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CASE REPORT

Listerial mesenteric lymphadenitis in 3 cats

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Abstract

Background: Listeriosis is a rare disease in cats with naturally occurring cases usually being identified in individual animals. Listerial mesenteric lymphadenitis has not been described previously in cats.

Objectives: To describe the clinical and histological features of listerial mesenteric lymphadenitis in cats as well as treatment outcome.

Animals: Listerial mesenteric lymphadenitis was confirmed in 3 cats by histology, immunohistochemistry, and bacterial culture.

Results: The affected cats were young to middle aged and were examined for various clinical signs. On both palpation and abdominal ultrasound examination, all cats had marked mesenteric lymphadenomegaly. Survival was prolonged in all 3 cases. Two of the 3 cats were fed a raw meat-based diet before they developed clinical illness.

Conclusions and Clinical Importance: Lymphadenitis caused by listeriosis has a protracted time course and should be a differential diagnosis for abdominal lymphadenopathy in young to middle-aged cats. Feeding of a raw meat-based diet may be a contributing factor for development of listeriosis in cats.

KEYWORDS

interleukin 10, listeria, lymphadenopathy, raw-fed

1 | INTRODUCTION

Listeria monocytogenes (Lm) is a facultative anaerobe, a Gram-positive bacillus capable of growing at a wide variety of temperatures, including under refrigeration (4-10°C).[1] A ubiquitous saprophyte, Lm can be found in soil, standing water, and production animal feeds (silage), as well as the meat products ultimately generated from these animals.[1] The medical literature describes 4 separate evolutionary lineages of Lm, with distinctive phenotypes that have variable pathogenicity.[2] The pathogenicity of Lm is a result of its ability to persist as a facultative intracellular organism within macrophages, enabling it to elude the humoral immune system of the host, and beta-hemolytic strains also produce the hemolytic toxin listeriolysin.[1,3]

In the veterinary literature, listeriosis most commonly has been described in ruminants, and manifests itself as 3 classical syndromes: meningioencephalitis, abortion and still birth, and septicemia.[3] In addition, there have been isolated reports of Lm infection of cattle and sheep resulting in enteritis and lymphadenitis of the mesenteric lymph nodes associated with the ingestion of Lm contaminated feed.[4-6] Listeriosis is a rare disease in cats.[7-11] Domestic cats traditionally were thought to be inherently resistant to infections with Lm, a hypothesis supported by previous experimental studies.[12,13] One of these studies identified feline immunodeficiency virus (FIV) infection as a risk factor for development of systemic listeriosis in cats.[12] A similar situation is encountered in humans, whereby healthy individuals rarely succumb to clinical disease when challenged with Lm, but immunocompromised

Abbreviations: 16S rRNA, 16S ribosomal ribonucleic acid; FeLV, feline leukemia virus; FIV, feline immunodeficiency virus; IL-10, interleukin 10; Lm, *Listeria monocytogenes*; MALDI-TOF MS, matrix-assisted laser desorption ionization time-of-flight mass spectrometry; MSU VDL, Michigan State University Veterinary Diagnostic Laboratory.

individuals, particularly those infected with human immunodeficiency virus, are at an increased risk of developing clinical disease.[2]

One study failed to isolate Lm from fecal specimens of healthy cats,[14] but another study isolated Lm from the feces of a single cat, generating an estimated prevalence of 0.4% fecal shedding in this species.[15] Natural infection of cats with Lm is usually secondary to ingestion of contaminated foods, but exposure does not always result in disease.[1] Once Lm penetrates the small intestine, it results in bacteremia, after which the organisms localize in the mononuclear phagocytic tissues, but also may embolize to other tissues, including the central nervous system, to which listeria has a particular predilection.[1,16]

Our goal was to describe the clinical presentation, pathology, and outcome of 3 cats with confirmed Lm infection, confined to the mesenteric lymph nodes.

2 | MATERIALS AND METHODS

Case 1 was examined by the Paddington Cat Clinic in Sydney, Australia, and cases 2 and 3 were examined by the Veterinary Specialist Group and the Animal Referral Centre, respectively, both in Auckland, New Zealand. The histological samples were evaluated by 3 different board-certified anatomical pathologists, and immunohistochemistry was carried out by Michigan State University Veterinary Diagnostic laboratory (MSU VDL).

