oesophagus



Fig 6.4 Retrieval forceps used to remove oesophageal foreign bodies

another foreign body of mineral bone density inside the stomach. There was no evidence of aspiration pneumonia and no evidence of mediastinitis or pneumomediastinum, so the foreign body did not appear to have penetrated the oesophagus.

DIAGNOSIS AND TREATMENT

Endoscopy

After re-hydration the dog was anaesthetized and oesophagoscopy was performed. A bone foreign body was visualized (Fig. 6.3).The foreign body was removed using retrieval forceps (Fig 6.4). The oesophageal wall

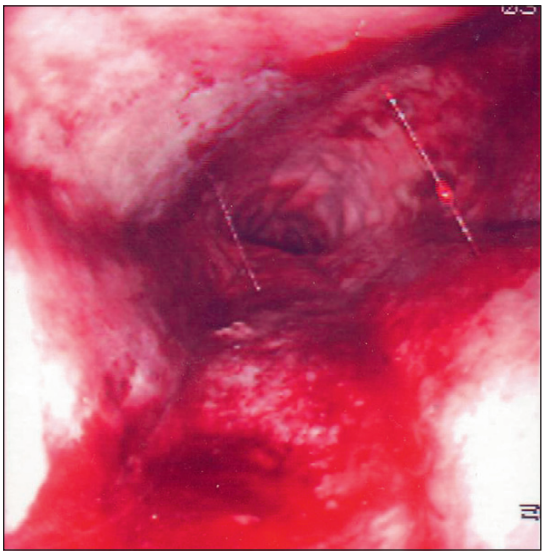


Fig 6.5 Endoscopic view of the oesophagus after removal of the bone, showing trauma and tearing of oesophageal mucosa (courtesy of Alison Ridyard)

showed ulcerations and mucosal tearing from the presence of the bone (Fig 6.5).

Medical management

After removal of the bone, the dog was treated with sucralfate suspension (4 ml po q 8 hours), ranitidine (2 mg/kg po q 12 hours) for its antacid and promotility effects and clavulanate-potentiated amoxicillin (15 mg/kg po q 12 hours). He was continued on intravenous fluids at a maintenance rate. At 24 hours post-procedure the dog was offered water and then later a liquid food, which he was able to swallow without discomfort or regurgitation.

Outcome

The dog was discharged 48 hours after removal of the bone, with instructions to continue the medications and feed soft food for a week. At a re-visit 2 weeks later, the dog was eating well and was able to swallow comfortably with no regurgitation.

DISCUSSION AND EPIDEMIOLOGY

Oesophageal foreign bodies are a frequent clinical problem in dogs and cats. They are more common in dogs than in cats, possibly because of the more discriminating eating habits of cats. Small breeds of dogs, especially West Highland white terriers and Yorkshire terriers

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are predisposed, although Bernese mountain dogs were over-represented in one study.

The most common oesophageal foreign bodies found in dogs are bones or bone fragments, such as found in this dog, and fish hooks. Other foreign bodies found in dogs include coins and hard food (e.g. uncooked or partially cooked potatoes, hard fruits), whereas toys or other play objects are more commonly found in cats. Many other objects have been found, including fabric, magnets, sticks, needles and just about anything an animal can attempt to swallow. Dental chews are increasingly being recognized for their potential to cause oesophageal obstruction.

Many foreign bodies are regurgitated or pass into the stomach or intestines, but those that are too large to pass through the oesophagus cause mechanical obstruction. The severity of oesophageal damage is dependent upon foreign body size, angularity or sharp points and the duration of obstruction. The most common locations are within the thoracic inlet, the base of the heart or the diaphragmatic hiatus, as these are the areas of least distensibility within the oesophagus. The pressure of the object can lead to pressure necrosis of the oesophageal wall, causing perforation or subsequent stricture.

In many cases there is a history of foreign body ingestion. In some cases the ingestion of the object goes unnoticed, particularly those associated with scavenging or rubbish ingestion. The onset of clinical signs depends upon the severity of oesophageal obstruction. Animals with complete oesophageal obstruction are often presented with acute signs, whereas animals with incomplete obstruction may be presented within days to weeks after the initial ingestion of the foreign body. Clinical signs include regurgitation, excessive salivation, odynophagia, anorexia, dysphagia, retching and respiratory distress.

Bone foreign bodies can occasionally be palpated if they become lodged in the cervical oesophagus, but definitive diagnosis requires radiography. Radiodense foreign bodies can be detected with survey radiography, but confirmation of radiolucent foreign bodies will require administration of contrast agents. Iodine contrast agents should be used instead of barium if oesophageal perforation is suspected. A foreign body can be confirmed and often removed during endoscopy.

The most important differential diagnoses would include oesophageal stricture, neoplasia, hiatal hernia and gastro-oesophageal intussusception. Each of these conditions can be differentiated with radiography and/or endoscopy.

Oesophageal foreign bodies should be removed promptly. The longer the foreign body is lodged in the oesophagus the greater the chances of oesophageal mucosal damage, ulceration and perforation. Rigid or flexible fibreoptic endoscopic retrieval should be the initial approach to treating an oesophageal foreign body although fluoroscopic-guided retrieval has been described. A rigid endoscope is most useful in retrieving large foreign bodies, particularly bones or bone fragments. Large grasping forceps are passed through the rigid endoscope to retrieve the foreign body. Large foreign bodies that cannot be safely removed through the mouth can occasionally be pushed into the stomach and removed by gastrotomy. Smaller foreign bodies are best managed with a flexible fibreoptic endoscope and basket, tripod or snare retrieval forceps. Flexible endoscopes are particularly useful in retrieving fish hooks. In one study, removal of oesophageal foreign bodies using an endoscope was successful in 90.2% of the dogs.

Careful assessment should be made of the damage to the oesophagus via the endoscope after foreign body removal. Survey radiographs should also be taken after removal to check for pneumothorax or pneumomediastinum secondary to oesophageal perforation.

Affected animals may be fasted for 24 hours after foreign body removal. Longer periods of fasting may be required if the oesophagus is necrotic, in which case, placement of a gastrostomy tube at the time of endoscopy facilitates feeding bypassing the oesophagus.

NURSING TIP ON FEEDING POST-OESOPHAGEAL FOREIGN BODY REMOVAL

Patients who have had an oesophageal foreign body removed are likely to initially have a painful oesophagitis. Liquid foods or canned food which has been made more liquid in a blender may be easier to swallow. If water is added to a canned food, the caloric content should be determined to make sure it is adequate. As these diets may be new to the patient and especially if the patient has not been eating adequately for the past 3 days or longer, the amount of food initially fed should not be more than one-third of the resting energy requirement per day. The amount can be gradually increased over 3 days. If the patient is reluctant to eat or swallows with difficulty, reassessment of the oesophagus and possibly the use of more analgesia may be necessary.

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Specific therapy for oesophagitis should include oral sucralfate suspensions (0.5–1.0 g po q 8 hours). Suspensions of sucralfate are more therapeutic than intact tablets. Anti-inflammatory doses of glucocorticoids have been recommended by some clinicians in those animals at risk for oesophageal stricture; however, there is no evidence that oral administration of glucocorticoids is of any benefit. In animals which have formed an oesophageal stricture, local oesophageal injections of corticosteroid (e.g. triamcinolone) via an endoscope after balloon dilation have shown some benefit and possibly this would be beneficial in animals at risk for oesophageal stricture, although it could also possibly delay healing. The risk of stricture is greatest in animals with a 180 degree or greater transmucosal ulceration. Broad-spectrum antibiotics should be considered in animals with severe ulceration and/or small perforations.

Minor oesophageal tears or lacerations less than 1 cm in length can usually be managed conservatively. Surgery is indicated if endoscopy fails or if there is evidence of larger oesophageal perforation. Gastrotomy is preferred to oesophagotomy for distal oesophageal foreign bodies because of the poorer healing properties of the oesophagus and the potential for stricture formation. However, oesophagotomy is indicated in those cases where the foreign body could not be removed through gastrotomy. Surgery is also indicated to repair oesophageal perforation.

Prognosis

The prognosis for most cases with oesophageal foreign bodies is generally good, especially if they are removed immediately, and one study reported 92% of dogs had no complications after discharge (although this study included gastric foreign bodies, which have a better prognosis). A worse prognosis is associated with foreign bodies that are large, have sharp points or are retained for a prolonged period of time. Immediate complications include complete obstruction or laceration, or aspiration pneumonia from the regurgitation. Late complications, which can occur a week or more after foreign body removal, include perforation, haemothorax, fistulation and diverticula, segmental hypomotility, or stricture formation.

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7 Persistent right aortic arch in a dog

INITIAL PRESENTATION

Regurgitating, especially food, since weaning
Signalment: 3-month-old uncastrated male Border collie, body weight 11 kg

CASE HISTORY

The puppy had been obtained from a farm about a month prior to presentation. Since then he had regurgitated frequently (several times a day), especially food but also occasionally water. The food he brought up was undigested with no bile present and the regurgitation occurred passively with no abdominal effort. Regurgitation usually occurred within minutes after eating, but recently it had occurred as long as an hour after meals. There was sometimes some retching after he regurgitated. He had no problem picking up his food and his initial swallowing appeared normal. He had been very bright, but was in poor body condition.

His initial diet was a dry puppy food, free choice. The owners had tried adding water to the food, but this did not improve the clinical signs. When they first obtained him he had a good appetite, but during the week previous to admission he had been less interested in eating. The owners had been trying to tempt him to eat with a variety of foods. His faeces had usually been normal, although since the change in diet, they occasionally were soft.

PHYSICAL EXAMINATION

The puppy was very bright and responsive, but not well grown. He was thin, with a body condition score of 2/9. His mucus membranes were pink and capillary refill time was less than 2 seconds. On oral examination his tonsils were slightly enlarged.

Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 140 beats per minute (bpm) and a respiratory rate of 24 breaths per minute. There was no evidence of pain on abdominal palpation and the only abnormality found was increased gas in the small intestines. Rectal temperature was 38.6° C.

PROBLEM LIST AND DIFFERENTIAL DIAGNOSES

Regurgitation was the puppy's primary problem. Differential diagnoses for regurgitation for this puppy included:

- Megaoesophagus
- Oesophageal stricture
- Oesophagitis
- Vascular ring anomaly (e.g. persistent right aortic arch (PRAA))
- Hiatal hernia
- Oesophageal dysmotility.

An oesophageal foreign body was unlikely (but not impossible) because of the duration of the clinical signs. Immune-mediated disorders (e.g. myasthenia gravis) and tumours were also unlikely in this young dog as they generally occur later in life.

Diagnostic aids

Diagnostic techniques that were indicated in this puppy included plain thoracic radiographs, which help diagnose megaoesophagus and foreign body. A positive contrast study using barium mixed with dog food would also help localize and characterize oesophageal disorders such as strictures, PRAA and dysmotility. Oesophageal endoscopy would also be useful to visualize the mucosa of the oesophagus.

CASE WORK-UP

Minimum data base

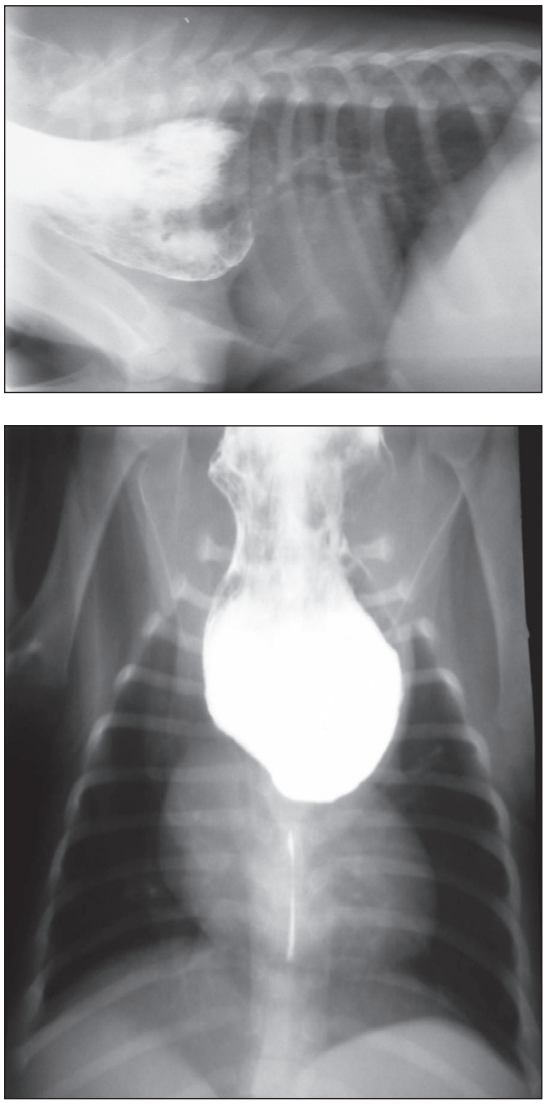
The dog was admitted to the hospital and a minimum data base including haematology, serum chemistry and

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routine urinalysis was performed. The results from these tests were unremarkable.

Imaging

Plain films indicated the possibility of a megaoesophagus in the cranial thoracic area. A positive contrast study with a barium meal revealed oesophageal constriction at the base of the heart and marked oesophageal dilatation extending cranially from that constricted area (Figs 7.1 and 7.2).



Figs 7.1 and 7.2 Lateral and ventrodorsal thoracic radiographs with contrast, showing enlarged oesophagus up to the level of the heart (courtesy of Dr Geoff Culshaw)

Endoscopy

Oesophageal endoscopy showed a pinched area of the oesophagus and also inflammation of the oesophageal mucosa.

DIAGNOSIS

A diagnosis of vascular ring anomaly and oesophagitis was made.

Treatment – surgical

The treatment consisted of transecting the bands forming the vascular ring. In this case a left thoracotomy was performed and a persistent right ligamentum arteriosum was identified, dissected and divided. A thoracostomy tube was placed for immediate post-operative management.

Treatment – medical

Treatment for the oesophagitis included omeprazole (1 mg/kg po q 24 hours) and sucralfate suspension (1.5 ml po q 8 hours given 1 hour before feeding or other medications).

CLINICAL TIP ON SUCRALFATE

Sucralfate binds to ulcerated mucosa forming a protective covering and stimulates repair mechanisms partially via stimulation of prostaglandin-mediated pathways. It has few side effects (constipation is reported in people but rarely seen in cats or dogs), but it can potentially affect the bioavailability of other medications and should probably be given separately. It may work best in the acid environment of the stomach, but does also work in a neutral pH and is therefore also indicated for use in oesophageal erosions or ulcers.

NURSING CARE

A liquid food was fed to try to ensure that adequate nutrition was maintained. The puppy's caloric requirements were determined and food provided in four meals per day. The initial amount of food on the first day provided one-third of his energy requirements and was increased to full feeding by day 3. The meals were provided from a height as with other megaoesophagus patients. Another option for nutritional support would have been placement of a gastrostomy tube.

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CLINICAL TIPS ON NUTRITION IN PUPPIES

Puppies need more calories per kilogram of body weight than do adult dogs or around 2.5 to 3 times resting energy requirements (RER). They also have higher protein requirements of 22 to 32% of dry matter and increased calcium requirements of 0.7 to 1.7% dry matter; these concentrations would be provided in a good puppy food.

For this 11 kg underweight puppy, initial RER was estimated to be 70 × body weight $(kg)^{0.75}$ or $70 \times 11^{0.75} = 443$ kcal. At 3 months of age he needs three times RER or about 1330 kcal. His initial feeding should be for his existing body weight, with increased amounts over time to increase his body condition score.

A typical puppy food contains around 3.8 kcal/g, so this puppy would need about 350 g of food per day.

Follow-up

The puppy was seen again 2 weeks after the surgery. He was still regurgitating, but it had decreased to once or twice per week. The puppy was still very bright and his body condition had improved to 3/9 and he had gained 2 kg. The owner was still feeding gruel.

DISCUSSION AND EPIDEMIOLOGY

Regurgitation after weaning is typical in patients with vascular ring anomalies. Nearly all affected dogs show signs before the age of 6 months. Vascular ring anomalies can be difficult to distinguish from idiopathic megaoesophagus, but contrast study with the vascular ring anomalies usually shows a pinching of the contrast just cranial to the heart base, whereas in idiopathic megaoesophagus there is usually oesophageal dilation caudal to the heart. Endoscopy will also show an area of stenosis at the point of constriction in cases with vascular ring anomaly. All patients with regurgitation are at risk for aspiration pneumonia and should be monitored for signs consistent with it. Poor body condition is consistent with inadequate food intake in many patients with oesophageal disorders.

The most common vascular ring anomaly is the persistent right aortic arch (PRAA). In normal embryonic development, the aortic arch develops from the left fourth aortic arch and left dorsal aortic root and the adult aortic arch is to the left of the oesophagus. There are other variations of abnormal development, for example retention of both arches or origination of the left subclavian artery from the right aortic arch. When the vessels develop abnormally, they may constrict the oesophagus (Fig 7.3).

The presence of oesophagitis in this case may have been due to the prolonged presence of food material irritating the oesophagus.

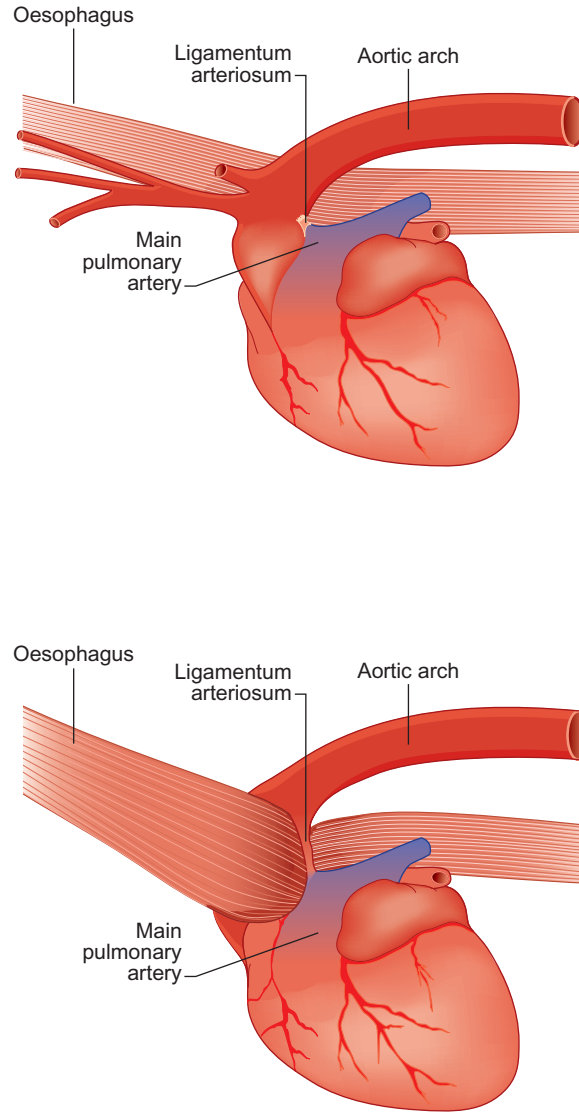


Fig 7.3 The normal heart and a heart with persistent right aortic arch

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It is likely that vascular ring anomalies have an inherited basis, as there are breed predispositions in German shepherd dogs, Great Danes, Irish Setters, Boston terriers and greyhounds. The disorders are much less common in cats, but have been reported.

Prognosis

It is estimated that only 10% of affected dogs will develop completely normal oesophageal function after surgery and up to 50% of affected dogs will continue to show occasional signs of regurgitation. Permanent oesophageal dilation cranial to the constriction is common due to irreversible degenerative changes. As many as 40% of these cases have been reported to have a short survival time due to malnourishment and/or aspiration pneumonia. This puppy's initial improvement in signs and body condition was encouraging, although he was still at risk for aspiration pneumonia.

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8 Feline dysautonomia

INITIAL PRESENTATION

Chronic dysphagia, vomiting, constipation, weight loss

Signalment: 2-year-old male neutered domestic shorthaired cat, body weight 3.75 kg

CASE HISTORY

The cat had a 5-week history of vomiting frothy fluid and partially digested food three to four times a day. He appeared interested in eating, but had difficulty swallowing and was occasionally retching. He had also regurgitated several times recently. His faeces were very dry. He had difficulty passing faeces and sometimes retched when attempting to defecate. His owners felt that the cat was drinking less and he was producing very small amounts of urine. He had also been sneezing and they said he often had a crusty discharge at his nostrils. The owners thought that he had lost about 1 kg in weight during this time.

The owners had owned the cat since he was a kitten. He lived with one other unrelated cat that was healthy and they had both been de-wormed and vaccinated approximately 3 months earlier. His previous diet was a combination of a commercial dry cat food and a commercial canned cat food, but he had recently only been able to swallow small amounts of the canned commercial food. To encourage him to try to eat, the owners had also been giving him small bits of canned tuna.

Previous diagnostic tests had been performed to check for toxoplasmosis, feline immunodeficiency virus and feline leukaemia virus (FeLV); the results of all of these tests were negative.

PHYSICAL EXAMINATION

The cat was quiet but responsive. He had evidence of weight loss, his body condition score was 3/9 and he had poor muscle mass. The mucous membranes of his mouth were dry and pink and capillary refill time was <2 seconds. His heart rate was 120 beats per minute (bpm), respiratory rate was 40 breaths per minute, rectal temperature was 37.9° C and systolic blood pressure was low normal at 140 mmHg. The thorax was normal on auscultation and compression. Palpation of the abdomen revealed a full bladder from which urine was easily expressed.

He had bilaterally dilated pupils which were not responsive to light and his third eyelids were elevated. He did respond to a menace test, although the response was decreased. He had normal postural reactions in all limbs and normal patellar reflexes. A Schirmer tear test showed bilaterally decreased tear production; the right eye was 10 mm and the left was 8 mm (reference range greater than 15 mm in 1 minute).

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The cat's problems included dysphagia, vomiting, constipation with tenesmus and non-responsive dilated pupils with decreased tear production. As he was unable to initiate swallowing, the dysphagia, appeared to be

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primarily oropharyngeal, although, as he had also regurgitated, a more general acquired neuromuscular disorder was likely.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses lists for the problems in this cat include the following.

Dysphagia and regurgitation

- Cranial nerve neuropathies, e.g. nerves VII, IX and/or XII
- Inflammation, e.g. caustic agents, infections, immune-mediated disorders
- Pharyngitis/oesophagitis
- Oropharyngeal/oesophageal neoplasia
- Retropharyngeal disorders, e.g. polyps, lymphadenopathy, haematoma, abscess, tumour
- Neuromuscular disorders, e.g. myasthenia gravis, dysautonomia, rabies, botulism
- Oropharyngeal/oesophageal foreign body
- Megaoesophagus (for the regurgitation, not for dysphagia).

Vomiting

- Adverse reactions to food
- Gastrointestinal inflammatory disorder, e.g. inflammatory bowel disease, lymphoma, gastritis, enteritis
- Gastrointestinal obstruction, e.g. foreign body, pyloric stenosis, ileus, tumour
- Constipation
- Intussusception
- Parasites, e.g. *Ollulanus triceps, ascarids, Physaloptera*
- Systemic disorders and disorders of other organs, e.g. uraemia, liver disease, congestive heart failure, pancreatopathy, metabolic disorders (diabetes mellitus, electrolyte imbalances)

Constipation/tenesmus

- Ingestion of indigestible material, e.g. cloth, hair
- Lack of exercise
- Painful defecation, e.g. trauma, pelvic fracture, inflammatory musculoskeletal disorders
- Obstruction, e.g. tumour, stricture
- Neurological disease, e.g. dysautonomia, idiopathic megacolon, sacral spinal nerve, pudendal nerve or perineal nerve disorder
- Dehydration

Dilated, non-responsive pupils

- Disorder of the visual pathways (likely to cause blindness)
- Disorders of the parasympathetic fibres that innervate the muscle fibres of the iris (oculomotor, CN III)
- Iris atrophy (unlikely in this young cat)
- Fluoroquinolone induced retinal degeneration – causes blindness but loss of pupillary light reflex can be an early sign (no history of its use in this cat)
- Anti-cholinergic drug toxicity
- Hypertension (although retinas appeared normal)
- Feline infectious peritonitis (previous negative test)
- Cryptococcosis
- Toxoplasmosis (unlikely as the serum test titres too low in this cat)
- Lymphoma
- Cuterebra migration (unlikely in the UK)
- Lead toxicity (no history consistent with lead ingestion)
- Spastic pupil syndrome has also been reported in FeLV+ cats due to ciliary ganglion lesions (FeLV negative on test)

Decreased Schirmer tear test (decreased tear production)

- Lesions of the afferent arm of the tearing reflex
  - loss of sensation from the cornea, conjunctive
  - trigeminal nerve disorder
- Lesions of the efferent arm of the tearing reflex
  - disorder of parasympathetic nerve supply to the lacrimal gland
  - facial nerve disorder
  - severe otitis media or middle ear neoplasia
  - keratoconjunctivitis sicca

CASE WORK-UP

The combination of clinical signs made dysautonomia a likely diagnosis as there appeared to be failure of the autonomic function in multiple organ systems.

Minimum data base

Haematology and serum chemistry results were unremarkable other than parameters elevated by

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dehydration, e.g. albumin was 41 g/l (reference range 28–39 g/l).

Urinalysis showed a urine specific gravity of 1.051, also consistent with dehydration. No abnormalities were noted on the chemical strip and the sediment showed some epithelial cells and white blood cells.

Pilocarpine test

Pharmacological testing is helpful for ruling out other potential diagnoses and a pilocarpine test performed in this cat showed a positive result (Figs 8.1 and 8.2).



Fig 8.1 The patient prior to pilocarpine showing dilated pupils (courtesy of Dr Danielle Gunn-Moore)



Fig 8.2 The patient after pilocarpine administration to one eye, showing constriction of the pupil of the treated eye (courtesy of Dr Danielle Gunn-Moore)

CLINICAL TIP ON PILOCARPINE TEST

Place one drop of diluted pilocarpine ophthalmic solution (0.05%) in one eye and observe the diameter of the pupil every 15 minutes for 1 hour. Most normal animals will not respond to this concentration or only show a minimal response at 60 minutes. Miosis of the treated eye demonstrates the denervation supersensitivity which occurs with dysautonomia. Toxicity with an anti-cholinergic drug would block the response of the pupil to pilocarpine.

Imaging

Thoracic radiography showed no abnormalities in this cat. Aspiration pneumonia can occur with regurgitation and sometimes megaoesophagus is seen in patients with dysautonomia. Abdominal ultrasound showed intestinal ileus, moderate gastric dilation, a large full bladder and a colon containing some faecal material.

TREATMENT AND OUTCOME

Treatment included intravenous crystalloid fluids for rehydration. Symptomatic treatment for the constipation and ileus included cisapride, a promotility drug with effects in the colon as well as the stomach and small intestine, at an initial dose of 0.1 mg/kg po q 12 hours. This dose was increased gradually to 0.5 mg/kg to produce a soft but not diarrhoeic stool. The cholinergic drug bethanechol (1 mg po q 12 hours) was initiated to aid bladder emptying and for the first 2 days his bladder was expressed 2 to 4 times per day. A lubricating eye ointment was applied to both eyes every 4 to 6 hours.

The cat was brighter after rehydration and the first day in hospital on medications. He urinated a small amount on his own without his bladder being expressed. He did not pass faeces until the third day and they were still fairly firm, so the dose of cisapride was increased.

An oesophageal feeding tube was placed for nutritional support (Fig 8.3) and a liquid nutritional supplement was provided, initially at one-third resting energy requirement (RER), with the intent to increase to full RER over 3 days. After 2 weeks the cat was discharged with the owners to feed using the feeding tube. He was continued on the cisapride, bethanechol and ocular lubricants. The cat was willing to eat small amounts of soft food and the owners were able to feed him successfully using the oesophageal feeding tube.

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Fig 8.3 The patient with an oesophageal feeding tube in place

NURSING TIP

Daily RER for this cat was 3.75 $kg^{0.75}$ × 70 = 188 kcal. The nutritional supplement contained 1 kcal/ml. On the first day the caloric goal was 63 kcal or ml, divided into six 10-ml increments. The feedings must be delivered slowly as with the distal end of the tube in the oesophagus it is not possible to check by aspirating on the tube how much remains in the stomach from the previous meal. Any evidence of refluxing or vomiting means that feeding should be stopped and either no food or smaller amounts of food should be fed during the next feeding period.

NURSING TIPS ON CAT CARE

A clean cat is a happier cat, so cats should always be gently groomed and any faeces or urine cleaned off of them. As this cat was not emptying his bladder fully, gentle manual expression of the urine was done several times a day. If urine accumulates in the bladder, there is an increased risk of urinary tract infections and of development of a flaccid, atonic bladder which cannot contract and empty efficiently.

With cats (or dogs) that have dry eyes, a mucus thread or discharge tends to form in the medial canthus of their eyes – this should be gently removed and a lubricant applied to the eye. If dry eyes are not lubricated, the animals are at risk of forming corneal ulcers.

The oesophageal tube was removed at a 3 week re-check.

At re-checks over the following 4 months, his demeanour and appetite improved, although he remained more comfortable eating canned rather than dry cat food. His pupillary response improved, but was not normal.

DISCUSSION

Dysautonomia is characterized by degeneration of the autonomic ganglia and failure of autonomic function and the clinical signs reflect this failure. Typical signs include dysphagia, vomiting, regurgitation, dysuria, diarrhoea or constipation, dry mucous membranes (eyes, nose and mouth), loss of pupillary light reflexes, third eyelid elevation and bradycardia or a lack of increased heart rate with sympathetic stimulation. A dilated anal sphincter is sometimes found, although this is skeletal muscle so the cause of this is not known. Diagnosis is based on typical signs and the use of a pilocarpine test helps rule out other differential diagnoses.

At post-mortem, there are no consistent gross pathological findings. Most animals are thin with a loss of muscle mass and some patients will have a megaoesophagus. Histological examination shows widespread degeneration of both sympathetic and parasympathetic nervous systems, especially in the autonomic ganglia. Neurones appear abnormal and may be reduced in density.

The cause of dysautonomia is unclear. Clostridial toxins have been implicated as the cause in horses. There is evidence that equine dysautonomia is associated with an infection with *Clostridium botulinum* type C.

A study has been done to investigate the presence of *C. botulinum* type C neurotoxin in the food, ileal content, faeces and serum of cats with dysautonomia. The toxin was detected directly in four of eight affected cats and after enrichment in seven of them. It was also detected in their dried food. No toxin was detected in healthy control cats or in their tinned food. The levels of IgA antibodies to the toxin and to surface antigens of *C. botulinum* type C in the faeces of the affected cats 14 weeks after the outbreak were significantly higher than in the faeces of the control cats. From this study, it appears that the botulinum neurotoxin is a factor in the aetiology of dysautonomia, although there may be a predisposition in some cats due to genetic or other factors.

Treatment primarily involves supportive care. Prokinetic drugs such as cisapride may improve gastrointestinal tract motility, including the colon. Metoclopramide may also be used, but is only effective for gastric

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emptying and duodenal motility. Cholinergic drugs such as bethanechol may help to prevent constipation and urinary retention. Bethanechol should be used with caution as it can cause bradycardia and arrhythmias and many of these cats present with bradycardia.

Parasympathomimetics are sometimes used to stimulate oronasal secretions. Although not used in this case, 1% pilocarpine (one drop in both eyes q 6 hours) may improve lacrimation. Ocular lubricants are also indicated for cats with dry eyes.

Supportive care also includes: rehydration, emptying of the bladder and colon if needed, monitoring for and treatment of urinary tract infections, nutritional support and possibly physical therapy involving flexing and extending joints and massage of muscles. Nursing care can make the difference in the outcome of these cases.

Epidemiology

Feline dysautonomia (also referred to as Key–Gaskell syndrome) was first reported in the United Kingdom in 1982. Initially it was rarely reported outside the UK; however, cases have been more recently reported in other countries, including the United States, where it is seen more in the Midwestern states. Where multiple cats in a household are affected, they are often related. The cases often seem to present in outbreaks, with multiple cases presenting over several months and then few cases seen in between these outbreaks. While there is a wide range of ages reported in affected cats, most commonly they are young adults.

In one report, six of eight pet cats living together developed signs of dysautonomia in 1 week. Two of these cats died and one was euthanased. In the two apparently unaffected cats, abnormal oesophageal motility was demonstrated by fluoroscopy, suggesting that there may be a subclinical form of the disease. The surviving cats had higher and more variable heart rates (mean 165 bpm) than the non-survivors (mean 121 bpm).

In dogs, being from a rural area and spending >50% of time outdoors have been recognized as risk factors, although indoor dogs are not immune. The effect of living outdoors or indoors has not been studied in cats.

Prognosis

Prognosis is generally thought to be poor, with a mortality rate estimated by some studies to be about 60%, although with dedicated nursing care it may be lower. Many survivors do not have complete recovery and some continue to show neurological signs, although they may still have an acceptable quality of life. Although severely affected cats carry a worse prognosis, even these cats may recover. Recovery often begins several months after the onset of clinical signs and complete reversal of signs may take up to a year or more.

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9 Gastrointestinal physiology – the normal stomach and small intestines

THE STOMACH

The stomach lies to the left of the median plane of the body. When empty, it is within the costal arch and a normal empty stomach cannot be palpated during physical examination. Even when full, the examiner may need to hook his or her fingers underneath the costal arch to feel a normal stomach.

The stomach is divided anatomically into five regions: cardia, fundus, body, antrum and pylorus (Fig 9.1). Physiologically, the stomach has a proximal part which stores food temporarily and a distal part which regulates the release of hydrochloric acid, grinds food particles and controls the emptying of the stomach. The fundus of the stomach dilates in response to the entry of food in receptive relaxation which results in a decrease in fundic motor activity and pressure. As dogs tend to eat large meals as opposed to cats, which tend to eat frequent small meals, the storage capacity of the stomach is likely to be of greater importance to dogs.

The stomach contributes to the initial stages of digestion by secreting hydrochloric acid and pepsinogen. Muscles of the antrum grind food particles and peristaltic waves move from the body of the stomach to the antrum towards a usually partially closed pylorus. A strong retrograde wave then moves the food back into the proximal antrum resulting in grinding into particles small enough to be allowed through the pylorus.

The pylorus and the antrum function as a unit to regulate the emptying of solid food. In dogs the particles of food are usually less than 2 mm in size before they move through the pylorus. Large indigestible particles of food do not leave the stomach until the interdigestive period (after digestion is complete). In fasted dogs an interdigestive motor complex (migrating motor complex) moves through the stomach and intestines to clear these larger particles (and sometimes also foreign bodies) into the intestines. This is also called a 'housekeeping wave'. The electrical impulse in cats differs from that of dogs; the wave is stimulated by a migrating spike complex, which likely serves the same function in the cat.

SMALL INTESTINE AND PANCREAS

Most of the enzymatic digestion of food occurs within the small intestine. The small intestine is divided into the duodenum, jejunum and ileum, although no anatomic distinction divides one section from another. The small intestine is 1.80 to 4.80 m long in the dog and about 1.3 m long in the cat (Fig 9.2). The pancreas lies near the duodenal flexure (Fig 9.3).

Like the oesophagus and the stomach, the intestine contains mucosal, submucosal and muscle layers (Fig 9.4). The mucosa consists of a single layer of epithelial cells with the lamina propria below it. Scattered throughout the epithelial cells are mucus-producing goblet cells. The luminal surface consists of a brush border made up of microvilli (Fig 9.5). The microvilli increase the surface area for digestion and absorption. They possess special mechanisms for transporting monosaccharides and amino enzymes and contain enzymes to digest disaccharides, oligosaccharides and some small peptides. The brush border also contains proteins that bind many other substances such as calcium, iron and cobalamin.

Among the villi are the crypts of Lieberkuhn which contain immature or stem cells which move up the villi as they mature into fully differentiated villus epithelial cells. Migration takes about 2 days and the cells are mature when about one-half to one-third of the migration is complete. Increased bacterial numbers, physical trauma or chemical trauma may shorten the epithelial cell survival time and lead to villus atrophy. Drugs that interfere with cell replication (e.g. many chemotherapeutic drugs) prevent normal cell renewal, as does fasting. A deficiency of either vitamin B_{12} (cobalamin) or folate will also result in mucosal atrophy. Maintenance of the

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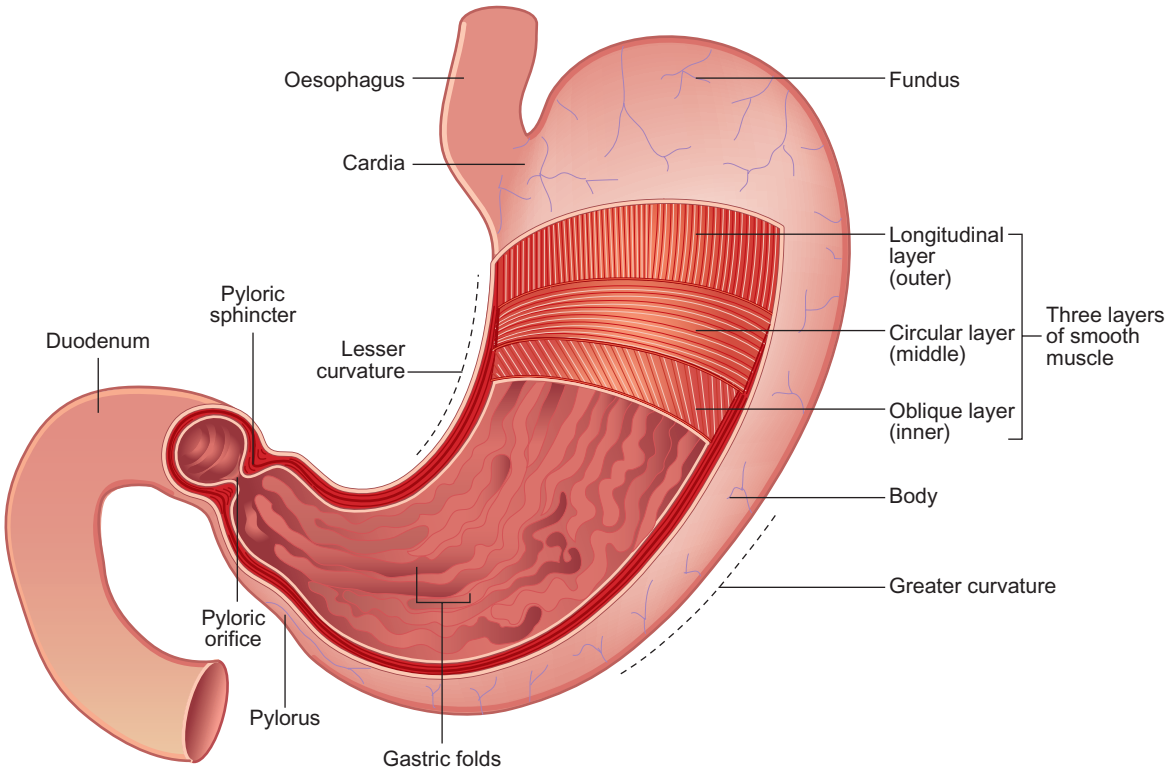


Fig 9.1 Anatomy of the stomach

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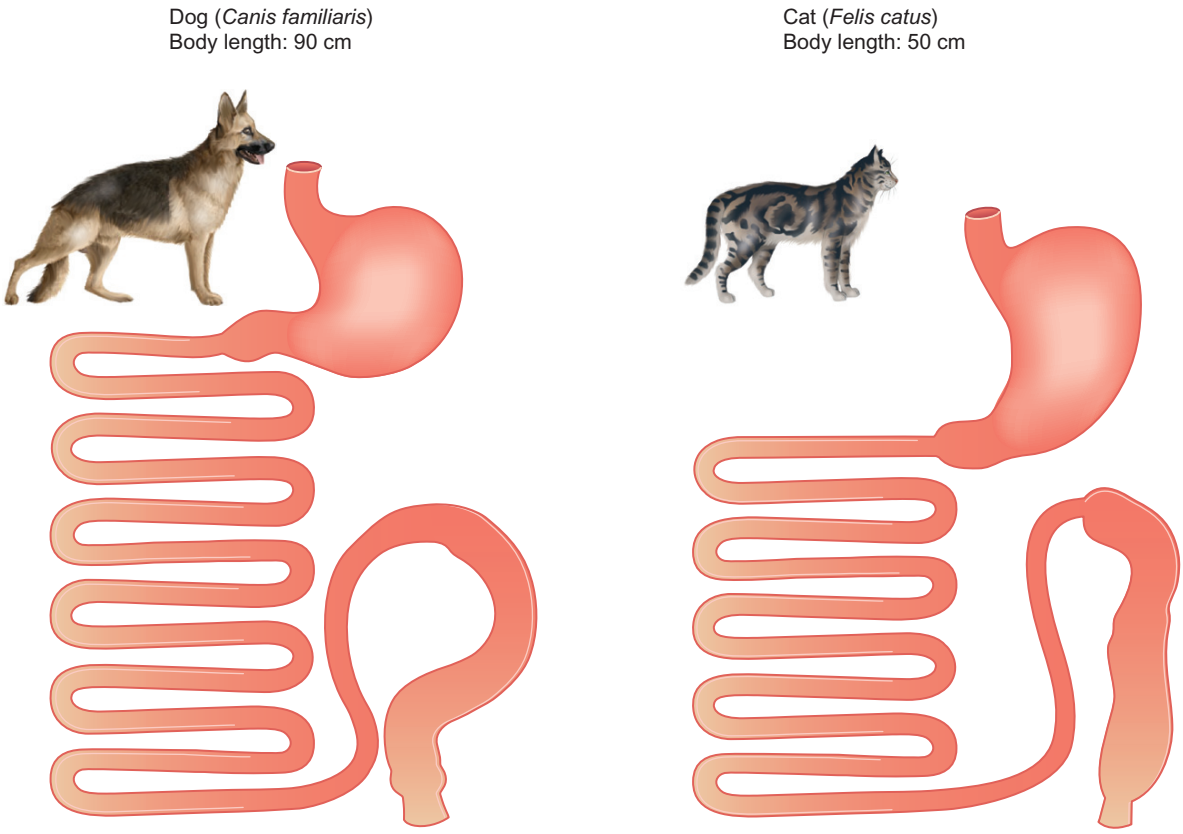


Fig 9.2 The small intestine of the dog and cat

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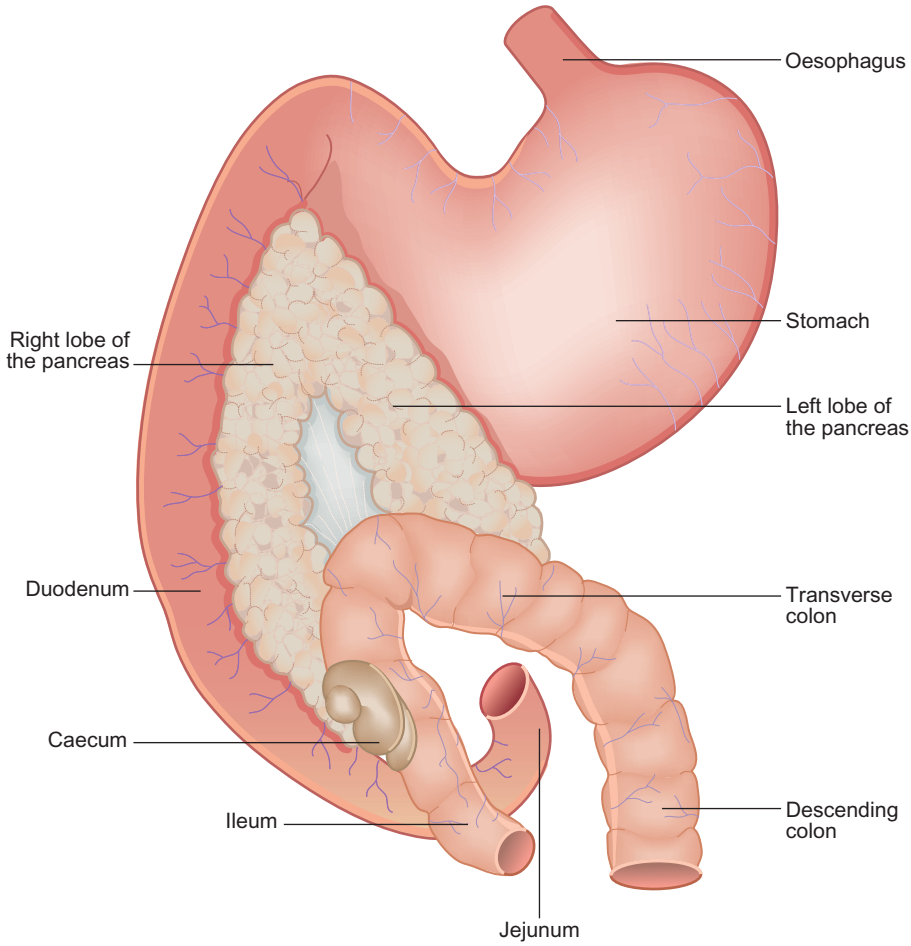


Fig 9.3 Relationship of the pancreas to the stomach, duodenum and colon

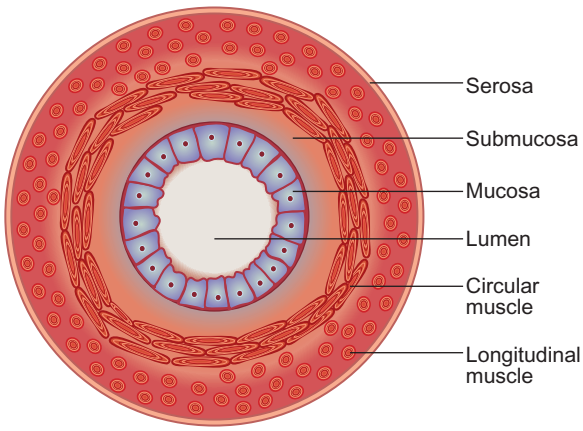


Fig 9.4 Layers of the small intestine

mucosal layer is vital for the barrier function of the intestine which prevents systemic spread of bacteria or other deleterious agents from within the intestine. The health of the barrier is stimulated by enteral feeding, especially by the dietary component glutamine.

Motility of the small intestine mixes and slows the passage of contents and also moves them aborally. Rhythmic contractions slow the movement, while peristalsis propels the contents aborally so that there is coordination of 'breaking and accelerating' effects (Fig 9.6). The transit time of food in the small intestine in dogs appears to be about 1 to 2 hours and in the cat about 2 to 3 hours.

The pancreas secretes enzymes that are important for the digestion of carbohydrates, proteins and lipids, and brush border enzymes of the small intestine further

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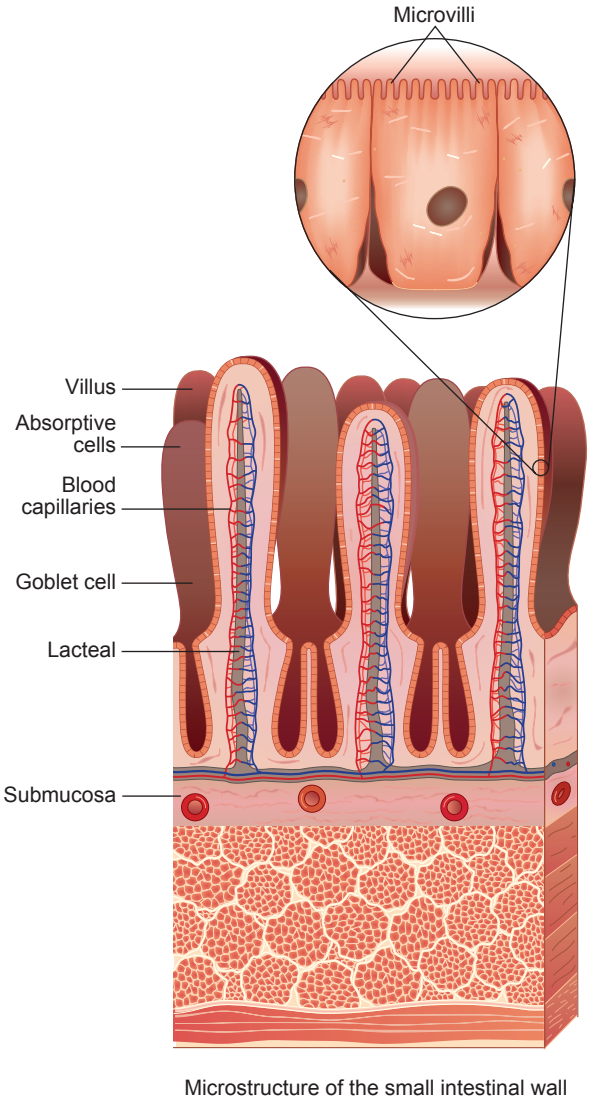


Fig 9.5 Microvilli of the small intestine

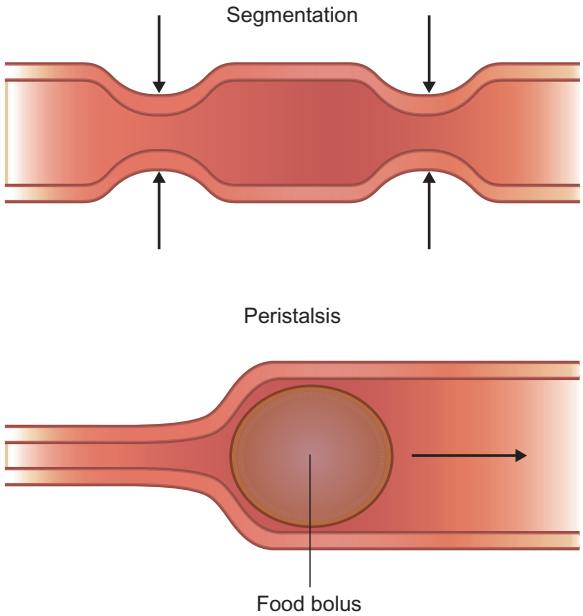


Fig 9.6 Intestinal motility showing the effects of segmentation and peristalsis

contribute to carbohydrate absorption. The brush border enzymes are affected by diet, disease and age. As animals mature, the amount of lactase is decreased, so that adult animals may not tolerate the disaccharide lactose in milk. When the diet is changed, it takes about 2 days for the enzymes to adapt (while the epithelial cells migrate up the villi) and abrupt dietary changes may lead to an increase in undigested carbohydrates causing osmotic diarrhoea prior to adaptation. Enteritis due to any cause can result in a loss of enzyme activity and resulting diarrhoea. In animals with large numbers of intestinal bacteria, bile salts may be deconjugated in large enough amounts to damage the microvilli. Fasting also decreases brush border enzymes so a return to feeding should be gradual enough to allow enzyme activity to increase.

In addition to assimilation of nutrients, the intestines are important in the secretion of fluid and electrolytes. As much as 8 to 10 l of fluid may move in and out of the intestine daily in a 20 kg animal. If absorption is compromised or secretion is excessive, diarrhoea may occur.

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10 Vomiting

PATHOPHYSIOLOGY OF VOMITING

Vomiting is a reflux of food and fluid from the stomach. It is a reflex act that involves prodromal signs of nausea, which may include anxiety, salivation, lip licking and restlessness.

Prior to vomiting, reverse peristalsis moves intestinal contents into the stomach and the animal begins to retch. There are then gastric antral and retrograde antral contractions with relaxation of the body of the stomach. The gastro-oesophageal and pharyngo-oesophageal sphincters relax. The driving force of vomiting is the contraction of the abdominal muscles and the diaphragmatic muscles.

Vomiting is a reflex act resulting from stimulation of the vomiting centre in the brain stem (Fig 10.1). The vomiting centre receives afferent input from peripheral receptors in the viscera from the chemoreceptor trigger zone (CRTZ) in the floor of the fourth ventricle of the brain, from the vestibular apparatus and from the higher centres of the brain such as the cerebral cortex. The peripheral visceral receptors are located throughout organs in the body, especially in the duodenum, sometimes referred to as 'the organ of vomition or nausea'. Distension or irritation of the intestinal mucosa may stimulate the vomiting centre and inflammation of other organs such as the pancreas can also result in vomiting. Afferent nerve fibres run from these organs in the vagal and sympathetic nerves.

The CRTZ is on the floor of the fourth ventricle and mediates vomiting from toxins such as those present in uraemia and also vomiting associated with some drugs, e.g. apomorphine or xylazine.

The vestibular apparatus mediates vomiting due to motion sickness or vestibular disease. Stimulation of the higher centres such as the cerebral cortex from acute stress may result in vomiting, although this may be more common in human beings than in animals.

DIAGNOSIS OF VOMITING

Several questions need to be answered early in the approach to vomiting cases. First, is it vomiting or regurgitation? Second, is the vomiting due to gastrointestinal (GI) disorders or due to disorders of other body systems? The signalment, history and physical examination often provide clues to these answers.

SIGNALMENT AND HISTORY

The patient's 'signalment' is the age, breed and sex. Knowing these factors often increases (or decreases) the index of suspicion for some disorders and helps the clinician order the differential diagnoses list.

The history should include the progression of the vomiting, the frequency, the timing with regards to eating and the time of day the vomiting most often occurs. Vomiting more than 12 hours after eating indicates delayed gastric emptying. Vomiting bile early in the morning can be part of the 'bilious vomiting syndrome' in dogs. A description of the contents of the vomitus, e.g. bile, foam, digested food, bright red blood or digested blood 'coffee grounds', is useful.

Careful questioning can usually allow the veterinary surgeon to determine if the patient is vomiting or regurgitating, although some cases do show both signs. Vomiting is the forceful ejection of gastric contents from the stomach, while regurgitation is the expulsion of ingesta from the oesophagus or pharynx. Regurgitation is described as a passive event; however, gagging may be present which can result in retching. With regurgitation, the animal should not show nausea, but drooling can be present if swallowing is impaired. Regurgitated food usually does not contain bile and should not have an acidic pH (unless refluxed from the stomach into the oesophagus). It is more common for animals to 're-eat' regurgitated food than vomited food. Regurgitated food

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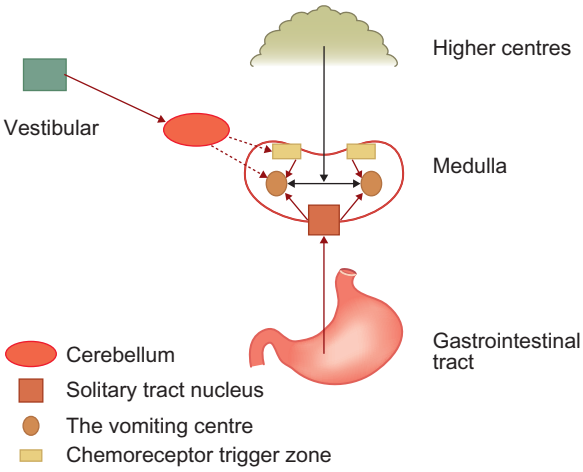


Fig 10.1 The vomiting centre

often appears undigested and may have the shape of the oesophagus. Regurgitation may take place immediately after eating, but can occur hours after eating, especially in cats where the normal oesophageal motility is slow.

Vomiting is usually accompanied by signs of nausea and a forceful ejection of food, with an abdominal effort present. The pH of vomitus may be acid, but can be alkaline, especially if duodenal contents are present. Expectoration – the ejection of material from the airway and laryngopharyngeal area – is also a forceful act and must be differentiated from vomiting and regurgitation. Expectoration and regurgitation are what cats do to remove hairballs from the laryngopharynx and oesophagus.

Environmental history should provide information regarding access to toxins such as lead, houseplants, cleaners, floor finishes, deodorants and organophosphates. The history of medication administered is important as many drugs can cause vomiting, e.g. non-steroidal anti-inflammatory drugs, corticosteroids, digitalis and some antibiotics. Asking questions regarding playing with toys, sticks, stones and string are important for possible foreign body ingestion, especially in young animals and retrievers.

During the history taking, particular attention should be paid to the diet, including information about scavenging, treats, snacks and food provided by neighbours or children. When questioned carefully, many owners provide evidence of a dietary influence on the patient's vomiting. A review of all body systems is always necessary. Anything that incites or exacerbates the signs should be noted, as well as abnormal posturing that may reflect abdominal pain.

PHYSICAL EXAMINATION

A complete physical examination is always necessary, including a thorough abdominal palpation. Examination of the teeth can sometimes indicate if a dog is a rock chewer and could have swallowed a stone. The underside of the tongue should be examined for string or other linear foreign bodies. The neurological status should be noted, as some neurological diseases can result in vomiting. Dehydration should be assessed. If the history is consistent with insufficient fluid intake and vomiting, dehydration is estimated to be at least 5% regardless of physical signs (i.e. subclinical dehydration). A rectal examination may reveal diarrhoea, melaena or haematochezia.

WORK-UP

If the vomiting is chronic and the animal is not ill and is eating well, a 1-month dietary trial with a restricted novel protein may be tried. Either a homemade or a commercial diet may be used. In dogs that have a history of early morning bilious vomiting, a late night snack or meal may reduce the vomiting. Some of these dogs are also helped by an evening dose of oral metoclopramide, although motility modifying drugs such as metoclopramide should not be used prior to ruling out foreign bodies. Ideally all animals at least have a minimum data base performed.

Minimum data base and other laboratory data

In most cases of chronic vomiting, further work-up is necessary. Causes of vomiting may be divided into those which are directly caused by GI disorders and those that are caused by disorders of other body systems. The minimum data base aids in deciding if a primary GI disorder or a non-GI disorder causes the vomiting. The initial diagnostic work-up includes haematology, serum chemistry, urinalysis and faecal flotation. If there is any indication of dehydration, parenteral fluids should be administered, as oral rehydration may not be effective.

Haematology is useful for detection of infection, toxaemia, eosinophilia associated with eosinophilic gastritis,

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parasitism, mastocytosis or hypoadrenocorticism, lead toxicosis, blood loss and determination of hydration status. A buffy coat smear may show mast cells in cases of mastocytosis, but normal animals may also have mast cells in their buffy coat, so this is not specific for mastocytosis.

Serum chemistry provides information about possible protein-losing enteropathies, azotaemia, liver disease, hypoadrenocorticism, diabetes mellitus, hydration status and electrolyte concentrations. A serum basal cortisol of greater than 70 μmol/l (or 2 μg/dl) makes hypoadrenocorticism unlikely.

Determination of serum amylase and lipase may provide an indication (albeit neither sensitive nor specific) of pancreatitis in canine patients. In acute pancreatitis, serum trypsin-like immunoreactivity (TLI) will be increased and may aid in the diagnosis. Tests specifically for serum canine and for feline pancreatic lipase immunoreactivity (cPLI and FPLI, respectively) are more highly sensitive and specific tests for pancreatitis than total serum lipase.

Puppies and kittens with congenital porto-systemic shunts (CPSS) may present with GI signs. Some of the clinicopathological changes associated with CPSS include microcytosis, decreased albumin and increased liver enzymes, although values of these parameters within the reference ranges does not rule out CPSS. A bile acid stimulation test should be performed whenever there is a possibility of CPSS. (Copper coloured irises in cats are nearly pathognomonic for this disorder.)

In older cats, a serum total T4 (thyroxine) should be taken to rule out hyperthyroidism. Over 50% of cats with hyperthyroidism may present with vomiting.

Determination of serum total carbon dioxide (TCO_2) can be useful. An increase in TCO_2 indicates alkalosis (as opposed to increased PCO_2, which indicates acidosis). Alkalosis in a vomiting small animal patient (who has not been administered alkalinizing agents such as sodium bicarbonate) nearly always means a pyloric or duodenal obstruction. Look for a foreign body!

Urinalysis provides information about renal and liver diseases, ketoacidosis, glucosuria and hydration. If possible, at least two drops of urine should be collected whenever initial serum chemistry is performed for chemical strip analysis and urine specific gravity. Ideally 5 to 10 ml should also be collected and centrifuged for a sediment examination.

Faecal examination for ascarids may be helpful, especially in puppies and kittens. De-worming with an effective agent such as fenbendazole would be recommended in some cases, even if parasite ova are not detected, as some parasites shed eggs only intermittently. *Ollulansus tricuspis* can cause vomiting in cats (and rarely in dogs) and is best diagnosed by a Baermann examination of the vomitus rather than faecal examination.

Further tests useful in some cases

Serum lead concentrations should be measured if there is a history consistent with lead ingestion, e.g. remodelling of an older house or neurological or behaviour signs associated with the vomiting. Plasma cholinesterase can be measured if history and clinical signs indicate possible organophosphate toxicity. Cats from areas endemic for *Dirofilaria immitis* heartworms should have a serologic test for *D. immitis* antigens and a thoracic radiograph, as this disease may present as vomiting in the cat.

Imaging

Survey (plain) radiographs of the abdomen are essential for any animal with chronic vomiting. They are used to look for radio-opaque foreign bodies, intestinal dilation, gastric size, position and content, liver and kidney size, uterine size (i.e. for pyometra), abdominal masses and bowel perforations. In cats, congestive heart failure can also cause partial anorexia and vomiting, so thoracic radiographs should be taken if the clinical examination indicates cardiac abnormalities.

Contrast radiography

Liquid gastrograms with barium or with iodinated compounds if a perforation is suspected may be useful for detection of gastric masses, gastric or intestinal foreign bodies, identifying the size and shape of the stomach and estimating the size of the liver. If enough barium is used, mucosal detail can be evaluated. Liquids and solids empty from the bowel differently, so liquid barium gives no information about most gastric motility disorders.

Barium mixed with a meal gives some indication of gastric emptying, which should begin within 30 minutes of ingestion. The barium may separate from the meal and be emptied separately as a liquid.

Barium impregnated polyethylene spheres (BIPS) are used to detect motility disorders and obstructions of the GI tract. There are 30 1.5-mm and 9 5-mm BIPS packaged in gelatin capsules (Fig 10.2). The small BIPS mimic the action of food particles; when mixed with food they

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Fig 10.2 Barium impregnated polyethylene spheres (BIPS)

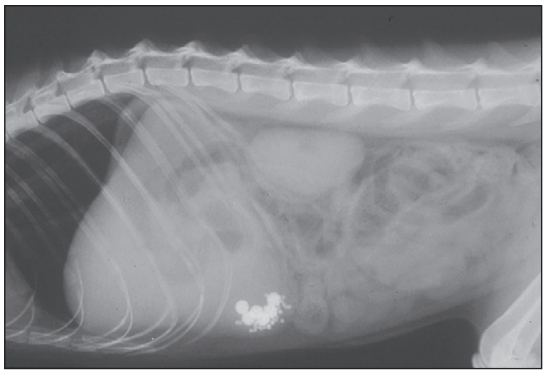


Fig 10.3 BIPS used to show an obstructive pattern where they have bunched together and are not being emptied from the stomach

can be used to estimate the GI transit time of food. The large BIPS are useful for detection of partial (or complete) obstructions (Fig 10.3). Determination of functional (i.e. functional ileus) vs. physical obstruction is made by a combination of history, clinical signs, laboratory findings, radiographic appearance and the BIPS radiographic pattern. Performing surgery or endoscopy after using BIPS is preferable to doing these procedures after barium has been used.

Ultrasound

Ultrasound is more sensitive than radiography for examining masses, intestinal mural thickenings, GI infiltrative diseases and mesenteric lymphadenopathy. The pancreas, liver, kidneys, prostate and uterus can be more fully evaluated by ultrasound than with radiography. In the hands of a good ultrasonographer, this technique is one of the best methods of diagnosing pancreatitis.

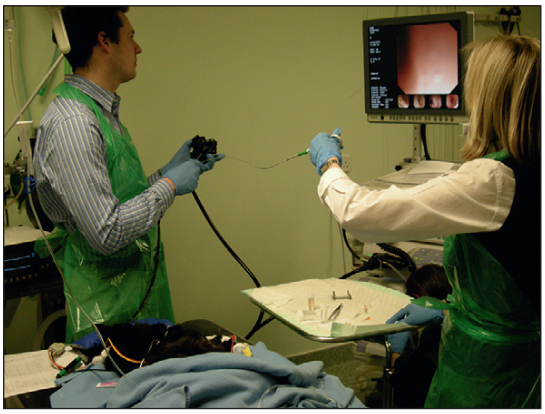


Fig 10.4 Use of endoscopy to take intestinal biopsies to aid in the diagnosis of vomiting in a cat

Intussusceptions can sometimes be clearly observed and intestinal peristalsis can be assessed. The organs can be seen through ascitic fluid, but gas, ingesta and faeces can obscure the view. Ultrasound guided fine needle aspirates or larger needle biopsies can sometimes provide useful information and may be preferred to surgically obtained samples in some animals (e.g. with severe hypoalbuminaemia).

Endoscopy

Endoscopy is not appropriate for every animal with GI disease. The more clinically ill patients, with severe signs (e.g. intractable vomiting or haematoemesis), weight loss, anorexia or ultrasonographic signs of infiltrative disease are candidates for endoscopy.

Endoscopic examination and biopsy of the stomach, duodenum and sometimes colon and ileum (Fig 10.4) is an aid in diagnosing inflammatory bowel disease (a common cause of vomiting in dogs and cats). It also aids in the diagnosis of some lymphomas, gastric adenocarcinoma and other gastric cancers, gastroduodenal ulcers, gastritis, pyloric stenosis and some foreign bodies. Some alimentary lymphomas require full thickness biopsy for diagnosis. Foreign bodies may sometimes be removed using the endoscope and forceps or snares.

Exploratory celiotomy

Exploratory surgery is necessary for the diagnosis of mural diseases (e.g. where mucosal biopsies obtained with an endoscope are not deep enough), for small

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bowel lesions distal to the reach of the endoscope and for removal of many gastric and nearly all small bowel foreign bodies. Surgery is necessary for the definitive diagnosis and treatment of pyloric stenosis. It is very helpful for the diagnosis of pancreatitis, especially in the cat. It allows visualization and biopsies of multiple organs, including the stomach, small intestine, liver and pancreas. No vomiting (or diarrhoeic) animal should undergo an exploratory surgery without biopsy of the stomach and intestines; performing endoscopy after an exploratory surgery performed without biopsy is an absurd diagnostic approach.

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11 A foreign body in the small intestine of a dog

INITIAL PRESENTATION

Vomiting and poor appetite

Signalment: 5-year-old male English springer spaniel, body weight 19.9 kg

CASE HISTORY

The dog had been healthy until 2 weeks prior to admission. He was de-wormed regularly with fenbendazole and vaccinated yearly. Two weeks prior to admission he had started vomiting and had a progressive decrease in appetite. He was now vomiting 2 to 3 times per day and the vomitus included food and bile but no blood. The dog's stools had been normal. The owner estimated that the dog had lost about 1 to 2 kg over this time period. The dog's water intake and urine output had not changed, but his exercise tolerance had decreased.

His usual diet was a mixture of dry and canned dog food and recently the owners had been hand feeding him with chicken and ham to encourage food intake. He was normally fed twice a day.

PHYSICAL EXAMINATION

The dog was dull but responsive. His body condition score was 4/9. There was evidence of recent weight loss, with signs of muscle and fat loss over the lumbar and flank areas. Mucous membranes were pink but slightly tacky; capillary refill time was less than 2 seconds. Peripheral lymph nodes felt normal.

Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 140 beats per minute and a respiratory rate of 24 breaths per minute.

There was no evidence of pain on abdominal palpation and the only abnormality found was increased gas in the small intestines. Rectal temperature was 39.1° C.

His prior treatment included clavulanate-potentiated amoxicillin, ranitidine and sucralfate (doses unknown), with no improvement in clinical signs. He was on no other medications or supplements.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting
- Tacky mucous membrane
- Poor appetite
- Weight loss
- Increased intestinal gas
- Raised body temperature

DIFFERENTIAL DIAGNOSIS

Vomiting is the primary complaint and the other problems were thought to be related to the vomiting. The differential diagnoses for the vomiting in this dog include:

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilation-volvulus
  - neoplasia
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease
  - neoplasia
  - parasites
  - intussusception (unlikely)

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- Disorders of the large intestine
  - colitis
  - obstipation
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism
  - diabetes mellitus
  - liver disorders
  - peritonitis
  - renal disease/uraemia (unlikely)
- Dietary causes
  - dietary sensitivity
  - dietary indiscretion

Most dogs (80%) with intussusception are less than 1 year of age, although it was still a possibility. He did not have any other signs of renal disease, so uraemia was an unlikely cause of the vomiting.

The slightly tacky membranes were likely indicative of mild dehydration. The dog had not drunk water that morning, had a 2-hour journey to the clinic and was also probably losing fluid in his vomitus.

CASE WORK-UP

The dog was admitted to the hospital and administered intravenous crystalloid fluids containing potassium, at a rate to correct 6% dehydration.

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed. Haematology results were unremarkable, although a packed cell volume of 0.47 l/l (reference range 0.37–0.55 l/l) is high in breeds of dogs other than sighthounds. This was thought to likely be due to haemoconcentration.

Serum chemistry showed an increase in liver enzymes, with alanine aminotransferase (ALT) at 1100 IU/l (reference range 21–102 IU/l) and alkaline phosphatase at 114 IU/l (reference range 20–60 IU/l). Serum chloride and sodium were decreased at 84 mmol/l (reference range 99–115 mmol/l) and 137 mmol/l (reference range 139–154 mmol/l), respectively, and potassium was at the bottom end of the reference range at 3.6 mmol/l (reference range 3.6–5.6 mmol/l). A basal cortisol was performed and was within the reference range at 97 nmol/l (reference range 20–230 nmol/l). If basal cortisol is greater than 70 nmol/l, hypoadrenocorticism is unlikely.

