Surgical treatment of gastric outflow obstruction in a five-week-old foal

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Introduction

Equine gastric ulcer syndrome (EGUS) is a common condition in foals, with a reported prevalence of 22–52% (Haggett 2020). This syndrome can lead to a variety of clinical presentations in foals. One sequala of this disease is gastric outflow obstruction. Gastroduodenal ulcer disease (GDUD) is one such EGUS condition and can lead to gastric outflow obstruction, most frequently affecting foals between two and six months of age (Elfenbein and Sanchez 2012). Severe cases of GDUD, causing obstruction, often necessitate surgical intervention (Zedler *et al*. 2009).

Various surgical techniques have been described for managing gastric outflow obstruction in foals and horses, including gastroduodenostomy, gastrojejunostomy (with or without jejunojejunostomy), pyloroplasty (Kent *et al.* 2009) and duodenojejunostomy (Auer *et al.* 2012). This case report details the clinical presentation, diagnostic approach, surgical technique, and outcome of a five-week-old foal with severe gastroduodenal ulceration and secondary gastric outflow obstruction.

Case presentation

An 80kg, five-week-old Thoroughbred filly presented with colic, ptyalism, and gastric reflux. The foal had a history of diarrhoea 10 days prior to admission. Before arriving at the hospital, the foal had been treated with dexamethasone (0.05mg/kg IV), meloxicam (0.6mg/kg IV), and butorphanol.

On admission, vital parameters were temperature 38.9°C, heart rate 84bpm, and respiratory rate 16bpm. Abdominal borborygmi were quiet but present in all four quadrants. Bruxism was noted, but no other overt colic signs were observed. The foal appeared dehydrated, with tacky, injected mucous membranes. Dried faecal staining was present, but no faecal output was noted prior to surgery.

Diagnostics

Blood analysis

A complete blood count (CBC), biochemistry panel, blood lactate and glucose were performed. The CBC revealed a marked leucocytosis ($36.2x10^{9}$/, reference value: $6.0\text{-}12.0x10^{9}$/L) and severe neutrophilia of ($32.75x10^{9}$/L, reference value: $2.7\text{-}6.7x10^{9}$/L). Mild hypoalbuminaemia of 23g/L (reference value: 30-36g/L) was noted (Knottenbelt and Malalana, 2014). The foal's lactate (3.8mmol/L) was consistent with dehydration. No other significant abnormalities were noted on laboratory parameters including blood glucose.

Nasogastric intubation and gastroscopy

Nasogastric intubation retrieved 2.2L of foul-smelling, haemorrhagic reflux suggestive of gastric outflow obstruction and mucosal compromise.

Gastroscopic examination revealed extensive oesophageal ulceration, with linear striations affecting the entire oesophageal mucosa and multifocal areas of hyperaemia, consistent with severe oesophagitis.

Initially the stomach could not be visualised due to excess food material. However, repeat gastroscopy demonstrated Grade IV squamous ulceration with sloughing of mucosa and haemorrhagic ulceration at the level of the lesser curvature of the stomach. The glandular mucosa was hyperaemic, and the pylorus showed a mild focal hyperaemic flat region with absent motility.

Abdominal ultrasonography and contrast radiography

Abdominal ultrasonography revealed a markedly distended stomach extending over ten intercostal spaces. The duodenal wall was thickened (4mm), with hyperechoic regions suggestive of mucosal necrosis or fibrosis. The remaining small intestine appeared normal, with adequate motility.

Contrast radiography was performed using Iohexol contrast medium (40ml omnipaque), administered via nasogastric intubation. A series of standing lateral and dorsoventral radiographs were obtained at 10-, 30-, and 90-minutes post contrast administration. The contrast remained in the stomach at 90 minutes post administration, confirming delayed gastric emptying (Blikslager *et al***.** 2017). The diagnostic findings confirmed severe gastric ulceration and strongly support a diagnosis of secondary gastric outflow obstruction and reflux oesophagitis in an immunocompromised foal.

Preoperative stabilisation

Medical stabilisation was initiated upon admission, with surgical intervention performed 24 hours later. The foal was rehydrated with a 2L Hartmann's intravenous (IV) bolus. Antibiotics were initiated based on laboratory findings and for preoperative prophylaxis (5mg/kg Ceftiofur IV BID and 6.6mg/kg Gentamicin IV SID). There were no signs of colic and lactate improve to 1.2mmol/L.

To prevent further gastric distension, the foal was muzzled and withheld from nursing. A 2L Hartmann's IV fluid bolus was administered every four hours to maintain hydration. Gastric decompression was performed at four-hour intervals, retrieving reflux volumes ranging from 200ml to 2L. Sucralfate (20mg/kg) was initiated as a gastroprotectant.

Surgical intervention

Prior to surgery, the foal received 1L IV of commercial plasma due to mild hypoalbuminaemia, intravenous antibiotics (as stated above), nonsteroidal anti-inflammatory medication (meloxicam 0.6mg/kg IV), and tetanus antitoxin. Gastric decompression was performed immediately before induction and the nasogastric tube (NGT) was left in situ during surgery.

