

Developmental Orthopaedic Disease: Problems of Limbs in Young Horses

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The term developmental orthopaedic disease (DaD) was coined in 1986 to encompass all orthopaedic problems seen in the growing foal and has become the generally accepted term. It came out of a Blue Ribbon Panel sponsored by the American Quarter Horse Association. It is a term that encompasses all general growth disturbances of horses and is therefore nonspecific. It is felt that one has to be nonspecific when talking about the various limb abnormalities of young horses because previous terms such as metabolic bone disease and osteochondrosis implied that they all had a common cause and pathogenesis (mechanism of disease development). It is still to be determined how closely related the various forms of DaD may be but it is important that the term not be used synonymously with osteochondrosis. It is considered inappropriate for all subchondral cystic lesions, physitis, angular limb deformities and cervical vertebral malformations to be presumed to be manifestations of osteochondrosis (a condition defined below). The spectrum of conditions currently classified as DaD was previously designated as metabolic bone disease. However, it is felt that this term is misleading because it refers specifically to bone, whereas many of these problems are seen essentially as joint and growth plate problems. Severe forms of DaD are seen sometimes when there is little or no aberration in bone histomorphometry, implying questionable change in bone metabolism.

Before defining the various syndrome of developmental orthopedic disease, some basic developmental anatomy is appropriate. Long bones develop from cartilage by a process of endochondral (within cartilage) ossification.

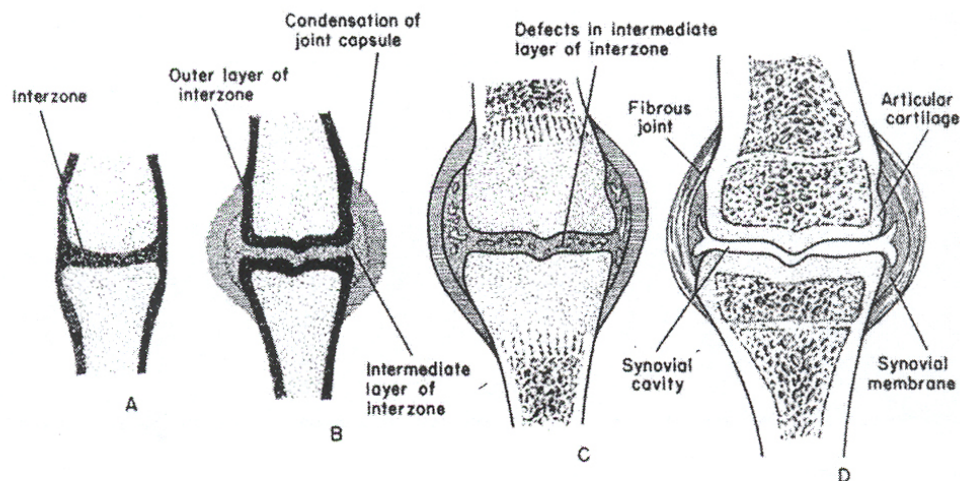


Figure 1. Diagrammatic representation of the development of a joint.

The centers of ossification (bone formation) develop in the center of the future long bone (diaphysis) and also at the ends of these long bones (epiphysis). As ossification proceeds, a bony epiphysis and a bony diaphysis form. Between these two centers of ossification is a metaphyseal growth plate and this is what enables the limb to lengthen after birth as the foal grows. There is a

second growth plate called the epiphyseal growth plate that forms as the epiphyseal ossification centre advances toward the ends of the bone and this is destined to be the articular surface of the joints. Joints form by condensation of connective tissue (Fig. 1)

The Different DOD's

When the term developmental orthopaedic disease was first coined, it was categorized to include the following:

1. Osteochondritis dissecans
2. Subchondral cystic lesions
3. Angular limb deformities
4. Physitis
5. Flexural deformities (these may have no defined cause, or may be secondary to osteochondrosis or physitis)
6. Cuboidal bone abnormalities
7. Juvenile osteoarthritis

Osteochondritis dissecans

This condition involves a dissecting lesion of the articular surface of a joint with the formation of a cartilage or cartilage and bone flap. Flaps may become detached and form joint mice. Blood vessels from the periphery of the joint frequently remain in communication with cartilage flaps or detached flakes, leading to calcification or ossification of the separated cartilage. It is commonly considered that release of debris from under the flap causes synovitis (inflammation of the synovial membrane) and pain, but we don't think it is that simple now because we find instances where the cartilage is intact but the horse still has lameness and synovial effusion (fluid filling of the joint).

