Leishmaniosis

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Abstract

Leishmania spp. affecting cats include *L. infantum*, *L. mexicana*, *L. venezuelensis*, *L. amazonensis*, and *L. braziliensis*. *Leishmania infantum* is the species most frequently reported in cats and causes feline leishmaniosis (FeL). Cats exposed to *L. infantum* are able to mount a cell-mediated immune response that does not parallel antibody production. Cats with *L. infantum*-associated clinical disease have positive blood PCR and low to very high antibody levels. About half of the clinical cases of FeL are diagnosed in cats with impaired immunocompetence. Skin or mucocutaneous lesions are the most common clinical findings; however, FeL is a systemic disease. Skin or mucocutaneous lesions and lymph node enlargement are seen in at least half of cases, ocular or oral lesions and some aspecific signs (weight loss, anorexia, lethargy) in about 20–30% of cases, and many other clinical signs (e.g., respiratory, gastrointestinal) are sporadically observed. Ulcerative and nodular lesions due to diffuse granulomatous dermatitis are the most frequent skin manifestations, mainly distributed on the head or symmetrically on the distal limbs. Diagnosis can be obtained by cytology and histology, and immunohistochemistry is useful to confirm the causative role of *Leishmania* infection in the dermopathological manifestations; however, other skin diseases may coexist with FeL. Polymerase chain reaction is used in case of suggestive lesions with lack of parasites and for *Leishmania* speciation. Comorbidities, coinfections, and chronic renal disease influence the prognosis and should be investigated. Treatment is currently based on the same drugs used for canine leishmaniosis, and generally clinical cure is obtained; however recurrence is possible.

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Introduction

Leishmaniases are protozoan diseases caused by *Leishmania* spp. affecting humans and animals, but leishmaniosis is the term used for diseases in animals. Leishmaniosis caused by *Leishmania infantum* is a severe, zoonotic, vector-borne disease endemic in areas of the Old and New Worlds, with dogs as the main reservoir [1]. In fact, the majority of infected dogs do not develop clinical signs or clinico-pathological abnormalities, but they are chronically infected and infectious to sand fly vectors. Dogs may, however, develop a mild to severe systemic disease, with frequent skin lesions usually associated with other clinical and clinico-pathological abnormalities. Therefore, much research interest has been focused on canine leishmaniosis (CanL), in order to prevent the infection, understand the pathomechanisms driving infection to disease, make early and accurate diagnosis, and treat affected dogs. Conversely, until about 25 years ago, the cat was considered a resistant host species to *Leishmania* infections, based on very rare case reports, occasional post mortem finding of the parasite in cats from endemic areas, and results from an experimental infection study demonstrating limited infection rates [2]. Over the last decades, an increasing number of clinical cases have been reported, and investigations with more sensitive diagnostic techniques detected a variable, but not negligible, infection rate in cats living in endemic areas. Therefore, feline leishmaniosis (FeL) appears nowadays as an emergent disease, and the cat's role as reservoir host is revalued. We now know that the epidemiology of leishmaniosis is complex and the vectorial transmission in endemic areas involves multiple host species infectious to sand flies, including the cat. Tegumentary leishmaniosis caused by dermotropic *Leishmania* spp. is rarely reported in both dogs and cats. Dermotropic species infecting cats are *Leishmania tropica* and *Leishmania major* in the Old World and *Leishmania mexicana*, *Leishmania venezuelensis*, and *Leishmania braziliensis* in the Americas. Main reservoir hosts for dermotropic species are wild animals, such as rodents.

Etiology, Diffusion, and Transmission

Leishmania genus (Kinetoplastea: Trypanosomatidae) includes diphasic and dixenous protozoans replicating as promastigotes in the gut of phlebotomine sand flies, their natural vectors. When inoculated into vertebrate hosts by sand fly bites, promastigotes change to the non-flagellated amastigote form that multiplies by binary fission in macrophages. *Leishmania* spp. detected in cats are able to infect also other mammals (including dogs and humans) and belong to the subgenus *Leishmania* (*L. infantum*, *L. mexicana*, *L. venezuelensis*, *L. amazonensis*) or *Viannia* (*L. braziliensis*).

