Canine Lameness Caused by Developmental Orthopedic Diseases: Osteochondrosis

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ABSTRACT: Osteochondrosis, a common developmental orthopedic disease in immature dogs, is caused by a disturbance in the process of endochondral ossification. The cause is undetermined, but numerous factors, including growth rate, genetics, and nutrition, have been implicated. Osteochondrosis typically affects the shoulder, elbow, stifle, or hock joint, causing lameness, joint effusion, and osteoarthritis. Clinical signs can develop in dogs as young as 4 months of age. Surgical treatment is recommended in most cases to debride the abnormal cartilage and encourage the ingrowth of repair tissue. NSAIDs, physical rehabilitation, and disease-modifying osteoarthritis agents are recommended after surgery to restore joint function. Medical management alone is often unrewarding.

Developmental orthopedic diseases (DODs) are a common cause of lameness and pain in young dogs. This group of spontaneous disorders of undetermined etiology commonly involves paired joints or bones of the skeleton in growing dogs. The resultant spectrum of structural changes in the limbs results in discomfort to the dog, and thus pet owners seek veterinary care for medical treatment or surgical correction.

DODs should be distinguished from the osteochondrodysplasias that produce generalized skeletal deformity involving bones and joints. Skeletal lesions of osteochondrodysplasia are the result of genetic defects in cartilage tissue of the cartilage model (e.g., achondroplastic dwarfism) and genetic defects in the cellular elements of bone tissue (e.g., osteopetrosis or osteogenesis imperfecta).

Because a variety of abnormalities are included in DODs, establishing a diagnosis can be challenging. Thorough knowledge of the patient’s signalment and history and a complete physical examination, including orthopedic and neurologic examinations, are essential to help localize the disease, identify all of the abnormalities present, and establish a differential diagnosis (see box on p. 837). Radiography is generally required to confirm the diagnosis of DOD and, in some cases, magnetic resonance imaging, computed tomography, or nuclear scintigraphy (bone scan)
provide useful information. A thorough understanding of the disease etiology, pathophysiology, and progression is needed to recommend the appropriate medical and surgical treatments. This article describes articular osteochondrosis (OC), outlining clinicopathology with an emphasis on pathogenesis, diagnosis, treatment, and prognosis.

**OSTEOCHONDROSIS OVERVIEW**

**Definition and Character of Lesion**

OC is a disturbance in the process of endochondral ossification. The term has been applied to a variety of disorders of the cartilage model in which the initial lesion is a focal injury of microscopic dimension that is located within the osteochondral junction of a developing articular surface of a weight-bearing joint or of both members of a joint pair. Only certain joints are affected, and lesion location in those joints is specific. The focal disturbance in the lesion of OC affects the growth and development of a normally shaped bony epiphysis or articular process covered by a smoothly contoured layer of articular cartilage of normal thickness and histologic structure. Loss of normal contour and sclerosis of the underlying subchondral bone plate are radiographic features of early symptomatic and asymptomatic lesions of articular OC before the effects of secondary degenerative joint disease alter radiographic images. Puppies from small breeds are not generally affected, whereas puppies from predisposed breeds are large, have rapid growth rates, and are often on high planes of nutrition.

Unfortunately, the term osteochondrosis has been altered and expanded from its original use in humans to describe a traumatic articular lesion. This type of joint lesion was spontaneously induced by blunt or rotary trauma to articular cartilage of the femoral condyle. Subsequently, early medical and veterinary surgeons progressively included, under the umbrella term osteochondrosis, almost any lesion in the cartilage model of humans and animals in which there is a focal delay or disruption of endochondral ossification. This large group of orthopedic conditions includes disorders of endochondral ossification centered in the growth plates of long bones, cuboidal bones, and apophyses. This discussion limits the use of the term osteochondrosis in dogs to lesions of the articular surface and does not include physeal lesions of long or cuboidal bones.

**Endochondral Ossification**

In normal long bone development, the growth plate located in the deep layer of the articular surface (i.e., epiphyseal physis) forms the cancellous bone of the epiphysis by endochondral ossification in a manner similar to that of the metaphyseal physis, which forms the metaphyseal spongiosa of the diaphysis. The most superficial layer of the enlarging epiphysis is the tangential layer (Figure 1), which forms the gliding articular surface but does not contribute to bone formation. The three deeper zones (transitional zone, radial zone, and the zone of calcified cartilage) are equivalent to the resting, proliferative, hypertrophic, and calcifying zones of the metaphyseal physis. However, chondrocytes and cartilage matrix of the several zones of the epiphyseal physis are retained at the time of skeletal maturity to form the joint surface, whereas the elements of the metaphyseal physis are removed during metaphyseal closure.

