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Normal cartilage development and the pathogenesis of osteochondrosis

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Epiphyseal cartilage and normal endochondral ossification

Most of the embryonic skeleton is first formed as cartilage. In fact, with the exception of the cranial vault, the skeleton appears in the embryo as an entirely cartilaginous structure. In the center of the shaft of a developing long bone, cartilage matrix calcifies and is invaded by blood vessels. Cells line up on the surface of the calcified cartilage and deposit a bony matrix. This process of cartilage calcification followed by vascular invasion and deposition of bone matrix is known as endochondral ossification and is the normal process by which cartilage is transformed to bone. In the fetus, the process of endochondral ossification continues until a considerable portion of the shaft of the bone has been converted into bony tissue and only the ends of the bone are still formed of cartilage. A secondary center of ossification then forms within the cartilaginous ends of the bone. Calcification occurs initially at the middle of the secondary center. This area is then invaded by blood vessels and the process of endochondral ossification occurs in this new location. The vessels leading to the calcified cartilage in the center of the epiphysis are carried in canals, termed cartilage canals, that develop from invaginations of the surface covering of the embryonic cartilage. As the secondary center of ossification grows, the only remaining cartilage is that which covers the articular end of the bone (articular-epiphyseal cartilage complex) and a thin layer or plate of cartilage lying between the secondary center of ossification and the main part of the bone shaft (growth plate or physis).

Epiphyseal cartilage, also termed growth cartilage, is the vascular hyaline cartilage that composes the growth plate and the epiphyseal cartilage portion of the articular-epiphyseal cartilage complex. The epiphyseal cartilage of the growth plate is responsible for longitudinal bone growth, whereas the epiphyseal cartilage of the articular-epiphyseal cartilage complex is responsible for forming the shape of the ends of the growing long bones. Epiphyseal cartilage is a temporary tissue that is present only in immature individuals. It is richly supplied with blood vessels. In the articular-epiphyseal cartilage complex, these vessels gradually decrease in number and extent with increasing age and weight of the individual as epiphyseal cartilage decreases in volume and becomes less dependent on a blood supply. In animals, this cartilage disappears completely by several months of age, depending on site and species. For example, it is absent from femoral condyles of pigs by 4-5 months of age. Normally, this attrition of vessels occurs through chondrification, a physiologic process whereby the lumens of cartilage canals narrow and become filled with cartilage. This process is not normally associated with pathologic changes in the epiphyseal cartilage. As growth ceases, the epiphyseal cartilage of the growth plate and the articular-epiphyseal cartilage complex is completely replaced by bone. The remaining cartilage at the ends of the long bones is termed articular cartilage and is a permanent tissue that remains throughout life. Articular cartilage is not supplied by blood vessels.

Pathogenesis of osteochondrosis

Osteochondrosis is a multifocal disorder of epiphyseal cartilage that occurs in both the articular-epiphyseal cartilage complex and the growth plate. The disease occurs in highly consistent predilection sites, and lesions often are bilaterally symmetrical. It occurs in humans and in a variety of animal species, most notably pigs, horses, and large-breed dogs. The prevalence in pigs is particularly high and approaches 100% in domestic swine. There is considerable confusion in the literature regarding osteochondrosis, primarily because it has been studied mainly in its chronic stages during which it causes significant problems due to lameness. Among all species in which they have been examined, however, the early (subclinical) lesions in the articular-epiphyseal cartilage complex and growth plate are remarkably similar, strongly suggesting that the pathophysiology of this condition is essentially the same in all species. Because epiphyseal cartilage is absent in the adult, the underlying lesions of osteochondrosis can only occur in growing individuals, although clinical signs may not be evident until adulthood.

Early lesions of osteochondrosis are composed of well-defined areas of cartilage necrosis (death) that are confined to the epiphyseal cartilage and do not involve either the overlying articular cartilage or the underlying subchondral bone. Many of these lesions are centered on necrotic cartilage canal blood vessels. Because dead carti-

lage cannot undergo endochondral ossification, a delay in this process occurs when the ossification front reaches the area of necrotic cartilage. The vast majority of these early lesions heal spontaneously; however, some do not. Because the necrotic cartilage is weaker than normal tissue, it is highly vulnerable to trauma. Given a large enough area of necrotic cartilage and sufficient trauma, a cleft extending from the articular surface to the subchondral bone will form. This leads to inflammation of the synovial membrane, increased joint fluid, and clinical signs of lameness. Once a cartilage cleft is formed, the disease proceeds down a "path of no return" with the only treatment option being surgical removal of the loose cartilage fragment.

