

## **Babesiosis in dogs: A report of two different cases**

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### **ABSTRACT**

*Canine babesiosis is a worldwide tick borne disease. In dogs, it is usually caused by Babesia canis. Based on the examination of peripheral blood smears, two dogs were found that they were affected with babesiosis. In this, case-1 had fever, decreased feed intake, dullness with anaemia and leucocytosis. Case-2 had fever, tachycardia, tachypnea, vomitions, oliguria, yellowish mucus membranes and low level of haemoglobin, erythrocyte count and platelet count. Both the dogs were treated with diminazine aceturate along with supportive therapy. In this first case was recovered after treatment due to uncomplicated condition. Second case was not responded to the treatment and died due to hepatic damage, anemia due to complication of the babesiosis.*

**Key words:** Clinical signs; babesiosis; dogs; hepatitis; treatment

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### **INTRODUCTION**

Canine babesiosis is a clinically significant and geographically widespread haemoprotozoan disease of domesticated dogs and wild canids [1]. The large *Babesia canis* and the small *Babesia gibsoni* are two organisms commonly known to infect the dogs. Both organisms have Ixodid tick vectors and are found throughout Asia, Africa, Europe, the Middle East, and North America, with *B. canis* being more prevalent [2]. A typical intra erythrocytic piroplasma is pear-shaped and often occurs in pairs [3]. The disease can be clinically classified into uncomplicated and complicated forms. Uncomplicated babesiosis has been suggested to be a consequence of haemolysis while complicated canine babesiosis has been suggested to be a consequence of the development of systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS) [4,5]. Present communication reports the babesiosis in two dogs in this one case are with uncomplicated and another one is complicated babesiosis.

#### **Case History and Observations:**

**Case-1:** An 8 months old, male Pomeranian dog was presented to Teaching Veterinary Clinical Complex, College of Veterinary Science with history of reduced intake of food and dullness. Clinical examination of the dog revealed rise in body temperature (104.4°F), increased heart rate (122/min), congested mucus membranes, dullness (Figure-1). Peripheral blood, whole blood with EDTA was collected for laboratory examination. Peripheral blood smear examination revealed presence of piroplasmic organisms in the RBC. Haematology revealed leucocytosis (9600/cumm) with neutrophilia (6912/cumm), Lymphocytosis (2496/cumm) and Eosinophilia (192/cumm). Decreased haemoglobin (8.8 g/dl), TEC ( $4.9 \times 10^6$ /cumm) was noticed.

**Case-2:** A 6 years old, female Pomeranian dog was presented to Teaching Veterinary Clinical Complex, College of Veterinary Science with a history of fever, in appetite, passing of yellowish urine and occasional vomitions from the past one week. Dog was under treatment at local dispensaries with antibiotics from the past 5 days, but no recovery was noticed. Dog was regularly dewormed and vaccinated against rabies and Canine Distemper, Canine Adenovirus 2, Canine Parainfluenza, Parvo Virus Infection, Leptospirosis (Canicola, icterohaemorrhagiae). Clinical examination of the dog revealed rise in body temperature (103.8°F), slight yellowish pale mucus membranes, increased heart rate (132/min) and respiratory rate (56/min) along with distress, bilateral enlarged lymph nodes. Dog

had tensed and slight yellowish discoloration of abdomen (Figure-2). Dog had decreased urine output, with passage of reddish colour urine along with constipation, vomitions. Peripheral blood, whole blood with EDTA was collected for laboratory examination. Peripheral blood smear examination revealed presence of piroplasmic organisms in the RBC (Figure-3). Haematology revealed decreased haemoglobin (6.3 g/dl), TEC ( $3.8 \times 10^9$ /cumm). Haematology revealed leucocytosis (10640/cumm), Neutrophils (7236/cumm), Lymphocytes (3192/cumm) and Eosinophils (212/cumm) and decreased platelet count of (82,000/ $\mu$ l). Serum biochemical parameters revealed decreased total protein (6.0 g/dL), serum albumin (2.2g/dL). Increased BUN (28 mg/dL), creatinine (1.8 mg/dL), SGPT (224 IU/L) levels and urine analysis revealed positive hays test and presence of RBC in the sediment of urine.

