Case Report

The equine gastrospenic ligament: Anatomy and clinical considerations

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Summary

A gelding was referred for colic and inguinal wound evaluation. Abdominal ultrasound showed distended, oedematous and immotile small intestinal loops abaxial to the spleen. An exploratory laparotomy revealed jejunal incarceration through a rent in the gastrospenic ligament (GSL). The rent in the GSL was opened and surgical correction (resection and anastomosis of non-viable jejunum) performed. The GSL was left open. The horse recovered uneventfully and one year after surgery no further episodes of colic have been observed.

Introduction

Incarceration of the small intestine through a rent in the gastrospenic ligament (GSL) represents only a minor proportion of all small intestinal colics. It is reported to account for 4.5% of all small intestinal lesions, with a prevalence of 1.5% of all horses undergoing exploratory laparotomy in one report (Jenei et al. 2007). Previous reports suggest that geldings are predisposed to the condition (Yovich et al. 1985; Marien and Steenhaut 1998; Rhoads and Parks 1999; Jenei et al. 2007).

The GSL is a broad, thin band of omentum that runs from the left part of the greater curvature of the stomach to the cranial edge of the spleen (Budras et al. 2003). Ventrally, the GSL is continuous with the greater omentum. The blood supply to the ligament is provided by the gastroepiploic artery, which is.split into left and right arteries (Budras et al. 2003). The role of the GSL is unclear, but it may provide structural support to the abdominal organs and viscera (Barone 1997). In the normal horse no small intestine lies abaxial to this ligament (Yovich et al. 1985; Rhoads and Parks 1999).

To the authors’ knowledge, there are no specific preoperative findings that indicate the presence of an entrapment of small intestine through a rent in the GSL and the diagnosis is usually made during surgical exploration of the abdomen at necropsy (Marien and Steenhaut 1998). Typical management of these cases involves manual traction and reduction of the incarcerated intestine through the GSL (Jenei et al. 2007). This report describes the ultrasonographic findings that led preoperatively to a suspicion of entrapment of small intestine through a rent in the GSL and some variations in the surgical technique used to reduce a portion of incarcerated jejunum through a tight rent in the dorsal portion of the GSL. In addition, a description of the normal anatomy in the gastrospenic region is given to help further treatment of lesions involving this area.

Case description

A 9-year-old Thoroughbred cross gelding was presented to our hospital for investigation of colic of 8 hours’ duration. The signs of pain had increased despite administration of nonsteroidal anti-inflammatory drugs by the referring veterinarian.

On presentation the horse was in good body condition, weighing 584 kg, and was of dull mentation. Heart and respiratory rates were elevated at 52 beats/min and 20 breaths/min, respectively. Mucous membranes were pink and slightly tacky, with a capillary refill time of 3 s. Intestinal borborygmi were absent in all abdominal quadrants on auscultation. Passage of a nasogastric tube did not reveal any net gastric reflux; however, a large volume of gas was relieved.

A deep laceration, which had occurred 4 days previously as a result of a hunting accident, was present in the right inguinal region. Primary closure of the wound had been performed by the referring veterinarian. This had largely dehisced and a large amount of malodorous, purulent material was discharging from the wound. Multiple loops of distended small intestine were present in the mid caudal abdomen on palpation per rectum. In addition, an accumulation of retroperitoneal fluid and air in the right inguinal area (which extended cranially) could be appreciated.

A complete ultrasonographic examination of the abdomen was performed (Reef 1998) using a 3.5 MHz curvilinear transducer (Sonoace Pico)1. Multiple loops of small intestine were seen in the left inguinal region and along the ventral abdomen. Most of the small intestinal loops in this region were normal in size and wall thickness. However, multiple loops of immotile, distended small intestine were seen between the left body wall and the spleen (Fig 1). Small intestinal wall thickness was also increased (1.29 cm, normal <0.3 cm, Reef 1998). The right inguinal area was difficult to evaluate due to the presence of air in the tissues creating artefacts. A moderate amount of peritoneal fluid was seen in the cranioventral aspect of the abdomen. Turbid, serosanguinous fluid was yielded readily on abdominocentesis and based on this appearance, samples were submitted for both cytology and bacterial culture. Total protein level was 38 g/l (reference range [rr] 20–90 g/l) and the white blood cell count was 0.32 × 109/l (rr <5 × 109/l) with 74% neutrophils. No bacteria were observed on cytological evaluation and no bacterial growth was yielded after 7 days of culture.

