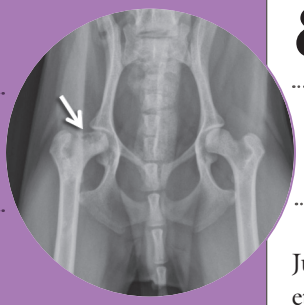

JUVENILE ORTHOPEDIC DISEASE IN DOGS & CATS

Part 1: Musculoskeletal Development & Pediatric Bone Diseases

Ingrid Balsa, MEd, DVM, and Duane Robinson, DVM, PhD, Diplomate ACVS (Small Animal)
University of California–Davis

Juvenile orthopedic diseases affect the musculoskeletal system of immature animals, and most of these diseases can be traced to pathologic events (eg, diseases, toxins, inappropriate nutrition, trauma) occurring in this period.

This 2-part series addresses the most common pathologic conditions affecting juvenile dogs and cats, including:

- **Congenital and neonatal orthopedic diseases:** Defined, for these articles, as diseases that occur in the prenatal period or within the first 3 to 4 weeks of life
- **Pediatric bone, cartilage, and joint diseases:** Diseases that occur in the skeletally immature dog.

Part 1 of this series presents an overview of musculoskeletal development and pediatric bone diseases (diseases that occur after 1 month of age and before skeletal maturity), which generally have a good prognosis. Part 2 will discuss pediatric joint and cartilage diseases, as well as congenital and neonatal orthopedic diseases.

DIAGNOSTIC APPROACH

In juvenile patients, determining the correct diagnosis can be challenging because the clinician must understand developmental physiology (**Tables 1 and 2**) and how errors in development affect particular body systems.

To properly diagnose juvenile orthopedic disease, it is important to obtain key information:

- Patient signalment: Age, breed, and sex
- Accurate history, including:
  - » Origin of the dog, which can indicate the general care the dam and puppies received
  - » History of any known trauma, which can help determine the presence of fractures or soft tissue injuries.
- Information about dam, sire, and siblings: Evidence suggests that many juvenile orthopedic conditions have a genetic basis and, thus, can be inherited
- Careful and thorough physical and orthopedic examinations.

Treatment options depend on the disease, and range from medical/nonsurgical management to surgical treatment. Similarly, prognosis is disease dependent and can vary from one patient to the next.

MUSCULOSKELETAL DEVELOPMENT

Embryonic Development

During embryonic development, mesenchymal cells—capable of developing into lymphatic, circulatory, and connective tissues—coalesce in the regions of future bones, progressing from cartilage

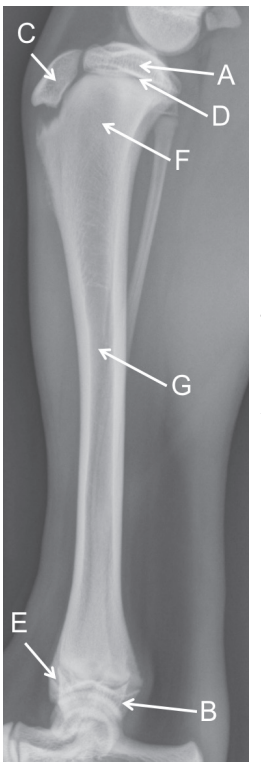

FIGURE 1. Anatomy of an immature long bone (tibia) in the dog: The immature long bone is characterized by radiolucent lines that represent the proximal and distal physeal growth plates (primary centers of ossification; the epiphyses are the locations of secondary centers of ossification). **A** = proximal epiphysis; **B** = distal epiphysis; **C** = apophysis; **D** = proximal physeal plate or physis; **E** = distal physeal plate or physis; **F** = metaphysis; **G** = diaphysis
Courtesy Veterinary Medical Teaching Hospital, University of California–Davis

TABLE 1.
Approximate Closure Times of Major Growth Plates in Long Bones of Average (25- to 30-kg) Dogs[2]

