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Fatal Canine Babesiosis in a Caucasian Shepherd Bitch from Northwestern Nigeria

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ABSTRACT

Canine babesiosis is an important tick-borne hemoparasitic disease associated with hemolytic anemia and, in severe cases, widespread systemic and organ-specific pathological changes in dogs. This report describes the clinicopathological and postmortem findings of a 2-year-old female Caucasian Shepherd dog presented to the D.I.K Osori Veterinary Teaching Hospital, Usmanu Danfodiyo University Sokoto, Nigeria, with weakness, anorexia, dyspnea, dehydration, jaundice, and heavy tick infestation. Blood smear examination demonstrated intraerythrocytic Babesia spp. organisms. Hematological evaluation revealed severe macrocytic hypochromic anemia (PCV: 15%, Hb: 48 g/L, RBC: $1.93x10^{12}$ L) with band neutrophilia ($0.42x10^{9}$). Serum biochemistry demonstrated marked hyperbilirubinemia (total bilirubin: 10.15 mg/dL), elevated liver enzymes (ALT: 118 U/L, AST: 170 U/L), and azotemia (urea: 56.2 mg/dL, creatinine: 2.6 mg/dL). Gross postmortem examination revealed generalized lymphadenomegaly, splenomegaly, and hepatomegaly with icterus, pulmonary congestion with areas of consolidation, renal petechiation, and multifocal hemorrhages. Histopathology demonstrated glomerular shrinkage, tubular epithelial degeneration, vascular congestion, and reactive lymphoid hyperplasia. The findings were consistent with severe canine babesiosis, characterized by hemolytic anemia and multisystemic pathological involvement. However, histopathological evaluation was limited to kidney and lymph node tissues, which may have restricted full microscopic characterization of lesions in other affected organs, including the spleen, liver, and lungs. Death occurred following rapid clinical deterioration despite treatment and supportive management. In conclusion, severe canine babesiosis induces fatal multisystemic failure through severe hemolysis, systemic inflammatory response, and multiorgan injury. Early diagnostic surveillance and prompt intervention remain critical to preventing mortality.

Keywords:
Canine babesiosis
Hemolytic anemia
Renal injury
Histopathology and
Necropsy Dog

INTRODUCTION

Canine babesiosis is a globally important tick-borne hemoparasitic disease caused by protozoan parasites of the genus Babesia, which infect erythrocytes and produce varying degrees of hemolytic anemia and systemic illness (Köster *et al.,* 2015). The disease is mainly transmitted by ixodid ticks, particularly Rhipicephalus sanguineus, although transmission through blood contamination and dog bites has also been documented (Begum *et al.,* 2020). Several species affect dogs, including Babesia rossi, Babesia canis, Babesia vogeli, and Babesia gibsoni, with marked variations in pathogenicity and geographic distribution (Köster *et al.,* 2015). The pathogenesis of canine babesiosis involves direct erythrocyte destruction, immune-mediated hemolysis, endothelial injury, inflammatory cytokine release, and circulatory compromise. Complicated babesiosis has been reported to occur with systemic inflammatory responses, vascular injury, acute kidney injury, hepatopathy, pulmonary edema, neurological complications, and shock (Köster *et al.,* 2015). Clinically, affected dogs commonly present with pyrexia, weakness, anorexia, pale or icteric mucous membranes, lymphadenomegaly, splenomegaly, dehydration, and respiratory distress (Begum *et al.,* 2020). The Diagnostic evaluation of hemoparasitic infections relies heavily on the microscopic examination of thin and thick blood films stained with Giemsa to detect intraerythrocytic parasite structures (Karibi & Ekine, 2026). Laboratory findings often reveal regenerative anemia, thrombocytopenia, neutrophilic leukocytosis, hyperbilirubinemia, elevated hepatic enzyme levels, azotemia, and electrolyte imbalances (Ettinger *et al.,* 2024). Gross pathological lesions include generalized icterus, splenomegaly, hepatomegaly, petechial hemorrhages, pulmonary congestion, nephrosis, and lymphoid hyperplasia (Zachary, 2022).