Leishmania infantum is the species most frequently reported in both dogs and cats in the Old World and in Central and South America. *Leishmania infantum* has been detected in cats in Mediterranean countries (Italy, Spain, Portugal, France, Greece, Turkey, Cyprus), Iran and Brazil [3–6]. Reported antibody and blood PCR prevalences are very variable (from nihil to >60%) and influenced by many factors

such as the local level of endemicity, selection of tested cats and analytical differences [3]. However, *L. infantum* antibody and molecular prevalence is usually lower in cats compared to dogs and cases of FeL are rarer [3, 7]. Cases of both CanL and FeL are diagnosed in non-endemic areas in dogs or cats rehomed from or travelling to endemic areas [1, 8–13].

Sand fly transmission is the most important way of transmission of *Leishmania* to humans and animals, and several studies about the feeding habit of sand flies suggest that this is likely also in feline infection, but it has never been investigated [3, 14–16]. Non-vectorial transmission (vertical, by blood transfusion, mating, or bite wounds) of CanL is well known and responsible for autochthonous cases in non-endemic areas in dogs, but we have no evidence of these ways of transmission to and in cats [1, 10, 17, 18]. However, blood transfusion could be a source of infection in cats as proven in dogs and humans. In fact, healthy cats – similarly to healthy dogs and humans – are found blood PCR positive in endemic areas [4–7, 19–22].

Pathogenesis

Leishmania infantum

A great number of both experimentally and field controlled prospective studies performed on CanL provided information about immunopathogenesis of CanL, but we do not have similar studies performed on cats. In dogs, T helper 1 (Th1) immune response responsible for protective CD4+ T cell-mediated immunity is associated to resistance to the disease [1]. Conversely, progression of *L. infantum* infection and development of lesions and clinical signs in dogs and humans are associated with a predominant T helper 2 (Th2) immune response and the consequent non-protective antibody production and T cell exhaustion [23]. Depending on a variable balance between humoral and cell-mediated immunity in the infected dog, a wide and dynamic clinical spectrum is seen in CanL, including subclinical infection, self-limiting mild disease, or severe progressive illness [1, 24]. Sick dogs with severe clinical disease and high blood parasitemia show a high antibody level and lack in specific IFN-γ production [25]. Similarly to what occurs in mouse experimental models, a complex genetic background modulates the dog's susceptibility or resistance to CanL [1, 24]. In cats, the adaptive immune response elicited by *L. infantum* exposure in endemic areas was recently explored with measurement of specific antibody and IFN-γ production [26]. Some cats produced *L. infantum*-specific IFN-γ and were found blood PCR negative and antibody negative or in few cases borderline positive [26]. This means that, similarly to other mammals, cats exposed to *L. infantum* are able to mount a protective cell-mediated immune response that does not parallel antibody production. The relationship between immunological pattern and severity of disease is still unexplored in cats; however, we know that cats with *L. infantum*-associated clinical disease have a high blood parasitemia and low to very high antibody levels [3, 27–32]. Moreover, longitudinal studies found that progression of the infection toward disease is associated in cats with increasing antibody titers, and, on the other hand, clinical improvement obtained by anti-*L. infantum*

therapy is associated with a significant reduction of antibody levels, similarly to CanL [33–36]. Coinfections with some vector-borne pathogens (e.g., *Dirofilaria immitis*, *Ehrlichia canis*, *Hepatozoon canis*) can influence parasite burden and progression of CanL [37–39]. In cats, the association between retroviral, coronavirus, *Toxoplasma*, or vector-borne coinfections and antibody and/or PCR positivity to *L. infantum* has been explored [5, 20, 40–50]. A significant association was found only between feline immunodeficiency virus (FIV) and *L. infantum* positivity in some cases [41, 46, 48]. Moreover, more than one third of cats with FeL and tested for retroviral coinfections were found positive to FIV [a few were also positive to Feline Leukemia Virus (FeLV)] [11, 12, 27–29, 31, 51–69]. Other FeL cases reported in FIV and FeLV negative cats were diagnosed in animals affected by immune-mediated diseases (and treated with immunosuppressive drugs), neoplasia, or diabetes mellitus, and we may assume that about half of the clinical cases of FeL were diagnosed in cats with impaired immunocompetence [12, 27–30, 34, 52, 59–61].

