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JOINT DISEASES IN CATS – WHAT DO WE KNOW?
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INTRODUCTION
Most cats that present with a problem of the musculoskeletal system have sustained trauma. Joint disease is not very commonly diagnosed as a cause for lameness. Although a variety of feline joint diseases has been described in the literature there is limited information regarding clinical significance of these diseases. The following text is a summary of the feline joint diseases that are seen more commonly and which we should have in mind when examining a cat for potential joint problem.

OSTEOARTHRITIS
A surprisingly high prevalence of radiographically evident osteoarthritis (OA) has been described in recent years, with up to 90% of older cats being affected. However, OA is only infrequently diagnosed clinically, maybe because cats commonly show non-specific clinical signs with OA that are not readily recognized as signs of pain. Cats suffering from OA may only show changes in demeanor, such as inactivity, difficulty or reluctance to jump or a stiff gait, and an actual lameness may not be observed. The spine is the most common site for degenerative joint disease in older cats. Of the joints of the appendicular skeleton, the elbow is most often affected, followed by the hip joint. Gonarthrosis is also relatively common and is usually associated with rupture of the cranial cruciate ligament.

Treatment of OA should address inciting causes where identified. Because an inciting cause is rarely apparent in cats, conservative treatment is usually chosen. Conservative management of OA involves environmental changes, weight loss in obese cats, and pain control. Weight loss is an important factor as obese cats are more likely to suffer from clinical signs associated with OA. Meloxicam is currently the only non-steroidal anti-inflammatory drug (NSAID) licensed in Europe for long-term use in cats. It has been shown to be an effective analgesic for feline locomotor disease. Other NSAIDs should be used very cautiously in the feline species. Cats have a reduced hepatic activity for glucuronide conjugation, and toxicity might be encountered with drugs and dosages that are considered safe in other species. NSAIDs are contraindicated in the presence of hypovolemia, dehydration, and kidney disease. Chondroprotective slow-acting disease-modifying agents may also have some benefits in the treatment of feline OA.

POLYARTHRITIS
Polyarthritis can be immune-mediated or septic. It is grossly classified into erosive and non-erosive forms, based on radiographic appearance. Affected cats are often systemically ill and reluctant to walk. Marked joint pain, joint effusion, elevated body temperature, and generalized muscle hypotrophy are common complaints. Cytological examination of the synovial fluid is the single most important method for diagnosing polyarthritis, and potentially differentiating between immune-mediated and infectious causes. Synovial neutrophil counts are elevated in both immune-mediated and septic polyarthritis. Degenerate neutrophils and intracellular bacteria can sometimes be seen in septic polyarthritis, but their absence does not rule out infection. A bacteriological culture needs to be performed when suspecting an infectious cause.

Erosive polyarthritis
Erosive polyarthritis includes septic polyarthritis (very rare in cats), and feline chronic progressive polyarthritis (FCPP). Two forms of FCPP have been described; a periosteal proliferative and a destructive form. The periosteal proliferative form is characterized by progressive formation of periosteal new bone, whereas areas of lysis that progress to marked joint destruction are seen with the destructive form. The tarsi and carpi are commonly affected joints. Cats with FCPP are generally young adult to middle-aged intact or neutered males. Prednisolone therapy can improve clinical signs and slow down the process of joint destruction but disease relapse is common and many patients have to receive continuous therapy. The addition of cytotoxic drugs, such as cyclophosphamide or chlorambucil is indicated with lack of improvement on prednisolone. Side effects in cats are common and potentially fatal with some cytotoxic drugs (eg azathioprine).

Non-erosive polyarthritis
Non-erosive forms of polyarthritis include immune-mediated non-erosive polyarthritis, drug- and vaccine-induced polyarthritis, and polyarthritis in association with systemic lupus erythematosus. Several inciting causes can activate a non-erosive immune-mediated polyarthritis: systemic inflammation or infection, diseases of the gastrointestinal, respiratory or urinary tract, and neoplasia. However, an underlying cause is not found in many cases and the polyarthritis is then classified as idiopathic. Treatment and prognosis of immune-mediated non-erosive polyarthritis depend on the inciting cause.

Feline calicivirus, and the respective vaccine can induce a polyarthritis that is generally seen in young cats that either have calicivirus infection or recently had their first vaccination. It is self-limiting and the prognosis is excellent.

Systemic lupus erythematosus is a rare multisystemic disease that causes polyarthritis as well as anemia, leukopenia, dermatitis, glomerulonephritis, and polymyositis. Prognosis with lupus erythematosus is guarded.

**SYSTEMIC CONGENITAL DISEASES AFFECTING JOINTS**

Systemic congenital diseases of joints are rare in cats. They include storage diseases (mucopolysaccharidosis, alpha-mannosidosis), and osteochondrodysplasia of the Scottish fold cat. Storage diseases lead to a variety of skeletal, joint, brain, eye, and internal organ dysfunction that are evident early in life.

