

Traumatic and Developmental Lesions of the Tarsus

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Traumatic and developmental lesions of the tarsus require treatment tailored to the peculiarities of the individual lesions. Surgical treatment is the treatment of choice for many traumatic and developmental lesions. This is especially true if the lesions involve the tarsal articulation, which is where degenerative joint disease is likely to result. Author's address: Rood and Riddle Equine Hospital, PO Box 12070, Lexington, KY 40580; e-mail: lbramlage@roodandriddle.com. © 2006 AAEP.

1. Introduction

Angular limb deformities of the tarsus are either valgus or varus. In tarsal-valgus deformities, the limb distal to the tarsus deviates laterally. In tarsal-varus deformities, the limb distal to the tarsal deviates medially. The deviation most commonly is at the level of the distal-tibial physis just above the tarsal joints. More correctly, it should be called distal-tibial valgus or varus; however, it is common to refer to all deformities in the area as "tarsal." Deviations through the tarsal joints can occur as a result of the crushing and/or malformation of the small tarsal bones, which is a much more serious condition. Mild distal-tibial angular deviations do not affect performance. Severe deviations can predispose the horse to abnormal wear and tear or directly interfere with performance. The diagnosis is confirmed radiographically, but a diagnostic exam that includes a visual examination of the conformation of the limb is most accurate.

The etiology of the deviation is normally developmental, although it can be traumatic when physiologic overload of the limb is a result of an injury to

the opposite limb. Treatment is based on the site and severity of the deformity, but in routine distal-tibial valgus or varus, periosteal transection and/or transphyseal bridging at the appropriate location on the distal tibia are the treatment options. Surgical correction of growth deformities in the distal tibia can resolve the problem if treated when there is sufficient growth left to accomplish correction; therefore, the prognosis is favorable for effective correction in young animals.

2. Malformation of the Tarsal Bones

Crushing or malformation of the tarsal bones can result in angular deformities, but malformation of the tarsal bones is even more significant because of its effect on the athletic soundness of the tarsal joints. Even mild asymmetry of the tarsal bones pre-disposes the articular surfaces of the heavily loaded tarsal joints to accelerated wear and tear. Malformation or misshaping of the tarsal bones results from the overload of immature cartilage precursors of the cuboidal bones; this causes malformation and then calcification of the cartilage precursors of the bones of the tarsus into an abnor-

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mal shape. In dysmature foals, the cartilage calcification is delayed, and the cartilage models, which should be fully calcified at birth, are in various stages of delayed bone formation. The retarded calcification makes the bones weaker than the bones of the normal newborn and vulnerable to overload from even normal exercise. Lack of soft-tissue tone and “sickle-hocked” conformation, common in dysmature foals, increases the load on the cranial aspect of the cuboidal bones and increases the chance for overload of the bones and malformation or fracture.

There is no good treatment for fracture or malformation of the tarsal bones. All efforts must be directed at prevention. After the bone precursors are damaged, the deranged anatomy is permanent, and the calcification adopts the malformed anatomy. The delayed calcification must be identified in the clinical examination, and the tarsus should be radiographed to assess the degree of maturity of the tarsal bones. Exercise must be restricted in dysmature foals to prevent injury to the cartilage templates before they calcify and fully develop. Exercise must be restricted to an area that does not allow the mare to gallop and subsequently, force the foal to “keep up” until the cuboidal bones have calcified and the foal is more mature.

For foals with plantar soft-tissue laxity, frequent episodes of walking exercise are helpful in strengthening the soft tissues, but no free-choice exercise should be allowed because of the danger of permanent derangement. After the cartilage models of the tarsal bones are fully calcified, stepwise increases in exercise using gradually increasing turnout areas and duration are indicated.

Various forms of treatment have been tried for cuboidal-bone injury/malformation. Most treatment efforts involve various forms of splinting and/or casting of the limb. None have been successful in restoring an articular surface that is functional for high-speed athletic activity. Therefore, all efforts must be directed at prevention of the problem, because treatment is generally futile.

The derangement or injury of the cuboidal bones does not prevent the horse from leading a normal life as a brood animal; however, it does preclude normal athletic activity, because it pre-disposes the horse to development of degenerative arthritis and lameness. In rare instances, complete degeneration and fusion of the lower tarsal joints will occur, but that is the exception. Normally, the horse’s athletic career is permanently limited.

