

Pyloric stenoses in horses: a seven case reports

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ABSTRACT: The seven cases presented here were diagnosed at the Equine Clinic, University of Veterinary and Pharmaceutical Sciences, Brno, Czech Republic between the years 2002 and 2008. The age range of affected horses was from 10 months to 10 years and included four Thoroughbreds and three warm blooded horses. The horses showed different clinical signs – recurrent or acute colic, poor appetite, frequent recumbency and poor performance. Physical examination revealed diverse results in each horse. Results of gastroscopic examination showed severe nonglandular mucosa ulceration and pathological changes along the pylorus in all horses. Two horses showed distal esophagitis. The duodenum was visible and endoscopically unchanged in two horses. Treatment was applied to four horses with different results. Follow up: Two horses are alive and they have partially or fully returned to their previous performance level. Five horses were euthanised because of clinical worsening and/or unsuccessful treatment. Four horses were examined *post mortem*. The final diagnosis of pyloric stenosis was made by gastroscopy in six horses. In the seventh horse it was confirmed by necropsy. Gastric or gastroduodenal ulceration was revealed as the cause of pyloric stenosis in five horses. In two horses the origins of pyloric changes were uncertain.

Keywords: horse; pyloric stenosis; gastric ulceration; colic

Pyloric stenosis is a rare disease in horses and other species (Sanchez, 2004; Venner, 2004). Sporadic cases have been described in horses (Barth et al., 1980; McGill and Bolton, 1984; Church et al., 1986; Morse and Richardson, 1988; Laing and Hutchins, 1992; Heidmann et al., 2004; Bezdekova et al., 2006). To our knowledge, a multiple study of pyloric stenosis in horses is missing from the annals of veterinary medicine. Affected horses are usually young (Sprayberry, 2003; Venner, 2004) but a breed predisposition has not been identified. The disease history is nonspecific and includes poor appetite, chronic weight loss, postprandial colic, exercise intolerance and frequent recumbency. Physical examination reveals poor body condition and depression. Clinical pathology is nonspecific, in some cases manifesting as low serum protein levels and metabolic imbalance. Usually treatment has only a temporary effect and long term follow ups are only partly successful (Sanchez, 2004;

Venner, 2004). Pyloric stenosis is formed by mucosal ulceration in most cases (Sanchez, 2004; Venner, 2004; Bezdekova et al., 2006).

MATERIAL AND METHODS

Seven horses with pyloric stenosis were diagnosed at the Equine Clinic in the University of Veterinary and Pharmaceutical Sciences, Brno in the Czech Republic between the years 2002 and 2008. The age range of affected horses was from ten months to ten years. Six horses were under three years of age. One mare was ten years old. Four thoroughbreds and three warm blood horses were included in this study. Four mares and three stallions were presented. The horses were reported to have manifested different clinical signs. A history of recurrent colic and poor appetite were presented in six

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horses. Five horses showed frequent recumbency and poor performance. One horse had poor hair coat whilst the remaining one displayed intermittent bruxism and salivation. Physical examination, laboratory examination (including hematology in all horses and blood biochemistry in two horses) and gastroscopic examination were carried out. A Dr.Fritz fibroendoscope (length 330 mm, diameter 13 mm) was used for gastroscopic examination. Food deprivation before endoscopic examination was prolonged from 16 hours to 30 hours in four horses because of the persistence of residual food in the stomach. Four horses were treated and in all cases a follow up was carried out. Four horses were examined by necropsy.

RESULTS

Physical examination revealed diverse results in each horse. Poor condition was found in five horses. Increased heart rate and respiratory rate were presented in three horses, whilst a reluctance to move was observed in two of them. Acute colic, acute diarrhoea or bruxism was found in one horse. Laboratory results (included hematological examination in all horses and biochemical examination in two horses) were normal, except for leucocytosis in one horse and an abnormal differential white cell count in another one.

The results of gastroscopic examination revealed severe ulceration on nonglandular mucosa in all horses. Two horses showed distal esophagitis (Figure 1). Lesions along the pylorus were visible in



Figure 1. Endoscopic view of distal esophagitis in a three year old gelding with pyloric stenosis

six horses. A single large ulcer covered with fibrin with thickened and reddened mucosa along the pylorus was present in four horses in the pyloric region (Figure 2). In two horses the pylorus had an unusual appearance. One horse had a rounded ulcerated thickening of the pylorus with polyp appearance (Figure 3). The other one had a nearly completely closed pylorus with thickened mucosa without superficial mucosal lesions (Figure 4). All horses examined endoscopically had impaired pyloric peristalsis. Endoscopic examination of the pyloric region in the last horse was not possible because of residual feed in the stomach. The duodenum was examined and endoscopically unchanged in two horses.

