Leishmanial polyarthritis in a dog

Sandra E. McConkey, Alfonso López, Darcy Shaw, Jill Calder

Abstract — A dog adopted as a stray in Spain and then brought to Canada 4 years prior to presentation was evaluated for polyarthritis. An electrophoresis showed a marked polyclonal gammopathy and synovial smears contained leishmanial organisms within macrophages.

Résumé — Polyarthrite à leischmanies chez un chien. Un chien égaré a été adopté en Espagne et amené au Canada 4 ans avant d'être examiné suite à une polyarthrite. Une électrophorèse a montré une gammapathie polyclonale et des frottis synoviaux contenant des leischmanies à l'intérieur des macrophages.

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A 5-year-old, castrated male, mixed breed dog was presented to the referring veterinarian for a lameness of 1-year duration, which had progressed to a non-ambulatory state. The dog had been splinted 1 y earlier by the veterinarian for a suspected left carpal bone fracture. The lameness continued after splint removal and appeared to have spread to several joints. Prednisone therapy was initiated 6 mo prior to presentation. The dog showed a good response initially, but a lesser one over time. According to the owner, the animal was adopted as a stray in Spain 4 y previously. Physical examination indicated swelling of several joints.

Whole blood and serum samples were submitted for analysis. The complete blood cell count revealed a mild leukocytosis consisting of a mature neutrophilia and mild left shift. A moderate, normocytic, normochromic, regenerative anemia was present. These changes were interpreted as being attributable to inflammation and hemorrhage or hemolysis.

Significant changes in the biochemical profile included a marked elevation in alkaline phosphatase (ALP) (523 U/L; normal, 23 to 87 U/L), alanine aminotransferase (ALT) (561 U/L; normal, 5 to 69 U/L), sorbitol dehydrogenase (SDH) (74 U/L; normal, 2 to 20 U/L), and a moderate elevation in aspartate aminotransferase (AST) (238 U/L; normal 20 to 50 U/L) levels. These could all be attributed to a steroid hepatopathy following 6 mo of glucocorticoid therapy. Serum electrophoresis showed that the marked hyperglobulinemia (82 g/L; normal, 22 to 44 g/L) was due to a polyclonal elevation of gammaglobulins. This was presumed to be associated with an antigenic stimulation. The significant but lesser elevation of the α_2 and β globulins indicated increased acute phase inflammatory proteins.

Given the dog's previous travel history and the hypergammaglobulinemia, infection by the genera *Ehrlichia* and *Leishmania* were included as possible etiologies for the polyarthropathy, as well as immune-mediated disease. The dog was referred to the Atlantic Veterinary College Teaching Hospital for further diagnostic tests.

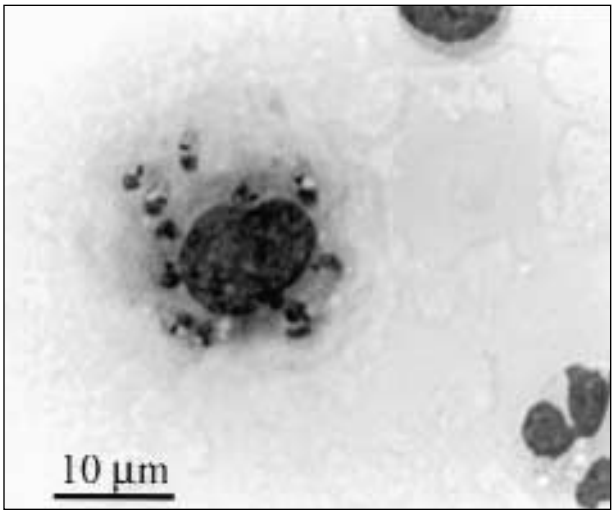

Figure 1. Photomicrograph of synovial fluid showing macrophage containing numerous leishmania; Wright's Giemsa stain; bar = 10 µm.

At the time of referral, the dog was normal on physical examination, except for showing pain on palpation of several joints. There was no lymphadenopathy, splenomegaly, or cutaneous lesion. Detailed orthopedic examination revealed a luxation of the left carpus, increased joint laxity, and pain involving the right carpus and both stifles, elbows, and hocks. Radiographs of the carpi and tarsi showed erosive polyarthritis with subluxation and collapse of the articular spaces. A complete blood profile confirmed the moderate, normocytic, normochromic, regenerative anemia and a mild leukocytosis characterized by a mild neutrophilia and mild regenerative left shift. A urinalysis indicated a urine specific gravity of 1.017 and trace protein. The serum was negative for rheumatoid factor.

