RESEARCH COMMUNICATION

Canine visceral leishmaniosis: first case in Zambia

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Visceral leishmaniosis was discovered in a male 12-year-old Australian cattle dog in September 1994. Canine leishmaniosis has not previously been reported in Zambia. At necropsy, splenomegaly, fatty degeneration of the liver and focal lesions in the renal cortex were observed. Histopathologically, focal diffuse proliferation of amastigote-laden macrophages (ALM) were found in the spleen and liver. Amastigotes were diffusely distributed in the pulmonary alveolar wall, and formed minute lesions in the adrenal cortex. Focal degenerative interstitial nephritis and myocarditis were observed, but ALM were hardly found in these lesions. The animal had lived in Lusaka for the previous 2 years at least, where it was likely to have had a *Leishmania* parasitaemia. Although the ecology of sandflies in Zambia is still veiled, the present case of canine leishmaniosis could be an indication of widespread leishmaniosis in this coutry, not only in dogs, but also in humans, particularly as human immunodeficiency virus (HIV) infection is prevalent in the country.

The subject was a male 12-year-old Australian cattle dog brought to the Veterinary Teaching Clinic at the University of Zambia on 5 September 1994, with a 5-d history of anorexia, polydipsia and longstanding ocular discharge. The dog was suspected to have been poisoned with horticultural chemicals. The animal had been kept in Lusaka for at least the previous 2 years, but no previous history was available.

Necropsy was performed within 2 h of death. Specimens from the spleen, liver, kidney, heart, lung, small intestine, pancreas, urinary bladder, prostate, testis, adrenal gland, thyroid and brain were taken and fixed in 10% buffered formalin, thin-sectioned after routine processing, and stained with haematoxylin and eosin (H & E).

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Physical examination of the dog revealed tachycardia, weak pulse, icterus and splenomegaly. Haematological data were as follows:

Packed-cell volume	29 %
Red-blood cells	3,7 x 10^6/µℓ
White-blood cells	14,6 x 10^3/µℓ
Neutrophils	13,090/µℓ
Lymphocytes	1,095/µℓ
Eosinophils	131 /µℓ
Monocytes	263 /µℓ
Basophils	29 /µℓ
Haemoglobin	9,2 g/dℓ
Total protein	8,0 g/dℓ
Serum protein	6,5 g/dℓ
Aspartate aminotransferase (AST)	39,7 [normal value (n.v.) quoted by Bistner & Ford 1995] 5–80] IU/ℓ
Alanine aminotransferase (ALT)	43,1 (n.v. 5–25) IU/ℓ
Alkaline phosphatase (ALP)	379,5 (n.v. 20–120) IU/ℓ
Gamma-glutamyltransferase (GGT)	13,9 (n.v. 1,4–11,5) IU/ℓ
Blood-urea nitrogen (BUN)	55,7 (n.v. 10–22) mg/dℓ
Creatinine	5,1 (n.v. 0,4–1,5) mg/dℓ

Body mass was 20 kg. Mild jaundice was observed. The spleen was swollen (30 x 5 x 2 cm) and the parenchyma was pulpy-hyperplastic and unevenly congested. The liver had a mass of 900 g and was light yellowish brown. Many irregular, whitish focal lesions were present in the kidney cortex. The heart was dilated and round in shape. The aorta had several hollowed scars in the intima caused by migration of *Spirocerca lupi*. The right testicle was atrophied with a thick capsule, while the left showed nodular hyperplasia.

The spleen was congested unevenly and ALM were scattered diffusely or as aggregates throughout the parenchyma (Fig. 1). The ALM were ovoidly swollen, and contained about 20 or more amastigotes. The average amastigote size was 3,0 x 2,4 µm, and had an eccentrically located, ovoid nucleus and a structure corresponding with that of a kinetoplast. Walls of some central arterioles were thickened with hyalin. ALM of various sizes were scattered diffusely or

as aggregates throughout the liver, accompanied by slight lymphocytic infiltration. In the heart, necrotizing inflammations with marked infiltration of lymphocytes and monocytes were observed. Some intramyocardial arterioles were thickened with hyalin. In the lung, amastigotes were spread diffusely or within macrophages in the alveolar walls (Fig. 2). In the kidney, the lesions observed macroscopically, were focal aggregates of degenerated monocytic cells in the interstitium, whereas ALM were hardly found. In the adrenal gland, minute degenerating lesions were found in the zona glomerulosa, accompanied by ALM and free amastigotes released from ruptured ALM. A few lymphocytes infiltrated into the leptomeninx. Fibrous thickening of arterial walls and demyelination were noted in the cerebellar white matter. The testes were atrophied and aspermatogenetic.

The amastigotes were round or ovoid in shape, and the size, 3,0 x 2,4 µm, ranged between the previously reported 2,5–5,0 x 1,5–2,0 µm (Slappendel 1988) or 2,4–3,1 x 1,5–2,6 µm (George, Nielsen, Shively, Hopek & Mroz 1976). The nucleus was round and located eccentrically, and a kinetoplast was observed in the cytoplasm. While the ALM in the present case contained about 20 or more amastigotes in the section (Fig. 1), George *et al.* (1976) reported that 25–30 amastigotes were found in macrophages, and Simpson, Harvey & French (1982) reported their finding of at least ten amastigotes per macrophage in the bone marrow.

