Esophago-gastroduodenal ulceration complicated by duodenal perforation in a three year old Thoroughbred colt: a case report

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ABSTRACT: A three year old Thoroughbred colt was admitted to our clinic because of chronic inapetence, weight loss and exercise intolerance. Clinical examination, blood examination and endoscopy were carried out. The endoscopic appearance of the distal esophagus was characterized by extensive and severe ulceration. Gastroscopy revealed severe diffuse ulceration of the entire nonglandular mucosa. A diagnosis of esophago-gastric ulceration and delayed gastric emptying was made. Shortly after endoscopic examination the colt developed acute severe colic with profuse diarrhea and was euthanized. Necropsy and histopathology were performed. The most notable abnormalities were severe and extensive esophageal, gastric and duodenal ulcerations. One duodenal ulcer was perforated and acute septic peritonitis developed. The diagnosis was thus confirmed and extended – esophago-gastroduodenal ulceration with complication of duodenal perforation.

Keywords: equine gastric ulcer syndrome (EGUS); duodenal perforation; diarrhoea; peritonitis

Equine gastric ulcer syndrome (EGUS) is a well described common problem in foals and adult horses (Sanchez, 2004). Duodenal lesions may appear in association with gastric ulceration and can be complicated by duodenal perforation or strictures (Borrow, 1993; Sanchez, 2004). Diverse cases of duodenal perforation in foals have been described (Orr, 1972; Acland et al., 1983; Borrow, 1993). The prevalence of duodenal perforation in adult horses is not well documented, but it appears to be low. Duodenal ulcers in two yearlings have been described (Ettlinger et al., 1990). To our knowledge, no published cases of duodenal perforation in horses older than two years are available. The case described below in detail was a part of study published by Bezdekova and Hanak (2009).

The aetiology of gastroduodenal ulceration in horses is unknown. The main factors that have been proposed in the aetiopathogenesis of EGUS are stress (especially training), nonsteroidal anti-inflammatory drug administration, diet and feeding behaviour (Murray et al., 1996; Buchanan and Andrews, 2003; Bezdekova et al., 2005).

Chronic inapetence, weight loss and exercise intolerance together with intermittent colic and poor quality of hair coat are other clinical signs characteristic of gastroduodenal ulceration in adult horses (Murray, 1992; Sanchez, 2004). Diarrhoea could accompany this syndrome in foals (Sanchez, 2004).

Clinical case

A three year old Thoroughbred colt with good deworming and vaccination history was referred to the Equine Clinic of University of Veterinary and Pharmaceutical Sciences Brno because of chronic inapetence, weight loss and exercise intolerance. The owner indicated that the colt was lying down more often in comparison with other horses. Clinical signs had been present for more than one

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year. The horse had been sporadically treated with a preparation containing pectin-lecithin-glycerol complex (Pronutrin; Boehringer Ingelheim Gmbh, Germany) without any obvious effect.

During clinical examination the colt was in poor body condition. The horse was slightly depressed with a reluctance to move. Rectal temperature was within reference ranges. Heart rate and respiratory rate were increased (62 beats/min, 28 breaths/min). The hair coat was glossy without any abnormalities. Mucous membranes were pink and capillary refill time was two seconds. No abnormalities were detected on auscultation of the lung fields. Gut sounds were present on both sides of the abdominal cavity dorsally and ventrally. Palpation of the xyphoid cartilage was painless. Rectal examination revealed a small amount of pasty faeces in the rectal ampoule and an enlarged caudally displaced spleen. Rectal examination was not well tolerated by the horse and the horse showed discomfort during palpation. Abdominocentesis was not performed. The rest of the clinical examination did not reveal any other pathological findings. Haematologic analysis showed a leucocytosis (18.2×10^9/l). PCV and other haematologic parameters were within reference ranges. Routine biochemical analysis was normal.

At the time of admission the colt had been withheld feed for 14 hours for gastroscopic examination. Endoscopy (videoendoscope Dr.Fritz, length 330 cm, diameter 13 mm, Munich, Germany) was performed after sedation with xylazine (0.5 mg/kg *i.v.*) (Rometar 2% inj. ad us. vet., Spofa PLC, Czech Republic). Endoscopy of the nasal passages, nasopharynx, larynx and proximal esophagus did not reveal any abnormalities. The endoscopic appearance of the distal esophagus was characterised by extensive and severe ulceration. The colt showed discomfort during the passage of the endoscope through this part of the esophagus. The endoscopic appearance of stomach was characterised by severe diffuse ulceration of the whole nonglandular mucosa. The dorsal part of the glandular mucosa adjacent to the *margo plicatus* showed linear haemorrhages. No ulceration was visible on the glandular mucosa along the *margo plicatus*. Residual feed and fluid was present in the ventral part of the stomach and precluded endoscopic examination of the entire glandular mucosa, pylorus and proximal duodenum. The endoscopic finding was categorised as grade 4 according to the Equine Gastric Ulcer Council lesion scoring system (Sanchez, 2004). As a result of the endoscopic mucosal findings and the finding of a large amount of residual feed after 14 hours without feeding a diagnosis of esophagogastric ulceration and delayed gastric emptying was made. Six hours after endoscopic examination the colt showed acute severe colic with profuse diarrhoea. Because of its state of deterioration and unfavorable prognosis the colt was euthanazied.