	AGE AT CLOSURE (MONTHS)	
GROWTH PLATE	Earliest	Latest
Hindlimb		
Greater trochanter	6	11
Proximal femur	6	12
Distal femur	6	11
Proximal tibia	6	12
Tibial tuberosity	10	12
Distal tibia	5	11
Medial malleolus	4	5
Proximal fibula	6	11
Distal fibula	5	11
Tuber calcaneus	11 wks	8
Forelimb		
Supraglenoid tuberosity	12 wks	5
Proximal humerus	10	12
Distal humerus	5	8
Proximal ulna	5	8
Anconeal process	4	5
Distal ulna	6	11
Proximal radius	5	8
Distal radius	6	11

TABLE 2.
Approximate Closure Times of Major Growth Plates in Bones of Average Cats[7]

	AGE AT CLOSURE (MONTHS)	
GROWTH PLATE	Earliest	Latest
Hindlimb		
Proximal femur	8	14
Distal femur	14	20
Proximal tibia	14	20
Distal tibia	8	14
Proximal fibula	14	20
Distal fibula	8	14
Ilioischial	4	8
Forelimb		
Proximal humerus	14	20
Distal humerus	4	8
Proximal ulna	8	14
Scapula	4	8
Distal ulna	14	20
Proximal radius	4	8
Distal radius	14	20
Spine	14	20

to bone via endochondral and intramembranous ossification.[1-3] Formation of joints and intraarticular structures follows; mesenchyme surrounding the bones and joints then gives rise to muscles. Tendinous insertions develop from cartilaginous primordia and secondarily connect to developing muscle masses during limb elongation.[1]

Postnatal Development

During postnatal skeletal development, the cartilaginous skeletal components enlarge, and mineralization occurs via intramembranous and endochondral ossification.[1,3]

Longitudinal bone development results from growth of the secondary centers of ossification in the epiphyses and the primary centers of ossification in the physes (**Figure 1**).[1] In these regions, new cartilage forms, increasing the area in length and width.[2]

Endochondral ossification then occurs, transforming the cartilage into bone, which is characterized as lamellar bone with Haversian systems.[3] In most long bones, physeal growth represents 75% to 80% of the final bone length, whereas epiphyseal growth represents 20% to 25%.[1,2]

By the end of this period, mineralization of the epiphyseal centers of ossification is complete and growth continues at the physes at a much slower rate. The cessation or closure of the physeal growth plates occurs at predetermined times and varies widely from bone to bone and across breeds of dogs and cats (**Tables 1 and 2**).[4-6]

Effect of Gonadectomy on Bone Growth

The most physeal activity and, thus, the majority of longitudinal bone growth, occurs from 12 to 26 weeks of age.[1] However, timing of long bone physeal closure is controlled, in part, by gonadal hormones, particularly estrogen, in both sexes.[4] Therefore, in both dogs and cats, gonadectomy before physeal closure delays this closure and is associated with significantly greater final length of associated long bones.[4–6] The role that delayed physeal closure plays in the development of orthopedic disease is an area of current research and discussion.

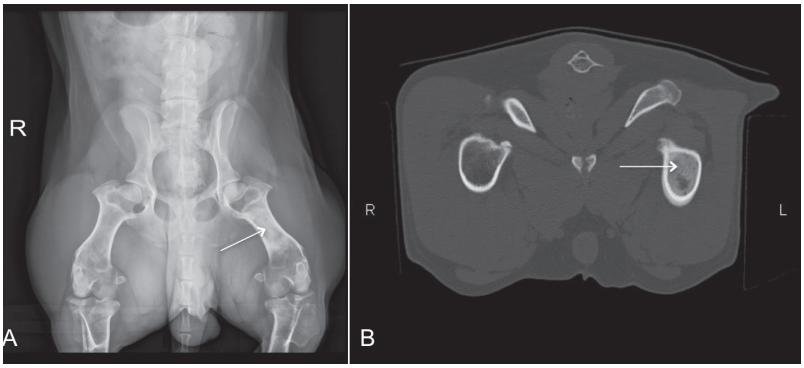

FIGURE 2. Ventrodorsal pelvic radiograph of a juvenile dog with panosteitis affecting the left femur (**A**); the **arrow** identifies nodular opacity similar to cortical bone opacity in the medullary canal. Transverse CT image of the femur of the same patient (**B**); the **arrow** identifies increased opacity/medullary sclerosis of the diaphysis of the left femur (compared with the right femur), which is consistent with panosteitis. *Courtesy Veterinary Medical Teaching Hospital, University of California–Davis*

PEDIATRIC BONE DISEASES

Panosteitis

Overview

Panosteitis—also known as enostosis, eosinophilic panosteitis, juvenile osteomyelitis, and osteomyelitis of young German shepherd dogs—is a self-limiting disease of the long bones of large- and giant-breed dogs. It most commonly affects the ulna, followed by the radius, humerus, femur, and tibia.

