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Acute canine Babesiosis in a dog at Basrah, Iraq (A case study)

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Abstract: A male, Mullinoise breed police (K9) dog, 25 kg weight and of 3 years old, had been brought to the Veterinary Clinical Consult Hospital, College of Veterinary Medicine, University of Basrah, Iraq, with patient history and signs of loss of appetite, emaciation, anemia, and hemoglobinuria. Complete clinical examinations were performed for the diseased dog. On clinical examinations, diseased animal show signs of Complete loss of appetite, Pale of mucus membranes, Severe emaciation and weakness, Panting with the abdominal type of respiration and Hemoglobinuria. Moreover, fever, increase respiratory and heart rate was also indicated in the diseased animal. Furthermore, Normocytic normochromic anemia with lymphocytosis and a total parasitemia of 5% as well as, Hypoproteinemia, Hyperbilirubinemia, and hypoglycemia were also detected as a clinical pathological change in the diseased dog. It has been concluded that canine Babesiosis is one of the important diseases that must be monitored due to its bad pathological effect on the health of the affected animal, which often ends with the death of the sick dogs. Therefore, early diagnosis of the disease and speeding up treatment may save the animal from inevitable death.

Keywords: Canine Babesiosis, a police (K9) dog, Basrah, Iraq.

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I. Introduction

Babesiosis is a disease of worldwide significance and was first recognized in 1888 as a cause of fever, hemolytic anemia, hemoglobinuria, and death of the diseased animal(1).

Canine babesiosis is a significant tick-borne disease caused by various species of the protozoan genus *Babesia*. (2). Parasites of this genus are primarily transmitted through tick bites and as such can infect a wide variety of domestic and wild animals as well as humans (3).

Members of the genus *Babesia* readily parasitize the red blood cells of dogs, causing progressive anemia. Canine babesia is morphologically classified into large and small forms, both exhibiting a worldwide distribution(4). *Babesia canis* and another novel, as yet unnamed *Babesia* spp. detected in the USA (large *Babesia*) and *Babesia gibsoni* and *Babesia annae* (small *Babesia*) have been documented to infect dogs(5). Three main species of large *Babesia* infect dogs, namely *Babesia vogeli*, *Babesia. canis* and *Babesia rossi*. These three species are antigenically distinct, transmitted by different vectors, and differ widely in pathogenicity and geographic distribution(6).

Traditionally, the morphology of the protozoan (piroplasm merozoites) within the red blood cell was used as the chief taxonomic determinant. This assessment, made by microscopic evaluation of a blood smear, can be used to classify these protozoa as either large (e.g. *Babesia canis*) or small forms (e.g. *Babesia gibsoni*). Subsequently, molecular techniques allowed the identification of several species of *Babesia* that can infect dogs(7).

Infection occurs when a *Babesia*-infected tick bites a dog and releases *Babesia* sporozoites into the dog's bloodstream. A tick must feed for two to three days to infect a dog with *Babesia*. The young *Babesia* organisms attach to red blood cells, eventually penetrating and making a new home within the cells for themselves. Inside the red blood cell, the *Babesia* organism divests its outer coating and begins to divide, becoming a new form called a merozoite that a new tick may ingest during a blood meal. Infected pregnant dogs can spread *Babesia* to their unborn puppies, and dogs can transmit the organism by biting another dog as well. In fact, for *Babesia gibsoni*, which is primarily a pit bull terrier infection, ticks are a minor cause of infection with maternal transmission, and bite wounds are the chief routes of transmission(8). The purpose of this report is to investigate a clinical case of acute babesial infection in a dog with complete clinical and some biochemical examinations performed.

II. Case Presentation

Animal presentation and case history:-

A male, Mullinoise breed a police (K9) dog, 25 kg weight and of 3 years old, had been brought to the Veterinary Clinical Consult Hospital, College of Veterinary Medicine, University of Basrah, Iraq, with patient history and signs of loss of appetite, emaciation anemia, and hemoglobinuria.

Clinical and laboratory examinations:-

1-Complete clinical examinations were performed for the diseased dog and included measuring the vital signs including body temperature, respiratory and heart rate, as well as a physical examination of other parts such as the kidneys, liver, and mucous membranes lining the eyes, Moreover, Auscultation of the lung and heart was also performed using the stethoscope.