A complete blood count (CBC) showed a mild leucocytosis (11.2 × 109/l, rr 5.5–9.2 × 109/l) with a mature
neutrophilia (6.72 x 10⁹/l, rr 2–6 x 10⁹/l) and a left shift with band neutrophils (2.69 x 10⁹/l, rr 0–0.1 x 10⁹/l). Fibrinogen was elevated at 6 g/l (rr 1–4 g/l). All other parameters were within normal limits. Biochemistry revealed a hypoproteinaemia (54 g/l, rr 57–79 g/l) with a mild hypoalbuminaemia (28 g/l, rr 29–37 g/l); in addition to an elevated creatinine kinase (826 iu/l, rr 0–70 iu/l), slightly increased creatinine level (199 µmol/l, rr 100–170 µmol/l) and mildly increased bilirubin (60 µmol/l, rr 5–51 µmol/l).

The presence of multiple loops of immotile, distended small intestine seen between the left body wall and the spleen was suggestive of an incarceration of small intestine through a rent in the GSL. Although the horse did not show overt signs of pain, the history and rectal palpation findings along with the macroscopic appearance of the peritoneal fluid prompted the decision to explore the abdomen surgically in order to confirm the clinical suspicion.

A 16 gauge i.v. catheter was placed in the left jugular vein. A 10 litre bolus of compound sodium lactate (CSL) fluids was administered over 45 min. CSL fluids at twice maintenance level (100 ml/kg bwt/day) were administered thereafter. Crystalline penicillin (22,000 i.u/kg bwt i.v.), gentamicin sulphate (6.6 mg/kg bwt i.v.) and subcutaneous tetanus antitoxin were administered prior to surgery.

The gelding was premedicated with xylazine (0.3 mg/kg bwt i.v.) and butorphanol (0.01 mg/kg bwt i.v.). Anaesthesia induction was achieved with administration of ketamine (2 mg/kg bwt i.v.) and diazepam (0.05 mg/kg bwt i.v.). A 30 mm diameter endotracheal tube was placed and anaesthesia was maintained with isoflurane in oxygen using positive pressure ventilation. CSL fluids, supplemented with 20 mEq of KCl/l, were administered i.v. throughout surgery. Dobutamine was administered at 0.1–0.4 µg/kg bwt/min to maintain mean arterial blood pressure at approximately 70 mmHg. Four litres of plasma (Hypermune) and 2 litres of 6% hetastarch (Voluven) were administered during surgery due to a worsening of the hypoproteinaemia. Lidocaine was given as a loading dose (1.3 mg/kg bwt i.v. over 10 min), followed by a continuous rate infusion (CRI: 0.05 mg/kg bwt/min i.v.); which was discontinued 30 min prior to the end of surgery. Morphine (0.1 mg/kg bwt i.v.) was administered at the end of surgery. Urine output was also measured during anaesthesia (2.5 ml/kg bwt/h).

The horse was positioned in dorsal recumbency. The abdomen was clipped and an aseptic preparation performed using a chlorhexidine based scrub⁴. A 30 cm ventral midline incision was made through the linea alba to access the abdomen. Abdominal exploration revealed a rent in the dorsal portion of the GSL. A right-to-left (caudal to cranial) herniation of jejunum through the rent was palpable. Despite multiple attempts, manual reduction (including enlargement of the rent), was not possible as the jejunum and caecum were also displaced and pulled cranially. The GSL was transected parallel to the gastroepiploic arteries using the Ligasure Vessel Sealing System (Ligasure™) in a ventral to dorsal direction, until the rent was reached (Fig 2a and d). A 6 m length of jejunum were deemed nonviable and a resection was undertaken. An end-to-end jejunojunostomy anastomosis was performed using a one layer Lembert suture pattern (Nieto et al. 2006) using USP 3-0 polydioxanone (PDS) on a 36 mm, half-circle round-bodied needle.