The purpose of endochondral ossification is to produce an expanding cartilage model of a bone or joint surface that undergoes orderly calcification, vascular invasion by osteogenic granulation tissue, and replacement of cartilage by bone tissue. During development of a long bone, the rate of cartilage production is matched by bone replacement until skeletal maturity, when the cartilage of metaphyseal physis is completely replaced by bone.

In articular cartilage, however, the interstitial growth of articular cartilage and its replacement by bone cease at skeletal maturity. Chondrocytes in the former proliferative zones of articular cartilage now maintain the joint surface and transfer energy to the underlying subchondral bone. What had formerly been a diffuse zone of calcified cartilage becomes a narrow zone that is a few chondro-
cytes in thickness. The microscopic demarcation between the superficial layers of uncalcified cartilage matrix and the deeper zone of calcified cartilage is recognized by the formation of one or more thin parallel lines called *tidemarks* (Figure 1). At skeletal maturity, the thin zone of calcified cartilage rests upon a thin cementing line that interdigitates with a continuous plate of subchondral bone.

In OC, the process of endochondral ossification is disrupted in a focal area of a developing articular surface centered at the osteochondral junction. Lesions are often bilaterally symmetric. The cartilage in the affected site fails to undergo physiologic calcification and replacement by bone, thereby leaving a thickened focal area of degenerative cartilage. The pathogenesis of this lesion is unclear (i.e., Is the initial lesion cartilaginous or vascular in origin?). It has long been proposed that chondrocyte degeneration in these deep articular lesions is due to failure of nutrient diffusion because of increased distance from the joint fluid on the joint surface. This theory does not indicate what caused the focal thickening of the cartilage in the first place.

A more reasonable explanation is that the bilateral lesions result from focal compression of the articular cartilage caused by conformational forces. When forces that are transmitted through the articular cartilage to the osteochondral junction exceed the capillary profusion pressure of the underlying osteogenic capillary bed in the subchondral spongiosa, vascular invasion and replacement of the cartilage matrix are slowed. The retained cartilage degenerates and is not physiologically prepared for mineralization. The immediately underlying and previously osteogenic capillary bed is diverted from bone to fibrous tissue production and forms a sheet-like barrier beneath the focal lesions of chondromalacia. Whatever the initiating cause, the OC lesion is a disruption of the normal developmental process of endochondral ossification.

When there is uncoupling of the sequence of endochondral ossification, the area of necrotic cartilage and fibrous tissue is vulnerable to even mild trauma and shearing forces encountered in normal weight bearing. If an episode of mechanical osteochondral separation occurs earlier in skeletal development before tidemarks have formed, a cleft will develop through this fibronecrotic

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**Figure 1A**

**Figure 1B**

*Figure 1—* Schematic (A) and histologic (B) representation of the different regions of articular cartilage. The middle and deep regions are separated by the *tidemark*, a thin line that separates noncalcified tissue from calcified tissue. (Figure 1A was illustrated by Julia Heard.)
interface. However, if the focal interface lesion occurs later, at or about the time of skeletal maturity when one or more of the linear tidemarks have formed between the uncalcified and calcified cartilage, a shearing force will follow along the tidemark for a variable distance.

The line of dissection developing at the osteochondral junction or tidemark may extend out through the superficial layers of uncalcified cartilage matrix to the articular surface, where it forms a flap. This lesion is referred to as osteochondritis dissecans (OCD). The fissure or cleft that forms can extend from the articular surface to the tidemark or osteochondral junction, depending on the age of the pup. This communication allows cartilage degradation products to reach the synovial fluid, causing synovitis, effusion, joint pain, and lameness. The resultant flap of cartilage may remain within the defect or may become dislodged. Cartilage flaps that remain in the defect may reattach to the underlying subchondral bone, as blood in the tidemark region allows the ingrowth of fibrous tissue and vascularized mesenchyma.

In many cases, however, the flap breaks free. These loose bodies of cartilage within the joint are referred to as joint mice. These bodies are sometimes resorbed in synovial recesses by granulation tissue arising from the adjacent irritated synovium. In other cases, they receive nutrients from the synovial fluid and may enlarge by radial growth of viable chondrocytes at the periphery of the nodule while central chondrocytes undergo necrosis and the matrix is mineralized (Figure 2). Most attached joint mice that contain cancellous bone do not revascularize their bony component but develop a foreign body reaction containing osteoclasts around necrotic bone fragments. Other cartilaginous nodules firmly attached to the synovium are found on histopathologic examination to be synovial chondromas. Synovial chondromas are cartilaginous nodules arising by metaplasia of chronically irritated synoviocytes. Unfortunately, joint mice contribute to synovitis and osteoarthritis (OA) within the joint.

