

Case Report

Spinal cord trauma in a recently foaled Friesian mare as a complication of ventral abdominal rupture

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Introduction

Rupture of the prepubic tendon or of ventral abdominal wall muscles occurs occasionally in mares during late gestation and most commonly in draught horse breeds (Adams 1979; Lofstedt 1993; Kawcak and Stashak 1995; Mirza *et al.* 1997; Wolfsdorf 2003). Although the precise aetiology of ventral abdominal rupture is not clear, extraordinary uterine weight as a result of hydrops of the fetal membranes or twins is sometimes a predisposing factor (Hanson and Todhunter 1986; Kaneps 1992; Lofstedt 1993; Kawcak and Stashak 1995; Wolfsdorf 2003).

In many cases, abdominal wall rupture is preceded by severe, progressive ventral oedema. In other cases, the abdominal wall remains intact but the attachment to the pelvis, the prepubic tendon, ruptures; this can be recognised by the characteristic cranial and ventral displacement of the udder so that it becomes confluent with the ventral abdomen. In early stages of abdominal wall rupture, palpation to determine muscular integrity can be difficult because of oedema and abdominal 'guarding' due to acute pain. However, once rupture is complete the deficit in the abdominal wall is usually readily palpable and the absence of the muscle layers can be confirmed by ultrasonographic examination. In the case of an incipient rupture, acute colic is a common reason for submission and/or hospitalisation, where pain results from muscular inflammation, the need for structures other than the abdominal muscles (e.g. the vertebral column) to support the viscera or, in rare cases, due to incarceration of viscera (Kaneps 1992).

Mares with a ruptured prepubic tendon tend to walk stiffly, prefer not to lie down and, if they do, often have trouble getting back up. Typically, affected mares adopt a lordotic 'sawhorse' stance with the tail head and sciatic tuberosities becoming elevated because there is no longer any ventral tension to maintain the normal anatomical

relationship between pelvis and spine (Hanson and Todhunter 1986; Arighi 1992; Kaneps 1992; Lofstedt 1993; Kawcak and Stashak 1995; Mirza *et al.* 1997; Wolfsdorf 2003).

In a case of ventral rupture, the decision to induce abortion/parturition immediately, to let pregnancy progress until there are clear signs of fetal 'readiness for birth', or to subject the mare to euthanasia, depend largely on the existence and nature of a predisposing cause, estimated extent of muscle/tendon damage, stage of gestation and severity of the pain. For example, in the case of hydrops of the fetal membranes, the recommended treatment is to initiate abortion, but to perform gradual transvaginal removal of the fetal fluids coupled with fluid therapy/circulatory support to prevent the mare suffering from hypovolaemic shock. When the cause of herniation is unknown but fetal survival is considered of paramount importance, the mare is confined to a stall and fed a low bulk diet, while an abdominal support device is often used to try and prevent progression of the muscle damage (Hanson and Todhunter 1986; Kaneps 1992; Lofstedt 1993; Kawcak and Stashak 1995; Wolfsdorf 2003).

When recovery of the mare is the primary concern, parturition is often induced in the hope of avoiding irreversible body wall damage; in the authors' experience, this is usually in vain. Parturition, however it is initiated, almost always requires assistance because the absence of an intact abdominal wall renders the mare incapable of useful abdominal contractions (Hanson and Todhunter 1986; Kaneps 1992; Lofstedt 1993; Kawcak and Stashak 1995; Wolfsdorf 2003). When abdominal wall damage is extensive or the prepubic tendon has ruptured the long-term prognosis for the mare is poor; future use as a broodmare (except via embryo transfer) or for any type of work is extremely unlikely.

This report describes a case of ventral rupture in which even the simple hope of managing the mare long enough to raise her foal was complicated by spinal cord trauma due to the unusual stress exerted by the unsupported abdominal viscera.

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Case details

History

An 11-year-old multiparous Friesian mare was presented at Utrecht University's equine clinic at approximately 320 days of gestation to investigate the cause and severity of sudden-onset, painful ventral abdominal oedema and enlargement of approximately 24 h duration.