2.1 | Case 1

A 6-year-old, male neutered, domestic shorthaired cat was examined at a routine annual appointment and an abdominal mass was palpated. The cat was not exhibiting any clinical signs at the time of this consultation. Pet-grade raw mince was prevalent in the cat's diet, but commercial tinned and dry foods also were consumed. The cat had free access to the outdoors. Retroviral testing found the cat to be negative for both FIV and feline leukemia virus (FeLV) using a point-of-care rapid immuno-migration assay (WITNESS FeLV-FIV Test kit, Zoetis, NSW, Australia). Serum biochemistry results were normal, and the PCV was within normal limits. On abdominal ultrasound examination, a large (5 cm), lobulated soft tissue mass was identified in the region of the jejunal lymph nodes, and the small intestine was diffusely thickened. Exploratory laparotomy was performed and a large mass was identified, firmly adhered to the omentum. The purulent contents of the mass were submitted along with a liver biopsy specimen for aerobic and anaerobic culture with susceptibility testing (Figure 1). Samples from this lesion, as well as from the liver and intestines, were collected for histopathology. Postoperatively, along with supportive care, the cat received antibiotics that included amoxicillin/clavulanic acid (Norclav 175 mg/mL, Norbrook, VIC, Australia) at 19.3 mg/kg SC q12h and enrofloxacin (Baytril 2.5% solution, Bayer, NSW, Australia) at 5.6 mg/kg SC q24h. Cytology of the purulent material identified colonies of slender Gram-positive rods, and upon aerobic incubation, the organisms ultimately were identified as Lm by a combination of gas chromatographic analysis of fatty acid methyl esters and

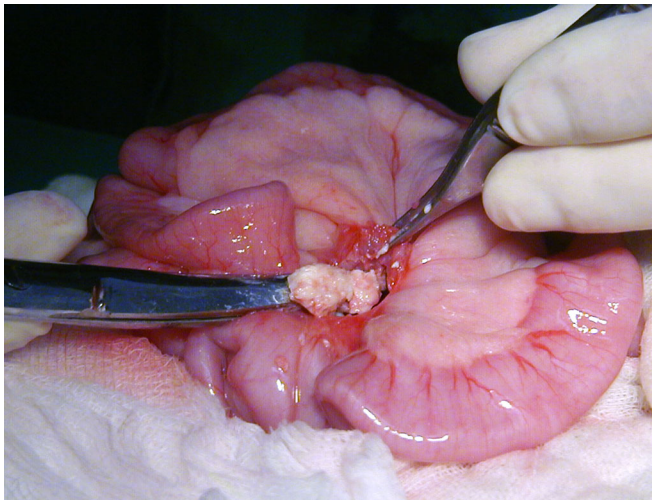

FIGURE 1 Case 1: Mesenteric lymph node undergoing debridement during first exploratory laparotomy

16S ribosomal ribonucleic acid (16S rRNA) gene analysis. The 16S RNA result was a 99% match to the GenBank library entry AL591983 for Lm. No aerobic or anaerobic bacterial growth was reported from the liver sample. The organism was found to be sensitive to penicillin, doxycycline, and vancomycin. Histology results indicated pyogranulomatous inflammation within the lymph nodes, mild cholangitis and hepatic degeneration, and mild neutrophilic enteritis. The cat was treated at home over the next 5 months with amoxicillin (Amoxil 200 mg tablets, Aspen, NSW, Australia) at 32.3 mg/kg PO q8h. The cat remained systemically well with the mass monitored by abdominal palpation during regular visits to the primary care clinic. Approximately 5.5 months after the exploratory laparotomy, the cat had a repeat abdominal ultrasound examination and the previously identified mass was estimated to be getting slightly larger and was intimately associated with the mesenteric vasculature. Approximately 1 month later, the cat underwent a second exploratory laparotomy and the abdominal mass was incised and debrided followed by lavage and capsular omentalization. Over the next few years, the cat was examined regularly at the practice with no reports of a palpable abdominal mass and the owners reported the cat to be clinically well. Fifty-six months later (63 months after initial diagnosis), the cat was examined for weight loss, poor appetite, and an abdominal mass was once again palpable. Serum biochemistry results included increased alanine aminotransferase activity (66 U/L; reference range, 0-48), low urea concentration (4.6 mmol/L; reference range, 5.6-12.9), and increased total T4 concentration (60 nmol/L; reference range, 6-52). A diagnosis of hyperthyroidism was made and carbimazole (Neomercazole 5 mg tablets, Amdipharm, NSW, Australia) was initiated at 1 mg/kg PO q12h. Response to medical management of the hyperthyroidism was unrewarding, and a month later the cat presented for progressive weight loss and lethargy. Abdominal ultrasound examination identified a large, heterogenous, cranial abdominal mass, in addition to the previously sampled abdominal mass. The next day, exploratory laparotomy identified a large, irregular, highly vascular mass, intimately associated with the liver (the omentalized abscess from the prior surgery

