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REVIEW ARTICLE

A CASE OF ACUTE RESPIRATORY DISTRESS SYNDROME IN A 2 YEAR OLD MALTESE TERRIER

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ABSTRACT

A case of acute respiratory distress syndrome following treatment of Babesiosis was reported in a 2 year old male Maltese terrier. The patient was admitted to the clinic following a history of anorexia and dullness with preferred recumbence. Main presenting signs on admission were severe pallor, prolonged clotting time, and dyspnoea, lethargy. Blood smears obtained from the ear revealed multiple *Babesia* parasites, severe regenerative anemia, neutrophilia and monocytosis. After meticulous clinical therapy for 5 days patient was discharged having become more active and with improved appetite. Within 24 hours of discharging from the clinic patient developed sudden dyspnoea. Patient went into respiratory shock within 2 hours of the onset of dyspnoea the patient died before arrival at the clinic.

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INTRODUCTION

Canine babesiosis is a tick borne protozoa disease caused by a number of species of *Babesia* parasites. These include: *Babesiagibsoni*, *Babesiacanis*, *Babesiavogeli* and *Babesiarossi* (Simões et al., 2011). The protozoa parasite infects the erythrocytes in the hosts body causing hemolysis. The disease which is also zoonotic is transmitted by the Damacator and Ripicephalus ticks. It has worldwide spread and affects all age groups and breeds of dogs although some genetic predisposition has been reported in the Grey hounds and Pit Bull terriers (Hines, 2012). Its incubation period ranges from 10-28 days. *B.canis* has a shorter incubation period compared to *B. gibsoni*. For infection to occur, the infected female tick bites and feeds on the host for about one week during which inoculation occurs. The severity of disease is influenced by the species of parasite, immune status of the host and age of the host (Schoeman, 2009). Although the disease affects many systems in the body, the main clinical signs include: Depression, pallor of mucous membranes, tachycardia, tachypnoea, weakness, splenomegaly and fever.

The condition can be classified as acute, subclinical, clinical and chronic depending on the presenting signs which may at times be vague i.e. fever, lethargy, vomiting and anorexia (Schoeman, 2009, Irwin, 2010). The disease condition presenting as above is usually manageable when intervention is prompt. However, other body systems may be involved in cases of acute and complicated forms of babesiosis. These complications may affect one or more systems in the body presenting as pancreatitis, dark coloration of urine and acute renal failure, vasculitis and diathesis, seizures, coma, stupors and respiratory failure resulting from pulmonary edema and hypotension. The clinical presentation of the disease is a result of hemolysis and in some cases the initial phase of disease may go unnoticed for long before the body's regenerative ability is overwhelmed (Schoeman, 2009, Irwin, 2010).

Hemogram of a patient with the disease is characterized by *Babesia* parasites, regenerative anaemia -which may not be present in chronic cases-, thrombocytopenia, leukocytosis, reduced PCV, MCHC and MHC, protein levels may also be lower than normal. Hemoconcentration may also be a finding despite hemolytic reaction. Patients with hemoconcentration are at a higher risk of suffering acute respiratory distress (Irwin, 2010). This study describes a case of ARDS in canine babesiosis in a 2 year old Maltese terrier.

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Case history findings and management

Alvin a 2 year old male Maltese terrier weighing 8kg was presented to the clinic with a history of anorexia, dullness, dyspnoea and listlessness for two days. On examination the patient was very weak, dull, dehydrated and with severe dyspnoea. Mucus membrane were paper white and capillary refill time was 6 seconds, body temperature was 39.0°C, heart was pounding, lung sounds were normal. Submandibular, prescapular and popliteal lymph nodes were enlarged. Blood smear examination indicated multiple *Babesia* parasites, severe anaemia characterized by anisocytosis. Severe anaemia, neutrophilia and monocytosis was also evident and blood clotting time was prolonged.

Given the anorexia and apparent weakness coupled with dehydration, the patient required rehydration to prevent shock. However the severe anemia constrained our ability of rapid rehydration. Normal saline and 5% Lactate Ringers solution (2:1 volume ratio) mixture was administered subcutaneously for two days at 35ml/kg twice daily before placement of an intravenous cannula which was used for fluid therapy for the next three days at 50ml/kg twice daily. The patient was also placed on Oxytetracycline 10% injections intramuscularly, vitamin B complex injections intramuscularly as well as dexamethasone 2% injections daily. The Imidocarb dipropionate 12% dose was split into 2 and was given 2 days apart to mitigate the effects that could result into shock. The patient was also placed on 1ml daily dose of Hemoton syrup (multi vitamin and iron combination).

This treatment was maintained for 5 days during which the patient was kept under close observation. The patient became more active, the appetite resumed and respiration was normal. The patient was then discharged with doxycycline and prednisolone for the remaining 9 days of treatment. On arrival at home the client reported no change in the state of the patient and activity and appetite were normal. 20 hours later the patient was reported to have become dull and anorexic, weak and with laboured breathing. Approximately 2 hours later, the patient died before arrival at the emergency clinic. Significant post mortem findings were generalised icterus with pulmonary edema and hydropericardium.

DISCUSSION

To the researchers understanding, there has been no documented case of Acute Respiratory Distress Syndrome in the Andys Veterinary Clinic. Cases of babesiosis diagnosed in the clinic before have been the uncomplicated forms which have previously been successfully treated. Common presenting cases of uncomplicated forms were as described by (Jacobson 2006, Irwin, 2010). Following infection of the erythrocytes, haemolysis occurs resulting in anemia and hence tissue hypoxia (Schoeman, 2009). Tissue hypoxia results in cellular damage and release of cytokines which trigger a generalised inflammatory response resulting in a Systemic Inflammatory Response Syndrome SIRS (Shetters and Klerskens, 2009). ARDS occurs as a result of pulmonary edema and intravascular migration of leukocytes. Tissue hypoxia results in accumulation of carbonic acid hence decrease in pH of body fluids, hence respiratory acidosis. Endothelial cells of lung

vasculature are also hypertrophied, and following the recruitment of inflammatory cells, endothelial cells develop trans-cellular channels that facilitate influx of protein-rich intra-alveolar fluid resulting in pulmonary edema. Increased intra-alveolar fluid impairs gaseous exchange hence respiratory failure (Wright *et al.*, 1989, Hemmer *et al.*, 1999). In some cases the body response may be overwhelmed resulting in Disseminated Intravascular Coagulopathies which subsequently result in Multiple Organ Dysfunction Syndrome (Mathe *et al.*, 2006). It is the opinion of the author that given the presenting signs, the patient died as a result of respiratory shock. The pulmonary edema resulted in severe tissue hypoxia to the vital organs including the heart (resulting in hypotension) and brain hence sudden death. The general prognosis of uncomplicated case of *Babesia* is good with about 85% survival rates. However, in complicated cases of babesiosis prognosis is poor and mortality rate ranges between 50-100% (Welzl *et al.*, 2001). The pathogenesis in this case seems to have been delayed by the initial therapeutic intervention but resumed following discharge of the patient from the clinic.

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