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CASE REPORT

Companion or pet animals

Diagnosis and treatment of sliding hiatal hernia and false gastric diverticulum in a British shorthair cat

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Abstract

A 7-year 9-month-old, neutered, male British shorthair cat was referred for chronic regurgitation and vomiting. Previous symptomatic medical management did not improve these clinical signs. Survey radiographs and abdominal ultrasound revealed no significant abnormalities of the gastrointestinal tract. Exploratory laparotomy was performed to obtain gastrointestinal biopsies. Sliding hiatal hernia and an abnormal gastric outpouching at the fundus, measuring 15 × 15 × 15 mm, were identified. Surgical treatment of the hiatal hernia included phrenoplasty, oesophagopexy and left-sided gastropexy. Additionally, the gastric outpouching was resected. Histopathologic examination of the excised fundic tissue revealed findings consistent with those indicative of a false gastric diverticulum. Postoperative examinations on Days 5 and 10 and telephone follow-up on Days 30 and 120 revealed a marked reduction in clinical signs. The combination of surgical techniques—phrenoplasty, left-sided gastropexy, oesophagopexy and gastric diverticulum resection—was found to effectively improve the cat's clinical signs.

BACKGROUND

To the best of the authors' knowledge, this is the first case report to describe the management of a feline gastric diverticulum (GD) with concurrent hiatal hernia in the field of veterinary medicine. This report also provides information regarding the possible pathophysiologic consequences of feline hiatal herniation.

CASE PRESENTATION

A 7-year 9-month-old, male, neutered British shorthair cat presented with a 4-month history of intermittent progressive regurgitation and vomiting. Abdominal ultrasound, performed by the referring veterinarian, did not reveal any abnormalities. Symptomatic medical management, including the administration of maropitant (Cerenia; Zoetis) and omeprazole (Omeprazol; 1A Pharma), was attempted. The cat continued to vomit infrequently, once every 2 weeks, but maintained a normal appetite. Subsequently, the cat was referred for further investigation.

INVESTIGATIONS

The results of a physical examination were unremarkable, apart from mildly increased respiratory effort.

Survey abdominal and thoracic radiographs were obtained and did not reveal any significant abnormalities, apart from uroliths within the urinary bladder (Figure 1). Abdominal ultrasound was performed, including a detailed evaluation of the gastrointestinal tract, which showed no abnormalities. Exploratory laparotomy was performed to obtain gastrointestinal biopsies. On exploration, signs of moderate serositis were found in the cardia and gastric fundus (Figure 2). Additionally, the gastro-oesophageal junction was observed to be sliding cranially through the diaphragm, and there was a subjective enlargement of the oesophageal hiatus. Furthermore, an outpouching of the gastric wall, measuring approximately 1.5 × 1.5 × 1.5 cm, was visible on the fundus (Figure 3). The remainder of the gastrointestinal tract was inspected, and no abnormalities were noted. Biopsies were obtained from the stomach, duodenum and jejunum.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses for regurgitation in cats include megaoesophagus related to neuromuscular disease (myasthenia gravis), oesophagus stricture, oesophageal foreign body, oesophageal neoplasm, persistent right aortic arc and hiatus herniation. Because the cat's appetite was well maintained over time, a dynamic disease process, such as hiatal herniation, was deemed to be the most likely cause of regurgitation in this case. The reported differential diagnoses for vomiting are many and include extragastrointestinal and gastrointestinal causes. The most likely extragastrointestinal causes in the present case were pancreatitis, cholangiohepatitis and cholecystitis. Gastrointestinal differential diagnoses, such as gastritis, enteritis, foreign bodies and ileus, were also considered possible given that lymphoplasmatic enteritis was

diagnosed through a histologic examination of gastroduodenal samples.

TREATMENT

Because of the cat's history and gastro-oesophageal abnormalities identified during exploratory laparotomy, the surgical interventions that were performed included phrenoplasty, oesophagopexy, left-sided gastropexy and surgical resection of the gastric outpouching.

