Clinical management of gastroduodenitis and equine gastric ulcer syndrome grade IV of unknown origin in an Arabian mare

Klinische benadering van gastroduodenitis en ernstige maagzweren van onbekende oorsprong bij een Arabische volbloed merrie

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ABSTRACT

A two-year-old Arabian filly was referred with symptons of colic. Clinical examination revealed signs associated with endotoxemia. Ultrasonographic examination of the abdomen demonstrated severe distention of the stomach and distended loops of small intestine with reduced motility. With nasogastric intubation, eight liters of hemorrhagic reflux were retrieved. Gastroscopic examination showed a severe degree of gastric ulceration scored as equine gastric ulcer syndrome (EGUS) grade IV. Based on these findings, EGUS and gastroduodenitis (GD) were diagnosed. Initially, the horse was treated using parenteral pantoprazole, which was supplemented by adding enteral ranitidine and sucralfate when the horse ceased refluxing. The horse was discharged eleven days after presentation with a 75%-healing of the gastric ulcers. A control gastroscopic examination was performed thirty days after discharge showing a 90%-healing (EGUS Grade I). To the authors' knowledge, this is the first case report in which pantoprazole in combination with ranitidine and sucralfate has been used to treat a severe degree of gastric ulceration.

SAMENVATTING

Een Arabische merrie van twee jaar oud werd doorverwezen naar Sharjah Equine Hospital met koliekverschijnselen. Op het klinisch onderzoek vertoonde ze tekenen van endotoxemie en echografisch onderzoek toonde een sterk opgezette maag aan met verschillende opgezette niet-motiele dunnedarmlussen. Tijdens een sondage van de maag werd er acht liter bloederige reflux bekomen. Op het gastroscopisch onderzoek werden ernstige maagzweren (graad 4) gevonden. Op basis van deze bevindingen werden "equine gastric ulcer syndrome" (EGUS) en gastroduodenitis (GD) gediagnosticeerd. Initieel werd het paard behandeld met parenterale pantoprazole. Nadat het paard geen reflux meer vertoonde, werd orale ranitidine en sucralfaat toegevoegd. Het paard verliet de kliniek na elf dagen, met een 75%-heling van de maagzweren. Dertig dagen later werd een controlegastroscopie uitgevoerd en hierbij was 90% heling zichtbaar (EGUS graad 1). Volgens de auteurs is dit de eerste casuïstiek waarin de combinatie pantoprazole, ranitidine en sucralfaat beschreven wordt voor het behandelen van ernstige maagzweren bij een paard.

INTRODUCTION

Gastroduodenitis (GD) is a gastrointestinal disease characterized by inflammation of the proximal part of the small intestine, colic, endotoxemia and nasogastric reflux (Freeman, 2000). This pathology is not just restricted to the small intestine, it may also affect other regions of the gastrointestinal tract including the stomach, pylorus and esophagus (White et al., 1987). In a review of the literature on the prevalence of GD in colic cases involving the small intestine, rates between 3 and 22% have been reported (Freeman, 2000). Typical lesions in horses with GD include ulceration of the mucosa of the proximal small intestine, which are often accompanied by lesions in the stomach consistent with ischemia-induced injury (Arroyo et al., 2017).

Equine gastric ulcer syndrome (EGUS) is de-

scribed as gastric ulceration in the horse (Andrews et al., 1999) and has recently been divided into two new syndromes called equine squamous gastric disease (ESGD) and equine glandular gastric disease (EGGD), the aim being to delineate the anatomical region affected (Sykes et al., 2015a). EGUS is very prevalent in performance horses, especially those in intensive training or active competition; a prevalence as high as 100% in such horses has been reported (Murray et al., 1996). However, EGUS has also been reported in untrained horses, pleasure horses and horses that rarely compete (Hartmann and Frankeny, 2003; Luthersson et al., 2009; Chameroy et al., 2006).

In the present case, the clinical management of severe GD and EGUS of unknown origin is described in an Arabian mare, using a previously undescribed combination of pantoprazole, ranitidine and sucralfate.

CLINICAL CASE

History and clinical signs

A two-year-old Arabian filly used for Arabian horse showing, was referred to the hospital with clinical signs of colic (restlessness, pawing at the ground, sweating and increased respiration rate) of a five-hour duration. She had not shown any colic signs for the past six months, during which her appetite and defecation had been normal. The horse had a body condition score of 4/5 (Carroll and Huntington 1988) and a history of frequent deworming, dental checks and moderate training exercise prior to showing. The horse was receiving 8 kg of grain daily, divided over four meals, being complemented with 8 kg of timothy hay divided over three meals.

Clinical findings and treatment

On presentation, the horse was showing signs of mild abdominal pain, had a heart rate of 90 beats per minute (bpm), respiratory rate of 28 breaths per minute (brpm), and a temperature of 38°C. The mucous membranes were congested and showed a capillary refill time of three seconds. Gastrointestinal borborygmi were absent in all the abdominal quadrants and digital pulses were absent in all the limbs.