In the caudal abdomen, an area of fluid and crepitus was present on the peritoneal surface overlying the right inguinal wound. No direct communication between the wound and abdomen was identified on palpation and no other abnormalities of the intestines or diaphragm were evident on exploration. No attempt was made to close the transected GSL due to poor access and friability. The abdomen was lavaged with 10 litres of CSL fluid and the abdominal incision routinely closed in 3 layers. The linea alba was closed with USP 6 polyglactin 910 (Vicryl) and the subcutaneous layer closed with USP 2-0 poliglecaprone 25 (Monocryl), both in a simple continuous pattern. The skin was closed with stainless steel staples (Auto Suture, APPOSE). After closure, the laparotomy wound was covered with sterile gauze and an iodine-impregnated adhesive dressing (Ioban)⁶ and was secured by skin staples.

The inguinal wound was then explored. Any remaining skin sutures were removed and the skin was aseptically prepared. The wound was lavaged with 5 litres of a 0.1% povidone iodine solution. Two deep tracks were identified: a cranioproximal tract in the direction of the inguinal space and an abaxial
Fig 2: Gross anatomical specimens showing anatomy of left cranial abdomen. 

a) Overview of GSL running from the left part of the greater curvature of the stomach to the cranial edge of the spleen (black open arrows). Labels: left lung (a), spleen (b), cranial (Cr), caudal (Ca), dorsal (Do); ventral (Ve). Black dashed line (--) indicating site of GSL transection. Note the thin appearance and continuation of the GSL with the greater omentum at the ventral aspect of the spleen. 

b) Dorsally, the gastrophrenic ligament joins the GSL to the diaphragm and is continuous as a reflection of peritoneum towards the spleen (open black arrows). Labels: left lung (a), spleen (b); diaphragm (c). 

c) Appearance of small intestine through a reconstructed rent (depicted by black open arrows) in the GSL to mimic findings in this case. Labels: left lung (a), spleen (b); diaphragm (c). 

d) GSL at ventral aspect of spleen with black dashed line (--) showing site of transection and Ligasure sealing system in place.
tract towards the medial aspect of the thigh. The skin edges were sharply debrided with a scalpel blade and the tracts were gently debrided with 0.1% povidone iodine impregnated gauze. The wound was left open and packed for recovery with laparotomy sponges. The horse was rope-assisted during recovery from anesthesia, which was uneventful.

Post operatively the horse received a loading dose followed by a CRI of lidocaine (dose as previously described) for 48 h. Intravenous fluids (CSL) were administered at 1.5x maintenance level (75 ml/kg bwt/day) for 12 h post operatively, maintenance level (50 ml/kg bwt/day) for 24 h and then tapered and discontinued over the subsequent 24 h. A follow-up creatinine measurement showed a return to normal concentration, confirming the suspicion of a mild prerenal azotemia at the time of presentation. Muscle enzyme levels gradually resumed normal levels over a 5 day period. Procaine penicillin (22,000 iu/kg bwt i.m. b.i.d.), gentamicin sulphate (6.6 mg/kg bwt i.v. s.i.d.) and metronidazole (15 mg/kg bwt per os t.i.d.) were administered for 4 days post operatively. Ceftiofur (2.2 mg/kg bwt i.v. b.i.d.) was then administered for 5 days followed by 10 days of trimethoprim sulphonamide (30 mg/kg bwt per os b.i.d.). Flunixin meglumine (1.1 mg/kg bwt i.v. b.i.d.) was administered for 72 h, followed by 0.5 mg/kg bwt i.v. b.i.d. for a further 48 h. Oral phenylbutazone was administered at a tapering dose over the subsequent 7 day period.

