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PROXIMAL SUSPENSO TORY DESMITIS IN THE HINDLIMB

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Summary

Proximal suspensory desmitis is a common cause of hindlimb lameness or poor performance. Accurate diagnosis requires careful interpretation of local analgesic techniques and high quality ultrasonographic images. Successful management is challenging.

Anatomy

The suspensory ligament (SL) (third interosseous muscle) can be divided into 3 separate regions which are subject to injury, the proximal part, the body and the branches. For clinical purposes the proximal part extends from approximately 2 to 10 cm distal to the tarsometatarsal (TMT) joint. In the forelimb the SL originates from 2 heads which rapidly fuse. In the hindlimb this division is less obvious. The SL contains a variable amount of muscular tissue (2 - 11%), which tends to be bilaterally symmetrical. In the hindlimb the SL originates principally from the proximoplantar aspect of the third metatarsal bone (Mt III). The SL in the forelimb is approximately rectangular in cross-section, but is more rounded in the hindlimb. The body of the SL descends between the second (Mt II) and fourth (Mt IV) metatarsal bones and divides into 2 branches at a variable site in the mid metatarsal region. The level of division is usually bilaterally symmetrical. Each branch inserts on the abaxial surface of the corresponding proximal sesamoid bone (PSB). Each branch detaches a thin extensor branch dorsodistally, that courses obliquely across the pastern to join the dorsal digital extensor tendon just above the proximal interphalangeal (PIP) joint. Each extensor branch also blends with the corresponding collateral sesamoidean ligament. Proximally there is a distinct fascial band in close apposition to the plantar aspect of the SL, with horizontally orientated fibres extending between Mt II and Mt IV.

The hindlimb SL is innervated by the plantar metatarsal nerves, branches from the deep branch of the lateral plantar nerve, which is derived from the tibial nerve. The proximal SL is closely related to the plantar outpouching of the TMT joint capsule.

The principal function of the SL is to prevent excessive extension of the fetlock joint. During weight bearing the relative tension in the SL and flexor tendons regulates the stresses applied to different aspects of the Mt III. When a limb is fully load bearing the distal part of the SL branches are closely apposed to the abaxial aspects of the metatarsal condyles and then move to the plantar aspect as the fetlock drops. During hyperextension, the PSBs move distally and dorsally, so the branches of the SL act as articular surfaces to balance the position of the Mt III. If the limb is loaded asymmetrically, so there is torque on the fetlock, the SL branches contribute to joint stability on the side opposite compression of the joint.

In the forelimb there is some evidence that training increases the strength of the SL; the mean absolute load to failure in a single load to failure compression test was significantly higher in horses that had been in racehorse training compared to those that had been confined to box or paddock rest (Bramlage et al. 1989). In the trained group failure was most likely to be by fracture of a PSB, whereas in the untrained group, the SL failed. However when six 2 year old Thoroughbred fillies underwent an 18 month controlled exercise programme including galloping, and were compared to 6 fillies which were restricted to walking exercise, there were no differences in the collagen fibril mass-average diameter (MAD) in the body of the SL (Patterson-Kane et al.1998). MAD is correlated with ligament strength. Similar studies have not been conducted in hindlimbs.

Proximal suspensory desmitis in the hindlimb

The diagnosis of proximal suspensory desmitis (PSD) in the hindlimb has increased in recent years unquestionably due to improved recognition, but possibly also due to increased frequency of occurrence. This may relate to a change in both training methods and training surfaces and the increasing athletic demands placed on upper level competition horses. It results in either an insidious onset, or sudden onset lameness, which may be mild or severe, either unilateral or bilateral. Some horses present with poor performance, rather than a recognised lameness. Complaints include loss of hindlimb...
impulsion; unwillingness to go forwards freely; stiffness; resistant behaviour; lack of power when jumping; refusing jumps uncharacteristically; difficulties in performing specific dressage movements e.g., canter pirouette; poor performance at high speed in racehorses; evasive behaviour such as bolting. In contrast to PSD in the forelimb, lameness may persist and remain severe, despite restriction to box rest. This is probably due to a compartment-like syndrome and pressure on the adjacent plantar metatarsal nerves (Dyson 1995b). In view of the chronicity of some lesions when first identified, and the finding of secondary radiological changes in sound horses, it is likely that some lesions exist sub-clinically, or are associated with a low grade lameness that goes unrecognised. The prevalence of bilateral lesions is higher than in forelimbs.

