Soft Tissue and Periarticular Conditions of the Plantar Tarsal Region

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The plantar aspect of the tarsus is a complex arrangement of tendons, ligaments, synovial structures, and bony processes. The region is often subjected to extreme, repetitive propulsive forces as well as direct external trauma. Injuries are relatively common in many types of performance horses. Characteristic gait deficits and physical-exam findings rarely exist. Accurate localization of pain by diagnostic anesthesia is important, but it can be complicated by overlapping innervations to the region. High-resolution radiography and ultrasonography remain invaluable to assess structural damage. Magnetic resonance imaging allows for diagnosis of many soft tissue and bone injuries that cannot be found by conventional imaging. Diagnostic and therapeutic advances seem to be improving the outcome of many orthopedic conditions that have traditionally warranted a guarded prognosis. Authors’ address: Oakridge Equine Hospital P.C., 6675 East Waterloo Road, Edmond, OK 73034; e-mail: mmajor@oakridgevet.com. © 2006 AAEP.

1. Proximal Suspensory Desmitis of the Hindlimb

Proximal suspensory desmitis (PSD) is an important, often overlooked cause of hindlimb lameness. Because of the prevalence of distal tarsitis and the proximity of the suspensory origin to the lower hock joints, proximal suspensory pain can be easily misdiagnosed as distal tarsitis and vice versa. PSD of the hindlimb can occur in horses of all breeds, ages, and uses. Increased incidence of hind suspensory injury has been associated with the disciplines of dressage, jumping, and harness racing.¹ In our practice, the Western performance Quarter Horse, especially the cutting horse, seems to be at increased risk for this injury. This impression is contrary to a previous survey,² and it may be caused by our regional prevalence of Western performance horses and number of referral lameness cases.

Clinical Signs

No characteristic gait abnormalities are observed with hindlimb PSD. Cranial stride may be shortened and arc of flight reduced so that the gait deficit is similar to that seen with distal tarsitis. Severity and onset of lameness can range from a subtle, insidious decrease in performance to an acute onset of severe lameness. Involvement of both hindlimbs is common in horses with chronic lameness, although severity of damage and lameness are usually asymmetric. Unilateral damage is more common with acute onset of lameness. Unlike PSD of the forelimb, the lameness is usually not accentuated by trotting in a circle with the lame limb on the outside. The severity of lameness tends to be consistent when the horse is trotting straight and in either circle. In horses with subtle lameness, the gait abnormality is often more obvious with a rider than
when jogging in hand or lunging. Responses to flexion tests of the upper or lower hindlimb are variable. In some cases, lameness is increased after the opposite hindlimb is held in flexion, likely from increased loading of the suspensory apparatus. Heat and swelling in the region are usually absent. Some horses with PSD show tenderness to deep palpation axial to the splint bones in the proximal metatarsal region. Lameness may be temporarily worse if the horse is jogged off immediately after digital pressure is applied to the suspensory origin. With more severe damage that extends distally into the body of the suspensory ligament, enlargement can be appreciated when the medial margin of the ligament is palpated.

Diagnostic Anesthesia

Because gait characteristics, physical examination, and conventional imaging modalities are often not diagnostic, accurate localization of pain with diagnostic blocks is essential. Before the proximal suspensory region is blocked, lower limb lameness diagnostic blocks is essential. Before the proximal suspensory ligament, enlargement can be appreciated when the medial margin of the ligament is palpated.

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pain is present in the most proximal extent of the suspensory origin, the lameness may not be completely abolished. Analgesia of the tibial nerve above the point of the hock completely desensitizes the suspensory ligament. Tibial nerve block without blocking the peroneal nerve should not completely alleviate pain from distal hock disease, although some improvement in lameness may result. In the authors’ opinions, the tibial nerve block is more dangerous and time consuming to the clinician than a subarticular approach for analgesia of the suspensory ligament.

Intra-articular anesthesia of the TMT joint is reported to completely eliminate pain from the suspensory origin only in rare occasions. In 24 cases of hindlimb PSD that became sound after subarticular analgesia, only two horses became sound when the TMT joint was blocked later. Optimally, a subarticular block should be assessed to eliminate suspensory pain before the TMT joint is blocked. If the TMT block is performed first, the desensitization of the suspensory origin can be assessed by the pressure test described above. Evaluation of lameness within 10 min of a TMT block should decrease the likelihood of inadvertently blocking the suspensory origin by diffusion from the plantar TMT pouch. A small volume (3 ml) of local anesthetic is adequate for the intra-articular TMT block. Injection of larger volumes under pressure increases the likelihood of anesthetic diffusion or joint capsule rupture, resulting in a ring block effect. Every attempt should be made to rule out PSD in the horse that “blocks sound” in the TMT joint but does not respond to intra-articular therapy.

