Osteochondrosis in the Horse
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Introduction

The term “osteochondritis dissecans” was originally proposed in 1887 by physician Franz König to describe joint lesions that resulted in formation of osteochondral fragments in the absence of significant trauma. The term was somewhat of a misnomer, since a primary inflammatory etiology has since been ruled out. This led to the suggestion of the term “osteochondrosis” (and by extension, osteochondrosis dissecans) by Howald in 1942, but the original name has stuck, as has the more common acronym “OCD.” In veterinary medicine, osteochondrosis (OC) is used for any focal abnormality of endochondral ossification affecting the articular-epiphyseal cartilage complex (AECC); if a fragment is present, then osteochondrosis dissecans (OCD) is appropriate. More variable terminology is used in the human clinical literature, where lesions of similar pathophysiology, but at different anatomical locations, are named by the person who first described them (e.g. Theimann’s disease in the fingers/toes, Panner’s disease in the elbow).

OC was traditionally considered to fall under the umbrella term of “developmental orthopedic disease” (DOD). This constellation of syndromes includes physitis, flexural limb deformity, incomplete ossification of cuboidal bones, angular limb deformity, and subchondral bone cysts. Recently, the term “juvenile osteochondral conditions” (JOCC) has been proposed for developmental disorders related to immature joints and bone plates. This slightly more specific term includes OC, cuboidal bone disease, and physitis.

OC is most simply defined as a failure of normal endochondral ossification, the process by which the ends of long bones transform from cartilage to bone. This condition affects individuals of multiple species, notably horse, pig, dog, chicken, cow, and human. Any diarthrodial joint can be affected by this condition, but there are some sites known to be predisposed to disease. In the horse, these include the fetlock, hock, and stifle. Less commonly affected are the cervical vertebrae, shoulder, and hip. Interestingly, some predilection sites are shared across species. Examples of these include (in order of frequency):

- Hock: distal intermediate ridge of the tibia (DIRT), lateral trochlear ridge of the talus (LTR), medial malleolus of the tibia (MM), medial trochlear ridge of the talus (MTR)
- Fetlock: dorsal distal mid-sagittal ridge MC/MTIII, dorsal margin P1
- Stifle: lateral trochlear ridge of the femur (LTR), medial trochlear ridge of the femur (MTR), distal patella, intertrochlear groove

The inclusion of palmar/plantar margin P1 fragments (commonly referred to as palmar/plantar osteochondral fragments [POF]) as manifestations of OC remains controversial, as does the inclusion of subchondral bone cysts (SBC) of the medial femoral condyle. It is not questioned...
that these are developmental disorders; rather, the crux of the debate focuses on whether they have the same pathophysiology as other OC lesions.

Multiple OC lesions in the same horse are not uncommon, although bilateral lesions are more common than lesions in multiple joints. Reports in the literature suggest that ~20% of horses with stifle OC, and 50% or more of horses with hock OC have bilateral lesions. In the fetlock, lesions can be bilateral or affect all four limbs. Multiple lesions within the same joint are particularly common in the hock.

Global prevalence of OC has been estimated at 7% to > 80%, based on radiographic surveys of yearling horses of various breeds. Prevalence varies by site and breed. For example, the average reported prevalence of hock OC lesions in Standardbreds is 14.7%, while in Thoroughbreds it is only 5.3%.

**Suggested Etiology/Pathogenesis and Risk Factors**

The etiology and pathogenesis of OC has long been debated. Historically, there have been two schools of thought, one supporting trauma as the primary underlying cause of OC, and the other supporting other processes, including inflammation, osteonecrosis, cartilage extracellular matrix abnormalities, and vascular abnormalities. Currently, research suggests that while trauma is the key precipitating factor for clinical signs, it cannot account for histologic changes during the course of disease or non-weightbearing predilection sites. The bulk of recent experimental evidence supports a disruption of the vascular supply to the AECC as being the precipitating event for the delay of endochondral ossification that eventually results in OC lesions. However, the precise mechanism by which these vascular abnormalities occur is still unknown, and it is possible that more than one etiologic process is involved. Histologically, the earliest OC lesions (*osteochondrosis latens*) are recognized by focal regions of necrotic cartilage surrounding necrotic blood vessels (cartilage canals). Once the ossification front passes beyond these necrotic islands they are visible radiographically as osseous defects (*osteochondrosis manifesta*). Under many circumstances, this necrotic cartilage heals with no or minimal remnants; however, in the presence of local trauma, a cleft can extend through the overlying articular cartilage into the necrotic epiphyseal cartilage, resulting in the formation of a fissure, flap, or osteochondral fragment (*osteochondrosis dissecans*).