Rectal palpation revealed mildly distended small intestinal loops in the right cranio-ventral area, and caudal displacement of the colon with some content and gas in the pelvic inlet. Ultrasonographic examination of the abdomen revealed stomach distention with a fluid line which could be identified between the 14th and 15th intercostal space, and a distended small intestine with reduced motility in the caudo-ventral region was seen. No free peritoneal fluid was observed. Nasogastric intubation gave eight liters of hemorragic reflux, which contained some undigested food (Figure 1). A blood sample and gas analysis revealed hemoconcentration (61%), metabolic acidosis (pH 7.25), hyperlactemia (6.3 mmol/L), and an anion gap of 22 mmol/L. Potassium, chloride and sodium levels were within normal ranges. Hematological and chemical examinations showed normal white blood cells, fibrinogen, creatinine and blood urea nitrogen levels, whereas the triglyceride concentrations were elevated at 422 mg/dL (normal range 17.7 -106.2 mg/dL).

Gastroscopic examination showed severe diffuse hyperemia of the dorsal squamous fundus with small areas of sloughing epithelium (Figure 2A). In addition, some extensive necrotic areas showed severe edema and thickening of the epithelium. Lesions with severe hyperkeratosis and multiple bullae formation of different sizes were also present in the squamous area alongside the margo plicatus (Figure 2B). The severity of the lesions was scored as EGUS grade IV (Andrews et al., 1999). The glandular region of the stomach showed some areas of hyperemia, but no obvious ulceration was observed.

As the horse was not showing active signs of colic after decompression of the stomach, medical treatment was initiated. A catheter was placed in the right jugular vein and the horse was given an initial intravenous bolus of two liters of hypertonic saline solution (Hypertonic Saline Solution 7.2%, Wellpharma Medical Solutions, Abu Dhabi, UAE) followed by Ringer's Lactate solution (Ringer's Lactate; Wellpharma Medical Solutions, Abu Dhabi, UAE) administered at a maintenance fluid rate of 3 ml/kg/day supplemented with 500 ml of 23% calcium borogluconate (C-B-Gluconat 24% plus 6%; Bela-pharm, Vechta, Germany). The stomach lesions were treated by intravenous administration of pantoprazole (Pantozol; Takeda GmbH, Konstanz, Germany) 1.5 mg/kg BW every 24 hours, diluted in 0.5 liters Ringer's Lactate solution and infused over ten minutes. This daily therapy was continued until the horse was discharged. In addition, the horse received quarter doses of flunixin meglumine (Banamine, MERCK Animal Health, Madison, New Jersey, USA) 0.27 mg/kg BW i.v. every six hours. Gastric decompression was performed periodically but only small amounts of reflux (≤ four liters) were obtained. It was therefore decided to administer the prokinetic, metoclopramide (Premosan; Gulf Pharmaceutical Industries, Ras Al Khaimah, UAE) subcutaneously at a rate of 0.04 mg/kg BW every six hours to stimulate intestinal motility and gastric emptying.

On day 2, the horse began to show an increased digital pulse in both front limbs and was reluctant to move. Radiographs revealed a mild degree of pedal bone (P3) rotation in both front feet (3° in the right front P3 and 4° in the left front P3). Supportive laminitis treatment was therefore commenced by administering pentoxifylline (Pentoxifylline; Apotex Corp, Weston, Florida, USA) 7.5 mg/kg BW per os every

twelve hours, applying digital cryotherapy on both front feet, and giving intravenous dimethyl sulfoxide (DMSO, Valhoma Corporation, Tulsa, Oklahoma, USA) 1 g/kg BW diluted in five liters Ringer's Lactate solution every twelve hours. Frog support was provided by applying removable strap-on hoof boots with foam rubber pads (Soft-Ride; Soft-Ride Inc, Bacliff, Texas, USA).

As no reflux was obtained from the stomach on day 4, the prokinetic medication was discontinued, and medication was given enterally: ranitidine (Ranitidine; Strides Shasun Limites, Puducherry, India) was administered orally using a dose of 6.6 mg/kg BW every eight hours, along with sucralfate (Sucralfate; Greenstone LLC, Peapack, new Jersey, USA) 20 mg/kg BW every six hours, to the gastric treatment.

The horse's condition improved, so the fluid therapy was discontinued and feeding was initiated.

On day 7, radiographs of both front feet revealed an increase in the degree of rotation of both pedal bones (4° in the right front P3 and 7° in the left front P3). Flunixin meglumine was replaced by phenylbutazone (Ilium Nabudone P; Troy Laboratories PTY. Limited, Glendenning, Australia) 2 mg/kg BW i.v. every twelve hours, plus acepromazine (Acepril-10; Troy Laboratories PTY. Limited, Glendenning, Australia) 0.03 mg/kg BW I. M. every eight hours. The horse started to be more comfortable and less reluctant to move. Blood analysis showed the triglyceride levels had declined to 312 mg/dl (normal range 17.7-106.2 mg/dL). Control gastroscopy revealed a significant improvement of the squamous region with a mild degree of hyperemia and decent integrity of the mucosa (Figure 3A) except for a band of ulceration adjacent to the margo plicatus extending from the greater to the lesser curvature (Figure 3B).