Despite the fact that skin or mucocutaneous lesions are the most common clinical findings, FeL is considered a systemic disease as CanL. Parasites can be detected in various other tissues, such as lymph nodes, spleen, bone marrow, eye, kidney, liver, and gastrointestinal and respiratory tract [8].

American Dermotropic *Leishmania* spp.

Some scanty information about adaptive immune response of cats toward American dermotropic *Leishmania* spp. can be inferred only from case reports of *L. mexicana* and from an experimental infection of cats with *L. braziliensis* [70–72].

Delayed-type hypersensitivity skin test with *L. donovani* antigen was repeatedly found negative in a cat affected by recurrent nodular dermatitis caused by *L. mexicana* infection, suggesting a lack of cell-mediated adaptive immune response in this cat [70]. Anti-*Leishmania* antibody production seems to be limited, as of five cats with *L. mexicana* tegumentary leishmaniosis only two were antibody positive at ELISA test, although Western blot test was positive in four [71]. Moreover, in cats intradermally infected with a human strain of *L. braziliensis*, a short-term antibody production was documented after the development of skin lesions, but, frequently, it appeared after the healing of lesions [72].

Clinical Picture

Leishmania infantum

Currently, in endemic areas FeL is far less frequently reported than CanL, but we are probably underestimating the disease, particularly the less frequent and less severe clinical presentations, as it occurred in the past with CanL. Furthermore, coinfections or comorbidities are frequently detected which can contribute to a clinical misrepresentation and misdiagnosis of FeL [3, 22, 27–32]. About a hundred of clinical cases were reported over the last 30 years – mostly in Southern Europe – and

they are at present the only source of knowledge about FeL. We are therefore aware of the current low level of evidence (III–IV) for statements and recommendations concerning this disease.

Age range of affected cats is wide (2–21 years); however, they are mostly mature cats (median age 7 years) at diagnosis, with very few being 2–3 years old [3, 27, 28, 32, 51, 57, 73]. Both genders are similarly represented and almost all cases are reported in domestic short-hair cats.

Some clinical manifestations are very frequent at diagnosis – found in at least half of the cases – such as skin or mucocutaneous lesions and lymph node enlargement. Common presentations – found in one fourth to half of the cats – are represented by ocular or oral lesions and some aspecific signs (weight loss, anorexia, lethargy). Finally, there are many clinical signs seen in less than one fourth of the cases. Usually affected cats display more than one clinical sign and often develop different lesions with time.

Skin and Mucocutaneous Manifestations

Skin or mucocutaneous manifestations were found in about two thirds of reported cases, but they rarely were the only abnormality detected [3, 8, 27–30, 73]. In a study from a pathology laboratory from Spain, FeL was diagnosed in 0.57% of all skin and ocular biopsies (n = 2632) examined over a 4-year period [73].

Several dermatological entities have been described, and different presentations often coexisted or developed subsequently in the same cat. Most lesions were observed on the head. Pruritus was rarely reported, and in most cats manifesting pruritus, a concurrent dermatological disease was identified such as flea allergy, eosinophilic granuloma, pemphigus foliaceus, squamous cell carcinoma (SCC), or demodicosis [12, 67, 75, 76]. In one case, however, pruritus stopped after starting anti-*Leishmania* therapy [77].