This report describes the clinical, laboratory, gross pathological, and histopathological findings in a fatal case of canine babesiosis, with particular emphasis on the extensive multisystemic lesions in a Caucasian Shepherd bitch presented to the D.I.K Osori Veterinary Teaching Hospital, Usmanu Danfodiyo University Sokoto, Nigeria.

Case Report

Signalment and History

On 16-04-2026, the carcass of a 2-year-old intact female Caucasian Shepherd dog (*Canis lupus familiaris*), weighing approximately 25kg, was referred from the Small Animal Unit to the Post-Mortem Unit of the Department of Veterinary Pathology, D.I.K Osori Veterinary Teaching Hospital, Usmanu Danfodiyo University Sokoto.

The dog reportedly presented with a three-day history of profound weakness, anorexia, dyspnea, dehydration, jaundice, and heavy tick infestation. Clinical examination revealed pale and icteric mucous membranes, pyrexia (40.6°C), delayed capillary refill time, and generalized lymphadenomegaly. Blood smear examination revealed intraerythrocytic Babesia spp. organisms, supporting a diagnosis of canine babesiosis.

Diagnostic Methodology

Diagnostic evaluations were performed using standard clinical pathology protocols at the D.I.K. Osori Veterinary Teaching Hospital. Parasitological screening for intraerythrocytic hemoparasites was conducted on thin blood films fixed in absolute methanol for 3 minutes, stained with 10% Giemsa solution for 30 minutes, and examined under oil immersion (1000× magnification). Complete blood counts were determined using an HB7021 Automated Hematology Analyzer, while serum biochemical parameters were measured using an automated serum chemistry analyzer. Urinalysis was performed on freshly collected urine using multi-parameter urinalysis reagent test strips, with specific gravity determined using a clinical hand refractometer. For pathological evaluation, tissue specimens collected during post-mortem

examination were fixed in 10% neutral-buffered formalin for 24–48 h, routinely processed through graded ethanol and xylene, and embedded in paraffin wax. Sections were cut at 4 µm using a rotary microtome, stained with Hematoxylin and Eosin (H&E), and examined under light microscopy.

Laboratory Findings

Parasitological examination of whole blood revealed intraerythrocytic Babesia spp. organisms, and ticks collected from the dog were identified as Rhipicephalus spp. (Plate V). Hematological findings (Table 1) demonstrated severe macrocytic hypochromic anemia, characterized by markedly reduced packed cell volume (15%), hemoglobin concentration (4.8 g/dL), and erythrocyte count (1.93×10^6 cells/mm³). Mild monocytopenia and circulating band neutrophils were also noted.

Serum biochemical and electrolyte findings (Table 2) revealed marked hyperbilirubinemia, elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities, azotemia characterized by increased urea and creatinine concentrations, hyponatremia, hypochloremia, and decreased bicarbonate concentrations.

Urinalysis findings (Table 3) demonstrated marked blood positivity (++++), proteinuria (++), bilirubinuria (+), trace urobilinogen, urine pH of 6.0, and specific gravity of 1.030.

Table 1. *Hematological Findings in a Caucasian Shepherd Bitch with Canine Babesiosis*

Parameter	Obtained Value	Reference Range	Interpretation
PCV (L/L)	0.15	0.36–0.55	**Decreased**
Hemoglobin (g/L)	48	120–200	**Decreased**
RBC ($\times 10^{12}$/L)	1.93	5.4–8.5	**Decreased**
MCV (fL)	77.72	60–77	Slightly increased
MCH (pg)	24.87	19–24	Slightly increased
MCHC (g/L)	32	33–36	**Decreased**
WBC ($\times 10^9$/L)	14.0	6–18	Within reference range
Neutrophils ($\times 10^9$/L)	11.62	3–12	Upper reference limit
Lymphocytes ($\times 10^9$/L)	1.12	1–5	Within reference range
Monocytes ($\times 10^9$/L)	0.14	0.2–1.5	**Decreased**
Eosinophils ($\times 10^9$/L)	0.70	0.1–0.8	Within reference range
Basophils ($\times 10^9$/L)	0	Rare	Within expected range
Band Cells ($\times 10^9$/L)	0.42	0–0.45	Present

Source: Generated from case patient laboratory analysis, 2026.

