TOPICAL REVIEW

Osteoarthritis in Cats

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Osteoarthritis is a common radiographic finding in older cats, with a prevalence of up to 90% in appendicular joints. Many cats experience impaired mobility from osteoarthritis; however, there are more and more treatment options available, including diet modification, environmental modification, medical therapy, and physical rehabilitation. Continuing challenges involve accurate diagnosis and outcome assessment of treatment, but considerable progress has been made in the last decade.

Keywords: cat, osteoarthritis, degenerative joint disease, appendicular joint

Feline osteoarthritis (OA) is an important and difficult problem to deal with in cats. Interest in feline OA has stemmed both from observations that older cats have difficulties with mobility, and that it is common to observe radiographic evidence of OA in multiple joints of older cats.[1] However, these observations leave us with more questions than answers. How do we know if the OA seen radiographically is painful? What causes it, at what age can it be diagnosed, and is it preventable? How do we best diagnose it, and what can we use to treat? Finally, if we do treat, how do we assess whether that treatment is effective?

OA is a degenerative process that leads to changes in all aspects of a synovial joint. A synovial joint is composed of the articular cartilage, synovial membrane, subchondral bone, synovial fluid, and periarticular soft tissues.[2] Although sometimes the terms OA and degenerative joint disease (DJD) are used interchangeably, DJD can refer to any abnormality of either synovial or cartilaginous joints, such as the intervertebral disks.[3] For purposes of this review, we will focus on appendicular synovial joints.

OA can be initiated in 2 ways: either by normal forces being placed on an abnormal joint (for example, hip joint laxity associated with hip dysplasia), or by abnormal forces acting on a normal joint (traumatic articular fracture). In addition, some authors describe primary versus secondary OA, with primary OA being idiopathic in origin and possibly associated with the "wear and tear" thought to occur with aging.[3] Until this decade, little attention has been paid to OA in cats, and it was thought to be rare. In one of the first major textbooks devoted solely to small animal orthopedics published in 1985, arthritis in cats was only mentioned in association with the breed-associated cartilage defects (Siamese mucopolysaccharidosis VI and Scottish Fold osteochondrodysplasia), and infectious causes.[4] However, a great deal of work has been done in the past 10 years, and feline OA has been increasingly recognized as a serious clinical problem, particularly in older cats, and is actively being researched as to prevalence,[1,5] underlying cause,[6,7] treatment,[8–11] and outcomes assessment.[12–15]

Diagnosis

The breed-related joint diseases can be spectacular both in their clinical presentations and radiographic appearance. The forward-folded ears of the Scottish Fold purebred cat are an outward sign of a generalized defect in cartilage formation. Scottish Fold osteochondrodysplasia is an autosomal incomplete dominant disorder characterized by skeletal deformities such as a short, thick, and inflexible tail, and shortened, splayed feet. The carpi and tarsi are irregular in size and shape and develop progressive, severe OA (Fig 1) that is associated with a variety of clinical signs including lameness, reluctance to jump, and stiff, stilted gait.[16–18]

Mucopolysaccharidosis type VI is an inherited autosomal recessive storage disease that has been identified in Siamese cats as well as domestic shorthair cats. Clinically, affected cats may have a broad and flattened face, corneal opacities, stunted growth, and clinically evident paraparesis or lameness. Radiographically, misshapen vertebral bodies with DJD of the articular facets as well as laxity and OA of the hip joints may be observed.[19] Two disease-causing mutations have been identified in cats, and DNA testing can be used to identify carriers.[20]

Hypervitaminosis A is a metabolic bone disease that was commonly identified in cats, associated with an all-liver diet, in the 1960s. Although classically the disease affects the spine, nonvertebral cases with only affected hip and stifle joints have been reported. A relatively recent case was reported in Spain in an 8-year-old female domestic shorthair on