Ulcerative dermatitis is the more commonly reported skin lesion and sometimes with a history of self-healing and recurrence of lesion. Crusty-ulcerative lesions with raised margins were seen on pressure points (hock, carpal, and ischiatic regions), often symmetrically, and were large up to 5 cm (Fig. 1) [27, 28, 54, 57, 64,

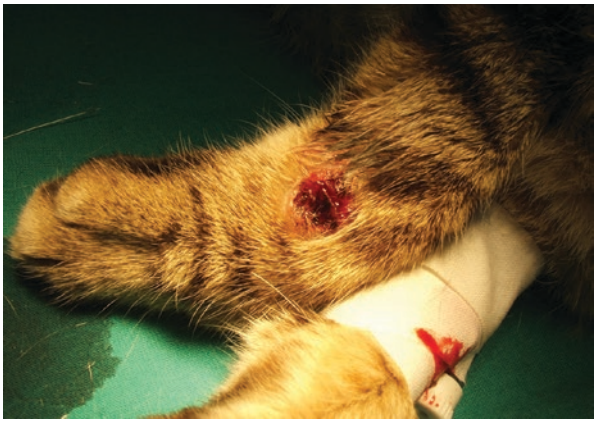

Fig. 1 Large ulcer with raised margins on right forelimb. A similar symmetrical lesion was present on the left forelimb

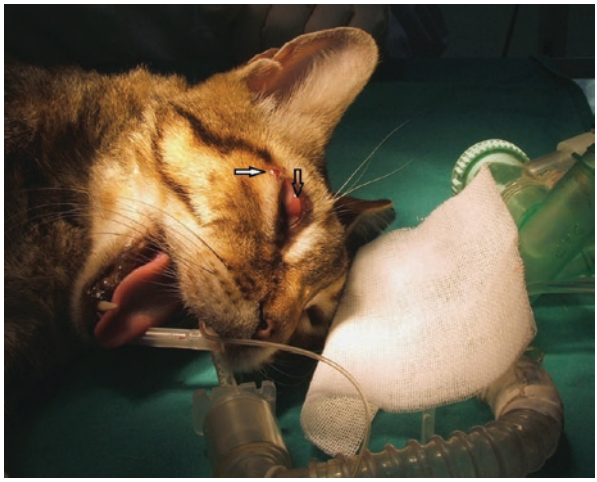

Fig. 2 Solitary focal ulceration on the face (white arrow) and conjunctival nodule (transparent arrow) observed in the same cat of Fig. 1

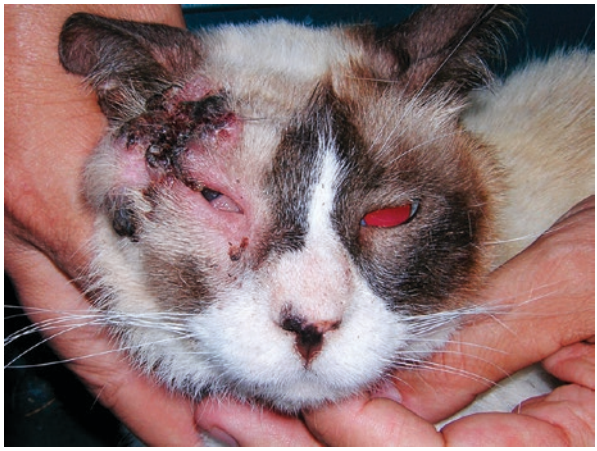

Fig. 3 Severe facial ulceration in a cat diagnosed with squamous cell carcinoma associated with *L. infantum* dermal infection

77]. Focal solitary or multiple smaller ulcers were reported on the face (Fig. 2), lips, ears, neck, or limbs [27, 28, 34, 64, 65, 73, 77–79]. In a few cases focal or diffuse ulcerative dermatitis affected face, trunk, or footpads [27, 63, 65, 79]. Ulceration of the *nasal planum* was also reported, and in one case it was associated with concurrent SCC [30, 54, 58, 67]. *Leishmania* infection and SCC were found associated in biopsied tissues obtained from a deep facial ulceration (Fig. 3) in other two cases [56, 76]. Unfortunately, the diagnosis of SCC was missed at first consultation in two cases when only *Leishmania* infection was detected by cytology or histology [30, 76]. Moreover, multifocal ulcerative dermatitis caused by *L. infantum* was diagnosed in a cat suffering from SCC at a different site [65]. Ulcerative dermatitis was found associated with eosinophilic granuloma complex, and in one other case *Leishmania* infection was confirmed (by serology and skin PCR) in a cat with pemphigus foliaceus [12, 73].