Ultrasonographic Evaluation
After the lameness is localized to the proximal suspensory region, ultrasound imaging is useful to estimate the degree of structural damage. The information can assist in formulating a plan for treatment and rehabilitation as well as prognosis of the injury. Differences in anatomy of the proximal suspensory region in the hindlimb from that of the forelimb must be considered to obtain quality images. In the proximal hindlimb, the lateral splint bone is positioned more caudal than the medial splint bone, and the plantar surface of MT III is on an oblique plane that angles from caudolateral to dorsomedial. The DDFT resides medial from midline, and the SDFT is located laterally. The proximal suspensory can best be imaged by positioning the transducer on the plantar-medial aspect of the limb. The suspensory origin is imaged through the DDFT with the beam at a dorsolateral angle that is perpendicular to the plantar surface of MT III. The hind suspensory is more round on cross section than in the forelimb, and the fiber density is normally more uniform throughout the most proximal portion. Normal thickness (plantar to dorsal) is ~2 mm greater in the hindlimb (10–12 mm). The most common abnormal findings in the suspensory origin are enlargement and diffuse loss of echogenicity with loss of defined margins in the dorsal portion. Central hypoechoic lesions are more rarely seen. Abnormalities are often seen best in a long axis image. On the longitudinal view, the plantar surface of the proximal hind suspensory is normally linear to slightly concave. Beginning ~4 cm distal to the TMT joint, a fluid density space can normally be seen between the dorsal aspect of the suspensory and plantar surface of MT III. With enlargement of the suspensory origin, the plantar surface becomes convex and the space between the suspensory ligament and MT III is lost. Examination of the opposite hindlimb can be helpful, but that limb cannot be assumed to be normal, even if no lameness has been observed.

Radiography
Radiographic changes in the proximal plantar metatarsal cortex are more common in hindlimb PSD than with the injury in the forelimb, but they are not a valid basis for diagnosis of active PSD. Sclerosis, attenuated trabecular pattern, and enthesophyte formation in plantar proximal MT III are the most common findings. Presence of radiographic changes suggests a chronic condition, even if onset of lameness was recent. Radiographic abnormalities usually persist unchanged and cannot be used to assess healing.

Nuclear Scintigraphy
Nuclear scintigraphy cannot be relied on to diagnose PSD. Less than one-half of horses with a clinical diagnosis of hindlimb PSD had abnormal radioisotope uptake on either the pool-phase or bone-phase examinations. If increased uptake in the plantar proximal metatarsus is found on a screening bone scan, diagnostic anesthesia should be used to localize pain to the suspensory origin region if the horse is lame. Ultrasound and radiographic evaluation are also indicated.

Magnetic Resonance Imaging
Magnetic resonance imaging (MRI) can identify suspensory ligament desmitis in horses that could not be identified using ultrasonography, radiography, and nuclear scintigraphy. MRI allows evaluation of the ligament itself and the origin on MT III. Cross-sectional images obtained using MRI allow specific evaluation of the ligament size and shape that is not possible with most other imaging modalities. On MRIs, the proximal suspensory ligament has a characteristic shape with the lateral bundle being substantially larger than the medial bundle. The ligament also contains discrete areas of high signal intensity because of the skeletal muscle and loose connective tissue within the ligament. The size and shape of these areas of normal high-signal intensity is relatively consistent between horses and should be bilaterally symmetrical. Because of the common occurrence of bilateral desmitis, compari-
son with the other hindlimb is not always as useful as comparison with normal ligaments. Injury to the proximal suspensory ligament can lead to enlargement of one or both bundles of the suspensory ligament and an overall shape change to a more round cross-sectional appearance (Fig. 1). Desmitis also results in an increased amount of high-signal intensity within the ligament, particularly on fat suppressed images. Fat suppressed images are also useful for detecting damage to the origin of the suspensory ligament on MT III. Medullary bone is normally of low signal intensity on fat suppressed images. When the origin of the ligament into bone is damaged, there can be abnormal high signal intensity within the medullary cavity on fat suppressed images. This abnormality is indicative of inflammation within the bone at the origin. These changes within the bone frequently cannot be identified on radiographs, although changes within the bone may be identified with nuclear scintigraphy in a portion of the cases. Although substantial damage to the suspensory ligament may be identified with ultrasonography, subtle changes can only be identified using MRI. Use of MRI to evaluate the area of the proximal suspensory ligament should be considered when a definitive diagnosis will change the treatment and it cannot be made with radiographs and ultrasound. A definitive diagnosis will change the treatment in most cases, because treating an injury to the suspensory ligament is usually different than treating for inflammation of the tarsal sheath, distal tarsal joints, and other potential diagnoses. Although the best treatment for each possible diagnosis is currently debatable, an accurate diagnosis is necessary to scientifically compare treatments.

