Review Article

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OSTEOARTHRITIS IN SMALL ANIMALS

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Keywords

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ABSTRACT

Osteoarthritis (OA) is a chronic disease that causes gradual damage to the joints. This disease causes problems such as pain, decreased mobility, and a decrease in overall quality of life. Regular check-ups are important for early detection of OA. Treatment options include pain management, slowing disease progression, increasing joint stability, and restoring normal joint function. Although there is no specific treatment for OA, a multimodal treatment approach is used. This treatment usually includes a combination of different modalities such as pharmacological interventions, surgical procedures, weight loss and physiotherapy.

INTRODUCTION

Osteoarthritis (OA) can be defined as a disorder of the joints characterized by deterioration of the articular cartilage. There is osteophyte formation in the joints, bone remodeling, changes in the periarticular tissues, and inflammation of varying severity [1]. Inflammatory arthritis includes immune-mediated or infectious causes. rheumatoid arthritis is considered more severe, progressive, and debilitating compared to osteoarthritis. Although osteoarthritis may limit normal function in severe cases, rheumatoid arthritis is recognized as a primary immune-mediated systemic condition with greater aetiological significance [2–4].

OA is a complicate disease and it is often misunderstood that it is only in cartilage. It is a disease involving the hyaline cartilage, synovial membrane, synovial fluid, subchondral bone and surrounding supporting tissues (muscle and ligament) in the joint. The joint can be considered as an organ with all the structures it contains [4].

Osteoarthritis is a degenerative condition that primarily affects movable joints. It is often caused by trauma, either abnormal force on a normal joint or normal force on an abnormal joint. Prolonged and vigorous use of normal joints does not typically lead to osteoarthritis, but abnormal joints are more susceptible to its development. Other less common causes in animals include metabolic, endocrine, and genetic disorders. Regardless of the cause, there is a common pathway leading to the breakdown of articular cartilage, subchondral bone, synovium, and joint capsule. Treatment of osteoarthritis requires a comprehensive understanding of the involved anatomy, physiology, and pathology [3].

OA is not a normal form of aging. Various methods have been developed in veterinary medicine to detect most diseases at an early stage. In fact, with the anatomical locations and predisposing factors, it is a specific disease [5]. Laxity, inconsistency, weight bearing variability, and joint damage increases OA predisposition by creating abnormal stress and chronic inflammation in the articular cartilage [6].

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OA is classified as idiopathic (primary) or secondary. It usually occurs secondary to the developmental anomaly, joint instability or trauma (hip dysplasia, osteochondritis dissecans, cruciate ligament rupture) in dogs [7]. Many factors increase the susceptibility to osteoarthritis. Systemic factors such as genetics, age, and obesity determine individual susceptibility.

Etiology

Idiopathic and generalized osteoarthritis is common and inherited. Many genetic factors can affect the incidence and severity of OA. Joint structure, gender and breed can affect it in different ways. Depending on gene diversity, susceptibility to OA may increase and even some genes alone can be effective in this regard [8,9].

It is known that age is effective in the formation of osteoarthritis. Aging affects joint structures, including cartilage tissue. The structure of normal articular cartilage is water, Type II collagen, aggrecan (respectively: 65-80%, 10-20%, 4-7%). In addition to these macromolecules, there are 5% small molecule proteoglycans, collagens, protein, fibronectin and lipids. With aging, chondrocytes are less synthesized, aggrecan molecules begin to lose their homogeneity, mitotic and synthetic activities, anabolic mechanical stimulants and growth factors decrease. As time passes, the cartilage gradually deteriorates, leaving the subchondral bone exposed and prompting its regenerative efforts to repair the damaged tissue. This leads to heightened bone density in the affected area and an uneven restructuring of the joint surface. Additionally, the formation of thick bony outgrowths known as spurs may occur. Consequently, the joint's ability to articulate becomes challenging. These changes are further exacerbated by a decrease in synovial fluid, which normally serves as a natural lubricant and cushion for the joint [10–12].