2-Blood sample was withdrawn from the cephalic vein using for examination of complete blood picture (EDTA sample) (Automatic digital cell counter from Beckman USA) and the remaining used for extraction of serum for evaluation of Total protein, total bilirubin and glucose according to manufacture instruction of (Roche Diagnostics, Indianapolis, GMBH, Germany).

3-Gimesa stain blood smears were used for identification of the Babesia spp.

4- Fecal samples were screened for internal parasite infection, using the standard coprological methods (2).

III. Results

The diseased animal exhibited different clinical signs which were represented as illustrated in **Table 1**.

Table 1. The clinical manifestations of a diseased dog

No	Clinical signs description
1	Complete loss of appetite
2	Pale of mucus membrane / Fig 1 .
3	Severe emaciation and weakness / Fig 2
4	Panting with the abdominal type of respiration
5	Hemoglobinuria

Moreover, the disease dog show signs of a fever (39.8C), the heart rate was 155/ min, respiratory rate of 47/ mint. **Table 2**. Besides, examination of blood parameters indicates Normocytic normochromic anemia with lymphocytosis and a total parasitemia of 5%. **Table 3**. Moreover, Biochemical changes reveal Hypoproteinemia, Increase total bilirubin value as well as hypoglycemia in disease dogs. **Table 4**.



Figure 1: Pale mucus membrane



Figure 2: Severe emaciation and weakness

Table 2: The Vital signs of the diseased dog

Parameters	Diseased animal (dog)	The normal reference*
Body temperature / °C	39.8	37.5-39.2
Heart rate/min	155	70-120
Respiratory rate/ min	47	15-30

**Ettinger and Feldman, 2005(6)*

Table 3: Hematological parameters of the dog with Babesial infection

Parameters	Diseased animal (dog)	The Normal Reference*
RBC x 10 ⁶	3.8	4.8-9.3
Hb g/dl	8.8	12.1-20.3
PCV %	25	37-55
MCV fl	65.78	64.77
MCHC %	35.2	32-63
TLC x 10 ³	15.3	4.0-15.5
Lymphocytes %	45	12-30
Neutrophils %	42	66-77
Eosinophiles %	5	2-12
Monocytes %	8	3-10
Basophiles %	0	0.5-0.7
Total platelet counts x10 ³	248	170-400
Percentage parasitemia	5%	0%

**Ettinger and Feldman, 2005(6)*

Blood smears stained with Giemsa indicated the Babesia spp infected dog as Pyriform in shape, large parasite with pointed one end, and round other. In a single RBC, more than one organism may be found sometimes. Fig 3.

