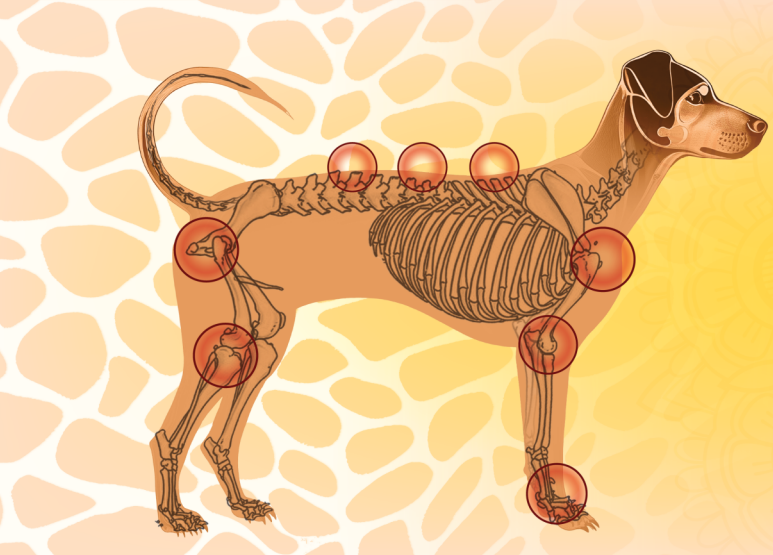

For Veterinarians only

PET POD

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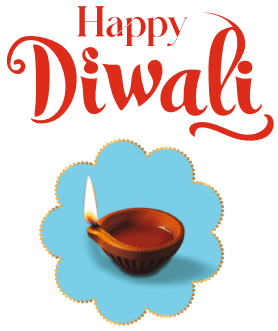
Canine Osteoarthritis: Disease Progression and Management



Dear Vets,

Canine osteoarthritis (OA) remains one of the most significant causes of chronic pain and reduced mobility in companion animals, affecting both longevity and quality of life. The pathophysiology of OA is multifactorial, involving a complex interplay of inflammatory, biochemical, mechanical, and structural changes within the joint. This evolving understanding has reshaped therapeutic approaches, encouraging Veterinarians to move beyond symptomatic relief and adopt comprehensive, multimodal management strategies. Given its multifaceted nature, successful management requires a lifelong, multimodal approach rather than a single therapeutic intervention. By integrating lifestyle modification, pain management, nutritional support, rehabilitation, environmental adjustments and disease-modifying therapies.

In recent years, advancements in pharmacological therapies have focused not only on controlling clinical signs but also on addressing the underlying structural changes associated with OA. Among these, Pentosan Polysulfate Sodium (PPS) has gained considerable attention for its potential disease-modifying effects. PPS may help reduce cartilage degradation, improve synovial and subchondral blood flow, stimulate hyaluronan and proteoglycan synthesis, and promote cartilage repair. By highlighting both the scientific understanding of disease pathogenesis and advances in therapeutic interventions, we aim to provide veterinarians with clinically relevant insights that support evidence-based decision-making in everyday practice.

We would also be delighted to learn about your approach to managing canine OA in routine practice. Kindly share your insights by scanning the QR code below or alternatively reach us *via* e-mail at petpod@ intaspharma.com.

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Osteoarthritis (OA) is a chronic, non-infectious inflammatory joint disorder characterized by progressive degeneration of articular cartilage, altered subchondral bone metabolism, periarticular osteophyte formation, and varying degrees of synovial inflammation. Rather than affecting cartilage alone, OA involves the entire joint structure, including articular cartilage, synovial membrane, subchondral bone, ligaments, joint capsule and periarticular tissues. These progressive structural and inflammatory changes contribute to chronic pain, joint stiffness, reduced mobility, swelling and lameness, ultimately impairing functional ability and quality of life in affected dogs. The management of OA primarily focuses on alleviating pain, preserving mobility and joint function, slowing functional deterioration and maintaining overall quality of life. In addition, the need to consider the potential sustained negative effects of pain, central sensitization, and activity impairment on the affective state (anxiety, depression and sleep impairment) and cognitive dysfunction of dogs is increasingly understood. Given its chronic and progressive nature and the complex mechanisms underlying OA-associated pain, understanding of its prevalence, pathophysiology, and contemporary management approaches is essential for early recognition and effective long-term care of affected dogs.

