Proximal Suspensory Desmitis in the Forelimb and the Hindlimb

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1. Introduction

The suspensory ligament (SL) can be divided into three separate regions which are subject to injury: the proximal part, the body, and the branches. For clinical purposes in the forelimb the proximal part extends from approximately 4 to 12 cm distal to the accessory carpal bone, and in the hindlimb from approximately 2 to 10 cm distal to the tarsometatarsal joint.

In the forelimb, the SL originates from two heads which rapidly fuse. In the hindlimb this division is less obvious. The ligament contains a variable amount of muscular tissue (2–11%), which tends to be bilaterally symmetrical.1 In the forelimb the SL originates from the palmar carpal ligament and the proximal aspect of the third metacarpal bone, whereas in the hindlimb it originates principally from the proximoplantar aspect of the third metatarsal bone. In the forelimb the SL is innervated by the palmar metacarpal nerves, derived from the lateral palmar nerve, which receives fibres from both the ulnar and median nerves.2 The hindlimb SL is innervated by the plantar metatarsal nerves, derived from the tibial nerve. The proximal SL is closely related to the palmar outpouching of the middle carpal joint in the forelimb,3 and the plantar outpouching of the tarsometatarsal joint in the hindlimb.4

Proximal suspensory desmitis (PSD) is a common injury in both forelimbs5–9 and hindlimbs10,11 of athletic horses, and may occur unilaterally or bilaterally.

2. Proximal Suspensory Desmitis in the Forelimb

PSD results in a sudden onset lameness, which can be remarkably transient, resolving within 24 h unless the horse is worked hard. Lameness varies from mild to moderate and is rarely severe unless the lesion is extensive. Bilateral PSD may result in loss of action rather than overt lameness. This occurs more commonly in flat racehorses, probably due to failure of recognition of earlier, subtle unilateral lameness. Lameness is usually worse on soft ground, especially with the affected limb on the outside of a circle, and, when subtle, may be more easily felt by a rider than seen by an observer. Lameness may not be apparent at working trot, but may be detectable at medium or extended trot. Recognition of these features in the history may be important since lameness often resolves rapidly and it may be undesirable to work the horse hard to reproduce lameness, with the inherent risk of worsening...
the injury. Lameness is often transiently accentuated by distal limb flexion.

In the acute phase there may be slight edema in the proximal metacarpal region, localized heat, and distension of the medial palmar vein, but these features may be transient or absent. Pressure applied to the SL against the palmar aspect of the third metacarpal bone may elicit pain. Forced extension and protraction of the limb may elicit pain.

The feet should be evaluated carefully since frequently foot imbalance is a predisposing factor.

Local Analgesic Techniques

If PSD is suspected, perineural analgesia of either the lateral palmar nerve (2 ml mepivacaine) or the medial and lateral palmar metacarpal nerves (2 ml per site) is indicated. This should result in substantial improvement in, or alleviation of, lameness within 10 min, assuming PSD is the only cause of lameness. However, neither technique is necessarily specific. Blockade of the lateral palmar nerve also has the potential to alleviate pain associated with a lateral source of pain in the more distal limb. The risks of influencing middle carpal joint pain are less than using the subcarpal approach, but local anaesthetic solution may diffuse and result in improvement in lameness associated with the middle carpal joint. Perineural analgesia of the palmar metacarpal nerves may alleviate pain associated with either the middle carpal or carpometacarpal joint, due to local diffusion or inadvertent deposition of local anaesthetic solution into the palmar outpouching of the middle carpal joint capsule. A false negative result may be achieved either due to inadvertent injection into the carpal sheath, or failure of the local anaesthetic solution to diffuse proximally to the most proximal extent of a lesion. Although the SL receives innervation from fibres from both the median and ulnar nerves, perineural analgesia of the ulnar nerve usually resolves lameness associated with PSD.

Intra-articular analgesia of the middle carpal joint may result in either partial improvement or complete alleviation of pain associated with the proximal suspensory ligament in some cases (15/25 horses, 60%). Using a dorsal approach to the middle carpal joint rather than a palmarolateral approach should theoretically reduce the risks of diffusion of local anaesthetic solution to the proximal SL and palmar metacarpal nerves; however, in practice there appears to be little difference. Comparison of the relative responses to middle carpal joint analgesia (6 ml mepivacaine; assessed 10 min after injection) and perineural analgesia of the lateral palmar nerve or the palmar metacarpal nerves...
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Diagnostic Ultrasonography

