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Medical management of *Babesia gibsoni* induced hepatopathy and acute renal injury in a dog

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Abstract

Canine babesiosis is a complex disease which may cause dysfunction of more than one vital organs of body, known as multi organ dysfunction syndrome (MODS). History and clinical examination of a six year old Labrador dog revealed high grade fever intermittently for7-8 days, anorexia, congested mucous membrane, increased pulse and respiration rates. Laboratory examination for haematobiochemical parameters revealed anaemia, eosinophilia, increased activity of hepatic enzymes, blood urea nitrogen and creatinine. Thin blood smear examination revealed *Babesia gibsoni*. The dog was diagnosed as suffering from *B. gibsoni* induced dysfunction of liver and kidney and successfully managed with combination therapy consisting of clindamycin, metronidazole and doxycycline for 21 days. The animal recovered uneventfully following therapy.

Keywords: *Babesia gibsoni,* Hepatopathy, Labrador dog, Doxycycline.

Introduction

Babesia gibsoni is a small form *Babesia,* an of apicomplexan, intraerythrocytic parasite of canines causing canine babesiosis characterised by fever, inappetence to anorexia, normocytic normochromic anaemia and apathy with the history of tick infestation on body (Terao *et al.,* 2015). Disease is manifested in two forms, uncomplicated and complicated (Jacobson and Clark, 1994). Clinical signs of uncomplicated babesiosis are generally related to acute haemolysis such as fever, anorexia, depression, pale mucous membrane, whereas complicated babesiosis is the result of systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS), which is manifested by acute renal failure, hepatopathy, pancreatitis, cerebral babesiosis, coagulopathy, immune mediated haemolytic anaemia (IMHA), acute respiratory distress syndrome (ARDS) and shock (Kules *et al*., 2016). Because of variability of complications of clinical syndrome in canine babesiosis, prognosis and therapeutic management differs accordingly. The present article describes successful management of babesiosis in a dog.

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Materials and Methods

A male Labrador retriever dog aged 6 years and weighing 20 kg body weight was presented to the Referral Veterinary Polyclinic-Indian Veterinary Research Institute, Izatnagar, Bareilly with the history of weakness, anorexia, fever, vomiting, oliguria and tick infestation. Clinical examination of the patient revealed rectal temperature (104.9ºF), increased respiration rate (52/ min), increased pulse rate (96/ min), congested mucous membrane, dehydration and abdominal pain. The condition was tentatively diagnosed as fever of unknown origin and treated with symptomatic and supportive therapy (Normal saline and Paracetamol @ 10 mg/kg BW, IM). Whole blood and serum samples were collected for haematological and serum biochemical studies, respectively. The blood smear was examined to rule out haemoprotozoal infection.

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Treatment of *B. gibsoni* was started with combination therapy consisting of clindamycin (Dalacin C, Pfizer @ 25 mg/kg BW, IV, BID), metronidazole (Baxter health care @ 15 mg/kg BW, IV, BID) and doxycycline (Dr. Reddy's Laboratories @ 5 mg/kg BW, PO, BID). Fiprofort Plus (Fipronil + S-methoprene) spot on was applied for ticks management. Fluid therapy consisted of dextrose normal saline (@10ml /kg BW, IV, BID) and supportive therapy included silymarin (70 mg PO, BID). After assessment of electrolytes fluid balance, fluid therapy was started with dextrose 25% @ 75 ml, IV, BID to provide energy and fluid low in sodium chloride NS 0.45% @ 500 ml IV BID. Pantoprazole 40mg IV, Ondansetron @ 0.2 mg/kg BW IV, Neohepatex @ 2 ml IM and Frusemide (Lasix(TM), Sanofi Aventis) @ 4 mg/ kg BW IV, BID was given for management of gastric, hepatic and renal damage for 3 days. From day 14th onwards, commercially available renal diet was started and animal was shifted on oral medication with tablets of clindamycin, metronidazole, doxycycline and Lasilactone @ 1 Tab. PO, BID, Pentocid 40 mg 1 Tab. PO, BID with syrup Sylimarin 1 TSP, PO, BID for next 10 days. Therapy was continued for 21 days.

Results and Discussion

Blood smear was stained with Giemsa stain revealed small pyriform organisms of *B. gibsoni* in singles and doubles, parasitizing 6-8 % RBCs. Haematology depicted anaemia (haemoglobin 8.5 g/dl, packed cell volume 23.2 %, total erythrocyte count $3.52x10^6/\mu l$, increased eosinophils 7%). Serum biochemical analysis revealed increased concentration of alanine aminotransferase (ALT) (228 IU/L), aspartate aminotransferase (ASP) (182 U/L), alkaline phosphatase (ALP) (222 U/L), BUN (84.2 mg/dl) and creatinine (8.6 mg/dl). Microscopic agglutination test (MAT) for *Leptospira* was found negative. Thus, on the basis of history, blood smear examination and haematobiochemical analysis, the case was diagnosed as acute liver and kidney damage induced by *B. gibsoni* infection.

Babesiosis is a tick-borne hemoprotozoan disease which is distributed throughout the world. Drug of choice for *B. gibsoni* is imidocarb dipropionate @ 5 mg/ kg BW SC or IM at 2 week apart (Suzuki *et al.,* 2007). Combination therapy (clindamycin + metronidazole + doxycycline) was the choice for management of *B. gibsoni* infection as reported by earlier authors (Suzuki *et al.,* 2007). The present case showed mild normocytic normochromic anaemia which showed recovery from the condition as therapy was started before substantial damage to erythrocytes and erythropoiesis resumes after 1 week. Hepatic dysfunction was evident as serum enzymatic activity of ALT, AST and ALP was high (Nel *et al*., 2004; Matijatko *et al*., 2009). Intravenous dextrose-25% provides sufficient energy to hepatic cells which acts as the energy of Liver. Azotaemia is a peculiar character indicating acute renal damage which was evident by abnormally high blood urea nitrogen (BUN) and creatinine (Jacobson *et al*., 2000). Fluid therapy on the basis of serum electrolyte balance and diuretic action of frusemide in combination play a major role in establishing the glomerular filtration rate thus improve the renal function.

Clinical examination on 3rd day revealed normal rectal temperature whereas slightly high pulse rate, respiration rate and animal showed fatigue and muscular weakness. Blood and serum sample was analysed to assess the haematobiochemical parameters which revealed decrease activity of serum ALT, AST, ALP, BUN, Creatinine, Na+ and moderate decrease in K+ (2.8 mmol/L). Because of hypokalaemia, the plan of fluid was changed to Dextrose 25 % 100 ml, NS 0.85 % 500 ml IV with KCl @ 21.2 mmol (deficit + maintenance) was given for 4 hr of infusion to combat hypokalaemia. After 5th day, the animal started taking little amount of milk and biscuits but fluid therapy was continued with same treatment. On 7th day slight increase in haemoglobin, TEC and PCV was evident, whereas ALT, AST, ALP, BUN, creatinine decreased and sodium, potassium and chloride found to be normal. On 14th day, animal was reassessed for haematobiochemical parameters which showed improvements in parameters, while creatinine was found to be slightly high. On 21st day blood smear was examined which revealed no evidence of *B. gibsoni* and haematobiochemical parameters were normal. Acute

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injury to renal parenchyma is reversible if diagnosed and management of dehydration commenced earlier in time. The animal recovered uneventfully following therapy.

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Conflict of interest

Author declared no conflict of interest.

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