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The suspensory ligament (**musculus interosseus medius**) can be divided into 3 separate regions that are subject to injury: the proximal part, which extends from 4–12 cm distal to the accessory carpal bone in the forelimb and from 1–10 cm distal to the tarsometatarsal joint in the hindlimb, the body, that runs from the proximal part to the bifurcation, and the lateral and medial branches, that extend from the bifurcation to their attachment on the abaxial surface of the proximal sesamoid bones.

In the author’s practice, initial management of suspensory desmitis consists of anti-inflammatory therapy with NSAIDs, corticosteroids, hydrotherapy, rest and support bandaging. After the initial inflammatory period, standard treatment involves at least 2 months of stall confinement and a course of focused shockwave therapy. The mechanism of focused shock wave therapy remains elusive but recent work has suggested that a topical increase in tissue growth factor levels may play a role. Following this initial 2 month period of rest, the horse starts an ultrasonographically monitored graduated exercise programme. There should be progressive decrease in the cross-sectional area (CSA) and improvement in echogenicity and fibre pattern of the affected part of the ligament as exercise is increased in 2 monthly increments of progressively longer periods of first walking, then jogging and finally cantering. Return to intended exercise level often takes 6–9 months. In all horses with suspensory desmitis special attention should go to maintaining well balanced feet. Egg bar shoes are usually the shoeing method of choice.

**Management aspects of desmitis of the suspensory body**

Desmitis of the body appears to be associated with exercise at high speed in racing Thoroughbreds (flat racing as well as jumping) or Standardbreds. Correlation between the extent of a lesion and the degree of lameness can be poor. Standardbred racehorses may be able to tolerate the presence of desmitis and continue athletic function while being managed symptomatically. According to a recent report, intraleSIONal injection of platelet-rich plasma (PRP) in 9 Standardbred racehorses with moderate to severe desmitis of the suspensory body allowed all 9 horses to return to racing. In other disciplines desmitis of the body tends to be associated with exostosis of the medial or lateral splint bone and surgical removal may be indicated. Suspected interference between a splint bone exostosis and the suspensory ligament can be difficult to confirm ultrasonographically and may require MRI. Splint bone fractures that occur secondarily to suspensory desmitis should be identified radiographically but rarely require surgical removal of the distal fragment, if it is unstable or significantly displaced.

One problem of serial ultrasonographic evaluation is that many body lesions persist long term after some initial improvement of the echogenicity and CSA. Consequently, it can be hard to determine the strength of the repair and appropriate time for return to athletic function. Re-injury rates are higher for racehorses than for other horses with suspensory body desmitis.

**Management aspects of suspensory branch desmitis**

Treatment may vary somewhat depending on the equestrian discipline and additional treatment modalities are currently being used for specific injury types.

Acute, large core lesions may benefit from a splitting procedure while this does not apply to peripheral or more diffuse lesions. Show horses with mild lesions and Standardbred racehorses may be managed successfully with a modified 8 week training programme and continuous aggressive anti-inflammatory treatment while being maintained in work. In speed horses (Eventers and racehorses) this regime is likely to result in progressive deterioration of the lesion. Apical or abaxial fragmentation of a proximal sesamoid bone accompanying a branch desmitis, is best removed arthroscopically. Persistent joint effusion of the fetlock joint in a horse with branch desmitis may reflect the presence of intra-articular insertional fibre tearing of the suspensory ligament branch and arthroscopic debridement of the torn fibres may be indicated.

Recently there has been considerable interest in the use of biologics including bone marrow, mesenchymal stem cells (MSCs) and platelet rich plasma (PRP) for the treatment of suspensory branch lesions. PRP is prepared from whole blood but contains more than 4 times the level of platelets. PRP may be able to deliver high concentrations of growth factors such as transforming growth factor beta 1 and platelet-derived growth factor beta. In vitro PRP has demonstrated anabolic effects on tendon explants and suspensory ligament desmocytes, although anabolic effects were less convincing when suspensory ligament explants were used. For clinical use, PRP has been combined with bovine thrombin to promote platelet degranulation and it has been suggested that PRP is best used after the inflammatory response has subsided (7–10 days) but no more than 35–40 days after injury. As in the body, lesions may be slow to change their ultrasonographic appearance which makes timing of a return to performance difficult. Significant periligamentous fibrosis is an undesirable trait associated with an increased recurrence rate.