The cause of panosteitis is unknown, but it may be the result of excessively high dietary protein or calcium administration that causes protein accumulation and/or vascular proliferation and local bone formation at nutrient foramina, which increases intraosseous pressures.[1]

In one study, incidence was estimated at 2.6 per 1000 patients.[8] Panosteitis also has regional and seasonal distribution differences, with more cases in the Northeast and north-central United States and in the summer and fall.

Signalment

Age at diagnosis is most often 5 to 12 months; however, panosteitis may affect younger dogs and those up to 5 years of age. Males are more commonly affected than females.

Breeds at risk of developing panosteitis include the Airedale terrier, Irish setter, German shorthaired pointer, Doberman pinscher, Afghan, Great Dane, Saint Bernard, Bernese mountain dog, Newfoundland, golden and Labrador retrievers, and German shepherd dog. Some small and medium-sized breeds also appear to be at risk, including the basset hound, Chinese shar-pei, miniature schnauzer, Scottish terrier, and American cocker spaniel.

Weight is a contributing factor, and young dogs weighing more than 23 kg are at increased risk compared with those weighing less than 23 kg. Females and males over 23 kg are 3 and 5 times, respectively, at greater risk.[9]

Diagnosis

Diagnosis is based on signalment, history, physical examination findings, and radiographic findings. There are generally no systemic signs of illness.

Physical examination findings include a shifting leg lameness and pain on palpation of the long bones. In some patients, pain is so severe that it impedes ambulation and can also result in hyporexia or unwillingness to eat and drink. Note that care must be taken during palpation to avoid eliciting pain by palpating/compressing a nerve or other structure.

In the acute phase, radiographs may be normal or show circumscribed cortical bone opacities within the medullary canal adjacent to the nutrient foramina (**Figure 2**). As the disease progresses, the medullary pattern changes to a coarser than normal trabecular pattern, and a progressive periosteal reaction may be present (**Figure 3**). Severity of radiographic signs does not necessarily parallel severity of clinical signs.

Differential diagnoses include hypertrophic osteodystrophy, osteochondritis dissecans, hip dysplasia, fragmented medial coronoid process, and united anconeal process.

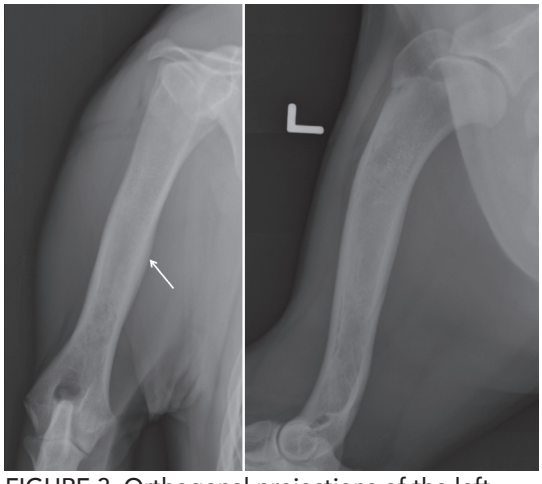

FIGURE 3. Orthogonal projections of the left humerus of a juvenile dog with a shifting limb lameness of 1.5 months' duration that was diagnosed as panosteitis; note the patchy increased medullary opacities that are visible throughout the length of the humeral diaphysis and smooth periosteal proliferation of the medial aspect of the diaphysis (**arrow**). *Courtesy Dr. Erik Wisner, University of California–Davis*

Treatment

In general, treatment does not appear to affect outcome.[5] Therapy includes exercise restriction and analgesics while the condition resolves. Nonsteroidal anti-inflammatory drugs (NSAIDs) are often sufficient to manage pain, but patients in severe discomfort require hospitalization with injectable opioid agonists and intravenous fluid support. It is generally accepted that steroid and antibiotic administration is not necessary and should be avoided.