Phrenoplasty was performed by inserting two horizontal mattress sutures dorsally and ventrally through the oesophageal hiatus using 2-0 polypropylene (Prolene; Ethicon), and care was taken to avoid the vagus nerve. Oesophagopexy was performed by securing the left side of the oesophagus to the medial left diaphragmatic crus with a simple continuous 4-0 polydioxanone (PDS; Ethicon) suture material. Subsequently, left-sided incisional fundic gastropexy was performed to secure the stomach to the left body wall caudal to the last rib using 4-0 polydioxanone (PDS; Ethicon).

The outpouching of the fundic tissue was resected using a scalpel blade. Following resection, gastric closure was performed in two appositional layers (mucosal and submucosal layers; muscular and serosal layers) using 4-0 polydioxanone (PDS; Ethicon). The resected tissue was submitted for histopathologic examination.

Routine cystotomy was performed to remove the previously identified uroliths, followed by routine ventral midline abdominal closure.

The cat recovered uneventfully after the surgery. Postoperative analgesia was achieved using methadone at a dose of 0.2 mg/kg administered intravenously every 4 hours for 12 hours. Subsequently, analgesia was maintained with a 25-μg/h fentanyl patch (Fentanyl; Hexal), which was applied transdermally during the postoperative period. The cat was discharged 24 hours postoperatively with a prescription for a proton-pump inhibitor (Omeprazol; 1A Pharma) and sucralfate (Sucrabest; Combustin), both to be administered for 7 days.

LEARNING POINTS/TAKE-HOME MESSAGES

- Gastric diverticula rarely occur in people and cats.
- Gastric diverticulum should be included in the list of differential diagnoses in case of cats with a history of chronic gastrointestinal signs.
- In human medicine, in most cases, gastric diverticula are asymptomatic and are discovered incidentally.
- If persisting gastrointestinal signs are present, surgical resection is indicated.

OUTCOME AND FOLLOW-UP

The referring veterinarian performed re-examinations on Days 3 and 10 postoperatively. On Day 10 postoperatively, the abdominal sutures were removed, and the cat's owners reported the complete resolution of the clinical signs. Telephone follow-up on Days 30 and 120 revealed a marked reduction in clinical signs at this time. Vomiting occurred less than once weekly, and regurgitation was not reported again by the owner.

A histopathologic examination of the gastric outpouching revealed mucosal and submucosal gastric tissue with absence of the normal gastric muscularis layer, consistent with the presence of a false GD. Examination of duodenal biopsies revealed lymphoplasmacytic enteritis. Urinary calculi analysis revealed calcium oxalate uroliths.

DISCUSSION

Reports regarding cases of GD in cats and dogs are rare in the literature. To the best of the authors' knowledge, this is the first case report of a GD with concurrent hiatal hernia in a cat. Recently, in a small case series of six cats, the occurrence of feline GD was reported for the first time in the literature.[1]

