Satellite Article

Internal (intra-abdominal) herniation in the horse

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Summary

Internal abdominal hernias are an uncommon occurrence in the horse. Protrusion of an abdominal organ or viscus may occur through a normal or pathological aperture within the peritoneal cavity, and incarceration of bowel through such an opening may lead to a strangulating obstruction of the intestine, characterised by luminal blockage and concurrent vascular compromise. As such, entrapment of bowel in internal hernias can represent an important indication for colic surgery in the horse.

Introduction

The accompanying report by Kelmer *et al.* (2008) describes an unusual case of small intestinal strangulation due to herniation of a segment of the distal jejunum through a rent in the greater omentum. The case report provides a detailed account of this rare lesion, which has previously been mentioned briefly in the literature by van den Boom and van der Velden (2001), who recorded 2 cases of omental herniation in a series of 224 horses undergoing colic surgery for small intestinal strangulating obstructions. Despite the unsuccessful outcome in this particular case, the report by Kelmer *et al.* (2008) documents another potential cause of small intestinal strangulation, highlights the difficulty in determining the specific cause of small intestinal strangulations preoperatively, and reinforces the need for early diagnosis and surgical treatment in such cases.

Obstructions of the small intestine are an important cause of colic in the horse caused by either mechanical or functional (adynamic ileus) interference of intestinal motility. Simple (mechanical) obstructions occur when the intestinal lumen is partially or completely occluded, but intestinal blood flow is preserved; ileal impaction is a well-recognised example of a mechanical obstruction. Strangulation obstructions develop when there is a luminal blockage and concurrent compromise to the blood supply of the affected intestine, leading to intestinal ischaemia and eventually necrosis and perforation (**Figs 1**–**11**). Strangulation obstructions of the small intestine are estimated to be responsible for between 19–32% of all surgical colic cases (van den Boom and van der Velden 2001). Early recognition of signs associated with strangulation obstruction of the intestine allows prompt surgical intervention and plays a key role in helping ensure a successful outcome.

Pathophysiology of strangulating obstructions of small intestine

The pathophysiology of strangulating obstructions of the small intestine has previously been reviewed by Mair and Edwards (2003).

Clinical presentation and diagnosis

In the acute stages clinical signs caused by strangulation obstructions are associated with severe, unrelenting abdominal pain, which shows no or only temporary response to analgesics. Later in the progression of the disease (after several hours), when the strangulated segment of bowel has become necrotic, signs of pain often subside and the horse becomes increasingly depressed (Edwards and Proudman 2001).

Diagnostic evaluation

Changes in cardiovascular parameters are associated with the ensuing development of endotoxaemia and hypovolaemic shock.

Clinically there is a progressive increase in heart rate and a reduction in pulse quality; respiratory rate is increased in response to the development of metabolic acidosis, and in the later stages of the disease the animal may be observed 'blowing' heavily with flared nostrils. Mucous membranes become congested and capillary refill time is prolonged. Eventually the oral mucous membranes develop a brick-red to purple colour and a 'toxic ring' may be observed. Sweating and muscle fasciculations may also be observed and could potentially be confused for signs of acute grass sickness. Intestinal borborygmi may be decreased, although abdominal

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auscultation is not a reliable indicator of small intestinal motility because sounds of large colon and caecal motility may still be present in the absence of small intestinal activity.
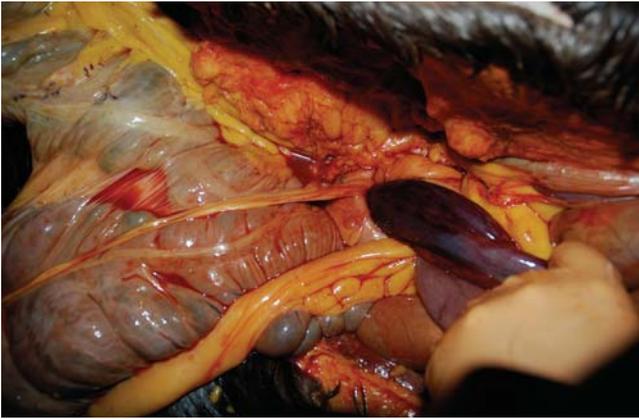

Fig 1a: Jejuno-ileo-caecal intussusception. Abdominocentesis was normal in this case because the ischaemic bowel was compartmentalised from the rest of the abdomen (post mortem appearance).