Serum amylase and lipase were not elevated, making a pancreatopathy less likely, although a test for canine pancreatic lipase immunoreactivity was not performed.

Urinalysis was unremarkable. The urine specific gravity was 1.048, consistent with good renal concentrating ability and also with the mild dehydration detected clinically.

Serum albumin, urea and creatinine can also be increased due to dehydration, although they were within the reference ranges in this dog.

Imaging

Barium impregnated polyethylene spheres (BIPS) were administered to the dog as an intestinal obstruction was considered to be likely. BIPS aid in diagnosing partial obstructions as the large (9 mm) BIPS will persistently bunch at the site of an obstruction.

Ventrodorsal and lateral abdominal radiographs were taken about 8 hours later. These showed bunching of the BIPS and very large gas filled loops of small intestine (Figs 11.1 and 11.2). Some areas of the intestine were greater than twice the height of the vertebral body of L5 in width. This pattern is highly suggestive of small intestinal obstruction, so the dog was sent for an exploratory laparotomy.

SURGERY

At surgery the intestines were dilated with gas and a foreign body was located within the jejunum (Fig 11.3). A peach stone was removed from the mid-jejunal area. Closure was routine and omentum was wrapped around the enterotomy site. Recovery was uneventful and a postoperative antibiotic (clavulanate-potentiated amoxicillin, initially at 20 mg/kg iv q 8 hours for the first day, then 19 mg/kg po q 12 hours for 4 days) was administered. Buprenorphine (20 μg/kg iv q 6 hours) was used for postoperative analgesia for the first day.

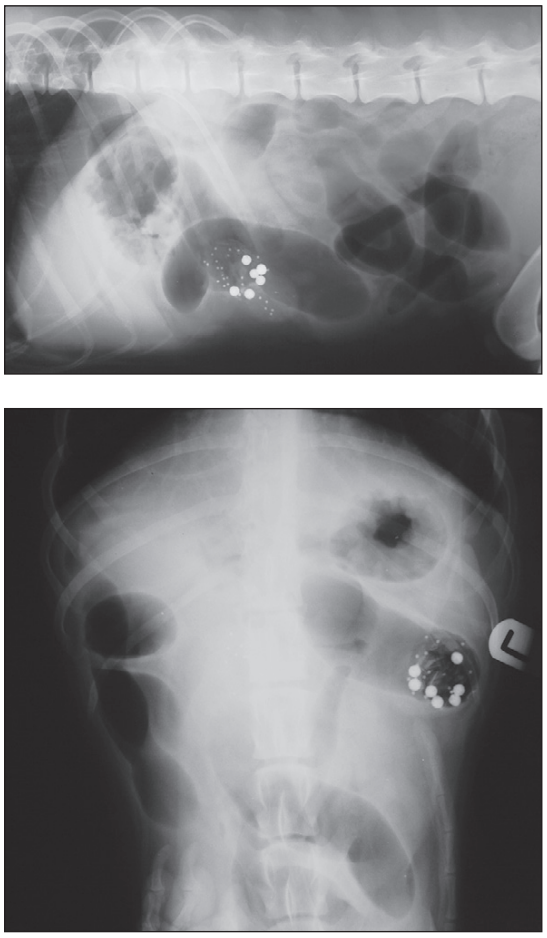
Follow-up

Five days postsurgery the serum chemistry values had improved with the ALT decreased to 290 IU/l and the AP decreased to 244 IU/l. Electrolytes were within the reference ranges. The dog had begun to eat, rectal temperature was normal; he was not vomiting and was discharged with no further medical treatment.

DISCUSSION

The radiographs were highly suggestive of the presence of an intestinal foreign body in this dog. The use of the

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Figs 11.1 and 11.2 Lateral and ventrodorsal radiographs showing grouping of BIPS and grossly dilated intestines characteristic of intestinal obstruction (courtesy of Dr Tobias Schwarz)

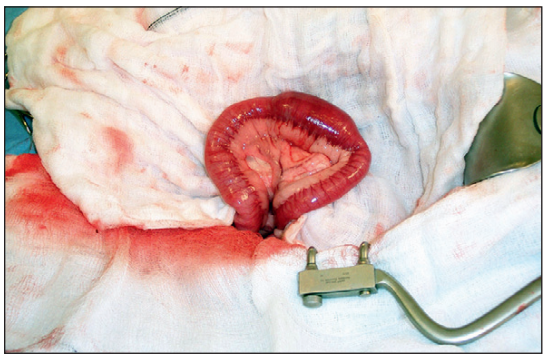


Fig 11.3 Small intestinal foreign body (courtesy of Dr Donald Yool)

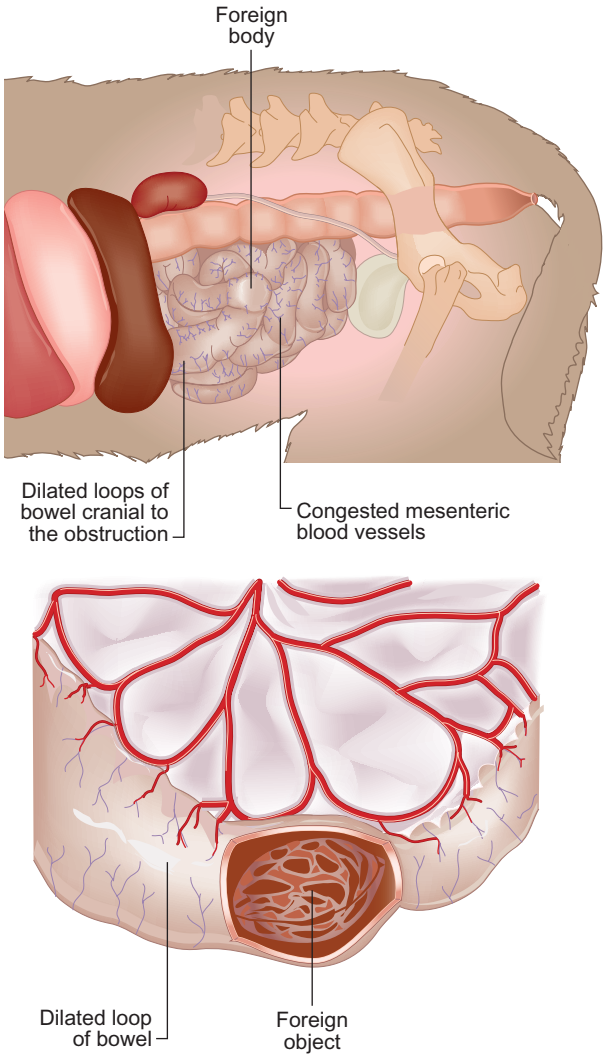


Fig 11.4 An intestinal foreign body causes dilatation of the bowel cranial to the obstruction, sometimes termed a 'stagnant' loop of bowel

BIPS added to the interpretation of the radiographs, although it would likely to have been possible to make the diagnosis without them. Peach pits are a fairly common canine intestinal foreign body and are not usually clearly radiopaque on plain films.

The low serum sodium and chloride and borderline potassium are consistent with intestinal obstruction and may be due to loss in the vomitus and/or sequestration within the intestines. Due to the dilation of the bowel cranial to the obstruction and the congestion of mesenteric blood vessels (Fig 11.4), after about 24 hours of obstruction the intestinal mucosa cranial to the

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obstruction secretes instead of absorbing fluids, although it is still able to absorb nutrients.

Colloids may have been a better fluid choice as they support oncotic pressure more effectively in dogs with intestinal obstruction than do crystalloid fluids, although there is no evidence that the use of colloids increases survival rates.

Bacterial numbers increase in obstructed intestinal segments and can result in endotoxic shock. This dog did not appear to be in shock, but he did have an elevated body temperature. The use of antibiotics is indicated in obstructed animals to decrease the bacterial load and help prevent bacterial intestinal translocation and endotoxaemia.

Epidemiology

In one study, English bull terriers, springer spaniels, Staffordshire bull terriers, Border collies and Jack Russell terriers were over-represented in presenting for gastrointestinal foreign bodies. In dogs, 63% of obstructions occurred in the jejunum but foreign objects were encountered at all points along the gastrointestinal tract.

Prognosis

A longer duration of clinical signs, the presence of a linear foreign body and multiple intestinal procedures are associated with significantly increased mortality. Neither the degree of obstruction (partial or complete) nor the location of the foreign body has been shown to have a significant influence on survival. In a study in cats, those without preoperative perforation from a foreign body have an 85% chance of survival while those with preoperative perforation have only a 50% chance of survival.

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12 Chronic partial gastric dilatation in a dog

INITIAL PRESENTATION

Vomiting

Signalment: 8-year-old male entire Labrador, body weight 24 kg

CASE HISTORY

The dog's previous history included arthritis, for which he was fed a diet designed for dogs with arthritis and was previously given the non-steroidal anti-inflammatory medication, meloxicam. He had been off the meloxicam for 3 weeks at admission.

The dog had become lethargic during the past month and was vomiting food and brown foam several times a day. He was also occasionally retching. His faeces were soft to watery. The owner had seen the dog in the 'praying position' several times during the last week. In this position the dog's head is down and front legs stretch forward. This usually occurs during episodes of abdominal pain. Prior to this he had had intermittent episodes of vomiting and diarrhoea every few weeks. He had lost about 3 kg during the past month.

The owner reported that during exercise the dog's breathing pattern had become more laboured and he was not as willing to exercise as he previously had been.

The dog was de-wormed regularly with fenbendazole, although the owner reported that he was 3 weeks overdue for de-worming. He was vaccinated yearly.

His current medication was cimetidine (200 mg po q 8 hours), which had not improved his condition.

PHYSICAL EXAMINATION

The dog was bright and responsive. His body condition score was 4/9. Hydration appeared to be adequate. Mucous membrane colour was pink and capillary refill time was less than 2 seconds. Peripheral lymph nodes were unremarkable.

Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 100 beats per minute. The respiratory rate could not be counted as the dog was panting. There was evidence of mild to moderate pain or discomfort on abdominal palpation. While this caused difficulty in performing a thorough abdominal palpation, no other abnormalities were detected. Rectal temperature was 38.9° C.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting
- Diarrhoea
- Decreased exercise tolerance
- Abdominal discomfort
- Possibly respiratory distress

DIFFERENTIAL DIAGNOSIS

Vomiting was the dog's main problem and it was the one initially addressed. Differential diagnoses for vomiting in this dog include the following disorders:

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilatation-volvulus
  - neoplasia
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease

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- neoplasia
- parasites
- intussusception (less likely due to the dog's age)

- Disorders of the large intestine
  - colitis
  - obstipation
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism
  - diabetes mellitus
  - liver disorders
  - peritonitis
  - renal disease/uraemia (unlikely)
- Dietary causes
  - adverse reaction to food (allergy or intolerance)
  - dietary indiscretion

Most dogs with intussusception are less than 1 year of age, although it was still a possibility. He did not have any other signs of renal disease such as polyuria or polydipsia, so uraemia was an unlikely cause of the vomiting.

CASE WORK-UP

The dog was admitted to the hospital for diagnostic investigation.

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed. Haematology results were unremarkable. Serum chemistry results including amylase, lipase and basal cortisol were within the reference range other than a mild elevation in bile acid concentrations to 19.4 μmol/l (fasting reference range 0–7 μmol/l).

A canine pancreatic lipase immunoreactivity test was within the reference ranges, as were serum trypsin-like immunoreactivity and folate. Serum cobalamin was slightly decreased at 268 ng/l (reference range 275–590 ng/l).

Urinalysis, including chemical strip and sediment, was unremarkable. The urine specific gravity was 1.037.

Imaging

Abdominal ultrasound showed decreased gastric motility and the pylorus and duodenum were shifted further towards the midline than normal. All other abdominal organs were considered normal. Contrast radiography was recommended.



Fig 12.1 Ventrodorsal abdominal radiograph with barium contrast showing the abnormal (medial) displacement of the pylorus (courtesy of Dr Tobias Schwarz)

Abdominal radiographs showed gas within the gastric fundus, which in the ventrodorsal (VD) view extended across the pylorus. Some gas was also seen in the proximal descending duodenum. The small intestines contained fluid and gas, but appeared to be normal in diameter.

Liquid barium was given to enhance visualization of the gastrointestinal tract. At 5 minutes post barium, a mixture of barium and particulate material was seen within the gas-filled fundus, with most of the barium pooling within the antrum, which appear rounded, dilated and medially displaced. On the VD view the angle between the fundus and the antrum was more acute than normal with the antrum moved toward the midline (Fig 12.1). On both views the pyloric region of the stomach appeared to terminate abruptly (Fig 12.2), with a narrow band of contrast present for several centimetres before the barium began to fill the duodenum.

At 30 minutes post barium, most of the barium had passed into the lower small intestine; however, the antrum still appeared to have an abnormal location.

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Fig 12.2 Lateral abdominal radiograph with barium contrast showing the narrow band of contrast present for several centimetres before the barium began to fill the duodenum (courtesy of Dr Tobias Schwarz)

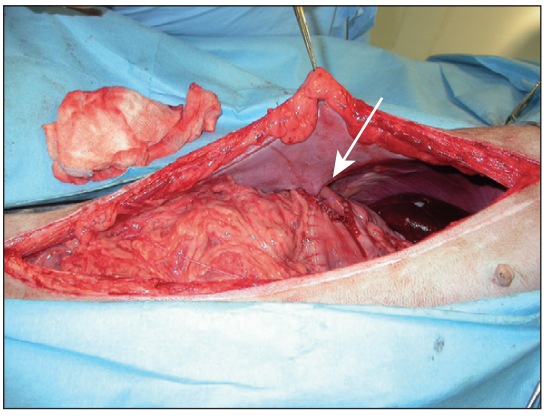


Fig 12.3 Belt loop gastropexy in place (arrow) (courtesy of Dr Donald Yool)

At 2 hours post barium, some barium was still retained within the stomach, predominantly in the antrum and pylorus. The antrum appeared less rounded and had a less acute angle with the lesser curvature than previously. The filling of the proximal duodenum remained incomplete with a narrow band of contrast in it.

Gastrointestinal endoscopy was attempted. The endoscopist had unusual difficulty passing the endoscope through the lower oesophageal sphincter into the stomach. The endoscopic view of the stomach was initially abnormal with the landmarks of the stomach (e.g. lesser curvature, antrum) appearing to be in the wrong place. During the endoscopy the stomach changed shape and landmarks appeared in the correct places. The duodenum was unremarkable. Routine biopsy samples were taken of the stomach and duodenum.

CASE ASSESSMENT

From the history, imaging and endoscopy it appeared that the dog had a chronic dynamic partial gastric torsion which was occurring intermittently. Exploratory surgery was scheduled.

Surgical management

At surgery a partial gastric torsion was identified. A belt loop gastropexy was performed to prevent reoccurrence or progression to gastric dilatation volvulus (Fig 12.3). Further full thickness biopsies were taken of the stomach and jejunum.

Histopathology

Histopathology of the biopsies showed that the stomach and duodenum were normal. The histopathology of the jejunum was diagnosed as mild, diffuse, chronic jejunitis.

Follow-up

The dog recovered well from the surgery and at 1 month was exercising at nearly normal levels. He was eating well and not vomiting.

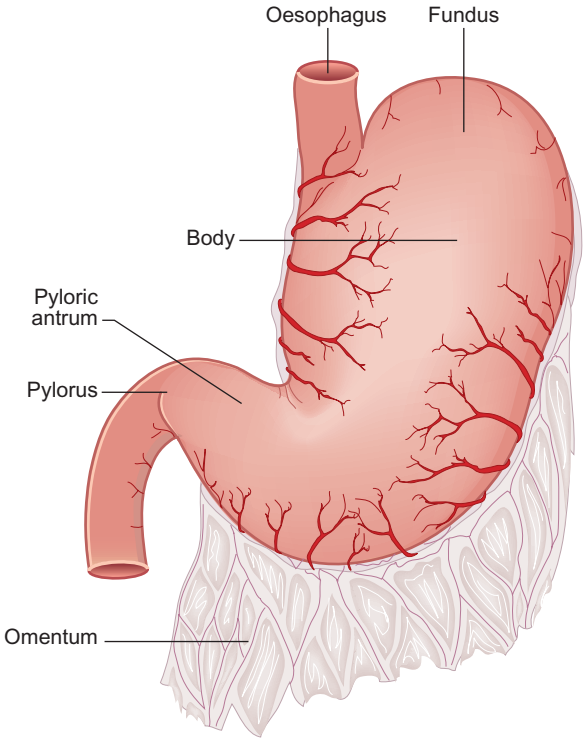
Medical management

While the surgery should prevent the stomach from torsing, some cases do continue to have episodes of gastric dilatation. Recommendations for these cases include the frequent feeding of small, low carbohydrate, low fat, high protein meals and the use of prokinetic drugs such as metoclopramide, cisapride or low dose erythromycin (0.5–1.0 mg/kg po q 8 hours, in between meals). In dogs, erythromycin at these low doses stimulates motilin release and promotes the interdigestive 'housekeeping' wave, promoting emptying of solids. Products containing peppermint have a mild prokinetic effect and may be useful. Anti-foaming agents such as dimethacone or simethacone have been shown to be of benefit in some animals.

CASE DISCUSSION AND EPIDEMIOLOGY

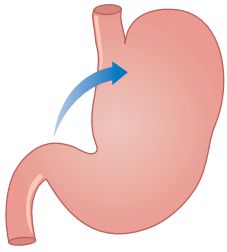
Dogs with chronic gastric distension and radiographic evidence of a twisted stomach may have a chronic intermittent form of gastric volvulus which usually corrects

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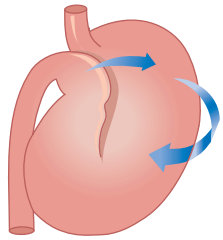


Sequence of gastric dilation with torsion

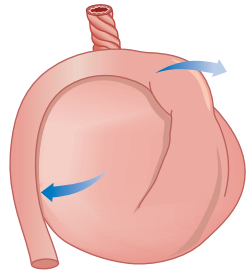
Clockwise rotation as viewed from a ventral position.



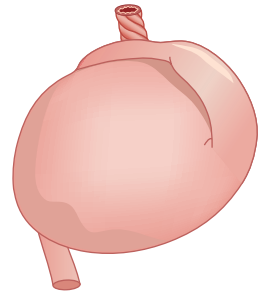
The pyloric antrum is displaced downward.



The pylorus crosses the midline, passes underneath the distended proximal part of stomach, and moves upward along the left abdominal wall.



The gastric fundus moves ventrally and becomes located in the ventral abdomen.



The continuing gastric dilation displaces the greater curvature ventrally.

Fig 12.4 An illustration of how the pylorus crosses the midline in a gastric torsion, causing the pylorus to be closed off and resulting in increased gas and fluid in the stomach

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spontaneously. The radiographic appearance is of the pylorus moving too close to the midline as the stomach fills with air. It is possible for this to progress to a gastric dilatation volvulus with torsion (Fig 12.4). The cause is unknown, but may be due to an impaired ability to eructate and possibly delayed gastric emptying.

Eructation should be initiated by gaseous distension of the stomach (especially the cardia) and is mediated by receptors in the cardia and vagal afferents to the brain. The response is a reduction in the pressure at the cardia and lower oesophageal junction to less than that of the rest of the stomach so that the gas can escape. Defective eructation could be due to a defect in the junction sphincter in the reflex, e.g. a disorder of the vagal innervation or a deficit in the sensory function of the cardia. The gastric cardia may be damaged by over-distension, so that dogs which have had gastric dilatation may be prone to recurrent episodes.

Gastric gas is usually from swallowed air, the reactions of gastric acid with secretions containing bicarbonate or from bacterial fermentation. Rapid eating and drinking increases the amount of swallowed air; nervous, excited or dysphagic (e.g. oesophageal motility disorders) animals are more likely to swallow air. Animals with respiratory distress or hyperventilation also tend to swallow air.

Composition of the diet has been suggested to increase the incidence of gastric dilatation; however, this has not been proven and gastric dilatation volvulus has been reported in dogs fed fresh meat, soy-based or cereal-based diets. The intake of a large amount of food rapidly will cause gastric dilatation and the stomach takes longer to empty. It is recommended that dogs at risk for gastric dilatation or that have a history of dilatation be fed several smaller meals per day instead of one large one.

Large breed deep-chested breeds of dogs appear to be at increased risk, but no age or sex predilection has been recognized.

Prognosis

Resolution of most of the clinical signs often follows gastropexy and management of gastric dilatation. Some animals continue to bloat, but with gastropexy the stomach should not be able to twist, so that overall the prognosis is good.

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13 Pancreatitis and alimentary lymphoma in a dog

INITIAL PRESENTATION

Vomiting

Signalment: 5-year-old male entire Beagle, body weight 12 kg

CASE HISTORY

The dog presented with a week long history of vomiting and decreased appetite. The vomitus contained food and bile and occurred at least once a day. The frequency of vomiting had increased over the past month. He had had a similar episode 5 months earlier which had responded to treatment with oral electrolyte solutions and antibiotics.

The owners thought that he had lost weight, but were unsure of how much. He was lethargic, although the owners reported that he was usually a quiet dog. His stools had been soft and somewhat dark in colour during the last couple of weeks, although also scant as he had not been eating well.

The dog had last been de-wormed 3 months previously, but had not been vaccinated for about 3 years. His previous diet was a dry food bought at the grocery store, plus snacks and treats of human food. He was being tempted to eat small amounts of chicken and ham at presentation. He was on no current medication.

PHYSICAL EXAMINATION

The dog's demeanour was dull but responsive. He was estimated to be about 6% dehydrated. His body condition score was good at 5/9, although there was evidence of muscle loss over his epaxial, lumbar and temporal muscles. Mucous membrane colour was pink and capillary refill time was less than 2 seconds. Peripheral lymph nodes were unremarkable.

Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 96 beats per minute and respiratory rate of 16 breaths per minute. There was evidence of mild to moderate pain on palpation of the cranial abdomen. Rectal temperature was decreased at 36.7° C.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting
- Abdominal discomfort
- Low rectal temperature

DIFFERENTIAL DIAGNOSIS

Vomiting was the dog's main problem and it was the one initially addressed. Differential diagnoses for vomiting in this dog include the following disorders:

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilatation-volvulus
  - neoplasia
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease (IBD)
  - neoplasia
  - parasites
  - intussusception (unlikely)
- Disorders of the large intestine
  - colitis
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism
  - diabetes mellitus

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- liver disorders
- peritonitis
- renal disease/uraemia (unlikely)

- Dietary causes
  - adverse reaction to food (allergy or intolerance)
  - dietary indiscretion

Most dogs with intussusception are less than 1 year of age, although it was still a possibility in a dog of this age. He did not have any other signs of renal disease, so uraemia was an unlikely cause of the vomiting.

Differential diagnoses for cranial abdominal pain in this dog included:

- Pancreatitis
- Gastritis
- Hepatopathy
- Peritonitis
- Pyelonephritis was unlikely as the pain was cranial and not over the area of the kidneys
- Occasionally back pain and abdominal pain may be difficult to differentiate, but this dog showed very localized pain which appeared unlikely to be coming from the spine or epaxial muscles

Low rectal temperature may be due to decreased ambient temperature, (which was not the case here) shock or other disorders of peripheral circulation, hypothyroidism, or to placement of the thermometer within a faecal ball in the rectum. One of the latter two differential diagnoses was thought to be most likely in this case.

CASE WORK-UP

The dog was admitted to the hospital for diagnostic investigation and fluid therapy to correct the dehydration.

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed. Haematology results showed an elevation in white blood cell with a neutrophil count of 23.5×10^9/l (reference range $3.6–12.0 \times 10^9$/l), band or meta neutrophils of 1.4×10^9/l (reference range 0 for this laboratory) and monocytes of 2.24×10^9/l 9 (reference range $0–1.5 \times 10^9$/l). His packed cell volume (PCV) was 0.54 l/l, within the reference range of 0.39 to 0.55 l/l, but high for a dog not of the sighthound breeds.

CLINICAL TIP ON PCVS

While the upper end of the range of PCV for most laboratories is above 0.50 l/l, the upper end of the reference range is more typically seen in greyhounds and other sighthounds. When values above 0.50 l/l are seen in other breeds, haemoconcentration should be considered, which is often due to dehydration. Values above 0.60 l/l may even be due to secondary polycythaemia or polycythaemia vera.

Serum chemistry results showed a decreased albumin of 21.9 g/l (reference range 26–35 g/l) and a globulin in the low end of the reference range at 19 g/l (reference range 18–37 g/l). Serum urea was mildly increased at 7.6 mmol/l (reference range 1.7–7.4 mmol/l) and sodium was decreased at 135 mmol/l (reference range 139–154 mmol/l) with potassium within the reference range at 4.0 mmol/l. Cholesterol was decreased at 3.1 mmol/l (3.8–7.0 mmol/l) and triglycerides were within the reference range at 0.73 mmol/l (range 0.57–1.14 mmol/l). Lipase was extremely increased at 2330 IU/l (reference range 13–200 IU/l), although amylase was within the reference range at 22.1 µmol/l (reference range 15–26 µmol/l). A canine-specific lipase was run to confirm this and was also highly elevated at 998 mmol/l (reference range less than 200 mmol/l).

Basal serum cortisol ruled out hypoadrenocorticism with a value of 202 nmol/l.

A serum thyroxine (total T4) concentration was decreased at 8 nmol/l (reference range 15–48 nmol/l).

Urinalysis was unremarkable on chemical strip and sediment and the specific gravity of urine was 1.047.

CLINICAL TIP

Serum amylase and lipase may reflect pancreatitis in the dog, although specificity of these tests is only about 50%. Serum amylase is particularly undiagnostic, as noticed in this case where the value was within the reference range in a severe case of pancreatitis. Serum amylase and lipase should be used only as preliminary screening tests if the more reliable specific canine pancreatic lipase immunoreactivity (cPLI) test is not immediately available. There is also an in-house qualitative kit form of the cPLI test. The sensitivity of the laboratory-based cPLI is above 80% and it is also highly specific.

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Imaging

Abdominal ultrasound showed an enlarged irregular pancreas with focal fluid accumulation and a corrugated appearance to the duodenum. The small intestine was considered to be otherwise normal in width and layering. There was no evidence of peristalsis in the proximal small intestine.

DIAGNOSIS

From the history, clinicopathological parameters and ultrasound results, a diagnosis of pancreatitis was made.

Medical treatment

Initial treatment included intravenous balanced crystalloid fluid therapy at a rate calculated to correct dehydration within 24 hours, intravenous clavulanate-potentiated amoxicillin (20 mg/kg iv q 8 hours), buprenorphine (20 µg/kg q 6 hours) for analgesia, ranitidine (2 mg/kg subcutaneous q 12 hours), metoclopramide in a constant rate infusion (1 mg/kg constant rate infusion in 24 hours in iv fluids) and sucralfate (1.5 ml per os (po) q 8 hours). The sucralfate had to be discontinued as the dog could not take anything po without vomiting.

The total T4 was decreased in this dog, which could have been due to hypothyroidism or to the euthyroid sick syndrome. It was planned to investigate this further after treatment of the more severe disorders. While the dog was lethargic and had a low rectal temperature, he did not have alopecia, high cholesterol, hypertriglyceridaemia or (even after rehydration) anaemia.

CLINICAL TIP ON ANTI-EMETICS

Oral or subcutaneously administered metoclopramide has a relatively short half life and it is more efficacious if given as a constant rate infusion. It may adversely affect pancreatic blood flow as it is a dopamine agonist and the blood flow to the pancreas is regulated via dopamine receptors. Maropitant, a neurokinin 1 antagonist with broad spectrum actions, may have been a better choice in this dog, although it was not yet available at the time this case presented. It does not have the promotility effects of metoclopramide, which were desired in this case. Another anti-emetic that may be useful in pancreatitis is dolasetron which is a 5HT antagonist and a very potent anti-emetic (but which was also not available at the time of this case).

CLINICAL TIP ON ANTIBIOTIC USE IN PANCREATITIS

There is much debate about whether or not to use antibiotics in cases of canine pancreatitis. In humans, infection is a common complication of pancreatitis, although there is limited evidence that human patients treated with prophylactic antibiotics have a better outcome. While most clinicians treat cats with pancreatitis with antibiotics, the decision is more difficult in dogs, as infection is thought to be less likely. In this case (rightly or wrongly) the decision was made to use an antibiotic, due to the presence of a left shift or increase in band neutrophils.

NURSING ASPECTS OF FEEDING IN CASES OF PANCREATITIS

It has previously been recommended to withhold food from patients with pancreatitis, which is sensible in patients that are vomiting. In humans there is no evidence that withholding nutritional supplementation is beneficial and even some that it may be detrimental. Current thinking is to withhold food only from dogs which are vomiting and then only for 2 to 4 days. If anti-emetics do not control vomiting, then parenteral support (e.g. partial parenteral nutrition via a peripheral vein) or enteral support via a jejunostomy tube are recommended. Parenteral nutrition has the advantage of not requiring anaesthesia for placement as does a jejunostomy tube. In dogs that are not vomiting, small amounts of low fat and moderate carbohydrate foods may be offered in small frequent meals. There is no scientific evidence that a high fat meal causes pancreatitis, but there is anecdotal evidence that the dogs appear to do better if offered low-fat foods during recovery. Feeding low-fat food also appears to cause resolution of signs in some chronic low-grade cases of pancreatopathy.

After 2 days of treatment, the dog's clinical signs had waxed and waned, but overall had failed to respond and he was still depressed and vomiting. Repeat haematology and serum chemistry was performed. This showed an improvement, in although still elevated, serum lipase

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to 875 IU/l (reference range 13–200 IU/l), but a fall in the albumin to 15.8 g/l (reference range 27–35 g/l) and also a decrease in globulin to 17.8 g/l (reference range 18–37 g/l). Serum urea was now within the reference range at 5.4 mol/l (reference range 1.7–7.4 mol/l) and PCV had decreased to 0.48 l/l. Urine protein to creatinine ratio was 0.3, effectively ruling out a protein losing nephropathy. Liver function tests other than albumin (e.g. serum bile acids and bilirubin) as well as liver enzymes were within reference ranges, making a hepatopathy unlikely.

CLINICAL TIP ON THE CLINICOPATHOLOGICAL EFFECT OF DEHYDRATION

Dehydration affects many clinicopathological parameters, notably increasing PCV, haemoglobin, urea, creatinine (prerenal azotemia), albumin, globulins and sodium. This can increase some parameters above the reference range, such as urea and creatinine; however, it can also mask low values for others, such as proteins and PCV. These parameters, especially if in the lower end of the reference range, should be re-evaluated after rehydration.

A gastrointestinal disorder in addition to the pancreatitis was suspected and gastrointestinal endoscopy was performed.

Endoscopy

At endoscopy, the stomach had thickening of the rugal folds (Fig 13.1) and the small intestinal mucosa was very roughened and abnormal in appearance with some bleeding present (Fig 13.2). Mucosal biopsies were taken of the stomach and small intestine.

Histopathology

Histopathology of the stomach showed oedema and congestion of the lamina propria, some focal haemorrhage and areas of erosions with underlying inflammation. In the gastric cardia, biopsies showed extensive fibrosis and atrophy of the gastric glands. This was interpreted as moderate to severe inflammatory gastritis.

The duodenum had severe diffuse and nodular infiltration of the lamina propria with immature, noncleaved lymphocytes. There was widespread villus blunting and atrophy. This was interpreted as alimentary lymphoma.

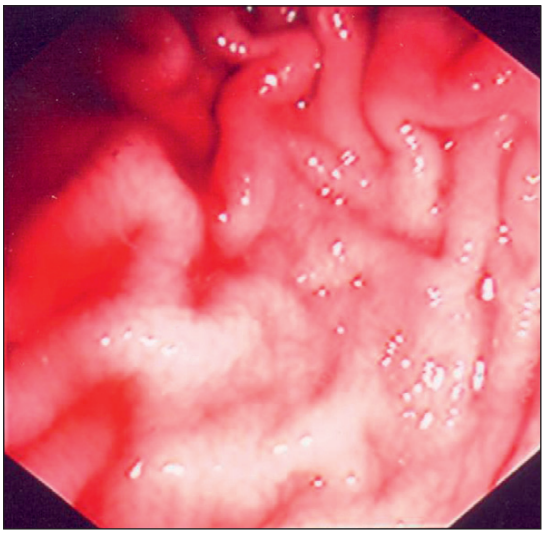


Fig 13.1 Endoscopic view of the stomach, showing lack of distension and a roughened appearance of the rugal folds

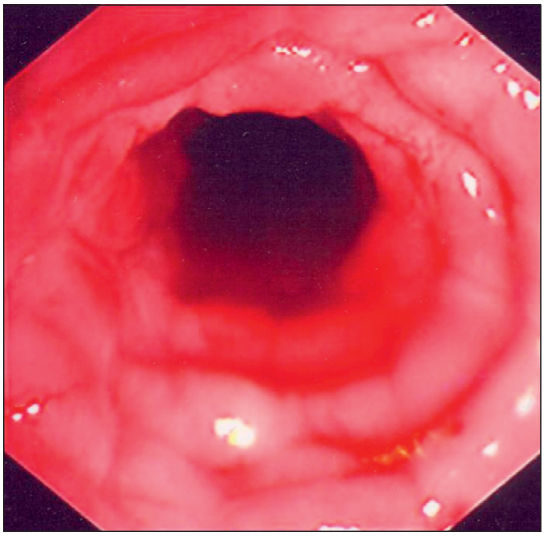


Fig 13.2 Endoscopic view of roughened small intestinal mucosa, with bleeding present

Follow-up

As these owners did not want to pursue therapy, the dog underwent euthanasia. A necropsy confirmed extensive infiltration of small lymphocytes within the villi, lamina propria and extending below the villi and into the submucosa in some areas. The mesenteric lymph nodes also showed effacement in flat fields of small

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lymphocytes. The subcapsular sinus of the lymph nodes was also heavily infiltrated with small lymphocytes.

The thyroid glands had atrophied follicles with nearly complete absence of colloid, but no inflammatory cells present. It appeared that the dog was hypothyroid due to idiopathic follicular atrophy rather than active lymphoid thyroiditis; in this diagnosis it is unclear whether the follicular atrophy is a distinct syndrome or the final result of previous lymphoid thyroiditis.

CASE DISCUSSION AND EPIDEMIOLOGY

This case demonstrates the pitfall of assuming that there is only one aetiology for a patient's clinical signs, in that he had more than one cause for his vomiting. As neither pancreatitis nor hypothyroidism are known risk factors for intestinal lymphoma, it is likely these diseases occurred independently in this unlucky dog. His pancreatitis was resolving and the hypothyroidism was treatable, so the final diagnosis is the intestinal lymphoma.

Alimentary lymphoma constitutes 7% of canine lymphoma cases and can occur diffusely as in this dog or in a more localized form. Signs usually include anorexia, vomiting, diarrhoea, weight loss and pyrexia; several of which were present in this case. The main differential is IBD and often full thickness biopsies, sometimes using immunohistochemistry, are necessary to differentiate between the two.

Other possible techniques for distinguishing the two may include flow cytometry and assessment of T-cell clonality by PCR. Primary canine gastrointestinal lymphoma is usually of T-cell phenotype. Hypoproteinaemia and hyperbilrubinaemia are more common in lymphoma than in IBD, but this is not consistent. It also appears that some cases of lymphocytic plasmacytic enteritis can eventually mutate into lymphoma.

There appears to be no age or sex predilection for the disorder. In one study, about half (23) of 44 dogs with gastrointestinal lymphoma were female, with 11 intact and 12 neutered: 21 of the dogs were male, with 12 intact and nine neutered. Sixteen breeds as well as individuals of mixed breeding were represented. The Boxer and the Chinese Shar-Pei were the most commonly represented breeds with six individuals each. The age range of the dogs was 1.5 to 14.6 years.

Treatment for focal lymphoma is usually by surgical resection, but this is not possible in the more generalized or diffuse form that this dog had. Dogs with this form respond poorly to chemotherapy and there is a risk of intestinal perforation. Chemotherapy with cyclophosphamide, vincristine and prednisolone (and sometimes doxorubricin) has been tried in dogs with diffuse alimentary lymphoma, but this form of lymphoma is more refractory to treatment than other forms in the dog. Response rates of 30% to 60% with a medium remission time of 4 to 8 months have been reported.

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14 Hypoadrenocorticism in a dog

INITIAL PRESENTATION

Vomiting, weakness and lethargy

Signalment: 6-year-old neutered female cocker spaniel, body weight 17 kg

CASE HISTORY

The dog had become lethargic during the past month and was vomiting digested food 3 to 4 times a week. Her exercise tolerance had decreased over the last 1 to 2 months. Her stools varied between normal and soft. The owner was unsure if the dog's drinking and urination patterns had changed.

The dog was de-wormed regularly with fenbendazole and she was vaccinated yearly.

Her usual diet was a high quality dry food and she was also given snacks and treats regularly. She was on no medications or supplements.

PHYSICAL EXAMINATION

The dog was very depressed at presentation. Her body condition score was 4/9 with evidence of recent weight loss. Her mucous membranes were tacky; capillary refill time was 3 seconds. Peripheral pulse quality was weak, but there was a pulse for every heart beat. There was a slight loss of skin elasticity. Based on these findings, she was estimated to be approximately 7% dehydrated.

Peripheral lymph nodes were unremarkable. Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 56 beats per minute (bpm). This heart rate was thought to be too slow for the degree of dehydration. She had a respiratory rate of 36 breaths per minute and her rectal temperature was 38.9° C.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting
- Decreased exercise tolerance
- Slow heart rate, especially for hypovolaemia

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for vomiting in this dog include the following disorders:

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilatation-volvulus
  - neoplasia
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease
  - neoplasia
  - parasites
  - intussusception (unlikely)
- Disorders of the large intestine
  - colitis
  - obstipation
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism

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  - diabetes mellitus
  - liver disorders
  - peritonitis
  - renal disease/uraemia (unlikely)
- Dietary causes
  - adverse reaction to food (allergy or intolerance)
  - dietary indiscretion

Most dogs with intussusception are less than 1 year of age, although it is still a possible diagnosis in a dog of this age.

Differential diagnoses for decreased exercise tolerance in this dog include:

- Cardiovascular disease
- Neuromuscular disorders such as myasthenia gravis, peripheral neuropathies, brain or spinal cord disease, myopathies
- Respiratory disorders
- Electrolyte disorders, e.g. hypo- or hyperkalaemia, hypo- or hypernatraemia, hypocalcaemia
- Endocrine diseases such as diabetes mellitus, hypothyroidism, hypoglycaemia, hypoadrenocorticism
- Infectious disease
- Anaemia
- Nutritional deficiencies or malassimilation of nutrients
- Neoplasia
- Medications or toxins; however, the dog had no history of ingestion of these
- Pain – although there was no evidence of pain on physical examination, this can be subtle

A heart rate of 56 bpm is considered bradycardic for a medium sized dog and the degree of hypovolaemia would be expected to have increased the rate. Possible causes for the bradycardia include:

- increased vagal tone
- hypothyroidism
- sinus node disease (sick sinus syndrome)
- sinus block due to atrial disease
- atrial standstill (e.g. due to hyperkalaemia) or an atrioventricular heart block

Very fit dogs sometimes have a slow heart rate, but this dog did not appear fit and also had not been exercising normally for the past month or longer.

CASE WORK-UP

The dog was admitted to the hospital for diagnostic investigation and treatment of her dehydration. Intravenous balanced electrolyte crystalloid fluids were initially started at 85 ml/hour, calculated to correct her dehydration over 24 hours.

NURSING TIP ON INTRAVENOUS FLUID THERAPY

Fluid therapy needs to take into account the amount needed for maintaining normal hydration and replenishing the amount lost. Maintenance fluids are usually calculated at 40 to 60 ml/kg per 24 hours or 1.6 to 2.5 ml/kg/hour. In this 17 kg dog, that was approximately 34 ml/hour. To replete the amount of fluid deficit, the dog's body weight is multiplied times the estimated amount of dehydration, e.g. 17 × 0.07 = 1.19 kg or 1.19 l in 24 hours. This is 1190 ml in 24 hours or about 50 ml/hour. Adding the 34 ml plus 50 ml is 84 ml/hour. The pet's hydration should be assessed several times during fluid administration as sometimes hydration improves before this total amount is given. If the pet is vomiting or has diarrhoea, sometimes additional fluid needs to be given to make up these ongoing losses.

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed. Haematology results showed an increased packed cell volume (PCV) of 0.56 l/l (reference range 0.37–0.55 l/l).

CLINICAL TIP ON PCVs

While the upper end of the range of PCV for most laboratories is above 0.50 l/l, the upper end of the reference range is more typically seen in greyhounds and other sighthounds. When values above 0.50 l/l are seen in other breeds, haemoconcentration should be considered, which is often due to dehydration. Values above 0.60 l/l may even be due to polycythaemia.

Serum chemistry results including basal cortisol were performed and showed a marked hyperkalaemia of

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6.1 mmol/l (reference range 3.6–5.6 mmol/l), a hyponatraemia of 131 mmol/l (reference range 135–154 mmol/l), an increased albumin of 38 g/l (reference range 26–35 g/l), increased calcium of 3.2 mmol/l (reference range 2.3–3.0), increased urea of 8.1 mmol/l (reference range 1.7–7.4 mmol/l) with a creatinine at the high end of the reference range at 131 µmol/l (40–132 µmol/l) and a decreased glucose of 2.7 mmol/l (reference range 3.0–5.0 mmol/l). Basal serum cortisol was decreased at less than 13.5 nmol/l (reference range 20–230 nmol/l; lowest detectable amount at 13.5 nmol/l).

CLINICAL TIP ON USE OF BASAL CORTISOL

While an adrenocorticotrophin hormone (ACTH) stimulation test is the definitive diagnostic test for hypoadrenocorticism, a serum basal cortisol concentration of greater than 70 ng/l effectively rules out hypoadrenocorticism. This test should probably be run in any dog with unexplained gastrointestinal signs or weakness, regardless of the electrolyte concentrations.

CLINICAL TIP ON SODIUM: POTASSIUM RATIOS

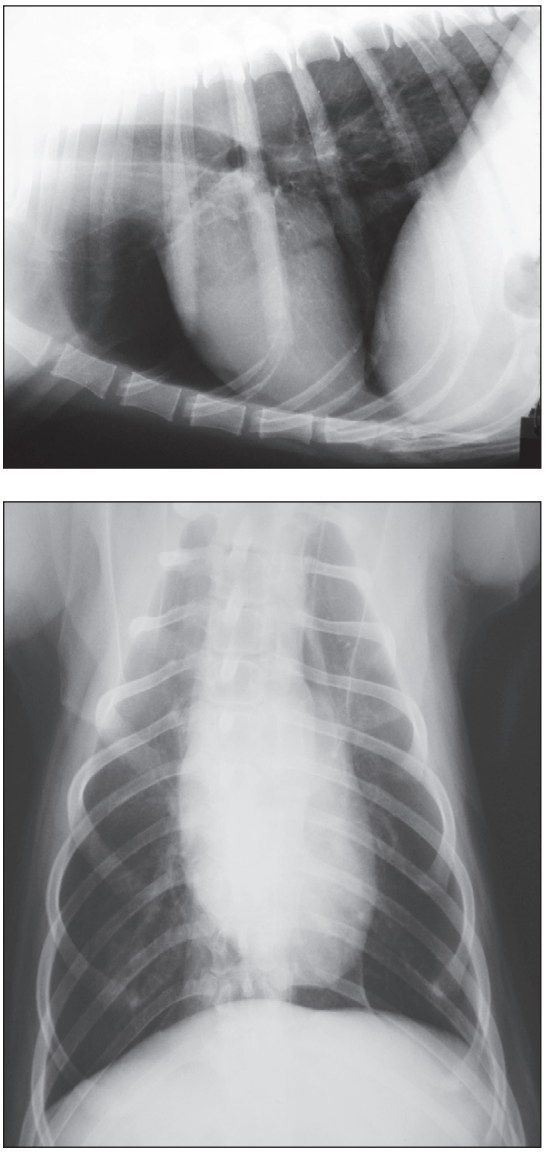
Sodium to potassium ratios of less than 27 have been used to diagnose hypoadrenocorticism, but other differential diagnoses for hyponatraemia with concurrent hyperkalaemia should include (as well as hypoadrenocorticism): renal failure, chylothorax and gastrointestinal tract disorders. The ACTH stimulation test is the gold standard for definitive diagnosis of hypoadrenocorticism.

Only a very small amount of urine was obtained by cystocentesis. The urine specific gravity was 1.019, which was inappropriately low for the degree of dehydration. The chemical strip showed a pH of 5.5 with no blood, bilirubin or glucose.

As these results are consistent with, but not diagnostic for hypoadrenocorticism and there was evidence of hypoglycaemia, the fluids were changed to 0.9% saline with 5% glucose and the rate was increased to 100 ml/hour.

Imaging

Thoracic radiographs showed microcardia and a small vena cava, consistent with dehydration (Figs 14.1 and 14.2). Abdominal ultrasound showed very small adrenal glands bilaterally (Figs 14.3 and 14.4).

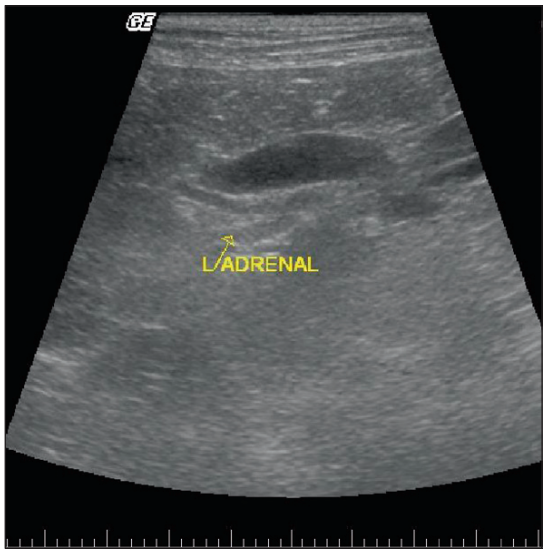


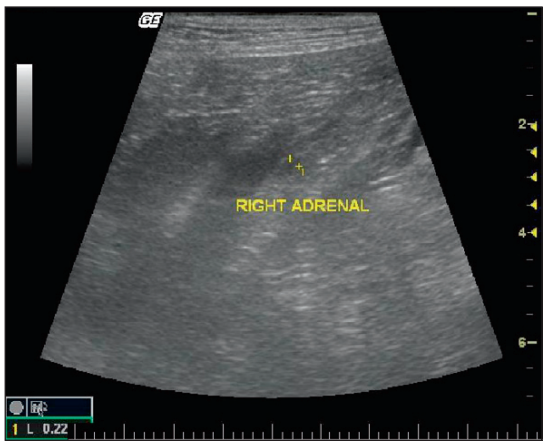
Figs 14.1 and 14.2 Lateral and ventrodorsal thoracic radiographs showing the decreased heart and vessel size consistent with dehydration (courtesy of Dr Tobias Schwarz)

Electrocardiogram

The dog started to deteriorate with a slower heart rate of 46 bpm prior to the results being available. An electrocardiogram (ECG) was performed which showed a heart rate of 48 bpm, spiked T waves and an absence of P waves, consistent with the hyperkalaemia (Fig 14.5).

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Figs 14.3 and 14.4 Ultrasound pictures of the left and right adrenal glands, both of which are decreased in size (courtesy of Carolina Urraca del Junco)

ACTH stimulation test

An ACTH stimulation test was performed. Serum results of the post-ACTH stimulation test were less than 13.5 ng/l, confirming the diagnosis of hypoadrenocorticism.

Initial treatment

The fluid rate was increased to 850 ml/hour for an hour (50 ml/kg/hour) using normal (0.9%) saline, and intravenous hydrocortisone sodium succinate was administered at 10 mg/kg every 6 hours.

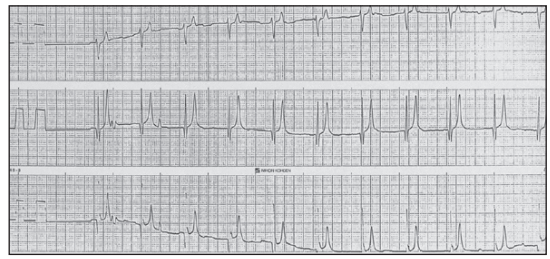


Fig 14.5 ECG showing bradycardia (heart rate 48 bpm), flattened P waves and spiked T waves typical of atrial standstill with hyperkalaemia (courtesy of Geoff Culshaw)

Follow-up

The dog responded very well to treatment and the following day was brighter and interested in eating. She was started on oral fludrocortisone at 0.2 mg (total dose) po q 24 hours and an initial dose of prednisolone at a physiological dose of 0.15 mg/kg po q 24 hours.

Outcome

The prednisolone was discontinued 1 week later; however, the owners kept a supply in case the dog suffered a stressful incident. Haematological and serum chemistry parameters returned to within the reference range other than a tendency to have potassium concentrations at the high end of the reference range and sodium just below the reference range.

She has continued to do well several months after diagnosis and her prognosis for a normal life is good, although she will need lifelong medication.

CASE DISCUSSION

In typical hypoadrenocorticism there is a deficiency of aldosterone and glucocorticoids. Aldosterone normally promotes sodium retention and water reabsorption as well as potassium excretion, especially in the renal tubules. The aldosterone deficiency leads to the electrolyte imbalances of increased potassium and decreased sodium, causing the clinical signs of impaired cardiac conduction and bradycardia, lethargy and nausea. Glucocorticoid deficiency causes loss of appetite, vomiting, diarrhoea and lethargy.

An atypical form of hypoadrenocorticism also occurs in which there is only a glucocorticoid deficiency and no change in the serum electrolyte concentrations. These dogs may still show gastrointestinal signs and weakness.

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Because hypoadrenocorticism can occur with or without electrolyte changes, this diagnosis should be considered in the differential diagnoses for any dog with any combination of weakness, gastrointestinal signs (vomiting or diarrhoea) or polyuria/polydipsia. The clinicopathological abnormalities of azotaemia, increased calcium and inappropriately low urine specific gravity may appear similar to kidney disease.

A basal cortisol is useful in ruling out hypoadrenocorticism as a value greater than 70 nmol/l will rule this out, while in a patient with a value less than that an ACTH stimulation test should be performed.

Concurrent hypovolaemia affects many of the parameters measured, in that albumin, urea, creatinine and the PCV appear higher than they will be after the dog is rehydrated. The inappropriately low urine specific gravity is caused by the impaired concentrating ability secondary to chronic sodium loss reducing the renal medullary concentration gradient.

The weakness may also be exacerbated by hypoglycaemia due to the lack of glucocorticoids, so the blood glucose should always be checked and treated if necessary. Mild to moderate hypercalcaemia is seen in about a third of dogs with hyperadrenocorticism; the cause of this is not yet known.

Medical management

Hyperkalaemia can be life-threatening and should be treated with aggressive fluid therapy with normal saline (0.9% sodium chloride). If hypoglycaemia occurs, either 5% glucose should be provided intravenously or a slow bolus of 50% glucose at 1 ml/kg should be given intravenously.

Glucocorticoid therapy should be administered early in the treatment. If there is a strong suspicion of hypoadrenocorticism, it should be given as soon as the ACTH stimulation test is completed rather than waiting for the results. If the dog is in a potentially fatal crisis, dexamethasone may be given prior to the test as it will not interfere with the assay, although it will affect the test results.

Mineralocorticoid treatment is not necessary in a crisis situation, although it will be needed for longer term management. Hydrocortisone does have some mineralocorticoid as well as glucocorticoid activities, although prednisolone sodium succinate or dexamethasone sodium succinate may also be used.

For longer term use, fludrocortisone acetate per os at an initial dose of 15 μg/kg q 24 hours is usually used in the United Kingdom, although injectable desoxycorticosterone pivalate is available in some countries. The dose of fludrocortisone often needs to be increased during the first year of treatment in many dogs to maintain the serum potassium concentration within the reference range and some dogs may need twice daily dosing.

Glucocorticoid (e.g. prednisolone) therapy is usually not needed on a daily basis in dogs treated with fludrocortisone (although may be needed in those treated with desoxycorticosterone pivalate). Owners are usually provided with a supply of prednisolone tablets to be given at a dose of 0.1 to 0.2 mg/kg/day if the dog appears to show clinical signs.

Salt supplementation may help the initial hyponatraemia, especially early in the treatment period. Many affected dogs do have a serum sodium concentration slightly below the reference range regardless of salt supplementation and this does not appear to affect them clinically if the serum potassium concentrations are well managed.

Epidemiology

While hypoadrenocorticism can occur in any breed, there has been a greater incidence reported in Great Danes, Portuguese water dogs, Rottweilers, standard poodles, West Highland white terriers, soft coated wheaten terriers, Nova Scotia duck tolling retrievers and bearded collies. It occurs most often in young to middle aged dogs (e.g. 4–6 years of age) and more often (70%) in female dogs (except in bearded collies and standard poodles, where both genders are affected equally). Intact female dogs appear to have a higher risk than spayed female dogs.

Prognosis

Hyporadrenocorticism can be life-threatening if not rapidly recognized and treated. The disorder requires lifelong management. Nevertheless, with good client compliance, the prognosis is excellent. In one study, 100% of 98 treated dogs survived to discharge.

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15 Lymphocytic inflammatory bowel disease/alimentary lymphoma in a cat

INITIAL PRESENTATION

2 months of intermittent vomiting, with recent increase in frequency
Signalment: 9-year-old neutered male domestic shorthaired cat; body weight 3.9 kg

CASE HISTORY

The cat presented with a 2-month history of vomiting and an increasingly poor appetite. The vomiting had been occurring initially two to three times per week, but during the 2 weeks prior to admission was occurring up to four times per day. The vomitus generally consisted of partially digested food, sometimes with bile present. There had been no diarrhoea, although the amount of faeces had decreased as the cat's appetite decreased.

His previous diet was a commercial dry food supplemented once a day with canned food. During the time his appetite was decreasing the owner had been tempting him with cooked chicken and ham.

His vaccinations were up to date and he had been treated for parasites 3 months prior to presentation. He had not travelled outside of the UK.

PHYSICAL EXAMINATION

On physical examination the cat was bright and alert, but seemed to tire quickly. His body condition score was 3/9. His mucous membranes were pink but slightly dry and capillary refill time was <2.5 seconds. Thoracic auscultation was unremarkable and lymph nodes were within normal limits. On abdominal palpation diffusely thickened loops of bowel were felt. His temperature was 38.7° C, respiratory rate was 30 breaths per minute and his heart rate was 187 beats per minute. His coat was unkempt in appearance.

PROBLEM LIST

- Chronic vomiting

DIFFERENTIAL DIAGNOSIS

- Dietary sensitivity (allergy or intolerance)
- Dietary indiscretion
- Stomach
  - gastritis
  - gastric ulcers
  - inflammatory bowel disease
  - foreign body
  - parasites
  - neoplasia
- Small intestinal disease
  - infectious, e.g. bacteria, viral ,fungal
  - gastrointestinal (GI) parasites
  - inflammatory bowel disease
  - infiltrative neoplasia, e.g. lymphoma, mastocytosis
  - high partial obstruction, e.g. intussusception, foreign body
- Systemic disease
  - uraemia
  - liver failure
  - sepsis
  - congestive heart failure
  - acidosis
  - hypoadrenocorticism
  - ketoacidotic diabetes mellitus
  - hyperparathyroidism
  - gastrinoma
- Neurological diseases (no other signs consistent with these in this cat)
  - dysautonomia

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- vestibular disease
- CNS disease
- Drugs, toxins (no known history of drug or toxin exposure)

CASE WORK-UP

Minimum data base

Haematology, serum chemistry, urinalysis and faecal examination were preformed.

Haematology results were within the reference ranges. Serum chemistry showed a mild decrease in albumin to 24 g/l (reference range 28–39 g/l), and mild hyperglycaemia of 6.7 mmol/l (reference range 3.0–5.0 mmol/l), which was thought to be likely due to stress. Serum total thyroxin was within the reference range at 17.5 nmol/l (reference range 13–48 nmol/l).

The serum cobalamin concentration was decreased to 177 ng/l (reference range 251–908 ng/l) and folate was decreased to 6.4 µg/ml (reference range 9.7–21.6 µg/l).

CLINICAL TIP: FOLATE AND COBALAMIN (VITAMIN B_{12})

Cobalamin is absorbed in the ileal part of the small intestine. It requires prior binding with intrinsic factor, which comes from the pancreas in cats and the stomach and pancreas in dogs. Deficiency may be caused by poor absorption due to intestinal disease, exocrine pancreatic insufficiency, excessive bacteria in the upper small intestine utilizing the vitamin, or a deficiency in intrinsic factor. In humans who are vegetarians (especially elderly humans), a dietary deficiency is possible; however, this is unlikely in cats as they cannot successfully be vegetarians.

Unlike most other B vitamins, cobalamin is stored in the body. In healthy cats the half life is 11 to 14 days and in cats with GI disease it is only 4.5 to 5.5 days. Clinical signs of cobalamin deficiency in humans include anaemia, thrombocytopenia, neuropathies and digestion abnormalities. In cats (and dogs) the clinical signs are not as well described; however, it has been suggested that response to treatment of intestinal disorders is improved with cobalamin repletion. A high serum concentration of cobalamin does not appear to have clinical significance and may occur with supplementation. The recommended dose for supplementation in humans exceeds the amounts needed to reach serum reference ranges and there is a consideration that there may be positive pharmacological effects.

Folate is absorbed only in the proximal intestine. A deficiency usually indicates small intestinal disease and if it is accompanied by a cobalamin deficiency it likely indicates widespread disease. A high serum folate can be caused by bacterial production, although serum concentrations of this vitamin are sensitive to food intake and a high dietary intake can also increase the serum concentration (Fig 15.1).

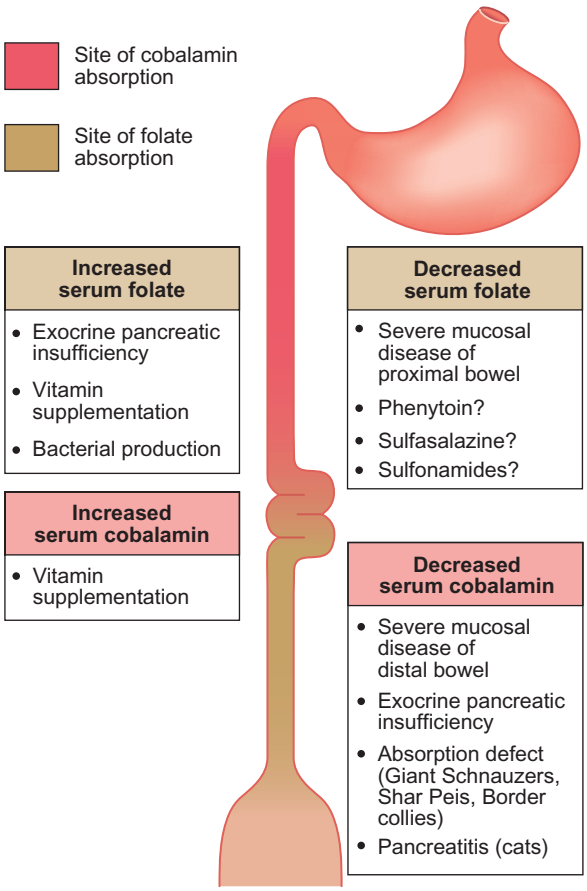


Fig 15.1 Intestinal absorption of cobalamin and folate

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Serum titres for *Toxoplasma gondii* were zero for IgM and 100 for IgG, inconsistent with the presence of this organism. Blood test results for feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV) were also negative.

Urinalysis showed a urine specific gravity of 1.026. The chemical strip analysis results were within reference ranges and the urine sediment was inactive. Urine protein to creatinine ratio was within the reference range (<0.4) at 0.12.

CLINICAL TIP

While a urine specific gravity of 1.026 is within the reference range for cats (1.015–1.050) it is lower than is expected for most healthy cats. This parameter should be repeated and if it is consistently below 1.030 (especially if the cat mostly eats dry food), the cause should be explored. Early chronic kidney disease may present with only an inappropriate low urine specific gravity.

Faeces were submitted for parasitology and culture of enteropathogens; both of these results were negative.

Imaging

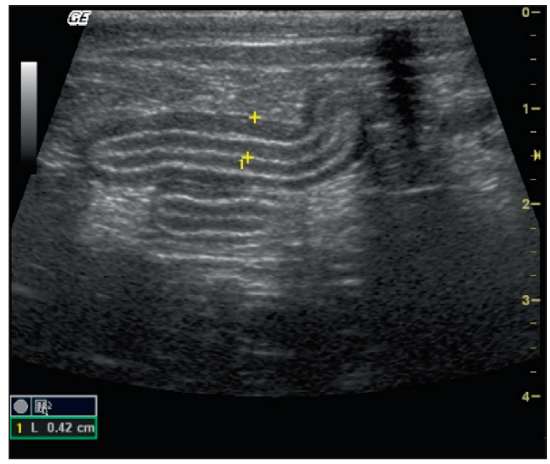
Abdominal radiographs showed no abnormalities.

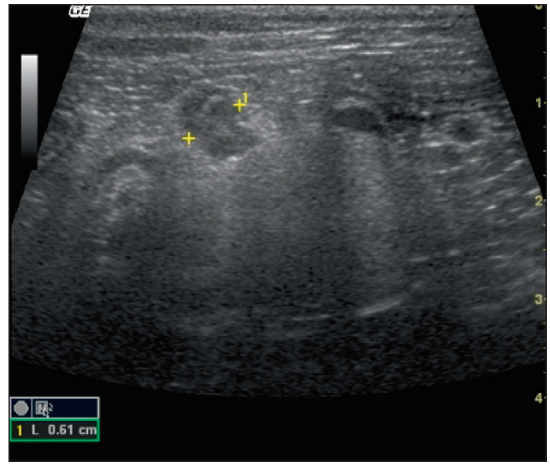
Abdominal ultrasound showed a diffusely irregular appearance and thickening to 0.55 cm (reference range <0.3 cm) of the mucosal layer of the small intestine. Loss of normal intestinal layering was present in some areas (Figs 15.2–15.5). Peristalsis appeared to be decreased. The mesenteric lymph nodes appeared prominent.

The right kidney had an irregular outline with a hyperechoic wedge-shaped lesion consistent with infarction.

Endoscopy and histopathology

At gastroduodenal endoscopy, there was digested blood present in the duodenum and the mucosa appeared much more roughened and hyperaemic than normal (Figs 15.6 and 15.7). Biopsies were taken of the gastric and duodenal mucosa.

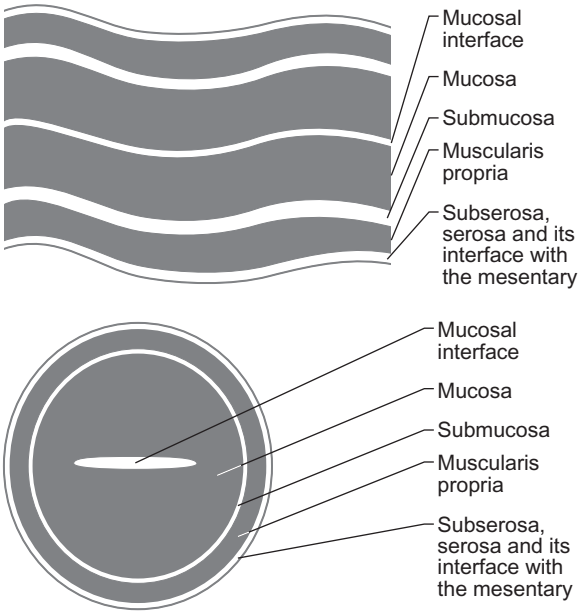




Figs 15.2 and 15.3 Sagittal and transverse ultrasound pictures of the small intestine, showing thickening and loss of normal layering (courtesy of Carolina Urraca del Junco)

Results of the histopathology of the gastric mucosa showed a moderate increase in lymphocytes and plasmacytes, which could be consistent with inflammatory bowel disease. The duodenal mucosa showed increased numbers of lymphocytes (Fig 15.8) which were thought to be possibly consistent with low-grade lymphoma, although severe lymphocytic inflammatory bowel disease could not be ruled out. The owners declined laparotomy in their cat for the purpose of obtaining full thickness intestinal samples and a more definitive diagnosis.

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Figs 15.4 and 15.5 Sagittal and transverse drawings of the ultrasonographic appearance of the normal intestine showing layering

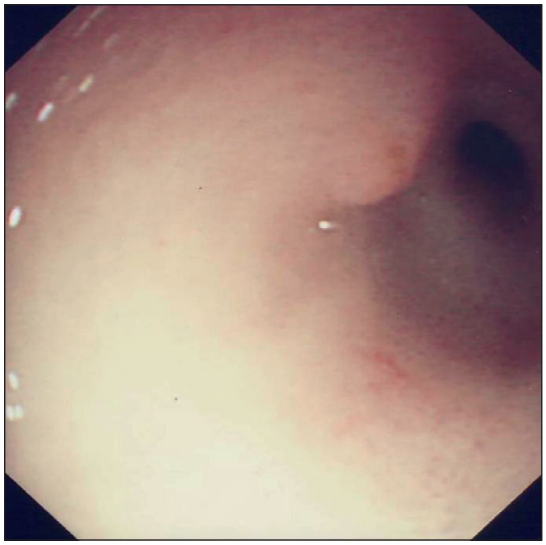


Fig 15.6 Endoscopic picture of a normal feline cranial small intestine

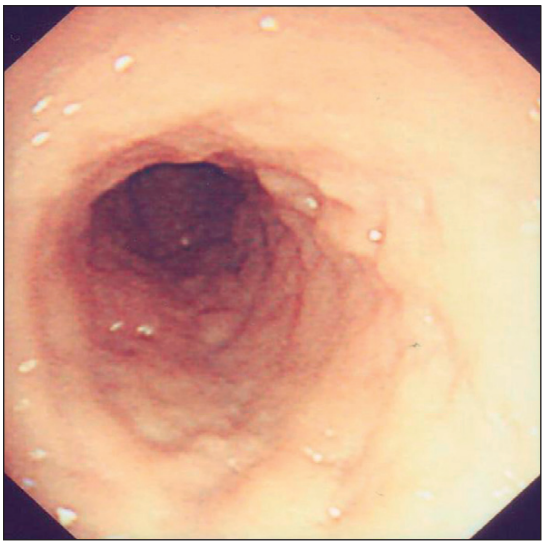


Fig 15.7 Endoscopic picture of an abnormally roughened feline small intestine due to cellular infiltration

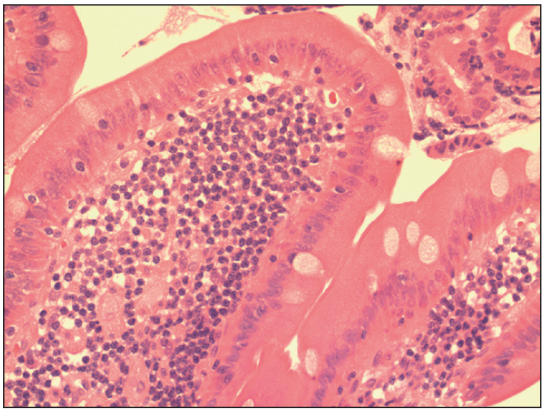


Fig 15.8 Histopathology of the duodenal mucosa showed increased numbers of lymphocytes

CLINICAL TIP

Gastric mucosal samples obtained via the endoscope may be useful for diagnosis of lymphoma in the stomach, but are not often adequate for differentiating between inflammatory bowel disease and lymphoma in the small intestine. The most common sites of GI lymphoma in the cat are the jejunum and ileum; areas which may require surgery (or laparoscopy) for obtaining intestinal samples. Intestinal lymphoma may also involve the deeper areas of the intestinal wall which may not be accessible by endoscopic biopsy instrumentation.

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Treatment and outcome

This cat was treated with oral chemotherapy with chlorambucil at 2 mg (total dose) po q 48 hours combined with prednisolone at 10 mg po q 24 hours and was provided with nutritional support (see below).

Cobalamin was administered subcutaneously at 250 µg once a week for 8 weeks and oral folate was supplemented at 0.5 mg (per cat) per day as an oral liquid.

NURSING ASPECTS

This cat had had an insufficient caloric intake for at least a month. As he presented with a much diminished appetite, nutritional supplementation was vital. Animals with GI disease require enteral nutritional support if possible, as the intestine is nourished by the luminal presence of nutrients, especially glutamate in the small intestine.

The estimation of caloric requirements for hospitalized patients is based upon the resting energy requirement, which is in kcal 70 × body weight in kilograms$^{0.75}$. The presenting weight is used initially, rather than the optimal weight. In this cat, this value was:

$$70 \times 3.9^{0.75} = 70 \times 2.77 = 194 \text{ kcal}.$$

The estimated amount to initiate feeding was decreased to one-third of this amount, or 65 kcal to decrease the risk of the re-feeding syndrome. The key tips for nutritional supplementation of sick animals are to feed early and to try to not feed too much.

As this cat was vomiting, the provision of oral or gastric nutrition could have been difficult. Constant rate intravenous metoclopramide was administered with his intravenous fluids at a rate of 1 mg/kg/24 hours.

