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Case Report

Squamous gastric ulceration complicated by gastric stenosis in a foal

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Summary

A 2-month-old Warmblood colt presented with recurrent colic and regurgitation. Gastroscopy, performed on several occasions, and barium-contrast radiography revealed severe squamous gastric ulceration and stenosis at the level of the margo plicatus. Treatment with omeprazole reduced the extent and severity of the gastric ulcers but did not affect the stenosis. The foal was euthanised because of a poor prognosis, and post-mortem examination confirmed the clinical diagnosis. Severe squamous gastric ulceration, granulation tissue formation and cicatrisation of deep gastric lesions were considered to have caused the stenosis. Gastroduodenal outflow obstruction is a recognised disorder in foals, but stenosis at the level of the margo plicatus has not been reported in foals or adult horses. To the authors' knowledge, this is the first case of severe squamous gastric ulceration, complicated by stenosis at the level of the margo plicatus, in a foal. Although rare, gastric stenosis should be considered in foals suffering recurrent colic and regurgitation.

Introduction

The term equine gastric ulcer syndrome (EGUS) has been in use since 1999 to describe stomach ulcers in horses, but more recently it has become clear that it encompasses two very different disease entities, now termed equine squamous gastric disease (ESGD) and equine glandular gastric disease (EGGD), with differing risk factors, epidemiology, clinical signs and prognosis (Sykes et al., 2015). Equine squamous gastric disease is the result of increased exposure of the squamous mucosa to acid and may be primary or secondary to duodenal outflow obstruction (Bezdekova & Hanak, 2009; Bezdekova et al., 2020; Sprayberry, 2015; Sykes et al., 2015; Zedler et al., 2009). Stress is often thought to play a role in the development of both ESGD and EGGD, both in foals and adult horses, although proof of the role of stress in ESGD is scarce and often indirect (Scheidegger et al., 2017; Sprayberry, 2015). Prinsloo et al. (2019) found a negative correlation between the severity of ESGD and hair cortisol concentrations, although this certainly does not prove causality, and a more recent study demonstrated that the severity of ESGD after transportation was correlated with stress-related behaviour (Padalino & Raidal, 2020).

Gastric ulceration is common in foals, with an estimated prevalence of 25 to 50%, increasing to as high as 95% around weaning, although the distinction between ESGD and EGGD is often not made (Hewetson et al., 2018; Murray et al., 1990).

Delayed gastric emptying in horses can occur because of functional or mechanical obstruction. Examples of functional obstruction include ileus or hypersecretion and distension of the duodenum (Sprayberry, 2015). Mechanical impairment of gastric emptying results from narrowing at the level of the pylorus or duodenum or, rarely, from an intraluminal mass (Bezdekova et al., 2020). Sárdi et al. (2013) reported a developmental abnormality in the stomach of a 9-month-old Warmblood filly that caused delayed gastric emptying. Most commonly, impaired gastric emptying is seen in foals with ulcers at the gastroduodenal junction (Bezdekova et al., 2020; Sprayberry, 2015; Zedler et al., 2009).

Strictures can form as a sequel to gastric ulceration, with tissue damage associated with ulceration leading to inflammation, scarring and the development of granulation tissue, and functional and mechanical obstruction (Zedler et al., 2009). In gastroduodenal ulcer disease (GDUD), which is mainly encountered in foals between 2 and 6 months of age, stricture formation at the pylorus and duodenum may occur, leading to gastric outflow obstruction (Camacho-Luna et al., 2018; McKenzie, 2018; Zedler et al., 2009). Affected foals are dull and may develop bruxism and ptyalism, and in some cases, they may present with gastric reflux (McKenzie, 2018).

Currently, a diagnosis of gastric ulceration can only be made by gastroscopy (Camacho-Luna et al., 2018; McKenzie, 2018), while transcutaneous ultrasonography and barium-contrast radiography can be used to confirm delayed gastric emptying (Camacho-Luna et al., 2018; McKenzie, 2018). If a stricture is present, complete emptying of the stomach takes more than 2 h (McKenzie, 2018; Sprayberry, 2015).

The treatment of foals with gastric ulceration and gastric outflow obstruction consists of the administration of gastroprotectants and/or proton-pump inhibitors (Bezdekova et al., 2020; Sprayberry, 2015). Various surgical techniques have been described for cases of gastroduodenal ulcer disease (GDUD) that fail to respond to medical treatment (Zedler et al., 2009). The procedures are reported to be associated with an excellent prognosis for foals with pyloric obstruction (Kent et al., 2020) and a fair to guarded prognosis for foals with duodenal obstruction (Zedler et al., 2009).

