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Osteochondritis Dissecans in the Dog

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Introduction

Osteochondritis dissecans (OCD) is a manifestation of osteochondrosis characterized by a focal thickening of joint cartilage and subsequent dissection of a flap of this thickened cartilage away from the underlying subchondral bone.[1] The etiology of this condition remains somewhat of a mystery; trauma, nutrition, ischemia, and hereditary abnormalities of ossification have all been suggested.[2] The disease is usually seen in the faster growing members of large and giant breed dogs. The first clinical signs of lameness are usually noted when the dog is between 5 and 9 months of age. OCD is most commonly recognized in the proximal humerus, but is also found in the distal humerus, distal femur, and tibial tarsal bone. One case of OCD of the distal radius has been reported.[3] Other manifestations of osteochondrosis include ununited anconeal process, fragmented coronoid process, and retained cartilage of metaphyseal growth plates.[1] Current research suggests there may be some relationship between osteochondrosis and the development of cervical spondylolisthesis, slipped femoral capital epiphysis, and hip dysplasia.[1]

Pathogensis

OCD is a pathological condition in rapidly growing cartilage caused by a disturbance of endochondral ossification. Growth of the epiphysis occurs in the articular cartilage in the same manner that growth of the long bones occurs in the cartilage of the metaphyseal growth plates.[4] Normal growth of the epiphysis takes place by proliferation of chondrocytes near the joint surface. As the cartilage continues to grow, these chondrocytes hypertrophy, vesiculate, degenerate, and become calcified. This calcified layer of the cartilage is invaded by vessels from the bone marrow. Some of the calcified cartilage is resorbed, but remnants of cartilage are used as a framework upon which osteoblasts lay down bone. This process is called endochondral ossification.[4]

In osteochondrosis, the normal chondrocytes differentiation process is disturbed.[5] Vesiculation, degeneration, and calcification do not occur in a normal fashion, and the cartilage gets thicker than normal. Vessels from the bone marrow cannot penetrate this thickened cartilage, and bone is not formed. Resorption of the basal layers of cartilage and replacement by bone on the diaphyseal side of the joint cartilage ceases. The chondrocytes continue to proliferate near the joint surface, resulting in a thickening of the joint cartilage.[4] If this process is localized to only a portion of the joint cartilage, and formation of bone continues in the calcified layer of the surrounding cartilage, the radiographic appearance will be that of an osseous defect. If the thickening of the joint cartilage is the only pathological change, there are no apparent clinical signs. There is little inflammatory reaction in the subchondral bone at this stage. After the thickened cartilage is resorbed and endochondral ossification then proceeds normally.

Since joint cartilage nutrition depends upon simple diffusion of nutrients from the synovial fluid, the deeper layers of the thickened cartilage are insufficiently nourished. These deeper layers of thickened articular cartilage die, necrose, and serve as a starting point for fissures.[5] When a developing fissure reaches the joint cartilage surface, synovial

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fluid enters the fissure and contacts the basal layers of the joint cartilage and the subchondral bone. An inflammatory reaction then takes place in the defect. The dog becomes painful and starts to limp. It is at this stage that the lesion becomes osteochondritis dissecans. Osteochondritis refers to the inflammation in the joint cartilage and subchondral bone, and dissecans refers to the flap of cartilage that is dissected away from the underlying subchondral bone. This flap may remain in the defect, or it may detach and form an intraarticular body. The fate of the flap is of great importance to further development of clinical signs.[5]

Etiology

A number of factors have been incriminated as the cause of OCD, but the etiology remains controversial and appears to be multifactorial. The most common factor incriminated in experimental and clinical studies is rapid growth and weight gain. In one study of nearly 300 patients with only a few dogs were not of medium or larger size. There were twice as many males as females in these cases. This difference may be explained by the fact that male dogs usually grow more rapidly than female dogs.[1]

There is probably a hereditary predisposition for OCD in the dog, although this has not been proven. A higher incidence of OCD was found in offspring of certain dogs and there were litters in which several or all of the puppies were affected.[1,2,6-9] Genetic factors that affect growth rate and weight gain, sexual development, behavior, and conformation undoubtedly play a role in the etiology.[10]