On day 11, gastroscopy showed an obvious improvement. The healing rate was at 75% and the dorsal squamous fundus had healed completely (Figure 4A). Nonetheless, a portion along the margo plicatus still showed a band of ulceration with severe loss of mucosal integrity, although there was an obvious ongoing healing process indicated by contraction of the borders (Figure 4B). The horse was discharged with diet recommendations that included ad libitum rough-

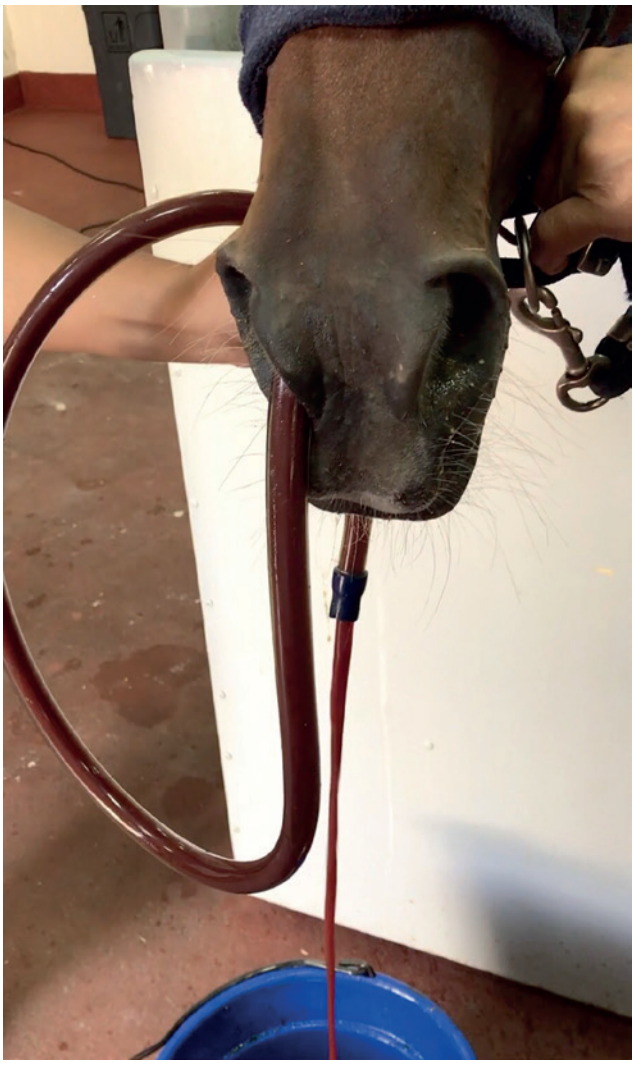

Figure 1. Nasogastric intubation at presentation giving a copious amount of hemorragic reflux.

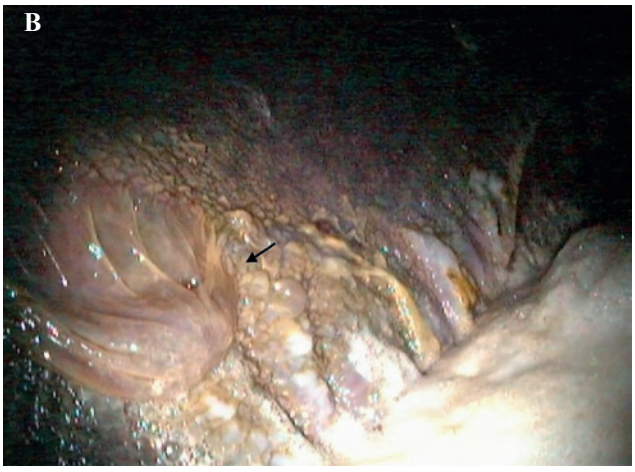

Figure 2. Gastroscopy on day 1 at presentation showing: A. The lesser curvature and dorsal squamous fundus with diffuse hyperemia, B. Extensive necrotic area in the squamous region along the margo plicatus with severe edema and thickening of the epithelium with the presence of a vesicle (arrow).

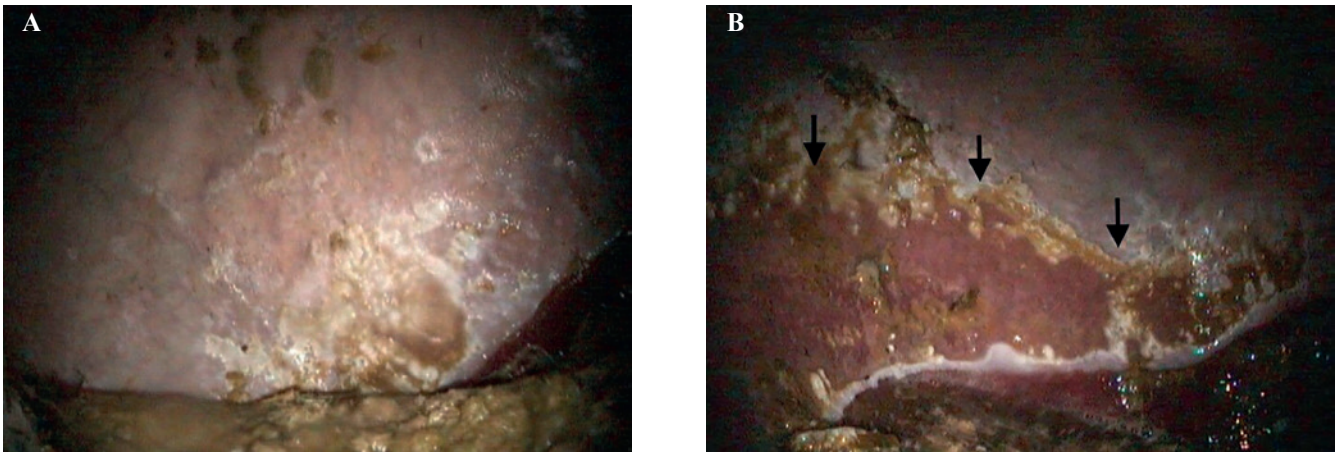

Figure 3. Gastroscopy control on day 7 showing: A. Squamous region with a mild degree of hyperemia and complete mucosa integrity, B. Band of ulceration (arrows) along the margo plicatus with loss of the mucosa integrity, extending from the greater to the lesser curvature.