This case report describes the clinical and histopathological features of a Warmblood foal with severe ESGD, complicated by gastric stenosis.

Case history

A 2-month-old Dutch Warmblood colt was presented to the Equine University Clinic of the Faculty of Veterinary Medicine of Utrecht University. The foal's dam was 16 years old and had previously had six foals, none of which had suffered any significant health problems. This foal was born after 331 days gestation, and no complications had occurred during foaling. The owners did not think the mare had suffered any stressful events or illness during gestation and had not observed any signs of disease in the foal during the first weeks of life. The foal had been dewormed with ivermectin 0.2 mg/kg at the age of 14 days and with fenbendazole 7.5 mg/kg a week before referral. The owners reported normal nursing behaviour, but the foal had refused to eat roughage and ate foal pellets slowly. The day before the first clinical signs were observed, the diet of the mare and foal were changed from pasture only to pasture plus haylage and pellets.

The foal had suffered an episode of colic and anorexia and had shown abnormal extension of the neck, 1 week before presentation. These signs had resolved after a single intravenous administration of butylscopolamine 0.2 mg/kg and metamizole 25 mg/kg (Buscopan Compositum, Boehringer Ingelheim) and flunixin-meglumine 1.1 mg/kg (Meflosyl, Zoetis). On the day of presentation to the Equine University Clinic, signs of colic and neck extension were reported to be more severe than observed previously and did not resolve with repeating the previously described medical treatment. Nasogastric intubation was performed by the referring veterinarian without difficulty, and approximately 2 L of reflux, consisting of milk and dissolved pellets, was obtained.

Clinical findings

On presentation, the foal was dull but nursing, and nasal discharge, containing food particles, was evident. The respiratory rate was 16 breaths/min, heart rate was regular at 60 beats/min, and the rectal temperature was 37.9°C. The foal showed no signs of dehydration, and auscultation of lungs and heart revealed no abnormalities. The abdomen was slightly distended, and gut sounds were decreased in frequency. Joints were not distended, and the foal was not lame. A nasogastric tube was passed, and 1 L of milk, with dissolved pellets, was obtained. The results of venous blood-gas analysis were unremarkable. The serum amyloid A (SAA) concentration was 6 μg/ml (ref <20 μg/ml). Haematology or further serum chemistry analysis was not performed.

Diagnosis

A transcutaneous abdominal ultrasound examination was performed using a 5–2 MHz curved-array transducer (Philips Lumify). There was no peritoneal effusion, several slightly distended loops of small intestine were visible, with normal motility, and the stomach could be visualised in intercostal spaces 7 to 16 on the left side. Gastroscopy was performed using a 1.80 m long endoscope (Storz). Severe ulceration of the squamous mucosa was noted, and the mucosa at the level of the margo plicatus appeared swollen and haemorrhagic (**Fig 1**) and the stomach seemed to have an abnormal anatomy. Some food particles and fluid were still present in the stomach, preventing visualisation of the entire stomach, and the pylorus could not be viewed.