Fig 1b: On examination there was severe necrosis of the ileum and distal jejunum (intussusceptum) intestine. This horse presented with persistent mild to moderate abdominal pain and signs of severe endotoxaemia.
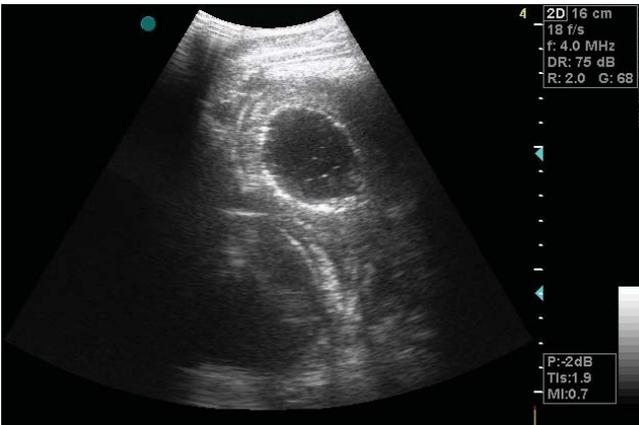

Fig 2: Ultrasonographic image demonstrating a distended loop of small intestine, which has marked mural thickening. When observed in real-time the loop of intestine was amotile.

In the early stages of the disease, laboratory findings may be normal, however with time packed cell volume (PCV) and total plasma protein (TPP) rise progressively reflecting haemoconcentration that occurs due to ongoing fluid and
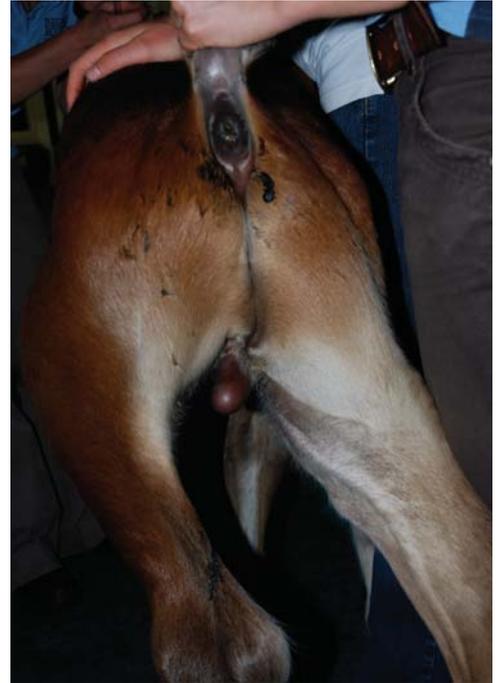

Fig 3: Inguinal hernia in a one-day-old Clydesdale colt. The contents were easily reducible and the condition had resolved spontaneously by age 3 months.

Fig 4a: Direct scrotal hernia in a one-day-old foal that presented showing signs of colic. The contents of the hernia were not reducible.

electrolyte losses. Increased vascular permeability and leakage of plasma proteins from the vascular space further compound these losses. If there is substantial protein loss into the

peritoneal cavity or into the tissues, TPP may not accurately represent the true degree of dehydration. Leucopenia is a common feature in acute severe gastrointestinal tract diseases and neutropenia is especially pronounced in the presence of endotoxaemia (Taylor 2001).

Abdominocentesis can be useful in the investigation of strangulation obstructions, although it can result in false positive and false negative results. Obtaining a serosanguinous peritoneal sample with an increased protein level and WBC is suggestive of a strangulation obstruction, although this does not distinguish between large and small intestinal pathology. It is important to recognise that results of abdominocentesis may be negative in some cases of strangulation obstructions, such as intussusceptions, inguinal hernias or diaphragmatic hernias, because the abnormal peritoneal fluid is compartmentalised

Fig 4b: Laparoscopic image of the foal in Figure 4a. Small intestine is seen passing through the internal vaginal ring.