Under general anaesthesia in dorsal recumbency the surgical site was aseptically prepared for a ventral midline celiotomy. Upon abdominal exploration, a tight fibrous band was identified at the pyloric-duodenal junction, causing an outflow obstruction. The remainder of the gastrointestinal tract appeared normal. A pyloric bypass procedure was performed, consisting of a gastrojejunostomy and jejunojejunostomy, as described by Zedler *et al.* (2009).

Gastrojejunostomy was performed by side-to-side anastomosis, orienting a segment of the proximal jejunum (oral to aboral) along an avascular region of the caudal ventral stomach. The jejunum was first sutured to the stomach, followed by parallel incisions into both structures. The cut edges were apposed using an absorbable suture with a continuous suture pattern (Lembert and simple continuous). Jejunojejunostomy was performed by aligning two jejunal segments approximately 10cm oral and aboral to the gastrojejunostomy site in an oral-to-aboral orientation. A stapling device was inserted through two stab incisions in either jejunal segment, which were subsequently closed using absorbable sutures in a continuous pattern (Lembert and simple continuous). The mesentery adjacent to the gastrojejunostomy was secured to the surrounding mesentery and jejunum to prevent potential intestinal entrapment or displacement. The foal recovered from anaesthesia uneventfully.

Post-operative care

Intensive post-operative management began immediately after anaesthesia. The foal received a continuous rate infusion (CRI) of IV fluids with glucose and dextrose for nutritional support. Initially, a 5% glucose solution (200ml/hr) and 5% dextrose in Plasmalyte (200 ml/hr) were administered at 5ml/kg/hr for 26 hours. Nutritional supplementation was then discontinued as glucose levels were stable and enteral nutrition increased. Fluid therapy was reduced to 1.6ml/kg/hr of Hartmann's for 12 hours. Hydration was monitored via urine specific gravity, which remained normal.

Lidocaine CRI (0.05mg/kg/min) was administered for 38 hours for analgesia (Blikslager *et al.* 2017) and potential prokinetic effects (Torfs *et al.* 2009). Enteral nutrition was withheld for six hours post-operatively and gradually reintroduced with 50ml/hr of mare's milk via an indwelling NGT. NGT was removed the next morning. No reflux was observed, and the foal's condition improved. By three days post-operatively the foal was nursing and grazing, however long fibres were avoided.

Post-operative gastroprotection and anti-ulcer medications were administered. Oral sucralfate (20mg/kg PO q6hrs) was continued. Intravenous omeprazole (4mg/kg slow IV infusion) was started after surgery for three doses. Oral omeprazole (2mg/kg PO SID) and misoprostol (5mcg/kg PO BID) were introduced the day after surgery.

The foal had episodes of pyrexia, managed with meloxicam (0.6mg/kg IV SID). Two days after surgery, the foal developed thrombophlebitis. Injectable antibiotics were continued for three days, followed by a transition to oral antibiotics (Doxycycline 10mg/kg PO BID) due to the thrombophlebitis. The foal was discharged five days post-operatively with oral medications (doxycycline, omeprazole, sucralfate, and misoprostol) for continued management.

Outcome

The foal presented dull 11 days post-operatively and on veterinary examination 3L of gastric reflux was present. The following morning the foal had deteriorated, and euthanasia was performed. Postmortem examination revealed gastric rupture on the dorsal aspect of the stomach suspected secondary to ulceration. On post-mortem the duodenum was non-patent, and the pylorus was acting as a blind ending sac. No dehiscence was noted at the anastomosis site.

Discussion

This case highlights the importance of early diagnosis, treatment, and prevention of GDUD in foals. Foals with concurrent gastrointestinal disease, such as this foal with prior diarrhoea, are at higher risk (Elfenbein and Sanchez 2012). While the pathogenesis remains unclear, this case demonstrates that EGUD can be fatal, emphasising the need for prevention. Further research is needed to improve understanding and develop effective prophylactic strategies.

Recent studies indicate improved survival rates for foals undergoing bypass surgery for gastric outflow obstruction, increasing from 35% to 69% (Campbell-Thompson *et al.* 1986; Zedler *et al.* 2009). These improvements may be attributed to better case selection, refined surgical techniques, and enhanced postoperative care (Zedler *et al.* 2009). However, despite the increasing success of bypass surgery, the risk of postoperative complications remains significant. Long-term risks such as cholangiohepatitis, anastomotic stricture, gastric rupture, and peritonitis must be considered (Coleman et al. 2009; Zedler *et al.* 2009).

In the most recent study, 76% of foals that survived long-term follow-up (>2 years) competed as racehorses (Zedler *et al*. 2009). However, there is limited literature examining persistent gastrointestinal disease and other long-term effects in these cases. Further research with extended follow-up periods is necessary to provide veterinarians and owners with a clearer understanding of long-term prognosis and management strategies.

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