

OCCURRENCE OF URAEMIA WITH CONCURRENT INFECTION OF HEPATOZOONOSIS IN DOG-CASE REPORT

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Introduction

Renal failure in dog is generally represented by failure to carry out normal metabolic and endocrine function. Renal failure result in to accumulation non-protein nitrogenous substances viz. urea, creatinine, uric acid, ammonia, etc. in blood which result in to is called as uremia. Hence, uraemia is a toxic syndrome associated with multicystic clinical signs and lesions due to renal failure (Confer and Panciera, 2001). Uraemia occurs due to loss of functional renal tissue and is usually progressive process which impairs the proper functioning of the kidney resulting into absorbance of waste materials in to blood which normally should form the urine (Shastry, 1983). The disease may occur in animals of all ages but is much more common in old age (Anonymous, 2008). Hepatozoonosis is a tick-borne protozoan disease of dog and other carnivores, caused by *Hepatozoon canis* (*H. canis*) (Craig 1990; Baneth 2001). Hepatozoonosis in dogs usually occur as an asymptomatic to mild disease, and is associated with a low level of *H. canis* infecting 1 to 5 per cent of the circulating leucocytes (Baneth and Weigler, 1997; Baneth *et al.*, 2003). Present paper put on record the occurrence of uremia with concurrent infection of *H. Canis* infection in Pomeranian dog.

Materials and Methods

A fifteen year old Pomeranian male dog was presented to Teaching Veterinary Clinical Complex (TVCC), Bombay Veterinary College (BVC), Goregaon, Mumbai for treatment. Laboratory investigation viz. serum biochemical analysis for kidney function test (urea, BUN-blood urea nitrogen and creatinine) and liver function test (SGOT and SGPT) was done at TVCC, BVC, Mumbai using Automatic analyser. In addition, complete blood count viz. haemoglobin (Hb), packed cell volume (PCV), total erythrocyte count (TEC), erythrocyte sedimentation rate (ESR), total leucocyte count (TLC), and differential leucocyte count (DLC)

was carried out using Haemo-autoanalyser at Department of Pathology, BVC, Mumbai. Detailed examination of dog was carried out and revealed numerous ticks on the body. The ticks were collected for confirmation.

Result and Discussion

Clinically, dog showed fever, polydipsia, polyuria, dehydration, vomition, lethargy, oral ulceration, rough skin coat and diarrhoea. Serum urea, BUN and creatinine was 212.85 (mg/dl), 99.46 (mg/dl) and 7.63 (mg/dl), respectively. From clinical signs such as vomition, ulceration in mouth and elevated serum levels of BUN and creatinine, the condition was diagnosed as uraemia. The clinical sign observed in the present investigation are in accordance with observation of earlier researchers (Anonymous, 2008; Shastry, 1983). The clinical signs such as polydipsia and polyuria attributable to renal dysfunction are which are not observed until the function of approximately two-thirds of the nephrons has been impaired (Anonymous, 2008). Elevated serum levels of BUN and creatinine indicates kidney damage (Benjamin, 2001). The liver function test parameters (SGOT and SGPT) were within normal physiological range.

CBC finding revealed decreased haemoglobin (7.0 gm %), packed cell volume (22.8%) and total erythrocyte count (3.41×10^6 / cu. mm). Erythrocytic indices revealed normocytic hypochromic anaemia. The occurrence of anaemia due to nephritis has been documented (Anonymous, 2008; Shastry, 1983). Kidneys produce hormones, namely erythropoietin, which stimulates the bone marrow to produce new red blood cells. Uraemia occurs when more than 70 per cent of kidney parenchyma is damaged (Benjamin, 2001). Renal damage results in to impaired production and release of erythropoietin, thus causing anaemia (Anonymous, 2008). The erythrocyte sedimentation rate (ESR) was increased (47mm/hr). Increased ESR has been

suggested in chronic (interstitial) nephritis (Benjamin, 2001) and has been of significance in generalized inflammatory condition as there was renal damage with stomatitis and possibly may be stomach and intestine as there was diarrhoea.

TLC analysis revealed leucocytosis (22.8×10^3 / cu. mm). Differential leucocytes count revealed neutrophilia (82%) and were hyper segmented indicating neutrophilia with shift to right. The other leucocytes were within normal range (L-10; M-05; E-03, B-00). The Leishman's stained blood smear revealed cigar shaped, pale-staining cytoplasmic bodies (gamonts) in the neutrophils (Fig. 1). These

bodies were identified as gametocytes of *H. canis* based on their morphological characteristics (Soulsby, 1982). The parasitemia of leucocytes was 4-5 per cent. Similarly, Ingole *et al.* (2011) reported 7.25 and 2 per cent parasitemia in two cases of hepatozoon *canis* infection, respectively. Rajamanickan *et al.* (1985) recorded 1-5 per cent parasitemia in canine hepatozoonosis, which is in support of our observation. The ticks from the body of dog were identified as *Rhipicephalus sanguineus*. Platelets count was 1.22 lakh/ cu. mm and were reduced on smear. Thrombocytopenia in canine hepatozoonosis has been reported (Ingole *et al.*, 2011).

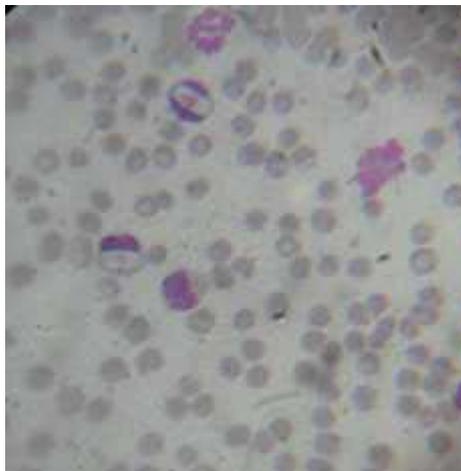


Fig. 01: Leishman stained blood smear showing gamonts in leucocyte

The dog was treated with Metranidazol orally (7-10@ kg b. wt), DNS (150 ml, I/V), and Ampoxime @ 10 mg/ kg b. Wt for three days. Clindamycin @10 mg/ kg b. wt. (I/M, twice daily for 14 days was suggested for treatment of *H. canis* infection. A supportive therapy of haematinic (Imferon) @ 1 ml twice a week for 15 days was suggested orally for recovery of anaemia. However, after three days of treatment, owner did not turn up.

Conclusion

A case of uraemia with concurrent infection of *H. canis* infection was diagnosed in Pomeranian dog on the basis of kidney function test and finding cigar shaped, pale-staining cytoplasmic bodies (gamonts) in the neutrophils of Leishman stained blood smear. Its detail clinical pathology with its therapeutic management is discussed.

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