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Management of Osteoarthritis in Small Animals Mini Series

Session One: What is osteoarthritis and how do we assess it?

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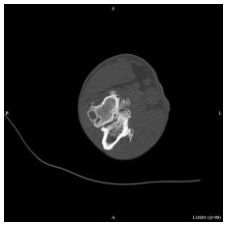

MINISERIES: OSTEOARTHRITIS IN SMALL ANIMALS

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SESSION 1

1. JOINT ANATOMY AND PHYSIOLOGY

ANATOMY

Diarthrodial joints include:

- Hyaline articular cartilage that covers the subchondral bone
- Joint capsule (fibrous capsule and synovial membrane, which contains Type A and B synoviocytes), and ligaments.
- Joint space that contains synovial fluid, which is a dialysate of blood plasma formed by plasma filtration through 2 layers, the vascular endothelium and the synovial interstitium.

Articular cartilage is made up of 70% water, being the remaining components collagen, proteoglycans and other glycoproteins, and a minimal proportion of cartilaginous cells (chondrocytes, chondroblasts and chondroclasts). Articular cartilage is moderately resistant to compression, and it deforms under loading as a results of its composition and distribution of structures. Articular cartilage has 3 distinct areas:

- Zone I (superficial of tangencial zone): It is superficial, collagen fibres are distributed tangencially to the surface and it is rich in cells.
- Zone II (transitional zone): It is located in the middle, with bigger and rounder cells. Collagen fibres are distributed perpendicularly to the surface, and small fibres branch out in a similar way to branches in a tree.
- Zone III (radiate zone): It is deep and contains bigger cells that are oriented longitudinally in columns (perpendicular to the surface), following the same direction of the collagen fibres. This deep layer is rich in proteoglycans.

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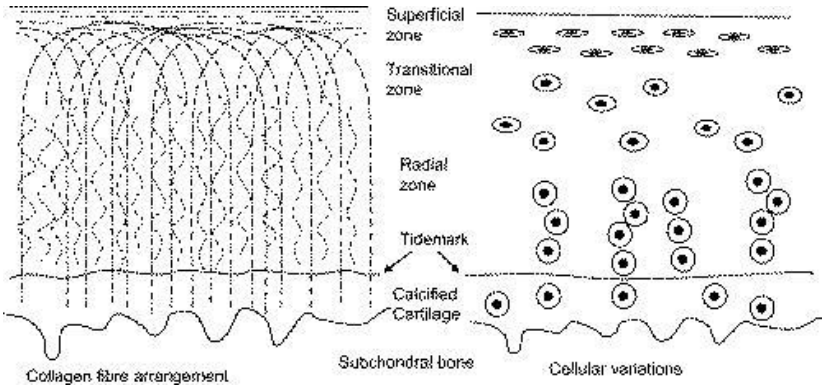
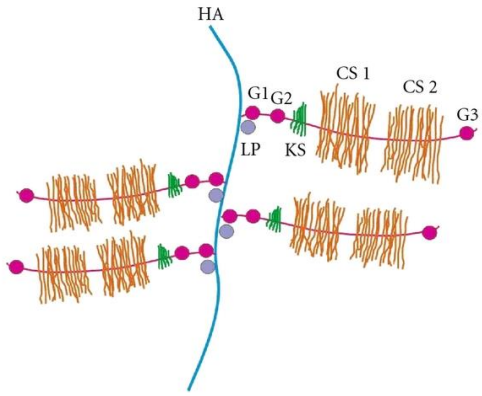




Most of the collagen present in articular cartilage is type II, although smaller amounts of collage types VI, IX, XI, XII and XIV are also present. Proteoglycans are important components of articular cartilage. They are glycosylated proteins, big in size, which consist of a central protein that is covalently linked to one or more glycosaminoglycans (GAG). These can be glucosaminoglycans (heparan or keratan sulphate) or galactosaminoglycans (chondroitin or dermatan sulphate). The most common and abundant proteoglycans are aggrecans (big conglomerates of proteoglycans), which have a significant affinity to water and maintain the articular cartilage hydrated, essential for its correct functionality.