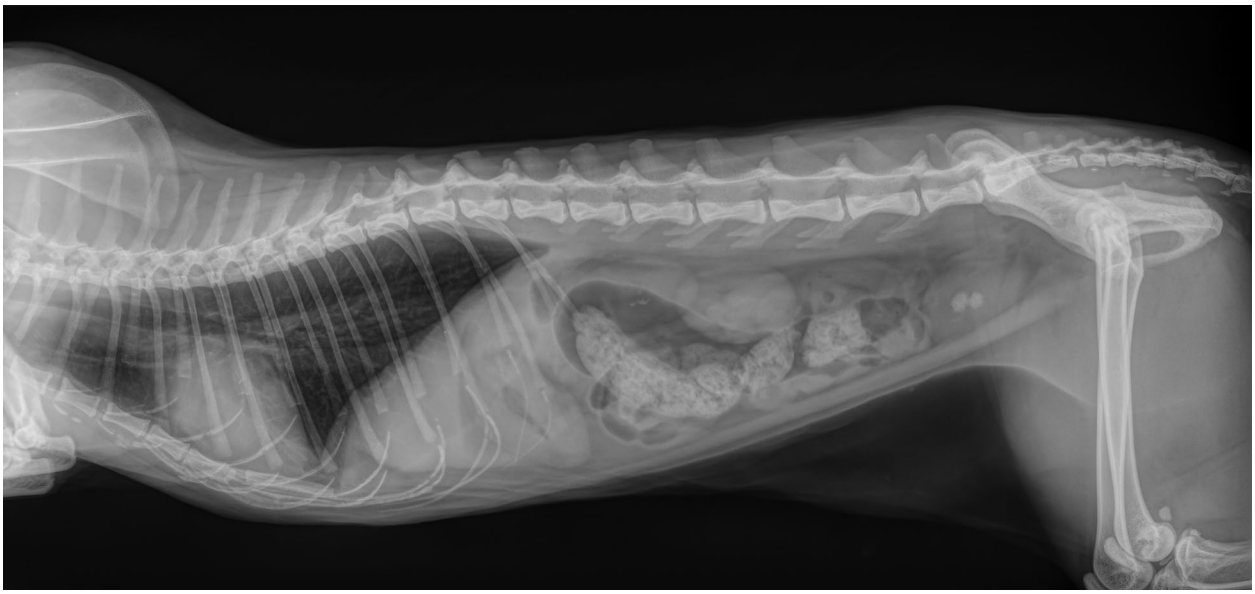

FIGURE 1 Left lateral survey radiograph failed to identify the hiatal herniation. Note the cystic calculi as incidental finding