The initial feeding attempted was the use of a complete and balanced liquid food containing 1 kcal/ml. This was administered by gentle oral administration at 8 ml given eight times per day. This was not completely successful, as the cat would often only tolerate about 5 ml, which provided 40 kcal per day. While not ideal, this is still satisfactory for initial feeding. The cat was also offered chicken and white fish and on the second day began to eat several grams per day, adding about 30 to 50 kcal to his intake.

A naso-oesophageal tube could have been placed to feed the cat; however, there was a risk of vomiting the tube and having it displaced into the trachea. If the cat had not started eating, placement of a jejunostomy tube and/or intravenous parenteral nutrition would have been indicated.

By the end of the first week, the cat was eating a complete canned food in adequate amounts and had only vomited on days 4 and 6. He was discharged on oral medications, with repeat visits for evaluation and cobalamin administration.

He gained 0.6 kg over the 2 months following diagnosis and the owner reported that he vomited only once or twice a week and was much brighter and more active. His urine specific gravity did not increase above 1.030; however, he did not become azotaemic and his kidney disease appeared stable. He was lost to follow-up 1 year after the tentative diagnosis of intestinal lymphoma.

DISCUSSION AND EPIDEMIOLOGY

As more cats are vaccinated for FeLV, the more common site of feline lymphoma has shifted from the mediastinal form to the GI form, which most often affects the small intestine. Cats with GI lymphoma have a lower incidence of FeLV (15%) infection than do cats with other forms of lymphoma (20–85% depending upon the site), although the true incidence might be higher if more sensitive tests were used (e.g. PCR testing).

The most common age range of cats with GI lymphoma is 9 to 13 years. There is no evidence of a breed predilection, but male cats are slightly more likely to develop the disease. As in this cat, the signs are chronic and often include a poor appetite and weight loss. Vomiting is reported in fewer than 50% of cases and diarrhoea is present in about 30%, so absence of GI signs does not rule out the disease. Cats with low-grade lymphoma are likely to have palpably thickened loops of bowel.

Some cases show a non-regenerative anaemia consistent with anaemia of chronic disease or bone marrow infiltration of the tumour in cases with FeLV. Mild to moderate hypoalbuminaemia is often present; however, this is common with many diseases of the small intestine. In some cases liver involvement in the neoplasia results in increased liver enzymes.

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Low serum cobalamin and folate are present in many cases, but again, this is true of many small intestinal diseases where absorption is decreased.

The increased intestinal wall thickness is characteristic, but does not differentiate GI lymphoma from inflammatory bowel disease. The disruption of normal intestinal wall layering is usually more pronounced with lymphoma than other inflammatory bowel disease. The increase in mediastinal lymph node size is a non-specific finding common in many abdominal disorders.

Histology of GI biopsies is needed for a definitive diagnosis, but even then the diagnosis can be difficult. There are three grades of GI lymphoma:

- Low – lymphocytic or small cell
- Intermediate
- High – lymphoblastic, immunoblastic or large cell.

It can be extremely difficult, even with histopathology, to differentiate low grade small cell lymphoma from lymphocytic inflammatory bowel disease. Many clinicians believe that inflammatory bowel disease may lead to lymphoma in some cases. CD79a (B-cell) and CD3 (T-cell phenotype) antibody stains can be used to assess B- and T-cell immunoreactivity to help differentiate between the disorders. A predominance of one type of cell is more likely to be tumour and a mixed population more likely to be IBD. While an aid in diagnosing, the type of tumour cell (B versus C) does not correlate to response to therapy or survival time in cats.

Treatment discussion

Various chemotherapy protocols have been used for GI lymphoma; these include various combinations of cyclophosphamide, vincristine, prednisolone, L-asparaginase, doxorubicin and methotrexate. Remission rates with these protocols are difficult to interpret as many studies do no include the histological grade of the disease and remission rates are better for low grade small cell lymphoma.

Cats with low grade lymphoma, such as this cat, can often have a good response to the less aggressive chemotherapy protocol of chlorambucil and prednisolone with less risk of serious side effects. A remission rate of 69% has been reported with this treatment in these cases, with a median survival time of 17 months. Cats which achieve complete remission have better survival times. It may be that some of the cats treated for small cell lymphoma have a form of inflammatory bowel disease; however, the treatment appears to be appropriate for either disorder.

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16 Ingestion of glue by a dog

INITIAL PRESENTATION

Vomiting and decreased appetite

Signalment: 15-month-old neutered female Bassett Fauve de Bretagne (Fig 16.1), body weight 9.1 kg

CASE HISTORY

The dog had been healthy up until 2 days prior to examination when she was thought to have been playing with a tube of glue. The owner reported that the dog had chewed off the cap and may have eaten some of the glue. Since then she had vomited twice, starting soon after access to the glue. She then had shown a decrease in appetite. The vomitus contained mostly a green fluid and foam. The owner reported that the dog had also passed a small amount of dark faeces.

The dog was de-wormed regularly with fenbendazole and she had received her vaccinations as a puppy. Her regular diet was a commercial dry dog food supplemented with treats.

PHYSICAL EXAMINATION

The dog was quiet but responsive. Her body condition score was 5/9. The mucous membranes were slightly tacky but the colour was pink and capillary refill time was less than 2 seconds. Peripheral lymph nodes were unremarkable.

Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 100 beats per minute. On abdominal palpation, a firm distended stomach was felt and the dog was uncomfortable during palpation. Respiratory rate was 24 breaths per minute. Rectal temperature was 38.4° C. She had been given no medications.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting and poor appetite

The dog's poor appetite was thought to be related to the vomiting and separate differential diagnoses were not considered.

DIFFERENTIAL DIAGNOSIS

Differentials for acute vomiting in this dog included:

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilatation-volvulus
  - neoplasia
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease
  - neoplasia
  - parasites
  - intussusception (unlikely)
- Disorders of the large intestine
  - colitis
  - obstipation
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism
  - diabetes mellitus
  - liver disorders
  - peritonitis
  - renal disease/uraemia (unlikely)
- Dietary causes
  - adverse reaction to food (allergy or intolerance)
  - dietary indiscretion

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Fig 16.1 Bassett Fauve de Bretagne dog at presentation

Because of the history of potentially having ingested glue and the fact that the dog appeared otherwise very healthy, glue ingestion was the top differential.

CASE WORK-UP

The dog was admitted to the hospital for diagnostic investigation.

Minimum data base

Haematology and serum chemistry were performed and the results were unremarkable. Albumin was 35 g/l, at the top of the reference range (26–35 g/l), which was likely due to haemoconcentration from mild dehydration.

Imaging

Abdominal radiographs showed a much distended rounded appearance to the stomach in both ventrodorsal and lateral views (Figs 16.2 and 16.3).

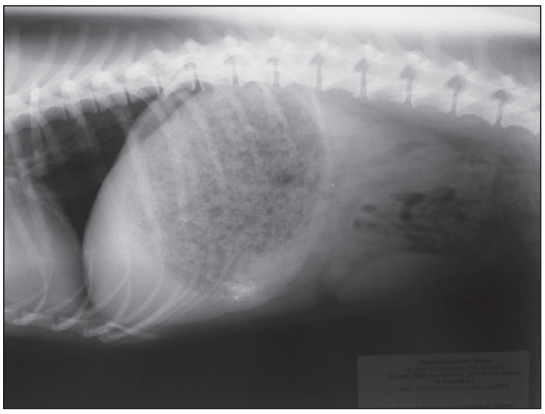
Abdominal ultrasound showed the pyloric antrum and pylorus filled with hyperechoic material. The material was not moving despite the presence of peristalsis. The duodenum was empty and the mass appeared to be causing a pyloric obstruction.

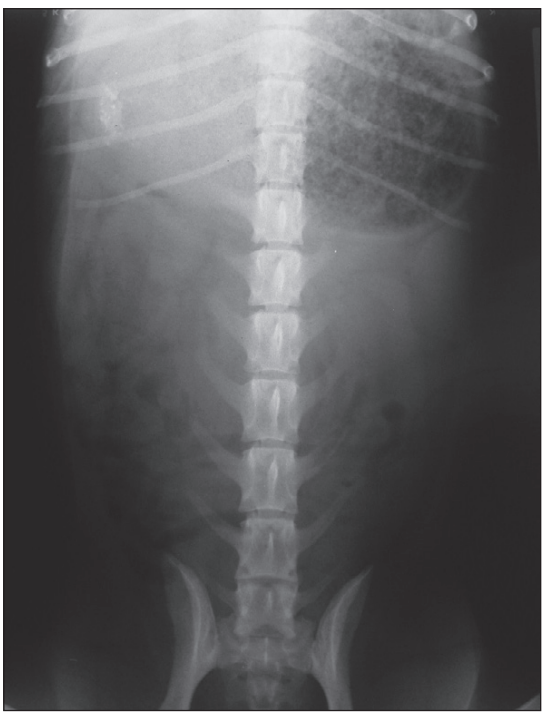
Case assessment

Initial case management included administration of intravenous fluids to maintain hydration and provide venous access. As there was the presence of a foreign mass, surgical exploration was performed.

Surgical management

At surgery a large gastrotomy was performed and a dense mass filling the stomach was removed (Fig 16.4).





Figs 16.2 and 16.3 Lateral and ventrodorsal abdominal radiographs of patient showing stomach distended with a heterogeneous appearing mass (courtesy of Dr Donald Yool)

An area of severe gastric ulceration was identified in the gastric mucosa, but the tissue appeared viable and resection was not needed. A prophylactic gastropexy was performed to prevent possible postoperative dilatation and volvulus from the mechanical stretching of the stomach by the mass.

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Fig 16.4 The mass of glue which was removed from stomach of the patient (courtesy of Dr Donald Yool)

Gastric biopsies of the ulcerated area and of a non-ulcerated area of the stomach were submitted for histopathology. There was mucosal loss with severe focal gastric ulceration and submucosal oedema. Moderate acute gastritis with large numbers of *Helicobacter*-like organisms were identified deep within the gastric glands in a non-ulcerated area of the stomach.

CLINICAL TIP: HELICOBACTER SPECIES

Many species of *Helicobacter* have been identified in dogs and cats: *H. felis*, *H. pylori* and *H. heilmannii* (*Gastrospirillum hominis*), *H. salomonis* and *H. bizzozeronii* have been the most common. Experimentally, infection can be established in both dogs and cats and a lymphoid follicular gastritis can be produced. However, in these experimental studies, clinical signs are absent or very mild. Several surveys of laboratory and pet populations have shown a very high prevalence rate. Peptic ulceration appears to be very rare in dogs and cats. The role of *Helicobacter* species in clinical cases of chronic gastritis is unknown, as is the optimal treatment. In approximately 25% of cases in which *Helicobacter* is identified, it appears to be the cause of or related to the clinical signs and should be treated. When the organisms are deep in the gastric glands (as in this case), rather than located more superficially, it is more likely that they are involved in the pathophysiology of vomiting.

There are several treatment protocols for *Helicobacter* infection, including 2 weeks of omeprazole 0.7 mg/kg po q 24 hours (or an H2 blocker such as ranitidine), amoxicillin at 10 to 20 mg/kg po q 8 hours, metronidazole 10 mg/kg po q 12 hours. Other suggestions include omeprazole with either azithromycin at 10 to 20 mg/kg po q 24 hours in dogs and 5 mg/kg po q 24 hours in cats or clarithromycin at 7.5 mg/kg po q 12 hours. Relapses have been seen. One study revealed that 70% of infected dogs can be cleared with triple antibiotic therapy with or without famotidine for 4 weeks. Vomiting decreased significantly in dogs in which the *Helicobacter* was cleared.

Follow-up and medical management

The dog recovered well after surgery, although she vomited once. Postoperative medical treatment included the anti-emetic maropitant at 1 mg/kg subcutaneously q 24 hours, omeprazole (1 mg/kg q 24 hours po), sucralfate (500 mg q 8 hours po) for their gastroprotectant effects and tramadol (2 mg/kg q 12 hours; initially intravenously, then po) for analgesia. Treatment for the *Helicobacter* species found in the gastric biopsy samples was clavulanate-potentiated amoxicillin (15 mg/kg po q 12 hours) and clarithromycin (7.5 mg/kg q 12 hours po) for 2 weeks.

CASE DISCUSSION AND EPIDEMIOLOGY

Most cases of glue ingestion by dogs (and less commonly, cats) result in gastritis and enteritis; however, expanding glues are associated with gastric obstruction with a foreign body. Gorilla glue is a urethane polymer and polymeric isocyanate liquid which expands to three to four times its original mass when used. The material is activated from a liquid to a solid mass in the stomach when ingested, which probably takes about 20 minutes. It will incorporate food in the stomach into the mass.

Dogs ingesting as little as 2 oz of the glue have developed gastric obstruction. In one report, eight of 10 cases of polyurethane glue ingestion required surgical removal of the mass. Clinical signs usually develop within 12 hours of ingestion (within 30 minutes in one reported case) and include vomiting, possibly haematemesis, anorexia, lethargy, abdominal distension, abdominal

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pain and secondary tachypnoea. Gastric hyperaemia and ulceration have been reported, as was present in this case, and in one reported case, gastric perforation occurred.

The radiographic appearance (see Fig 16.2) is fairly distinct, with the presence of a distended, stomach filled with a mottled radiopaque substance. Radiographic evidence may be present 4 hours after ingestion, although is more obvious in radiographs taken at 24 hours.

Because of the solid nature of the mass, induction of emesis should not be considered; it is also ill advised as having the glue expand within the oesophagus would cause serious complications. Unlike in most toxin ingestion cases, administration of charcoal is also not advised as it would be incorporated in the mass rather than preventing absorption of the glue (which is unlikely anyway).

Prognosis

If the glue mass is removed without delay and if gastric perforation does not take place, the prognosis for recovery should be good.

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17 Lead ingestion in a puppy

INITIAL PRESENTATION

Vomiting and seizures

Signalment: 5-month-old entire male West Highland white terrier, body weight 3.1 kg

CASE HISTORY

The puppy had been vomiting bile and occasionally digested food once a day for 4 weeks. Two weeks prior to consultation he had a seizure which was described as beginning with barking and running around the room before becoming recumbent, unconscious, salivating and paddling. The episode was thought to have lasted about 10 minutes. There had been no further seizures.

The owner thought that the puppy's appetite was reduced and the puppy was reportedly more flatulent. His stools were reported as normal in consistency, but only passed every other day. The puppy's drinking and urination were unchanged.

The puppy did play with toys and the owner did not know if he could have ingested any of them.

The owner had owned the patient and his litter mate since they were 12 weeks old and the other puppy appeared healthy. The affected puppy was smaller than his littermate. Both puppies were fully vaccinated and had been de-wormed with fenbendazole several weeks prior to presentation. Both puppies were fed a commercial puppy food supplemented with commercial 'puppy milk', chicken and rice. They had not travelled out of the United Kingdom.

PHYSICAL EXAMINATION

The puppy was alert and responsive, but quieter than normal for a 6-month-old terrier puppy. His body condition score was 4/9. His mucous membranes were pink and moist with a capillary refill time of <2 seconds. His heart rate was 152 beats per minute with matched good quality pulses. Respiratory rate was 28 breaths per minute and thoracic auscultation revealed no abnormalities. Abdominal palpation was unremarkable, as were his peripheral lymph nodes. Rectal temperature was 38.6° C.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Seizure
- Vomiting

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for vomiting

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilatation-volvulus
  - neoplasia (unlikely at this age)
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease
  - neoplasia (again, unlikely at this age)
  - parasites
  - intussusception
- Disorders of the large intestine
  - obstipation
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism

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- diabetes mellitus
- porto-systemic shunt (PSS) and other liver disorders
- renal disease/uraemia (unlikely, no concurrent signs)

- Toxins
  - NSAIDs
  - lead
  - mercury
  - organochlorines. organophosphates, carbamates
  - toxic plants, e.g. philodendron, diffenbachia, daffodil bulbs
- Dietary causes
  - adverse reaction to food (allergy or intolerance)
  - dietary indiscretion

Differential diagnoses for seizures

- Intracranial disorders
  - congenital and hereditary disorders, e.g. brain malformations, Chiari-like malformations of the foramen magnum, hydrocephalus, inborn errors of metabolism (the puppy was probably too normal after the seizure for these differential diagnoses to be highly likely)
  - inflammation, e.g. encephalitis, vascular disease, granulomatous meningioencephalopathy (GME), neoplasia, abscess/granuloma (other than a resolving vascular disorder or GME), the puppy was too normal after the seizure for most of these differential diagnoses
  - epilepsy – possible, as can start at a young age
  - intracranial haemorrhage – possible if resolving
- Extracranial (systemic) disorders affecting the brain
  - Infectious agents, e.g. distemper, *Parvovirus*, *Anaplasma/Ehrlichia*, rabies, bacterial, tetanus; there was no evidence of these in the history or at physical examination
  - Metabolic disease – many of these are possible as other than hypothyroidism, sometimes the signs are intermittent
    - hypoglycaemia
    - congenital hypothyroidism (usually presents as cretinism in puppies)
    - hypocalcaemia
    - hypernatraemia
  - Toxins
    - lead
    - organophosphate
    - metaldehyde
  - Hepatic encephalopathy, e.g. with portosystemic shunt (PSS)
  - Parasites – *Toxoplasma*, migrating larvae of *Cuterebra* (unlikely in the UK) or *Toxocara*

In this case, considering the two problems (vomiting and neurological signs) together made PSS or toxins the most likely cause, although the possibility of two separate unrelated problems had to be considered.

CASE WORK-UP

Minimum data base

A minimum data base of haematology, serum chemistry and urinalysis was obtained. The packed cell volume was 0.34 l/l (reference range 0.37–0.55 l/l), with a decreased mean corpuscular volume (MCV) of 55 fl (reference range 60–77 fl) and decreased mean cell haemoglobulin concentration (MCHC) of 31% (reference range 32–36%). The blood smear was also consistent with a mild microcytic hypochromic anaemia. Moderate numbers of polychromatophils and normoblasts (nucleated red blood cells (nRBCs)) were seen and some basophilic stippling was present on some of the polychromatophils (Fig 17.1).

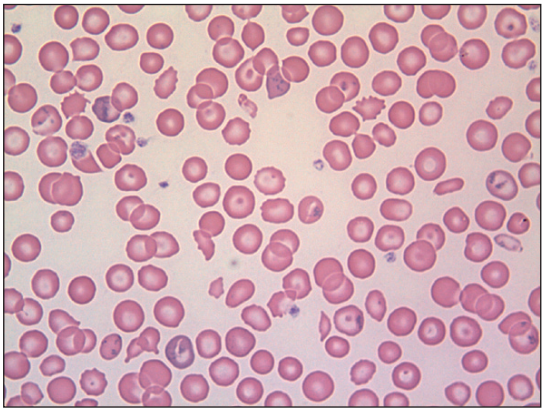


Fig 17.1 Basophilic stippling in red blood cells from lead toxicity (courtesy of Prof Elspeth Milne)

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CLINICAL TIP ON MICROCYTOSIS

Microcytosis demonstrated by smear and reduced MCV may be caused by chronic blood loss, anaemia of chronic disease or chronic inflammation, or PSSs. It is also seen in healthy Japanese Akita dogs. Hypochromia is most often seen in iron deficiency anaemias.

Basophilic stippling is neither specific nor sensitive for lead toxicity as it can also occur in regenerative anaemias. The presence of the combination of basophilic stippling and normoblasts (nRBCs) did indicate that blood lead concentration should be determined.

Serum chemistry showed an elevated alkaline phosphatase (AP) of 134 IU/l (adult reference range 20–60 IU/l); however, for a puppy of this age this is within expectations of two to three times adult ranges. The inorganic phosphate was also elevated at 2.77 mmol/l (reference range 0.9–2.0 mmol/l) but also within reference range for a growing puppy.

A bile acid stimulation test was performed as PSS was high on the differential diagnoses lists for both problems. The pre-prandial serum bile acid concentration was 6.7 µmol/l (reference range 3.8–7.9 µmol/l) and the post-prandial serum bile acid concentration was 28.2 µmol/l. Generally, post-prandial values over 30 µmol/l are suggestive of PSS, but a lower value cannot completely rule it out.

Urinalysis revealed a urine specific gravity of 1.030, pH of 5.8, with negative results on other chemical strip parameters. A few white blood cells and epithelial cells were seen on the urine sediment. The finding of urate crystals would indicate a possible PSS, but again, the absence of this finding cannot rule it out.

Imaging

Abdominal radiographs were taken, primarily to look for evidence of a foreign body, and no abnormalities were detected on these other than the presence of a moderately small sized liver.

Abdominal ultrasound confirmed the small liver size, but no shunting vessel was found. The rest of the abdominal organs appeared normal.

Blood lead concentration

A blood sample was submitted to measure the lead concentration and the result was 21.9 µmol/l with a reference range of <2.9 µmol/l. As no lead foreign bodies were found on abdominal radiographs, another source of lead ingestion had to be explored. The other puppy in the household was also tested and his blood lead concentration was 3.77 µmol/l, which is above the reference range but as he was not symptomatic, he was not treated. The owner was also tested and her blood lead concentrations were not elevated. As only the dogs were affected, it was likely that there was an environmental contamination, e.g. in the garden or from a food container, rather than the in-house water supply.

Treatment and outcome

Treatment used in this case was chelation of the lead with calcium edetate at 25 mg/kg by subcutaneous injection every 6 hours for 5 days. The 250 mg/ml calcium edetate solution was diluted in 5% glucose to a concentration of 10 mg/ml prior to injection. The dog was monitored in hospital during the treatment and tolerated it well. His blood lead concentration 1 week after chelation finished was 4.59 µmol/l. He did not show any further clinical signs after chelation treatment and no further treatment was used.

CLINICAL TIP ON CALCIUM EDETATE TREATMENT

In animals with chronic lead toxicity, treatment may result in a post-chelation rebound effect, where blood lead concentration increases, due to redistribution of the lead from bone and tissue. In cases where the blood lead concentration increases, it is necessary to ensure that the patient is not getting re-exposed to lead. It is also necessary to make sure any lead present in the gastrointestinal (GI) tract has been removed by excretion, which sometimes can be aided by the use of a magnesium or sodium sulphate cathartic or by endoscopy or surgery.

DISCUSSION

Pathophysiology of lead toxicity

Lead is absorbed from the GI tract in varying amounts depending upon the form of the lead, the age of the patient and any intestinal disease present. The acidity of the gastric contents results in ionization of the lead, which is then absorbed from the duodenum, although most of it is passed in the faeces. Lead shot embedded

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in muscles is not commonly a cause of lead toxicity as it is not well absorbed, although if lead is in the joints it can be released into the body.

Lead is carried on the red blood cells and the unbound form is distributed widely throughout the tissues of the body. It is stored in bone, where it can replace calcium in the bone matrix. It may be released from storage during chelation treatment, fracture repair or lactation.

Lead in the blood is filtered through the kidneys and can accumulate in the renal tubular epithelium.

Lead interferes with calcium ions in the body and alters vitamin D metabolism. It interferes with haem synthesis, affects the stability of the red blood cell membrane causing increased fragility, shortens red blood cell life span and causes decreased oxygen carrying capacity.

The GI signs may be caused by alteration of the smooth muscle contractions.

It also causes neuronal damage, decreased cerebral blood flow, cerebral oedema, demyelination and decreased nerve conduction.

Clinical signs and laboratory findings

The GI signs seen with lead toxicity most commonly include vomiting, diarrhoea, abdominal discomfort and anorexia. Megaoesophagus may also occur. Neurological signs include ataxia, tremors, agitation, hysteria, seizures and blindness. Dogs have also been reported to develop aggression, dementia, pica and may even progress to being in a coma. Polyuria and polydipsia have also been reported and appear to occur more in older than in younger animals.

Normoblasts or nRBCs (greater than 40 nRBCs per 100 WBCs) have been found in about half of the blood smears of affected animals, occurring more often in dogs than in cats. Basophilic stippling of red blood cells is found in about one-quarter of the blood smears from affected dogs. An increase in white blood cells and an increase in liver enzyme (AP and alanine aminotransferase) activities are found in some patients.

Radiography may show a lead foreign body within the GI tract. In chronic cases of lead toxicity, lead lines, which are linear opacities in the epiphysis of the long bones, are seen in lead toxicosis of humans but are not commonly found in dogs and cats.

Diagnosis

The neurological and GI signs associated with lead toxicity are non-specific and have many differential diagnoses. Haematological changes are helpful but not consistently present, so blood lead concentrations should be checked in patients with the clinical signs. However, the blood lead concentrations can fluctuate and are often not consistent with the severity of clinical signs. Blood lead concentrations of 1.45 to 1.7 mmol/l (30–35 μg/dl) are suggestive of toxicity and levels greater than 2.9 mmol/l (60 μg/dl) are generally diagnostic of toxicity.

Medical treatment

When necessary, supportive treatment such as diazepam or barbiturates may be needed for control of seizures. Attempts should be made to find and remove the source of lead and it may be necessary to have the water supply checked.

If lead is present in the GI tract, removal is necessary prior to chelation therapy as calcium edetate increases the absorption of lead from the GI tract. Sulphate-containing cathartics may be given to aid emptying the GI tract; these also precipitate the lead as lead sulphate which is poorly absorbed. Larger lead objects may require removal by surgery or endoscopy.

Chelation is used to bind the lead to soluble complexes that are excreted in the urine. Most chelators are nephrotoxic, so renal parameters should be checked prior to use and monitored during use. Chelation of animals without signs, such as the litter mate of this puppy, is not recommended as the chelation may increase the blood lead concentration and result in clinical signs. When the source is removed, these animals will slowly eliminate the lead.

Calcium edetate was used as the chelator in this case, after dilution to a 10 mg/ml solution in 5% glucose. Sodium EDTA should not be used as it may cause hypocalcaemia. As the blood lead concentrations may increase during chelation, initial monitoring should be done by clinical signs instead of blood lead concentration. The most serious potential side effect of calcium edetate is a reversible nephrotoxicity. GI side effects may also been seen and reportedly these may be reduced by oral zinc supplementation, although no exact dose is available.

British anti-lewisite or dimercaprol is sometimes used as an adjunct therapy with calcium edetate to increase urinary and biliary excretion of lead. It is nephrotoxic and causes pain on injection and potentially may also cause vomiting and hypertension. Penicillamine may be used orally as a chelator but it will also bind zinc, iron and copper and its chelation products may be nephrotoxic.

Succimer is an analogue to dimercaprol which may be given orally. It is less likely to cause nephrotoxicity than the other chelators and does not bind with the

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nutritionally essential minerals. It probably does not increase the absorption of lead from the intestine and has a lower incidence of GI side effects. However, the drug is not widely available in the United Kingdom.

Following chelation, blood lead concentrations may rebound within 2 to 3 weeks due to redistribution of lead from bone and tissue. If this is not associated with clinical signs, further chelation therapy is not required. The animal's environment should be checked to ensure that there has not been further exposure.

Epidemiology

Younger animals are at greater risk for lead toxicity due to increased blood–brain barrier permeability and up to five times greater absorption rates following ingestion.

Usually the affected animals are less than 5 years of age and about a third of them are less then 1 year old. The more commonly affected gender is male and, for reasons that are unclear, poodles are one of the most commonly affected breeds.

Although lead is less commonly used than in previous decades as sources of lead are being replaced, there is still exposure to it. One of the most common sources is oil- and lead-based paints which were used before 1959 and may cause exposure during remodelling of buildings. Other sources include putty, caulking material, roofing materials, batteries, lubrication grease, fishing sinkers, lead shot, linoleum, improperly fired ceramic bowls, artists' paints, curtain weights, golf balls, wine cork covers, lead arsenate pesticides and soft water in lead pipes. One dog developed signs after licking water dripping from a tap which contained lead. Lead intoxication from water pipe sources may increase in the warmer months and with any cause of increased acidity. Lead contamination of soil can also be a source of exposure.

When lead poisoning is diagnosed in a patient where there is not an obvious source (e.g. lead foreign body in the GI tract), the owner should still be informed about the public health risks for human exposure. Children, like puppies and kittens, are at increased risk for lead poisoning. Local agencies should be contacted to check the water supply and investigate the source of contamination.

Prognosis

In animals showing mild to moderate signs that are treated appropriately, the prognosis is good as long as the source of lead is removed. In animals with severe nervous system signs the prognosis may be more guarded.

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18 Gastric adenocarcinoma in a dog

INITIAL PRESENTATION

Vomiting

Signalment: 11-year-old male entire Border collie, body weight 22.1 kg

CASE HISTORY

The dog had been vomiting intermittently for about 8 months. For the previous 2 months the frequency of vomiting had increased to at least once a day, usually in the morning. The vomiting occurred either before or after eating. The vomitus usually contained food and bile, but no blood. He was also reported to recently be gulping, retching and drooling. His appetite was still fair, although he was sometimes less interested in food than previously. His faeces were normal in appearance and frequency. He had lost some weight over the past 2 months.

He had previously been diagnosed with early congestive heart failure and was on treatment with furosemide (2 mg/kg po q 12 hours) and enalapril (0.45 mg/kg po q 12 hours). His urination and drinking were increased due to the diuretic treatment. His previous signs of weakness, coughing and tachypnoea appeared to be well controlled by the medication.

He was vaccinated yearly, but had not been treated for endoparasites for a couple of years. He was fed three times a day on a diet which included dry dog food as well as fried chicken, pasta, tuna and rice.

PHYSICAL EXAMINATION

The dog was bright, alert and responsive. His body condition score was 4/9. Thoracic auscultation revealed a IV/VI left apex parasystolic heart murmur, with normal lung sounds. Heart rate was 132 beats per minute and he was intermittently panting. His pulse quality was good. The mucous membranes were pink and slightly tacky with a capillary refill time of <2 seconds. He was estimated to be about 6% dehydrated. Abdominal palpation revealed no abnormalities, although the dog swallowed several times during palpation. Rectal temperature was 38.5° C.

CLINICAL TIP ON ABDOMINAL PALPATION

In some cases, dogs with abdominal discomfort will not tense their abdomen during palpation, but careful observation of the head and face may show swallowing, a sharp intake of breath (catching breath) or turning of the head. This should be gently repeated to check that it is a consistent finding. It is also valuable to observe the dog's head during palpation as an otherwise gentle dog may nip if a sensitive area is palpated!

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for vomiting in this dog included:

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilation-volvulus
  - neoplasia

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- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease
  - neoplasia
  - parasites
  - intussusception (unlikely)
- Disorders of the large intestine
  - colitis
  - obstipation
  - neoplasia
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism
  - diabetes mellitus
  - liver disorders
  - peritonitis
  - renal disease/uraemia (unlikely)
- Dietary causes
  - dietary sensitivity
  - dietary indiscretion

Most dogs (80%) with intussusception are less than 1 year of age, although it was still a possibility. He did not have any other signs of renal disease, so uraemia was an unlikely cause of the vomiting.

CASE WORK-UP

The dog's diet included a variety of foods, increasing the possibility of a dietary aspect to the aetiology of the vomiting. However, as he showed abdominal discomfort, an initial dietary trial was not an appropriate approach to the case.

Minimum data base

A minimum data base of haematology, serum chemistry and urinalysis was obtained.

Haematology showed a packed cell volume at the high end of the reference range at 55% (reference range 39–55%). While still in the reference range, this high value is uncommon in breeds other than sighthounds and was likely due to dehydration caused by the vomiting plus diuretic treatment. The other haematological parameters were within the reference ranges.

Serum chemistry parameters were within the reference range other than a serum creatinine of 162 μmol/l (reference range 40–132 μmol/l), a urea of 9.4 mmol/l (reference range 1.7–7.4 mmol/l) and lipase of 296 IU/l (reference range 13–200 IU/l). A SNAP cPL (qualitative canine pancreatic specific lipase test) was negative.

CLINICAL TIP

While an elevated serum lipase can be consistent with pancreatitis in dogs, it is neither sensitive nor specific. As lipase is excreted through the kidneys a decrease in glomerular filtration with increased urea and creatinine will also increase lipase. In this case the SNAP cPL (canine pancreatic specific lipase) also confirmed that pancreatitis was unlikely to be the cause of the dog's vomiting. The SNAP cPL is positive in ranges that are equivalent to a Spec cPL levels >200. (The Spec cPL is a quantitative test.) This value is considered to be consistent with pancreatitis. The correlation of the SNAP cPL to the Spec cPL is about 95%.

Urinalysis showed a urine specific gravity of 1.038, consistent with the dog's ability to concentrate urine when dehydrated, which was also being affected by the use of furosemide (and may have reflected a missed furosemide dose the morning of urine collection). The urine chemical strip results were unremarkable, with a pH of 7.4, and only a few struvite crystals seen on sediment.

CLINICAL TIP

Struvite crystals are normal in dog urine and should not be interpreted as an indication that uroliths (stones) are present. If uroliths are present, then the crystals can be an indicator of the type of stone, but the presence of crystals without stones is not an indication for treatment.

Imaging

A barium impregnated polyethylene spheres (BIPS) study was performed to detect any possible foreign body. A radiograph taken 14 hours after administration showed that all the BIPS had moved out of the stomach into intestines, showing that no obstruction was present (Fig 18.1).

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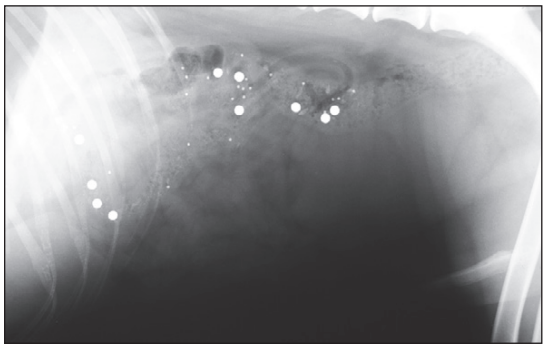


Fig 18.1 Lateral abdominal radiograph showing normal movement of BIPS ruling out a complete gastric obstruction (courtesy of Dr Tobias Schwarz)

CLINICAL TIP

BIPS are barium impregnated polyethylene spheres packaged with 9 or 10 large (5 mm) BIPS and 30 small (1.5 mm) BIPS. The small ones are meant to mimic the action of small food particles and move through the intestinal tract similarly to food. The large BIPS are similar to non-digestible particles which are only moved into the duodenum during the fasting period from the migrating motility or 'housekeeper wave' of contractility. If there is an obstruction present, the BIPS will bunch proximal to it. They do not show gut wall thickness, mucosal detail or outline foreign bodies.

Endoscopy

Gastroduodenoscopy was performed. A large raised mass with a crater ulcer was present at the lesser curvature of the stomach (Fig 18.2). The mucosa around the mass was inflamed, thickened and firm and the mass bled easily when biopsied. The proximal small intestine appeared normal.

As neoplasia was suspected, thoracic radiographs were taken. There was no evidence of pulmonary tumour metastasis present on the radiographs.

Histopathology of the samples of the gastric mass was described as gastric ulceration with associated intense granulation reaction. As there was a suspicion of neoplasia, a laparotomy was performed and a surgical biopsy was taken. The histopathology of this biopsy sample was interpreted as gastric adenocarcinoma.

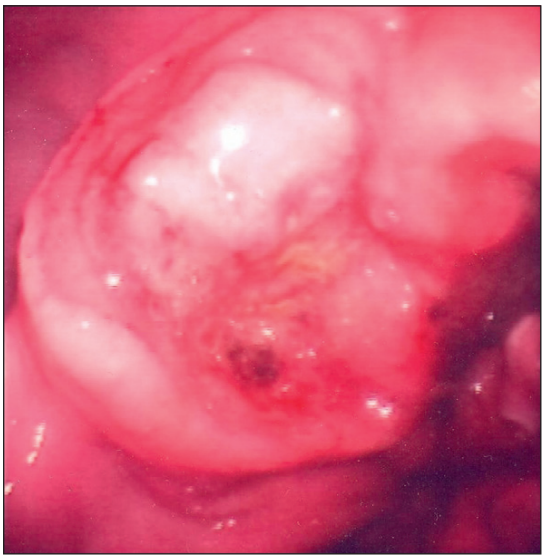


Fig 18.2 Endoscopic view of raised ulcerated lesion on lesser curvature of the stomach

Treatment and outcome

Gastric neoplasms are generally treated by surgical resection; unfortunately in this case the surgeons determined that the extent of the tumour was too great to excise. Chemotherapy for gastric adenocarcinomas is generally unrewarding. In humans with some types of gastric tumours, tumour growth is enhanced by histamine and histamine 2-receptor blockers such as ranitidine or cimetidine may inhibit tumour growth.

CLINICAL TIP ON GASTROINTESTINAL TUMOUR BIOPSY

These cancers often cause an inflammatory reaction and biopsies taken only from the mucosa may be interpreted as inflammatory or, if the tumour is affecting the submucosa or muscularis, the mucosa may appear fairly normal. The gastric wall will often feel rigid and stiff and be difficult to distend. If the gastric biopsy results from samples obtained via the endoscope are not consistent with the appearance of the stomach, a full thickness sample obtained by surgery may be required. As the stomach wall is abnormal, this must be done with care and skill, as dehiscence is possible when working with diseased tissue.

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Prior to obtaining the second biopsy results this dog was treated with palliative care using ranitidine and sucralfate. As no further treatment was available, the owners elected to euthanize the dog.

Epidemiology

The prevalence of gastric cancer is thought to be low in dogs and cats, accounting for less than 1% of canine tumours. Of the gastric tumours, malignant adenocarcinoma is the most common in dogs and comprises 47 to 72% of gastric malignancies. Many British clinicians believe that the prevalence may be higher in the United Kingdom, although that has not been documented. While gastric adenocarcinomas or adenomas are the most common gastric tumour in dogs, leiomyomas and lymphosarcoma, as well as leiomyosarcoma, polyps, fibromas, fibrosarcomas, squamous cell carcinomas and plasmacytomas also occur. Some of these tumours have a better outcome than gastric adenocarcinomas, so the tumour type should be characterized. Primary gastric adenocarcinomas are frequently located on the incisura angularis of the lesser curvature (as was this one) or the pyloric antrum of the stomach, although gastric ulcers from other causes may occur here as well. There are three morphological patterns of distribution of gastric carcinomas: diffusely infiltrating non-ulcerating lesions that involve most of the stomach that cause a tough, rigid (leather bottle) appearance, localized, raised thickened plaques usually containing a raised central ulcer (as in this case) and raised, polypoid, sessile lesions projecting into the lumen of the stomach.

As with many tumours, primary gastric tumours occur more frequently in older dogs, usually around 8 to 10 years of age with a peak incidence of gastric carcinomas at 11 to 12 years of age, similar to this patient. Male dogs are affected more frequently than female dogs and a higher risk has been suggested for Rough collies, Staffordshire terriers and Belgian shepherd dogs.

About 70% of gastric tumours are malignant and these spread to the regional lymph nodes, liver and lungs. Before surgical treatment is undertaken, the lungs should be checked for evidence of metastasis.

In humans, diet is thought to influence the development of gastric tumours. The prevalence of the tumours in dogs is so low that dietary influences would be difficult to study. Gastric tumours in humans are also related to the presence of *Helicobacter* spp., but no association with this has been made in dogs.

Prognosis

The prognosis for most patients with malignant gastric cancer is poor. Even in those dogs in which surgery can be performed, most patients are dead within 6 months due to recurrent or metastatic cancer.

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19 Pancreatitis in a cat

INITIAL PRESENTATION

Vomiting, poor appetite and lethargy

Signalment: 14-year-old male neutered Burmese cat, body weight 4.4 kg

CASE HISTORY

The cat had been vomiting for 2 days and was refusing to eat. The owner had tried to encourage him to eat, but he refused any type of food. He was very lethargic and the owner thought that he had been urinating larger volumes than usual because the litter box was very wet and she had observed this cat in the litter box frequently.

The owner had owned the cat since he was a kitten. He lived with a littermate sibling, who was clinically healthy. Both the cats had been vaccinated approximately 18 months earlier, but had not been treated for endoparasites for several years. They were both primarily indoor cats, although occasionally had access to a fenced garden. Neither cat hunted nor scavenged food. They did play with toys, although the patient had arthritis in both elbows, which decreased his mobility. His previous diet was a combination of a commercial dry cat food and a commercial canned cat food.

PHYSICAL EXAMINATION

Physical examination revealed the cat to be quiet and dull, but responsive. He was in slightly overweight body condition (body condition score 6/9). His oral mucous membranes were mildly jaundiced and tacky, with a capillary refill time of 2 seconds. He appeared approximately 7 to 8% dehydrated.

The sclera was mildly jaundiced bilaterally with no other ocular changes noted. Examination of the oral cavity, ears and nose showed no abnormalities. All peripheral lymph nodes were within normal limits. Auscultation of the lung fields as well as compression and percussion of the thorax were normal. The respiratory rate was elevated at 60 breaths per minute. Cardiac auscultation revealed a heart rate of 180 beats per minute with matched pulses. Rectal temperature was 39.1° C. Abdominal palpation indicated marked cranial abdominal pain. The reminder of the physical examination was unremarkable.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The cat's problems included vomiting, poor appetite and lethargy. The poor appetite and lethargy were thought to be related to the cause of the vomiting. He also was suspected to have polyuria, although it was possible that the other cat in the household had increased urine output. He was also jaundiced.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for vomiting

- Dietary sensitivity (allergy or intolerance)
- Dietary indiscretion
- Stomach
  - gastritis
  - gastric ulcers
  - inflammatory bowel disease
  - foreign body
  - parasites
  - neoplasia
- Small intestinal disease
  - infectious, e.g. bacterial, viral, fungal
  - gastrointestinal parasites
  - inflammatory bowel disease

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  - infiltrative neoplasia, e.g. lymphoma, mastocytosis
  - high partial obstruction, e.g. intussusception, foreign body
- Systemic disease
  - uraemia
  - liver failure
  - sepsis
  - congestive heart failure
  - acidosis
  - hypoadrenocorticism
  - ketoacidotic diabetes mellitus
  - hyperparathyroidism
  - gastrinoma
- Neurological diseases (no other signs consistent with these in this cat)
  - dysautonomia
  - vestibular disease
  - CNS disease
- Drugs, toxins (no known history of drug or toxin exposure)

Differential diagnoses for polyuria

- Diabetes mellitus
- Acromegaly (usually concurrent with diabetes mellitus in cats)
- Chronic kidney disease (sometimes acute renal failure) – the signs were too acute for chronic, unless the owner had not observed a gradual increase in urine output
- Pyelonephritis
- Electrolyte imbalances, e.g. hypercalcaemia, hypokalaemia, hyperkalaemia
- Hyperthyroidism
- Liver disorders
- Polycythaemia
- Drugs (no history of or known access to drugs or medications)

Other causes of polyuria/polydipsia, which are uncommon to extremely rare in cats, include:

- Hyperadrenocorticism
- Primary hyperaldosteronism
- Hypoadrenocorticism
- Tubular disorders (e.g. renal glucosuria)
- Central diabetes insipidus
- Psychogenic polydipsia

Differential diagnoses for jaundice

These include pre-hepatic disorder, hepatic disorders and post-hepatic disorders.

- Pre-hepatic jaundice is caused by intra- or extravascular haemolysis
- Hepatic causes of jaundice are due to liver disease, e.g.
  - cholangiohepatitis complex
  - hepatic lipidosis
  - diffuse neoplasia
  - feline infectious peritonitis
  - toxins, e.g. paracetamol (acetaminophen)
  - cirrhosis
  - idiopathic drug reactions
- Post-hepatic causes of jaundice are caused by biliary obstruction or rupture, e.g.
  - pancreatitis
  - enteritis
  - biliary neoplasia
  - calculus
  - ruptured bile duct or gallbladder

CASE WORK-UP

Intravenous fluid therapy with crystalloids at a rate to correct 8% dehydration was initiated.

Minimum data base

Haematology revealed a mild leukocytosis due to mild neutrophilia (13.52×10^9/l; reference range: 2.5–12.5 × 10^9/l), a left shift, with 1.5×10^9/l band neutrophils and moderate monocytosis (3.07×10^9/l; reference range: 0.15–1.7 × 10^9/l). A mild normocytic, normochromic anaemia was present (PCV: 0.237 l/l; reference range 0.30–0.45 l/l). A blood smear analysis revealed the presence of toxic neutrophils and reactive lymphocytes.

Serum biochemistry revealed mild hypoalbuminaemia (20 g/l; reference range 23–39 g/l) and hyperbilirubinaemia (20 μmol/l; reference range 0–15 μmol/l), elevated alanine aminotransferase (ALT) of 104 IU/l (reference range 6–83 IU/l) and moderate hypokalaemia (2.0 mmol/l; reference range 2.9–4.2 mmol/l). Alkaline phosphatase (ALP) was within the reference range at 35 IU/l (reference range 10–100 IU/l). Serum urea and creatinine were both elevated at 17.2 mol/l (reference range 2.9–9.8 mmol/l) and 161 μmol/l (reference range 40–177 μmol/l). Hyperglycaemia (26.9 mmol/l; reference range 3.94–8.83 mmol/l) was also present and serum fructosamine was 471 μmol/l (reference range 159–295 μmol/l).

Cholesterol was mildly increased at 5.5 mmol/l (reference range 2.0–3.4 mmol/l) and triglycerides were increased at 3.3 mmol/l (reference range 0.57–1.14 mmol/l).

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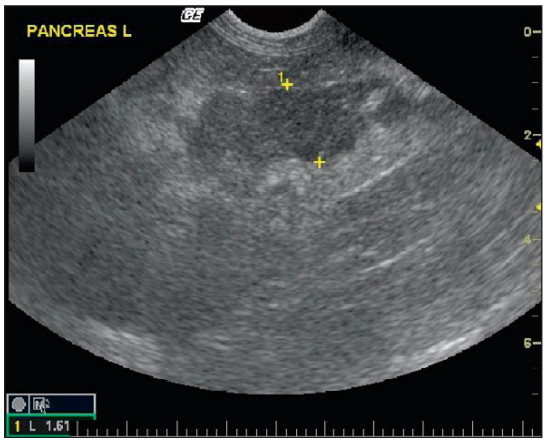


Fig 19.1 Ultrasound picture of pancreas showing uneven appearance (courtesy of Carolina Urraca del Junco)

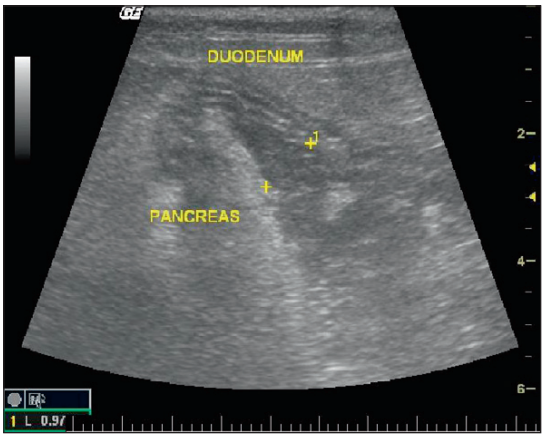


Fig 19.2 Ultrasound picture of duodenum showing corrugated appearance due to pancreatitis (courtesy of Carolina Urraca del Junco)

Urinalysis revealed a urine specific gravity (USG) of 1.014 and dip stick analysis was 1+ positive for protein, 2+ positive for blood, bilirubin and leukocytes and 3+ positive for glucose. Ketone bodies were not detected. Sediment examination showed the presence of occasional red blood cells, numerous white blood cells and bacteria (cocci) and a sample submitted for bacterial culture was later found to be positive for *Staphylococcus.*

The cat was diagnosed at this point with diabetes mellitus and a urinary tract infection. The azotaemia present was thought to be due either to pre-renal causes (dehydration) or kidney disease or a combination of both.

Imaging

Abdominal ultrasonography revealed an enlarged hypoechoic pancreas surrounded by a small amount of free abdominal fluid (Fig 19.1). The duodenum appeared corrugated (Fig 19.2), which is often seen with pancreatitis. The liver appeared hyperechoic, but was homogenous and normal in size. There was loss of demarcation between cortex and medulla of both kidneys (Fig 19.3). The right kidney was of similar appearance and contained a small cyst within the cortex. A large amount of sediment was seen in the urinary bladder.

The enlarged hypoechoic pancreas with free abdominal fluid was considered the clinically most significant finding and was consistent with pancreatitis, with evidence of chronic kidney disease or renal age related changes. The changes in the liver may have been due to a degree of hepatic lipidosis, which is often seen with diabetes mellitus. The small cyst in the right kidney was thought to be an incidental finding.

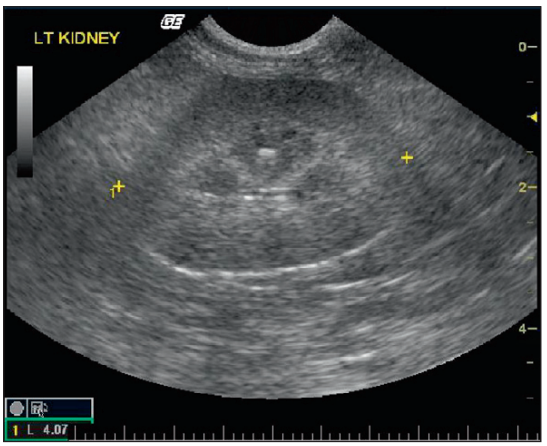


Fig 19.3 Ultrasound of kidney showing decreased corticomedullary junction (courtesy of Carolina Urraca del Junco)

Further testing

Serum was submitted for a feline pancreatic lipase immunoreactivity (fPLI) test and cobalamin concentrations. The fPLI was increased at 16.9 µg/l (reference range 0.1–3.5 µg/l), consistent with a diagnosis of pancreatitis. Serum cobalamin concentration was within the reference range at 946 ng/l (reference range 290–1499 ng/l).

The elevated fPLI plus the ultrasonographic changes in the pancreas confirmed an additional diagnosis of pancreatitis. The jaundice was thought to be post-hepatic in origin and due to the pancreatitis.

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CLINICAL TIP ON SERUM COBALAMIN IN CATS

Cobalamin is absorbed in the ileal part of the small intestine and requires prior binding with intrinsic factor, which comes from only the pancreas in cats, while in dogs it comes from both the stomach and pancreas. Because of this, cats with pancreatitis are at risk for hypocobalaminia. Other causes of cobalamin deficiency include poor absorption due to intestinal disease, exocrine pancreatic insufficiency, excessive bacteria in the upper small intestine utilizing the vitamin or a deficiency in intrinsic factor.

Treatment and outcome

The cat was stabilized with intravenous fluids (0.9% saline supplemented with potassium chloride at a rate to correct dehydration). Analgesia was provided using buprenorphine (initially at 20 μg/kg iv q 8 hours, later 10 μg/kg iv q 8 hours). Maropitant was administered as an anti-emetic (1 mg/kg sc q 24 hours) and ranitidine (2 mg/kg sq q 12 hours) was used as an antacid and promotility agent. Clavulanate-potentiated amoxicillin (20 mg/kg iv q 4 hours for 2 days, then 15 mg/kg po q 12 hours) and clindamycin (5.5 mg/kg po q 12 hours) were administered. He also received 1 IU of Caninsulin subcutaneously every 12 hours.

CLINICAL TIP ON MAROPITANT USE IN CATS

While maropitant is not approved in cats (at the time of writing), studies have shown that at 1.0 mg/kg it is highly effective in preventing motion-induced emesis in cats. These studies indicate that the NK-1 receptor antagonist maropitant is well tolerated, safe and has excellent anti-emetic properties in cats.

The cat responded very well to this treatment. His demeanour improved and there was no longer evidence of abdominal pain. He accepted syringe feeding very well and no further vomiting was noted.

Repeat measurements of electrolytes were performed and showed improvement and resolution of the hypokalaemia. Blood glucose levels ranged from 10.9 to 18.5 mmol/l.

The cat was discharged on 1 IU of Caninsulin sq q 12 hours, a high protein, low carbohydrate diet and 2 weeks worth of the antibiotics. At a re-visit 1 week later, his blood glucose was 11.2 mmol/l and other haematological and most serum chemistry parameters had improved, although a moderate azotaemia was still present, with a urea of 14 mmol/l and a creatinine of 132 μmol/l. His USG was 1.016, there were no ketones present and the sediment was 'inactive'. A repeated urine culture was negative.

NURSING TIP ON EXAMINATION OF URINE SEDIMENT

To check urine sediment, centrifuge the urine sample for 3 to 5 minutes at 2000 to 3000 rpm. Remove all but a small volume (0.25–0.5 ml) with a pipette or by pouring off the fluid on top. Re-suspend the sediment in the remaining supernatant by gently tapping the tube with your finger. Overzealous shaking may break cells and casts. Transfer a drop of the re-suspended sediment to a microscope slide and cover it with a cover slip. Decrease the microscope light intensity by lowering the condenser and partially closing the diaphragm. Scan the entire sample via the low power objective and then record the average number of elements (e.g. cells, crystals, casts, bacteria) per several high-power fields. Use of stains on wet preparations is by personal preference. Air-dried urine sediment slides may be stained for additional microscopic evaluation.

Urine collected by cystocentesis normally contains only a few cells and sometimes some struvite or oxalate crystals. When there is nothing abnormal in the sediment it is sometimes termed 'inactive'. Cells seen in healthy animals include zero to three white blood cells and zero to three red blood cells per high powered field and a small number of epithelial cells. A few struvite or oxalate crystals may also be seen in the urine of healthy animals and entire male animals may have some sperm in their urine. Abnormal findings include seeing more white or red blood cells, more than two hyaline casts per high powered field, any bacteria (from a cystocentesis sample) or any other types of crystals.

The cat was monitored every 2 to 3 weeks for the next 6 months. His insulin requirements decreased over

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Fig 19.4 The patient at 16 years of age (2 years after diagnosis)

time and at the 6 month re-visit appointment he no longer required insulin injections. He was relatively healthy without recurrence of pancreatitis or diabetes mellitus (Fig 19.4) until 18 years of age when he was euthanized due to the effects of chronic kidney disease (CKD) and debilitating arthritis.

NURSING TIP ON NUTRITION

This cat's dietary requirements were complicated. High protein diets have been recommended for cats with diabetes, however, as high protein diets are generally high in phosphate and he may have had early chronic kidney disease (CKD), this possibly had to be used with caution. Monitoring his serum phosphorus would be a good indicator for adjusting the diet or addition of a phosphate binder to his diet.

A low fat diet was indicated as he was overweight and extra body weight contributes to insulin resistance and aggravates arthritis. Losing weight is extremely difficult on a high fat diet. With the possibility of early CKD, the use of non-steroidal anti-inflammatory drugs (NAIDs) is also questionable, so he was treated with glucosamine and omega-3 fatty acids. Omega-3 fatty acids have been shown in the dog to decrease the required dose of NSAIDs and also appear to be beneficial in other inflammatory states. Glucosamine has been reported to increase insulin resistance; however, this is usually not a clinical problem as the doses used in the rodent trial showing this were much higher than used clinically.

DISCUSSION AND EPIDEMIOLOGY

This cat's combination of urinary tract infection and pancreatitis may have predisposed him to developing diabetes mellitus. He was also overweight, which increases the risk, and a Burmese cat. Burmese cats in the United Kingdom, New Zealand and Australia have a much higher risk of developing diabetes mellitus than other breeds. The development of diabetes mellitus implies that there was possibly chronic inflammation of the pancreas, which had been destroying the function. Pancreatic inflammation is a not an uncommon finding in cats, with significant pancreatic lesions seen in 1.5 to 3.5% of cats at necropsy.

Pancreatitis often occurs in conjunction with diseases of other organs in cats including the liver, kidney, intestine, endocrine pancreas and lungs. In most cases of pancreatitis in cats, an underlying cause or instigating event cannot be determined, leading to classification as idiopathic. Abdominal trauma, infectious diseases (toxoplasmosis, liver flukes, FIP and FIV), lipodystrophy and organophosphate administration have been associated with feline pancreatitis, but appear to account for a very small percentage of cases. Hypercalcaemia precipitated pancreatitis in one experimental study in cats and aspirin induced pancreatic cell damage in another. Potential risk factors for pancreatitis in dogs, such as obesity, dietary indiscretion, a high fat meal, high fat diets and pre-existing endocrine diseases (diabetes mellitus, hyperadrenocorticism, hypothyroidism and hyperlipidaemia) do not appear to be risk factors in cats. It is not known whether dietary factors, e.g. a high fat diet, predispose to pancreatitis in cats.

Inflammatory bowel disease (IBD) may be an important risk factor for the development of pancreatitis (and inflammatory liver disease) in cats, although there was no evidence of that in this cat, other than the vomiting, which is the most common clinical sign of IBD in affected cats. Vomiting can raise intraduodenal pressure and predispose to reflux of enteric contents into the pancreatic duct. Cats may be more susceptible than dogs to pancreatic pathology as a result of such reflux because the feline sphincter of Oddi is a common channel for the pancreatic and biliary outflow at the duodenal papilla and cats have much higher numbers of bacteria in the proximal duodenum than dogs (10^8 vs. 10^4 organisms/ml).

Pancreatitis is classified as acute or chronic (relapsing). Acute pancreatitis is further sub-classified as acute necrotizing or suppurative. Pancreatic biopsy samples for

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histopathology are required for accurate classification and this cat, like many patients with pancreatitis, was not a good candidate for anaesthesia. Both acute and chronic pancreatitis may be involved in the development of transient or permanent diabetes mellitus in cats, as in this cat. Some cases of acute pancreatitis are associated with severe clinical syndromes including shock, disseminated intravascular coagulation and multi-organ failure, which he fortunately did not appear to develop.

No significant age or gender predisposition has been recognized in cats with pancreatitis. This varies from the 'fat, five and female' concept of pancreatitis in dogs. In cats, a wide age range for pancreatitis (5 weeks to 20 years) has been reported, but some authors suggest that cats older than 7 years are more likely to be affected.

Lethargy (86–100%) and partial to complete anorexia (95–97%) are the most common clinical signs in cats diagnosed with pancreatitis. All other clinical signs occur only sporadically. Classic signs of pancreatitis, such as vomiting and abdominal pain, which occur in dogs are usually absent in cats. Vomiting occurs in about 35% of cats compared to 90% of dogs, abdominal pain occurs in 25 to 52% of cats compared to 58% of dogs, diarrhoea in 15% of cats compared to 33% in dogs and fever in 7% of cats compared to 32% in dogs. Other reported clinical findings in affected cats include dehydration (92%), tachypnoea (74%), hypothermia (68%), tachycardia (48%) and dyspnoea (20%). Almost one-quarter (23%) of cats with pancreatitis have a palpable abdominal mass that may or may not appear painful and can be easily misdiagnosed as a lesion of other intra-abdominal structures such as intestinal tract or mesenteric lymph nodes. Tachypnoea or dyspnoea may occur due to pain, but pleural effusion and pulmonary thrombosis have been diagnosed concurrently with pancreatitis in cats and should not be overlooked as possible causes. In one study of 21 cats with pancreatitis, 66% had complicating conditions.

Diagnosis

The diagnosis of pancreatitis in cats can be challenging. Haematologic abnormalities are uncommon and non-specific and may include a mild anaemia that may be non-regenerative and a leukocytosis, possibly with a left-shift or toxic white cells. Other hematologic changes reflect fluid loss and haemoconcentration. Serum chemistry abnormalities include mild to moderate elevations in ALT, ALP and bilirubin and may reflect concurrent hepatic disease (hepatic lipidosis or cholangiohepatitis) or biliary stasis. Azotemia may be pre-renal, secondary to dehydration, in many cases, although underlying chronic kidney disease should also be considered. Hyperglycaemia is commonly seen in cats due to concurrent stress or diabetes mellitus. Hypocalcaemia is occasionally seen due to saponification of peripancreatic fat.

Changes on abdominal radiographs are often subtle and subjective. Decreased contrast in the anterior abdomen, dilated and gas filled small intestines and transposition of the duodenum, stomach and colon are commonly reported.

Abdominal ultrasound may reveal an enlarged hypoechoic pancreas surrounded by hyperechoic mesentery, with or without dilated pancreatic ducts. Ascites is occasionally observed. The sensitivity of ultrasound is obviously operator and equipment dependent, with reported sensitivities ranging from 35% to 75%. These results underscore the point that a normal ultrasound does not rule out pancreatitis. The clinician should also consider differential diagnoses aside from pancreatitis, including pancreatic neoplasia and pancreatic oedema (associated with hypoproteinaemia or portal hypertension). Computed tomography has not shown any benefit compared to ultrasound for the diagnosis of pancreatitis.

Serum lipase and amylase activities are nearly useless in the diagnosis of pancreatitis in cats, with serum concentrations being neither specific nor sensitive. Determination of serum trypsin-like immunoreactivity (fTLI) measures antibodies against circulating trypsin and trypsinogen. TLI is cleared by the kidneys, so elevations can occur with renal dysfunction. Increases in TLI, which can be quite high, often return to the reference range within 2 to 3 days.

fPLI is more sensitive than any other non-invasive test for the diagnosis of feline pancreatitis and is the test of choice for diagnosis. As it stays elevated while there is ongoing inflammation, it appears to be useful for both acute and chronic pancreatitis.

The theoretical 'gold standard' for diagnosing pancreatitis is pancreatic biopsy for histologic evaluation. Peripancreatic fat necrosis is a typical finding in cats with pancreatitis, with variable amounts of pancreatic acinar cell necrosis and inflammation. Chronic pancreatitis is characterized by interstitial fibrosis with acinar atrophy and lymphocyte infiltrates. The disease can have a 'patchy' or multifocal distribution and pancreatic biopsies should always be procured during laparotomy even if the gross appearance of the organ is normal. Unfortunately, the lack of pancreatic inflammation on a single biopsy does not rule out pancreatitis conclusively.

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Therapy

The old concept of nil per os or withholding food from patients with pancreatitis has little evidence for effectiveness and in humans is thought to possibly have negative consequences for outcome. In cats that may have hepatic lipidosis, nutritional support is especially important. In cats that are not vomiting, feeding a palatable diet is recommended, although anorexia is a common problem. In cats that are not vomiting but still not willing to eat, placement of a nasogastric/nasooesophageal tube is a good alternative. There is no evidence that feeding a low fat diet is beneficial in cats with pancreatitis, so most liquid diets formulated for cats can be used. Anti-emetics are also useful and may also decrease the nausea associated with pancreatitis in cats that are not eating even though they are not vomiting. In cats that are vomiting, ideally a jejunostomy tube would be placed for nutritional support; however, due to the anaesthesia required for this procedure this is often not feasible.

Abdominal pain may be present in cats with pancreatitis even if the cat is not showing obvious signs. Cats often mask signs of pain. Routine analgesia is recommended, as the previous concerns that morphine and related drugs may exacerbate pancreatitis have been discounted.

In dogs, intravenous plasma has been recommended to replete the alpha-macroglobulin, a scavenger protein for activated proteases, which is depleted in pancreatitis. While in humans, the use of plasma has not shown improved survival, many clinicians believe that it is useful. Fresh frozen plasma can be used if available or whole blood (after blood typing the cat and using matched blood) may be used if it is not.

The use of antibiotics in pancreatitis is controversial. There is no evidence that infectious agents cause pancreatitis, however, many clinicians believe in using antibiotics, especially in cats. One rationale is the potential for bacterial infection of the biliary system and pancreatic duct because of the direct communication of the common bile duct and pancreatic duct in the cats. Some clinicians also feel that corticosteroids are useful in cats with chronic (but not necessarily acute) pancreatitis, but this is also controversial, especially in a case with concurrent diabetes mellitus.

Prognosis

The prognosis for cats with pancreatitis depends upon the severity of the disease and the extent of any pancreatic necrosis, the complications and the presence of concurrent disease. Some chronic cases are subclinical and may flare up from time to time. Some patients with chronic disease may develop diabetes mellitus, as did this case, and some have also been reported to develop exocrine pancreatic insufficiency, although the incidence of these sequelae has not been reported. In cats which develop diabetes mellitus, a percentage of them are able to eventually maintain their blood glucose without insulin, a state termed diabetic remission. Some of these will relapse and need insulin, but may be able to achieve another remission with insulin therapy and careful monitoring.

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20 Linear foreign body in a cat

Case co-written by Carolina Urraca del Junco

INITIAL PRESENTATION

Vomiting

Signalment: 9-month-old neutered male domestic shorthaired cat body weight 3.3 kg

CASE HISTORY

The cat had been vomiting for 5 days. The vomitus initially had contained food and then contained primarily liquid. The cat was not eating and had reportedly lost weight. There had been no diarrhoea, although he had not passed faeces for 2 days.

He had been given his vaccinations as a kitten and had last been de-wormed 4 months previous to presentation. He had not been given any treatments for ectoparasites. His usual diet was canned cat food, plus cooked ham, chicken and tuna.

Previous treatment had included intravenous fluids, maropitant, clavulanate-potentiated amoxicillin and ranitidine. After this treatment, he had initially vomited less and was brighter. A feline pancreatic lipase immunoreactivity test for pancreatopathy had been negative. He then vomited a large amount of greenish bile-stained fluid several times and became lethargic and was referred to a specialist centre.

CLINICAL TIP ON HISTAMINERGIC H_2 BLOCKERS

Ranitidine, unlike cimetidine, has a gastrointestinal promotility effect by inhibiting acetylcholinesterase inhibitors and is used in some cases for this effect. It also is reported to produce fewer adverse drug interactions, have less systemic toxicity and greater potency than cimetidine. Cimetidine is the only H_2 blocker which inhibits the P450 enzymes in the liver, causing the metabolism of other drugs to be decreased. Cimetidine induced leukopaenias and thrombocytopaenia have also been reported.

CLINICAL TIP ON MAROPITANT USE IN CATS

While maropitant is not approved in cats (at the time of writing), studies have shown that at 1.0 mg/kg it is highly effective in preventing motion-induced emesis in cats. These studies indicate that the NK-1 receptor antagonist maropitant is well tolerated, safe and has excellent anti-emetic properties in cats.

PHYSICAL EXAMINATION

On physical examination the cat was quiet but alert. He was in slightly thin body condition (body condition score 4/9) and appeared well hydrated. His mucous membranes were pink and his capillary refill time was less than 2 seconds. Thoracic auscultation revealed a heart rate of 185 beats per minute with matched pulses. Heart and lung sounds and percussion and compression of the thorax were unremarkable. The cat appeared to be uncomfortable on abdominal palpation. His respiratory rate was normal at 32 breaths per minute. Rectal temperature was 38.9° C.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The cat's primary problem was vomiting; the decreased appetite, weight loss and abdominal discomfort were thought to be related to the vomiting.

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DIFFERENTIAL DIAGNOSIS

Differential diagnoses for vomiting in this cat included:

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilation-volvulus
  - neoplasia
- Disorders of the small intestine
  - foreign body
  - inflammatory bowel disease
  - neoplasia
  - parasites
  - intussusception
- Disorders of the large intestine (although there was no history consistent with large intestine disorders)
  - colitis
  - obstipation
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism
  - diabetes mellitus
  - liver disorders
  - peritonitis
  - renal disease/uraemia (unlikely)
- Dietary causes
  - dietary sensitivity
  - dietary indiscretion

CASE WORK-UP

As this cat was clinically unwell, a dietary food trial was not appropriate.

Minimum data base

Haematology results were within the reference ranges. Serum chemistry results showed decreased potassium of 3.0 mmol/l (reference range 3.5–5.8 mmol/l). Other serum chemistry parameters were within the reference ranges. Urine was not obtained at admission. Intravenous fluid therapy at a rate of 2 ml/kg/hour, with potassium added at 12 mmol/l of fluids, was initiated.

NURSING TIP ON INTRAVENOUS FLUIDS THERAPY AND POTASSIUM SUPPLEMENTATION

Moderate or severe hypokalaemia (≤3.0 mmol/l) requires parenteral administration of potassium chloride (KCl) for correction. It is usually recommended that the rate of administration does not exceed 0.5 mmol/kg body weight per hour (1 mEquiv of KCl = 1 mmol KCl). Potassium supplementation is contraindicated in oliguric or anuric states as the kidneys are unable to excrete it. Guidelines for estimating the dose of KCl to add to intravenous fluids are listed in Table 20.1. Remember that Ringer's solution and lactated Ringer's contain 4 mmol/l of KCl.

Imaging

Abdominal radiographs showed a small intestinal corrugation cranial to the urinary bladder, at the level of the 5th to 6th lumbar vertebrae. There was an abnormal plicated appearance to the small intestine ventral to the descending colon, cranial to and overlying the bladder shadow (L4–6 level) and a small focus of gas within the small intestinal lumen suggested a linear foreign body (Fig 20.1).

Abdominal ultrasound also showed plication of the descending duodenum with a linear foreign body evident in the lumen of the duodenum (Fig 20.2). The small intestinal motility appeared to be increased.

Table 20.1 Amount of potassium chloride (KCl) to add to crystalloid fluids based on the serum potassium (K^+) concentration of the patient. Remember to subtract the amount already in the fluids.

