Proximal Metatarsal Lameness in Sports Horses: 
A Clinical Approach to Diagnosis

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Proximal metatarsal pain is most commonly caused by desmopathy or enthesopathy of the proximal part of the suspensory ligament. Diagnosis of the causes of proximal metatarsal pain has improved markedly with the use of more specific regional analgesic techniques, objective techniques of lameness quantification, and cross-sectional imaging methods. However, even diagnostic anesthesia of the deep branch of the lateral plantar nerve cannot be considered totally specific for the diagnosis of proximal suspensory desmitis. Treatment methods are often unsuccessful, and neurectomy of the deep branch of the lateral plantar nerve may be necessary to regain soundness. Author’s address: Clinéquine, Campus Vétérinaire de l’Université de Lyon, VetAgro Sup, 69280 Marcy L’Etoile, France; e-mail: michael.schramme@vetagro-sup.fr. © 2013 AAEP.

1. Introduction
Proximal plantar metatarsal pain can be defined as lameness that improves after anesthesia of the deep branch of the lateral plantar nerve or other forms of subtarsal anesthesia. Proximal plantar metatarsal pain appears to have become the most commonly diagnosed cause of hind limb lameness in sports horses, even more common than distal tarsal joint pain. Recent high-field magnetic resonance (MR) imaging studies of horses with proximal plantar metatarsal pain have indicated that proximal suspensory desmopathy (PSD) and/or enthesopathy was identified as the cause of lameness in 55% to 80% of these horses, whereas in the remaining cases the diagnosis was unrelated to the suspensory ligament.1,2

2. Anatomy and Risk Factors
The proximal portion of the hind suspensory ligament is contained within a restricted canal composed of the plantar surface of the third metatarsal bone, the large head of the fourth metatarsal bone, and the overlying deep lamina of the flexor retinaculum that connects the plantar borders of the second and fourth metatarsal bones. This particular anatomical arrangement has led to suggestions that proximal metatarsal pain may arise from a compartment-like syndrome with compression of the lateral and medial plantar metatarsal nerves rather than from the suspensory ligament itself.3 Although the prevalence of PSD in sports horses appears to have increased in recent years, the reasons for this remain poorly understood. Improved recognition not only by veterinarians but also by equestrian professionals certainly has played a role. Modern training demands, regimes, and surfaces may also contribute. Predisposing factors have been identified as dressage training not only at advanced levels but also at lower, non-elite levels (in particular piaffe, passage, and pirouettes),4 straight hock and hyperextended fetlock conformation,5 and
long toe-low heel conformation, especially in combination with a negative palmar angle of the distal phalanx.5

3. History and Clinical Signs
A diagnosis of proximal metatarsal pain is based on the history, evaluation of risk factors and conformation, clinical signs, response to diagnostic anesthesia, and imaging.

PSD is frequently though not always associated with a straight hock and hyperextension of the metacarpophalangeal/metatarsophalangeal (MCP/ MTP) joint. It is not always clear if this appearance is a primary risk factor or a secondary postural change caused by loss of strength of the stay apparatus in affected horses. The reason for presentation of the horse to a veterinarian may vary from subtle loss of performance to marked, unilateral lameness. Loss of performance during ridden work may present as bilateral stiffness, loss of hind limb impulsion, difficulties in transitions, resistance to lateral exercises, flying changes or canter pirouettes, evasive behavior, or reduced power when jumping. Horses tend to warm out of early injuries fairly quickly. Lameness may be absent or present as mild with an insidious progression or severe with an acute onset. Bilateral lameness is common and can be mild to moderate in degree.

Clinical signs of acute inflammation (swelling, heat, and pain over the affected ligament) may be evident in acute cases but are more frequently absent. The presence of swelling is best identified by palpation in the standing limb. Swelling will result in some loss of the concave profile of the skin on the plantarolateral aspect of the limb, between the plantar border of the lateral splint bone and the lateral margin of the superficial digital flexor tendon in the proximal metatarsal region (Fig. 1). This is a highly specific clinical finding even when mild, and it should always be compared with the same area in the contralateral limb. Pain on palpation is easier to detect in the raised limb, even though the proximal part of the suspensory ligament is difficult to palpate in the hind limb because it is largely covered by the heads of the lateral and medial splint bones and lies deep to the digital flexor tendons. The best technique in this case is to exert pressure on the proximal part of the suspensory ligament by compressing the flexor tendons dorsally. Horses with PSD may also resent pressure between the head of the lateral or medial splint bone and the lateral or medial margin of the superficial digital flexor tendon. Some clinicians believe the Churchill test to be particularly useful in identifying pain in the proximal part of the hind suspensory ligament.

