Case Report

Treatment of a horse following rupture of the colon during surgery

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Introduction

Rupture of the large colon of horses during colon surgery is considered to be a fatal complication, and affected horses are subjected routinely to euthanasia (Adams 1982; Dabareiner and White 1995; Johnston and Freeman 1997). **This report describes the apparently successful surgical and postsurgical management of a 2-year-old American Quarter Horse gelding whose right dorsal colon was ruptured during surgery to correct a colonic displacement.**

Case details

History

The horse had shown signs of abdominal pain for 36 h, initially thought to be caused by feed impaction of the ascending colon. Exploration of the abdomen was prompted by lack of response to administration of a laxative (i.e. 4 litres mineral oil administered via nasogastric tube), administration i.v. of a balanced electrolyte solution and analgesic drugs, and increasing gaseous distension of the ascending colon, determined by abdominal palpation *per rectum*. Procaine penicillin (22,000 u/kg bwt, i.m., q. 12 h) and gentamicin sulphate (5 mg/kg bwt i.v., q. 12 h) were administered prior to surgery.

Surgical procedure

The ascending colon was found to be displaced to the right and dorsally when the abdomen was explored through a ventral midline celiotomy. During an attempt to exteriorise the left dorsal and ventral colons, the right dorsal colon incurred a transmural, nearly 360° rent resulting in spillage of at least 2 litres ingesta mixed with mineral oil into the abdominal cavity. Only a 6 to 8 cm wide section of dorsal colon at the mesenteric surface remained intact. While manually apposing the edges of the colonic rent to prevent further contamination, the celiotomy was extended cranially and a portion of the left ascending colon, including the pelvic flexure, was exteriorised.

Ingesta were removed from the exteriorised portion of the colon through an enterotomy created at the pelvic flexure. With the torn segment of colon still within the abdomen, the colonic rent was closed with 2-0 polyglycolic acid suture using a simple continuous pattern. More ingesta were removed by lavaging the colon with water administered using a garden hose inserted through the pelvic enterotomy.

Fig 1: A Foley catheter was inserted into the abdominal cavity. The catheter was held in place by inflation of a 30 ml bulb. A fluid administration set was attached to the catheter. After administration of fluids, the fluid bag was lowered and fluid drained from the abdomen into the bag.

After ingesta were evacuated, more of the large colon, including the torn segment, was exteriorised. The suture was removed from the colonic rent and the colonic wound and enterotomy site were lavaged with sterile 0.9% saline solution. The enterotomy and rent were each closed with 2-0 polyglycolic acid suture using a continuous Lembert pattern, which was oversewn using a Cushing pattern.

All portions of intestine that could be exteriorised were removed from the abdomen and most of the omentum, which was heavily contaminated with

ingesta, was amputated. The abdominal cavity and viscera were copiously lavaged for about 40 min with warm tap water administered through a garden hose. Clumps of ingesta were suctioned from all regions of the abdominal cavity during lavage. Lavage was discontinued when ingesta were no longer observed floating through the celiotomy or retrieved by suction. The abdominal cavity and viscera were then lavaged with 20 litres sterile 0.9% saline solution, containing 10^3 units heparin/l, which was removed by suction. A 30 Fr. Foley catheter was inserted 3 cm to the right of the midline at the cranial-most aspect of the celiotomy. The *linea alba* was sutured with No. 3 polygalactin 910 using a simple continuous pattern and the subcutaneous tissue was apposed with 2-0 polyglycolic acid suture using a simple continuous suture pattern. The skin was apposed with staples.

Postoperative course

Immediately after recovery from anaesthesia, the horse displayed severe muscle fasciculations and had a 3 or 4 mm wide band of dark blue mucous membrane at the apices of the premaxillary incisor teeth. The horse was administered 7.2% saline solution (2 ml/kg bwt i.v.) and lidocaine hydrochloride (1.3 mg/kg bwt i.v.) followed by continuous infusion i.v. of balanced polyionic fluids and lidocaine hydrochloride (0.05 mg/kg/min) for 5 days. Sodium heparin (40 u/kg bwt sub cut., q. 12 h) was administered for 2 days. Gentamicin sulphate (5 mg/kg bwt q. 12 h) and potassium penicillin (22,000 u/kg bwt i.v., q. 6 h), were administered for 5 days, after which trimethoprim sulphadiazine (20 mg/kg bwt *per os*, q. 12 h) was administered for an additional 35 days. Metronidazole (25 mg/kg bwt *per os*, q. 12 h) and flunixin meglumine (0.33 mg/kg bwt i.v. or *per os*, q. 8 h) were administered for 16 days.