Smears of synovial fluid showed a moderately increased cellularity. Cells consisted of 23% nondegenerative neutrophils, 16% mononuclear cells, and 61% small lymphocytes. Many macrophages and a few

Department of Pathology and Microbiology (McConkey, Lopez), Department of Companion Animals (Shaw), Atlantic Veterinary College, University of Prince Edward Island, 550 University Avenue, Charlottetown, Prince Edward Island C1A 4P3; Jordan Branch Road Veterinary Clinic, P.O. Box 1181, Shelburne, Nova Scotia B0T 1W0 (Calder).

Address all correspondence and reprint requests to Dr. Sandra McConkey.

neutrophils contained numerous magenta-staining organisms, 2 to 3 μm in length with a distinct kinetoplast (Figure 1). The morphological and staining characteristics of these organisms were compatible with *Leishmania* spp. amastigotes.

The owners elected to have the dog euthanized and a complete postmortem examination was done. At necropsy, the dog was found to be in good body condition with abundant subcutaneous fat and pale skeletal musculature. The size of the lymph nodes appeared normal.

Microscopic examination revealed that the bronchoalveolar and perivascular spaces in the lung were filled with eosinophilic edematous fluid. The spleen, liver, lymph nodes, and bone marrow contained numerous macrophages filled with small, Giemsa stain-positive bodies consistent with amastigotes of *Leishmania* spp. In sections of kidney, Bowman's capsule appeared slightly thickened and the renal interstitium contained focal aggregates of lymphoplasmacytic cells. Most of these renal infiltrates also contain mononuclear cells with amastigotes in the cytoplasm. Sections of skeletal musculature and peripheral nerves showed many well-defined foci of mononuclear infiltrates containing parasites, mainly around blood vessels. The most spectacular microscopic change was seen in the synovial membranes, which appeared thickened with notable villus hyperplasia. Large numbers of plasma cells, lymphocytes, macrophages, and fibroblasts infiltrated the synovium and many of these cells contained parasites. The articular cartilage adjacent to the inflamed synovium showed early stages of fibrillation. The microscopic changes in this dog confirmed the diagnosis of visceral leishmaniasis with marked involvement of joints.

Leishmania spp. are diphasic protozoa found in both the New World, mainly Central and South America, and in the Old World in parts of Africa, India, and the Mediterranean. In the United States, *Leishmania* spp. are endemic in Ohio, Oklahoma, and Texas (1). The most common species that can affect dogs are *L. infantum* and *L. tropica* (both are subspecies of *L. donavani* (1,2)), *L. mexicana,* and *L. brazilensia* (1).

The vectors for *Leishmania* spp. are sandflies of the genera *Phlebotomus* in the Old World and *Lutzomyia* (found in the USA) in the New World. *Leishmania* spp. circulate between its vertebrate host, as an amastigote, and the sandfly, as a promastigote (flagellate). In vertebrates, the amastigotes, with their telltale kinetoplasts are found predominantly in macrophages, where they multiply by binary fission until the cells rupture, allowing them to escape and spread to new cells (3).

Not all animals exposed to *Leishmania* spp. develop leishmaniasis. It has been reported that 20% of cases resolve spontaneously (3). Development of clinical disease depends on whether a host mounts a predominant thymus helper (Th) 1 or Th 2 helper T-cell response. The Th 1 lymphocytes are activated by interleukin (IL)-12 and secrete the cytokines IL-2, interferon-γ, and tumor necrosis factor-β. These, in turn, primarily stimulate the cell-mediated immune response with the activation of macrophages (4). This activation enhances the macrophages' ability to phagocytize and destroy the *Leishmania* organisms.

Clinical signs for leishmaniasis generally develop between 3 mo and 7 y postinfection (2,3). There are 3 major clinical presentations of leishmaniasis: cutaneous, mucocutaneous, and visceral. Clinical features vary with the phase of the disease, state of the animal's immunity, and previous therapy (5). Most cases show dullness, fatigue, pyrexia, weight loss, anorexia, and exercise intolerance, which then culminates in a wasting disease (1,3,5). Lymphadenopathy and splenomegaly are common (3). Articular involvement is also relatively common, and 37.5% of cases may show a reluctance to walk and an abnormal gait. These animals usually have arthralgia with osteolytis or peripheral proliferative periosteal reactions (2,6). Internally, the main histological changes are infiltration of the spleen and lymph nodes by macrophages containing amastigotes (8).

Most dogs with leishmaniasis have a hyperproteinemia due to a hyperglobulinemia. Immunoelectrophoresis shows increases in immunoglobulin G. The antibodies are produced in response to both organism and self (damaged tissue). The gammopathy is usually polyclonal but can appear more monoclonal (5,8), resembling a plasma cell myeloma or ehrlichiosis. There is a decrease in albumin (as it is a negative acute phase protein) in 94% of dogs. Proteinuria is seen in 85% of infected animals due to the glomerulonephritis resulting from the deposition of immune complexes in glomeruli. Other changes in the blood include nonregenerative anemia (60% of dogs), thrombocytopenia, (20%), and leukocytosis (24%) (5).

