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Short Communication

HISTOPATHOLOGIAL ALTERATIONS IN *BABESIA GIBSONI* INFECTION- A CASE REPORT

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ABSTRACT: An eight-year-old male Labrador with high fever was brought for treatment in the Veterinary Clinical Complex, Faculty of Veterinary and Animal Sciences, WBUAFS, Kolkata. *Babesia gibsoni* was identified in microscopical examination of the blood smear taken from the animal. The dog could not survive despite treatment and the post mortem examination revealed remarkable changes in the liver, lungs, kidneys and spleen. Histopathological examination showed congestion and edema in lungs, liver and spleen along with remarkable necrotic and degenerative changes in the tubular epithelial cell of kidneys.

Key words: *Babesia gibsoni*, Haemoglobin, Alkaline phosphatase, Histopathology.

Canine babesiosis is caused by tick- transmitted apicomplexan parasites of Babesia spp., which parasitize erythrocytes. Based on host specificity and morphology of the intra-erythrocytic forms, canine piroplasms have been originally recognized belonging to two distinct species, the large *Babesia canis* and the small *Babesia gibsoni* (Alvarado-Rybak *et al.* 2016). Clinical signs exhibited by the affected animals are letharginess, anorexia, pale mucous membranes, vomition, brown urine, jaundice, splenomegaly, weight loss, tachycardia, tachypnea and lymph node enlargement. Sometimes involvement of kidney and liver causes organ dysfunction/ failure (Jacobson 2006, Sivajothi *et al.* 2014).

In complicated form, some cases show cerebral form of babesiosis, coagulopathy, immune-mediated haemolytic anaemia (IMHA), acute renal failure, hepatopathy and/ or icterus (Koster *et al.* 2015). The present case was investigated to see the account of biochemical functions as well as involvement of various organs and their histo-structure.

Case history and observations

An eight years old male Labrador dog weighting 35 kg was presented in the Veterinary Clinical Complex, Faculty of Veterinary and Animal Sciences, Kolkata, West Bengal. The dog was suffering from high temperature (105 ºC), heavy tick infestation, respiratory distress and visible pale mucous membranes of eyes. Following clinical examination, blood sample was collected to rule out haemo-protozoa as well as for biochemistry.

The results of Giemsa stained thin blood smear examination revealed that presence of *Babesia gibsoni* infection in the erythrocytes. The serum biochemistry was performed as per standard method by semi auto-analyser (Table 1). The biochemical profile revealed increased levels of blood urea nitrogen (BUN), creatinine, aspartate aminotransferase (AST), bilirubin and alkaline phosphatase (ALP) whereas levels of alanine aminotransferase (ALT) and total protein recorded a fall. Furthermore, hematological profiling (Table 2) revealed fall in the levels of hemoglobin (Hb), total erythrocyte count (TEC) and packed cell volume (PCV). Significant ($p<0.05$) elevations in the levels of bilirubin, ALP, BUN and creatinine has been observed in both *B. gibsoni* and *B. canis* infected dogs along with a significant ($p<0.01$) decrease in values of Hb, TEC and PCV, in *Babesia gibsoni* infection (Gonde *et al*. 2016).

However, during the course of treatment, the dog could

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Table 1. The serum bio-chemical profile of the dog.

Sl. No.	Bio-chemical profile	Values	Reference range (Blood *et al.* 2007)
1.	BUN (Blood Urea Nitrogen)	58 mg/dl	(7-25) mg/dl
2.	Creatinine	3.1 mg/dl	(0.5-1.5) mg/dl
3.	ALT (Alanine Aminotransferase)/SGPT	27 IU/L	17-69 IU/L
4.	AST (Aspertate Aminotransferase)/SGOT	140 IU/L	12-37 IU/L
5.	TP (Total Protein)	3.4 mg/dl	(5.2-7.56) mg/dl
6.	Bilurubin	0.92 mg/dl	(0.01-0.5) mg/dl
7.	Alkaline Phosphatase (ALP)	965 IU/L	10-80 IU/L

Table 2. The Hematological profile of the dog.