Immediately after recovery from anaesthesia, and then twice daily, the abdomen was lavaged with 5 l of sterile, balanced, polyionic fluid (**Fig 1**). The day after surgery, the horse had a good appetite for both hay and grain but showed mild signs of abdominal pain that included pawing and odontoprisis. Because odontoprisis can be a sign of gastric ulceration, the horse was administered ranitidine hydrochloride (1.3 mg/kg bwt i.v., q. 12 h) for 3 days.

After recovery from anaesthesia, the horse's packed cell volume (PCV) was 40%, and plasma protein 45 g/l. The heart rate remained consistently and markedly elevated (60–80 beats/min) for 10 days. Serum concentrations of sodium, potassium and chloride were within the reference range at 6 and 24 h after surgery. On Day 2, the horse's rectal temperature became elevated (39.5°C) and remained so for 12 more days (range 38.8–40.0°C).

The day after surgery, the plasma protein concentration was 40 g/l. The serum concentration of calcium was 1.72 mmol/l and that of creatinine was 203.3 µmol/l, and the white blood cells (WBC) count was 4.3 x 10^9cells/l. Four litres of plasma and 500 ml calcium dextrose solution were administered i.v. On Day 3, the horse was bright and alert. Peritoneal fluid collected before abdominal lavage contained 15% macrophages and 85% neutrophils, only a few of which showed signs of degeneration. Bacteria were not observed. On Day 3, the horse was neutropenic (PMNs 1.4 x 10^9/l), its neutrophils were toxic and plasma protein remained low (39 g/l), but serum creatinine concentration was within reference range. On Day 4, the horse was no longer neutropenic (PMNs 4.4 x 10^9/l) and toxic neutrophils were less evident.

On Day 5, peritoneal fluid had a protein concentration less than 25 g/l and a nucleated cell count of 13.7 x 10^9/l, 20% of which were macrophages and lymphocytes and the rest morphologically normal neutrophils. The peritoneal drain tube was removed after a final lavage. The catheterised jugular vein was thrombosed and exudate dripped from the abdominal incision at several sites. The horse appeared depressed and showed mild signs of abdominal pain. During abdominal palpation *per rectum*, the small colon was found to be impacted. The horse was treated with mineral oil administered by nasogastric tube and with warm water enemas administered t.i.d., and the impaction and signs of abdominal pain and depression resolved 36 h later.

On Day 10, the horse showed signs of mild abdominal pain, prompting palpation of the abdomen *per rectum*. Intestinal segments that could be palpated were adhered together but could be separated, indicating that abdominal adhesions were fibrinous. On this day, the horse's WBC count was 27.0 x 10^9 cells/l, PCV 29%, plasma protein 56 g/l and plasma fibrinogen 8 g/l. Weight loss was estimated to be about 100 kg. The horse remained hospitalised for an additional 2 weeks and, during this period, no further signs of abdominal pain were observed, appetite improved and further weight loss was not apparent. At discharge from the hospital, exudate discharged from the celiotomy site and the jugular vein that had been catheterised was thrombosed.

Eight months after discharge, the horse was returned for repair of an incisional hernia. At this time, the horse was in good condition and both jugular veins were patent. Results of haematology and serum biochemical analyses were within reference range. The horse had been kept at pasture for the previous 8 months, and its diet supplemented with alfalfa hay. The owner reported that, on 3 occasions, the horse had shown signs of mild abdominal pain which, in each case, resolved either with no treatment or after i.m. administration of 500 mg flunixin meglumine. No signs of abdominal pain had been observed during the previous 4 months. The hernia was repaired without complication using nylon mesh implanted subcutaneously without opening the abdomen.

Discussion

A large, ingesta-filled ascending colon is predisposed to rupture during manipulation because, when the abdomen is opened, transmural pressure in the colonic wall increases dramatically due to elimination of intra-abdominal pressure (Johnston and Freeman 1997). The right dorsal colon is the site most likely to rupture when tension is applied to the colon, because this section of

colon is fixed at one end by mesentery. In this case, correction of the displacement, repair of the colonic rent, intra-operative abdominal lavage and subsequent treatment of the horse for septic peritonitis resulted in a favourable outcome despite complications of incisional infection and herniation and apparent formation of abdominal adhesions.