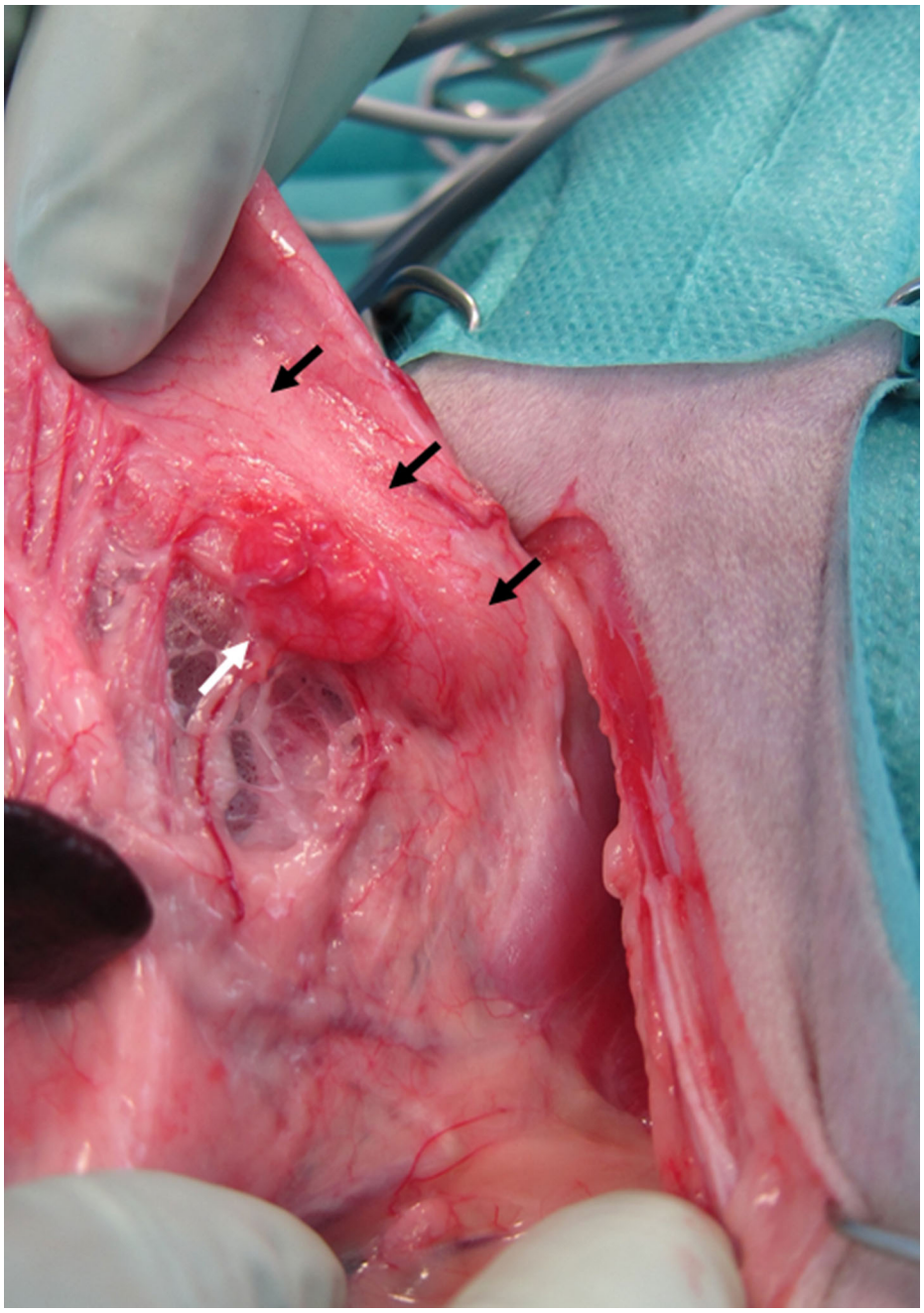

FIGURE 2 Intraoperative picture of the stomach. A marked serositis is seen along the cardia and fundus (black arrow). An outpouching structure at the fundic region is visible (white arrow)

In human medicine, GD has been identified throughout the gastrointestinal tract, including in the oesophagus, stomach, small intestine and colon. The estimated prevalence in previous reports ranges between 0.1% and 2.6%.[2–5]

GD is thought to develop within the gastrointestinal wall due to the development of abnormal pressure within the organs, defects in the bowel wall or dysfunctional peristalsis.[6]

Two different types of GD occur in people: true and false GD. True GD is congenital and histologically involves all four gastric layers (mucosa, submucosa, muscle and serosa). By contrast, false GD is acquired and develops from mucosal and submucosal outpouchings through the gastric wall. In human medicine, GD is most commonly asymptomatic, and diagnoses are often made incidentally by oesophagogastroduodenoscopy. Abdominal signs associated with GD include nausea, vomiting, early satiety, anorexia and epigastric pain and discomfort. Infrequently, complications, such as ulceration, haemorrhaging, upper gastrointestinal bleeding and malignant transformation, can develop. These complications may become life threatening and are indications for surgical management. The treatment of choice includes partial gastrectomy along with the treatment of the underlying disease. Medical management includes the use of a proton-pump inhibitor, histamine H2 receptor antagonist or antacid medication.

In the case described here, the GD was located in the gastric fundus, which is similar to the findings in previous reports of GD located in the fundic region of the stomach in all the affected cats.[1]

In human medicine, although false GD could potentially develop at any gastric location, it is typically found near the antrum.[2] This anomalous location may be explained by the