Nutrition seems to be an important factor in the development of OCD. In an experimental study in Great Dane puppies, free choice feeding resulted in increased growth rate accompanied by skeletal abnormalities similar to those seen with OCD.[11]

Trauma may also contribute to the development of OCD. Biomechanical studies have shown that OCD lesions typically develop in those areas of the joint cartilage that are most subjected to the stress and trauma of weight bearing. Trauma probably enhances the opportunity for the abnormally thickened and slow-to-mature cartilage to cleave further once a fissure is formed.[12]

In conclusion, most evidence indicates that dogs with a genetic capacity for rapid growth and development, and are "pushed" nutritionally during their most active growth period have the greatest risk of developing OCD.[1]

OCD of the Shoulder

OCD of the shoulder joint is seen in large and medium size dogs, predominantly in the male. The clinical signs are first noticeable between 4 to 7 months of age and are usually insidious in onset. Lameness on one or both forelegs, which worsens after exercise, is the most prominent clinical sign. Stiffness after periods of rest is also an important clinical sign. Pain can usually be elicted by palpation, hyperflexion or hyperextension of the shoulder joint. The clinical signs may vary in severity over periods of weeks to months. The condition is often bilateral.

The definitive diagnosis of OCD is made by radiology. The view that best demonstrates the lesion is the medial-lateral with slight extension of the limb.[13] The affected limb should be placed down on the film cassette and pulled cranial. The unaffected or upper limb should be pulled caudally. The image of the humeral head will thereby be superimposed over the radiolucent lumen of the trachea to enhance delineation of any lesions present. Radiographs will demonstrate a defect, usually in the caudal aspect of the humeral head. In mild or early cases only a flattening of the dorso-caudal contour of the humeral head is seen. In advanced lesions there may be sclerosis of the subchondral bone and calcification of the cartilagenous flap. A radiograph of the shoulder taken with the primary beam directed at a slightly oblique angle from the lateral may be needed to visualize the lesion. This view may assist in a diagnosis because in many cases the lesion is slightly to the caudolateral instead of the caudal side of the humeral head.[13] Both shoulders should be radiographed, even if there is no history or clinical sign of bilateral lameness.[1]

It is generally easier to diagnose OCD of the shoulder than it is to decide what therapy to use. If the flap mineralizes, it is usually visible on radiographs. The cartilagenous flap may remain connected to the adjacent normal articular cartilage and within the defect. If the flap remains cartilagenous, radiographs will reveal only the defect in the subchondral bone. Because animals show pain and lameness, restriction of exercise has been recom-

mended as a part of the therapy by many investigators. In contrast, another researchers believes that a dog with OCD of the shoulder should be allowed to move around as much as possible because this will increase the chances that the flap will become dislodged.[1] If necessary, the dog can be given analgesics. In cases in which clinical signs are not severe or may have subsided, or if there is no improvement after 4 to 6 weeks, a repeat radiographic exam is recommended. If the defect in the humeral head has not been filled, an arthrogram should be made to determine whether or not a flap or loose piece of cartilage remains within the defect. If the arthrogram is positive, surgery is indicated. Surgery is usually not necessary if there is no loose cartilage or flap in the defect, because healing in this case can occur spontaneously.[1]

Once the pedicle of the flap breaks, the flap becomes dislodged and floats freely in the synovial cavity. The flap is then referred to as a joint mouse. The joint mouse is usually resorbed by the synovial fluid thru enzyme activity. Once the flap dislodges it takes one or two weeks before the pain subsides. It is in these cases where conservative medical therapy is indicated. Sometimes the loose flap will grow while bathed in the synovia and may then mineralize and become visible on radiographs in the posterior compartment of the joint cavity. Occasionally the joint mouse will migrate and lodge in the bicipital tendon sheath resulting acute pain and requiring surgical removal.[1] Surgery is indicated when a mineralized joint mouse is detected to prevent secondary degenerative joint disease.