PSD in the hindlimb occurs in horses in all athletic disciplines and of all ages. It is a particular problem in dressage horses working at advanced level. Horses with either straight hock conformation, and or hyperextension of the metatarsophalangeal (MTP) joint, appear predisposed to injury. Such conformational abnormalities were identified in 9 of 42 horses (21%) with hindlimb PSD, but in only 4 of 50 (8%) horses examined consecutively with hindlimb lameness unrelated to the suspensory apparatus (Dyson 1994, 1995a). Hyperextension of the MTP joint may develop as a sequel to PSD. A long toe and low heel conformation may also be a predisposing factor, especially if associated with abnormal orientation of the distal phalanx, with the plantar aspect lower than the toe (Dyson and Genovese 2003).

**Clinical features**

In horses with acute hindlimb PSD there may be localised heat and swelling and pain on pressure applied to the SL, but frequently there are no localising clinical features. At the walk there may be reduced extension of the MTP joint of the lame limb, unless the integrity of the SL is severely compromised resulting in hyperextension of the joint. Lameness is often characterised by a reduced height of arc of foot flight, with or without intermittent catching of the toe. The cranial phase of the stride may be shortened. Lameness may be accentuated by either proximal or distal limb flexion. Bilateral lesions may result in poor hindlimb action, with poor hindlimb impulsion and engagement, rather than obvious hindlimb lameness, and may compromise the movement of the entire horse. Lameness may be more obvious on a circle on the lunge, but unlike forelimb PSD, the lameness is not necessarily worse with the lame or lamer limb on the outside. Like many hindlimb lamenesses, lameness is usually more obvious when the horse is ridden, especially when the rider sits on the diagonal of the lame or lamer limb, and in some horses gait abnormalities are only evident when ridden.

**Local analgesic techniques**

It is important to recognise that perineural analgesia of the plantar nerves (at the junction of the proximal 2/3 and distal 1/3 of the metatarsal region) and plantar metatarsal nerves may result in slight improvement in lameness, due to proximal diffusion of the local anaesthetic solution, and if a ‘4 point’ or ‘6 point’ block is done more proximally the risk of false positive results increases. Lameness is usually substantially improved by perineural analgesia of either the deep branch of the lateral plantar nerve distal to the tarsus (3 - 5 ml mepivacaine 2%), or by local infiltration axial to the second and fourth metatarsal bones, but may not be alleviated fully. Improvement is usually seen within 10 minutes of injection. In some horses it may be difficult to deposit the local anaesthetic solution as proximal as ideal, due to the shape of the base of Mt IV. This may result in only partial improvement in lameness. False negative results may also be obtained due to inadvertent injection into either the tarsal sheath, or the TMT joint capsule (Dyson and Romero 1993). In a horse presenting with poor hindlimb impulsion it might theoretically be expected that if pain was alleviated from one limb the horse should then show overt lameness on the other. Although this sometimes happens, it does not always and this can result in false negative results. In such horses a much more dramatic improvement in gait may be seen if perineural analgesia of the deep branch of the lateral plantar nerve is performed bilaterally. This technique should theoretically only remove ligamentous pain and not osseous pain at the ligament’s origin. Osseous pain is more likely to be abolished by directing the needle in a dorsal direction and infiltrating as deeply as possible. However, diffusion of local anaesthetic solution from the site for perineural analgesia of the deep branch of the lateral planar nerve may produce some confusing results and nuclear scintigraphy may be a more reliable means of establishing whether there is active concurrent bony pathology at the SL’s origin.
Sub-tarsal analgesia can influence TMT joint pain, and occasionally (2/24 horses, 8%; Dyson 1994) intra-articular analgesia of the TMT joint alleviates pain associated with PSD. If a horse which had lameness abolished by intra-articular analgesia of the TMT joint, but with only minor radiological change of the TMT and centrodistal joints, fails to respond adequately to treatment consideration should be given to the presence of PSD. Therefore a comparison of the responses to intra-articular analgesia and subtarsal analgesia can be useful. Perineural analgesia of the tibial nerve alone alleviates pain associated with PSD, without significantly influencing tarsal pain. However this is a larger nerve, therefore it may take 20 minutes before analgesia is effectively achieved. This is an extremely useful block to perform to differentiate between distal hock joint pain and PSD, especially in horses in which false negative results to subtarsal analgesia have been obtained. Occasionally PSD occurs together with pain associated with osteoarthritis of the TMT joint.