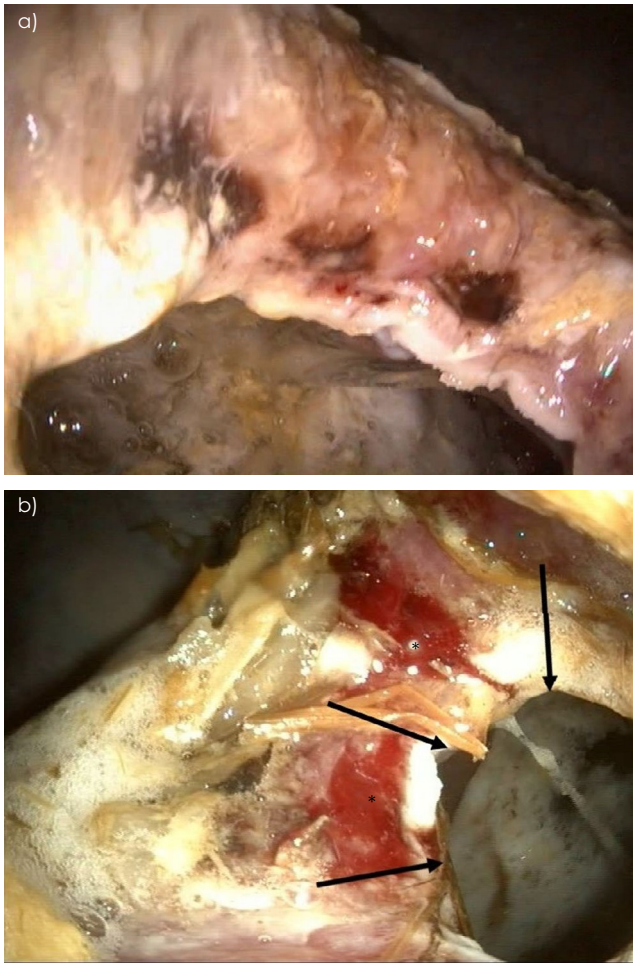

Fig 1: Gastroscopic images of the margo plicatus of a 2-month-old Warmblood foal, at admission (a) and after 6 days of treatment (b), showing severe ulceration of the squamous mucosa, with a swollen and haemorrhagic appearance. The mucosa was very fragile and bled easily after the endoscope touched the mucosa (*). There appeared to be a stricture at the level of the margo plicatus (arrows).

Following 6 days of treatment, gastroscopy was repeated after 12 h of fasting and no access to milk or water for 3 h. The stomach was emptier than during the previous gastroscopy, allowing better inspection of the entire stomach. The ulcers had partially healed, but bleeding occurred whenever the endoscope touched the ulcerated mucosa. There appeared to be a stricture at the level of the margo plicatus. The endoscope could easily be passed through the constricted portion of the stomach. The corpus of the stomach appeared normal, and the glandular mucosa was intact and normal in appearance. The pylorus appeared to function normally, and no evidence of an outflow obstruction was observed.

To further evaluate the stomach and to confirm the diagnosis of gastric stricture, contrast radiography was performed immediately after the second gastroscopy. Barium sulphate (30 ml in 2 L of water) was administered through a nasogastric tube into the cervical part of the oesophagus. Serial standing lateral radiographs were taken, several minutes apart, before and starting immediately after administration of the barium. Images showed the presence of barium in the stomach and revealed a distinct constriction in the horizontal plane in the middle of the stomach (**Fig 2**).

Treatment and outcome

The foal was treated with oral omeprazole (4 mg/kg q 24 h; Gastrogard, Boehringer Ingelheim) and was allowed to nurse, to drink water and to eat a limited amount of a watery mash. Intake of roughage or bedding was prevented by the use of a muzzle that was removed every 2 h to allow supervised nursing. On this regime, the foal nursed well, no extension of the neck or low head carriage were seen, and no spontaneous nasogastric reflux was present. The foal did not pass any faeces during the first 24 h of hospitalisation.

After the second gastroscopy, one of the options suggested to the owner was to continue medical treatment of the gastric ulcers, combined with dietary changes (milk, water, and watery mashes). Surgical intervention was discussed but not pursued by the owners of the foal. The prognosis was considered to be poor, both for a normal life and future athletic performance, and the owners agreed to euthanasia.

Post-mortem findings

Macroscopic evaluation of the stomach in situ revealed a severely reduced diameter at the level of the margo plicatus, dividing the stomach into a smaller orad (squamous), and a larger aborad (glandular) compartment (**Fig 3a,b**), this disproportion may have been the result of delayed gastric emptying. Both compartments were moderately gas distended. There was severe ulceration of the squamous mucosa at the level of the margo plicatus, with multifocal, coalescing, dark red, depressed ulcers that extended into the submucosa, with smooth, firm, white edges (fibrosis/granulation tissue, **Fig 3c**). As a result of the stenosis, the gastric lumen in this region was reduced to approximately 3 cm in diameter. Macroscopically and microscopically, no pathological changes were visible in the oesophagus, glandular mucosa of the stomach or duodenum.

Microscopic evaluation showed multifocal thickening of the squamous epithelium, adjacent to the margo plicatus, with abnormal, layered hyperkeratosis (**Fig 4**). There were multiple areas of ulceration along the margo plicatus, covered with amorphous eosinophilic debris, containing moderate numbers of, frequently degenerate, neutrophils (**Fig 5**). The submucosa was greatly expanded by large amounts of collagenous matrix with blood vessels oriented perpendicularly to the collagen fibres (granulation tissue). Within the muscularis, the myofibres were frequently separated by increased collagen deposition (**Fig 6**).

