THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
April 2024 Vol.4(4), 1559- 1562

Popular Article

An Overview of Canine Babesiosis

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https://doi.org/10.5281/zenodo.11098138

Introduction

Tick-borne haemoparasites are one of the most important vector-borne infections of dogs. They are numerous and are caused by several etiological agents such as bacterial, protozoan and rickettsial organisms. Among them, Tick-borne haemopathogens such as Babesia, Ehrlichia, Anaplasma, Borrelia and Hepatozoon are of major health concern to dogs and some of which are of zoonotic significance. Canine babesiosis is an important world-wide disease caused by intra-erythrocytic protozoan parasites of the genus Babesia including B. canis and B. gibsoni which are the two predominant species that cause canine babesiosis and strains of these organisms are found worldwide. Babesiosis, caused by infection with organisms from the genus *Babesia*, is characterized by hemolytic anemia, fever, and splenomegaly.

Etiology and epidemiology

Babesia spp. are intraerythrocytic protozoan parasites and species have been named and identified based on the vertebrate host and the size of the parasite (large or small *Babesia* species). Large *Babesia* spp. are 3 to 7 µm in length, whereas small *Babesia* spp. are 1 to 3 µm in length.

Tick vectors are the most important means of transmission of *Babesia* species. However, for some *Babesia* species, such as *Babesia gibsoni* direct transmission between dogs, through fighting (and exchange of blood), or congenital transplacental transmission, is believed to be the most common route of transmission. Canine babesiosis is a disease of worldwide importance. Initially, two species of *Babesia* were recognized in dogs (*Babesia canis* and *B. gibsoni*); however, now at least nine genetically distinct canine piroplasms have been described.

Clinical Signs and Their Pathogenesis

Clinical features include fever, thrombocytopenia, haemolytic anaemia, and splenomegaly. Nonspecific signs: such as lethargy, anorexia, and weakness. Occasionally owners note jaundice, mucosal

pallor, or discoloration of the urine caused by bilirubinuria or haemoglobinuria.

Parasite antigens are incorporated on the erythrocyte surface and may leads to their opsonization by antibodies, with or without complement, and account for haemolytic anaemia and thrombocytopenia. Mechanisms other than immune-mediated destruction that contribute to erythrocyte damage include increased erythrocyte osmotic fragility, direct injury of erythrocytes by *Babesia* parasites, accumulation of cyclic nucleotides, and oxidative injury. Lipid peroxidation increases erythrocyte rigidity and slows the passage of erythrocytes through capillary beds. Soluble parasite proteases activate the kallikrein system and induce fibrinogen- like protein formation. These proteins make erythrocytes more "sticky," and they sludge in capillary beds, which contributes to anaemia and many of the other potential clinical signs. The most severe sludging occurs in the central nervous system (CNS) and muscles. Thrombocytopenia may result from immune-mediated or coagulatory consumption of platelets. Despite severely decreased platelet counts, bleeding is rarely observed in dogs infected with most *Babesia* strains, and other abnormal coagulation test results are uncommon. Other possible complications include membranoproliferative glomerulonephritis, which may have an immune-mediated pathogenesis.

Severe Babesiosis

Virulent *B. canis canis* and especially *B. canis rossi* strains induce a profound systemic inflammatory response, which can result in a severe sepsis-like syndrome with multiple organ dysfunction. Severe clinical illness in dogs infected with *B. canis rossi* results from hypotension, acute renal failure (ARF), neurologic complications, disseminated intravascular coagulation (DIC), a hepatopathy, and acute respiratory distress syndrome (ARDS).

Red biliary syndrome" is a paradoxical phenomenon of severe intravascular hemolysis (manifested as hemoglobinemia and hemoglobinuria) in combination with hemoconcentration (high-reference-range or elevated hematocrit). Neurologic complications result from sludging of parasitized erythrocytes in CNS capillary beds, with congestion and macroscopic and microscopic hemorrhages. Severe hypoglycemia can also result in neurologic signs.

Other complications of severe babesiosis include pancreatitis, rhabdomyolysis, ocular involvement, upper respiratory signs, cardiac arrhythmias, necrosis of the extremities, and fluid accumulation. Persistent lactate concentrations above 40 mg/dL are a poor prognostic indicator for survival.

Physical Examination Findings

Physical examination abnormalities in most dogs with babesiosis consist of fever, mucosal pallor, lethargy, splenomegaly, and bounding pulse. Tachycardia and tachypnea may be present in severely anemic dogs. Mucosal hemorrhages and/ or epistaxis may be present in dogs with

B. conradae infection and excessive bleeding from venipuncture sites may be noted. Dogs with severe babesiosis may show CNS signs such as incoordination, pelvic limb paresis, muscle tremors, nystagmus, anisocoria, intermittent loss of consciousness, seizures, stupor, coma, aggression, or vocalization.63 Dogs with red biliary syndrome may have congested mucous membranes or icterus. Other

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Published 26.04.2024

clinical abnormalities in dogs with severe babesiosis include tachypnea, increased lung sounds, and cardiac arrhythmias.