Evaluation of lameness should be performed with the horse trotting in straight lines and lunging in circles both on a hard surface and a soft surface. As with many horses with hind limb lameness, there is often a reduced arc of foot flight, reduced extension of the fetlock joint, and shortening of the cranial phase of the stride. When examining horses with mild lameness caused by proximal plantar metatarsal pain, the use of a wireless, inertial, sensor-based system of lameness quantification can be of tremendous help to quantify the degree of lameness objectively and to characterize the nature of the asymmetry in vertical displacement of the pelvis between strides. Horses with PSD often show push-off rather than impact lameness. The effect of circling and surface variation is less predictable than in horses with forelimb PSD. There is frequently but not always a moderately positive response to both distal and proximal flexion tests. In some horses with PSD, lameness is only obvious under saddle, especially when the rider is sitting on the diagonal of the lame limb.

4. Diagnostic Anesthesia
There are many different ways of removing sensation from the proximal plantar metatarsal region, in particular from the origin and body of the suspensory ligament. Techniques include the high six-point nerve block, the tibial nerve block, direct infiltration of local anesthetic solution around the origin of the suspensory ligament, and anesthesia of the deep branch of the lateral plantar nerve. This latter technique has a better chance of improving the specificity of diagnostic anesthesia compared with the other techniques.6 The horse is restrained with a twitch, the affected hind limb is lifted, and the fetlock is supported on the clinician’s knee with
the hock flexed at 90° and the third metatarsal bone positioned vertically. The superficial digital flexor tendon (SDFT) is deflected medially, and a 25-mm, 23-gauge needle inserted perpendicular to the skin surface, 15 mm distal to the head of the fourth metatarsal bone, on the plantarolateral surface of the metatarsal region. The needle is advanced in a slightly dorsomedial direction between the fourth metatarsal bone and the lateral border of the SDFT up to the hub, and 3 to 4 mL of mepivacaine is injected without resistance. Occasionally, blood is seen to flow freely from the needle, indicating puncture of the venous portion of the (proximal) deep plantar arch, in which case the needle should be re-directed slightly more dorsolaterally to avoid intravascular injection. The lateral placement of the needle in this technique reduces the risk of inadvertent penetration of the tarsometatarsal joint and the tarsal sheath when compared with other methods of subtarsal anesthesia. However, in up to 20% of horses in which 2.5 mL of mepivacaine was injected at this site, the lateral plantar nerve also appeared to have been desensitized. Therefore, it is always advisable to assess the effect of anesthesia of the distal limb with a low six-point nerve block first, before performing diagnostic anesthesia of the deep branch of the lateral plantar nerve.

Lameness is assessed 10 to 15 minutes after injection. Critical evaluation of the degree of improvement can be performed accurately and objectively with a wireless, inertial, sensor-based system of lameness quantification. This is essential when considering treatment by neurectomy. It is also important when comparing the degree of improvement with that seen after intra-articular anesthesia of the distal tarsal joints. Subtarsal anesthesia may improve tarsometatarsal joint pain and vice versa, but most improvement in suspensory pain is usually seen after anesthesia of the deep branch of the lateral plantar nerve. It has been suggested that pain is less successfully alleviated by anesthesia of the deep branch if enthesopathy of the proximal plantar portion of the metatarsal cortex is present. In these cases, direct deeper infiltration of 2 to 4 mL of mepivacaine at the bone surface may be more effective in abolishing lameness. Anesthesia of the tibial nerve alone eliminates suspensory ligament pain without completely removing sensation from the distal tarsal joints.

