Functional Anatomy

Osteochondrosis

Submitted by Donna Stokell

In the fetus, at an early period, the greater part of the skeleton is cartilaginous: as this cartilage is afterward replaced by bone, it is call temporary, in contrast to that which remains unossified during the life, and is called permanent. In this paper I will discuss the temporary cartilage, the ossification process and how the disease Osteochondrosis develops.

Osteochondrosis (OC) is a multifocal disorder of epiphyseal cartilage that occurs in both the articular-epiphyseal cartilage complex and the growth plate (C Carlson, 1998) About 60% of the time OC will develop when the horse is a year of age or less (Trotter Gayle, 2003).

Cartilage is a type of dense connective tissue composed of cells called chondrocytes, which are dispersed, in a gel-like substance, called the matrix. Cartilage contains no blood vessels and nutrients are diffused through the matrix. Chondrocytes are the only cells found in the cartilage and are responsible for the maintenance of the matrix.

There are three different types of cartilages, each with special characteristics adapted to the local needs of the horse: Hyaline Cartilage, Elastic Cartilage and Fibrocartilage. The cartilage that I will be addressing in this paper is the Hyaline Cartilage, which is found lining bones in joints (articular cartilage) and inside bones, serving as the centre of ossification.

Most types of cartilage have a tissue layer called the perichondrium that lines the cartilage. In the hyaline cartilage that will be transformed into bone, the perichondrium will become the periosteum (the vascular tissue lining the marrow cavity of a bone).

There are two different types of growth that can occur in cartilage: appositional and interstitial. The appositional growth is the increase of the diameter of the cartilage with new cells deriving from the perichondrium on the outer surface of the cartilage. The interstitial growth results in an increase of cartilage mass from within. Chondrocytes undergo mitosis and remain in the matrix, which will result in clusters of cells called isogenous groups.

The ossification process develops in the center of the future long bone (diaphysis) and also at the ends of these long bones (epiphysis). Endochondral ossification occurs in the bones of the extremities, the vertebral column, pelvis, and base of the skull. Bone is formed from a hyaline cartilage matrix, which becomes ossified. First the cells that absorb cartilage (chondrocytes) mature and enlarge and the intracellular matrix begins to thin. The mature chondrocytes produce alkaline phosphatase, causing the intracellular matrix to calcify. The calcification of the matrix in turn causes the chondrocytes to die. This creates cavities with in the bone. A periosteal bud, which consists of blood vessels, lymph vessels and nerves, invades the cavity left by the chondrocytes.

The vascularization ultimately carries hemopoietic cells, osteoblasts and osteoclasts inside the cavity. The hemopoietic cells later form the bone marrow.

Cartilage is retained in the epiphyseal plate, located between the diaphysis (shaft) and the epiphysis (end) of the bone. These areas of cartilage are known as secondary centres of ossification. As the growth progresses, the proliferation of cartilage cells in the epiphyseal plate slows and eventually stops. The continuous replacement of cartilage by bone results in the obliteration of the epiphyseal plate. Then only articular cartilage remains.

If the ossification process fails, osteochondrosis will result. OC develops when: cartilage cells that have proliferated in a normal manner, mature and become abnormal; a failure of the cartilage matrix to calcify; a vascular penetration does not occur, or a lack of nutrition may cause necrosis in the deeper layers. This condition mainly affects articular growth cartilage, but the metaphysis may also be involved (MerckVet). Another scenario is that thickened cartilage persists without fissuring and becomes surrounded by subchondral bone as adjacent cartilage continues endochondral ossification; the retained cartilage dies resulting in a subchondral cyst-like lesion (Turner Tracy).

The viability of the epiphyseal cartilage highly depends on the blood supply from the cartilage canal vessels. In contrast, articular cartilage derives almost all of its nutrition from synovial fluid, with only a minor supply from vessels.

In both the articular-epiphyseal cartilage complex and the growth plate, lesions of OC are composed of focal areas of retained cartilage that are not converted to bone by endochondral ossification. (Carlson, 1998) Growth plate lesions are not evident until there is a failure in endochondral ossification.

Etiology of Osteochondrosis

Trauma is often considered a cause of OC when the sites are in the predilection areas of increased biomechanical stress. Adequate exercise for a foal is imperative, however, over exercising or high intensity exercise is also responsible for the onset of OC. A young horse that overuses a joint affected by OC can cause the defective cartilage to separate from the bone to which it is attached. Separation of cartilage from bones causes pain and joint instability initiating the development of secondary arthritis. The housing of a horse on hard flooring also appears to increase the prevalence and severity of OC. Traumatic osteochondral fractures are characterized by sudden onset of pain and a history of significant trauma, and should be considered in a separate category from OC.

The tendency to develop OC is inherited, with larger, fast-growing breeds of horses being more likely to develop the condition. However, not all horses whose parents have the condition will develop it themselves. Genetic factors and rapid growth clearly have a role in OC. In all species in which the disease occurs, OC occurs during the period of rapid

growth, and most commonly involved where rapid growth is emphasized.

A number of nutritional factors have been implicated: including high-energy, high-protein diets; excessive intake of calories, calcium and phosphorus; and imbalances of calcium, phosphorus, vitamin A, vitamin D and iron. Copper deficiency has also been reported to produce OC like lesions in horses.

Overfeeding a weanling which was not allowed to creep feed while nursing can cause a compensatory growth spurt that might predispose the foal to alterations in the endochondral ossification process. Excessive dietary energy becomes an even greater problem when certain nutrients are lacking while others are fed in excess. The excessive dietary energy leads to rapid bone growth, and rapid growth tends to be one of the major factors for OC (Kemper, 1996).

Improper shoeing can cause many joint and muscle problems and is believed to be a significant factor in the progression of OC.