Serum K^+ of the patient in mmol/l	mmol KCl to add per 250 ml of fluids	mmol KCl to add per litre of fluids
>3.5	5	20
3.0–3.5	7	28
2.5–3.0	10	40
2.0–2.5	15	60
2.0	20	80

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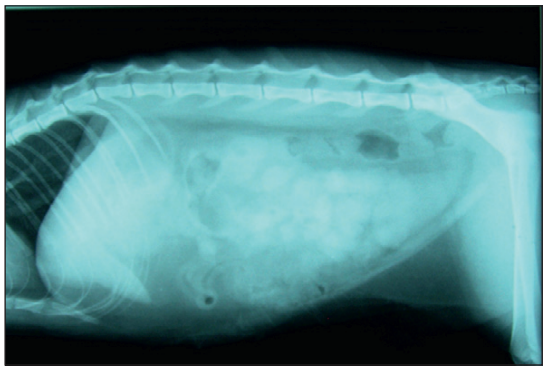


Fig 20.1 Lateral abdominal radiograph showing small intestine bunched mid-abdomen (courtesy of Carolina Urraca del Junco)

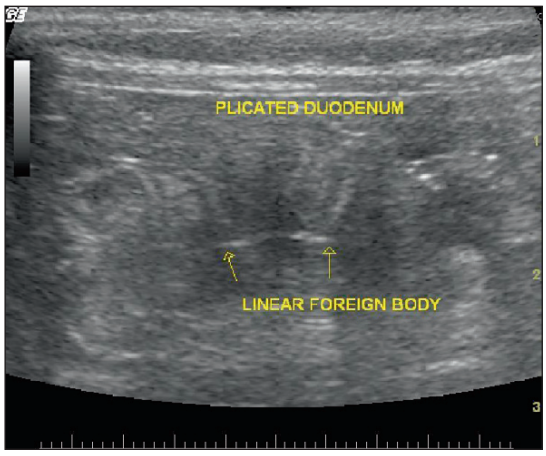


Fig 20.2 Ultrasound picture showing the plication of the small intestine around a linear foreign body (courtesy of Carolina Urraca del Junco

Treatment

Surgical treatment

The cat was referred to the surgery service for management of a suspected linear foreign body. At surgery, the linear foreign body (thread) was removed by one gastrotomy and four enterotomies. During these procedures, one area of the jejunum perforated at the mesenteric border where the thread had been cutting into the mucosa. The distal two enterotomy sites were excised with adjacent intestine and an end-to-end anastomosis was performed. A more proximal area which appeared to have some compromise to the serosal surface near an enterotomy site had a serosal patch applied.

Follow-up medical treatment

Immediate postoperative treatment included intravenous clavulanate-potentiated amoxicillin (20 mg/kg iv q 8 hours), analgesia with buprenorphine (20 μg/kg iv q 8 hours), ranitidine (2 mg/kg SQ q 12 hours) for its antacid and promotility effects and intravenous fluids.

Outcome

At 24 hours postoperatively the cat became lethargic, pyrexic and had vomited. A guarded prognosis was given as it was suspected that the patient had injuries to the mucosa along much of his intestinal tract. However, by 4 days post surgery, he was bright and alert and was drinking and eating without assistance. He did not pass faeces until 5 days post-surgery, but they were normal at that time. His treatments were changed to oral medications and he was discharged on 1 week of oral ranitidine (2 mg/kg po q 12 hours) and clavulanate potentiated amoxicillin (15 mg/kg po q 12 hours). He has continued to do well.

DISCUSSION

Often, linear foreign bodies such as pieces of string, cord or fabric will pass through the intestines with few clinical signs; however, if one part lodges within the intestine an obstruction and potentially a perforation will result. Linear foreign bodies most often lodge in the oral cavity or in the pylorus. The free end may eventually come lose from the oral cavity or pylorus and travel to the small intestine. Within the intestine the foreign body causes the intestine to be pleated or bunched up around it. The peristalsis of the intestine causes the pleating. The linear foreign body then abrades the wall of the intestinal loops and eventually multiple perforations and peritonitis occurs.

Linear foreign bodies are more common in cats than in dogs, likely due to cats' fascination with string and similar objects. Clinical signs include anorexia, vomiting and abdominal pain, as in this young cat and sometimes diarrhoea. If the obstruction is complete, the signs will be acute. If it is a partial obstruction, signs may be more subtle and last for weeks before diagnosis. On palpation of the abdomen, bunched loops of bowel may be felt.

Imaging is often diagnostic as linear foreign bodies produce characteristic signs. On survey radiographs the affected loops of intestine appear gathered or clumped

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and may be situated to one side of the abdomen. Contrast studies may show the pleating of the intestine. Linear foreign bodies can be difficult to identify on ultrasound, but the corrugated appearance of the intestine may be seen. The small intestinal loops of the intestine proximal to an obstruction may be dilated. In this case the foreign body was actually visible.

In some cases where a linear foreign body is looped around the tongue, it can be cut and allowed to pass through the intestines, which will usually take 1 to 3 days. This conservative management is contraindicated if the cat shows any gastrointestinal signs or depression or if the cat cannot be monitored very carefully. In one study, 9 out of 24 cats were successfully managed conservatively, with passage of the linear foreign body occurring 1 to 3 days after presentation. The cats that were managed successfully via conservative treatment had a shorter duration of clinical signs before presentation, decreased incidence of abdominal pain, less palpable intestinal plication and less severe haematological abnormalities.

Surgery is indicated if there is plication of the intestines or any other indication that the foreign body is not moving through the intestines without damage. At surgery, multiple enterotomies are often necessary to remove the foreign body. Perforated areas of the intestine are repaired and treatment with broad spectrum antibiotics is indicated.

Prognosis

In one study on linear foreign bodies in cats, surgery (gastrotomy and multiple enterotomies) was necessary in 96.9% of the cases and most (83.9%) of the cats so treated did well. Approximately 50% of the cats with linear foreign body-induced bowel lacerations recovered.

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21 Porto-systemic shunt in a dog

INITIAL PRESENTATION

Vomiting and diarrhoea

Patient details: 14-month-old female entire Cairn terrier, body weight 5.7 kg

CASE HISTORY

The dog had a history of vomiting digested food about an hour after eating. The vomiting had been occurring for about a month. Because she did play with sticks the owners had been concerned about a possible gastrointestinal foreign body.

She had also had intermittent diarrhoea for the past 6 months. Her stools were occasionally soft, with no blood or mucous present and no straining to defecate. This was thought to most likely be diarrhoea of small intestinal origin.

The owners also reported weight loss over the past several months and episodes of staring vacantly or pacing. When put in her pen in this state she became agitated and starting biting the wires. These episodes lasted about 9 to 10 hours and started post-prandially. The dog was fed a canned specialty puppy food twice a day. She had always been a picky eater and the owners often encouraged her to eat with treats and human food.

The owners did not report that the dog had polydipsia or polyuria, but she had been difficult to house train. She had been an active puppy, but had been quieter during the last couple of months. The dog was dewormed regularly with fenbendazole and had been vaccinated as a puppy.

She had been to the veterinary surgery previously for the vomiting and had been administered intravenous fluids. She responded to this treatment temporarily, but the vomiting reoccurred the next day after she went home.

PHYSICAL EXAMINATION

On physical examination the dog was alert but quiet. She was in thin body condition with a body condition score of 2/9, but appeared to be adequately hydrated. Oral examination identified an undershot jaw with laterally displaced upper canines. The tongue showed evidence of previous trauma with a V-shaped split to the tongue tip and an erythematous, horseshoe-shaped scar. Her mucous membranes were pink and moist and her capillary refill time was 2 seconds.

Thoracic auscultation was unremarkable. On abdominal palpation the liver could not be identified, but the caudal pole of the left kidney could be palpated easily. Her small intestines were easily palpated and felt normal. She did not show any evidence of discomfort during palpation. Her heart rate was 120 beats per minute, respiratory rate 24 breaths per minute and rectal temperature was 38.7° C.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Vomiting
- Diarrhoea
- Decreased exercise tolerance
- Episodes of abnormal behaviour.

The decreased exercise tolerance was thought to be related to the other disorders and was not addressed separately.

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DIFFERENTIAL DIAGNOSIS

For the vomiting

- Disorders of the stomach
  - foreign body
  - gastritis
  - ulceration
  - chronic partial dilatation-volvulus (less likely due to the breed and body shape of the dog)
  - neoplasia (less likely, although lymphoma can occur in younger animals)
- Disorders of the small intestine
  - foreign body
  - parasites
  - intussusception

The small intestinal disease differentials below were possible but less likely due to the dog's young age:

  - inflammatory bowel disease
  - neoplasia
- Disorders of the large intestine
  - colitis
- Systemic disorders
  - liver disorders, including porto-systemic shunt
  - pancreatopathy

The differentials below were possible but less likely due to the dog's age:

  - hypoadrenocorticism
  - diabetes mellitus
  - renal disease/uraemia
- Dietary causes
  - adverse reaction to food (allergy or intolerance)
  - dietary indiscretion

For the chronic small intestinal diarrhoea

- Dietary
  - adverse reaction to food: intolerance or allergy
  - gluttony – unlikely in this picky dog
  - changes of diet
- Gastric disorders, rapid gastric emptying
- Small intestinal disease
  - infectious, e.g. bacteria, viral, fungal
  - partial obstruction, e.g. intussusception, foreign body
  - gastrointestinal parasites
  - brush border defects
- Less likely small intestinal disorders due to her age and clinical presentation:
  - inflammatory bowel disease
  - infiltrative neoplasia (e.g. lymphoma, mastocytosis)
  - lymphangiectasia
- Pancreatic disease
  - chronic pancreatitis
  - exocrine pancreatic insufficiency
  - pancreatic neoplasia (less likely due to age)
- Liver disease
  - porto-systemic shunt
  - bile duct obstruction
  - liver failure
- Kidney disease
- Miscellaneous
  - congestive heart failure – no signs consistent with this
  - immunodeficiencies
  - hypoadrenocorticism (less likely at this young age)

As the dog showed no other neurological signs, the abnormal behaviours were most likely to be cerebral in origin, either from the cerebrum itself or extra-cranial disorders affecting brain function. A behavioural problem was also possible but less likely.

For the abnormal behaviours

- Primary brain disorders include:
  - congenital diseases such as malformation of the brain or Chiari-like malformation
  - hydrocephalus
  - inborn errors of metabolism
  - granulomatous meningoencephalomyelitis
  - intracranial or brain tumours
  - epilepsy
- Systemic disorders affecting brain function include:
  - Porto-systemic shunts (PSS) causing signs of hepatic encephalopathy (HE)
  - hypoglycaemia
  - toxins
  - nutritional deficiencies or imbalances

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CASE WORK-UP

The dog was admitted to the hospital for diagnostic investigation.

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed. Results of routine haematology showed a slightly decreased packed cell volume (PCV) of 0.35 l/l (reference range 0.37–0.55 l/l) due to a microcytosis with an mean corpuscular volume (MCV) of 52% (reference range 60–77%). The red blood cell count was within the norm at 5.6 × 10^{12}/l (reference range 5.5–8.5 × 10^{12}/l) and haemoglobin was slightly low at 11.8 g/dl (reference range 12–18 g/dl).

CLINICAL TIP ON HAEMATOLOGY

When the PCV is decreased, check the cell size, i.e. the mean corpuscular volume (MCV). Microcytosis can decrease the PCV while the number of red blood cells stays within the reference range. Microcytosis can occur with iron loss (e.g. with gastrointestinal bleeding), with iron sequestration (as occurs with PSS or anaemia of chronic inflammation) and in some normal Akita dogs.

Serum biochemistry results showed a moderate increase in alanine aminotransferase of 233 IU/l (reference range 21–102 IU/l) and in alkaline phosphatase (ALP) of 116 IU/l (reference range 20–60 IU/l). The fasting bile acids were markedly increased at 243 µmol/l (reference range 0–7 µmol/l) and post-prandial bile acids rose to 521 µmol/l. Serum albumin was at the lower end of the reference range at 22.3 g/l (reference range 26–35 g/l) and urea was also at the lower end of the reference range at 1.9 mmol/l (range 1.7–7.4 mmol/l).

Basal serum cortisol concentration was assessed to screen for hypoadrenocorticism and was within the reference range at 221 nmol/l (reference range 20–230 nmol/l). A coagulation profile was performed in case liver biopsies were required; the prothrombin time, activated partial thromboplastin time (APTT) and fibrinogen were all within reference ranges.

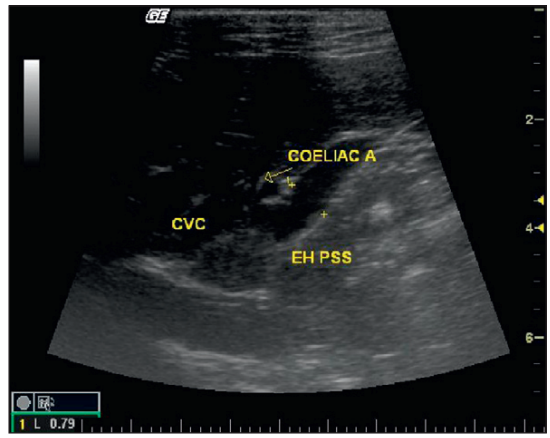


Fig 21.1 Ultrasound picture showing a large extrahepatic portocaval shunt (coelic a, coelic artery; EH PSS, extrahepatic porto-systemic shunt; CVC, caudal vena cava) (courtesy of Carolina Urraca del Junco)

CLINICAL TIP ON COAGULATION PROFILES IN DOGS WITH PORTO-SYSTEMIC SHUNTS

While this dog's coagulation profile was within the reference range, serious post-operative haemorrhage has been reported in dogs after closure of congenital porto-systemic shunts (PSS). Dogs with PSS often have lower platelet counts, lower activity of clotting factors II, V, VII and X, and increased factor VIII and APTT compared to healthy dogs. Surgical attenuation of the shunt results in increased frequency of abnormalities in coagulation times and factors immediately after surgery and there may be a risk of bleeding. Haemostasis does become normal in dogs where there is complete attenuation of shunting, in contrast to dogs with persistent shunting.

Urinalysis showed a urine specific gravity of 1.008. The chemical strip showed a pH of 6.5 and the sediment showed urate crystals.

Imaging

Abdominal radiographs showed a small liver size and the kidneys appeared to be large.

Abdominal ultrasonography supported the liver being small in size. The hepatic veins were small and a single extrahepatic PSS was identified, curving round the caudal pole of the right kidney and coursing craniodorsally to enter the caudal vena cava close to the entry site

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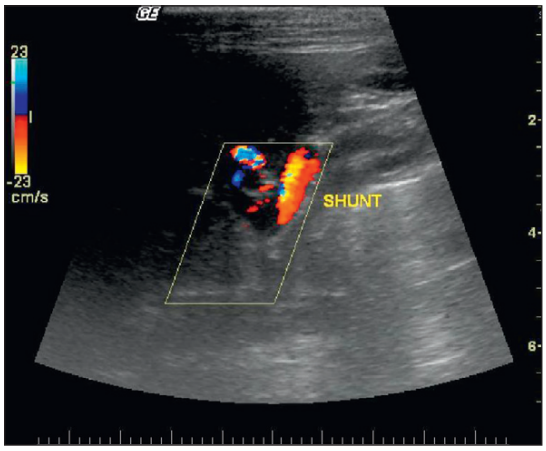


Fig 21.2 Ultrasound of colour Doppler showing blood flowing through the extrahepatic porto-systemic shunt (courtesy of Carolina Urraca del Junco)

of the right renal vein (Fig 21.1). Doppler ultrasound was used to confirm the presence of blood flow within the shunt (Fig 21.2). The kidneys were both enlarged at 6 cm and small calculi were identified within the urinary bladder; as there were urate crystals present in the urinalysis, these were presumed to be ammonium urate in origin.

CLINICAL TIP ON IMAGING FOR PSS

Computed tomography can be useful to better determine the location of a shunting vessel, but was not used in this case. Transplenic portal scintigraphy has also been used as a diagnostic aid in some cases. Many surgeons like the portogram to be performed prior to surgery as this gives them information about the location of the shunting vessel so they can plan their surgical approach.

Case assessment

From the history, clinicopathological results and imaging a diagnosis of extrahepatic PSS was made.

CLINICAL TIPS

Microcytosis can lower the PCV as the smaller cells result in a lower percentage of the blood being cellular. Causes of microcytosis include iron deficiency and PSS. The reason for the microcytosis in animals with PSS is thought to be due to iron sequestration or ineffective iron transport, which have been proposed as the causes of iron deficient erythropoiesis.

Serum urea is often at the low end of or below the reference range in animals with PSS as ammonia is not being efficiently converted to urea within the liver. Hypoglycaemia can be seen in some cases and should be addressed therapeutically.

Urate crystals are found in many dogs with PSS due to hyperammonaemia and a decreased ability of the liver to convert uric acid to allantoin.

The presence of urate crystalluria is never normal and should prompt the clinician to look for PSS or other hepatic disorders. Some Dalmatian dogs also have urate crystals in their urine due to an inborn metabolic defect and they may form urate uroliths.

Many animals with PSS also have a low urine specific gravity and may present for polyuria/polydipsia. This poor urine concentrating ability may be due in part to decreased medullary tonicity from the low urea, increased adrenocorticotrophin hormone (ACTH) secretion and hypercortisolism, and there also appears to be a possible decrease in the synthesis or release of antidiuretic hormone from the pituitary for reasons that are not fully elucidated.

NURSING TIP

As high protein diets can worsen clinical signs, animals with suspected PSS should be fed protein restricted diets, e.g. 15 to 20% protein on a dry matter basis or 2 g of protein/kg/day to decrease ammonia formation from colonic bacteria. If a puppy is still growing, commercial low protein diets designed to decrease the risk of HE may be too low in protein to support growth (22% protein on a dry matter basis). In that case, supplement the diet with cottage cheese at 100 g per 400 g of canned dog food. Cottage cheese is high in branched chain amino acids and has less tendency to cause encephalopathic problems than meat proteins. Vegetable proteins or soy proteins are also less likely to cause problems than meat proteins and can be used. These patients should also be fed small meals frequently, as that decreases the risk of hypoglycaemia.

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Initial (pre-surgical) medical management

Initial medical treatment to decrease signs of HE were started as soon as the diagnosis was made. This included dietary management as mentioned above plus oral lactulose syrup (1 ml po q 12 hours). The dose was adjusted so that the faeces were soft but formed, as an overdose can cause diarrhoea. Metronidazole was started at 7 mg/kg po q 12 hours.

CLINICAL TIP ON HE TREATMENT

Lactulose decreases intestinal transit time and acidifies colonic content which aids in trapping ammonia in the colon and decreasing absorption. In cases with severe signs of HE, lactulose enemas may be helpful. Oral antibiotics such as metronidazole, ampicillin or neomycin may be given to decrease intestinal urease-producing bacteria, although low dosages are recommended, e.g. 7 mg/kg metronidazole po q 12 hours.

One-third of animals do well with medical management alone, especially dogs which are older at diagnosis; however, over half of dogs treated with medical management alone undergo euthanasia within the first year after diagnosis.

This patient was sent home for 3 weeks on a low protein diet with cottage cheese supplementation, lactulose at 2 ml po q 12 hours and ampicillin (10 mg/kg po q 8 hours) to stabilize her HE and improve her body condition prior to surgery. She was then readmitted for placement of an ameroid constrictor on the shunting vessel and liver biopsy.

Surgical treatment

Surgical treatment is recommended in most patients with PSS to improve long-term outcome. There are several methods of occluding the shunt, including gradual occlusion with ameroid constrictors, cellophane banding, embolism with coils and suture ligation. The traditional surgical treatment involved placement of a ligature around the anomalous vessel, which is gradually tightened during the surgical procedure while measuring portal pressure and observing the splanchnic viscera. If the ligature is placed too tight, there is a significant risk of inducing portal hypertension, a severe and often fatal post-operative complication. Up to 68% of dogs with single extrahepatic PSS cannot be completely ligated and surgery results in partial shunt attenuation. Partial PSS attenuation is associated with an increased risk of persistence or recurrence of clinical signs. Recurrence of clinical signs is reported in 32 to 50% of partially ligated and 0 to 12% of completely ligated extrahepatic PSS. Therefore, gradual rather than acute occlusion of PSS is advocated as a surgical technique. Theoretically, this allows development of the portal circulation and hepatic architecture in response to the increased vascular supply, while reducing perioperative complications associated with portal hypertension.

The ameroid ring constrictor used in this case (Figs 21.3 and 21.4) is commonly used for gradual PSS



Fig 21.3 Ameroid constrictor (courtesy of Dr Donald Yool)

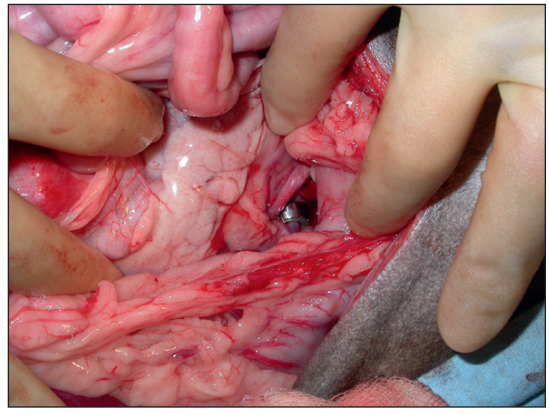


Fig 21.4 Ameroid constrictor in position in a dog with an extrahepatic porto-systemic shunt (courtesy of Dr Donald Yool)

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attenuation, although actually it produces a delayed acute reduction in blood flow associated with vascular thrombosis rather than the gradual attenuation it was originally thought to cause. The clinical results of ameroid constrictor placement in dogs with single extrahepatic PSS is much better than in dogs with intrahepatic PSS.

Post-operative care

Post-operative care included monitoring for hypothermia, hypoglycaemia and especially for signs of portal hypertension. Occlusion of the shunting vessels forces blood through the liver, opening previously closed vessels and some cases do not tolerate this very well. Signs of portal hypertension include an increasing abdominal effusion. If evidence of portal hypertension had been present, treatment with intravenous colloids would have been tried, although many of these dogs need to return to surgery to have the constriction lessened or removed.

Medical management was continued after surgery for 7 weeks; once serum bile acid concentrations were within the reference range, indicating improved liver function, the dietary protein was gradually increased.

NURSING TIPS ON DIET AFTER SURGERY FOR PSS

The adult canine requirement for protein is not high (18% dry matter basis), so it may be satisfactory for the dog to remain on a moderately restricted protein diet. Especially in dogs which continue to have some shunting of the vessel a moderately restricted protein diet may be beneficial.

Follow-up

The dog recovered well from the surgery, although she was rather lethargic for the first few days. Difficult or slow recovery from anaesthetic drugs is common in dogs with hepatic compromise.

The histopathology results for the liver biopsy taken at surgery showed periportal arteriolar and biliary duplication with lobular atrophy and lipogranulomas and marked diffuse hepatocellular fatty change. These changes are all consistent with a PSS and correlated well with the clinical and surgical findings. The degree of abnormality was thought to explain the poor body condition and neurological signs.

At the 7 week recheck the dog was very bright and there had been no further episodes of vomiting, diarrhoea or neurological problems. Haematological and serum chemistry parameters were within the reference ranges and at ultrasound scan the ameroid constrictor was still in place and occluding the vessel. The urate cystic uroliths were not detected on ultrasound. The restricted protein plus cottage cheese diet was continued while the lactulose and ampicillin were discontinued.

Pathophysiology

In the embryonic dog, the ductus venosus carries oxygenated placental blood to the circulation, bypassing the hepatic circulation. This vessel normally closes within the first 1 to 2 weeks after birth. Abnormal patency of this vessel or other abnormal vasculature formation connecting the portal venous system to the systemic circulation result in the signs seen in PSS. Congenital shunts are generally one vessel, whereas shunts acquired due to liver disease (e.g. cirrhosis and fibrosis) are more often multiple.

Common types of shunts include intrahepatic porto-caval shunts and extrahepatic porto-caval (Fig 21.5) or portal-azygos shunts.

The degree of change in the hepatic histology is probably a function of the amount of portal blood that is diverted from the liver and may vary between liver lobes depending of the site of the shunt vessel, particularly with intrahepatic shunting associated with a persistent ductus venosus. Typical histopathologic changes in the liver include arteriolar hyperplasia, loss of portal vein profiles and hepatocellular atrophy with increased lipogranuloma formation. Similar histopathological changes can also be found in cases of hepatic microvascular dysplasia, only in those cases no shunting vessel is found. Pathologic changes may be present in the central nervous system, especially in encephalopathic animals.

Since the liver relies on a substantial amount of nourishment from the portal circulation, decreased blood flow and subsequent decreased nourishment results in hepatic atrophy. Decreased insulin supply due to the shunting may also contribute to atrophy since insulin aids in the development of hepatic parenchyma. Atrophy of the liver further reduces hepatic function.

Gastrointestinal signs are seen in about 30% of dogs with PSS and include vomiting, diarrhoea and anorexia. Other signs often seen include poor growth and abnormal neurological behaviours, as in this case. High portal pressure is not usually a feature of PSS, so abdominal effusion does not usually develop unless the serum albumin is very low and a transudate forms due to decreased oncotic pressure.

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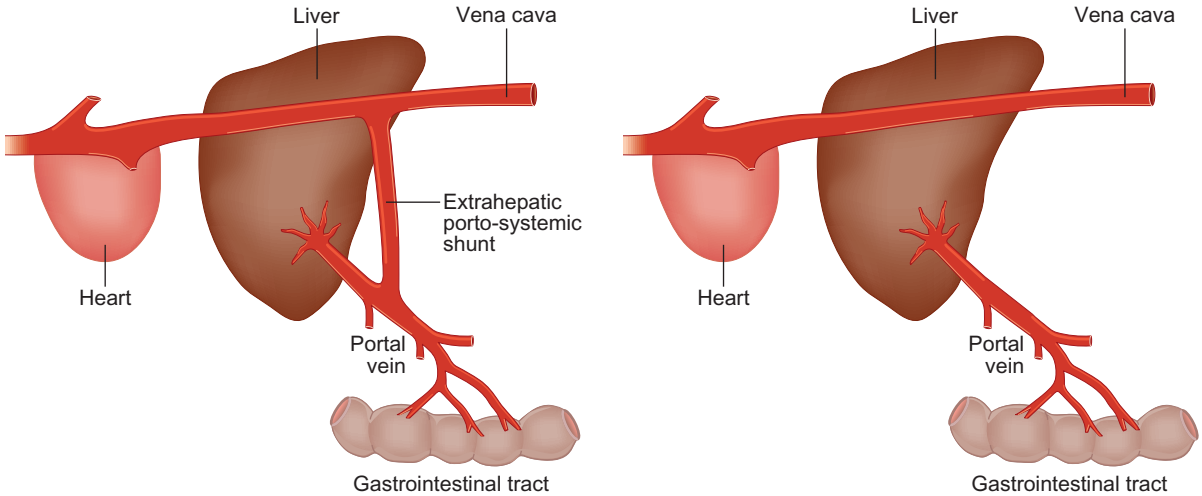


Fig 21.5 A liver with an extrahepatic porto-systemic shunt and a normal liver.

An increase in plasma cortisol is common in dogs with PSS and may account for the elevations in ALP levels and hepatic hydropic degeneration as well as the polydipsia/polyuria. Increased plasma cortisol is thought to be due to dysregulation of pituitary ACTH secretion by GABAergic mechanisms. Hypercortisolaemia regresses with correction of the shunt.

Epidemiology

Congenital PSS has been reported in 0.18% of all dogs and 0.05% of mixed-breed dogs. The proportion of diagnoses of congenital PSS has increased from 5 in 10,000 dogs in 1980 to 5 in 1000 dogs in 2001. In one study, the greatest numbers of shunts were found in Havanese (3.2%), Yorkshire terriers (2.9%), Maltese (1.6%), Dandie Dinmont terriers (1.6%) and Pugs (1.3%). In another study of 242 cases from Australia, breeds that were significantly over-represented included the Maltese terriers, Silky terrier, Australian cattle dog, Bichon Frise, Shih Tzu, miniature schnauzer, Border collie, Jack Russell terrier, Irish wolfhound and Himalayan cat. Bichon frises with shunts were significantly more likely to be female than male.

Congenital PSS appears to be hereditary in Yorkshire terriers, but the exact mechanism of inheritance has not been elucidated. In Cairn terriers, extrahepatic shunts are inherited as an autosomal trait with variable expression. Familial lines with increased incidence of PSS have been reported in miniature schnauzers, Irish wolfhounds and old English sheepdogs. Multiple extrahepatic PSSs have been found in related female American cocker spaniels and breed predispositions for multiple extrahepatic shunts include the German shepherd dog and Doberman pinscher.

Single intrahepatic shunts are most commonly found in large breed dogs such as Irish wolfhound, old English sheepdog, golden and Labrador retrievers and Samoyed and in medium-sized breeds such as Australian shepherd dogs and Australian cattle dogs.

Most dogs with PSS are diagnosed at less than 2 years of age, although sometimes the signs are subtle and the cases are not brought in for veterinary attention until they are older. Intrahepatic PSS may occur more commonly in male dogs than in female dogs.

Prognosis

Nearly one in five dogs has a seizure after shunt ligation, for reasons not yet well understood. Treatment has been tried with intravenous phenobarbitone or continuous infusion of propofol (0.025–1 mg/kg/minute) for 12 to 24 hours. Prognosis is poor for animals with seizures post-operatively.

In one study of dogs treated for single extrahepatic shunts with an ameroid constrictor post-operative mor-

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tality rate was 7.1%. Predictive factors for post-operative death included high preoperative WBC count and post-operative complications such as seizuring. In another study using silk ligatures to close the shunting extrahepatic vessel, short-term and long-term mortality were 27% and 2.9%, respectively. Clinical recurrence occurred in 10% of dogs. The degree of closure was significantly associated with mortality, but not with clinical recurrence. The preoperative size of the liver (in relation to the dog's weight) also appears to be an indicator of prognosis, with the greater the liver size, the better the prognosis.

Twenty-one percent of dogs after surgery using ameroid constrictors and three of 10 dogs using ligatures had continued shunting. Predictive factors for persistent shunting included low preoperative plasma albumin concentration, high portal pressure after complete occlusion and high portal pressure difference (post-occlusion minus baseline).

In a study of 168 dogs with single extrahepatic shunts treated by placement of an ameroid constrictor, there was a low prevalence of post-operative complications (10%); the most common complications were seizures, ascites and haemoabdomen. The prophylactic use of an anticonvulsant drug, such as potassium bromide, is recommended by some clinicians in dogs undergoing PSS surgery. Twenty-one percent of dogs had continued porto-systemic shunting, usually associated with the development of multiple extrahepatic shunts, though many dogs with persistent shunting were clinically normal. The long-term outcome was classified as excellent in 80% of dogs, good in 14% and poor in 6%.

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22 Hepatic lipidosis in a cat

Case contributed by Nicki Reed

INITIAL PRESENTATION

Anorexia, weight loss, vomiting, lethargy, gulping

Signalment: 8-year-old, male neutered domestic shorthair cat, body weight 5.8 kg

CASE HISTORY

The cat presented with an eight-day history of anorexia and rapid weight loss. The cat had also become very dull and lethargic and did not always appear to be aware of his surroundings. He had vomited brown fluid twice in the 24 hours prior to presentation. In addition, the owners reported that the cat seemed to make exaggerated swallowing/gulping motions. The cat's defecation appeared normal.

In the 3 days prior to presentation the cat had become progressively quieter and less interactive with the owner. The owner felt the cat seemed weak, particularly on its hind limbs. No seizures were reported.

The cat had initially been presented to the referring veterinary surgeon 4 days previously who, unable to find anything specific wrong with the cat, had administered dexamethasone and long-acting penicillin (doses not known). When no improvement was seen after 48 hours, the penicillin injection was repeated and a blood sample obtained, but the results had not been received when referral was deemed necessary.

The cat had been in the owner's possession since he was a kitten, was fully vaccinated (including against feline leukaemia virus) and wormed every 6 months with a combination of pyrantel and praziquantel.

He was the only cat in the household and had outdoor access via a cat flap. He was fed a complete dried diet ad lib. The cat had always eaten well and had been overweight at 7 kg when last weighed 3 months prior to presentation. The cat's appetite had declined gradually for about a week prior to him becoming completely anorexic 8 days before presentation.

Although water was always available, the cat was rarely seen to drink and the owner assumed that the cat drank from the pond outside. Recently the cat had developed the habit of drinking out of the toilet. A litter tray was offered in the house, but was rarely used. In the week prior to presentation the cat had become reluctant to go outside and had used the litter tray daily. The owner did not notice any abnormalities in the cat's urine.

PHYSICAL EXAMINATION

The cat appeared obtunded (reduced mental awareness) but was still responsive. His body condition score was still reasonable (body condition score 4/9), but the cat weighed 5.8 kg, indicating a 17% body weight loss. The oral cavity and muzzle appeared moist.

His mucous membranes appeared slightly icteric and the capillary refill time was prolonged at approximately 3 seconds. There was marked gingivitis, with osteoclastic resorptive lesions associated with the upper pre-molars and some evidence of pyorrhoea. His skin tent test result was approximately 1.5 seconds, giving an estimated 7% dehydration.

His submandibular lymph nodes were palpably enlarged, but the pre-scapular and popliteal lymph nodes felt normal. Thoracic auscultation was unremarkable (heart rate was 188 beats per minute and respiratory rate was 24 breaths per minute). On abdominal palpation the liver was palpably enlarged and renal palpation elicited an apparently painful response. The bladder was small and the intestines felt empty. Rectal temperature was normal at 38.7° C.

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With the exception of weakness and altered mentation, no neurological deficits were detected, although a full neurological examination was not possible. The cat was bruised around its previous venepuncture site.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

This cat had an extensive problem list, namely: anorexia, weight loss, possible polydipsia, vomiting, exaggerated swallowing/ptyalism, altered mentation, weakness, lethargy, dental disease, dehydration, icterus, hepatomegaly, renal pain and bruising. Whilst some of these problems are non-specific, such as lethargy, others, such as dehydration and weakness, are likely to be a consequence of the disease process.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses lists were done for the anorexia, weight loss, vomiting, hepatomegaly, icterus, ptyalism, altered mentation, renal pain and ecchymoses. For the most significant problems in this cat these include:

- Anorexia
  - oral disease
  - oesophagitis
  - pancreatitis
  - neoplasia
  - hepatic disease
  - renal disease
  - unpalatable diet
  - anosmia
- Weight loss
  - secondary to anorexia
  - hyperthyroidism
  - diabetes mellitus
  - liver disease
  - renal disease
  - intestinal malassimilation (malabsorption, maldigestion)
  - neoplasia
- Vomiting
  - gastrointestinal disease
  - hepatic disease
  - renal disease
  - pancreatitis
  - diabetes mellitus (ketoacidosis)
  - neoplasia
  - CNS disease
  - drugs (e.g. antibiotic, corticosteroids, non-steroidal anti-inflammatory drugs)
- Hepatomegaly
  - hepatic lipidosis (HL)
  - cholangiohepatitis
  - hepatic neoplasia
  - hepatic cysts
  - lymphoma
  - feline infectious peritonitis
- Icterus
  - pre-hepatic causes, i.e. haemolysis
  - hepatic disease (e.g. HL, cholangiohepatitis, feline infectious peritonitis)
  - post-hepatic biliary obstruction (e.g. bile duct carcinoma, pancreatitis)
- Ptyalism
  - dental disease
  - oral ulceration
  - oesophagitis
  - nausea
  - hepatoencephalopathy
- Altered mentation
  - hepatoencephalopathy
  - hypoglycaemia
  - thiamine deficiency
  - post-ictal
  - meningitis
  - encephalitis
  - space-occupying brain lesion
  - head trauma
- Renal pain
  - pyelonephritis
  - nephrolithiasis
  - hydronephrosis
  - feline infectious peritonitis
  - lymphoma or other tumour
- Bruising/ecchymoses
  - thrombocytopenia
  - thrombocytopathia
  - acquired coagulopathy (e.g. rodenticide poisoning, liver disease, vitamin K malabsorption)
  - hereditary coagulopathy (e.g. haemophilias, unlikely in this neutered cat with no previous history of bleeding)
  - disseminated intravascular coagulopathy
  - vasculitis

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CASE WORK-UP

Blood was obtained by venepuncture from the medial saphenous vein for haematology, serum chemistry and a coagulation profile, and a urinalysis was performed.

Minimum data base

Routine haematology results were within the reference range. Serum biochemistry results showed elevations in alanine aminotransferase to 551 IU/l (reference range 6–83 IU/l), alkaline phosphatase to 360 IU/l (reference range 10–100 IU/l) total bilirubin to 20.6 µmol/l (reference range 0–6.8 µmol/l) and bile acids to 26.8 µmol/l (reference range 0–7 µmol/l). Gamma glutamyl transferase (GGT) was not elevated (4.8 IU/l; reference range 1–5 IU/l).

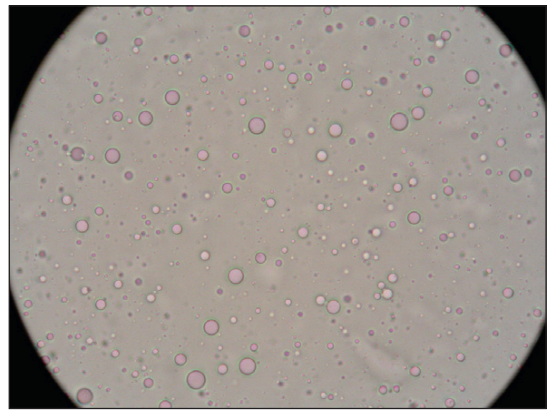


Fig 22.1 Lipid droplets in the urine

CLINICAL TIPS ON DIAGNOSING HL

These are typical findings in HL, but can also occur with other liver or pancreatic disorders. Serum GGT is elevated with necroinflammatory diseases e.g. cholangitis, therefore a serum GGT concentration within the reference range can serve as a useful indicator of HL. An elevated GGT does not, however, rule out HL, as HL may be present concurrently with inflammatory liver disease.

As these patients can have coagulation abnormalities, blood should ideally be obtained from a peripheral vein, rather than the jugular vein, until the coagulation status has been ascertained. Similarly, cystocentesis is best avoided until the coagulation status has been ascertained.

Total proteins were elevated, with albumin at the top end of the reference range at 38.4 g/l (reference range 28–39 g/l) and globulin above the reference range at 56.7 g/l (reference range 25–50 g/l). The high value for the albumin was attributed to dehydration with hyperglobulinaemia possibly relating to the dental disease or another inflammatory condition. Urea was elevated at 16.7 mmol/l (reference range 2.8–9.8 mmol/l) and creatinine was elevated at 210 µmol/l (reference range 40–177 µmol/l), which could reflect dehydration, renal disease or, much less likely, a post-renal cause of azotaemia. Potassium was low at 3.5 mmol/l (reference range 4–5 mmol/l), which is a common finding in anorectic cats. Glucose was moderately elevated at 15.9 mmol/l (reference range 3–5 mmol/l), which was attributed to stress from the illness, but could also have been due to diabetes mellitus or renal tubular disorders.

In the coagulation profile, prothrombin time was 16 seconds (reference range 5–12 seconds) and activated partial thromboplastin was 25 seconds (reference range 10–20 seconds) and fibrinogen was at the low end of the reference range at 2.1 g/l (reference range 2–4 g/l). As the liver is responsible for synthesis of the majority of the clotting factors, prolonged clotting times were likely due to hepatic dysfunction.

Urinalysis showed a urine specific gravity of 1.025, which was considered inappropriately low for a dehydrated cat. Dipstick analysis revealed 2+ glucose, trace ketones, 1+ blood, pH 7.0 and 2+ bilirubin. Sediment analysis showed an average of 6 WBC per high powered field (normal <3), 4 red blood cells per high powered field (normal <3) and numerous lipid droplets (Fig 22.1). Occasional granular casts were also observed. Urine culture was negative.

Blood pressure measurement

The systolic blood pressure in this cat was low normal at 115 mmHg.

CLINICAL TIP

Anorexic, dehydrated cats can be fragile patients. Assessment of blood pressure can be useful to guide fluid therapy, with shock doses (50 ml/kg/hour) potentially required in collapsed, hypotensive patients.

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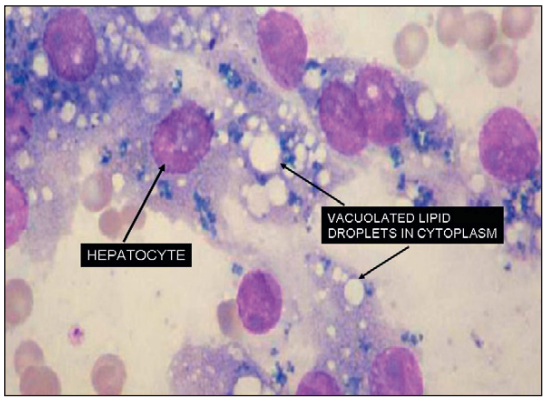


Fig 22.2 Hepatocytes showing increased lipid accumulation due to hepatic lipidosis

Imaging

Ultrasonography showed an enlarged hyperechoic liver and also reduced renal corticomedullary definition with widened renal pelves, consistent with pyelonephritis.

Liver tissue histopathology

A fine-needle aspirate of the liver was obtained under ultrasound guidance. The sample obtained from this cat was supportive of a diagnosis of HL (Fig 22.2).

CLINICAL TIP

A sample of liver tissue is required to confirm the presence of HL. Fine-needle aspiration is a reasonably safe option, particularly with ultrasound guidance. If more than 50% of the hepatocytes show vacuolation, then HL is likely to be present. Confirmation that the vacuoles are filled with fat and not glycogen, requires staining with an appropriate stain such as oil red O, but this is rarely considered necessary. Although lipidosis can be relatively easily detected on fine-needle aspiration, concurrent hepatic disease such as cholangitis may be missed, therefore if there is a suspicion of inflammatory disease, a biopsy (Tru-Cut or surgical) is a better option for confirmation. The potential complications such as haemorrhage, hypotension, bile peritonitis and inadequate sampling should be borne in mind if this procedure is performed.

Medical treatment options

As the severity of HL can vary from mild, subclinical disease to severe, life-threatening metabolic abnormalities, the treatments administered require to be individualized to the case, although nutritional support is a key factor for all cases of HL. Any specific treatments for the underlying disease, e.g. antibiotics, analgesics or insulin, should obviously be provided.

NURSING ASPECTS OF NUTRITIONAL SUPPORT

In milder cases of HL where an underlying disease is identified that should respond rapidly to therapy, offering small quantities of palatable foods or syringing a liquid diet may be adequate. However, it may cause food aversions and continued anorexia. Placement of feeding tubes should therefore be considered sooner rather than later (see Appendix 9). The calorie requirements of the cat should be calculated in order that the appropriate quantity of food is fed. A balanced feline diet should be fed, although consideration should also be given to underlying disease conditions e.g. moderately restricted, high quality protein content in renal disease or if hepatic encephalopathy is present.

The nutritional requirements are based on resting energy requirements (RER), which may be calculated as follows BW, Body weight:

$$\text{RER(kcal)} = [30 \times \text{BW(kg)}] + 70$$

The calorie requirement should be split into four to six feeds per day, with a maximum of 50 ml at any feed (allow for fluid flushing the tube!).

Only one-third of the calorie requirement should be fed on the first day, two-thirds on the second day and the full amount not fed until the third day. This reduces the risk of re-feeding syndrome and GI upset. Re-feeding syndrome arises when there is an increase of insulin released in response to food. This causes intracellular movements of potassium and phosphate and a shift from a catabolic state to an anabolic state with increased utilization of glucose and thiamine.

Fluid therapy

The majority of cats with HL are dehydrated; therefore rehydration with crystalloids is indicated. As cats with

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compromised liver function may have difficulty metabolizing lactate, the author's preferred initial choice is normal saline. In addition, this is a useful fluid for enabling supplementation with other therapies as required. In severe cases, consideration should be given to placement of a multi-lumen catheter to facilitate blood collection and fluid administration, but peripheral catheters are adequate in the initial stages and may be preferable until the coagulation status of the cat is known.

Electrolytes

Potassium supplementation is almost certain to be required and should be based on measured electrolytes where possible. If unable to measure potassium, then a 'maintenance' dose of 20 mmol potassium chloride should be added per litre of fluids. Phosphate may also be low and should be supplemented as potassium phosphate, with the amount of potassium provided subtracted from any potassium chloride supplementation given. Even if potassium and phosphate are normal on initial assessment, their values are prone to fall rapidly, particularly when feeding is re-introduced. Regular assessment should therefore be performed (at least daily) and changes to supplementation made as required. Magnesium is harder to measure, but is worth checking as supplementation may be required, particularly if potassium values are difficult to maintain in the normal range.

Vitamin supplementation

A high percentage of cats with HL have coagulation abnormalities, therefore supplementation with Vitamin K is likely to be beneficial, particularly if clotting times cannot be obtained rapidly. Water soluble multivitamin preparations should be added to the intravenous fluids, although this is not likely to give adequate levels of thiamine. Thiamine is not available as a single-source parenteral vitamin and oral supplementation may be required in cats that show continued weakness or neurological signs. Cobalamin may be injected subcutaneously once weekly as required. Vitamin E could be considered for its antioxidant properties.

Anti-emetics

Many cats with HL have underlying conditions that may make them vomit or feel nauseous. In addition, with some gastrointestinal conditions, or if hypokalaemia is present, ileus may be a feature which can contribute to anorexia. Maropitant, metoclopramide or ranitidine may all be considered. (See tip on maropitant in Ch 19).

S-adenosyl methionine

Supplementation with S-adenosyl methionine (SAMe) promotes formation of glutathione and L-carnitine and may be considered hepato-protectant. Oral administration may be difficult in extremely sick animals, in which case consideration can be given to using intravenous *N*-acetylcysteine, which is also a glutathione precursor.

Taurine and L-carnitine

Taurine and L-carnitine supplementation may be beneficial, although consideration must be given as to how many medications the cat can tolerate.

CLINICAL TIPS ON TREATMENT OF HL

Dextrose or glucose containing solutions are inappropriate fluid solutions:

- They may potentiate hepatic triglyceride accumulation and inhibit fatty acid oxidation.
- They stimulate release of insulin, which will cause intracellular movement of potassium and phosphate.
- They do not provide anywhere near the calorie requirement for maintenance. The calorie content of 5% dextrose is 170 kcal/l, therefore a 5 kg cat would require 1 to 1.5 l/day to meet its maintenance energy requirement.

FOLLOW-UP

The cat was given intravenous fluids supplemented with B vitamins, a naso-oesophageal tube was placed and feeding initiated, and vitamin K was administered for 3 days. Intravenous antibiotics were also started, a subcutaneous injection of cobalamin given and SAMe and L-carnitine administered orally. After 48 hours stabilization, when coagulation times were normalized, the cat was anaesthetized with alphaxalone/sevofluorane. A jugular catheter was placed to facilitate monitoring (Fig 22.3) and an oesophagostomy tube placed to facilitate feeding. The cat was hospitalized for 10 days, at which point he was consuming 80% of his caloric requirements voluntarily. He was discharged home on oral SAMe (200 mg per cat po q 24 hours) and antibiotics

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Fig 22.3 Jugular catheter in place for blood sampling and intravenous access for fluids or medications. Jugular catheters may also be used for parenteral nutrition.

(clavulanate-potentiated amoxicillin at 15 mg/kg po q 12 hours), which were continued for a further 5 weeks. At that point repeat biochemistry indicated all liver parameters within reference ranges. Dental surgery was performed 2 months later to decrease the risk of HL recurrence.

DISCUSSION

Hepatic lipidosis is a complex metabolic condition arising following inadequate food consumption. The starvation that induces the condition may be considered 'simple', where there is no underlying medical condition (e.g. a cat that gets shut in a garage for a week) or 'stressed', which is secondary to an underlying medical disease. Mobilization of fat from adipocytes leads to deposition of fat within the hepatocytes, hence the term HL or fatty liver. Cats are much more susceptible to HL than dogs due to their unique nutritional requirements and obligate carnivore status. Any cat that has not eaten for more than 3 days should be considered at risk of developing HL, with obese cats being at an increased risk compared to lean cats due to the greater amount of fat that can be mobilized.

The serum chemistry findings of elevated liver parameters other than GGT, the large, hyperechoic liver on ultrasound and the vacuolated hepatocytes obtained on a fine-needle aspirate confirmed the diagnosis of HL in this case. The presumptive underlying cause was thought to be pyelonephritis. The ultrasonographic findings were consistent with this, despite the negative urine culture. This was attributed to the cat having received antibiotics prior to culture and the dental disease was considered a potential source for haematogenous infection. The bruising was consistent with the finding of prolonged clotting times and the altered mentation was attributed to hepatoencephalopathy, although an assessment of ammonia levels may have helped to support this assumption.

While this cat was not anaemic, anaemia is frequently present in cases of HL. The anaemia may be due to blood loss secondary to coagulopathies, haemolysis due to oxidative stress on the red blood cells, in which case Heinz bodies may be seen or non-regenerative from underlying disease processes. Abnormalities in clotting times have been identified in 45% of cats with HL.

Pathogenesis

The pathogenesis of HL is complex and relates to the unique nutritional requirements of cats. Cats are obligate carnivores, with a high requirement for essential amino acids from protein, particularly arginine, taurine and methionine. When deprived of protein (starvation), cats are unable to down-regulate the aminotransferases involved in protein metabolism. Protein metabolism continues, producing ammonia, which would normally enter the urea cycle. However, arginine is required for the urea cycle and as this falls with inadequate protein intake, the levels of ammonia increase, which can contribute to hepatoencephalopathy. Taurine is required for conjugation of bile acids. Its deficiency leads to a build up of bile acids. Methionine is involved in many metabolic pathways, including the synthesis of SAMe. This is a precursor for the synthesis of glutathione and L-carnitine and is an important antioxidant and detoxifying agent within the liver.

Vitamin deficiencies are also of importance. The water-soluble B vitamins are not stored to any great extent and rapidly become depleted in anorexic cats. Deficiency of vitamin B_1 (thiamine) can lead to neurological signs such as altered mentation, vestibular signs or seizures. Vitamins B_6 (pyridoxine) and B_{12} (cobalamin) are important co-factors for metabolic pathways in the liver including metabolism of SAMe. The fat soluble vitamins are affected by lack of bile acids contributing to malabsorption as well as inadequate intake. As already mentioned, vitamin K deficiency can lead to coagulopathies and vitamin E deficiency may contribute to oxidative damage.

Sick animals have higher circulating levels of the stress hormones cortisol, adrenaline and growth hormone,

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which in turn stimulate hormone sensitive lipase. This hormone initiates mobilization of lipids out of adipocytes into the circulation. From the circulation the lipid is taken up into the hepatocytes of the liver, where its metabolism is compromised due to lack of L-carnitine. In cases of HL up to 50% of the weight of the liver may be due to fat. The swollen hepatocytes can in turn cause obstruction of bile canaliculi.

Epidemiology

Overweight or obese cats are at greater risk of developing HL due to the increased quantity of lipid that can be mobilized at times of starvation. As such, there is no breed or sex predisposition. The same factors that predispose to obesity may therefore predispose to HL, namely, high carbohydrate diet, lack of exercise, neuter status and middle age.

Prognosis

Although some cats suffering from HL can be extremely fragile patients, the outcome with intensive management is generally good. Recovery rates are quoted as 60 to 85% depending on the severity of the condition. Owners should, however, be warned of the potential risks, the costs that may be involved and that an extended hospitalization period may be required.

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SECTION 3

DIARRHOEA

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23 Intestinal disorders: diarrhoea

Diarrhoea is defined as a change in the frequency, consistency or volume of bowel movements. Increased frequency may be caused from diseases of the colon or rectum or those that cause larger faecal volume. Larger faecal volume is usually due to increased water or nutrients which have not been assimilated and can be affected by the amount of fibre in the diet. Faecal water content increases when there is increased secretion or decreased absorption of fluid. The water content of faeces which appear normal is 60 to 80%; in unformed and watery faeces the water content is 70 to 90%.

In a normal 20 kg dog, 2.5 l of fluids enter the duodenum and over 98% of this is usually reabsorbed. The colon can increase its absorptive capacity three-fold over normal, so the volume of fluid entering the colon must exceed this capacity before diarrhoea is seen. If there is colonic disease, however, only small changes in the absorptive capacity may result in diarrhoea.

Animals with diarrhoea may become faecally incontinent or be unable to retain faeces; however, these two conditions do not necessarily occur concurrently. Animals may be incontinent without diarrhoea or have diarrhoea without incontinence.

Other signs of intestinal disease include the appearance in the stool of bright red blood, melena, mucus and malassimilated nutrients (e.g. steatorrhoea). Patients may show dyschesia (discomfort on passing stools) or tenesmus (straining to pass stools). Abdominal pain, bloating, increased eructation, borborygmi or flatulence, salivation and anorexia may occur. Intestinal diseases can occur without diarrhoea and should be considered when any of these other signs are present.

CLASSIFICATION OF DIARRHOEA

Diarrhoea is often classified as originating from the large or small intestine and as acute or chronic (see below) which helps the clinician decide upon the appropriate diagnostic approach. Classification according to pathophysiology also helps in the understanding of the cause of the diarrhoea. The usual pathophysiological classifications include osmotic diarrhoea, secretory diarrhoea, diarrhoea resulting from increased permeability and diarrhoea resulting from deranged motility. More than one of these mechanisms can occur in a patient simultaneously.

Osmotic diarrhoea is caused by an increase in unabsorbed solutes in the faeces causing an increase in faecal water content. With the increased solutes, water diffuses across the duodenal mucosa and sodium diffuses with the water, causing even more water to be drawn into the lumen. Causes of osmotic diarrhoea include overeating, sudden diet changes, gastric dumping (sudden flow of ingesta into the duodenum) or malassimilation. Malassimilation may be due to maldigestion, for example, with exocrine pancreatic insufficiency or malabsorption such as occurs with small intestinal mucosal or intramural disease. Lactose intolerance due to lactase deficiency may also cause osmotic diarrhoea due to the unabsorbed lactose in the intestines.

Secretory diarrhoea is due to an increase in the intestinal secretion of fluid greater than the absorption and can occur either due to an increase in secretion or a decrease in absorption, or both. Causes of secretory diarrhoea include bacterial enterotoxins, deconjugated bile acids, hydroxyl fatty acids, gastrointestinal hormones, cholinergic agonists and enteric system neuropeptides.

Bacteria that can produce enterotoxins include *Escherichia coli*, *Clostridium perfringens*, *Campylobacter* species, *Salmonella typhimurium*, *Staphylococcus aureus*, *Klebsiella pneumoniae* and *Yersinia enterocolitica*.

Diarrhoea from increased permeability is caused by a defect in the intestinal permeability which causes electrolytes which have been absorbed to flow back into the intestinal lumen. This results in a decrease of absorption of water from the bowel. If the intestinal barrier is

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further damaged, albumin and other plasma proteins may leak into the intestinal lumen and with enough damage blood may also be lost. Causes of increased intestinal permeability include mucosal inflammation, erosion or ulceration, cellular infiltration of the lamina propria (e.g. with neoplastic cells) and compromised blood or lymphatic circulation. Mucosal inflammation is present in chronic inflammatory bowel disease, gluten enteropathy and milk protein intolerance.

Deranged intestinal motility may be primary or occur secondarily to other intestinal disorders. A more rapid intestinal transit time can be caused by either decreased segmentation contractions or, much less commonly, increased peristaltic contractions. Decreased segmentation contractions are sometimes termed a garden hose gut, as there is less slowing of the ingesta and less time for absorption. Causes include hookworm infections, canine dysautonomia and some types of colitis.

DIAGNOSIS

Diarrhoea is a clinical sign and not a final diagnosis. In order to treat a patient effectively for diarrhoea, the underlying cause must be found.

Many dogs and cats with acute diarrhoea do not require full investigation as their diarrhoea is most likely to be dietary induced and will be self-limiting. These patients receive empiric therapy and frequently improve within several days.

Where diarrhoea persists for more than 2 weeks (i.e. is chronic) and is unresponsive to suitable empiric therapy or if there are signs of systemic illness in any patient, they require further investigation.

The approach to investigating patients with chronic diarrhoea should always start with the patient's signalment, a detailed history and full physical examination.

Young patients are more likely to have nutritional, parasitic or microbial causes of diarrhoea. There are also some breed predispositions to certain types of diarrhoea, for example, exocrine pancreatic insufficiency in young German shepherd dogs. A patient who has been in a boarding kennel or otherwise exposed to new animals has a higher chance of getting an infectious disease.

Assessment should be made of the diet and any changes in the diet and any history of scavenging or hunting or being give treats or snacks or anything other than pet food. Adverse reactions to food are a common cause of diarrhoea and may still occur even when the diet has been fed for a long time. Any change in appetite should be noted, as a poor appetite may signify severe disease.

The duration of diarrhoea and the frequency of abnormal or normal appearing stools should be noted, as well as the character of the stools. Many animals will show progressively worsening diarrhoea during the day, as during the night more water is absorbed from the faeces and increased activity promotes gastrointestinal (GI) motility. These signs are also important in differentiating small intestinal diarrhoea from large intestinal diarrhoea (see Table 23.1).

Other questions that should be asked during the history taking include:

- Vaccine history and worming status
- Are there other pets or persons in the household with diarrhoea or has the animal been exposed to other animals (e.g. boarding, playing in the park)?
- Previous history of GI signs
- Presence of vomiting (see section on vomiting for more information)
- Presence of weight loss

As mentioned above, diarrhoea is often classified into small bowel or large bowel disorders. Table 23.1 indicates the parameters which are used to make this distinction.

Table 23.1 Differentiating diarrhoea caused by small bowel disorders from that caused by large bowel disorders

Symptom	Small bowel	Large bowel
Faecal volume	Large	Small
Faecal frequency	Increased four-fold	Increased eight-fold
Haematochezia	None or changed blood	None or fresh blood
Faecal mucus	No	Often present
Steatorrhoea	Possible	No
Tenesmus	No	Frequent
Dyschezia	No	Frequent
Flatus/borborygmi	Present	Present
Vomiting	Possible	Possible
Weight loss	Common	Rare

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Fig 23.1 Dog showing a hunched posture due to abdominal pain



Fig 23.2 Abdominal palpation of a cat

Many patients exhibit symptoms which fall into both categories. This may be due to small bowel disorders affecting the large bowel or that disease involves both regions of the intestine. Up to 30% of dogs with chronic diarrhoea have diffuse disease of the GI tract. Many dogs with colitis or enteritis will vomit, but may or may not have gastric pathology. Visceral stimulation through inflammation causes reflex vomiting.

PHYSICAL EXAMINATION

The physical examination should be used to examine the GI tract and to look for evidence of systemic disease. The pet should be assessed unrestrained initially if possible to evaluate attitude and postures. Animals with a painful abdomen may demonstrate a hunched posture (Fig 23.1) and cats with hyperthyroidism may be hyperactive and difficult to examine.

In particular the clinician should palpate the abdomen for evidence of abnormal intestines, 'mass' lesions, pain or fluid/gas accumulations (Fig 23.2). The perineum should be examined for the presence of faeces and for evidence of perineal hernias.

The physical examination is critical for determining the severity of the patient's disease; warning signs for severe disease include fever, depression, anorexia, weight loss, weakness, dehydration, pale or discoloured mucous membranes and abdominal pain, masses or effusions.

After the history and physical examination it should be possible for the clinician to determine if there is likely to be systemic disease or primary GI disease – if the latter it is likely to be associated with the small or large bowel or both.

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24 Small intestinal diarrhoea

Diarrhoea may be determined to be of small intestinal origin when there are large volumes of faeces produced without evidence of tenesmus or dyschezia and there is no bright red blood or mucus present on the stools. Weight loss may accompany diarrhoea of small intestinal origin.

Some of the differential diagnoses of small intestinal diarrhoea include:

- Dietary
  - food poisoning
  - adverse reaction to food (allergy or food intolerance)
  - dietary indiscretion (e.g. raiding the rubbish bin)
  - sudden change of diet
- Gastric
  - dumping syndrome, where too much food is presented to the duodenum rapidly
- Small intestinal diseases
  - infectious (viral infections, bacterial, e.g. *Salmonella* spp., *Campylobacter* spp., *E. coli* enterotoxins).
  - parasites (e.g. *Giardia*, strongyloides, ascarids, hookworms)
  - inflammatory bowel disease
  - infiltrative neoplasia (e.g. lymphoma, mastocytosis)
  - lymphangiectasia
  - brush border enzyme defects
  - antibiotic responsive diarrhoea (ARD) (or antibiotic responsive enteritis)
  - partial obstruction (e.g. intussusception, neoplasia, foreign body)
  - extraluminal obstruction
  - ischaemic disease (infarction, torsions)
  - haemorrhagic gastroenteritis
- Secondary systemic disorders
  - pancreatitis
  - exocrine pancreatic insufficiency (EPI)
- Liver disease, including biliary diseases and portosystemic shunts, intrahepatic cholestasis or bile duct obstruction
- Kidney disease
- Hyperthyroidism in cats
- Hypoadrenocorticism
- Congestive heart failure
- Feline viruses (feline infectious peritonitus (FIP), feline leukaemia virus (FeLV))
- Miscellaneous causes
  - toxaemia (e.g. pyometra, peritonitis)
  - toxins and drugs (e.g. non-steroidal anti-inflammatory drugs, lead, organophosphates)
  - APUDomas (e.g. gastrinoma)
  - autoimmune disorders (e.g. systemic lupus erythematosus)

FURTHER INVESTIGATION OF CHRONIC SMALL INTESTINAL DIARRHOEA

Dietary trial

If there are no abnormalities detected during the physical examination and the animal is bright and eating well, feeding an exclusion diet with a novel protein source or a hydrolyzed protein source for 2 to 6 weeks may be tried prior to further testing. In cats with diarrhoea, many respond to a restricted novel protein diet within a week. If the restricted diet is successful in improving the clinical signs, ideally a challenge with the previous food or snacks should be tried to prove that dietary sensitivity is the cause of the diarrhoea. Many owners are understandably reluctant to try this challenge and a new diet may be designed using the trial food. The owners should understand that the animal may become sensitized to the new diet and again develop diarrhoea.

Minimum data base

For cases in which the patient is systemically unwell or has not responded to dietary therapy and an appropriate de-worming programme, a minimum data base of haematology, serum biochemistry including basal cortisol and urinalysis should be performed. In cats, serologic

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tests for FeLV, FIP and a total serum thyroxine concentration should be included. These tests evaluate the patient for systemic diseases such as kidney disease, hepatopathies, hypoadrenocorticism, hyperthyroidism and viral diseases. A low albumin may be present in hepatopathies or protein losing enteropathy or nephropathy. If low protein is combined with low serum cholesterol and lymphopenia, lymphangiectasia may be present. Eosinophilia may be present with eosinophilic gastroenteritis, parasitism and hypoadrenocorticism. The minimum data base also aids in assessing hydration status and electrolytes which will guide the clinician in the choice of fluid therapy.

Faecal tests

Further testing may include faecal analysis for parasites and enteropathogenic bacteria, e.g. *Salmonella*, *Campylobacter* and *Yersinia*. Diarrhoea due to *Campylobacter* is more common in younger animals and the bacteria can also be isolated from the faeces of healthy animals. *Salmonella* can also be present in the faeces of animals without clinical signs and should probably not be treated unless serious or systemic signs are present as treatment may prolong the carrier state.

Toxin producing *Clostridium difficile* may cause acute or chronic diarrhoea. Faecal cultures for *C. difficile* are of no value because it is found in faeces from healthy animals and the presence of it does not prove that it is producing a toxin. Diagnosis of enterotoxigenic clostridia is best supported by a positive enterotoxin assay.

A fresh saline smear may detect *Giardia* trophozoites, although zinc sulphate flotation which detects *Giardia* cysts is more accurate. If giardiasis is suspected but cannot be confirmed a therapeutic trial with fenbendazole may be indicated.

Faecal alpha-1-antiprotease

Faecal alpha-1-antiprotease has been measured in the USA as an indicator of gastrointestinal (GI) protein loss and is useful to determine that intestinal loss is the cause of hypoproteinaemia.

Trypsin-like immunoreactivity and pancreatic lipase immunoreactivity

If EPI is a possibility, a trypsin-like immunoreactivity (TLI) test should be done. This is the test of choice for EPI, in which the serum concentration is decreased. The TLI test may be also useful as an adjunctive test in some cases of acute pancreatitis where TLI is temporarily increased, although the pancreatic lipase immunoreactivity (PLI) tests are more specific and have a more prolonged increase in cases of pancreatitis. There are species specific PLI tests for dogs and cats and an in-house serum SNAP cPLI test for dogs (Fig 24.1).



Fig 24.1 The SNAP® cPL test used for in-house screening for pancreatitis

Folate and cobalamin (vitamin B_{12})

Serum folate and cobalamin (vitamin B_{12}) may be decreased in small intestinal disease due to poor absorption. In some cases where there are excessive bacteria in the proximal small intestine the cobalamin may be low due to bacterial consumption and the folate increased due to bacterial production. Excessive bacteria in the intestine has been termed small intestinal bacterial overgrowth (SIBO), a disease process documented in people with $>10^5$ colony forming units (CFU) per millilitre of fasting duodenal intestinal fluid. The accuracy of the term SIBO in small animal patients is dubious. In many dogs with bacterial disorders of the small intestines, it is probably an incorrect description. Studies have shown that clinically normal dogs can have $>10^8$ CFU/ml of bacteria in the upper small intestine and this number is clearly in excess of what was initially accepted as being definitive for SIBO ($>10^5$ CFU/ml).

The terms antibiotic responsive enteropathy (ARE) or antibiotic responsive diarrhoea (ARD) are now more commonly used. The terms ARE or ARD seem to be preferable to SIBO in most dogs because essentially this is a syndrome in which the patient has small intestinal disease which responds to the administration of antibiotics.

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Histopathology and cytology of the intestinal mucosa are extremely insensitive at detecting ARE. Serum cobalamin and folate concentrations have been used for diagnosis and finding both a low serum cobalamin and an increased serum folate concentration had been considered to be relatively specific for ARE.

Dietary folate supplementation contributes to increased blood folate values. Measuring serum cobalamin and folate concentrations is relatively insensitive at detecting ARE or SIBO. There are many dogs with chronic GI disease that respond to antibiotic administration but which have normal cobalamin and/or normal folate concentrations. However, they should be measured in animals suspected to have intestinal disease as hypocobalaminaemia or low serum folate should be treated.

In cats, low serum cobalamin may occur with EPI, intestinal disease (e.g. IBD or LSA), hepatobiliary disease or pancreatic disease. Cats appear to be highly susceptible to cobalamin deficiency as it has a short half-life in this species (12.75 days in healthy cats, 5 days in cats with intestinal disease).

Serum lead concentrations should be determined if there is a history consistent with exposure to lead (e.g. remodelling an older house, lead pipes), especially if central nervous system or behaviour changes are present. Serum organophosphate concentrations should be determined if there has been possible access to these toxins.

Gastrointestinal permeability testing with inert sugar analysis

During some intestinal diseases the mucosal cells are destroyed, decreasing the number of cellular small pores and increasing the number of large pores between the cells, thereby changing the ratio of the two types (Fig 24.2). By using oral administration of sugars of different sizes which move through the different types of pores and measuring the urine recovery of the sugars, a ratio of the recovered sugars can be determined. This appears to correlate with the degree of pathological increase in intestinal permeability.

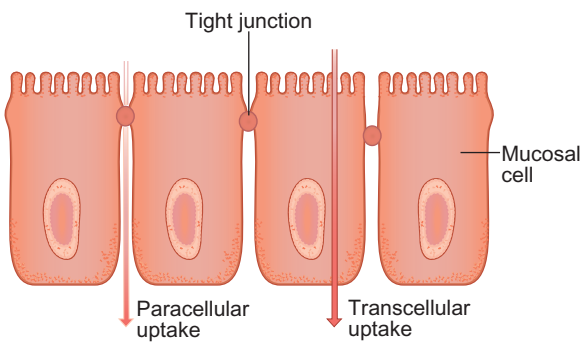


Fig 24.2 Testing of intestinal permeability testing is based on the difference between uptake of sugars via the transcellular route and via the paracellular route

Imaging

Radiography

While generally survey radiographs are of limited value in patients with chronic diarrhoea, contrast studies may help in some cases. Abdominal radiography can aid in the diagnosis of complete or partial obstructions (especially when combined with contrast study using barium suspension or barium impregnated polyethylene spheres), assessment of ileus and diagnosis of an intussusception.

Radiography also helps assess liver and kidney size, presence of abdominal fluid, organ torsions, peritonitis and sometimes pancreatitis.

Ultrasonography

Abdominal ultrasound is more sensitive than radiography for the detection of masses, assessing the pancreas and liver parenchyma, mesenteric lymph nodes and intestinal wall thickness and irregularity.

In some cases ultrasonography can provide additional information, in particular to evaluate the GI peristalsis, and also can allow the identification of the cause of the ileus such as an intussusception or an intestinal foreign body.

Ultrasonographic examination of the small intestine includes evaluation of wall thickness and layering, evaluation of luminal contents and measurement of peristaltic function. In the normal dog, the gastric wall is 3 to 5 mm thick when the stomach is moderately distended and may be slightly thicker when the stomach is not distended. The normal thickness of the small and large bowel wall is 2 to 3 mm when measured during bowel relaxation.

Pathologic thickening should be suspected when wall thickness exceeds 6 to 7 mm in the stomach and 5 mm in the bowel.

Ultrasonography allows differentiation of the layers of the intestine, which alternate in echogenicity. Under optimal conditions, five separate layers can be identified (see Figs 15.4 and 15.5). These include the mucosal surface (hyperechoic), mucosa (hypoechoic), submucosa

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(hyperechoic), muscularis (hypoechoic) and serosa (hyperechoic). The submucosa and subserosa-serosa are hyperechoic because of the presence of relatively more fibrous connective tissue. Examination of the layers of the stomach and bowel may be helpful in determining the severity and location of pathologic processes.

