TREATMENT OF HINDLIMB PROXIMAL SUSPENSORY DESMITIS IN HORSES

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considers diagnostic methods for this ligament injury prevalent in performance horses, and treatment choices, including surgery

ABSTRACT
Proximal suspensory desmitis (PSD) is usually diagnosed using local analgesic techniques in combination with ultrasonography and radiography. Scintigraphic examination is useful for assessing osseous pathology. MRI may be required in some cases. Hindlimb PSD is a difficult condition to manage, and is associated with a poor prognosis with rest alone. Intervention, such as surgery, is often therefore warranted.

Keywords: horse, suspensory ligament, desmitis, neurectomy and fasciotomy

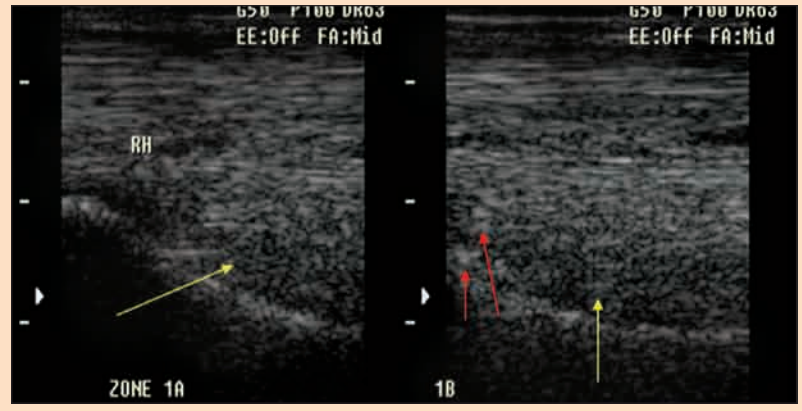

Figure 3. Longitudinal ultrasonographic image of the right proximal metatarsal region of a nine-year-old, working hunter pony at Zones 1A and 1A/1B. Proximal is to the left. Note loss of long fibre pattern in the dorsal aspect of the suspensory ligament (yellow arrows). There are also hyperechogenic foci (red arrows), most likely indicating chronicity.

PROXIMAL suspensory desmitis (PSD) is a well recognised cause of lameness[1,2].

The proximal aspect of the hindlimb suspensory ligament (SL) is defined as the region 2cm to 10cm distal to the tarsometatarsal joint (TMTJ)[2]. In the hindlimb, the SL originates from the proximoplantar aspect of the third metatarsal bone (MTIII); there is a proximally extending band originating on the plantar aspect of the fourth tarsal bone.

The function of the SL is to prevent overextension of the metatarsophalangeal joint[3]. Innervation is via the plantar metatarsal nerves – branches of the deep branch of the lateral plantar nerve (DBLPN), which is a branch of the lateral plantar nerve, derived from the tibial nerve.

Horses present with lameness or poor performance (unwilling to go forward, reduced hindlimb impulsion, evasive behaviour, reduced power when jumping, refusing fences). PSD is a common condition in all types of horses[1,4]; dressage and jumping horses are particularly prone[5,6]. Pain causing lameness may originate from the SL itself or be associated with compression of the adjacent nerves[7].

Clinical examination

There are frequently no localising clinical signs[4], reflecting the often chronic nature of the injury. It is not possible to palpate the proximal suspensory ligament (PSL) because of the position between the second and fourth metatarsal bones. In acute injury, distension of the medial plantar vein, localised pain[8] and oedema are sometimes found.

PSD is often accompanied by mediolateral foot imbalance[9,10]. Hindlimb PSD may result in secondary back pain, including development of pain from pre-existing dorsal spinous processes[4].

Horses with metatarsophalangeal hyperextension or straight hock conformation (**Figure 1**) are predisposed[1,11], as are those with long toes and low heels[2]. Metatarsophalangeal hyperextension is found in breeds such as the Peruvian paso[12,13] and in old brood mares[14], which may indicate age-related degeneration.