A number of risk factors for OC have been proposed. The most widely studied of these are genetics, nutrition, and exercise; however, others include conformation, biomechanical forces, trauma, stress response, *in utero* environment, and hormonal interactions. It is most likely that a combination of factors play a role; in fact, OC is widely acknowledged to be a complex disease, with both genetic and environmental risk factors.

The role of genetics is supported by the observation that certain lines of horses seem to be predisposed to disease. In studies, up to 70% of foals from a single sire were affected, and offspring of affected sires were twice as likely to be affected as offspring of unaffected sires.
Estimates of heritability for hock OC range from 25-50%, depending on the breed. The specific genes involved in disease risk are currently unknown, but this is an active area of research.

Nutrition has long been suggested as a risk factor for OC, with high dietary energy, high protein, and mineral imbalances most commonly suggested as culprits in anecdotal reports. There is some evidence that larger foals have increased risk of disease, but mixed evidence for a direct connection between increased feed and lesion formation. Studies looking specifically at “overfeeding” protein have not found any negative effects. There is a known link between copper deficiency and severe, multi-focal OC. A study has also shown that foals fed high amounts of phosphorus developed more OC lesions than those on a normal ration; here, it seems that the ratio of calcium to phosphorus is most important (Ca:P 1.3:1). A large study looking at the effects of ration adjustments on OC prevalence did find mild improvement in farm lesion scores in response to decreased feed intake and increased copper intake. The overall conclusion from this body of work is that nutrition can play an important role in disease development, but it interacts with genetics and other factors.

The role of exercise is even less clear-cut than that of nutrition. In a group of foals bred to have a genetic predisposition to OC, those that were stall-rested had greater severity, but not greater numbers, of lesions. Similarly, in a large random survey of foals irregular exercise was associated with a higher risk of OC compared to regular moderate exercise. Based on these findings, it seems likely that exercise plays a modifying role in disease.

At the end of the day, the available research suggests that fairly straightforward recommendations for management can be made in most cases. Foals should be fed a balanced diet, with appropriate mineral ratios and micronutrients, at a level to support consistent moderate growth. Regular, moderate exercise (turnout) on even ground should be encouraged. However, even with the best management, some foals will likely still be affected with OC, due to genetic risk factors.

**Diagnosis, Treatment, and Outcome of OC**

Depending on the breed, there are a number of common scenarios for diagnosis of OC. In racehorses, the condition is commonly recognized in yearlings going to (or at) a sale. In sport horses, it is often diagnosed at a slightly older age (2-4 years), when training commences. Of course, OC may be found incidentally in any horse undergoing a comprehensive pre-purchase exam that includes radiographs. Frequently, OC is diagnosed radiographically in the absence of clinical signs. However, joint effusion (sometimes transient) is not uncommon. Lameness, if present, is generally mild, but may be severe depending on location and size of lesion. Lameness is generally accompanied by effusion and is most common in horses that have just been put into work.

Often, there is a high index of suspicion for OC based on clinical signs combined with appropriate signalment; however, as previously mentioned, a significant number of affected horses may have no clinical signs. Radiographs or fluoroscopy are the most common methods
to diagnose OC lesions, but can result in false negative findings. Ultrasound has been suggested as an alternative modality that may be more sensitive than radiographs for certain lesions (e.g. trochlear ridge of the femur, medial malleolus of the tibia, cervical vertebrae, shoulder, hip). However, arthroscopy is considered the gold standard for diagnosis, and is often recommended even if radiographic evidence for a lesion is equivocal, particularly in the face of clinical signs.