Evaluation of intestinal motility should be included in the examination. The mean number of peristaltic contractions in the stomach and proximal duodenum is four to five per minute.

In some cases of intestinal neoplasia, a focal thickening of the intestinal wall can be observed, with intestinal dilatation present proximal to it and often fluid accumulation. In the area of wall thickening, the normal layered pattern of the intestinal wall is usually absent. Intestinal lymphomas are often characterized by a diffuse neoplastic infiltration of the wall rather than a focal mass.

Endoscopy and intestinal biopsy

Endoscopy allows visual assessment and biopsy of the intestinal mucosa and is safer and less invasive than surgery, which is especially useful in hypoalbuminaemic patients. The investigation of small bowel diarrhoea, protein losing enteropathies, chronic vomiting (intermittent or persistent) and melaena are the major indications for duodenoscopy. The prior work-up should first rule out underlying metabolic, parasitic, infectious and pancreatic causes before pursuing primary intestinal disorders with endoscopy. The diffuse nature of many intestinal causes of small bowel diarrhoea and protein losing enteropathies means that duodenoscopy may enable a diagnosis to be obtained without recourse to surgery.

Endoscopy allows the clinician to assess the mucosal colour, presence of haemorrhage, texture (e.g. friability or granularity) and detect luminal masses. Friability describes the ease with which the mucosa is damaged by contact with the endoscope or the biopsy forceps and, along with granularity (sometimes referred to as a cobble-stone appearance), is a good indicator of increased cellularity, although differentiation between inflammation and neoplasia cannot be made without histopathology. Gross abnormalities such as undigested food, large amounts of fluid, tumours, foreign bodies and parasites are easily detected. Deflation and inflation of the duodenum enables detection of alterations of distensibility and mucosal irregularities.

Histopathology of intestinal biopsies is frequently required to make a diagnosis and should be part of the endoscopic procedures in dogs and cats with diarrhoea. The biopsies are restricted to the mucosa unless multiple samples are taken in the same location. The endoscopist rarely is able to proceed much further than the duodenum, so focal lesions in the jejunum may not be accessible. For these lesions and for full-thickness biopsies, an exploratory laparotomy or laparoscopy is required.

Exploratory laparotomy

If the patient is a good surgical risk, surgical exploration of the abdomen provides a good, although invasive, method of examining the abdominal organs, obtaining full thickness intestinal biopsies and potentially treating some intestinal disorders such as focal tumours, intussusception and foreign bodies. In diarrhoea cases, biopsies of the small intestine should always be performed (even if it grossly appears normal).

INVESTIGATION OF ACUTE DIARRHOEA

The majority of cases of acute diarrhoea are dietary induced and self-limiting. Dietary problems can include ingestion of excessive amounts of food, especially fatty or high carbohydrate foods to which the animal is not accustomed, ingestion of spoiled foods or carcasses or ingestion of a food to which the animal is allergic or intolerant.

Viral infections, bacterial infections and metabolic disorders are also possible causes of acute diarrhoea. Ingestion of toxins, exacerbation of hypoadrenocorticism, pancreatitis, ischaemic disease or haemorrhagic gastroenteritis can cause the acute onset of diarrhoea.

Differential diagnosis of infectious causes of diarrhoea in dogs and cats include:

- *Parvovirus*, *Coronavirus*
- Salmonellosis
- *Campylobacter* infection
- *Yersinia* spp. (uncommon)
- *Clostridium perfringens*
- *E. coli*

Tests for these are the same as for chronic diarrhoea (see page 116).

Many patients remain bright and may continue to eat. Depending on the cause, they may or may not vomit. The diarrhoea may be soft to liquid in consistency.

Treatment for dietary indiscretion (e.g. patient ate from the rubbish bin) is supportive. If the animal is vomiting or dehydration is present intravenous or subcutaneous fluid administration is advised. If osmotic diarrhoea is suspected (e.g. food overload) withhold food for 12

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to 48 hours followed by the use of a highly digestible, low fat diet fed frequently and in small amounts. Anti-emetics (e.g. maropitant or metoclopramide) may be used if needed. Anti-diarrhoeal drugs (e.g. loperamide or diphenoxylate) may be used if an infectious cause is not suspected; these are relatively contraindicated with infectious diarrhoea as they may prolong the presence of the bacteria within the intestinal tract. Antibiotics should be given if there is a known bacterial cause of the diarrhoea and are contraindicated for acute diarrhoea cases otherwise. Spasmolytics are also contraindicated as they decrease intestinal segmentation. The use of probiotics or prebiotics may have some beneficial effects in the treatment of diarrhoea.

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25 Protein losing enteropathy in a dog

INITIAL PRESENTATION

Diarrhoea and weight loss

Signalment: 9-year-old neutered male Jack Russell terrier, body weight 5.3 kg

CASE HISTORY

The dog presented with a 3-month history of diarrhoea, which had initially been intermittent, but had become more persistent during the previous month. The faeces were described as soft to watery, containing no blood or mucus. He produced large volumes two to four times a day. He did not strain to defecate. He had lost about 0.5 kg during the previous month. He rarely vomited, but his appetite had decreased. His exercise tolerance was decreased and the owners thought he had become a bit quieter and less active in the house.

He had previously been a healthy dog and was fully vaccinated yearly. He had been treated for internal parasites with a commercial product (unknown type) about 4 months prior to consultation. His usual diet was a commercial dry dog food and during the month prior to his consultation the owners had been adding canned tuna and other foods to encourage him to eat.

PHYSICAL EXAMINATION

On physical examination the dog was quiet but alert and responsive. His body condition score was 4/9, with evidence of recent weight loss.

His mucous membranes were pink with a capillary refill time of <2 seconds and his hydration appeared adequate. His abdomen was slightly distended and had a fluid thrill. On abdominal palpation, the intestines felt thickened and 'ropey'. Thoracic auscultation and percussion revealed dullness in the ventral thorax and muffled heart sounds. His heart rate was 106 beats per minute. Peripheral pulse quality was adequate and pulses were matched with the heart rate. The respiratory rate was moderately elevated at 48 breaths per minute, with an increased inspiratory effort. Rectal temperature was 37.9° C and soft faeces were present on the thermometer.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The dog's primary problem was diarrhoea, which was thought to be small intestinal in origin due to the presence of weight loss, the lack of blood or mucus in the faeces and to the lack of tenesmus during defecation. There was an increase in frequency of defecation, which is commonly seen in diarrhoea of large intestinal origin, but can also be seen when the storage capacity of the colon is overwhelmed by the volume of faeces. Another problem was the apparent presence of fluid in the abdomen.

DIFFERENTIAL DIAGNOSIS

Differential lists for the diarrhoea (small intestinal):

- Dietary
  - gluttony – unlikely as the dog's appetite was decreased rather than excessive
  - changes of diet – possible with a variety of food being fed; however, the diarrhoea started prior to the variations in diet
  - adverse reaction to food: intolerance or allergy
- Gastric disorders
  - rapid gastric emptying

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- Small intestinal disease
  - infectious (e.g. bacteria, virus, fungus)
  - gastrointestinal parasites
  - infiltrative neoplasia (e.g. lymphoma, mastocytosis)
  - partial obstruction (e.g. intussusception, foreign body)
  - lymphangiectasia
  - brush border defects
  - inflammatory bowel disease (IBD)
- Pancreatic disease
  - chronic pancreatitis
  - exocrine pancreatic insufficiency
  - pancreatic neoplasia
- Liver disease
  - bile duct obstruction
  - liver failure
- Kidney disease – unlikely as he did not have polyuria/polydipsia (pu/pd), although glomerular disorders may present without pu/pd
- Miscellaneous
  - congestive heart failure – no other signs consistent with this
  - immunodeficiencies (e.g. IgA deficiency)
  - hypoadrenocorticism

Another problem in this case was the increased fluid palpated in the abdomen (and suspected in the thorax). Differential diagnoses for ascites rely on first determining the type of fluid:

- Transudate
- Modified transudate
- Exudate
- Chyle
- Pseudochyle
- Blood

CASE WORK-UP

Minimum data base

Routine haematology revealed a moderate leucocytosis with a total WBC count of 23 × 10^9/l (reference range 6.0–15.0 × 10^9/l), due predominantly to a mature neutrophilia of 20 × 10^9/l (reference range 3.6–12.0 × 10^9/l). Lymphopenia was present with a lymphocyte count of 0.46 × 10^9/l (reference range 0.7–4.8 × 10^9/l).

Serum chemistry results showed panhypoproteinaemia with a total serum protein concentration of 28 g/l (reference range 58–75 g/l), due to a decreased albumin concentration of 13.7 g/l (reference range 26–35 g/l) and decreased globulin concentration of 15.3 g/l (reference range 18–37 g/l). Total serum calcium concentration was decreased to 2.0 mmol/l (reference range 2.3–3.0 mmol/l), likely due to the decrease in the albumin bound fraction of calcium.

CLINICAL TIP ON HYPOCALCAEMIA WITH HYPOALBUMINAEMIA

Normally about 40% of the total serum calcium is bound to albumin, with about 10% chelated to anions and about 50% ionized and unbound. The ionized fraction is the biologically active fraction. With decreased albumin, the bound fraction is decreased, but the ionized fraction is often within the reference range and therefore clinical signs of hypocalcaemia are usually not seen. Ionized calcium can be measured to ensure that it is within the reference range.

The total calcium can also be 'corrected' for hypoalbuminaemia and while the accuracy of this calculation is questionable, it is useful as an estimation. The SI units must first be converted to conventional units, e.g. for this case:

$$2\ \text{mmol/l calcium} \times 4 = 8\ \text{mg/dl}$$

$$13.7\ \text{g/l albumin} \times 10 = 1.37\ \text{g/dl}$$

$$\text{Corrected calcium (mg/dl)} = \text{calcium (mg/dl)} - \text{albumin (g/dl)} + 3.5$$

$$= 8 - 1.37 + 3.5 = 10.13\ \text{mg/dl calcium}$$

To convert this back to SI units, divide by 4, e.g. 10.13/4 = 2.54 mmol/l, which is within the reference range.

Serum cholesterol concentration was decreased to 1.7 mmol/l (reference range 3.8–7.0 mmol/l). Serum liver enzyme concentrations were moderately increased, with an alkaline phosphatase of 242 IU/l (reference range 20–60 IU/l) and ALT of 174 IU/l (reference range 21–102 IU/l).

Serum cobalamin concentration was decreased at 112 ng/l (reference range 215–908 ng/l) as was serum folate at 4.5 µg/l (reference range 7.7–24.4 µg/l).

Urinalysis results were unremarkable, with a urine specific gravity of 1.040. Urine protein to creatinine ratio was 0.05 (reference range <0.5).

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Table 25.1 Determination of the type of fluid present in an effusion

	Transudate	Modified transudate	Exudate
Specific gravity	<1.017	1.017–1.025	>1.025
Total protein (g/l)	<25	25–50	>30
Nucleated cells per microlitre	<1,000	500–1,000	>5,000
Predominant cell type	Mononuclear, mesothelial	Lymphocytes, monocytes, mesothelial, RBCs, neutrophils	Neutrophils, mononuclear, RBCs
Bacteria	Absent	Absent	Variable

Faecal analysis was negative for parasites and also negative on culture for enteropathogenic bacteria.

Imaging

Abdominal ultrasound revealed changes (speckling and increased thickness) in the small intestinal mucosa, consistent with lymphangiectasia and a protein losing enteropathy. Free fluid was noted within the pleural and peritoneal spaces. Thoracocentesis was performed and 340 ml of a clear fluid was removed from the pleural space. Analysis of this was consistent with a pure transudate.

CLINICAL TIP ON FLUID ANALYSIS

The type of fluid present in an effusion gives an indication of the source. Modified transudates, transudates and exudates are distinguished by the specific gravity, total protein, number of nucleated cells, the cell type and the presence of bacteria (see Table 25.1).

With an albumin concentration of <15 g/l in this dog, the pure transudate found in the abdomen was consistent with decreased oncotic pressure as the cause for the effusion.

Endoscopy

Gastroduodenoscopy was performed. The gastric mucosa appeared normal, but the duodenal mucosa was grossly abnormal with a very irregular, cobble-stone appearance (Fig. 25.1).

Histopathology of gastric mucosa was interpreted as within normal limits. The histopathological analysis of the duodenal mucosa revealed moderate infiltration of the lamina propria with inflammatory cells, mostly lymphocytes and plasmacytes and distension of the lacteals.

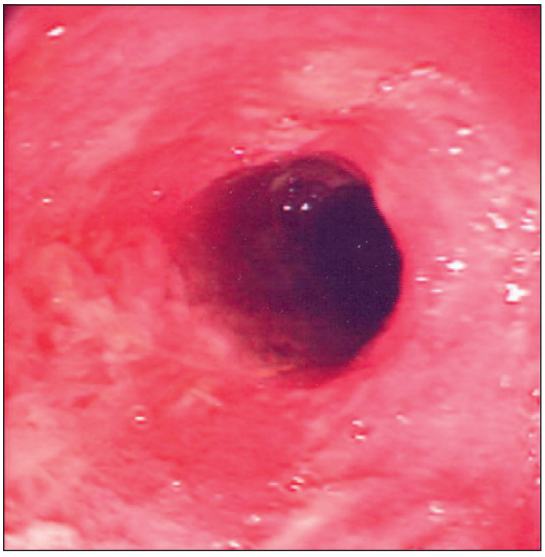


Fig 25.1 Endoscopic of the small intestine in a dog with severe protein losing enteropathy. There is loss of the normal mucosa with haemorrhage and inflammation present.

Diagnosis

A diagnosis of protein losing enteropathy due to lymphocytic plasmacytic IBD and lymphangiectasia was made. The term protein losing enteropathy is a description of the pathology and is not a final diagnosis. Where

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there is low protein thought to be due to gastrointestinal loss, the underlying cause should be determined.

CLINICAL TIP

A scoring index for canine IBD (the canine IBD activity index or CIBDAI) has been developed. The canine IBD activity index includes:
A. attitude/activity
B. appetite
C. vomiting
D. stool consistency
E. stool frequency
F. weight loss
These six variables are scored 0 to 3:
0 = normal
1 = mild change
2 = moderate change
3 = severe change

The summary of these six variables gives the total composite CIBDAI score, which is interpreted as:
0 to 3 = clinically insignificant disease
4 to 5 = mild IBD
6 to 8 = moderate IBD
9 or greater = severe IBD

The canine IBD index for this dog was calculated as:

Attitude and activity = 2

Appetite = 2

Vomiting = 1

Stool consistency = 3

Weight loss = 2

for a score of 10, which is severe IBD.

Treatment and outcome

An elimination diet of home cooked chicken and rice was initiated, along with metronidazole at 10 mg/kg po q 12 hours, prednisolone at 5 mg (1 mg/kg) po q 12 hours and weekly subcutaneous cobalamin injections at 125 mg per week.

One month later, the dog was re-admitted to the hospital. He had continued to have diarrhoea and weight loss. He had begun to vomit two to three times per day and was dull. On physical examination, he showed discomfort on palpation of the cranial abdomen. A Snap cPL (canine pancreatic specific lipase) test was positive and a quantitative Spec cPL was 600 µg/l (reference range <200), consistent with a concurrent diagnosis of pancreatitis. His serum albumin and globulin concentrations had decreased to 12.8 g/l and 13.3 g/l, respectively (reference ranges 26–35 g/l and 28–37 g/l).

He was treated with intravenous fluids, analgesia (buprenorphine at 0.02 mg/kg iv q 8 hours) and an antiemetic (maropitant at 1 mg/kg sq q 24 hours) for 3 days. He began to eat small amounts by the second day, but was still dull and was developing ascites.

Parenteral nutrition was initiated via a jugular catheter to support him nutritionally and was continued for 3 days.

CLINICAL TIP ON PARENTERAL NUTRITION

Parenteral nutrition in veterinary patients involves supporting the patient's nutritional requirements via the parenteral route. Compounding (correctly mixing together the ingredients in the right proportions in a sterile manner) parenteral solutions is beyond the scope of most veterinary practices, however, there are three-in-one products containing glucose, amino acids and lipids which are suitable for most patients on a short-term basis (e.g. 3–7 days). Solutions with an osmolarity of greater than 650 mOsm/l should be administered via a central line, i.e. a long catheter placed in the jugular vein or threaded up the saphenous vein to the vena cava. Solutions of 400 to 650 mOsm/l may be administered via a standard catheter in a peripheral vein using a 1.2 micron in-line filter. When using a central line, the nutritional goal is to meet but not exceed the patient's resting energy requirement, as calculated by body $wt^{0.75} \times 70$ = daily kcal. Peripheral parenteral nutrition is an excellent way to provide partial nutrition for several days and either peripheral or central can be combined with partial enteral nutrition.

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Further medical support included azathioprine at 2 mg/kg po q 24 hours, ranitidine at 2 mg/kg po q 8 hours and budesonide at 1 mg po q 24 hours. Budesonide is a locally acting corticosteroid which has high first-pass metabolism in the liver and therefore has minimal systemic side effects compared to other corticosteroids. Prednisolone was continued at a reduced dose of 1 mg/kg po q 24 hours. The dose was reduced as corticosteroids may be associated with an increased risk of pancreatitis. As the restricted protein home-cooked diet had not improved his clinical signs, he was changed to a canned low fat diet to decrease the risk of recurrent pancreatitis. Weekly subcutaneously injections of 250 µg cobalamin were given.

CLINICAL TIP ON COBALAMIN

Many cats and dogs with small intestinal disease are deficient in cobalamin (vitamin B_{12}) and it is often necessary to supplement this parenterally. Weekly subcutaneous injections of cobalamin at 250 to 500 µg per patient are efficient in repleting serum cobalamin concentrations. Adequate serum cobalamin may be necessary for repair of the inflamed bowel.

The dog responded very well to the change in medication and the treatment with parenteral nutrition. His serum albumin concentration increased to 20.8 g/l and his serum globulin concentration increased to 19.2 g/l. His ascites resolved with the increase in serum protein concentrations. His appetite improved and his faecal consistency improved to a soft formed consistency.

Over the next 2 months (month 3 after diagnosis), the dog continued to improve in his appetite, stool quality and exercise tolerance. His weight increased to 5.65 kg. The jugular vein where the central line catheter had been placed did not fill when occluded and the presence of a thrombus was suspected. His serum proteins had improved, with an albumin concentration of 26.1 g/l and serum globulin concentration of 16.0 g/l.

NURSING ASPECTS OF JUGULAR CATHETERS

The main issues with indwelling catheters are the accumulation of thrombus and the risk of infection. Thrombus formation starts the moment you puncture the endothelium of the vein and forms a sleeve around the catheter that either breaks and sloughs off or plugs the end. Thrombosis also starts away from the insertion site because the catheter tip irritates the vessel wall.

Bacteria can be carried in during venepuncture or can be introduced to the catheter during injections, line changes or by having hair contaminating the hub at the time of iv line connection. Thrombi favour the accumulation of bacteria, which form biofilm that itself favours thrombosis. To reduce these sources of damage to the vessel and catheter, you can

1. use minimally traumatic venepuncture technique
2. use soft catheters
3. use sterile technique meticulously for every catheter placement and every subsequent use of the catheter.

It is often recommended to leave jugular catheters in for no longer than 3 days; however, if you are diligent with the care (and lucky), you can extend the catheter life for much longer than 3 days. If the clinical staff is not careful and meticulous, then even 3 days can be too long.

One month later (month 4 after diagnosis) the prednisolone dose was decreased to 0.8 mg/kg po q 24 hours while maintaining the other medications and diet. A month after that he was seen for a re-visit appointment. After decreasing the prednisolone dose the owners reported that the dog's appetite had decreased, he had become more lethargic and his faeces had become softer. He had also lost 0.3 kg in body weight. The prednisolone dose was increased to 1 mg/kg alternating with 0.5 mg/kg every other day.

A month after the increase in the prednisolone dose the dog was seen for a re-visit appointment and the owner reported that he was brighter, exercising well and

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his stool quality had improved again. His weight had increased to 5.7 kg. His serum albumin concentration was 24.9 g/l and his serum globulin concentration was 15.6 g/l.

A month later the prednisolone dose was decreased to 0.5 mg/kg po q 24 hours and the dog remained fairly stable on this (plus the other medications and diet) for a further 5 months. When he was seen at that time, he was showing signs of corticosteroid side effects, with mild abdominal distension, a sparse hair coat and hyperpigmentation on his ventral abdomen. The owners reported that the dog did urinate and drink more than prior to corticosteroid administration, but not excessively. His stools were reported to be soft and his weight was slightly reduced at 5.5 kg.

At this visit his haematology showed a mild neutrophilia 19.6 × 10^9/l (reference range 3.6–12.0 × 10^9/l), likely due to the corticosteroid administration. Serum albumin concentration was 16.9 g/l and serum globulin concentration was 14.9 g/l. Abdominal ultrasound showed an increase in the small intestinal wall thickness to 6.8 mm (previously 6.0 mm), suggesting that potentially the inflammation had increased. The prednisolone dose frequency was decreased to 0.5 mg/kg po q 48 hours. At a re-visit 1 month (month 11) later the owners reported that the dog's appetite had decreased, he had became lethargic, his faeces were softer and he had lost a further small amount (0.2 kg) of weight. The prednisolone dose was again increased so that he received 1 mg/kg one day alternating with 0.5 mg/kg po every other day. Four months later (month 15) the dose was again decreased to 0.5 mg/kg po per day.

The dog was maintained on these medications and diet for a further 6 months (to month 21 after diagnosis) with a relatively good quality of life. Further attempts to decrease the dose to every other day resulted in reoccurrence of clinical signs.

At the 21 month revisit, the dog was found to again have diarrhoea, weight loss and a variable appetite. On physical examination a 3/6 systolic murmur was detected over the tricuspid valve. His serum albumin concentration was still decreased at 19.6 g/l.

There were decreases in his serum concentration of calcium (2.0 mmol/l; reference range 2.2–3.0 mmol/l), phosphorus (0.7 mmol/l; reference range 0.9–2.0 mmol/l) magnesium (0.55 mmol/l; reference range 0.69–1.18 mmol/l) and potassium 3.2 mmol/l; reference range 3.6–5.6 mmol/l).

Echocardiography confirmed the presence of mild pulmonary hypertension; which may have been due to pulmonary thromboembolism. None of the chambers of the heart were enlarged and there was no evidence of hypertrophy.

Additional treatment at this time included a vitamin and mineral supplement plus oral calcitriol daily (2.5 ng/kg po q 24 hour). Weekly serum calcium concentration tests were scheduled to ensure that hypercalcaemia did not develop.

Outcome

The dog did not respond well to treatment at this time and remained thin and lethargic. Unfortunately at the next visit 2 weeks later to the hospital the dog had a cardiac arrest and died. Post mortem revealed a very large thrombus filling the right ventricle and pulmonary artery, which were highly likely to be the cause of death.

DISCUSSION

Protein losing enteropathy (PLE) refers to the excessive loss of proteins into the gastrointestinal tract, which, if severe enough, causes a decrease in the plasma albumin and globulins. When serum albumin concentration is decreased, there is an increased risk of ascites and pleural fluid accumulation due to the loss of oncotic pressure. Causes of PLE include IBD (lymphocytic-plasmacytic, eosinophilic or granulomatous), neoplasia, foreign bodies, intussusception, lymphangiectasia, immune-mediated and allergic diseases.

Inflammation of the bowel increases the permeability, causing loss of proteins from the plasma and interstitium. In this dog's case, there was also evidence of lymphangiectasia, which in adult animals is usually associated with obstructive lesions in the lymphatic vessels. The lymphatic stasis causes lymphatic hypertension, which causes interstitial fluid to leak into the abdomen and into the intestinal lumen. Lymphocytes are also lost into the intestinal lumen and dogs with lymphangiectasia often have a low lymphocyte count, although this finding is not very specific as stress or illness often causes lymphopenia.

Treatment of inflammatory bowel disease in small animals is somewhat empirical as there is a poor understanding of the underlying cause of the disorder, which may indeed be a collection of disorders with a commonality of histopathological changes. The mainstays of treatment include dietary therapy, corticosteroids, azathioprine, ciclosporin, antibiotics (e.g. tylosin or metronidazole) and possibly pre- or probiotics, in varying combinations. Where the side effects of corticosteroids

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become severe enough to decrease the dog's quality of life, other medications (e.g. azathioprine or ciclosporin) should be added to try to decrease the corticosteroid dose without losing remission of disease.

As in this case, hypocalcaemia has been reported in protein losing enteropathies, which can be due to the low albumin; however, these changes can also occur due to malabsorption of vitamin D. Unabsorbed fatty acids can also bind dietary calcium to form insoluble salts which are not absorbed. Treatment with the calcitriol form of vitamin D was initiated in this case to try to increase intestinal calcium absorption.

Epidemiology

Most dogs with lymphocytic plasmacytic enteritis are middle aged to older, although it can occur in younger dogs. Breed related immunoproliferative enteropathy is recognized in Basenjis and breed related protein losing enteropathies have been reported in soft coated wheaten terriers and Lundenhounds. Sharpei dogs also have an increased risk for inflammatory enteritis, likely related to IgG or IgA deficiencies. Many German shepherd dogs also have IgA deficiency which likely plays a role in chronic enteropathies. Anecdotally, some clinicians do believe that Jack Russell terriers may also be at risk for enteritis.

Prognosis

Inflammatory bowel disease can be difficult to control; in one study only 26% of dogs with IBD were considered to be in remission at a 6 month follow-up check. The presence of hypoalbuminaemia is a negative prognostic indicator and an elevated canine pancreatic lipase concentration is also associated with a negative outcome.

The long-term prognosis for dogs with PLE due to inflammatory causes is also guarded and most require long-term therapy. The prognosis for lymphangiectasia is also guarded.

As with other causes of protein loss, there can be loss of antithrombin, which has a similar molecular weight to albumin. This loss, along with vascular damage from immune complexes, may predispose these cases to the formation of thrombi. In this dog's case, he had an additional risk factor due to the formation of a large thrombus in his jugular vein, which could have seeded other thrombi.

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26 Inflammatory bowel disease and adverse reaction to food in a dog

INITIAL PRESENTATION

Six months of soft to watery diarrhoea and weight loss and episodes of vomiting
Signalment: 4-year-old entire male Sharpei, body weight 15.5 kg

CASE HISTORY

The dog presented with a chronic, 6-month history of diarrhoea and weight loss. He was producing large volumes of soft faeces, with no increase in frequency of defecation, no blood or mucus in the faeces and no tenesmus while defecating. There had been episodes of vomiting occurring nearly every day. The dog usually vomited digested food and bile and this often occurred soon after eating. The owner reported that the dog had become quieter than he had been previously.

CLINICAL TIP

The presence of weight loss, large volumes of faeces and lack of tenesmus, mucus or haematochezia indicate diarrhoea of small intestinal origin. Localizing the source of the diarrhoea aids the clinician in making a differential diagnoses list and deciding upon diagnostic techniques.

CLINICAL TIP

The presence of digested food and bile in the vomited material indicates that it is likely to be true vomiting rather than regurgitation. The length of time after eating that an event takes place does not necessarily indicate whether it is vomiting or regurgitation.

The dog had been fed a variety of diets in an attempt to control the gastrointestinal signs, including a highly digestible diet for gastrointestinal disorders and a premium dry dog food.

The current diet being fed was a commercial dry diet based on lamb and rice. The dog had a good appetite while on this food, but there was no improvement in the diarrhoea or vomiting. In addition to eating dog food, the dog also scavenged from rubbish and was fed treats of human food.

His vaccinations were upto date and he had been treated for parasites 6 months prior to presentation. He had not travelled outside of the UK.

The diagnostic tests and treatments from the previous veterinary surgeon included:

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- Faecal examination for parasites: negative
- Treatment with sulfasalazine and metronidazole for 1 week (doses not reported)
- Dietary therapy: a dry veterinary diet designed for intestinal disease for 2 weeks, then a dry commercial premium dog food for 2 weeks

There had been an initial improvement on these treatments and then the diarrhoea reoccurred 2 months later. He was treated again with sulfasalazine, metronidazole (doses not reported) and prednisolone at 0.6 mg/kg, with no improvement.

CLINICAL TIP

Sulfasalazine is used for large intestinal diarrhoea so would not be expected to improve the signs in this dog. Metronidazole does help in some cases of diarrhoea. Prednisolone should probably not be used without a confirmed diagnosis; if it was being used for presumptive inflammatory bowel disease, the dose was too low as it is between the physiological dose for dogs (0.15–0.25 mg/kg/day) and the anti-inflammatory dose for dogs (0.5–1 mg/kg/day).

CLINICAL TIP

While the diets that were used in this case are highly digestible they do include several protein sources and are not appropriate as elimination diets. The first ingredient in the lamb and rice diet was wheat, showing that it is necessary to read the labels. The diets at the time of this case contained the following main ingredients:

a. Veterinary intestinal diet: maize, gluten meal, chicken and turkey meal
b. Premium dry diet: chicken, rice, maize, poultry meal, gluten meal, wheat, fish oil, egg
c. Lamb and rice based diet: wheat, lamb, rice, bran

On physical examination the dog was quiet and alert and his hydration was adequate. His body condition score was 3/9, with evidence of muscle loss. His mucous membranes were pink and capillary refill time was <2.5 seconds. His temperature, pulse and respiration parameters were within normal limits, thoracic auscultation was unremarkable and lymph nodes were within normal limits. On abdominal palpation there were thickened loops of small intestines which felt fluid filled. He had a dry dull hair coat.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Chronic small intestinal diarrhoea
- Weight loss with good appetite
- Occasional vomiting

DIFFERENTIAL DIAGNOSIS

Chronic small intestinal diarrhoea

- Dietary
  - gluttony – the dog did have a good appetite but this is unlikely to cause chronic diarrhoea
  - changes of diet – possible with scavenging and snacks
  - adverse reaction to food: intolerance or allergy
- Gastric disorders
  - rapid gastric emptying
- Small intestinal disease
  - infectious, e.g. bacterial, viral, fungal
  - gastrointestinal parasites
  - inflammatory bowel disease
  - infiltrative neoplasia, e.g. lymphoma, mastocytosis
  - partial obstruction, e.g. intussusception, foreign body
  - brush border defects
  - lymphangiectasia
- Pancreatic disease
  - chronic pancreatitis
  - exocrine pancreatic insufficiency
  - pancreatic neoplasia
- Liver disease
  - bile duct obstruction
  - liver failure
- Kidney disease – unlikely as he did not have polyuria/polydipsia (pu/pd), although glomerular disorders may present without pu/pd
- Miscellaneous
  - congestive heart failure – no signs consistent with this
  - immunodeficiencies – e.g. IgA deficiency, which has been reported in the Sharpei dog
  - hypoadrenocorticism

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Weight loss with good appetite

- Dietary
  - unbalanced or poor quality diet
  - insufficient food provided
- Malassimilation
  - intestinal disorders as above for diarrhoea
  - exocrine pancreatic insufficiency
  - liver failure
- Nutrient loss
  - diabetes mellitus (unlikely as not pu/pd)
  - protein losing enteropathy
  - protein losing nephropathy
- Increased nutrient requirements
  - fever (not present)
  - hyperthyroidism (unlikely)

Occasional vomiting

- Dietary, small intestinal, liver and pancreatic disorders as above
- Stomach
  - gastritis
  - gastric ulcers
  - inflammatory bowel disease
  - foreign body
  - parasites
- Systemic disease
  - uraemia
  - liver failure
  - sepsis
  - congestive heart failure
  - acidosis
  - hypoadrenocorticism
  - ketoacidotic diabetes mellitus
  - hyperparathyroidism
  - gastrinomas
- Neurological diseases (no other signs consistent with these in this dog)
  - dysautonomia
  - vestibular disease
- CNS disease
- Drugs, toxins (no known history outside of medications provided by referring veterinary surgeon)

CASE WORK-UP

Minimum data base

Haematology, serum chemistry and urinalysis were performed. The most significant findings were a hypoalbuminaemia of 15.2 g/l (reference range 26–35 g/l) and borderline hypoglobulinaemia of 21.9 g/l (reference range 22–45 g/l). He also had a peripheral eosinophilia of 1.7×10^9/l (reference range 0–1×10^9/l). His urine had a specific gravity of 1.040 with a pH of 6.5. The urine protein to creatinine ratio was 0.8 (slightly elevated above the reference range of 0.5) and there were calcium oxalate crystals present in the sediment.

Low albumin may be caused by intestinal loss, urinary loss, decreased liver production due to liver disease, third spacing, a shift from the vascular space into the interstitial space or decreased liver production during an acute phase reaction from inflammation. With a concurrent decrease in globulins and only a mild increase in urine protein and a history of diarrhoea, intestinal loss was the most likely diagnosis in this dog.

Eosinophilia may be caused by hypersensitivity (e.g. food, parasites), eosinophilic diseases (eosinophilic inflammatory bowel disease, eosinophilic pneumonitis, eosinophilic granulomatous disease, eosinophilic leukaemia) and is also sometimes seen with mast cell tumours, carcinoma and hypoadrenocorticism.

Faeces were submitted for parasitology and culture of enteropathogens; both of these results were negative.

CLINICAL TIP

Many intestinal parasites shed eggs only intermittently, so a negative faecal examination does not rule out parasites. Doing three faecal examinations from three different stool samples has been recommended especially in cases with gastrointestinal signs, de-worming is usually recommended.

Adrenocorticotrophin hormone stimulation test

An adrenocorticotrophin hormone (ACTH) stimulation test was performed and the results were within the reference range (pre-ACTH stimulation of 223 mmol/l increasing to a post-stimulation of 443 mmol/l), ruling out hypoadrenocorticism.

Endoscopy

Endoscopy showed an increased duodenal granularity, hyperaemia and friability (Fig 26.1). Results of the histopathology of the gastric and small intestinal biopsies showed an increased in filtration of eosinophils, consistent with eosinophilic inflammatory bowel disease.

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CLINICAL TIP ON INFLAMMATORY BOWEL DISEASE SCORING

A scoring index for canine inflammatory bowel disease (IBD) (the canine IBD activity index or CIBDAI) has been developed. The canine IBD activity index includes:
A. attitude/activity
B. appetite
C. vomiting
D. stool consistency
E. stool frequency
F. weight loss

These six variables are scored 0 to 3:
0 = normal
1 = mild change
2 = moderate change
3 = severe change

The summary of these six variables gives the total composite CIBDAI score, which is interpreted as:
0 to 3 = clinically insignificant disease
4 to 5 = mild IBD
6 to 8 = moderate IBD
9 or greater = severe IBD

The CIBDAI has shown correlation with the acute phase protein reactant C reactive protein and severity of histology changes. Following treatment for IBD both the CIBDAI and serum C reactive protein decreased.

To calculate the CIBDAI score in this dog the following were used: a mild decrease in his activity (1), no change in his appetite (0), a moderate change in his faecal quality (2) moderate amount of vomiting (2) and moderate weight loss (2), for a score of 7 or moderate IBD.

Treatment and outcome

This dog was initially treated with prednisolone at 2 mg/kg po q 24 hours, subcutaneous cobalamin injections of 500 µg per week for 4 weeks and fenbendazole at 50 mg/kg po q 24 hours for 3 days. The owner did not wish to change the food, so he continued to feed the commercial lamb and rice diet.

At 19 days, there was no response to treatment and the dog had vomited after being fed pancakes. Prednisolone was continued and the dog was changed on to a single novel protein, gluten-free, highly digestible diet.

At 37 days, the dog was still occasionally vomiting and the stools were loose. The owner said he had not been giving the prednisolone consistently.

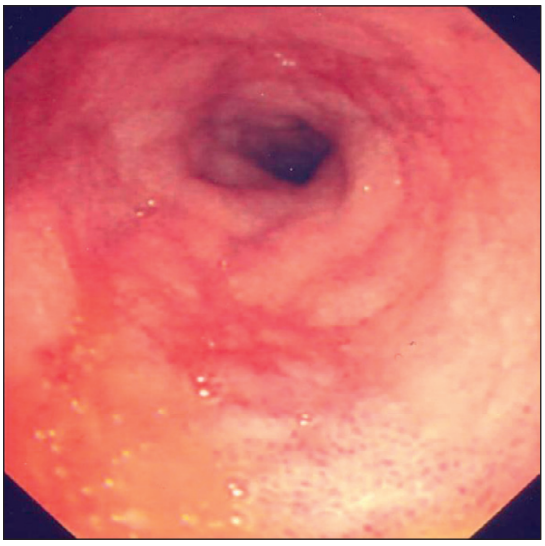


Fig 26.1 Endoscopy picture of the small intestine of a dog with eosinophilic inflammatory bowel disease. There is roughening of the mucosa with small pinpoint haemorrhagic areas

At the day 42 check up, the owner had been giving prednisolone, but had added potatoes, gravy, bread and poultry to the diet and there was no improvement in the clinical signs. The prednisolone dose was decreased to 1.6 mg/kg po q 24 hours.

At the day 56 check up, the owner reported that he had complied with the dietary and medication therapies. The dog had gained 1 kg and his stools were soft but formed. The prednisolone dose was decreased to 1.25 mg/kg po q 24 hours and a therapeutic plan was made to decrease the dose about 20% every 3 weeks, until the dose was less than 0.5 mg/kg q 24 hours, when it was to be changed to an every other day regime.

At the day 120 check up, the owner reported that he had stopped giving prednisolone. The dog had gained 2.5 kg and the stools were still soft to firm and he was much brighter and more active. The serum albumin and globulins were within the reference range.

DISCUSSION

This dog did not improve until wheat products were eliminated from the diet and the medical treatment (prednisolone) did not work without dietary intervention.

Because of the response to an elimination diet, it is highly likely that this dog had an adverse reaction to food. In order to determine what ingredient caused the reaction and to confirm that the response was due to an adverse reaction to food, a food challenge would have been necessary.

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CLINICAL TIP

Elimination-challenge dietary trials confirm or rule out adverse reactions to food, but do not establish an immune mediated basis (hypersensitivity) for the reaction. It is however, probably not important to establish an immune mediated basis as it does not change the management of the case.

The elimination trial involves putting the patient on a restricted diet of a single protein source and a single carbohydrate source. The diet used should contain ingredients to which the animal has not previously been exposed. There is no universal 'hypoallergenic diet' containing intact proteins, as allergenicity depends upon the foods that an individual has previously been fed. Homemade diets are often recommended; however, 20% of the dogs having no signs when fed a homemade diet show signs when fed a canned diet with the same constituents. Another option is to use a commercial diet with hydrolyzed protein. Supplements with n-3 or n-6 fatty acids should not be given, as they may cause allergic reactions in animals that are allergic to fish.

In a study on food sensitive cats with gastrointestinal signs there was an immediate cessation of vomiting and a decrease in diarrhoea within 3 to 4 days, in most of the cats after feeding a novel protein diet. Most of these cats also showed a recrudescence of signs after 3 to 5 days of an antigenic challenge.

After a 2-week period with no clinical signs, a small amount of a second protein source (e.g. one-half to two tablespoons of powdered milk) to which the animal has been previously exposed can be added to the controlled diet and fed for 3 days – or less if signs are observed. This procedure is repeated weekly until a suspect food appears to cause the signs. After the suspect food is identified, remove that protein source from the diet and observe for evidence of resolution of signs. After 1 to 2 weeks of no signs, challenge the animal with the suspect food again.

This owner, as are many, was reluctant to challenge the animal after the resolution of signs and it was necessary to design a diet without confirmation of the allergen. The diet fed in cases like this should be palatable, complete and balanced. Use of a high quality commercial gluten free, moderate to low fat, lactose free diet with limited antigens and few additives may work well in these cases. In some cases it is difficult to find a protein source which has not been fed; in these cases use of a hydrolyzed protein diet or one which has not been fed for the previous 6 months may be tried. While many of these patients are underweight, as was this one, excess dietary fat should be avoided during small intestinal malassimilation as malabsorbed fatty acids and bile acids can cause secretory diarrhoea.

It may have been possible to manage this case with dietary therapy alone; however, most cases of eosinophilic inflammatory bowel disease also require therapy with immune suppressive drugs such as prednisolone.

The lack of owner compliance with diet and medications in this case complicated the management and prolonged the time to recovery. Many medications are not given according to directions and clinicians must be sensitive to the owners' ability and willingness to administer medications and control the diet. Asking clients in a non-judgmental manner if they are having any problems giving the prescribed medication or if they have missed any doses is one way to assess compliance and identify any difficulties clients may have in giving medications to pets. If a client does not admit to missing any doses but compliance is still in doubt, a follow-up appointment for re-examination and a pill count may reveal a problem with compliance.

Prognosis

In one retrospective study, only 26% of 80 cases of IBD were considered to be in remission at a 6-month follow-up. Many of the dogs (about 50%) showed intermittent signs of disease and most of these (65%) were still on treatment. The presence of hypoalbuminaemia is a negative prognostic indicator and an elevated canine pancreatic lipase concentration is also associated with a negative outcome.

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27 Intussusception in a cat

INITIAL PRESENTATION

Diarrhoea and poor appetite

Signalment: 2-year-old female entire domestic shorthaired cat, body weight 3.2 kg

CASE HISTORY

The cat lived at a rescue centre and was brought in with a 3-day history of poor appetite and having watery diarrhoea. She had been at the rescue centre for about a month and had not been vaccinated or de-wormed during that time as she was pregnant when admitted there. She was nursing two 3-week-old kittens at admission to the hospital and was producing an adequate amount of milk for them.

There was no blood or mucus present in the cat's diarrhoea. It was not known how frequently she was defecating, but she had not been observed to be straining to defecate. The staff at the rescue centre did not believe that she had been vomiting, but were unsure about that part of the history. She did not appear to be drinking or urinating more than previously.

Her previous history was not known as she had come to the rescue centre as a stray cat.

PHYSICAL EXAMINATION

The cat was bright and alert. There was evidence of recent weight loss and she was estimated to be about 6% dehydrated. Her body condition score was 3/9. Her mucous membranes were pink with a capillary refill time of <2 seconds. Her heart rate was 170 beats per minute and rectal temperature was 38.8° C. Respiratory rate was not determined as she was purring loudly and persistently. At thoracic auscultation, there were increased ronchii heard, but no other abnormal lung sounds. It was thought that the increased airway sounds may have been due to her thin body condition.

She had large mammary glands, appropriate for lactation, but there was no heat or abnormal swelling in them. A firm structure was palpated within her abdomen, which was thought to be possibly faeces within the colon, an intussusception or a uterine disorder.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The cat's problems included diarrhoea, poor appetite, which was thought to be related to the diarrhoea and the firm structure palpated within the abdomen.

DIFFERENTIAL DIAGNOSIS

For the diarrhoea

The diarrhoea was thought to be small bowel in origin; differentials include:

- Intestinal disorders
  - inflammatory bowel disease, e.g. plasmacytic/lymphocytic, eosinophilic, other
  - infectious (viral, bacterial [enterotoxins], fungal – although fungal unlikely in the UK)
  - intussusception

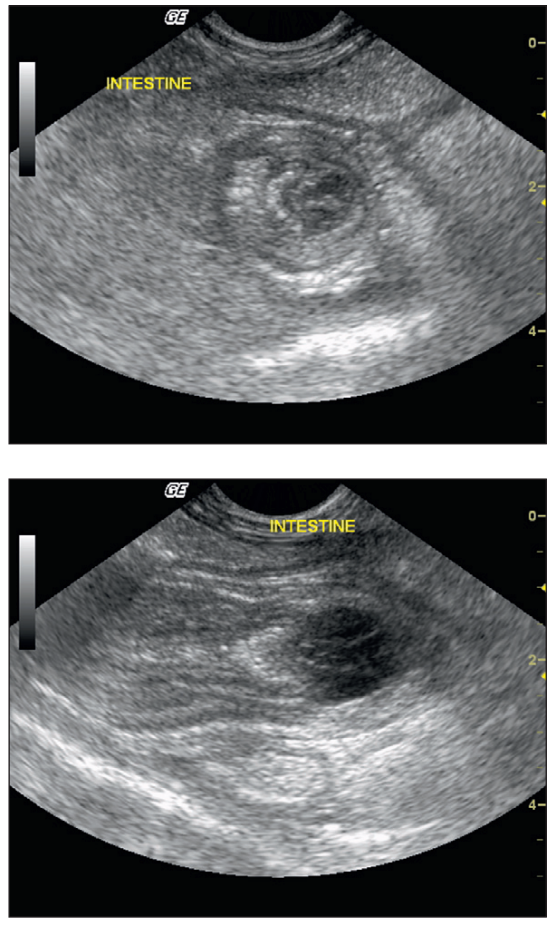
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- parasites, e.g. *Giardia*, cryptosporidium, Strongyloides, ascarids, hookworms, *Toxoplasma gondii*
- neoplasia, e.g. lymphoma, mastocytosis
- lymphangiectasia (uncommon in cats)
- ileus, e.g. hypokalaemia, enteritis, dysautonomia
- antibiotic responsive diarrhoea

- Pancreatic disorders
  - pancreatic exocrine insufficiency
  - chronic pancreatitis
- Liver disorders
  - liver failure
  - intrahepatic cholestasis
  - bile duct obstruction
- Kidney disease and uraemia
- Hyperthyroidism
- Congestive heart failure
- Hypoadrenocorticism (uncommon in cats)

For the firm structure

- Intussusception
- Uterine disorder, e.g. infection, retained kitten or placenta
- Colonic disorder or retained faeces



Figs 27.1 and 27.2 Transverse and sagittal ultrasound pictures of an intussusception in a cat showing the 'doughnut' appearance in the transverse, and the double layering of the intestine in the sagittal (courtesy of Carolina Urraca del Junco)

CASE WORK-UP

Minimum data base

Intravenous fluids at a rate to correct 6% dehydration were initiated. Haematology showed a neutrophilia of 16.3 × 10^9/l (reference range 2.5–12.8 × 10^9/l) and monocytes of 2.14 × 10^9/l (reference range: 0.07–0.85 × 10^9/l) thought to have been consistent with an inflammatory process. Serum chemistry parameters were within the reference ranges. Tests for feline leukaemia virus and feline immunodeficiency virus were negative using a SNAP Combo™ test.

Imaging

Thoracic radiographs showed no abnormalities. Abdominal ultrasound showed a 6-cm long section of small intestine in the mid-cranial abdomen with the characteristic multiple layering of an intussusception (Figs 27.1 and 27.2). At the tip of the intussusceptum there was a round septated hypoechoic to anechoic structure present within the intussuscipiens which was suspected to be an abscess.

Treatment and outcome

Presurgical medical management

The cat was started on intravenous clavulanate-potentiated amoxicillin (20 mg/kg q 8 hours) and intravenous fluids were continued until she was thought to be stable enough for anaesthesia and surgery.

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CLINICAL TIP

Medical management of intussusception is generally not possible, so the primary treatment is surgical. Surgical management of intussusception involves reduction and/or resection and anastomosis. Upon identifying the intussusception, it is isolated and packed off from the peritoneal cavity. When possible, reduction is facilitated by gentle milking of the intussusceptum from the intussuscipiens. The ensheathing layer is gently compressed over the apex of the intussusceptum while gentle traction is placed on the ileum. In relatively acute cases, reduction is usually accomplished and bowel viability is closely scrutinized. When mature adhesions have formed between the invaginated and ensheathing layers, as in this cat, reduction is usually not possible and resection and anastomosis are performed.

In some cases, following reduction and/or resection and anastomosis of the intussusception, a bowel plication or enteropexy technique is performed.

Surgical treatment

In this case, surgery proceeded with a routine midline incision through the linea alba from just caudal to the xiphoid to just cranial to the pubis. An exploratory coeliotomy located a jejuno-jejunal intussusception. The intussusception proved to be non-reducible, as the serosa started to tear when reduction was attempted. The affected piece of bowel was resected with an oblique cut made on the oral side and a straight cut on the aboral side. The oral side of the intestine was spatulated and an end-to-end anastomosis was performed. A routine ovariohysterectomy was performed at the same surgery. The excised loop of intestine was submitted to histopathology to look for an underlying cause.

The histopathology of the biopsy of the resected intestine was described as appearing to be a classic intussusception with a necrotic intussuscepted portion of bowel surrounded by degenerating compressed proximal bowel. There were many bacterial colonies present thought to be commensal bacteria proliferating in the hypoxic and necrotic environment. There was a granulation-like reaction in part of the invaginated part of the intestine that was thought to have been a scarred or healing segment that could have been predisposed to abnormal peristalsis and be the cause of the intussusception.

Postoperative care

After surgery the cat was continued on intravenous fluids, antibiotics and analgesia with morphine. Her rectal temperature increased that night to 40.3° C, but decreased the next morning to 39.0° C without intervention. Enrofloxacin (5 mg/kg iv q 24 hours) was added to her antibiotic treatments.

CLINICAL TIP ON USE OF ENROFLOXACIN IN CATS

In cats, use of enrofloxacin has caused rare incidences of ocular toxicity characterized by mydriasis, retinal degeneration and blindness. These effects were generally seen at higher dosage ranges (>15 mg/kg) and have necessitated a reduction in dosage recommendations in cats to a maximum of 5 mg/kg/day. Other rare adverse effects seen in cats may include: vomiting, anorexia, elevated hepatic enzymes, diarrhoea, ataxia, seizures, depression/lethargy, vocalization and aggression.

After 2 days, the cat was bright and had a good appetite. She was initially offered one-third of her resting energy requirement (RER) of food. Initially it was planned to increase her feed to 100% of resting energy requirement by day 3, but after the first day of feeding she had diarrhoea again, so she was kept at one-third of RER. The diarrhoea resolved 3 days later.

Outcome

Prior to discharging the cat back to the rescue centre, she was de-wormed with 5 days of fenbendazole at 50 mg/kg/day po. At her re-visit appointment 5 days later she was very bright and eating well and the diarrhoea had resolved.

DISCUSSION AND EPIDEMIOLOGY

Intussusceptions occur less often in the cat than in the dog. When they do occur, intussusceptions are most often seen in immature cats, although the results of one

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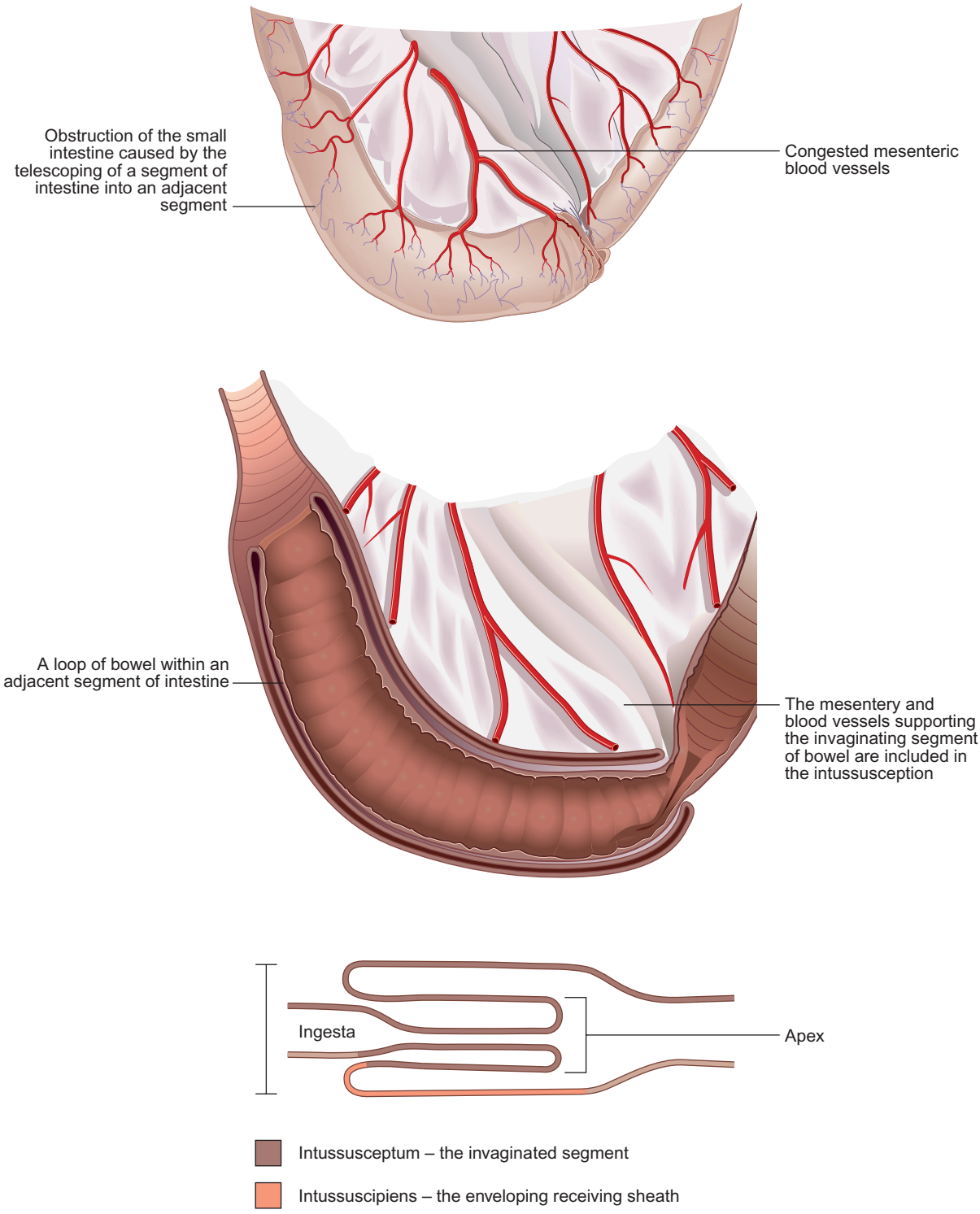


Fig 27.3 Illustration of an intussusception showing the invaginated intussusceptum and the invaginating intussuscipiens

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study suggested that in cats, intussusception has a bimodal age distribution and half of 20 cases were seen in cats less than 1 year of age and nearly half were over 9 years of age. In the older cats it was often associated with alimentary lymphoma or inflammatory bowel disease. In dogs, 80% of the cases of intussusception are in patients less than 1 year of age. Generally there is no increased risk for any breed reported, although in one study over-represented breeds included Siamese and Burmese cats. There is no increased risk reported with gender or neutering.

The exact biomechanical cause of the condition is unknown and it has not been reproduced experimentally. It appears that there is a focal area with inhibited normal propulsive and segmental motility. When a wave of peristalsis moves over the affected area, a proximal bowel segment invaginates (becoming the intussusceptum) into a distal section of bowel (the intussuscipiens). Three layers of bowel wall are essentially created, with two from the intussusceptum and one layer from the intussuscipiens (Fig 27.3).

Heavy intestinal parasitism with ascarids or coccidia as well as severe enteritis are possible predisposing causes and as a rescue cat, the chances of parasitism in this case were thought to be high. Other causes include intestinal viral infection, linear foreign bodies, intestinal tumours and adhesions. Intussusceptions are also seen with increasing frequency after laparotomy on elective or non-elective intra-abdominal procedures.

Clinical signs depend on the completeness and level of obstruction. The majority of intussusceptions in dogs occur at the ileocecocolic junction but jejuno-jejunal intussusceptions or pylorogastric and gastro-oesophageal intussusceptions are reported. In one study the location was most commonly jejuno-jejunal in cats.

Patients with high intussusceptions usually have profuse vomiting, rapid dehydration and early death. Ileocolic intussusceptions often present with a history of sporadic vomiting, inappetence or bloody stools. If jejuno-jejunal intussusceptions like this one do not cause a complete obstruction, it is possible for them to be chronic and have subtle clinical signs. With more complete obstructions, fluid and electrolyte imbalances occur with the distension of the intestines and the number of bacteria proximal to the obstruction increases dramatically resulting in potential endotoxaemia.

Ultrasonography aids in the diagnosis of intussusception of the intestine. The sonographic appearance of an intussusception in a transverse plane has been described as a 'target lesion' or as the 'multiple concentric ring sign', reflecting the concentric layers of bowel wall within the intussuscepted segment (Fig 27.1). On longitudinal scan, an intussusception has the appearance of a thickened segment of bowel with an excessive number of layers that alternate in echogenicity (Fig 27.2). It should be noted that other gastrointestinal disorders can also cause 'target' or 'bull's eye' type lesions. Therefore, when intussusception is suspected on a transverse image, the lesion must be examined in a longitudinal plane as well.

In most cases, the obstruction is incomplete and a chronic course of poor appetite, diarrhoea or bloody tenesmus is seen for several weeks.

In the majority of reported feline intussusception cases, resection and anastomosis of the intestine was necessary. Enteroplication of the intestines after surgery for intussusception used to be routinely recommended, although one study found no significant difference in the follow-up of canine cases where this procedure was or was not done and it is not without potential complications.

Although the invaginated bowel may become devitalized, perforation resulting in peritonitis is relatively rare because the outer ensheathing layer retains its viability and fibrinous adhesions seal the proximal border of the intussusception. Occasionally spontaneous recovery occurs when the nonviable intussusceptum is sloughed and patency of the intestinal lumen is re-established.

Prognosis

Prognosis for a successful outcome after surgery is good; with over 80% of reported cases doing well, although the number of cases reported is low.

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28 Feline exocrine pancreatic insufficiency

Case contributed by Nicki Reed

INITIAL PRESENTATION

Weight loss, diarrhoea, steatorrhoea and vomiting

Signalment: 7-year-old, male neutered domestic shorthaired cat, body weight 3.33 kg

CASE HISTORY

This cat presented for investigation of weight loss, diarrhoea, steatorrhoea and vomiting, which were accompanied by a good appetite.

The cat had been acquired 5 years previously and lived indoors. He had received core vaccinations and regular boosters, but no endo- or ectoparasiticides. His episodes of diarrhoea had occurred since acquisition. The diarrhoea was usually semi-formed and brown and would usually resolve without treatment after 2 to 3 days. The episodes occurred approximately six to eight times per year. The nature of the diarrhoea had changed approximately 3 months prior to presentation. The faeces were described as voluminous, greasy and malodorous and had also previously been white in colour. During defecation the cat did not strain and there was no blood or mucus in the faeces.

Occasional episodes of vomiting (one to three times per year) were also reported. The vomitus generally comprised semi-digested food. In the 3 months prior to presentation the cat's appetite had increased. He was eating a 450 g tin of commercial canned cat food and approximately 50 g of dried biscuits per day.

The cat had always been overweight (previously 6 kg) but there had been a steady weight loss since his last vaccination 8 months previously.

PHYSICAL EXAMINATION

The cat was in poor body condition (body condition score 2/9), but otherwise bright and well. His coat was greasy, matted and unkempt. On abdominal palpation the intestines felt fluid filled, but the cat did not appear uncomfortable during palpation. Thoracic auscultation was unremarkable. His heart rate was 160 beats per minute with matched good quality pulses, respiratory rate 28 breaths per minute and rectal temperature 37.9° C. There was faecal soiling around the perineum.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The primary problems were weight loss and diarrhoea, but the presence of a good appetite helped to narrow the differential diagnoses list and the vomiting and weight loss were thought to be related to the diarrhoea in this case. The clinical signs were consistent with diarrhoea due to small intestinal disease.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for diarrhoea in this cat include:

- Maldigestion
  - exocrine pancreatic insufficiency (EPI)
- Malabsorption
  - inflammatory bowel disease (lymphocytic-plasmacytic, eosinophilic, granulomatous)
  - lymphoma
- Metabolic disorders
  - hyperthyroidism
  - diabetes mellitus
- Gastrointestinal parasites
- Pancreatitis
- Lymphocytic cholangitis

The presence of steatorrhoeic, malodorous faeces suggested a maldigestion problem was most likely.

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CASE WORK-UP

Routine haematology, serum biochemistry and urinalysis were performed as a minimum data base. Total thyroxine was also measured to screen for hyperthyroidism. Results were all within the reference range, with the exception of alanine aminotransferase (ALT), which was markedly elevated at 865 IU/l (reference range 20–75 IU/l) and alkaline phosphatase which was elevated at 124 IU/l (reference range 20–80 IU/l). Faecal analysis for parasites was repeated and was again found to be negative.

More specific investigations for the gastrointestinal tract were performed. Feline pancreatic lipase immunoreactivity (fPLI) and feline trypsin-like immunoreactivity (fTLI) were performed to assess pancreatic function. Folate and cobalamin were measured to assess for malabsorption of these vitamins. Folate and fPLI were both within their reference range. Cobalamin was low at 99 ng/l (reference range 290–1499 ng/l) and fTLI was also low at 9.2 μg/l (reference range 12.0–82.0 μg/l).

Abdominal ultrasonography was performed, primarily to evaluate the liver and pancreas. The liver appeared grossly normal and although the pancreas was identified, it appeared small. In light of the raised liver enzymes, a liver biopsy was felt to be useful; however, prior to this being performed an assessment was made of clotting times. Whilst the activated partial thromboplastin time (APTT) was normal, the prothrombin time (PT) was prolonged at 27.7 seconds (reference range 7.0–12.0 seconds). The liver biopsy was thus deferred.

Diagnosis

The history, clinical examination findings and the subnormal fTLI were supportive of a diagnosis of EPI. Cobalamin deficiency is frequently reported in association with EPI in cats and prolongation of the PT is attributed to Vitamin K deficiency.

Treatment options

Pancreatic enzyme replacement

A number of commercial pancreatic enzyme replacement products are available in powder or capsule form. Supplementation should be in a non enteric-coated formulation, as enteric coating has been shown to reduce the amount of available enzyme in the duodenum. The amount administered should relate to the amount of food fed, rather than the body weight of the animal and should be provided with each meal fed. Palatability appears to be more problematic with cats than with dogs.

This cat was given two capsules of a powdered pancreatic supplement opened and sprinkled on each meal of canned food and he was willing to eat the supplemented food.

A number of cats have refused to eat food containing enzyme replacement powder, despite the ravenous appetite induced by the condition. Fresh frozen pancreas is an alternative if it can be sourced. Bovine pancreas is not covered by the Specified Bovine Offal Order of 1995 and therefore may be used. Pig pancreas may be more appropriate in the United Kingdom where bovine prion disease could be a concern for owners. In countries where Aujesky's disease is present bovine pancreas is more appropriate. Informed written consent should be obtained from owners, as the risk of contracting food poisoning organisms such as *Salmonella*, *Escherichia coli* and *Toxoplasma gondii* remains, although freezing greatly reduces the risk. Approximately 30 to 40 g of pancreas should be mixed with each meal. Although no published data exist regarding the duration of efficacy following freezing, in the author's experience, pancreas that has been frozen for up to 8 months still appears to retain its enzymatic function.

Cobalamin

Replacement of pancreatic enzymes does not address the lack of intrinsic factor (see Clinical Tip in Ch 5). Cobalamin replacement is therefore required on a permanent basis and as the lack of intrinsic factor prevents absorption from the ileum this must be replaced parenterally. Variable doses have been reported. This cat was given 250 μg subcutaneously weekly for 6 weeks. His serum cobalamin concentration was still low at 179 μg/l, so the dose was increased to 500 μg subcutaneously weekly. His serum cobalamin concentration was within the reference range at the next revisit 1 month later, so the frequency of the injections was decreased to monthly. Many owners can be taught to do these injections at home, in the same way diabetic owners will administer insulin.

Folate

Cobalamin deficiency can affect the intestinal villi, leading to malabsorption, which may cause folate deficiency. Folate levels should therefore be assessed and supplemented orally if found to be low (400 μg/day), although in this cat they were within the reference range at diagnosis.

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Vitamin K

If a prolonged PT (and/or APTT) is identified, as in this cat, where the PT was prolonged, then supplementation of vitamin K_1 may be required in the short term to prevent potential bleeding problems. Vitamin K_1 was administered subcutaneously using a fine gauge needle to minimize the risk of haematoma formation, at a dose of 2.5 mg/kg. Oral supplementation was not initially used at it may be less satisfactory because absorption may be compromised from lipase deficiency. The prolonged PT was within the reference range within 24 hours. As in most cases, once enzyme replacement is instituted enteric Vitamin K absorption was satisfactory.

Diet

If the above management strategies had not been fully effective, then consideration would have been given to dietary manipulation. Whilst use of a low fat diet has been proposed, there is little evidence to support that this is necessary. Inflammatory bowel disease is often associated with pancreatitis. This may arise from bacteria being forced up the pancreatic duct from the duodenum when cats vomit or from lymphocytic plasmacytic infiltrate of the pancreas in association with lymphocytic plasmacytic infiltrate of the intestine (and liver). The combination of pancreatitis, inflammatory bowel disease and hepatopathy is sometimes termed triaditis. Cats with EPI may therefore benefit from management of inflammatory bowel disease with a hypoallergenic diet or novel protein diet.

DISCUSSION

EPI is a functional disorder of the pancreas, whereby inadequate production of digestive enzymes results in signs of maldigestion. The primary digestive enzymes involved are trypsin for the digestion of proteins, amylase for the digestion of carbohydrates and lipase for the digestion of fats. Deficiency of these enzymes gives rise to maldigestion of food, production of voluminous fatty stools and subsequent weight loss.

In dogs the condition is most frequently caused by selective atrophy of the acinar glands responsible for the production of digestive enzymes. It is proposed that in some breeds this may be the result of immune-mediated destruction. In cats, EPI is attributed to destruction from chronic pancreatitis and the history of this case is suggestive of bouts of pancreatitis (with likely concurrent cholangitis).

Lipase deficiency in EPI prevents the breakdown of dietary triglycerides into monoglycerides and fatty acids, resulting in the production of steatorrhoea. It is the rancid fat in the faeces that gives rise to their malodorous nature. In addition, absorption of fat-soluble

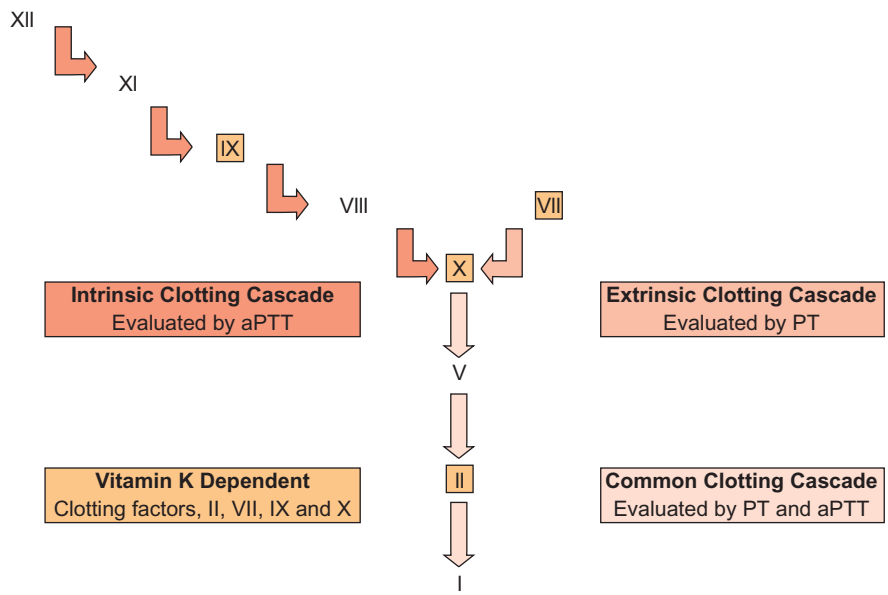


Fig 28.1 The clotting cascade

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vitamins (A, D, E and K) may be compromised. Vitamin K is necessary for the synthesis of clotting factors II, VII, IX and X. Of these, factor VII has the shortest half-life, therefore the extrinsic clotting cascade, as measured by the PT, is affected before the intrinsic clotting cascade, as measured by the APTT (Fig 28.1).

Cobalamin absorption requires intrinsic factor. In dogs, intrinsic factor is produced partially by the stomach, whereas in cats it is produced exclusively by the pancreas. In addition to deficiency of digestive enzymes, cats with EPI are also deficient in intrinsic factor, which in turn leads to an inability to absorb adequate cobalamin. Cobalamin deficiency may be found in association with canine EPI, and may be worth checking in poorly responsive patients, as deficiency of cobalamin can lead to villous atrophy and malabsorption.

Epidemiology

The incidence of EPI is much rarer in cats than dogs. The condition is common in German shepherd dogs and Rough collies, but may be under-recognized in cats. One study gave an incidence of only 11 cases out of 180,648 submissions (0.006%), although this was a post-mortem based study. The majority of cats with EPI are middle aged to older, which reflects the underlying pathogenesis of chronic pancreatitis. No breed or sex predisposition has been identified.

Prognosis

Although this is a lifelong condition, the prognosis for control is usually reasonable, provided the cat tolerates the enzyme replacement. Typical of many cats, this cat responded rapidly to treatment (Fig 28.2). Owners should be advised of the potential for the cat to develop diabetes mellitus as both conditions share the same aetiology, i.e. recurrent/chronic pancreatitis. Diabetes mellitus would appear to be the more common outcome with chronic pancreatitis and whilst it is possible for the cat to develop both conditions, this rarely appears to happen. The reason why some cats appear to have damage to the exocrine pancreas more than the endocrine pancreas is not known.



Fig 28.2 The cat after 6 weeks of treatment showing weight gain and improvement in coat condition

NURSING ASPECTS

Cats with EPI develop a greasy coat as a result of fat malabsorption and cobalamin deficiency. In addition, their coats may become soiled with faecal matter, particularly if longhaired. Bathing with an anti-seborrhoeic shampoo may be beneficial until clinical signs are controlled.