### RESULTS AND DISCUSSION

Based on the clinical signs, and laboratory examination, the condition was diagnosed as babesiosis in both the dogs. Initially treatment was given with inj.Nurobion forte @ 2 ml, inj. Meloxicam @ 0.5 mg/kg body weight, on the day of presentation. After confirmation of the condition inj. diminazine aceturate @ 5 mg/kg body, IM, body weight was given to both the dogs. For case-1 supportive therapy was given with inj.Nurobion forte @ 2 ml, inj. Meloxicam @ 0.5 mg/kg body weight for three days and advised parental administration of four doses of iron dextron @ 2ml/animal at weekly twice, daily supplementation of sharkoferol pet syrup @ 5 ml per day as a general supplementation. After completion of two weeks of therapy Case-1 was responded well and attains its normal activities. For cae-2 inj. ondansetron @ 2ml/dog, 5%DNS @ 5 ml /kg body weight along with above therapy and Liv-52 was oral daily 5 ml was advised. But case was not responded to the therapy and died on the 3<sup>rd</sup> day of therapy.

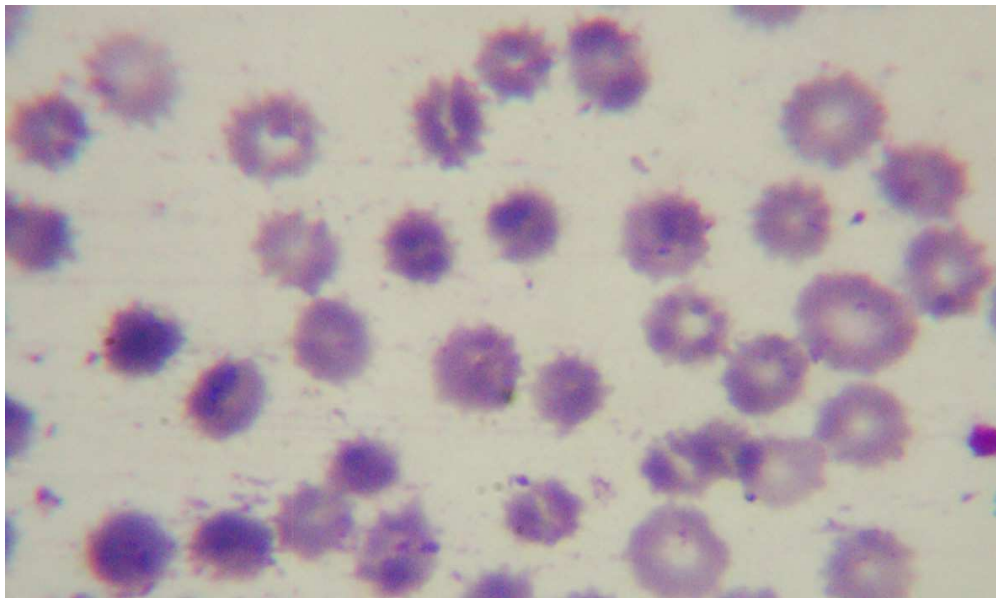


**Figure-1: Congested congenital mucus membranes (Case-1)**

Dogs with uncomplicated babesiosis (case-1) showed the signs of fever, anorexia, depression, and these findings in accordance with the Taboada and Merchant [2]. In complicated form of babesiosis clinical manifestations depend up on the type of particular complication that develops. Clinical signs observed in the present cases include depression, tachy cardia, tachypnoea, anorexia, weakness and fever. It is thought that the clinical signs are the result of tissue hypoxia following the anaemia and a concomitant systemic inflammatory response syndrome caused by marked cytokine release [6]. In the severe form of the disease (case-2) can observe marked haemolytic anaemia, severe acid-base abnormalities with frequent secondary multiple organ failure and complications such as acute renal failure (ARF), hepatopathy with marked icterus, hypoglycaemia [7,8]. Dogs with haemoconcentrated babesiosis and cases developing acute renal failure, acute respiratory distress syndrome or cerebral babesiosis have the worst prognosis and mortality can be greater than 50 % in some cases approaching 100 %, despite intensive, technically advanced interventions [5]. Oliguria is an ominous sign in dogs affected with renal impairment due to babesiosis presently observed in the case-2 [9]. Present observed clinical signs were in agreement with the previous reports [10] and observed haematological and serum biochemical values differ from the local apparently healthy dogs values [11]. Diagnosis of Babesia was done based on the peripheral blood smear examination and same procedure was used for diagnosis of *T.evansi* in different animals previously [12].



