Successful management of nervous signs due to babesiosis in a dog

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Abstract
Six years old Spitz was presented to the clinic with the history of ataxia, anorexia and severe tick infestation. Wet film examination of the blood did not reveal any motile parasites but, stained peripheral blood smear examination revealed *Babesia canis* organisms in the erythrocytes. Haematobiochemical findings revealed anaemia, low packed cell volume, leucopenia, neutropaenia and eosinophilia. Increased total protein, alanine aminotransferase, alkaline phosphatase and globulin levels noticed. Dog was treated with two doses of diminazene aceturate @ 7.0 mg/kg body weight, deep intramuscular with three days interval along with oral doxycycline. The dog showed uneventful recovery after following the therapy.

Keywords: Babesia canis, dog, doxycycline, nervous signs

Introduction
Babesiosis is one of the important haemoprotozoan diseases of the dogs transmitted mainly by ticks. Severity of the disease depends up on the type of the *Babesia* species involved and the host’s immune response. Different clinical signs in babesiosis were reported earlier including anaemia, lethargy, pale gums, red colour urine, jaundice, enlarged lymph nodes described previously (Reddy et al., 2016) [5]. Cerebral babesiosis is characterized by nervous symptoms such as limb weakness, muscular pain, paresis and sudden death (Schoeman, 2009) [6]. But, nervous signs due to babesiosis were not well documented. Present communication reports about the successful management of nervous signs due to babesiosis in a dog.

Case History and Observations
Six years old Spitz was presented to the clinic with history of lateral recumbence, depression, ataxia, anorexia, walking in circles, changes in behavior and severe tick infestation. Vaccinations were up-to-date and no exposure to toxic drugs or history of travel was reported. Detailed clinical examination revealed, pyrexia (103.8°F), increased heart rate (126/min), respiratory rate (38/min), ataxia, enlarged popliteal lymph nodes, severe tick infestation, reduced response to the stimuli, seeming off balance and stumbling in the legs Mucous membranes were pale pink, capillary refill time was three seconds and dehydration was 5%. Whole blood, serum and urine were collected for laboratory analysis. Complete blood count abnormalities includes microcytic non regenerative anemia (haemoglobin 8.9 g/dL), total erythrocyte count (4.2 x 10⁶/µL), reduced packed cell volume (26%), leukopenia (total white blood cells count 5200/cumm), thrombocytopenia (platelet count 0.96 x10⁶/µl), neutrophil (40 %), lymphocyte (56 %) and eosinophil (4%). Microscopic evaluation of stained peripheral blood smear showed low numbers of spherocytes and characteristic ring shaped *Babesia canis* organisms in the erythrocytes (Fig.1). Biochemical changes noticed were reduced serum albumin (2.2 g/dL), increased total protein (7.5 g/dL), globulin (5.3 g/dL), alanine aminotransferase (68 IU/L) and alkaline phosphatase (258 IU/L).

Treatment and Discussion
The dog was treated with two doses of inj. diminazene aceturate @ 7.0 mg/kg body weight, deep intra muscular at three days interval, dextrose normal saline (@ 10 ml/kg BW), diazepam (@ 0.5 mg/kg BW IM) and ranitidine (@ 2.0 mg/ kg BW IM) on the first day of presentation. Tab. gabapentin (@ 10 mg/kg body weight) and tab.
Doxycycline (@ 10 mg/kg body weight), ranitidine (@ 0.5 mg/kg body weight orally) along with multi vitamin syrup (5 ml PO, BID) were administered for two weeks. It was advised that application of fipronil spray all over the body to control tick infestation. Following therapy dog had uneventful recovery, very active and free from nervous signs. By the third day of therapy it showed improvement in the walk, able to take water voluntarily, on the tenth day it was able to take food regularly and after completion of therapy it was free from nervous signs completely.

Fig 1: *Babesia canis* organisms within the red blood cells (100X).

Babesiosis is one of the most important tick transmitted haemoprotozoan diseases in animals including dogs. In dogs, babesiosis was reported worldwide including various parts of India (Wadhwa, 2011) [9]. Variations in the clinical signs may be due to extent of parasite replication in the host’s red blood cells with subsequent cell lysis. Recorded clinical signs include anorexia, lethargy, haemolytic anaemia, icterus, vomiting and marked loss of body condition (Sivajothi et al., 2014) [8]. Recorded pathological abnormalities including haemoglobinuria, hypoglycaemia, acid-base disturbances, azotaemia and elevations in the levels of liver enzymes (Irwin, 2010) [2]. Observed clinical signs in the present study were due to results of tissue hypoxia following anaemia and a concomitant systemic inflammatory response syndrome caused by marked cytokine release (Lobetti, 2006) [4]. Lymphopenia and thrombocytopenia was reported and it could be due to platelet sequestration in the spleen or immune mediated platelet destruction (Shah et al., 2011) [7]. Pathogenesis involved in development of cerebral babesiosis is related to parasitized erythrocytes that become sequestrated in the central nervous system microvasculature and the release of inflammatory mediators and tissue hypoxia, which can lead to neurological signs (Isbael, 2008) [3]. Clinical signs observed such as ataxia, altered consciousness and recumbency were in accordance with the previous studies. Other neurological signs of observed were paresis, muscle tremor, anisocoria, and vestibular signs (Boozer and Macintire, 2003) [1]. In the present study, early diagnosis and treatment favours the good prognosis. Present case was differentiated from other causes of nervous signs development by collection of history related to type of food offered, vaccination, deworming, toxic drug administration and trauma. Dogs with babesiosis may be able to flare up again in times of stress or reduced immune function so present case was advised not for breeding purpose.

**Summary**

Present communication reports about the cerebral babesiosis and it was successful treatment with diminazene aceturate and doxycycline.

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**References**