A caudolateral approach to the shoulder joint can be used to expose the humeral head.[14] Surgery consists of removing the cartilage flap or piece of cartilage in the defect and trimming the edges of the defect. The base of the lesion should be curretted to the bleeding subchondral bone. In addition, some authors advocate drilling 1 mm holes through the defect towards the metaphysics to stimulate neovascularization and accelerate healing.[15] Any free joint mice or periarticular osteophytes should be removed. Postsurgical care consists of cage rest for 7 to 10 days and restricted exercise for 4 to 6 weeks.

If bony sclerosis is minimal or absent adjacent to the lesion and there is no secondary osteoarthrosis, the prognosis is considered good. Following surgery, the defect will fill with functional fibrocartilage and restore a smooth contour over the articular defect. This fibrocartilage is not as biomechanically stable as the hyaline cartilage that was originally present, but is functionally adequate. Removal of the diseased tissue allows for better healing, and greater freedom from lameness.[15]

OCD of the Elbow

Three manifestations of osteochondrosis in the elbow are ununited anconeal processes, fragmented coronoid process, and OCD of the medial condyle of the humerus.[1] Fragmented coronoid process is the most common and OCD of the medial condyle of the humerus is the second most common of these elbow lesions. These two lesions are seen most frequently in Golden and Labrador Retrievers, but can be seen independently or together in most large breeds of dogs.[1] Although the clinical signs of these three elbow lesions are very similar in the early stages, an ununited anconeal process is usually the only lesion that can be radiographically diagnosed at five months of age. The owner usually complains of a stiff foreleg gait for the first few minutes after a long rest. This sign is usually first demonstrated when the dog is four to five months of age. The lesion is often bilateral, which makes it more difficult for the owner to observe. The lameness progresses to a stiff stilted gait on the forelegs, which are held externally rotated with the elbows close the chest. Physical examination reveals joint crepitation and pain upon palpation, flexion, or extension of the elbow.

It is sometimes difficult to differentiate lameness caused by pain in the shoulder from lameness caused by pain in the elbow on physical examination. Therefore, in doubtful cases both elbow and shoulder should be radiographed. No radiographic abnormalities will be visible in the elbow of a dog with OCD of the medial condyle of the humerus until the dog is about seven months of age, even though the clinical signs may be present intermittently for two to three months. A young dog with slight clinical signs of elbow lameness should have a repeat radiographic examination in four to eight weeks following the first examination to allow time for radiographic changes to develop. A flexed lateral and an anteroposterior radiograph are necessary to evaluate the elbow. OCD of the me-

dial condyle of the humerus has a characteristic radiographic appearance. A small triangular defect can be seen in the weight bearing surface of the medial condyle on an anteroposterior projection. This defect is often surrounded by a sclerotic zone. In later stages, this defect may be obscured by osteophytes formed at the margin of the medial humeral condyle and the medial aspect of the proximal ulna. In such cases, an anteroposterior medial to lateral oblique view should be obtained for better interpretation.[13] Cartilagenous flaps and joint mice may also be observed. Signs of secondary degenerative joint disease vary with the extent and severity of the lesion, age of the patient upon presentation, and possible coexistence of additional bony disease, i.e. ununited anconeal process and fragmented coronoid process.[2]

If left untreated, OCD of the medial condyle of the humerus will lead to severe osteoarthrosis with decreased range of motion of the elbow joint. Treatment is surgical and should be done as soon as a diagnosis is made. A medial approach to the elbow should be used. Two techniques for approaching this area have been described in veterinary literature. Flexor muscle tenotomy with radial collateral ligament severance, or osteotomy of the medial epicondyle an be utilized to gain surgical exposure to the medial humeral condyle.[14] In early cases of OCD of the medial condyle of the humerus there is a defect in the weight bearing surface covered by a flap of cartilage. The flap should be removed, the edges of the defect trimmed, and the base of the defect curetted to bleeding subchondral bone. A flap is usually not present in more advanced cases. It may be present as a large cartilagenous body adherent to the joint capsule, or it may have been resorbed. In a joint with a defect and no flap, the defect should be trimmed and curetted and all fragments removed.[2] The medial ulnar coronoid process should also be carefully inspected, as OCD of the medial condyle of the humerus is frequently associated with a fragmented coronoid process. Following surgery the dog is cage rested for ten days and kept on restricted exercise for 4 to 6 weeks.

