Equine Osteochondrosis: Predict the Outcomes for Your Clients

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Osteochondrosis (OC) is a developmental orthopedic condition defined as a disturbance of the process of endochondral ossification. This critical process is responsible for the coordinated cellular differentiation that leads to the formation of cartilage. Any disruption of this sequence of events may result in the retention of poorly differentiated growth cartilage. The structure of the joint surface is weakened by the presence of retained, immature cartilage resulting in necrosis and fissure formation extending to the articular surface. Damage to the articular surface causes inflammation and the term osteochondritis defines this condition. In some cases, a cartilage flap will detach from the underlying bone creating lesions known as osteochondritis dissecans (OCD). A “dissecans” lesion forms commonly as a result of trauma acting on poorly developed cartilage and leads to the formation of a cleft from the articular surface to the subchondral bone. These cartilage flaps have been described as type I lesions and develop at or near the center of a convex joint surface. At some locations, such as the distal intermediate ridge of the distal tibia, abnormally developed cartilage may fracture along the cleft line resulting in the formation of an osteochondral fragment. These are described as type II lesions.

Epiphyseal cartilage is destined to turn to bone through the process of endochondral ossification. This process is accomplished as chondrocytes (cartilage producing cells) progress through four histologic zones to produce the extracellular matrix that defines this specialized tissue. Chondrocytes must progress through the resting, proliferation, hypertrophic, and calcification zones in an orderly fashion to allow normal development and function. If any of the events involved in the process of endochondral ossification are altered to a significant degree, OC can occur. Histologically, an area of necrotic cartilage is typically the first identifiable sign of an OC lesion.

When the epiphyseal cartilage becomes necrotic, ossification cannot occur, the "cartilage template" is retained, and progresses to a focus of thickened cartilage. Because cartilage is avascular, it depends on diffusion of synovial fluid for its metabolic needs. Chondrocytes in the deeper zones of abnormally thickened cartilage are deprived of nutritional support because of the increased distance synovial fluid must diffuse. This further compromises normal cartilage development. At the cellular level, important factors that are thought to be responsible for OC include abnormal chondrocyte metabolism, abnormal coding for cartilage matrix production, and biomechanical forces leading to irregular articular loads. It is likely that these elements participate concurrently in the etiological process, therefore, one should consider OC a multifactorial disease.
Direct blood supply from the systemic circulation and diffusion of nutrients through the synovial fluid are the two sources for the metabolic support of the process of endochondral ossification. Articular cartilage has no blood supply and must receive its nutrients by diffusion through the synovial fluid. In contrast, epiphyseal cartilage has a direct blood supply from vessels located in the perichondrium, a thin continuation of the periosteum over the extremities of bone. Perichondrial blood vessels penetrate and supply the epiphyseal cartilage through cartilage canals. This source of blood supply is vitally important during the early stages of cartilage development. If it is disrupted prior to the normal time frame required for epiphyseal development, ischemia and necrosis can result and produce lesions consistent with OC.

Appropriate and specific extracellular matrix production is essential to the development of the epiphyseal cartilage and the formation of bone. Chondrocytes are capable of producing many different types of matrix molecules such as proteoglycans and glycosaminoglycans. These molecules must be produced in the right order, location and arrangement to allow for successful completion of endochondral ossification. Disruption of this complex process due to abnormal genetic makeup or cellular injury may result in lesions consistent with OC. Because OC lesions are noted to occur in consistent locations within each affected joint, biomechanical influences are thought to play a role in their development. Some investigators hypothesize that physiologic trauma during development results in focal stresses on specific areas of developing articular cartilage which leads to abnormal development of the tissue.

Etiology

Osteochondrosis is thought to have a multifactorial etiology. Genetic factors that affect weight gain and growth, behavior, sexual development and conformation undoubtedly are involved in the etiology of osteochondrosis.

Osteochondrosis occurs with specific breed predisposition such as large warmblood breeds and thoroughbreds. Rapidly growing representatives of these breeds seem to be predisposed. The genetic capacity for rapid growth and overfeeding may be influential during the period of rapid growth.

This is an important aspect in athletic animals as often it is the largest, most rapidly growing animal that is desired. In addition the selective breeding of performance animals may be based on performance data, conformation, and other desired qualities without knowledge of the presence of articular disease that may be subclinical in nature or affected siblings.

Biomechanical forces are another important etiologic factor in the development of osteochondrosis. Osteochondrosis lesions develop in areas of articular cartilage that are subject to increased loads. Normal joint stresses and focal trauma likely are inciting perpetuating factors involved in the pathogenesis of this condition. The strenuous activity often imposed on performance animals at very young ages may partially account for the high incidence of osteochondrosis in many breeds of working and racing species.
Overnutrition in the form of excessive amounts of food and/or nutritional supplements has been incriminated as another important etiologic factor. This is evidenced by higher caloric intake in rapidly growing animals resulting in greater incidence of osteochondrosis. Excessive calcium supplementation has been definitively established to increase the development of osteochondrosis.