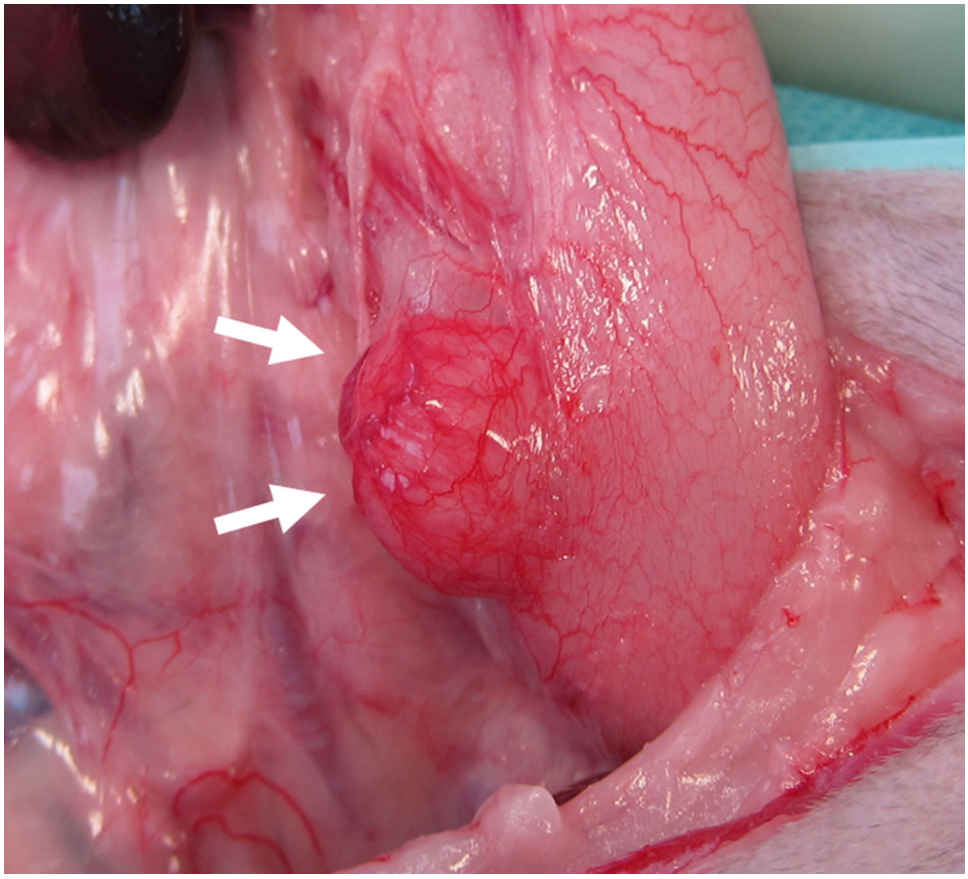

FIGURE 3 Intraoperative picture of the gastric diverticulum (white arrow)

pathophysiology of false GD. False GD can be further subdivided into traction and pulsion diverticula. Pulsion diverticula develop as a result of increased intraluminal pressure caused by chronic coughing or vomiting, as seen in the present case, whereas traction diverticula result from adhesive forces induced by an adjacent inflammatory process (e.g., pancreatitis, cancer or cholecystitis).[2]

In the present case, the exploratory laparotomy also revealed a marked area of serositis along the gastric cardia and fundus. Additionally, a subjectively enlarged oesophageal hiatus was noticed, which pushed the gastro-oesophageal junction into a position cranial to the diaphragm. These findings were consistent with a diagnosis of type I sliding hiatal hernia. In dogs and cats, hiatal hernia is defined as the protrusion of the abdominal contents, most often the cardia and fundus of the stomach, through the oesophageal hiatus into the caudal mediastinum.[7] Although limited data exist in the case of cats, type I sliding hiatal hernia is the most common (85.7%) type of feline hiatal hernia according to a recent retrospective study.[7] Types II and III were found in 3.6% and type IV in 7.2% of the cats in the same investigation. In type I hiatal hernia, the gastro-oesophageal junction intermittently moves craniocaudally from the abdominal cavity into the thoracic cavity, whereby type II hiatal hernia is characterised by the physiologic position of the gastro-oesophageal junction, but parts of the gastric fundus herniate cranial to the diaphragm. In type III hiatal hernia, both the gastro-oesophageal junction and parts of the gastric fundus herniate through the diaphragm, and in type IV hiatal hernia, the abdominal organs, apart from the stomach, herniate cranial to the diaphragm into the thoracic cavity.[8]

In the same retrospective study, gastrointestinal signs, including vomiting, weight loss and anorexia, were reported in 96% of cats with hiatal hernia. Interestingly, this study also reported an increased respiratory rate (>40 breaths per minute) and effort in 96% of cats with hiatal hernia. The authors of the study suggested that conditions that cause airway obstruction play an important role in the development of hiatal hernia in cats, similar to in brachycephalic dogs.[7] The literature indicates that there is a high incidence of sliding hiatal hernia in brachycephalic dogs. Reeve et al. reported that dogs with brachycephalic obstructive syndrome (BOAS) presented with gastro-oesophageal reflux and sliding hiatal hernia in 75% and 44.4% of cases, respectively.[9]

There are two possible explanations for the development of hiatal hernia in brachycephalic dogs. First, the increased supraphysiologic inspiratory pressure required to overcome upper airway resistance creates an increased negative intrathoracic pressure. Consequently, the gastro-oesophageal junction is pulled intrathoracically, leading to an axial cranial displacement. It is suggested that the movement of the gastro-oesophageal junction through the oesophageal hiatus is responsible for hiatus enlargement. Second, an enlarged oesophageal hiatus is another congenital anatomical characteristic of brachycephalic breeds and may be responsible for a functional alteration of the oesophagus and gastro-oesophageal junction, which could lead to regurgitation, sliding hiatal herniation and gastro-oesophageal reflux.[10]

