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CASE REPORT

Brunner's glands hyperplasia and recurrent gastric impaction in a horse

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Abstract

While Brunner's gland hyperplasia may rarely be considered as a cause of chronic gastric impaction in horses, it should be included in the differential diagnoses. A 7-year-old Cob cross-breed gelding was presented with weight loss over a 3-week period, and a diagnosis of chronic gastric impaction was made. Initial medical management was successful; however, the impaction recurred a further three times, at progressively shorter intervals, over the following 3 months. The owners subsequently elected euthanasia, and the horse was submitted for post-mortem examination. A focal nodule within the cranial part of the duodenum was seen during the post-mortem exam. Histopathologic evaluation of the duodenum revealed a marked increase in Brunner's glands admixed with distinct lobules of exocrine pancreatic tissue.

BACKGROUND

The duodenal glands or Brunner's glands are located within the submucosa of the small intestine of all mammals. These glands are composed by two different cell types: serous and mucinous, and usually the latter one is predominant.[1] They are involved in digestion through the secretion of alkaline fluid with high content of mucin.[2] Here, we describe a previously unreported manifestation of focal Brunner's gland hyperplasia in a horse with gastric impaction. To date, the pathogenesis of this condition is still poorly understood, and it had been hypothesised to be a duodenal reaction to acid hypersecretion.[3] In order to better characterise the pathogenesis and possible treatments for this condition, further studies and investigations are required.

CASE PRESENTATION

The horse initially presented with a 3-week history of sudden weight loss, despite an increase in feed quality and quantity being consumed, lethargy and reluctance to walk. Previous parasite management and anthelmintic administration was deemed appropriate, and there had been no recent changes in husbandry. Clinical examination was unremarkable other than poor body condition score (2/5) and sensitivity over the left side of the ribs/girth area. Faecal worm egg count, haematology and biochemistry were within normal limits. Gastroscopy following a 16-h food witholding diagnosed gastric impaction. Abdominal ultrasonography confirmed gastric distension with the stomach wall visible over seven intercostal spaces reaching the 15th intercostal space. Following clearance of this impaction with carbonated soft drink, secondary grade 3 equine squamous gastric disease was observed. The horse was initially managed on a grass and soaked hay diet with additional hard feed which was replaced by complete grass and grass pellet diet after recurrence of impaction. Repeat gastric impaction was diagnosed by gastroscopy following a 16-h fast on three further occasions over the next 3 months, initially after 4 weeks, then 9 days and latterly 4 days, necessitating humane euthanasia.

On the post-mortem examination, the stomach was moderately distended, with no evidence of displacement or obstruction. The stomach contained a large amount of dry, solid, green, ingesta (grass), and within the small intestine, there was abundant soft green matter (grass) admixed with a dark yellow liquid. There was a focal, raised nodular lesion within the duodenum (Figure 1) and few rare round worms (compatible with *Parascaris equorum)*.

Multiple sections of stomach, duodenum and pancreas were examined. Microscopically, expanding the duodenal submucosa and the lamina propria, there was a marked increase of the Brunner's glands. Multifocally, interspersed between the hyperplastic tissues, there were larger distinct lobules (up to 2 x 5 mm) of exocrine pancreas (Figure 2). One section of pancreas was analysed, but no structural alterations were noticed.

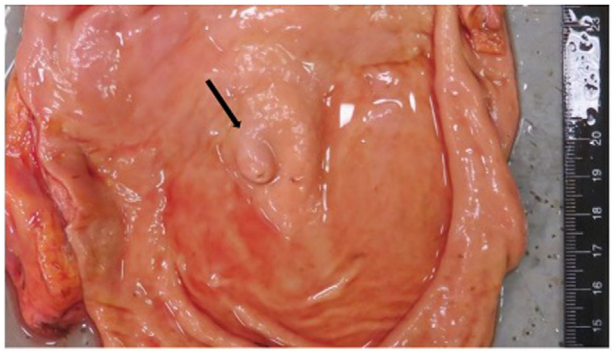

FIGURE 1 Cranial duodenum (luminal surface) with a focal, round, raised pink nodule histologically identified as Brunner's glands hyperplasia (black arrow)