Definitive diagnosis is usually by demonstration of the protozoon in macrophages in lymph nodes, spleen, liver, or, as in this case, in the synovial fluid. Amastigotes are typically seen in macrophages, but they have also been reported in neutrophils, eosinophils, endothelial cells, and even fibroblasts (9). A serological test can be done if the organisms cannot be found, but the presence of antibodies merely shows exposure to the protozoon and not clinical disease (3). At postmortem, most dogs show cachexia, generalized lymphadenopathy, hepatosplenomegaly, and various skin lesions.

The clinical presentation of this dog was atypical for leishmaniasis, since there was no evidence of cachexia, anorexia, or cutaneous lesions accompanying the polyarthritis (6). *Leishmania* induced polyarthritis can occur due to an inflammatory reaction to either the organisms or the immune complexes in the synovium.

To our knowledge, this was the first case of canine leishmaniasis to be reported in the Maritimes. It constitutes a good example of the international nature of many diseases because of the increased travel of people and their pets throughout the world.

Acknowledgment

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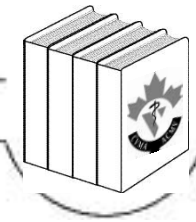
BOOK REVIEW



COMPTE RENDU DE LIVRE

Eisner C. ***Diagnostic Testing and Imaging — Creating a Profit Centre: Blueprints for Your Bottom Line.*** American Animal Hospital Association, Lakewood Colorado, 2001, 136 pp, ISBN-58326-014-5, US$59.00.

This book is the 2nd of the *Blueprints* series by the same author. The author, a veterinary practice manager, is a CVT with an MBA degree who has worked in the veterinary industry for over 20 years. This 137-page paperback consists of 115 pages of text, tables and photographs written within 9 chapters, followed by 3 appendices and an index. The book took a relatively short time to read, as approximately half of the 9 chapters was comprised of tables, forms, and black-and-white photographs. Some extra time was required, however, to digest the tables, which were not laid out as simply as they might have been.

The theme of the book is that with sufficient management, one can create a very profitable service or group of services within a veterinary hospital — a "profit center." The author describes the steps that are necessary to ensure that the diagnostic laboratory and imaging areas of a veterinary hospital are run in a business-like fashion.

In chapters 1 and 2, compelling reasons are given for offering more diagnostic services within a hospital and explain the profit center concept is explained. In chapter 3, a business plan for implementation of these services is outlined, while in chapter 4, the training required to obtain the participation of the entire veterinary team in the implementation of the business plan is explained. Chapters 5 and 6 are primers in designing a diagnostics area and a radiology department.

In chapter 7, the need for excellent records once the decision to create a profit center has been made is detailed and the need to charge appropriately for these services is stressed. Chapter 8 provides a marketing plan for the profit center and chapter 9 is a summary. The appendices are primarily lists of references and resources.

I liked the book, though I suspect many accomplished practice managers and owners would not glean a great deal of new information from it. Much of the book contains what would be common knowledge or common sense to this group. I did, however, take away a few "golden nuggets," as the author calls them, from this book. Many hospital owners and managers look at the presence or absence of profitability in their businesses from the perspective of the entire hospital. The profit center approach allows one to ascertain whether a particular service is indeed profitable as a stand-alone service. Once these principles are applied, some may be surprised to learn that although their business is profitable they are actually losing money in the provision of a certain service, unbeknownst to them.

The book did an excellent job of underscoring the importance of pricing services appropriately. Modest increases in fees can have a dramatic impact on the bottom line without offending clients. The book provides a nice reference list of manufacturers and suppliers of diagnostic equipment.

I found the many tables somewhat confusing. The corresponding columns of words and numbers often required a little extra thought and some arithmetic that could have been provided by the author.

This book would be a great resource for a novice practice manager or owner. It would also be mutually beneficial to associate veterinarians and their employer. The book would give an associate a good appreciation of the high cost of running a veterinary hospital. Furthermore, associates who promote and perform more diagnostic testing, when appropriate, as a result of reading this book would help their patients, themselves, and their employers as they hone their diagnostic skills.

Finally, there may be veterinarians who do not offer diagnostic testing as often as perhaps they should. This may be out of a sense of pride in their physical examination skills or due to a belief that their clients will not pay for the services as they are offered. If this book were the key to rekindling the zeal for veterinary practice by stimulating a practitioner to develop as a diagnostician and perform more diagnostic testing, then it would be priceless.

Reviewed by ***Stephen Noonan, DVM,*** *Woodlawn Animal Clinic, 650 Portland Street, Dartmouth, Nova Scotia.*