appeared quiescent). Based on the gross appearance of the hepatic lesion, which was consistent with a primary neoplasm, euthanasia was recommended and performed approximately 64 months after initial presentation for listeriosis. Necropsy was not performed to confirm the nature of the hepatic mass.

2.2 | Case 2

A 4-year-old, male neutered, Tonkinese cat was examined at a primary care practice for anorexia and weight loss and was subsequently referred to a radiologist for further evaluation of an abdominal mass identified on palpation. The cat was fed a pet-grade raw meat diet alongside a commercial dry diet and was allowed free access to the outdoors. Hematology and serum biochemistry testing identified mild neutrophilia (17.5×10^9/L; reference range, 2.5-12.5), hyperproteinemia (98 g/L; reference range, 58-80), mild hyperglycemia (7.2 mmol/L; reference range, 3.9-6.9), and mildly increased serum creatinine concentration (179 μmol/L; reference range, 53-177). Retroviral testing was negative for both FIV and FeLV using a point-of-care immunochromatographic assay (Anigen Rapid FIV Ab/FeLV Ag Test Kit, Gyeonggi-do, Korea). Abdominal ultrasound examination identified abdominal lymphadenomegaly and fine-needle aspirates of the abdominal lymph nodes identified pyogranulomatous inflammation and extracellular bacteria. At this time, the owner did not pursue any treatment. Four months later, the cat was examined by a referral clinic for further investigation and management of the abdominal lymphadenomegaly. Abdominal ultrasound examination at this time identified progressive, marked abdominal lymphadenomegaly, and intestinal wall thickening. Exploratory laparotomy identified thickened jejunum and markedly enlarged ileocecal lymph nodes, measuring up to 4 cm in diameter. Incisional biopsies were performed of the mesenteric lymph nodes as well as the duodenum, jejunum, ileum, and liver. Histological examination of the lymph node samples identified Gram-positive bacilli within the necrotic areas (Figure 2), and the normal architecture of the node was effaced by necrosis, fibrosis, and granulation tissue (Figure 3). The cat also had lymphoplasmacytic and suppurative enteritis and perivascular pyogranulomatous hepatitis. Samples of the lymph nodes were submitted for aerobic and anaerobic bacterial culture and susceptibility testing. The cultures yielded a scant growth of Lm, identified using matrix-assisted laser desorption ionization time-of-flight mass spectrometry (MALDI-TOF MS). Samples of the lymph node also were submitted for immunohistochemistry for Lm to the MSU VDL, which confirmed the MALDI-TOF MS results (Figure 4). The cultured Lm was reported to be sensitive to amoxicillin/clavulanic acid, cephalothin, penicillin, tetracycline, and trimethoprim/sulfamethoxazole. The cat initially was treated with amoxicillin/clavulanic acid (Clavulox 50 mg tablets, Zoetis) at 20 mg/kg PO q12h and trimethoprim/sulfamethoxazole (Deprim 200 mg sulfamethoxazole, 40 mg trimethoprim per 5 mL