Recurrence is possible, although severity appears to diminish as the patient ages. The main recommendation when attempting to treat and prevent panosteitis is ensuring that affected dogs and at-risk puppies are fed an appropriate, complete, and balanced large-breed puppy diet without excessive protein.

Prognosis

Prognosis is generally good to excellent. Few dogs have long-term ramifications from panosteitis.

Hypertrophic Osteodystrophy

Overview

Hypertrophic osteodystrophy—also known as skeletal scurvy, canine scurvy, Moller-Barlow's disease, osteodystrophy type 2, metaphyseal osteopathy, and metaphyseal dysplasia—is a developmental disease of young large- and giant-breed dogs. The most commonly affected bones are the radius, ulna, and tibia, and the disease is often bilateral.

Although several causes have been suggested in the literature, no single cause of hypertrophic osteodystrophy has been identified. Infectious causes, including canine distemper virus and *Escherichia coli*, have been suspected but not validated.

Incidence is estimated at 2.8 per 100,000 cases.[9,10] Similar to panosteitis, hypertrophic osteodystrophy has geographic and seasonal distribution, with more cases in the northeastern U.S. and in the fall.

Signalment

Age at diagnosis is typically 2 to 6 months.[9,10] Male dogs appear to be predisposed compared with females. At-risk breeds include the Great Dane, Chesapeake Bay retriever, Irish setter, boxer, German shepherd dog, golden and Labrador retrievers, and Weimaraner; this disease is heritable in Weimaraners.[11]

Diagnosis

Diagnosis is based on signalment, history, physical examination findings, and radiographic findings.

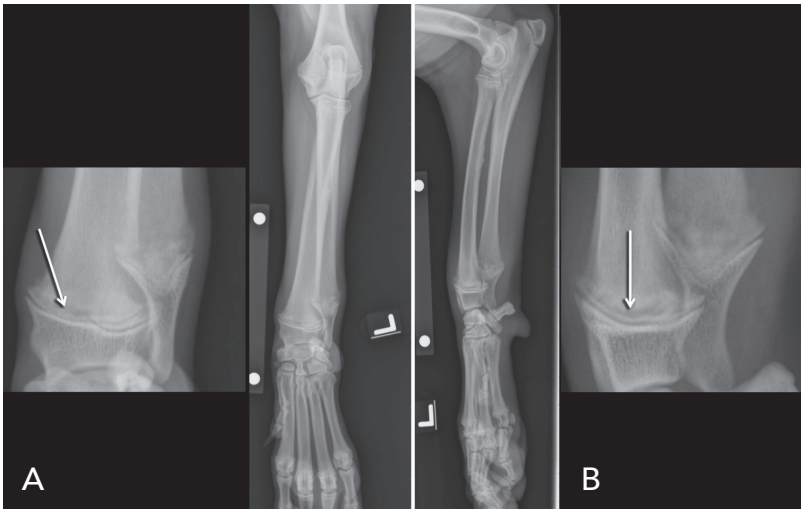

FIGURE 4. Craniocaudal (**A**) and lateral (**B**) views of the left radius/ulna of a juvenile dog with hypertrophic osteodystrophy; both are enlarged views of the affected metaphysis. The **arrows** identify mild sclerosis and heterogeneity of the distal radial and ulna metaphyses (**A**), and faint irregular lucent lines in the distal metaphysis of the radius and ulna (**B**).
Courtesy Veterinary Medical Teaching Hospital, University of California–Davis

Hypertrophic osteodystrophy is characterized by painful swelling of the metaphyseal region of long bones in the appendicular skeleton. Patients are often systemically ill with fever, lethargy, inappetence, or diarrhea.

Radiographs are critical in confirming the diagnosis:

- The condition is characterized by a lucent line in the metaphyseal region parallel to the physis, also referred to as a *double physis*.[12]
- Periosteal or endosteal reaction in the region of the physis is typically present (**Figure 4**).
- Subperiosteal hemorrhage can result in new bone formation, which is noted palisading along the periosteum and, in some patients, bridging the physis (**Figure 5**, page 42).