Table 2. *Serum Biochemical and Electrolyte Findings in a Caucasian Shepherd Bitch with Canine Babesiosis*

Parameter	Obtained Value	Reference Range	Interpretation
Total Protein (g/dL)	6.4	5.5–9.0	Within reference range
Albumin (g/dL)	4.0	3.5–5.5	Within reference range
Total Bilirubin (mg/dL)	10.15	0.3–1.0	**Increased**
Direct Bilirubin (mg/dL)	1.6	0.1–0.3	**Increased**
AST (U/L)	170	10–40	**Increased**
ALT (U/L)	118	10–40	**Increased**
Creatinine (mg/dL)	2.6	0.7–1.3	**Increased**
Urea (mg/dL)	56.2	8.0–20.0	**Increased**
Chloride (mmol/L)	76.56	98–106	**Decreased**
Sodium (mmol/L)	130	136–145	**Decreased**
Potassium (mmol/L)	4.1	3.5–5.0	Within reference range
Bicarbonate (mmol/L)	22	23–28	**Decreased**

Source: Case laboratory records, D.I.K Osori Veterinary Teaching Hospital, 2026.

Table 3. *Urinalysis Findings in a Caucasian Shepherd Bitch with Canine Babesiosis*

Parameter	Result	Interpretation
Blood	++++	**Marked blood positivity**
Urobilinogen	Trace	**Present**
Ketones	Negative	Within normal limits
Bilirubin	+	**Bilirubinuria**
Glucose	Negative	Within normal limits
Protein	++	**Proteinuria**
Nitrite	Negative	Negative
Ph	6.0	Within expected range
Specific Gravity	1.030	Concentrated urine

Source: Case urinalysis report, 2026.

Case Management

Following clinical and laboratory diagnosis of canine babesiosis, the patient was placed on intensive supportive and specific therapeutic management. Fluid therapy was administered using 5% dextrose saline administered intravenously at a rate of 5 mL/kg/hour to correct dehydration and maintain circulatory support. Antimicrobial therapy was provided with crystalline penicillin at 20,000 IU/kg intravenously every 6 hours. Due to the heavy tick infestation, ivermectin (1%) was administered subcutaneously at 0.4 mg/kg as a single dose for ectoparasite control. Supportive therapy included intramuscular administration of vitamin B-complex (containing thiamine, riboflavin, pyridoxine, nicotinamide, and D-panthenol) at 1 mL/10 kg body weight and 10% iron dextran at 10 mg/kg intramuscularly to support hematopoiesis. Specific anti-babesial treatment was initiated using 12% imidocarb dipropionate administered subcutaneously at 6.6 mg/kg as a single dose. Despite treatment and supportive care, the patient's condition deteriorated progressively, and death occurred the following morning.

Gross Pathology

On external examination, the carcass was in poor body condition, with marked dehydration characterized by sunken eyes (Plate II) and reduced skin elasticity. Pale oral mucous membranes and generalized icterus were observed (Plate II). Numerous ticks were attached to various body regions (Plate IV), and mild epistaxis was present. Generalized lymphadenomegaly involved the prescapular (Plate VI), submandibular, inguinal (Plate VII), and mesenteric lymph nodes (Plate VIII). The spleen was markedly enlarged (Plate IX). Within the thoracic cavity, the lungs were swollen, heavy, and failed to collapse, with areas of consolidation and congestion (Plate X). The heart appeared mildly globular (Plate XI). Examination of the abdominal cavity revealed diffuse hepatomegaly with generalized icterus and multifocal petechiae on the hepatic surface (Plate XII). The gallbladder contained a black, hard, and non-odorous calculus (Plate XIII). Petechiatic hemorrhages were present on the pancreas (Plate XIV). The kidneys, particularly the left kidney, showed multifocal petechial hemorrhages involving the cortex and medulla (Plate XVI). Petechiation was also observed on the urinary bladder mucosa (Plate XV).



