Proceedings of the
World Small Animal Veterinary Association
Sydney, Australia – 2007

Hosted by:

Australian Small Animal Veterinary Association (ASAVA)

Australian Small Animal Veterinary Association (ASAVA)

Australian Small Animal Veterinary Association (ASAVA)

Next WSAVA Congress

ESOPHAGEAL DISEASES
DIAGNOSTIC AND THERAPEUTIC APPROACH

Thomas Spillmann, Dipl. vet. med., Dr. med. vet.
Professor of Small Animal Internal Medicine
Department of Equine and Small Animal Medicine
Faculty of Veterinary Medicine
University of Helsinki, Finland
PO 57 (Viikintie 49)
00014 Helsinki University / Finland

Introduction

Esophageal diseases show a limited amount of clinical signs and are therefore relatively strait forward to diagnose. Main symptom is regurgitation which has to be differentiated from dysphagia and vomiting by taking a thorough history. However, in cases with severe morphological changes in the cranial esophagus patients can also show painful dysphagia. Severe esophagitis with or without gastritis can cause both vomiting and regurgitation.

Further diagnostic efforts aim at the differentiation of primary esophageal diseases from secondary involvement of the esophagus in other diseases processes. Intraluminal esophageal diseases are foreign bodies and gastroesophageal reflux. Intramural esophageal disorders include megaesophagus, diverticle, esophagitis, stricture, gastroesophageal intussusception, granuloma, tracheoesophageal fistula, and tumors. Extraluminal narrowing of the esophageal lumen can be caused by vascular ring anomalies, hiatal hernia and mediastinal masses.

General diagnostic approach

The diagnostic workup starts with a thorough physical examination of oral cavity, ventral neck region and auscultation of the thorax in order to search for oropharyngeal disorders, dilation or mass effects in the neck part of the esophagus and signs of aspiration pneumonia as a feared complication of esophageal diseases.

Plain and contrast radiographs are the next necessary step to reveal esophageal foreign bodies, dilation and strictures, as well as thoracic masses, hiatal and diaphragmatic hernia, and gastroesophageal intussusception. Aspiration pneumonia, pleural effusions, and pneumothorax as serious complications of esophageal diseases can also be found. Radiographic contrast imaging is indicated, when plain radiography did not give enough proof for an esophageal disorder. Barium sulfate or iodine containing contrast media are used. Latter especially when esophagel perforation or tracheoesophageal fistula is suspected. In case of esophageal strictures fluid contrast media might show no abnormalities. Prestenotic dilation caused by intramural or extramural strictures can be visualized by mixing food with the contrast medium. With this procedure it has to be taken care, that the patient does not aspirate the food-contrast medium mixture when positioned on the X-ray table for radiography.

Endoscopic examination of the esophagus is the next step and the most diagnostic tool examining for esophageal diseases. It allows visualization of intraluminal disorders, gastroesophageal reflux, esophagitis, strictures, diverticles, granuloma, and neoplasia.

In case of pathologic findings it can be combined with sampling of tissue biopsies. The mucosa of the healthy esophagus is very difficult to biopsy but in case of inflammation or tumor sampling is easier to perform.

Limitation of endoscopy is the assessment of esophageal function especially in dogs with suspected megaesophagus. Deep anesthesia can cause a relaxation of the striated esophageal muscles and therefore be misinterpreted as dilation.

Thoracic or transesophageal ultrasound is indicated when intrathoracic or mediastinal masses compress the esophagus and shall be closer evaluated.

Laboratory tests including complete blood cell count and clinical chemistry are used to assess the severity of possible systemic complications and the possible risks of anesthesia. Special laboratory tests are indicated when ruling out different causes of canine megaesophagus.

General treatment recommendations

The aims of symptomatic treatment are maintenance of energy and fluid requirements of the patient by ensuring adequate food and water supply. If oral intake is not possible for a limited time or dehydration occurred percutaneous gastric tube feeding and intravenous fluid administration can be indicated.

Further attempts are needed to avoid or treat secondary aspiration pneumonia. General feeding principle of patients with esophageal disease is to offer food and water in small portions several times per day out of an elevated position in order to decrease the esophageal passage time and the amount of food and fluid that remains in the esophagus between the feedings.

Medical treatment such as motility modifiers, antibiotics and drugs that block gastric acid secretion are mentioned in the following paragraphs about specific primary esophageal diseases.