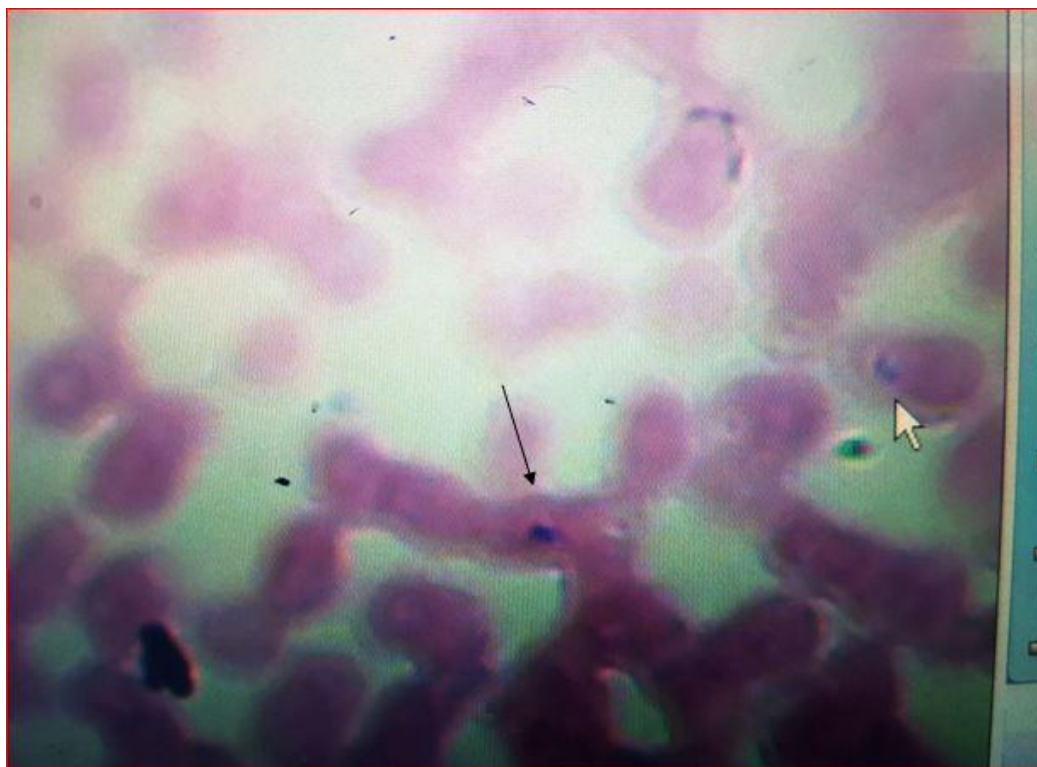


Figure 3: Babesia spp inside the erythrocyte of the diseased dog (Giemsa stain)x100

Table 4: Biochemical analysis of the diseased dog

Parameters	Diseased animal (dog)	The normal reference*
Total protein g/dl	4.2	5.1-7.8
Total bilirubin mg/dl	0.6	0.1-0.3
Glucose (mg/dL)	51	70-138

*Ettinger and Feldman, 2005(6)

IV. Discussion

It was documented that Babesiosis is a tick-borne disease affecting humans and many domestic and wild animals. Domestic animals showing appreciable morbidity and mortality include dogs, cats, cattle, and horses. Moreover, Both canine and feline babesiosis are diseases characterized by hemolytic anemia, icterus, and hemoglobinuria. Canine babesiosis can range from chronic or subclinical to peracute and fatal, depending on the virulence of the species and the susceptibility of the host(9).

Various species of ticks such as *Rhipicephalus sanguineus*, *Dermacentor* spp. and *Haemaphysalis ellipticum* can transmit the large babesia of dogs, whereas *B. gibsoni* is transmitted by *Haemaphysalis bispinosa* and *Haemaphysalis longicornis*. *Babesia annae* is thought to be transmitted by *Ixodes hexagonus*, Both transstadial and transovarial transmission can occur and ticks are believed to remain infective for several generations. *Babesia* spp. can also be transmitted by blood transfusion. Strong circumstantial evidence exists that *B. gibsoni* is transmitted by dog bites whilst transplacental transmission from the dam to offspring has recently been proven as an additional mode of transmission(10).

The geographical distribution of *Babesia* spp. infections in the world is highly variable and largely dependent on the distribution of the competent tick vector. Besides, the prevalence of *Babesia* spp. infections vary likely because of the various diagnostic techniques used for detection, the country and population analyzed, and the species of *Babesia* under investigation(11).

None of the *Babesia* species that affect dogs and/or cats are considered to be of zoonotic importance, Moreover, there is a lack of evidence that *Babesia* spp. known to be zoonotic can infect dogs. However, the data for addressing this topic are incomplete given that some cases of human babesiosis are reported without any firm identification of the causative protozoan species (12)

The wide range of clinical manifestations depends very much on the species of *Babesia* causing infection and other factors that affect the severity of the disease, including age, splenectomy, immune competence, and concomitant infection or disease, Besides, disease severity has been associated with parasite density, However, limited information is available regarding disease severity and parasite density in other

Babesia species. In a recent study, parasite density was not different between survivors and non-survivors in dogs infected with *B. canis*. (9)

In the current case study, the animal shows different clinical manifestations which are also mentioned by (1,5,7,8). There are clinical signs and clinicopathological abnormalities that are common across all *Babesia* species infecting dogs, As, frequent clinical signs associated with canine babesiosis are apathy, weakness, anorexia, pale mucous membranes, and a poor general condition. All *Babesia* species can cause fever, enlarged lymph nodes, and splenomegaly, anemia, thrombocytopenia, jaundice, and pigmenturia. Although thrombocytopenia, to a varying extent, is frequently detected, the presence of petechiae or ecchymosis is less common. Thrombocytopenia, when present, varies from mild to severe, as does anemia. Other abnormalities that can be detected include hypoalbuminemia and hyperbilirubinemia.