Plate I. Carcass on presentation



Plate II. Sunken eyes



Plate III. Pale and slightly yellowish oral mucous membranes discoloration



Plate IV. Numerous ticks present on the carcass body



Plate V. A stereomicrograph of the tick present (*Rhipicephalus spp*.)



Plate VI. Enlarged prescapular lymph node



Plate VII. Enlarged inguinal lymph node.



Plate VIII. Enlarged mesenteric lymph node.



Plate IX. Markedly enlarged, congested spleen (splenomegaly).



Plate X. Swollen, heavy lungs with multifocal areas of consolidation and congestion.

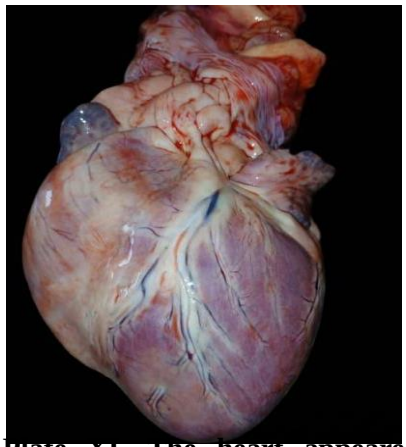


Plate XI. The heart appeared mildly globular.

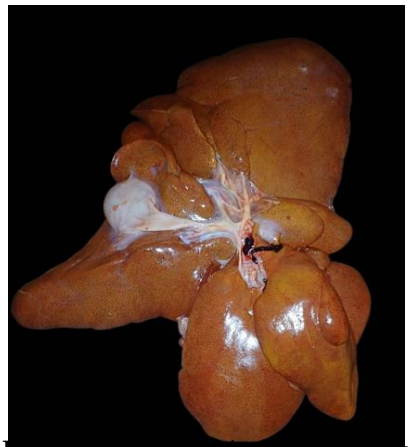


Plate XII. Icteric (yellow-orange) liver.



Plate XIII. A black, hard, non-odorous calculus found inside the gallbladder



Plate XIV. Petechial hemorrhages on the pancreas

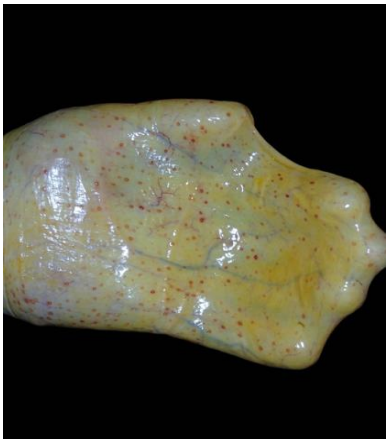


Plate XV. Petechiation on the urinary bladder mucosa.

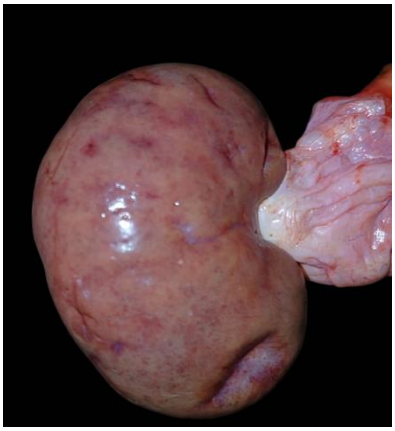


Plate XVI. Kidney showing multifocal petechial hemorrhages.