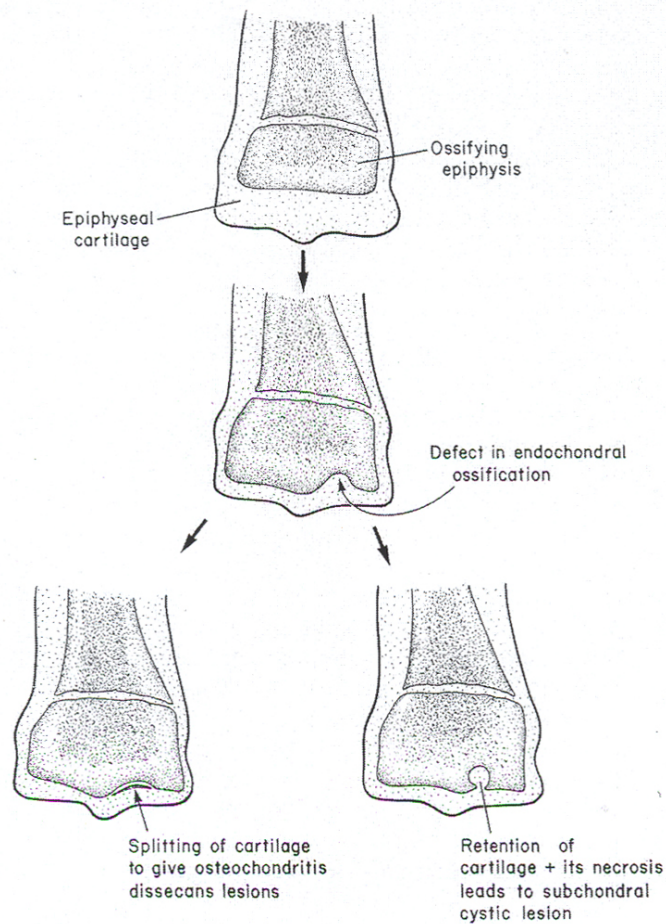


Figure 2. Diagram of pathogenesis of osteochondritis dissecans and subchondral cystic lesions in relation to the generalized condition of osteochondrosis.

Osteochondritis dissecans (OCD), as well as subchondral bone cysts, are commonly considered to be a manifestation of osteochondrosis (Figure 2). As mentioned previously, the epiphyseal ossification center advances out until ossification ceases, leaving a layer of cartilage. This layer of cartilage becomes the articular cartilage. If there is a disturbance in endochondral ossification, an area of retained cartilage can be formed with a consequent defect in the bone. Cracking can then proceed in this retained cartilage to give a flap or fragment of cartilage that may contain bone. These flaps and fragments on the surface of the joint result in osteochondritis dissecans.

Subchondral Cystic Lesions

Subchondral cystic lesions occur in various locations, but the most common place is in the medial condyle of the femur in the stifle, followed by the distal metacarpus in the fetlock joint. Subchondral cystic lesions have often been proposed as a manifestation of osteochondrosis by a number of authors and there is pathologic evidence to support this. When we have a yearling horse with a large subchondral cystic lesion in one stifle and a defect in the other, we feel comfortable to assume the cause to be osteochondrosis. However, we also get subchondral cystic

lesions that develop in older horses and in some instances we have noted them developing after a cartilage surface defect.

Recently in a project funded by the American Quarter Horse Association, we demonstrated that subchondral cystic lesions could develop from defects in the joint surface and in mature animals. Also, in other research from our laboratory, examination of the contents of these bone cysts after their removal have shown that they have high levels of degradative enzymes and can actively resorb bone. This probably expands how cysts expand.

Angular Limb Deformities

Angular limb deformities involve deviations as looked at from the front or back, such that the deviation is excessive from side to side. Many angular limb deformities cause no problems and resolve themselves. The most common one, carpal valgus, is a normal conformation for a young foal. The legs gradually straighten on their own. On the other hand, if the deformity is in the other direction (carpal varus), this is a severe problem and needs immediate attention. Most of these cases in the knee, if excessive, are treated with transphyseal bridging to stop growth on one side of the growth plate and allow the other side to catch up. When the deformity involves rotation below the level of the fetlock joint, these are usually handled with trimming and/or hoof extensions. There is quite good evidence that periosteal stripping does little to correct angular limb deformities.

Phyinitis

This is a swelling around the growth plates of the distal radius and distal metacarpus. Recent research by Erica Gee and Elwyn Firth at Massey University calls into question whether much of this swelling is indeed a clinical problem. If swelling of the growth plates is associated with lameness, radiographs are taken to ensure that there is not a significant problem within the growth plate. These problems are managed conservatively.