Osteochondrodysplasia of the Scottish Fold results from an inherited defect in cartilage metabolism in this breed that affects formation of joint cartilage as well as bone growth. Clinical symptoms include shortening of bones (eg metatarsi, metacarpi, vertebrae) and an ankylosing polyarthropathy of the carpal and tarsal bones.

**SELECTED DISEASES OF SPECIFIC JOINTS**

**Elbow joint**

Östeoarthritis of the elbow joint is approximately four times more common than OA of other joints of the appendicular skeleton. Possible underlying disease processes have not yet been identified. A variety of other diseases such as synovial cysts, congenital elbow luxation, and elbow epicondylitis are also encountered.

Synovial cysts occur infrequently and seem to be associated with elbow OA. The cysts can be palpated as large fluid filled structures adjacent to the elbow joint. Retrieval of synovial fluid out of the cyst supports the diagnosis. Treatment involves periodical needle drainage of the cyst and conservative treatment of concurrent OA.

Avulsion and calcification of the antebrachial flexor tendons at their insertion on the medial epicondyle of the humerus (elbow epicondylitis) is a condition that has only recently been diagnosed in cats. It is a periarticular disease and should not be mistaken for OA although radiological signs may appear similar at first sight. Radiographs of both elbows should be obtained, as the condition can be bilateral. Treatment consists of surgical removal of calcified tendon and muscle tissue if the cat is lame. The debrided flexor tendons are then reattached to the surrounding tendons and muscle fascia with sutures.

**Hip joint**

Common diseases of the hip joint include hip dysplasia (HD) and/or coxarthrosis. Coxarthrosis may be secondary to HD or to a previous trauma. The reported incidence of feline HD ranges from 7-32%, with purebred cats having a higher incidence of HD compared to domestic shorthair cats. Maine Coon, Himalayan, Siamese, Abyssinian, Devon Rex, and Persian cats seem to be overrepresented. Cats with HD can have concurrent patella luxation. Radiographic features of HD are somewhat different in the cat than in the dog; the most obvious radiological findings of feline HD are a shallow acetabulum and subluxation of the femoral head. Degenerative changes seem to develop later and to a lesser extent than in dogs and tend to appear first at the cranial margin of the acetabulum. The normal acetabulum of cats is shallower than in dogs, but less than 50% coverage of the femoral head should be considered abnormal. Cats with obvious changes should not be used for breeding even though many cats with HD and coxarthrosis do not display obvious clinical signs.

Two conditions involving the femoral head and neck have been described in cats: slipped capital femoral physis, and femoral neck metaphyseal osteopathy. Both conditions seem to occur predominantly in male castrated, overweight cats at an age of 1-2 years. Both diseases causes marked clinical symptoms and hip pain. Radiographic changes include spontaneous fractures of the femoral capital physis, pathological fractures of the femoral neck, and bone remodeling and necrosis of the femoral neck. It is suspected that delayed phsyseal closure due to early castration is involved in the pathogenesis. Considering the similarities between the two conditions, it seems possible that they reflect the same disease at a different stage of chronicity. Treatment involves femoral head and neck excision or potentially, total hip replacement.
Stifle joint

Common diseases of the stifle joint include congenital patella luxation, cranial cruciate ligament rupture, and mineralization in the cranial aspect of the stifle joint.

Congenital patellar luxation has been reported to occur more commonly in certain breeds, such as Abyssinian and Devon Rex, but other cats are affected as well. The patella usually luxates medially and both stifle joints are often affected. Cats have a larger patella and a higher physiological laxity of the patella than dogs and the patella can be manually moved onto the trochlear ridge of the femoral condyles in many normal cats. Laxity of the patella should therefore always be interpreted in conjunction with clinical signs. Clinical complaints include a crouched gait, inactivity, and inability to jump. Some cats show intermittent lameness or sudden onset distress with vocalization and unwillingness to use the affected leg. Several surgical procedures, used alone or in combination, clinically improve stifle function in most cats. The techniques include tibial crest transposition, sulcoplasty, and soft tissue corrections. Soft tissues correction alone is rarely successful.

Although cranial cruciate ligament rupture is commonly caused by trauma in cats, there is evidence that, similar to the condition in dogs, ligament degeneration may precede rupture of the cranial cruciate ligament in some cases. Such cats are typically older and overweight, present with preexisting OA at the time of clinical presentation, and may have bilateral involvement. Although conservative treatment of cranial cruciate ligament disease in cats has been described to result in good limb function, it seems reasonable to assume that surgical stabilization will facilitate recovery and reduce instability-induced chronic pain.

Mineralisations in the cranial compartment of the stifle joint are common. Small mineralisations in the absence of stifle OA are likely to be of no clinical significance. Large mineralisations are commonly associated with OA and/or cranial cruciate ligament rupture and should be considered abnormal.

REFERENCES