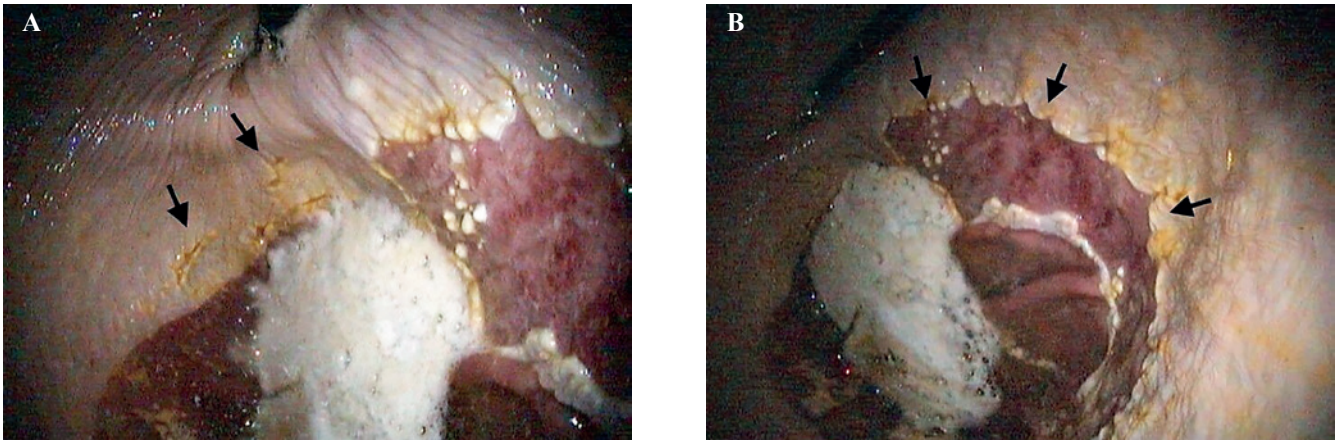

Figure 4. Gastroscopy control on day 11 showing: A. Squamous region with some areas of hyperkeratosis (arrows) but complete mucosa integrity, B. Band of ulceration along the margo plicatus with loss of mucosa integrity and contraction of the borders (arrows).

age, plus grain or concentrate meals spaced at least six hours apart (< 2g/Kg BW starch intake daily). Constant access to fresh water was recommended (Videla and Andrews, 2009) and daily low doses of omeprazole (Gastrogard, Merial Inc., Duluth, Georgia, USA) 1mg/kg BW per os for fourteen days. Control gastroscopy performed thirty days after discharge revealed a complete healing of the area along the margo plicatus with some areas of hyperemia in the squamous mucosa scored as Grade I (Figures 5A and 5B).

DISCUSSION

Gastroduodenitis is an acute intestinal disease characterized by an inflammatory process leading to gastrointestinal ileus, accumulation of fluid, distention of the proximal part of the small intestine and stomach, and absorption of bacteria into the bloodstream. Extending from the intestinal mucosa to the serosa, lesions in GD can begin with hyperemia that may lead to edema and hemorrhage (Freeman, 2000). The cause of GD remains unknown. Experimental studies on the origin of GD have been conducted, but a causal microbiologic agent has not been proved (Arroyo et al., 2018). However, it has been speculated that different agents could be associated with the disease including *Salmonella spp*., mycotoxins, *Clostridium spp*., *Clostridium perfringens* and *Clostridium difficile* (Arroyo et al., 2018). In the present study, bacteriological examination was not performed to identify possible pathogens responsible for the disease as the clinical signs improved after gastric decompression and as the hematological results were within normal limits. However, it would have been prudent to take a bacterial culture to rule out the presence of possible pathogens.

The diagnosis of GD is based on the clinical signs, the response to medical treatment, blood analysis, ultrasound and rectal examination. It is important to rule out other potential causes of small intestinal distention and reflux, such as small intestine strangulating obstruction (SISO). In the present case, the horse presented with signs of mild abdominal pain

and depression and displayed relief of this pain after gastric decompression. On rectal examination, horses with SISO have marked firm bowel distention while distention of the intestinal bowel is less in GD. Ultrasonographic examination may give inconsistent findings but usually, horses with GD show a reduction in the overall diameter coupled with an increase in wall thickness of the intestinal bowel compared to SISO cases (Arroyo et al., 2018). Peritoneal fluid analysis has been reported to have some diagnostic value, with marked changes suggesting a strangulating lesion (Arroyo et al., 2018). However, this procedure was not performed in the present case due to the lack of free peritoneal fluid.

Since the cause of GD is unknown, its treatment relies on supportive therapy, the aim of which is frequent gastric decompression, correction of fluid and electrolyte losses and restoration of normal intestinal function (Freeman, 2000). Prokinetic drugs such as metoclopramide have been used for their beneficial action in increasing the emptying of liquid from the stomach and increasing gastric and small intestine mechanical activity (Cohen et al., 1995). In most situations, gastric reflux usually ceases within three days (Freeman, 2000), as occurred in the present case. Supportive therapy for endotoxemia and ileus is also important. The combination of low doses of flunixin meglumine, and pentoxifylline has been reported to be effective in reducing the production of cytokines and thromboxane, thereby interfering with the inflammatory cascade and hence, decreasing the endotoxemic effects (Moore and Barton, 1999).