**Treatment**

Historically, prognosis for horses with PSD of the hindlimb has been guarded to poor. Resolution of lameness and evidence of ultrasonographic healing has been extremely slow. Recurrence of lameness on return to work was high, even after prolonged rest and rehabilitation programs. In a review of 42 cases, only 14% of the horses had resolution of lameness and successfully returned to work for at least 1 yr without recurrence. In a survey of 22 U.S. practices, recurrence of injury was estimated to be 65%. Acute injuries have had a much better outcome than cases with chronic lameness. The primary treatment in these reports was prolonged rest and progressive controlled exercise with healing monitored by serial ultrasound scans. With rest as the primary treatment, the average healing time was >14

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Fig. 1. (A) Transverse proton-density image of a normal hindlimb 3 cm distal to the tarsometatarsal joint. The bundles of the suspensory ligament are normally asymmetric in size and contain discrete areas of high signal intensity. (B) Transverse proton density image of a horse with desmitis of the proximal suspensory ligament. There is enlargement of the ligament with an overall “rounding” of the bundles. There is also an increased amount of high signal intensity within the suspensory ligament.
mo. Intralesional or periligamentous injections with corticosteroids, hyaluronate, polysulfated glycosaminoglycan (PSGAG), or counterirritants have not proven to be of benefit in cases with demonstrable structural damage.

Extracorporeal shockwave therapy (ESWT) seems to reduce healing time and improve prognosis. In a retrospective study on chronic or recurrent cases of hindlimb PSD, 41% of horses became sound and returned to full work within 6 mo of three treatments with radial pressure wave therapy (RPWT). The authors concluded that RPWT significantly improved the prognosis for hindlimb PSD. Another study evaluated the effects of focused ESWT on 10 horses with collagenase induced suspensory desmitis of both hindlimbs. One hindlimb was treated three times with focused ESWT, and the opposite hindlimb was untreated. The treated suspensory ligaments showed significant reduction in size of the lesion and suspensory body on serial ultrasound scans. Biopsies at week 14 showed more new collagen fibrils and greater expression of transforming growth factor beta-1 in the ESWT treated ligaments. Although the only two published reports of ESWT for hindlimb PSD are encouraging, many practitioners who successfully employ ESWT for treatment of forelimb PSD have experienced frustration in treating hindlimb PSD.

The poor healing associated with hindlimb PSD was initially attributed to chronic accumulation of subclinical damage that had finally progressed to severe damage by the time that the horse displayed lameness. An additional mechanism was proposed by Dyson10 after post-mortem examinations were performed on eight horses with non-responding hindlimb PSD. Gross enlargement of the suspensory ligament, thickening of the plantar fascia, and evidence of compression of the plantar metatarsal nerves were found. Histologic changes included fibrosis, chondroid metaplasia, and hyalinization of collagen. The changes supported the theory that a local compartment syndrome can result from enlargement of the suspensory origin in the hindlimb. The plausibility of a compartment syndrome has prompted many clinicians to surgical treatment by longitudinal incision of the deep fascia that covers the plantar surface of the suspensory origin (fasciotomy). Multiple anecdotal reports suggest that fasciotomy markedly improves the recovery rate.

Tibial neurectomy was mentioned by Dyson10 as being successful in returning three horses to full performance. Compression of the plantar metatarsal nerves or entrapment by periligamentous fibrosis could conceivably result in persistent lameness. A surgical technique that combined fasciotomy with neurectomy of the deep branch of the lateral plantar nerve was presented by Bathe11 in 2003. Results of the neurectomy/fasciotomy on 20 chronic cases that had not responded to rest were encouraging. Nineteen of twenty returned to previous work without lameness, and only two cases had re-injury. We have experienced similar results since adopting this treatment. Rare cases of severe tearing of the suspensory origin and fetlock hyperextension have occurred early in the post-operative period. This complication may be minimized if the neurectomy is omitted in cases that have evidence of severe fiber damage on ultrasound examination or that display abnormal fetlock hyperextension before or after subarticular anesthesia. Average post-operative recovery period before full work is 4 mo. The recovery time and exercise protocol should be adjusted according to the severity of damage and progress in healing.