Diagnostic ultrasonography is essential for accurate diagnosis of PSD. The limb should be evaluated in both transverse and longitudinal planes and careful comparison should be made with the contralateral limb. High quality images are required since lesions can be subtle and easily missed if the gain controls are too high and the transducer is not focused on the SL. Cross-sectional area measurements may be extremely valuable since, especially in acute cases, enlargement of the ligament may be the only detectable ultrasonographic abnormality. Bear in mind that muscular tissue appears less echogenic than ligamentous tissue and that proximally the SL originates in two halves. Therefore, the proximal aspect of a normal SL is not necessarily of uniform echogenicity in transverse images. In longitudinal images in the most proximal part of the metacarpal region it may be difficult to orientate the transducer perpendicular to the line of the fibers, thus the SL may appear less echogenic than further distally. Previous injuries to the SL may not resolve fully to restore normal, uniform echogenicity. Be aware that poor local anesthetic technique may result in aspiration of air, which will create artefacts.

Abnormalities associated with PSD include:

- Enlargement of the cross-sectional area. This may result in reduction of space between the SL and the palmar cortex of the third metacarpal bone, or reduced space between the SL and the accessory ligament of the deep digital flexor tendon.
- Poor demarcation of the margins of the SL, especially the dorsal margin.
- Focal or diffuse areas of reduced echogenicity. These may extend less than 1 cm proximodistally and occupy from less than 10% to up to the entire cross-sectional area of the ligament.
- Focal anechoic core lesions.
- Reduced strength of fiber pattern.
- Focal mineralization.

In a three-year-old Thoroughbred that had had sustained PSD at two years of age, there may be recurrent mild lameness and it may not be possible to discern any structural abnormality other than enlargement of the SL.

The degree of ultrasonographic abnormality (cross-sectional area involved and proximodistal extent of the lesion) usually reflects the severity of the lameness. In acute cases the ultrasonographic abnormalities may be very subtle. They may deteriorate over the next 10 to 14 days and re-evaluation may be useful to confirm the diagnosis.

In horses with an avulsion fracture of the origin of the suspensory ligament, the avulsed fragment is usually readily identifiable and is generally associated with only a very focal lesion in the SL itself, usually restricted to the dorsal aspect.

Radiography

There are usually no detectable radiographic abnormalities of the third metacarpal bone in acute cases of PSD. In chronic cases increased opacity of the proximal aspect of the third metacarpal bone may be seen in dorsopalmar views. This sclerosis should be differentiated from that associated with a palmar cortical fatigue fracture. In a lateromedial projection there may be sub-cortical sclerosis in the
proximal palmar aspect of the third metacarpal bone. These secondary bony changes in a forelimb are associated with a more guarded prognosis.

Nuclear Scintigraphy

Nuclear scintigraphy is generally unnecessary for diagnosis provided that good quality ultrasonographic images are obtained, but may give additional information about associated bone turnover at the insertion of the SL. Pool phase images using technetium 99m MDP are rather insensitive, and may be negative. Abnormal uptake may actually reflect very early bone uptake. Bone phase images may or may not show increased uptake of the radio-pharmaceutical in the palmar aspect of the third metacarpal bone. Negative scintigraphic images do not preclude the presence of PSD. Abnormal uptake in the bone phase seen in the absence of ultrasonographic abnormality is more likely to reflect primary bony pathology.

Treatment

Most cases of acute forelimb PSD respond well to box rest and controlled walking exercise for 3 months. Attention to correct foot balance is important. A premature resumption of work usually results in recurrent injury. Approximately 90% of horses resume full athletic function without recurrent injury. More chronic cases may require more prolonged rehabilitation, and in a small proportion lameness is persistent. Extracorporeal shock wave treatment (3 treatments at 2 week intervals) has been successful in some chronic cases, which had failed to respond to conservative management.

In some horses the lesions disappear completely ultrasonographically. In others there may be some increase in echogenicity, but uniform echogenicity is never restored. Rest should be continued until the ultrasonographic appearance remains stable.

3. Proximal Suspensory Desmitis in the Hindlimb

PSD in the hindlimb results in either an insidious onset or sudden onset lameness which may be mild or severe. In contrast to 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. 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 unrecognized. The incidence of bilateral lesions is higher than in forelimbs. Horses with either straight hock conformation and/or hyperextension of the metatarsophalangeal joints appear predisposed to injury. Such conformational abnormalities were identified in 9 of 42 horses (21%) with hindlimb PSD, but in only 4 of 50 consecutive horses (8%) with hindlimb lameness unrelated to the suspensory apparatus. Hyperextension of the metatarsophalangeal joint may also develop as a sequel to PSD.

In acute cases there may be localized heat and swelling and pain on pressure applied to the SL, but frequently there are no localizing clinical features. Lameness is often characterized 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 rather than obvious hindlimb lameness. Like many hindlimb lamenesses, lameness is often more obvious when the horse is ridden, especially when the rider sits on the diagonal of the lame limb.