Clinical recovery was uneventful. Water was reintroduced gradually in increasing amounts. Food was also offered 24 h post operatively in the form of grass and small bran mashes. The amount fed and intervals between feeding were gradually increased to normal levels over a period of 5 days.

The inguinal wound was flushed twice daily with 5 litres of 0.1% povidone iodine solution. The wound edges were sharply debrided again 6 days post operatively and allowed to heal by secondary intention. The horse was discharged from the hospital 10 days post operatively. Recommendations for healing of the abdominal incision included 2 months of strict box rest, followed by one month of in-hand grazing. Instructions to clean the inguinal wound and perform hydrotherapy twice daily were made. Box rest and restricted exercise were advised until the wound had healed, to be gradually increased to normal levels over a period of 5 days.

Seven months after surgery, the horse was presented for a chronic wound in the right inguinal area. Local debridement of the wound edges was performed to reactivate epithelialisation. Follow-up with the owner after an additional 2 months revealed that the wound had completely healed. One year after surgery the horse is back in work and no further episodes of colic have been observed.

Discussion

Abdominal ultrasonography in the colic patient can be a very useful diagnostic tool. Previous studies have confirmed its usefulness for the evaluation of horses with signs of colic including nephro-splenic entrapment of the large colon, a large colon volvulus, right dorsal colon displacements (McGladdery 1992; Santschi et al. 1993) and for distinguishing between large and small intestinal lesions (Klohnen et al. 1996). A previous study also showed that abdominal ultrasound was useful for the evaluation of horses with signs of acute abdominal pain, with a 100% sensitivity and specificity in diagnosing strangulating obstructions of the small intestine (Klohnen et al. 1996); however, abdominal ultrasound could not be used to determine the actual cause of the strangulating obstruction (Klohnen 2008).

To date, there are no papers that describe the specific ultrasonic findings associated with GSL entrapment or the sensitivity associated with this technique. In clinically normal horses and in medical colic cases, small intestine is frequently imaged on the ventromedial surface of the spleen from the left side. Loops of small intestine can also be found close to the ventral edge of the spleen (Klohnen 2008), but not normally abaxial to it. This is in contrast to this case, where multiple loops of distended and thickened small intestine were seen between the left body wall and the spleen. Previous reports have described the intraoperative findings with GSL incarceration involving the small intestine; including a portion of small intestine lying between the spleen and body wall (Yovich et al. 1985). One report of small colon incarceration through a rent in the GSL also described a degree of splenomegaly and displacement of the ventral portion of the spleen towards midline (Marien and Steenhaut 1998). Ultrasound findings of oedematous, small intestine lateral to the spleen preoperatively were suspicious for a GSL entrapment and were confirmed on exploratory laparotomy. Prospective evaluation of a greater number of cases would be necessary to determine if these ultrasound findings are indicative of GSL entrapment.

The anatomy in the region of the GSL is poorly described in the literature. It would be extremely useful to obtain a clearer understanding of the anatomy in this region, especially as this is not easily accessible during dorsal recumbency and exploratory laparotomy. In particular, an extensive blood supply runs within the GSL (Figs 2 and 3) and details of this...
anatomy are important to know for proper haemostasis. This is extremely important as one is working blindly within the abdominal cavity, particularly if a rent is running close to the hilus of the spleen in close proximity to the gastropiploic arteries.