Intralesional therapy with a number of agents has shown promise in stimulation of tendon and ligament healing. The goal is complete restoration of tissue strength and elasticity by maximizing the rate and quality of healing. The treatments are designed to improve healing by adding precursor cells that can differentiate into fibroblasts, by supplying growth factors and cytokines to attract and stimulate fibroblasts, or by providing an internal scaffold for migration of fibroblasts. Autogenous, adipose derived adult stem cells are being used for a number of orthopedic conditions with reports of success. A protocol for harvesting, isolating, multiplying, and re-injecting bone marrow derived stem cells has shown promise in the United Kingdom, and is being investigated in the United States. This procedure should produce a larger number of viable stem cells than that harvested from adipose tissue, but it has a disadvantage of a 2- to 3-wk delay to allow for the cells to expand in culture. Autogenous bone marrow injection has been used widely in the United States for proximal suspensory desmitis, but the success rate on hindlimb cases has not been reported. Only a very low number of mesenchymal stem cells are harvested by this procedure, but growth factors contained in the bone marrow aspirate may also contribute to healing. A fasciotomy is often performed in combination with the bone marrow injection. Intralesional injection of a particulate formulation of a porcine urinary bladder matrix product has been used to treat suspensory ligament and flexor tendon injuries by many clinicians in recent years. The mixture of proteins provides a biodegradable scaffold to support migration and activity of fibroblasts. Little scientific data and no specific results on hindlimb PSD are available. The product is currently not commercially available. Some questions about adverse reactions and liability concerns have been raised. Intralesional therapy with growth factors such as insulin-like growth factor and platelet derived growth factor have been shown to benefit healing of induced tendon damage, but no results on suspensory origin injuries are available. Hopefully, future research and clinical trials will help define the best combination of these treatment modalities. At this time, our approach to treating PSD in the hindlimb is determined by the severity of the injury. In acute cases with minimal ultrasound
abnormalities, a combination of rest, focused ESWT, and local and systemic anti-inflammatory therapy can be used. In chronic cases and acute cases that fail to respond quickly to non-surgical therapy, the combined fasciotomy/neurectomy is recommended. If severe ultrasound damage and/or fetlock drop is seen, the neurectomy is omitted, and intrasional stem cell or platelet-rich plasma injection is performed along with the fasciotomy.

2. Conditions of the Tarsal Sheath

Applied Anatomy
The tarsal sheath is the tendon sheath of the lateral digital flexor tendon that courses along the plantar aspect of the tarsus. The lateral digital flexor tendon (LDFT) is the proper anatomic term for the proximal portion of the DDFT that passes along the planar aspect of the tarsus and proximal metatarsus. The LDFT changes direction as it passes distally through the tarsal canal, which is formed by the sustentaculum tali of the calcaneus dorsally, the tuber calcaneus laterally, and the flexor retinaculum plantar and medially. The thin medial digital flexor tendon courses along the medial aspect of the tarsus in its own separate tendon sheath and then merges with the large LDFT ~3 cm distal to the tarsometatarsal joint to form the DDFT. The tarsal sheath, ~18 cm long, starts at the musculotendinous junction of the LDFT at the distal caudal crus and ends in the proximal one-third of the metatarsus. The large proximal pouch of the tarsal sheath is between the common calcaneal tendon and tibia. The generic term for tarsal sheath distension is thoroughpin. Fluid distension of the tarsal sheath is most evident on the lateral aspect of the proximal pouch and to a lesser extent, on the medial aspect. Distension of the tarsal sheath must be distinguished from tarsocrural joint distension. The plantar pouches of the tarsocrural joint are located distal and slightly dorsal to the proximal pouch of the tarsal sheath. If the tarsocrural joint is distended, fluid can be moved from the plantar synovial pouches to the dorsal pouches by manual compression. Increased distension in the dorsal joint pouches can be seen and felt.

Idiopathic Thoroughpin
Mild to moderate idiopathic effusion of the tarsal sheath of one or both hindlimbs is not uncommon and usually not associated with lameness. The distension often decreases with exercise and worsens with shipping or stall confinement. Effusion will often resolve spontaneously. Treatment is unnecessary if no lameness is present. Drainage through needle centesis and injection of anti-inflammatory medications for cosmesis may provide only temporary resolution and carries slight risk of iatrogenic infection or intrathecal hemorrhage.