Systemic antimicrobial treatment could have been established as part of the initial treatment. However, antimicrobial treatment in such cases is controversial because an infectious agent as a cause of GD has not been clearly established (Hanson and Albanese, 2015; Murray, 1998). Due to the lack of pyrexia and due to the hematological results, in the present case, it was decided not to establish a course of antibiotics. In severe neutropenic horses, the use of broad spectrum antimicrobials can be recommended to prevent secondary infections (Hanson and Albanese, 2015). The measurement of serum amyloid A (SAA) and/or comparison between serum iron concentration and fibrinogen have been reported to be useful to identify the presence of an infection (Borges et al., 2007). In this case report, SAA concentrations could not be established because the test was not available at the hospital at that time. On the other hand, the authors considered the measurement of serum iron concentration to be unreliable due to the frequent iron supplementation that Arabian horses receive in this geographic region (Borges et al., 2007).

Laminitis has been reported to occur secondary to GD in 30% of cases, 8 to 192 hours after the onset of the disease (Cohen et al., 1994). In the present case, the horse started to show signs of discomfort and reluctance to move 24 hours after presentation, and intensive supportive therapy was established to minimize the effects of the laminitis. The authors believe that it can be helpful to start prophylactic treatment earlier, in case there is a high risk of endotoxemia.

EGUS has been reported to have a 17-to-100% incidence in different breeds of horses involved in different equine disciplines (Sykes et al., 2015a). EGUS shows a wide variety of clinical signs, which include poor appetite (Vatistas et al., 1999), behavioral changes (McClure et al., 1999), poor body condition and/or weight loss (Dionne et al., 2003), chronic diarrhea (Andrews and Nadeau, 1999), bruxism (Bell et al., 2007), poor coat condition (Vatistas et al., 1999), poor performance (Franklin et al., 2008) and colic (Vatistas et al., 1999; Andrews and Nadeau, 1999). Gastric ulcers are usually present in horses that show recurrent colic, and in a study by Murray (1992), 83% of the horses with gastric ulcers were reported to show recurrent colic while 28% with colic signs exhibited gastric ulcers.

In the present case report, the hemorrhagic reflux obtained by nasogastric intubation on arrival may have resulted from a combination of severe damage

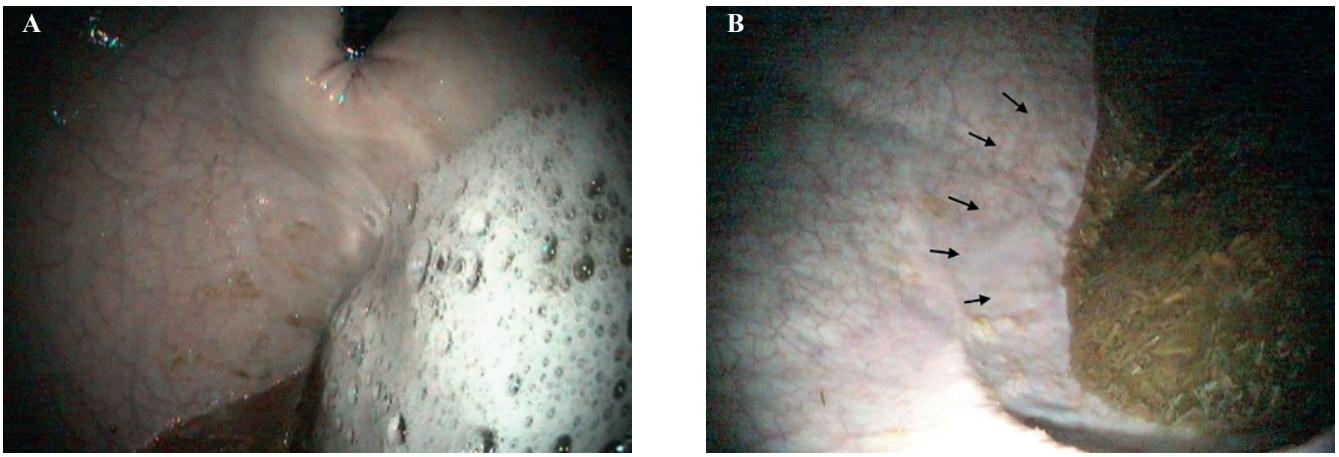

Figure 5. Gastroscopy control on day 30 showing: A. Cardia and squamous region with a mild degree of hyperemia, B. Area along the margo plicatus, where the band of ulceration was presented, showing complete healing (arrows).

in the gastric and small intestinal mucosa. The lesions presented in this case (i. e. hyperkeratosis, bullae formation, extensive damage of the gastric mucosa) could be compatible with emphysematous gastritis, an uncommon form of infectious gastritis previously reported in humans and horses (Delesalle et al., 2013). In humans, predisposing factors to develop this condition are gastric ulceration and GD (Moosvi et al., 1990), both present in this case. However, emphysematous gastritis usually has an acute onset and is fatal in most cases (Delesalle et al., 2013), making it an unlikely diagnosis in the present case.