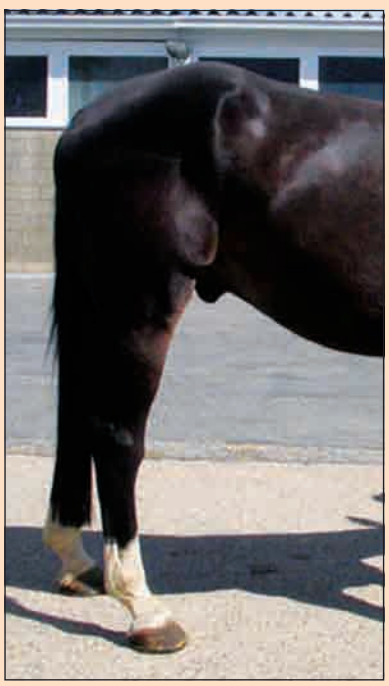

Figure 1. The hindlimbs of a four-year-old warmblood potential event horse. Note marked straight hock conformation. This horse had bilateral hindlimb proximal suspensory desmitis. This is an example of a poor candidate for surgical intervention with a plantar neurectomy and fasciotomy.

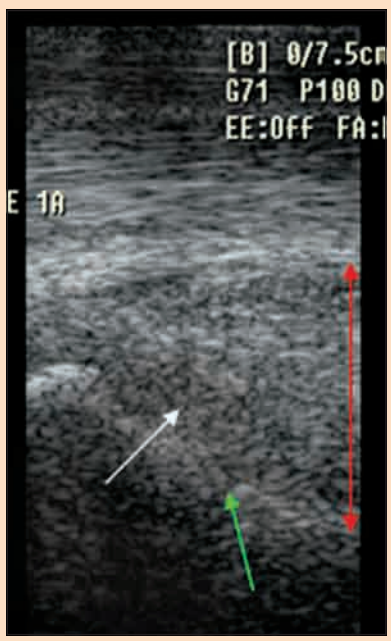

Figure 2. Longitudinal ultrasonographic image of the right proximal metatarsal region of a five-year-old, warmblood, dressage horse. Proximal is to the left. There is a convex contour of the plantar aspect of the SL reflecting swelling (red arrows), loss of long fibre pattern (white arrow) and mildly irregular bone (green arrow) at the origin of the suspensory ligament.

A positive response to both distal and proximal limb flexion is not unusual[1,4]. Many horses present with lameness most evident when the affected limb is on the outside of the circle on a soft surface. However, there is no pathognomonic gait abnormality[10] or pattern in lameness for hindlimb PSD[4].

Analgesia

Ultrasonographic and radiographic changes are sometimes subtle, resulting in heavy reliance on diagnostic analgesia for diagnosis. Pain resulting from PSD can be partially improved with "low-4(6)-point" analgesia[4] probably due to proximal diffusion of local anaesthetic solution.

Proximal suspensory ligament desensitisation can be achieved by analgesia of the proximal medial and lateral metatarsal nerves[15], tibial nerve[8], DBLPN using a nerve block (DBLPnb)[16], and local infiltration[15]. The author uses the DBLPnb due to its simplicity and option of neurectomy if response is marked. There is also a reduced chance of inadvertent entry into the TMTJ and tarsal sheath[16].

If the response is unclear, it is useful to perform tibial nerve analgesia, which alleviates PSL pain without significantly influencing tarsal pain[4].

There has been evidence of DBLPnb causing desensitisation of the lateral heel bulb to the distal aspect of the fourth metatarsal bone on the plantarolateral aspect of the limb, indicating analgesia of the lateral plantar and plantar metatarsal nerves[16]. It is, therefore, advisable that analgesia of the distal aspect of the limb be undertaken first.

When there is a significant response (greater than or equal to 75 per cent) to DBLPnb, the author undertakes TMTJ analgesia on a separate occasion due to the possible inadvertent entrance of the TMTJ plantar pouch[17], and results compared. If there is a positive but less than 75 per cent improvement following DBLPnb, then TMTJ analgesia and/or infiltration of the proximoplantar MTIII region can be added to assess any coexistence of distal tarsal or proximal MTIII entheseous pain and PSD respectively.

