Pathologic Changes and Diagnostic Workup of Palmar/Plantar Metacarpal/Metatarsal Condylar Disease in the Thoroughbred Racehorse

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1. Introduction
The palmar aspect of the third metacarpal condyles (PMC) and the plantar aspect of the third metatarsal condyles (PMT) are common sites of injury in the Thoroughbred racehorse. Several disease processes can occur at these sites including condylar fracture, traumatic arthrosis, and osteoarthritis. The PMC and PMT articulate with the proximal sesamoid bones at maximal extension, causing high pinpoint loading at that site. The dorsal aspect of the condyles also articulate with the proximal aspect of the first phalanx, probably adding to the compressive stress across the condylar surface. The purpose of this presentation is to review the pathologic processes that occur in the PMC and PMT, the clinical techniques used to diagnose such diseases, and the various therapies used for their treatment.

2. Normal Adaptation and Pathologic Processes
The PMC and PMT undergo normal adaptive bone remodeling and modeling to withstand the intense loading of training and racing. At maximal extension, it appears that the bone in the PMC and PMT becomes thickened in the subchondral and trabecular areas between the articulations of the proximal sesamoid bone and proximal first phalanx. However, the quality of bone produced and its integration with parent bone can be influenced by the intensity of training.

Most racehorses do not have PMC or PMT disease, but, probably because of normally adaptive bone, there are indications that some diseases of the PMC and PMT could begin to develop before training. Lucencies and defects within the condyles and parasagittal groove have been seen in samples from young Thoroughbred horses before training. Ar eas of thin and fragmented calcified cartilage with subchondral bone resorption are often present, with areas of hypermineralized matrix protruding into articular cartilage, indicative of pathologic changes during development. The hypermineralized tissue within the calcified cartilage layer may create a density gradient predisposing the area to fracture. Degenerative changes in articular cartilage matrix overlying these areas of subchondral bone changes are also seen.

In horses that have trained and raced, the pathologic changes often progress. Abnormal bone ma-
horses with pain in this area often can have edema. Significant bone density can be apparent. Some even in a horse training without this problem, significant cartilage changes. The articulation between the palmar/planter aspect of the third metacarpal/metatarsal bones and the proximal sesamoid bones undergoes high stress. The proximal sesamoid bones articulate with both the condylar surface and the abaxial surface of the transverse ridge at this site. Consequently, in the young horse, there is good trabecular pattern in both the condylar surface and the abaxial surface of the transverse ridge; however, in the parasagittal groove, a highly remodeled/odeled trabecular pattern is usually apparent. This is a site of relatively low subchondral bone density even in a young, trained horse. It has been shown in young Thoroughbred horses that this is a site of poor articular cartilage integrity and strength and a site of significant subchondral bone density gradient in some horses. Microdamage is often present in fractured joints and at sites predisposed to fracture. The severity of subchondral bone changes appears to be associated with overlying articular cartilage changes.

Condylar fractures typically occur within the parasagittal groove, originating at the palmar/plantar aspect of the third metacarpal/metatarsal condyles. This correlates well with the pathologic changes mentioned above. However, condylar fractures can commonly occur more abaxially on the condylar surface. Therefore, the study of pathologic changes leading to fracture must include changes on the condylar surface. In addition, there may be associated changes on the dorsal aspect of the condyle that are worth investigating.

Factors that can affect the incidence of fracture include the shape of the bone, bone quality, the intensity of the remodeling/modeling cascade (often times affected by training intensity), bone quality, and the shear stresses at the interface between old bone and new remodeled/odeled bone. One must consider that conformation may be involved as a factor in these cases.

Significant articular cartilage erosion and subchondral bone exposure can occur at the PMC and PMT. This has been referred to by several names, including palmar arthrosis, overload arthrosis, and palmar/planter osteochondral disease. Postmortem specimens of this disease often demonstrate significant subchondral bone remodeling/modeling, which can often lead to significant sclerosis at that site. In some cases, the bone mineralization front can enter into and through the non-calcified articular cartilage, leading to erosion. However, even in a horse training without this problem, significant bone density can be apparent. Some horses with pain in this area often can have edema at the site without any structural damage at that point. However, structural damage can occur with progression of this disease. This commonly manifests as subchondral bone microdamage and consequently, necrosis. The necrosis can continue to occur while the articular cartilage is intact. This cascade often leads to subchondral bone modeling, which advances through the calcified cartilage and into the articular cartilage. Once the articular cartilage either collapses into a cyst-like structure or subchondral bone completely models through the cartilage layer, then the horse can become refractory to treatment.

