

OESOPHAGEAL DISEASE: CORRECT INTERPRETATION OF CLINICAL SIGNS

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DAVID WALKER provides an outline to diagnosis of several presentations of oesophageal disease and discusses treatment and management

Summary

This article outlines the approach to the patient with suspected oesophageal disease. Regurgitation is frequently misinterpreted as vomiting, and it is essential to identify the clinical signs of oesophageal disease to ensure the appropriate diagnostic path is followed. The diagnosis and management of some of the more common oesophageal diseases is discussed.

Key words

oesophagus, oesophagitis, regurgitation, stricture, foreign bodies, hiatal hernia, neoplasia, megaoesophagus

OESOPHAGEAL disease is not seen as commonly as gastrointestinal disease; however, correct interpretation of the animal's clinical signs is essential to guide further diagnostics.

Regurgitation is often mistaken for vomiting, and a thorough history is essential. [Table 1](#) outlines the signs associated with vomiting and regurgitation. It is important to remember that some animals can be both regurgitating and vomiting at different times (for example, inflammatory bowel disease and secondary oesophagitis).

The animal's signalment can sometimes be helpful when considering the differential diagnoses for oesophageal disease ([Table 2](#)), for example, vascular ring anomalies are often seen in young animals. It should be determined when questioning the owner how quickly the clinical signs appeared, as this may also help to order the differential diagnosis list.

Acute onset clinical signs are often seen with oesophageal foreign bodies and acute oesophagitis, while the clinical signs are normally longer standing in animals with megaesophagus and neoplasia. Animals with hiatal hernias and reflux oesophagitis often have intermittent clinical signs. Recent anaesthesia should increase the index of suspicion for oesophagitis, while concurrent neurological signs may increase the index of suspicion for secondary megaesophagus.

Clinical signs of oesophageal disease include regurgitation, hypersalivation, pain on eating and anorexia. Aspiration pneumonia is a common sequel to oesophageal disease as the airway is unprotected during regurgitation and thoracic radiography is indicated in all patients with suspected oesophageal disease. Diagnostic investigation of suspected oesophageal disease may include blood work, plain and contrast radiography and oesophagoscopy. Oesophagoscopy is the only means of diagnosing some conditions, such as oesophagitis, but is normally unhelpful in others, such as megaesophagus.

A detailed physical examination should be performed in every patient with oesophageal disease and should include oesophageal palpation and respiratory assessment, due to the possibility of aspiration pneumonia. A complete neurological examination should be performed. Body condition may be reduced as a consequence of aspiration pneumonia, or reduced calorie intake (despite a ravenous appetite) due to severe regurgitation or pain.

Oesophagitis

Oesophageal inflammation can be acute or chronic and often results in erosion and/ or ulceration. [Table 3](#) outlines common causes of oesophagitis. Oesophagitis causes muscular weakness, a secondary motility disorder and regurgitation. It can only be definitively diagnosed by endoscopic examination.

Treatment of oesophagitis involves the following.

- Small, high-protein, lowfat meals to minimise reflux. Very unwell patients are likely to require hospitalisation and intravenous fluid therapy.
- Oral sucralfate suspension to act as a chemical bandage binding to erosions and ulcers. It may stimulate mucosal defence and reparative mechanisms.
- Omeprazole. This proton pump inhibitor is preferred over H₂ antagonists (ranitidine, cimetidine and famotidine) by many clinicians, under the cascade. Omeprazole inhibits gastric acid production

by any secretagogue (histamine, acetylcholine and gastrin). This is in contrast to H2 antagonists that only suppress gastric acid production stimulated by histamine. The duration of drug therapy is empiric. The author treats moderate to severe oesophagitis for three to four weeks.

- Broad-spectrum antibiotics may be required if aspiration pneumonia is present. Pending culture and sensitivity results on a bronchoalveolar lavage (performed at the same time as endoscopy), clavulanatepotentiated amoxicillin would be appropriate. This has a good spectrum of activity against most gram-positive organisms and penicillinase producing *Staphylococcus* species. It also has some activity against gram-negative organisms.
- A percutaneous endoscopic gastrostomy tube may be required in some severely affected patients that cannot maintain their calorie intake.
- Prokinetic agents (metoclopramide) may be advocated in oesophagitis secondary to gastroesophageal reflux.