The exact aetiopathogenesis for the development of sliding hiatal hernia in brachycephalic dogs has not been explained yet, but similarities with brachycephalic cats may exist.[7]

Currently, there is no gold standard for the diagnosis of hiatal hernia in cats. In the present case, before exploratory laparotomy, additional diagnostic procedures, such as contrast oesophagography and upper gastrointestinal contrast radiographic examinations, as well as videofluoroscopic swallowing studies, were not performed because the owners had cost restrictions. A comprehensive diagnostic workup may have provided further information regarding the aetiopathogenesis of GD. This may also support the hypothesis that sliding hiatal hernia is underdiagnosed in cats and is important in the differential diagnosis of cats with gastrointestinal and concomitant upper respiratory symptoms. A previous study demonstrated that survey radiographs could aid in diagnosing hiatal hernia in only 38% of cats, but contrast oesophagography and upper gastrointestinal radiographic studies increased the diagnostic sensitivity to 89% in affected cats.[7] This may explain why the diagnosis of hiatal hernia in this case was made during exploratory laparotomy rather than through the thoracic radiographs received from the referring veterinarian. Exploratory laparotomy was performed to obtain gastrointestinal biopsies in order to identify the underlying aetiology for the clinical signs of vomiting and regurgitation exhibited by the cat. A histopathologic analysis of the duodenal biopsies revealed lymphoplasmatic enteritis. These results are consistent with the findings of a recent retrospective study in which 76% of cats treated surgically for hiatal hernia had concurrent abnormalities with aetiologies unrelated to hiatal hernia (e.g., hyperthyroidism, thyroid adenoma, thyroid carcinoma, pulmonary adenocarcinoma, chronic renal disease and diabetes mellitus).[7] Furthermore, a recent case series of feline GD reported significant comorbidities, including inflammatory bowel disease (3/6) and alimentary small cell lymphoma (2/6), in four out of six cats.[1]

Given the diagnostic findings, reported gastrointestinal signs and respiratory effort in the cat in our case, it may be possible that sliding hiatal hernia developed as a comorbidity of the brachycephalic nature of the cat's breed. The occurrence of sliding hiatal hernia may have been influenced by gastrointestinal peristalsis and an altered thoracoabdominal pressure gradient compared to that in unaffected cats.

Furthermore, chronic vomiting causes temporary excessive intra-abdominal pressure and abnormal gastrointestinal peristalsis, which could be factors that led to the development of false GD in the cat.

Although the cause of GD is most likely multifactorial, it could be hypothesised that one of the factors that contributed to the development of GD and hiatal hernia in this cat was upper airway obstruction. GD was primarily diagnosed at the time of exploratory laparotomy as an incidental finding. At this time, it is also unclear if the histopathologically diagnosed lymphoplasmacytic enteritis had an influence on the clinical signs and genesis of GD. Although histopathologic findings tend to support a diagnosis of false 'acquired' GD, a congenital cause cannot be excluded.

Additionally, it is not possible to definitively state whether the remission of gastrointestinal signs was due to the resection of the GD or surgical treatment of hiatal hernia. Bahlmann et al. reported reduced clinical signs following surgery; therefore, it is likely that the resection of the GD contributed to the improvement in this case.[1]

In conclusion, this report describes a case of sliding hiatal hernia and false 'acquired' GD in a brachycephalic cat. Further investigations are warranted for determining the prevalence of sliding hiatal hernia and concurrent GD in cats. Negative contrast radiography before exploratory laparotomy may be a helpful tool for detecting GD.[1]

FUNDING INFORMATION

The authors received no specific funding for this work.

CONFLICT OF INTEREST

The authors declare they have no conflicts of interest.

ETHICS STATEMENT

The case report describes a client-owned cat with a high standard of veterinary medicine. The owner was informed about diagnostic and therapeutic steps and confirmed these with permission.

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