3. Osteochondrosis–Osteochondritis Dessicans

Osteochondrosis has become the commonly accepted term for developmental abnormalities of the articular surface of growing bones. When the developmental abnormality is anatomically deranged and a “flap” or fragment of joint surface develops, the term osteochondritis dessicans (OCD) applies. The three most common sites of osteochondrosis in the

tarsus are (1) the distal-intermediate ridge of the tibia, (2) the distal-medial malleolus of the tibia, and (3) the distal-lateral trochlear ridge of the talus. The OCD lesions of the tarsi are normally dessicans lesions, because they normally form separated fragments.^{1–4} These fragments cause inflammation of the underlying parent bone, which results in inflammatory debris being shed into the joint.

The presence of a lesion of osteochondrosis causes synovial effusion within the articulation by means of physical debris shedding and/or hemorrhage and exit of biologically active compounds from the interface of the malformed portion of the articular surface with the parent bone into the joint. This continuous shedding of physical and biochemical substances into the joint causes synovial effusion or “bog spavin,” chronic distention, and resultant interference with performance. In routine tarsal osteochondrosis, it is most often the synovial effusion, which restricts flexion and extension, that causes the interference with performance. However, degenerative arthritis caused by damage of the articular surface would apply equally to the tarsal-articular surfaces as it would to any joint if the debris shedding were prolonged enough to do permanent damage to the articular cartilage.

Diagnosis of osteochondrosis of the tarsal joints is made radiographically. The examination is done with routine views for examination of the tarsus. The etiology of the disease is multi-factorial. Supra-physiologic loads on normal bone or physiologic loads on abnormal bone disrupt normal bone growth. Most commonly in the tarsus, the insult is an abnormally large physiologic load on a normally developing joint surface; this results in disturbance of bone growth and osteochondrosis-fragment formation. The treatment for osteochondrosis of the tarsal joints is arthroscopic removal of the damaged area of bone. The removal of the unstable articular surface (the dessicans lesion) stops the inflammatory response of the parent bone to the unstable fragment and stops the debris shedding into the joint. If the surgical removal occurs before permanent damage to the articular cartilage, the prognosis is excellent for resolution of the synovial effusion and restoration of normality to the joint.^{3,4} There are virtually no lesions of the tarsus that involve an important articular surface that surgical removal cannot resolve. Most of the osteochondrosis lesions, fortunately, involve the margins of the joint that are not heavily loaded during exercise. Therefore, the lesions can be removed, and the joint surface involved can be sacrificed in the interest of prevention of inflammatory degeneration of the joint.

There are other sites of OCD or fragmentation that occur in the tarsus, such as the distal-medial trochlear ridge. However, this lesion in the distal-medial trochlear ridge area is within the synovial attachment of the tibial-tarsal joint to the distal aspect of the talus and therefore, does not shed

debris within the joint. This lesion rarely requires removal unless it is large enough to shed debris through the damaged synovial lining.

Other OCD sites can occur. The posterior-medial trochlear ridge will occasionally be a site of OCD formation. In these other sites, the guidelines of treatment, including surgical removal through an arthroscopic approach, apply in the same way as to the more common locations.

4. Traumatic Injuries

Traumatic luxations rarely occur in the tibio-tarsal joint. If they do occur, they are accompanied by such severe trauma and the soft-tissue structures and/or fractures of the bone that accompany the luxation are so disabling that salvage of the horse is virtually impossible. Fortunately, they are rare and therefore, are seldom indications for treatment or clinical intervention. Traumatic luxations of the distal-tarsal joints, however, are more common, although they generally occur infrequently. When a luxation of the tarsus does occur, it usually occurs through one of the distal-tarsal joints. Luxation through the tarsal-metatarsal, proximal, or distal intertarsal joints is athletically disabling.

The diagnosis is normally not difficult clinically and is substantiated through radiographs. Treatment requires stabilization in some fashion, usually surgical and possibly with an accompanying cast, to enable the horse to reestablish functional weight bearing. Surgical arthrodesis and stabilization of the luxated limb is aimed at preservation of breeding animals. The prognosis is guarded, because two common complications occur with this injury. Vascular disruption of the arterial supply to the distal limb is an occasional complication as is the more frequent laminitis of the opposite limb during the treatment period.

