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## OSTEOCHONDROSIS

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### **OSTEOCHONDROSE**

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Osteochondrosis is a disturbance of endochondral ossification which is seen in the growing dog and may occur either in the epiphyseal or the physeal sites.

Epiphyseal osteochondrosis (articular osteochondrosis), develops in the articular cartilage which acts as a growing cartilage in young dogs. As a consequence, the condition takes place within the affected joints. The most frequent sites are the humeral head (shoulder osteochondrosis), the medial humeral condyle (elbow osteochondrosis), the lateral – rarely medial- femoral condyle (stifle osteochondrosis), the medial - rarely lateral - ridge of the talus (hock osteochondrosis).

Physeal osteochondrosis (non-articular osteochondrosis), takes place in the growing part of some long bones (physe), between the metaphyse and the epiphyse. The distal ulnar metaphyseal growth plate is the more affected site, though the situation may be encountered in other places, like the distal fibular or distal radial metaphyseal growth plate.

Articular osteochondrosis may remain a reversible and non-clinical situation, or lead to a clinical problem, with lameness on the affected limb. The appropriate term for this second situation is « osteochondritis dissecans ».

Non-articular osteochondrosis generally leads to abnormality of the forelimb rather than lameness, though it may also remain a reversible and non-clinical situation .

### **I/ EPIPHYSEAL OSTEOCHONDROSIS (ARTICULAR OSTEOCHONDROSIS)**

The most frequent situation is seen in the scapulo-humeral (shoulder) joint. We will take shoulder osteochondrosis as an example, then compare with other articular osteochondrosis.

### **SHOULDER OSTEOCHONDROSIS**

#### **Epidemiology**

Shoulder osteochondrosis is seen in young dogs (4 to 10 months of age), of large size and rapid growth (generally with individuals weighing more than 20kg), though the condition has also seldom been described in the beagle and the miniature poodle. Males are more often affected than females (ratio 2/1). The condition is generally bilateral (27 to 68% depending on the authors), though only 5% of radiographically affected dogs present with bilateral forelimb lameness.

## **Pathogenesis**

The increase of epiphyseal volume, in growing animals, occurs through endochondral ossification within the epiphyseal cartilage. Multiplication of cartilage cells within a germinal layer leads to thickening of the growth cartilage towards the metaphysis. The new cartilage is progressively replaced by bone tissue.

Thus, normal growth of long bones results from a precise balance between cartilage growth and its gradual replacement by bone. Osteochondrosis (or chondrodystrophy) is a failure of the replacement of the cartilage by bone. Consequently, the cartilage tissue becomes abnormally thickened in that area. The process can be self-limiting. Should this happen, the condition remains clinically silent, although it may be detected as an incidental finding on survey radiographs. On the other hand, it may, evolve into a more significant lesion, via the development of a fissure in the deeper portion of the thickened cartilage. This fissure progresses towards the cartilage surface and creates a cartilage flap. As soon as the cartilage is fissured, there is an inflammation and clinical symptoms : this is referred to as « osteochondritis dissecans » (OCD).

In shoulder OCD, the caudal aspect of the humeral head is usually affected.

The flap may become completely detached and loose, migrating within the joint, forming one or more « joint mice ». The joint mice may become localised in the caudal recess or in the bicipital groove of the shoulder (which may produce a severe tenosynovitis).

A joint mouse can occasionally (very rarely infact...) be gradually resorbed , stay the same in size, or increase in size and become mineralised.

Cartilage fissuring releases degradation products that have a pro-inflammatory effect. This lead to the initial signs of the clinical condition, but it is also the biochemical mechanism wich will initiate the development of a secondary degenerative joint disease.

## **Etiology**

It is more likely multifactorial. Several hypotheses have been proposed. The growth rate and weight gain appear to be major predisposing factors, other underlying factors being frequently suggested.

- The occurrence of this condition in heavier breeds and in specific bloodlines provides a strong suspicion that this condition is the result of genetic predisposition on the part of the parents, and that rapid skeletal growth is a contributing factor. Thus in several countries, shoulder osteochondrosis must be radiographically looked at and affected dogs are excluded from the breeding program.

- Nutritional factors in growing pups : overfeeding, i.e. excess energy, protein, calcium, phosphorus and vitamin D intake (HEDHAMMAR et al. 1974) causes an increased incidence of OCD when compared to a normal diet. Neither excess energy (LAVELLE 1989) nor excess protein (NAP et al. 1993) in the diet appear to be involved. Excess calcium, conversely, has been shown to be a significant factor (HAZEWINDEL 1985). The potential roles of excess phosphorus and vitamin D have not been demonstrated.

- The involvement of traumatic factors has been reported. A primary involvement of trauma is, however, unlikely. The thickened cartilage is admittedly a « weak point », which may be particularly prone to injury from mechanical stress, especially once fissures have developed.

## **Clinical and diagnostic significance**

Clinically, shoulder OCD is most commonly diagnosed in animals aged 6 to 8 months. A sudden or more insidious onset forelimb lameness is observed. There may be episodes of spontaneous improvement for one or several weeks but the pain persists despite anti-inflammatory or analgesic therapy. Rapid muscle atrophy develops in shoulder muscles. Manipulation of the limb yields marked pain upon hyperextension of the scapulo-humeral joint and, to a lesser degree, upon forced flexion or deep palpation of the caudal joint recess. Diagnosis is based upon radiography, a mediolateral projection being most useful.

Both shoulders must be radiographed.