There appear to be multiple contributing causes for osteochondrosis, including trauma, hereditary factors, rapid growth, nutritional factors, and failure in blood supply. Trauma is the most widely proposed etiology, partly because the appearance of the chronic lesions is highly suggestive of trauma. In addition, this disease occurs in highly specific predilection sites, some of which are located in areas of increased biomechanical stress. In addition, housing animals on hard flooring appears to increase the prevalence and severity of osteochondrosis, as does rough handling during loading and unloading for transport. In human beings (the only species able to report a history of trauma), however, most cases have an insidious onset. In all species in which the disease occurs, osteochondrosis develops during the period of rapid growth, and the most commonly involved animal species are those in which rapid growth is emphasized. The prevalence of lesions, however, is not altered significantly by reducing growth rate by restricted feeding or by breeding animals with fast growth rates with those with slower growth rates. Genetic factors, however, clearly have a role in osteochondrosis. Many familial cases of this disease have been reported in humans, including closely similar lesions in identical twins. One reason for the high prevalence of osteochondrosis in domestic pigs may be that these animals are genetically selected for body characteristics that show a positive correlation with the presence of lesions of osteochondrosis. Interestingly, the disease is absent from wild pigs and miniature pigs. A number of nutritional factors, including high-energy, high-protein diets; excessive intake of calcium and phosphorus; and imbalances of calcium, phosphorus, vitamin A, and vitamin D have been implicated in osteochondrosis; however, there is no clear evidence that any of these has an important role in its cause. The preponderance of the evidence regarding the cause of osteochondrosis is supportive of a defect in vascular supply to epiphyseal cartilage. In the femoral condyles of pigs, lesions of cartilage necrosis are first seen in the first areas of epiphyseal cartilage to become avascular and are associated with necrotic blood vessels. Experimentally, lesions similar to those that occur naturally have been produced by surgically interrupting blood supply to epiphyseal cartilage, supporting the hypothesis that these lesions occur secondary to a failure in blood supply from cartilage canal blood vessels. Because epiphyseal cartilage becomes avascular prior to adulthood, a vascular etiology also explains why osteochondrosis only develops during the period of skeletal growth. A vascular etiology also explains why lesions occur in multiple and predictable locations as well as why they often are bilaterally symmetrical. Although a defect in vascular supply appears to be very important in the pathogenesis of osteochondrosis, the precise nature of that defect is unclear and is the subject of current study.

Osteochondrosis and gilt rearing practices

Although osteochondrosis appears to be associated with increased rate of gain, it does not appear to be possible to eliminate or even reduce these lesions in individual animals by restricting weight gain or by other dietary manipulations. Given our lack of understanding regarding the underlying cause of this disease at the present time, the most realistic approach in rearing gilts may be to make every effort to minimize joint trauma in these animals. This would include the use of soft flooring as well as using care during transport, particularly loading and unloading. Although trauma is not the cause of this disease, it appears to have an important role in the conversion of subclinical lesions to clinical disease. The most probable role of trauma is as a final insult to a locally extensive area of vulnerable epiphyseal cartilage. Even physiological forces could result in cleft formation and clinical disease and should be avoided to whatever extent is possible. The bottom line is that the great majority of subclinical lesions of osteochondrosis heal; therefore, if the joint can be protected from trauma during the time frame during which it is vulnerable (particularly between the ages of two and five months), it is possible that clinical disease could be reduced.

Predilection sites of osteochondrosis in pigs

Articular-epiphyseal cartilage complex

- Medial femoral condyle
- Humeral condyles
- Humeral head
- Dorsal acetabulum

Growth plate

- Distal ulna and femur
- Costochondral junction

- Femoral head
- Humeral head
- Ischial tuberosity

References

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