# HIATAL DISEASES: CAUSES, COMPLICATIONS AND TREATMENT

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## INTRODUCTION

The term "hiatal disease" indicates a number of diseases affecting the lower esophageal sphincter (LES) or the esophageal hiatus of the diaphragm that in most cases result in reflux esophagitis. Hiatal disease may result from: 1) defects in the embryonic development of the esophageal hiatus (congenital form); 2) factors altering the pressure of the LES (hiatal hernia, chronic vomiting, delayed gastric emptying, general anesthesia, surgical Trendelembur position, nasogastric and pharingogastric tube placement); 3) abdominal trauma causing a sudden increase in intra-abdominal pressure. Hiatal disease can cause clinical signs itself, but of more significance is its role in the development of gastroesophageal reflux and esophagitis. Factors responsible of reflux esophagitis are many and include the composition of the refluxed material, volume and frequency of reflux, contact time of reflux with the esophageal mucosa, integrity of the esophageal mucosal barrier and efficiency of the esophageal clearance. Clinical signs of reflux esophagitis depend on the severity of the esophageal lesions and on the nature of the underlying disease, and include anorexia, dysphagia, odynophagia, excessive salivation and regurgitation of thick, white saliva. Vomiting resulting from the hiatal disease may be associated. Among the different hiatal diseases, we consider cardial incompetence, hiatal hernia (sliding hernia and para-esophageal hiatal hernia) and gastroesophageal intussusception. Severe complications such as stricture and ulceration of the esophageal wall are frequent in chronic gastresophageal reflux secondary to cardial incompetence and/or hiatal hernia. A complication not reported in animals but well described in man has been observed recently by authors in cats affected by gastresophageal reflux. In the distal esophagus, the normal squamous epithelium was replaced by wide areas of metaplastic columnar epitheliu http://www.vin.com/ECVIM/2004/m; the lesion is comparable to Barrett's esophagus in men.

## CARDIAL INCOMPETENCE

This condition may result from lack of coordination of the muscle of the LES and the diaphragmatic crura, which normally work together to protect the esophagus against reflux. This uncoordination leads to the reflux of gastric content in the esophageal lumen. In the author's opinion, cardial incompetence may be considered the most common cause of gastroesophageal reflux in the dog and cat. Clinical signs vary with the severity of esophageal damage and include anorexia, dysphagia, excessive salivation, regurgitation, cough, vomiting, haematemesis and dyspnea. Respiratory symptoms are often the initial signs of this condition and in mild forms they may be the only signs. Reflux in fact is not necessarily associated with esophageal lesions. The diagnosis is not always easy and is based on clinical signs and endoscopic examination. Contrast radiography is not sufficiently sensitive for diagnosing this disorder. The correct endoscopic evaluation of cardial continence is not always comfortable and its quantification largely relies on the experience of the operator. Factors that can elicit gastroesophageal reflux during the endoscopic examination should also be considered, including lateral recumbency, air inflation and preanesthetic and anesthetic drugs (diazepam, thiopental, alotane). In cardial incompetence, the degree of opening of the cardia varies from partial to complete; in the letter case the gastric cavity may be directly seen from the esophagus. Often, the LES opens with the simple air inflation. This condition can be associated with a constant reflux of gastric fluid towards the esophagus and signs of esophagitis. In mild cases or when the described endoscocpic signs (particularly esophagitis), are lacking, the diagnosis can be really challenging.

## HIATAL HERNIA

A hiatal hernia is a protrusion of the stomach through the esophageal hiatus into the thorax. Two types of hiatal hernia have been recognized in the dog and cat: sliding hiatal hernia and paraesophageal hiatal hernia. In the sliding hiatal hernia the abdominal esophagus and part of the stomach are displaced cranially trough the esophageal hiatus, so that the LES is located within the thoracic cavity. This malpositioning and the loss of support of the LES lead to a reduced LES pressure and secondary gastroesophageal reflux. In the para-esophageal hiatal hernia a portion of the stomach (usually the fundus) herniates into the mediastinum alongside the thoracic esophagus, while the LES is located in a normal position. The sliding hernia is more common then the para-esophageal hernia and can be permanent or intermittent. Hiatal hernia may be congenital or acquired. Congenital forms are more frequent and it is suggested that they result from a malformation of the esophageal hiatus. Acquired hiatal hernias may result from increased intra-abdominal pressure caused by chronic vomiting or by persistently high negative intrathoracic pressure (causing a low-pressure zone in the esophagus) in animals with upper respiratory obstruction. Clinical signs are usually more severe with congenital forms than in acquired forms. Many patients may be asymptomatic. The primary clinical signs include vomiting, regurgitation, excessive salivation, dysphagia, anorexia, weight loss and dyspnea. Signs may be intermittent in a number of cases. These signs are completely superimposable to signs of cardial incompetence. Physical examination is usually unremarkable but can include weight loss, fever and pulmonary wheezes and crackles. Hiatal hernia can usually be diagnosed by survey thoracic radiographs, which reveal a gas filled soft tissue density in the caudodorsal thoracic region referable to the herniated stomach, and megaesophagus of variable degrees (permanent hernia). The diagnosis of intermittent sliding hernia may require a positive pressure applied to the abdomen to induce the displacement, or fluoroscopic studies. Contrast esophageal study will confirm the diagnosis and is useful to further delineate the LES, the esophageal hiatus and the esophageal dilation and motility, especially when performed under fluoroscopy. Chest radiographs can also reveal signs of aspiration pneumonia. The definitive diagnosis of para-esophageal hiatal hernia is usually made by esophago/gastrography and fluoroscopy. Endoscopy is useful to confirm the diagnosis of sliding hernia and to assess secondary reflux esophagitis. This is not costantly possible in case of intermittent hernia. Different degrees of distal esophagitis can be seen. Endoscopically, the cardia is followed by a dilated region covered by gastric mucosa (the intratoracic stomach) which ends with a narrowing of he lumen (the imprint of the esophageal hiatus) which simulate the LES. A more accurate diagnosis is obtained reaching the gastric lumen with the endoscope and observing the gastric side of the cardia ("J" maneuver). From this position an opening of variable size simulating the cardia but given by the imprint of the diaphragmatic crura is seen. Trough this opening a tract of distal esophagus and the dislocated cardia wraping the endoscope are seen. To demonstrate the hiatal disorder, the endoscope can be retracted in retroflexed position for up the whole distal third of the esophagus. In para-esophageal hernia, the gastroesophageal junction appears in the normal position, but with the endoscope retroflexed in the gastric lumen a portion of the stomach dislocating cranially trough the esophageal hiatus may be seen. In his experience, the author has never documented this type of esophageal hernia.

