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Canine Visceral Leishmaniasis in a Doberman

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ABSTRACT

Canine visceral leishmaniasis was diagnosed in a six years old male dog presented with progressive weight loss, desquamative dermatitis, anemia, hepatosplenomegaly, lymphadenopathy, conjunctivitis and pale mucus membrane. Biopsy of lymph nodes and tissue staining with H&E and Giemsa revealed canine visceral leishmaniasis. To confirm the diagnosis, direct agglutination test showed 1:20,480 titer, serum electrophoresis demonstrated hyperproteinemia, hypergammaglobulinemia (6.7 g/dl) and hypoalbominemia (1.37 g/dl) and A/G was 0.15. After 7 days of cultivation in NNN plus LIT media, the promastigote form of parasite was observed. At necropsy findings, the liver and spleen were swollen and hyperemic which Leishman bodies were shown into the cytoplasm of kupffer cells. The kidneys were swollen and there were acute interstitial nephritis, membranoproliferative glomerulonephritis and so amyloidosis. Afterwards in a serological survey, three cases of seropositive dogs were found. Finally the specimens were sent to Tropical Disease and Hygiene Section in London School, and *Leishmania infantum* was specified.

INTRODUCTION

Canine visceral leishmaniasis (CVL) is a disease caused by flagellated dimorphic protozoan of the genus *Leishmania* (Huss and Ettinger 1992). The disease is endemic in the Mediterranean area and other parts of the world that is transmitted via the bite of the subfamily phlebotominae (Font et al. 1993). CVL is also a zoonosis in which the dog is considered to be the source of the parasite, but it can be infect the man, rodents and other canidae (Huss and Ettinger 1992; Font

et al. 1993). CVL generally is caused by the species *L. infantum* (Guy et al. 1993; Rodriguez et al. 1996). Signs of CVL include desquamative or ulcerative dermatitis, recurrent fever, anterior uveitis, conjunctivitis, hepatosplenomegaly, lymphadenopathy, loss of weight, anorexia, epistaxis, lameness, renal and hepatic insufficiency, hyperglobulinemia, anemia, thrombocytopenia, leukopenia with relative monocytosis, diarrhea and cachexia. As many as 90% of untreated patients die as result of secondary infections or bleeding disorders. Diagnosis is possible by direct observation of Leishman body in smears of bone marrow, lymph node or via skin biopsy in cases of cutaneous presentation (Font et al. 1993a; 1993b; Huss and Ettinger 1992).

CASE REPORT

A six year old male Doberman was referred to small animal hospital of veterinary faculty of Tehran University with a history of progressive weight loss, desquamative or ulcerative and alopecic dermatitis, anemia, hepatosplenomegaly, lymphadenopathy, conjunctivitis and pale mucus membrane. Results from biochemical, hematological and electrophoretic analysis of blood showed anemia (RBC=4.1 ± 10^6, Hb=10.8, HCT=27.8). Findings of urine analysis indicated proteinuria, high creatinine (0/8) and uremia (49) (Fig. 1, Table 1).

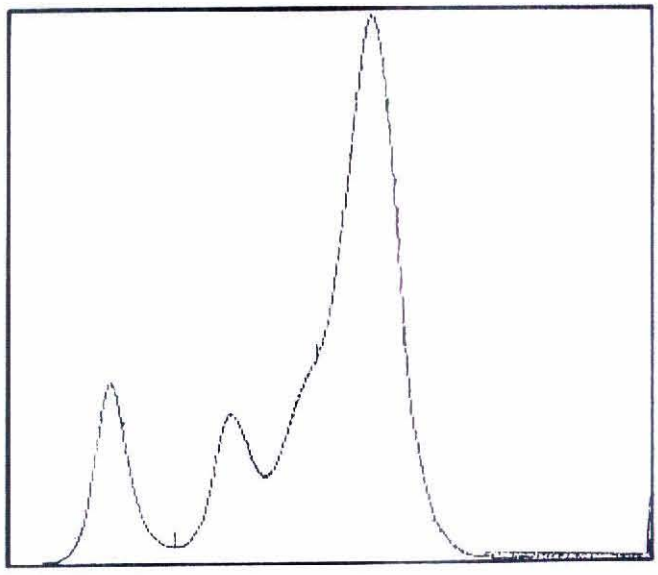

Figure 1. Hypoalbominemia and hypergammaglobulinemia. The amount of albumin was 1.37 g/dl, gammaglobulin was 6.7 g/dl, and A/G was 0.15. Normal level of albumin is 2.2-3.5 g/dl, gammaglobulin is 0.5-1.8, and A/G is 0.7-1.1.

Table 1. Biochemical and hematological analysis.