Bilateral DBLPnb can be undertaken in horses with poor hindlimb impulsion. Analgesia of one limb in these cases may not result in visible lameness in the contralateral limb, as would be expected[4]; yet bilateral analgesia can result in marked ridden improvement.

Diagnostic imaging

– Ultrasonography

For ultrasonography of the PSL, the limb must be approached from the plantaromedial aspect[18]. The plantar aspect of MTIII must be visualised to ensure SL changes are not artefactual. The size of this "window" can be narrow, affecting image quality. Due to the depth of the ligament and position between the second and fourth metatarsal bones, the medial and lateral margins are not always appreciable. Use of a convex-array transducer, "virtual convex" application or "stand-off" pad may alleviate these problems. Pathology seen on ultrasonography may include enlargement (**Figure 2**), poor margin definition, loss of fibre pattern (**Figure 3**), areas of hypoechogenicity, both centrally (**Figure 4**) and peripherally (**Figure 5**), hyperechogenic foci (**Figure 3**), short fibre pattern on longitudinal images (**Figures 2** and **3**) and MTIII plantar cortex irregularity (**Figure 2**).

Analgesic techniques can cause air artefacts. Because of this it is advisable to leave 24 hours between analgesia and ultrasonography. Presence of muscular tissue and shadowing artefacts[10] resulting from fluid-filled structures (such as blood vessels) and round structures (such as overlying flexor tendons[19]) may also complicate interpretation. Longitudinal images highlight short fibre pattern and may be more sensitive than radiography at detecting plantar MTIII pathology[20].

Increased proximal cross sectional area (greater than 1.5cm^2) of the PSL has been recorded[21], but obliquity causes inaccuracy in measurements. With unilateral lameness it is useful to compare with the contralateral limb. A study comparing SL measurements (cross sectional area, width and thickness) using ultrasonography, MRI and histology found ultrasonography had poor accuracy for cross sectional area measurement compared to MRI and histology[22,23]. Zauscher et al[24] has also found poor intra and interobserver agreement for measurement of PSL width, cross-sectional area and circumference. Reduction in space between the SL and the plantar MTIII cortex may be useful subjectively to represent SL enlargement[4] (**Figure 5**).

– Radiography

Radiography may reveal endosteal new bone on the dorsal aspect of the plantar cortex of MTIII (**Figures 6** and **7**) or entheseous new bone on the proximal aspect of MTIII. This may indicate chronicity, possibly with subclinical injury prior to detectable lameness/poor performance[8]. It must be noted, however, that increased radiopacity of the proximal aspect of MTIII (**Figure 8**) can be present in sound horses[4,25] and so should be interpreted with care. Radiology may underestimate the presence of new bone, which may be more reliably detected using computed tomograph[26] or MRI[27,28].

– Scintigraphy

Scintigraphy may determine if there is active pathology at the origin of the SL, which is not evident radiographically[20]. This is especially important if ultrasonographic changes do not fit the degree of lameness. Analgesia of the DBLPN may result in diffusion of local anaesthetic solution to the proximal aspect of MTIII. In the absence of ultrasonographic abnormality, scintigraphic examination may help to define active osseous lesions as the principal cause of pain, altering prognosis and treatment. Lack of increased radiopharmaceutical uptake (IRU) is a common finding with PSD unless there is an avulsion injury[29]. In a study of 126 horses, only 12 per cent

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with hindlimb PSD had IRU identified subjectively in bone phase images[20].

– Magnetic resonance imaging

If there is a positive response to analgesia, but ultrasonography is negative or equivocal, MRI should be undertaken of the distal hock and proximal metatarsal regions[27,28,30]. Some horses with osseous injury at the origin of the suspensory ligament where lameness is abolished following DBLPnb, have only been noted on MRI[27].

Treatment

Horses with hindlimb PSD respond poorly to rest alone[1,31], although horses with loss of support to the metatarsophalangeal joint need long-term rest regardless of therapy[6]. Only 6/42 (14 per cent) of horses were able to resume work without lameness for a year, all of which had presented with lameness less than five weeks in duration[1].