In summary, the stomach showed severe, chronic, nonperforating ulceration, hyperkeratosis and granulation tissue of the gastric mucosa at the level of the margo plicatus, leading to stenosis of the stomach.

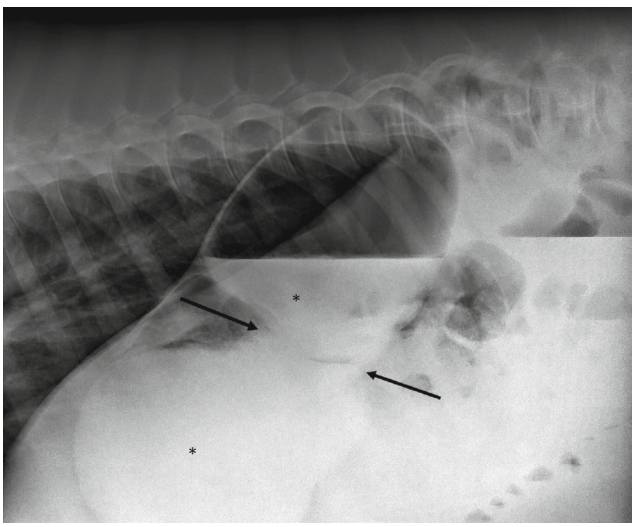

Fig 2: Gastrointestinal contrast radiography of a 2-month-old Warmblood foal, showing the presence of barium (*) in the stomach and constriction in the horizontal plane in the middle of the stomach (arrows).

Discussion

This case report provides a description of the clinical investigation and post-mortem findings in a case of severe ESGD, complicated by gastric stenosis at the level of the margo plicatus, in a 2-month-old Warmblood foal. To the authors' knowledge, this is the first description of a foal with such a gastric stricture.

The stricture found in the stomach of this foal was most likely caused by severe squamous gastric ulceration at the level of the margo plicatus, leading to chronic inflammation and granulation tissue formation. During the healing process, fibrosis and scar tissue possibly developed, leading to stricture formation. A developmental abnormality, as reported by Sárdi et al. (2013), with the apparent absence of squamous mucosa in the stomach, was considered to be less likely considering the histological findings. No known risk factors for ESGD development were considered to have been present in this foal. There was no history of illness or treatment with nonsteroidal anti-inflammatory drugs, which may contribute to ESGD (Castagnetti & Mariella, 2015; Cook & Blikslager, 2015). It seems likely that the foal had had ESGD at the level of the margo plicatus for some time before obvious clinical signs became apparent. The foal only developed clinical signs when it started to eat significant amounts of fibrous food material, such as haylage, which could not easily pass through the constricted region of the stomach. No problems were seen when the foal was on a diet of milk, water and mashes, and ingestion of bedding was prevented. Fluids, such as milk and water, are not retained at the pylorus, as solid feed is, and normally pass the stomach in less than 2 h (Sprayberry, 2015). Barium contrast was used to confirm the clinical diagnosis. Radiographs can be repeated after 2 h to reveal delayed gastric emptying (Camacho-Luna et al., 2018). In the current case, the abdominal radiographs taken directly after barium administration already showed the