Two types of OC lesions have been described:

- **Type I (classic form) lesions** occur in or near the center of a convex joint surface, and the resulting cartilage flap has no contact with vascularized tissue, such as the joint capsule or ligaments. Type I lesions in dogs include OCD of the humeral head, the medial condyle of the humerus, and the femoral condyle.

- **Type II lesions** occur at the periphery of a joint surface in direct contact with the joint capsule or ligaments. Type II lesions in dogs include OCD of the talus.

Four gradations of OCD lesions have been described:

- **Grades I to III** are rarely diagnosed in dogs because of an absence of clinical signs. OC in dogs is usually not painful until a fissure or fracture reaches the articular surface of the cartilage. In Grade I (the mildest form), the articular surface is grossly normal but a small defect is noted in the subchondral bone.

- **Grade IV** is the most common form of OCD in dogs presented with lameness. In this grade, a vertical fissure or fracture of the articular cartilage and separation of the cartilage flap from the subchondral bone are present.

**Etiology and Incidence**

The cause of OC has not been determined, but a complex of factors, including genetics, rapid growth, overnutrition, excess dietary calcium, trauma, ischemia, and hormonal influences, has been implicated. OC is most commonly observed in rapidly growing, large- and giant-breed male dogs. The dis-
The most common clinical sign of shoulder OC is mild to moderate unilateral forelimb lameness. Bilateral lameness is uncommon, even if both shoulders are affected. Owners usually report a gradual onset of lameness that improves after rest and worsens with exercise. Dogs typically stand with the affected or more painful limb somewhat externally rotated, with the elbow abducted. Joint capsule swelling along the bicipital groove and mild atrophy of the supraspinatus, infraspinatus, and deltoideus muscles are seen occasionally. Hyperextension and flexion of the scapulohumeral joint usually elicits a painful response. Care should be taken to avoid manipulating the elbow joint while testing the shoulder for pain. Elbow disease and panosteitis may occur concurrently with shoulder OC in young dogs.

Diagnosis
A presumptive diagnosis of shoulder OC is based on the history and physical examination, but radiographs are necessary to confirm the diagnosis. Radiographs of both scapulohumeral joints should be taken. The typical OCD lesion appears as a subchondral bone defect in the caudal aspect of the humeral head (Figure 3). Most lesions are readily observed on the mediolateral radiographic view because the lesion is centrally located on the caudal humeral head. Right and left limbs are equally affected.
Despite the radiographic appearance of bilateral lesions, most dogs demonstrated lameness in only one limb, with only 21% of dogs with bilateral radiographic lesions exhibiting signs in both forelimbs. If no apparent mineralized cartilage flap is visible, the best way to distinguish OC from OCD radiographically is to perform contrast arthrography. This can be helpful in confirming the presence of free flaps within the biceps tendon sheath and in diagnosing additional articular cartilage defects. (Figure 4).

**Medical Treatment**

Conservative therapy may be indicated in dogs younger than 7 months of age with small lesions evident radiographically and no clinical pain or joint mice. Conservative treatment of dogs with clinical signs of lameness and joint pain is often unrewarding. Conservative therapy consists of strict rest for up to 6 weeks, restricted diet, NSAIDs, disease-modifying osteoarthritis (DMOAs), and analgesics. Alterations of the diet include decreasing caloric intake and stopping calcium supplementation. Appropriate nutrition and breeding programs are essential for minimizing the occurrence of OC. If lameness resolves with medical therapy, surgery may not be required. However, if lameness persists for more than 4 to 6 weeks, surgery should be performed. In the past, authors have recommended analgesics, anti-inflammatories, and forced exercise with the intention of dislodging the cartilage flap and thus facilitating healing. The hope was that the flap would fall into the caudal cul-de-sac of the joint and be resorbed. Unfortunately, flaps are often not resorbed but instead remain in the joint and incite an inflammatory response.

**Surgical Treatment**

Surgical treatment is recommended in dogs if a flap is present, the dog has been lame for more than 6 weeks, the dog is older than 8 months of age, a joint mouse is evident radiographically, or the lesion is large. In our experience, most dogs with clinical signs of pain and lameness do not respond to medical management and early surgical intervention is preferred. Surgical treatment provides a more rapid return to function and minimizes the development of OA.