Nodular dermatitis is also a frequent dermatological manifestation, and single, multiple or diffuse, firm, alopecic, non-painful nodules were detected. They are usually small (<1 cm), mainly distributed on the head and, in descending order of frequency, on the eyelid, ear, chin, nose, lips, and tongue [11, 27, 28, 31, 55, 64, 66, 73, 80–83]. Nodules can be found also on limbs or rarely on the trunk or the anus [12, 55, 73]. In rare cases nodules were ulcerated [12, 66, 84].

Differently from CanL, facial or diffuse scaling and alopecia are less frequently reported in FeL, and in few of these cases, histopathological evaluation confirmed the presence of amastigotes in the affected skin [29, 63, 73]. Digital hyperkeratosis was found in one case only [27].

An atypical FeL presentation that is not reported in CanL is development of hemorrhagic bullae, observed in three cases, respectively, on the nasal planum, head, and margin of the pinna [34, 76]. However, the lesion developed on the nasal planum was histologically diagnosed as hemangioma [76]. The other two cases were cytologically evaluated and amastigotes were found [34].

Visceral Manifestations

Lymph node enlargement is the most frequent non-dermatological finding [3]. It is usually multicentric and can be symmetrical. Lymph nodes are firm and non-painful, and enlargement can be relevant mimicking neoplasia. Monolateral or bilateral ocular lesions were reported in about one third of cases, but a specialistic ophthalmic examination was not performed in all cats with FeL; therefore, some less severe ocular findings could have been missed. Conjunctivitis (including also conjunctival nodules) and uveitis are the most frequent ocular manifestations [11, 27, 31, 34, 60, 62, 64, 68, 73]. Keratitis, keratouveitis, and chorioretinitis were diagnosed in a few cats [27, 31, 34, 67, 78]. Panophthalmitis is the consequence of progressive extension of diffuse granulomatous inflammation in case of late diagnosis [60, 73].

Apart from single cases of gingival ulceration, nodular glossitis, or epulid-like lesions, chronic stomatitis and faucitis was found in about 20% of cats, and the parasite was detected in the inflamed oral tissue [27, 31–34, 52, 58, 60, 62, 66, 78, 83].

Non-specific manifestations as weight loss, anorexia, or lethargy were not very frequent [3], and occasionally gastrointestinal (vomiting, diarrhea) or respiratory (chronic nasal discharge, stertor, dyspnea, wheezing) signs were reported [3, 74]. Rare manifestations were icterus, fever, spleen or liver enlargement, and abortion [3]. Interestingly, chronic leishmanial rhinitis was confirmed in some cases [58, 64, 73–75].

American Tegumentary Leishmaniosis (ATL)

A limited number of cases of feline cutaneous leishmaniosis caused by dermotropic *Leishmania* species were reported in the Americas [70, 71, 85–91]. Not always *Leishmania* speciation was obtained from affected cats, and *L. mexicana* could be identified in nine cases [70, 71, 91], *L. braziliensis* in five [85–88], *L. venezuelensis* in four [90], and *L. amazonensis* in one [89]. They were all domestic short-hair