Prevalence

OA is one of the most prevalent musculoskeletal disorders and major cause of lameness and joint disease in companion animals. It commonly affects the weight-bearing joints of medium- to large-sized dogs, although any synovial joint can be affected. The prevalence of OA increases with age, with approximately 20% of middle-aged dogs and up to 90% of older dogs reported to have OA in one or more joints. The prevalence also varies depending on the joint involved, with reported rates of 39.2% in shoulder, 57.4% in elbow, 35.9% in hip and 36.4% in stifle. Breed predisposition has also been observed, particularly among large-breed dogs viz. Golden Retrievers (7.7%), Labrador Retrievers (6.1%), Rottweilers (5.4%) and German Shepherds (4.9%).

The exact pathogenesis of OA remains unclear, though it is understood to be multifactorial, encompassing genetic, environmental, and immunological factors that contribute to its progression. Although the hallmark of the disease process is abnormal repair and progressive degradation of articular cartilage, involving the entire synovial joint, resulting in changes to synovial membrane, synovial fluid and subchondral bone. The anatomical differences between a normal and an osteoarthritic joint are illustrated in Fig. 1. OA progression involves four stages, as described below.

- **Stage One:** In this, minor bone spurs begin to develop. The cartilage matrix begins to break down due to chondrocyte's metabolism being affected and increasing the production of matrix destroying enzymes, metalloproteinases (MMPs). The severity of cartilage lesions can be correlated with the levels of MMP-1. Cartilage lesions disrupt the function of cartilage, increasing friction and inflammation in joints, resulting in pain.
- **Stage Two:** It is considered the mild stage. This stage involves erosion of bone due to cartilage lesions. This can cause new bone growth, osteophyte, also called bone spur, which affect normal joint movement. In this stage, proteoglycan and collagen fragments are released into synovial fluid. In adult dog, protecoglycan turnover is quicker (300 days) than estimated collagen turnover (120 years). Marked protecoglycan loss of articular cartilage is irreversible and results in joint degeneration.

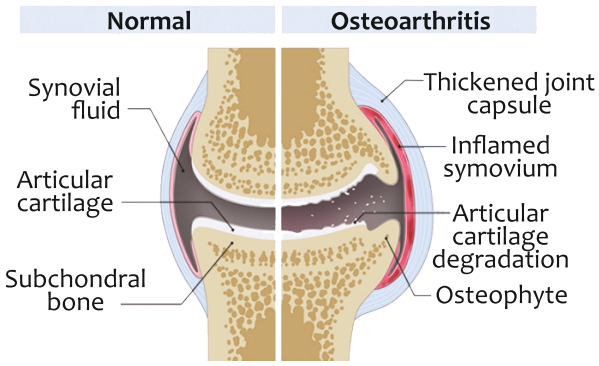

Fig. 1: Anatomy of normal and osteoarthritic joint

- **Stage Three:** It is considered the moderate stage. The cartilage, in-between bones thins out and loses cushion. The space between bones is also narrowing, causing grinding between the adjacent subchondral bones. During this stage, symptoms are more severe and inflammation begins to occur. Production of synovial macrophages occurs, including MMP, cytokines (interleukin 1), and tumor necrosis factor-alpha (TNF-α). Once the synovial macrophages are produced, they can destroy tissues by diffusing back into the cartilage and can also stimulate chondrocytes.
- **Stage Four:** It is the final stage and considered the severe stage. In this, joint space is dramatically reduced, cartilage is almost thinning and joint mobility is reduced greatly. Early diagnosis becomes key to prevent further damage to joint and alleviate symptoms.