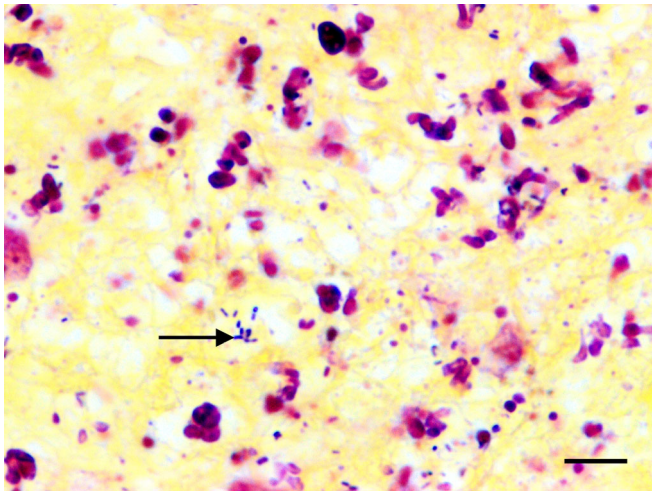

FIGURE 2 Case 2: Mesenteric lymph node. Scattered intracellular and extracellular Gram-positive bacilli (arrow [to clump]). ×100. Modified Brown-Hopps Gram (scale bar = 50 μm)

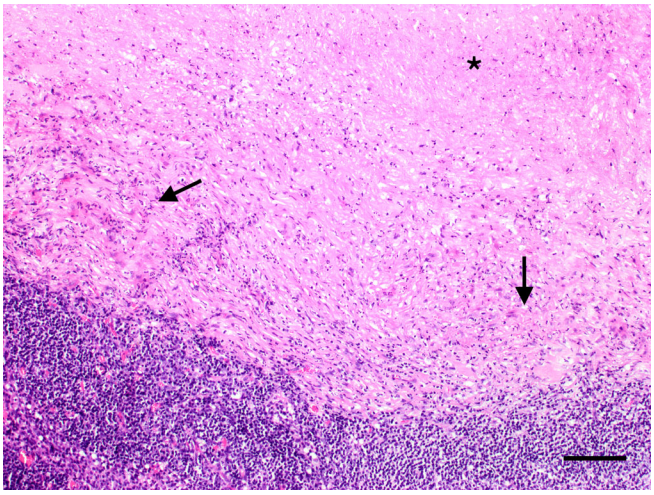

FIGURE 3 Case 2: Mesenteric lymph node. Extensive coagulative necrosis (*) attended by infiltrating neutrophils and fewer macrophages (arrows). ×10. Hematoxylin an Eosin (scale bar = 400 μm)

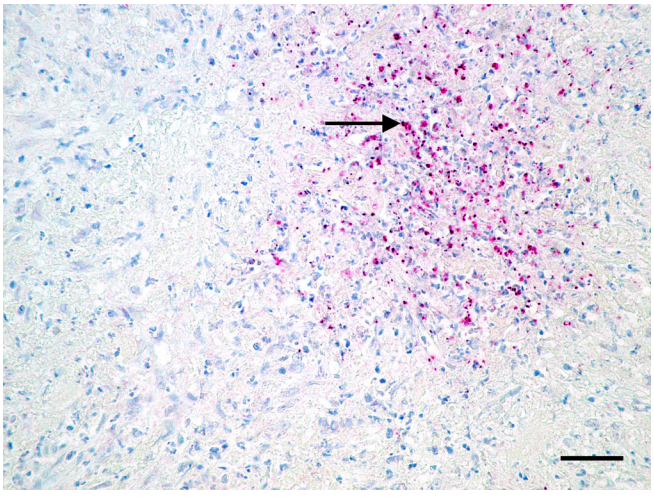

FIGURE 4 Case 2: Immunohistochemistry against *Listeria monocytogenes* detected large numbers of bacilli (red) in centers of coagulative necrosis (arrow). ×40. Alkaline phosphatase red, hematoxylin counterstain (scale bar = 200 μm)