Many approaches to the scapulohumeral joint have been described, but most surgeons use a cranial, lateral, or caudal approach (the latter two are more common). The cranial approach to the shoulder requires tenotomy of the infraspinatus tendon and provides excellent exposure to 62% of the articular surface with minimal retraction. Unfortunately, the tenotomy incision often heals by fibrosis and could affect joint range of motion after surgery. Muscle-separating approaches require more intraoperative retraction and are more difficult to perform without surgical assistance. One caudal approach (infraspinatus and teres minor muscles retracted craniodorsally) was found to expose 23% to 48% of the articular surface, whereas a second technique (infraspinatus and teres minor muscles separated and retracted individually) exposed 35%. (Figure 5). Both were considered ade-
quate for treating most OCD lesions. The caudomedial aspect of the joint can be reached when the muscles are retracted craniodorsally, which has been reported as an advantage of the caudal approach. Because muscle-separating approaches are less invasive and cause less periarticular fibrosis than approaches that involve tenotomies, they are preferred in athletic dogs.

The use of arthroscopy to explore the shoulder joint, remove cartilage flaps, and curette and forage the defect has become more common. Arthroscopy is less invasive and allows complete exploration of the joint and excellent lavage. It is technically more difficult and requires specialized equipment and training. In humans, arthroscopic lavage has been one of the most basic traditional techniques for treating subchondral defects. However, long-term clinical results were generally insufficient for athletic or young patients, providing only short-term symptomatic relief. In humans, debridement has been shown to be superior to lavage.

In veterinary medicine, arthroscopy, when available, has become the treatment of choice for shoulder OCD lesions. The objectives of surgery are to remove the cartilage flap or joint mice, remove cartilage in the periphery of the lesion that is not adhering to underlying tissue, and stimulate the defect to heal by fibrocartilage formation. Healing of the defect requires bleeding from the subchondral bone to bring in mesenchymal cells and a fibrin clot. Fibrocartilage eventually fills the defect.

Three methods commonly used in veterinary medicine to expose the bleeding subchondral bone are curettage, forage, and abrasion arthroplasty. Curettage, also called debridement, is commonly recommended to remove necrotic cartilage from the defect and allow ingrowth of repair tissue. If the bed of the lesion is sclerotic, the curette may be used to access the subchondral bone until bleeding occurs. However, the lesion should not be over-curetted. Doing so reduces the quality of the repair tissue, delays healing, and removes calcified cartilage that contributes to healing. Therefore, many surgeons prefer forage or abrasion arthroplasty.

Forage (subchondral drilling) uses a small Kirschner wire to drill numerous small, shallow holes into the subchondral bone plate beneath the bed of the defect. Forage allows neovascularization without disturbing the cartilage elements already present in the defect bed. This technique is particularly useful in cases in which the subchondral bone is eburnated and curettage is difficult. Some believe that forage provides better long-term results than curettage by allowing the repair tissue to anchor in the small holes.

In abrasion arthroplasty, the defect bed is simply abraded (usually to a depth of 1 to 3 mm) until bleeding occurs. This is usually done using a small bone curette. The edges of the cartilage defect should be checked to ensure...
that there are no loose fragments. The edges should be perpendicular to the surface of the cartilage and not beveled. Beveling the edges makes the wound larger and creates a partial-thickness cartilage defect, which will not heal well. Once the flap is removed, the defect is treated, the edges are addressed, joint mice are removed, and the joint is copiously lavaged.

Additional techniques for treating OC lesions, including microfracture, mosaicplasty, perichondrial/periosteal grafting, and chondrocyte transplantation, have been reported in humans and animals. Microfracture is a similar stimulation technique in which the subchondral bone is exposed and gently abraded but left intact while adjacent cartilage is debrided to healthy cartilage. The subchondral bone is then broached using small picks or awls so that it communicates with the marrow and clot formation is stimulated. Mosaicplasty involves using special instrumentation to harvest sections of autogenous osteochondral cartilage and implanting them in mosaic fashion. The transplanted, small-diameter, cylindrical grafts are taken from a non–weight-bearing surface. They have been shown to incorporate at the insertion sites without evidence of loosening or subsidence. Human studies have demonstrated that perichondrium taken from the cartilaginous covering of a rib could be placed in a joint, where it would develop into hyaline cartilage. Animal studies have shown that neochondrogenesis of hyaline cartilage is also possible using autologous periosteal or perichondral grafts sutured or glued with the cambium layer facing the joint. Autologous chondrocyte implantation has shown success in reconstituting cartilage, indicating that the implanted cells were partly or fully responsible for repairing subchondral tissue defects. A majority of these techniques have been used primarily in lesions involving the human knee. Animal studies were initially conducted, and several studies in horses and sheep were later documented, but all of these techniques remain in their infancy with small animal patients.