Fig 5a: Scrotal hernia in an adult stallion. The horse presented with signs of acute severe colic. On physical examination the scrotum was oedematous and cold. Incarcerated small intestine is being held on the left side of the picture. The pampiniform plexus (right side of the picture) is swollen/congested due to venous obstruction.
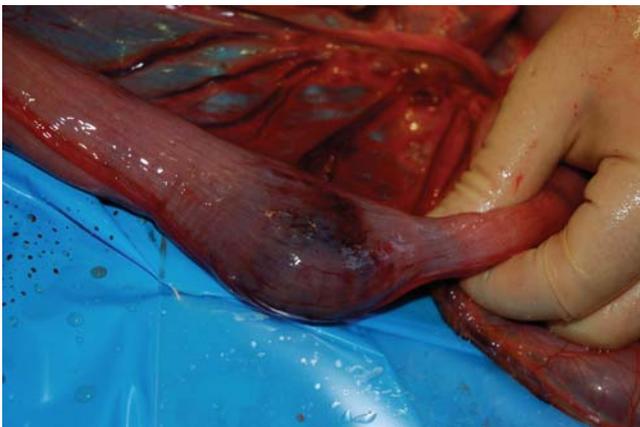

Fig 5b: Image of the incarcerated segment of ileum following open surgical reduction.

Fig 6a: Strangulating obstruction due to epiploic foramen entrapment of the small intestine. Haemorrhagic strangulating obstruction of multiple loops of small intestine. The affected bowel is congested due to occlusion of the veins occurring before occlusion of the arterial supply.
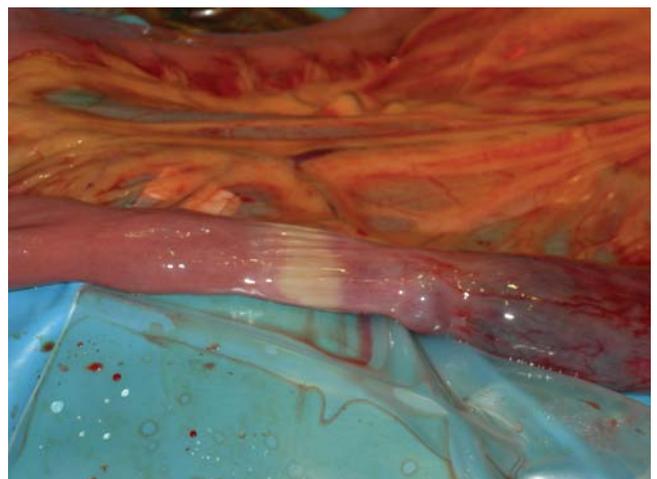

Fig 6b: A circumferential band of ischaemic jejunum was observed at the site of constriction by the epiploic foramen. The blanched area indicates ischaemic strangulating obstruction due to occlusion of both the veins and arterial supply simultaneously.

from the rest of the abdominal cavity (**Fig 1**). In addition, other diseases such as anterior enteritis or nonstrangulating intestinal infarction can also cause similar discolouration of the peritoneal fluid.

Progressive fluid and gas distension of the small intestine is a principle feature of small intestinal obstructions. Build-up of fluid in the bowel proximal to the obstruction eventually results in distension of the stomach. Obtaining a net volume of more than 2 l of gastric reflux on passage of a nasogastric tube is considered abnormal; however, significant volumes of gastric reflux can also be obtained with both strangulating and nonstrangulating obstructions and therefore is not a definitive indication for surgical intervention in all cases.

Distended loops of small intestine can be detected on rectal palpation (Kopf 1987; White 1998). Multiple loops of taut, distended small intestine may be obvious in the caudal abdomen and pelvic inlet region if the obstruction has been present for several hours. However, in the acute stages, distended loops may be too far cranial and ventral to palpate. In such instances careful palpation over a period of several minutes may facilitate detection of one or 2 distended loops (Edwards and Proudman 2001).

In recent years transabdominal ultrasonography has become an invaluable tool in the investigation of abdominal pain in the horse. The technique is noninvasive, relatively inexpensive and with experience is quick and easy to perform. In horses with acute abdominal pain, it has been shown that abdominal ultrasonography is more sensitive than rectal palpation for diagnosing small intestinal strangulation obstructions, reflecting the fact that more of the abdominal cavity can be examined using ultrasonography and therefore earlier detection of lesions cranial to the limits of one's reach may be possible (Klohnen *et al.* 1996; Klohnen 2008). In addition, abdominal ultrasonography is valuable for evaluation of colic in patients whose size precludes rectal examination. Ultrasonographic evidence of small intestinal strangulation obstruction includes the presence of loops of distended, amotile small intestine with increased mural thickness (**Fig 2**).