Treatment was applied to four horses. One horse has been administered with a preparation containing a pectin-lecithin complex (Pronutrin®). Two horses have been treated with omeprazol (Omeprazol®) (1–2 mg/kg s.i.d.) repeatedly and a pectin-lecithin complex continuously. The fourth horse has been treated intensively with omeprazol (1–2 mg/kg s.i.d.), a pectin-lecithin complex, sucralfate, doxycyclin, metronidazol and repeated ulcer debridement under endoscopic guidance. Two treated horses were euthanised because of clinical deterioration after six months. One treated horse was euthanised due to acute colic three months after the end of treatment and a pathomorphological examination was not carried out. One untreated horse was euthanised on the day of admission due to duodenal ulcer perforation and consequent septic peritonitis. One untreated horse was euthanised one week after diagnosis at the owner's request.



Figure 2. Endoscopic view of pyloric ulcers covered with fibrin in a three year old stallion with pyloric stenosis



Figure 3. Unusual polyp-like appearance of an ulcerated pyloric lesion in a 10 year old warm blooded mare

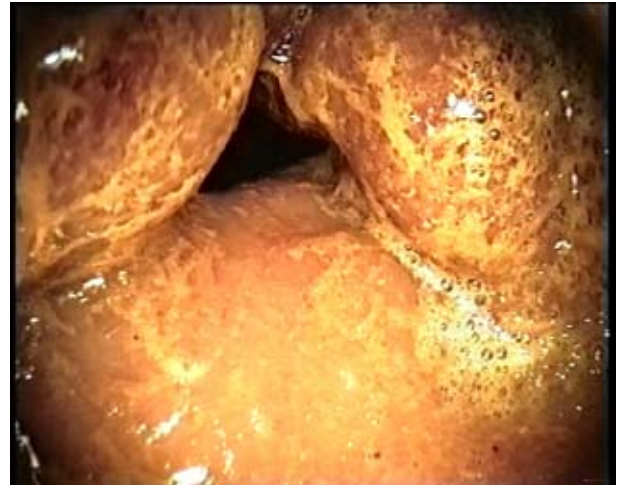


Figure 4. Unusual appearance of pylorus changed by mucosal thickening without superficial lesions in a three year old Thoroughbred mare

One untreated horse is alive and used for pleasure riding and one treated horse is alive and fully used in racing at the time of writing this paper. Four of the five dead horses with pyloric stenosis were examined by necropsy. The *post mortem* examination revealed chronic pyloric ulceration in all horses (Figure 5). Distal esophagitis was present in two horses. Severe nonglandular mucosa ulceration was found in all cases. Duodenal ulceration and duodenal ulcer perforation was presented in one case (Figure 6). Chronic inflammation in the pylorus was confirmed by histopathologic examination in all cases.

In five horses stenosis was caused by the formation of a pyloric ulcer. In two horses pyloric changes had uncertain origins. The final diagnosis of pyloric

stenosis was made in six horses through gastroscopy and in the seventh horse this was confirmed by necropsy.

DISCUSSION

Pyloric stenosis is an uncommon disease in horses. The seven horses diagnosed at the Equine Clinic in Brno represent 0.07% of all cases admitted between 2002 and 2008. This fact confirms the rareness of this condition in horses, although we have previously published such a case (Bezdekova et al., 2006). Affected horses are usually under five years of age (Venner, 2004). The horses in six of the seven cases described here were younger than

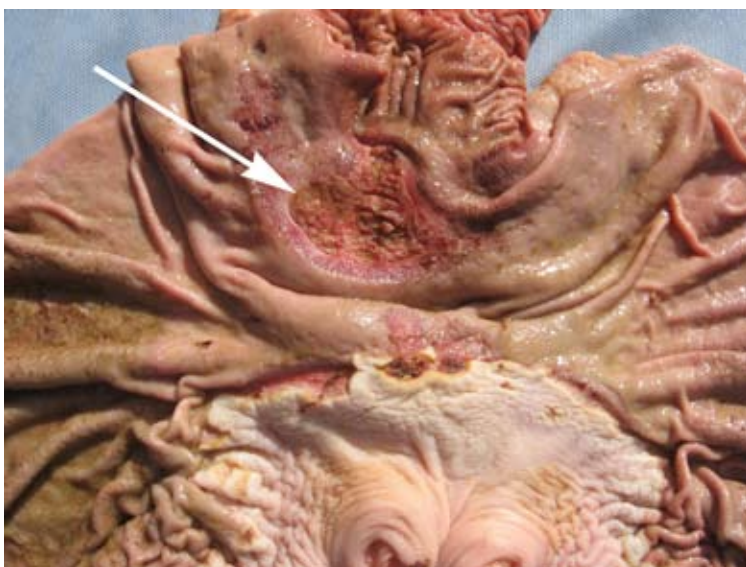


Figure 5. *Post mortem* view of pylorus with a large chronic ulcer in a two year old mare with pyloric stenosis



Figure 6. *Post mortem* view of ulcerated duodenum with ulcer perforation in a three year old gelding

three years old. Five of them were diagnosed with pyloric ulceration and showed clinical signs of delayed gastric emptying. Delayed gastric emptying which relates to the occurrence of pyloric stenosis has already been described in young horses (Sprayberry, 2003).