There is no clear information about a definite relationship between weight and osteoarthritis, but the risk of developing diseases that cause OA, such as hip dysplasia, increases depending on weight gain. Cruciate ligament rupture is especially more common depending on the increase in weight. Obesity is a risk factor for OA [13].

Studies have shown that in male and female animals, anterior cruciate ligament rupture causing OA is at the same level. According to a study, male dogs were found to have 1.47 times higher odds of developing elbow joint disease compared to females. Neutered dogs had 1.69 times higher odds of elbow joint disease compared to intact (non-neutered) dogs [14].

The initial onset of OA is believed to be caused by an imbalance between cartilage degradation and repair [15,16]. The specific events that trigger the disease are a topic of debate. One hypothesis suggests that pro-inflammatory cytokines released into the joint lead to the production of matrix metalloproteinases, which break down the cartilage matrix, resulting in bone remodelling and synovitis [17-19]. However, studies propose that synovitis and subchondral bone remodelling occur before articular degeneration in the early stages of OA [20,21]. Another theory suggests that meniscal degeneration, characterized by tissue fibrillation and reduced collagen levels, contributes to the progression of OA [22,23]. In later stages, osteophytes, subchondral cysts and sclerosis form as a direct consequence of cartilage degradation, bone remodelling, and synovitis [24,25].

Joint fractures, elbow and hip dysplasia, rupture of the cranial cruciate ligament (CCL), and joint instability because of trauma or angular limb deformity are predisposing factors. In disease processes affecting the joint, there is a permanent state of inflammation in the affected joint. In long-lasting inflammation, the body responds by trying to stabilize the area through the formation of new bone. This leads to the development of OA. Arthritis can also affect joints along the vertebral column. In some cases, for example, hip dysplasia and spondylosis are the same, and their clinical manifestations may be similar. If only hip dysplasia is detected and spondylosis is overlooked, clinical symptoms may not decrease. Therefore, it is important to look for pain and signs of spondylosis (spinal degeneration) in the cervical, thoracic and lumbar spinal segments, as well as in the thoracolumbar and lumbosacral junctions [5].

Diagnosis

Instability, uneven load bearing, and joint injury lead to abnormal stress on the intra-articular cartilage and the development of chronic inflammation, resulting in osteoarthritis. OA-related symptoms; pain, swelling, stiffness and crepitation of the affected joints are often reported as decreased joint function or limited range of motion [5].

Regular check-ups and pain assessments are important to detect and intervene in the development of pain and arthritis early. Often animals with OA can adapt to the condition and compensate for their pain, so a thorough examination is necessary to identify the source of the discomfort. A thorough physical examination

helps locate the source of the pain. For this, an orthopedic and neurological evaluation may be useful [5,26].

The diagnosis of OA can be supported by history, orthopedic and neurologic examination findings, cytology, or imaging. Physical examination signs of arthritis are not specific to OA and need to be combined with further diagnostics and patient history. Radiographs can show various signs of OA depending on its severity, including fluid accumulation, osteophytosis, and subchondral sclerosis (Figure 1). Researchers have explored the use of biochemical markers for diagnosing arthritis before clinical signs appear or predicting disease progression, but practical clinical applications of these markers have been limited so far [5,26,27].