The severity and duration of the disease influence the extent of new bone formation and, in some patients, may extend to the level of the diaphysis.[12]

When affected bone is examined histologically, necrosis of trabecular bone, inflammatory cellular infiltration, hemorrhage, and hemosiderin deposits are present. Subclinical fractures are common. Subperiosteal hemorrhage not only leads to new bone formation, but can also result in a synostosis; both can lead to disturbance of appropriate growth and resulting deformities.[1,9]

Differential diagnoses include septic arthritis, septic physitis, secondary nutritional hyperparathyroidism, retained cartilage cores, hypertrophic osteopathy, and panosteitis. It is important to differentiate this

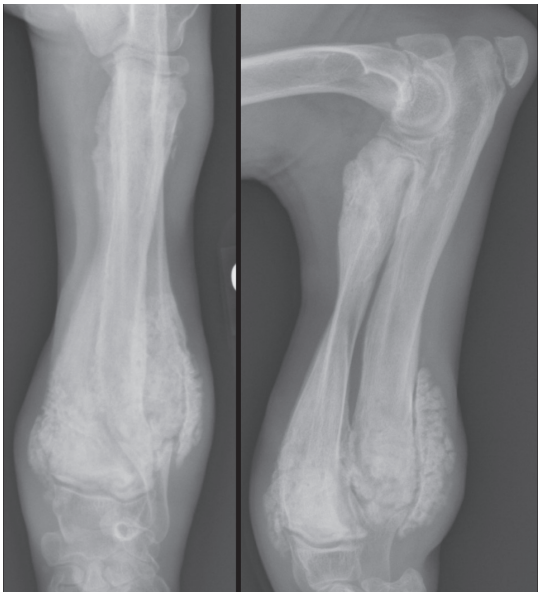

FIGURE 5. Orthogonal projections of the right radius and ulna in a 4-month-old dog diagnosed with hypertrophic osteodystrophy; note the marked, irregularly marginated periosteal proliferation that is present circumferentially, involving the distal metaphyses of the radius and ulna. Both projections show bridging of the distal ulnar physis. *Courtesy Dr. Erik Wisner, University of California–Davis*

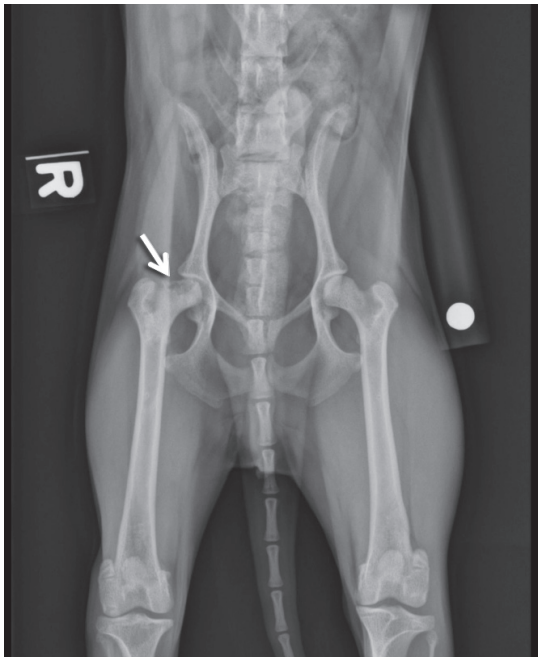

FIGURE 6. Ventrodorsal pelvic radiograph of a juvenile dog with avascular necrosis of the right femoral head; note the patchy areas of radiolucency present within the right femoral head and neck (**arrow**), with narrowing of the femoral neck. *Courtesy Veterinary Medical Teaching Hospital, University of California–Davis*

disease from hypertrophic osteopathy, which is characterized by deposition of periosteal new bone, most typically in the distal extremities and long bones. Hypertrophic osteopathy is thought to be a secondary disorder most often associated with pulmonary neoplasia (primary or metastatic).[5]

Treatment

Hypertrophic osteodystrophy is most commonly self-limiting in days to months; however, signs may last for months. For mild cases, supportive care with NSAIDs is recommended, while severe cases may require hospitalization for more aggressive supportive care, fluid and nutritional therapy, and injectable opioid analgesics.

