Clinical Commentary

Inguinal herniation and rupture in horses

J. Schumacher* and J. Perkins†

*Department of Large Animal Clinical Sciences, 2407 River Drive, College of Veterinary Medicine, University of Tennessee, Knoxville, Tennessee 37996, USA; and †Department of Veterinary Clinical Sciences, Royal Veterinary College, Hawkshead Lane, North Mymms, Hatfield, Hertfordshire AL9 7TA, UK.

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Introduction

Inguinal herniation occurs when an abdominal viscus, nearly always intestine, protrudes through a vaginal ring into the inguinal canal. The condition is often referred to as scrotal herniation if the contents of the hernia extend into the scrotum, but the terms scrotal herniation and inguinal herniation are used interchangeably (Schneider et al. 1982; Cox 1988). Inguinal or scrotal herniation is sometimes also referred to as 'indirect herniation', terminology taken from descriptions of the analogous condition in men (Cox 1988). The hernia is referred to as a ruptured inguinal hernia if the contents of the hernia protrude through a tear, or rupture, in the vaginal sac so that the contents of the hernia protrude through a tear, or rupture, in the vaginal sac so that the contents of the hernia lie in fascia in the inguinal or scrotal region adjacent to, but outside of, the vaginal sac (van der Velden 1988a).

The opening in the abdomen through which the herniated viscus protrudes into the inguinal canal is the vaginal ring (Fig 1), which is the opening of the vaginal sac, or vaginal process, which is a diverticulum of the abdominal peritoneum that occupies the inguinal canal and scrotum. This diverticulum is composed of a visceral tunic (tunica vaginalis propria), which is that portion of the diverticulum that envelops the testis and spermatic cord, and the parietal tunic (tunica vaginalis communis), which is continuous with the abdominal peritoneum at the vaginal ring. The parietal tunic lines the scrotal (vaginal) cavity, which is the cavity between the parietal and visceral tunics (Sisson and Grossman 1953; Rooney et al. 1967).

Inguinal rupture describes a condition characterised by protrusion of a viscus through a tear, or rupture, in the abdominal peritoneum adjacent to the vaginal ring, causing the viscus to become trapped in fascia adjacent to, but outside of, the vaginal sac (Fig 2) (Cox 1988). Inguinal ruptures have been referred to as 'direct hernias' (Vasey 1981), but this terminology is derived from descriptions of hernias, not ruptures, in men and wrongly describes the condition in horses (Cox 1988). In men suffering from a direct hernia, the peritoneum and fascia next to the vaginal ring are weak, causing formation of a peritoneum-lined, viscus-filled diverticulum, whereas in horses suffering from an inguinal rupture, the viscus has passed through a rupture in the peritoneum adjacent to the vaginal ring, and consequently, is surrounded by scrotal fascia, rather than by peritoneum. The so-called 'direct hernia' of horses is a rupture, not a hernia (Kersjes et al. 1985; Cox 1988), and the viscus that protrudes through the rent should not be referred to as being 'herniated' (Figs 3 and 4).

Aetiology

The usual cause of inguinal herniation is a congenitally enlarged vaginal ring, and the mechanism by which congenital inguinal herniation occurs may be excessive growth of the extra-abdominal portion of the gubernaculum resulting in a vaginal sac with an unusually wide neck (Wensing et al. 1980; Budras et al. 1994). Although inguinal herniation has been reported to occur in geldings (Wright 1963; Schneider et al. 1982; Bickers et al. 1998) and in a mare (Umstead et al. 1986), it occurs nearly exclusively in stallions (Schneider et al. 1982; van der Velden 1988a,b). Geldings are at low risk of developing an inguinal hernia because their vaginal rings reduce in size soon after castration.

Inguinal hernias of foals are congenital, and most are probably hereditary (Roberts 1971). Inguinal herniation occurs much more commonly on the left side, but right inguinal herniation and bilateral inguinal herniation occur occasionally (Hutchins and Rawlinson 1972). The herniated viscus of a foal usually retains adequate blood supply and can usually be reduced temporarily into the abdomen. If the hernia is long-standing, the testis may atrophy (Wright 1963). Compression of the foal’s abdomen during parturition may cause the vaginal sac to rupture resulting in escape of the hernial contents into the surrounding scrotal fascia (i.e. ruptured inguinal hernia) (Spurlock and Robertson 1988; van der Velden 1988a).
Inguinal hernias of adult horses are usually acquired, but probably occur because one or both vaginal rings are congenitally enlarged (Sembrat 1975). Because they result from a congenitally enlarged vaginal ring, most occur on the left side. The incidence of acquired inguinal herniation is greater in Standardbreds, draught breeds and Andalusian horses than in the general equine population (Sembrat 1975; Shoemaker et al. 2004; Munoz et al. 2008). Another breed that may have an increased incidence of acquired inguinal herniation is the Tennessee Walking Horse (authors, personal observation). Inguinal herniation can be acquired during strenuous exercise or copulatory movements, either of which may alter the configuration of the vaginal rings and inguinal canals and increase abdominal pressure (Schneider et al. 1982). Small intestine is the most commonly herniated viscus, but inguinal herniation of small colon and large colon has been reported (Wheat 1975; Ivens et al. 2009).