Sl. No.	Hematological parameter	Values	Reference range (Blood *et al.* 2007)
1.	Hemoglobin (Hb)	6.6 gm/dl	12-18 gm/dl
2.	Red blood cell (RBC)	2.4×10^6 g/L	(6-9 10^6) g/L
3.	Packed cell volume (PCV)	16 %	(37-55) %
4.	Mean corpuscular volume (MCV)	66 fL	(60-75) fL
5.	Mean Corpuscular Hemoglobin (MCH)	27 pg	(20-28) pg
6	Mean Corpuscular Hemoglobin Concentration (MCHC)	41 %	(32-36) %

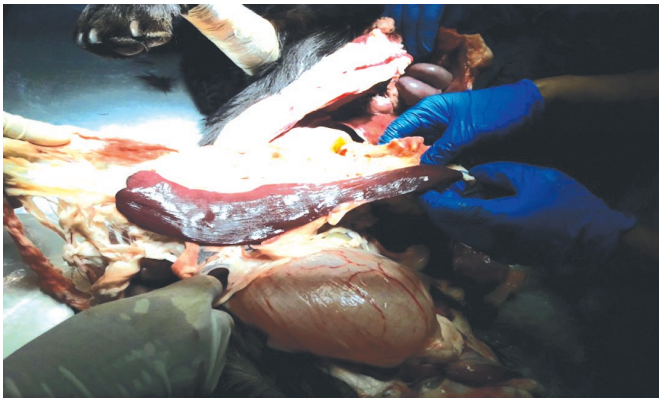

Fig. 1. Enlarged spleen after postmortem.

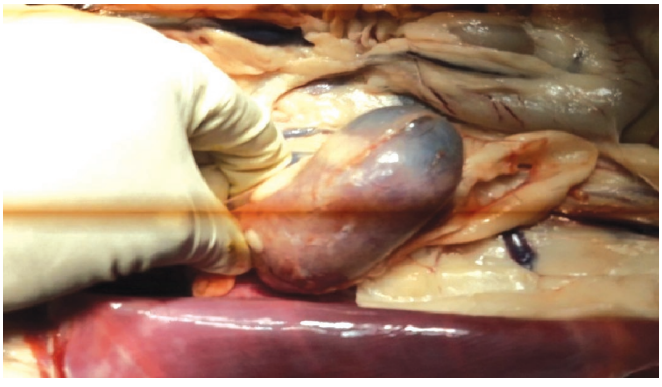

Fig. 2. Enlarged kidney.

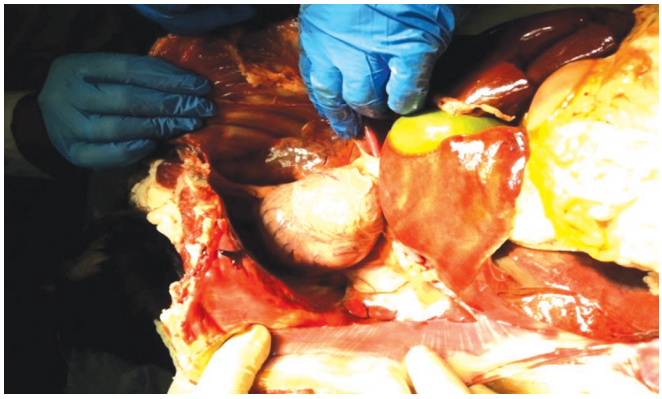

Fig. 3. Enlarged liver seen after post-mortem.

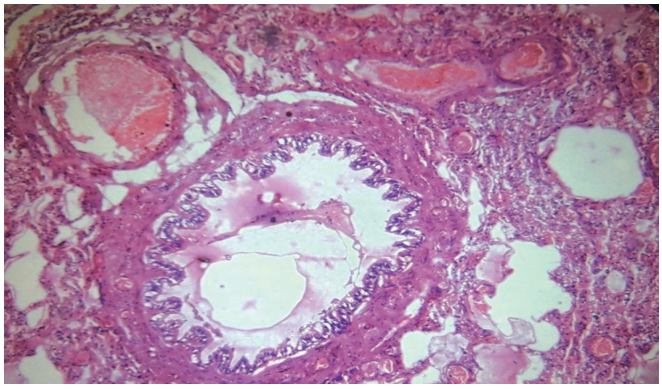

Fig. 4. The microphotograph of lungs (H & E 100X).

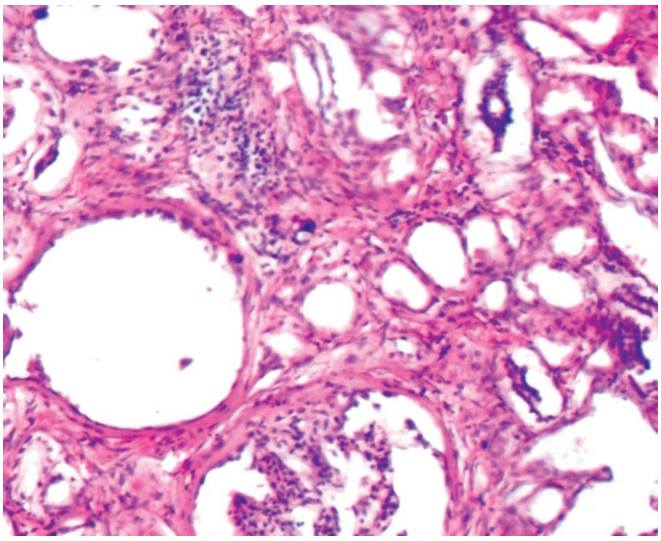

Fig. 5. The microphotograph of kidney (H & E 400X).