Considering the extent of abdominal contamination in this horse and the need for a large quantity of lavage solution applied to serosal surfaces, the use of commercial isotonic solutions seemed inadequate. With use of a garden hose it was possible to lavage, with pressure, regions of the abdomen that could not have been lavaged by merely pouring bottled, commercial, isotonic solutions into the abdomen. Use of a liquid proportioner for large volume isotonic lavage of the equine colon during pelvic flexure enterotomy has been described (Bohn *et al.* 1994), but this device was not available. The systemic or local effect of water applied to the peritoneum has not, to our knowledge, been investigated in detail in any species. In one study, lavage of the serosal surface of the ascending colon of horses with tap water during evacuation of ingesta from the colon did not seem to have adverse clinical effects (Markel *et al.* 1998). Histological comparison of the ascending colon of 2 horses lavaged for 20 min 8 days previously with either tap water or lactated Ringer's solution (LRS), showed that the serosa of the colon lavaged with tap water had 3 to 5 times more fibrosis than did the serosa of the colon lavaged with LRS (Bohn *et al.* 1994).

Injection of 20 ml sterile distilled water into the peritoneal cavity of rats caused no clinical signs of disease and significantly enhanced the rats' resistance to septic peritonitis induced 2 weeks later by colonic perforation (Shin *et al.* 1988). Enhanced resistance to septic peritonitis by intraperitoneal administration of distilled water was thought to be the result of disruption of mast cells. The chemotactic and phagocytic activities of peritoneal macrophages were evidently enhanced by the uptake of extruded mast cell granules. Attenuation of the peritoneal inflammatory response was also noted in these rats and attributed to an absence of peritoneal mast cells caused by intraperitoneal administration of distilled water. A 2 week interval between uptake of mast cell granules by macrophages and colonic perforation, however, was required for development of increased resistance to septic peritonitis.

The systemic effects of tap water lavage in this horse appeared to be minimal, but serum electrolyte concentrations were not determined until 6 h after recovery from anaesthesia and after hypertonic saline solution had been administered. Except for serum creatinine and electrolyte concentrations, postoperative serum biochemical analyses were not performed.

The peritoneal cavity of this horse was lavaged with a balanced polyionic fluid postoperatively to remove bacteria and debris that may have failed to be removed during intra-operative lavage, and to reduce the likelihood of formation of intra-abdominal adhesions. In some clinical investigations, peritoneal lavage for treatment of horses suffering from septic peritonitis was considered ineffective (Dyson 1983; Hillyer and Wright 1997), but some investigators considered it to be an important component of treatment (Valdez *et al.* 1979; Hardy and Rakestraw 1999). Postoperative abdominal lavage of horses has been shown significantly to reduce the incidence of adhesions, possibly by removing fibrin from serosal surfaces and mechanically separating intestine at the time of lavage (Hague *et al.* 1996).

Other measures taken to prevent formation of adhesions in this horse included administration of heparin in the intraoperative lavage solution and subcutaneously. In one study, heparin was shown to reduce formation of adhesions in ponies when administered at a dose of 40 u/kg bwt at the time of surgery and then every 12 h for 48 h (Parker *et al.* 1987). The omentum was excised in this case, because it was grossly contaminated and because excision of omentum has been reported to decrease formation of abdominal adhesions in horses (Kuebelbeck *et al.* 1994). Perioperative administration of a combination of gentamicin, penicillin and flunixin meglumine, as was used in this horse, was perceived, in a survey of equine surgeons, to prevent abdominal adhesions in the horse by ameliorating inflammation (septic and nonseptic) of the peritoneal cavity (Southwood *et al.* 1997).

Stimulation of intestinal motility by using prokinetic drugs may also help to prevent the formation of abdominal adhesions (Southwood and Baxter 1997). Because lidocaine (lignocaine) hydrochloride has been shown to retard bacterial killing *in vitro*, use of this prokinetic drug may be contraindicated when the abdomen is grossly contaminated at surgery (Hardy and Rakestraw 1999). Abdominal sepsis in this horse resolved despite this putative adverse clinical effect of lidocaine hydrochloride.

The episodes of colic observed during the first 4 months postoperatively may have been caused by abdominal adhesions. Prognosis for long-term survival of horses with signs of abdominal adhesions has been shown to be guarded if signs of colic are observed during the first 2 months after celiotomy (Baxter *et al.* 1989).

Although the prognosis for long-term survival of this horse remains guarded, the initial favourable response of the horse to therapy for rupture of the colon demonstrates that this complication of colic surgery need not always be immediately fatal.

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