Local ischemia to the epiphyseal cartilage leads to the formation of highly vulnerable zones of necrotic epiphyseal cartilage, which later causes a delay in endochondral ossification, with the extension of necrotic cartilage into the subchondral bone. Early lesions of OC in horses have been associated with abnormalities in the cartilage canal blood vessels, which are hypothesized to cause local ischemia and condronecrosis.

Common Sites of Osteochondrosis

Articular-epiphyseal cartilage complex

- -Dorsal edge of sagittal ridge of distal tibia
- -Articular processes of cervical vertebrae
- -Medial condyle and trochlear ridges of femur
- -Medial ridge of trochlea of tibial tarsal bone

Growth plate

- -Distal radius
- -Distal tibia
- -Distal end of third metacarpal bone
- -Distal end of third metatarsal bone
- -Proximal end of proximal phalanx

Symptoms/ Diagnosis of Osteochondrosis

The most common sign of OC is a nonpainful distension of an affected joint (e.g. gonitis, bog spavin). The main clinical signs in foals under six months old are that they have a tendency to spend more time lying down. Joint swelling, stiffness, and difficulty keeping up with other animals may accompany this. In some foals a sign may be the development

of upright conformation of the limbs, presumably as a result of rapid growth.

The signs of OC in yearling or older horses are usually stiffness of joints, flexion responses, and varying degrees of lameness. These signs are usually associated with the onset of training and, therefore, suggest a biomechanical influence and an activation of subclinical or "silent" lesions (Merk Vet).

A horse with OC may try to compensate for the lameness by restricting movement of the affected joint. For example if the leg is affected, the horse may swing its leg outward in a circular motion to avoid bending the leg. OC usually affects both sides, however, one leg is often worse than the other. If the horse takes extra weight on the better leg this can affect the healthy limb and cause alterations in bone development and the endochondral ossification causing additional developmental problems.

Clinical diagnosis requires the use of some specific clinical aids such as radiographic examination, Ultrasound examination of the swollen joints, or the most accurate way to confirm the diagnosis is by arthroscopy, except in the case of the cervical articulations which are not accessible.

Treatment Options for Osteochondrosis

All treatment for OC is based on further inhibiting breakdown of the joint cartilage and bone, thus decreasing the pain that the horse may be experiencing..

Nonsurgical methods may consist of stall rest from four to six weeks for foals with incomplete ossification and straight limbs. Foals with incomplete ossification of the carpal and tarsal bones may be managed effectively with splints or casts to maintain the limb in proper alignment and to allow normal endochondral ossification to progress without damage by uneven loading. The casting period may range from two to four weeks.

Restricting the amount and intensity of a horse's activity has been shown to reduce the incidence of OC. Flaps of cartilage that have not yet broken away from the underlying bone may heal back if the affected joint use is not intense or prolonged. Regular short walks are recommended for horses with OC combined with added stall rest.

Overfeeding contributes significantly to the development of many orthopedic conditions in horses, including OC. Research shows that horses fed 130% of what National Research Council (NRC) recommends for carbohydrates consistently produced OC in weanling foals compared to a controlled diet based on 100% of NRC requirement. The results of a study performed by Colorado State University (1999) did not support the commonly held belief that high-protein diets are a problem.
A foal must not only receive adequate amounts of each nutrient, it must also receive the proper ratios between nutrients to ensure bone development. A foal should receive a ratio or two parts calcium to one part phosphorus. Calcium should make up 0 .8% of a weanling's dry matter intake, and 0.5% of a yearling's dry matter intake. Phosphorus should make up 0.5% of a weanlings dry matter and 0.3% of a yearling's dry matter (Kemper Tina, 1996). Calcium is imperative to proper bone growth, excessive calcium

can inhibit the absorption of phosphorus, which will cause an imbalance and can lead to OC.

The amounts of copper and zinc in the foals diet can also be problematic, Copper is needed for bone collagen and elastin synthesis, which make up the lattice-work for bone formation. One method to ensure the foal has a proper copper level is to monitor the mare during gestation, as the foal's liver will accumulate the copper and stores it for use after foaling.

Protein is another nutrient that must be monitored. A foal's diet should consist of 14% protein. Excessive amount of protein alone will not increase a foal's growth rate, but in combination with an excess of other nutrients it can cause rapid bone growth and predispose a foal to OC. Rapid bone growth can be genetically based and not always a result of nutritional imbalances.

Analgesic and anti-inflammatory medications should only be used for the short term, when necessary to encourage movement and relieve severe pain.

Surgery is an option in the treatment of OC. The common surgical method is aimed at accelerating growth of the long bones or to retard growth of the long bones. This procedure is based upon the location of the problem, the degree of the deformity and the age of the foal. The goal of the surgical procedure is to remove any loose pieces of cartilage from the joint surface and scrape the cartilage defect to encourage the body to refill the desiccated area. Surgery is always considered to be the last resort for OC.

Summary

Etiology of OC appears to be multifactorial, with trauma, hereditary factors, and rapid growth, nutritional factors, and ischemia all having a role in the pathogenesis. "I don't think anyone really knows how much the manifestation of osteochondrosis sites has to do with genetics, and how much has to do with a function of what the animal does for a living" (Kemper,Tina 1996) Genetically we may not be selecting for OC, but we are selecting for rapid growth and increased body weight in many of our performance horses. By doing this, we may very well, unconsciously, be breeding horses, which may be predisposed to developing Osteochondrosis.

A take-home message for horse owners would seem to be this: feed a growing horse a balanced diet and do not push the high-energy feeds, make sure the horse ingests the proper balance of minerals. If OC shows up in several foals by the same stallion, consider that genetics could be the problem, and, most importantly, if a young horse shows signs of lameness or swelling in any joint call your veterinarian.