OSTEOCHONDRITIS DISSECANS OF THE SHOULDER JOINT IN DOGS

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Summary

Within the last several years, a developmental disease process named "osteochondritis dissecans" has been diagnosed with increasing frequency in the veterinary medical fields. A generalized metabolic disease process disrupting the normal sequence of cartilage calcification and ossification, osteochondritis has become a major problem in many breeds of dogs. The purpose of this study is to describe pathophysiology of disease, clinical signs, physical examination findings, specific radiographic abnormalities and to indicate specific treatment of osteochondritis dissecans of the shoulder joint in dog.

Key words: osteochondritis dissecans, shoulder joint, dog

Osteochondritis represents a systemic disease, characterized by alteration in the endochondral ossification processes of the epiphisal cartilage and also in the metaphiseal and apophiseal growth cartilages (3, 12, 24).

Disturbances of the equilibrium between the articular cartilage growth and ossification, lead to a series of lesions, which can be divided into four degrees of evolution. The first three degrees evolve asymptomatically, usually being diagnosed accidentally (by chance). The fourth degree of osteochondritis dissecans evolves with pain and lameness, as a result of a fragment of cartilage being detached. The clinical and radiological aspects met in the fourth degree are defined using the term of osteochondritis dissecans (OCD) (10, 12, 22). König, in 1887, first used the term OCD in human medicine, although references regarding a similar symptomatology existed since 1726 (22). In veterinary medicine, one of the first descriptions of OCD in dogs belongs to Brass (1956) cited by Probst and Johnston (22).

Osteochondritis dissecans affects the articular cartilage, in different locations: shoulder, elbow, knee, stifle, vertebrae (3, 4, 10, 16), also having a part in the fragmentation of the ulnar choronoid process and in the nonunion of the medial humeral condyle (3, 16). Humeral head OCD represents the most frequent localisation of OCD (about 36% of all the cases in a study carried out on 626 dogs) in the USA dogs (3, 16).

In case of the metaphiseal OCD (growth cartilage/plates), the osteochondritis is responsible for the nonunion of the ulnar anconated process and for the hypertrophic osteodistrophy – persistence of the growth nuclei – radial and ulnar angular deformities (3, 17).

The normal epiphiseal growth of the long bones is due to the germinativ layer cells multiplication. This layer is placed in the mid area of the cartilage (22). The

cartilage cells are fed indirectly through osmotic imbibition from the sinovial liquid of the respective joint. When the cells from the profound layer are matured, they are mineralised – endochondral ossification. In the mature animal, when the growth process ceases, a fine and clear line appears between the calcified area and the cartilage itself. In young animals (under 9 months of age) this line is missing. The equilibrium between the multiplication processes of the chondral lineage cells and the ossification of the deep cell layers ensure a normal growth (12).

In the pathogenesis of OCD a series of perturbing factors are involved which determining a slower ossification of the deep layers of the cartilage. In these conditions, the germinativ layer cells that are multiplying, determine an excessive thickening of the cartilage. Due to the thickness of the cartilage, the nutrition of the deeper layer is deficitary, which determine the random positioning of the chondrocites from those layers. Endochondral ossification stops and rifts between the affected cartilage and the calcified epiphiseal are appearing. These rifts release degradation substances which have a proinflammatory effect, initially causing joint inflammation and later on, degenerative lesions of the joint (10, 11). This stage of osteochondrosis (IV) corresponds to the initial phase of the osteochondritis dissecans. The pathologic process continues with autodissection and detachment of the affected cartilage fragment. In this phase, the intervention of different traumatic factors favorises the cartilage detachment. The importance of the traumatic factors is questionable. If the traumatism is not powerful enough to produce vertical fracture lines in the cartilage, the spontaneous reattachment of the damaged fragment is possible. The haemorrhage produced by traumatising the capillary vessels of the subchondral osseous area and the haematoma in the rift space, allows the organisation of a fibrin net which is rich in undifferentiated mesenchymal cells, which will repair the damaged cartilage fragment, reattaching it to the healthy one around it.

The loose cartilage fragments are known as *joint mice*, and they are at first floating free in the sinovial fluid, enhancing the inflammatory response, and then they usually sink in the caudal sack of the shoulder joint and in the sinovial sheath of the brachial biceps muscle (3, 5, 9, 10, 12, 19). The cartilage fragments can be resorbed, or, they can grow through mineralization (12).

The healing is not possible if the detached cartilage fragment is not surgically reattached and stabilised or excised (3, 10, 12, 17). After the cartilage is excised, the healing of the barren site takes place depending on its size – the granular tissue layer that fills the area is populated with chondroblastes and it transforms into fibrocartilaginous tissue and then, gradually in hyaline cartilage (11, 15).

The aetiology of OCD is controversial. Generally accepted is the polifactorial aspect (12, 22). The literature presents various hypotheses.

The high incidence of OCD in certain lineages of some races, imply a genetic factor or a hereditary predisposition for the condition (25).

The hormonal influence over the ossification process – the excessive administration of STH and TRH determine a substantial thickening of the articular

cartilage, similar to that encountered in OCD. Testosterone acts sinergically with the growth hormone, while estrogens favour calcification, which explains the higher incidence of the conditions in males rather than in females (22).

The physiological incongruence of the shoulder joint bones leads to excessive thickening of the caudo-medial cartilage on the humeral head, which compensates the accentuated concavity of the glenoid cavity, redistributing the intraarticular forces on a larger cartilage surface. The excessive thickening of the caudal humeral head cartilage slows down the endostal ossification and forces the cartilage to stand traumatic mechanical forces that act repeatedly, detaching it from the bone surface.

