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Devlopmenal orthopedic disorders of the dog and cat

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The imaging findings associated with developmental orthopedic disease are as diverse as the etiologies of the disorders themselves. Developmental lesions are frequently bilateral when they affect a specific bone or joint, multifocal when they are not site specific, or generalized. Secondary degenerative joint disease is often a sequelum to developmental orthopedic disease, especially when the primary lesion involves joints or causes alterations in long bone growth resulting in conformational abnormalities. Frequently the most obvious radiographic findings are these secondary degenerative changes that can often mask the original developmental lesion. From the standpoint of understanding etiology of a problem it is important to keep separate the cause (developmental lesion) from the effect (degenerative lesion).

REGIONAL DISORDERS OF ARTICULAR AND PHYSEAL CARTILAGE

Osteochondrosis (Syn. Osteochondritis dissecans, OCD)

Clinical Findings: Osteochondrosis is the result of a disruption of normal endochondral ossification of sub-articular cartilage in large, immature animals. More than one joint may be affected and lesions are often bilateral. Affected animals are typically lame in the affected limb or limbs. Lamelessness may be accompanied by joint capsule distention and pain on manipulation of the joint.

Radiographic Findings: Lesions most often appear as a defect within subchondral bone. Depending on the severity of the lesion, these defects can be small or large, smoothly or irregularly margined, and shallow or deeply cavitating. Surrounding bone is often sclerotic. In some instances separate mineralized fragments can be seen within the joint space either within the defect or some distance from it. Synovitis may occur due to migration of mineralized fragments and osteoarthritis frequently occurs secondarily. In dogs, the most common locations include the caudal aspect of the humeral head, the medial aspect of the humeral condyle, the medial and lateral femoral condyles, and the medial and lateral trochlear ridges of the tibiotarsal bone.

Metaphyseal Dysplasia due to Retained Cartilage Core in the Distal Ulna

Clinical Findings: This lesion is often found as an incidental finding on radiographic examinations of the antebrachium of large breed dogs. Dogs may show evidence of lameness in the affected limb. When the lesion is large or when it interferes with elongation of the ulna by inhibiting normal endochondral ossification, angular limb deformities can occasionally occur. Lesions are often bilateral.

Radiographic Findings: A lucent cartilage core can be seen centrally within the medullary region of the distal ulnar metaphysis adjacent to the physeal line.

Osteonecrosis of the Femoral Head (Syn. Avascular Necrosis of the Femoral Head, Legg-Calve-Perthes Disease)

Clinical Findings: This is a disease of small breed dogs approximately 3-11 months of age. Dogs are presented with hind limb lameness that may be either unilateral or bilateral. The disorder may be initiated by mild trauma of the coxofemoral joint.

Radiographic Findings: The early radiographic changes consist of mottled lucency within the affected femoral head and widening of the coxofemoral joint space. As the disease progresses the femoral head will collapse and flatten. Fragmentation of the femoral head may also occur. Sclerosis of the femoral head and neck and periarticular osteophyte formation becomes evident as secondary osteoarthritis progresses.

METABOLIC AND GENERALIZED DISORDERS OF THE IMMATURE SKELETON

Osteochondral dysplasias

Osteochondral dysplasias include chondrodysplasia, osteochondrodysplasia, enchondrodystrophy, multiple enchondromatosis, oculoskeletal dysplasia, hypochondroplasia, multiple epiphyseal dysplasia, and pseudoachondroplasia. Chondrodysplasia and osteochondrodysplasia result in disproportionate dwarfism and have been reported in a number of breeds of dogs and cats. Chondrodysplasia associated with ocular defects has been described in Labrador retrievers and other breeds. Although these disorders may sometimes appear clinically and radiographically similar, they represent a histologically and biochemically heterogeneous group of diseases. Characterization is further complicated by the variation in terminology used to describe these lesions.

In almost all instances in which microscopic findings have been described, marked alterations in chondrocyte morphol-
ogy and cartilage architecture are present. Although many of these disorders are inherited and known to be single autosomal recessive defects, others have not been adequately characterized. In some instances, the genetic defect can also be variably expressed, resulting in a wide variation in severity of clinical signs. It is important to distinguish between chondrodystrophoid dogs, those that have been bred for many generations to establish a defect as a breed characteristic, and chondrodysplastic dwarves that sporadically arise from normal parents.

**Radiographic Findings:** Imaging characteristics vary depending on the specific disorder but, in general, long bones of the appendicular skeleton are short, bowed, and have widened metaphyses in comparison to non-chondrodystrophic animals. These characteristics may result in angular limb deformities. In some form of this disorder, the axial skeleton is somewhat spared relative to the appendicular skeleton, in others vertebral segments may be short and box-like and marked skull deformities may develop.

**Congenital hypothyroidism**

**Clinical Findings:** Congenital hypothyroidism is an uncommon developmental disorder that has been reported in Boxers, Scottish deerhounds, Giant schnauzers, Affenpinschers, and Great Danes and is due to thyroid aplasia or hypoplasia. Clinically, the dogs are disproportionate, short-limbed dwarves with bowed limbs and long necks and trunks.