The PSL and distal DBLPN are confined between the second, third and fourth metatarsal bones and the deep laminar plantar metatarsal fascia. PSD is theorised to result in compartment syndrome and neural compression in some horses[7,11,32]. Abnormal innervation can follow SL injury, resulting in chronic pain[31].

Treatments for PSD include radial pressure wave therapy. Forty-one per cent of 43 horses with hindlimb PSD and lameness greater than or equal to three months' duration returned to full work six months after this treatment[33]. Focused shockwave therapy has achieved similar results[34,35]. Desmoplasty and fasciotomy for core injuries resulted in 87 per cent of 23 horses resuming work[36]. Bioscaffold therapy (A-Cell) in combination with fasciotomy resulted in 84 per cent of 77 horses with fore or hindlimb PSD returning to full work[37]. The two latter studies had a poorly defined follow-up period. Stem cell therapy[38] and osteostixis undertaken when there is concurrent osseous pathology of the proximoplantar aspect of MTIII[39,40] has also been used. Stem cell therapy (and bioscaffold treatment) may be an advantageous treatment for return of SL structure, but may not alleviate pain and lameness as the result of neural compression and compartment syndrome[40]. Such treatments may be useful in conjunction with neurectomy and fasciotomy to improve ultrasonographic fibre pattern, which is often not achieved following this surgery. Use of bisphosphonates has also been reported as being useful in horses with enthesis-related pain[41]. Tibial neurectomy has been used as a treatment for PSD with 6/8 horses (75 per cent) returning to full athletic function for at least two years post-surgery[2]. Neurectomy of the DBLPN and fasciotomy in combination[7,31,42] has resulted in success rates between 62 per cent and 91 per cent, with good long-term follow-up (greater than one year postoperatively[43]) and no loss of sensation or proprioceptive deficits[44].

In most cases, the author recommends treatment of PSD with a neurectomy and fasciotomy if there is a diagnosis of primary PSD causing pain and lameness, significant response to DBLPnb (greater than or equal to 75 per cent) and SL enlargement probably leading to compartment syndrome and neural compression.

Lameness and response to analgesic techniques is sometimes only seen unilaterally. Some horses become lame on the contralateral limb after treatment such as surgery when strenuous exercise resumes[7].

A histopathology study of the DBLPNs of 16 PSD cases that underwent neurectomy found myxomatous expansion with Renault bodies in the subperineurium and nerve fascicles, and axonal degeneration.

This was also found in non-lame limbs if surgery was undertaken bilaterally[7]. In the non-lame limb these findings may indicate the presence of neural compression caused by an enlarged SL. Therefore it may be of benefit to undertake surgery in the non-lame contralateral limb if there are any ultrasonographic abnormalities.

Success of neurectomy and fasciotomy can be assessed four to eight weeks following surgery[7]. Continued lameness at this point carries a guarded prognosis.

Ultrasonographic improvement often lags behind clinical improvement because structural improvement requires greater time. Ideally, ultrasonographic improvement is gained before any increase in exercise, regardless of clinical improvement. In some cases there is no ultrasonographic improvement, despite soundness. Reduction in cross-sectional area is often seen, which may be resolution of desmitis, or the result of neurogenic atrophy of the SL muscular tissue[45]. Painful neuromas did not occur in a study involving more than 250 horses[31], suggesting a reduced risk compared with palmar digital neurectomy[46].

Farriery should also be addressed, such as reducing the dorsal hoof wall angle. This decreases the break-over reducing the requirement for limb flexion and decreases strain within the suspensory ligament[47]. Mediolateral foot imbalance should be addressed to prevent further PSL aggravation.