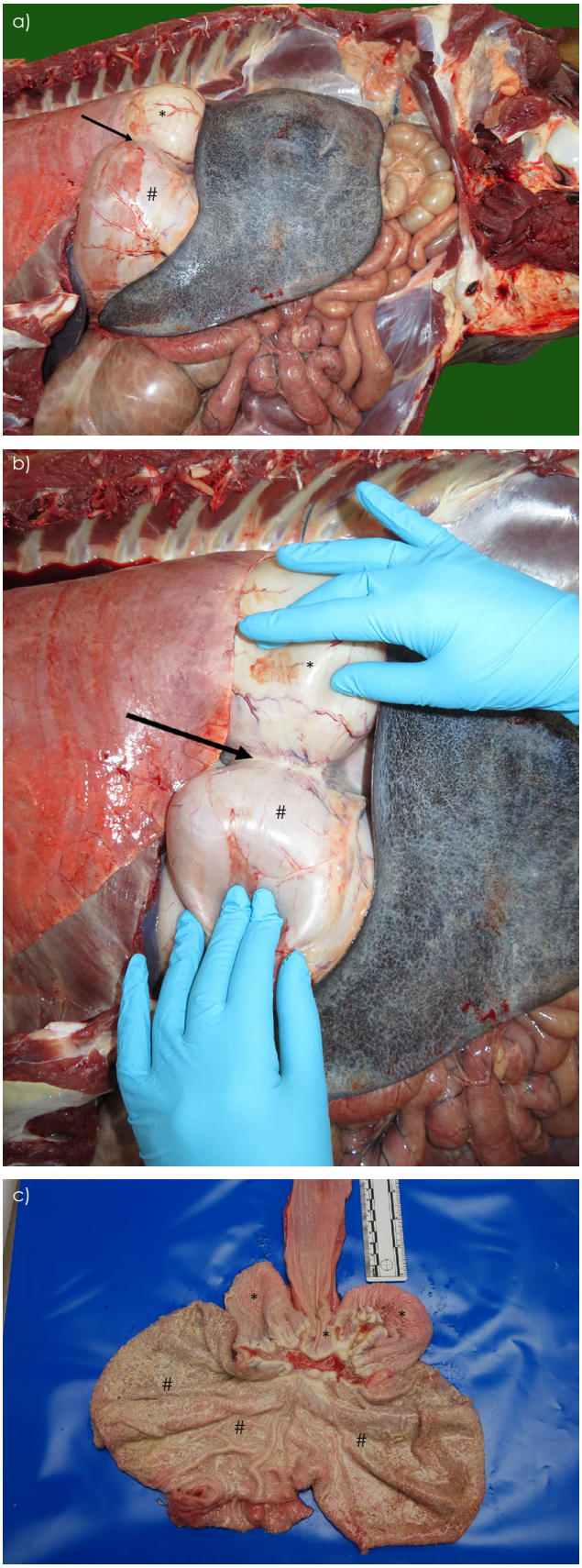

Fig 3: Post-mortem macroscopic evaluation of a 2-month-old foal showing a severely reduced circumferential diameter of the stomach at the level of the margo plicatus (arrow), dividing the stomach into a smaller (*) and larger (#) compartment (a and b). Severe ulceration and fibrosis with granulation tissue, at the level of the margo plicatus (c).

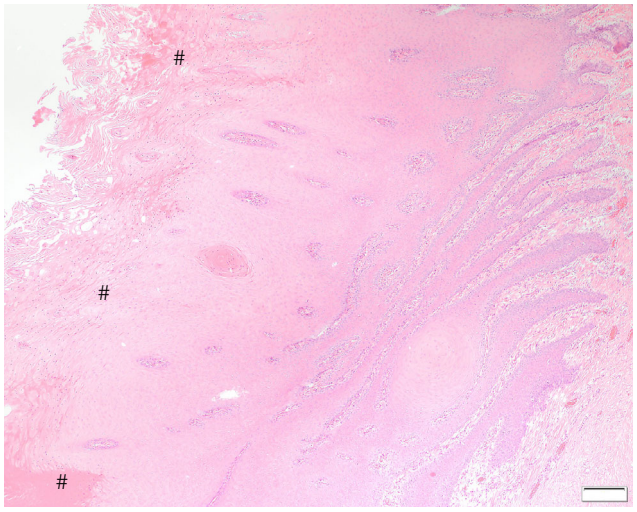

Fig 4: Stratified squamous epithelium, adjacent to the margo plicatus, showing orthokeratotic and parakeratotic hyperkeratosis (#). HE 40×. Bar = 200 μm.

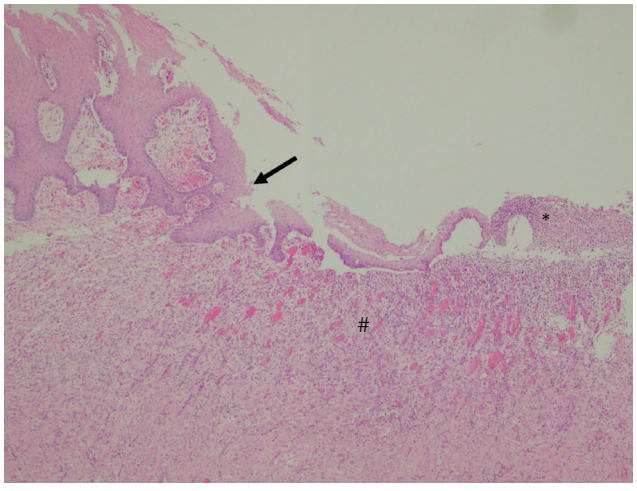

Fig 5: Erosion and ulceration of the squamous portion of the stomach: thinning and loss of stratified squamous epithelium (black arrow), covered with neutrophils and fibrin (*). The exposed lamina propria is expanded by granulation tissue, low numbers of mononuclear cells, few neutrophils and shows moderate hyperaemia (#). HE 100×. Bar = 100 μm.

abnormal shape of the stomach; therefore, no further images were taken.