Previously, OA was predominantly considered as a degenerative condition resulting from mechanical stress and the aging process. However, recently a substantial immune-mediated dimension has unveiled in its advancement. The field of osteoimmunology has redefined OA as a low-grade, chronic inflammatory disease involving dysregulated immune-bone crosstalk (Fig. 2). In which, development of OA involves, immune cells such as macrophages, dendritic cells (DCs), neutrophils, mast cells (MCs), natural killer (NK) cells, T cells and B cells orchestrate destructive immune-bone crosstalk within the joint microenvironment. This engagement instigates and perpetuates inflammatory and catabolic processes

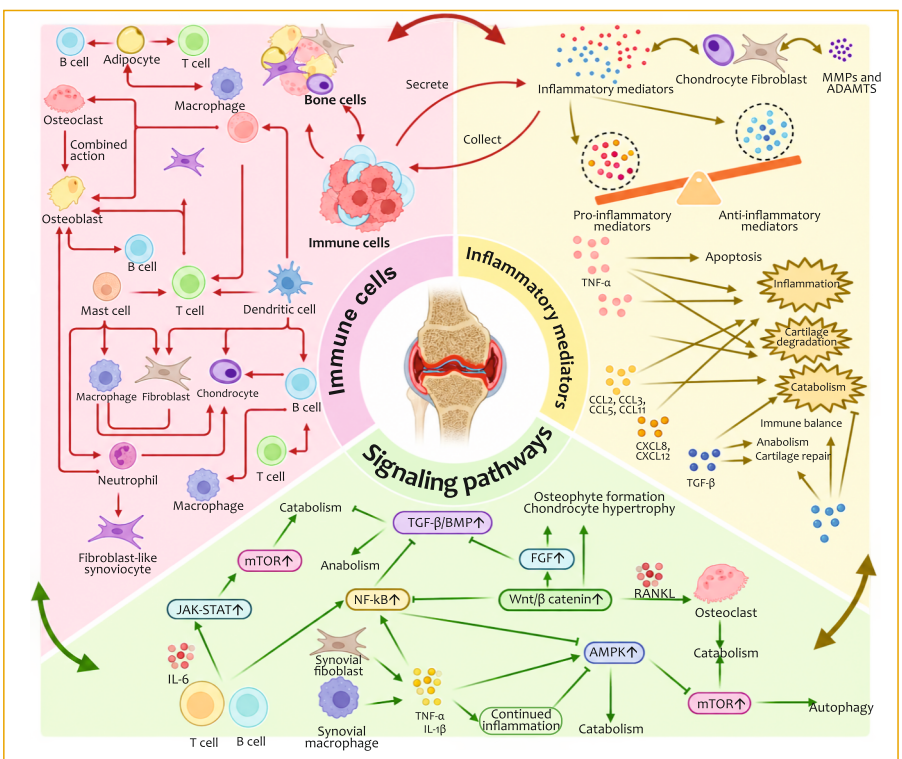

Fig. 2: Immune-bone crosstalk in OA pathogenesis

within joint. These cells release a broad-spectrum of inflammatory mediators; such as cytokines (e.g., TNF-α, IL-1β) and chemokines, that act on chondrocytes, synoviocytes, and bone-resident cells, thereby perpetuating cartilage breakdown and subchondral bone changes. Moreover, pathogenic signaling pathways, such as nuclear factor-kappa B (NF-κB), Wingless-related integration site (Wnt), adenosine monophosphate-activated protein kinase (AMPK), mammalian target of rapamycin (mTOR), TGF-β/bone morphogenetic protein (BMP), and fibroblast growth factor (FGF), integrate these immune signals and coordinate cellular responses that drive disease progression.(NK) cells, T cells and B cells orchestrate destructive immune-bone crosstalk within the joint microenvironment. This engagement instigates and perpetuates inflammatory and catabolic processes

Classification

The heterogeneous etiology of OA makes its classification challenging. Based on etiology, OA can be broadly classified as primary and secondary.

- **Primary OA:** It develops without an identifiable underlying initiating cause and is therefore also referred to as idiopathic OA. It is generally associated with progressive degenerative changes within the joint. Primary OA may be further classified as localized, involving a single joint, and generalized, involving multiple joints. Factors contributing to primary OA can broadly include age-related joint degeneration and stiffness in older large-breed dogs.
- **Secondary OA:** It develops as a consequence of abnormal mechanical stresses on articular cartilage or pre-existing joint disease. Factors contributing to secondary OA can broadly include may broadly include genetic predisposition, advancing age, nutritional factors, obesity, joint injury or trauma, excessive or repetitive physical activity, and developmental orthopedic diseases.

