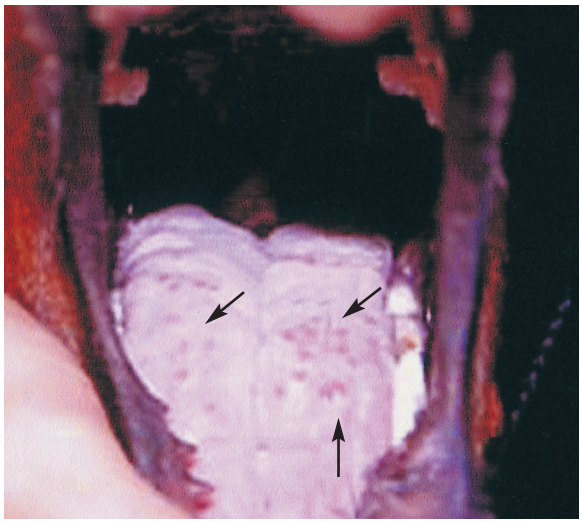

FIG 1: Numerous red, lentil-like lesions on the tongue surface (arrows) are evident

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Papular-like glossitis in a dog with leishmaniosis

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CANINE leishmaniosis is a severe systemic disease caused by the kinetoplastid protozoan *Leishmania infantum*, an obligatory intracellular parasite of mammalian macrophages transmitted by the bite of haematophagous phlebotomine sandflies. Domestic dogs represent the main reservoir hosts for zoonotic human visceral leishmaniosis in both the Old and New Worlds. Canine leishmaniosis is characterised by chronic viscerocutaneous signs, such as lymphadenopathy, skin lesions (furfuraceous dermatopathy, ulcers and nodular lesions), splenomegaly, onychogryphosis, and renal and ocular damage due to immunocomplex deposition (Ciaramella and others 1997). Atypically the parasites are found in the muscular system, central nervous system, endocrine glands (Cortese and others 1999) or gonads, with or without functional damage. Oral and lingual localisations are rarely described in dogs (Lamothe and Poujade 2002). This short communication describes the oral lesions in a dog with leishmaniosis.

A four-year-old female dobermann was diagnosed as suffering from leishmaniosis in February 2002. Clinical and haematological examinations showed lymphadenomegaly, weight loss, moderate anaemia and thrombocytopenia. The indirect immunofluorescent antibody test (IFAT) for *L infantum* and *Ehrlichia canis* was positive for both infections at a titre of 1:1280 and 1:1600, respectively. Serum protein electrophoresis showed elevated total proteins (10·8 g/dl), with hypoalbuminaemia, hyperglobulinaemia and a low albumin:globulin ratio (0·32). Bone marrow aspirate smears revealed numerous *Leishmania* species amastigotes. The dog was treated with a combination of meglumine antimoniate (Glucantim; Aventis) (50 mg/kg subcutaneously twice a day) and allopurinol (Zyloric; GlaxoSmithKline) (10 mg/kg orally twice a day) for 60 days, and doxycycline (Vibravet; Pfizer) (10 mg/kg orally) for 30 days. At the end of the treatment the animal showed full remission of clinical signs and a reduction of the antileishmanial IFAT titre (1:640).

Six months later, in October 2002, the dog was referred because of ocular disease, weight loss and hypersalivation. Clinical examination showed a severe uveitis with hyphema on the left eye and a nodular lesion on the right eyelid. Examination of the oral cavity revealed the presence of multiple red, lentil-like lesions on the tongue surface (Fig 1). The haematological parameters and IFAT titre were unchanged from the previous examination, and urinalysis revealed the slight proteinuria. Biopsy of lingual and eyelid lesions was performed by fine-needle aspiration and a brushing technique. In both lesions, cytological examination demonstrated the presence of numerous non-degenerated neutrophils, macrophages with foamy cytoplasm, epithelial cells, lymphocytes and a few multinucleated giant cells (Fig 2). Bacteria were not seen. Numerous *Leishmania* species amastigotes were seen in macrophage cytoplasm or extracellularly (Fig 3), and a culture from the left prescapular lymph node aspirate was positive for *Leishmania* species.

A specific therapy for uveitis, consisting of subconjunctival injections of triamcinolone (Uvitriam; Centralvet) weekly for two doses and orally administered prednisone (Delta

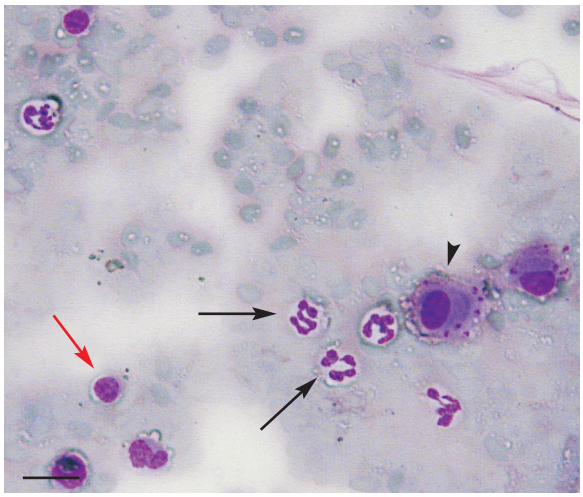

FIG 2: Non-degenerated neutrophils (black arrows), macrophages containing *Leishmania* species amastigotes (arrowhead) and some lymphocytes (red arrow) in biopsy material from the lingual lesions. May-Grünwald-Giemsa Quick. x 100