It was considered unlikely that medical management of the current case would result in the foal being able to lead a normal life and consume a normal diet. Gastric bypass

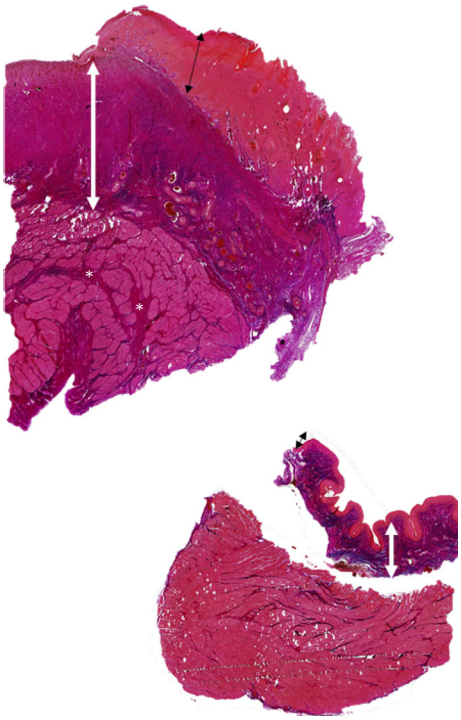

Fig 6: Van Giesson stain (to highlight collagen) of the affected area of the margo plicatus (above) and unaffected nonglandular stomach (below) showing the extent of the submucosal collagenous tissue (white arrow) and collagen deposition between myofibres of the muscularis layer (*). The thickness of the mucosa shows the extent of the hyperkeratosis (black arrow).

procedures, such as gastroduodenostomy or gastrojejunostomy, or pyloroplasty have been described in foals suffering strictures at the level of the duodenum, but these would not have provided relief in the present case, since the pyloric region was not affected (Kent et al., 2020; Zedler et al., 2009). Creating a bypass above the level of the stricture would have resulted in a very small functional stomach and feed not being able to pass through the glandular fundus. This might have had uncertain effects on the digestive process and the ability of the foal to consume adequate quantities of food. Also, surgical access would have been extremely challenging, with a high risk of contamination of the abdomen and the development of peritonitis.

Gastric stenosis in an adult horse has been described previously. In 1972, Peterson et al. described a case of gastric stenosis located at the proventricular part of the stomach, without involvement of the margo plicatus in an 11-year-old Morgan stallion. The origin of this stricture was not definitively determined, but given the histological appearance, gastric irritation or ulceration seem plausible. Since the horse did not show any signs consistent with gastric stenosis during the first 10 years of its life, a congenital cause was considered unlikely. Surgical treatment using a trans-thoracic technique was performed in this case, but the horse died as a result of severe post-surgical complications (Peterson et al., 1972).

In human medicine, various shapes and positions of the stomach have been described and may result from congenital or acquired conditions. Clinical signs in humans are similar to those exhibited by the foal in the present case: regurgitation and vomiting, or gastric reflux in the foal, and difficulty processing solid and/or liquid food. Acquired stenosis of the stomach can occur as a result of gastric ulceration or as a complication after laparoscopic sleeve gastrectomy in obese individuals (Burgos et al., 2013; Floate & Duggan, 1976) and results in delayed gastric emptying (Fukumoto et al., 1987). People have been successfully treated using endoscopic balloon dilatation of the stricture or corrective surgery (Burgos et al., 2013).

This case suggests that gastric stenosis is a rare complication of severe ESGD in foals and should be considered in a foal exhibiting gastric reflux and/or recurrent colic when eating roughage.

Authors' declaration of interests

No conflicts of interest have been declared.

Ethical animal research

This is a report of a case presented to the Equine University Clinic of the Faculty of Veterinary Medicine of Utrecht University. Informed consent was received from the owner for performing post-mortem examination and for the use of clinical data and images for research purposes and publication. The images were taken during the clinical and post-mortem examination.

Off-label antimicrobial use

None.

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Authorship

All authors have read and approved the manuscript, and all authors agree to the submission of the manuscript to the journal. All named authors have made an active contribution to the conception, design and the drafting of the paper, and all have critically reviewed its content and approved the final version submitted for publication.

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