The typical radiographic appearance of OCD consists of an altered subchondral bone contour in the caudal aspect of the humeral head : an 1 to 2cm area of decreased radiodensity and irregular contour is observed. It may be surrounded by a sclerotic bone area characterised by increased radiodensity and loss of trabecular pattern.

Occasionally, a thin radiopaque line, corresponding to mineralisation of the cartilage flap, is visible.

The radiographic image only translates the failure of mineralisation of the epiphyseal bone but yields little information regarding the nature and severity of cartilage damage.

If osteochondrosis without osteochondritis is suspected, arthrography or arthroscopy may be necessary to confirm or rule out the presence of cartilage flaps.

## **Treatment**

It is a general agreement that the only rational treatment of an OCD is a surgical one, possibly after a 6 week maximum conservative treatment.

If the equipment and skills are available, arthroscopic treatment (minimal invasive surgery) of OCD can be performed.

Conventional surgery is performed via a limited caudo-lateral approach. The minimal surgical trauma caused by this approach allows a good and rapid post-operative recovery. The cartilage flap is cut free and all the abnormal cartilage around the lesion is trimmed. A forceful lavage of the joint helps to flush out any remaining debris, including free fragments of cartilage which should always be looked for in the caudal cul-de-sac of the joint. If joint mice have been identified in the bicipital groove, a cranial approach is performed to remove them.

The leg is usually left unbandaged and very minimal activity (house confinement and short leash walking) is suggested for 4 to 6 weeks after surgery.

Usually the results are quite good, particularly if the treatment has been performed precociously. In some rare cases (most often with big lesions) it may take up to 6 months before the dog becomes sound.

Some animals, seemingly hyperactive ones, develop a seroma, all of them clearing spontaneously (we never aspirate nor drain even the largest ones).

Late surgery or conservative treatment are likely to cause a chronic synovitis and favour the development of osteoarthritis. However, shoulder OCD is the condition, among all epiphyseal osteochondrosis, with the more favourable prognosis, as it is the less prone to degenerative joint disease.

## **OTHER TYPES OF EPIPHYSEAL OSTEOCHONDROSIS**

Pathogenesis, etiology and treatment principles are very similar to the ones just described with shoulder osteochondrosis.

## **ELBOW OSTEOCHONDROSIS**

The condition is part of elbow dysplasia. It was proven by GUTHRIE & PIDDUCK (1990,1991), then by PADGET & all (1995) that it was a polygenic multifactorial inheritable condition. Though some dogs may remain sound, generally, affected dogs exhibit a unilateral or bilateral frontleg lameness with median onset between 4 and 8 months. With time, particularly if the surgical treatment is delayed, elbow osteoarthritis will develop. The prognosis of elbow osteochondrosis is guarded.

## **STIFLE OSTEOCHONDROSIS**

Most affected breeds belong to large breeds, including the Irish Wolfhound, German Shepherd Dog and Great Dane. Clinically the dog exhibit a unilateral or bilateral pelvic limb lameness. Age of onset ranges from 3 to 9 months. In bilaterally affected dogs, the animal may be suspected of having hip dysplasia. Both pathologies are often associated in the German Shepherd, but in the Irish wolfhound and Great Dane, most of the time it is clearly a stifle lameness. Development of stifle osteoarthritis is anticipated in affected dogs, but cases with minimal degenerative joint disease treated surgically have a good prognosis.

## **HOCK OSTEOCHONDROSIS (Osteochondritis of the talus)**

This is the more complicated situation because of late detection, and generally development of secondary arthrosis at the time of diagnosis. Surgical approach to the hock is also more complicated than to the shoulder, stifle, and even elbow joint. In cases treated surgically early enough, with minimal degenerative change already present, the outcome is better. Anyhow the overall prognosis must be guarded.

The condition has a low frequency and is mainly seen in Labrador retrievers and Rotweilers. There is a strong likelihood of osteochondrosis of the talus being an inherited disease (littermates reported to be affected, sire producing several affected dogs), so it is wise to avoid the use of affected dogs in the breeding program.

## **II/ PHYSEAL OSTEOCHONDROSIS (NON-ARTICULAR OSTEOCHONDROSIS)**

The most frequent situation is a retained distal ulnar cartilage core. Giant breeds are more often affected. The lesions are unilateral or bilateral. They looked radiographically as an enlargement of the normal cartilage physis, in a shape of a radioluscent cone ("cartilage retention") extending proximally into the ulnar metaphysis.

Pathogenesis is similar to the one depicted in epiphyseal osteochondrosis, except that no necrotic area develop within the cartilage (no "cartilage flap" formation). Retained distal ulnar cartilage core occurs frequently in a sub-clinical stage, as the efficient portion of the growth plate is large enough to sustain normal ulnar growth. In some dogs however, ulnar growth is slowed and clinical signs (cranio-lateral bowing of the radius, lateral deviation of the foot) are seen between 4 and 8 months of age. Elbow and carpal joint may be involved secondarily (sub-luxation, and development of secondary degenerative joint disease), and the gait become painful. Heritability of distal ulnar lesions has not been thoroughly studied, and the major evidence for the condition being inherited is found in its occurrence in certain breed and family lines.

Early treatment relies on partial ulnar ostectomy to allow normal radius development. The prognosis is fair to good when the degree of bowing is minimal and the elbow joint remains intact. In event of severe deformity, surgical treatment is difficult to achieve. Rotational deformity, radial bowing, shortening of the leg, malalignment of weight-bearing planes in the elbow and carpus must be addressed. The prognosis is very much dependent on the severity of change present at the time of presentation, and on the skill of the surgeon.