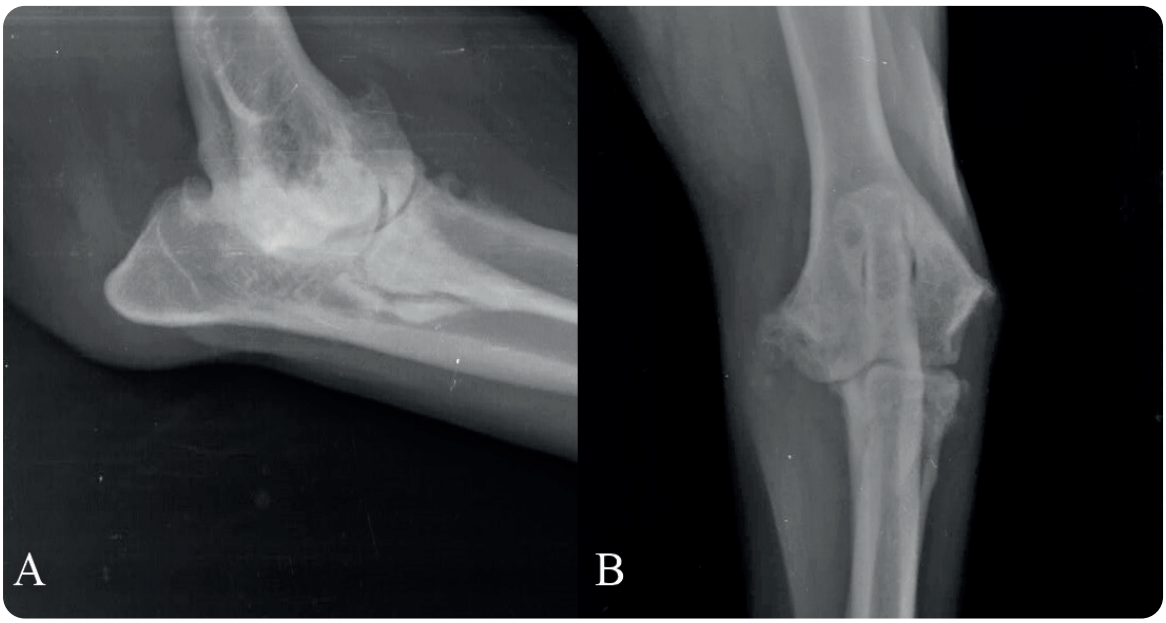

Figure 1. Anteroposterior (A) and mediolateral (B) projections of a mix dog elbow with marked degenerative joint disease. Osteolysis and periarticular new bone formation are present.

It is also important to understand the underlying cause of pain in aging animals and to exclude other potential diseases such as septic synovitis, osteosarcoma, bursitis, osteomyelitis and tendinitis/tendovaginitis. History and physical examination are helpful in the differential diagnosis of some diseases [5].

The clinician's role in diagnosing OA is not just about recognizing the disease but also about ruling out other possible causes. Many animals may have OA in one or more joints. However, determining whether OA is the primary source of clinical symptoms can sometimes be challenging. A thorough physical examination is crucial in eliminating the possibility of other problems [5,26].

In some cases, the presence of arthritic changes is evident and it is clearly the cause of the patient's issues. However, it's important to differentiate between different types of arthritis, such as immune-mediated arthritis or infectious arthritis, as they require different management approaches. Patient history and signalment (patient characteristics like age, breed, etc.) may be sufficient for making a diagnosis. Additional diagnostic steps involve evaluating the joint fluid through cytologic examination, specifically looking at the number and type of nucleated cells present. Generally, OA is considered a "noninflammatory" arthritis because of the cell types found in the joint fluid. Animals with OA typically have elevated cell counts but lower than 5000 cells/mL. The fluid in OA primarily contains mononuclear cells, while inflammatory arthritides have primarily polymorphonuclear cells [5,26,28].

Radiography is a very good method for imaging joints and bones [29,30]. To see the difference between normal and abnormal joint structures, radiography details, sharpness and contrast must be good. While high-quality radiographs provide maximum information, poor-quality radiographs are open to misinterpretation [31,32]. Radiographic examination plays a very important role in the diagnosis of OA. Radiography can reveal bone changes in and around the joint. In addition, it can rule out other abnormalities that may be indicative of diseases other than OA, such as excessive bone destruction, bone proliferation, or suspicious findings of other diseases [5].

Tomography is a better imaging tool than radiography for early diagnosis. Transversal images are taken from multiple anatomical planes so that changes in the joint can be detected by three-dimensional analysis. Magnetic resonance imaging (MRI) is superior to tomography in imaging periarticular and intraarticular soft tissues. Compared to radiography, new bone formations and bone destruction are displayed in more detail on tomography (Figure2 and 3) [33,34].