Postoperative Care
After surgery, the dog’s activity is limited for 4 weeks to allow adequate healing of the incision and ingrowth of repair tissue into the defect. Mild physical therapy, such as passive motion exercises and swimming, are often recommended. Postoperative complications may include seroma formation, wound dehiscence, infection, and chronic lameness due to preexisting OA or joint mice that were not retrieved. Seroma formation is reduced by careful hemostasis, appropriate tissue-handling techniques, elimination of dead space in the closure, and controlled exercise after surgery. Failure to completely close the joint capsule has not been shown to contribute to seroma formation.

Prognosis
The prognosis after surgical treatment of shoulder OCD is good to excellent, although mild OA often develops in the shoulder over time. One study retrospectively evaluated 44 dogs after surgery and found that 75% were not lame, 23% were minimally lame, and only 2% were consistently lame 3 years after surgery. Older dogs with chronic lameness and OA have a more guarded prognosis; however, most dogs return to normal function within 4 to 8 weeks after surgery.

OSTEOCHONDROSIS OF THE ELBOW
OC of the cubital joint occurs less commonly than shoulder OC. However, the clinical presentation is similar in both diseases. Affected dogs are usually large to giant breeds, with the most common breeds being the Bernese mountain dog, Labrador retriever, and golden retriever. Ages of onset and presentation for clinical signs vary, but most dogs are younger than 1 year of age. Lameness most commonly develops at 5 to 7 months of age. Males are more commonly affected than females by a 2:1 ratio. Right and left elbows are equally affected, and bilateral disease occurs in 20% to 50% of patients.

Clinical Signs
Owners typically report that the dog appears stiff in the morning or after periods of rest. A mild or intermittent lameness is present that becomes more appreciable after strenuous activity. On physical examination, the dog may stand with the affected elbow slightly adducted. Pain may be elicited on extension and lateral rotation of the elbow. A decrease in the range of motion, particularly during flexion, is indicative of secondary OA. Rarely is muscle atrophy observed, but crepitation can be palpated. Joint effusion and periarticular swelling may be observed, especially if OA is present. It is important to avoid manipulation of the shoulder and carpal joints while evaluating the elbow for pain. Pain can also be elicited during manipulation of the forelimbs in dogs with concomitant skeletal lesions of canine panosteitis.

Diagnosis
A diagnosis of elbow OC is often confirmed with radiography. Several radiographic views, including mediolateral, flexed mediolateral, and craniocaudal
(with elbow flexed 90° and slight medial rotation), are needed to identify an OC lesion. Standard practice should include views of both elbows because of the propensity for this condition to occur bilaterally. Definitive radiographic diagnosis of OCD is made when a radiolucent concavity is observed on the distal trochlear ridge of the medial humeral condyle. It can be extremely difficult to distinguish an OCD lesion on the medial humeral condyle from a “kissing lesion” on the condyle caused by a fragmented medial coronoid process. A “kissing lesion” is often narrower and longer than an OCD lesion, but radiographic differentiation is difficult. Computed tomography can improve or enhance radiographic diagnosis.

**Treatment**

The treatment options for OCD of the elbow include medical management or surgery. Medical therapy consists of rest, NSAIDs, and DMOAs and is used primarily for small lesions. Surgical therapy involves removal of the cartilaginous flap with or without curettage of the defect bed. Several surgical techniques exist, but most surgeons prefer a medial approach and arthroscopy. Arthroscopic techniques are becoming more popular and allow exploration of the entire joint, removal of the cartilage flap, and forage or curettage of the defect. Because of the limited invasiveness and improved visibility provided by magnification, arthroscopic treatment is preferred.

**Postoperative Care**

Following surgery for elbow OCD, the limb is placed in a light-pressure bandage for 24 to 48 hours. The dog is confined to leash walking for 4 weeks. Gradual return to full activity is then started. Physical therapy is typically recommended during the recovery phase and includes passive range-of-motion exercises and swimming.

**Prognosis**

The prognosis for medical or surgical treatment of elbow OCD is guarded. Progression of secondary degenerative joint disease is common after medical management. Early surgical treatment of the OCD lesion decreases lameness but may not prevent the progression of OA.