The GSL is a broad, thin band of omentum that runs from the left part of the greater curvature of the stomach to the cranial edge of the spleen (Budras et al. 2003, Fig 2a). Dorsally, the gastrophrenic ligament joins the GSL to the diaphragm. This is constantly as a reflection of peritoneum towards the spleen, and becomes the phrenicocolic and splenorenal ligaments (Fig 2b). The GSL is therefore more fibrous and thicker at the dorsal attachment and becomes progressively thinner and continuous with the greater omentum at the ventral aspect (Fig 2a). The blood supply to the ligament is complex and is largely provided by the gastropiploic artery, which is split into left and right branches (Budras et al. 2003; Fig 3). The origin of this blood supply is the coeliac artery, which branches from the aorta in the cranial abdominal cavity. After a short distance, the coeliac artery branches further into the hepatic artery (travelling to the right side of the abdomen), the left gastric artery (which further subdivides and courses over the surface of the stomach) and the splenic artery travelling to and along the axial surface of the spleen (Barone 1997). The left gastropiploic artery is a continuation of the splenic artery, where it exits at the tip of the spleen and courses through the greater omentum. Multiple branches of the splenic artery, the short gastric arteries, arise at frequent intervals (much like the branches of a tree) along its entire length. These branches travel in both directions into the parenchyma of the spleen. In the axial plane of the spleen, the lesser curvature of the greater omentum. Some of these arteries also track deeper through and underneath the GSL into the adjacent greater omentum (Barone 1997; Fig 3). The role of the GSL is unclear, but it appears to act as a suspensory apparatus and fibroelastic reinforcement between the peritoneal phases; providing structural support to the abdominal organs and viscera (Barone 1997). In the normal horse, small intestine does not normally lie abaxial to this ligament (Yovich et al. 1985; Rhoads and Parks 1999).

Currently, there are no specific diagnostic indicators described that would suggest the presence of a GSL entrapment, only those that indicate involvement of the small intestine and a requirement for surgical correction (Yovich et al. 1985; Trestle and Markel 1993; Jenei et al. 2007). Palpation of distended small intestine on rectal abdominal palpation is an indication of small intestinal involvement; however, this is an inconsistent finding with GSL entrapment (Marien and Steenhaut 1998; Jenei et al. 2007) and is variable with any strangulating small intestinal lesion depending on the time between onset and presentation. Abdominal ultrasound provides an earlier indication and in one study was found to be more accurate than abdominal palpation per rectum in diagnosing a small intestinal strangulating lesion (Klohn et al. 1996). Despite the short duration of clinical signs, resection of 6 m of jejunum was necessary in this horse. In a recent study, the length of small intestinal resection required was found to be a significant risk factor for subsequent development of post operative ileus (Holcombe et al. 2009).

Common to all horses that present with abdominal discomfort, the decision to take the horse to surgery was made following consideration of the history and clinical findings. The history of extreme pain (unresponsive to analgesics), in addition to the ultrasonographic and abdominal palpation per rectum findings, were highly indicative of a strangulating small intestinal lesion and sufficient to justify an exploratory laparotomy in this horse. In particular, the ultrasound findings (oedematous, small intestine lateral to the spleen) were suspicious for a GSL entrapment (Fig 2d) as small intestine would normally be contained within the greater omentum and/or GSL in this area.

Broad spectrum antibiotic cover (including anaerobes) and an adequately long course of antibiotic therapy were considered appropriate in this horse due to the nature of the inguinal wound. An area of fluid accumulation and crepitis had been evident on the peritoneal surface overlying the inguinal wound. No direct communication between the wound and abdomen had been found during surgery and the abdominocentesis performed prior to surgery was indicative of peritoneal inflammation rather than septic peritonitis. However, due to the close proximity of the wound and the potential risk of peritonitis development subsequent to bacterial translocation, appropriate and prolonged antibiotic therapy was considered necessary in this case. In view of this, continued oral antibiotics were selected due to ease of administration and the horse’s disposition to injections. Flunixin meglumine was administered for 5 days post operatively, followed by oral phenylbutazone, at a tapering dose for 7 days. Both were administered for their analgesic and anti-inflammatory effects. In particular, the horse remained quite painful and reluctant to allow flushing and cleaning of the wound. Further debridement was also required 6 days post operatively. The horse was more comfortable and more amenable with continued, low dose phenylbutazone administration.

As described by Marien and Steenhaut (1998), opening rather than enlarging of the GSL rent was necessary to exteriorise the strangulated jejunum in this horse. This was carried out with the Ligasure Vessel Sealing System to prevent haemorrhage from the vessels within the GSL and would be recommended by the authors, especially in cases where the ligament appears more vascular and/or where correction of the incarceration (as in this case) is not possible.

