Canine Osteoarthritis

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Osteoarthritis (OA), also known as *degenerative joint disease* (DJD), is a debilitating disorder that can affect many animal species.

Overview

In dogs, OA commonly causes joint dysfunction with stiffness, loss of mobility, and varying degrees of inflammation and pain. OA is typically a result of joint instability from ligament laxity, strains, direct or indirect injury, or faulty bone and cartilage development.

Less efficient repair processes in older patients make age a contributing factor, and the condition may be exacerbated by obesity and/or overexertion. OA may affect up to 20% of dogs over 1 year of age,[1] and nearly 50% of musculoskeletal disorders identified in a 10-year span in 16 veterinary hospitals resulted from joint disease.[1]

Clinical Signs

Once joint cells are stressed or damaged, enzymes that fray and ulcerate joint cartilage and compromise the lubricants of the joint fluid are released. This damage causes the joint lining and capsule to become inflamed and bone that is underlying the cartilage to become less resilient.

How I Treat Canine OA

- ❑ Treat and remove underlying joint pathology.
- ❑ Administer slow-acting, disease-modifying OA agents.
- ❑ Consider intraarticular steroid injections.
- ❑ Provide or refer for rehabilitation therapy.
- ❑ Recommend weight loss and exercise.
- ❑ Consider alternative therapies.
- ❑ Institute medical management.

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DJD = degenerative joint disease, OA = osteoarthritis

When the sensitive tissues of both joint capsule and bone are affected (usually after significant articular cartilage damage), signs of pain, lameness, swelling, stiffness, and muscle atrophy are typically apparent. The body then reacts with an ossification process (ie, laying down bone) in the attempt to stabilize the joint, reduce movement, and potentially lessen pain. However, this degenerative process may still result in lifelong pain.

Diagnosis

The initial stages of OA are not readily apparent, but once deterioration has reached the synovial membrane and/or bone beneath joint cartilage, painful inflammation begins. The first visible signs of OA pain may include lameness; apparent pain with range of motion (ROM); sensitivity to palpation of the affected area; decreased activity; stiffness (especially after rest); difficulty rising, lying down, or climbing stairs; or inability or reluctance to jump.

Radiographs can reveal and help confirm preliminary indicators of OA (eg, joint effusion) or more advanced changes (eg, calcification within and around the joint, often appearing as osteophytes).

Arthrocentesis can help rule out other causes of joint effusion or pain. For example, if nondegenerate neutrophils are present on joint fluid cytology and analysis, immune-mediated and/or tick-borne diseases are more likely. In contrast, the presence of significant monocytic inflammation (ie, high mononuclear cells) often confirms DJD or classic OA.

It is important to identify and treat underlying joint instabilities or pathologies, including fragmented coronoid process, osteochondrosis dissecans, ruptured cranial cruciate ligament, meniscal injury, or collateral ligament injury.

Diagnosis Checklist for Canine OA

- ❑ Observe for lameness.
- ❑ Evaluate radiographs.
- ❑ Conduct arthrocentesis with fluid cytology and analysis.
- ❑ Identify underlying joint instabilities or pathology.

How I Treat Canine OA

✓ Treat and remove underlying joint pathology to ensure treatment success.

✓ Administer slow-acting, disease-modifying OA agents.

- Injectable products include polysulfated glycosaminoglycan (PSGAG; Adequan, adequancanine.us) and intraarticular hyaluronic acid (HA; Hylartin V, zoetis.com).
  - As OA advances, weekly to monthly injections of PSGAG can help lessen joint pain.
  - Intraarticular HA injections can help reduce joint pain and inflammation and reestablish normal joint environment.
- Oral products include glucosamine hydrochloride and chondroitin sulfate, methylsulfonylmethane, and long-chain omega-3 fatty acids (eg, docosahexaenoic acid, avocado soybean unsaponifiables, eicosapentaenoic acid [Cosequin, Dasuquin, or Welactin; nutramaxlabs.com]).
  - Start patients on oral glucosamine-chondroitin sulfate and omega-3 fatty acid supplements at the first signs of OA.
  - Athletic patients can be started earlier in life to help lower the incidence of OA from joint overexertion.

✓ Consider intraarticular steroid injections (eg, methylprednisolone, triamcinolone).

- These injections can be used to reduce pain and inflammation in refractory patients.
- Evidence suggests that intraarticular steroids can protect articular cartilage in experimental canine OA; however, repeated use may also have deleterious effects on joint tissue from suppression of cartilage matrix synthesis.[2-4]
  - Benefits typically outweigh risks.
  - Strict aseptic technique is essential to avoid iatrogenic septic arthritis.

✓ Provide or refer for rehabilitation therapy.

- Therapy should begin with modality treatment, such as cold laser and transelectrical neuromuscular stimulation, to help reduce inflammation, effusion, and pain.
- Home exercise programs should include passive ROM to improve and maintain full-joint ROM, as well as muscle-building exercises to improve potential muscle atrophy.
- Once pain and discomfort have been reduced and controlled, hydrotherapy (eg, underwater treadmill, deep

DJD = degenerative joint disease, HA = hyaluronic acid, OA = osteoarthritis, PSGAG = polysulfated glycosaminoglycan, ROM = range of motion

water or swim therapy) can help increase joint ROM and muscle building, and hydrostatic pressure and warm water temperatures may help lessen joint effusion and chronic pain.

☑ Recommend weight loss and exercise, if indicated.

☑ Consider alternative therapies:

- Acupuncture to decrease chronic pain
- Stem cell and/or platelet-rich plasma therapy

☑ Institute medical management.

- NSAIDs are commonly used to treat OA.
  - Many NSAIDs may produce adverse effects (eg, GI, liver, kidney damage) with long-term use.
  - NSAIDs should be used sparingly but can be offered for breakthrough pain or when other therapies are insufficient.
  - Although NSAIDs should not be used concurrently with systemic steroids, they can be used concurrently with intraarticular steroid injections.
- Tramadol (an opioid derivative that acts on serotonergic and α-adrenergic systems) and gabapentin or amantadine (neuropathic, musculoskeletal pain cascade-blocking agents [N-methyl-D-aspartate] receptor antagonists) can be used in conjunction with NSAIDs when refractory pain is suspected. ■ cb

See **Aids & Resources**, back page, for references & suggested reading.