**Incidence**

The incidence of osteochondrosis is estimated to around 5 to 20%, although depending on the breed the occurrence rate may be much higher. Recently it was shown that 50% of horses operated on for osteochondrosis of the femoropatellar joint were Thoroughbreds. OC primarily affects diarthrodial joints and, in the horse, is usually found more frequently in males than females. All breeds of horses may be affected, however the majority of cases have been recognized in racing breeds. It is important to remember that any articular surface may be affected, including vertebral articulations, with more than one joint frequently involved. For this reason any lameness in young horses, particularly if bilateral, should include OCD as a differential diagnosis. The most common sites are the distal intermediate ridge of the tibia, the lateral and medial trochlear ridge of the talus, the lateral and medial trochlear ridge of the femur and less commonly the humeral head, the glenoid surface of the scapula, the metacarpo/tarso-phalangeal and proximal interphalangeal joints.

**Clinical signs and diagnosis.**

The appearance of clinical signs associated with OC can sometimes be delayed many years, particularly if only mild lesions have occurred, and the horse has been allowed to mature before vigorous exercise has commenced. Cases appearing in older animals can often present as a degenerative arthritis, however the location and appearance of lesions is indicative of a longstanding OC lesion.

The most prominent clinical sign in cases of OC is effusion in at least one affected joint. Effusion may not always be evident in the contralateral joint, even though radiographic evidence of disease exists. Lameness is variable, but is usually only mild to moderate. The lameness may be difficult to completely abolish with intra-articular local anaesthesia. Muscle wasting is often evident in longstanding OC of the shoulder and stifle.

The age of the horse, its size and sex, and the presence of joint effusion should be factors suggesting a diagnosis of OC. Confirmation of a diagnosis of OC is usually made by radiography. If clinical signs are only apparent in one limb, the contralateral joint should always be radiographed. The horse should also be closely examined for abnormalities in other joints. A neurological examination may be indicated to determine if cervical vertebrae have been affected. In OCD, articular cartilage becomes thickened and usually does not mineralize, therefore it appears radiographically as a defect in the articular surface. Common radiographic signs of osteochondrosis include, discrete osteochondral fragments, defects in the contour of the articular surface (flattening or depression), irregularly shaped lucent areas in the subchondral bone, subchondral bone sclerosis surrounding the lucent areas, secondary degenerative joint disease (DJD). Despite the presence of lameness and effusion, OC lesions of the shoulder and stifle may not be readily identified radiographically. In these cases the diagnosis can ultimately be made by investigating the joint via arthroscopy. It is important to note that lesions found on a radiographic
study are not always clinically significant. Radiographs must be interpreted in light of clinical signs. Generally, lesions which cause clinical lameness occur in horses less than three years of age. Occasionally, horses remain asymptomatic until later in life.

Treatment

Conservative therapy consists of restricting the horse’s exercise, slowing the growth rate, and ensuring that there are no dietary vitamin or mineral deficiencies. Excessive feeding of grain should be avoided. Conservative therapy is successful in approximately 20–50% of cases, dependent upon the site and severity of the lesion, but up to 12 month convalescence may be required, and joint effusion and lameness may be present for a considerable period of this time. Intra-articular Hyaluronic acid and systemic administration of polysulphated glycosaminoglycans (PSGAGs) do not appear to have any effect on the long-term outcome if combined with conservative treatment. Corticosteroids are contraindicated.

Surgical treatment is usually recommended for OCD in the horse. Surgical treatment consists of removal and debridement of detached or abnormal cartilage to induce subchondral bone capillary bleeding. This facilitates the initiation of a repair process which will eventually lead to the formation of functional fibrocartilage in the majority of cases. Arthroscopic surgery is preferred to arthrotomy, because of the ability to observe and operate on multiple sites within a joint, reduce surgical trauma, and minimize postoperative complications. Approximately 70–80% of cases respond favorably to arthroscopic surgery. Improvement in clinical signs is often evident within weeks of surgery. Postoperative care consists of 4 weeks of stall confinement, then 4 weeks of hand-walking, followed by paddock rest for a further 8–16 weeks. This can be modified to suit the individual lesions and training program of the affected horse. If required, some horses, primarily those with small lesions of the distal intermediate ridge of tibia, can resume exercise within 10–14 days of surgery. Because OC is very often associated with some degree of osteoarthritis, clients need to be informed of the potential for the development of lameness and need for medical management after surgery. Early surgical management will help retard the development of the osteoarthritis and produce a less painful and more functional joint. The prognosis in appropriately managed patients is generally favorable.