**Management aspects of proximal suspensory desmitis**

In a series of cases of chronic hindlimb proximal suspensory desmitis (PSD) with long-term follow-up, less than 20% of horses returned to full work following this treatment regimen. Experience with different treatment modalities has resulted in the following treatment recommendations.

Horses with acute PSD (4–6 weeks) may benefit from local periligamentous infiltration of corticosteroids and rest. Reduction
of acute inflammation may reduce the risk of a resulting compartment syndrome and persistent chronic lameness. Extracorporeal shock wave or radial pressure wave therapy has been helpful in the management of subacute or chronic PSD. Forty-one percent of 43 horses with chronic PSD were sound and in work 6 months after treatment. However, many horses remain lame either due to on-going desmitis or due to neuropathy of the deep branch of the lateral plantar nerve that has been associated with the possibility of a compartment syndrome. Although some horses with mild lameness due to chronic PSD can work satisfactorily with phenylbutazone without apparent deterioration of clinical signs, most horses with persistent lameness require neurectomy of the deep branch of the lateral plantar nerve to regain soundness. The results of this procedure in 331 horses were recently reported by Bathe (2007). Two-hundred-and-seventy-one horses underwent neurectomy and fasciotomy alone. Twenty-five cases were additionally injected with bone marrow or ACell, while 35 underwent an additional osteostixis procedure. Long-term (median 17 months) follow-up for horses that underwent neurectomy and fasciotomy alone revealed that 79% had resumed and maintained their previous level of work. The success rate for combination therapy consisting of neurectomy, fasciotomy and bone marrow or ACell injection was 84%, while a combination of neurectomy, fasciotomy and osteostixis rendered 55% of horses sound.

NOTES
11.35–12.00
How to manage proximal sesamoid bone injuries

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No abstract submitted
Diagnosis and management of distal sesamoidean ligament injuries

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There are 4 distal sesamoidean ligaments. The (paired) short and cruciate ligaments run from the proximal sesamoid bone to the adjacent P1 (ipsilateral for the short ligaments and contralateral for the cruciate ligaments). They are small ligaments, hard to image, injury is seldom diagnosed and they will form no part of this discussion. The (paired) oblique sesamoidean ligaments run from the distal border of the sesamoid bones, heading axially, to insert on the palmar/plantar surface of P1 in the mid to distal third. The straight sesamoidean ligament is not paired, and runs axially, from the sesamoid bones to insert on the middle scutum.

Radiography is seldom helpful in the diagnosis of distal sesamoidean ligament desmitis. Enthesiitis new bone in the insertion of the oblique sesamoidean ligaments (on the palmar/plantar aspect of the pastern) is a common finding. This is seldom associated with lameness and is generally considered insignificant, including at prepurchase examination. Dorsal subluxation of the pastern joint can be associated with injury to many of the soft tissue structures of the palmar pastern, including rupture or strain of the distal sesamoidean ligaments.

The straight and oblique distal sesamoidean ligaments can be visualised by ultrasonography. The straight sesamoidean ligament is an echogenic triangular structure running down the axial aspect of the pastern, becoming more oval distally. It is highly prominent on ultrasound scans, an inexperienced operator might mistake it for the deep digital flexor tendon. In the proximal extreme of the ligament it is not possible to have all the ligament ‘in plane’ which does result in a consistent hypo-echoic artefact that should not be mistaken for a core lesion. Injury of the straight sesamoidean ligament is usually imaged as a core lesion, slightly peripheral in the ligament.

