Case Report

Perforating jejunal diverticulosis in 2 horses

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Keywords: horse; colic; peritonitis; diverticulosis; jejunum

Introduction

Small intestinal diverticula have been recognised in several species, including man, horse, pig and sheep. Jejunal diverticulosis has been described mainly in man (Zager et al. 2000) and is usually asymptomatic, but may lead to perforation and resulting peritonitis. In horses, diverticula of the small intestine have been reported along the mesenteric border of the ileum and can be associated with muscular hypertrophy (Chaffin et al. 1992). Full thickness rupture of ileal diverticula can lead to diffuse peritonitis in some of these cases. Because most horses with small intestinal diverticula present with a history of chronic intermittent colic, the clinician is presented with a diagnostic challenge and must decide when it is appropriate to send the horse to a referral centre. In horses showing signs of colic, pyrexia, persistent tachycardia and reduced borborygmi, referral is recommended. In this report, we describe 2 cases of perforating jejunal diverticulosis associated with muscular hypertrophy and eosinophilic enteritis of the jejunum.

Case details

Case 1

History

A 9-year-old Peruvian Paso gelding weighing 450 kg was admitted to the Veterinary Teaching Hospital at Oregon State University for evaluation of acute abdominal pain of 5 h duration. The gelding had a history of low-grade colic episodes for several years and had been previously treated for gastric ulceration. The referring veterinarian administered 500 mg flunixin meglumine and 8 mg butorphanol tartrate i.v. and 4 l mineral oil via nasogastric tube.

Clinical examination

On presentation, the horse was bright and alert, with no obvious signs of colic. Abnormal signs included: pyrexia (39°C), persistent tachycardia (60 beats/min) and tachypnoea (44 breaths/min). Borborygmi were normal in all quadrants. No reflux could be obtained following nasogastric intubation. Rectal examination was initially without abnormal findings. However, a second rectal examination performed 12 h later revealed multiple loops of distended small intestine.

Haematology

Results included leucopenia, total white blood cell count (WBC) 5.06 x 10⁹/l (reference range [rr] 6–12 x 10⁹/l), hyperfibrinogenaemia (5 g/l [rr 1–4 g/l]) and hyperglycaemia (1.21 g/l [rr 0.79–0.99 g/l]). Twelve hours post admittance, a second sample revealed leucopenia (5.8 x 10⁹ cells/l), band neutrophilia (1.044 x 10⁹/l [rr <10 x 10⁹/l]), hypocalcaemia (0.105 g/l [rr 0.115–0.133 g/l]), hypophosphataemia (0.016 g/l [rr 0.019–0.041 g/l]), hypomagnesaemia (0.008 g/l [rr 0.017–0.029 g/l]) and hyperglycaemia (1.56 g/l).

Peritoneal fluid

Abdominocentesis yielded serosanguinous peritoneal fluid; cytology revealed a nucleated cell count of 74.75 x 10⁹/l (rr <2.5 x 10⁹/l) consisting of 94% nondegenerate neutrophils, 6% large mononuclear cells and total protein 38 g/l (rr <25 g/l). Blood gas analysis revealed high lactate (4.34 mmol/l [rr 0.5–2.0 mmol/l]). Culture of a peritoneal fluid swab was negative 48 h post sampling. Repeat abdominocentesis after 24 h revealed a serosanguinous cloudy sample with a nucleated cell count of 148 x 10⁹/l consisting of 78% neutrophils and 22% large mononuclear cells and total protein 43 g/l. Analysis of the second peritoneal fluid sample showed low pH (6.76), high lactate (12.68 mmol/l) and low glucose (<0.10 g/l [rr 0.79–0.99 g/l]). Intestinal alkaline phosphatase (ALP) levels in the peritoneal fluid before and after incubation with l-phenylalanine were 687 and 564 iu/l, respectively (rr 3.0–39 iu/l).

Treatment

Initial treatment included i.v. polyionic fluids (4 l/h), potassium penicillin (22,000 u/kg bwt i.v. q. 6 h), gentamicin (6.6 mg/kg bwt i.v. q. 24 h) and flunixin meglumine (1.1 mg/kg bwt i.v.)

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q. 12 h). The horse remained comfortable overnight, but continued to have tachycardia (56–60 beats/min). Gastric reflux (4 l each time) was obtained twice.

**Outcome**

Due to the increased nucleated cell count, low glucose and high lactate in the abdominal fluid, a diagnosis of septic peritonitis was made. Surgical intervention was recommended to determine the source of the peritonitis and to treat the condition. The owner elected to have the horse subjected to euthanasia due to economic constraints and guarded prognosis.