solution, AFT, Auckland, New Zealand) at 24 mg/kg PO q12h. After approximately 6 weeks of treatment, progressive lymphadenomegaly was observed and the cat was switched to gentamycin (Gentamycin Injection BP 40 mg/mL solution, Pfizer, Auckland, New Zealand) at 7.4 mg/kg SC q24h in combination with amoxicillin/clavulanic acid at the previous dose and dose interval. The cat was evaluated after finishing this second antibiotic course to which it responded favorably, with a good appetite and weight gain, but the abdominal lymphadenomegaly had not improved on sonographic evaluation. The cat's antibiotic treatment then was changed to amoxicillin (Alphamox 25 mg/mL solution, Mylan, Auckland, New Zealand) at 15 mg/kg PO q12h and it was started on prednisolone (Redipred 5 mg/mL solution, Aspen) at 0.6 mg/kg PO q24h based on presumption of an exuberant inflammatory response contributing to the clinical syndrome. Subsequently, the owner was able to manage flare-ups of clinical signs (eg, anorexia and vomiting) with short, tapering, anti-inflammatory doses of prednisolone (<4 weeks) in conjunction with amoxicillin/clavulanic acid. The cat continued to do well according to a phone conversation with the owner 27 months after initial diagnosis.

2.3 | Case 3

A 6-year-old, male neutered, Burmese cat was examined at an after-hours clinic for acute onset of vomiting and anorexia and was treated supportively overnight. The cat was fed a commercial dry diet and had free access to the outdoors. After referral to a specialty clinic the next day, a mass was palpated in the cranial abdomen, and subsequent abdominal ultrasonography identified a 2.8 cm hypoechoic mass in the cranial abdomen. Biochemistry test results included mild hyponatremia (138 mmol/L; reference range, 142-164), mild hypophosphatemia (0.8 mmol/L; reference range, 1.1-2.74), mild hyperglycemia (8.8 mmol/L; reference range, 3.9-8.3), and a CBC was normal. The cat was negative for both FIV and FeLV using a point-of-care immunochromatographic assay (Anigen Rapid FIV Ab/FeLV Ag Test Kit). An exploratory laparotomy identified a mass in the region of the mesenteric lymph node, and a wedge biopsy of this tissue was performed as well as biopsy of the liver, stomach, and intestines. The cat was discharged from the hospital with a course of amoxicillin/clavulanic acid (Clavulox 50 mg tablets, Zoetis) at 15.6 mg/kg PO q12h. Samples of the abdominal mass were submitted for aerobic and anaerobic bacterial culture and susceptibility testing that resulted in no growth. Histology of the abdominal mass disclosed severe necrotizing and pyogranulomatous lymphadenitis with intralesional Gram-positive bacilli. Sections of liver and stomach were normal, but marked lymphoplasmacytic, eosinophilic, and neutrophilic inflammation was observed in sections of duodenum and jejunum. Immunohistochemistry of the abdominal mass confirmed infection with Lm. The cat was examined 3 weeks after surgery and was eating well and gaining weight with no further vomiting noted. Clinical examination at this time identified persistence of the cranial abdominal mass and repeat abdominal ultrasound examination determined the lesion to be of similar size (2.8 cm). Amoxicillin/clavulanic acid was continued for 2 more weeks at the same dose and dose interval. Subsequent examinations by the referring veterinarian documented no clinical signs referable to the abdominal mass, and the size of the cranial abdominal mass was decreased based on serial abdominal palpation. The cat was last examined 20 months post-surgery and the abdominal mass remained palpable, but the cat was clinically well aside from intermittent vomiting.

3 | DISCUSSION

In our case series, all 3 cats presented with similar manifestations of chronic listeriosis, best described as the “mesenteric lymphadenitis” phenotype with an unexpectedly protracted temporal progression. Infection likely resulted from either gastrointestinal translocation, or possibly bacteraemia. *Listeria monocytogenes* has been reported to adopt an L-form phenotype, which may alter its antibiotic susceptibility, particularly to beta lactams.[17] Histology of the affected lymph nodes in 2 of the cats disclosed large areas of necrosis, which may have contributed to the persistence of the lymphadenitis by resistance to phagocytosis, essentially behaving as sequestra.