**OSTEOCHONDROSIS OF THE STIFLE**

OC of the femorotibial joint is seen infrequently in dogs. Clinically affected dogs usually present between 4 and 9 months of age. The condition is common in Great Danes, German shepherds, and Labrador and golden retrievers. Seventy-six percent of reported cases have involved male dogs. The condition was bilateral in 72% of reported cases.

**Clinical Signs**

Dogs with stifle OCD usually present with a history of gradually developing, intermittent lameness. The lameness may be mild to severe and worsens with exercise. Disuse muscle atrophy and joint effusion are present in some cases. Palpation is usually unrewarding, although crepitation, pain, and mild joint laxity may be present. Stifle range of motion (particularly extension) is decreased, and a clicking sound may be audible when the limb is manipulated. The dog may have a crouched stance if the disease is bilateral.

**Diagnosis**

The diagnosis of stifle OCD is based on radiographic evaluation. Radiographs of both stifles should be taken. A typical lesion appears as a flattening of the subchondral bone of the femoral condyle. Lesions may occur on either the lateral (96% of cases) or medial (4% of cases) condyle (Figures 6A and 6B). The medial aspect of the lateral femoral condyle is the most common location reported. The normal fossa of the long digital extensor tendon and aberrant attachment of the cranial cruciate ligament can be confused with OCD lesions. Secondary changes within the joint, including joint effusion, mineralized cartilage flaps, and osteophyte formation, may also be evident radiographically.

**Treatment**

Medical management of stifle OCD consists of rest, NSAIDs, and DMOAs. It is most successful in patients with mild lameness and only a small radiographically evident subchondral lesion. Surgical treatment is preferred in patients with persistent lameness, joint mice, or larger radiographic lesions. Arthrotomy or arthroscopy may be used to explore the joint, excise the cartilage flap and joint mice, and encourage healing of the defect (Figures 6C and 6D). The edges of the lesion and underlying subchondral bone are treated with curettage, abrasion arthroplasty, or forage. Additional techniques previously described to treat shoulder OCD can be applied to the stifle. With large defects, cancellous bone grafting has been used after curettage.

**Postoperative Care**

Postoperative care consists of restricting the patient’s activity, NSAIDs, and DMOAs. Physical therapy is important for improving function after surgery.

**Prognosis**

The prognosis for dogs with stifle OC is guarded to fair because progression of OA is common even after surgery. The severity of OA before surgery, size and location of the defect, and quality of postoperative physical therapy all affect the prognosis.
Figure 6—Lateromedial (A) and craniocaudal (B) radiographic views of an OCD lesion of the lateral condyle of the femur in a 7-month-old rottweiler. Note the flattening of the subchondral bone and radiolucent areas of lysis (arrows). (C) Intraoperative view of the same dog. A probe is pointing to the large subchondral defect along the lateral condyle. (D) Intraoperative arthroscopic view of the stifle joint. Note the cartilage defect (a) and portion of the OCD lesion (b).
OSTEOCHONDROSIS OF THE HOCK

OC of the tibiotarsal joint typically occurs in large-breed dogs, with over 70% of reported cases occurring in Rottweilers and Labrador retrievers. Cases of tarsal OCD have also been reported in Australian cattle dogs, bullterriers, and bullmastiffs. Most dogs develop clinical signs between 5 and 7 months of age, although up to 36% of dogs present for veterinary care later in life with a chronic history of lameness. Males and females are equally affected. The condition is observed bilaterally in 40% of cases.

A majority (79%) of tarsal OCD lesions involve the medial trochlear ridge of the talus. Eighty percent of medial ridge lesions occur on the plantar aspect of the ridge. Twenty-one percent of tarsal OCD lesions involve the lateral trochlear ridge. Over 70% of these lesions were on the dorsal aspect of the ridge. Approximately 90% of dogs with lateral trochlear ridge lesions were Rottweilers, and 63% were affected bilaterally. Multiple lesions in a single joint have also been reported.

Clinical Signs

Patients with tarsal OCD typically present with a history of intermittent non-weight-bearing lameness, exercise intolerance, and swelling of the tarsus. The signs are usually progressive and of several months’ duration. Hyperextension of the affected tarsal joint, pain on flexion and extension, decreased range of motion, joint effusion, joint thickening, crepitus, and muscle atrophy may be evident on physical examination.

Diagnosis

The diagnosis of OCD of the tarsus is based on radiography. Radiographic evaluation should include standard lateral, flexed lateral, and dorsoplantar views of both tarsi. In addition, several special views can assist in the diagnosis. Craniocaudal views of the proximal trochlear ridges and with the tarsus flexed allow visualization of the cranial portion of the trochlear ridges (Figure 7A). They also allow visualization of the lateral condylar ridge without superimposition of the calcaneous. Oblique views can
also provide additional information. The dorsolateral–plantaromedial oblique view (D45˚L-PLMO) has been reported to be the most useful in identifying lesions of the medial trochlear ridge (Figure 7B). A dorsomedial–plantarolateral oblique view (D45˚M-PLLO) provides the best visualization of the lateral trochlear ridge.