Clinical findings

A general clinical examination revealed elevations in both the pulse and respiratory rates (64 beats/min and 32 breaths/min, respectively) and a grossly oedematous ventral abdomen (Figs 1 and 2) with abdominal contents palpable on both sides. The swelling on the left was larger than that on the right (approximately 40 x 20 x 10 cm vs. 15 x 10 x 4 cm) and, although the extent of the muscle damage was difficult to palpate, ultrasonographic examination using a 3.5 MHz curved array probe demonstrated the presence of small intestine beneath the skin in the swelling on the left hand side. On the right hand side, it appeared that at least one muscle layer was still intact. *Per rectum* examination revealed a live foal and a firm, narrow cervix. The udder was moderately enlarged but the mammary secretions were serum-like in both appearance and electrolyte composition ($[Na^+] > 100$ mmol/l; $[K^+] < 20$ mmol/l; $[Ca^{2+}] < 4$ mmol/l).

Clinical follow-up

Initial pain was controlled with an i.v. injection of 600 mg flunixin meglumine (1 mg/kg bwt i.v. b.i.d.; Finadyne)¹, the mare was confined to a foaling stall and a leather harness

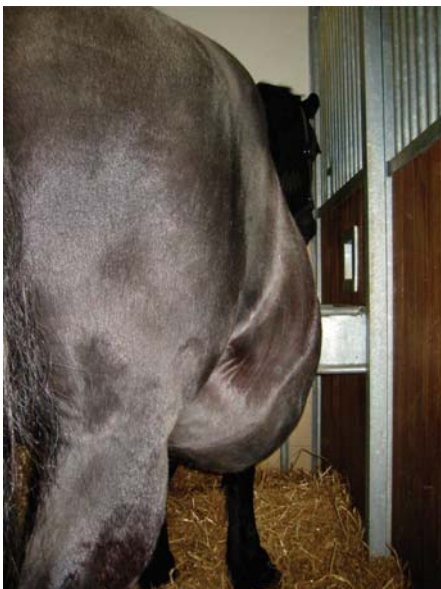


Fig 1: Lower right flank of an 11-year-old Friesian mare with a ventral hernia.



Fig 2: Posture of an 11-year-old Friesian mare with a ventral hernia.

used to support the abdomen. The harness was well padded and removed daily to prevent pressure necrosis, a previously described complication. The roughage content of the mare's diet was reduced and she was fed a laxative diet. During the following 2 days, the mare was comfortable with no obvious signs of abdominal pain or need for pain relief, even though the extent of the oedema and herniation increased. The udder developed rapidly and 2 days following admission the mare began to run milk, although the milk electrolyte values had not yet changed significantly. A further 3 days later, the mare lost her appetite, became restless and showed signs of mild abdominal discomfort. Milk electrolyte evaluation revealed a marked elevation in the $[Ca^{2+}]$ to > 10 mmol/l. *Per rectum* examination revealed a flaccid uterus which suggested that labour was not yet in progress, however a *per vaginam* examination revealed that the cervix had begun to relax and the decision was made to induce parturition with a single i.v. injection of 20 iu oxytocin (Oxytocin)². Thirty minutes later the foal was delivered with surprisingly little need for veterinary assistance. The foal was alive and apparently mature but categorised as 'high risk' because of the mare's history and the induced parturition. Since the mare had lost colostrum over the course of 3 days and had not been vaccinated against influenza and tetanus, the foal was given supplementary frozen-thawed mare colostrum by stomach tube, treated with 6000 iu tetanus antitoxin (Tetanuserum)², and provided with antibiotic cover in the form of ceftiofur sodium (5 mg/kg bwt i.m. b.i.d; Excenel)³. Twelve hours *post partum* the foal's plasma IgG level was only 2 g/l and he was therefore transfused with 600 ml plasma from an unrelated donor.