The gastric ulceration was identified by gastroscopic examination, which allowed prompt treatment. Gastroscopy has been reported to be the only reliable ante mortem diagnostic method to identify gastric ulceration (Sykes et al., 2015a). The common prevention and treatment therapies for EGUS are suppression of gastric acid production and increasing intragastric pH (Lester et al., 2005; Sykes et al., 2015b). Omeprazole has been the cornerstone in the treatment of EGUS for the past twenty years (Sykes et al., 2015b). Omeprazole and pantoprazole both belong to the family of proton pump inhibitors (PPIs) that block the production of gastric H+/ K+ ATPase by the gastric parietal cells, thereby inhibiting gastric acid secretion. Because H+/ K+ ATPase production is the final step of the acid secretion route, PPIs are more effective in suppressing gastric acid secretion than H-2 antagonists (Shin and Sachs, 2008). Pantoprazole has widely been used in human medicine as an effective therapy for gastric ulcers, peptic ulcer bleeding and gastroesophageal reflux disease (Witzel et al., 1995; Liang et al., 2012; Van Rensburg and Cheer, 2012; Scholten, 2007).

In a study by Ryan et al., (2005), no adverse reactions during or after enteral or parenteral administration of pantoprazole in foals were noted. Similarly, Smith et al., (2010) treated alpacas successfully with pantoprazole. Hence, pantoprazole provides a new option for the treatment of gastric and duodenal ulceration in horses, in which oral administration is not tolerated due to reflux. This potentially gives parenteral pantoprazole an advantage over enteral omeprazole, of which inconsistent and variable reactions have been reported (Andrews et al., 2006).

Due to the severity of the horse's condition in the present case, it was decided to commence the gastric treatment with intravenous pantoprazole and add an adjunct therapy with ranitidine and sucralfate on day 4. Based on the positive experience of the authors in using the pantoprazole treatment, gastroscopy was not performed at that time point. Moreover, ranitidine and sucralfate have been shown to suppress intragastric acidity and increase blood flow. (Murray and Schusser, 1993; Murray, 2009). Ranitidine, as a histamine type-2 receptor antagonist, decreases acid secretion by blocking the histamine production involved in acid secretion from the parietal cells (Kitchen et al., 1998). Ranitidine has been reported to be effective in the treatment of gastric ulcers (Murray and Eichorn, 1996). Sucralfate is a hydroxyl aluminum salt of sucrose octasulfate. It acts as a buffer against hydrochloric acid, stimulating the secretion of prostaglandins and adhering to the ulcer bed (Borne and MacAllister, 1993). Sucralfate alone has no beneficial effects in the treatment of gastric and duodenal ulcers but it can be used in combination with acid-suppression drugs as an adjunct therapy (Videla and Andrews, 2009).

Diets with a high concentration of energy and volatile fatty acids can significantly increase the production of acid by the stomach, leading to upper duodenal mucosal ulceration and gastric ulceration that can disrupt the gastrointestinal activity. Based on the body condition of the horse (4/5) of the present case, the authors considered that the presence of large amounts of concentrate in the feeding schedule of the horse had probably led to the presence of both diseases (GD and EGUS). Such a feeding regime frequently leads to increased gastric acid production which, in turn, leads to ulceration of the small intestinal and gastric mucosa, and to disruption of the motility and function of the small intestine (Cohen et al., 2006).

Furthermore, it has been reported that some horses affected by EGUS do not show clinical signs (Luthersson et al., 2009) until an extensive and severe loss of mucosal integrity has been reached, thereby activating sensory nerves and causing pain (Raybould et al., 1992). Taking this into account and considering the acute nature of GD, the authors suggest that the ulcerative lesions in the horse of the present case had occurred chronically although the clinical signs had not been obvious until the gastric and intestinal activity was disrupted.

Significant improvement in the lesions was observed during the control gastroscopies performed on days 7 and 11. The stomach was scored as Grade III EGUS due to the presence of a band of ulceration along the margo plicatus, while the remainder of the squamous region showed obvious healing with some areas of hyperkeratosis. The authors consider that, disregarding the persisting margo plicatus lesions, a high rate of healing was achieved because the integrity of the remaining squamous mucosa and its hyperkeratotic areas gave the stomach an overall score of Grade I.

The horse was discharged with a low dose of oral omeprazole (1mg/kg BW per os) given every 24 hours for 14 days to complete the ongoing healing process. Low doses of omeprazole (1mg/kg per os every 24 hours) have been reported to have the same efficacy as higher doses (4mg/kg per os every 24 hours) (Sykes et al., 2015b), thereby reducing the amount of drug required for the treatment as well as the cost.

Given the severe and extensive lesions seen in the initial gastroscopy plus the clinical signs on arrival, the authors believe that with the therapy used in the present case, the aim of healing was achieved from its initiation. To their knowledge, the combination of

the medications used in this case can be applied in the clinical management of cases of severe duodenal and gastric ulceration. Even though the cause of the onset and severe lesions remain unclear and although other factors may lead to the clinical signs presented in this case, the authors believe that based on the body condition of the horse and the feeding schedules that Arabian show horses typically receive in the Middle East, a high concentrate feeding schedule was probably the most likely cause of the pathologies.

REFERENCES

Andrews, F., Bernard, W., Byars, D., Cohen, N., Divers, T., MacAllister, C., McGladdery, A., Merritt, A., Murray, M., Orsini, J., Snyder, J., Vatistas, N. (1999). Recommendations for the diagnosis and treatment of equine gastric ulcer syndrome (EGUS). *Equine Veterinary Education 11(5)*, 262-272.