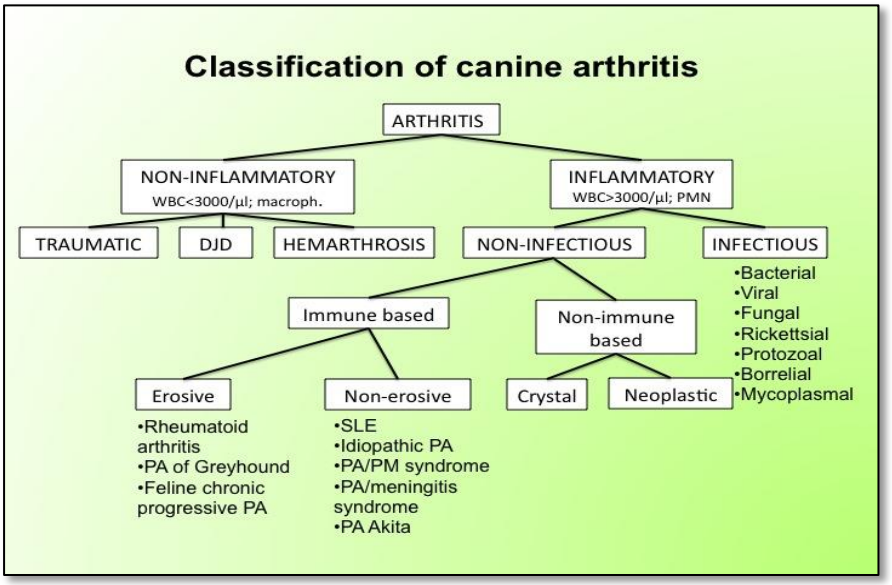
PATHOPHYSIOLOGY

Specific insults to the joint (i.e. bacteria, mechanical factors, etc.) cause an inflammatory reaction. Synovium is extremely vascular, with no basilar membrane, so that bacteria and inflammatory cells can cross it easily and have access to the joint space. Inflammatory cells synthetize and secrete cytokines that attract more proinflammatory cells. With time, bone and articular cartilage destruction occurs secondary to the proteolytic enzymes induced by cytokines.

CLASSIFICATION OF JOINT PATHOLOGIES

Joint pathologies can be classified in 2 big categories:

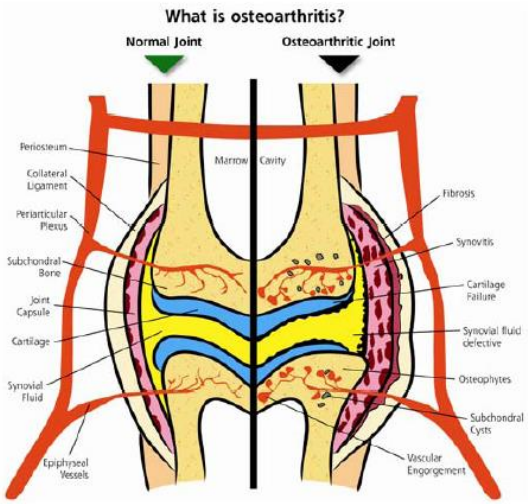
- Inflammatory arthropathies
- Non-inflammatory arthropathies



OSTEOARTHRITIS

1. **Pathophysiology**

Osteoarthritis (OA) is the most frequent joint condition in small animals, affecting 20% of adult dogs and 60% of adult cats. Expenses derived from the diagnosis and management of OA in small animals has been estimated in millions of euros every year.



OA can be primary (no obvious cause) or secondary, being the latter most common in companion animals. It is a multifactorial condition inducing articular cartilage destruction, remodelling of subchondral bone and inflammation of synovial membrane. There are some predisposing factors in the development of OA (i.e. genetics, age, systemic factors such as obesity, reproduction status, etc.), and some determinant factors causing OA (i.e. biological, mechanical, infectious factors, etc.)