cats and younger (age range: 8 months–11 years; median age 4 years) than cats with disease caused by *L. infantum*. The most common manifestation consisted in solitary or multiple firm nodules as large as 3 × 2 cm. They were alopecic, variably erythematous, or ulcerated and mainly distributed on the pinnae and the face (eyelids, *nasal planum*, muzzle) and rarely on the distal limbs or tail. A larger (6 cm) interdigital ovoid lesion was reported in a cat with *L. braziliensis* infection [88]. Nasal or ear ulcerations were seen in two cats with *L. mexicana* infection and in two others (*nasal planum* or medial canthus) with *L. braziliensis* infection [71, 86, 87]. Mucosal nodules may develop in the nasal cavity causing sneezing, stertor, and inspiratory dyspnea [71, 85]. No other manifestations were reported in cats with ATL; however, some followed up cases of *L. venezuelensis* or *L. mexicana* infections developed new nodular lesions at other sites [70, 90].

Diagnosis

Diagnostic testing of symptomatic cats aims to confirm *Leishmania* infection and to establish a causal relationship with the clinical picture. In case of dermatological or mucosal lesions, the cytological evaluation of impression smears from erosions and ulcers, of scrapings from margins of deep ulcers, and of fine needle punction of nodules can show a pyogranulomatous pattern and the presence of amastigotes (in the cytoplasm of macrophages or extracellularly) (Fig. 4) [3, 71]. Amastigotes have an elliptic shape with pointed ends, measure about 3–4 × 2 μm, and are characterized by the rod-shaped basophilic kinetoplast set perpendicular to the large nucleus. Morphology of amastigotes does not allow to differentiate between *Leishmania* species. In cats with leishmaniosis caused by *L. infantum*, amastigotes can be found also in cytological samples from enlarged lymph nodes, bone marrow, nasal exudates, liver, and spleen and rarely in circulating neutrophils [3].

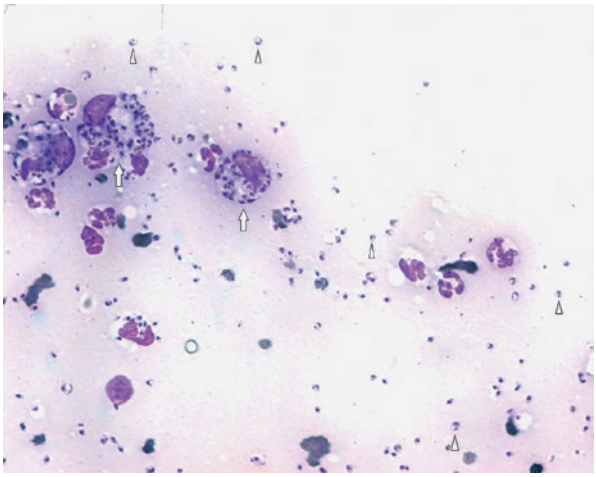

Fig. 4 Cytology from the cutaneous lesion in Fig. 1. Macrophagic–neutrophilic inflammation with numerous intracellular (arrows) and extracellular amastigotes. In some extracellular amastigotes the basophilic rod-shaped kinetoplast is clearly visible (arrow heads) (May Grünwald–Giemsa stain 1000×)

Biopsy of skin or mucosal lesions is required when cytology is inconclusive and in any case when the clinical presentation is compatible with neoplastic or immune-mediated diseases. Amastigotes are not easily detected by the conventional histological staining, and in suspected cases they should be investigated by immunohistochemistry (Fig. 5). However, immunohistochemistry does not allow the speciation of *Leishmania* amastigotes, which can be obtained by polymerase chain reaction (PCR) and sequencing of amplicons. PCR can be performed also from cytological slides, formalin-fixed and paraffin-embedded biopsies. Quantitative real-time PCR is very sensitive and can provide parasite load of samples.