Visceral leishmaniosis in the Old World is caused by parasites of *L. donovani* complex (*L. donovani*, *L. infantum* and *L. chagasi*) (WHO 1990). In Africa, endemic areas of visceral leishmaniosis are located in northern and eastern Africa (WHO 1984, 1990). In Zambia, only two cases of human leishmaniosis have been reported; the first was in Southern Province, by Hira, Naike & Egere (1973), and the other in 1976 (WHO 1990). The disease has been found in adjoining countries such as Tanzania, Namibia, Malawi, Zaire and Zimbabwe (WHO 1990). In South Africa, cutaneous leishmaniosis was diagnosed in sheep in 1989 and 1990, and the visceral form was diagnosed in two dogs, one in Durban, in 1964 (of which the life history was uncertain), and the other (which had never left the country) in the Orange Free State, in 1987 (Van der Lugt & Stewart 1994).

Because a full history was not available for the present case, it is not known where the animal had become infected. Even if the dog had been infected in another country, it had been kept in Zambia for at least 2 years, where it could have been a parasitaemic carrier. The acute onset of disease in this case was suspected to have been caused by poisoning which resulted in hepatic fatty degeneration. Ferrer (1992) mentioned that elevation of ALT and AST could occur in canine visceral leishmaniosis. Elevations of AST, ALT, ALP and GGT in the present case, were probably related to both poisoning and hepatic *Leishmania* infection. Increases of BUN and creatinine were apparently related to the renal lesions (Ferrer 1992).

In the present case, the dog was 12 years old, and in the histological findings there were apparently senile changes such as testicular atrophy and fibrous thickening of the arterial walls in the brain. Hyaline degeneration in the spleen and heart might be amyloid degeneration, as described by George *et al.*

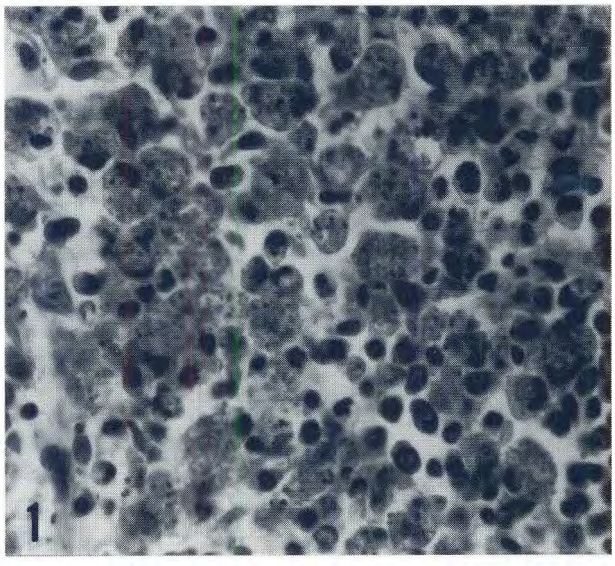

FIG. 1 Amastigote-laden macrophages are gathered in the red pulp of the spleen

x 600, H & E stain

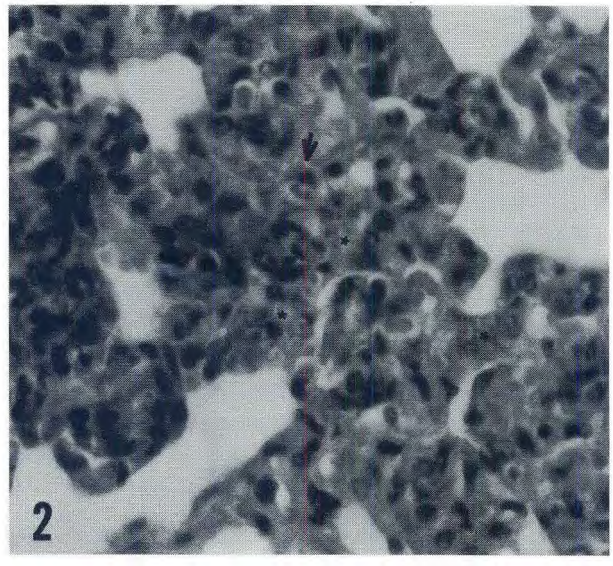

FIG. 2 Amastigotes are observed in macrophage (arrow) or diffusely (around star marks) in the pulmonary alveolar walls

x 600, H & E stain

(1976), regarding visceral leishmaniosis. Mild lymphocytic infiltration in the leptomeninx might be related to generalized leishmaniosis, while the demyelination in the cerebellar white substance would be a senile change.

Ecology of sandflies, vectors of leishmaniosis in Zambia, is not described in the textbook of Ashford & Bettini (1987) nor by Lewis & Ward (1987). *Leishmania* is transmissible by other blood-sucking arthropods or by direct contact (Longstaffe, Jefferies, Kelly, Bedford, Herrtage & Darke 1983; Huss & Ettinger 1992; Slappendel 1988). Leishmaniosis is known to be an opportunistic infection, and is prevalent among HIV-infected patients with acquired immune deficiency syndrome (AIDS) (Dedet, Lambert & Pratlong 1995; Baily & Nandy 1994). Numazaki, Luo & Suzuki (1994, 1995, 1996) reported that the HIV-positive rate in Zambia was high:

Patients in the University Teaching Hospital (UTH)	54,0 %
Mothers of children in the urban community of Lusaka	22,3 %
Children admitted to UTH	43,6 %

The occurrence of canine visceral leishmaniosis could indicate a risk of canine and human leishmaniosis spreading in Zambia.

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