The involvement of nutritional factors – overfeeding, protein/energy/calcium/ phosphorus/vitamin D excesses – was mentioned as a factor that raises the incidence of OCD [Hedhammer et al. (1974) cited by Genevoise (12)]. Of all these factors involved in the OCD pathogenesis in dogs, but only the involvement of excess calcium was proven to lead to raise calcitonin levels, delaying mineralization and endochondral ossification (13).

The traumatic factors play a major role in OCD pathogenesis, but less probable as a primary factor (12). Repeated mechanical stress can lead to excessive cartilage thickening as well as to their incomplete fracture, especially if the bones are abnormally developed (12). Cook (4) emphasises the importance of the conditions that the animals is maintained in (avoiding rough/rigid floors) and of the movement regime in the fist year of life (avoiding jumps), which if ignored lead to a higher incidence and gravity of OCD lesions.

The cartilage structure alterations based on local metabolic disorders are a plausible theory for OCD aetiology (6, 7, 8, 26).

Shoulder joint OCD develops in dogs of 3 months to 5 years of age (17), more frequently (75% of the cases) in young dogs at 4-10 months of age (4,10, 12, 16, 22). Especially affected are large breed dogs, weighing over 20 kg (10), but shoulder joint OCD was reported also in medium and small breed dogs (5). The most commonly affected races are Labrador, Golden Retriever, Rottweiler, Great Dane, Saint Bernard and German Shepard. Males are more often affected than females, at a rate of 2:1 (12) to 5:1 (25). The literature data show that bilateral lesions are predominant, with an incidence between 20% (24) and 85% (4), and of these dogs only 5% show bilateral lameness (12).

At first, the OCD symptomatology evolves insidiously characterised by bilateral lameness (22) often in episodes (3, 12, 17). Other inconstant symptoms are: atrophy of the shoulder blade muscles, articular swelling, and outward turn of the shoulder and elbow adduction.

Mobilising the limb and hyper-flexion/extension movements constantly produce pain, sometimes accompanied by a specific sound “click-clack” (3, 12, 16, 17, 19, 22).

The diagnostic is based on X-ray imaging of the shoulder joint. For this, the animal must be sedated. Exposing the scapulae-humeral joint in a medio-lateral

incidence, with the elbow slightly pointing outwards, allows visualisation of the central articular surface of the humeral head, which makes out one of the predilect areas for cartilage detachment. On the radiography, the affected area appears as being flattened, as a small plateau (1-2 cm), radio transparent (3, 10, 12, 22). In some cases, the mineralization of the loosened cartilage fragment can be observed as a radio opaque line (10, 12). Also, there can be noticed the presence of mineralised cartilage fragments in the caudal articular sack or in the biceps brachial muscle sinovial sheath (22). If the mineralization isn't present, joint mice presence can only be suspected and confirmed using contrast radiography. Both sides must be examined due to the frequent bilateral localisation of OCD. Simple radiography or stress radiography (23), usually evidentiate only one defect, and the increased perilesional radio density and bone sclerosis offer very few hints on the severity of the lesion. For an accurate assessment and treatment, contrast arthrography (3, 10, 12, 24) or arthroscopy is necessary (2, 12 ,18, 21, 27).

Injection of 1-4 ml of contrast substance – 25% solution of sodium meglumine diartrisoate (Renographin®) or of another mielografic contrast solution in a concentration of 33 mg iodine/ml – allows radiographic mapping of the lesion and localisation of the detached cartilage and/or joint mice (1, 3 ,10, 28, 29). MR – magnetic resonance contrast arthrography after intraarticular injection of 5 ml dimeglumine gadopentate (Gd-DTPA) allows an accurate assessment of humeral OCD lesions (30).

Testing of the sinovial fluid shows inflammatory alterations (10).

In choosing the right treatment, a few aspects must be considered. The conservatory treatment, initially recommended as a start of treatment in all OCD cases (5, 11, 14, 19), now is only indicated for dogs under 6-7 months of age, with mild symptomatology and small lesions, without completely detached cartilage fragments. The surgical treatment is only recommended in dogs of over 6-7 months with medium o high severity symptoms, and with radiographically evident loose cartilage fragments. In chronic cases, usually associated with degenerative alterations, in those with no progress after 6 weeks of conservatory treatment or in dogs under 6 months and detached cartilage, it is recommended the surgical approach (2, 3, 12, 22).

The conservatory treatment

Consists of total locomotors break in association with administration of anti-inflammatory and/ or analgesic drugs for 4-6 weeks; it represent the recommended approach for bilateral lesions (10, 22). Kerstetter (14) recommends treatment supplementation with hyaluronic acid products (Adequan, Glycoflex, SynoviCare), and absolute body weight control. Healing is in some cases spontaneous, as a result of the cartilage fragment reattachment or of its resorbtion. The absence of radiographic healing after 6 weeks of treatment imposes the surgical approach thru arthroscopy or arthrotomy (1, 2, 3, 10, 16, 22, 26, 27).

Surgical treatment

Surgical treatment has as purpose eliminating the cartilage flap (humeral head arthroplasty) or fragment, including joint mice and stimulation of defect covering with fibrocartilaginous tissue (22). Several surgical approaches of the scapulae-humeral joint were proposed (cranial-lateral, caudal-lateral, caudal and cranial-medial, their comparative study being the object of numerous research (3, 10, 20, 22). In bilateral lesions, the recommended approach is arthroscopy (2, 4, 18, 21, 27). If this approach is impossible, first will be operating the leg with severe lameness, and after 4-6 weeks, the other leg will be subject to classic arthrotomy (17).

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