5. Differential Diagnosis
Recent high-field MR imaging studies of horses with proximal plantar metatarsal pain have indicated that PSD and/or enthesopathy was identified as the cause of lameness in the majority of them (55–80%), whereas in 20% to 25% of horses, a pathologic process unrelated to the suspensory ligament was documented, and in 10% to 20% of cases, no reason for the lameness could be found in the proximal metatarsal or distal tarsal regions. Lesions that were considered responsible for lameness but were unrelated to the suspensory ligament included osteoarthritis of the distal tarsal joints, osseous cyst-like lesions of the tarsal bones, tarsal bone edema, enthesopathy of the intertarsal ligaments, osseous injury of the third or fourth metatarsal bones, ten- dinopathy of the deep or superficial digital flexor tendon, and desmopathy of the plantar ligament. Other injuries that should be considered in the proximal plantar metatarsal region are stress fractures of the plantar metatarsal cortex and avulsion fractures of the origin of the suspensory ligament. Neuropathy of the deep branch of the lateral plantar nerve may be the cause of pain in horses without imaging abnormalities.

6. Imaging
An accurate imaging diagnosis of proximal metatarsal pain is of great importance because recommended options for management are costly and time-demanding. This diagnosis can be based on radiographic, scintigraphic, sonographic, and MR imaging findings. Radiographic and scintigraphic findings are useful for the detection of bone injuries but frequently nonspecific for PSD. Sonographic assessment of the proximal portion of the suspensory ligament is difficult. High-field MR imaging was recently shown to be the most reliable technique for accurate diagnosis of the causes of proximal metatarsal pain.

It is recommended that a complete radiographic examination of the tarsus and proximal metatarsal regions is always performed because distal hock joint pain and PSD may coexist in horses with proximal metatarsal pain. Accurate radiographic assessment of the proximal aspect of the third metatarsal bone for the presence of increased radiopacity or avulsion fractures requires that radiographic views be centered at this level. In the dorsoproximal image of the proximal aspect of the third metatarsal bones of sound horses, there can be a variable amount of increased radiopacity that should not be interpreted as a pathologic stress reaction at the origin of the suspensory ligament. In the dorsoproximal image, increased opacity is frequently most obvious laterally. On lateromedial images, remodeling changes may include thickening of the plantar cortex, endosteal new bone, alteration of the trabecular pattern of the proximoplantar aspect, and enthesophyte formation on the plantar aspect of the third metatarsal bone. Although increased radiopacity in this region may be more extensive in horses with chronic PSD, these radiographic findings are frequently not specific. In a recent report, features of PSD in 155 horses showed that 21% of lame limbs had a spur on the dorsoproximal aspect of the third metatarsal bone; 30% had mild, diffusely increased radiopacity proximolaterally in the third metatarsal bone; 3% had focal areas of intensely increased radiopacity; and 6% had low-grade osteoarthritis of the distal tarsal joints.
Ultrasonographic examination of the proximal portion of the suspensory ligament is performed with the use of a ≥7.5-MHz linear-array transducer and must always include comparison with the contralateral limb. A delay of 1 to 2 days after diagnostic anesthesia is useful to avoid imaging artifacts caused by air in the tissues. Alternatively, the ultrasonographic examination may precede the use of nerve blocks. Even with careful attention to detail, ultrasonographic examination of the proximal metatarsal region is a difficult technique. The superficial and deep digital flexor tendons and the inferior check ligament are superimposed over the suspensory ligament. The plantar soft tissue structures all have a different echogenicity, slightly different fiber orientation, and acoustic impedances. Overlying tendons and large vessels also cause refraction and enhancement artifacts, adding to the heterogeneity in the echogenicity of the suspensory ligament itself. The margins of the splint bones interfere with visualization of the lateral and medial borders of the ligament. These factors significantly compromise the ability to interpret the morphology of the proximal portion of the suspensory ligament accurately. Modifications to ultrasonographic techniques that have been suggested to improve accuracy include a plantaromedial position for the ultrasound probe, holding the limb in a non–weight-bearing position, and the use of an off-incidence ultrasound beam. In addition, the use of stand-off pads and convex-array or virtual convex-array transducers may offer a wider field of view on the proximal portion of the suspensory ligament. The suspensory ligament has a more heterogeneous echogenicity than the flexor tendons and a less linear fiber pattern on longitudinal views. At its origin, the echogenicity is most variable and can include normal lateral and medial hypoechoic regions that must be differentiated from pathology. These normal variants are caused by areas of looser connective tissue within the ligament containing fat and vascular elements. Unlike the normal echogenicity variants, pathological changes tend to be asymmetrical and are associated with other changes such as altered linear fiber pattern and enlargement (Fig. 2). The suspensory ligament separates from the plantar metatarsal cortex at 4 cm distal to the level of the tarsometatarsal joint. Distal to this point, the dorsal border of the ligament should be distinct and separated from the underlying metatarsal cortex by a small anechoic gap. It has been suggested that the presence of injury of the proximal part of the suspensory ligament is most commonly recognized by the presence of ultrasonographic enlargement, with poor demarcation of the borders and diffuse reduction of the echogenicity rather than by the presence of focal areas of hypoechoogenicity. It should be pointed out that this suggestion is not in accordance with the focal nature of many lesions as described in recent MR imaging studies (Fig. 3). An irregular contour of the plantar aspect of the third metatarsal bone may indicate enthesophyte formation, but this finding may not be specific for the presence of PSD. It has also been suggested that there is little use in measuring the cross-sectional area of the suspensory ligament at this level because only the central third of the proximal portion of the ligament can be visualized with the routine ultrasonographic technique. In addition, the cross-sectional area of the suspensory ligament changes markedly in its most proximal 6 cm, the area in which most lesions occur. The cross-sectional area of normal suspensory ligaments was measured on MR images as 0.86 cm² at the level of the tarsometatarsal joint, 2.08 cm² at 2 cm, 1.81 cm² at 4 cm, 1.69 cm² at 6 cm, and 1.57 cm² at 8 cm distal to the level of the tarsometatarsal joint. Even so, it has been suggested that ultrasonographic cross-sectional area measurements of >1.5 cm² are suggestive of PSD.