Severe, long-standing inflammation can result in an oesophageal stricture or ulceration and oesophageal perforation.

Oesophageal strictures

Oesophageal strictures are an abnormal circumferential narrowing of the oesophageal lumen that occur secondary to severe oesophagitis. They are the result of deep wall injury and healing by fibrosis.

Contrast radiography may reveal narrowing of the oesophageal lumen and oesophageal dilation proximal to the stricture site. Endoscopy should be performed in all animals to confirm the site and severity of the stricture. Oesophagitis has generally resolved by the time the animal is showing clinical signs secondary to the stricture.

Oesophageal strictures can be dilated with a balloon catheter or bougie. Bougies are flexible rubber tubes of increasing diameter that are introduced through the stricture to try to achieve dilation. Balloon catheter dilation is thought to be preferable to bougienage, as there is less chance of perforation and a longer clinical response between dilations¹. Multiple dilation procedures are often required and it may be preferable to plan several less aggressive dilation procedures (causing minimal trauma each time) rather than one very aggressive procedure. It is unrealistic to expect the oesophageal lumen to return to a normal diameter, but there should be a significant improvement in lumen size. If after a minimum of two procedures significant difference has been made, then repeat procedures can be based on clinical signs. The goal at the outset should be for the animal to return to its old diet; however, in some animals that have multiple procedures the goalposts end up moving.

There is a risk of perforation and subsequent pneumothorax with oesophageal stricture dilation, and the equipment necessary to perform thoracocentesis should always be at hand. Thoracic radiography should be performed if the procedure was very difficult or there is any degree of respiratory compromise. Oesophageal perforation generally requires thoracotomy and the owners should be warned of this risk prior to any dilation procedure. Postprocedure oesophagitis should be managed aggressively, as outlined previously. Additionally, an anti-inflammatory dose of prednisolone is often used. Glucocorticoids are thought to help retard fibrous connective tissue proliferation, but their effectiveness is uncertain.

Foreign bodies

Oesophageal foreign bodies are usually bones ([Figure 1](#)). They usually lodge at the thoracic inlet, heart base or hiatus. Plain radiographs are normally sufficient for diagnosis. Oesophageal perforation may occur after ingestion of a foreign object, and the presence of pleural effusion, pneumomediastinum and pneumothorax on a thoracic radiograph may suggest perforation.

Oesophageal foreign bodies are an emergency and should be removed as soon as the patient is stable, to reduce the risk of perforation. They can be removed with the help of endoscopy or fluoroscopy. As a last resort, foreign bodies are sometimes pushed into the stomach. Endoscopy has the advantage that the oesophageal mucosa can be assessed for damage. Large, rigid grasping forceps are passed alongside the endoscope and the foreign body is firmly grasped. Care must be taken not to grasp the oesophageal wall. The foreign body is gently manipulated and, if movement is evident, it is withdrawn. If a bone foreign body has to be manipulated into the stomach, it will be rapidly digested and gastrotomy is not needed. Remember that the aborad (closer to the stomach) side of the foreign body may be sharp and could damage the mucosa. Oesophagotomy is required in rare cases when the foreign body cannot be removed endoscopically.

Fish hooks ([Figure 2](#)) are sometimes challenging. It is worth checking the tongue for fishing line and it is useful not to cut the line, as it can be helpful during manipulation. Fish hooks normally need to be pushed aborally to disengage them from the oesophageal wall. A small hole is left, but there are rarely any complications. The point of the hook needs to face aborally for removal.

Radiographs should be taken after the procedure and secondary oesophagitis is managed as previously outlined. The major complications with foreign body removal are perforation and stricture formation.

Megaoesophagus

Megaoesophagus refers to a large, dilated oesophagus with severely impaired motility. It is mostly an acquired disease process, but congenital megaoesophagus can be seen.