The clinical presentation of the case described by Kelmer *et al.* (2008) was consistent with strangulation obstruction of the small intestine. However, there was no indication of the cause of the primary lesion. The indication for surgery was enforced by level of pain, duration of signs and positive findings on physical examination; rectal palpation; ultrasonography and nasogastric reflux.

Such positive findings may not be obvious in all cases, perhaps due to the stoical nature of the individual, or in the early stages of the disease or due to masking of clinical signs following administration of analgesics. In these cases repeated systematic examinations may be necessary before a decision to perform surgery is reached.

Strangulation obstructions of the small intestine

There are many potential causes of strangulation obstruction of the small intestine in the horse. In 2 large retrospective studies the most common cause of strangulation obstruction of the small intestine varied depending upon the population of horses treated. Mair and Smith (2005) reported strangulation by pedunculated lipomas as the most common lesion whilst van den Boom and van der Velden (2001) reported epiploic foramen entrapment as the most common cause. In the second study the authors suggested that this was because there were only a small number of ponies seen in their caseload. Based upon these 2 studies the commonest causes included (listed in approximately decreasing frequency):

Pedunculated lipomas
Epiploic foramen entrapment
Incarceration in inguinal/scrotal hernia
Volvulus
Incarceration in mesenteric rent
Incarceration by fibrous band/adhesions
Intussusception
Incarceration in gastroplenic ligament
Diaphragmatic hernia
Incarceration in umbilical hernia
Other (including omental hernia)

Abdominal hernias

A 'true' hernia comprises of 3 parts: the orifice (ring), the sac and the contents, whilst strictly speaking the passage of abdominal viscera through a mesenteric or peritoneal aperture without a sac results in a prolasus or procidentia (Armstrong *et al.* 2007). The nomenclature used to describe specific hernias usually indicates the anatomic location of the responsible hernia orifice, rather than the nature tissue that is prolapsed (e.g. inguinal and umbilical hernias or epiploic foramen entrapment, etc.). Hernias may be further characterised as congenital or acquired, depending on whether they are present at birth. The contents of the hernial sac may be described as reducible when they can be freely manipulated back through the hernial ring, incarcerated when they cannot be reduced and strangulated if the vascular supply to the herniated tissue contents becomes compromised (Stick 2006).

There are many causes of abdominal hernias. External hernias (i.e. abdominal wall hernias) are caused by the prolapse of intestine, omentum or mesentery through a defect in the abdominal wall. Defects in the body wall may arise secondary to trauma (e.g. lateral abdominal hernias) or as a result of incisional complications (ventral midline hernias); however, strangulation obstructions rarely develop as a result.

Umbilical hernias are the most common type of external hernia in the horse and are estimated to occur in 0.5–2.0% of foals. Most umbilical hernias are congenital, but they may also develop within the first 2 months after birth (Enzerink *et al.* 2000). Most umbilical hernias are considered a cosmetic defect. Strangulation of the intestine in an umbilical hernia is a rare and serious complication.

Inguinal hernias may be congenital or acquired. Congenital herniation of small intestine through the inguinal canal into the

vaginal tunic (so-called 'indirect' inguinal or scrotal hernia) is often observed in young colts and the contents are usually reducible (**Fig 3**). Intestinal obstruction is rare and the condition usually resolves spontaneously during the first few months of life. Occasionally intestine may herniate through a tear in the vaginal tunic distal to the vaginal ring or through a rent in the peritoneum and transverse fascia, adjacent to the vaginal ring (so-called 'direct' inguinal or scrotal hernia). The intestine lies in the subcutaneous space of the scrotum and prepuce, resulting in severe oedematous swelling and skin necrosis; although strangulation is uncommon this condition represents a surgical emergency (Freeman 2006) (**Fig 4**).

Acquired inguinal hernias in adult stallions usually result in strangulation obstructions and represent an important differential diagnosis for acute colic in entire males. Usually a small segment (10–15 cm) of ileum or jejunum is incarcerated and the testicle becomes enlarged and cold due to compression of the testicular vessels. Prompt surgical correction with or without resection of the affected intestine is indicated, and castration is usually performed (**Fig 5**). Recently, encouraging results have been reported using a manual closed nonsurgical reduction technique followed by delayed laparoscopic closure of the vaginal ring (Wilderjans 2007).