Osteoarthritis (OA) can result from any of the above pathologic processes. In some cases, the origin of OA is idiopathic and the horse typically shows significant erosions, wear lines, osteophytes, and joint collapse, which are typical of OA.

3. Diagnostic Techniques

Depending on the type of injury in the joint, the horse can either show overt signs of clinical disease within the fetlock joint or in more subtle cases the joint may have no outward signs of disease. Horses that show no outward signs of disease other than a history of training sore can be a diagnostic challenge. For disease processes that do not involve the actual synovial environment, there may be no synovial effusion and the horse may be lame and may or may not be positive to digital flexion. Often, these horses will not improve with intra-articular analgesia of the joint; however, a low four-point nerve block often alleviates the lameness. More specifically, diagnostic analgesia of the lateral and medial palmar metacarpal nerves may alleviate the lameness in the forelimb, and a block of the lateral plantar metatarsal nerve may alleviate the lameness in the hind limbs. A full series of radiographs should be performed, and, especially in the Thoroughbred racehorse, radiographic imaging of the palmar/plantar aspect of the joint should also be performed. This entails a 35° dorsodistal-palmaroproximal oblique projection and a flexed 30° plantarodistal-dorsoproximal oblique projection. Downward oblique images (a dorsoproximolateral-palmarodistomedial plantarodistomedial oblique projection and a dorsoproximomedial-palmarodistolateral plantarodistolateral oblique projection) may also be performed to highlight the palmar or plantar aspect of the third metacarpus/metatarsus.

Nuclear scintigraphy often will demonstrate radioisotope uptake in the palmar/plantar aspect of the joints. However, one must remember that in the young Thoroughbred racehorse, there may be significant increase in radioisotope in this area caused by training. However, for horses that have disease at that site, the degree of radioisotope uptake can be significant. Trope et al. found that increased radiopharmaceutical uptake in the PMC and PMT was the most common abnormality in
Thoroughbred racehorses but was not sensitive for screening fracture risk.

Magnetic resonance imaging to characterize disease in the PMC and PMT area is now the gold standard. Powell et al. have demonstrated all of the disease processes characterized in this report. This includes demonstrating subchondral bone edema, sclerosis, necrosis, and fracture. Although a high-field MRI is typically preferred, general anesthetia is needed. However, Powell et al. have demonstrated the use of low field standing MRI to demonstrate these lesions. In a postmortem study, Tranquille et al. found MRI to be sensitive for detecting small incomplete lateral condylar fractures.

Computed tomography has also been used to characterize diseases in the PMC and PMT. Incomplete fractures can be detected as well as subchondral sclerosis and necrosis. Treatment of diseases in the PMC and PMT varies, depending on the disease process present. For condylar fractures, an incomplete or a complete fracture that involves the joint should be surgically reduced through the use of the lag screw technique to give the best prognosis. Arthroscopic visualization of the joint surface is suggested because the prognosis is often determined by articular cartilage damage in the joint. In general, retrospective studies of condylar fracture repair demonstrate that horses with incomplete fractures carry a better prognosis than those with complete fractures and that prognosis diminishes with increasing pathologic change within the joint.

Palmar arthrosis, depending on its severity, often will respond to rest in the early stages; however, with more advanced disease the pain becomes more difficult to control. Intra-articular medication is thought to work in some cases; however, in those horses, a point is reached of which that is no longer effective. Systemic non-steroidal anti-inflammatory drugs can also be used to help control pain in this area.

For osteoarthritis, medical therapy is used to help control pain and disease progression in these areas. Intra-articular stem cell therapy may have some benefit; however, this is still unproven in the metacarpophalangeal/metatarsophalangeal joints.

References