Dermopathological evaluation (Fig. 6) shows dermal periadnexal to diffuse granulomatous inflammation with a diffuse infiltration of macrophages, a moderate number of amastigotes, and a variable number of lymphocytes and plasma cells [12, 73]. The overlying epidermis is affected by hyperkeratosis, acanthosis, and ulceration [73]. In nodular lesions giants cells may be seen [73]. A low number of parasites were found in nodular lesions, characterized by perifollicular granulomatous

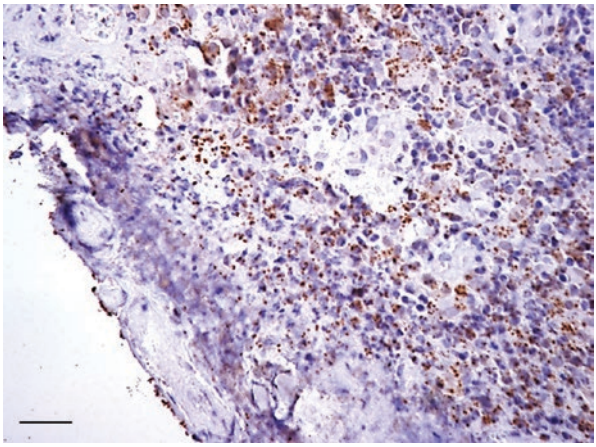

Fig. 5 Dark brown amastigotes evidenced by immunohistochemistry. Mayer's hematoxylin counterstain. Bar = 10 µm. (Courtesy of R. Puleio, IZS Sicilia, Italy)

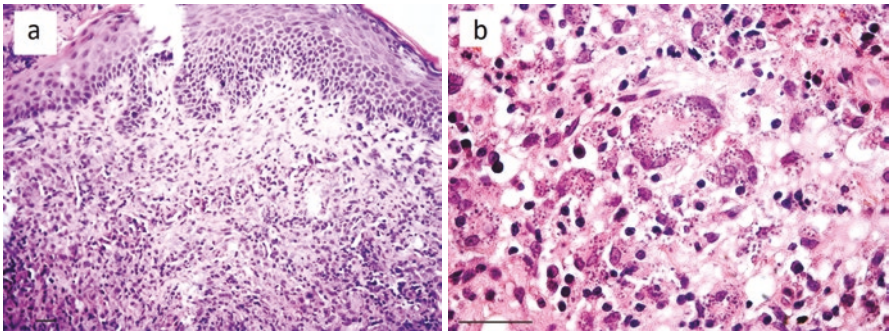

Fig. 6 Diffuse pyogranulomatous dermatitis (**a**) with numerous amastigotes within macrophages (**b**). HE. Bar = 10 µm. (Courtesy of R. Puleio, IZS Sicilia, Italy)

dermatitis and in a lichenoid interface dermatitis in a cat affected by scaly dermatitis [73]. Mucosal (and mucocutaneous) lesions harbor a higher parasite load and submucosal diffuse granulomatous inflammation is seen [62, 68, 73]. In some cases, a dermal, diffuse, granulomatous inflammation was found associated with lesions characteristic of feline eosinophilic granuloma complex [54, 73]. A transepidermal inflammatory infiltrate with parasitized macrophages was reported in the neoplastic tissue of a cat diagnosed with concurrent SCC [56]. In another case, a stromal infiltration of parasitized macrophages was observed adjacent to islands of SCC [30]. Nodular to diffuse granulomatous dermatitis with hyperkeratotic, hyperplastic, and often ulcerated epidermis is described in cases of ATL [71, 85, 91].

Anti-*L. infantum* antibody detection is performed by quantitative serology (IFAT, ELISA, or DAT) and Western blot (WB) techniques [3]. Cutoff setting for IFAT is established at 1:80 dilution, and almost all cats affected by clinical FeL caused by *L. infantum* have low to very high antibody levels [43, 92]. Conversely, sick cats with ATL may not have detectable circulating antibodies [71].

Culture of infected tissues provided feline strains that in most cases showed the same zymodemes and genotypes detected in dogs or humans [3, 30].

Clinico-pathological abnormalities more frequently reported at diagnosis in cats with FeL caused by *L. infantum* consisted in mild to moderate non-regenerative anemia, hyperglobulinemia, and proteinuria [3]. Chronic kidney disease (CKD), in most cases at an early stage (International Renal Interest Society [IRIS] stages 1 or 2), is often documented when a renal profile including urinalysis and the urine to protein concentrations ratio is performed [32, 75].