Three hours after foaling, the mare had not delivered her placenta and was treated with 20 iu oxytocin i.m. to accelerate this process. The placenta had still not been expelled 5 h *post partum* and 50 iu oxytocin diluted in 1 l of 0.9% NaCl⁴ was infused slowly i.v. A *per vaginam* examination 1 h later revealed that the placenta was still firmly attached to the uterine wall, and at this point the placenta was removed by careful manual 'unzipping' of the utero-



Fig 3: Hoisting of the recumbent mare. It transpired that the mare was unable to support herself with her hindlimbs.

placental interdigitation, followed immediately by uterine lavage with 10 l tap water containing 90 g salt. The mare was also treated with 15,000 iu tetanus antitoxin i.m. and, since the risk of metritis was considered great, broad-spectrum antibiotic cover was initiated with trimethoprim and sulphadoxine (5 and 25 mg/kg bwt i.v. s.i.d.; Borgal)² and continued with trimethoprim and sulphadiazine (1 ml/20 kg bwt *per os* b.i.d.; Sultrisan oral paste)⁵ for 5 days. Uterine involution was further assisted by daily uterine lavage with salinated tap water and i.m. injections of oxytocin (20 iu), every 4 h on the first day and twice daily on the following 2 days.

After foaling, the subcutaneous ventral swellings were even larger and lordosis more pronounced. However, because the tailhead was not elevated and the udder had not been displaced cranio-ventrally, there was no clear evidence of prepubic tendon rupture. In other respects, the mare was clinically stable, not in obvious pain and was eating, drinking, nursing and caring for her foal. Although the long-term prognosis for the mare was poor, it was hoped to keep her alive and pain-free long enough to rear her foal. Two days later, however, the mare showed signs of acute abdominal pain, which was relieved sufficiently by a single i.v. injection of flunixin meglumine to allow the mare to regain her appetite. During the next 4 days, the mare remained stable without the need for further pain relief. On Day 7 *post partum*, however, the mare was found recumbent in her box apparently unable to stand and, although not distressed, appeared to have no deep-pain sensation in her distal hindlimbs. Attempts to help her to stand were unsuccessful, and she was dragged on a tarpaulin to an anaesthetic recovery-box where a harness was used to hoist her to her feet (**Fig 3**) in the hope that the lack of abdominal tension was the only factor rendering her unable to stand. Unfortunately, after hoisting it was clear that the mare was unable to support herself with her hind legs and she was, therefore, subjected to euthanasia with i.v. bolus injection of 100 mg sodium pentobarbitone (Euthesate)⁶.

Following euthanasia of his dam, the foal had to be hand-reared, but 3 months later was progressing well.

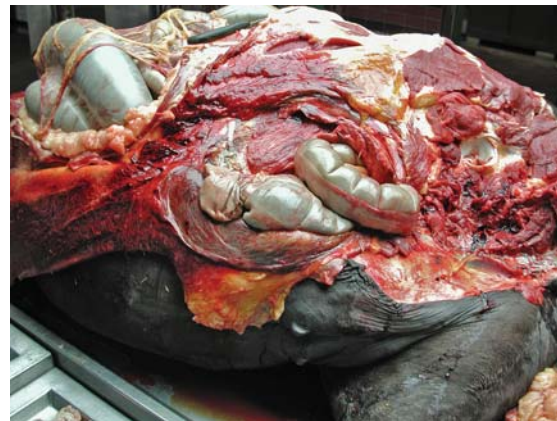


Fig 4: Small intestines herniated through the abdominal muscles to lie just beneath the skin on the left hand side of the abdomen.

Post mortem examination

An extensive tear was present in the abdominal oblique muscles on the left hand side, but the *transversus abdominus* was largely intact. On the right hand side, small intestine was found immediately beneath the *cutaneus trunci*, but was found to have migrated to this position via a tear in the aponeurosis of the *rectus abdominis*, just cranial to the pubis, and a 20 cm subcutaneous 'tunnel'. In fact, the *rectus abdominis* had torn from its attachment to the pelvis on both sides, and small intestinal loops had passed through both defects to lie under the udder and skin (**Fig 4**; the mare's hind end is on the right hand side of the picture and the left hand side of the abdominal cavity is displayed). Extensive subcutaneous bleeding and oedema were evident in the region of the torn aponeurosis but the prepubic tendon was intact. The peritoneal fluid was lightly haemorrhagic and there were fibrin strands on many intestinal loops, indicating a mild, reactive peritonitis.