Partial luxations with fracture of the proximal metatarsi is a consistent fracture pattern seen when horses kick solid structures with a significant amount of force. The kick impact bends the plantar metatarsi without the support of the flexor structures that normally present during weight bearing. The kick by the horse to the solid object usually fractures the metatarsi approximately one-third of the way down the plantar cortex. The fracture then propagates proximally and obliquely into the tarsal-metatarsal joint. Stabilization is required, usually with implants and a cast. The objective of treatment is usually to retain the animal for breeding, because the prognosis for return to athletic performance is not favorable.

Less severe fractures of the tarsal joints are more common. The most commonly encountered fracture is the lateral-malleolar fracture. A traumatic force from lateral to medial on the limb fractures the lateral malleolus. This fracture sheds debris within the joint, similar to an osteochondrosis lesion. The diagnosis is made radiographically; however, if one suspects this injury, multiple diagnostic

radiographic views may be necessary to establish the presence of the fracture. Healing of this lesion is nearly impossible, and therefore, removal is preferable for most of the fractures. Occasionally, a fragment will be large and then internal fixation and stabilization of the fracture fragment is necessary, but this is rare. The treatment is arthroscopic removal. Separation of the lateral-malleolus fracture from the ligamentous attachments by careful dissection and extraction of the large fragment is a more difficult procedure than removal of an OCD fragment, but it is possible with careful maneuvering. The prognosis is favorable for athletic activity if one removes the fragments and stops the debris shedding before significant articular-cartilage damage of the rest of the joint occurs. Occasionally, fractures of the medial malleolus or some other site within the tarsus will be encountered. The treatment approach would be similar with all similar traumatic-fracture fragments.

5. Exercise-Induced Fractures

Exercise-induced fractures include the sagittal-talus fracture that fits the category of a stress fracture of the tarsus. These fractures are more commonly seen in the Standardbred racehorse, although they also will be encountered occasionally in other breeds. The diagnosis is suspected from the clinical signs of synovial effusion and significant lameness, and it is confirmed radiographically. Stall rest until the horse becomes sound and then 2 mo of paddock exercise will normally result in resolution of the problem and a favorable outcome.

Tarsal-slab fractures of the central or third tarsal bones will occasionally occur.^{5,6} These fractures are exercise-induced fractures and result from an accumulation of damage with eventual gross fracture. They are normally found on the dorsal and dorsolateral aspects of the bones. They can be suspected when the clinical exam reveals a much more severe response to flexion of one tarsus compared with the opposite tarsus. Slab fractures of the tarsus often have minimal or no edema or synovial effusion accompanying the fracture, unless the fracture enters the proximal-intertarsal joint. The diagnosis can be difficult to establish, because in some instances, the curved nature of the fracture plane makes it difficult to image. Multiple or repeated radiographic sessions may be necessary to identify the presence of the fracture. Surgical treatment consists of lag-screw fixation, but horses with less high-speed exercise demands will return to exercise with stall rest alone.^{6,7} Horses that have heavily loaded tarsal joints and undergo very strenuous activity will likely do better with stabilization. Occasionally, similar fractures are seen in the proximal aspect of the third metatarsus.

6. Collateral-Ligament Desmitis

The short collateral-ligament attachment to the talus is sometimes a site of painful inflammation

in horses with obscure lamenesses accompanied by synovial effusion and minimal radiographic change. Collateral-ligament desmitis is a traumatically induced disease usually caused by exercise, and is often seen when the horse repeatedly exercises over an area with turns of a very small radius. The clinical signs are that of synovial effusion and lameness. The desmitis is performance limiting because of the inflammation of the ligamentous attachment and the pain and lameness that accompanies it. The diagnosis is most commonly made radiographically by identifying the proliferative calcification at the ligamentous insertion on the talus; then, the area can be imaged ultrasonographically for verification of the diagnosis. This lesion, however, most often occurs very near the insertion of the short collateral ligament into the talus, and therefore, the ligament often has minimal ultrasonographic changes. The treatment for the disease is restriction of exercise for 2–3 mo. The prognosis is favorable for resolution after a period of rest.

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