Etiology

OA in dogs is a multifactorial, progressive joint disorder that develops through interactions among mechanical stress, joint instability, abnormal joint development, trauma, inflammation, and age-related changes. In dogs, OA commonly develops secondary to an underlying orthopedic abnormality or joint injury, which alters normal joint biomechanics and ultimately contributes to progressive cartilage damage and changes in other joint tissues. Predisposing factors include;

Genetic

Multiple genetic factors influence the development and severity of OA, varying by joint, breed and gender. Many genes involved in cartilage extracellular matrix proteins contribute to early-onset of OA, including mutations in type II collagen and other collagen genes (types IV, V and VI). Additionally, a 14-bp deletion in proopiomelanocortin (POMC) gene, present in about 25% of Labrador Retrievers, is strongly associated with obesity characterized by increased body weight, higher fat composition, and greater food motivation which further predisposes these dogs to OA.

Age

Aging is associated with progressive alterations in articular cartilage composition, chondrocyte function, and the ability of joint tissues to respond to mechanical stress and injury. With advancing age, chondrocytes exhibit reduced proliferative and synthetic capacity, resulting in decreased synthesis and turnover of essential extracellular matrix components, including aggrecan. Their responsiveness to anabolic mechanical stimuli and growth factors also declines. In addition, aging is associated with changes in the size, structure, and uniformity of aggrecan molecules, which may compromise the biomechanical properties of cartilage and increase its susceptibility to degeneration.

Overweight and Obesity

It is observed that obese dogs affected by OA often exhibit more severe clinical symptoms than non-obese dogs. It may increases mechanical stress on weight-bearing joints and contribute to cartilage degeneration

Exercise, Diet and Housing

Repetitive high-impact exercise, sporting activity, or physical stress may contribute to joint injury and OA, particularly when underlying joint abnormalities are present. Puppies on slippery surfaces during development have a higher incidence of hip dysplasia and osteoarthritis. Diets high in fat and protein during developmental phase have been identified as a risk factor for elbow osteoarthritis.

- **Osteochondritis dissecans (OCD):** It is a manifestation of osteoch-ondrosis, characterized by disturbance of normal cartilage maturation and endochondral ossification. OCD is a condition that affects the shoulder joint in dogs; the condition is also called as shoulder dysplasia. In this, cartilage covering the surfaces of bones in shoulder joint fails to develop correctly. Abnormal cartilage growth leads to the formation of small pieces of cartilage that break off and float inside the joint, causing pain and stiffness. OCD lesions predominantly occur in adolescent large-breed dogs. Radiogra-phically, shoulder OCD is typically charac-terized by mineralized flap over the caudal region of humeral head with associated subchondral sclerosis (Fig. 5).