Andrews, F.M., Frank, N., Sommardahl, C.S., Buchanan, B.R., Elliot, S.B., Allen, V.A. (2006). Effects of intravenously administered Omeprazole on gastric juice pH and gastric ulcers scores in adult horses. *Journal of Veterinary Internal Medicine 20,* 1202-1206.

Andrews, F.M., Nadeau, J. (1999). Clinical syndromes of gastric ulceration in foals and mature horses. *Equine Veterinary Journal 31(Suppl29),* 30-33.

Arroyo, L.G., Costa, M.C., Guest, B.B., Plattner, B.N., Lillie, B.N., Weese, J.S. (2017). Duodenitis-proximal jejunitis in horses after experimental administration of *Clostridium difficile* toxins. *Journal of Veterinary Internal Medicine 31,* 158-163.

Bell, R.J.W., Mogg, T.D., Kingston, J.K. (2007). Equine gastric ulcer syndrome in adult horses: A review. *New Zealand Veterinary Journal 55,* 1-12.

Borges, S.A., Divers, T.J., Stokol, T., Mohammed, O.H. (2007). Serum iron and plasma fibrinogen concentrations as indicators of systemic inflammatory diseases in horses. *Journal of Veterinary Internal Medicine 21,* 489-494.

Borne, A.T., MacAllister, C.G. (1993). Effect of sucralfate on healing of subclinical gastric ulcers in foals. *Journal of the American Veterinary Medicine Association 202(9),* 1465-1468.

Carroll, C.L., Huntington, P.J. (1988). Body condition scoring and weight estimation of horses. *Equine Veterinary Journal 20,* 41-45.

Chameroy, K.A., Nadeau, J.A., Bushmich, S.L., Dinger, J.E., Hoagland, T.A., Saxton, A.M. (2006). Prevalence of non-glandular gastric ulcers in horses involved in a university riding program. *Journal of Equine Veterinary Science 26,* 207-211.

Cohen, N.D., Faber, N.A., Brumbaugh, G.W. (1995). Use of bethanechol and metoclopramide in horses with duodenitis/proximal jejunitis: 13 cases (1987-1993). *Journal of Equine Veterinary Science 15*, 493-494.

Cohen, N.D., Parson, E.M., Seahorn, T.L., Carter, G.K. (1994). Prevalence and factors associated with development of laminitis in horses with duodenitis/proximal jejunitis: 33 cases (1985-1991). *Journal of the American Veterinary Medicine Association 204,* 250-254.

Cohen, N.D., Toby, E., Roussel, A.J., Murphey, E.L., Wang, N. (2006). Are feeding practices associated with duodenitis-proximal jejunitis? *Equine Veterinary Journal 38(6)*, 526-531.

Delesalle, C., Deprez, P., Vanbrantegem, L., Vaneechoutte, M., Decostere, A., Ducatelle, R. (2003). Emphysematous gastritis associated with *Clostridium septicum* in a horse. *Journal of Veterinary Internal Medicine 17*, 115-118.

Dionne, R., Vrins, A., Doucet, M. (2003). Gastric ulcers in standardbred racehorses: prevalence, lesion description, and risk factors. *Journal of Veterinary Internal Medicine 17,* 218-222.

Franklin, S.H., Brazil, T.J., Allen, K.J. (2008). Poor performance associated with equine gastric ulceration syndrome in four Thoroughbred racehorses. *Equine Veterinary Education 20,* 119-124.

Freeman, D.E. (2000). Duodenitis-proximal jejunitis. *Equine Veterinary Education 12(6)*, 322-332.

Hanson, R.R., Albanese, V. (2015). Small intestine colic. In: Sprayberry K.A., Robinson N.E. (editors). *Equine Internal Medicine*. Seventh edition, Saunders, Missouri, p. 328-332.

Hartmann, A.M., Frankeny, R.L. (2003). A preliminary investigation into the association between competition and gastric ulcer formation in non-racing performance horses. *Journal of Equine Veterinary Science 23,* 560-561.

Kitchen, D.L., Merritt, A.M., Burrow, J.A. (1998). Histamine-induced gastric acid secretion in horses. *American Journal of Veterinary Research 59(10),* 1303-1306.

Lester, G.D., Smith, R.L., Robertson, I.D. (2005). Effects of treatment with Omeprazole or Ranitidine on gastric squamous ulceration in racing thoroughbreds. *Journal of the American Veterinary Medicine Association 227,* 1636-1639.

Liang, C.M., Lee, J.H., Kuo, Y.H., Wu, K.L., Chiu, Y.C., Chou, Y.P., Hu, M.L., Tai, W.C., Chiu, K.W., Hu, T.H., Chuah, S.K. (2012). Intravenous non-high-dose Pantoprazole is equally effective as high-dose Pantoprazole in preventing rebreeding among low risk patients with a bleeding peptic ulcer after initial endoscopic hemostasis. *BMC Gastroenterology 12,* 28.

Luthersson, N., Nielsen, K.H., Harris, P., Parkin, T.D.H. (2009). The prevalence and anatomical distribution of equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark. *Equine Veterinary Journal 41,* 619-624.

McClure, S.R., Glickman, L.T., Glickman, N.W. (1999). Prevalence of gastric ulcers in show horses. *Journal of the American Veterinary Medicine Association 215,* 1130-1133.