**Post mortem findings**

Necropsy findings included diffuse peritonitis and marked muscular hypertrophy of the small intestine with multiple diverticula (2 cm diameter) present along the mesenteric surface, which were associated with the vasculature and extended into the adjacent mesentery. In the proximal jejunum, one markedly enlarged diverticulum (5 cm diameter), with a thin necrotic wall and fibrin adhered to the serosal surface, had ruptured. In the adjacent mesentery, there was marked haemorrhage and oedema associated with the diverticulum. Haemomelasma ilei-like lesions were present throughout the jejunum. Histopathology of the duodenum, jejunum and ileum revealed moderate accumulations of eosinophils in the lamina propria and submucosa (Fig 1). The jejunum and ileum had marked diffuse hypertrophy of the smooth muscle layers. A section of a jejunal diverticulum showed that it was lined by a smooth muscle border. No Salmonella spp. were cultured from the markedly enlarged diverticulum.

**Case 2**

**History**

A 9-year-old Morgan gelding weighing 400 kg was admitted for evaluation of mild progressive colic signs of 12 h duration. The gelding had experienced a mild episode of colic 6 weeks earlier and resolved after the administration of 550 mg flunixin meglumine i.v. The referring veterinarian had administered 18 l polyionic fluids with 200 ml DMSO and 300 mg flunixin meglumine i.v., 2 l mineral oil via nasogastric tube and abdominal acupuncture prior to referral.

**Clinical findings**

The gelding appeared bright and alert on presentation with no apparent signs of colic, but pyrexia (39.5°C), tachycardia (60 beats/min) and tachypnoea (24 breaths/min) were present. Abdominal auscultation revealed absence of borborygmi. No gastric reflux was obtained on nasogastric intubation and rectal palpation was within normal limits. Eight hours after presentation, abdominal ultrasonography using a 5 MHz linear probe along the ventral midline revealed multiple loops of hypomotile small intestine with thickened walls (>5 mm) and a lumen diameter within normal limits (1.7 cm). Rectal examination confirmed the ultrasound findings of thickened small intestine without concurrent distension.

**Haematology**

Leucopenia (2.8 x 10^9 WBC/l [rr 6–12 x 10^9/l]) and hyperglycaemia (1.59 g/l) were found.
Peritoneal fluid

Abdominocentesis yielded peritoneal fluid that was cloudy and serosanguinous with a nucleated cell count of 196.2 x 10^9/l with 90% degenerate neutrophils containing intracellular rod- and cocci-shaped bacteria, and total protein 62 g/l. Culture of a peritoneal fluid swab produced *Clostridium* spp. and coliform bacteria 72 h post sampling. Abdominal fluid analysis using a blood gas analyser revealed low pH (7.083), low glucose (0.23 g/l) and high lactate (10.74 mmol/l). Intestinal ALP levels in peritoneal fluid before and after incubation with l-phenylalanine were 2260 and 1900 iu/l, respectively.

Treatment

Following jugular catheterisation, 1 l hypertonic saline and polyionic fluids (2 l/h) were administered i.v. The gelding received potassium penicillin (22 x 10^3 u/kg bwt i.v. q. 6 h), amikacin sulphate (12 mg/kg bwt i.v. q. 24 h), flunixin meglumine (0.375 mg/kg bwt i.v. q. 6 h), polymyxin B (5000 u/kg bwt in 1 l NaCl i.v. q. 12 h), 4 l plasma i.v. and DMSO (1 g/kg bwt) in 5 l lactated Ringer’s solution q. 24 h. Eight hours after presentation, metronidazole (25 mg/kg bwt per os q. 8 h) was added and the dose of flunixin meglumine adjusted (1.1 mg/kg bwt i.v. q. 12 h).

Outcome

The horse remained comfortable during the first 8 h after presentation despite continued tachycardia (60–80 beats/min) while passing watery faeces with traces of mineral oil. Small quantities of wet grass hay were then introduced hourly and readily consumed. Sixteen hours following presentation, the gelding developed muscle fasciculations and intermittent colic episodes with a heart rate fluctuating between 52 and 120 beats/min over the next 2 h. Surgical exploration of the abdomen was elected due to deterioration of cardiovascular parameters and further colic episodes.

Histopathology

Histopathology of the jejunum featured hypertrophy of the muscularis, which was accompanied by scattered eosinophils in the *lamina propria* and submucosa. Sections of a jejunal diverticulum revealed normal appearing and arrayed epithelial, connective tissue, and muscular layers forming the walls of the diverticulum.