**Diagnostic ultrasonography**

High quality ultrasonographic images, in both transverse and sagittal planes, are essential for accurate diagnosis. Transverse images are best acquired from the plantarolateral aspect of the metatarsal region (Dyson 1998). Large vessels plantarolateral to the SL may result in broad linear anechogenic artefacts within the SL. Air artefacts after local analgesia may also be a problem. In large Warmblood horses in particular, the SL is situated deeply and the ultrasound transducer must be focused accordingly so that a clear image of the plantar cortex of Mt III is obtained. In both transverse and longitudinal images the most proximal part of the SL in a normal horse may appear slightly less echogenic than the DDFT. Detection of subtle abnormalities requires careful comparison with the contralateral limb and measurement of cross-sectional area.

In hindlimb PSD focal anechogenic areas are relatively unusual, except in the Standardbred racehorse. More commonly there is enlargement of the SL, with poor demarcation of its borders, especially the dorsal border and a diffuse reduction in echogenicity of part, or all, of the cross sectional area of the ligament. Ectopic fibrosis or mineralisation occurs more often in hindlimbs compared with forelimbs. Lesions are easily missed unless the most proximal aspect of the SL is examined. Familiarity with the normal ultrasonographic appearance is crucial for recognition of mild lesions. An irregular contour of the plantar aspect of the Mt III may reflect entheseophyte formation. In the author’s experience, in the vast majority of horses with a positive response to subtarsal analgesia, ultrasonographic abnormalities are detectable, unless it is a very acute lesion (days), or fibrosis is the predominant change in a chronic injury, or in the less lame limb of a bilaterally lame horse.

In some horses, especially those with abnormal conformation, the lesions may progress despite box rest.

**Radiography**

Diagnosis should never be based on radiography alone, since some sound horse have some sclerosis of the proximal aspect of the Mt III. In horses with chronic active PSD this may be more extensive. In the dorsoplantar view there is increased opacity of the proximal aspect of the Mt III, often more obvious laterally. In a lateromedial projection there may be sub-cortical sclerosis and alteration of the trabecular pattern of the proximoplantar aspect of the MT III due to endosteal new bone, extending up to 4cm proximodistally. The plantar cortex may itself be thickened and in addition there may be entheseophyte formation on the plantar aspect. However in many horses no radiological abnormality is detectable.

**Nuclear scintigraphy**

Recognition of normal patterns of radiopharmaceutical uptake (RU) is crucial for accurate image interpretation. In normal horses, it is normal in plantar images to see relatively greater RU in the proximoplantar aspect of Mt III compared with medially or dorsally (Murray et al. 2005; Weekes et al. 2005). Nuclear scintigraphy is not a sensitive means of detecting PSD in hindlimbs. Pool phase images were positive in only 25% of 20 horses with ultrasonographic evidence of PSD (Dyson and Genovese 2003). In bone phase images there was increased RU (IRU) in the proximoplantar aspect of the Mt III in 42%. However, in a more recent, much larger study of more than 200 horses the frequency of IRU in the proximoplantar aspect of Mt III associated with PSD was considerably less (20%) (unpublished data). IRU associated with PSD should be differentiated from those horses with primary bony pathology, with no detectable
ultrasonographic abnormality of the SL and no radiographic change associated with enthesisopathy.

**Magnetic resonance imaging**

The interpretation of magnetic resonance (MR) images is complicated by the high signal intensity of muscle within the ligamentous structure. In the author's experience MR imaging is more useful for explaining concurrent bony pathology in horses in which the degree of ultrasonographic abnormality is not commensurate with the degree of lameness. In most of these horses there is IRU in the region of origin of the SL or slightly distal to this. This has been associated with endosteal mineralisation and fluid accumulation in T1 and T2 weighted MR images and increased signal intensity in fat suppressed images.

**Differential diagnosis**

PSD should be differentiated from pain associated with the TMT joint, an avulsion fracture of the Mt III at the origin of the SL, and primary stress reactions in the Mt III.

**Treatment**

Treatment depends on time constraints, athletic expectations, rules for medication control, degree of lameness and architectural disruption of the SL, conformation, chronicity of the lesion, age of the horse and the number of limbs affected. The prognosis for PSD in the hindlimb has generally been poor. Only 6/42 horses (14%) seen in a referral practice were able to resume full work without detectable lameness for at least 1 year, all of which had been lame for less than 5 weeks (Dyson 1994). All these horses showed marked improvement in clinical signs within 3 months of the onset of lameness. Two additional horses resumed full work, but suffered lameness in another limb. Seven horses improved markedly and were able to work, despite persistent mild lameness. Twenty-four horses (57%) had persistent or recurrent lameness. Results from a first opinion practice were also disappointing with only 10 of 17 horses (58%) resuming work (Dyson 1998).