Mossvi, A.R., Saravolatz, L.D., Wong, D.H., Simms, S.M. (1990). Emphysematous gastritis: Case report and review. *Reviews of Infectious Diseases 12*, 848-855.

Moore, J.N., Barton, M.H. (1999). An update on endotoxaemia. *Equine Veterinary Education 11*, 30-34.

Murray, M.J. (1992). Gastric ulceration in horses: 91 cases (1987-1990). *Journal of the American Veterinary Medicine Association 201,* 117-120.

Murray, M.J. (1998). Duodenitis-proximal jejunitis. In: Reed S.M., Bayly W.M. (editors). *Equine Internal Medicine.* Saunders, Philadelphia, p. 623-627.

Murray, M.J. (2009). Diseases of the stomach. In: Smith B.P. (editors). *Large Animal Internal Medicine*. Third edition, Mosby Elsevier, Missouri, p. 695-702.

Murray, M.J., Eichorn, E.S. (1996). Effects of intermittent feed deprivation, intermittent feed deprivation with ranitidine administration and stall confinement with ad libitum access to hay on gastric ulceration in horses. *American Journal of Veterinary Research 57(11),* 1599-1603.

Murray, M.J., Schusser, G.F. (1993). Measurement of 24-h gastric pH using an indwelling pH electrode in horses

unfed, fed and treated with ranitidine. *Equine Veterinary Journal 25,* 417-421.
Murray, M.J., Schusser, G.F., Pipers, F.S., Gross, S.J. (1996). Factors associated with gastric lesions in thoroughbred racehorses. *Equine Veterinary Journal 28*, 368-374.
Raybould, H.E., Li, D.S., Guth, P.H. (1992). Calcitonin gene-related peptide mediates the gastric hyperemic response to acid back-diffusion. *Annals of the New York Academy of Sciences 657,* 536-537.
Ryan, C.A., Sanchez, L.C., Giguere, S., Vickroy, T. (2005). Parmacokinetics and pharmacodynamics of Pantoprazole in clinically normal neonatal foals. *Equine Veterinary Journal 37(4),* 336-341.
Scholten, T. (2007). Long-term management of gastro-esophageal reflux disease with Pantoprazole. *Therapeutics and Clinical Risk Management 3(2),* 231-243.
Shin, J.M., Sachs, G. (2008). Pharmacology of proton pump inhibitors. *Current gastroenterology reports 10(6),* 528-534.
Smith, G.W., Davis, J.L., Smith, S.M., Gerard, M.P., Campbell, N.B., Foster, D.M. (2010). Efficacy and pharmacokinetics of Pantoprazole in Alpacas. *Journal of Veterinary Internal Medicine 24,* 949-955.
Sykes, B.W., Hewetson, M., Hepburn, R.J., Luthersson, N., Tamzali, Y. (2015a). European college of equine internal medicine consensus statement - equine gastric ulcer syndrome in adult horses. *Journal of Veterinary Internal Medicine 29,* 1288-1299.
Sykes, B.W., Sykes, K.M., Hallowell, G.D. (2015b). A comparison of three doses of Omeprazole in the treatment of gastric ulceration in the horse: a randomized, blinded clinical trial. *Equine Veterinary Journal 47,* 285-290.
Van Resburg, R.J., Cheer, S. (2012). Pantoprazole for the treatment of peptic ulcer bleeding and prevention of re-bleeding. *Clinical Medicine Insights Gastroenterology 5,* 51-60.
Vatistas, N.J., Snyder, J.R., Carlson, G., Johnson, B., Arthur, R.M., Thurmond, M., Zhou, H., Lloyd, K.L. (1999). Cross-sectional study of gastric ulcers of the squamous mucosa in Thoroughbred racehorses. *Equine Veterinary Journal 31(suppl 29),* 34-39.
Videla, R., Andrews, F.M. (2009). New perspectives in equine gastric ulcer syndrome. *Veterinary Clinics of North America: Equine Practice 25,* 283-301.
White, N., Tyler, D., Blackwell, R., Allen, D. (1987). Hemorrhagic fibrinonecrotic duodenitis-proximal jejunitis in horses: 20 cases (1977-1984). *Journal of the American Veterinary Medicine Association 190,* 311-315.
Witzel, L., Gutz, H., Huttemann, W., Schepp, W. (1995). Pantoprazole versus Omeprazole in the treatment of acute gastric ulcers. *Alimentary Pharmacology & Therapeutics 9,* 19-24.

Uit het verleden

Castratie, niet afgeleid van castor (bever)

In een nog niet zo ver verleden, en hier en daar nog steeds, werd het woord castratie – castreren afgeleid van de Latijnse benaming, van de Europese bever *Castor fiber*. De oorsprong daarvan is te vinden in het volksgeloof dat dit beest in nood zijn eigen testikels afbijt. De bever werd daarmee eigenaardig genoeg symbool van zowel overspel als kuisheid en ascetisme. In het oude Egypte stond dit dier als voorbeeld van de man verzwakt door overspel. Vandaar dus.

Aanleiding voor dit volksgeloof in zelfcastratie is meer dan waarschijnlijk een eigenschap die dit dier deelt met tal van andere zoogdiersoorten: het vermogen om zijn testikels in de buikholte op te trekken. Bij konijnen bijvoorbeeld is dat een probaat middel om te verhinderen dat rivaliserende rammen die zouden afbijten. Zo gaat het er immers aan toe in de lieflijke ongerepte natuur …

Luc Devriese