One year after surgery, the horse is clinically normal and the wound in the right inguinal region has fully healed. No further episodes of colic have been noted. This is in agreement with previous cases where reincarceration of small intestine following transection (Marien and Steenhaut 1998) or enlargement (Yovich et al. 1985; Jenei et al. 2007) of the GSL has not been reported. Hypothetically, loss of structural support from the GSL could result in lateral displacement or torsion along the long axis of the spleen. Small intestine could also become displaced around the tip of the spleen and lie adjacent to the left body wall. However, to date, these findings have not been reported.

Transection of the GSL is not frequently performed in man, equids or other species. However, anatomical differences between species, in particular the depth of the body cavity, allows for reasonable access and vision in this region in man and small animals compared to the equine species. The use of laparoscopy has advanced surgical capabilities immensely and many human procedures are now performed via hand-assisted laparoscopies that spare transection of the gastroplenic ligament (Hirota et al. 2009). Therefore, the dilemma as to whether one should repair the GSL or not following transection is rarely an issue and it is also difficult to
make comparisons across species as to the likelihood of potential complications associated with not repairing the GSL. In man, gastric volvulus is unlikely as the stomach is suspended relatively securely by the gastric ligaments (the gastrohepatic ligament along the lesser curvature, the gastrocolic and gastroepiploic ligaments along the greater curvature and the gastrohepatic ligament along the posterior aspect of the fundus). In addition, the oesophagus and duodenum hold the stomach in place superiorly and inferiorly, respectively (Mmicha et al. 1979). If the ligaments are absent or unusually lax, abnormal mobility of the stomach could potentially occur. However, gastric volvulus and abnormal motility of the stomach are most commonly associated with an underlying diaphragmatic defect (diaphragmatic or paraesophageal hernia, Shivanand et al. 2003), and have also been reported as a rare complication of liver transplant surgery (Franco et al. 2005). To the authors’ knowledge, there are no existing reports in the literature that describe gastric volvulus secondary to gastroplenic ligament transection. In dogs, repeated overstretching or abnormalities of the hepatogastric or hepatoduodenal ligaments may allow transposition of the stomach and predispose to gastric dilatation volvulus (GDV) and could also potentially predispose to splenic torsion; however, a recent report investigating any association between splenectomy and any subsequent episode of GDV was discounted (Goldhammer et al. 2010).

In horses, geldings appear to be predisposed to GSL entrapment (Yovich et al. 1985; Marien and Steenhaut 1998; Rhoads and Parks 1999; Jenei et al. 2007); however, there is no anatomical explanation for this finding (Marien and Steenhaut 1998). Other causes of GSL entrapment have also been proposed, including trauma, in addition to congenital defects within the GSL (Jenei et al. 2007). This horse had sustained an inguinal injury 4 days previously, so a traumatic aetiology was suspected; however, a pre-existing congenital defect could not be completely ruled out. An increase in abdominal pressure as a result of trauma, strenuous exercise, breeding, dystocia and severe gastrointestinal distension, may be important in the aetiology of this condition (Trostle and Markel 1993).

The current report is of interest as it highlights the potential future use of ultrasonography in the diagnosis of GSL entrapment. The case also shows that partial transection of the GSL can be beneficial in exteriorisation of incarcerated small intestine and may be considered as an alternative to rent closure. Additionally, the nonrepair of the transected GSL, as in this horse and previously reported cases, does not appear to be associated with any deleterious effects or problems related to recurrence of colic or future athletic performance.

Authors’ declaration of interests
No conflicts of interest have been declared.

Manufacturers’ addresses
1 BCF Technology, Livingston, West Lothian, UK.
2 Veterinary Immunogenics Ltd., Penrith, Cumbria, UK.
3 Fresenius Kabi Ltd., Bad Homburg, Germany.
4 Regent Medical Overseas Ltd., Braunschweig, Germany.
5 Tyco Healthcare UK Ltd., Gosport, Hampshire, UK.
6 3M Health Care, Bracknell, Berkshire, UK.

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