Acquired megaesophagus generally occurs in middle-aged to older dogs, particularly large breeds. An underlying cause is often not identified (idiopathic disease). However, megaesophagus can occur secondary to several disease processes including, but not limited to, myasthenia gravis (generalised or focal), thymoma, polyneuropathy, polymyopathy and hypoadrenocorticism. The pathophysiologic mechanism for idiopathic megaesophagus is unknown, but a defect in the afferent neural pathway is suspected².

Diagnosis is normally made with survey radiography. This must be performed without sedation or anaesthesia, as both can induce oesophageal dilation. Aspiration pneumonia is commonly seen in association with megaesophagus. Diagnostic tests should try to identify an underlying cause and these may include complete blood cell count, serum biochemistry, urinalysis, thorough neurological examination, an adrenocorticotrophic hormone stimulation test and an acetylcholine receptor antibody titre. Oesophagoscopy is rarely required to diagnose megaesophagus except when obstructive disease or oesophagitis is suspected.

Treatment is aimed at managing the underlying disease process if one is identified. Treatment of idiopathic megaesophagus is supportive. Small, frequent meals should be fed from an elevated position and the patient would ideally be held vertical for 10 to 15 minutes post-feeding. Some patients will regurgitate less with solid food, while others tolerate gruel better. Manage secondary aspiration pneumonia with broad-spectrum antibiotics as outlined in the management of oesophagitis.

Oesophageal dysmotility can occur without megaesophagus as a consequence of some of the disease processes listed above. Fluoroscopy is required to make a definitive diagnosis. Temporary oesophageal dysmotility has also been identified in young terrier dogs, and affected animals may be symptomatic or asymptomatic³.

Neoplasia

Oesophageal neoplasia is rare. Tumours may be of oesophageal, perioesophageal or metastatic origin. Common primary malignant tumours include fibrosarcoma, leiomyosarcoma and osteosarcoma in dogs ([Figure 3](#)) and squamous cell carcinoma in cats. Benign tumours (papillomas and leiomyomas) have also been reported.

Initial diagnosis is based on the results of survey radiographs, contrast studies and endoscopy. Oesophageal masses are often submucosal and are covered in normal-looking mucosa. The final diagnosis requires histologic examination of the suspected mass, but this is not always straight forward. The oesophagus is difficult to biopsy endoscopically and cytology brushing is sometimes useful. Surgery is often required to obtain a definitive diagnosis.

Vascular ring anomaly

Vascular ring anomalies are caused by congenital arterial malformations (most often due to a persistent right aortic arch) that entrap the oesophagus, causing obstruction. Clinical signs are often seen around weaning and, radiographically, oesophageal dilation cranial to the heart base is seen. Prompt surgical intervention is needed before chronic dilation leads to irreversible oesophageal hypomotility ([Figures 4](#) and [5](#)).

Hiatal hernia

A hiatal hernia is a protrusion of the oesophagus, lower oesophageal sphincter and/ or part of stomach through the oesophageal hiatus into the thoracic cavity. Intermittent, sliding hiatal hernias are most common. These cause cranial displacement of the distal oesophagus and stomach into the mediastinum through the oesophageal hiatus of the diaphragm. Sometimes, hiatal hernias are easy to diagnose but they are occasionally a real challenge. They are probably not always clinically significant.

Hiatal hernias can be seen congenitally in a number of breeds and can occur as a consequence of altered intrathoracic pressures in brachycephalic airway obstruction syndrome. Clinical signs are normally the result of secondary reflux oesophagitis. Hiatal hernias are diagnosed on the basis of plain radiography, contrast radiography or fluoroscopy. Applying pressure to the cranial abdomen during fluoroscopic imaging can sometimes help. They may be identified endoscopically, but other imaging modalities are preferred as they can be easily missed.

Medical management for oesophagitis can initially be attempted and will lead to an improvement in some animals. Reconstructive surgery is necessary in those animals that develop recurrent oesophagitis.

References and further reading

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