Cuboidal Bone Abnormalities

This problem is generally one of immaturity and is most commonly seen in premature foals. The normal small bones of the carpus (knee) and tarsus (hock) ossify initially in a circle and later form a bony cube. If a foal is born early, sometimes wedging can occur in these bones. They will usually appear as an angular limb deformity in the carpus or, in the case of tarsal bone collapse, as a sickle hock conformation. Radiographs confirm the diagnosis. These problems should always be considered in obvious angular limb deformities that are not improving.

Incidence of DOD and Radiographic Surveys

There has been a problem or a point of confusion between reports of clinical instances of DOD and radiographic surveys of horses not necessarily showing clinical signs. This has led to some questionable conclusions, not only regarding the causative factors of DOD but also the effect of treatment or the significance of various problems. There are many instances where radiographs

on a pre-purchase examination will show QCD, for instance, but that QCD may not be causing clinical problems because the fragment has not separated. That is why it is important to distinguish between the two. Most of the major radiographic studies have involved Standardbred horses.

In one study in Quebec, each horse's racing performance at two years of age was related to the radiographic lesions diagnosed at approximately 17 months of age and before training (there were two generations of 41 and 32 yearlings, respectively); five stallions and 46 mares, were also radiographed). No complete clinical examinations or lameness diagnoses were made. Radiographic lesions were found in 31 (25%) of the horses (8 adults and 23 yearlings), of which 60% had a single problem, and 40% had between two and four radiographic problems. Subchondral bone cysts were detected in 14 (11.3%) horses (6 carpus, 5 fetlock, 4 pastern, 2 hock, and 2 stifle). Juvenile osteoarthritis lesions were diagnosed 78 times in 35 (47.9%) of the yearlings (40 pastern, 13 fetlock, 11 carpus, 8 coffin, 6 hock) based primarily on the basis of osteophytes. Sesamoiditis was also diagnosed in yearlings. The average winnings and number of starts were compared between radiographically normal horses, the OCD or subchondral bone cyst horses, and the juvenile OA horses; no significant differences were found. Although the radiographic lesions did not seem to be associated with poor racing performance, the authors of the study noted the lack of clinical data and the relatively small numbers. What we need are improved studies in which clinical signs are correlated with radiographs and possibly more important, all horses are radiographed and then followed so we know how many of those horses with x-ray changes develop clinical problems. This would solve many of the current issues at the yearling sales.

A high incidence of clinically apparent physitis and deformities was emphasized in another study in Canada. Mild to moderate physitis and flexural deformities (concurrent with physitis in most cases) occurred in 88% of 42 weanlings between weeks 6 to 8 of a study looking at the effect of dietary energy and phosphorus on blood chemistry and development of growing horses. In these instances, the clinical signs largely resolved on their own by five weeks. Dietary treatment did not influence the incidence, nor was it related to daily weight gain.

There has been another study defining incidence of DOD in Thoroughbred horses in Ireland over a period of 18 months. It was found that angular limb deformities and physitis together constituted 72.9% of the cases treated. The peak incidence of DOD problems occurred between weaning and the end of December. In a retrospective study, 193 of 1,711 (11.3%) were treated for DOD (21 had more than one type) and they are detailed as follows: angular limb deformities - 92, physitis - 64, flexural deformities - 18, wobbler's - 7, and osteochondritis dissecans, juvenile arthritis or other joint problems - 28. More than half the animals treated (53.9%) recovered completely (that is, they achieved expected sale value as yearlings), 27.5% showed incomplete recovery and mild to moderate loss of sale value, and 18.7% were either killed or lost much of their sale value. It was also noted that 67.7% of the animals showed some evidence of DOD, but only 11.3% were deemed to need treatment. This study was a good start and points out the need to have definition of what disease process we have. I think it can be seen from these studies that when "developmental orthopedic disease" occurs, it commonly involves angular limb deformities and physitis problems that spontaneously self-correct. It is the ones that often do not self-correct, such as OCD or subchondral bone cysts, that we need to investigate further.

Osteochondritis Dissecans - Causative Factors

As mentioned previously, many cases of OCD are associated with the pathologic process of osteochondrosis. Certain etiologic factors have contributed to the development of these lesions. These factors have varied, but the idea that there is a multifactorial etiology has generally been accepted. The information here is from clinical and pathology reports, as well as experimental studies in the horse. Several factors that seem to predispose the horse to osteochondrosis problems have been identified and include rapid growth, nutrition, excesses or imbalances and superimposed trauma on the cartilage.