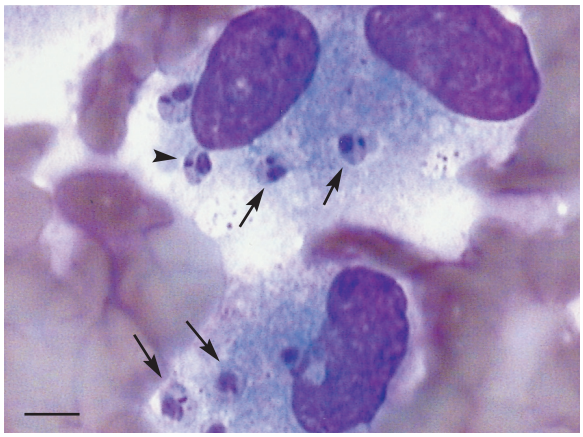

FIG 3: Macrophages containing numerous *Leishmania* species amastigotes (arrows) in biopsy material from the lingual lesions. Kinetoplasts (arrowhead) of parasites are evident. May-Grünwald-Giemsa Quick. x 1000

Cortene; Lepetit) (1 mg/kg) was started. Four days after glucocorticoid therapy, both eyelid and lingual lesions disappeared (Fig 4). A new antimonial therapy was started at the same dosages as before. Two weeks after beginning treatment, the dog's clinical condition worsened and it developed vomiting, weight loss and polyuria/polydipsia. Laboratory findings showed moderate normocytic, normochromic, hyporegenerative anaemia and severe increases of blood urea nitrogen (176 mg/dl) and creatinine (3·35 mg/dl). Cytological examination of a bone marrow aspirate showed erythroid hypoplasia (myeloid:erythroid ratio 3·86) and the presence of numerous *Leishmania* species amastigotes. Six months later the dog died.

Oral *Leishmania*-positive granulomatous lesions, particularly on the tongue, are unusual in dogs. To the authors' knowledge, only one case has been described in the veterinary literature (Lamothe and Poujade 2002); concerning nodular ulcerated lesions in a dog. It was suggested that the lesions were a probable consequence of systemic dissemination of parasites. In human beings, oral lesions caused by *L infantum* are frequently seen in severely immunosuppressed patients, such as individuals coinfected with HIV or graft recipients (Aliaga and others 2003). Borzoni and others (1991) described a 60-year-old immunocompetent patient affected by leishmaniasis who showed a single lingual nodule and several palatine nodular lesions. The patient was not affected by other diseases. The absence of clinical, parasitological and immunological signs of generalised leishmaniasis led to the conclusion that the lesions originated at the site of parasite entrance, probably after the patient had crushed an infected sandfly.

In the present case the macroscopic aspect of the lingual and eyelid lesions, and some peculiar microscopic features common to both (the presence of numerous neutrophil granulocytes and a high parasite load), suggest that the lesions could have been of recent origin. The time of appearance (early October) is also coincident with the period of highest rate of natural *L infantum* infections of sandflies in southern Italy. As with the case in a human being described by Borzoni and others (1991), the lingual lesions could represent entrance sites of parasites after ingestion or crushing of infected sandflies. A clinical feature of the oral lesions described in human beings is their persistence (as long as one year before the correct diagnosis of leishmaniasis), the specific drugs (pentavalent antimony and amphotericin B) and the long duration of therapy (40 to 60 days) required for clinical improvement. On the contrary, in this case the clinical remission of lesions was fast (three to four days) and associated only with glucocorticoid administration.

It was therefore believed that the clinical signs observed in this dog could have originated from distinct events: uveitis and renal damage resulting from the previous chronic infection and localised mucosal lesions from a new infection. The re-infection of dogs that had had previous contact with *Leishmania* species is assumed to be possible, as shown by experimental work (Santos Gomes and others 2003). However, dogs that have developed an asymptomatic 'resistant' condition following first infection do not develop signs of disease at re-infection. In the dog in the case described here, the new entrance of parasites could have been favoured by the anergic clinical response as demonstrated by the progressive evolution of the disease and the poor response to treatment. However, the possibility that the lingual lesions resulted from the reactivation and dissemination of previous infection cannot be excluded.

In conclusion, it is suggested that the unusual glossitis described in this case was not a mucocutaneous form of leishmaniosis, never before described in dogs, or a dissemination from a visceral form, but a localised mucosal leishmaniosis resulting from a new infection.

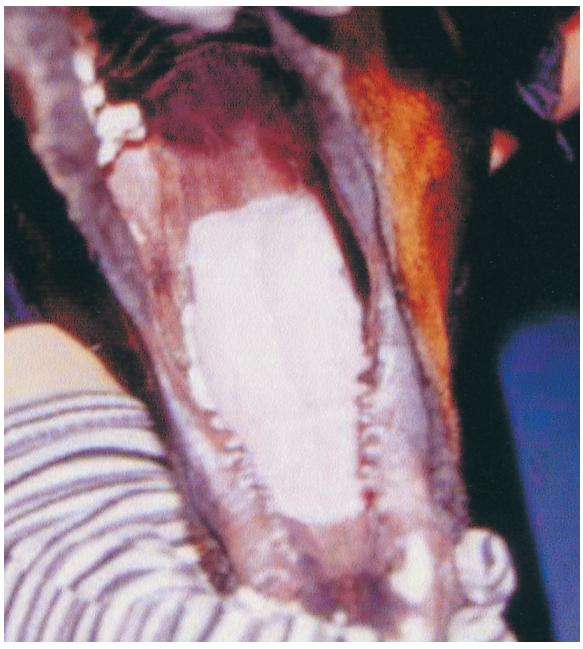

FIG 4: Total clinical resolution of the tongue lesions after treatment with a combination of meglumine antimoniate, allopurinol and doxycyline

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