Standard radiographic views are often sufficient for diagnosis, and it is helpful if a fragment can be visualized on more than one view. For specific lesions, special views have been recommended. These include a 30 degree dorsolateral-plantaromedial oblique of the hock for medial malleolus lesions, flexed and/or oblique views of the stifle for trochlear ridge lesions, and a skyline view of the patella. Given the large number of horses with bilateral lesions (particularly in the hocks), it is recommended to take bilateral films even if there is unilateral effusion. There has been some recent debate regarding the appropriate timing of radiographs. If there are concerns about OC because of genetics, clinical signs, etc., it is not a bad idea to screen early (i.e. 6 months of age). However, OC lesions can heal spontaneously, so early intervention is rarely recommended. Radiographic studies suggest that lesions become permanent after 5 months of age in the fetlock and hock and after 8 months of age in the stifle, but this may vary slightly by breed. There are “windows of susceptibility” for each joint related to the timing of epiphyseal cartilage ossification. Any healing that will occur is typically complete by 11 months of age, so radiographs taken at this time or later should be sufficient for making decisions about treatment options.

When evaluating diagnostic imaging findings and determining treatment approach, a number of points should be considered. These include distinguishing between OC and traumatic fragments, and determining the intended use/owner expectations for the horse. Clinical significance of lesions can be difficult to determine; interpretation relies on multiple factors including location, severity, and duration (i.e. how long has it been there) of the lesion, age of the horse, and clinical signs. Based on these, conservative and/or surgical therapy can be considered.

Conservative treatment consists of some combination of rest, dietary modification (i.e. reduce dietary energy), systemic non-steroidal anti-inflammatory drugs, intra-articular (or systemic) hyaluronic acid and/or polysulfated glycosaminoglycans, and oral joint supplements (i.e. chondroitin sulfate). This approach is most likely to be successful in mild disease with minimal effusion and no lameness and/or disease in very young horses.

Arthroscopic removal of fragments with curettage of the fragment bed/abnormal tissue is generally recommended if a fragment or flap is present. Reattachment of selected large osteochondral fragments with PDS pins has been reported, but is not widely performed. Arthroscopic surgery allows for complete evaluation of the joint, which may be particularly important for horses with clinical signs or longstanding disease, since the presence of other joint pathology will affect prognosis. Arthroscopy is also frequently chosen for horses being prepped for sale even if they have no clinical signs, because the presence of an OC lesion can decrease sales price. Early intervention is preferred in the face of effusion, because chronic
effusion may not resolve even with surgical correction of the lesion; this is often a particularly important factor for show horses.

Reports of treatment outcomes for conservative therapy are mixed, and it is likely that outcome depends on the severity and location of the lesion. Lesions that are less likely to do well with a conservative approach are stifle lesions (compared to hock/fetlock), large lesions (e.g. > 15mm), and lesions with osteochondral fragmentation. In two different studies, racehorses performed poorly with conservative treatment compared to non-racehorses (good outcomes in < 40% of racehorses compared to 50-80% of horses competing in other disciplines). In contrast, another study reported equivalent race performance in conservatively-treated Standardbred racehorses compared to unaffected individuals; however, horses with more severe lesions in this study tended to be treated surgically.

In general, surgical intervention for OC has excellent outcomes reported. As for conservative therapy, return to work is better for non-racehorses than for racehorses; in various studies 62-76% of racehorses and 67-83% of non-racehorses returned to their intended work after surgery. Not surprisingly, larger lesions or joints with other pathology are less likely to do well even with surgical intervention. The best timing of surgical intervention has been the subject of some debate in the literature. One study showed that affected racehorses that were treated as 2-year-olds were less likely to race in that year compared to their unaffected siblings, likely due to lost training days. In contrast, another study showed that Standardbred racehorses undergoing surgical intervention prior to yearling sales performed equivalently to related unaffected horses, both as 2-year-olds and over several race seasons. Improvement of lameness and effusion can be expected within the first few weeks after surgery although, as mentioned previously, chronic effusion may not completely resolve.

The question of whether you should breed a horse with OC is extremely contentious, and regulations vary widely among breed organizations. OC does have a strong genetic component, but it is difficult to make breeding decisions when specific major risk genes are unknown. On the other hand, OC does have major environmental risk factors, but optimizing management does not eliminate all disease. Horses with OC can go on to perform at elite levels if treated, but severely affected or untreated horses can have career- or life-limiting consequences to disease. Research in OC is ongoing, and advances are being made in our understanding of pathogenesis and risk factors. New diagnostic approaches are also being developed. In the future, these advances will provide breeders and trainers with additional tools that should help to identify high-risk individuals and reduce the overall prevalence of disease.

Selected references and recommendations for further reading