### Complications

Reflux of gastric fluid into the esophagus is the most common cause of esophagitis in small animals. In hiatal disease a reduction in lower esophageal sphincter pressure is responsible of the reflux because of the function of the lower esophageal sphincter is mechanically impaired. The frequency of reflux, the contact time with the mucosa and the composition of the refluxed material determine the severity of the lesion. The lesions are usually localized at the distal third of the esophagus. The endoscopic finding of mild forms is characterized by mucosal hyperemia arranged in linear streaks on the top of mucosal folds or round localized hyperemic areas. Increased mucosal friability and easily bleeding mucosa are seen in more severe cases; bleeding can be spontaneous or it

can be elicited by the passage of the endoscope. Most severe cases are characterized by the presence of erosions and ulcers. Erosions may disseminate on most of the esophageal mucosa. Ulcers may appear as oval shape, shallow lesions with indistinct margins or as deeper lesion with raised and edematous margins. The letter more severe forms are generally located next to or at the cardia and are usually fewer in number (from 1 to four), deeper and have raised and edematous margins. Another complication is caracterized by the presence of esophageal strictures. They probably originate from an abnormal, irregular healing of large and deep lesions of the esophageal mucosa. By endoscopy they may appear as a white ring of fibrous tissue narrowing the esophageal lumen at various degrees and not distending in response to air inflation. In the author's experience, benign fibrous strictures can be classified upon their endoscopic aspect in five main types: annular, mucous branches, semilunar, tortuous and tubular strictures.

Among complications of chronic gastroesophageal reflux secondary to cardial incompetence and/or hiatal hernia, the author has observed an intestinal metaplasia of the distal esophageal epithelium in three cats, similar to Barrett's esophagus in men. The suspected pathogenesis of this condition is the replacement of the native squamous epithelium by a metaplastic epithelium offering greater resistance to the effects of gastresophageal reflux. The clinical and endoscopic aspects in these cats were not specific and referable to chronic esophagitis.

## GASTROESOPHAGEAL INTUSSUSCEPTION

This is a rare hiatal disorder resulting from invagination of the stomach into the lumen of the esophagus; other abdominal organs (duodenum, spleen, pancreas etc.) may or not invaginate as well. It is more common in young animals and in dogs than in cats. The onset is usually acute. The etiology is not completely known, but predisposing factors are idiopathic megaesophagus, cardial incompetence and acute and chronic vomiting. Regurgitation, hematemesis and dyspnea can be followed by more severe and dramatic signs such as respiratory and cardiac arrest, shock and death due to the greatly enlarged esophagus compressing the pulmonary parenchima and causing circulatory collapse. In some cases however, chronic intermittent gastroesophageal intussusception with a mild degree of invagination can cause mild and intermittent signs. Survey radiographs will reveal a soft tissue density with gastric rugal folds in the distal esophagus; a ventrally deviated trachea and aspiration pneumonia may be seen. Contrast study may also demonstrate the intussusception. Esophagoscopy reveals a dilated esophagus filled with gastric mucosal folds. Esophagitis is usually evident . Advancement of the endoscope may be restricted by the edematous and congested gastric mucosa or by other invaginated organs.

## TREATMENT

In case of small intermittent hiatal hernias and/or moderate cardial incompetence, medical treatment (ranitidine 2-5 mg/kg PO, BID, methoclopramide 0.2 mg/kg PO, BID, commercial low fat diet) is beneficial for gastroesophageal reflux or esophagitis. Surgery is required in cases unresponsive to medical treatment or in presence of large hernias. A number of surgical techniques have been described for correction of hiatal hernia, including diaphragmatic hiatal reduction and plication, esophagopexy, left sided fundic gastropexy. In patients with gastroesophageal reflux secondary to severe cardial incompetence, an antireflux procedure is mandatory (Nissen fundoplication). Surgery should be performed early after diagnosis of gastroesophagal intussusception. The technique involves the reduction of the intussuscepted stomach, resection of any devitalized tissue, reduction of the size of the esophageal hiatus and incisional gastropexy at the gastric fundus to prevent relapses.