The history in our cases is similar to a previous report of Venner (2004). Recurrent colic and poor appetite were the most common problems, which relates to intermittent gastric dilation due to a partly or completely obstructed pylorus region. No difference between history in cases with pyloric stenosis and cases with gastroduodenal ulceration is usually presented except in more emergent (acute) histories in stenotic cases. Clinical signs such as the poor condition in all cases described here correlates with the chronicity of the disease. Acute diarrhoea was present in the case with a perforated duodenum and probably relates to septic peritonitis in this case. Bruxism and salivation were present in the case with distal esophagitis. Esophagitis is connected with these clinical signs (Heidmann et al., 2004; Venner, 2004). Gastroesophageal reflux is typical for pyloric stenosis, however it could be present also in different cases (Baker et al., 2004). Laboratory results were nonspecific in all cases. Venner (2004) describes hypoalbuminemia in such cases, which was not confirmed in our study. Unfortunately, only two horses were examined biochemically and the biochemical parameters obtained were not suitable for a full comparison.

Gastroscopic examination is the most important procedure for establishing a diagnosis. In one case, which was included in this study, the diagnosis of

pyloric stenosis was supported by the finding of thickening pylorus during exploratory laparotomy (Bezdekova et al., 2006). Distal esophagitis is a common finding in pyloric stenosis (Venner, 2004). It was confirmed in only two horses in this study possibly because of the slow progression of the disease. Severe chronic nonglandular mucosa ulceration was found in all cases. These changes are typical for pyloric stenosis, when food and gastric fluid stagnation causes chronic nonglandular mucosal irritation (Venner, 2004). Because of delayed gastric emptying, a prolonged period of food deprivation was necessary to enable visualization of the pylorus. In cases with advanced pyloric lesions it was not possible to pass the gastroscope through the pylorus because of narrowing and thickening of the pyloric mucosa. Duodenal lesions were presented in one case and they were found *post mortem*.

Treatment was applied to four horses. Satisfactory improvement in pyloric stenosis is described by Venner (2004). However the author recommends continuous treatment with omeprazol for a period of years which is not economically effective under our conditions. Our treatment experience was similar. The particular history of one long term treated case from this study has been described by us previously (Bezdekova et al., 2006). Under omeprazol treatment horses were stable, but clinical signs relapsed soon after withdrawal of the drug. Moreover, the dose of omeprazol used in our patients (1–2 mg/kg s.i.d.) is lower than the generally recommended dose (4–6 mg/kg s.i.d.), but to our experience still effective. White et al. (2003) described the use of a lower omeprazol dose, but

their study was focused on the prevention of gastroduodenal ulceration.

Five pyloric stenoses in this study were formed by mucosal ulceration. Detailed progression of the pyloric ulceration has been described previously in one case (Bezdekova et al., 2006). A pyloric ulcer was presented together with severe nonglandular ulceration in all cases. Pyloric ulceration is the main cause of pyloric stenosis in horses (Venner, 2004). Duodenal ulceration was present in one adult horse, which is unusual. The true cause of gastric ulceration remains obscure in horses (Sanchez, 2004).

Two horses with unusual pyloric appearance were not examined by necropsy. One survived and the other was euthanised out of the clinic. A congenital origin of pyloric stenosis is discussed by Barth et al. (1980). It is not possible to confirm this in our cases. At least in the ten year old mare from our study we can exclude a congenital form of the disease. Benign masses and polyps were previously described in the pyloric region in horses (McGill and Bolton, 1984; Church et al., 1986; Morse and Richardson, 1988; Laing and Hutchins, 1992). However, some of them were later diagnosed as chronic ulceration causing gastric wall thickening similarly to horses in our study (McGill and Bolton, 1984). It is possible that the ten year old mare included in this study was presented with a pyloric polyp or adenoma. In these cases proliferation of mucin glands is present (Murray, personal communication). Unfortunately a pathomorphological examination was not carried out and therefore this theory could not be confirmed. The role of sand irritation in pyloric stenosis origin was discussed by Heidmann et al. (2004). Pathomorphologic examination did not reveal the presence of sand in the proximal gastrointestinal tract in our cases.

The results obtained in our study point out the necessity for complete endoscopic examination of the stomach including the pyloric region when pyloric stenosis is suspected. Not all cases of pyloric stenosis in horses are caused by mucosal ulceration and atypical changes can occur. Treatment is partly successful, but the long term prognosis in cases of pyloric stenosis in horses is usually poor.

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