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Case Report

Clinico-Pathological and Molecular Characterization of Fatal Canine Leptospirosis: A Case Report from Nigeria

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ABSTRACT

Introduction: Spirochetes of *Leptospira* spp. are responsible for leptospirosis, a zoonotic bacterial infection that affects various animal species, particularly canines. The present study aimed to report the detailed clinical presentation, management, post-mortem examinations, and histopathological findings of a 5-year-old male Boerboel dog displaying signs of acute illness, including anorexia, constipation, and emesis.

Case report: The dog was presented to the small animal clinic, Department of Veterinary Services, Ministry of Agriculture and Food Systems, Ikeja, Lagos State, Nigeria. Initial physical examinations revealed lethargy, pale mucous membranes, and constipation; laxatives and enemas were administered. The dog's condition worsened, and then an exploratory laparotomy was performed, revealing a distended, obstructed intestine. Post-surgical care included antimicrobial and fluid therapy; however, the dog died a few days after the surgery. Necropsy revealed significant findings, including fatty liver, haemorrhagic enteritis, and lymphadenomegaly. Histopathological findings of the liver exhibited moderate hepatic lipidosis, inflammatory cell infiltration, and cholestasis. Renal samples exhibited interstitial nephritis and tubular degeneration. Reactive changes in gut-associated lymphoid tissues were observed in the intestinal samples. The polymerase chain reaction result for the *Leptospira 16S* rRNA gene was positive in the liver, confirming the diagnosis of canine leptospirosis.

Conclusion: The present study highlighted the importance of timely diagnosis and intervention in suspected cases of leptospirosis. The combination of clinical assessment, post-mortem findings, histopathological evaluation, and molecular diagnostics provided a comprehensive understanding of the disease process and emphasized the need for increased awareness and improved management strategies for canine leptospirosis in veterinary practice.

1. Introduction

The *Leptospira* (*L.*) sp. comprises pathogenic spirochetes responsible for leptospirosis, a prevalent zoonotic disease in the world[1]. These aerobic, gram-negative bacteria are classified in the Leptospiraceae family and can infect a wide variety of mammalian hosts, including humans, dogs, cats, and wildlife animals[1,2]. In canine populations, leptospirosis has been historically categorized as a clinically significant infection, frequently linked to renal and hepatic damage,

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and remains a notable contributor to morbidity and mortality internationally[3,4].

Transmission of leptospirosis primarily occurs through direct or indirect exposure to urine from infected animals[3]. Contaminated water, soil, or feed are major routes of transmission to incidental hosts such as canines[3]. The entry of *leptospira* sp. is facilitated through mucous membranes, abraded skin, or bite wounds, resulting in leptospiremia, leading to systemic distribution and then localization in the renal tubules[3].

Multiple factors influence the pathology of leptospirosis. Tissue damage occurs not only due to bacterial invasion but also from viral factors such as sphingomyelinases, hemolysins, porins, and lipopolysaccharides, which lead to endothelial injury, vasculitis, and inflammation responses[5]. Organ tropism differed based on the infecting serovar; *L. icterohaemorrhagiae* is more hepatotropic, whereas *L. canicola* primarily affects the kidney[5]. Clinical leptospirosis in dogs may range from per-acute, often fatal septicemia to acute or chronic forms characterized by fever, vomiting, dehydration, polyuria, polydipsia, and icterus; renal and hepatic failure are prominent manifestations[3,4].

Despite its significant clinical importance, leptospirosis remains underdiagnosed in resource-limited settings. Molecular techniques, such as polymerase chain reaction (PCR), now enable the rapid identification of *Leptospira* spp.[6]. Moreover, histopathological findings help identify lesion patterns, thereby improving diagnostic accuracy and precision. The present study aimed to provide a detailed history of a 5-year-old male Boerboel dog that presented with clinical signs consistent with acute leptospirosis, highlighting the importance of the diagnostic process, surgical intervention, and subsequent postmortem and histopathological findings.