Inguinal rupture of horses is rare, based on the scarcity of reports describing horses with this condition. Even though rupture must be associated with a traumatic event, this event is not always obvious, as in the case of inguinal rupture described by Cousty et al. (2010) in this issue. The cause of inguinal rupture of the pony of this report was not determined, but rupture was thought to have occurred...
during a normal parturition 3 months before the pony was presented to the authors for evaluation. A stallion reported by Vasey (1981) developed signs of severe colic caused not only by escape of intestine through a rent in the peritoneum adjacent to the vaginal ring (i.e. inguinal rupture) but also by protrusion of a loop of intestine through the vaginal ring into the vaginal cavity (i.e. inguinal herniation). The inguinal rupture was thought to have occurred 4 weeks previously when the horse fell. The case of inguinal rupture reported by Cousty et al. (2010) is interesting not only because so few horses with the condition have been reported but also because of the apparently long duration of the condition and because the viscus that entered the rent was bladder rather than intestine.

**Fig 6:** Transcutaneous ultrasonogram showing oedematous small intestine (arrow) in right inguinal region, proximal to right testis. SC = spermatic cord.

**Fig 7:** A parietal inguinal hernia. The antimesenteric surface of the ileum had become incarcerated by the vaginal ring causing small intestinal distension. Intestinal resection was not required.

**Fig 8:** Laparoscopic view of the left inguinal region of a one-month-old foal with a nonreducible inguinal hernia. B = bladder; SI = small intestine; M = mesentery of the herniated small intestine; VR = vaginal ring.

**Fig 9:** Laparoscopic view of the left inguinal canal of a one-month-old foal with a nonreducible inguinal hernia. Traction is being applied with Babcock forceps to return the small intestine to the abdomen. B = bladder; SI = small intestine.

**Fig 10:** Laparoscopic view of the left inguinal region of the foal in Figure 9. The testis has been pulled into the abdomen. Note that the vaginal tunic has been partially inverted into the abdomen (IVT). T = testes; UA = umbilical artery.
Diagnosis

The history and clinical findings displayed by a horse suffering from an inguinal hernia or an inguinal rupture may include acute onset of signs of severe colic caused by strangulation of the herniated viscus. The inguinal or scrotal region may enlarge because of the presence of a viscus within or outside of the vaginal sac and because of enlargement of the testis caused by partial occlusion of the spermatic vasculature. Occlusion of the spermatic vasculature results in oedema of the testis and eventually in testicular degeneration. The testis may degenerate even after the incarcerated viscus has returned to the abdomen. Physical examination may reveal differences between the right and left testes in size, shape, texture, temperature and tenderness to palpation. Although an acute increase in testicular size is suggestive of inguinal herniation, it is also suggestive of torsion of the spermatic cord, orchitis or thrombosis of the testicular vasculature. The clinician should also be mindful that the left testis of a normal stallion is usually slightly larger and slightly more pendulous than the right testis (Fig 5). A stallion that develops signs of colic after copulation or exercise should be suspected of having a strangulated inguinal hernia or rupture, especially if the signs of colic are accompanied by a swollen and tender testis and spermatic cord.

Palpation of nonstrangulated intestine within the inguinal canal or scrotum may elicit a sensation of crepitus, and peristalsis of the intestine may occasionally cause the skin overlying the intestine to move (van der Velden 1988b). Borborygmi may be heard during auscultation of the affected spermatic cord. A viscus protruding through a rent adjacent to the vaginal ring (i.e. an inguinal rupture) or within the vaginal sac (i.e. a ruptured inguinal hernia) may separate the skin from its vasculature. The clinician should also be mindful that the spermatic cord, orchitis or thrombosis of the testicular vasculature is less echogenic than its normal counterpart. Ultrasonography is rarely helpful in identifying whether the small intestine has gone through the inguinal canal or through a rupture in the peritoneum (authors’ observations). Failure to palpate or image small intestine within the scrotum does not rule out the presence of a parietal inguinal hernia, where only the antimesenteric side of the small intestine is incarcerated by the vaginal ring (Fig 7).