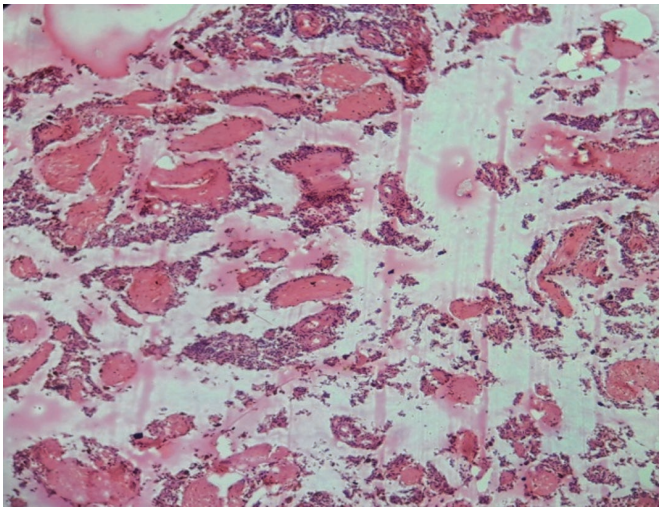

Fig. 6. The microphotograph of spleen (H & E 100X).

not survive and the post mortem examination was carried out. In the post mortem examination, animal showed gross signs of enteritis and splenomegaly (Fig. 1), kidney enlargement (Fig. 2) and hepatomegaly (Fig. 3). Pulmonary edema, kidney damage, hepatomegaly splenomegaly and inflammation of intestine with patches of hemorrhages in the mesentery and cardiac musculature have reported previously in *B. gibsoni* infection (Ubah *et al*. 2019).

The tissue samples of liver, lungs, spleen and kidney were collected and processed for histopathology by standard techniques. Histopathological examination of the lungs showed congestion and edema in the lung parenchyma (Fig. 4). Microscopic changes observed in kidney were focal interstitial hemorrhage, atrophy of glomerular tuft even focal dissolution of endothelial

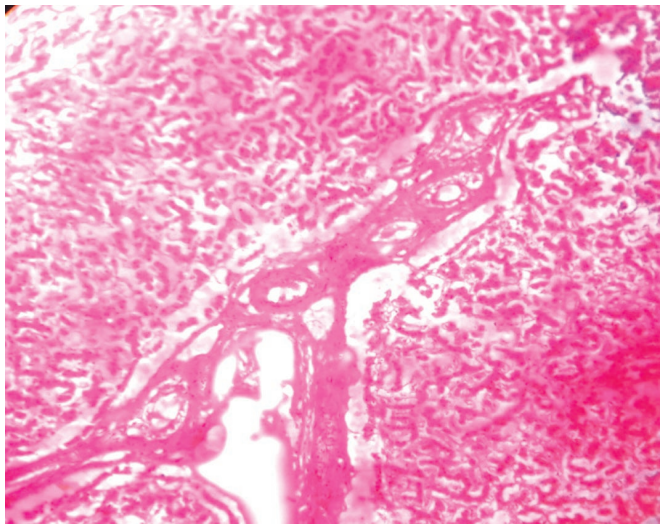

Fig. 7. The microphotograph of liver (H &E 100X).

network (Fig. 5). Necrotic and degenerative changes in tubular epithelial cell, fibrosis in the interstitium and focal desquamation of tubular epithelium from basement membrane were also recorded. There was marked depletion of lymphocyte along with proliferation of trabiculae and edema in spleen (Fig. 6). Disarrangement of hepatic chord along with lysis of hepatocytes was observed and there were periportal fibrosis in liver (Fig. 7). These histopathological changes were in corroboration with a study where the lungs showed enlarged and edematous alveoli, which were mostly fused with the presence of marked inter-septal cellular infiltration (Ubah *et al*. 2019). Vacular-hydropic degeneration and necrosis were also observed in the glomerular and interstitial capillary endothelium along with necrosis occurred in renal tubular epithelial (Mathe *et al*. 2007).

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