Case Report

Bilateral degenerative suspensory desmitis with acute rupture in a Standardbred colt

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Summary

A 3-month-old Standardbred colt was examined for acute, bilateral hindlimb swelling and lameness. Serum chemistry demonstrated elevated muscle enzymes (AST, ALT, LDH and CK). Radiographs of the hindlimbs demonstrated intact proximal sesamoid bones that were displaced distally and subluxation of the pastern joints. Ultrasonography of the affected areas revealed large, diffuse hypoechoic areas in the bodies of both hind suspensory ligaments consistent with bilateral rupture. Histology of the lesions was consistent with degenerative suspensory desmitis with acute rupture.

Introduction

Suspensory ligament collapse can be secondary to either chronic suspensory ligament desmitis or due to acute rupture. Degenerative suspensory ligament desmitis is most common in aged horses, especially Paso Finos (Halper et al. 2006). Acute ruptures of the suspensory apparatus most commonly occur with fracture of the proximal sesamoid bones or due to complete avulsion of the distal sesamoidean ligaments. Acute breakdown injuries are most common in the forelimbs of racing Thoroughbreds. The least common cause of collapse is a complete tear of the suspensory ligament body or both branches (Dyson and Ross 2002). This case represents an unusual manifestation of degenerative suspensory desmitis with acute, bilateral rupture in a foal.

Case details

Case history and physical examination

A 3-month-old, Standardbred colt was examined for bilateral hindlimb swelling and lameness. The colt was reported to have had normal conformation since birth. Three days prior to presentation at the hospital, the colt was noted to be lying down for an extended period of time in a turn-out pasture. At that time, bilateral hindlimb swelling was visible from the caudal aspect of the tarsometatarsal joint extending distally to below the metatarsophalangeal joint. The colt was able to ambulate, although hyperextension of both metatarsophalangeal joints was noted (Fig 1). No other abnormalities were noted during physical examination.

Fig 1: Rear quarters of a 3-month-old Standardbred colt. Notice hyperextension of metatarsophalangeal joints as well as straight hock conformation.
Clinicopathological findings

A complete blood count was within normal limits. Abnormalities in the serum chemistry panel included elevated aspartate aminotranserase (AST; 846 u/l; reference range [rr] 174–480 u/l), increased alanine aminotransferase (ALT; 75 u/l; rr 3–20 u/l), increased lactate dehydrogenase (LDH; 1032 u/l; rr 150–370 u/l) and increased creatinine kinase (CK; 1653 u/l; rr 10–350 u/l).

Radiographs

Lateral radiographs of the metatarsophalangeal joints of both hindlimbs were taken (Fig 2). The proximal sesamoid bones were displaced distally. The sesamoid bones were intact and no avulsion fractures were visible. Subluxation of the proximal interphalangeal joint was noted in both hindlimbs.

Ultrasound examination

A 12 mHz linear array transducer was used to examine the flexor tendons and suspensory ligaments of both hindlimbs. Imaging of the superficial and deep digital flexor tendons on both hindlimbs was unremarkable. Transverse images of the suspensory ligament body indicated marked enlargement, with a cross-sectional area of 6.7 cm². This area was diffusely hypoechoic with loss of definition of the majority of the margins of the ligaments (Fig 3).

Outcome

The owner elected euthanasia due to the grave prognosis for sound athletic function. The colt was subjected to euthanasia 7 days after presentation to the clinic. A post mortem examination was performed at this time.

Post mortem findings

Radiographic and ultrasonographic findings were confirmed at necropsy. The proximal sesamoid bones in both hindlimbs were found to be intact. The straight, oblique and cruciate sesamoidean ligaments were found to be intact in both hindlimbs. The site of the lesion in the left hindlimb was noticeably larger than the lesion in the right hindlimb (Fig 4). Haemorrhage was present centrally within the ligament several cm proximal to the site of the rupture (Fig 5). Histology from the site of the lesions demonstrated multifocal, coalescing extensive fibrovascular proliferation as well as disruption of cell architecture with haemorrhage within the collagen fascicles. Inflammation was minimal with mild, multifocal perivascular and interstitial plasma cell accumulation. A sample taken distal to the lesion in the right hindlimb displayed fibrovascular proliferation that partially dissected the ligament into irregular compartments. Histology of the suspensory ligament proximal to both lesions indicated mild fibrovascular proliferation with minimal inflammation. At a site proximal to the left lesion multifocal...
myofibre degeneration was noted. Proteoglycan accumulation was not noted on haematoxylin and eosin stain (Figs 6 and 7).