Local Analgesic Techniques

Perineural analgesia of the plantar nerves (mid metatarsal level) and plantar metatarsal nerves may result in slight improvement in lameness due to proximal diffusion of the local anaesthetic solution. Lameness is usually substantially improved by perineural analgesia of the medial and lateral plantar metatarsal nerves distal to the tarsus, but may not be alleviated fully. It is difficult to deposit the local anaesthetic solution as proximal as ideal due to the shape of especially the fourth metatarsal bone. False negative results may be obtained due to inadvertent injection into either the tarsal sheath or the tarsometatarsal joint capsule. Sub-tarsal analgesia can influence tarsometatarsal joint pain, and occasionally (2/24 horses, 8%11) intra-articular analgesia of the tarsometatarsal joint alleviates pain associated with PSD. Perineural analgesia of the tibial nerve alone alleviates pain associated with PSD, without influencing tarsal pain.

Diagnostic Ultrasonography

High quality ultrasonographic images are essential for accurate diagnosis. Large vessels plantarolateral to the SL may result in broad linear anechoic artefacts within the SL, which complicate interpretation. In large Warmblood horses in particular the SL is situated deeply and the ultrasound transducer must be focused accordingly. The SL should be imaged in both transverse and longitudinal planes. To examine the most proximal part of the SL in transverse images it may be helpful to rock the transducer slightly medially and laterally in order to obtain the best quality images. In a normal horse the proximal part of the hindlimb SL is more uniformly echogenic than in the forelimb.

In PSD in the hindlimb focal anechoic areas are relatively unusual. More commonly there is enlargement of the SL, with poor demarcation of its borders and a diffuse reduction in echogenicity of part or all of the cross-sectional area of the ligament (Fig. 1). Ectopic mineralization occurs more often in hindlimbs compared to forelimbs. An irregular contour of the plantar contour of the third metatarsal bone may reflect enthesophyte formation. 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 horses have some sclerosis of the proximal aspect of the third metatarsal bone. In horses with chronic active PSD this tends to be more extensive. In the dorsoplantar view there is increased opacity of the proximal aspect of the third metatarsal bone, often more obvious laterally. In a lateromedial projection there is sub-cortical sclerosis and alteration of the trabecular pattern of the proximoplantar aspect of the third metatarsal bone due to endosteal new bone, extending up to 4 cm proximodistally. The plantar cortex may itself be thickened and in addition there may be entheseophyte formation on its plantar aspect. However in some acute cases no radiological abnormality is detectable.

Nuclear Scintigraphy
Not all horses with PSD have detectable abnormalities if examined using nuclear scintigraphy (Fig. 2). Pool phase images appear to be rather insensitive. In bone phase images there may be increased uptake of the radiopharmaceutical in the proximoplanar aspect of the third metatarsal bone in some, but not all cases of PSD. This should be differentiated from those horses with primary bony pathology with no detectable ultrasonographic abnormality of the SL (Fig. 3).

Differential Diagnosis
PSD should be differentiated from pain associated with the tarsometatarsal joint, and primary stress reactions in the third metatarsal bone.

Treatment
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. All these horses showed marked clinical improvement within 3 months of the onset of lameness. Two additional horses resumed full work, but suffered lameness in another limb. Seven horses improved substantially 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.

Acute cases (less than 4–6 weeks duration) of PSD respond reasonably to local infiltration with corticosteroids, aimed to reduce inflammation and therefore swelling and thus minimize the risk of the development of a compartment syndrome (see below). Chronic cases have a very guarded prognosis regardless of the treatment. Lameness tends 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, glycosaminoglycan polysulphate, sodium hyaluronan or actovegin and Traumil has given disappointing results. Some horses have worked satisfactorily while being treated with phenylbutazone, without apparent deterioration of clinical signs. Tibial neurectomy performed in three horses enabled all to return to full athletic function (show jumping and horse trials). Extracorporeal shock wave therapy (lithotripsy) appears to be helpful in some cases. Fasciotomy has also been successful in some horses.

Gross Pathology and Histopathology
Post mortem examinations have been performed on both hindlimbs of eight horses, 6 with unilateral lameness and 2 with bilateral lameness. 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, hyalinization of collagen, an increased number of fibrous septae, some with blood vessels, neovascularization, 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 5 horses. Abnormalities of the plantar metatarsal nerves included thickening of the perineurium, perineural fibrosis, reduction or absence of nerve fibers, and Renaut bodies. These changes support the theory of PSD in the hindlimb resulting in a compartment syndrome.

References and Notes

*b Boening, J. Personal communication 1999.
*c Ross, M. Personal communication 2000.