**Radiographic Findings:** Radiographic findings consist of epiphyseal dysplasia that appears as reduced or delayed ossification of the epiphyseal cartilage model. This is most easily seen in the proximal tibia and the humeral and femoral condyles. Cuboid bone ossification in the carpus and tarsus is also delayed. Vertebral bodies appear shorter than normal owing to end-plate dysplasia. The skull may appear shorter and broader than normal. Secondary degenerative joint disease may be seen.

**Mucopolysaccharidosis**

**Clinical Findings:** The mucopolysaccharidoses represent a loosely related group of uncommon autosomal recessive inherited disorders that result in reduction or absence of glycosaminoglycan catabolism. Lysosomal degradation of these mucopolysaccharides is necessary for normal growth in developing animals, and the abnormal metabolism leads to chronic, progressive multisystemic disease. More than 10 forms are recognized in humans, each produced by a different enzyme defect. Many of these have also been identified in dogs and cats. The most pronounced clinical manifestations involve the musculoskeletal, ocular, neurologic, hepatic, and cardiovascular systems. Affected animals are often stunted and lame and have visual deficits. Clinical manifestations include disproportionate dwarfism and facial dysmorphism, which includes a broad maxilla, widespread eyes, a flat nose, and short ears. Hyperextension of the distal extremity joints occurs as a result of joint laxity.

**Radiographic Findings:** Radiographic changes of mucopolysaccharidosis involve both the axial and the appendicular skeleton. There is generalized epiphyseal dysplasia involving long bones and vertebral end plates. Findings include delayed and incomplete mineralization of the epiphyseal cartilage model. Ossified regions of the epiphyses are smaller than normal and have a nonuniform opacity with a granular appearance. Vertebral bodies appear cuboid and shorter than normal. The maxilla is short and flattened; the frontal sinuses may be small or absent. Progressive degenerative joint disease occurs as a sequel to the epiphyseal malformations. Hip subluxation or luxation may result from femoral head epiphysis remodeling. Ventral, bridging spondylosis is seen in older animals.

**DISORDERS OF BONE TISSUE OF UNKNOWN CAUSE**

**Panosteitis (Syn Eosinophilic Panosteitis)**

**Clinical Findings:** Canine panosteitis is a self-limiting disease of the long bones of large breed dogs with the German Shepherd dog over represented. Panosteitis generally occurs in dogs 5 to 18 months of age although older dogs may also be affected. Clinically, these dogs develop a shifting leg lameness that is mild to severe.

**Radiographic Findings:** Lesions can be seen in one or more long bones and may occur in more than one leg. Lesions are diaphyseal in location and often appear at the level of the nutrient canal of the affected bone. There is typically an ill-defined increase in intramedullary opacity with an associated loss of trabecular (spongy) bone detail. Due to this increase in medullary opacity and an endosteal productive response, the internal cortical margin may also become less distinct. In more severe lesions a mild to moderate, smooth, productive periosteal response is also evident which may extend the length of the diaphysis. Radiographically, there is no destructive component to this disease. The severity of the clinical signs do not always correlate well with the severity of the radiographic lesions and radiographic changes usually lag behind the clinical progression of the disease. Radiographic changes usually resolve a few weeks after resolution of the clinical signs with productive periosteal changes requiring somewhat longer to resolve.

**INFLAMMATORY DISORDERS SPECIFIC TO THE IMMATURE SKELETON**

**Hypertrophic Osteodystrophy**

**Clinical Findings:** This inflammatory disease develops in young (3 to 6 months), rapidly growing, large breed dogs. Affected dogs are typically febrile and lame in one or more limbs. The metaphyseal regions of affected limbs are frequently swollen and painful to palpation. The distal radius and ulna are usually the most obviously affected regions. In severe cases, primary lesions can lead to angular limb deformity and persistent metaphyseal thickening from bone remodeling. The cause of hypertrophic osteodystrophy is not known but general dietary over-supplementation, dietary calcium/phosphorus imbalance, and vitamin C deficiency have all been suggested.

**Radiographic Findings:** Radiographic findings are often best visualized in the distal radius and ulna although all
metaphyses are involved. Regional soft tissue swelling around affected metaphyses is often evident. An irregularly marginated radiolucent line is seen within the metaphysis adjacent and parallel to the normal physeal line and metaphyseal bone may appear sclerotic. Irregular periosteal new bone formation occurs around the affected metaphysis with the extent of new bone formation dependent on the severity of the lesion. The appearance (smooth to irregular) of the periosteal bone depends on whether the periosteal response is active or inactive. In more severe cases, premature physeal closure can sometimes occur as a sequela to the primary disease with subsequent development of angular limb deformities. Extensive periosteal bone production may also lead to persistently thickened metaphyses.

**Craniomandibular Osteopathy**

*Clinical Findings:* This disease affects immature dogs usually at 4 to 12 months of age. West Highland White, Scottish and Cairn Terriers are most commonly affected but the lesion has also been seen in other breeds. Dogs may be febrile and display evidence of mandibular or temporomandibular joint pain. Although the etiology is unknown, some believe this may be a different clinical manifestation of hypertrophic osteodystrophy.

*Radiographic Findings:* Typically, there is bilaterally symmetrical productive periosteal bone response that most often involves the mandible and less commonly the calvarium (specifically the temporal, parietal and occipital bones). The extent of new bone formation may vary but in the most severe cases, ankylosis of the temporomandibular joints can occur. New bone production ceases when normal bone growth ceases.

**Additional reading**