2. Case report

A 5-year-old male brindle Boerboel was presented to the Department of Veterinary Services, Ministry of Agriculture and Food Systems, Lagos State, Nigeria, from May 25 to July 20, 2025. The present study was conducted in accordance with the ethical guidelines of the University of Ibadan, Ibadan, Oyo State, Nigeria, and all ethical guidelines were followed. Clinical observation was carried out along with a primary history of anorexia lasting approximately five days and constipation persisting for about seven days, accompanied by emesis. On initial clinical examination, the dog was lethargic, with a temperature of 38.8°C, a weight of 31 kg, and slightly pale mucous membranes. An abdominal X-ray was conducted, revealing no obstructions, but an impaction was observed in the lower gastrointestinal tract (Figure 1a).

2.1. Blood sampling

A total of 5 mL blood sample was collected from the cephalic vein of the dog using an Ethylenediaminetetraacetic acid (EDTA)-laden sample tube on the first day of presentation. Haematologic evaluation, such as packed cell volume (PCV, %), hemoglobin concentration (Hb, g/dl), red blood cell count (RBC, × 10^{12}/L), erythrocyte indices, leukogram, and thrombogram data, revealed a marked leukocytosis characterized by marked neutrophilia with a left shift and numerous immature neutrophils, accompanied by monocytosis (Table 1). The erythrogram demonstrated borderline low hematocrit (HCT, %), which might be suggestive of a mild non-regenerative anaemia. The reference values used were from Duncan and Prasse's Veterinary Laboratory Medicine[7].

Table 1. Haematological data of a 5-year-old male brindle Boerboel dog on the first day of presentation to the hospital, Nigeria

Parameters	Result	Reference interval *	Remarks
WBC count	43.7 × 10^9/L	5.0-14.1 × 10^9/L	High
Lymphocytes	2.0 × 10^9/L	0.4-2.9 × 10^9/L	Normal
Monocytes	7.9 × 10^9/L	0.1-1.4 × 10^9/L	High
Neutrophils	33.6 × 10^9/L	2.9-12.0 × 10^9/L	High
Eosinophils	0.1 × 10^9/L	0-1.3 × 10^9/L	Normal
Basophils	0.1 × 10^9/L	0-0.14 × 10^9/L	Normal
RBC count	6.4 × 10^{12}/L	4.95-7.87 × 10^{12}/L	Normal
Hemoglobin concentration	12.5 g/dl	11.9-18.9 g/dL	Normal
HCT	35.8 %	35-57%	Normal
MCV	55.7 fL	66-77 fL	Low
MCH	19.4 Pg	21.0-26.2 Pg	Low
MCHC	34.9 g/dL	32.0-36.3 g/dL	Normal
Platelet count	152.0 × 10^9/L	211-621 × 10^9/L	Low
MPV	11.5 fL	6.1-10.0 fL	High

*Reference value from Duncan and Prasse's Veterinary Laboratory Medicine[7]. WBC: White blood cells, RBC: Red blood cells, HCT: Haematocrit, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, MPV: Mean platelet volume

2.2. Serum chemistry

A total of 5 mL of blood sample was collected from the cephalic vein into a plain tube on the first day of presentation, and serum was separated for biochemical and electrolyte analysis. The biochemical parameters included sodium (mmol/L), potassium (mmol/L), chloride (mmol/l), bicarbonate (mmol/L), total calcium (mmol/L), ionized calcium (mmol/L), pH, anion gap (mmol/L), urea (mmol/L), creatinine (mg/dL), total protein (g/L), albumin (g/L), globulin (g/L), aspartate aminotransferase (AST, U/L), alanine aminotransferase (ALT, U/L), alkaline phosphatase (ALP, U/L), total bilirubin (µmol/L), direct bilirubin (µmol/L), indirect bilirubin (mmol/l), and GGT (U/L; Table 2). Hyponatraemia, hyperkalaemia, and hypochloraemia, with a metabolic acidosis resulting from elevated anion-gap and reduced bicarbonate levels, were observed. Azotaemia

was present, suggestive of both pre-renal and renal damage. There was a true hypocalcaemia (low total and ionized calcium) and a high level of AST. The reference values used were from Duncan and Prasse's veterinary laboratory medicine[7].