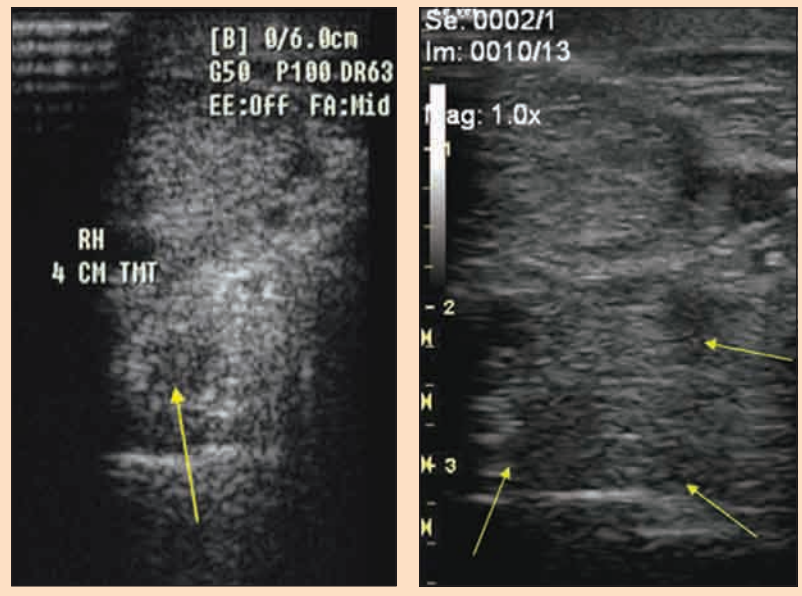

Figure 4 (left). Transverse ultrasonographic image of the proximal aspect of the right suspensory ligament, four centimetres distal to the tarsometatarsal joint in the same pony as in Figure 3. Medial is to the left. Note the large acentric hypoechogenic core-type lesion (yellow arrow).

Figure 5 (right). Transverse ultrasonographic image of the proximal aspect of the suspensory ligament, four centimetres distal to the left tarsometatarsal joint in an 16-year-old, grade A, warmblood, showjumper gelding. Medial is to the left. There is no space between the plantar aspect of the third metatarsal bone and the suspensory ligament, and the ligament is subjectively markedly enlarged. There is poor fibre pattern of the ligament with three large hypoechogenic areas (yellow arrows).

Hyperextension of the metatarsophalangeal joint can be a sequel to PSD, especially in horses that appear to have progressive degeneration of the SL[4]. In these horses, the SL may continually degenerate despite surgery. Horses with hyperextension of the metatarsophalangeal joint and straight hock conformation are at greater risk of surgical failure[43], lameness reoccurrence, SL deterioration and SL breakdown and should not be candidates for neurectomy and fasciotomy. Horses that undergo surgery for PSD with concurrent local pathology have a reduced prognosis for return to full athletic function[43]. Surgical cases should be chosen appropriately and owners advised accordingly.

Concurrent tarsal pain is reported with PSD[48]. Surgical management of horses with PSL and tarsal pain may improve the overall prognosis. The accessory ligament of the SL originates on the plantar aspect of the calcaneous and fourth tarsal bone and merges with the SL in the proximal metatarsal region[49]. As neurogenic atrophy of the muscular tissue within the SL may occur following neurectomy, it is possible the biomechanics of the distal hock joints may be altered, which may predispose to the development of distal hock joint pain[43].

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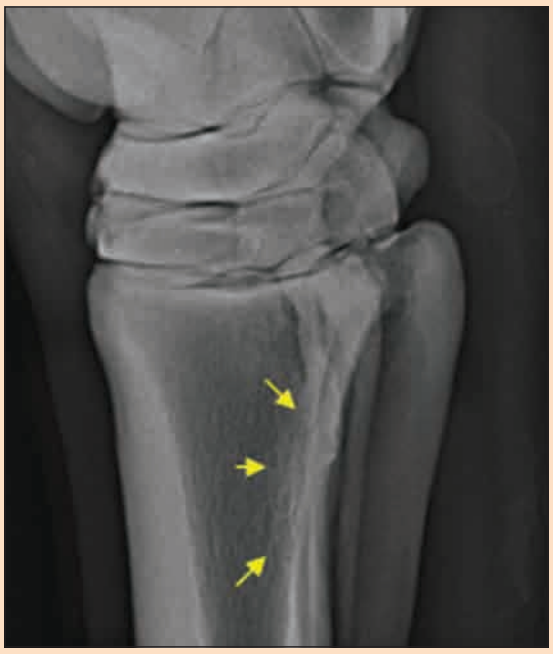