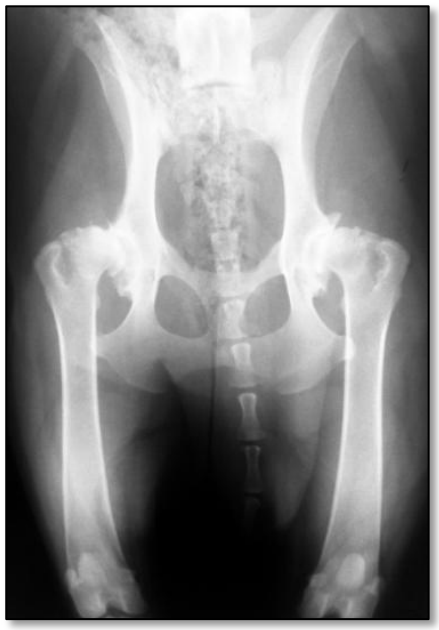
Changes in the composition of articular cartilage occur during OA, causing a decrease in resistance to tensile and compressive forces. This leads to fibrillation of the articular surface, progressing to loss of articular cartilage and formation of cracks and defects. OA progression takes place in 3 overlapping stages:

- *Stage 1:* There is degradation of the extracellular matrix with increase in water content, decrease in size of the aggrecan molecules, and injury of the

collagen molecules. All this leads to decreased rigidity of the articular cartilage.

- *Stage 2:* Chondrocytes try to compensate these changes by means of proliferation and increased metabolic activity. This situation can last for months or years.
- *Stage 3:* Chondrocytes cannot maintain this level of repairing activity and it ends up with loss of cartilaginous tissue.

The beginning of the degenerative process is caused by an imbalance between the anti-inflammatory factors/cytokines (IGF, TGFb, BMPs, IL-4, IL-10) and the proinflammatory cytokines (IL-1, TNF, IL-8, IL-15, IL-17, IL-18, IL-21, LIF). This imbalance generates the arrival of inflammatory cells that, together with the degenerated chondrocytes, synthetize more proinflammatory cytokines, which attract more inflammatory cells. This is a vicious circle that promotes the continuous degeneration of the joint. The cytokines stimulate the synthesis of metalloproteinases and other proteolytic enzymes. Damage of the subchondral bone and synovial membrane triggers the nociceptors, which through a variety of pathways reach the central nervous system, generating a pain sensation. The synovial membrane participates actively in the development of OA, suffering from different degrees of synovitis and synovial hyperplasia. The remains of cartilage tissue stimulate the secretion of proinflammatory cytokines and proteolytic enzymes by the synovium.



2. Diagnosis

Animals with OA suffer from a chronic and constant pain that induces variable signs such as exercise intolerance, unwillingness to jump, lameness, stiffness and behavioural changes. This chronic pain can cause sensitization of the central and peripheral nervous system, which could lead to hyperalgesia or alodynia. It is therefore essential chronic pain is diagnosed, evaluated and treted.

History and signalment of the patient provide important information regarding the possible diagnosis of OA and the degree of pain the patient may be suffering. During orthopaedic examination the clinician may found crepitus, pain, decreased range of motion or joint effusion. Radiographs taken of the affected joint generally show degenerative changes in the joint with formation of osteophytes, enthesiophytes, subchondral sclerosis, joint effusion, soft tissue swelling, bone cysts, and in some occasions, intraarticular mineralised bodies.

However, it is important to mention that there is no direct relationship between the radiographic severity of OA and the severity of the clinical signs, and therefore it is essential the clinician determines a management plan based on the degree of clinical signs of the patient. Advanced imaging techniques, such as CT, can also be used to diagnose OA in small animals. The next step in our diagnostic work up may be arthrocentesis and analysis of the synovial fluid, including cell count and cytology, chemical analysis and culture.

Feline osteoarthritis has a similar pathophysiology as canine OA, although the clinical signs may vary. Cat are lighter and more agile, which help this species be able to compensate better. Cats usually present with changes in behaviour and mobility rather than lameness.

It is essential the clinician is able to diagnose and grade chronic pain in small animals, which is challenging. There are objective (heart rate, respiratory rate, blood pressure, pupil dilation, cortisol levels) and subjective methods (vocalization, posture, behaviour, etc) to evaluate pain in animals. Several methods have been reported to grade pain in animals:

- Scales: numerical vs VAS
- Quality of Life Questionnaires (i.e. CSOM, CBPI, Helsinki, Galsgow, etc)

Lameness would be a manifestation of pain, and this can also be evaluated and graded through gait analysis. This can be performed in a subjective or objective way:

- Subjective gait analysis:
    - Visual
    - Numerical scales vs VAS
    - Slow motion video
- Objective gait analysis:
    - Kinetics (force plate vs pressure mat)
    - Kinematics
    - Activity monitors