Examination of the thoracic and lumbar regions of the vertebral column revealed that the spinous process of L1 had broken off and that the articulation between T13 and L1 had been distorted. In addition, there was subdural bleeding around the spinal cord at the T13–L1 level, and the corresponding intervertebral disc was torn. Finally, the iliosacral ligaments and articulation were very 'loose', although this was probably a normal peripartum change.

Discussion

Abdominal wall and prepubic tendon rupture are infrequent but life-threatening complications of late equine gestation. This was a typical case in that it affected an older, multiparous, draught-type mare and, while initial clinical onset was reported to be unusually sudden, subsequent abdominal enlargement exhibited typical progression (Adams 1979; Jackson 1982; Hanson and Todhunter 1986; Kawcak and Stashak 1995; Mirza *et al.* 1997). Since abdominal wall damage appeared to be extensive at presentation, it was estimated that the prognosis

for the mare was at best guarded. Furthermore, since the mare responded rapidly to pain-relief and showed no obvious signs of impending parturition, it was decided to manage the mare until there was clear evidence of 'readiness for birth' (e.g. milk $[Na^+] > [K^+]$ and elevated $[Ca^{2+}]$: Ousey *et al.* 1984; Ousey 2003) to maximise the foal's chances of survival. During this period, abdominal support was provided via a harness because such devices have been reported to protect the mare's back from damage caused by having to suspend the abdominal contents in the absence of ventral abdominal tension (Adams 1979; Hanson and Todhunter 1986; Arighi 1992; Kaneps 1992; Lofstedt 1993; Kawcak and Stashak 1995; Mirza *et al.* 1997; Wolfsdorf 2003).

In the present case, there was no overwhelming reason to attempt early induction and risk delivery of a nonviable foal. However, if abdominal pain is severe or abdominal damage appears to be minimal, and survival of the mare is the priority, immediate induction is often considered to prevent worsening of the abdominal damage; however, in our experience, this is often insufficient to salvage the mare's career as a broodmare. In the present case, foaling required surprisingly little assistance (Kaneps 1992; Lofstedt 1993; Wolfsdorf 2003) but, *post partum*, the abdominal damage appeared to be worse and the posture of the mare suggested that the support provided by the ventral abdominal wall had deteriorated further. Nevertheless, it was still a surprise when the mare's clinical condition deteriorated 4–5 days after foaling; *post partum* deterioration is not normally expected because the weight of the foal and fetal fluids have been removed (Kaneps 1992; Mirza *et al.* 1997; Wolfsdorf 2003). Abdominal discomfort at this time was possibly a result of temporary intestinal entrapment combined with inflammation of the torn muscles, since the pain was readily relieved by NSAID administration (Jackson 1982; Hanson and Todhunter 1986; Kawcak and Stashak 1995). Although less likely, it is also possible that the pain originated from the vertebral column, damage to which was the eventual reason for euthanasia. The unsupported weight of the abdominal viscera may have exerted sufficient stress on the vertebral column to avulse the spinal process of L1, tear the intervertebral disc between T13 and L1 and cause intradural haemorrhage and disruption of the spinal cord. However it is possible that, since the time that the mare was found recumbent was the first time that she had been seen to lie down since foaling, the vertebral column damage occurred during attempts to stand up.

In conclusion, this report describes *post partum* spinal cord trauma in a Friesian mare as a complication of abdominal wall rupture, and emphasises that the prognosis in cases of extensive muscular disruption is poor even when the mare can be managed to deliver a viable foal at term by a combination of exercise restriction, pain relief and careful monitoring and assistance of foaling.

Manufacturers' addresses

¹Schering-Plough Animal Health, Maarsse, The Netherlands.

²Intervet, Boxmeer, The Netherlands.

³Pfizer Animal Health, Capelle aan de IJssel, The Netherlands.

⁴Braun, Melsungen, Germany.

⁵Anisane Pet Health Products, Raamsdonksveer, The Netherlands.

⁶CEVA Sante Animale BV, Naaldwijk, The Netherlands.

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