Traumatic Tarsal Tenosynovitis
Acute onset of moderate to severe effusion of the tarsal sheath with inflammation and lameness can result from direct external trauma to the sustentaculum or from injury of the LDFT or tendon sheath by trauma or stretching. The medial edge of the sustentaculum tali is a prominent structure on the medial contour of the tarsus and is prone to fracture from direct trauma. Pain results from movement of the fragments because of compression by the LDFT or distraction by the fibrous retinaculum attachments. The LDFT is unaffected if bone damage is confined to the medial edge of the sustentaculum, but larger fractures can disrupt the fibrocartilagenous gliding surface. Intrathecal hemorrhage from synovial trauma induces marked inflammatory response, and it can lead to formation of restrictive intrasynovial adhesions.

Signs of skin trauma are often absent. Lameness associated with tarsal-sheath injuries varies in severity but is worsened by hock-flexion test. More severe cases manifest pain on palpation and with attempted flexion of the hock. The horse may walk on its toe to avoid loading the LDFT. Radiographic examination is indicated to rule out sustentaculum fracture. The dorsomedial-plantarolateral oblique and flexed skyline views are essential to evaluate the sustentaculum. Ultrasound evaluation of the tarsal sheath requires expertise and high-resolution equipment. The LDFT and the planar aspect of the sustentaculum are imaged from the plantar medial surface of the tarsal sheath, which is aided by sharply debulking the chestnut flush with the skin surface. Lesions of the LDFT usually occur on its dorsal surface adjacent to the sustentaculum. Normal ultrasound appearance does not rule out superficial fraying or longitudinal tears, which are the most common lesions. The thickness and integrity of the fibrocartilage and bony surface of the sustentaculum can be evaluated sonographically. The synovial lining should be evaluated for thickening, adhesions to the LDFT, mineralization, and fibrous masses. The appearance of the synovial fluid is assessed in the distended proximal pouch of the tarsal sheath. Homogenous echogenic swirling fluid is characteristic of recent hemorrhage. More chronic hemorrhage can be difficult to distinguish from sepsis, because both can manifest as hypoechoic fluid with admixed hyperechoic fibrin strands, clots, and sheets adhered to the synovium. Synoviocentesis and fluid analysis is helpful to distinguish hemorrhage from sepsis. With either condition, obtaining a sample can be difficult because of needle occlusion by congested synovial lining, blood clots, or fibrin.

Treatment of acute tenosynovitis should include rest, non-steroidal anti-inflammatory drug (NSAID) therapy, and cold therapy if no bone or tendon damage is found. Intrathecal injection of hyaluronan and short-acting corticosteroid should help resolve...
acute synovitis, but corticosteroids should be used with caution if tendon damage is found. Management of intrasynovial hemorrhage is a dilemma. Fresh hemorrhage without evidence of clots or fibrin may best be managed by needle drainage and lavage to reduce inflammation and development of adhesions. However, centesis drainage and lavage may cause additional hemorrhage. If inflammation and lameness do not steadily improve or if bone or tendon damage is suspected, tenoscopy of the tarsal sheath is indicated. Tenoscopy allows direct assessment of the entire tarsal sheath through a single arthroscopic portal. Advantages of tenoscopic approach to evaluate and treat many conditions of the digital flexor sheath and tarsal sheath have been documented. The tarsal sheath is roomier and less complex than the digital flexor sheath. Clots and debris can only be effectively removed through tenoscopy. The gliding surfaces of the LDFT and sustentaculum can be evaluated and debrided. Synovial masses and fibrous adhesions can be resected. Fragmentation of the sustentaculum is best managed through tenoscopic fragment removal and fracture site debridement. If the fragments are outside the retinaculum on the medial aspect of the sustentaculum, they can be removed by making a separate extrasynovial incision directly over the fragment. If no significant damage to the LDFT or fibrocartilage is found, prognosis for soundness is good with adequate rest. Prognosis is guarded to poor with tendon damage.

