CEREBRAL BABESIOSIS IN A DOG: A CASE REPORT

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INTRODUCTION

Babesiosis is caused by intraerythrocytic protozoan parasites of the genus Babesia. A wide range of domestic and wild animals and occasionally man are affected by this disease which is transmitted by ticks and has a world wide distribution. B. gibsoni is a small (2.5 µm), ring-shaped organism and characteristically causes a chronic disease, with progressive anemia as the main sign, in most infected adult dogs and transmitted by brown dog tick Rhiphicephalus sangueinus which is common in Africa and Asia. Cerebral form of babesiosis is limited which involves multiorgan failure. The clinical management of a case of cerebral babesiosis in a dog is presented here.

CASE HISTORY AND OBSERVATION:

A 3 year old male Doberman pincher dog was reported to Small Animal Out-Patient Unit of Madras Veterinary College Teaching Hospital with a history of high fever, anorexia, dull, debilitated and with occasional muscle twitching. On clinical examination the animal was found dehydrated showed blanched mucous membrane with capillary refilling time delayed by 4 seconds. Lymphopenia (15%) and neutropenia (55%) were observed in haemogram. Peripheral blood smear revealed *B. gibsoni* in the erythrocytes. Ulratrasonographic evaluation revealed splenomegaly with left kidney arbitar indistinct Serum alkaline phosphatase (287 I.U/l)activity was found elevated.

TREATMENT AND DISCUSSION

The animal was treated with Diminazene aceturate @ 3.5 mg/kg single intramuscular injection (Taboada, 1998). Injection Metronidazole was administered intravenous @ 3.5 mg/kg for every 8 hours interval for seven days as a treatment protocol. Intravenous colloids with bicarbonate to prevent metabolic acidosis due to tissue hypoxia were administered. Nerve tonics with iron preparations were administered as supportive therapy. In polymerase chain reaction (PCR), corneal smear sample was found negative for canine distemper. The animal was treated intensively but developed weakness, ataxia, seizures, vestibular and cerebellar signs and turned to lateral recumbency.

Since, the animal was found to have multiple organ involvement and did not respond to therapy, the animal was put to sleep after 20 days of ailment. On postmortem examination splenomegaly and chronic nephritis were noticed. Cerebral blood smear examination revealed *B. gibsoni* organisms in erythrocytes.

Symptoms include lethargy, fever, splenomegaly, pallor, icterus and haemoglobinuria and presence of ticks were the most common observations. Thrombocytopenia, lymphopenia and neutropenia were frequent haemogram changes. The clinical manifestations are hyperacute stage: where the hypotensive shock, hypoxia, tissue damage; Hypothermia, coma, disseminated intravascular coagulation (DIC), and death are most common in puppies; an acute form with lethargy, pyrexia, anemia, anorexia, thrombocytopenia, hemolytic anemia, lymphadenopathy, splenomegaly and vomiting and the chronic form by *B. canis* in South Africa. *B. gibsoni* in US reported to cause chronic babesiosis. This involves anorexia, loss of body condition, intermittent pyrexia and lymphadenopathy (Farwell et al., 1982). Cutanous manifestations of *B. gibsoni* were reported by Tarello (2003).

"Cerebral babesiosis" occurs with the sludging of RBC's within central nervous system capillaries leading to tissue hypoxia, weakness, ataxia, seizures, vestibular or cerebellar signs. It should be differentiated from other neurological disorder and systemic diseases which include canine distemper, poisoning and renal involvement leading uremic neuropathy. Old age was a predisposing factor for multiple organ complications. Multiple organ manifestations had poor prognosis. Hepatopathy, pancreatitis, acute renal failure (ARF) and DIC were frequent complications, with immune-mediated haemolytic anaemia (IMHA), acute respiratory distress syndrome (ARDS) and cerebral babesiosis.

Diagnosis can be made from the symptoms and clinical signs and demonstration of small, round, ring-shaped, or teardrop-shaped organism sometimes occurring in pairs in the peripheral blood. Hematological changes include anemia which becomes nonregenerative; inconsistent leukocyte changes and thrombocytopenia are more severe in dogs with ehrlichiosis but usually normal serum chemistry hyperglobulinemia and bilirubinemia may be present.

Treatment administration includes antibabesial drugs, Diminazene acetate @ 3.5 mg/kg b.wt single dose and Imidocarb dipropionate once a week for 2-3 weeks and was found to be most effective. No drug has been completely effective in clearing *B. gibsoni* parasites from erthrocytes (Ruff et al., 1973).Transfusion of packed RBC's or fresh whole blood was indicated if the hemotocrite falls below 15%. Supportive therapy with parental fluids, antibiotics amikacin or clindamycin to control secondary bacterial infection and Vitamin B6 and B12 as nerve tonics anabolic steroids can be used to assist erythrocyte production and to improve general condition. Spontaneous recovery can achieve within 1-2 weeks in babesiosis. In case of multiple organ dysfunction syndromes (MODS) the prognosis is poor. Mathe et al. (2006) reported that systemic inflammatory response syndrome (SIRS) and DIC are two possible pathways leading to multiple organ dysfunction syndromes (MODS) in babesiosis. DIC was found to predict MODS more sensitively than SIRS.

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