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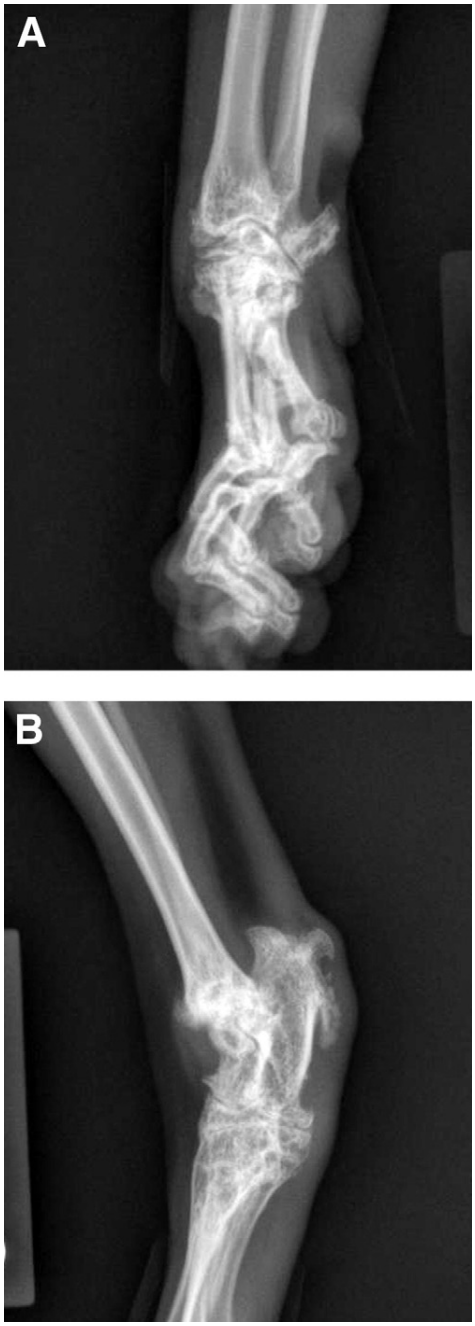

Figure 1. Lateral radiographic projection of the carpus (A) and hock (B) in a 7-year-old female spayed Scottish fold cat. The owner of this cat did not perceive any lameness or mobility problems.

a diet of raw liver and commercial cat food.[21] The cat had bilateral ankylosis of the hip and stifle, was poorly groomed and, radiographically, massive bony proliferations were observed.

Several retrospective studies have identified a high prevalence of appendicular OA in cats. One study evaluating 100 cats over 12 years of age found that 64 had DJD in one or more appendicular joints, with the elbow being the most severely affected (based on radiographic appearance, not numbers of elbows affected).[1] A second study evaluated radiographs of 292 cats over 1 year of age where at least one synovial joint could be visualized.[5] In that study, 22% of cats showed evidence of radiographic OA, with only 11% of affected cats having a potential cause for their OA identified. The population of cats with radiographic evidence of OA was significantly older than the control population, and elbow, hip, and stifle joints were most frequently affected, with

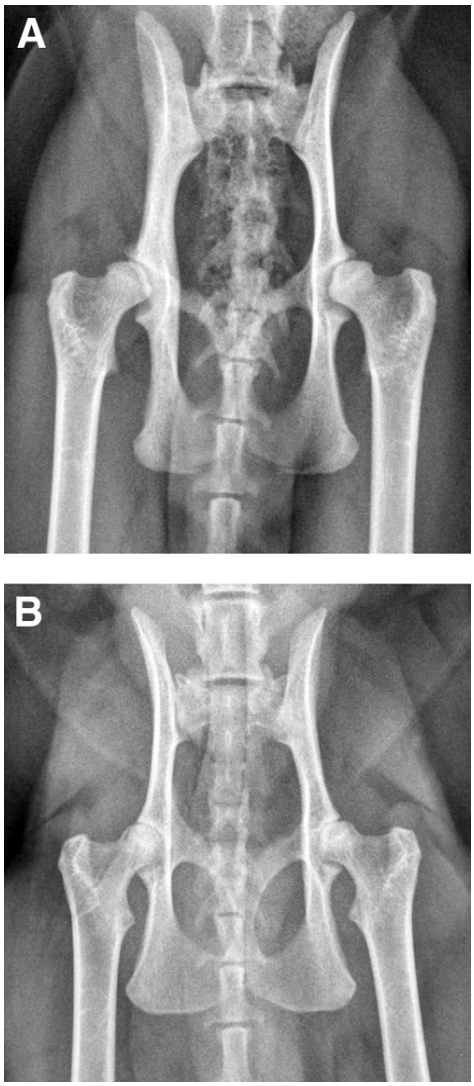

Figure 2. Ventrodorsal projection of the pelvis of 2 different cats. The cat in part A is a 2-year-old female spayed domestic shorthair cat with radiographic evidence of hip dysplasia. This cat exhibited pain on extension of the hips. Note the laxity and incongruity of both hips, as well as the proliferative changes of the craniodorsal acetabulae. This cat also had a littermate with hip dysplasia. The cat in part B is a 6-year-old intact male Maine Coon with normal hip conformation as a comparison.