Discussion

Small intestinal diverticula have been reported infrequently in several species. In man, a form called jejunal diverticulosis (JD) is considered a rare entity that can lead to intestinal bleeding, perforation or small bowel obstruction (Zager et al. 2000). The diverticula are found usually along the mesenteric border, similar to the 2 cases presented here. Unlike our cases, however, human JD usually occurs as mucosal herniation through the muscular layer (false pulsion diverticulum) rather...
than containing all layers of the bowel (true pulsion diverticulum). In addition, the human form is not usually associated with muscular hypertrophy of the small intestine.

In horses, an association between muscular hypertrophy of the small intestine and diverticula formation has been established (Chaffin et al. 1992). In that study, 45% of horses with muscular hypertrophy had false pulsion diverticula along the mesenteric aspect of the ileum and one horse had linear diverticula on the antimesenteric surface of the jejunum. Another study reported hypertrophy and diverticula of the duodenum in 2 horses (Cordes and Dewes 1971). Our cases were different from the previous studies in that our horses had multiple true pulsion diverticula along the mesenteric border of the jejunum. True pulsion diverticula result probably from prolonged intraluminal pressure (Chaffin et al. 1992). It is believed that muscular hypertrophy occurs first, followed by secondary diverticula formation. Inherent weakness of the bowel wall due to larger than normal interruptions in the thickened muscularis can lead to diverticula formation, tearing or rupture (Cordes and Dewes 1971; Chaffin et al. 1992).

Causes of primary small intestinal hypertrophy have not been determined, but may involve visceral larval migrans, inflammatory bowel disease, uncontrolled spasmodic contractions, or congenital hormonal influences (Chaffin et al. 1992). Both of our cases had evidence of eosinophilic infiltrates in the lamina propria and submucosa of the jejunum, incriminating a potential parasitic or inflammatory aetiology. One case had haemomelasma ilei-like lesions along the length of the jejunum, an association that has been noted previously in horses with small intestinal hypertrophy (Chaffin et al. 1992). This association may be related to a serosal response to chronic distension and inflammation of the intestinal wall.

Diagnosis of perforating jejunal diverticulosis in our cases was not possible without exploratory laparotomy or post mortem findings. However, clinicians can utilise abdominocectensis, ultrasound and rectal examination to determine the presence of septic peritonitis in the horse. Abdominocectensis is a rapid, minimally invasive, cost-effective way of evaluating the abdomen (Freden et al. 1998). Hallmark diagnostic indicators of septic peritonitis include increased nucleated cell count (>5 x 10^9 cells/l), high protein levels (>25 g/l), increased fibrinogen (>2 g/l), low pH (<7.3), low glucose levels (<0.3 g/l) and the presence of bacteria identified by cytology or culture of abdominal fluid samples (Van Hoogmoed et al. 1999). Increased lactate levels in abdominal fluid suggest the presence of anaerobic metabolism resulting from neutrophil glycysis or potentially compromised bowel (Stassen et al. 1986; Reef 1991). Elevated ALP concentrations in the peritoneal fluid can also indicate compromised bowel, as ALP is found in the intestinal mucosa (Jones et al. 1967). Nonintestinal phosphatases can be inhibited by the addition of L-phenylalanine to allow improved evaluation of the degree of intestinal damage (Blackmore and Palmer 1977). Abdominal ultrasonography is the noninvasive method of choice for evaluation of bowel wall thickness, degree of bowel distension, and the quantity and character of peritoneal fluid (Spaulding 1993). In one study, horses with peritonitis had some degree of small intestinal motility on ultrasound with a wall thickness of 0.5–1.3 cm and diameter of 2.0–5.1 cm (Klohnen et al. 1996). Rectal examination further aids in evaluating intestinal wall thickness, motility and distension. By combining these diagnostic tests, the clinician can determine the severity of the condition and the need for transferring a case to a referral centre.

The decision for surgical intervention in cases of septic peritonitis relies on clinical parameters, response to treatment, severity of the condition, and clinician experience.

Inability to control pain, intermittent or constant pain without a diagnosis, abnormal distention found by ultrasound or rectal examination, or increasing levels of protein and nucleated cells in the abdominal fluid indicate a need for surgical exploration of the abdomen. Also, if the nucleated cell count in the abdominal fluid is >40 x 10^9 cells/l, abdominal lavage may be indicated, either by nonsurgical or surgical means (Hanson 1999). In the cases described here, early surgical intervention and resection of the affected jejunum may have improved the prognosis. However, rapid deterioration of clinical parameters due to bowel perforation, diffuse serosal inflammation and adhesions precluded a successful outcome in these horses.

References