Clinico-pathological abnormalities of cats with ATL were rarely investigated and only eosinophilia and neutrophilia were found in one cat with *L. braziliensis* infection [70, 85].

Treatment and Prognosis

Treatment of cats with clinical FeL caused by *L. infantum* is empirical and based on off-label use of the most common drugs prescribed to dogs with CanL [3]. Long-term oral administration of allopurinol (10–20 mg/kg once or twice daily) as monotherapy or as maintenance treatment after a course of subcutaneous injections of meglumine antimoniate (50 mg/kg once daily for 30 days) are the most frequently used regimens. Clinical cure is usually obtained, but efficacy and safety of used protocols have never been evaluated in controlled studies; therefore cats should be monitored very carefully for adverse effects during treatment (particularly cats affected by renal disease) and for possible clinical recurrence after stopping the therapy [3, 27–32, 34, 74]. A cutaneous adverse drug reaction (head and neck erythema, alopecia, exfoliation, and crusting) was suspected few days after starting allopurinol in a cat [75]. The skin reaction rapidly solved after stopping allopurinol [75]. Increases in liver enzymes were observed in another cat, and they resolved

after lowering dosage to 5 mg/kg twice a day [12]. In two further cases, acute kidney injury was diagnosed few weeks after starting allopurinol administration [32]. In another cat with concurrent IRIS stage 1-CKD at the time of FeL diagnosis, azotemia developed after meglumine antimoniate and afterward to miltefosine (2 mg/kg orally once daily for 30 days) administration. [75] This latter cat was hereafter maintained with a dietary supplementation of nucleotides and active hexose correlated compounds that was recently found effective in dogs as CanL maintenance treatment [75, 93].

Domperidone (0.5 mg/kg orally once daily) was recently used in two cats in association with allopurinol, and miltefosine was given in one other case [27, 29, 30].

Surgical removal of nodules was performed but generally they recurred [12, 27, 54, 81]. In one case an integrated approach between surgery and chemotherapy was needed for treating large ulcerations [28].

Clinical recurrence is associated with raised antibody titer and parasite load [34].

Cats with clinical FeL may live for several years after diagnosis, even those untreated and/or FIV positive, unless concurrent conditions (neoplasia) and complications (chronic kidney disease) occur or develop [32, 68].

Scant information is available about treatment and prognosis of ATL. Some cats with *L. mexicana* ATL were cured after surgical excision of nodules [91]. However, radical pinnectomy was not effective in a FIV- and FeLV-negative cat and lesions recurred at pinnectomy site in about 2 years [70]. Subsequently new lesions progressively involved the muzzle and finally the nasal mucosa, and the cat was euthanized over 6 years after ATL diagnosis due to a mediastinal lymphosarcoma [70].

Prevention of *L. infantum* Infection

Individual protection of exposed cats reduces their risk to be infected by sand fly bites and to develop the clinical disease [3, 22]. *Phlebotomus perniciosus* and *Lutzomyia longipalpis*, proven vectors of *L. infantum*, respectively, in the Old and New Worlds, were found infected after feeding on one single sick cat with FeL [33, 94]. This means that protection of cats at population level contributes to the regional control of *L. infantum* infection. In fact, the percentage of antibody and/or PCR-positive cats is often not negligible in endemic areas [3–6, 20, 21, 41, 42, 45, 47].

Pyrethroids are used in dogs for preventing the bites of sand flies, but most of them are toxic to cats [3, 95]. Collars containing a combination of 10% imidacloprid and 4.5% flumethrin are the only pyrethroid formulation licensed also for cats, and it was effective in reducing incidence of *L. infantum* infection in cats living in endemic areas [22].

According to current knowledge, testing of blood donors by antibody detection and blood PCR is the only advisable measure for preventing non-vectorial transmission in cats [96].

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