CLINICAL TIPS ON TREATMENT OF FELINE EPI

- Regular monitoring of cobalamin and folate levels with appropriate supplementation is recommended. Measurement of cobalamin by competitive chemiluminescent enzyme immunoassay appears to be more reliable than measurement by radioimmunoassay, therefore checking which method the laboratory uses may be worthwhile.
- If pig pancreas can be sourced, then approximately one-third of a pig pancreas is an appropriate portion per meal. Wrapping each pancreas in cling film prior to freezing means an individual pancreas can be removed from the freezer for defrosting each day.
- Feline TLI can be increased by renal disease or feeding, therefore this should be borne in mind if a low normal value is obtained in a cat with typical clinical signs. Re-evaluation of a fasted sample should be performed if there is strong clinical suspicion. Values <8 µg/L has a reported specificity of 85 to 100%.

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29 Parvovirus infection in a dog

INITIAL PRESENTATION

Acute haemorrhagic diarrhoea and vomiting with some streaks of blood in vomitus
Signalment: 5-month-old entire male Rottweiler puppy, body weight 20.0 kg

CASE HISTORY

The puppy presented out of hours as an emergency. Thirty-six hours prior to presentation he had become dull. He had started to retch and then to vomit bile stained fluid. This progressed to vomiting blood stained fluid and the presence of diarrhoea. The diarrhoea had become progressively bloody, with both dark digested appearing blood (melaena) and some bright red blood present. There was no mucus in the stools and the puppy did not strain to defecate. The puppy had not eaten since the onset of the vomiting and was also not drinking.

He had been de-wormed with febantel and praziquantel 1 month prior to presentation. He had been vaccinated against parvovirus, adenovirus, parainfluenza and distemper once after the owner obtained him from the breeder at 6 weeks of age. He had not received a booster vaccination. His usual diet was a commercial dry puppy food.

PHYSICAL EXAMINATION

On presentation the puppy was extremely depressed. He had a body condition score of 4/9. His mucous membranes were slightly dry and pale pink, with a capillary refill time of 3 seconds. A skin tent was present. He was drooling, which was thought likely to be due to nausea. His heart rate was 90 beats per minute with somewhat weak peripheral pulses. Respiratory rate was 40 breaths per minute and rectal temperature was 39.6° C. There were bloody faeces present on the thermometer.

PROBLEM LIST DISCUSSION OF PROBLEMS

The puppy's main problems were acute haemorrhagic diarrhoea and vomiting. While there was some fresh blood present in the faeces, the diarrhoea was thought to be primarily small intestinal in origin. The vomiting and diarrhoea were combined for the differential lists in this puppy as the concurrent onset suggested a single disorder.

DIFFERENTIAL DIAGNOSIS

Potential causes of acute vomiting and haemorrhagic diarrhoea in this puppy included:

- Parvovirus infection
- Intussusception or intestinal volvulus
- Hookworms
- Caustic agents
- Medications (e.g. corticosteroids, NSAIDs)
- Haemorrhagic gastroenteritis
- Severe acute pancreatitis
- Neoplasia and exocrine pancreatic insufficiency are unlikely due to his young age

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CLINICAL TIP ON HAEMORRHAGIC DIARRHOEA

Haemorrhagic diarrhoea has a characteristic odour – once smelled, not easily forgotten. This odour is associated with other causes of haemorrhagic diarrhoea and is not specific for parvovirus enteritis

CASE WORK-UP

Emergency diagnostic tests and treatment

The puppy was admitted for diagnostic tests and treatment and was housed in an isolation unit to prevent spread of a possibly contagious disease to other animals.

NURSING TIP FOR INFECTIOUS DISEASES

With cases of possibly infectious disease, patients should be kept in an area separate from other patients, especially those patients that may be immunocompromised (e.g. on corticosteroids or chemotherapeutic agents, elderly or young patients). Gloves and protective clothing should be worn to handle the patient and the same materials should not be worn when handling other patients. Scrupulous washing of hands and a dip bath or covers for shoes should also be used. When possible, different staff members should handle a potentially infected case than those handling other patients, especially immunocompromised patients. A thermometer should be kept for use in the potentially infected patient only and a cover used on the thermometer. Stethoscopes and any other equipment used should be cleaned after use or kept only for that patient.

Parvovirus may survive outside the host and is resistant to many disinfectants. For parvovirus, kennel cleaning should be done with bleach at 1:32 dilution (half cup bleach per gallon water). Also note that hand sanitizers are probably not viricidal since they are alcohol-based.

The puppy was estimated to be approximately 8% dehydrated. A balanced intravenous crystalloid fluid therapy (lactated Ringers) was initiated at 116 ml/hour.

CLINICAL TIP: CALCULATION OF INTRAVENOUS FLUID AMOUNTS

Fluid therapy is calculated based on the amounts needed for maintenance, rehydration and to replace ongoing losses. Maintenance requirements are 40 to 60 ml/kg/24 hour. In this puppy, they were calculated as: 20 kg × 60 ml/kg = 1200 ml/24 hour or 50 ml/hour. The upper end of the maintenance requirement was used in this dog as he was a puppy and they have a larger percentage of body weight as fluid compared to increased body fat in adult animals. Usually the lower end is used for large breed dogs.

To correct his hydration over 24 hours, his body weight times his percent dehydration was used: 20 kg × 0.08% = 1.6 l (1600 ml)/24 hours or 66 ml/hour. This was added to the maintenance amount so that the initial fluid rate was 116 ml/hour. The dehydration could also have been corrected in a shorter time period, e.g. 10 to 12 hours with a faster fluid rate (210 ml/hour for 10 hours or 183 ml/hour for 12 hours).

Estimates of the amount of fluid lost in vomiting or diarrhoea may be added to the maintenance plus rehydration rates. The hydration status should be re-evaluated frequently (hourly while on the fast rates) to determine if the rate should be changed and to assess for overhydration.

CLINICAL TIP: CHOICE OF FLUIDS

For rehydration a crystalloid fluid containing some electrolytes is usually appropriate. With dehydration due to diarrhoea and most cases of vomiting (other than those caused by upper gastrointestinal tract obstruction), the patient is in a state of metabolic acidosis due to decreased tissue perfusion. Buffered crystalloid intravenous solutions such as lactated Ringers are more alkalinizing than 0.9% sodium chloride and may help correct the acidosis, although reperfusion of the tissues is the most crucial part of the treatment.

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Emergency minimum data base

Haematology showed a marked neutropenia of 0.06 × 10^9/l (reference range 3.0–12.0), a lymphopenia of 0.4 × 10^9/l (reference range 0.5–4.99). His packed cell volume (PCV) was 0.322 l/l, at the bottom end of the reference range used (0.32–0.55 l/l); however, he was dehydrated so the true value of his PCV would likely have been lower upon rehydration. Haemorrhagic diarrhoea can result in a rapid decrease in PCV.

An in-house electrolyte analysis showed a decreased serum potassium concentration of 2.9 mmol/l, reference ranges 3.6–5.6 mmol/l, with the serum sodium and chloride within the reference ranges. A decreased serum potassium concentration is common in gastrointestinal disorders and can result in further gastrointestinal ileus. Treatment was potassium chloride added at 15 mEquiv/l of fluid.

CLINICAL TIP ON POTASSIUM SUPPLEMENTATION

For maintenance fluids, the rates of potassium supplementation based on serum concentrations are:

Serum concentration	mEquiv KCl to add per litre
<2.0 mEquiv/l	80
2.1–2.5 mEquiv/l	60
2.6–3.0 mEquiv/l	40
3.1–3.5 mEquiv/l	20

As this puppy's fluid rate was over twice maintenance, his added amount of potassium was decreased proportionally. There are 4 mEquiv/l of KCl in lactated Ringers, so the addition of 15 mEquiv brought the amount in his fluids to 19 mEquiv/l (run at over twice maintenance), which is approximately equivalent to adding 40 mEquiv/l as required by his serum potassium of 2.9 mEquiv/l.

Additional medical treatment

In addition to rehydration and correction of hypokalaemia, his treatment included intravenous ampicillin (20 µg/kg q 8 hours), metronidazole (10 mg/kg slow iv q 12 hours), ranitidine (2 mg/kg sq q 12 hours), buprenorphine (0.02 mg/kg sq q 8 hours) and constant rate metoclopramide (1 mg/kg over 24 hours in the iv fluids). Antibiotics are indicated in cases with diarrhoea when there is blood present in the diarrhoea, as there is an increased risk of bacterial translocation across the intestinal wall and sepsis.

CLINICAL TIP ON ANTIBIOTIC USE IN PARVOVIRUS

While ampicillin was used in this case and is recommended by some clinicians, others believe that trimethoprim sulfa drugs are a better broad spectrum choice as ampicillin may have a negative effect on the normal gut flora and cause an overgrowth of bacteria such as *Salmonella*.

NURSING TIP

Blood glucose should have been checked on this puppy! Puppies are at risk for hypoglycaemia, especially with anorexia and gastrointestinal disease.

CLINICAL TIP ON ANTI-EMETIC DRUGS

Maropitant may have been a good additional choice of anti-emetic for this puppy, but was not yet available when the puppy was presented. When metoclopramide is used for severe vomiting, a constant rate infusion is better than SQ administration as the half-life is not long enough for consistent efficacy. Metoclopramide and ranitidine both have promotility effects, which were important in this case as ileus was present.

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NURSING TIP

Intravenous catheters should be well bandaged to keep them clean, especially in cases with vomiting and diarrhoea. They should be checked and flushed several times a day for patency. Catheter infections are a common cause of pyrexia so good hygiene is imperative. A rise in rectal temperature in patients with intravenous catheters is often associated with inflammation at the catheter site.

A faecal SNAP™ Parvo test was performed and was positive. As the signalment and clinical signs were also consistent in this puppy, a diagnosis of parvovirus gastroenteritis was made.

CLINICAL TIP ON PARVOVIRUS TESTING

The ELISA faecal SNAP™ Parvo test is the most common in house test for parvovirus. The test does have some limitations. Recent vaccination with a live vaccine may interfere with the test results causing false positive readings. This interference usually occurs 5 to 12 days after vaccination so if a positive faecal ELISA SNAP™ test is obtained within this period after vaccination, additional tests may be recommended. The test is highly sensitive (100%) and specific (99.9%).

The puppy was a bit brighter the following day, but still had diarrhoea containing blood and was vomiting.

A full haematology and serum chemistry was performed at a veterinary laboratory. Haematology showed a decreased PCV of 0.30 l/l (reference range 0.35–0.55 l/l). White blood cell counts had improved, but were still decreased with a neutrophil count of 2.1 $\times$ 10^9/l (reference range 3–12 $\times$ 10^9/l) and a lymphocyte count of 0.47 $\times$ 10^9/l (reference range 0.7–4.8 $\times$ 10^9/l).

The puppy's ALP was increased to 422 IU/l. The reference range for ALP for this laboratory is 20 to 60 IU/l for adult dogs. In 6-month-old puppies the value can be expected to be increased, but usually in the range of two to three times the adult range (i.e. probably not much greater than 180 IU/l). Gastrointestinal disease can also increase ALP, so there was likely an increase due to illness in this puppy. Serum phosphorus was also increased to 2.4 mmol/l, with the adult canine reference range for this laboratory being 0.4 to 2.0 mmol/l. He was, however, within the reference range for a growing puppy at 6 months of age, which is about 1.65 to 2.84 mmol/l. Serum glucose was within the reference range at 5.0 mmol/l (reference range 3.0–5.0 mmol/l).

CLINICAL TIP ON HAEMATOLOGY AND SERUM CHEMISTRY IN PUPPIES AND KITTENS

The reference ranges for several clinicopathological parameters are different in young growing animals compared to adults and most laboratory reference ranges are those for adults. See Appendix 5 for a table of some juvenile blood values.

Serum potassium increased to 4.4 mmol/l (reference range 3.6–5.6 mmol/l) after supplementation and all other electrolytes were also within the reference ranges.

Serum albumin concentration was decreased at 17.6 g/l (reference range 26–35 g/l) as was the globulin concentration of 17.5 g/l (18–37 g/l). Serum albumin is decreased in young puppies, but by the age of 4 months should be within adult reference ranges. The decrease in this puppy is likely due to intestinal loses.

Outcome and further treatment

The puppy gradually improved over the next several days. He was first offered ice cubes to lick and then small amounts of water. By the fourth day he was eating small amounts of cooked chicken. An oral balanced electrolyte solution containing glutamate was also given to him in small amounts by syringe.

Over the next few days his lymphocyte and neutrophil counts improved and serum albumin and globulins increased.

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NURSING TIP ON NUTRITION OF VOMITING PATIENT

If a patient that has been vomiting is offered water or food too soon, they may start vomiting again. After 12 to 24 hours of no vomiting, allowing them to lick ice cubes is a good way to gently start them back on oral intake, as they can only swallow very small amounts at a time. Use of oral fluids that contain glutamate may provide some nutrition for the cells lining the intestinal tract, which have usually been depleted during severe diarrhoea. The first foods offered need to be highly digestible and low in fat, as the intestinal tract needs some time to adjust and re-develop digestive enzymes. Some puppies that have had parvovirus may require a highly digestible diet for months or even for the rest of their lives.

Partial parenteral nutrition has been used successfully in puppies with parvovirus and provides good short-term nutritional support. This may be administered through a peripheral vein and can be utilized in a practice setting.

It has been shown that early enteral nutrition in dogs with parvovirus results in an earlier clinical improvement and possibly an improved gut barrier function. Some people have recommended enteral feeding by naso-oesophageal tube, but this should be well monitored to make sure the dog does not vomit the tube and cause feeding in to the trachea.

Recombinant feline interferon (type omega) has also been recommended for use in dogs with parvovirus and one study showed an improvement in clinical signs and decreased mortality rates when this was administered at 2.5 million units/kg intravenously once a day for 3 days.

The puppy was sent home after 5 days in hospital. He continued to have diarrhoea for about another week, although the vomiting stopped and he was eating well.

DISCUSSION AND EPIDEMIOLOGY

Canine parvovirus is widespread in the dog population and most dogs are exposed to the virus either by vaccination or from contact with other dogs or dog faeces. The virus can survive for about 5 to 7 months in faeces and contact with infected faeces is the most common route of infection.

The incubation time is 3 to 8 days and shedding of the virus may occur prior to the onset of clinical signs. Following ingestion of the virus, it replicates in the lymphoid tissue of the oropharynx and then spreads to the bloodstream. The virus is deposited in the rapidly dividing cells of the body, including the bone marrow and the epithelial cells of the intestinal tract. The replication of virus in the lymphopoietic system and bone marrow results in the lymphopenia and neutrophilia usually seen. The presence of the replicating virus in the intestinal cells (Fig 29.1) causes depletion of the intestinal villi and epithelial necrosis, resulting in the typical severe haemorrhagic diarrhoea. Intestinal bacteria such as *Clostridium perfringens* and *Escherichia coli* may translocate across the compromised intestine into the body and can cause sepsis and death.

Rottweilers, Labrador retrievers and Doberman pinschers appear to be predisposed to the infection. Most cases occur in dogs less than 1 year of age and most often in dogs from 6 to 18 weeks of age.

While this dog had been vaccinated, vaccine 'breaks' (disease in the face of vaccination) can occur. This is sometimes due to the effect of antibodies from the dam's milk (maternal antibodies) interfering with the response to vaccination. Immunity is also not likely to be sufficient in a dog where only one vaccination has been given.

A first parvovirus vaccination should be given between 6 and 9 weeks of age and a second parvovirus vaccination should be given 3 to 4 weeks later. There is some debate about whether the second vaccine should be given at 10 or 12 weeks of age. If it is delayed until 12 weeks, this may mean keeping puppies away from other dogs until then and that time period is thought to be an important age for puppies to interact with other dogs for socialization purposes. However, maternal antibodies are more likely to interfere with vaccines in puppies less than 12 weeks of age. In breeds or individuals at risk, another vaccination at 14 to 16 weeks of age is appropriate. A booster should be given 1 year later and then subsequent booster vaccinations every 3 years.

A diet supplemented with antioxidants, e.g. vitamins E and C, β-carotene and selenium was shown in one study to result in an increased response to parvovirus (and distemper) vaccination in puppies.

New strains of parvovirus (CPV-2a, 2b, 2c) have emerged since the original (CPV-2) strain was identified in 1978. Novel CPV variants have also emerged in cats and dogs. Vaccines against the original strain can confer protection against the new strains, but disease outbreaks have been reported. Generally dogs with

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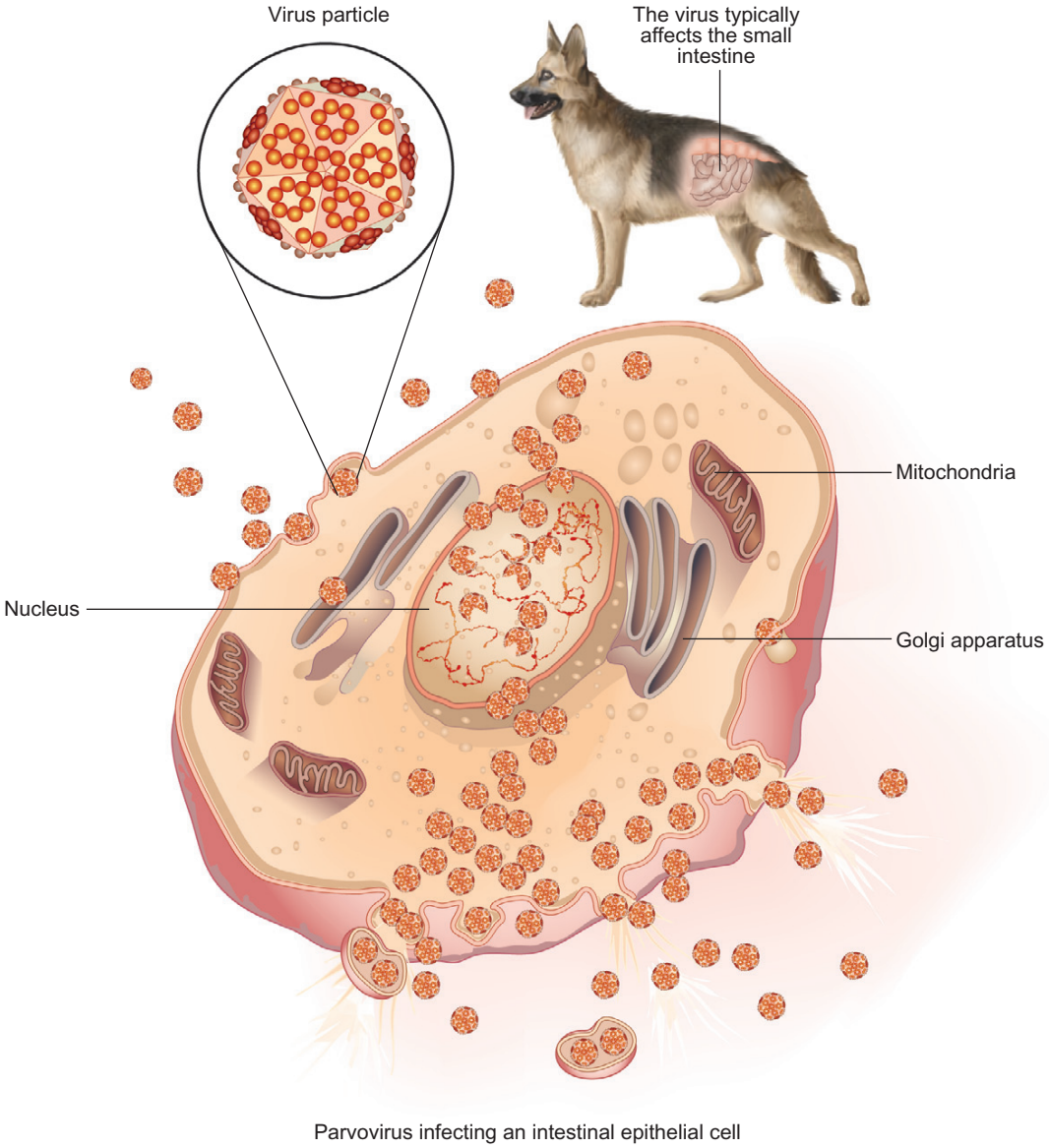


Fig 29.1 Illustration of parvovirus infecting an epithelial cell.

partial immunity from vaccination have a less severe form of the disease.

Prognosis

Most dogs that survive the initial 2 to 3 days of treatment will recover. The mortality rate without treatment has been estimated to be as high as 91%, but with aggressive supportive therapy decreases to 5 to 20%. Deaths are now less common than when the disease first arose in the 1970s. The improvement in mortality may be due to improved immunity, decreased virulence and/or improved treatment.

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SECTION 4

HAEMATOCHEZIA AND MELAENA

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30 Introduction to haematochezia and melaena

Blood may be present in diarrhoea of either large or small intestinal origin; however, sometimes the passing of blood is the predominant presenting clinical feature. Melaena refers to a dark tarry appearance to the stool which is caused by the presence of digested blood. The dark colour is due to the oxidation of haemoglobin and the tarry appearance is due to bacterial breakdown of haemoglobin. In dogs 300 to 500 mg/kg of haemoglobin must be present in the upper gastrointestinal (GI) tract before melaena is visually present, so its absence does not rule out GI bleeding.

In humans, blood must be present in the GI tract for at least 8 hours to turn a black colour, so a slow colonic transit time can cause melaenic stools from bleeding from a colonic area. Generally though, melaena is due to bleeding from the proximal GI tract or swallowed blood from a nasal, oral, oesophageal, pharyngeal or respiratory tract lesion. The use of salicylates, bismuth or charcoal can colour the faeces black as well.

Haematochezia is the presence of bright red blood in the faeces. If fresh blood is present on the outside of the faeces it is more likely to be from the distal colon or rectum.

Confirming the presence of melaena should be performed using a faecal occult blood test after the patient has been fed a meat-free diet for 3 days. In patients with undiagnosed anaemia, a faecal occult blood test should also be performed, as smaller amounts of GI bleeding can result in an apparently normal coloured stool.

Differential diagnoses for melaena include:

- Swallowed blood, e.g. from oro-nasal lesions
- Oesophageal disorders, e.g. ulcers, erosions, neoplasia
- Gastric causes, e.g. erosions or ulcers, severe gastritis, sharp foreign bodies, neoplasia, drugs (NSAIDs, corticosteroids, iron overdose)
- Small intestinal disorders, e.g. ulcers, severe inflammation, neoplasia, foreign bodies, hookworms
- GI ischaemia, e.g. due to shock, intussusception, volvulus
- Liver disease
- Pancreatitis (severe acute)
- Kidney disease causing uraemia

Differential diagnoses for haematochezia include:

- Colitis or proctitis
- Colonic or rectal tumour/polyp, stricture, infarct or foreign body
- Caecal inversion
- Colonic vascular ectasia
- Ileocaecal intussusception
- Parasites, e.g. hookworms
- Small intestinal bleeding with rapid transit time (i.e. insufficient time for oxidation of haemoglobin)

DIAGNOSING THE CAUSE OF GI BLEEDING

The patient should be examined for evidence of non-GI tract bleeding such as a cough resulting in haemoptysis or epistaxis. The mouth and pharynx should be examined for oral causes. Regurgitation may occur with oesophageal disease and haematemesis with gastric bleeding.

Haematology may reveal anaemia. When chronic, an anaemia due to GI bleeding may become microcytic, hypochromic and non-regenerative. Even occult bleeding can cause a rapid drop in the packed cell volume and severe bleeding can cause a dramatic decrease within hours. The blood urea nitrogen may increase due to digestion of blood. A platelet count, buccal mucosal bleeding time and coagulation panel should be

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performed to check for primary or secondary bleeding disorders. Unlike most other cases of GI bleeding, cases of idiopathic haemorrhagic gastroenteritis often have an increased packed cell volume. This is likely because the fluid losses are more pronounced than the loss of red blood cells, although the diarrhoea often appears severely haemorrhagic and there is often not obvious clinical dehydration.

Procedures to localize the bleeding may include ultrasound, endoscopy and exploratory surgery.

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31 Haemorrhagic gastroenteritis in a dog

INITIAL PRESENTATION

Acute onset of haemorrhagic diarrhoea and vomiting
Signalment: 2-year-old neutered female German shorthaired pointer, body weight 29.1 kg

CASE HISTORY

The dog had an acute onset of haemorrhagic diarrhoea and vomiting starting the evening before presentation. On the evening of presentation she was lethargic and was not willing to eat. The diarrhoea was liquid and contained large amounts of frank blood. There was a little tenesmus, but she was producing a large volume of diarrhoeic fluid. The vomitus initially contained digested food and then only bile tinged fluid.

The dog was last vaccinated 4 months earlier and had been dewormed with fenbendazole 2 months prior to presentation. Her usual diet was a mixture of commercial dry complete dog food and commercial canned food and she received treats. She also scavenged and the owners said that it was possible but unlikely that she could have eaten something from the rubbish.

PHYSICAL EXAMINATION

On physical examination she appeared very quiet, but was responsive. She was in good body condition (body condition score 5/9). Mucous membranes were moist, but hyperaemic, with a relatively slow capillary refill time (CRT) at 3 seconds. Thoracic auscultation revealed no abnormalities other than an increased heart rate of 200 beats per minute. Her respiratory rate was 16 breaths per minute. Peripheral pulse quality was poor, with weak thready pulses. Her peripheral lymph nodes appeared normal.

She appeared uncomfortable on palpation of her small intestines and the colon felt enlarged and doughy. Rectal temperature was normal at 38.2° C, but there were dark bloody faeces present on the thermometer.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The dog's problems included:

- Haemorrhagic diarrhoea
- Vomiting
- Decreased appetite
- Lethargy
- Tachycardia

The major problems for this dog were the profound haemorrhagic diarrhoea and the vomiting. Lethargy, decreased appetite and probably tachycardia were likely to be secondary to the underlying disorder. As the onset was acute, more chronic disorders were not on the original differential diagnoses lists.

DIFFERENTIAL DIAGNOSIS

For the acute haemorrhagic diarrhoea

- Idiopathic haemorrhagic gastroenteritis (HGE)
- Infectious causes: Parvovirus, *Salmonella, Clostridium perfringens*
- Amanita mushroom toxicity
- Warfarin toxicity
- Hypoadrenocorticism
- Intestinal volvulus
- Intussusception
- Shock

For acute onset vomiting

- Disorders of the stomach
  - foreign body

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- gastritis
- ulceration
- Disorders of the small intestine
  - foreign body
  - intussusception
  - intestinal volvulus
  - idiopathic HGE
  - infectious diseases as for the diarrhoea (above)
- Disorders of the large intestine
  - colitis
- Systemic disorders
  - pancreatopathy
  - hypoadrenocorticism
  - peritonitis
- Dietary causes
  - dietary sensitivity
  - dietary indiscretion

CASE WORK-UP

The high heart rate and weak thready pulses were consistent with the onset of shock. Treatment was initiated with shock doses of intravenous crystalloid fluids (60 ml/kg/hour) for the first hour. This was decreased to 30 ml/kg/hour after the first 30 minutes as her CRT and pulses improved.

Emergency minimum data base

As the dog presented as an emergency after regular opening hours, full laboratory evaluation was not possible. A packed cell volume (PCV) run in house was 0.60 l/l (reference range 0.37–0.55 l/l but usually only greyhounds and other sighthounds are over 0.50 l/l). The serum albumin concentration was 26 g/l (reference range 22–39 g/l), globulin concentration was 27 g/l (25–45 g/l), serum urea was 13.1 mmol/l (reference range 2.5–9.6 mmol/l) and creatinine was 147 µmol/l (reference range 40–132 µmol/l). Other parameters measured included serum alanine aminotransferase, calcium, glucose, phosphorus and total bilirubin. The electrolytes sodium, potassium and chloride were all at the higher end of the reference range. She did not urinate that evening so a urinalysis was not performed. Faeces were collected for testing the following day.

CLINICAL TIP ON DEHYDRATION EFFECTS ON HAEMATOLOGY AND SERUM CHEMISTRY PARAMETERS

Dehydration resulting in haemoconcentration can affect many parameters on the haematology and serum chemistry results, including increasing the PCV, white cell count, albumin, globulins, urea, creatinine and sodium. These should be re-assessed after the patient is rehydrated. Abnormal values may have been masked, e.g. a low albumin, and pre-renal azotaemia (an increase in serum urea and creatinine due to decreased renal perfusion) may be improved.

The dog was maintained on intravenous fluids and administered the gut protectants sucralfate at 5 ml po q 8 hours and ranitidine at 2 mg/kg sq q 8 hours. For analgesia she was given buprenorphine at 20 µg/kg slowly iv q 8 hours and maropitant (1 mg/kg sq q 24 hours) was used as an anti-emetic. She was also given metronidazole at 10 mg/kg slowly iv q 12 hours.

CLINICAL TIP ON USE OF ANTIBIOTICS IN HAEMORRHAGIC DIARRHOEA

Many cases of diarrhoea are not responsive to antibiotics and their use is often not indicated. In cases where there is melaena or haematochezia, the use of antibiotics is indicated, because bacterial translocation from the intestine into the system is possible. Further, as *Clostridium perfringens* may be part of the aetiology of HGE, antibiotics may help in treatment.

The following day the dog was brighter and had gained 2.8 kg from fluid replacement (weight 31.9 kg). She still had diarrhoea containing blood, but had not vomited since being administered maropitant.

Minimum data base

Full haematology and serum chemistry were performed. The PCV following rehydration had decreased to 0.42 l/l (reference range 0.37–0.55 l/l). Serum creatinine and urea were within the reference ranges at 75 µmol/l and 4.1 µmol/l, respectively. Serum albumin and globulins had dropped below the reference ranges to 19.3g/l (ref-

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erence range 26–35 g/l) and 13.7 g/l (reference range 18–37 g/l). Calcium had also decreased to 2.02 mmol/l (reference range 2.3–3.0 mmol/l), likely due to the decrease in albumin and concurrent decrease in albumin bound calcium.

Urine was negative for all parameters other than bilirubin (+1) on a chemical strip, with a pH of 6.5. The urine specific gravity was 1.015 and the sediment was not active. The low urine specific gravity was likely to be due to the aggressive rehydration causing a diuresis.

Faecal analysis

Faecal analysis for parasites including *Giardia* was negative. Faecal culture was positive for *Clostridium perfringens*, but the faeces were negative for the *Clostridium perfringens* toxin test. A faecal IDEXX SNAP® test for parvovirus was negative.

CLINICAL TIP ON PARVOVIRUS TESTING

The ELISA faecal SNAP® Parvo test is the most common in-house test for parvovirus. The test does have some limitations. Recent vaccination with a live vaccine may interfere with the test results causing false positive readings. This interference usually occurs 5 to12 days after vaccination so if a positive faecal ELISA SNAP test is obtained within this period after vaccination, additional tests may be recommended. The dog could also be infected but no longer shedding virus in its stool or the virus particles may be so thoroughly coated with antibodies that they cannot react with the chemicals of the test. The test is highly sensitive (100% sensitivity) and specific (99.9% specificity).

Adrenocorticotrophin hormone stimulation test

An adrenocorticotrophin hormone (ACTH) stimulation test was performed, which ruled out hypoadrenocorticism with values within the reference ranges, i.e. a basal value of 70.1 nmol/l and a post-stimulation value of 262 nmol/l (reference range for basal 20–230 nmol/l; a basal value of 70 nmol/l or more effectively rules out hypoadrenocorticism).

Outcome

The dog's clinical signs improved progressively over the next 2 days, with a decrease in the amount of faecal blood and an improved consistency (to soft but formed) of the faeces. The dog did not have any further vomiting. She was offered small amounts of a low fat, highly digestible canned food in small amounts and was willing to eat.

NURSING TIP ON NUTRITION

Dogs which have had gastrointestinal disorders and have not been eating need to be re-introduced to food very gradually. The brush border enzymes of the small intestines have often been decreased in severe intestinal disease and the ability to digest a large amount of carbohydrates is often decreased. Fats can also be difficult for these patients to digest. Offering small amounts of a highly digestible food is indicated. The initial food offered to a patient which has not been eating should be at one-third of resting energy requirement (RER). In this dog, that would be:

$$\text{RER} = \text{body weight in kg}^{0.75} \times 70 = 31.9^{0.75} \times 70$$
$$= 13.4 \times 70 = 939 \text{ kcal}$$

One-third of that is 313 kcal/day; this could be divided into four to six meals or roughly 50 to 80 kcal per meal.

DISCUSSION

HGE usually presents as a peracute or acute condition, as in this case, with the patient suddenly developing profoundly haemorrhagic diarrhoea and possibly haematemesis. The faeces are often said to look like dark raspberry jam. Very large amounts of fresh blood are passed in the diarrhoea and sometimes in the vomitus, so the animal rapidly becomes hypovolaemic, shocked and collapsed. Even though the blood loss is dramatic, there is usually an increase in the PCV (e.g. above 50% or 0.50 l/l) along with normal or low total serum proteins (albumin and globulins). The discord between the increased PCV and low or normal serum proteins is suspected to be due to plasma protein loss. The intercellular junctions are leaky allowing loss of protein, electrolytes and fluid, but the spaces are not big enough to allow red blood cells to leak through; thus the loss of proteins, electrolytes and fluid exceeds cellular loss.

The aetiology is not known although viral infection and endotoxin have been implicated. Most patients are

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young adults and although most recover, there is a risk of relapse in about 15% of cases. Most often small breeds of dogs are affected.

While the clinical signs are typical, the differential diagnoses should include sharp or penetrating foreign bodies, intussusception, ingested poisons, parvovirus and other infections, internal parasites, coagulopathies, hypoadrenocorticism, disseminated intravascular coagulation or neoplasia.

Treatment

The mainstay of treatment is aggressive supportive care with intravenous crystalloid fluids (40–90 ml/kg). Colloids may need to be added if plasma proteins are very low and oncotic pressure needs to be supported.

Broad spectrum antibiotic cover should be provided, as these patients are at risk for intestinal bacterial translocation and *Clostridium perfringens* may be part of the aetiology. Gastroprotectant drugs are indicated, as are parenteral anti-emetic drugs.

Avoid the use of anticholinergic agents as they suppress gut motility and could worsen ileus. Opioids are also contraindicated as they may prolong gastrointestinal transit time which may encourage bacterial overgrowth.

Prognosis

The prognosis is usually good if the case is seen early and treated appropriately. One study of 15 dogs showed a good recovery in all 15 cases with fluid therapy, antibiotics and dietary management.

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32 Colonic vascular ectasia in a dog

INITIAL PRESENTATION

Collapse and haematochezia

Signalment: 5-year-old neutered male Bichon Frise, body weight 8.5 kg

CASE HISTORY

The dog had presented to the referring veterinary surgeon 1 month previously with a history of having collapsed. Clinical examination at that time had been unremarkable. The only abnormal finding was highly regenerative anaemia, with a packed cell volume (PCV) of 0.22 l/l (reference range 0.37–0.55 l/l) and a reticulocyte count of 335 × 10^9/l (reference range >60 × 10^9/l, indicates a regenerative anaemia). Empiric treatment had been prednisolone and ferrous sulphate (doses not known).

Since then the dog had been increasingly lethargic and his appetite had been poor. His faeces were of normal consistency, but frequently were dark in colour and also contained bright red blood (haematochezia) and some mucus. The owners reported that he had lost about 0.5 kg during this time.

He was de-wormed every 6 months and his vaccinations were current. His regular diet was a commercial dry dog food.

PHYSICAL EXAMINATION

The dog was responsive but lethargic and quiet. His body condition score was 4/9 with evidence of recent weight loss. His mucous membranes were very pale and slightly dry. His capillary refill time was <3 seconds. Abdominal palpation and thoracic auscultation were unremarkable other than a II/VI left sided systolic murmur. His heart rate was 142 beats per minute (bpm) and pulse quality was bounding. His respiratory rate was 32 breaths per minute. His rectal temperature was 38.7° C and there was evidence of blood on the thermometer.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The primary problems in this dog were the pale mucous membranes and the evidence of melaena and haematochezia. The murmur was possibly due to the anaemia. As he was known to have a regenerative anaemia, this was the likely reason for his pale mucous membranes. Major categories for regenerative anaemia are blood loss and haemolysis; as he had evidence of gastrointestinal blood loss this was presumed to be the likely cause of the anaemia.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for the melaena and haematochezia causing anaemia in this dog include:

- Swallowed blood from oral, nasal, pharyngeal or respiratory system disorders (melaena)
- Oesophageal disorders, e.g. ulcer or neoplasia
- Coagulopathies, e.g. vitamin K deficiency, thrombocytopenia, von Willebrand's disease
- Drugs, e.g. NSAIDs, corticosteroids (no history of drug administration prior to first episode)
- Toxins, e.g. caustic agents, heavy metals, Amanita (magic) mushrooms
- Internal parasite, e.g. hook worms

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- Neoplasia, e.g. adenocarcinoma, mast cell tumour, gastrointestinal lymphoma, multiple myeloma, gastrinoma
- Inflammatory bowel disease with ulceration
- Pancreatitis
- Hepatopathy
- Sharp or penetrating foreign body
- Uraemia (no evidence in previous serum chemistry)
- Pancreatitis
- Disseminated intravascular coagulopathy (unlikely)
- Vascular disorders, e.g. colonic vascular ectasia

CASE WORK-UP

Minimum data base

An in-house PCV was performed, which was 0.11 (reference range 0.37–0.55 l/l). The dog was blood typed as dog erythrocyte antigen (DEA) 1.1 negative and transfused with 250 ml of fresh whole blood from a DEA 1.1 negative donor. Routine haematology from a pre-transfusion sample confirmed the PCV. There was a hypochromia with an mean haemoglobin concentration of 29.5% (reference range 23–36%), although the blood was normocytic with a mean corpuscular volume of 68 fl (reference range 60–77 fl). The anaemia was still regenerative with a reticulocyte count of 17% (reference range >1%). His platelet count was within the reference range, as were coagulation parameters (activated partial thromboplastin time and prothrombin time). Serum chemistry results were within the reference range other than a slightly decreased albumin of 23.6 g/l (reference range 26–35 g/l) which may have been due to an intestinal disorder causing malabsorption or from the blood loss.

After transfusion his PCV increased to 0.27 l/l and the murmur disappeared. His heart rate deceased to 60 bpm, although an ECG showed no abnormalities other than a wandering pacemaker (not considered to be pathological).

Faecal samples were negative for parasites and enteropathogenic bacteria, but highly positive for faecal occult blood.

Imaging

Radiography and abdominal ultrasonography showed no abnormalities other than food present in the stomach after 12 hours of fasting, indicative of delayed gastric emptying.

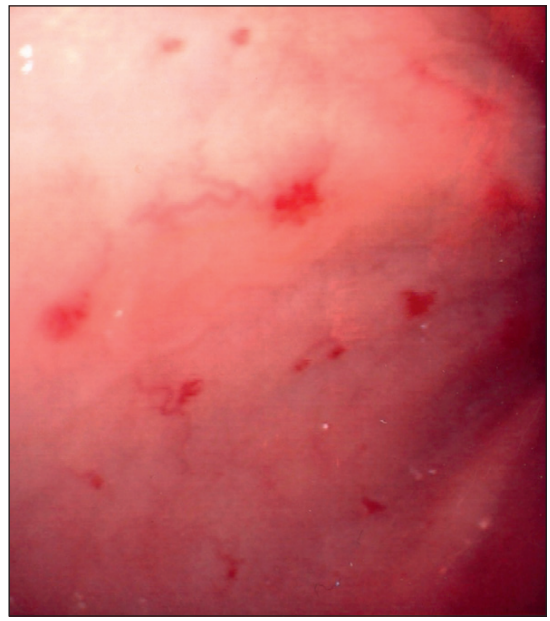


Fig 32.1 Endoscopic view of colon showing abnormal vessels

Endoscopy

On endoscopy, the gastric and duodenal mucosa appeared grossly normal. The colon had numerous areas of raised, tortuous, dilated blood vessels which were diffusely spread throughout the length of the colon (Fig 32.1). Histology of the mucosal sample from the duodenum was consistent with mild, chronic duodenitis. Histology of the sample from the colon was interpreted as moderate chronic active colitis.

Diagnosis

The severity of the dog's signs and the severity of the gross appearance of the colon, along with the lack of response to corticosteroids, were not consistent with inflammatory bowel disease. A diagnosis of colonic vascular ectasia was made from the characteristic gross morphologic abnormalities visualized during colonoscopy.

Treatment and outcome

Palliative therapy with tylosin in addition to the prednisolone was initially tried in this case, but with no success. As the lesions were diffusely distributed throughout the colon, laser coagulation or partial colonic resection were

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not feasible options. A subtotal colectomy was discussed with the owners. This surgery in dogs is not as successful as it is in cats with dilated idiopathic megacolon as reservoir faecal incontinence is a common complication in dogs. Further medical management with hormone therapy was discussed; however, the dog had another severe bleeding episode and unfortunately the owners chose to have him undergo euthanasia.

DISCUSSION

Colonic vascular ectasia, also known as angiodysplasia, is the presence of dilated arteries, veins and lymphatic vessels lined with a single layer of endothelial cells within the mucosa and submucosa of the colon. Haemorrhage is caused by disruption of the blood vessels, which often results in chronic haematochezia and an iron deficiency anaemia characterized by hypochromia and microcytosis. Melaena may also be present if the lesions extend into the small intestine or if the transit time in the colon is slow enough to allow digestion of the haemoglobin.

The aetiology is unknown, although theoretically colonic distension from chronic constipation could lead to submucosal venous occlusion and dilation. However, it has not been reported in cats with acquired megacolon due to obstipation and neither this case nor most of the reported cases had a history of constipation or tenesmus.

Diagnosis of the disorder is based upon assessment of the colonic mucosa at endoscopy and the characteristic appearance. The lesions are not usually visible from the serosal surface and would be missed at exploratory laparotomy.

Subtotal colectomy and partial colectomy (aided by intra-operative endoscopy to locate the lesions in one case) have been used successfully to treat the disorder.

Successful use of oral oestrogen-progesterone therapy has been used in humans with the condition and has been reported in two canine cases. In humans, this therapy does not lead to regression of the lesions, but it was associated with a reduction in the number of blood transfusions necessary to maintain stable to normal haemoglobin concentrations. The mechanism of action of the drug combination is not well understood, but theories include an induction of squamous metaplasia, restoration of the continuity of the endothelium of abnormal vessels, primary effects on blood coagulation and stasis of blood in the mesenteric microcirculation. In reported canine cases, while the lesions persisted as in humans, the haematochezia resolved and no further transfusions were necessary. The dose used was 2 μg/kg ethinyl oestradiol po q 24 hours and 23 μg/kg of norethindrone acetate po q 24 hours. Ferrous sulphate at 18 mg/kg po q 24 hours was also administered.

The potential risk of the oestrogen-progesterone therapy is bone marrow toxicity resulting in pancytopenia, as well as alopecia, cystic endometrial hyperplasia and pyometra, although the doses used in these two dogs appeared to be low enough to avoid these side effects.

Prognosis

There have been insufficient cases reported to predict the prognosis for these cases. Surgical management for focal lesions has been reported to be successful and in limited numbers, medical management has also been successful for cases in which surgery was not possible. It is likely that many of these cases die or are euthanized without a diagnosis.

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33 Intestinal leiomyoma in a dog

Case contributed by Carolina Urraca del Junco

INITIAL PRESENTATION

Intermittent melaena

Signalment: 12-year-old neutered male, English springer spaniel, body weight 19.8 kg

CASE HISTORY

The dog presented to the hospital for investigation of 2 to 3 weeks of intermittent melaena, lethargy and pale mucous membranes. His faeces were formed, but varied in colour from dark brown to tarry and black. There was no bright red blood or mucus present in the faeces and the dog did not have an increase in urgency of defecation. The dog's appetite had recently decreased, although he was still eating. He was thought to have lost weight, although the amount was not known. He had not been vomiting.

The dog's usual diet was a high quality commercial dry dog food. He was not fed scraps or extra treats and did not have a history of scavenging. He was current on his vaccinations and had been de-wormed 5 months prior to presentation.

PHYSICAL EXAMINATION

On presentation, the dog was quiet, but alert and responsive. His body condition score was 5/9. His oral mucous membranes were very pale, but capillary refill time was normal. His mucous membranes were moist and he appeared to be well hydrated. His heart rate was mildly elevated at 134 beats per minute and his respiratory rate was also slightly elevated at 36 breaths per minute. The rectal temperature was within normal limits at 38.2° C. Auscultation of the thorax and abdominal palpation were unremarkable and his peripheral lymph nodes felt normal.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The dog's main problems were the lethargy, pale mucous membranes and intermittent melaena. Pale mucous membranes can be caused by either poor perfusion or anaemia. The dog had no evidence of cardiovascular disorders and was well hydrated. The lethargy and likely anaemia (confirmed by packed cell volume (PCV)) were thought to be due to intestinal blood loss resulting in the melaena.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for melaena in this case included:

- Swallowed blood from oral, nasal, pharyngeal or respiratory system disorders (melaena)
- Oesophageal disorders, e.g. ulcer or neoplasia
- Internal parasites, e.g. hook worms
- Gastrointestinal neoplasia, e.g. adenocarcinoma, mast cell tumour, gastrointestinal lymphoma, multiple myeloma, gastrinoma, leiomyoma, leiomyosarcoma
- Inflammatory bowel disease with ulceration
- Sharp or penetrating gastrointestinal foreign body
- Vascular disorders, e.g. colonic vascular ectasia, although it would have to be in the proximal colon as there was no haematochezia

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Other less likely differential diagnoses include:

- Hepatopathy (no other consistent clinical signs)
- Uraemia (no polyuria present)
- Pancreatitis (unlikely with no vomiting or evidence of abdominal pain)
- Disseminated intravascular coagulopathy (unlikely)
- Coagulopathies, e.g. vitamin K deficiency, thrombocytopenia, von Willebrand's disease (less likely as no signs of bleeding elsewhere and no previous history of bleeding)
- Drugs, e.g. NSAIDs, corticosteroids (no history of drug administration)
- Toxins, e.g. caustic agents, heavy metals, Amanita (magic) mushrooms (no known history of ingestion of toxins)

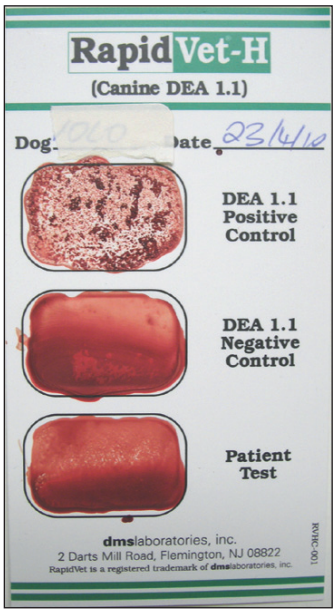


Fig 33.1 In-house blood typing showing a DEA 1.1 positive sample.

CASE WORK-UP

Minimum data base

Routine haematology revealed a severe anaemia with a PCV of 0.11 l/l (reference range 0.39–0.55 l/l, with moderate red blood cell regeneration (corrected reticulocyte count 2.76%). There was a low mean corpuscular volume (MCV) of 56 fl (reference range 60–77 fl) and low mean cell haemoglobin concentration (MCHC) of 30.2% (reference range 32–36%). The microcytosis and hypochromia were consistent with iron deficiency anaemia. Blood smear evaluation revealed marked microcytic hypochromic anaemia with moderate numbers of polychromatophils and small numbers of normoblasts. There was a moderate RBC anisocytosis and mild hypochromia. The platelet number appeared adequate. There was a moderate mature neutrophilia and a mild monocytosis, with a few reactive lymphocytes.

As the dog had a history of melaena, the regenerative anaemia was thought to be likely to be secondary to gastrointestinal blood loss, although haemolysis was possible. A slide agglutination test was negative and Coombs tests for IgG, IgM and complement were also not consistent with immune mediated haemolysis. The haematological parameters were consistent with an iron deficiency anaemia, also likely due to gastrointestinal blood loss.

The dog was dog erythrocyte antigen (DEA) 1.1 blood type positive on an in-house test, which also showed no evidence of auto-agglutination (Fig 33.1).

Serum biochemistry revealed mild hypoproteinaemia with a total protein of 46.4 g/l (reference range 58–73 g/l) due to decreased albumin of 20.4 g/l (reference range 26–35 g/l) and globulins at the low end of the reference range at 20 g/l (reference range 18–37 g/l). Decreased albumin can be caused by ongoing intestinal loss, urinary loss, third spacing or decreased hepatic production due to liver disorders or decreased production during an acute phase protein reaction (albumin is considered a 'negative' acute phase protein).

Urinalysis was unremarkable with a urine specific gravity of 1.032. Faecal analysis was negative for parasites and for enteropathogens.

The microcytic, hypochromic, mildly regenerative anaemia, reduced total protein and albumin, with globulins in the lower part of the reference range, suggested chronic blood loss.

Imaging

Right lateral and ventrodorsal thoracic and abdominal radiographs were obtained. In the thoracic radiographs, several well-defined 1- to 2-mm opaque nodules characteristic of pleural plaques were identified within the lung fields (Fig 33.2). The cardiac silhouette was within normal limits. There was no evidence of thoracic metastatic disease. Both shoulders showed evidence of degenerative joint disease. Abdominal radiographs

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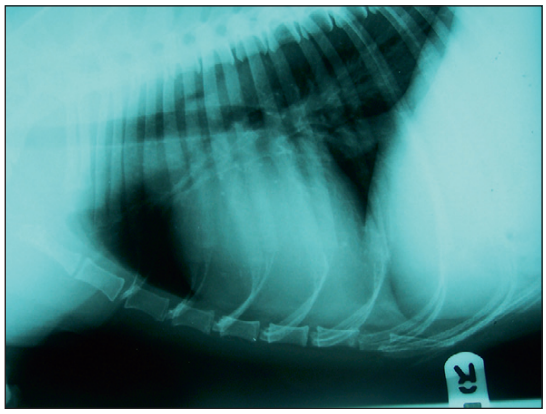


Fig 33.2 Lateral thoracic radiograph showing nodules characteristic of pleural plaques (courtesy of Carolina Urraca del Junco).

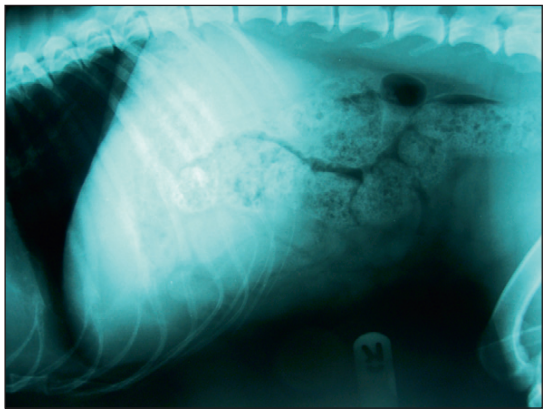


Fig 33.3 Lateral abdominal radiograph showing full colon (courtesy of Carolina Urraca del Junco).

revealed a faeces-filled normal size colon (Fig 33.3). The transverse colon was partially displaced dorsally by a poorly marginated soft tissue mass.

Ultrasonography of the abdomen showed a midline mass identified caudal to the stomach, in the ventral abdomen, with normal jejunal loops entering both ends. The mass was well vascularized and protruded into the intestinal lumen, with evidence of luminal dilatation and a partial loss of wall layering. At the centre of the mass, there was a rounded vascularized out-pouching of intestinal wall tissue, which appeared to connect to the intraluminal tissue. This out-pouching showed no evidence of intestinal wall layering (Fig 33.4). The location of the mass was consistent in location with the radiographically identified soft tissue mass. The duodenum,

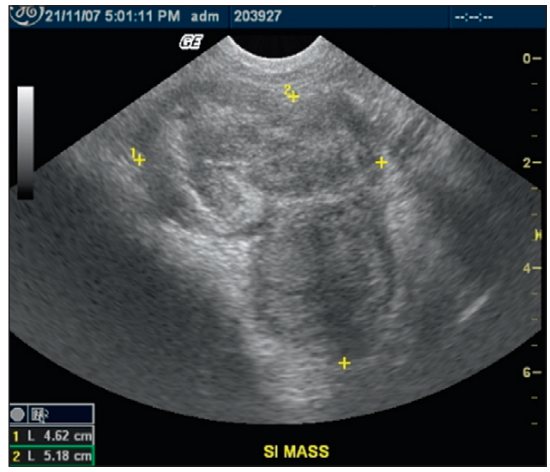


Fig 33.4 Ultrasound picture of jejunal mass with an outpouching of intestinal wall (courtesy of Carolina Urraca del Junco).

ileum and the large intestines were within normal limits and the remainder of the ultrasound examination was unremarkable.

Based on these findings a tentative diagnosis of jejunal neoplasia was made. Adenocarcinoma, leiomyoma, leiomyosarcoma, other sarcomas, metastatic disease involving the jejunum and lymphoma were considered to be the most likely differential diagnoses.

Surgical treatment

After imaging, the dog received a blood transfusion with one unit of DEA 1.1 positive blood and underwent enterectomy to remove the mass, which measured 4 cm in diameter and was localized within the jejunum. The appearance of regional lymph nodes and other abdominal organs was unremarkable. A jejunal end-to-end anastomosis was performed using surgical staples and the ventral midline incision was closed routinely.

Histopathology

The mass was submitted for histopathology. Macroscopically, the external appearance of the mass was contiguous and well-encapsulated, with a firm serosal surface.

The solid mass identified on ultrasound as expanding eccentrically from the jejunal lumen consisted of irregular swathes, whorls and streams of intensely eosinophilic bundles of uniform fibres, consistent with smooth muscle. These smooth muscle cells contained occasional ellipsoidal pale nuclei, with a finely stippled chromatin pattern. No mitotic figures were noted in the examined

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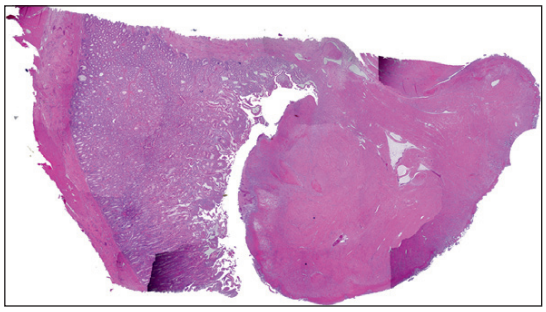


Fig 33.5 Histopathology of intestinal mass consistent with smooth muscle tissue (courtesy of Carolina Urraca del Junco).

sections and no inflammatory cells were seen within the mass.

The solid mass was found to infiltrate the submucosa and mucosa and extend in an annular fashion around the lumen. Normal mucosa was partially replaced with whorls and streams of eosinophilic tissue and there was an oedematous inflammatory reaction at the mucosal surface. Dystrophic calcification was identified on one section at a junction between normal and abnormal mucosa (Fig 33.5).

The appearance of well-differentiated smooth muscle cells and the absence of mitotic figures were consistent with a diagnosis of a benign leiomyoma, likely originating from the jejunal muscularis propria. As this was a solitary mass, the long-term prognosis was considered as very good.

Outcome

One week after surgery, the dog was discharged with instructions to restrict exercise until removal of skin staples. A 10-day course of clavulanate-potentiated amoxicillin (250 mg po q 12 hours) was prescribed.

In addition, iron supplementation (ferrous fumerate) was started at 200 mg po q 24 hours. At discharge the dog's PCV was 0.17 l/l. Iron supplementation was continued until erythrocyte parameters were back within the reference range.

Six months later the dog had good exercise tolerance, had gained weight and was no longer anaemic.

DISCUSSION AND EPIDEMIOLOGY

Melaena and microcytic, normochromic or hypochromic anaemia are common findings in cases with gastrointestinal ulceration due to tumours. Leiomyomas and leiomyosarcomas, in particular, may cause especially dramatic bleeding due to their propensity to ulcerate. Other signs of these intestinal tumours may include obstruction, vomiting, anorexia, diarrhoea, abdominal pain and weight loss. Hypoglycaemia associated with intra-abdominal leiomyoma has been reported. This was thought to be potentially due to an increase in insulin-like growth factor produced by the tumour. Leiomyomas are usually slow growing tumours and they may reach a large size before clinical signs are seen.

Intestinal tract neoplasia may be diffuse or nodular, with a variable incidence of local lymphadenopathy as a result of metastatic spread or reactive change. Therefore if the intestinal tract is abnormal, surgical biopsies should be taken from the tract itself and not just from the enlarged lymph nodes. Benign tumours are characterized by small cells, with small nuclei and little variation in cell and nuclear size. Leiomyoma is a benign tumour of smooth muscle origin and it is less common than leiomyosarcoma.

Several ultrasonographic features of gastrointestinal smooth muscle tumours are helpful in differentiating them from other types of gastrointestinal neoplasia. Leiomyosarcomas and leiomyomas are often large (over 3 cm) intramural lesions growing out of the serosa as large eccentric or extraluminal masses. Uncommonly, as in this case, they also invade or project into the gastrointestinal lumen. Anechoic and hypoechoic foci within the mass may correlate with the areas of central degeneration and necrosis frequently found in these lesions.

Gastrointestinal leiomyomas that show phenotypic features of typical smooth muscle cells occur most commonly in the oesophagus in humans. Published case series in humans show median ages of 30 to 35 years and these tumours are very rare in the stomach and intestines. In dogs, by contrast, benign leiomyomas occur most commonly in the stomach of geriatric dogs, while a higher incidence of malignant tumours with smooth muscle appearance are reported in the small intestine and caecum.

The signalment of this case, a 12-year-old male dog, is in keeping with published data – most leiomyomas occur in older male dogs. In one study, 82% of leiomyomas occur in male dogs, with a mean age of 11 years (range 8–17 years) at presentation. Only 31% of the cases had associated clinical signs. In another study, the mean age for dogs with small intestinal smooth muscle tumours (9.1 ± 2.3 years) was lower than for dogs with caecal tumours (10.7 ± 1.9 years). In contrast to dogs with intestinal tumours, which all showed clinical signs

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in this study, four dogs with caecal tumours showed no clinical signs at all and the mass was an incidental finding. In dogs with intestinal tumours, weight loss was observed more often, whereas intestinal perforation and peritonitis were observed more commonly in dogs with caecal tumours. In general the caecal tumours showed higher scores for necrosis and bleeding. No breed predilection has been reported.

In humans, many of these tumours were formerly diagnosed as smooth muscle tumours but have been reclassified as gastrointestinal stromal tumours. Apart from the surgical therapy in humans, an additional medical treatment under investigation is selective receptor tyrosine kinase inhibitor (imatinib mesylate) therapy.

Prognosis

An increased tumour diameter has been observed to be an important negative prognostic indicator and castrated or spayed dogs have been reported to have better survival statistics than intact dogs. In one study, in the 42 dogs with small intestinal tumours that were surgically-treated, the overall 1- and 2-year survival times were 63% and 52%, respectively. For the 19 dogs with caecal tumours that were surgically treated, the respective survival times were 84% and 66%. Of the surgically treated dogs, nine (of 42) with a small intestinal tumour and one (of 19) with a caecal tumour, died within 15 days because of surgery or tumour-related complications. For the remaining dogs with small intestinal tumours, the 1- and 2-year recurrence free period was 80% and 67% whereas, for the dogs with the caecal tumours, this was 83% and 62%, respectively.

No significant influence on prognosis after surgical removal has been identified based on tumour type or histological characteristics. Even dogs with tumours with very malignant histological appearance have a relatively good prognosis for long-term survival if complete excision of the tumour is accomplished.

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34 Corticosteroid induced gastrointestinal ulceration in a dog

INITIAL PRESENTATION

Weakness, lethargy, muscle wasting, soft dark faeces
Signalment: 7-year-old neutered female Labrador retriever, body weight 21.8 kg

CASE HISTORY

This dog had a history of immune mediated thrombocytopenia the previous year, which had responded to oral methylprednisolone. Two months prior to admission her platelet count had dropped to 136 × 10^9/l (reference range 200–500 × 10^9/l) and the referring veterinary surgery had again initiated oral methylprednisolone at 1.5 mg/kg body weight divided into two treatments per day. She had been maintained on this dose. Serum chemistry performed by the referring veterinary surgery had documented increased alanine aminotransferase (ALT) and alkaline phosphatase (AP), although the values were not recorded.

The owner was concerned that the dog was very lethargic and had very poor exercise tolerance. She reported that the dog had increased urination and thirst and a very good appetite. She was not vomiting, although had had several episodes of diarrhoea with soft, dark faeces.

The dog's usual diet was a low calorie dry food and she was normally fed twice a day. She had previously been on glucosamine and chrondroitin sulphate for arthritis, but these had not been administered during the previous 2 months. She was on no other medications or supplements. She was current on her vaccinations but had not been de-wormed for about a year.

PHYSICAL EXAMINATION

The dog was quiet but responsive. Her body condition score was approximately 5/9, although this was hard to determine as she had moderate muscle wasting with retention of body fat.

Her capillary refill time was less than 2 seconds. Her mucous membrane colour was pale pink and her membranes were tacky. She was estimated to be about 6% dehydrated.

Thoracic auscultation revealed normal heart and lung sounds, with a heart rate of 76 beats per minute and a respiratory rate of 14 breaths per minute. There was no evidence of pain on abdominal palpation, but the abdomen was pendulous and felt soft. Her rectal temperature was 38.4° C. Her coat was dry and showed evidence of seborrhoea.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The dog's problems included:

- Lethargy and poor exercise tolerance
- Pale, tacky mucous membranes
- Muscle wasting
- Pendulous abdomen
- Possible polyuria and polydipsia
- Elevated liver enzymes

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DIFFERENTIAL DIAGNOSIS

Many of this dog's problems may have been due to the administration of corticosteroids, but they were still explored as this assumption can result in missing a disorder.

Lethargy and exercise intolerance have many differential diagnoses, but for this dog the list included anaemia, hepatopathy, electrolyte disturbances, hypoglycaemia, corticosteroid induced myopathy, cardiopulmonary disorders and renal disease.

The slightly tacky membranes were likely indicative of mild dehydration due to the polyuria.

The pale mucous membranes may have been due to poor perfusion or anaemia. The heart sounds were regular and on palpation her pulses felt normal, making anaemia the more likely diagnosis. Anaemia may be divided into non-regenerative or regenerative, which is determined by performing a reticulocyte count. Non-regenerative anaemias are due to systemic diseases suppressing bone marrow function or primary bone marrow disorders. Regenerative anaemias are due to blood loss or haemolysis. Gastrointestinal blood loss is always a potential problem in animals on corticosteroid treatment.

The muscle wasting was typical of that induced by corticosteroids, but can also be caused by myopathies or weight loss due to illness, where muscle loss can be more prominent than fat loss.

The pendulous abdomen on this dog may have been due to the administration of corticosteroids, which cause muscle weakness and abdominal deposition of fat. Other causes for the abdominal enlargement included ascites or organomegaly.

The polyuria and polydipsia are common side effects of corticosteroids, but other common or concurrent disorders like renal disease, urinary tract infection, endocrine disorders or electrolyte disturbances were also included in the differential diagnoses list.

The elevation in liver enzymes may be due to corticosteroids or primary or secondary liver disorders.

Dark faeces can indicate melaena (the presence of digested blood in the faeces). Usually the stools are described as being dark and tarry. The black colour is a result of the oxidation of haemoglobin and the tarry appearance is from the bacterial breakdown of haemoglobin. Generally melaena is thought to be from bleeding from the upper intestinal tract or from the ingestion of blood (e.g. from epistaxis or oral lesions). Blood must be present in the gastrointestinal tract for several hours (8 hours in humans) before the colour turns black, so a rapid transit time of blood may not show melaena and a very slow transit time may show as melaenic faeces from a bleed in the lower gastrointestinal tract.

CLINICAL TIPS FOR DIAGNOSING GASTROINTESTINAL BLEEDING

In dogs 350 to 500 mg of haemoglobin per kilogram of body weight must enter the gastrointestinal tract before melaena is seen, so gastrointestinal bleeding without the overt presence of melaena is very possible. A faecal occult blood test is useful to rule out melaena.

With chronic gastrointestinal bleeding, iron is lost from the body and an iron deficient anaemia may develop. Iron deficiency anaemias become microcytic and hypochromic. More acute bleeding can cause a significant drop in the packed cell volume (PCV), even within hours.

The serum urea nitrogen to creatinine ratio increases with upper gastrointestinal bleeding due to the absorption of blood protein. An increased serum urea with a creatinine within the reference range (or other similar disproportionate changes) should prompt the clinician to check for gastrointestinal bleeding. The use of diuretics can also cause this change.

Faecal occult blood tests using orthotolidine or guaiac tablets are qualitative and only moderately sensitive. It is recommended to feed a meat-free diet (e.g. cottage cheese and rice) for 3 days prior to collecting the faecal sample as the myoglobin from meat in the diet may interfere with the test, causing a false positive.

CASE WORK-UP

The dog was admitted to the hospital and rehydrated with intravenous crystalloid fluids.

Minimum data base

Haematology, serum chemistry and routine urinalysis were performed. Haematology showed a regenerative anaemia with a PCV of 0.277 l/l (reference range 0.39–0.55) and an uncorrected reticulocyte count of 2%. The reticulocyte corrected to 1.23% as determined by correction for her PCV and indicated a mild degree

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of regeneration. The platelet count was within the reference range at 218 × 10^9 (reference range 200–500 × 10^9/l).

CLINICAL TIP ON CORRECTING RETICULOCYTE COUNTS

Reticulocyte counts are necessary for an objective assessment of the degree of RBC regeneration, but the count should be assessed with consideration of the degree of anaemia. One way to 'correct' the reticulocyte count is with the following formula:

Corrected reticulocyte percent =
[reticulocyte count (%) × PCV]/
[average PCV for species (45%) for dogs]

Results:
1 to 4%: Slight regeneration
5 to 20%: Moderate regeneration
>20%: Marked regeneration

Another way to assess the degree of regeneration is to multiply the % reticulocytes × RBC × 10. This is greater than 60,000 cells/μl in regenerative anaemias.

Serum chemistry confirmed the elevation in liver enzymes, with AP of 1454 IU/l (reference range 20–60 IU/l) and ALT of 815 IU/l (reference range 21–102 IU/l). Serum fasting bile acid concentration was also increased to 43.9 μmol/l (reference range 0–7 μmol/l) and serum bilirubin was increased to 10.9 mmol/l (reference range 0–6.7 mmol/l).

Total calcium was decreased to 1.9 mmol/l (reference range 2.3–3.0 mmol/l) with albumin just below the reference range at 25.2 g/l (reference range 26–35 g/l). The true albumin value may have been lower than this as haemoconcentration could have increased the apparent serum albumin concentration. The serum ionized calcium was normal at 1.1 mmol/l (reference range 1.1–1.5 mmol/l). Although serum amylase and lipase were within the reference ranges, a canine-specific lipase test was submitted. This result, which was available about 2 weeks later, was 271 μg/l (reference range 0–200 μg/l), indicating a mild to moderate pancreatopathy. Clotting times (activated partial thromboplastin time and prothrombin time) were within the reference ranges.

Urinalysis was unremarkable other than a urine specific gravity of 1.011, which was inappropriately low for a dog with dehydration. This again was likely to have been affected by the administration of corticosteroids, but could also have been caused by renal disease or other disorders affecting urine concentrating ability. Urine culture was negative.

Coombs test

A Coombs test, which was run to help rule out immune mediated haemolysis, was negative, although corticosteroid administration can cause false negative results. The increase in bilirubin can be consistent with haemolysis, but also with liver disorders. There was no haemoglobinuria, which can occur with haemolysis, but haemolysis was still a possibility.

Faecal analysis

Faecal analysis for parasites was negative and faecal culture for enteropathogens was also negative. A faecal occult blood test was performed after feeding the dog cottage cheese and rice for 3 days and this was positive for occult blood. A positive faecal occult blood test is indicative of either gastrointestinal bleeding or swallowed blood. As there was no evidence of nasal or oropharyngeal bleeding in this dog, gastrointestinal blood loss was most likely.

Differentials for a positive faecal occult blood in this dog included:

- Clotting disorders, although this dog had normal coagulation parameters and platelet count, a platelet function disorder was possible.
- Gastrointestinal ulcers, which can be caused by drugs, especially non-steroidal anti-inflammatory or corticosteroid administration, inflammation, neoplasia such as adenocarcinoma or lymphoma, sharp foreign bodies, gastrointestinal ischaemia (e.g. volvulus, shock), abnormal vasculature.
- Gastrointestinal parasites may also cause GI blood loss and although the faecal parasitology was negative, she was treated with 3 days of fenbendazole at 50 mg/kg/day po.

Imaging

Abdominal ultrasound examination showed a large hyperechoic homogeneous liver, an enlarged right pancreatic lobe and free peritoneal fluid (Figs 34.1 and 34.2). The adrenals were both small (Fig 34.3). The stomach appeared hypomotile. A sample of the fluid was obtained and a Tru-Cut liver biopsy was taken.