Post mortem

Necropsy was performed. The abdominal cavity contained approximately 10 litres of predominately clear peritoneal fluid. There was minimal intestinal content on the peritoneal surface. The most notable abnormalities were severe and extensive esophageal, gastric and duodenal ulceration complicated by duodenal ulcer perforation. The stomach contained approximately 2 kg of fine-grained sand. The esophageal mucosa was ulcerated in the distal 25 cm of its length. Individual ulcers were 1–5 mm in size. The nonglandular mucosa was diffusely ulcerated. The glandular mucosa along the *margo plicatus* contained multiple linear haemorrhages. A large pyloric ulcer was noted (3 × 3 cm). This ulcer caused pyloric stenosis. Multiple small ulcers were evident in the proximal part of the duodenum. The duodenal mucosa was ulcerated along the whole length (85 cm) with single circular and oval ulcers. Perforation of the biggest duodenal ulcer (length 2.5 cm) was found 20 cm distal to the pylorus. There was a fibrinous adhesion on the serosal side of the bowel. The rest of the gastrointestinal tract including the right dorsal colon was without pathological changes. No other pathology was found during *post mortem* examination. The diagnosis of esophago-gastroduodenal ulceration with the complication of duodenal perforation and following peritonitis was confirmed.

Histopathology

Histopathology was performed. The esophageal ulcers extended into the *lamina propria* and onto the internal tunica of the *muscularis mucosae*. Necrosis with mixed inflammatory cell infiltration was found. The external tunica of the *muscularis mucosae* of the esophagus contained deposits of calcification. Gastric ulcers of the nonglandular mucosa extended into the submucosa. There was loss of mucosal epithelium and eosinophilic de-

bris. Duodenal ulcers extended into the submucosa. Adjacent to the ulcers were diphteroid-necrotic inflammation and capillary thrombosis. Fibrin-necrotic enteritis was found in the duodenum. The perforated ulcer had a necrotic intestine wall with fibrin adhesion. Microbiological examination of the duodenal samples identified the presence of *Clostridium perfringens.*

DISCUSSION

Gastric ulceration of the glandular mucosa and duodenum occurs more often in suckling foals than in adults and perforation of gastric or duodenal ulcers is a possible complication of this disorder (Sanchez, 2004). In adults ulceration of the glandular part of equine stomach mucosa (especially the antrum and pylorus) is increasingly being identified (Murray et al., 2001; Begg and O'Sullivan, 2003; Bezdekova et al., 2007). In 1972 Orr reported perforation of a duodenal ulcer probably associated with Ascarid infection in a four month old colt. Duodenal strictures due to ulcerative duodenitis in two 18 months yearlings have also been described (Ettlinger et al., 1990). Borrow (1993) reported three cases of foals (seven days, two months and five months old) with gastroduodenal ulcers. Two of these foals developed perforation of the duodenum. Ackland et al. (1983) described strictures in the duodenum associated with intraabdominal adhesions together with cholangiohepatitis and pancreatitis in the foal. Cholangiohepatitis and pancreatitis and duodenal stenosis secondary to gastroduodenal ulceration in a two month old colt were also reported by Buote (2003). Exsanguination due to gastric ulceration in a nine week old foal has been described (Traub-Dagartz et al., 1985). No evidence of intestinal parasites, duodenal stricture, cholangiohepatitis, pancreatitis or severe intraabdominal bleeding was found in our case. To the author's knowledge, there is no published information about duodenal perforation in a horse of this age.

A history of chronic inapetence, weight loss and exercise intolerance together with other clinical signs are characteristic for gastroduodenal ulceration in adult horses (Murray, 1992; Sanchez, 2004) and these were all present in this case. Intermittent colic and poor quality of hair coat in association with EGUS are also described (Murray, 1992; Sanchez, 2004). The horse described here had a good hair coat and no history of colic, although frequent recumbency was reported. Spontaneous gastric reflux, bruxism and ptyalism all suggested gastroesophageal lesions (Sanchez, 2004). Although distal esofagitis was present in this case the symptoms described above were absent. Diarrhea is not typically associated with EGUS in adults, but gastroduodenal ulceration can occur concurrently with primary diarrhea (Sanchez, 2004). The condition of the colt worsened quickly and acute colic with profuse diarrhea followed gastroscopic examination few hours later. It is likely that the severe colic attack and acute diarrhea were a result of septic peritonitis development because of bowel perforation. Clinical signs of peritonitis in the horse include increased heart rate, diarrhea and reluctance to move in peracute and acute cases (Dyson, 1983; Dickinson, 2004). Pain, which was obvious during rectal palpation of the abdominal cavity in this case, could also be associated with developing peritonitis. Reluctance to move, depression and increased heart and respiratory rate could also be caused by inflammation in the abdominal cavity. Duodenal perforation followed by acute fibrinous peritonitis in foals has been reported (Acland et al., 1983). Ulcerative duodenitis in two yearlings with watery diarrhea has also been described. These horses were diagnosed and treated with proximal enteritis and a final diagnosis of ulcerative duodenitis was made on the basis of laparotomy following necropsy (Ettlinger et al., 1990). There were no symptoms of proximal enteritis during clinical and pathological examination in this case.

