Student Paper Communication étudiante

Chronic progressive polyarthritis in a domestic shorthair cat

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Abstract — A 6-year-old, neutered male, domestic shorthair cat was presented with shifting leg lameness and palpable effusion of the carpal and tarsal joints. Blood work, arthrocentesis, and radiographs identified an immune-mediated erosive polyarthritis. The cat was positive for feline syncytia-forming virus, and with his signalment, was diagnosed with feline chronic progressive polyarthritis.

Résumé — Polyarthrite progressive chronique chez un chat commun. Un chat commun mâle stérilisé âgé de 6 ans a été présenté avec une boiterie changeante et une effusion palpable des articulations carpienne et du tarse. Une analyse sanguine, une arthrocentèse et des radiographies ont identifié une polyarthrite érosive à médiation immunitaire. Le chat s'est avéré positif pour le virus félin induisant le syncytium et, avec ce signalement, a été diagnostiqué avec la polyarthrite féline progressive chronique.

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A 6-year-old, neutered male, domestic shorthair cat was presented to the Atlantic Veterinary Collage (AVC) for a shifting leg lameness of 3-years duration. Although the referring veterinarian was unable to identify its exact location, the pain appeared worse in the hind end when the patient's gait was evaluated. In the 3 y prior to presentation, the patient had been prescribed meloxicam, gabapentin, buprenorphine, and prednisolone, but none of these treatments had improved his condition. In fact, the patient's clinical signs had worsened leading up to the appointment at the AVC, at which time he could walk only about 5 continuous steps before sitting and resting. The patient did not appear to feel pain when he was handled by his owners.

For the 5 wk immediately prior to presentation at the AVC, the patient had been prescribed meloxicam, 0.1 mg/kg body weight (BW), PO, q24h, and gabapentin, 3.5 mg/kg BW, PO, q24h. The patient was bright, alert, and responsive; he weighed 6.6 kg and his body condition score was 6/9. All physical examination parameters were within normal limits. Gait abnormalities were clearly visible and the patient seemed reluctant to walk. When walking, the patient had a hunched posture, and would sit after only a few steps. A musculoskeletal examination was performed, and revealed pain on palpation of both the carpal and tarsal joints bilaterally. Bilateral carpal and tarsal joint effusion was also noted. No pain was elicited on palpation of the spine. A neurological examination revealed no abnormal findings.

The reluctance of the patient to walk, the noticeable discomfort in response to palpation of the patient's carpal and tarsal joints, as well as the apparent effusion within these joints, localized the suspected cause of pain and gait abnormalities to the joints. The differential diagnoses included immune-mediated polyarthritis, infectious polyarthritis, degenerative joint disease, polymyositis, osteomyelitis, and synovial cell sarcoma. The diagnostic plan included a complete blood (cell) count (CBC) and serum biochemistry tests to evaluate for systemic inflammation, organ dysfunction, and to assess the patient's anesthetic risk.

The CBC and serum biochemistry revealed no significant abnormalities. The patient was sedated with dexmedetomidine (Dexdomitor; Zoetis, Kirkland, Ontario), 0.05 mg/kg BW, IV, and butorphanol (Torbugesic; Zoetis), 0.2 mg/kg BW, IV, and radiographs were taken of both the front limbs and hindlimbs to assess for bony changes within the joints. Abnormalities were noted in the carpal and tarsal joints, bilaterally. The right and left carpi, and the right tarsus showed severe subchondral bony degeneration and severe osteophyte production, as well as a marked amount of soft tissue swelling surrounding these joints. There appeared to be subluxation of some of the small joints of the extremities, and many of these joint spaces were narrowed. The left tarsus showed mild subchondral bony degeneration and mild osteophyte production.