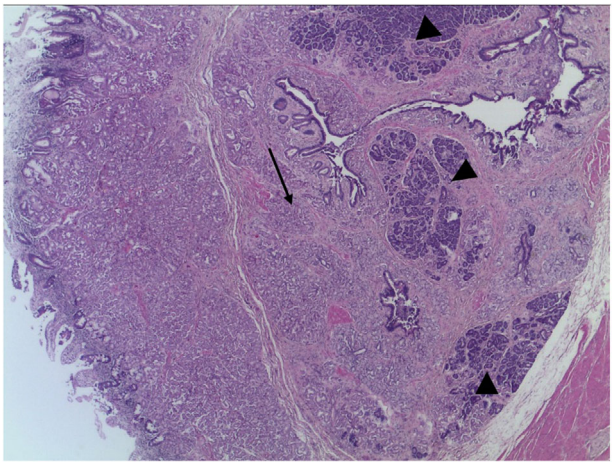

FIGURE 2 Duodenum (H&E 2.5X HPF): There is marked increase of the Brunner's glands expanding the submucosa and multifocally extending into the lamina propria(arrow). The glands are predominantly mucinous type. Small nests of serous type glands are present resembling exocrine pancreatic tissue (arrow head)

The lamina propria of the stomach was infiltrated by moderate numbers of lymphocytes, plasma cells and eosinophils, surrounding and separating the gastric glands.

INVESTIGATIONS

For each of the episodes of impaction gastroscopy, following a 16-h fast, and abdominal ultrasonography were performed to confirm the diagnosis. Gastric mucosal biopsy was performed at second presentation of impaction, due to a cobblestone appearance of the squamous mucosa, which revealed mucosal hyperplasia and hyperkeratosis. Haematology and biochemistry, faecal worm egg count, abdominocentesis and peritoneal fluid analysis and a glucose absorption test were all within normal parameters. Increased wall thickness of the proximal duodenum (0.6 cm) adjacent to the stomach and liver was observed on abdominal ultrasonography at the first repeat examination and thereafter. The owner declined duodenal biopsy.

DIFFERENTIAL DIAGNOSIS

Several differential diagnoses were needed to be considered in this case, including the presence of a foreign body within the pyloric region, a partial outflow obstruction due to pyloric hypertrophy and granuloma due to parasitic infection. Important to mention are also neoplastic diseases such as lymphosarcoma, adenocarcinoma or squamous cell carcinoma. A primary motility disorder was also considered.

LEARNING POINTS/TAKE-HOME MESSAGES

- Nodular Brunner's gland hyperplasia is rarely considered as a cause of chronic gastric impaction.
- Biopsy is essential to distinguish this condition from its differential diagnoses but would only be possible between impaction episodes and would be challenging using endoscopic grab biopsies given the submucosal pathology.
- Further study aimed to understand the pathogenesis is essential in order to determine treatment.

TREATMENT

Each episode of gastric impaction was treated by nasogastric intubation and administration of 2 L of Coca-Cola Zero (Coca Cola European Partners, Uxbridge, UK) and 2 L of water followed by starving. After the initial diagnosis of secondary equine squamous gastric disease (ESGD), treatment was started using omeprazole (Gastrogard, Boehringer Ingelheim, Bracknell, UK) at 4 mg/kg per os once a day (SID). At the first repeat examination, omeprazole was continued at the reduced dosage of 2 mg/kg per os SID, with the addition of sucralfate (Antepsin, Chugai Pharma UK Ltd, Turnham Green, UK) at 10 mg/kg per os twice a day (BID). Prednisolone (Accord-UK Ltd, Barnstaple, UK) at 1 mg/kg per os q24hrs was added in case of an inflammatory component to the impaction given the small intestinal distension seen on ultrasonography. Following resolution of the third gastric impaction, metoclopramide (Accord-UK Ltd) was initiated at 0.1 mg/kg PO BID as a motility stimulant.

OUTCOME AND FOLLOW-UP

Euthanasia and post-mortem examination.

DISCUSSION

Although some horses with recurrent gastric impaction have been successfully managed with long-term dietary modification (removal of long fibre forage) and motility stimulant administration,[4] others fail to respond and require euthanasia.