Specific primary esophageal diseases

Canine megaesophagus

Megaesophagus is mainly a canine disease and in most cases primary idiopathic. Idiopathic megaesophagus can be congenital with higher prevalence in young dogs of certain breeds such as miniature schnauzer, fox terrier, German shepherd dog, Great Dane, German pointer, rottweiler, bobtail, and Irish setter. Acquired idiopathic megaesophagus occurs in adult dogs and is not breed associated. Defects in the afferent nerve reaction to food stimulated dilation of the esophagus and disturbances of the enteric

nervous system within the esophageal wall are seen to be of pathophysiological importance.

The diagnosis of idiopathic megaesophagus can only be made by exclusion of causes for secondary megaesophagus. In puppies, diseases that have to be ruled out are primary myasthenia gravis, polyneuropathy, hiatal hernia (congenital, acquired), and storage diseases (glycogen, lipid). In adult dogs, secondary megaesophagus can be caused by acquired (local) myasthenia gravis, polymyositis, polymyopathy, polyneuropathy, endocrine disorders (hypoadrenocorticism, hypothyroidism [?]), intoxications (lead, thallium, organophosphates), mechanical obstructions (gastric dilation / volvulus, hiatal hernia, tumor at the lower esophageal sphincter), esophagitis, infections (distemper, tetanus), systemic lupus erythematodes, dermatomyositis, and thymom with secondary myasthenia gravis.

The management of megaesophagus includes the treatment of the underlying disease if possible and is otherwise performed according to the general therapeutic guide lines of esophageal diseases. Broad spectrum antibiotics are indicated when aspiration pneumonia occurs. There are currently no possibilities for medical management of idiopathic megaesophagus. Treatment with calcium channel blockers such as nifedipine or the application of cisaprid as a motility modifier is of no help due to the mainly striated muscle fibers in the canine esophagus and lower esophageal sphincter region. Therefore, the treatment can be only symptomatic.

The prognosis is guarded to poor. The mortality rate is 63-73% for congenital idiopathic esophagus. Most animals die or are euthanized approximately one year after diagnosis due to aspiration pneumonia or malnutrition.

Esophageal foreign bodies

Esophageal foreign bodies are easily recognized by endoscopy. A breed often presented is the West Highland white terrier. We have seen an increase in esophageal foreign bodies when BARF feeding was started to be used in Finland.

The treatment is immediate removal of the foreign body due to the high risk of local necrosis and esophageal perforation. Endoscopic withdrawal or placing of the foreign body into the stomach is possible in most cases by using a rigid Stadler's foreign body forceps (modified by Christoph) parallel to the endoscope. Through-the-scope foreign body forceps's are too weak for this procedure. Possible complication is esophageal rupture if swallowed bones have very sharp edges or the foreign body has been in the esophagus for several days. Surgical management is rarely necessary but of good prognoses.

Post operative management contains the application of H2-blockers (e.g. ranitidine) or proton pump inhibitors (e.g. omeprazole) depending on the severity of esophageal mucosal damage. Oral sucralfate as liquid solution between the feedings supports mucosal healing. In cases of severe mucosal damage, metronidazol can give antibiotic coverage against invasion of food

associated or oral anaerobic bacteria. In order to decrease pain during food intake, initial pain management can be performed by using butorphanol. We start to feed our patients a commercial canned food about 12-18 hours after the procedure.

The prognosis is very good if the foreign bodies are removed immediately. Delay in treatment increases the risk for stricture formation even despite successful removal and appropriate post operative management.

Esophagitis and gastroesophageal reflux

Causes of esophagitis are sharp foreign bodies, strong acid or alkaline fluids, and gastroesophageal reflux caused by incompetence of the lower esophageal sphincter, chronic vomiting, and hiatal hernia. Iatrogenic reflux can occur in dogs with cardia incompetence after long lasting abdominal surgeries. The inflammation can vary from mild to ulcerative hemorrhagic and suppurative hyperplastic. One cause of granulomatous esophagitis in dogs living in subtropic/tropic areas can be *Spirocerca lupi*.

Diagnosis is made by endoscopy revealing the severity of the inflammation and the occurrence of esophageal strictures as a secondary complication. Gastroesophageal reflux is difficult to assess due to esophageal relaxation during anesthesia causing opening of the lower esophageal spincter. It is likely the cause of esophagitis, when the inflammation mainly affects the caudal part of the esophagus. Gastroscopy with retrograde assessment of the lower esophageal sphincter allows to diagnose and to assess the severity of a hiatal hernia as a cause for reflux esophagitis.

Treatment is the combination of dietary management (low fat food, no feeding very late in the evening) and medical therapy using metoclopramide to increase lower esophageal sphincter tonus, antacida such as ranitidine, famotidine or omeprazole, and mucosa protectants such as sucralfate. In case of hiatal hernia and inadequate response to dietary and medical treatment within 30 days, surgical esophagopexy can be performed.