Horses with acute (less than 4 – 6 weeks duration) hindlimb PSD respond reasonably well to local infiltration with corticosteroids, aimed to reduce inflammation and therefore swelling and thus minimise the risk of the development of a compartment syndrome (see below). Foot imbalance is corrected and egg bar shoes are used to reduce extension of the fetlock. Historically, horses with chronic PSD have had a very guarded prognosis regardless of the treatment. Lameness often tended to persist unchanged even following prolonged box rest, which is unusual for a primary soft tissue lesion. In some cases lesions are progressive. Local infiltration with corticosteroids, polysulphated glycosaminoglycan, hyaluronan or homeopathic drugs, such as actovegin and traumil has given disappointing results.

In some horses an initial improvement in lameness is seen after box rest and controlled walking exercise for 2 to 3 months and then no further improvement is seen. Increasing the exercise despite the lameness, sometimes results in further improvement. Some horses have worked satisfactorily while being treated with phenylbutazone, without apparent deterioration of clinical signs.

Extracorporeal shock wave or radial pressure wave therapy appears to be helpful in some cases (Boening et al. 2000; Crowe et al. 2004). Forty-one percent of 43 horses with lameness of more than 3 months duration associated with hindlimb PSD were sound in work 6 months after treatment using radial pressure wave therapy (Crowe et al. 2004). Response was not related to duration of lameness, but outcome was inversely related to lesion severity. Injection of 2% iodine in almond oil resulted in 12 of 22 (54%) horses returning to work (Soule. S.; personal communication 2000). Local infiltration with A cell has resulted in 31 of 38 (82%) of horses with either forelimb or hindlimb PSD returning to full work (Mitchell, R., 2005 personal communication).

Tibial neurectomy performed in 8 horses enabled 6 to return to full athletic function (show jumping and horse trials) for at least 2 years after surgery, with no post operative complications (Dyson and Genovese 2003). Neurectomy of the deep branch of the lateral plantar nerve has been combined with incising the thin plantar fascial and was successful in 80% of more than 100 horses (Bathe, A., personal communication 2005). This is now my treatment of choice (provided that there is no bone pathology involved) and 37 of 53 horses (70%) have returned to full athletic function. Horses have been restricted to box rest for 10 - 14 days post operatively and have then been walked for 45 minutes (in hand, ridden,
or on a horse walker) for 6 weeks, prior to clinical and ultrasonographic reassessment. If horses were sound at this stage full work has been progressively resumed. However, in some horses with very straight hindlimb conformation PSD has been progressive following surgery, and in my opinion these horses are poor surgical candidates. Fasciotomy of the deep plantar metatarsal fascia alone has been successful in some cases (Ross, M. personal communication). Injection of approximately 30 ml bone marrow is also claimed to be successful, especially if combined with fasciotomy, allowing 87% of horses to return to their former level of function (Herthel 2003), however it is not clear from the report what proportion of these horses had hindlimb or forelimb injuries. Nor is it clear what the duration or severity of lameness was.

**Gross pathology and histopathology**

Post mortem examinations have been performed on both hindlimbs of 14 horses, 8 with unilateral lameness and 6 with bilateral lameness (Dyson 1995, 2003). Abnormalities of the SLs were confined to the lame limbs. There was gross enlargement of the SLs, with thickening of surrounding fascia and periligamentous tissues, especially on the plantar aspect. Histological changes in the SL included hypercellularity and acellular areas, haemosiderin deposition, fibrosis, hyalinisation of collagen, an increased number of fibrous septae, some with blood vessels, neovascularisation and chondroid metaplasia. Although chondroid metaplasia was seen at the ligament bone interface in both lame and sound limbs, intra-ligamentous chondroid metaplasia was only seen in the lame limbs. There was evidence of compression of adjacent peripheral nerves in the lame limb of 12 horses. Abnormalities of the plantar metatarsal nerves included thickening of the perineurium, perineural fibrosis, reduction or absence of nerve fibres and Renaut bodies. These changes support the theory of PSD in the hindlimb resulting in a compartment syndrome.

**Concurrent injuries**

Hindlimb PSD may also be accompanied by injury of the lateral or, less commonly, the medial branch of the SL. This may be unapparent at the time of initial clinical examination, unless there is gross swelling. However, ultrasonographic examination may reveal evidence of damage. Several horses had been successfully treated surgically only to incur recurrent lameness due to desmitis of the lateral or, less commonly, the medial branch of the SL that was not recognised at the time of initial examination (unpublished data). One horse with acute PSD was managed successfully conservatively and 1 year later had recurrent lameness due to desmitis of the proximal aspect of the lateral oblique sesamoidean ligament. Chronic PSD has also been seen in association with sacroiliac joint region pain.

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