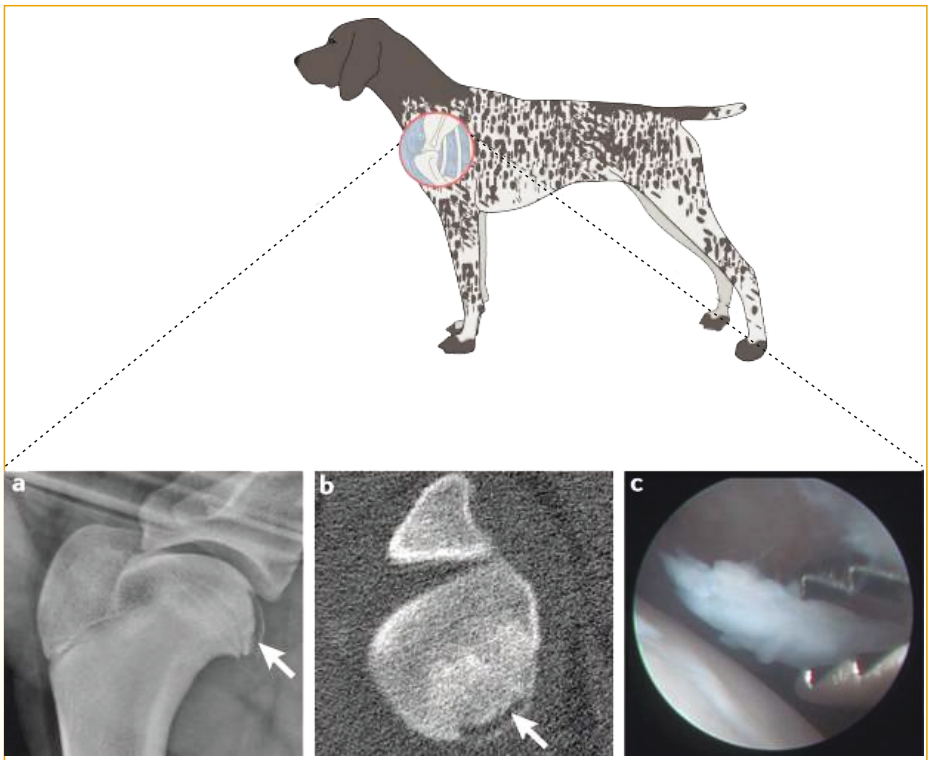

Fig. 5: Osteochondritis dissecans in dogs- a,b; Lateral radiograph (part a) showing a classical mineralized flap over the caudal region of humeral head, which can also be seen in a transverse CT (part b) of the same shoulder with associated subchondral sclerosis (arrows mark the lesion), c; Osteochondritis dissecans flap being removed arthroscopically

- **Avascular necrosis of femoral head:** It is also known as Legg-Calvé-Perthes disease (LCPD), a painful condition that affects hip joint of young, growing dogs, particularly small breeds. It occurs due to disruption of the blood supply to the developing femoral head, resulting in avascular necrosis. As the disease progresses, femoral head becomes misshapen. This creates an irregular hip joint and eventually leads to OA. Radiographic findings may include, abnormal articular surface of femoral head, showing typical focal lucencies and new bone formation, as seen in advanced lesions (Fig. 6).

