Pyloric ulceration and stenosis in a two-year-old thoroughbred filly: a case report

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ABSTRACT: The case of the two year old thoroughbred filly presented here has been diagnosed with and treated for equine gastric ulceration syndrome (EGUS). The results of her examination are documented over a five month period. The patient was finally hospitalised with acute severe colic. Absence of chronic clinical problems common for pyloric stenosis can be attributed to the permanent antiulceration medication of the filly. The diagnosis of pyloric stenosis was made by exploratory laparotomy and confirmed by necropsy.

Keywords: horse; pyloric stenosis; gastric ulceration; thoroughbred; colic

Pyloric stenosis is one of the less frequent diseases in horses (Sanchez, 2004; Venner, 2004). Congenital and acquired forms of this disease are described. Acquired form is usually caused by gastric ulceration and accompanied by a typical history and clinical signs (Venner, 2004). Pyloric stenosis commonly results in chronic deterioration in general state (Heidmann et al., 2004; Venner, 2004). The diagnostic process of pyloric stenosis *ante mortem* is difficult (Heidmann et al., 2004). Medical treatment of horses with pyloric stenosis usually leads to good clinical improvement while interruption of this therapy leads to the status rapidly worsening (Venner, 2004).

Clinical case

A two-year-old thoroughbred filly (weighing 362 kg) was admitted to the Equine Clinic (University of Veterinary and Pharmaceutical Sciences, Brno, Czech Republic) on 24th April 2004 with acute colic, which had started during the night prior to admission. The filly had a history of intermittent colics, indicated by the filly lying down, looking at her flank and periods of inappetence. Such problems had been reported not only for the last five months during the stay at the racing stables, but even in a group of yearlings. The referring veterinarian was unable arrive at the precise diagnosis.

The filly showed no signs of colic during physical examination at admission on 24th April 2004. The filly was in good body condition without changes in her general state of health. Her skin was dry with some excoriations in the supraorbital area. Temperature, heart rate and respiratory rate were within reference ranges. Peristaltic sounds were continuous through out all four quadrants of the abdominal cavity. Rectal examination revealed a medially and caudally displaced spleen and no other pathological findings. Haematological parameters were within reference ranges (Table 1).

The filly showed inappetence, and no signs of colic during four days of hospitalisation. As no obvious cause of colic was found, the filly was examined using gastroscopy. Gastroscopy revealed erosions and circular ulcerations of the nonglandular mucosa affecting area along *margo plicatus* as well as the whole area of *saccus caecus*. Profound linear

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Table 1. Results of haematology and acid-base examination

Day	24.4.2005	21.9.2005
Haemoglobin (g/l)	112	193
PCV (l/l)	0.3	0.56
Erythrocytes (10^{12}/l)	7.3	12.4
Leucocytes (10^9/l)	9	18.9
Platelets (10^9/l)	163	135
TPP (g/l)	50	68
Band neutrophiles (%)	0	2
Segmented neutrophiles (%)	57	91
Lymphocytes (%)	39	7
Monocytes (%)	4	0
Eosinophiles (%)	0	0
Basophiles (%)	0	0
Blood pH	7.412	7.421
Base excess (mol/l)	3.1	3.1
pCO_2 (kPa)	6.47	6.24
HCO_3 (mmol/l)	26.9	26.3

ulceration of the nonglandular mucosa was found in the lesser curvature of the stomach (Figure 1). The pyloric glandular mucosa was inflamed without any erosions or ulcerations (Figure 2). Therapy by cimetidine and omeprazole was initiated with respective doses of 7.7 mg/kg i.v. t.i.d. and 1 mg/kg p.o. s.i.d. for 16 days.