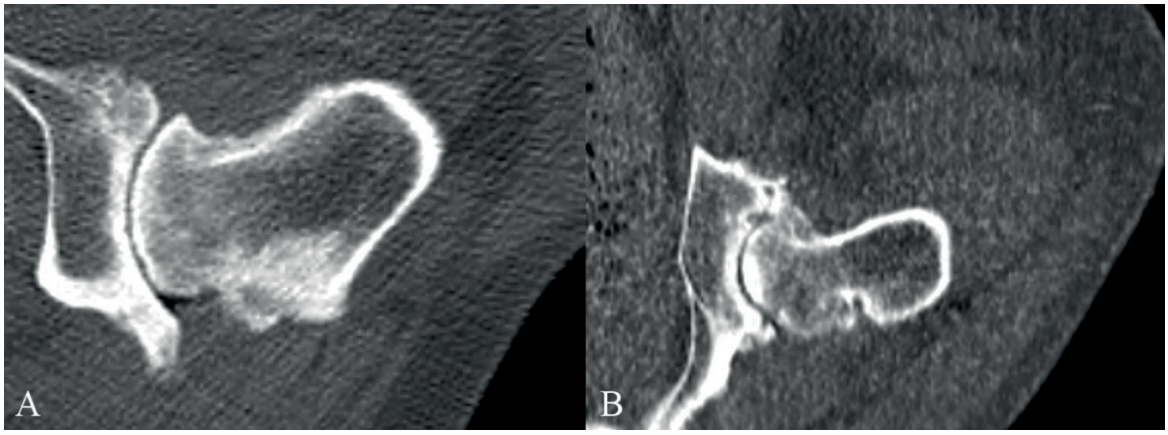

Figure 2. Transversal (A) and dorsal (B) right pelvic CT image of 11 years old Kangal with marked osteoarthritis. diffuse sclerosis of the caput femoris and acetabulum.

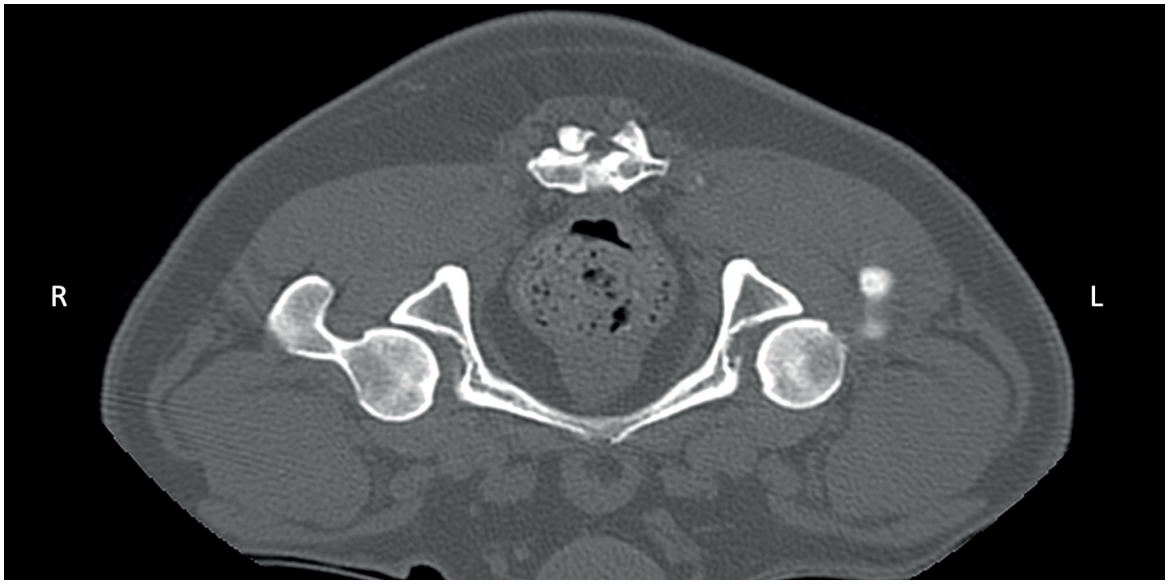

Figure 3. Transversal pelvic CT image of 7 years old, male, Golden Retriever. bilateral hip dyplasia and modarate osteoarthritis at acetabulum. R: Right, L: Left.