Nuclear scintigraphy of the proximal metatarsal region of sound horses has shown a characteristic distribution of radiopharmaceutical uptake. Maximum uptake occurred at the central to plantar aspect of the proximal metatarsal region in the lateral images, with peak activity over the lateral portion of the proximal metatarsal region on plantar images. In addition, there was a significant difference between left and right proximal metatarsal regions, with higher radiopharmaceutical uptake in the right hind limb. However, nuclear scintigraphy cannot be considered a sensitive tool for the detection of...
PSD in the hind limbs of lame horses. Both pool and bone phase images were found to be abnormal in only 12% of horses with ultrasonographic evidence of PSD. Increased radiopharmaceutical uptake in the proximoplanter aspect of the third metatarsal bone without detectable ultrasonographic or radiographic abnormalities probably represents primary osseous pathology such as stress injury or enthesopathy at the origin of the suspensory ligament rather than PSD per se. In a recent study of 155 horses with PSD, there was increased radiopharmaceutical uptake in the proximoplanter aspect of the third metatarsal bone, unilaterally or bilaterally, in 15% of horses.

MR imaging is the best-suited imaging technique for assessment of the proximal portion of the suspensory ligament, thanks to its superior soft tissue resolution and detail. MR imaging is able to visualize inflammatory fluid within bone, tendons, or ligaments when gross pathologic changes have not yet occurred and cross-sectional images allow accurate assessment of the size of the ligament. High-field MR imaging is a more sensitive imaging technique than is ultrasonography for lesions that have bone edema, mild to moderate sclerosis, fiber disruption, or ligament enlargement. Objective comparisons of ultrasonography, MR imaging, and histology have been performed for the proximal part of the suspensory ligament of hind limbs of sound horses. MR imaging allowed accurate quantification of suspensory ligament dimensions and accurate identification of tissue bundles containing muscle, adipose, and loose connective tissue throughout the entire length of the origin and body of the suspensory ligament, whereas ultrasonography was unable to distinguish these bundles from surrounding dense collagenous tissue.

High-field MR imaging findings in lame horses indicated that lesions of the proximal part of the suspensory ligament consisted predominantly of focal areas of signal increase that extended on average from 14.2 mm to 50.4 mm distal to the level of the tarsometatarsal joint, with lesion length varying from 4.3 mm to 107 mm. When comparing ultrasonographic with MR imaging findings, ultrasonography was found to have a sensitivity of 66% and a specificity of 31% for the diagnosis of confirmed PSD. Because of the high incidence of false-positive ultrasonographic diagnoses, ultrasonography was considered of limited value for the detection of PSD. In comparison with high-field magnets, low-field MR imaging on standing horses only has a limited ability to show anatomic detail of the proximal portion of the suspensory ligament and to detect soft tissue lesions accurately, mainly because of imaging artifacts caused by movement of the horse. Consequently, the most common primary abnormalities detected with low-field MR imaging in this region are osseous injuries.

References and Footnote


*“Lameness Locator, Equinosis, 1141 South 7th Street, St. Louis, MO 63104.