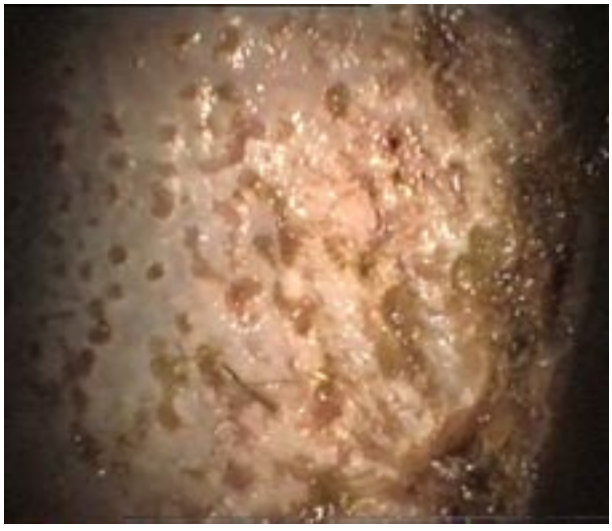

Figure 1. Endoscopic view of erosions and circular ulcerations of the nonglandular mucosa along *margo plicatus*. Linear ulceration of the nonglandular mucosa is seen in the lower right part of the picture (foto by Bezdekova, 2004)

The filly was discharged after five days of hospitalisation (on 28th April 2004) for treatment at home. She was presented for re-examination on 17th May 2004. The owner reported a mild increase of appetite and absence of colic symptoms. Repeated gastroscopy of the nonglandular mucosa revealed less frequent circular ulcerations, and no change of linear ulcerations along *margo plicatus*. There were no pathological changes of the glandular mucosa of *fundus* along the greater curvature of the stomach. As a result of these findings, the filly was hospitalised again and treated with cimetidine (7.7 mg/kg i.v. t.i.d.) for five days, and then with omeprazole (1 mg/kg s.i.d.) in combination with a preparation containing the pectin-lecithin-glycerol complex (300 g divided into two daily p.o. doses [Pronutrin; Boehringer Ingelheim]).

Further gastroscopy was performed on 26th May 2004, and there were only minor improvements found in the gastric mucosa. Therefore, the dose of omeprazole was increased to 2 mg/kg s.i.d. It was confirmed by gastroscopy on 4th June 2004, that the lesions of the cutaneous mucosa of the *saccus caecus* had healed completely. The linear ulceration along *margo plicatus* was smaller and showed signs of healing such as marginal epithelization. The treatment using a combination of omeprazole (2 mg/kg s.i.d.) with a preparation containing pectin-lecithin-glycerol complex was continued for another 8 days.

A further gastroscopy performed on 11th June 2004 showed healing lesions and linear ulceration of the nonglandular mucosa still present. As result of the improvement, the administration of omeprazole was discontinued. The filly was discharged on 13th June 2004 for treatment at home with only the prescribed preparation containing pectin-lecithin-glycerol complex. The pyloric part of stomach was only examined during the first session, and was not re-examined further.

During the following three months, the filly was in good body condition and in full training for her first race. She showed no signs of change in her general state of health or appetite. The treatment with a preparation, containing pectin-lecithin-glycerol complex, was discontinued on 17th September 2004. Four days later she was again found in the stable again showing colic symptoms. The severity of colic was indicated by multiple wounds on

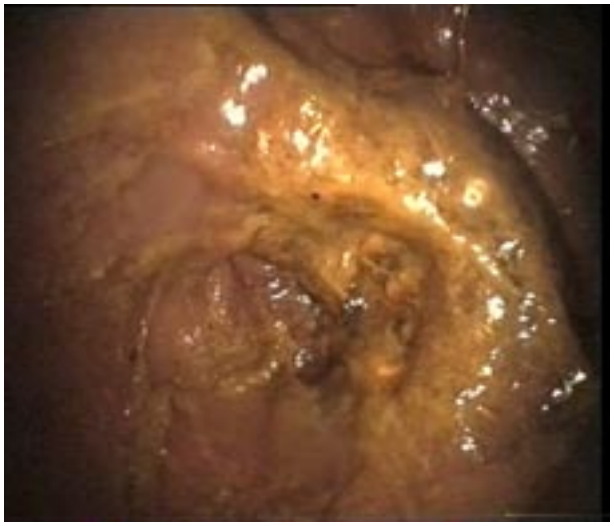

Figure 2. Endoscopic view of inflamed pyloric glandular mucosa (foto by Bezdekova, 2004)

her legs and head, together with a high pulse rate (80 beats per min).