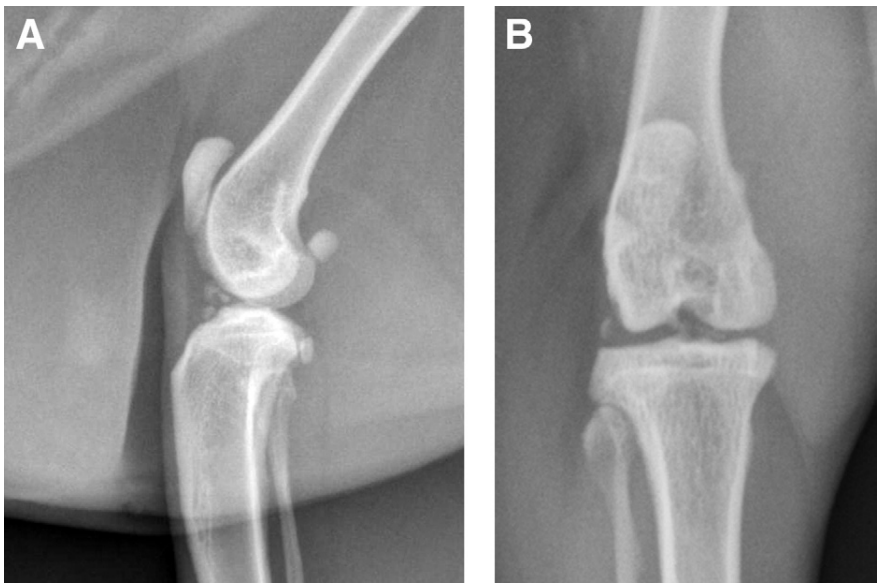

Figure 3. Lateral (A) and ventrodorsal (B) radiographic projections of the stifle joint of an 8-year-old female spayed domestic shorthaired cat. Note the moderate meniscal mineralization of the cranial aspect of the medial meniscus. Some osteophyte formation on the lateral aspect of the joint on the craniocaudal view indicates that this cat may have some DJD.

bilateral symmetry common.[5] A similar study published the same year observed appendicular OA in 36 of 218 cats (16.5%).[22] All of these studies were performed retrospectively, and the radiographs were not obtained with the intent of studying the joints (many were routine chest and abdominal radiographs), so drawing conclusions regarding true prevalence of OA in cats based on these studies is difficult.

However, a newly published study prospectively evaluated 100 cats from a single practice database of 1640 cats equally distributed across four age groups (6 months to 5 years, 6-10 years, 11-15 years, 16-20 years). Cats were randomly selected (regardless of health or lameness status) to participate in a cross-sectional prevalence study of DJD. The cats were examined and sedated, and orthogonal radiographs were obtained of every appendicular joint as well as the axial skeleton. Ninety-one percent of the cats were found to have at least one appendicular joint affected with DJD, with the most frequently affected joints the hip, stifle, tarsus, and elbow. Only age was significantly correlated with DJD. This study confirms the previous impression from other studies that the incidence of DJD in cats is high.[11,23]

In the retrospective studies evaluating appendicular OA cited above, the authors noticed a "mismatch" between identification of clinical signs of lameness or joint pain in the medical record versus the identification of radiographic signs of OA. Possible reasons for this included the perception that cats were able to mask or cope with more severe orthopedic disease than dogs, that it was difficult to confidently identify pain on feline orthopedic examination, and that clinical signs of feline OA included weight loss, anorexia, depression, abnormal elimination habits, aggressive behavior, reduction in ability to jump, and decreased grooming; with overt lameness being relatively rare.[1,22,24] A prospective study evaluating 28 cats with both radiographic and clinical signs of OA was conducted, involving treatment with meloxicam, to attempt to identify common signs of disease. The elbow and hip joint were the most frequently affected, with 71% of the cases having no identified underlying cause of the OA. Treatment resulted in significantly improved ability to jump, height of jump, lameness, stiff gait, and activity level.[22,24]

Etiology

Almost nothing is known about etiology of feline OA. Although there is evidence in humans and dogs that obesity is linked to the development of OA,[25] many osteoarthritic cats are actually underweight, although one study has shown a link between body condition score and lameness, with heavy cats being almost 3 times as likely to be presented to a veterinarian for lameness than cats with optimal body weight.[26] Hip dysplasia has been reported in the cat both based on radiographic surveys (with an incidence of 6.6% in one study)[7,27] and clinical studies[28,29]; little is known about risk factors, genetics, or treatment (Fig 2).