The most common route of transmission of listeriosis is by ingestion of contaminated food, and raw meat diets have been shown to cause listeriosis in humans.[18] Two of the cats in our series had been fed raw meat before developing listeriosis, suggesting raw meat as the source of the infections. A recent communication from the United Kingdom reported 3 exclusively indoor cats developing tuberculosis with infection potentially associated with a commercial raw meat-based diet.[19] Even diets including raw meat intended for human consumption can present a risk of bacterial infection to pets, with up to 34% of raw chicken samples acquired from retail grocery stores in a study containing Lm.[20] Slightly higher rates of contamination with Lm were identified in a 2018 analysis of raw food pet diets where 54% samples tested positive for the bacterium.[21] Raw meat-based pet diets are becoming increasingly popular, and Lm may be yet another infectious agent that presents a risk to the animals receiving these diets. There has been at least 1 report of *Listeria grayi* contamination of pet jerky treats and dry cat food,[22] and although this nonpathogenic *Listeria* species is not implicated in foodborne disease, the detection of a nonpathogenic *Listeria* spp. acts as an indicator of conditions appropriate for the presence and growth of Lm.[23] Thus, there is also the potential for commercially processed pet foods to act as the source of Lm infection.

Two of the cats in our study were oriental breeds (Tonkinese and Burmese), and 1 of the first case reports of systemic listeriosis in a cat was a young female spayed Siamese cat.[7] This finding may suggest a possible vulnerability of these breeds to infection with Lm. Siamese cats also have been shown to have a higher incidence of cryptococcosis and disseminated mycobacteriosis compared to other breeds.[24,25] Interestingly, all of these infections induce a primarily histiocytic and granulomatous immune response. It is unknown whether oriental cat breeds are less immunocompetent than other breeds or potentially harbor a genetic defect affecting the ability of dendritic cells to counter these diseases.

Studies in mice have shown that resistance to infection with Lm is impaired when interleukin 10 (IL-10) concentrations are increased.[26,27] An increase in IL-10 augments the immune response in a number of ways, particularly through the down-regulation of interferon-gamma, a cytokine central to cell-mediated immunity, and clearance of Lm infection in murine models.[28,29] Chronically FIV-infected cats have been shown to have marked upregulation of IL-10 compared to healthy cats, and this cytokine abnormality has been implicated in decreased resistance to infection with Lm in these retrovirus-positive individuals.[12] According to the sufficient cause model, a minimum set of factors when present in an individual will produce disease.[30] The sufficient cause for disease in each individual can be made up of different factors or mechanisms, and thus immunocompromise may not be required for every instance of listeriosis in cats. Recent research using multilocus sequence typing has categorized Lm into subtypes known as clones.[31] These data have identified hypervirulent clones of Lm that are able cause severe disease in immunocompetent humans.[31] One would expect a similar stratification of virulence in Lm to which our veterinary patients are exposed and thus this stratification may account for the unpredictable incidence of infection, which can include immunocompetent animals.

Although the excretion of Lm in the feces of cats has been reported,[15] the zoonotic risk that cats with listerial mesenteric lymphadenitis pose has not been established. The feces of the cats in our case series was not cultured to determine excretion of Lm, but we suspect that the risk lies instead with the owners of these cats being exposed to a common source of contamination, namely raw meats. This may be of particular concern if the pet's diet is a homemade preparation using ingredients that also are being consumed by the owners.

Although rare, listerial mesenteric lymphadenitis should be a differential diagnosis for abdominal lymphadenomegaly in young to middle-aged cats with or without clinical signs. Cats with the "mesenteric lymphadenitis" phenotype of Lm infection can have extended survival times, and the role of medical treatment in the life history of the disease is difficult to determine. With regard to treatment, a sensible recommendation would be surgical extirpation of affected nodes with omentalization followed by use of empirically chosen antibiotics. Amoxicillin, as a single agent, would be the antibiotic of choice because it has shown in vitro superiority to ampicillin against Lm, is safe in cats, and is widely available in a veterinary setting.[32] The role of raw meat-based diets in the development of listeriosis cannot be demonstrated definitively, but such diets may be a risk factor.

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CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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