Following treatment with butylscopolamine and flunixin-meglumine by a practicing veterinarian, she was referred again to the clinic. At admission, the filly was in racing body condition, weighing 348 kg. Her general body state had changed and the filly was apathetic, with no colic symptoms. Her skin was dry, with many excoriations, lacerated wounds and contusions on the legs and head. The pulse rate was 50 bpm, and the respiratory rate and rectal temperature were within reference ranges. The conjunctival and oral mucosa were clearly inflamed. Intestinal motility was decreased in all four abdominal quadrants. Rectal examination revealed an enlarged and caudally displaced spleen.

The rectal ampoule was filled with a small volume of soft faeces and the abdominal cavity was difficult to examine. The content of left colon was soft and small, and intestinal loops were not palpable. Rectal findings were comparable to that made five months earlier during the first hospitalisation of the filly. Insertion of a naso-gastric tube did not yield any gastric reflux. Transabdominal sonography revealed mild distention of the small intestine. Haematology and blood biochemistry findings showed a rise in the PVC, leucocytosis (Table 1), hypokalemia, an increased activity of muscle enzymes and higher level of bilirubin. As result of the recurrent colic, an exploratory laparotomy was performed on 21st September 2004. During surgery palpation of the pylorus revealed a markedly thickened wall with a prominence into the gastric lumen. There were small amounts of digesta and mild thickening of intestinal wall of cranial jejunum and duodenum, together with multiple haemorrhages; including the mesentery. No other pathological findings were noted.

The laparotomy finding confirmed the diagnosis of pyloric stenosis. After intraabdominal application of antibiotics (penicillin), heparin and dextran, the laparotomic incision was closed routinely. Usual postoperative care included parenteral hydration with 0.9% saline in maintenance doses and antibiotics (a combination of penicillin and streptomycin), neostigmine, heparin and flunixin-meglumine. As inflammatory aetiology of pyloric stenosis was suspected, postoperative therapy included cimetidine (7.7 mg/kg i.v. t.i.d.), and subsequently omeprazole (1 mg/kg s.i.d.). Intermittent inappetence was noted in the postoperative period. The filly produced soft faeces. The filly had mild

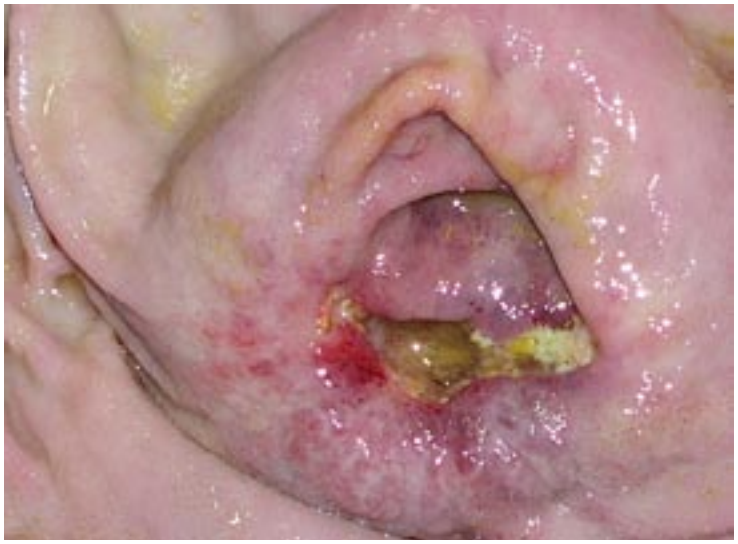

Figure 3. *Post mortem* view of pylorus with large chronic ulcer (foto by Bezdekova, 2004)

colic associated with feeding on the third postoperative day. Apathy and abdominal discomfort were repeatedly noted during the following days. Lack of response to the conservative therapy and poor prognosis resulted in the filly being euthanized ten days after laparotomy.