Affected patients should consume a complete and balanced, age-appropriate diet. Vitamin C and vitamin D therapy have been described, but there is no evidence that supplementation is helpful in disease treatment.

Blood cultures are indicated for immunocompromised patients. If cultures are positive, appropriate antibiotic support may be warranted.[13]

A report in 6 Weimaraner puppies noted a better response when corticosteroids were used; however, one must be prudent when applying this information to the general population of dogs because 5 of the 6 puppies in this report were genetically related.[10]

Prognosis

In mildly affected patients, the prognosis is excellent, but in severely affected patients, the disease may be fatal. Recurrence is possible, usually within months of the primary episode. As noted previously, development of angular limb deformities is possible in severe cases and owners should be warned of this possibility. Client education is essential because they will need to monitor the pet for growth deformities.

Avascular Necrosis of the Femoral Head

Overview

Avascular necrosis of the femoral head—also known as Legg-Calvé-Perthes disease and osteochondritis dissecans of the femoral head—is characterized as noninflammatory local ischemia of the femoral head and neck, resulting in deformation and lameness.

The vascular nature of this disease is suspected due to pathologic changes, which include necrosis of the trabecular bone followed by fragmentation

after continued loading of the joint. Bilateral disease occurs in 12% to 16.5% of dogs.[9]

In addition to a potential hereditary component (see **Signalment**), other possible causes include conformation, infarction of the femoral head, hormonal influence, and increased intracapsular pressures, although none have been uniformly accepted as a definite underlying cause.

Signalment

Most dogs present between 4 and 11 months of age, and there is no sex predilection. Small-breed dogs are most commonly affected, with toy and terrier breeds being at increased risk. Due to the strong breed predilection, a hereditary component is suspected. In miniature poodles and West Highland white terriers, the trait is autosomal recessive; therefore, owners of affected puppies should be counseled against breeding.

Diagnosis

Diagnosis is based on signalment, history, physical examination findings, and radiographic findings.

Physical examination findings include a mild to severe, non–weight-bearing lameness of the pelvic limbs. Mild cases may be subclinical; therefore, careful and thorough orthopedic examination is essential.

Radiographs are usually diagnostic (**Figure 6**), but some chondrodystrophic breeds are challenging. Computed tomography (CT) may be advised. Similarly, flexed ventrodorsal ("frog-leg") radiography of the hip/pelvis may be preferred to the traditional extended-hip radiography.

Radiography or CT shows progressive radiopacity of the lateral epiphyseal area of the femoral head, followed by lysis of the femoral head in a "moth-eaten" or "apple-core" appearance. After lysis, the femoral head flattens, creating the potential for femoral neck fractures.

Differential diagnoses include capital physeal trauma, epiphysitis, septic physitis, osteomyelitis, and neoplasia.

Treatment

In less than 25% of cases, lameness resolves with rest and NSAID therapy.[5] With this in mind, avascular necrosis of the femoral head is most often considered a surgical disease. Surgical options are femoral head and neck ostectomy or total hip replacement.

Given the lytic appearance of the femoral head, samples should be submitted for culture and histopathologic assessment to rule out osteomyelitis and neoplasia.