Chronic Tarsal Tenosynovitis
In chronic cases, effusion and lameness persist and are accompanied by progressive fibrous thickening of the tarsal sheath. Ultrasound examination may reveal LDFT damage, adhesions, fibrous masses, adhesions, or mineralization associated with the synovial lining. Adhesions from the synovial membrane to the LDFT most commonly develop in the proximal pouch secondary to recurrent intrasynovial hemorrhage or sepsis. Radiographic exam can reveal flexor surface irregularity or marginal osteophyte formation of the sustentaculum or osteitis of the medial aspect of the tuber calcaneus. If no significant pathology is found on ultrasound or radiographic exams, treatment by fluid drainage and intra-synovial injection with hyaluronan and corticosteroid can be tried. If injection provides incomplete or temporary relief, the tarsal sheath and associated structures should be more thoroughly investigated by MRI or tenoscopy. Intrasynovial adhesions, fibrous masses, and ossicles can be best evaluated and resected using tenoscopic visualization. Repeated injections of corticosteroid has been associated with LDFT rupture, synovial mineralization, or ossicle formation.

Septic Tenosynovitis
Penetrating wounds or iatrogenic contamination from intrasynovial injection can lead to infectious tenosynovitis. Sepsis of the tendon sheath is characterized by severe lameness, cellulitis, and suppurative effusion. The common site of trauma and penetration is along the medial edge of the sustentaculum or through the adjacent flexor retinaculum. Iatrogenic infection may not manifest clinically until 2–3 wk post-injection because of suppression of inflammation by the medications injected. Synovio-centesis is useful to confirm suppurative inflammation and for culture and antimicrobial sensitivity testing. Radiography and ultrasound imaging should be performed to identify fragmentation, osteomyelitis, and tendon pathology. In some cases, osteomyelitis or bone necrosis may not manifest radiographically for 14–21 days. Treatment should include aggressive antimicrobial therapy, surgical removal of fragments and devitalized tissue, and copious lavage of the entire length of the tarsal sheath. We believe debridement and lavage is best accomplished through tenoscopy. The entire sheath can be inspected, and fragments, frayed soft tissues, fibrin, and foreign bodies can be removed. The penetrating wound works well as the scope portal if it is adjacent to the sustentaculum. The portals can be left open and used for subsequent standing lavage in chronic cases. The main limitation to tenoscopic debridement is limited exposure of the medial aspect of the sustentaculum. This area is best approached through a medial incision directly over the damaged bone. IV regional antibiotic perfusion is well tolerated in most standing, well-sedated patients. We commonly perform regional perfusions on post-operative days 1, 3, and 5, and we believe that it has resulted in shortened hospitalization stays and improved outcomes. Degeneration of the LDFT, fibrocartilage, or calcaneus from severe trauma or delayed treatment of sepsis warrants a guarded to poor prognosis. Tenectomy of the entire intrasynovial portion of the LDFT or tenotomy of the DDFT at the mid-metatarsal level has been successful as a salvage procedure in cases that remain severely lame.

False Thoroughpin
Development of a fluctuant or firm swelling exterior to the tarsal sheath on the caudolateral aspect of the distal crus is called a false thoroughpin. Causes are variable and poorly understood. Most cases are unilateral. The swelling can be a synovial lined cyst, sometimes containing a pedunculated fibrous mass. These structures might originate from a rupture or herniation of the tarsal sheath, calcaneus bursa, or gastrocnemius bursa. In other cases, the mass appears to be an organizing hematoma, most likely from repeated episodes of hemorrhage. A false thoroughpin can be a cause of lameness or merely a cosmetic blemish. Unlike tarsal tenosynovitis, the swelling is usually confined to the lateral aspect of the distal crus and cannot be balloted to the medial pouch or distomedial portion of the tarsal sheath. Ultrasound can reveal the interior architecture of the mass and its relationship to the tarsal sheath and adjacent structures. Positive contrast
radiography can be used to rule out communication with the tarsal sheath and calcaneal bursa. If present, lameness can be characterized by decreased weight bearing or a mechanical restriction of hock flexion. Treatment is unnecessary if no gait deficit is present. If lameness is present, rest and anti-inflammatory administration should be tried. If rest is unsuccessful, surgical excision of the mass and associated adhesions can improve the lameness. Residual cosmetic blemish is likely, regardless of treatment.21