Diagnosis

Haemato- biochemical profile: In dogs with babesiosis, the primary hematologic abnormalities are anemia and thrombocytopenia. Thrombocytopenia is often present, even when anemia is absent. A mild, normocytic, normochromic anemia is generally noted in the first few days after infection, which becomes macrocytic, hypochromic, and regenerative as the disease progresses. Leukocyte abnormalities are inconsistently observed but may include leukocytosis (with or without a left shift), leukopenia, neutrophilia, neutropenia, lymphocytosis, and/ or eosinophilia.

There are no pathognomonic biochemical findings in dogs with babesiosis. Common findings in North American dogs include hyperglobulinemia (which may be present in the absence of other laboratory abnormalities), mildly increased liver enzyme activities, and, less commonly, hyperbilirubinemia.

Urinalysis: Urinalysis abnormalities in dogs with babesiosis are variable but include bilirubinuria, hemoglobinuria, proteinuria, and, in rare cases granular casts.

Microbiologic Testing: There are three basic methods available for specific diagnosis of *Babesia* infections: microscopic identification, serologic testing, and nucleic acid–based detection methods.

Cytologic Diagnosis: Microscopic identification: *Babesia canis* are large, pyriform organisms and usually exist singly or in pairs. Smaller single intracellular organisms are likely to be *B. gibsoni* or *B. conradae.*

Serologic Diagnosis: Immunofluorescent antibody (IFA) assays are used most commonly to detect antibodies to *Babesia* species. As a general guideline, titers to *B. canis* or *B. gibsoni* of 1:64 or greater on a single specimen are supportive of exposure.

Molecular Diagnosis Using the Polymerase Chain Reaction: Genetic methods such as PCR assays are the most sensitive and specific means of detecting active infection with *Babesia.*

Pathologic Findings: These include staining of tissues with hemoglobin or bilirubin, hepatosplenomegaly, lymphadenomegaly, and kidneys that are a dark-reddish color. Edema and hemorrhage, which may indicate vascular injury and poor tissue oxygenation in severely affected dogs, are often most severe in the lungs. Pathologic changes in the brain of these dogs include congestion, macroscopic and microscopic hemorrhages, sequestration of parasitized erythrocytes in capillary beds. Microthrombi of many tissues may be evident in animals exhibiting signs of DIC. Impression smears of the spleen may substantiate the diagnosis of babesiosis at necropsy.

Treatment

Antiprotozoal Treatment

Dogs generally show clinical improvement within 24 to 72 hours of treatment with antibabesial drugs, but some animals take as long as 7 days to respond. Imidocarb dipropionate is active against *B. canis*. A single dose of 7.5 mg/kg or a single dose of 6 mg/kg given the day after a dose of diminazene (3.5 mg/kg) also clears infection.

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Published 26.04.2024

Atovaquone (@ 13.3 mg/kg PO q8hr) and azithromycin (@ 10mg/kg PO q24hr) combination therapy for 10 days is the most effective treatment for *B. gibsoni* and *B. conradae* infections. Atovaquone must be administered with a fatty meal to maximize drug absorption. An alternative treatment strategy can be used to treat *B. gibsoni* infections that fail to respond to atovaquone and azithromycin. This regimen involves a combination of clindamycin (@ 25 mg/kgPO q12hr), metronidazole (@15mg/kf PO q12 hr) , and doxycycline (@ 5mg/kg PO q 12 hr) for a minimum of 3 months. Aggressive supportive care and monotherapy with clindamycin (25 mg/kg PO q12h for 7 to 21 days) has been recommended if specific antibabesial drugs are not available.

Supportive Care: Other supportive care measures that may be required to treat animals with babesiosis are packed red blood cell transfusions and intravenous crystalloid fluid therapy.

Immunity and Vaccination: The duration of protective immunity against *B. canis* infection is limited. Antibody titers may gradually decline between 3 and 5 months after infection. Cross- protection between strains does not occur. A vaccine produced from cell-culture–derived exoantigens of *B. canis canis* is available in Europe.

Prevention: Treatment of babesiosis is expensive and may be ineffective, so prevention is of paramount importance. The primary means of prevention is tick control by use of amitraz- impregnated collars. Prevention of aggressive interactions with other dogs may also prevent infection. All prospective canine blood donors should be tested for babesiosis with serology and PCR assays and animals positive by either or both methods should not be used for blood donation.

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Published 26.04.2024