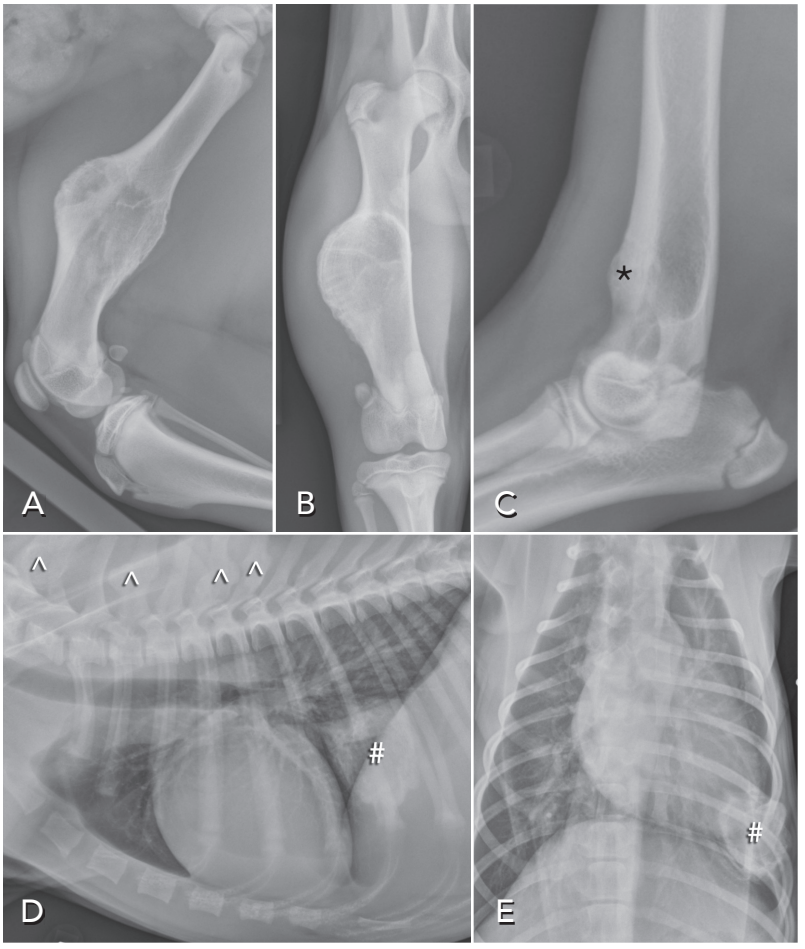

FIGURE 7. Radiographs of a dog diagnosed with multiple cartilaginous exostosis: Lateral (**A**) and craniocaudal (**B**) views of the right femur; note the expansile lesion visible in the mid-diaphysis with marked cortical thinning. Minimal smooth periosteal reaction is visible surrounding this lesion. Lateral view of the right distal humerus (**C**); note the smooth, mildly expansile lesion visible at the cranial aspect of the distal humerus (*****). Lateral (**D**) and dorsoventral (**E**) views of the thorax; note the well-defined, rounded expansile lesion associated with the seventh rib on the left side (**#**). This lesion appears to impinge on the lung margin. Multiple well-defined expansile and rounded lesions are visible in the spinous processes of the cranial thoracic spine (**^**). *Courtesy Dr. Dan Bucy, Veterinary Medical Teaching Hospital, University of California–Davis*

After femoral head and neck ostectomy, early return to function and aggressive physical rehabilitation are not only encouraged, but essential to a successful outcome.

Prognosis

Postoperative prognosis is good, with lameness resolving in 84% to 100% of cases.[5] Physical rehabilitation after surgery may help ensure a positive outcome.

Multiple Cartilaginous Exostosis

Overview

Multiple cartilaginous exostosis—also known as osteochondromatosis and multiple hereditary osteochondromata (or osteochondroma, if singular)—is a benign bone disease of multiple, cartilage-capped bony protuberances that arise from the surface of any bone formed by endochondral ossification.

The bones most frequently affected are vertebrae, ribs, and long bones. The cause is unknown, and the condition may affect both dogs and cats. Exostosis may undergo malignant transformation to chondrosarcoma or osteosarcoma.[14,15]

Signalment

There are no known sex or breed predilections, although Great Danes, Saint Bernards, and hounds appear overrepresented.

In dogs, the disease is inherited as an autosomal dominant trait, and is seen in young, growing patients. Exostosis appears and enlarges before skeletal maturity.

In cats, the disease has been associated with feline leukemia virus. Exostosis occurs more commonly after skeletal maturity.

Diagnosis

Diagnosis is based on physical examination and radiographic findings, with excisional biopsy and histologic examination important for definitive diagnosis.

Physical examination findings depend on the location of the lesions and may indicate interference with locomotion. Pain may be present if exostosis is associated with a tendon, ligament, vessel, or spinal cord compression.

Radiographic findings include single or multiple bony masses with a thin cortex and a medullary cavity that is confluent with the host bone and has a distinct trabecular pattern (**Figure 7**, page 43). In dogs, growth of the exostosis should cease with closure of the adjacent growth plate and, in animals diagnosed with exostosis, full-body radiography is recommended as a monitoring tool.

