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Cerebral Babesiosis in a Geriatric Pomeranian- A Case Report

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Abstract

Canine babesiosis is a globally significant haemoprotozoan disease caused primarily by *Babesia gibsoni* and *Babesia canis,* with clinical presentations ranging from subclinical carriers to fulminant, life-threatening illness. While hematological and systemic manifestations are well documented, central nervous system (CNS) involvement is an uncommon and often overlooked presentation, particularly in geriatric animals, where clinical signs may be non-specific. The current case describes a ten-year-old female Pomeranian exhibiting progressive ataxia, systemic lethargy, and prolonged anorexia in the absence of overt haematologic crisis. Peripheral blood smear examination revealed intraerythrocytic piroplasms consistent with *Babesia gibsoni*. Treatment with atovaquone and azithromycin led to significant clinical improvement, supported by hepatoprotective and metabolic therapy.

Keywords: Canine- *Babesia gibsoni* - cerebral babesiosis

Introduction

Babesiosis is a clinically significant vector-borne protozoan infection in domestic dogs, with *Babesia gibsoni* being a prominent causative agent. Classic clinical signs include pyrexia, lethargy, hemolytic anemia, icterus, and thrombocytopenia. However, in chronic or atypical cases, especially those involving elderly or immuno-compromised animals, the disease may progress insidiously and present with vague systemic or neurological abnormalities. This case report describes a rare instance of subclinical cerebral babesiosis in a geriatric Pomeranian, where the diagnostic challenge was compounded by the absence of visible ectoparasites and the lack of hallmark hematologic derangement. This present case describes the neurological manifestation of babesiosis in Pomeranian dog.

Case History and Observations

A ten-year-old, unspayed female Pomeranian weighing 9.5 kg was presented to the Veterinary Clinical Complex at ANDUAT with a history of progressive neurological deterioration, including hind limb ataxia and impaired motor coordination, accompanied by systemic signs such as persistent anorexia, intermittent low-grade fever, and marked lethargy. The duration of the clinical signs extended over nearly three months, during which the dog exhibited gradual weight loss, reduced voluntary movement, and increasingly impaired proprioception. The owner reported that the dog had not consumed solid food in the previous three days and had only ingested a single biscuit. The patient had no known exposure to ticks in recent months, and her vaccination and deworming records were complete and up to date. There was no history of trauma, recent travel, toxin exposure, or prior systemic illness. Clinical examination revealed a dull and depressed demeanor, mild dehydration, poor body condition, and a rectal temperature of 101.6 °F. The mucous membranes were pink with adequate capillary refill time, and no icterus or petechiae were observed. Neurological assessment revealed generalized ataxia, delayed proprioceptive positioning, and reduced awareness of environmental stimuli, suggestive of central nervous system involvement. The absence of spinal pain or asymmetry indicated that the signs were unlikely to be due to orthopedic or spinal cord pathology. Initial considerations included the neurological form of canine distemper, tick-borne encephalitis, ehrlichiosis, intervertebral disc disease (IVDD), hepatic encephalopathy, and cerebral Babesia. Canine distemper was ruled out based on the current vaccination, absence of characteristic signs such as myoclonus, ocular/nasal discharge, or hyperkeratosis, and lack of history of exposure. IVDD or spinal trauma was considered less likely because of the symmetrical limb deficits, absence of spinal pain, and no focal neurological deficits.

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Metabolic encephalopathies, such as hypoglycemia and hepatic encephalopathy, were ruled out following normal blood glucose levels and the absence of hepatic insufficiency signs. A complete blood count revealed mild anemia (Hb: 10.2 g/dL) and moderate neutrophilic leukocytosis (78%), consistent with ongoing inflammatory or infectious processes. Biochemical parameters indicated mild elevations in SGPT (112 IU/L) and blood urea nitrogen (26 mg/dL Giemsa-stained peripheral blood smear revealed small, intraerythrocytic ring-form piroplasms morphologically consistent with *Babesia gibsoni*, establishing the final diagnosis of subclinical cerebral babesiosis. The diagnostic challenge was compounded by the absence of thrombocytopenia, overt hemolysis, or visible tick burden, which are typically expected in acute babesiosis. However, the neurological signs combined with haematological and parasitological findings confirmed a case of cerebral involvement by *B. gibsoni*, manifesting in a subacute, atypical form.

Fig 1. --

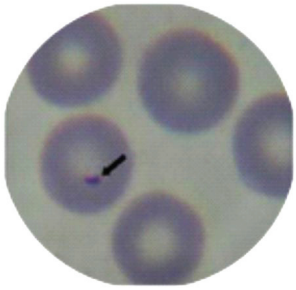

Fig 2. Giemsa-stained smear intraery throcytic piroplasms

Treatment and Discussion

Following confirmation of the diagnosis, treatment was initiated with a combination of Atovaquone and Azithromycin, a regimen known for its high efficacy against *Babesia gibsoni*. Atovaquone was administered orally at a dose of 13.3 mg/kg every eight hours, while Azithromycin was administered at 10 mg/kg once daily for ten days. Intravenous isotonic fluids (Lactated Ringer's solution) were administered to correct dehydration and support renal perfusion. Nutritional supplementation, including energy-dense palatable diets and B-complex vitamins, was provided to counteract anorexia and support hematopoiesis. Within 48 h of treatment initiation, the patient exhibited noticeable improvements in mentation and appetite. By the fifth day, proprioceptive reflexes improved, and the animals were ambulatory with reduced ataxia. By the tenth day, neurological signs had fully resolved, and peripheral blood smears were negative for parasitemia.

This case underscores the increasingly recognized but underreported phenomenon of neurological babesiosis in dogs, particularly in geriatric patients. The pathogenesis of cerebral babesiosis is believed to involve the sequestration of parasitized erythrocytes within the cerebral microvasculature, resulting in microthrombi formation, localized hypoxia, and perivascular inflammation. Multiple treatment options have been proposed for *Babesia gibsoni*, including traditional agents such as Diminazene aceturate and imidocarb dipropionate, both of which have shown limited efficacy in persistent or neurologically involved cases. Multi-drug regimens, such as clindamycin-metronidazole-doxycycline (CMD) and PCR-monitored protocols involving doxycycline with fluoroquinolones or rifampin, have also been employed with inconsistent success and prolonged treatment durations. In contrast, the combination of Atovaquone and Azithromycin has emerged as a superior therapeutic strategy owing to its dual mechanisms of action and favorable tissue penetration, including in the central nervous system.

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