76,81,82 Radiographic findings may include subchondral radiolucency, increased joint space, or a radiolucent concavity on the medial or lateral trochlear ridge of the talus.

Medical Treatment
Medical management for tarsal OCD is recommended in older dogs with severe degenerative changes. Medical therapy consists of restricted activity, NSAIDs, and DMOAs. Although most reports suggest that surgical intervention is preferred for treating tarsal OC, two previous studies found no significant differences in long-term outcome between joints treated medically and those treated surgically.70,83 This is likely to be particularly true in dogs with chronic disease and significant OA.

Surgical Treatment
Surgical exploration and removal of the cartilage flap or osteochondral fragment can allow ingrowth of fibrocartilage from underlying subchondral bone. Early intervention with a minimally invasive approach is preferred.

Several minimally invasive surgical approaches (e.g., dorsomedial,84 planteromedial,84 caudal,85 dorsolateral,76 plantorolateral73,76) have been described to provide access to the medial and lateral trochlear ridges of the talus for treating OC lesions. When used in combination, these approaches allow good visualization of the trochlear ridges without desmotomy or osteotomy. The location of the lesion determines which approaches are used. The plantaromedial surgical approach is used most commonly and allows exposure of 40% of the plantar aspect of the medial trochlear ridge, which is the most common site for tarsal OC74 (Figure 8). The dorsomedial surgical approach exposes the dorsal 50% of the medial trochlear ridge.74 The caudal surgical approach allows exposure to the entire plantar talar articular surface. The dorsolateral surgical approach exposes the dorsal 60% of the lateral trochlear ridge, whereas the plantarolateral approach exposes the plantar 60% of the lateral trochlear ridge.74 Accurate radiographic assessment of the lesion's location is required to ensure the proper approach is used to expose and treat the defect.

Once the lesion is exposed, the cartilage flap or osteochondral fragment is excised. Care should be taken not to overdebride the defect because this will enlarge the defect and lead to joint instability. Overall function is better with minimal curettage. Because of the poorer prognosis associated with tarsal OC, several attempts have been made to reattach the fragment (using tissue adhesives or absorbable implants) or replace it with a graft (from the phalanx).78 Unfortunately, these attempts have yielded only limited success.

Arthroscopy of the tarsus has also been described for evaluating and diagnosing tarsal diseases.79,86,87 Arthroscopic removal of OCD fragments is possible in the dorsal aspect but can be very difficult on the plantar aspect of the talus.86

Postoperative Care
After surgery, the limb is covered with a soft padded bandage for 3 to 5 days. The dog is confined to short leash walks for 4 to 6 weeks, then gradual exercise is instituted. Physical therapy can begin during confinement with passive range-of-motion exercises. Swimming and incline walking can begin after confinement. Long-term use of NSAIDs and DMOAs is generally started if they have not been previously used.
Prognosis

The prognosis for OC of the tarsus following conservative therapy is guarded because patients continue to have intermittent lameness and progression of secondary degenerative changes. Even with surgery, degenerative changes are likely and require medical management. However, most dogs are functional. Recovery time varies from 2 to 4 weeks. Recovery was found to be faster in dogs with lesions involving the non–weight-bearing dorsal aspect of the lateral trochlear ridge. Despite the progression of OA noted radiographically after surgical treatment of tarsal OC, many dogs are clinically improved. Unfortunately, the prognosis remains guarded because joint pain and lameness may recur as the OA progresses. Several factors may influence the success of both medical and surgical treatments, including the age of the dog, the presence of OA, the size of the osteochondral defect, the presence of joint instability, the site of the lesion, and whether the lesions are unilateral or bilateral. Dogs older than 12 months of age have a worse prognosis, likely due to the presence of OA at this late stage of the disease. Dogs with unilateral, smaller lesions have a better prognosis, particularly if the joint is stable. In a study of 55 joints with a 52-month follow-up, 18% to 40% were normal, 27% had intermittent lameness, and 42% to 55% had chronic lameness. Excellent short-term outcomes were reported in two dogs treated arthroscopically.