Treatment

Treatment depends on size and location of the lesion and associated clinical signs. Nonsurgical management may be attempted and includes rest and NSAID analgesics. Surgical management is often indicated for single or large exostosis, specifically those compressing the spinal cord.

Prognosis

Prognosis depends on the site and size of the lesions, ranging from excellent to guarded. In dogs, the prognosis is better if lesions stop increasing in size at cessation of growth. After malignant transformation, prognosis may be poor, depending on location and type of tumor.[15]

Slipped Capital Femoral Epiphysis

Overview

Slipped capital femoral epiphysis—also known as femoral neck metaphyseal osteopathy, spontaneous femoral capital physeal fracture, and femoral capital physeal dysplasia syndrome—is nontraumatic, slow, progressive displacement of the proximal femoral metaphysis from the femoral epiphysis through the physis.

This disease is most common in cats but has also been reported in dogs, and the cause is unknown. It is not the same disease as a Salter-Harris type 1 fracture of the femoral capital physis, which is acute and traumatic in origin.

Signalment

The most common age at presentation is 4.5 to 42 months. Maine coon and Siamese cats may be overrepresented; in one study, Maine coon cats were 12 times more likely than other breeds to develop this condition.[9,16] Overweight, neutered male cats appear to be at risk.[9,17]

Diagnosis

Diagnosis is based on signalment, history, physical examination findings, and radiographic findings.

History and physical examination findings include acute or chronic pelvic limb lameness, pain on manipulation of the coxofemoral joint, and an inability to jump. Approximately one quarter of cases are bilateral.

Radiographic findings include a widening and lateral displacement of the capital femoral growth plate with progressive resorption and sclerosis of the femoral neck.

Treatment

Surgical treatment is indicated and results in better long-term outcome than medical management.

- Early in the disease process, reduction with internal fixation is possible with use of multiple divergent wires to stabilize the physis.
- With more chronic disease, femoral head and neck ostectomy or total hip replacement is indicated.

Among surgical patients, post discharge analgesia may consist of sublingual buprenorphine or oral tramadol. After femoral head and neck ostectomy, early return to function and aggressive physical rehabilitation should be encouraged and are essential to a successful outcome.

Prognosis

Prognosis with surgical treatment, assuming no surgical complications, is good with primary fixation in acute cases or femoral head and neck ostectomy or total hip replacement in chronic cases.[17-19] Complications associated with a particular surgery are outside the scope of this article and should be completely discussed with the owner before surgery. Physical rehabilitation after surgery may help ensure a positive outcome.

IN SUMMARY

Proper musculoskeletal development must proceed in an ordered manner. Deviation from normal development as a result of insults to growing bones in utero or after birth may lead to a variety of orthopedic diseases affecting juvenile bones.

These diseases are often diagnosed according to patient signalment, history, physical and orthopedic examination findings, and appropriately positioned radiographs. Although treatment of the diseases varies, many affect multiple limbs. Therefore, attention should be paid to appropriate analgesia as described in both medically and surgically managed patients.

The prognosis of juvenile bone diseases is generally good, although surgery may be required for some conditions to achieve the best outcome. Due to strong breed predilections for many of these conditions, until the specific cause of the disease is fully elucidated, it may be wise to counsel owners of affected individuals against breeding.

CT = computed tomography; NSAIDs = nonsteroidal anti-inflammatory drugs

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INGRID BALSA

Ingrid Balsa, MEd, DVM, is a small animal surgical resident at University of California–Davis School of Veterinary Medicine. Dr. Balsa's research interests include minimally invasive surgery and the medical management of surgical diseases. She received her DVM from University of Minnesota and completed a small animal rotating internship at North Carolina State University.

DUANE ROBINSON

Duane Robinson, DVM, PhD, Diplomate ACVS (Small Animal), is an assistant professor of orthopedic surgery at University of California–Davis School of Veterinary Medicine. He received his DVM from University of Guelph Ontario Veterinary College and his PhD (infectious diseases) from University of Minnesota. He completed a rotating internship in medicine and surgery at Ontario Veterinary College; surgical internship at Affiliated Veterinary Specialists, Orange Park, Florida; research fellowship at Iowa State University Veterinary Orthopedic Research Laboratory; and surgery residency at University of Minnesota.