The prognosis depends on the severity of inflammation and the development of esophageal strictures.

Esophageal strictures

Intramural esophageal strictures are diagnosed by contrast radiography and endoscopy and have to be differentiated from extramural compression of the esophagus. In most cases they are benign and appear as singular or multiple scar-like circular narrowing of the esophagus.

The treatment is endoscopic guided balloon dilation with or without fluoroscopic control using through the scope balloons or second hand cardiac balloon catheters. It is advisable to use hydraulic pressure and balloon catheters should be filled with water or iodine contrast medium when fluoroscopy is performed parallel to the endoscopy procedure. The diameter of the balloon should be chosen according to the diameter of the stricture and the size of the patient in order to avoid esophageal rupture as a serious

complication of the procedure. The balloon is filled under vision control. After dilatation the dilated area has to be controlled for longitudinal bleeding tears as a sign for effective controlled rupture of the stricture. The procedure should be repeated every 2 - 3 days for about 3 – 4 times to make sure that the scar formation during the healing process does not lead to severe re-stenosis of the esophagus.

For post operative treatment some recommend the placement of a PEG tube to avoid oral passage of food. This is opposed by the fact that this leads to a lack of peristaltic boluses which can increase the likelihood of severe scar strictures. We do feed our patients with food of a particle size that can pass easily through the esophagus at its narrowest point. Medical management includes pain medication (butorphanol), omeprazole and sucralfate similar to the treatment of esophagitis. Whether the systemic or local application of glucocorticoids influences the re-stenosis of esophageal strictures after balloon dilation is under debate. There is currently no strong evidence for their benefit. The repeated stricture dilation procedures are stopped when the diameter of the stricture is about 1.0 – 2.0 cm so that a large sized endoscope (9-13 mm diameter) can easily be passed through the esophagus.

The prognosis is good. However, normal anatomic conditions will never be reached again and affected patients may be repeatedly presented either to remove foreign material that got stuck in front of the stricture or for a new series of balloon dilations.

Esophageal tumors

Esophageal tumors are very rare and diagnosed by endoscopy with biopsy when the tumor is erosive and friable as it is the case for carcinomas and sarcomas. Benign tumors at the lower esophageal sphincter such as submucosal leiomyoma can be treated successfully by surgery. Malignant tumors are often metastatic and of poor prognosis.

Gastroesophageal intussusception

Intussusception of the stomach into the esophagus can sometimes be diagnosed by radiography and confirmed by endoscopy. In patients with incompetence of the lower esophageal sphincter, vomiting during anesthesia for endoscopy can lead to temporary gastroesophageal intussusception that resoles spontaneusly.

In case of stationary intussusception it can be tried to push the intraesophageal part of the stomach back into the normal anatomic position. In cases with local necrosis or unsuccessful attempts of re-positioning surgical intervention is indicated.

References

1. Washabau RJ. Oropharyngeal and esophageal diseases. In: Gorman N (ed.): Canine Medicine and Therapeutics, 4th ed. Blackwell Science, Oxford, 1998: 437-55

2. Washabau RJ. Diseases of the esophagus. In: Ettinger SJ, Feldman EC (eds.): Textbook of Veterinary Internal Medicine, 4th ed. Vol 2, WB Saunders, Philadelphia, 2000:1142-1154
3. Gualtieri M. Esophagoscopy. Vet Clin North Am Small Anim Pract 2001; 31(4): 605-30
4. Sellon RK, Willard MD. Esophagitis and esophageal structure. Vet Clin North Am Small Anim Pract 2003; 33(5): 945-67
5. Han E, Broussard J, Baer KE. Feline esophagitis secondary to gastroesophageal reflux diseases: clinical signs and radiographic, endoscopic, and histopathological findings. J Am Anim Hosp Assoc. 2003; 39(2): 161-7
6. Jergens AE. Diseases of the esophagus. In: Ettinger SJ, Feldman EC (eds.): Textbook of Veterinary Internal Medicine, 5th ed. Vol 2, Elsevier Saunders, St.Luis, 2005:1298-1310
7. Hall EJ. Diagnosis and treatment of oesophageal diseases.Proceedings of the 18th annual congress of the VÖK, Salzburg, Austria, 2003: 53-61
8. Gualtieri M, Olivero D. Reflux esophagitis in three cats associated with metaplastic columnar esophageal epithelium. J Am Anim Hosp Assoc. 2006; 42(1): 65-70
9. Spillmann T. Erkrankungen des Ösophagus. In: Grünbaum EG, Schimke E. Klinik der Hundekrankheiten, 3rd ed., Enke, Stuttgart, 2007, 468-73