Internal (intra-abdominal) hernias result from the protrusion of an abdominal organ or viscus through an aperture in the peritoneum or mesentery, leading to its encapsulation within another compartment of the otherwise intact abdominal cavity. From an anatomical perspective, intraperitoneal openings may be classified as normal or abnormal. Normal intra-abdominal apertures in the horse include the epiploic foramen (foramen of Winslow) and the nephrosplenic space.

Abnormal or pathological openings include defects or tears in the mesentery (Gayle *et al.* 2000), greater omentum (Kelmer *et al.* 2008) or intra-abdominal ligaments including: gastrosplenic ligament (Trostle and Markel 1993; Jenei *et al.* 2007), broad ligament (Becht and McIlwraith 1980), lateral ligament of the urinary bladder (Hawkins *et al.* 1993) and caecocolic fold (Gayle *et al.* 2001) or form secondary to congenital mesodiverticular bands (Abutarbush *et al.* 2003).

Eplploic foramen entrapment (EFE) is the most common cause of internal herniation in the horse and reportedly accounts for 5–23% of all strangulating lesions of the small intestine (Archer *et al.* 2008). The epiploic foramen is a slit-like aperture that forms the entrance from the peritoneal cavity into the omental bursa and is located in the right dorsal abdomen. The caudate lobe of the liver and the caudal *vena cava* form the dorsal anatomical boundary, whilst the pancreas, hepatoduodenal ligament and the portal vein contribute to the ventral border (Sisson 1975).

Most frequently strangulation obstruction occurs when small intestine (ileum and jejunum) herniates through the epiploic foramen (**Fig 6**). A variable length of intestine usually passes from left to right through the foramen, to lie in the right side of the abdomen above the duodenum. Consequently the intestine must tear through the omentum before passing through the foramen (Vachon and Fischer

1995). Horses usually present with signs of severe abdominal pain, although some horses may not show evidence of pain, particularly if analgesic drugs have been administered (Vachon and Fischer 1995).

Archer *et al.* (2008) recently reported risk factors associated with development of the disease. Crib-biting/windsucking behaviour was associated with the greatest increase in likelihood of EFE. Other factors associated with increased likelihood of EFE included: greater height; a previous episode of colic within the previous 12 months; increased duration of stabling within the previous 28 days.

Transmesenteric rents

Abnormal defects in the mesentery may be congenital or acquired. Acquired tears can arise spontaneously, presumably secondary to trauma (e.g. during pregnancy), excessive stretching of the mesentery due to another lesion (e.g. ileal impaction) or rough manipulation of the mesentery during previous abdominal surgery (Hance *et al.* 1991). Most mesenteric hernias involve defects in the mesentery of the jejunum but can also occur in the mesentery of the ileum, duodenum or the mesocolons (**Fig 7**). Horses of all ages can be affected and the condition is more common in females (Gayle *et al.* 2000). Congenital defects can be secondary to mesodiverticular bands, which result from persistence of the left vitelline artery and associated mesenteric folds during embryological development. These fibrous bands extend from the mesentery to the antimesenteric border of the distal jejunum or proximal ileum, forming a potential triangular space, through which small intestine can herniate (Freeman *et al.* 1979; Abutarbush *et al.* 2003) (**Fig 8**).

Surgical management of mesenteric hernias can be challenging because access to the lesion via a ventral midline approach may be limited. In addition, reduction of the hernia can be extremely difficult and can lead to severe intra-abdominal haemorrhage (Gayle *et al.* 2000). Laparoscopy, particularly in the standing horse offers better surgical access to structures in the dorsal abdomen and the technique has been used successfully to repair a defect in the mesoduodenum of a Thoroughbred mare (Sutter and Hardy 2004).

Although considered separately, iatrogenic, post surgical hernias can occur through residual defects in the mesentery following resection and anastomosis of the small intestine (**Fig 9**).

Gastrosplenic ligament

The gastrosplenic ligament is a broad attachment between the left part of the greater curvature of the stomach and the hilus of the spleen. Herniation of bowel through a defect in the ligament is occasionally encountered. Small intestine is usually involved and passes from caudal to cranial through the defect, resulting in the strangulated loop being located lateral to the stomach and craniolateral to the spleen. Herniation of small or large colon has been reported (Trostle 1993; Rhoads and Parks 1999; Jenei *et al.* 2007).