Some clinical signs and clinicopathological abnormalities differ among *Babesia* species infecting dogs, However, Many dogs could present other clinical signs that are not directly related to hemolysis by piroplasms but that demonstrate the involvement of other organs. These complications are especially prevalent following infection by *B. rossi*. A non-exhaustive list includes weight loss, acute or chronic nephropathy, glomerulonephritis, coagulation disorders (disseminated intravascular coagulation), jaundice from liver disease, immune-mediated hemolysis or thrombocytopenia, hemoconcentration, shock, metabolic and/or respiratory alkalosis, and/or acidosis, gastrointestinal disorders (vomiting or diarrhea), pancreatitis, ascites, ocular lesions (uveitis or blindness), myalgia, rhabdomyolysis and respiratory problems (edema or acute respiratory distress) (13).

It was also documented that, Clinical signs include pale mucous membranes, depression, tachycardia, tachypnoea, anorexia, weakness, splenomegaly, and fever. It is thought that the clinical signs are the result of tissue hypoxia following the anemia and a concomitant systemic inflammatory response syndrome caused by marked cytokine release (10). The pathogenesis of the anemia is incompletely understood, intravascular and extravascular hemolysis takes place, but other mechanisms such as poor bone marrow response are thought to play a role as well(5,8). The anemia is (maybe, the opposite of expected) not correlated to the degree of parasitemia and dogs start to improve after parasitocidal treatment, even though their hematocrits generally drop further, before starting to rise (14). Some cases show additional immune-mediated break- down of red blood cells and dogs that show in-saline-positive red blood cell agglutination have to be carefully monitored for rapid decreases in hematocrits (10).

The severe form of the disease is characterized by marked hemolytic anemia, severe acid-base abnormalities (13) with frequent secondary multiple organ failure and complications such as acute renal failure (ARF), Hepatopathy with marked icterus, Hypoglycemia(which indicated in the current case study) (8,13), acute respiratory distress syndrome (ARDS), cerebral pathology and additional immune-mediated red blood cell destruction (IMHA)(5). Moreover, the mild hypoproteinemia may occur due to digestive disturbances and severe limitation of protein intake in the diet, destruction of proteins due to fever as macrophages activated in the liver and spleen secrete tissue necrotic factor (TNF-alpha) into the bloodstream resulting in hypoproteinemia, Moreover decrees production and synthesis from liver especially albumin and when more plasma proteins loss in urine due to renal disease and nephritic syndrome reflecting from the disease (11,14).

The pathogenic effect of babesia started After penetration of the cell, *Babesia* multiplies via repeated binary fission within the erythrocyte, resulting in up to 16 merozoites. Multiplication of the parasites damages the erythrocyte cell membrane, causing increased osmotic fragility and subsequent intravascular and extravascular hemolysis. Indirect pathways of cell destruction are also important contributors to the pathogenicity of *Babesia*-induced anemia, which is the predominant clinical syndrome. Immune-mediated hemolytic anemia is assumed to occur with all *Babesia* spp. following the production of anti-erythrocyte membrane antibodies

(1, 7). *Babesia* activates antibody-mediated cytotoxic destruction of erythrocytes, leading to anemia, hemoglobinaemia, hemoglobinuria, thrombocytopenia, and, in cases of massive infection, to death caused by multiple organ dysfunction syndromes (5,8). Tissue hypoxia is found in severe babesiosis in both dogs and ruminants (1).

Hyperbilirubinemia due to significant increase in indirect bilirubin was also indicated in the current case study which might result from excessive destruction of erythrocytes and the indirect hepatocellular damage, Furthermore, Solano-Gallego and Baneth(9) and Camacho-Garcia(15) were added that measurement of indirect bilirubin might be helpful in measurement the increased breakdown of hemoglobin as in hemolytic anemia which will increase the production of unconjugated bilirubin, which is presented to the liver in excess, and can result in increased total bilirubin in the blood.

V. Management and treatment

Clinical cure and a good therapeutic response are much more likely achieved for infections by large-sized *Babesia* species than infections by the small-sized species, the latter of which tend to be more refractory to

conventional treatments (7). Several therapeutic protocols aimed at infections caused by small Babesia species are used, although parasitological cures are considered rare. The persistence of *B. gibsoni* in dogs following treatment with different protocols using clindamycin, metronidazole, doxycycline, diminazene, imidocarb dipropionate, atovaquone, and azithromycin is testament to the resilience of this parasite (2,4).

Furthermore, The, same dog can be re-infected by identical Babesia species or co-incidentally with a second species. Although the clinical consequences of re-infections are not well defined, in endemic regions, dogs can be chronically infected, in a premunition phase, without clinical consequences, this phase may even be beneficial in terms of protecting against future infection (9,10).

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