Arthrocentesis of the joints and a joint culture were suggested to assess for inflammation or infection within the joint. The patient was placed in sternal recumbency and the skin over his left carpus, left stifle, and left tarsus was clipped and disinfected. Synovial fluid was aspirated from the left carpus and the left stifle, and was submitted to the diagnostic laboratory

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for cytology. A swab of the joint fluid from the left carpus was submitted for aerobic bacterial culture. Cytological examination of both samples showed marked increases in nucleated cells, predominantly non-degenerate neutrophils, and a low number of red blood cells. One slide contained very small foci of extracellular coccus-shaped bacteria; however, this slide also contained a small number of keratinocytes, so the bacteria were possibly a contaminant. These findings were consistent with marked non-degenerate neutrophilic inflammation of the joints. The bacterial culture was negative for microbial growth.

The patient's blood was taken to assess for the presence of 3 viruses: feline leukemia virus (FeLV), feline immunodeficiency virus (FIV), and feline syncytia-forming virus (FeSFV). The patient was positive for FIV on enzyme-linked immunosorbent assay (ELISA) (Idexx Canada, Markham, Ontario), and negative for FeLV on ELISA (Idexx Canada). An antibody titer to assess the presence of antibodies to FeSFV was positive (titer = 192 units of antibody) (Cornell University, Ithaca, New York, USA). These results indicated that the patient had been infected or was currently infected with both FIV and FeSFV.

The results of the radiographs and joint fluid cytology were consistent with an erosive polyarthritis, which was thought to be immune-mediated because of an unrecognized underlying cause, such as infection or neoplasia. Treatment of any immune-mediated disease, including polyarthritis, involves suppression of the immune system to decrease the amount of inflammatory mediator; this is frequently done with a glucocorticoid. The patient was therefore prescribed prednisolone (Rafter 8, Calgary, Alberta), 2 mg/kg BW, PO, q24h. The plan was to bring the disease under control while using the lowest dose of glucorticoid necessary in order to minimize the side effects on the patient (1). Therefore, cyclosporine (Atopica; Novartis, Mississauga, Ontario), 5 mg/kg BW, PO, q24h, was also prescribed, to lessen the amount of steroid needed. Since the patient had been receiving meloxicam, a non-steroidal anti-inflammatory drug, a 5-day washout period was required to decrease the risk of gastrointestinal upset. The patient was placed on buprenorphine (Vetergesic; Champion Alstoe, Whitby, Ontario), 0.01 mg/kg BW, transmucosally, q8h, for pain control during this time.

The patient has been doing well since being discharged from AVC. Six months after his diagnosis, the patient's owners reported that the cat's level of comfort had improved, and he was now able to walk continuously without needing to sit and rest. Since returning to his referring veterinarian, the patient's medications have changed slightly according to his perceived level of pain. The cat is now receiving prednisolone (1.5 mg/kg BW, PO, q12h), cyclosporine (7 mg/kg BW, PO, q24h), and buprenorphine (0.01 mg/kg BW, PO, q8h). He has also been prescribed gabapentin (compounded by Murphy's, Charlottetown, Prince Edward Island), 5 mg/kg BW, PO, q24h for additional pain management. No attempt has been made thus far to decrease the amount of steroid the patient is receiving, but if he continues to do well on this protocol, an attempt may be made in the future.

Discussion

Polyarthritis is inflammation of 2 or more joints. The diagnostic test of choice to confirm polyarthritis is arthrocentesis and analysis of the aspirated synovial fluid (2). With any type of polyarthritis, neutrophils are the most prevalent cell type within the synovial fluid, with total counts > 3000 cells/mL (2). There are 3 main subtypes of polyarthritis: non-infectious, non-erosive immune-mediated polyarthritis (IMPA); non-infectious, erosive IMPA; and infectious, erosive, and non-erosive polyarthritis. Different forms of disease have also been established within each subtype (2). The discussion of this report focusses on the autoimmune diseases, as the patient herein had no evidence of an underlying neoplastic or infectious cause to his polyarthritis, and therefore his disease was diagnosed as non-infectious and immune-mediated.

Non-infectious, non-erosive IMPA is the most common form of polyarthritis, and most of these cases are idiopathic (2). Possible pathologic mechanisms suggested include immune complex formation in response to a microbial infection, with subsequent deposition in the joints; or genetic predisposition in which antibodies to certain antigens cross-react with joint antigens (2). Radiographs are needed to distinguish the non-erosive from erosive form of IMPA. Non-erosive polyarthritis shows no bony radiographic abnormalities; however, signs of joint effusion and soft-tissue swelling are often present (2).