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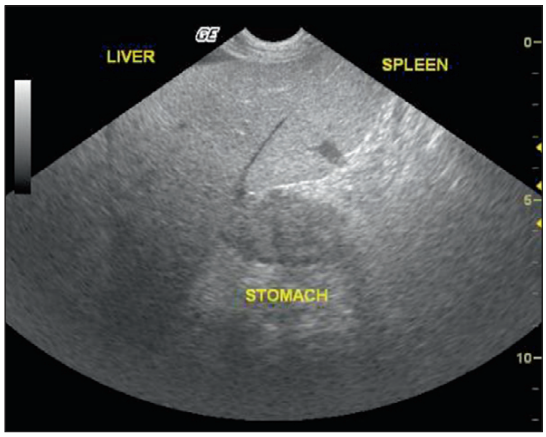


Fig 34.1 Ultrasound picture of the liver and spleen, showing a mottled hyperechoic appearance to the liver (courtesy of Carolina Urraca del Junco).

Abdominal fluid and liver biopsy analysis

The abdominal fluid had a low specific gravity of 1.015, low protein content of 2.4 g/l and moderate cellularity with a count of 6.1 × 10^9/l. Neutrophils were predominant, comprising 80% of the cells, with 20% macrophages and the rest lymphocytes and mesothelial cells. The neutrophils were hypersegmented. The poor correlation of the cellularity and the low protein content made it impossible to classify the fluid into a distinct category (Table 34.1). The fluid was cultured and was negative for bacterial growth.

The liver biopsy results showed severe diffuse, midzonal to periportal hepatocellular swelling and clearing consistent with a corticosteroid hepatopathy.

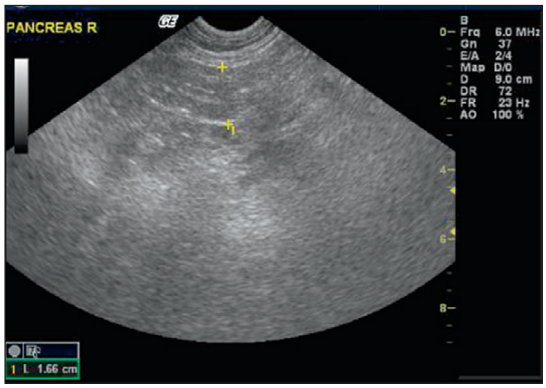


Fig 34.2 Ultrasound picture of the pancreas, showing an enlarged right pancreatic lobe (R = right) (courtesy of Carolina Urraca del Junco).

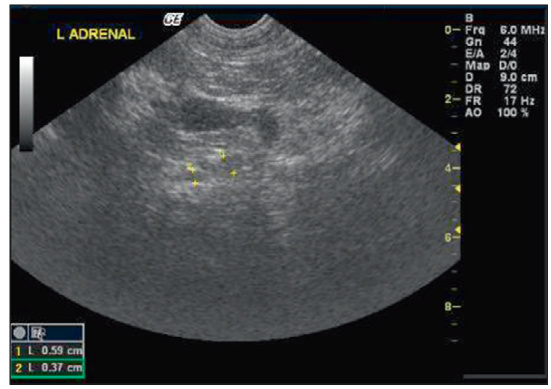


Fig 34.3 Ultrasound picture of the left adrenal gland, which is very small in size (as was the right adrenal gland) (courtesy of Carolina Urraca del Junco).

Table 34.1 Clinical tip: determining the type of ascites

	Specific gravity	Nucleated cell count	Protein	Predominant cell type	Common cause
Transudate	<1.017	<1,000 cells/μl	<2.5 g	Mesothelial/macrophage; up to 60% neutrophils	Hypoalbuminaemia; portal hypertension
Modified transudate		<5,000 cells/μl	>2.5 g	Mesothelial/macrophage; up to 60% neutrophils	Portal hypertension (pre-hepatic, hepatic or post-hepatic); heart failure
Exudate	>1.017	>5,000 cells/μl	>2.5 g	Neutrophils/macrophage; >60% neutrophils	Many, e.g. peritonitis, trauma, neoplasia, chylous effusions, blood

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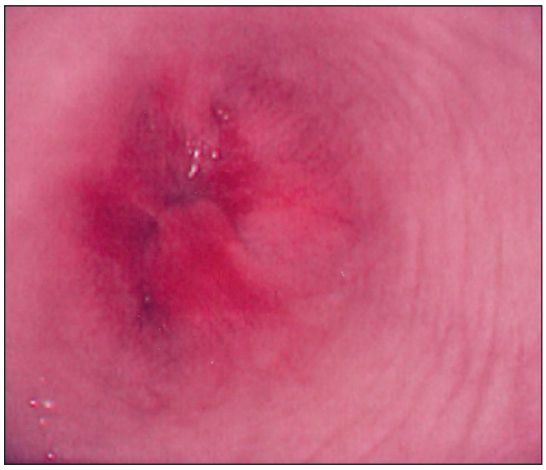


Fig 34.4 Endoscopic picture of oesophagitis, showing reddening of the distal oesophagus at the junction with the gastro-oesophageal sphincter.

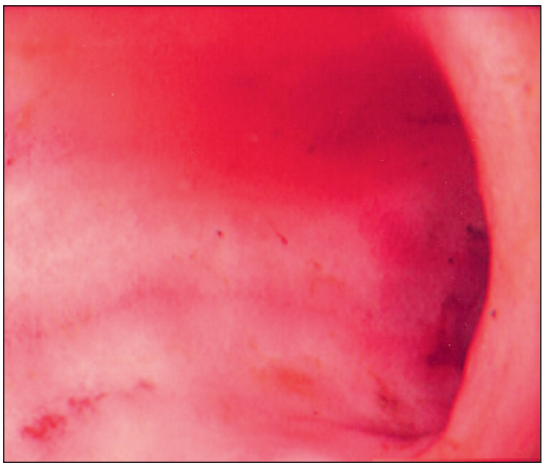


Fig 34.6 Endoscopic picture of linear ulcers in the ilium.

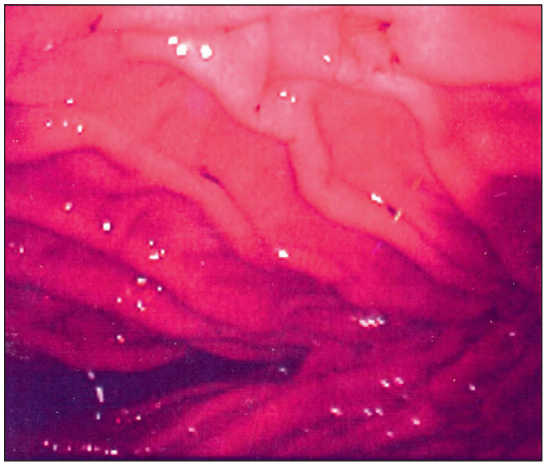


Fig 34.5 Endoscopic picture of pinpoint ulcers in the cardia of the stomach. The retroflexed endoscope can be seen in the lower left of the picture.

Gastrointestinal endoscopy

At endoscopy the lower oesophagus was found to be inflamed (Fig 34.4) and there were multiple areas of ulceration within the stomach (Fig 34.5), duodenum and ileum (Fig 34.6). Histopathology of pinch biopsies of the stomach, duodenum, ileum and colon all showed signs of inflammation.

CLINICAL TIP ON CORTICOSTEROIDS

While corticosteroids are an appropriate treatment for many disorders, they can also affect and confound many diagnostic tests (e.g. liver enzymes, serum bile acids concentration, Coombs, serum thyroxin, urine specific gravity) and cause many negative side effects such as polyuria/polydipsia (pu/pd), muscle wasting, vomiting and GI ulceration.

CLINICAL TIP ON GI HISTOPATHOLOGY VS. GROSS APPEARANCE

It is not uncommon for gastrointestinal histopathology to appear to be inconsistent with what is seen grossly at endoscopy; the information from both techniques needs to be combined as they look at different aspects of diseases.

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CLINICAL TIP ON THROMBOCYTOPENIA

Many dogs with immune mediated thrombocytopenia do not achieve a platelet count within the reference range. They should be monitored and if the platelet count is continuing to fall after they are off medication, diagnostic and therapeutic interventions may be necessary. The treatment goal is to prevent clinical signs of thrombocytopenia, not achieve a number within the reference range.

Medical treatment options

Two aspects of the treatment of the case were addressed: the oesophagitis and gastric bleeding, and the hepatopathy.

The oesophagitis and gastric bleeding were treated with ranitidine (2 mg/kg po q 12 hours) for its antacid and promotility effects and liquid sucralfate (3 ml po q 8 hours) for its ulcer binding and prostaglandin secretion effects. A short course of clavulanate-amoxicillin (16 mg/kg po q 12 hours) was administered, as gastrointestinal bleeding can predispose to bacterial translocation.

The dose of oral methylprednisolone was decreased over about a 1-month period while monitoring the platelet count, which continued to be stable. As soon as the dose was approximately 0.5 mg/kg, every other day dosing was administered, as this allowed the animal to have a 'recovery' day from the effects and usually markedly decreases the side effects. The hepatopathy was treated with ursodeoxycholic acid (10 mg/kg po q 24 hours), S-adenosyl methionine and vitamin E (Hepatosyl™) at three capsules twice a day po for their positive effects on the hepatocytes.

Outcome

The dog recovered very well, with the PCV rising to 0.33 l/l and the liver enzymes dropping to approximately half the values at presentation within 6 weeks. The owner reported an improvement in energy, muscle mass, pu/pd and coat quality.

DISCUSSION

Drugs are a very important cause of gastrointestinal ulceration in the dog. While there is some controversy whether all corticosteroids are severely ulcerogenic, most clinicians agree that high doses of dexamethasone have the potential for significant gastric erosion. Prednisolone by itself is not generally severely ulcerogenic unless it is used in high doses (e.g. >1–2 mg/kg/day) or for long terms; however, some individual dogs are especially sensitive to the effect. Many clinicians feel that larger breeds of dogs are more at risk. Any dog which becomes anaemic, has melaena or an increased serum urea, should be investigated for potential gastrointestinal bleeding, especially if it is on corticosteroid treatment.

Prognosis

The prognosis for dogs with gastrointestinal ulceration depends upon the severity of the ulcers. Some cases can rapidly develop perforating ulcers which can cause a fatal peritonitis and some resolve with cessation of the inciting drug. In any patient on corticosteroids or non-steroidal anti-inflammatory medications gastrointestinal signs, pale mucous membranes or a decrease in PCV should be addressed promptly.

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SECTION 5

THE COLON AND COLONIC DISORDERS

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35 Introduction to the colon and colonic disorders

The large intestine comprises the caecum, colon, rectum and anus (Fig 35.1). The primary role of the colon is the resorption of water and electrolytes. Colonic bacteria break down fermentable fibre to produce short chain fatty acids – acetate, butyrate and proprionate. Butyrate is an essential energy source for colonocytes.

Diarrhoea associated with the colon occurs when there is interference with water or electrolyte resorption, abnormal motility or presence of abnormal luminal substrate following pancreatic, hepatic or small intestinal disease.

Diseases of the colon include:

- Diarrhoea
- Constipation and megacolon
- Neoplasia
- Rectal disease; neoplasia, strictures, foreign bodies
- Colonic vascular dysplasia
- Typhilitis – rare

Causes of large bowel diarrhoea include:

- Inflammatory disease
  - acute non-specific colitis
  - chronic colitis, non-specific colitis, inflammatory bowel disease
  - infectious colitis (bacteria as for small intestinal disorders)
  - parasites, e.g. Trichuris, Coccidia
- Obstructive disease
  - intraluminal obstruction, e.g. tumour, constipation, foreign body, intussusception, stricture
  - extraluminal obstruction (hernia, masses, adhesions)
- Ischaemic disease – infarct, intussusception
- Neoplasia
  - adenocarcinoma
  - lymphoma
  - benign polyps
- Non-inflammatory disease
  - motility disorders, e.g. irritable bowel syndrome (IBS)
  - congenital malformations
- Systemic disorders
  - toxaemias (e.g. pyometra, peritonitis)
  - uraemia
  - pancreatitis
- Dietary causes
  - food poisoning
  - intolerance or allergy
  - foreign material (bones, hair, plastic)
- Secondary to unabsorbed nutrients from small intestinal diarrhoea

Clinical signs of large intestinal disease include:

- Chronic or acute diarrhoea
- Tenesmus
- Dyschezia
- Haematochezia
- Mucus production
- Abdominal pain
- Intermittent vomiting (20% of cases)

INVESTIGATION OF LARGE INTESTINAL DISORDERS

An initial minimum data base, especially faecal testing, as for small intestinal diarrhoea (see page 116) may be helpful in chronic cases. In cats, a fresh saline faecal preparation should be examined for *Tritrichomonas foetus* under the microscope and a sample sent for PCR test.

Imaging

Radiology and ultrasound may be helpful for diagnosing megacolon, foreign bodies, bowel thickening or focal masses, mesenteric lymphadenopathy (usually a non-specific finding) and intussusceptions. Computerized tomography is also useful for localizing and assessing focal or generalized bowel thickening.

Colonoscopy and biopsy

Colonoscopy allows for visualization of the colonic mucosa (if preparation is adequate) (Fig 35.2) and

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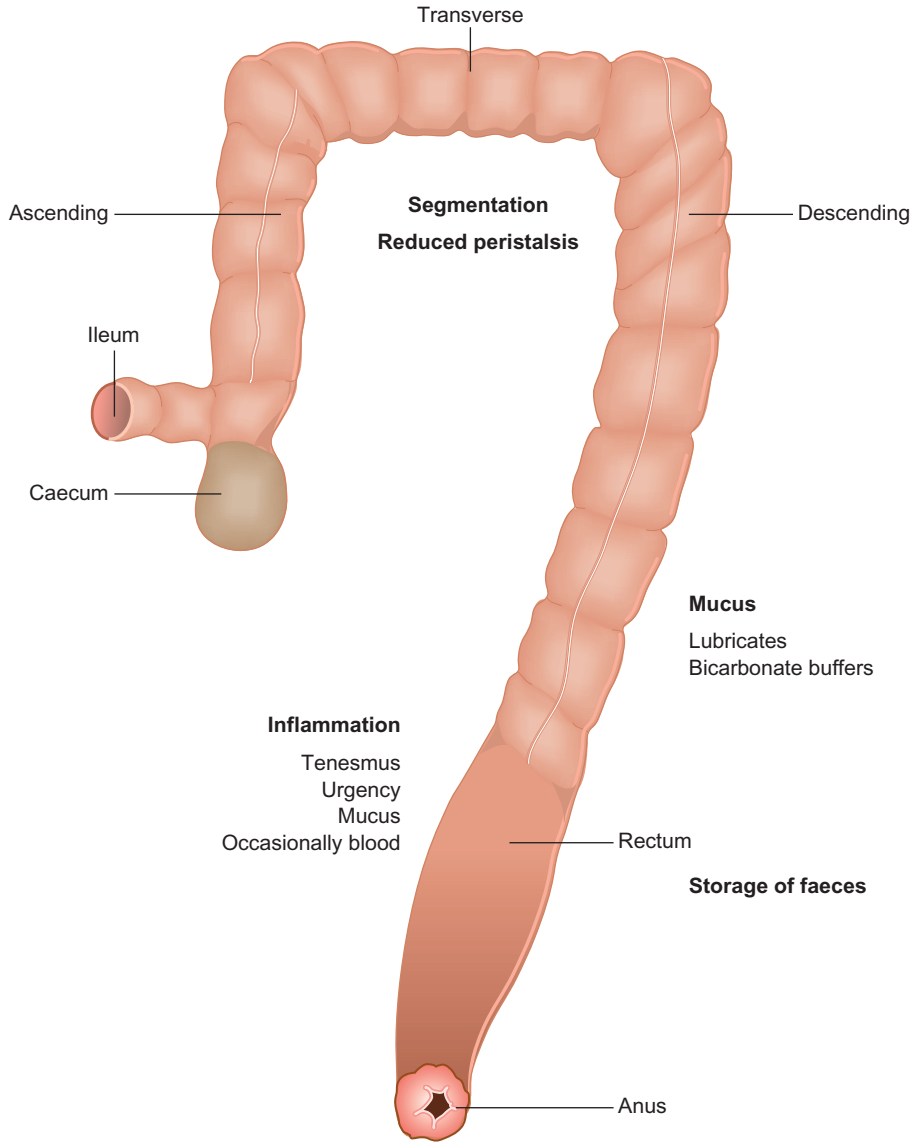


Fig 35.1 Illustration of the large intestine, which comprises the caecum, colon, rectum and anus.

observation of intraluminal masses, mucosal texture and colour. Submucosal vessels should be visible; if they are not visible, it indicates thickening of the mucosa. Generalized or focal haemorrhage is seen in some cases.

Biopsy may confirm the presence of inflammatory or neoplastic cells. Often in cases of large bowel diarrhoea the histology of the colonic biopsy shows little or no inflammation. These cases may be analogous to the human condition of IBS.

Irritable bowel syndrome

Irritable bowel syndrome (IBS) is thought have an underlying motility disorder and possibly the patient has an increased sense of colo-rectal pain. The patient presents with diarrhoea or constipation, often intermittent, and may also show vomiting, abdominal pain, borborygmi and flatulence. No pathological lesion is found at colonoscopy or on histopathology of colonic mucosa. It may be associated with stress and is sometimes detected in

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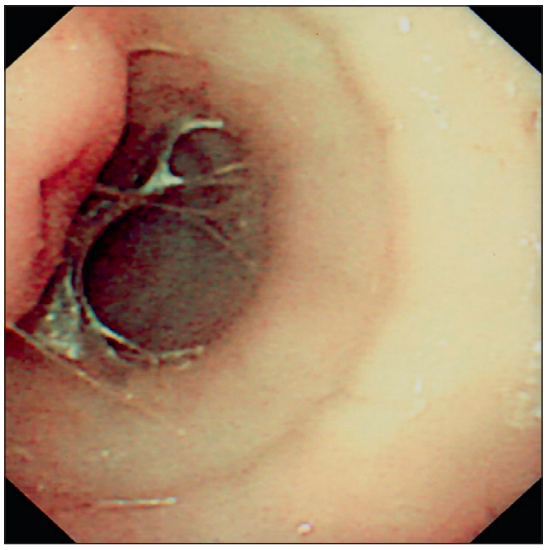


Fig 35.2 Colonoscopy allows visualization of the colonic mucosa.

working dogs. There is no definitive diagnostic test for IBS, it is diagnosed by exclusion of other disorders.

Dietary management with increased soluble fibre helps some cases. Motility modifying drugs such as loperamide, buscopan and mebeverine may also be tried.

Constipation

Constipation may be defined as infrequent or difficult passage of faeces. This may be an acute or chronic problem, but there is no permanent loss of function, whereas with obstipation the constipation is refractory and no longer responds to medication.

Megacolon, which literally translates as huge colon, is often a clinical finding in association with obstipation. Colonic impaction is the presence of hard faeces within the colon that are no longer moving caudally. It can be associated with constipation, obstipation or megacolon, but does not necessarily mean loss of function.

The clinical signs may include tenesmus, dyschezia, small volume and increased frequency of defaecation. Blood may appear on the surface of faeces.

The aetiology of constipation is extensive and not confined to disorders of the colon. It is therefore very important to examine the patient thoroughly as part of the investigation of this disorder.

Causes of constipation include:

- Ingestion of indigestible material, e.g. bones, hair, cat litter, fibrous plants
- Lack of exercise, e.g. obesity, orthopaedic disease, hospitalization
- Painful defecation, e.g. orthopaedic disease, perianal disease
- Mechanical obstruction
  - colorectal mass
  - congenital anorectal lesions
  - healed pelvic fracture with stenosis
  - intrapelvic mass
  - perineal hernia
  - enlarged prostate
  - pseudocoprostasis
  - rectal diverticulum
  - strictures
- Neurological disease
  - dysautonomia
  - idiopathic megacolon (most common cause of obstipation in cats)
  - paraplegia
  - sacral spinal cord deformity (e.g. in some Manx cats)
- Miscellaneous, e.g. dehydration, drugs, hypokalaemia, hypothyroidism, hypoparathyroidism

Treating the impaction may involve giving enemas, oral laxatives, mineral oils and, if necessary, manual removal of faecal material under general anaesthesia. Most cases should receive intravenous fluids prior to starting other treatments.

The prognosis is guarded as the problem may recur, especially if the underlying problem has not been resolved. Failure to correct the constipation may lead to the development of megacolon.

Megacolon

Congenital and acquired cases of megacolon occur (Fig 35.3). Acquired cases of dilated megacolon are often the end stage condition of idiopathic colonic dysfunction. There is an abnormality of smooth muscle function in cats with idiopathic dilated megacolon.

Hypertrophic megacolon may occur due to obstructions such as pelvic fracture and stenosis, neoplasm or chronic foreign body. Feline dysautonomia and canine dysautonomia are also acquired causes of constipation that may lead to megacolon.

Congenital cases are thought to be associated with failure of adequate myenteric plexus development.

Patients may present with reduced, absent or painful elimination of hard faeces. Dehydration may occur. Radiographs usually show an enlarged colon, with

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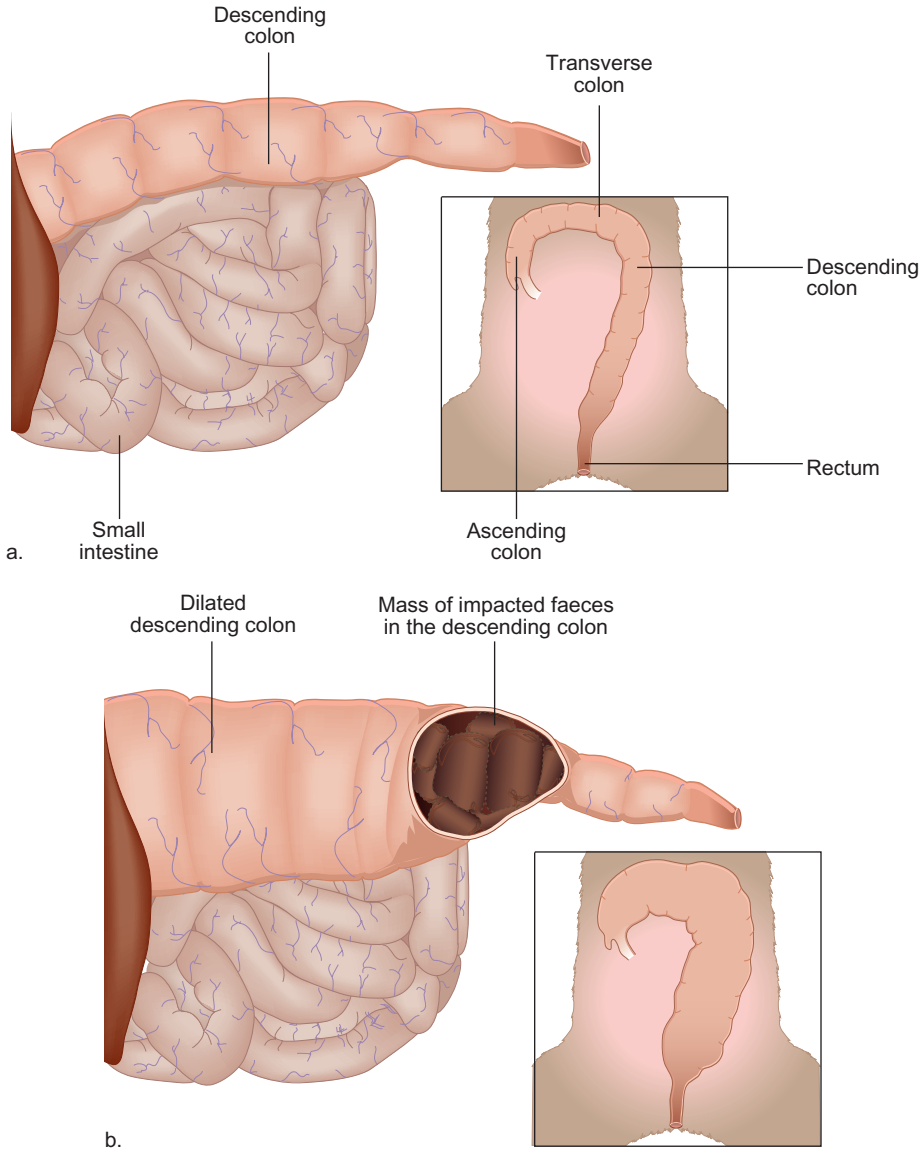


Fig 35.3 (**a**) Normal colon. (**b**) Megacolon.

distension occurring throughout the colon. A healed pelvic fracture with malunion or other obstruction may be seen with hypertrophic megacolon. The diagnosis is based on history, clinical findings and radiography.

There are five aspects to managing the patient with megacolon: optimal systemic hydration, removal of impacted faeces, increased dietary fibre (early in disease) to increase faecal bulk and stimulate motility, laxatives and colonic prokinetic agents such as cisapride (where available), prucalopride and tegaserod.

Colonic neoplasia

The most common tumours seen in the large intestine include lymphomas, adenocarcinomas and rectal adenomatous polyps.

Diagnosis depends on rectal examination, imaging studies (e.g. barium enema and radiography or CT scan, ultrasound of the more proximal colon) and colonoscopy and biopsy with histopathology.

Treatment depends on the type of tumour. If an adenocarcinoma is focal and surgically accessible, it may be resected. Piroxicam either as a rectal suppository or

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orally or other non-steroidal anti-inflammatory drugs may decrease the clinical signs of rectal adenocarcinomas and adenomas.

RECTAL DISEASES

Rectal stricture

Rectal strictures are surprisingly common in dogs but rare in cats. The aetiology is often not determined but may be the result of previous trauma or associated with neoplasia.

Patients present with haematochezia and dyschezia. They may become constipated due to avoiding painful defecation.

On rectal examination the stricture will usually be palpated easily and where neoplasia is present, a roughening or obstruction may be felt. Endoscopy and biopsy with histopathology are essential to rule out neoplasia.

Treatment involves surgical correction where possible. Stretching may also be considered in those cases unsuitable for surgery.

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36 Feline constipation and megacolon

Case contributed by Nicki Reed

INITIAL PRESENTATION

Tenesmus, haematochezia and abdominal distension

Signalment: 8-year-old, female neutered Devon Rex cat, body weight 2.5 kg

CASE HISTORY

The cat was presented for tenesmus, haematochezia and abdominal distension, accompanied by anorexia and lethargy. Since being acquired 4 years earlier she had had intermittent episodes of constipation, manifested as straining to pass hard faeces which were occasionally coated with fresh blood or mucus. The episodes had initially occurred two or three times per year, but for the past 6 months had been occurring monthly. During these episodes the cat would defecate outside the litter tray and she appeared to have abdominal bloating. Symptomatic treatment with laxatives generally improved the condition within 1 to 2 days.

Occasional episodes of small volume diarrhoea were reported (once to twice a year). At the time of presentation, she had passed no faeces for 4 days and had had a poor appetite for 2 days.

The cat was acquired from a rescue centre 4 years previously and lived indoors with one other unrelated Devon Rex cat. She was vaccinated against feline herpesvirus-1, calicivirus and feline parvovirus and dewormed every 6 months with praziquantel and pyrantel. The cat was fed a proprietary canned cat food.

She was thought to have previously been used for breeding and was reported to have had surgery for an intussusception at 2 years of age.

PHYSICAL EXAMINATION

The cat appeared slightly thin (body condition score 3/9) and had a distended abdomen (Fig 36.1). Her hydration appeared adequate and mucous membrane colour was pink with a capillary refill time of 2 seconds. Thoracic auscultation revealed a Grade I to II/VI systolic murmur, with point of maximal intensity at the left sternal border. No pulse deficits or arrhythmias were detected. Respiratory rate was mildly increased at 30 breaths per minute, attributed to stress, as no abnormal lung sounds were detected. Abdominal palpation revealed a large colon, filled with hard faeces and a moderately distended bladder.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The cat's primary problem was thought to be constipation, based on the history and physical examination findings.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for constipation include:

- Dietary causes, e.g. ingestion of bones or foreign material.
- Environmental causes, e.g. decreased exercise, litter box problems (dirty, change in type of litter or location of box; competition from other cats; inability to use the box due to arthritis or other locomotor disorders), stress
- Traumatic disorders, e.g. fractured pelvis; fractured or dislocated hip; bite wound; abscess; abdominal bruising; saccro-coccygeal luxation

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Fig 36.1 Dorsal view of the cat showing distended abdomen.

(tail-pull – unlikely as there was no history of trauma in this cat and the problem was chronic)

- Obstructive disorders, e.g. anal stricture; anal sac infection; rectal foreign body; rectal or colonic tumour; pelvic fracture malunion; perineal hernia; rectal diverticulum; pseudocoprostasis
- Neuromuscular disorders, e.g. spinal cord disease; congenital spinal cord anomaly (Manx cats); paraplegia/paralysis; central nervous system dysfunction; dysautonomia (Key–Gaskell syndrome); idiopathic megacolon
- Pharmacological causes, e.g. opioids; diuretics; anticholinergics; antihistamines; barium sulphate

With the chronic history and the lack of clinical signs consistent with most of the other possible differential diagnoses for constipation, the most likely cause in this cat was thought to be idiopathic megacolon, although other neurological causes and neoplasia could not be ruled out

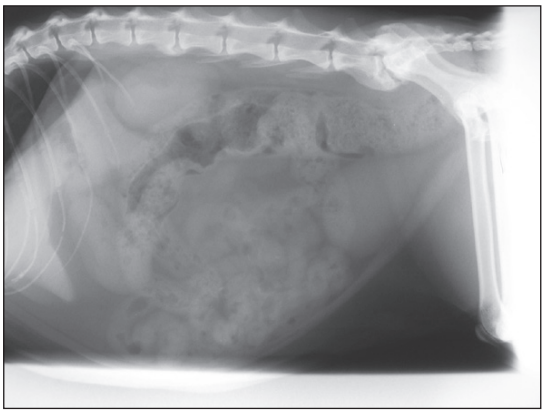


Fig 36.2 Lateral abdominal radiograph showing impaction of the transverse and descending colon with faeces at initial presentation.

CASE WORK-UP

A neurological examination was performed with no abnormalities detected. A rectal examination showed no evidence of herniation, anal sac disease, strictures or foreign bodies. A few small pieces of hard dry faeces were present in the rectum.

Minimum database

Routine haematology showed a moderate neutrophilia (26.2×10^9/l; reference range $2.5–12.8 \times 10^9$/l). This most likely represented a non-specific inflammatory response. Serum chemistry parameters were within the reference ranges.

Imaging

Abdominal radiography showed impaction of the transverse and descending colon with faeces (Fig 36.2). In addition, ventral spondylopathy was present at the lumbosacral junction. Two views were taken to fully evaluate narrowing of the pelvic canal.

Further investigations that could have been beneficial include urinalysis for assessment of hydration, ultrasonography if intraluminal or extraluminal masses were suspected, colonoscopy if intraluminal disease is suspected and barium enema if a stenotic lesion was suspected. If the neurological examination had been abnormal, further evaluation with MRI may have been useful.

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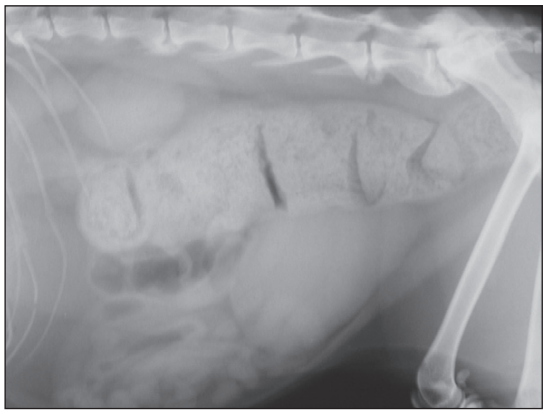


Fig 36.3 Lateral abdominal radiograph showing a grossly dilated impacted colon at revisit.

Diagnosis

A diagnosis of colonic impaction was made. A diagnosis of obstipation could not initially be made, as it was not known whether the condition was refractory to therapy. Initial management was commenced with manual evacuation of faeces with the cat anaesthetized and a warm water enema was given.

Oral therapy with lactulose (initial dose 0.5 ml po q 8 hours) and cisapride (1.5 mg/kg po q 12 hours) was then started and the owner advised to feed a low residue diet. The clinical signs improved initially, although the frequency of defecation remained decreased, occurring only every second day.

Follow-up

Over the following 18 months the cat required increasing doses of lactulose and cisapride and micro-enemas with sodium alkylsulphoacetate on three occasions. The cat subsequently presented after having not defecated for 4 days. She also again had a poor appetite and was lethargic. Further radiographs were obtained which showed a grossly dilated impacted colon (Fig 36.3) and as the condition was no longer responding to management a diagnosis of idiopathic megacolon was made.

Treatment options

The most appropriate treatment depended on the underlying cause of constipation and the severity of the problem. Potential therapy includes:

- Ensure adequate hydration and correct electrolyte imbalance if present
- Evacuation of faeces (enemas)
- Dietary alteration and bulking agents
- Laxatives
- Prokinetics
- Surgery

Evacuation of faeces

When the cat was anaesthetized, an endotracheal tube was in position in case colonic manipulation induced vomiting. A combination of digital evacuation and enemas was typically used. Digital evacuation was performed gently, with copious lubrication. A warm water enema was used in this cat, although a variety of enema agents are available:

- Warm water or saline (5–10 ml/kg)
- Mineral oil, e.g. liquid paraffin (5–10 ml/cat)
- Lactulose (5–10 ml/cat)
- Sodium citrate + sodium alkylsulphoacetate
- Dioctyl sodium sulphate

CLINICAL TIP ON ENEMAS IN CATS

Enema should be given slowly as rapid administration can induce vomiting. In addition, rapid administration reduces the ability to soften the stool and can increase the risk of colonic perforation. Enemas containing sodium phosphate should not be given to cats; they can cause hyperphosphataemia and an associated hypocalcaemia that may be fatal. Dioctyl sodium sulphate can increase the absorption of mineral oil therefore these two products should not be used together.

Dietary changes

Alterations in diet consist primarily of feeding increased fibre or low residue diets. Bulking agents consist primarily of poorly digested celluloses and polysaccharides (insoluble fibre). The insoluble fibre draws water into the colon and stimulates colonic motility and thus defecation. Either proprietary high-fibre diets can be used or bulking agents such as sterculia may be added to the diet. Cats should be adequately hydrated before using

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bulking agents and this approach is only appropriate for mild cases. In cats with megacolon, increasing dietary fibre may be contraindicated, as the fibre is incapable of inducing colonic motility and may contribute to obstipation. Highly digestible/low residue diets are more appropriate in these circumstances as the faecal volume is reduced.

Laxatives

Emollient

Emollient laxatives are detergents that improve the mixing of lipids (oils) with water. This improves the absorption of lipids from the colon, but decreases the absorption of water, hence softening the stool.

- Docusate plus dantron

Clinical experience with oral emollient laxatives is limited and efficacy not yet proven. Hepatotoxicity and carcinogenicity have been reported in people resulting in one of these products only being licensed for use in the terminally ill. Long-term use should therefore be avoided.

Lubricant

Lubricant laxatives reduce water absorption and facilitate passage of the faeces. Effects are generally mild, but they may be useful in mild cases of constipation.

- White paraffin
- Liquid paraffin (5–10 ml/cat per os)

Care should be taken if giving liquid paraffin orally, as it can be aspirated into the lungs. Chronic use of mineral oil laxatives can result in decreased absorption of fat-soluble vitamins.

Hyperosmotic

This group comprises sugars, magnesium salts and polyethylene glycol that, due to their concentrated nature, draw fluid into the lumen of the colon. The most commonly used is lactulose (0.5 ml/kg po q 8–12 hours), which also undergoes fermentation in the colon to produce organic acids. The organic acids stimulate fluid secretion in the colon. The magnesium salts are less well tolerated in cats than they are in humans and should *NOT* be used in cats with renal failure (due to impaired magnesium excretion). Polyethylene glycol is used commonly in the human field; however, the large volume required with some products makes them unfeasible to administer to cats. Further, polyethylene glycol is contraindicated in cases of mechanical or functional bowel obstruction (i.e. most cases of feline megacolon) and there are some concerns that polyethylene glycol can cause renal failure, therefore this author would caution against its use.

Stimulant

Stimulant laxatives cause secretion of fluid from the cells lining the colon and also stimulate the nerves that cause contraction of the smooth muscle. This latter effect may damage the nerves if used long-term, therefore continuous daily use is not recommended.

- Senna
- Castor oil
- Bisacodyl 5 mg po q 24 hours

Prokinetics

Prokinetic drugs act to improve the contractions of the bowel, thereby propelling digesta from the stomach to the rectum. Different areas of the gut have different receptors, therefore some prokinetic drugs are more effective in the stomach than the colon. Cisapride belongs to the group of drugs known as benzamide prokinetics and is particularly effective in the colon of cats because of its action on the $5HT_4$ receptors that are present there. Although it is not readily available in the United Kingdom, it may be imported under a Special Treatment Authorization (see Appendix 7). New drugs in this group (renzapride, prucalopride, mosapride) are being developed, as well as non-benzamide prokinetics (tegaserod). The latter was licensed in the USA, but has been recently withdrawn due to concerns about cardiac side effects and is therefore unlikely to be available to the veterinary market.

The H_2 antagonists ranitidine and nizatidine have some prokinetic effect, although they are not as potent as cisapride. Their mode of action is via inhibition of acetylcholinesterase, resulting in accumulation of acetylcholine at the neuromuscular junction and potentiation of the action potential. Efficacy has been demonstrated in vitro, but not in vivo and related H_2 antagonists cimetidine and famotidine do not share this prokinetic effect.

- Cisapride 0.1–1.0 mg/kg po q 8–12 hours
- Ranitidine 1.0–2.0 mg/kg po q 8–12 hours
- Nizatidine 2.5–5.0 mg/kg po q 24 hours

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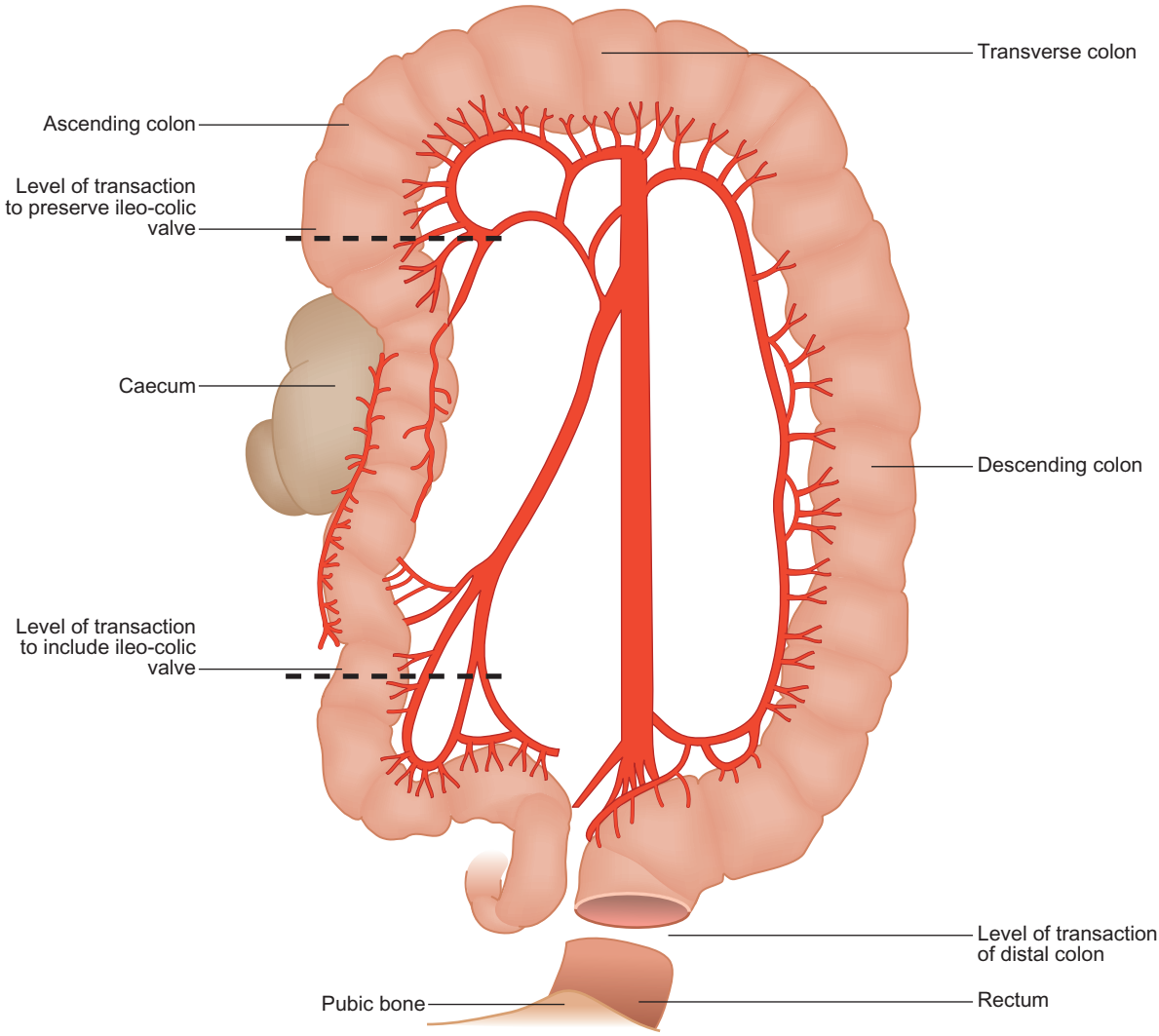


Fig 36.4 Illustration of the locations of the surgical options for colectomy.

Surgery

Although mild cases of constipation will respond to medical management, cases of obstipation usually require correction by means of surgery to remove the distended and non-functional colon. This procedure is known as a sub-total colectomy and may be performed with or without preservation of the ileo-caeco-colic valve (Fig 36.4). The advantages and disadvantages of each procedure are given in Table 36.1.

CLINICAL TIP ON CONSTIPATION IN CATS

Owners of constipated cats may occasionally perceive the problem to be diarrhoea, rather than constipation. Faecal concretions within the colon are irritating to the mucosa causing secretion of fluid which can leak past the impacted faeces.

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Table 36.1 Advantages and disadvantages of sub-total colectomy

Surgical procedure	Advantages	Disadvantages
Ileo-colic valve preserved	Reduced risk of colonic bacteria refluxing into small intestine and causing diarrhoea	May be recurrence of constipation in the segment of colon not removed. The bowel at the site of repair may be under greater tension
Ileo-colic valve removed	More colon is removed, therefore it is less likely to have recurrence of constipation Some surgeons feel it is an easier procedure to perform, as the area where the two ends of bowel are joined is under less tension	There may be a greater risk of small intestinal diarrhoea as a result of colonic bacteria migrating to the small intestine
Both procedures	The vast majority of cases have permanent resolution of constipation problems, thereby improving the cat's quality of life and removing the necessity for permanent medication	*Surgical*: Anaesthetic risk Wound infections, breakdown Blood loss Peritonitis *Postoperative*: Recurrence of constipation Diarrhoea Increased frequency of defaecation

NURSING ASPECTS

Standard enema pumps may be too wide bore to effectively introduce into a constipated cat. Use of a dog urinary catheter and syringe may facilitate introduction of enema solutions more proximally into the colon, enabling break-up of faecal material.

Many cats when hospitalized will become constipated. Reasons for this may include drugs (e.g. opioids), unfamiliar litter substrate (especially if normally outdoor cats), anxiety and lack of exercise. Trying a variety of litters, providing privacy and allowing the cats to exercise, particularly after feeding, may encourage defaecation.

Lack of water intake may be a feature in a number of cats that become constipated and cats are often reported to drink little at home. Reasons for this may be the water bowl is beside the food bowl, the bowl is an inappropriate size, the cat cannot see the water level or the cat prefers running water. Moving the water bowl away from the food bowl and trying larger bowls of a different substance may encourage drinking, as can use of a water fountain.

Follow-up

With a diagnosis of idiopathic megacolon no longer responsive to medical management the cat underwent surgery for a sub-total colectomy. Histopathological findings were consistent with idiopathic megacolon. Following surgery her demeanour and appetite improved, although she had a tendency to pass diarrhoeic faeces. The cat has remained well for 6 years post-surgery and has been maintained on a highly digestible diet.

Pathophysiology

The colon has two main functions: absorption of water and electrolytes and storage of faeces. The wall of the colon is made up of two types of smooth muscle: the concentric circular muscle on the luminal aspect and the longitudinal muscle on the serosal aspect. The muscular layers receive stimulation from the intrinsic nerves whose cell bodies lie in the submucosal (Auerbach's) and myenteric plexuses (Fig 36.5). These nerves cause 'slow-wave' contractions that occur at a rate of approximately five per minute. The contractions are initiated by a 'pacemaker' located at the junction of the transverse and descending colon. The slow wave contractions mix the digesta. When the colon becomes distended with digesta more coordinated contractions (segmentation

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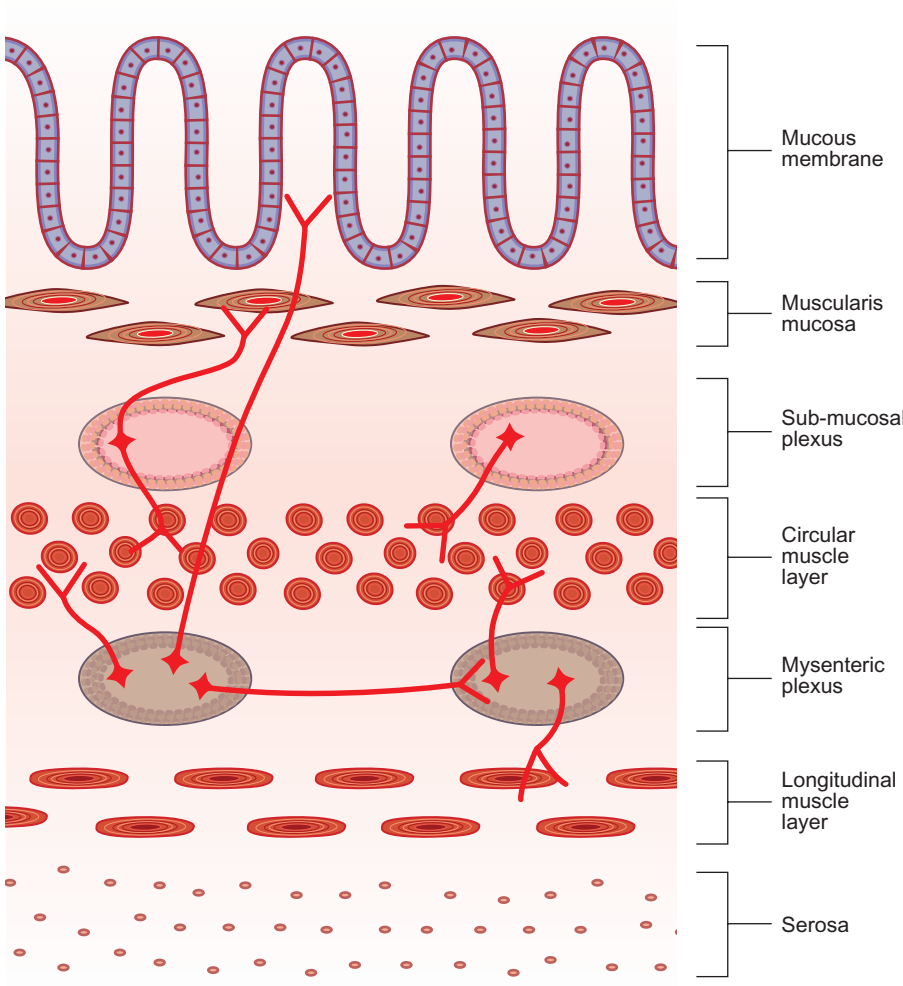


Fig 36.5 Illustration of the muscular layers of the colonic wall.

and peristalsis) are initiated, which pushes the digesta aborally towards the rectum. Here, defecation is controlled by the extrinsic nerve supply from the pelvic and pudendal nerves (Fig 36.6). The extrinsic nerve supply can also have an effect on segmentation, either stimulating it (parasympathetic) or inhibiting it (sympathetic – adrenergic).

The cause of megacolon is not well understood. It is typically recognized in middle-aged to older cats, with a history of progressively worsening constipation. Proposed mechanisms have included abnormalities with the nervous supply to the lower colon (intrinsic or extrinsic), an abnormal neuromuscular junction or abnormal smooth muscle. No anatomical abnormalities have been demonstrated with histopathological examination, but the contractile ability of the smooth muscle has been shown to be altered. It is not known whether this altered contractility is the cause of megacolon or simply an effect of prolonged stretch.

Two forms of megacolon are described – dilated and hypertrophic. Idiopathic megacolon is a dilated form of megacolon, with permanent loss of function. Hypertrophic megacolon may arise when the colon dilates proximal to an obstruction (e.g. fractured pelvis) and may be reversible if the obstruction is relieved in sufficient time (<6 months). If the underlying causal agent is not addressed, hypertrophic megacolon will progress to irreversible dilated megacolon.

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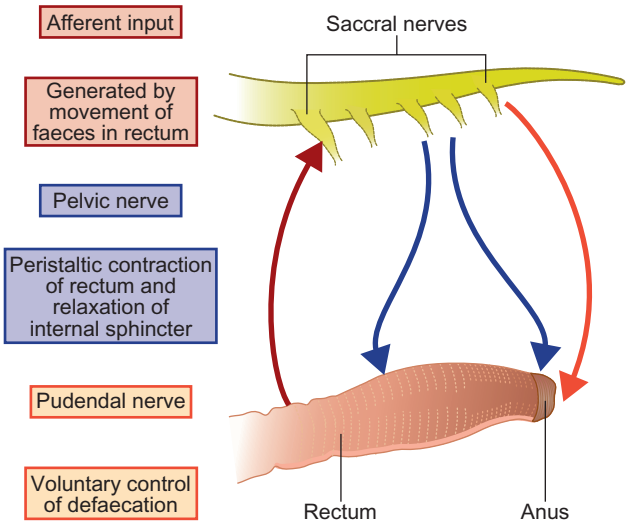


Fig 36.6 Illustration of the nerve supply to the colon.

Epidemiology

Constipation may be observed in cats of any age, breed or sex. The literature suggests that it tends to be a problem of middle-aged cats, with male cats more commonly affected (70% cf 30% females). Domestic shorthaired cats (46%), domestic longhair cats (14%) and Siamese cats (11%) have been most commonly reported as being affected, but breed prevalence was not taken into account to establish whether these breeds are truly over-represented (Siamese) or under-represented (DSH). In addition, the literature focuses on cases of obstipation or megacolon and therefore the true incidence of *constipation* in the feline population is not known.

Prognosis

The prognosis for simple cases of constipation or where an underlying aetiology can be addressed is generally good. The majority of cases of recurrent constipation develop some degree of colonic hypomotility with subsequent development of megacolon. Although medical management is successful initially, the condition is often progressive therefore owners should be warned that their cat may become refractory to medical management and that surgery may well become necessary. In some cases the condition may lead to premature euthanasia of an animal, either because the owner does not wish surgery or because following surgery the cat develops intractable diarrhoea or recurrence of megacolon.

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37 *Tritrichomonas* infection in two kittens

INITIAL PRESENTATION

Profuse watery diarrhoea

Signalment: Two 6-month-old littermate male entire Siamese kittens, body weights 3.18 kg and 3.4 kg

CASE HISTORY

The kittens had been obtained from a breeder 2 months earlier and both had had diarrhoea since then. The kittens had a profuse, watery diarrhoea containing mucus. They showed an increased urgency to defecate and increased frequency of defecation.

They were eating a high quality commercial kitten food and both were still eating well and had not shown any vomiting. They had been fully vaccinated and dewormed with fenbendazole. They were indoor cats with occasional supervised access to a fenced garden.

PHYSICAL EXAMINATION

The kittens were both bright and in good body condition (BCS 5/9). Mucous membranes were pink and moist and CRT was <2 seconds. Auscultation and compression of the thorax of both were normal. Abdominal palpation was mostly unremarkable, although their colons felt fluid filled. There was inflammation of the perianal area of one kitten. The rectal temperatures, heart rates and respiratory rates were within normal limits and the pulse quality was good in both kittens.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Diarrhoea of large intestinal origin

Differential diagnosis

Differential diagnoses for chronic large intestinal diarrhoea in these kittens included:

- Inflammatory diseases
  - chronic colitis
  - inflammatory bowel disease
  - infectious colitis, e.g. *Salmonella* spp., *Campylobacter* spp., *Clostridium perfringens*
  - parasites, e.g. *Tritrichomonas foetus*, *Giardia*, *Cryptosporidium*, coccidia
- Dietary
  - food poisoning
  - dietary intolerance or allergy
- Toxins, including toxic plants
- Congenital malformations
- Small intestinal malabsorption and the production of secretagogues such as unconjugated bile acids and hydroxylated fatty acids
- Other causes such as obstruction, ischaemia or neoplasia were less likely to involve two kittens. Infections and environmental (diet, toxin) factors were the most likely causes of the diarrhoea.

CASE WORK-UP

Minimum data base

Haematology and serum chemistry were performed and the results were unremarkable. Serum feline trypsin like immunoreactivity (TLI), cobalamin and folate were within the reference ranges, consistent with the presumed large intestinal origin of the diarrhoea. Screening tests for FeLV and FIV were negative. Routine urinalysis was unremarkable in both kittens.

Imaging

Abdominal ultrasound showed mildly enlarged mesenteric lymph nodes but no other abnormalities. Intestinal wall layering and thickness were within normal limits.

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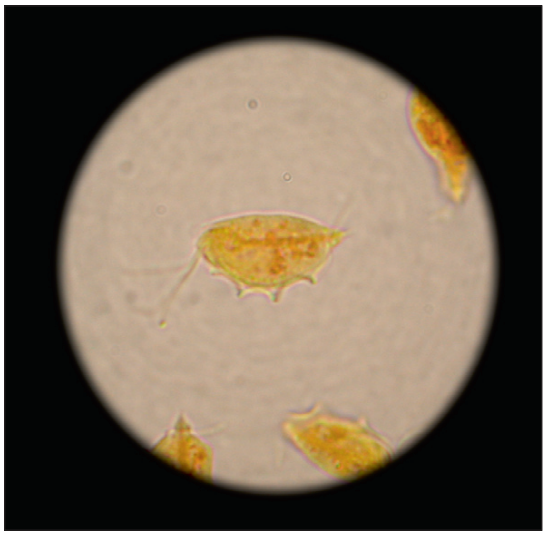


Fig 37.1 Microphotograph of *Tritrichomonas foetus* (courtesy of Dr Andy Sparkes)

Faecal tests

Faecal flotation (performed on three samples) showed no parasitic ova and faecal culture for enteropathogens was negative. Zinc sulphate centrifugation-flotation preparations were negative for *Giardia* and *Cryptosporidium*.

A fresh faecal smear was collected by inserting a swab into the anus of each of the kittens. The faeces and mucus obtained was diluted with an equal volume of normal saline and examined under the microscope. Large numbers of motile spindle shaped trophozoites were observed (Fig 37.1), which had flagellae and moved with a jerky movement. This appearance is typical of *Tritrichomonas foetus*. To confirm the infection, a sample of faeces from each kitten was submitted for *T. foetus* polymerase chain reaction (PCR), which was positive in both kittens.

Treatment and outcome

The kittens were treated with ronidazole at 30 mg/kg q 12 hours po for 14 days. Both kittens improved on the treatment. Their diarrhoea completely resolved and they showed no adverse side effects from the treatment.

DISCUSSION AND EPIDEMIOLOGY

T. foetus is a flagellated protozoan parasite that causes large bowel diarrhoea in infected cats. Clinical signs vary among cats, but often include increased frequency of defecation, passing of semi-formed to liquid diarrhoea often described as foul-smelling and containing mucus and blood, and flatulence. The anus may become inflamed due to the severe diarrhoea, as happened in one of these kittens. Affected cats are usually well otherwise and do not usually show significant weight loss.

The protozoal induced diarrhoea appears to be more common in younger cats (often less than a year of age), cats from households containing pedigree cats or other multi-cat households and young cats from rescue centres. It has been reported that the Siamese and Bengal breeds of cats may have an increased risk and purebred cats in general seem to be over-represented. It may be that genetic factors increase the risk in these cats, although the high density management likely increases the risk of infection.

Cats shed the organism in their faeces and infection is thought to be faecal–oral. The distribution of the organism is not completely understood yet, but it has been reported in the USA, UK and Germany and there are indications that it is also present in other European countries, China and South America.

Fresh faecal smears diluted with saline may be examined microscopically for the presence of typical motile trophozoites. If any mucus has been passed with the faeces this should be included in the smear as it is the most likely place to find the organisms. A cover slip should be pressed over the smear and then the slide can be examined under ×200 and ×400 magnification.

In most clinically affected cats, large numbers of organisms can be seen. They have short tails and an undulating membrane that runs over the length of the body. They are described as having a jerky, forward motion. Examination of multiple smears and multiple faecal samples will improve the rate of detection of the organism. Rectal swabs, as used in this case, can also be examined for the organism. A cotton swab is inserted into the anus and rotated over the colonic mucosa. It is then withdrawn and a smear made on a microscope slide which is again diluted with saline and examined. The organism should to be distinguished from *Giardia*. With *Giardia* infection the trophozoites tend to be far fewer in number, they are binucleate with a concave ventral sucker, sometimes described as a 'monkey face'. *Giardia* do not exhibit the same forward motion as *T. foetus*. If a cat has had recent antibiotic therapy, the number of *T. foetus* trophozoites shed may be decreased which can make the diagnosis more difficult. In such cases, more sensitive diagnostic techniques may be preferable.

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Two other diagnostic tests are available which are both more sensitive and specific for this organism than examination of fresh faeces. The first test is PCR (polymerase chain reaction) which can detect the presence of the genetic material of the organism. This is a highly sensitive test. In the USA, samples can be submitted to the College of Veterinary Medicine, North Caroline State University for this test. Information on this is available in Appendix 6. The PCR test is available in the UK from Capital Diagnostics in Edinburgh (phone 0131 535 3145). See Appendix 6 for further information about testing for *T. foetus*.

Treatment

Treatment with ronidazole at 30 to 50 mg/kg has been shown to resolve the diarrhoea and eradicate infection (on the basis of the PCR test). However, ronidazole is not licensed for use in cats and although it appears to be relatively safe from limited studies, it should be used with caution and only with informed and signed owner consent. Neurotoxic side effects have been reported with ronidazole, including agitation, facial tremors and trembling, seizures, ataxia and anorexia. In two reported cases, the signs stopped when the drug was withdrawn. Metronidazole has also been used, but has shown poor efficacy. Tinidazole has been trialled as it is thought to possibly have less risk of neurotoxicosis compared to ronidazole. While tinidazole decreased the detection and shedding of *T. foetus*, it appeared to be less efficacious than ronidazole.

Prognosis

The clinical prognosis for most infected cats is good, although many owners report subsequent infrequent and short-duration relapses of diarrhoea. It is not known if these relapses correspond with faecal shedding of *T. foetus.* As the diarrhoea does resolve over time and is often more of an inconvenience than being associated with significant adverse effects in affected cats, it may not be necessary or advisable to treat all affected cats with ronidazole. Using a simple, highly digestible diet frequently results in improved faecal consistency and this alone may allow sufficient control of clinical signs in some cats.

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38 Colorectal neoplasia in a dog

INITIAL PRESENTATION

Haematochezia, diarrhoea and faecal tenesmus

Signalment: 2-year-old entire male German shepherd dog, body weight 29 kg

CASE HISTORY

The dog had initially begun to have diarrhoea 4 months prior to presentation. The diarrhoea had become progressively more haemorrhagic and increased in frequency and urgency to defecate. At presentation the dog strained to defecate almost continuously when taken on walks. He produced small amounts of soft to liquid faeces and large amounts of blood when defecating. The dog only rarely vomited. The owner said the dog had lost about 7 kg during the last 4 months.

His current diet was a restricted protein dry diet, with no snacks or treats. The dog's vaccinations were current and he had recently been treated with 50 mg/kg fenbendazole for 5 days.

He was on meloxicam for discomfort due to hip dysplasia. This had been stopped for a month 2 months earlier and there had been no resolution of clinical signs of diarrhoea or haematochezia. Other than the hip dysplasia, he had been a healthy dog until the onset of the diarrhoea.

PHYSICAL EXAMINATION

The dog was bright, alert and responsive (Fig 38.1) although he was quite thin (body condition score 3/9) with muscle loss over the lumbar area (Fig 38.2). His mucous membranes were pink and moist with a capillary refill time of <2 seconds and hydration appeared adequate. No abnormalities were noted on thoracic auscultation. His heart rate was 82 beats per minute with good matched pulses and respiratory rate was 32 breaths per minute. Peripheral lymph nodes were normal in size. Due to the history of haematochezia, taking the rectal temperature was delayed until analgesia (e.g. topical lignocaine) could be used. When taken later it was 38.2° C.

On abdominal palpation several large firm masses were identified. One of these was especially prominent in the caudal abdomen and was felt to be at least 4 to 5 cm in diameter. The masses were not attached to the body wall and could be moved fairly freely. A tubular mass was present in the ventral abdomen, which was thought to be either a ventrally displaced colon or an enlarged loop of small intestine. The dog appeared uncomfortable during abdominal palpation.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

The dog's primary problems were haematochezia and tenesmus consistent with large intestinal disease. The frequency and urgency of the diarrhoea was also consistent with disease of the colon and/or rectum, although weight loss is less common in large intestinal disease.

DIFFERENTIAL DIAGNOSIS

A differential list for the haematochezia, diarrhoea and tenesmus includes disorders of the colon and rectum. There was no melaena and no evidence of bleeding anywhere else (e.g. haematuria, epistaxis). Generally differential diagnoses lists should be made for each problem; in this case it was thought that the problems were likely to be related. The differential diagnoses include:

- Colitis
- Vascular dysplasia

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Fig 38.1 Dog showing bright and alert attitude



Fig 38.2 Dog showing loss of muscle mass

- Intussusception
- Foreign body
- Aneurysm
- Infarction
- Neoplasia
- Parasites (hookworms, whipworms)
- Non-steroidal anti-inflammatory drug (NSAID) use

Severe acute pancreatitis can cause melaena and haematochezia, but this dog was too bright to have severe acute pancreatitis. The dog had been given an adequate dose of fenbendazole, making gastrointestinal parasites less likely. While he had previously been on meloxicam, there had been a period of about a month where it had not been given and there had been no change in clinical signs.

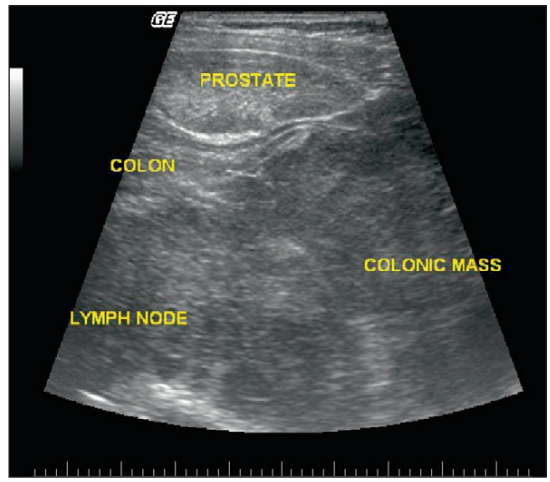


Fig 38.3 Ultrasound of the colonic mass (courtesy of Carolina Urraca del Junco)

The other problem was the presence of masses or thickened areas found during palpation of the abdomen. The most likely causes of these were thought to be neoplasia, lymph node enlargement or intussusception(s).

CASE WORK-UP

Minimum data base

Haematology, serum chemistry and urinalysis parameters were unremarkable. Faecal parasitology was negative but faecal culture was positive for *Clostridium perfringens.*

Imaging

On abdominal ultrasound, there was gross enlargement of medial iliac lymph nodes, with a highly heterogeneous hypoechoic internal appearance. The largest lymph node was 8 × 4 × 4 cm. The cranial mesenteric node close to the greater curvature was grossly enlarged at 6 × 4 cm and the hepatic or pancreaticoduodenal node was also enlarged at 3 × 2 cm. A 1-cm round hypoechoic nodule was also found in the colonic wall at the level of the medial iliac lymph nodes and a much larger colonic tumour a few centimetres caudally, at the level of the pubic brim. The mass was at least 6-cm long and was hypoechoic, involving mucosa, with loss of layering and an irregular luminal interface (Fig 38.3).

Thoracic films were taken to check for metastasis and the pulmonary fields appeared normal.

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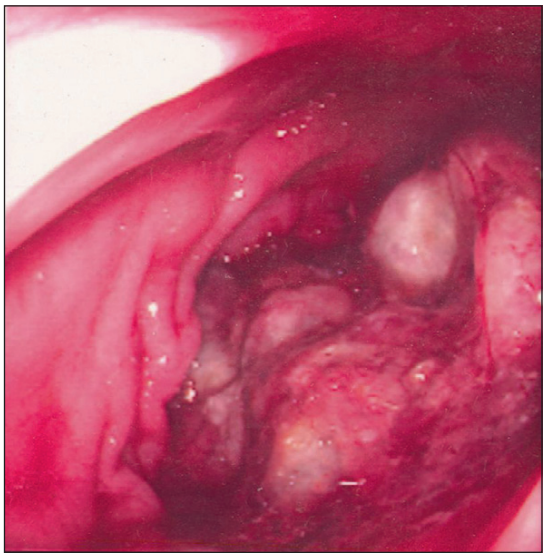


Fig 38.4 Endoscopic picture of colorectal mass

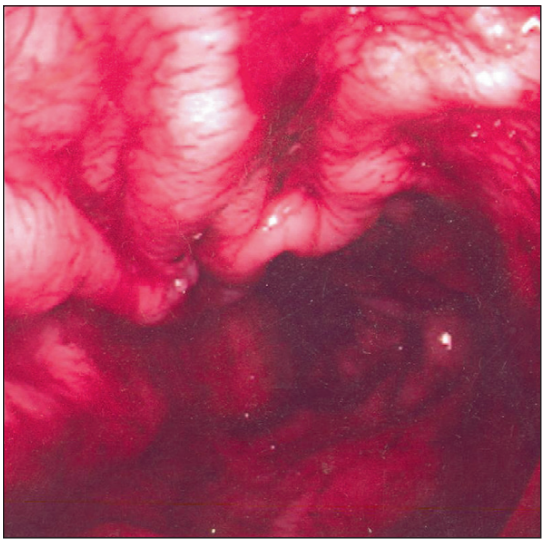


Fig 38.5 Endoscopic picture of colonic mucosa proximal to the mass

Lymph node cytology

A fine needle aspiration of the iliac lymph node was obtained; cytology of this was interpreted as lymphoma.

Endoscopy

At colonoscopy, a large, irregular, mass was found in the colorectal area approximately 2–3 cm from the anus (Fig. 38.4). It extended about 4 cm proximally and filled about 90% of the lumen. Proximal to this mass the colonic mucosa appeared swollen and irregular (Fig 38.5). Multiple biopsies were taken of the mass and of the colonic mucosa proximal to the mass to a distance of about 35 cm from the anus, where faecal material prevented further colonoscopy.

Histopathology

Histopathology of the mass was described as extensive necrotizing inflammatory reactions and the mucosa of the colon proximal to the mass was described as containing a mixed inflammatory cell population. Tumours often have a superficial layer of inflammation and this can be all that is obtained from a mucosal biopsy, so there may have been lymphoma underlying the mass or there may have been concurrent inflammatory bowel disease. Often full thickness biopsies can provide further information; however, as the lymph nodes of this dog were diagnostic of lymphoma, this was not pursued, as addressing the lymphoma was required regardless of the intestinal disease. The prognosis was affected by the presence or absence of alimentary lymphoma and an argument can be made for pursuing the exact diagnosis.

Treatment and outcome

As lymphoma was known to be present in the abdominal lymph nodes of this dog and suspected in the large intestine, a chemotherapy protocol was planned. This was initiated using prednisolone only, at 1 mg/kg twice a day, for 5 days. Aggressive chemotherapy in intestinal lymphoma can result in lysis of the lymphocytes to a degree that the intestine perforates.

The dog coped well with the prednisolone and after 2 days the abdominal lymph nodes felt smaller. He continued to have haematochezia, although it (subjectively) appeared to be less severe. He did continue to strain to defecate. On the fifth day, the prednisolone was decreased to 1 mg/kg once a day and vincristine was administered intravenously as the initiation of a chemotherapy protocol (by an Oncology Service) including vincristine, cyclophosphamide, prednisolone and doxorubicin (CHOP protocol).

In addition to the chemotherapy, the dog was treated for the *Clostridium perfringens* with metronidazole at 400 mg po q 12 hours for 3 weeks. His diarrhoea resolved within a week of initiation of this treatment and the haematochezia decreased with the chemotherapy.

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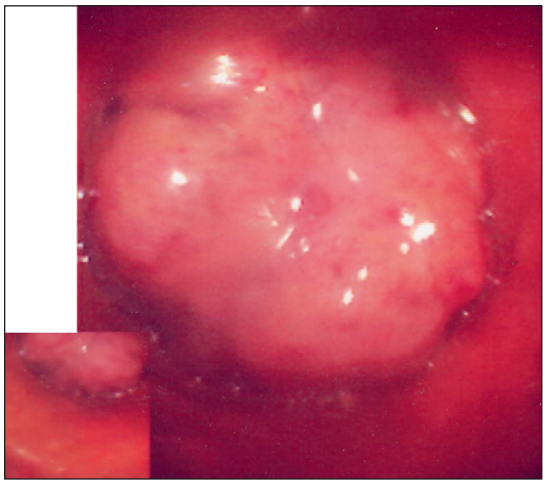


Fig 38.6 Endoscopic picture of a colonic adenoma

At 1 month after diagnosis the dog was reported to have gained 1 kg in weight. There was no organomegaly present, although there was a persistent caudal abdominal lymphadenopathy. On digital rectal examination the previous mass could not be felt. The dog's faeces were soft but formed and no blood was present. His status was described as partial remission. At 4 months after diagnosis the dog was doing very well and had gained another 3 kg in weight.