Genetic Predisposition

Radiographic studies in Swedish Trotters and Warmbloods have shown progeny of one stallion from each breed having a significantly high frequency of QCD amongst his progeny, compared with the progeny of the other stallions. In another study in Denmark, radiographic evidence of a significantly high proportion of osteochondrosis in the progeny of one of eight stallions, even though the stallion itself did not show radiographic signs of osteochondrosis, was seen. Since that time, there have been two more studies on the heritability of osteochondrosis in the hock of Standardbred Trotters showing a reasonably high heritability in hock QCD.

There has been little work done in the United States with regard to heredity and we certainly haven't been able to develop any type of screening program for osteochondrosis in stallions and mares that will ensure freedom from that condition. However, it would appear very likely that there are genetic components to this disease. Individual instances of certain stallions and mares producing these individuals have been seen.

Growth and body size

Fast growth was implicated with a high incidence of osteochondrosis in dogs and pigs. There have been anecdotal reports of this in the horse.

Mechanical stress and trauma

It has certainly been recognized that mechanical stresses often precipitate clinical signs with QCD and it is presumed that this is by separating the QCD flap or fragment from the parent bone. Whether trauma or physical stress is involved in the primary induction of an QCD lesion is controversial. However, some people do tend to feel this is the case and we do recognize there are certain predisposing sites for the occurrence of QCD, suggesting possible mechanical factors. A notable veterinary bone and joint pathologist, Dr. Roy Pool, feels that shear forces may disrupt capillaries in the subchondral bone (bone under the cartilage) and give rise to chondrocyte or cartilage cell damage. This is based on histologic observations of various lesions.

Nutrition

There has been an increased incidence of QCD lesions noted in horses fed 130% of what the National Research Council (NRC) recommends for carbohydrate and protein. A second study in

Australia by Dr. Kate Savage, which was very well controlled, showed that high energy diets (120% NRC requirements) consistently produced lesions of osteochondrosis in weanling foals compared to a control diet based on 100% NRC requirements. Some people have focused on "high protein" being a problem but this has not been demonstrated.

Mineral imbalances

Various mineral imbalances have been implicated as causative factors with QCD, including high calcium, high phosphorus, low copper and high zinc. Although high calcium levels have been implicated, experimental research in the horse with three times the NRC level of calcium in the diet failed to produce lesions of osteochondrosis. High phosphorus diets (five times NRC) did produce lesions of QCD in young foals.

Low copper has been implicated as a cause. An epidemiologic study on clinical cases of DQD implicated low copper levels as the most consistent factor. In experimental studies, it has been noted that a marked copper deficiency (1.7 ppm - a very artificially low level) produced QCD-like lesions and flexural deformities. In another study in Thoroughbred foals in which osteochondrosis developed before weaning, seven had serum copper and ceruloplasmin concentrations below normal. In a third controlled experiment in Canada with high (30 ppm) and low (7 ppm) copper diets, there was a much higher incidence of lesions seen in the foals fed the low copper diet. However, it is to be noted that most of the changes were present in the cervical vertebrae rather than the limbs where we commonly see clinical problems.

Excessive zinc intake has been related to equine osteochondrosis. Generalized osteochondrosis has been seen in foals raised near a zinc smelter. The relationship between zinc and copper (it has been suggested that high zinc suppresses copper levels) is still being elucidated.

Endocrine Factors

It has been postulated by one investigator that the production of osteochondrosis lesions in association with overfeeding is mediated by the endocrine system. Certainly the long-term administration of dexamethasone has produced osteochondrosis-type lesions and it is considered that glucocorticoids induced a parathyroid hormone resistance at the level of the osteocyte causing an inhibition of normal remodeling. Glucocorticoids also induced decreased GAG levels and this decrease in turn inhibits capillary penetration of the cartilage, which is a very important step in forming bone from cartilage. The failure of ossification could also be mediated through induced defects in vitamin D metabolism. Corticosteroids are also a potent inhibitor of lysyl oxidase, which is involved in cross-linking of collagen in cartilage and bone. It is felt this could be a way of inducing lesions.

Site Vulnerability

Because the lesions of equine osteochondrosis occur at specific anatomic sites, this obviously suggests site vulnerability. This predilection could be related to an ossification defect or trauma caused by excessive stress in that region. In nearly all instances, the sites of occurrence of QCD are very close to the limits of articulation and we know from basic research that the makeup of

the cartilage between articulating and non-articulating surfaces is different. QCD lesions are frequently bilateral in the stifle and hock and quadrilateral in the fetlock joint, although they frequently involve different joints in the same animal. It is felt this may suggest a "window of vulnerability" in the endochondral ossification of that specific joint when an environmental insult may have occurred. If the causative factor were present intermittently or for a transient period during the foal's growth period, this would³⁰ explain the development of the disease in only one pair of joints. It is not possible from this data to ascertain different periods of onset of the disease process in different joints.