Post mortem

During *post mortem* examination, small amounts of solid feed were found in the stomach. The principle finding was a large (4.5 × 1 cm) pyloric ulceration with fibrosis of submucosa, causing pyloric stricture (Figure 3). Multiple erosions and ulcerations with hyperkeratosis were found in the nonglandular mucosa of the stomach. The oesophageal and duodenal mucosa were without any pathological changes.

Histopathology

Sites of ulceration of the nonglandular mucosa of the stomach could histologically be described as purulent inflammation. Histopathology of the pyloric ulcer confirmed severe neutrophilic infiltration and fibrosis of the submucosa. The stroma of duodenal villi from the proximal duodenum was infiltrated by plasmocytes. The final pathological diagnosis was *gastritis erosiva et ulcerative* and *duodenitis chronica.*

DISCUSSION

Pyloric stenosis is rather a rare disease in horses. It is more prevalent in young animals (Venner, 2004) such as our filly. Clinical signs such as ptyalism, bruxism, inappetence and colic symptoms are commonly present in affected animals (Sanchez, 2004). Signs of the disease are mostly progressive, and lead quickly to deterioration in the state of health of the animal. The evaluation period of patient with pyloric stenosis is dependent on the duration of the clinical signs associated with delayed gastric emptying, and is normally no longer than two weeks (Baker et al., 2004; Heidmann et al., 2004).

Our patient was examined regularly for a period of five months. The history of mild colic and inappetence dates from the post-weaning period and continued during her stay in a herd of yearlings. The syndrome of gastric ulceration affecting both types of the gastric mucosa was diagnosed during the first hospitalisation (on 24th April 2004). Pyloric passage seemed to be unaffected.

Examination of the pyloric area of stomach is a technically demanding process (Murray et al., 2001). It may be difficult to insert the endoscope into the pyloric anthrum due to digesta remaining in the stomach, a short endoscope, or lack of skill in gastroscopic procedures (Baker et al., 2004; Heidmann et al., 2004; Murray et al., 2001). The residual content of feeds in the stomach during repeated gastroscopy made it impossible to examine the pylorus and the proximal part of duodenum. As ulcerations of both types of the gastric mucosa are treated using the same medication, it was not expedient to examine the pylorus and duodenum each time.

Based on the gastroscopy, the filly was treated using H_2 receptor antagonist, an inhibitor of proton pumps, and stomach mucosa protecting substances. Omeprazole in the daily dose of 1mg/kg or 2mg/kg was found to have preventive antiulcerative effects in the horse. Therapeutic effect of these doses, however, has not yet been studied (White et al., 2003). The prescribed doses also depend on economic constraints. Even though the permanent administration of stomach protectants had been discontinued four days prior to the last hospitalisation of the filly with acute colic, a possible association of this discontinuation of treatment by preparation containing pectin-lecithin-glycerol complex with the subsequent colic is merely speculative.

As shown by repeated gastroscopies, the selected treatment promoted healing of the nonglandular mucosa. The lesions were not eradicated completely, although the number and affected area were reduced. Ongoing medication of the filly was also not successful in preventing such complications as the pyloric stenosis.

Pyloric stenosis may be either congenital or acquired. The congenital form of pyloric stenosis has been thoroughly studied in human twins (Szmytkowska, 2000), as well as other infants (Bodnar et al., 2002). It has also been reported in foals (Crowhurst et al., 1975; Barth et al., 1980). The onset of clinical signs later in her life excluded congenital form of pyloric stenosis.

The acquired form may be due to mechanical obstruction of the pyloric sphincter of inflammatory or neoplastic origin (Mackay et al., 1981; McGill and Bolton, 1984; Laing and Hutchins, 1992).