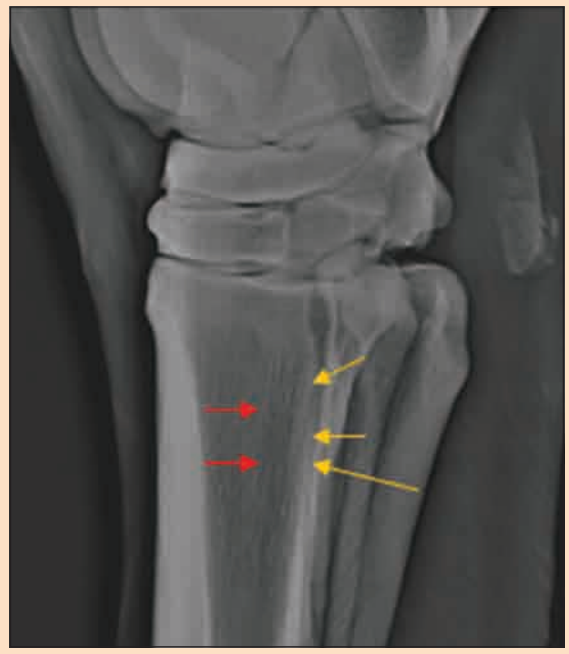

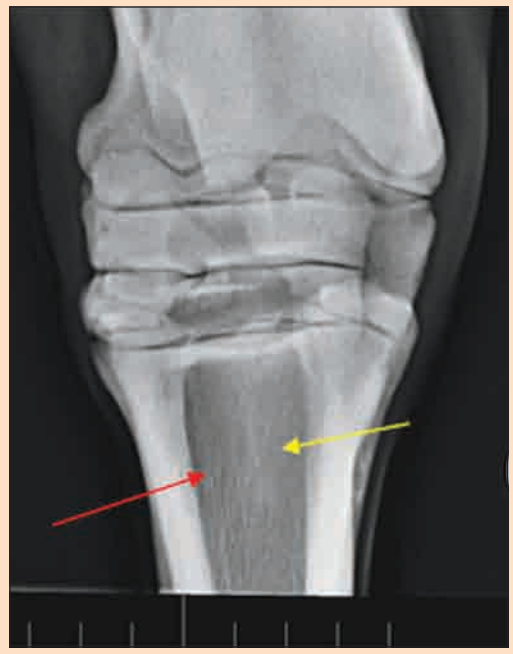

Figure 6 (far left). Lateromedial radiographic image of the right proximal metatarsal region of the same pony as in Figure 3. Dorsal is to the left. There is endosteal new bone on the dorsal aspect of the plantar cortex of the third metatarsal bone (yellow arrows). Figure 7 (centre). Lateromedial radiographic image of the right proximal metatarsal region of an eight-year-old, thoroughbred-cross horse used for general purpose. Dorsal is to the left. There is sclerosis (yellow arrows) and increased radiopacity and alteration of trabecular architecture (red arrows) of the proximoplantar aspect of the third metatarsal bone. Figure 8 (right). Dorsoplantar radiographic image of the right proximal metatarsal region of the same pony as in Figure 3. Medial is to the left. There is increased opacity (yellow arrow) of the axial aspect of the third metatarsal bone. This can be a normal finding in clinically sound horses. There is alteration in trabecular pattern (red arrow) in the region of the origin of the suspensory ligament.

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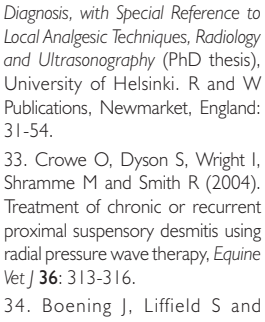

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