MRI has been established as a reliable non-invasive method for assessing cartilage in osteoarthritis, supported by studies. It is potentially capable of detecting early degeneration using high-resolution techniques [34,35].

Treatment

OA is a lifelong condition that cannot be cured but can be effectively managed. The management approach depends on the severity and location of the disease. Clinicians can guide the therapy by considering factors such as pain, normal activities, mobility, pet owner's goals. Establishing a pyramid of pain management, joint protection, nutritional support, and strengthening can be a natural progression in managing OA.

Surgical or conservative methods may be preferred in the treatment of OA. Surgical management aims to address the primary cause or perform salvage procedures. Weight control, physical therapy, and medications are components of conservative management. Specific protocols for conservative management may vary based on the animal's needs and the owner's abilities or preferences [36,37].

Surgical options are most effective in the early stages of the OA, such as repairing torn ligaments or treating conditions like osteochondrosis, hip dysplasia, elbow dysplasia, articular fractures, and growth deformities.

Surgical options are most effective in the early stages of the OA, such as repairing torn ligaments or treating conditions like osteochondrosis, hip dysplasia, elbow dysplasia, articular fractures, and growth deformities. However, if the disease has progressed significantly, surgery may still be performed to alleviate symptoms, but the patient will likely have signs consistent with OA [38].

Salvage procedures can also be performed surgically to relive the arthritis symptoms. These procedures involve eliminating the affected joint or limb, ranging from simple procedures like excision arthroplasty to more complex ones like joint replacement or arthrodesis. The clinical outcomes of these treatments can vary and can be performed at any stage of the disease. Surgical procedures may be preferred when conservative treatment is unsuccessful [39].

Weight loss is crucial in managing OA. Overweight is a common issue in small animals and a significant risk factor for the development of OA. In older dogs, obesity can also increase the injuries such as damage to the CCL. Excess weight adds strain to already painful joints and contributes to chronic inflammation throughout the body. Adipose tissue, or body fat, is proinflammatory and produces active cytokines. Animals with excessive body fat are more likely to be inactive, and this chronic inflammatory state negatively affects joint health. Research has demonstrated that reducing caloric intake and achieving weight loss can decrease the clinical signs of OA and improve mobility [36,37,40].

A study mentioned the benefits of exercise in reducing joint pain, particularly supervised aerobic exercise performed at least three times a week [41]. Swimming has also shown positive effects on joint function in dogs with OA [42]. However, these therapies may not be readily accessible or affordable for all dog owners, particularly those with mild OA. Walking and running exercises have shown benefits similar to aquatic therapy in humans and are more accessible for dogs. A tailored exercise prescription based on the severity of OA in dogs may be necessary, and activity monitors can help veterinarians understand the animal's activity patterns and make exercise recommendations [43].

Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used for the treatment of OA due to their effectiveness in relieving pain and their ease of administration. Multimodal therapy, combining pharmacologic and nonpharmacologic approaches, is often recommended for the complex nature of pain in OA. It is suggested to use the lowest effective dose of any drug, especially when it is a part of multimodal therapy, to minimize potential side effects [3]. NSAIDs such as carprofen, etodolac, deracoxib, meloxicam, tepoxalin, and firocoxib can be used for OA management. The analgesics such as tramadol, amantadine, gabapentin, and others can be used in multimodal treatments [3,44,45].

CONCLUSION

OA is a chronic disease that causes gradual damage to the joints, causing pain, decreased mobility, and a decline in overall quality of life. Regular examinations are important for early diagnosis of the disease. There is no certain treatment for OA, multimodal treatment options aim to manage pain, slow disease progression, increase joint stability and restore normal joint function. These treatment approaches typically include a combination of modalities such as pharmacological interventions, surgery, weight loss and physiotherapy.

Conflict of interest statement

The authors declare that they have no conflicts of interests.

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