Histopathology

Representative tissue samples from the kidneys and lymph nodes were routinely processed and stained with hematoxylin and eosin (H&E). Among the examined tissues, renal lesions were the most prominent microscopic findings and were indicative of significant renal involvement in the disease process. The kidneys showed marked glomerular shrinkage with widening of Bowman's spaces, diffuse vascular congestion, multifocal interstitial hemorrhages, and tubular epithelial degeneration, consistent with acute renal tubular injury. Sections of the lymph nodes revealed marked cellular proliferation within the germinal centers and paracortical regions, indicating reactive lymphoid hyperplasia associated with systemic immune stimulation.

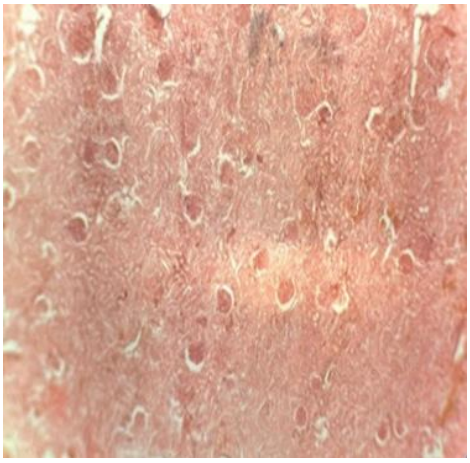


Plate XVII. A Photomicrograph (Mag. X100) showing shrunken glomeruli with noticeably widening of capsular spaces.

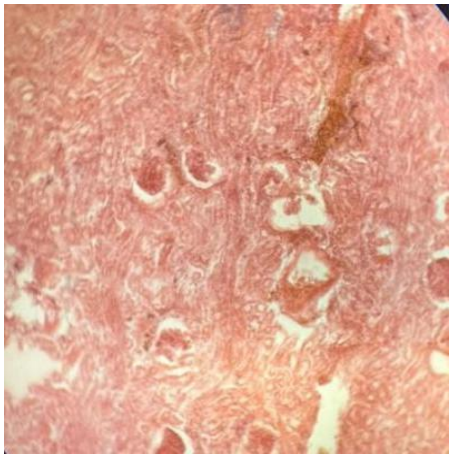


Plate XVIII. A Photomicrograph (Mag. X100) showing severe blood pooling (vascular congestion), bleeding into tissue (interstitial hemorrhages), and tubular epithelial degeneration.

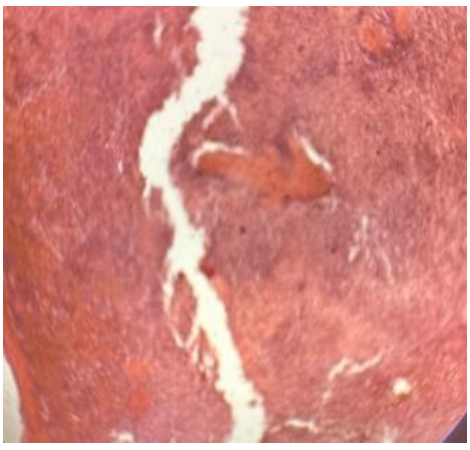


Plate XVIII. A Photomicrograph (Mag. X100) showing marked cellular proliferation within the germinal center and paracortical region.

Morphological Diagnosis

1. Severe hemolytic anemia associated with babesiosis
2. Generalized lymphadenitis with reactive lymphoid hyperplasia
3. Marked splenomegaly
4. Diffuse hepatomegaly with icterus
5. Multifocal renal petechiation with acute renal tubular injury
6. Pulmonary congestion and consolidation.
7. Multisystemic gross pathological involvement is characterized by hepatic, renal, pulmonary, splenic, and lymphoid lesions.

DISCUSSION

The clinicopathological, gross pathological, and histopathological findings in this case were consistent with severe complicated canine babesiosis. The presence of heavy tick infestation, marked anemia, jaundice, hemoglobinuria, and detection of intraerythrocytic *Babesia spp* confirmed an active hemoparasitic infection. Canine babesiosis is a globally significant tick-borne disease with variable severity ranging from mild self-limiting infection to fatal multisystemic failure, depending on host immunity, parasite virulence, and inflammatory response (Solano-Gallego *et al*., 2016; Köster *et al*., 2015).