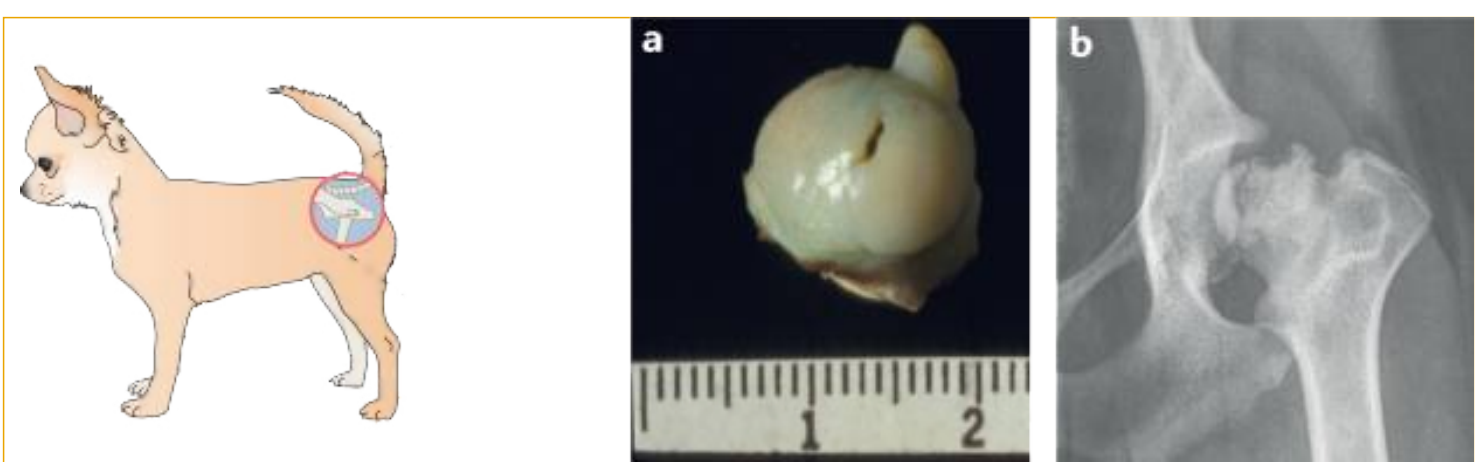

Fig. 6: Avascular necrosis of femoral head- a; Excised femoral head with an abnormal articular surface morphology and a central dark line indicating an articular surface defect, b; Ventrodorsal radiograph of a dog hip joint showing typical focal lucencies and new bone formation, as seen in advanced lesions

Clinical Signs

The signs of OA may vary from mild to severe based on chronicity and number of joints affected. Signs may include any of the following:

- Pain
- Stiffness
- Waddle or truncal sway
- Bunny hopping
- Lameness or a change in gait
- Decrease in activity or reluctance to walk, run or exercise
- Difficulty navigating stairs or jumping on couches
- Difficulty getting up from lying down
- Muscle wasting
- Sudden change in behaviour, such as irritability or aggression
- Decreased activity

Diagnosis

It is based on multimodal approach including history, physical examination, diagnostic imaging, and assessment of pain and mobility. Other potential causes of lameness should also be considered and ruled out where it is appropriate.

a. Physical examination: The examination includes assessment of gait, posture, weight-bearing and joint function. Each affected joint is evaluated through its range of motion to identify pain, stiffness, swelling, warmth, crepitus, joint instability or reduced mobility, which may indicate underlying joint disease.

b. Radiography and other diagnostic imaging: Radiography is commonly used to identify structural changes associated with OA, including osteophyte formation, subchondral bone changes, joint-space alterations and joint remodelling. Advanced imaging techniques such as CT scan or MRI may be considered when more detailed assessment of bone, cartilage or surrounding soft tissues is required.

c. Mobility and Pain assessmen: Validated clinical assessment tools, such as the Canine OsteoArthritis Staging Tool (COAST), can support systematic evaluation and staging. Changes in mobility, pain, comfort, activity, and overall function can also be monitored over time to assess disease progression and response to management.

d. Ruling out other causes: Lameness, pain and stiffness are not specific to osteoarthritis. Differential diagnosis may include ligament or tendon injuries, fractures, joint infection or inflammation and neurological conditions. Appropriate diagnostic investigations help differentiate these conditions and establish the underlying cause.

Management

The pathophysiology of OA is complex and involves multiple interacting inflammatory, biochemical, mechanical and structural processes. Consequently, it is unrealistic to expect a single therapeutic intervention to provide optimal clinical improvement. OA management represents a lifelong commitment, with the primary goals of reducing pain and inflammation, slowing disease progression maintaining or improving muscle strength and endurance, preserving joint range of motion, enhancing mobility and functional performance, and ultimately improving quality of life. These objectives can be achieved through an integrated combination of lifestyle modification, pain management, nutritional supplements, physical rehabilitation, environmental modifications, disease modifying osteoarthritic drugs and other.

- **Lifestyle modification:** The primary objectives are to reduce excessive joint stress, maintaining muscle strength and mobility and improving overall quality of life. It includes weight management with controlled, low-impact exercise, such as short frequent walks, swimming, or appropriately supervised range-of-motion exercises, can help preserve joint mobility, muscle mass, and functional capacity while avoiding excessive stress on compromised joints.