SUMMARY

OC is caused by abnormal endochondral ossification and is a common cause of lameness in immature dogs. A thorough physical examination and proper radiographic technique are important for diagnosing OCD. Treatment usually requires surgical intervention to remove the cartilaginous flaps and promote the ingrowth of repair tissue. However, medical treatment is used instead of surgery in some cases and may be beneficial after surgery to help control clinical signs of OA. The prognosis for OCD varies with the joint involved.

ACKNOWLEDGMENT

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1. OC of the tibiotarsal joint most commonly affects the
   a. medial malleoli.
   b. lateral malleoli.
   c. medial trochlear ridge of the talus.
   d. lateral trochlear ridge of the talus.
   e. tibial condyle.

2. Which of the following is not a common cause of lameness and pain in young dogs?
   a. OCD of the stifle
   b. bicipital tenosynovitis
   c. panosteitis
   d. avascular necrosis of the femoral head and neck
   e. ununited anconeal process

3. Which of the following is part of the multifactorial complex implicated in the development of OC?
   a. trauma
d. hormones
   b. genetics
e. all of the above
c. overnutrition

4. The most common grade of OCD in dogs is
   a. I.
d. IV.
b. II.
e. V.
c. III.

5. Which statement regarding OC is true?
   a. Endochondral ossification is interrupted in focal areas of the metaphysis.
   b. The tidemark separates the superficial and middle regions of mature articular cartilage.
   c. Fissures that form can extend from the articular surface to the subchondral bone.
   d. Three types of lesions have been described.
   e. none of the above

6. Which statement regarding OC of the scapulohumeral joint is true?
   a. It commonly involves the cranial central aspect of the humeral head.
   b. Female dogs are affected three times more frequently than male dogs.
   c. Small dogs and cats are overrepresented.
   d. The condition is reported as a bilateral disease in less than 10% of cases.
   e. Most patients develop clinical signs between 5 and 10 months of age.

7. Which radiographic view provides the best visualization of the medial trochlear ridge of the talus?
   a. dorsolateral-plantaromedial oblique (D45˚L-PLMO)
   b. dorsomedial-plantarolateral oblique (D45˚M-PLLO)
   c. craniocaudal
   d. flexed lateromedial
   e. skyline

8. Which statement regarding surgical approaches to access the tibiotarsal joint is true?
   a. The size of the lesion determines which approach to use.
   b. The plantarolateral approach is the most commonly used.
   c. The dorsomedial approach exposes the ventral 50% of the medial trochlear ridge.
   d. The dorsolateral approach exposes the dorsal 40% of the lateral trochlear ridge.
   e. The plantarolateral approach exposes the plantar 60% of the lateral trochlear ridge.

9. Postoperative care for most OC lesions includes
   a. a soft padded bandage for 3 to 5 days.
   b. short leash walks for 4 to 6 weeks.
   c. passive range-of-motion therapy, swimming, and incline walking.
   d. a, b, and c
   e. none of the above

10. OC lesions of the __________ have a favorable prognosis.
    a. shoulder
d. stifle (medial condyle)
b. elbow
e. stifle (lateral condyle)
c. hock

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Ask six veterinarians what causes developmental orthopedic disorders in foals and you might get six different answers. According to Tina Kemper, DVM, there could very well be six causes, and possibly more. Kemper said the incidence of developmental orthopedic disorders (DOD) in horses ranges anywhere from 10% to 50%, depending on whose research you accept. The opinion of whether developmental disorders are preventable will again depend on whose research you accept. The most common developmental diseases found in foals today are: osteochondrosis (OCD), physeitis, cervical vertebral malformation, angular limb deformities, flexural limb deformities, and club feet. Developmental orthopedic diseases of horses are an important group of conditions that occur in growing horses. Examples include osteochondrosis, physeal dysplasia, acquired angular limb deformities, flexor tendon deformities, and cuboidal bone malformations. Osteochondrosis (Osteochondritis Dissecans). Horses affected by osteochondrosis do not typically become lame, except in cases of damage to particular sites (such as the shoulder or stifle). In severe cases, signs typical of other developmental orthopedic diseases also may be present. It causes joint instability and arthritis of the shoulder. Although it is caused by improper development of the shoulder, an affected horse may not show signs until it is an adult. A severe lameness may also appear suddenly. Osteochondrosis is a family of disorders that affect bone growth in children and adolescents. The disruption of blood flow to the joints is often the cause. Though certain diseases in this family can affect older adults, they’re most likely to affect children and teenagers whose bones are still growing. Osteochondroses may cause pain and disability. What are the types? A number of diseases fall into the category of osteochondrosis. They affect different parts of your body. They’re typically grouped into one of three categories based on where they occur.