The severe anemia observed in this case was attributable to both intravascular and extravascular hemolysis. This occurs through direct erythrocyte destruction by the parasite and immune-mediated mechanisms, including erythrophagocytosis, complement activation, and oxidative damage to red blood cells (Köster *et al.*, 2015; Zygner *et al.*, 2023). The resulting hemolytic crisis leads to tissue hypoxia, jaundice, and progressive clinical deterioration. Hyperbilirubinaemia and associated biochemical changes further reflect excessive hemoglobin breakdown and hepatic overload (Köster et al., 2015; Zygner et al., 2023).

Multisystemic involvement observed at necropsy indicates a systemic inflammatory response and progressive organ dysfunction, which are well documented in severe babesiosis (Bloch *et al*., 2022; Kuleš *et al*., 2017). Gross lesions involving the spleen, liver, lungs, kidneys, and lymphoid tissues highlight the widespread nature of this disease. However, histopathological confirmation was limited to the kidneys and lymph nodes due to sample constraints, which restricted full microscopic evaluation of other affected organs.

Renal lesions characterized by glomerular shrinkage, vascular congestion, tubular degeneration, and interstitial haemorrhage are consistent with acute kidney injury secondary to haemodynamic instability, hypoxia, and haemoglobinuria-associated nephrotoxicity (Köster *et al.*, 2015). Such renal involvement is commonly reported in complicated babesiosis and is associated with poor prognosis in severe cases.

Pulmonary congestion and consolidation observed grossly suggest significant respiratory involvement, likely resulting from vascular injury, inflammatory mediator release, and circulatory disturbances associated with systemic disease (Solano-Gallego *et al*., 2016). However, the lack of lung histopathology limited the definitive characterization of these lesions.

Marked splenomegaly and lymphoid hyperplasia reflect intense immunological activity and sequestration of parasitized erythrocytes, which are typical features of severe babesiosis (Köster *et al.*, 2015). These findings further emphasize the role of both innate and adaptive immune responses in disease progression.

The gallbladder calculus observed in this case was to be considered incidental. However, severe

intravascular hemolysis associated with babesiosis can lead to excessive bilirubin production and hepatobiliary overload, which may contribute to biliary stasis and pigment accumulation (Köster *et al.*, 2015).

Diagnosis was based on compatible clinical signs, hematological abnormalities, and microscopic identification of *Babesia spp*. on Giemsa-stained blood smears. Although this remains a practical diagnostic tool in field and clinical settings, its sensitivity is limited in cases of low parasitemia. Molecular diagnostic methods, such as PCR, offer higher sensitivity and allow species-level identification, which is important for epidemiological and therapeutic purposes (Krause *et al.*, 2021; Swanson *et al.*, 2023).

The combined effects of severe hemolytic anemia, systemic inflammatory response, and multiorgan dysfunction likely contributed to rapid clinical deterioration and death in this case. A limitation of this report was the absence of molecular confirmation and incomplete histopathological sampling, which restricted full characterization of the disease process.

CONCLUSION

This report describes a fatal case of canine babesiosis in a Caucasian Shepherd bitch characterized by severe hemolytic anemia, generalized lymphadenomegaly, splenomegaly, hepatopathy, pulmonary lesions, and renal injury. This case illustrates how canine babesiosis can be associated with widespread pathological changes affecting multiple organ systems, particularly when diagnosis and intervention occur late in the course of the disease. The combined clinical, laboratory, necropsy, and histopathological findings provided important insights into the disease process and its progression in this case. This case further emphasizes the importance of thorough clinicopathological evaluation when investigating fatal cases of canine babesiosis. A limitation of this report was the absence of molecular diagnostic testing, which prevented definitive species-level identification of the Babesia organism. Additionally, microbiological investigations for possible concurrent infections were not performed.

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