Adhesions

Intra-abdominal adhesions can potentially cause obstruction of the small intestine. Simple obstructions can result from extraluminal compression or 'kinking' of the bowel, which leads to obstruction of intestinal transit (**Fig 10**). On rare occasions adhesions may form a hole, through which bowel can become incarcerated or strangulated, but most adhesions are asymptomatic.

Adhesion formation is an important complication in all horses that have undergone previous colic surgery. In one study

Fig 8b: Strangulation of the small intestine due to a ruptured congenital mesodiverticular band.

Fig 7: Incarceration of small intestine through a rent in the ascending mesocolon. Courtesy of Professor Christoph Lischer.

Fig 9: In this case prolapse of the small intestine occurred through a mesenteric defect present following a previous jejunocaecostomy procedure.
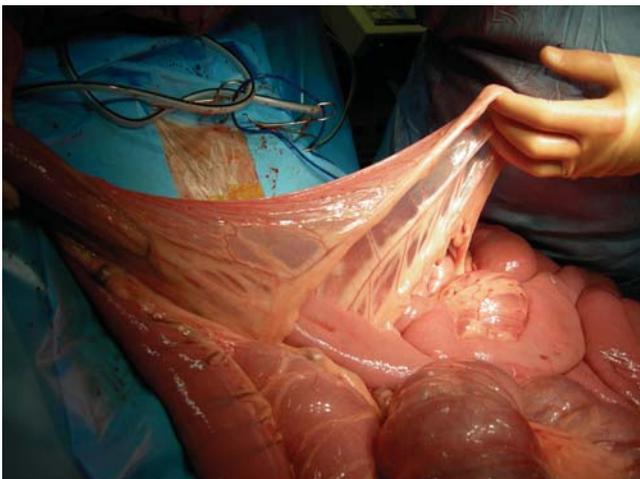

Fig 8a: Example of a congenital mesodiverticular band between the mesentery and the antimesenteric border of the jejunum (incidental surgical finding in this case).
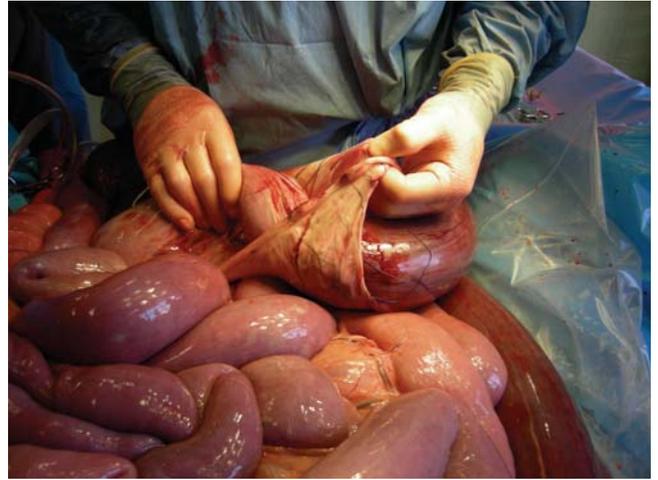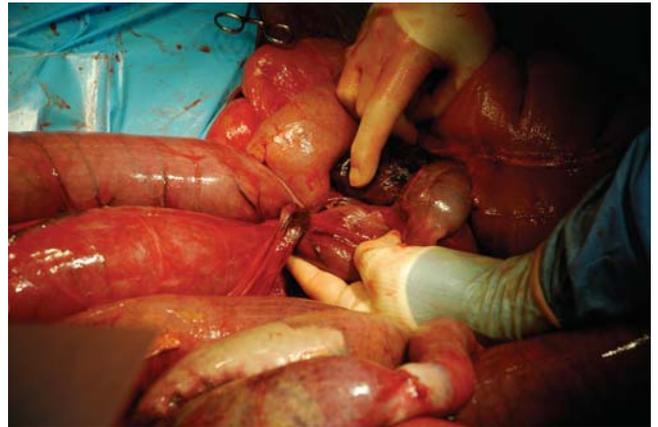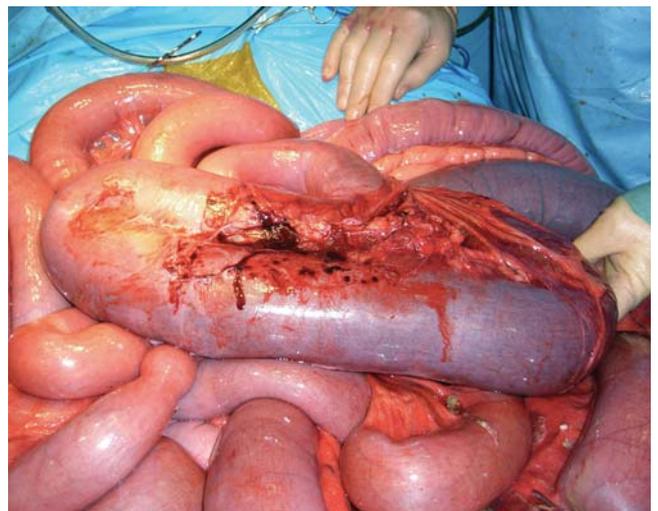