In cases of OCD of the medical condyle of the humerus, the prognosis is guarded even if surgery is done early. However, surgery should always be attempted, because an untreated animal usually develops very severe secondary degenerative joint disease. Surprisingly, many untreated dogs can function well in spite of their handicap, but even slight trauma to the elbow results in transient lameness.[5]

OCD of the Stifle

OCD of the stifle usually affects large dogs between three and nine months of age, and has been reported in several instances to occur in littermates.[2,6,9] As high as 15% of all cases of osteoarthrosis of the knee in large dogs is secondary to OCD of the stifle joint.[1] Diagnosis is often difficult because the clinical signs are vague. The hip joint rather than the knee is apt to be suspected as the cause of lameness. The dog presents with a disturbed gait pattern of the hind legs similar to the "slinky gait" of hip dysplasia. Sometimes there is only a temporary "locking."[5] Physical examination may or may not reveal joint distension and pain upon manipulation of the affected joint. However, the physical examination is valuable in ruling out other causes of stifle lameness in young dogs, such as ruptured cruciate ligaments or a patellar luxations.

Lateral and anterioposterior radiographs are needed to confirm the diagnosis. The most common site of OCD of the stifle is the medial aspect of the weight bearing surface of the lateral femoral condyle. The radiographic appearance can vary from a flattening of the affected femoral condyle to the presence of a radiolucent concave defect on the articular surface, often bordered by a sclerotic margin.[13] Joint distension and joint bodies may be observed.

In some dogs, the pathologically thickened cartilage of the condyle does not lead to OCD, as ossification may be resumed and the radiographic defect may resolve. Other dogs develop OCD, but healing can take place in the same way it does in the humeral head. In most cases, however, only the floor of the defect fills with fibrocartilage and severe secondary osteoarthrosis develops.[5]

Surgery has not proven very effective in the treatment of OCD of the stifle. Even animals that have been operated on early by removal of the flap do not have a good prognosis. However, surgery should be attempted in animals that present with an acute case of stifle lameness and radiographically show a large flap or a joint mouse. In such cases an exploratory arthrotomy should be done and the

flap or joint mouse should be removed. Once osteoarthorsis develops there is little that can be done to improve the condition other than conventional medical or physical therapy.[1]

OCD of the Hock

OCD of the hock is the least common form of the osteochondroses. It is most frequently seen in Golden and Labrador Retrievers.[1] Vague clinical signs that worsen following exercise begin at four to five months of age. The dog presents with a slightly shorter step on the affected leg, with pain upon flexion and extension of the hock. The range of flexion is reduced and the dog typically stands with its leg in a hyperextended position.[7] The joint capsule of the tibiotarsal joint is usually distended, and in later stages a firm swelling can be palpated on the medial side of the tarsus.

Anterioposterior and lateral radiographs are needed for the diagnosis. The lesion is located on the medial trochlear ridge of the talus and is best demonstrated as a defect in this ridge on an anterioposterior projection. The height of the medial trochlear ridge of the talus is reduced due to displacement of an osteochondral fragment and lysis of subchondral bone. The width of the joint space over the medial trochlear ridge of the talus is increased. In later stages, the subchondral bone of the distal tibia opposite the medial trochlear ridge of the talus is eroded and the medial malleolus undergoes some lysis.[7] Displaced or undisplaced osteochondral fragments can be found associated with the medial trochlear ridge of the talus. Cartilage flaps and joint mice frequently contain bone because these structures usually remain attached to soft tissues such as the medial collateral ligaments or the joint capsule. This maintains their blood supply and allows them to continue endochondral ossification.[1] Sometimes a flexed lateral view is necessary to demonstrate these intraarticular bodies in the medial or caudomedial compartment of the joint capsule.[7]

Surgery is the treatment of choice for OCD of the hock. Approach to the hock joint by osteotomy of the medial malleolus permits direct entry into the joint and gives good exposure to the medial trochlear ridge of the talus.[14] Surgical debridement of the articular cartilage is similar to that described previously.