3. Calcaneal Bursitis

The proximal plantar aspect of the tuber calcis is associated with two to three bursae. The calcaneal bursa (intertendinous calcaneal bursa) is dorsal to the SDFT and plantar to the gastrocnemius tendon, tuber calcis, and long plantar ligament. This large bursa is ~10 cm long and 5 cm wide with medial and lateral outpouchings located proximal and distal to the retinaculum that attaches the SDFT to the medial and lateral sides of the calcaneus at the point of the hock. The smaller gastrocnemius bursa (subtendonous calcaneal bursa) is between the gastrocnemius tendon and the tuber calcis and communicates laterally with the calcaneal bursa in many horses. An acquired SC bursa may develop over the point of the hock (SC calcanean bursa) in response to repetitive trauma such as stall kicking. Accumulation of fluid in an acquired SC bursa is commonly referred to as capped hock. Lameness associated with a capped hock is absent or transient unless the SDFT is damaged or the acquired bursa becomes septic. The fluid can be aseptically drained for cosmesis, but pressure bandaging of the hock becomes septic. The swelling and effusion will generally resolve if the cause of trauma can be eliminated. If the SC bursa is infected, it should be lanced and flushed. Placement of a Penrose drain and systemic antibiotic administration are helpful.

Calcaneal bursitis involves the deeper calcaneal bursa and possibly, the gastrocnemius bursa. Effusion and/or synovial thickening of the calcaneal bursa manifests as enlargement of the lateromedial dimension of the point of the hock that is most apparent from behind the horse. Non-septic bursitis can result from blunt trauma, resulting in primary bursitis or more commonly, secondary bursitis from damage to the gastrocnemius ligament insertion, tuber calcis, SDFT, or plantar ligament origin. Associated lameness is usually moderate to severe and is increased by flexion of the hock. Radiographic and ultrasound imaging should be performed to assess bone and soft tissue damage. Synoviectomies may be needed to rule out sepsis. Treatment consists of rest and systemic anti-inflammatory administration. Intra-bursal injection of hyaluronan and a short-acting corticosteroid may be beneficial if significant non-suppurative effusion is present. Tendonitis may be helped by ESWT, and healing is monitored by serial ultrasound evaluation. Gastrocnemius tendonitis requires a prolonged rest period and has been associated with high recurrence of lameness.19 Cyst-like osteolysis of the tuber calcis has been attributed to damage at the gastrocnemius tendon insertion, and it also has a guarded prognosis for soundness.22 Endoscopy of the calcaneal bursa is helpful to diagnose and debride surface damage to the gastrocnemius tendon, SDFT, or plantar ligament that is not apparent ultrasonographically.23

Septic calcaneal bursitis is a serious condition with a guarded prognosis.24 Early diagnosis and aggressive treatment is critical. Involvement of the calcaneal bursa should be investigated with any penetrating wound to the region of the tuber calcis. Kick wounds are commonly reported, but the most common cause of calcaneal bursa infection in our practice is a deep abrasion to the lateral aspect of the calcaneus sustained when a horse gets loose onto a paved or graveled surface, loses footing, and slides onto its side when trying to turn a corner. Synoviocentesis and fluid sample collection is difficult in recent cases with an open, draining bursa. Communication of the wound with the calcaneal bursa can be confirmed by ultrasonography or contrast radiography. Lameness may be mild if the bursa remains open, but if infection persists, it will worsen dramatically as the wound closes. Surgical debridement and copious lavage of the wound and involved structures should be performed as soon as bursal infection is suspected. An endoscopic approach allows the best visualization and minimizes potential complications from wound dehiscence or iatrogenic damage to the retinaculum.23 However, large penetrating wounds to the bursa can prevent adequate fluid distension for endoscopic examination.24 Broad spectrum antibiotics are administered systemically for a minimum of 3 wk with antibiotic changes dictated by culture results and clinical response. Bony involvement of the tuber calcis significantly worsens the prognosis, but it may not be evident radiographically for up to 2 wk post-injury. A flexed skyline view of the calcaneus is very helpful, but it is often difficult to obtain because of pain on hock flexion. Follow-up radiographs are essential if clinical response is unsatisfactory. Osteitis may be evidenced by a roughened bone surface on ultrasound exam before it is visible radiographically. We believe that repeated post-operative regional antibiotic perfusions have significantly improved our results. One cause of poor response may be extension of infection into the gastrocnemius bursa and sequestration of bacteria.

4. Curb

Curb is defined as desmitis of the long plantar tarsal ligament, which results in swelling of the plantar distal aspect of the tarsus.25 The long plantar ligament attaches to the plantar aspect of the calca-
neus, fourth tarsal bone, and MT IV. With the advent of ultrasound evaluation of soft tissue damage, clinicians have learned that the characteristic “curb” appearance often results from enlargement of other structures besides the plantar ligament, including the SDFT and thin peritendinous and periligamentous fascia. Enlargement of the proximal end of MT IV can also produce a similar appearance. Radiographic and ultrasound examination are essential to accurately identify the structure(s) involved, evaluate severity of damage, and develop a therapeutic plan.