Fig 10: Small intestinal obstruction caused by serosal and omental adhesion formation. This horse had no prior history of abdominal surgery.

Fig 11: Post mortem appearance of the thorax in a horse with a diaphragmatic hernia. The large colon occupies most of the thoracic cavity. The horse presented with severe colic but died on induction of general anaesthesia.

22% of 172 horses that had undergone surgery for small intestinal obstruction developed abdominal adhesions that required additional surgery or euthanasia (Baxter *et al.* 1989). In a recent study by Gorvy *et al.* (2008) adhesions were found in 32.3% (32/99) of horses undergoing repeat laparotomy and were considered pathological in 84.4% of these cases. Interestingly, in this study, adhesion formation was not associated with the site of the primary lesion (small intestine vs. large intestine), having resection of bowel performed or evidence of endotoxaemia, supporting the theory that surgical trauma is an important stimulus for adhesion formation.

Adhesions can also be found in horses that have not undergone previous abdominal surgery (**Fig 10**). Intestinal inflammation is thought to be the most likely stimulus. Potential causes include: intra-abdominal abscesses; parasite migration or peritonitis (possibly following castration) (Mair and Edwards 2003).

Diaphragmatic hernias are a separate rare form of internal hernias. Diaphragmatic defects may be congenital or acquired following trauma (Spiers and Reynolds 1976; Dabrainer and White 1998; Wilkins 2007). Congenital defects usually have smooth, rounded edges and can be quite small. Traumatic rents tend to be larger with irregular and rough borders and are usually located in the tendinous portion of the diaphragm. They usually occur subsequent to a recent event that caused a severe and sudden increase in intra-abdominal pressure, such as severe exercise, parturition, blunt trauma or a fall. Small or large intestine can pass through the diaphragmatic defect into the thoracic cavity leading to variable clinical signs. Horses with diaphragmatic hernias may present with signs of respiratory distress if the large colon is displaced into the thoracic cavity (**Fig 11**). Displacement of small intestine through the rent invariably leads to signs of simple or strangulation obstruction. In a horse showing these clinical signs, the index of suspicion of a diaphragmatic hernia may be raised if the caudal abdomen appears unusually 'empty' on rectal examination and obvious borborygmi are detected on auscultation of the thorax, although these findings are not definitive. It is important to realise that results of abdominocenteis may be negative because abnormal peritoneal fluid associated with the ischaemic bowel is sequestered in the thoracic cavity. Confirmation of the diagnosis is most accurately achieved using ultrasonograpic or radiographic examination of the thorax. Occasionally diaphragmatic hernias result in low grade, chronic recurrent pain or may even be detected as an incidental finding at *post mortem* examination.

Conclusion

As demonstrated in the case report by Kelmer *et al.* (2008) there are few clinical features that help distinguish between the different causes of internal herniation resulting in strangulating obstructions of the small intestine, and in the majority of cases a diagnosis has to be made at surgery. In this case it could be speculated that the successful surgical treatment may have been possible if the horse had been referred earlier, prior to development of widespread vascular embarrassment of the small intestine. By the time the horse was taken to surgery, evidence of severe colic had been present for 2 days. Early surgical treatment may not be possible in some cases due to the lack suitable surgical facilities within a short distance or may be due to a number of owner related factors, such as financial restrictions or lack of transport. In some cases owners may initially decline the option for surgery only to change their mind and it is therefore important for the primary attending surgeon to effectively communicate the potential implications of delaying referral for surgical treatment.

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