Biochemistry		Hematology	
Test	Result	Test	Result
SGOT (AST)	38	WBC	13,900
SGPT (ALT)	44	RBC	4,130,000
ALK. PHOS	1,127	HGB	10.8
CREAT.	0.8	HCT	27.8
UR. AC	0.2	MCV	0.67
UREA	49	MCH	25.8
GLUC.	89	MCHC	38.5
CHOL.	200	POLY	68%
		LYMPH.	29%
		MONO.	1%
		EOS.	2%

Leukocyte, RBC and epithelial cells were in urine. With failure of symptomatic treatment, biopsy of prescapular lymph node was performed. The tissue sections were stained with H&E and Leishman bodies were seen in cytoplasm of macrophages. Then, the parasite with Giemsa staining was observed. To confirm the diagnosis, serum analysis with direct agglutination (DA) test revealed 1:20,480 titer. Also aspiration of bone marrow and lymph node showed an abundance of parasites of *Leishmania* (Figs. 2 and 3).

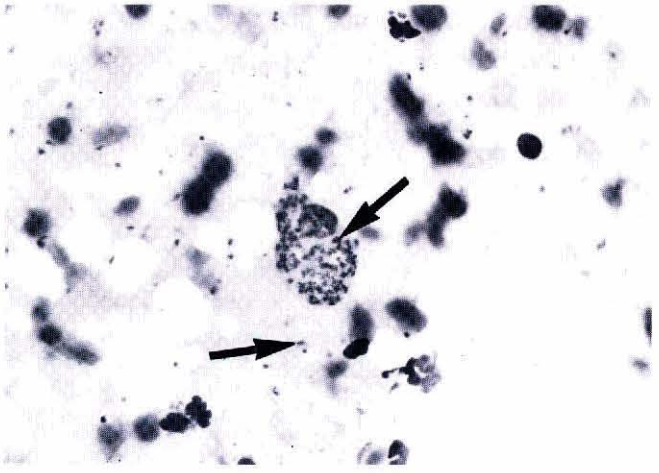

Figure 2. Numerous leishmanial amastigotes in the cytoplasm of macrophages staining with Giemsa solution.

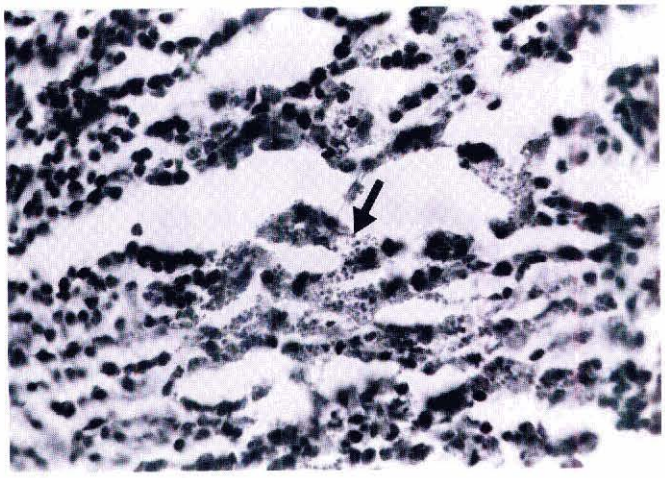

Figure 3. Intracytoplasmic protozoa organisms (arrow) were seen into macrophages derived from lymph node (H&E staining).

After 7 days of cultivation in NNN plus LIT media, the promastigote form of parasites were observed. Also the specimens were sent to Tropical Disease & Hygiene Section in London School and Leishmania infantum was specified.

At necropsy findings, exfoliation and desquamative dermatitis with infiltration of lymphocytes and plasma cells were noticed, and there was adhesion of derma and hypodermic to muscles by exudate. The liver was swollen and hyperemic which Leishman bodies were observed into the cytoplasm of kupffer cells. Spleen was swollen and proliferation of macrophages, lymphocytes and plasma cells were obvious. The kidneys were swollen and there acute interstitial nephritis, membranoproliferative glomerulonephritis and amyloidosis.

Prescapular, precrural and mesenteric lymph nodes were swollen and the parasites were frequent within the cytoplasm of macrophages. There were conjunctivitis with exudation and lacrimation, lymphoplasmacytic enteritis and so DIC in vessels of intestine, brain, heart and other tissues.

DISCUSSION

Two subjects were important in present case. The first was incidence of CVL. The second was wrong clinical diagnosis of lymphosarcoma, which tissue staining differentiated CVL from other diseases. It is important to know which leishmaniasis must be differentiate from lymphosarcoma, multiple myeloma, systemic lupus erythematosus and systemic mycosis. Bone marrow biopsy is very useful for differential diagnosis (Font et al. 1993a; 1993b). Afterwards, in a serological survey three cases of seropositive dogs were found. Pathologically in CVL, normal tissue replaces by extensive proliferation of reticuloendothelial cells, especially in the spleen, liver, lymph nodes, bone marrow and GI tract (Huss and

Ettinger 1992).

Leukopenia and anemia are outcome of this process (Font et al. 1993a; 1993b; Huss and Ettinger 1992). *Leishmania* species avoid immune system responses and a direct *Leishmania* antigen specific immunosuppression as measured by T cell blastogenesis and lack of IL-2 or gamma-interferon production has been proven (Huss and Ettinger 1992). Renal lesions were noticable with membranoproliferative glomerulonephritis and acute diffuse interstitial nephritis with amyloidosis which were due to antigen-antibody complex (Font et al. 1993a; Nieto et al. 1992).

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