DISCUSSION AND EPIDEMIOLOGY

Colorectal tumours are uncommon in dogs. The most common tumours in this location are adenomas or adenocarcinomas, which often have a more rounded appearance than this tumour (Fig 38.6) and are often found as a focal growth which does not metastasize. Adenomas and adenocarcinomas are most often tumours of older dogs. If not surgically resectable, the clinical signs from adenomas and adenocarcinomas often improve with the use of NSAIDs, such as piroxicam given as rectal suppositories or per os.

Alimentary lymphoma accounts for about 5% of cases of lymphoma and is often more difficult to diagnose than the more common multicentric form. In this dog, the presence of a colorectal lymphoma was not definitively proven, although he had lymph node involvement. This could either be a spread of the colorectal tumour or the dog may have had two diseases. In one study, the median age for GI lymphoma was about 8 years, with a spread of about 3 to 13 years, so this dog was a fairly young victim of the disease. Six of 30 dogs in that study had enlarged lymph nodes.

There appears to be no clear sex predilection for lymphoma, although some studies have shown more male than female dogs affected.

Prednisolone was used in this dog prior to the initiation of a chemotherapy protocol. Most oncologists have recommended against using glucocorticoids for treating dogs suspected of having lymphoma. If the lymphoma has not been diagnosed, the rapid lymphocytolytic effects of glucocorticoids may have masked the diagnosis. Another reason for avoiding corticosteroid USC is the potential of multidrug resistance (MDR). MDR is a common cause for chemotherapeutic failure in canine lymphoma. It involves P-glycoprotein, a transmembrane protein which acts as an efflux pump for some chemotherapeutic agents, causing the drugs to be extruded from the tumour cells. Other mechanisms of resistance to multiple agents have been discovered and MDR now refers to a phenotype of simultaneous resistance to chemotherapeutic agents which differ in structure and also includes the aptly named MDR associated protein (MRP). Many chemotherapeutic drugs are able to induce P-glycoprotein or MRP expression; however, the ability of dexamethasone to upregulate P-glycoprotein expression may be tissue and species dependent. There is still some suggestion that pre-treatment with glucocorticoids may adversely affect the success of treatment with a multidrug chemotherapy protocol; the answer is not definitive. In one study dogs pretreated with glucocorticoids had a significantly shorter remission (134 days) compared to those that did not receive glucocorticoids (267 days). However, in the present case, initial treatment with a more rigorous protocol may have resulted in excessive cytolysis and intestinal breakdown, so a more gradual approach was used.

Prognosis

In one study the overall median survival time of dogs with GI lymphoma was 13 days; however, two dogs were alive at 31 and 84 months and both of these dogs had colonic or rectal neoplasia. Overall, alimentary lymphoma provides a fair prognosis for short to medium term remission, but a guarded prognosis for a long remission.

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39 Colonic inflammatory bowel disease in a cat

INITIAL PRESENTATION

Diarrhoea, decreased appetite and weight loss

Signalment: 11-year-old, male neutered, domestic shorthaired cat, body weight 3.8 kg

CASE HISTORY

The cat had a 1-month history of decreased appetite and weight loss, along with increasingly softer faeces. There was some increase in the frequency of defecation and the owner reported that mucus and fresh blood had been present on some occasions. The consistency of the faeces was described as 'cow-pat', i.e. soft but formed. He sometimes vomited, although the owners reported that this had been occurring for years.

The cat had been vaccinated within the past year, but had not been de-wormed for several years. His usual diet was a commercial canned cat food, fed two to three times per day. During the past month the owner had also been feeding him cooked ham and canned tuna to encourage his appetite.

PHYSICAL EXAMINATION

Physical examination revealed the cat to be bright, alert and responsive. He was in slightly thin body condition (body condition score 4/9) and appeared well hydrated. His mucous membranes were pink with a capillary refill time of less than 2 seconds. Oral examination showed that most of his teeth were missing but was otherwise unremarkable.

Thoracic auscultation revealed a heart rate of 200 beats per minute with matched pulses. Heart and lung sounds and percussion and compression of the thorax were unremarkable. The respiratory rate was normal at 32 breaths per minute. Abdominal palpation was not resented. There was possible abdominal lymph node enlargement. Rectal temperature was 38.3° C. The remainder of the physical examination was unremarkable.

PROBLEM LIST AND DISCUSSION OF PROBLEMS

- Diarrhoea
- Weight loss
- Decreased appetite

The diagnostic approach to diarrhoea usually begins with determining if the cause is large or small intestinal in origin. The diarrhoea in this case appeared to have a large intestine component due to the increased frequency and the presence of fresh blood and mucus. However, concurrent involvement of the small intestine could not be ruled out, especially with the presence of weight loss. The decreased appetite and weight loss were thought to be related to the diarrhoea. Differential diagnoses for decreased appetite include those which cause the animal to be unable to eat (can't eat) and those in which the animal does not want to eat (won't eat); in this case the cat was able to eat so the decreased appetite was likely due to illness.

DIFFERENTIAL DIAGNOSIS

Large intestinal diarrhoea

- Inflammatory conditions
  - non-specific colitis
  - chronic inflammatory colitis, e.g. plasmacytic/lymphocytic, eosinophilic, granulomatous, suppurative
- Infectious causes
  - FIP
  - feline leukaemia virus (FeLV)
  - *Salmonella*

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- *Campylobacter*
- *Clostridium perfringens*
- Parasitic
  - whipworms
  - hookworms
  - *Giardia*
  - *Tritrichomonas foetus*
  - *Toxoplasma gondii*
- Diet related
  - dietary sensitivity (allergy or intolerance)
  - fibre responsive diarrhoea
- Neoplasia, e.g lymphoma, adenocarcinoma, others
- Systemic disorders
- Uraemia
  - toxicities
  - pancreatopathy
- Secretagogues, e.g. unconjugated bile acids, hydroxylated fatty acids secondary to small intestinal disorders

Small intestinal diarrhoea

- Diet related
  - dietary sensitivity
  - food poisoning
  - sudden change of diet
- Small intestinal disease
  - inflammatory bowel disease, e.g. plasmacytic/lymphocytic, eosinophilic, other
  - infectious (viral, bacterial enterotoxins, fungal – although fungal disorders of the small intestine are unlikely in the UK)
  - parasites, e.g. *Giardia*, cryptosporida, Strongyloides, ascarids, hookworms, *Toxoplasma gondii*
  - neoplasia, e.g. lymphoma, mastocytosis
  - lymphangiectasia (uncommon in cats)
  - ileus, e.g. hypokalaemia, enteritis, dysautonomia
  - antibiotic responsive diarrhoea
- Pancreatic disorders
  - pancreatic exocrine insufficiency
- Chronic pancreatitis
- Liver disorders
  - liver failure
  - intrahepatic cholestasis
  - bile duct obstruction
- Kidney disease and uraemia
- Hyperthyroidism
- Congestive heart failure
- Hypoadrenocorticism (uncommon in cats)

CASE WORK-UP

A 4- to 6-week dietary trial with a highly digestible restricted novel protein or hydrolyzed diet is appropriate in some cases of diarrhoea. With cases of colitis, the addition of mixed soluble and insoluble fibre sources may also be of benefit. The use of probiotics and prebiotics are also being investigated in intestinal disorders of dogs and cats. In this case, however, as the cat was not eating well and had been losing weight, prompt investigation rather than waiting for the results of a dietary trial was indicated.

Minimum data base

Routine haematology was performed and revealed a moderate mature neutrophilia (21.7×10^9/l; reference range: $2.5–12.8 \times 10^9$/l) and a mild monocytosis (1.06×10^9/l; reference range: $0.07–0.85 \times 10^9$/l). Blood smear analysis confirmed these findings and showed atypical, reactive appearing lymphocytes, suggestive of antigen stimulation.

Serum biochemistry results revealed a marginal increase in urea (9.9 mmol/l; reference range: 2.8–9.8) and bile acids (7.2 mmol/l; reference range 0–7) and a marginal hypophosphataemia (1.39 mmol/l; reference range: 1.4–2.5). Mild hypoalbuminaemia (24.0 g/l; reference range: 28–39) was also present.

CLINICAL TIP ON DECREASED SERUM ALBUMIN

Decreased serum albumin can be due to decreased production by the liver, increased loss via the intestines or kidneys or a 'third space shift' (e.g. into an obstructed loop of intestine, the pleural space or the peritoneal space). Production may also be decreased in inflammatory states, as it is a negative acute phase protein and decreases whereas production of some of the other proteins increases. After checking for urinary loss (a urine protein to creatinine ratio), further diagnostics involve looking for evidence of hepatic or gastrointestinal disorders or inappropriate fluid accumulation.

To assess the pancreas, serum samples were submitted for measurement of feline pancreatic lipase (fPL) and trypsin-like immunoreactivity (TLI) which were both found to be within the reference range (fPL: 1.2 µg/l;

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reference range: 0.1–3.5; TLI: 22.5 µg/l; reference range: 12–82).

Both serum folate (4.2 µg/l; reference range: 9.5–20.2 µg/l) and serum cobalamin (186.0 ng/l; reference range: 270–1000 ng/l) concentrations were decreased and supplementation was initiated.

CLINICAL TIP ON SERUM FOLATE AND COBALAMIN

Cobalamin is absorbed in the ileal part of the small intestine. It requires prior binding with intrinsic factor, which comes from the pancreas in cats (and the stomach and pancreas in dogs). Deficiency may be caused by poor absorption due to intestinal disease or exocrine pancreatic insufficiency, excessive bacteria in the upper small intestine utilizing the vitamin or a deficiency in intrinsic factor. In humans who are vegetarians (especially elderly humans), a dietary deficiency is possible; however, this is unlikely in cats as they cannot successfully be vegetarians.

Unlike most other B vitamins, cobalamin is stored in the body. In healthy cats the half-life is 11 to 14 days and in cats with gastrointestinal disease it is only 4.5 to 5.5 days. Clinical signs of cobalamin deficiency in humans include anaemia, thrombocytopenia, neuropathies and digestion abnormalities. In cats (and dogs) the clinical signs are not as well described; however, it has been suggested that response to treatment of intestinal disorders is improved with cobalamin repletion. A high serum concentration of cobalamin does not appear to have clinical significance and may occur with supplementation. The recommended dose for treatment of gastrointestinal disease in humans exceeds the recommended daily amounts and there is a consideration that there may be positive pharmacological effects.

Folate is absorbed only in the proximal intestine. A deficiency usually indicates small intestinal disease and if it is accompanied by a cobalamin deficiency it likely indicates widespread disease. A high serum folate can be caused by bacterial production, although serum concentrations of this vitamin can also be increased by dietary intake.

Serum T4 was at the low end of the reference range at 15.3 nmol/l (reference range: 15–48 nmol/l). An feline immunodeficiency virus (FIV)/FeLV ELISA was performed which was negative for FeLV but positive for FIV antibodies.

Toxoplasma serology showed a negative IgM titre (<20) and an IgG titre of 200, which is most consistent with previous exposure to *Toxoplasma gondii* rather than active infection.

CLINICAL TIP ON TOXOPLASMOSIS TITRES

Toxoplasmosis titres can be positive in healthy animals and the IgM titres correspond best with clinical toxoplasmosis as this antibody class is rarely detected in the serum of healthy animals. In an animal with clinical signs, demonstration of an IgM antibody titre of >1:64 or a four-fold or greater increase in paired (2 to 3 weeks apart) IgG titres is likely to indicate disease. The absence of the titre increases does not rule out toxoplasmosis as some cats and dogs with the disease will undergo an antibody class shift from IgA to IgM. A response to treatment (e.g. clindamycin) is diagnostically supportive (although not definitive of diagnosis) if there is a suspicion of disease.

Urinalysis showed a urine specific gravity of 1.048, indicating good urine concentrating ability. The chemistry strip was unremarkable, and the urine sediment was inactive. Urine protein to creatinine ratio was 0.13 (reference range <0.4 for cats), indicating that the hypoalbuminaemia was not due to urinary loss.

Tests for intestinal parasites, including *Giardia* and *Tritrichomonas*, were negative. Faecal culture for enteropathogenic bacteria (*Salmonella*, *Campylobacter*, *Yersinia*) was negative. In cases of suspected *Clostridium perfringens* induced colitis, an enzyme-linked immunosorbent assay should be performed to identify CPE A and CPE B in faeces after a faecal bacterial culture is performed.

Imaging

Abdominal ultrasonography was performed with the cat sedated. This revealed enlarged, hyperechoic and

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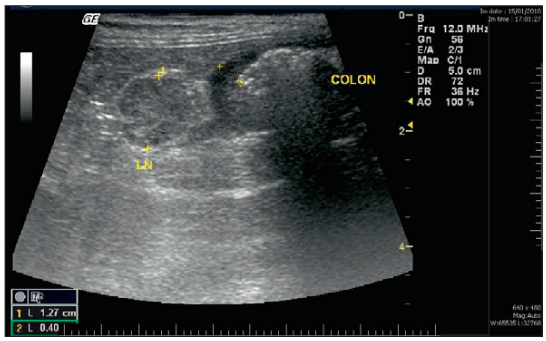


Fig 39.1 Ultrasound picture of enlarged, hyperechoic and irregularly outlined colonic and jejunal lymph nodes (courtesy of Carolina Urraca del Junco)

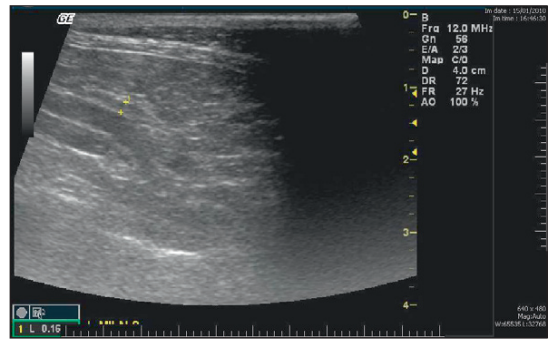


Fig 39.2 Ultrasound picture of colon showing marked thickening of the mucosa with loss of normal wall layering (courtesy of Carolina Urraca del Junco)

irregularly outlined colonic and jejunal lymph nodes (Fig 39.1). The muscularis layer of the small intestine was prominent. There was also marked thickening of the mucosa with loss of normal wall layering in the transverse and ascending colon (Fig 39.2).

CLINICAL TIP ON ULTRASOUND OF THE INTESTINE

Ultrasound imaging of the small intestine is very useful in defining small intestinal masses and interrogation of the wall layers of the bowel. The most prominent wall layer of the bowel is the mucosal layer and this is universally very hypoechoic. On ultrasound exams the normal width of the small intestinal wall is up to 5 mm and in the cat it is closer to 4 mm. Bowel wall width changes are often accompanied with an increase in the size of one or more layers of the small intestine. Disruption of the normal layering pattern of the intestinal wall can also be seen in some disorders.

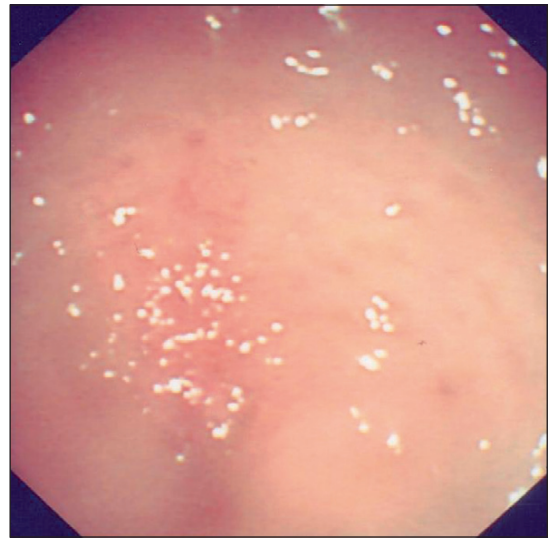


Fig 39.3 Endoscopic picture of colonic mucosa showing thickening and loss of visualization of submucosal vessels

At gastroduodenoscopy, the stomach and small intestine appeared grossly normal. At colonoscopy, the colonic mucosa appeared thickened and the submucosal vessels could not be clearly seen (Fig 39.3); however, there were no obvious mass lesions or ulcerated areas. Biopsy samples were collected from the stomach, duodenum and colon. While the cat was anaesthetized for endoscopy, fine needle aspirates of an enlarged mesenteric lymph node and the thickened colonic wall were also taken under ultrasound guidance and submitted for cytological evaluation.

Cytology of the lymph node aspirate was suggestive of reactive hyperplasia. The colonic wall cytology was, unfortunately, non-diagnostic.

Histopathological examination of the gastric mucosal biopsies revealed the presence of some *Helicobacter*-like spiral organisms at the surface of the gastric mucosa. There was no associated gastric inflammation and it was therefore considered an incidental finding.

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CLINICAL TIP ON GASTRIC HELICOBACTER

A causal role of *Helicobacter* in chronic gastritis and vomiting in dogs and cats has not been established, although there are cases in which treatment improves the clinical condition. As in this case the spiral bacteria present were only on the surface of the mucosa and there was no gastric inflammation or clinical signs; treatment was not warranted. Where there are clinical signs, evidence of inflammation and the organisms are located within the gastric pits or submucosally, treatment is probably indicated.

The small intestinal mucosal biopsies revealed evidence of mild, diffuse chronic enteritis. Histopathology of the colonic mucosal biopsies showed severe, multifocal, granulomatous ulcerative colitis (Fig 39.4). There was no evidence of any aetiological agents and Ziehl–Neelsen stains of the colonic biopsies for the presence of acid fast (i.e. mycobacterial) organisms were negative.

Treatment and outcome

This cat was diagnosed with a granulomatous ulcerated from of colonic inflammatory bowel disease, complicated by a positive FIV status. The FIV may either have caused or contributed to the cat's clinical condition. The enteritis in the small intestine was thought to be of less significance, although may have contributed to decreased nutrient absorption.

Initial treatment included:

- Prednisolone: 5 mg po q 24 hours
- Marbofloxacin: 7.5 mg po q 24 hours
- Folic acid: 2.5 mg po q 24 hours
- Vitamin B_{12}: 300 μg sq every 7 days for 3 weeks
- A novel restricted protein, highly digestible canned diet

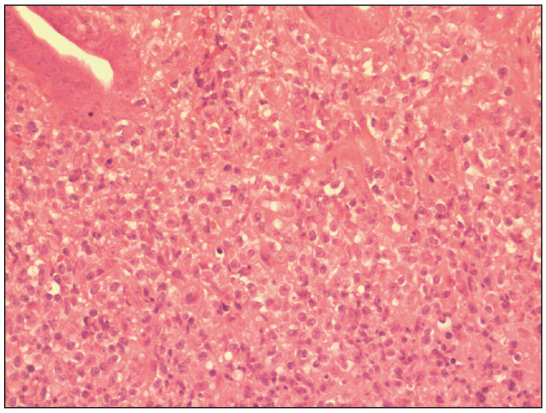


Fig 39.4 Histopathology of the colon showing severe, multifocal, granulomatous ulcerative colitis (courtesy of Professor Elspeth Milne)

CLINICAL TIP ON FLUOROQUINOLONE USE IN CATS

In some cats given enrofloxacin, retinal degeneration leading to partial, temporary or total blindness has occurred. This reaction is not common even with very high doses, but there is no way to predict which cats will react. This reaction has only been reported with enrofloxacin and not with other fluoroquinolones, as it is theorized that the biochemical structure of enrofloxacin leads to especially high concentrations in the feline eye. Because of the potential adverse reactions to enrofloxacin in cats, marbofloxacin was initiated in this cat prior to the results of the periodic acid–Schiff tests in the chance that similar pathophysiology to the histiocytic colitis of Boxer dogs might be present.

At the time of discharge the cat was very bright, with a good appetite, having gained 500 g since admission and with only intermittently soft stools.

Two weeks later the owners reported that the cat had done very well; his faecal quality was improving and he had gained some weight. After the marbofloxacin was stopped, the cat immediately showed signs of diarrhoea again and it was re-instituted 2 days later. The cat again improved and has remained well while continuing on marbofloxacin.

Pathophysiology and prognosis

In general, the more common forms of inflammatory bowel disease, with infiltration of lymphocytes and plasmacytes or even eosinophils, carry a relatively good prognosis for improvement in clinical signs.

Granulomatous colitis or enteritis, however, has a more guarded prognosis. Granulomatous colitis is characterized by the presence of aggregates of histiocytes in the lamina propria. In this cat the infiltration of the

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mucosa and lamina propria was described as predominantly large macrophage like cells, which may have been a histiocytic type of cell. The presence of underlying infectious causes (e.g. mycobacterial organisms, parasitic organisms, feline infectious peritonitis or fungal infections, although the latter are uncommon in the UK) should be ruled out before treatment with corticosteroids or other immunosuppressive drugs is used. Metronidazole is commonly used in feline chronic colitis, as it has multiple effects: antibiotic, antiprotozoal and inhibition of some aspects of cell mediated immunity.

An ulcerative histiocytic colitis has been reported in young Boxer dogs, which presents with thickened colonic mucosa and haemorrhage. This disorder shows a cellular infiltrate of plasma cells, lymphocytes and periodic acid–Schiff positive macrophages. The presence of the periodic acid–Schiff positive macrophages is a discriminating feature between this form of colitis and granulomatous colitis.

An infectious aetiology is suspected for this histiocytic disorder and treatment of cases early in the course of disease with enrofloxacin has been successful. Treatment is lifelong in these cases.

Cats with FIV show a significant incidence of dental disease (50–52% of seropositive cats) and most of the teeth in this cat had been lost or removed. A significant number (15–20%) of seropositive cats show chronic enteritis or enterocolitis. Clinical signs include anorexia, chronic diarrhoea, dehydration and weight loss. The diarrhoea may be persistent and, as in this cat, intestinal thickening develops. A high mortality rate is seen in FIV positive cats with gastrointestinal signs.

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Multiple choice questions

1. **In cases suspected of having regurgitation**
   - **a.** the pH of the food brought up is always acidic
   - **b.** episodes always occur within an hour after eating
   - **c.** the disorder is generally located in the oesophagus
   - **d.** the food brought up usually contains bile

2. **In a dog which is suspected of regurgitating, which of the following imaging procedures should be performed** first **to help confirm the diagnosis?**
   - **a.** CT of the thorax
   - **b.** Fluoroscopy of the thorax
   - **c.** Radiographs of the thorax
   - **d.** Ultrasound of the thorax

3. **Acquired myasthenia gravis in dogs is an**
   - **a.** idiopathic disorder with no known cause
   - **b.** immune mediated disorder with antibodies against ACh molecules
   - **c.** immune mediated disorder with antibodies against ACh receptors
   - **d.** infectious disease caused by *Bartonella* spp.

4. **The prognosis for a dog diagnosed with acquired myasthenia gravis is**
   - **a.** very poor with only 10% of dogs living for a year
   - **b.** guarded as only about 50% of the dogs survive
   - **c.** excellent with nearly 100% of dogs responding well
   - **d.** unknown as insufficient case numbers have been examined

5. **In cases of idiopathic megaoesophagus, the most common cause of death is**
   - **a.** starvation
   - **b.** systemic infection
   - **c.** oesophageal rupture
   - **d.** aspiration pneumonia

6. **Oesophageal strictures are usually best treated using**
   - **a.** balloon dilation, with repeated procedures if needed
   - **b.** solely medical management with antacid drugs
   - **c.** high dose oral corticosteroids to decrease inflammation
   - **d.** surgical repair performed immediately

7. **In a regurgitating 4-month-old puppy, a barium swallow shows abrupt discontinuation of the barium just cranial to the base of the heart with gross dilatation of the oesophagus cranial to that area. The most likely diagnosis is**
   - **a.** oesophageal foreign body
   - **b.** idiopathic megaoesophagus
   - **c.** oesophageal stricture
   - **d.** persistent right aortic arch

8. **What is the prognosis for dogs with persistent right aortic arch which have surgery?**
   - **a.** 80% have good swallowing function after the surgery
   - **b.** The megaoesophagus will still be present but most dogs won't regurgitate
   - **c.** About half the dogs will continue to regurgitate
   - **d.** Almost all of the dogs will still regurgitate

9. **There is evidence that dysautonomia in cats**
   - **a.** may have an aetiology related to botulinum type C neurotoxin
   - **b.** is likely to be caused by an autoimmune response

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c. may be related to a viral infection that is spread via faeces
d. is due to a congenital disorder as it occurs mostly in related cats

10. Diagnoses of feline dysautonomia is based primarily on
a. clinical signs
b. imaging results
c. a specific blood test
d. the response to treatment

11. Treatment of feline dysautonomia
a. is usually unrewarding as nearly all affected cats die
b. includes use of interferon for the viral component of the disease
c. is based on supportive, nutritional and nursing care
d. is primarily based on antibiotics effective against anaerobic bacteria

12. The pilocarpine test used to aid in a diagnosis of dysautonomia is based on
a. mydriasis of the treated eye due to denervation supersensitivity
b. miosis of the treated eye due to denervation supersensitivity
c. mydriasis of the treated eye due to lack of sensitivity
d. miosis of the treated eye due to lack of sensitivity

13. Large rocks or bones can enter the small intestine of a dog from the stomach
a. by moving through the pylorus with food particles
b. by exerting pressure on the pylorus so that it opens
c. due to 'housekeeping waves' which open the pylorus during fasting
d. by entering the duodenum with liquids during drinking

14. Dogs that chronically vomit bile in the early hours of the morning are likely to have
a. bilious vomiting syndrome and may respond to a late night snack
b. irritable bowel syndrome which will respond to probiotics
c. a motility disorder involving gastric emptying of food
d. alimentary lymphoma and need chemotherapy

15. Copper coloured irises in cats are nearly pathognomonic for
a. food sensitivity
b. persistent right aortic arch
c. dysautonomia
d. porto-systemic shunt

16. Metabolic alkalosis in a dog or cat which is vomiting is usually associated with
a. a pyloric or duodenal foreign body
b. gastrointestinal parasites
c. *Helicobacter* infection
d. pyometra

17. Barium impregnated polyethylene spheres (BIPS) are most useful for
a. looking for intestinal mucosal disorders
b. outlining the gastric shadow
c. checking for an intestinal obstruction
d. checking for megaoesophagus

18. The 'organ of vomition' is the
a. oesophagus
b. stomach
c. duodenum
d. colon

19. The chemoreceptor trigger zone is in
a. the floor of the fourth ventricle
b. the cerebrum
c. the vestibular apparatus
d. the duodenum

20. Endotoxic shock may occur in animals with intestinal obstruction with a foreign body due to the
a. presence of bacteria within the foreign body
b. consequences of dehydration due to vomiting
c. increased numbers of bacteria within the stagnant loop of intestine
d. body's response to the dilated intestine distal to the foreign body

21. A gastrointestinal barium contrast study is performed in a 10-year-old dog that has been vomiting intermittently for four months. On a

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ventrodorsal view, the pylorus is positioned more medially than normal and there is a decreased amount of contrast seen in the duodenum. While not absolutely diagnostic, this pattern is most suggestive of

a. linear foreign body obstruction
b. partial gastric dilatation and volvulus
c. mucosal pyloric stenosis
d. granulomatous inflammatory bowel disease

22. Which of the following drugs has a pro-motility intestinal effect at low doses in the fasting dog?

a. Amoxicillin
b. Clindamycin
c. Erythromycin
d. Enrofloxacin

23. Bradycardia is most likely to occur in dogs with hypoadrenocorticism due to

a. hyperkalaemia
b. hypocalcaemia
c. hyponatraemia
d. cortisol deficiency

24. A definitive diagnosis of hypoadrenocorticism should be made based on the results of which test(s)?

a. Basal cortisol
b. Electrolyte imbalance
c. Low dose dexamethasone suppression test
d. ACTH stimulation test

25. The intravenous fluid of choice for correcting dehydration in a patient with hypoadrenocorticism is

a. 0.9% saline
b. 0.18% saline with 4% glucose
c. Lactated ringers
d. hypertonic saline

26. Cats with pancreatitis may have low serum cobalamin concentrations because

a. ileal absorption is decreased due to ileus
b. there is a dietary deficiency due to anorexia
c. they lack the enzyme necessary for absorption
d. they have decreased intrinsic factor

27. Folate is absorbed primarily in the

a. stomach
b. proximal intestine
c. distal jejunum and ileum
d. colon

28. Differentiating feline low grade small cell lymphoma from lymphocytic inflammatory bowel disease

a. is based on the endoscopic appearance of the small intestine
b. is based on ultrasonographic measurement of intestinal wall thickness
c. is always possible based on cytology
d. can be difficult even with histopathology

29. In a dog suspected of having eaten Gorilla glue, which is the best treatment?

a. The initial treatment should be inducing emesis
b. The dog should be treated with prokinetic drugs
c. The dog should be treated with activated charcoal
d. The glue mass should be removed surgically

30. Basophilic stippling of red blood cells on a haematoxylin and eosin stained blood smear from a dog

a. can occur with any regenerative anaemia
b. is suggestive of possible lead toxicity
c. is suggestive of possible zinc toxicity
d. is suggestive of porto-systemic shunting

31. Microcytosis (small red blood cells) can be found in the blood of normal healthy dogs of which breed?

a. Akita
b. Beagle
c. Poodle
d. Rottweiler

32. If ingested and absorbed, lead may be stored in the

a. liver
b. bone
c. brain
d. prostate

33. The potentially most serious side effect of treating lead toxicity with calcium edentate is reversible

a. nephrotoxicity
b. liver failure

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c. retinal damage
d. osteoporosis

34. The most common gastric tumour in dogs is
a. lymphoma
b. leiomyosarcoma
c. adenocarcinoma
d. fibroma

35. The cat breed at highest risk for developing diabetes mellitus in the United Kingdom is the
a. Burmese
b. Persian
c. Siamese
d. Ragdoll

36. The most sensitive and specific test for diagnosis of pancreatitis in a cat which has been ill for a week is
a. lipase
b. amylase
c. trypsin-like immunoreactivity
d. feline pancreatic specific lipase

37. In cats, which is the most common sign of pancreatitis?
a. Anorexia
b. Vomiting
c. Diarrhoea
d. Fever

38. Compared to round (e.g. ball or rock) foreign bodies, linear intestinal foreign bodies
a. are less serious as they rarely cause obstruction
b. cause more electrolyte imbalance
c. are more likely to perforate the intestine
d. are usually located within the stomach

39. The urinary crystal or urolith caused by porto-systemic shunts is
a. calcium oxalate
b. cystine
c. struvite
d. urate

40. The ductus venosus usually closes
a. before birth
b. within a week or two after birth
c. at 4 months of age
d. at 9 months of age

41. A breed commonly recognized to have congenital extrahepatic porto-systemic shunts is the
a. Affenpinscher
b. Beagle
c. Chow
d. Yorkshire terrier

42. Diets with a high meat protein content are contraindicated for dogs with porto-systemic shunting due to the increased risk of
a. hepatic encephalopathy
b. vomiting
c. diarrhoea
d. polydipsia

43. A good source of supplemental protein for a growing puppy with a porto-systemic shunt is
a. cottage cheese
b. turkey
c. salmon
d. ham

44. Lactulose is used to treat patients with porto-systemic shunting because it
a. has an antibiotic effect in the colon
b. increases intestinal transit time
c. acidifies colonic contents thereby trapping ammonia
d. decreases bile acid concentration

45. A cat predisposed to hepatic lipidosis would be one which
a. is fat and then becomes anorexic
b. is thin and then becomes anorexic
c. is on a high fat diet
d. is on a high protein diet

46. Which of the following abnormalities on serum chemistry would indicate that a cat may have hepatic lipidosis rather than cholangitis?
a. Elevated AP and GGT
b. Decreased AP and GGT
c. Normal AP and increased GGT
d. Elevated AP with normal GGT

47. Regarding gastrointestinal bleeding, which of the following is most correct?
a. If the faeces colour is normal, there is no bleeding

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b. Dark faecal colour can only be caused by GI bleeding
c. Melaena in faeces is caused by oxidation of haemoglobin
d. Haematochezia is often caused by ingested blood

48. Acute haemorrhagic gastroenteritis usually differs from other forms of gastrointestinal bleeding in that
a. the PCV increases
b. the PCV decreases
c. the PCV is not affected
d. the albumin increases

49. Colonic vascular dysplasia is diagnosed
a. based on haematology and serum chemistry
b. by a typical appearance of the colon at colonoscopy
c. on the typical cytology appearance
d. on the typical histopathological appearance

50. Irritable bowel syndrome in dogs is diagnosed by the
a. ultrasonographic appearance of the colon
b. endoscopic appearance of the colon
c. histopathology of the colon
d. exclusion of other disorders and response to treatment

51. The most common colorectal tumours in the dog are
a. adenoma/adenocarcinoma
b. fibroma
c. lymphoma
d. sarcoma

52. In cats with *Tritrichomonas foetus*
a. diagnosis is made by routine faecal examination
b. diagnosis is made by zinc flotation of faeces
c. the organisms are too small to be seen by light microscopy
d. trophozoites can sometimes be seen in a fresh wet faecal preparations

53. Histiocytic colitis in Boxer dogs responds to
a. amoxicillin
b. enrofloxacin
c. clindamycin
d. metronidazole

54. What is the most common cause of EPI in cats?
a. Pancreatic acinar atrophy
b. Exocrine pancreatic hypoplasia
c. Chronic pancreatitis
d. Pancreatic neoplasia

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Multiple choice answers

1. **In cases suspected of having regurgitation,**
   c. the disorder is generally located in the oesophagus

2. **In a dog which is suspected of regurgitating, which of the following imaging procedures should be performed** first **to help confirm the diagnosis?**
   c. radiographs of the thorax

3. **Acquired myasthenia gravis in dogs is an**
   c. immune mediated disorder with antibodies against ACh receptors

4. **The prognosis for a dog diagnosed with acquired myasthenia gravis is**
   b. guarded as only about 50% of the dogs survive

5. **In cases of idiopathic megaoesophagus, the most common cause of death is**
   d. aspiration pneumonia

6. **Oesophageal strictures are usually best treated using**
   a. balloon dilation, with repeated procedures if needed

7. **In a regurgitating 4-month-old puppy, a barium swallow shows abrupt discontinuation of the barium just cranial to the base of the heart with gross dilatation of the oesophagus cranial to that area. The most likely diagnosis is:**
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8. **What is the prognosis for dogs with persistent right aortic arch which have surgery?**
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   a. may have an aetiology related to botulinum type C neurotoxin

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11. **Treatment of feline dysautonomia**
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18. **The 'organ of vomition' is the**
    c. duodenum

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19. The chemoreceptor trigger zone is in
a. the floor of the fourth ventricle

20. Endotoxic shock may occur in animals with intestinal obstruction with a foreign body due to the
c. increased numbers of bacteria within the stagnant loop of intestine

21. A gastrointestinal barium contrast study is performed in a 10-year-old dog that has been vomiting intermittently for four months. On a ventrodorsal view, the pylorus is positioned more medially than normal and there is a decreased amount of contrast seen in the duodenum. While not absolutely diagnostic, this pattern is most suggestive of
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d. urate

40. The ductus venosus usually closes
b. within a week or two after birth

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41. A breed commonly recognized to have congenital extrahepatic porto-systemic shunts is the
d. Yorkshire terrier

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a. hepatic encephalopathy

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b. by a typical appearance of the colon at colonoscopy

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d. exclusion of other disorders and response to treatment

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52. In cats with *Tritrichomonas foetus*
d. trophozoites can sometimes be seen in a fresh wet faecal preparation

53. Histiocytic colitis in Boxer dogs responds to
b. enrofloxacin

54. What is the most common cause of EPI in cats?
c. Chronic pancreatitis

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1 APPENDIX

Abbreviations list

AchR	anticholinesterase receptor
ACTH	adrenocorticotrophin hormone
Ad lib	ad libitum (i.e. free choice)
ALT	alanine aminotransferase
AP	alkaline phosphatase
APTT	activated partial thromboplastin time
ARD	antibiotic responsive diarrhoea
ARE	antibiotic responsive enteropathy
BCS	body condition score
BIPS	barium impregnated polyethylene spheres
bpm	beats per minute
CFU	colony forming units
CI	contraindicated
CK	creatinine kinase
CKD	chronic kidney disease
cPLI	canine pancreatic lipase immunoreactivity
CRT	capillary refill time
CRTZ	chemoreceptor trigger zone
CT	computed tomography
DEA	dog erythrocyte antigen
dL	decilitre
DSH	domestic short hair (cat)
EDTA	ethylenediaminetetra-acetic acid
FeLV	feline leukaemia virus
EPI	exocrine pancreatic insufficiency
FIP	feline infectious peritonitus
FIV	feline immunodeficiency virus
fPLI	feline pancreatic lipase immunoreactivity
g	gram
GABA	*gamma*-Aminobutyric acid
GGT	gamma (γ) glutamyltransferase
GI	gastrointestinal
H_2	histamine (type 2) (e.g. for receptors)
Hb	haemo globin
HL	hepatic lipidosis
KCl	potassium chloride
kg	kilogram
IBD	inflammatory bowel disease
L	lumbar, e.g. lumbar vertebra (other than in imaging pictures, where L refers to left)
LSA	lymphoma
MCHC	mean cell haemoglobin concentration
MCV	mean corpuscular volume
mg	milligram
min	minute
ml	millilitre
MRI	magnetic resonance imaging
NSAID	non-steroidal anti-inflammatory drug
ng/l	nanograms/litre
nRBC	nucleated red blood cell
oz	ounce
PCR	polymerase chain reaction
PCV	packed cell volume
PLE	protein losing enteropathy
PLI	pancreatic lipase immunoreactivity
po	per os
PRAA	persistent right aortic arch
PSS	porto-systemic shunt
PT	prothrombin time
pu/pd	polyuria/polydipsia
q	every (used for frequency of dosing of medications)
R	right
RER	resting energy requirement
SAMe	S-adenosyl methionine
sec	second
si	small intestine
SIBO	small intestinal bacterial overgrowth
T4	thyroxine
TLI	trypsin-like immunoreactivity
µg	microgram
UK	United Kingdom
µl	microlitre
USA	United States of America
WBC	white blood cell
wt	weight

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2 APPENDIX

Estimating the degree of dehydration

The degree of dehydration can be subjectively estimated based on a patient's mucous membrane dryness, skin turgor, the degree of sunken eyes and mentation.

Skin tenting is performed by lifting the skin on the torso into a fold and evaluating for the approximate time required for the skin to return to its normal position. Ideally, the same individual should perform this test each time using the same location on the patient's body to eliminate some of the subjectivity of the test. Skin tenting is affected by the amount of subcutaneous fat – very thin animals will tent more than fat ones.

Nauseous animals may be salivating and this can affect the detection of dry oral mucous membranes. Nervous or affectionate cats and some breeds of dogs may also salivate.

These estimations are rarely exact, so the patient should be evaluated frequently during iv fluid therapy to ensure adequate hydration and prevent overhydration.

When a patient's hydration is adequate the urine production should be >1–2 ml/kg/hour and the heart rate and respiratory rate should be within normal limits. The CRT should return to 1.5–2 seconds and the mucous membranes should be moist and pink (unless other conditions affect the membrane colour).

Hypoproteinaemic and oliguric/anuric patients are susceptible to overhydration and pulmonary oedema during intravenous fluid therapy. All patients receiving iv fluid therapy should be monitored for signs of overhydration (anxiety, tachycardia, tachypnoea, increasing body weight, clear nasal discharge and pulmonary crackles or harsh lung sounds).

Estimated degree of dehydration	Signs
<5%	No clinical abnormalities detectable, but possibly a history of vomiting/diarrhoea or decreased fluid intake
5–6%	Mild skin tenting (can vary with age and body condition) and ± dry mucous membranes
7–8%	Tenting of skin, prolonged capillary refill time (CRT), dry mucous membranes, slight enophthalmos
9–12%	Skin remains tented, dry mucous membranes, dry appearing eyes, rapid weak pulse, slow CRT, likely in ventral recumbency
13–15%	Severe life threatening dehydration, marked shock, lateral recumbency, may die

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3 APPENDIX

Urgent vs non-urgent gastrointestinal cases

It can be difficult to decide the appropriate intervention and when to perform surgery on gastrointestinal cases. The guidelines below are only guidelines and a decision must be made on each case individually. The lists are not inclusive of all disorders.

I. Regurgitation

A. Many cases of chronic regurgitation are treated medically without surgical treatment. These include:
   1. oesophagitis
   2. megaoesophagus, which can be idiopathic or due to an underlying cause
   3. oesophageal dysmotility
B. The regurgitation case needing the most urgent intervention is oesophageal obstruction with a foreign body. These may often be removed with endoscopic guidance, but may require urgent surgery in some cases.
C. Oesophageal strictures also require intervention, but again endoscopic guided balloon dilation is the preferred method.
D. Cases of regurgitation which will likely require surgery after stabilization include:
   1. hiatal hernia
   2. vascular ring anomalies
   3. some oesophageal diverticula

II. Vomiting

(may also have diarrhoea)

A. Many causes of vomiting can be treated without surgery and may potentially be worsened by anaesthesia and surgery. These include:
   1. acute and chronic pancreatitis (without abscess or biliary obstruction)
   2. acute or chronic gastroenteritis
   3. parvovirus infection
   4. parasites
   5. acute liver disease
   6. gastrointestinal ulceration (without perforation)
   7. toxin ingestion or dietary indiscretion
   8. adverse reactions to food
   9. colitis
   10. diabetic ketoacidosis
   11. uraemia
   12. hypoadrenocorticism
   13. vestibular disorders
B. Emergency surgery – critical cases that require immediate surgery include:
   1. gastric-dilatation volvulus
   2. intestinal volvulus
   3. acute peritonitis
   4. incarcerated bowel
   5. diaphragmatic hernia with stomach or strangulated intestine
   6. linear foreign bodies
   7. complete high intestinal foreign body obstruction.
C. Critical cases that require surgery as soon as they are stabilized:
   1. gastric dilation without volvulus
   2. gastric obstruction, e.g. pyloric stenosis or foreign body
   3. distal or partial intestinal obstruction
   4. obstructed or ruptured biliary tree
   5. pyometra
   6. pancreatic mass/abscess
   7. intussusception

III. Small intestinal diarrhoea

(see also vomiting cases above as many present with either or both clinical signs)

A. Cases which are treated medically include:
   1. enteritis
   2. inflammatory bowel disease
   3. food responsive disorders (adverse reactions to food)

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4. parasites
5. infectious diarrhoea
6. antibiotic responsive diarrhoea
7. parvovirus infection
8. parasites
9. acute liver disease
10. gastrointestinal ulceration (without perforation)
11. toxin ingestion or dietary indiscretion
12. diabetic ketoacidosis
13. uraemia
14. hypoadrenocorticism
15. vestibular disorders

B. Cases which may present with small intestinal diarrhoea that require urgent surgery include:
   1. any case with a perforated intestine
   2. mesenteric torsions
   3. intestinal foreign bodies (although usually present with vomiting)

C. Cases that require surgery as soon as they are stabilized include:
   1. intussusception
   2. intestinal masses/focal tumours

IV. Generally not urgent, but surgery often indicated for

1. some porto-systemic shunts

V. Colonic disorders

A. Cases which usually are not treated surgically:
   1. most cases of colitis
   2. constipation
   3. irritable bowel syndrome
   4. colonic vascular dysplasia if generalized
   5. non-resectable tumours

B. May require surgery as soon as the case is stabilized include:
   1. obstructive colitis (e.g. granulomatous)

C. Generally not urgent, but may require surgery:
   1. obstipation (may require colectomy)
   2. focal colonic tumours
   3. colonic vascular dysplasia if focal

VI. Exploratory celiotomy for diagnostic purposes

A. Exploratory surgery is necessary for biopsies for diagnosis of:
   1. some intestinal mural diseases (e.g. where mucosal biopsies obtained with an endoscope are not deep enough)
   2. small bowel lesions distal to the reach of the endoscope or if endoscopy is not available
   3. hepatopathies
   4. some pancreatic masses

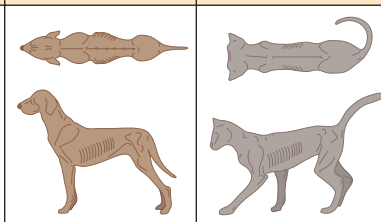
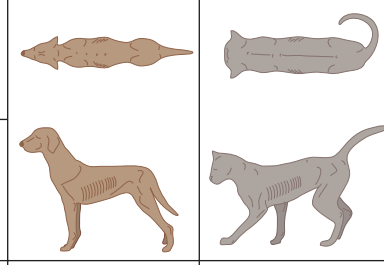
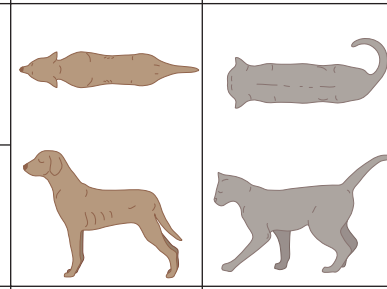
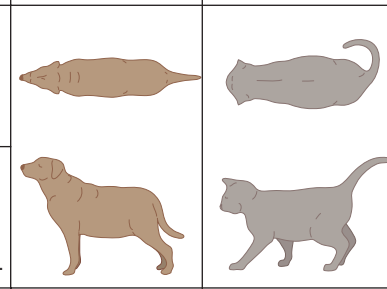
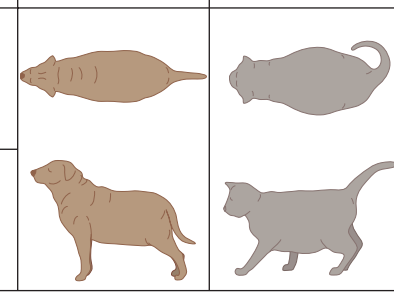
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4 APPENDIX

Body condition scores

A body condition score (BCS) is a subjective assessment of body composition. Several BCS systems have been proposed with 3, 5, 6 or 9 points – the 9-point system has been used in this book. The BCS system was developed and validated to provide a reasonable estimate of body fat. The 9-point BCS system was validated for dogs and cats using dual energy X-ray absorptiometry and was shown to provide a reasonable estimate of body fat. Using BCS charts and showing the owner how to palpate the ribs of their pet can be useful in convincing them of their pet's body condition. Ideal BCS for dogs is 4 to 5 depending upon breed and is 5 for cats. The BCS systems can be used to estimate percentage of body fat as in the table over leaf.

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9pt scale	% body fat	Body condition scoring	Dogs	Cats
1	≤5	**Emaciated.** Ribs and bony prominences are visible from a distance. No palpable body fat. Obvious abdominal tuck and loss of muscle mass.		
2	6 – 9	**Very thin.** Ribs and bony prominences visible. Minimal loss of muscle mass, no palpable fat.		
3	10 – 14	**Thin.** Ribs easily palpable, tops of lumbar vertebrae are visible. Obvious waist and abdominal tuck. Minimal fat covering.		
4	15 – 19	**Lean.** Ribs eaily palpable, waist visible from above. Abdominal tuck present. Abdominal fat pad is absent in cats.		
5	20 – 24	**Ideal.** Ribs palpable without excess fat covering. Waist and abdominal tuck present in dogs. Cats have a waist and a minimal fat pad.		
6	25 – 29	**Slightly overweight.** Ribs have slight excess fat covering. Waist is discernible from above, but not obvious. Abdominal tuck still present in dogs. Abdominal fat pad is apparent, but not obvious in cats.		
7	30 – 34	**Overweight.** Difficult to palpate ribs. **Dogs:** fat deposits over lumbar area and tail base. Abdominal tuck may be present, but waist is absent. **Cats:** moderate abdominal fat pad and rounding of the abdomen.		
8	35 – 39	**Obese.** Ribs not palpable and abdomen may be rounded. **Dogs:** heavy fat deposits over lumbar and base of tail. No abdominal tuck or waist. **Cats:** prominent abdominal fat pad and lumbar fat deposits.		
9	40 – 45+	**Morbidly obese.** **Dogs:** large fat deposits over thorax, tail base and spine with abdominal distension. **Cats:** heavy fat deposits over lumbar area, face and limbs. Large abdominal fat pad and rounded abdomen.		

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5 APPENDIX

Reference ranges for haematology and serum chemistry values for adult cats and dogs

The laboratory used for the cases in this book for haematology and serum chemistry was the University of Edinburgh Veterinary Pathology Unit although some emergency cases also had in-house laboratory parameters measured.

Parameter	Unit	Dog	Cat
Haematology			
RBC	$\times 10^{12}$/l	5.5–8.5	5.5–10.0
WBC	$\times 10^{9}$/l	6.0–15.0	7.0–20.0
Neutrophils (segmented)	$\times 10^{9}$/l	3.6–12.0	2.5–12.8
Neutrophils (bands)	$\times 10^{9}$/l	0–0	0–0
Lymphocytes	$\times 10^{9}$/l	0.7–4.8	1.5–7.0
Monocytes	$\times 10^{9}$/l	0.0–1.5	0.07–0.85
Eosinophils	$\times 10^{9}$/l	0.0–1.0	0.0–1.0
Basophils	$\times 10^{9}$/l	0.0–1.0	0.0–1.0
PCV	l/l	0.39–0.55	0.24–0.45
Hb	g/dl	12.0–18.0	8.0–14.0
MCV	fl	60–77	28–55
MCHC	%	32–36	30–36
Platelets	$\times 10^{9}$/l	200–500	300–600
Prothrombin time	sec	5–12	5–12
APTT	sec	10–20	10–20
Serum chemistry			
Albumin	g/l	26–35	28–39
Amylase	IU/l	225–990	525–960
Bile acids (fasting)	µmol/l	0–7	0–7
Bilirubin (total)	µmol/l	0–6.8	0–6.8
Chloride	mmol/l	99–115	117–140
Cholesterol	mmol/l	3.8–7.0	2.0–3.4
Cortisol	nmol/l	20–230	10–250
Creatinine	µmol/l	40–132	40–177
Fibrinogen	g/l	2.0–4.0	2.0–4.0
Fructosamine	µmol/l	49–225	100–350
Glucose	mmol/l	3.0–5.0	3.3–5.0
Lead (blood)	µg/ml	0.5–2.4	0–1.21
Lipase	IU/l	13–200	0–83
Total protein	g/l	58–73	69–79
Urea	mmol/l	1.7–7.4	2.8–9.8
Enzymes			
AP	IU/l	20–60	10–100
ALT	IU/l	21–102	6–83
AST	IU/l	15–65	15–50
CK	IU/l	50–200	50–200
GGT	IU/l	2–8	1–5
Minerals/trace elements			
Calcium	mmol/l	2.3–3.0	2.1–2.9
Magnesium	mmol/l	0.69–1.18	0.82–1.23
Inorganic phosphate	mmol/l	0.9–2.0	1.4–2.5
Sodium	mmol/l	139–154	145–156
Potassium	mmol/l	3.6–5.6	4.0–5.0
Vitamins (from Texas GI Lab)			
Cobalamin	ng/l	251–908	290–1500
Folate	µg/l	7.7–24.4	9.7–21.6

Abbreviations: ALT, alanine aminotransferase; AP, alkaline phosphatase; APTT, activated partial thromboplastin time; AST, aspartate aminotransferase; CK, creatine kinase; GGT, γ-glutamyl transferase; Hb, haemoglobin; MCHC, mean cell haemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume.

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LABORATORY VALUES FOR KITTENS AND PUPPIES

While few laboratories give separate reference ranges for kittens and puppies, there are some important differences that should be taken into consideration.

Haematology

The packed cell volume (PCV) of kittens and puppies is lower than that of adult cats and dogs. The PCV is lowest in kittens at around 3 to 4 weeks of age and puppies at around 4 to 5 weeks of age. The values increase to adult ranges when they are around 3–4 months old. The white blood cell count of puppies and kittens may be in the high adult range or slightly higher at 3 months of age and then should be within the adult reference range.

Serum chemistry

Serum inorganic phosphate and calcium are higher in young growing animals. Serum inorganic phosphate may be as high as 3.5 to 3.8 mmol/l and calcium values may be about 0.5 mmol/l higher than the top of the adult reference range.

Total serum alkaline phosphatase (AP) is often two to three times the adult reference range during growth due to the bone isoenzyme.

Serum total protein is usually below the adult reference range until about 4 to 5 months of age. Serum albumin concentration should reach adult concentrations by about 2 months of age. Serum globulin concentration increases more slowly.

The concentration of serum creatinine, which is derived from muscle tissue, is usually low in puppies and kittens. Fasted puppies and kittens may have lower blood urea nitrogen than adults, although diet can affect this parameter.

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6 APPENDIX

Resources for tests for gastrointestinal disorders

GI Laboratory

The GI Lab at Texas A&M University is an excellent resource, although some samples cannot be shipped successfully from overseas. Contact details are:

e-mail: gilab@cvm.tamu.edu
Shipping address:
Gastrointestinal Laboratory
4474 TAMU
College of Veterinary Medicine
Texas A&M University
College Station, TX 77843-4474
Tel: (979) 862 2861; Mon-Fri 8:00 AM to 5:00 PM (Central Time, USA)
Fax: (979) 862 2864

Tests available:

- Serum Trypsin-Like Immunoreactivity (TLI): For exocrine pancreatic function testing (i.e. to diagnose EPI)
- Serum Cobalamin (Vitamin B_{12}) and Folate: Test of small intestinal function (not species specific)
- Pancreatic Lipase Immunoreactivity (PLI): Test for diagnosis of pancreatitis in dogs and cats (species specific)
- Faecal Alpha$_1$-Proteinase Inhibitor: Test for protein-losing enteropathy – dogs and cats
- Serum Bile Acids: Test for hepatic function – dogs, cats and other species
- Canine C-reactive Protein: Test for systemic inflammation – dogs only
- *Tritrichomonas foetus*: PCR testing
- *Campylobacter* spp.: PCR test for differentiation and species specific identification of *Campylobacter jejuni*, *C. upsaliensis*, *C. coli*, *C. helveticus*
- Canine schistosomiasis: PCR test for *Heterobilharzia americana*
- Enterotoxigenic *Clostridium perfringens*: PCR test for the gene encoding *C. perfringens* enterotoxin
- Thyroid hormones and cortisols: Total T4, TSH, free T4 by equilibrium dialysis, cortisols
- Serum gastrin

IDEXX Laboratories

IDEXX, as well as a full service veterinary laboratory, IDEXX offers the Spec cPL and Spec fPL for quantitative specific pancreatic lipase in dogs and cats for diagnosis of pancreatitis. They also have a range of in house tests available, e.g. SNAP tests for Giardia and Parvovirus. Their contact details in the UK are:

IDEXX Laboratories Ltd
Milton Court
Churchfield Road
Chalfont St Peter
Buckinghamshire SL9 9EW
United Kingdom
Tel: +44 (0)175 389 1660
Fax: +44 (0)175 389 1520

Myasthenia gravis

The 'gold standard' for the diagnosis of acquired MG in both dogs and cats is the documentation of antibodies against Ach receptors by immunoprecipitation radioimmunoassay. This test is available through the Comparative Neuromuscular Laboratory, University of California, San Diego; Available at: http://medicine.ucsd.edu/vet_neuromuscular; E-mail: musclelab@ucsd.edu).

Tritrichomonas foetus

In the USA, samples can be submitted to the College of Veterinary Medicine, North Caroline State University for this test. Information on this is available at: www.cvm.ncsu.edu/mbs/gookin_jody.htm

In the UK the PCR test is available from Capital Diagnostics in Edinburgh (+44 (0)131 535 3145).

The organism can also be cultured from faecal samples using a system developed for diagnosis in cattle. The 'In Pouch tm TF' test (BioMed Diagnostics, Oregon, USA) uses a liquid culture system in a sterile plastic pouch. The pouch can be inoculated with 0.05 g of faeces (about half the size of a small pea). The pouches are incubated at room temperature and can be exam-

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ined microscopically for the motile organisms every 2 days for 12 days. This test is more sensitive than direct examination of faeces and helpful for detecting infections where direct smears are negative. *Giardia* and other similar organisms will not grow in this specific culture medium. In the UK, this system is available from Capital Diagnostics in Edinburgh (+44 (0)131 535 3145).

Most tests in this book were run at the:
Veterinary Pathology Unit
Easter Bush
Midlothian EH25 9RG
United Kingdom
Tel: +44 (0)131 650 6403
E-mail: vpu.enquiries@ed.ac.uk

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7 APPENDIX

Obtaining cisapride

Since withdrawal of cisapride from the human market, it has become increasingly difficult, but not impossible to obtain this drug. The drug may still be imported from abroad, on a named patient basis as follows:

- Obtain a Special Treatment Certificate (STC) from the Veterinary Medicines Directorate
  - A written application must be made in the first instance to the Veterinary Medicines Directorate, Woodham Lane, New Haw, Addlestone, Surrey, KT15 3LS (01932 336911).
  - Repeat applications can be made online
  - Further information available at: www.vmd.gov.uk
- The drug can then be imported via IDIS Ltd, IDIS House, Churchfield Road, Weybridge, Surrey, KT13 8DB; Available at: www.idispharma.com
- The brand name is Gasprid and it comes as 10 mg tablets made by a Polish company Teva Polska. Polfa Kutno, ul. Sienkiewicza 25, 99-300 Kutno, Tel: +48 24 355 01 00, Fax: +48 24 355 03 50; Available at: www.teva.pl/u235/navi/90
- The 10 mg tablets may be reformulated into a more cat-friendly size (e.g. 2.5 mg capsules) by Nova Laboratories Ltd, Martin House, Gloucester Crescent, Wigston, Leicester LE18 4YL; Available at: www.novalabs.co.uk
- Cisapride suspension has a shelf life of 28 days.
- If a European source of drug is no longer available, then it may be possible to import from veterinary pharmacies in the USA (e.g. www.vetrxdirect.com; www.diamondbackdrugs.com), but currently European sources must be used in the first instance.

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8 APPENDIX

Canine inflammatory bowel disease activity index (CIBDAI)

An index to estimate the severity of inflammatory bowel disease (IBD) in dogs has been developed to help in the management of patients and to aid in collaborative multicentre studies. An index for feline IBD is also being developed. The current criteria for assessment of canine inflammatory bowel disease activity index are below:

A. attitude/activity
B. appetite
C. vomiting
D. stool consistency
E. stool frequency
F. weight loss

These six variables are scored 0 to 3:

0 = normal
1 = mild change
2 = moderate change
3 = severe change

The summary of these six variables gives the total composite CIBDAI score, which is interpreted as:

0–3 = clinically insignificant disease
4–5 = mild IBD
6–8 = moderate IBD
9 or greater = severe IBD

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9 APPENDIX

Feeding tubes

Feeding tube	Advantages	Disadvantages
Naso-oesophageal 	Easy to place Minimal equipment Not contraindicated with coagulopathy	Contraindicated with nasal, facial, oesophageal disease or frequent vomiting Tubes easily become blocked Short-term (3–5 days)
Oesophagostomy 	Larger bore, therefore feeding easier Moderately easy to place Can be used for >1 week	Requires general anaesthetic Contraindicated with coagulopathy, vomiting, oesophageal disease
Gastrostomy (peg) 	Larger bore, therefore feeding easier Not likely to become dislodged with vomiting Medications can be given through the tube	Requires general anaesthetic Contraindicated with coagulopathy Higher level of skill and equipment Must stay *in situ* a minimum of 7 days

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10 APPENDIX

Medications used in the treatment of gastrointestinal disorders

These lists of medications are not complete and while sometimes or often used, many of the medications are not licensed for use in cats and dogs.

Medication	Dose	Comments
Anti-emetics (check for obstruction before using as they may mask signs)		
Metoclopramide	1–2 mg/kg/24 hr CRI or 0.2–0.5 mg/kg im, sq or po	D_2 dopaminergic antagonist. Upper GI prokinetic, short half life when given orally or sq
Domperidone	0.1–0.3 mg/kg im, iv q 12 hr or 1–2 mg/animal po	D_2 dopaminergic antagonist Not commonly used
Maropitant	0.5–1.0 mg/kg q 24 hr sq or 2 mg/kg po q 24 hr	No known prokinetic effect. Blocks NK–1 receptors in the medullary vomiting centre
Chlorpromazine	0.2–0.4 mg/kg sq q 8 hr	Alpha2 agonist. Affects CRTZ and emetic centre
Ondansetron	0.5–1.0 mg/kg po q 12–24 hr	Affects CRTZ, vagal afferent neurones
Cisapride	*Dogs*: As a promotility agent: 0.5 mg/kg q 8 hr For oesophagitis: 0.25 mg/kg po q 8–12 hr *Cats*: For chronic constipation (e.g. megacolon): In combination with a stool softener (e.g. lactulose) 0.1–1 mg/kg q 8–12 hr po or 5 mg per cat (total dose) po q 8–12 hr	Not a true anti-emetic, but promotility effect may decrease vomiting by enhancing gastric emptying and intestinal transit. Also used for reflux oesophagitis and constipation/megacolon (cats) No longer commercially available, must be obtained from a compounding pharmacy or imported Contraindications: hypersensitivity, GI perforation/obstruction, haemorrhage
Drugs for diarrhoea		
Diphenoxylate	0.1–0.2 mg/kg po q 8 hr	Narcotic analgesic
Loperamide	0.1–0.2 mg/kg po q 8 hr	Narcotic analgesic
Antibiotics sometimes used for bacteria induced diarrhoea and other GI disorders		
Ampicillin	10–20 mg/kg iv q 6–8 hr, sq, po	Anaerobic infections
Cephalathin	22–44 mg/kg q po 6–8 hr	Gram-negative organisms
Clarithromycin	4–12 mg/kg po q 12 hr	As part of treatment for *Helicobacter* spp.

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Medication	Dose	Comments
Clavulanate-potentiated amoxicillin	8.75 mg/kg iv q 8 hr 8.75 mg/kg im or sc of 24 hr 12.5 mg/kg po q 8–12 hr	As part of treatment for *Helicobacter* spp.
Enrofloxacin	3–5 mg/kg po q 12 hr	Gram-negative organisms and Boxer histiocytic colitis. Caution with use in cats due to potential blindness.
Erythromycin	10 mg/kg po q 8 hr	*Campylobacter* only, may cause diarrhoea
Erythromycin	0.1–50 mg/kg po q 8 hr	Low dose to promote fasting motility, mimics motilin in dogs
Gentamicin	2.2 mg/kg sc, iv, q 8 hr	For sepsis only, nephrotoxic, ototoxic
Tylosin	10–20 mg/kg po q 12 hr	Aids in some cases of colitis/enteritis
Metronidazole	7–15 mg/kg po q 8–12 hr	IBD, anaerobic bacteria, *Giardia*. Teratogenic and may be carcinogenic.
Immunosuppressive or anti–inflammatory drugs		
Prednisolone	0.5–3 mg/kg po q 12–48 hr depending upon condition	Taper higher doses over several months (e.g. 20% decrease q 2–3 weeks)
Ciclosporin	Dogs (older than 6 months): 5 mg/kg po q 12 –24 hr	Contraindicated or caution with drugs that inhibit cytochrome P450 enzymes, e.g. cimetidine. CI with hyperkalaemia Potential nephrotoxicity, especially if used with other potentially nephrotoxic drugs
Azathioprine	2 mg/kg po q 24 hr until remission then 0.5–2 mg/kg po q 48 hr	Bone marrow suppression is the most serious potential adverse reaction, monitor haematology
Olsalazine	10–20 mg/kg po q 12 hr	Local colonic NSAIDs. Less risk of KCS cf sulfasalazine
Sulfasalazine	*Dogs*: 15–30 mg/kg po q 8–12 hr (max 6 g/day) *Cats*: 1–20 mg/kg po q 8–12 hr	Local NSAIDs. Risk of KCS
H_2 receptor blockers (used to decrease gastric acidity)		
Cimetidine	*Dogs*: 5–10 mg/kg iv, im, po q 8 hr *Cats*: 2.5–5.0 mg/kg iv, im, po q 12 hr	Slows metabolism of other drugs by binding to hepatic microsomal cytochrome P450 and may increase their plasma concentrations. No known promotility effect
Ranitidine	2 mg/kg sc, iv or slow iv 8–12 hr	Some prokinetic effect via muscarinic AchR
Famotidine	0.5–1.0 mg/kg po q 12–24 hr	Does not have the interaction of cimetidine, may or may not have prokinetic effects
Cytoprotective drugs		
Sucralfate	*Dogs*: 500 mg/dog po q 6–8 hr for dogs up to 20 kg, 1–2 g/dog po q 6–8 hr for dogs >20 kg *Cats*: 250 mg/cat po q 8–12 hr	Aluminium ion binds to proteinaceous exudates forming a barrier over ulcerated areas. Promotes bicarbonate and prostaglandin E production which aids mucosal defense and repair. May decrease bioavailability of other drugs, especially tetracyclines
Misoprostol	*Dogs*: 2–7.5 µg/kg po q 8–12 hr *Cats*: 5 µg/kg po q 8 hr	Aids in protection of the GI ulcers due to NSAIDs. Side effects include diarrhoea, vomiting, abortion

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11 APPENDIX

Medications used in the treatment of feline hepatic lipidosis

Medication	Dose	Comments
0.9% NaCl	Depends on degree of dehydration, concurrent cardiac disease, etc	Lactate-containing fluids (e.g. Hartmann's) should be avoided, as lactate requires hepatic metabolism
Potassium chloride	Depends on degree of hypokalaemia.	Hypokalaemia may develop with re-feeding. Monitor daily for first 72 hr. Do not exceed 0.5 mmol/kg/hr
Potassium phosphate	0.01–0.06 mmol/kg/hr Give as constant rate infusion in NaCl	Hypophosphataemia may develop with re-feeding. Supplementation indicated if <0.7mmol/l. Re-evaluate after 6 hr. Will precipitate with Ca-containing fluids
Magnesium sulphate	0.3–0.5 mmol/kg/day Give as constant rate infusion in NaCl	Hypomagnesaemia may develop with re-feeding and may contribute to hypokalaemia. Monitor q 12–24 hr. Will precipitate with calcium containing fluids
Thiamine (Vitamin B_1)	50–100 mg/cat/day	Anaphylaxis may be seen with injectable preparations – use oral instead
Cobalamin (Vitamin B_{12})	1 mg/cat sq q 7–14 days	Liver cobalamin stores may be depleted in the face of normal serum cobalamin
Multivitamin preparations	1–2 ml per 500 ml bag of fluids	Protect from light once added to bag
Vitamin K_1	0.5–1.0 mg/kg q 12 hr for 3 doses	Give orally or by subcutaneous injection using a 25 G needle. Vitamin K required for clotting factors II, VII, IX and X
Maropitant	0.5–1.0 mg/kg q 24 hr	Not licensed for use in cats
Metoclopramide	1 mg/kg/24 hr Constant rate infusion	Central anti-emetic effect in cats questionable due to lack of dopaminergic receptors, but peripheral prokinetic effects
Ranitidine	2 mg/kg q 8–12 hr	Not licensed for use in cats, but preferred over cimetidine due to prokinetic effect
Vitamin E	10 IU/kg/day	Antioxidant often depleted with anorexia or malabsorption
L-carnitine	250–500 mg/cat/day	Promotes utilization of fatty acids by mitochondria
Taurine	250–500 mg/cat/day	High dietary requirement, as used for bile acid conjugation
s-adenosyl methionine	20–40 mg/kg/day 90 mg/cat/day	Higher dose required if enteric coated tablets are crushed for administration via feeding tube. Precursor for L-carnitine and glutathione; antioxidant and hepatoprotective properties
N-acetyl cysteine	140 mg/kg; dilute 1:4 with NaCl and give over 20 min	Subsequent doses may be given after 8–12 hr at 70 mg/kg. Precursor for glutathione

Abbreviations: AchR, anticholinesterase receptor; Ca, calcium; cf, compared to; CI, contraindicated; CRI, constant rate infusion; CRTZ, chemoreceptor trigger zone; g, gram; ga, gauge; GI, gastrointestinal; hr, hour; iv, intravenous; KCS, keratoconjunctivitis sicca; kg, kilogram; mg, milligram; min, minute; NaCl, sodium chloride; NSAIDs, non-steroidal anti-inflammatory drugs; po, per os; sc, subcutaneously; µg, microgram.

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Recommended reading

Swallowing and regurgitation

Jergens AE, 2010. Diseases of the esophagus, In: Ettinger SJ, Feldman EC (eds), Textbook of Veterinary Internal Medicine, 7th edn. Elsevier Saunders, St. Louis, MO, p. 1487-1499.

Lecoindrea P, Cadore JL, 1994. Disorders of the oesophagus in domestic carnivores. Pratique Médicale et Chirurgicale de l'Animal de Compagnie 29(1):25-43.

Strombeck DR, Guilford WG, 1996. Pharynx and esophagus: normal structure and function, In: Guilford WG, Center SA, Strombeck DR, Williams DA, Meyer DJ (eds), Strombeck's Small Animal Gastroenterology, 3rd edn. WB Saunders, Philadelphia, PA, p. 202-210.

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