Desmitis of the Plantar Ligament

Plantar ligament injury usually results from excessive strain, but it can also be caused by direct trauma. Disciplines that involve explosive acceleration, pulling, or jumping can pre-dispose to curb. Sickle-hock conformation imposes added stress to the plantar ligament. Horses with tarsal misalignment from neonatal tarsal crush syndrome have a curb deformity and are at greater risk of developing curb because of abnormal strain on the plantar ligament. Risk of reinjury of the plantar ligament is much greater in horses with weak tarsal conformation. Gait deficit secondary to curb can vary from subtly performance limiting to severe. In the acute stage, the swelling is warm, moderately soft, and tender to palpation. As the injury heals, the swelling becomes firm, and other signs of inflammation subside. Ultrasound evaluation of the entire length of the plantar ligament as well as adjacent structures should be performed. Damage to the plantar ligament usually manifests as diffuse enlargement with normal to mildly hypoechoic fiber pattern. Shape, size, and fiber density can be assessed by comparing measurements with those made at identical sites on the opposite hindlimb. Acute inflammation is managed by cold therapy and anti-inflammatories. The horse should be taken out of work for 60–120 days to allow the ligament to heal. The horse should be confined with a slowly progressive controlled exercise program. Results of ESWT for curb have not been reported, but empirically, the treatment seems to speed healing and resolution of lameness. Healing can be assessed with follow-up ultrasound scans if fiber damage is apparent. Some degree of firm, non-painful enlargement will usually persist. The potential role of chronic plantar desmitis in a horse with lameness or decreased performance should not be discounted, especially if hock conformation is weak. After pain in the lower limb, subtarsal region, and tarsal joints has been ruled out by diagnostic analgesia, the plantar ligament can be desensitized by SC infiltration of 20–30 ml of local anesthetic along the lateral, plantar, and medial aspects of the curb. Alternatively, a tibial nerve block can be performed but is less specific. Treatments for chronic plantar desmitis have included pin firing, cryotherapy, topical counterirritants, SC injection of corticosteroids or iodine, or ESWT with no treatment producing consistent results.

Superficial Digital Flexor Tendonitis

Damage to the superficial digital flexor tendon at the distal tarsal region is a common cause of curb-like swelling. The proximity of the SDFT to the plantar ligament and concurrent peritendinous or periligamentous swelling makes diagnosis difficult without ultrasound imaging. Severity and duration of lameness associated with SDFT injury is variable. On ultrasound examination, the SDFT is enlarged, with focal or diffuse hypoechoic fiber pattern. Peritendinous edema is present acutely, progressing to fibrosis in chronic cases. Aggressive anti-inflammatory treatment in the early phase is paramount to reduce swelling and vascular compromise. Cold therapy, NSAID administration, and topical anti-inflammatory treatment is indicated but must be accompanied by rest. Peritendinous corticosteroid injection is probably not advisable because of potential long-term delay of tissue repair. Prognosis and healing time vary with the area and length of tendon damaged. If the damage is confined to the plantar hock region, the injury generally heals more rapidly and with less recurrence than injuries to the same tendon on the forelimb. ESWT seems to speed healing and improve swelling. Limited tendon splitting may be indicated for anechoic core lesions. Other intralesional therapies such as stem cells, platelet rich plasma, and insulin-like growth factor should have merit in more severe cases.

Peritendonous and Periligamentous Inflammation

SC soft tissue edema is nearly always present with acute curb-like swelling, often without apparent damage to the plantar ligament or flexor tendons. The cause of the swelling is usually unknown. If no evidence of skin trauma is found, the swelling is likely from stretching or tearing of the SC plantar fascia and/or early, mild strain of the plantar ligament or SDFT. MRI is more sensitive than ultrasound, and it could provide additional information about the condition of all of the structures. Because the fascia is the most plantar structure and is thin, it may be the first tissue in progression to be injured. For this reason, rest is warranted to allow healing and prevent more severe injury. Corticosteroid injection or pin firing have been used with success, but should be combined with 3–4 wk of rest or until swelling and lameness have resolved. The owner or trainer should be educated to closely monitor for recurrence of inflammation to avoid more serious structural damage.

References and Footnotes


*Benoit P. Personal communication, 2005.

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