Medial patellar luxation has also been associated with OA in cats, and in some cases, with a more severe radiographic appearance than dogs. The authors also noted that low-grade luxations can be associated with the same severity of lameness as high-grade luxations.[30] A new study has documented the incidence of meniscal mineralization in cats, with 46% of 100 client-owned cats having radiographic evidence of meniscal mineralization (Fig 3). A second portion of the same study evaluated stifles with feline cadavers, examining the relationship between meniscal mineralization (radiographi-

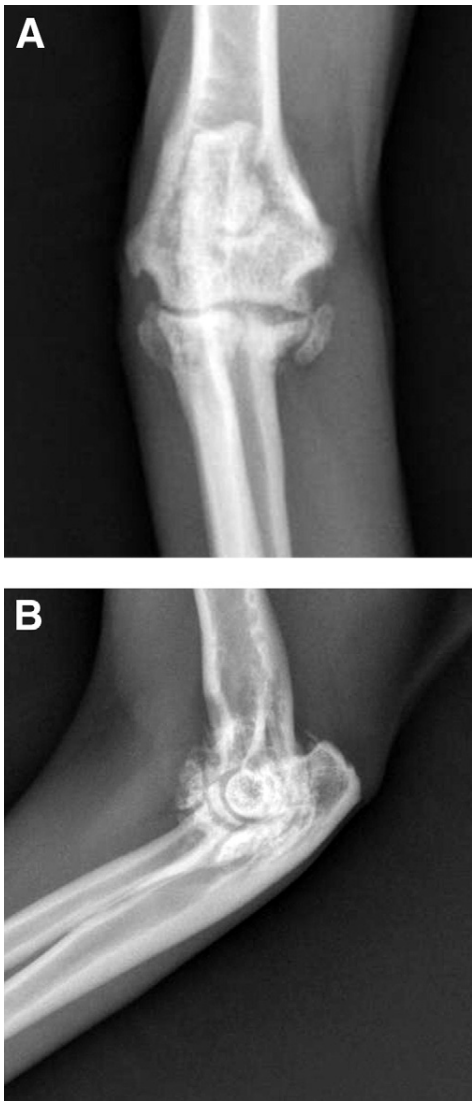

Figure 4. Craniocaudal (A) and lateral (B) radiographic views of the elbow in a 19-year-old female spayed domestic shorthair cat. Although the owners viewed this cat as clinically normal, there was loss of flexion of the elbows on orthopedic examination and this cat had a stiff, stilted gait.

cally and histologically) with radiographic appearance of DJD and cartilage damage. Interestingly, they found that percentage mineralization of the menisci (which were affected in roughly half of the cadaver cats examined) correlated strikingly with cartilage damage observed on the medial femoral and tibial condyles. Also of interest is that cartilage damage could be present in the absence of radiographic signs, suggesting that we may be limited in our ability to detect OA in cats radiographically, particularly early in the disease process.

Although elbow OA is the most common presentation in some studies, very little is known about the etiology (Fig 4). One case report described arthroscopic debridement of a feline elbow[9] and speculated that cats may get fragmented medial coronoid process disease; however, to date no published reports of "elbow dysplasia" exist for the cat. Much has been speculated about the role of nutrition, exercise, early-age neutering, and other environmental factors in the development of feline OA; however, to date there is very little known about risk factors and underlying etiologies as compared with what is known in dogs.[31]

Treatment

Many veterinarians identify OA in older cats, yet are hesitant to treat it, particularly with nonsteroidal anti-inflammatory drugs, for fear of side effects, as many older cats may have comorbidities such as chronic renal failure, hyperthyroidism, or diabetes. However, there are an increasing number of options for the clinician that can provide a better quality of life for these cats. One simple remedy is to consider modifying the environment for the arthritic cat. Making it easier (by moving furniture or providing ramps or steps) for cats to gain access to preferred heights (and in some cases, better access to food and water) or to the litter box can help the mobility-impaired cat to get in and out. Encouraging more activity (increased activity is shown to improve mobility and decrease pain in arthritic humans) by providing environmental enrichment, such as toys, cat towers, playing with laser-pointers, or hiding snacks or catnip is an easy way to improve quality of life.[11]