The aetiology of gastroduodenal ulceration in horses is unknown. The main factors that have been proposed in the aetiopathogenesis of EGUS are stress (especially training), nonsteroidal anti-inflammatory drugs administration, diet and feeding behaviour (Murray et al., 1996; Buchanan and Andrews, 2003; Bezdekova et al., 2005). *Helicobacter pylori* has been demonstrated to be a major cause of gastric ulcers in man (Mertz and Walsh, 1991). Recently the question of the role of *Helicobacter* sp. infection in gastroduodenal ulceration in horses has been reopened (Scott et al., 2001; Contreras et al., 2007; Murray, personal communication). Typical histopathologic changes for *Helicobacter* spp. were not found in this case. No nonsteroidal anti-inflammatory drugs had been administrated in this case. Being a racehorse, it is possible to speculate on the influence of training stress. A high prevalence of EGUS in horses in training has been reported (Murray et al., 1996; Bezdekova et al., 2005). Stress

could accompany transport to the clinic together with withdrawal of feed in this case. However, the real cause of the ulcer disease remains unknown in this colt.

The endoscopic appearance of the esophagus and nonglandular mucosa showed diffuse extensive ulceration. Reflux esophagitis can occur along with EGUS, motility disorders, increased gastric volume from gastric outflow obstruction, gastric paresis, intestinal ileus and impaired lower esophageal sphincter function (Campbell-Thompson and Merrit, 1990; Vatistas et al., 1994; Bezdekova and Hanak, 2009). Spontaneous regurgitation causing esophageal ulceration is rare in horses (Laing and Hutchins, 2002; Baker et al., 2004). Flaccid lower esophageal sphincter (LES) in a horse with reflux esophagitis was described by Heidmann et al. (2004). The lower esophageal sphincter had usual appearance in this case. Gastro-esophageal reflux disease (GERD) in humans is caused by acid gastric juice irritation on esophageal mucosa but it is not accurate to compare the ethiopathogenesis of reflux esophagitis in horses and humans in consideration of horse stomach anatomy. Extensive nongladular ulcerations without esophageal lesions are usual in horses; however, reflux esophagitis usually occurs together with diffuse ulceration of the nonglandular mucosa (Sanchez, 2004). It is likely that chronic exposure of mucosa around the cardia to acid has a functional impact on the equine LES and gastroesophageal reflux doesn't result from mechanical distention (due to delayed gastric emptying) only and furthermore, that reflux esophagitis in this case was present as a consequence of the gastroduodenal ulceration.

Delayed gastric emptying may have resulted from mechanical obstruction because of the presence of the extensive pyloric ulceration. Pyloric ulceration is the most common way of acquiring this form of the disease in horses (Titus et al., 1972; McGill and Bolton, 1984; Church et al., 1986; Laing and Hutchins, 2002; Bezdekova et al., 2006; Bezdekova and Hanak, 2009). Heidmann et al. (2004) described a case of pyloric stenosis in a Thoroughbred filly where the passage of sand through the stomach may have produced inflammation with secondary fibrosis of the pylorus. Sand was found during *post mortem* examination in this case too. The pathogenesis of chronic sand irritation of pylorus could be the same. Another motility disorder was not identified.

The increased volume of peritoneal fluid could result from developing peritonitis following duodenal perforation. However, in spite of the duodenal perforation, minimal gastrointestinal contents were found in the abdominal cavity in this case. Intestinal perforation was localized in the proximal part of duodenum. This section descends along the right dorsal body wall before passing caudally around the base of the cecum. The relatively inaccessible and fixed location of the duodenum could be a reason for the limited development of peritonitis and presence of minimal abdominal contents in the abdominal cavity. Histopathologic examination confirmed the diagnosis and is similar to cases described by Borrow (1993). Emphysematous gastritis caused by *Clostridium perfringens* has been reported (Weldon et al., 1991). *Clostridium perfringens* was found in the duodenal samples of this horse, but *Clostridium perfringens* occurs also in the feces of normal horses. However, an association with enterocolitic disorders has been statistically confirmed (Weese et al., 2001).

Gastroduodenal ulceration in adult horses can be an obscure disease. Complete gastroscopy including pyloroscopy and duodenoscopy is needed in such cases. In this report we describe a three year old male horse diagnosed with severe ulceration of the proximal gastrointestinal tract complicated with duodenal ulcer perforation and subsequent peritonitis. History and clinical signs were characteristic for EGUS. Unusually diarrhoa was also present in this case. The etiology of pyloric ulceration and stenosis could be connected with sand presented in the horse's stomach. Duodenal perforation as a complication of EGUS is rare in adults and should be considered as a differential diagnosis in horses with acute diarrhea.

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