- **Pain management:** Effective analgesia is an essential component of OA management. Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used to control inflammation and nociceptive pain associated with OA. E.g., Carprofen, Firocoxib, Robenacoxib, Meloxicam **(Melonex)** and other analgesics or adjunctive agents viz. Grapiprant, Amantadine, Pregabalin and Gabapentin **(Gabapin Pet)**.
- **Nutritional supplements:** Nutraceuticals can be incorporated as an adjunct to multimodal management of OA in dogs, with the aim of supporting joint health, mobility and overall quality of life. Commonly used supplements include Omega-3 fatty acids, Glucosamine, Chondroitin sulfate, Undenatured type II collagen (UC II), Green-lipped mussel, Avocado/soybean unsaponifiables and Boswellia. Glucosamine combined with Chondroitin sulfate (**Synopet** and **SynoPet HA**) is commonly used chondroprotective nutritional supplement in the management of OA. Glucosamine exert anti-inflammatory and cartilage-supportive effects by modulating the activity of enzymes involved in cartilage degradation, supporting the synthesis of extracellular matrix components and reducing apoptosis of articular chondrocytes. Chondroitin sulfate, an important component of cartilage extracellular matrix, contributes to cartilage structure and function and inhibit enzymatic degradation of cartilage matrix components. Together, Glucosamine and Chondroitin sulfate are intended to support cartilage integrity, joint function and mobility.
- **Environmental modifications:** A soft, well-padded bed should be provided, along with good flooring to prevent slips and falls, minimize climbing of stairs, introducing portable ramps, harnesses and support devices may be helpful for managing arthritic dogs.
- **Other treatments:** Additional therapies may be considered in selected patients based on disease severity, affected joints, and response to conventional treatment. These may include surgical management, intraarticular injections such as, platelet rich plasma, hyaluronic acid, stem cell therapy, Methylprednisolone, monoclonal antibodies, acupuncture, chiropractic, pulsed electromagnetic field therapy, prolotherapy, joint mobilization and manipulation, radiation therapy, and nuclear magnetic resonance therapy.
- **Disease modifying osteoarthritic drugs (DMOADs):** Over the past decades, increased understanding and knowledge on the molecular mechanisms driving the progression of the disease has led to the use of alternative treatments designated as disease modifying osteoarthritic drugs (DMOADs). These agents are anticipated to target the pathophysiological processes of OA thereby preventing, retarding progression of or reversing morphologic changes associated with OA cartilage joints. Includes; Polysulfated Glycosaminoglycan (PSGAG) and Pentosane Polysulfate (PPS). PPS, a heparin-like, low-molecular-weight compound is semisynthetically derived from beechwood hemicellulose. Therapeutic intervention with sodium, as NaPPS **(Synocur)** possesses anti-infammatory, anti-arthritic and chondroprotective properties. These beneficial effects arise from: Direct anti-inflammatory activity, Inhibition of neutrophil migration into joints, Stimulation of hyaluronic acid synthesis by synovial fibroblasts which results in a marked increase in synovial volume and viscosity, Stimulation of chondrocytes and biosynthesis of proteoglycans, and Strong fibrinolytic activity which improves the circulation of subchondral bone and peri-articular structures.

Conclusion

Canine Osteoarthritis is one of the most common degenerative joint diseases, involving progressive changes in the articular cartilage, synovial membrane, ligaments and subchondral bone. It is characterized by pain, low-grade chronic inflammation, and progressive structural and functional deterioration of the affected joint. The complex etiology, multifactorial influences, substantial individual variation in disease burden, and chronic progressive nature of OA make its management particularly challenging. Successful management of canine OA cannot rely on a single therapeutic intervention. Instead, it requires a patient-specific, multimodal, and continuously reassessed approach, integrating appropriate pharmacological and non-pharmacological strategies to control pain, maintain mobility, and preserve long-term quality of life. In recent years, therapeutic development has increasingly focused not only on symptomatic relief but also on modifying the underlying structural progression of OA. In this context, disease-modifying osteoarthritis drugs (DMOADs) have gained considerable interest in Veterinary medicine because of their potential to protect joint tissues, support cartilage repair, and slow further structural deterioration. Thus, incorporation of disease-modifying strategies alongside comprehensive multimodal management represents a promising approach toward improving long-term outcomes in dogs affected by OA.

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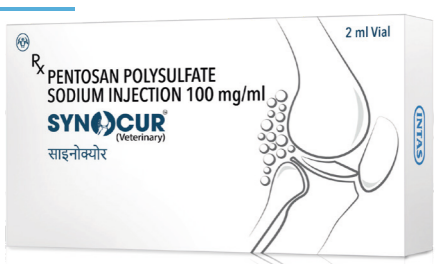

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