Many drugs are currently available that can be used successfully as part of the management of feline OA. Two excellent reviews have recently been published, and the International Society of Feline Medicine and the American Association of Feline Practitioners have published a consensus guideline on the long-term use of nonsteroidal anti-inflammatory drugs in cats.[8,11,32] Although meloxicam is not approved in the United States for long-term (unlimited) use to treat feline OA, it has been approved in Europe and there is a great deal of collective experience using this drug in cats. One recommended protocol is 0.1 mg/kg by mouth (PO) on day 1, followed by 0.05 mg/kg PO daily for 4 days, then 0.05 mg/kg every other day thereafter, or 0.025 mg/kg daily. Meloxicam should be dosed accurately with syringes because drop size dispensed from the bottle varies.[11] One study has evaluated the long-term (6-month) efficacy and safety of low-dose meloxicam, given with food, in arthritic cats with good results.[33] Although there is little in the veterinary literature regarding the use of chondroprotective drugs, such as polysulfated glycosaminoglycan, glucosamine/chondroitin sulfate plus or minus combination with avocado/soya extracts, there is anecdotal evidence that these medications can provide additional analgesia and are likely to be safe in the cat. Alternative medications that have been reported in managing feline DJD include amantadine, buprenorphine, gabapentin, and tramadol.[11]

"Joint diets" have been available for dogs for some time, under the premise that the addition of long-chain fatty acids of the n3 series would replace n6 fatty acids in the cell membranes, resulting in a less inflammatory pathway should tissue damage occur. A new randomized, controlled, double-

Table 1. Differential Diagnoses for Osteoarthritis in Cats[39–41]

Septic arthritis
Feline progressive polyarthritis
Femoral neck metaphyseal osteopathy
Calicivirus
Mycoplasma gatteae
Histoplasmosis
Synovial tumors
Osteosarcoma
Multiple myeloma
Feline rheumatoid arthritis

blinded prospective clinical study has examined a diet high in fish oil, green-lipped mussel extract, and glucosamine/chondroitin sulfate in 40 client-owned osteoarthritic cats.[10] The investigators found that osteoarthritic cats receiving the diet, as compared with a control group, were more active as measured objectively with a collar-mounted accelerometer over a 9-week period. Although this is not direct evidence of improvement, it was theorized that increased activity reflected decreased pain associated with OA.[10]

Physical rehabilitation has been described for the treatment of OA in dogs, and rehabilitation and weight loss improve clinical signs in osteoarthritic dogs.[34] Little research in this area has been reported in cats. Anecdotally, we have received reports from clients documenting increased height of jump and overall activity level in overweight, arthritic cats after weight loss. Clinical reports show that cats may be treated with standard methods used in dogs, such as passive and active exercises, massage, water treadmill, laser therapy, and acupuncture.[35,36]

Surgery may have a role in treatment of some cases of OA. Although relatively little is known about long-term outcomes of stabilization in the feline cruciate-deficient stifle, it makes sense that stabilizing an unstable joint will aid patient comfort. Femoral head and neck ostectomy is a well-established treatment for advanced OA of the hip, and total hip replacement of the osteoarthritic hip has recently been successfully reported in 8 cats and may be superior to femoral head and neck ostectomy.[37]

Outcome Assessment

As discussed above, if making a definitive diagnosis of OA can be challenging, how can we then evaluate whether treatment is effective? Diagnosis is based on a combination of careful history, owner observations, lameness, and orthopedic examination and radiographs, and documenting the response to treatment can use these techniques as well, although it is not expected that the radiographic appearance will improve, but may stabilize.

In addition, goniometry and muscle circumference may be helpful in documenting changes in range of motion or muscle mass in response to treatment.[38] Work is being done to develop and publish a validated owner questionnaire that relies on client-specific outcome measures, using items such as walking, running, ability to jump up and down, climbing stairs, playing with toys, and so on.[14] Using novel monitoring devices, such as collar-mounted accelerometers to measure activity levels, is another way that outcomes can be measured in client-owned cats.[10]

In the meantime, simply recognizing that there are a variety of behaviors, other than overt lameness, that may indicate the presence of painful OA can help the clinician obtain the important information from the client.

Summary

OA in cats is very common, causes dysfunction in many cats, and can be successfully treated, ideally incorporating a multimodal approach (Table 1).

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