Discussion

In this case, histology was consistent with the diagnosis of degenerative suspensory desmitis with acute rupture. Degenerative suspensory desmitis is a condition that leads to chronic and incurable lameness, recognised in specific equine breeds including Paso Fino, Peruvian Paso, Arabians, American Saddlebreds, Thoroughbreds and American Quarter Horses (Mero and Pool 2002; Haiper et al. 2006). One publication suggests that this is a systemic condition characterised by generalised proteoglycan accumulation in connective tissues including the
suspensory ligaments, aorta and pulmonary arteries, as opposed to a primary defect in collagen fibres (Halper et al. 2006). This conclusion was not verified in a more recent study (Schenkman et al. 2009). Horses with degenerative suspensory desmitis are thought to have a healing response that is abnormal (Gibson and Steel 2002). This abnormal healing mechanism is seen as degeneration within the ligament, followed by coalescing collagen bundles that eventually become large bundles isolated from their blood supply (Mero and Pool 2002; Halper et al. 2006). A grossly normal tissue sample taken proximal to one of the lesions in this foal had multifocal coalescing fibrovascular proliferation that partially dissected the ligament into irregular compartments - evidence of the abnormal healing process. Histopathology of the lesions in this case demonstrated coalescing and focally extensive fibrovascular proliferation with minimal inflammation and accumulation of proteoglycan was not noted in any of the samples analysed in this case. This histopathology is consistent with early to intermediate stages of degenerative suspensory ligament desmitis described by Mero and Pool (2002).

Acute rupture of the suspensory apparatus is an injury most commonly seen in the forelimbs of racing Thoroughbreds (Dyson and Ross 2002; McIlwraith 2002). This injury is most commonly associated with fractures of the proximal sesamoid bones. Collapse of the suspensory apparatus happens most commonly in the forelimbs of all breeds except Standardbreds, where it can occur in the forelimbs or hindlimbs. Failure of the suspensory apparatus can also be seen in foals (Honnas et al. 1990). The injury most commonly occurs in either neonates or foals who are allowed access to a large pasture after a prolonged period of stall confinement (Dyson and Ross 2002; Knottenbelt et al. 2004). These injuries are thought to be due to joint laxity and the resulting strain on the ligamentous structures of the limb resulting in fracture of the proximal sesamoid bones or rupture of the distal sesamoidean ligaments. Radiographs are often diagnostic for either circumstance with fractures being visible on radiographs or proximal displacement of the proximal sesamoid bones due to avulsion or rupture of the distal sesamoidean ligaments. One case report describes a foal with bilateral forelimb suspensory apparatus collapse where the distal sesamoidean ligaments ruptured (Harrison and May 1992). That foal was noted to have severe tendon laxity since birth that probably led to abnormal strain on soft tissue structures supporting the fetlock. The foal in our case was conformationally normal since birth, and hand walked to a pasture for turnout daily and had never been confined to a stall for an extended period. Foals of heavier breeds, such as Shires have also been noted to suffer from collapse of the suspensory apparatus either due to fracture of the proximal sesamoid bones or rupture of the suspensory body. This probably happens due to concurrent tendon laxity and/or the large stature of this breed causing excessive strain on the ligament (Knottenbelt et al. 2004).

The composition of the suspensory ligament may explain the increased muscle enzymes seen on the biochemistry panel in this foal. The equine suspensory ligament contains of varying amounts of muscle fibres. Standardbreds have 40% more muscle than Thoroughbreds, with more muscle fibres present in the hind- than the forelimbs (Wilson et al. 1991). Skeletal muscle fibres as well as myofibre degeneration and regeneration were visualised on histology of samples taken from this foal. No histology samples were taken from the large muscles of the hindlimbs. Since the foal was found down in the field, it is likely that some of the increased muscle enzymes could be due to trauma of large hindlimb muscles. The changes seen on histology of the affected ligament could partially explain the increased muscle enzyme values.

Degenerative suspensory desmitis is most common in older horses and although acute ruptures of the suspensory ligament can occur in foals, this case is a unique manifestation of degenerative suspensory ligament desmitis combined with acute rupture. To the best of our knowledge this has not previously been reported in a foal of this age.

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References