Non-infectious, erosive IMPA includes 2 forms of disease. The first is rheumatoid arthritis, which is rare in cats and is associated with autoantibodies to immunoglobulin G, or rheumatoid factor (2). The second form of this disease in cats is feline chronic progressive polyarthritis (FCPP), the condition that affected the cat in this report. Except for 1 report of this disease in a female cat in 2009 (3), FCPP primarily affects young adult, male cats. It is characterized by periosteal new bone formation and bony erosions within the joints, mainly the carpal and tarsal joints (2). On radiographs, erosive polyarthritis shows subchondral bone destruction, which is seen as an irregular joint surface or erosion of bone at the joint space. If severe, loss of mineralization of the epiphysis and calcification of soft tissues of the joint may be evident (2).

Feline chronic progressive polyarthritis was first reported as a specific disease entity in 1975 (4). In 1980, a case series was published wherein 20 cats with FCPP were evaluated at the University of California, Davis, and 2 types of FCPP were described: the periosteal proliferative and erosive forms (5). The periosteal proliferative form was suggested to be more common, being diagnosed in 17 of the 20 cats in the study. The radiographic signs include periarticular soft tissue swelling, periosteal new bone formation, perichondral enthesophyte production, perichondral and subchondral erosion, subchondral cysts, osteopenia of bone adjacent to affected joints, and narrowed joint spaces (6). With this form, periarticular erosions and collapse of joint spaces occurred with chronicity, but joint instability and deformities were not seen (5). Histopathologically, the periosteal proliferative form shows changes in the synovial membrane such as hyperemia and edema, and cellular infiltrate characterized by neutrophils, lymphocytes, and plasma cells, and can progress to pannus formation over time (5).

The erosive form of FCPP, seen in only 3 of the 20 cats in the UC-Davis study, is characterized radiographically by severe subchondral bone erosion, perichondral bone erosion, and

subchondral cyst formation (6). Perichondral enthesophyte formation, bone destruction at points of ligamentous insertion to bone, and subluxation of small joints of the extremities also occur (6). The patient in this case had the erosive form of the disease, as he had severe subchondral bony degeneration and severe osteophyte production, as well as subluxation of the bones of the small joints. Histopathology of the synovial membrane in cats with erosive FCPP shows pronounced villus hypertrophy and the synovial infiltrate consists of dense accumulations of lymphocytes and plasma cells (5). Samples were not submitted for histopathology of the synovial membrane for the patient herein.

The etiology of FCPP is unknown, but appears to be related to viral infection with either one or both FeLV and FeSFV. However, the 1980 UC-Davis study was unable to reproduce FCPP in a healthy cat inoculated with either or both of these viruses. Therefore, the relationship between FCPP and the 2 viruses is considered purely statistical (5). Of the 20 cats in the UC-Davis study, all were found to have FeSFV, with 60% having co-infections with FeLV. There have been additional reports of FCPP in cats co-infected with FIV and FeSFV (7). The co-infection has been shown to cause an enhancement of the effects seen in either of the 2 forms of the disease (7). However, most cats with FeSFV do not show signs of polyarthritis, so it is thought to be an uncommon manifestation of this virus infection (5). The patient in this case was positive for FeSFV and FIV, but negative for FeLV.

Although a primary cause is unknown, the disease appears to be facilitated by immune-mediated means. The neutrophilic inflammation within multiple joints suggests that there is a local antigenic stimulus within the joint itself (5). Glucocorticoids have been efficacious in cases of polyarthritis, as they help to reduce joint swelling, pain, and fever (1). However, glucocorticoids have not prevented progression of the disease, and bone erosion will continue. Some research has shown that cytotoxic drugs in combination with glucocorticoids may be able to put this disease into remission, although it will not decrease the amount of bone erosion already present (5). In this case, the patient was prescribed the glucocorticoid prednisolone, and an additional immunosuppressant, cyclosporine, which dampens cell-mediated immune responses (8). CVJ

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