The oblique sesamoidean ligament is much harder to visualise ultrasonographically. The ligament is visible in its distal half from palmar/plantar approach, with the ligaments as paired triangular structures adjacent to the reflective surface of P1, flanking the straight distal sesamoidean ligament. However, this region is seldom injured and to examine the oblique sesamoidean ligament accurately an oblique scan is necessary. The plane of the ligament requires the probe to be directed from the palmar/lateral aspect of the limb (for the lateral forelimb ligament), proximally up underneath the sesamoid bone. When preparing the pastern for scanning, it is surprising how far lateral/medial this approach is, so more area should be prepared than would be anticipated as necessary. Oblique sesamoidean desmitis is reported to be associated with suspensory ligament desmitis so consideration should be given to concurrent examination of these structures.

The length of the oblique sesamoidean ligament that is not in contact with P1 is remarkably limited. It is also surprising how hairy the skin overlying the area is - while it is often possible to scan the flexor tendons of horses without clipping the hair, it is exceptional that reliable images can be obtained of the oblique sesamoidean ligaments without complete preparation. Even with clipping a wide area, ultrasonography of the oblique sesamoidean ligaments is difficult. Injury is usually imaged as diffuse enlargement with regions of reduced echogenicity, rather than the more definitive core lesion, adding to the difficulty of reliable imaging.

Magnetic resonance imaging allows examination of the sesamoidean ligaments. The straight sesamoidean ligament can appear to have 2 parts to it - a deep part which is the sagittal component of the oblique sesamoidean ligament, and a superficial and wider part, the straight sesamoidean ligament itself. The separation into these 2 different ligaments is clearly visible on MRI scans while it is seldom evident on ultrasound scans.

There is a magnetic resonance effect known as the ‘magic angle’. At approximately 54.7° to the magnetic field (B₀), the dipolar bonds between protons fail to zero, resulting in a prolongation of the T2 time. For medical MRI this is a menace, resulting in artefactual hyper-intensity in solid structures at 50–60° to the magnetic field (B₀). With the Hallmarq MR units B₀ is perpendicular to the long axis of the limb. Two key structures are at 50–60° to this plane, the collateral ligaments of the coffin joint (particularly the lateral) and the oblique distal sesamoidean ligaments (particularly the medial). Scans that have a longer echo time are more resistant to this effect, but are still not immune to it. In practical terms the longest echo time is the T2 Fast Spin Echo sequence (84 ms). Thus any diagnosis of oblique sesamoidean desmitis, which does not include abnormalities on T2 FSE sequences, is dubious. Swelling of the ligament is helpful as an indicator of disease, but the lateral and medial ligaments are not symmetrical.

Distal sesamoidean ligament desmitis typically presents as lameness. Swelling of the pastern is seldom recognised, probably because the ligaments are quite deep. Lameness is usually exacerbated by flexion. The nerve block response is variable and confusing. Many cases will respond to intra-articular anaesthesia of the fetlock joint and some to intrasynovial anaesthesia of the tendon sheath. Most cases will not respond to a palmar digital nerve block, do respond to a 4-point nerve block and the response to abaxial sesamoid nerve block is variable.

Distal sesamoidean ligament desmitis has been reported in 27 horses following MRI diagnosis (Sampson et al. 2007). The authors reported that the prognosis was good, with 76% of horses returning to athletic activity following 6 months convalescence. In most cases the tendon sheath was medicated with corticosteroids.

We have diagnosed distal sesamoidean desmitis by MRI in 13 horses (not the same as seeing 13 horses with distal sesamoidean desmitis). Swelling was present in one horse and a tendon sheath effusion in another. Of these horses, 9 have follow-up for >6 months. Only one has returned to its previous level of athletic activity. A further 6 horses are back in exercise, but have failed to reach the same level of activity. One horse progressed on to subluxation of the pastern joint.

A variety of treatments have been used, including shockwave therapy, intralesional medication either under ultrasound or arthroscopic guidance, with platelet rich plasma, IGF-1 and bone marrow supernatant. We have concluded that the prognosis may be more guarded than has been suggested.

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