**Figure-2: Yellowish discoloration of the abdomen (Case-2)**



**Figure-3: Presence of *Babesia* organisms in peripheral blood smears  
(100X with 4x camera magnification)**

Dogs with babesiosis treated with a single intramuscular injection of Diminazene aceturate at a dose of 5 mg/kg [2, 13]. In dogs affected with babesiosis early diagnosis and treatment, the prognosis is good, but severely affected or untreated animals may die. Current chemotherapeutic agents used to treat canine babesiosis would be incapable of completely eliminating the disease at the recommended dose; they only are capable of limiting mortality and the severity of clinical signs [13]. The most common abnormality in the investigated parameters was thrombocytopenia. The mechanisms of the thrombocytopenia are not yet fully understood in babesiosis[14]. *Babesia* initiates a mechanism of antibody-mediated cytotoxic destruction of circulating erythrocytes. Auto-antibodies are directed against components of the membranes of infected and uninfected erythrocytes. This causes intravascular and extravascular haemolysis, which leads to anaemia. Furlanello et al recorded the anaemia in 74% of dogs with babesiosis and in all the cases the anaemia was normocytic and normochromic [15]. Current treatment strategies for babesiosis often ameliorate the clinical signs of infection, but these hemoparasites are seldom completely eliminated, and when immunocompromised, recrudescence may occur [16]. So, advised the owners Regular control of the ticks was done by regular spraying of cypermethrin preparations to prevent recurrence and spreading of infection in case-1.

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**REFERENCES**

- [1] Irwin PJ, *Parasit Vectors*, **2009**, 2: S4.
- [2] Taboada J, Babesiosis. In: Greene, C. (Ed.), *Infectious Diseases of the Dog and Cat*. WB Saunders, Philadelphia, **1998**, pp. 473–481.
- [3] Homer MJ, Aguilar-Delfin I, Telford SR, Krause PJ, Persing DH, *Clin Microbiol Rev*, **2000**, 13, 451-469.
- [4] Jacobson LS, Clark I, *Journal of the South African Veterinary Association*, **1994**, 65, 134–145.
- [5] Welzl C, Leisewitz AL, Jacobson LS, Vaughanscott T, Myburgh E, *Journal of the South African Veterinary Association*, **2001**, 72:158–162.
- [6] Lobetti RG, Babesiosis, in *Infectious diseases of the dog and cat*, 3rd ed., edited by C.E. Greene. Philadelphia: W.B. Saunders., **2006**.
- [7] Leisewitz AL, Jacobson LS, De Morais HS, Reyers F, *Journal of Veterinary Internal Medicine*, **2001**, 15:445–452.
- [8] Keller N, Jacobson LS, Nel M, De Clerq M, Thompson PN, Schoeman JP, *Journal of Veterinary Internal Medicine*, **2004**, 18:265–270.
- [9] Lobetti RG, Jacobson LS, *Journal of the South African Veterinary Association*, **2001**, 72:23–28.
- [10] Reddy BS, Kumari KN, Sivajothi S, *Comp Clin Pathol*. **2014**, DOI 10.1007/s00580-014-1893-y.
- [11] Reddy BS, Sivajothi S, Reddy LSSV, Raju KGS. *J Parasit Dis*, **2014**, DOI 10.1007/s12639-014-0491-x
- [12] Sivajothi S, Rayulu VC, Malakondaiah P, Sreenivasulu D, *International Journal of Livestock Research*, **2013**, Vol 3(3), 48-56.
- [13] Birkenheuer AJ, Levy MG, Savary KC, Gager RB, Breitschwerdt EB, *J. Am. Anim. Hosp. Assoc.*, **1999**, 35, 125–128.
- [14] Boozer AL, Macintir DK, *Vet. Clin. N. Amer. Small Anim. Pract.*, **2003**, 33, 885-904.
- [15] Furlanello T, Fiorio F, Caldin M, Lubas G, Solano Gallego L, *Italy. Vet. Parasitol.*, **2005**, 134, 77-85
- [16] Irwin PJ, *Vet Clin Small Anim.* , **2010**, 40. 1141–1156.