Reportedly, the most common tumour of the proximal gastrointestinal tract is classified as sq-

uamous cell carcinoma, originating from the non-glandular mucosa of the stomach and oesophagus and capable of producing metastases in later stages (Deegen and Venner, 2000). However, metastasizing tumorous diseases of the stomach are associated with severe changes in the general state of health of the horse (Tennant et al., 1982). Morse and Richardson (1988) reported an exceptional case of a hyperplastic polyp in the stomach of a horse leading to duodenal obstruction. Thickening of the pyloric wall was confirmed in our patient, who survived without significant clinical signs for a longer period. Therefore neoplastic aetiology of pyloric stenosis was less probable. The possibility of a tumour growing in the pyloric area was excluded definitively by histopathology.

Gastroduodenal ulcerations are the most common cause of pyloric obstruction and decreased gastric emptying in the horse (Titus et al., 1972; McGill and Bolton, 1984; Church et al., 1986; Laing and Hutchins, 1992). This syndrome is more frequent in foals less than four months old, rather than in adult animals (Moore and Watson, 1993). Apart from pylorus, sites predisposed for obstruction are cardia, pyloric anthrum and proximal duodenum (Orsini and Donawick, 1986). It is known that duodenal ulcers in two- to six-month-old foals may occur with a higher prevalence (Becht and Byars, 1986; Campbell-Thompson and Merrit, 1987).

Clinical signs associated with secondary obstruction in foals include depression, weakness and anorexia. Radiography may reveal gastric and oesophageal distension (Orsini and Donawick, 1986). Decreased gastric emptying related to gastric ulcerations in foals may also result in cachexia, poor hair coat, oral candidosis and gastro-oesophageal reflux (Becht and Byars, 1986). Our patient had a history of intermittent colic episodes during her early life.

Characteristic clinical signs are associated with pyloric stenosis in adult horses. Partial pyloric obstruction and the resulting decreased gastric emptying can be manifested as chronic weight loss, inappetence, colic symptoms after feeding and exercise intolerance (Venner, 2004). Serious complications of the gastric emptying failure in the horse are represented by the formation of erosions and ulcerations of the distal third of oesophagus, due to the gastro-oesophageal reflux (Baker et al., 2004; Heidmann et al., 2004). Characteristic clinical signs related to this reflux include ptyalism, bruxism and possible nasal discharge of gastric ingesta (Laing and Hutchins, 1992; Baker et al., 2004; Heidmann et al., 2004). Our patient had a history of inappetence and colic episodes prior to the first hospitalisation, but no clinical signs associated with gastric reflux during this and following period. According to the owner's information the filly was without any health problems and tolerated full training exercise well for three months. Nevertheless, at admission on 21st September she was 14 kg lighter in comparison to the first hospitalisation on 24th April 2004. It may, therefore, be supposed that signs characteristic of pyloric stenosis were suppressed by the permanent medication of the filly.

We suppose that the acute colic episode the filly was presented with during the last hospitalisation could be caused by acute gastric dilatation, due to pyloric obstruction together with mechanical participation of the pyloric ulceration and fibrosis. Acute gastric dilatation can be responsible for severe colic symptoms in horses (Todhunter et al., 1986). The filly had been treated by a practicing veterinarian with spasmolytics, which could relax the pyloric obstruction and cause the gastric contents to be moved into the proximal duodenum. Time of formation and duration of the pyloric obstruction could not be determined in our case. However, it cannot be excluded that the first colic on 24th April 2004 was primarily due to gastric distension too.

Exploratory laparotomy is considered to be a suitable procedure to diagnose pathological thickening of the stomach and proximal duodenum (Boy et al., 1992). The laparotomy performed in our patient excluded problems of the intestinal tract, and resulted in a definitive diagnosis. In respect of the possibility of conservative therapy and stabilization of patients with pyloric stenosis (Venner, 2004), the filly was treated with omeprazole in the postoperative period. However, the dose selected was not sufficient for the patient's stabilization. Gradual deterioration in her state of health and poor prognosis for athletic use were the reasons which lead the owner to decide for euthanasia. Surgical treatment of pyloric stenosis is considered risky and challenging (Venner, 2004). It was not included in the list of possible therapeutic measures in the case of this patient.

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