Gastric impaction can occur as a primary disease or secondary to an inciting cause; the reader is referred to Freeman, 2011[5] for a complete review of the causes of gastric impaction. Briefly, primary causes are obstructions caused by expanded ingesta, dental disease or poor water intake, while secondary gastric impaction is associated with ragwort toxicity or intestinal obstruction. Clinical signs associated with

gastric impactions are generally related to the onset of colic and may vary from mild lethargy and anorexia to severe colic signs (such as recumbency and rolling).[6] In this case, signs associated with colic were not reported, and the recurring reason for clinical examination was a depressed demeanour and lethargy; progression to colic signs may have been avoided by factors such as an insidious increase in size of duodenal glands or by increased vigilance of the owners following the diagnosis of gastric impaction at preliminary examination, allowing for prompt veterinary intervention. Initial medical treatment for gastric impactions is aimed towards softening the impacted bolus and is achieved by nasogastric intubation and repeated administration of water or a carbonated drink.[7] Duodenal biopsy during remission of gastric impaction may have aided pre-mortem diagnosis of Brunner's gland hyperplasia but endoscopic grab biopsies usually only retrieve superficial mucosa. Surgical approaches have been described[8] however, due to the relatively high anaesthetic risk in horses,[9] are only considered as a last resort if medical management is insufficient to alleviate clinical signs.

Duodenal glands (also known as Brunner's glands) were first histologically described in horses in 1989 by Takehana et al.[1] They are responsible for producing an alkaline fluid that acts as a buffer for gastric acid once digesta enter the small intestine. While there are species-specific differences in composition, duodenal glands are broadly arranged in coiled tubules composed of a combination of mucous and serous glandular cells. In horses, both cell types are present in the proximal 10 cm of duodenum closest to the pyloric sphincter, while the more distal segments contain predominantly mucous glands.

In medical literature (both human and veterinary), published reports of pathology of the duodenal glands are restricted to proliferative diseases categorised as either hyperplasia or adenoma. Carcinomas are reported in medical literature but are considered very rare.[10] Duodenal gland pathology has been reported in sand rats, a dog and a horse.[11–13]

In medical literature, Brunner's gland hyperplasia most commonly manifests as a focal, often pedunculated, nodule or polyp within the duodenal lumen; however there are rare reports of a more focally extensive nodular thickening as the presentation.[14,15] Brunner's gland hyperplasia is often an incidental finding on gastrointestinal endoscopy in human medicine; clinical signs are more commonly associated with larger masses with obstruction being seen in masses greater than 2.1 cm.[16]

In horses, Brunner's gland hyperplasia has previously only been described as a multifocal nodular thickening of the duodenal mucosa (Kullman, 2013). This case appears to be the first report of a single, focal, circumscribed thickening, more similar to that commonly seen in humans. Due to the low incidence of duodenal gland pathology in veterinary medicine, there are no reports of surgical management of these conditions. In the case presented above, it is unlikely that surgery to correct the Brunner's gland hyperplasia would have been useful as the mass was diffuse and situated across the pyloric outlet.

The association between the recurrent gastric impactions and Brunner's gland hyperplasia is unknown. The nodule itself appears too small to have had a direct obstructive effect; however gland hyperplasia may have affected intestinal motility. It is plausible that the chronic infiltrates observed within the lamina propria of the stomach had a negative impact on gastric motility. In human medicine, recent studies have confirmed an association of coeliac disease with a chronic lymphocytic gastritis.[17] Delayed gastric emptying has been documented in coeliac patients,[18] which some studies report to be associated with increased plasma concentrations of neurotensin, a hormone which inhibits upper gastrointestinal motility.[19,20] Possible gluten sensitive enteropathy has been described in two horses but neither of these presented with gastric impaction.[21,22] To our knowledge, little is known about gastric motility and chronic gastritis in horses, further studies are required to better understand the pathogenesis of gastric impactions.

Alternatively, the observed surrounding inflammatory cell infiltrates may have been responsible for clinical signs in this horse. Inflammatory small bowel disease can be associated with weight loss but an effect on intestinal motility has not previously been described to the authors' knowledge. It is also possible that the Bruuners' gland hyperplasia was an incidental finding in this case.

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