Treatment of horses for inguinal herniation or rupture

Foals that have an inguinal hernia usually suffer no distress from the hernia (Fig 3) and experience spontaneous resolution of the hernia by the time they are 3–6 months old (Wright 1963; Goetz et al. 1981; van der Velden 1988b). Frequent manual reduction or applying a truss after the hernia has been reduced manually may promote spontaneous resolution of the hernia (Varner 1991). Care must be taken when applying the truss to avoid pressure that could damage the skin. The owner should be warned that congenital inguinal herniation is often heritable so that the foal, once matured, is not used for breeding.

Horses with an acquired inguinal hernia, a ruptured congenital inguinal hernia, or an inguinal rupture must usually be treated immediately after the condition is recognised because the contents of the hernia or rupture are likely to rapidly lose blood supply. An incarcerated inguinal hernia can sometimes be reduced nonsurgically by external manipulation or by traction per rectum, provided that reduction is attempted soon after the viscus becomes entrapped, before the vascular supply to the herniated viscus becomes severely compromised (Goetz et al. 1981). Incarcerated intestine can be assessed ultrasonographically for viability by measuring the thickness of the intestinal wall and by observing the intestine for motility.

To return the herniated viscus into the abdomen using external manipulation, the horse is sedated, and traction is placed on the testis of the affected spermatic cord so that the vaginal sac is tensed into a straight, rigid tube (Goetz et al. 1981). The spermatic cord is grasped above the testis (Schneider et al. 1982; van der Velden 1988b); therefore, palpation of the vaginal rings per rectum should be part of the physical examination of every adult stallion displaying signs of colic. Clinical signs of acquired inguinal herniation are usually those associated with obstruction of small intestine, such as nasogastric reflux, after nasogastric intubation, and distension of small intestine, detected during examination of the abdomen by palpation per rectum or during transabdominal or transrectal ultrasonographic examination of the abdominal viscera. Peritoneal fluid obtained by abdominocentesis is often normal, even in horses with a strangulating lesion of the small intestine. Ultrasonographic examination of the scrotum and inguinal region can be helpful in confirming the presence of small intestine adjacent to the testis and may provide an indication of intestinal viability (Fig 6). The affected testis of a stallion suffering from inguinal herniation is less echogenic than its normal counterpart. Ultrasonography is rarely helpful in identifying whether the small intestine has gone through the inguinal canal or through a rupture in the peritoneum (authors’ observations). Failure to palpate or image small intestine within the scrotum does not rule out the presence of a parietal inguinal hernia, where only the antimesenteric side of the small intestine is incarcerated by the vaginal ring (Fig 7).
desensitised with an anaesthetic agent administered into the epidural space. The horse should be observed closely for signs of colic and endotoxaemia if the hernial contents have been reduced by rectal traction or by external manipulation because the viability of the reduced viscus and testis cannot be assessed directly. Periodic analysis of peritoneal fluid obtained by abdominocentesis may allow early diagnosis of intestinal necrosis, but a normal analysis of abdominal fluid should not be relied upon.

A congenital inguinal hernia of a foal should be reduced surgically if the hernia cannot be reduced by external manipulation, fails to resolve or enlarges, is so large that spontaneous reduction is unlikely, or has migrated from the vaginal cavity into scrotal sac through a rupture in the vaginal sac (Marien et al. 2001). To surgically reduce an inguinal hernia, the foal is anaesthetised and positioned in dorsal recumbency. The skin over the superficial inguinal ring of the affected side is incised to expose the vaginal sac containing the testis and the herniated viscus. The vaginal sac is bluntly separated from surrounding fascia, and the scrotal ligament, which attaches the vaginal sac to the scrotum, is transected. The vaginal sac is tensed by placing traction on the testis, and the contents of the hernia are pushed back into the abdomen. Twisting the vaginal sac may facilitate return of the contents of the hernia. The vaginal sac is incised to expose the testis and the contents of the hernia. Another method used to prevent herniation, while preserving the testis, is to laparoscopically release a flap of peritoneum adjacent to vaginal ring and then to transpose and staple or suture this flap over the vaginal ring (Wilderjans et al. 2007).

References


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