Conclusion

In recent years, the increasing number of reports describing osteochondrosis in dogs has lead to a greater awareness of the disease. Osteochondrosis is a generalized disease affecting endochondral ossification of cartilage in young dogs of the large and giant breeds that have the egenetic capacity for rapid growth. The possible causes of OCD include rapid growth and weight gain, overnutrition, trauma, and hereditary factors. OCD should be suspected in any large, young dog with a lameness of insidious onset that becomes worse after exercise and periods of rest. OCD is more frequently seen in males and the lesions are often bilateral. The shoulder, elbow, stifle, and hock joints are the primary sites of occurance. Diagnosis is based on signalment, history, physical examination and radiography. Treatment must be individualized and based upon age, frequency and severity of the lameness, and radiographic appearance of the lesion. Surgery is usually the treatment of choice. Prognosis depends upon the site involved, the severity of the lesion, the presence or absence of secondary degenerative joint disease, and the stage at which the lesion is diagnosed and treated. Finally, it seems that genetic factors influence development of the disease, and the owner should be made aware that the condition may be hereditary.

If a dog has a family history of OCD or has produced offspring with a high prevelance of the disease, breeding should be discouraged.

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Hope for the Peregrine

by Danny Brass*

In the midst of the high Sierra stands Yosemite Valley, surrounded by glacial domes and enormous walls of stone soaring thousands of feet upward above a tree-laden landscape. Snugly nestled in its granite sanctuary, the valley and the accompanying high country of Tuolumne Meadows constitute Yosemite National Park, one of the earliest of our country's pristine wilderness areas to be so designated and thus conserved as an enduring natural heritage.

Although well-known for the grandeur of its mountainous expanse and the splendor that its limitless hiking trails provide, its worldwide reputation is, perhaps, predominantly derived from its wealth of high granite walls, which beckon to rock climbers from the farthest reaches of our globe. It is a mecca to which all of the world's climbers ultimately aspire; a shrine, at whose temple of stone, mettle, skill, and character can all be tested against the resplendant backdrop and airy exposure of the valley floor below.

On an otherwise calm and still morning, when the chink of hardware and the grunts of his own exertions are the only sounds to assail a climber's ears, a great wind may suddenly well up overhead, transfix him to the rock and, in the wink of an eye, disappear. It is as though some unseen force had thrust through the atmosphere and a small measure of sky had been abruptly sucked out of existence. A slight quivering of air and flesh may linger for some moments after the reality has passed. The event, of course, has marked the swift and sure stoop of the peregrine falcon along the cliff face, in its unending quest for food to feed its young.

Observation of the peregrine in its natural state is an experience well beyond belief and from his precarious perch high above the mainstream of the valley's activities, the climber is privy to an unparalleled avian display, not generally available to more earthbound visitors to the park. Their flight along the cliff face is difficult to describe in a manner that adequately conveys the full impact of so memorable an event. It has been superbly depicted by J. A. Hagar (Cade, 1982).

"The patient watcher will see an exhibition of flying that is literally breath-taking... again and again the tiercel started well to leeward and came along the cliff against the wind, diving, plunging, saw-toothing, rolling over and over, darting hither and yon like an autumn leaf until he would swoop up into the full current of air and be borne off on the gale to do it all over again...Nosing over suddenly, he flicked his wings rapidly 15 or 20 times and fell like a thunderbolt. Wings closed now, he shot down past the north end of the cliff, described three successive loop-the-loops across its face, turning completely upside down at the top of each loop, and roared out over our heads with the wind rustling through his wings like rippling canvas. Against the background of the cliff his terrific speed was much more apparent than it would have been in the open sky. The sheer excitement of watching such a performance was tremendous; we felt a strong impulse to stand up and cheer."

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