Table 2. Serum chemistry data for a 5-year-old male brindle Boerboel dog on the first day of presentation

Parameters	Result	Reference interval*	Remarks
Sodium	137.9 mmol/L	142-152 mmol/L	Low
Potassium	6.3 mmol/L	3.9-5.1 mmol/L	High
Chloride	94.9 mmol/L	110-124 mmol/L	Low
Bicarbonate	17.9 mmol/L	17-24 mmol/L	Normal
Total calcium	2.0 mmol/L	2.3-2.9 mmol/L	Low
Ionized calcium	1.0 mmol/L	1.2-1.4 mmol/L	Low
PH	7.4	7.32-7.42	Normal
Anion gap	25.5 mmol/L	8.0-16.0 mmol/L	High
Urea	2.7 mmol/L	2.9-10.0 mmol/L	Low
Creatinine	1.9 mg/dL	0.5-1.7 mg/dL	High
Total protein	67.4 g/L	54-75 g/L	Normal
Albumin	24.9 g/L	23-31 g/L	Normal
Globulin	42.5 g/L	27-44 g/L	Normal
AST	90.8 U/L	13-15 U/L	High
ALT	13.4 U/L	10-109 U/L	Normal
ALP	112.0 U/L	1-114 U/L	Normal
Total bilirubin	8.9 µmol/L	1.71-10.3 µmol/L	Normal
Direct bilirubin	4.8 µmol/L	0.0-5.1 µmol/L	Normal
Indirect bilirubin	4.1 µmol/L	1.7-5.1 µmol/L	Normal
GGT	6.8 U/L	1.0-9.7 U/L	Normal

*Reference value from Duncan and Prasse's Veterinary Laboratory Medicine[7]. AST: Aspartate aminotransferase, ALT: Alanine transaminase, ALP: Alkaline Phosphatase, GGT: Gamma-glutamyl transferase

2.3. Case management

While a consultant veterinary radiologist who specialized in radiography analyzed the radiological results, conservative management was started in accordance with the clinical presentation and recommendations of the Plumb's veterinary drug handbook[8]. The case management therapy included the administration of laxatives; 15 mL of paraffin oil (Moko, Nigeria) administered hourly, three tablets of 5 mg of bisacodyl orally, and an enema (Unicure, Nigeria) with warm soapy water. Following the enema, the animal voided bloody and mucoid faces.

2.4. Surgical procedure

Despite conservative treatment, no significant improvement was observed, leading to a decision to perform an exploratory laparotomy, which was carried out as described by Pastore et al.[9]. During the surgical procedure, the large intestine was found to be distended with gas, although no obstructions were noted. Post-surgical care included the administration of 3 mL penicillin-streptomycin at 10 mg/kg (Kepro, Holland) and 100 mL dextrose 10% Intravenously (Tuyil, Nigeria) as recommended in the Plumb's veterinary drug handbook[8]. On July 15, 2025, the animal's temperature dropped to 37.4°C, and it passed watery, bloody faces. Unfortunately, the dog died on July 16, 2025, at 8:10 AM, leading to a post-mortem examination.

2.5. Post-mortem findings

The post-mortem examination revealed significant findings consistent with severe systemic disease. The liver was diffusely yellow and firm (Figure 1b). The entire length of the intestine was dark, tarry blood mixed with mucus. The spleen had a wrinkled parenchyma. The rectal tonsils were hemorrhagic, swollen, and embedded within the mucosa (Figure 1c).

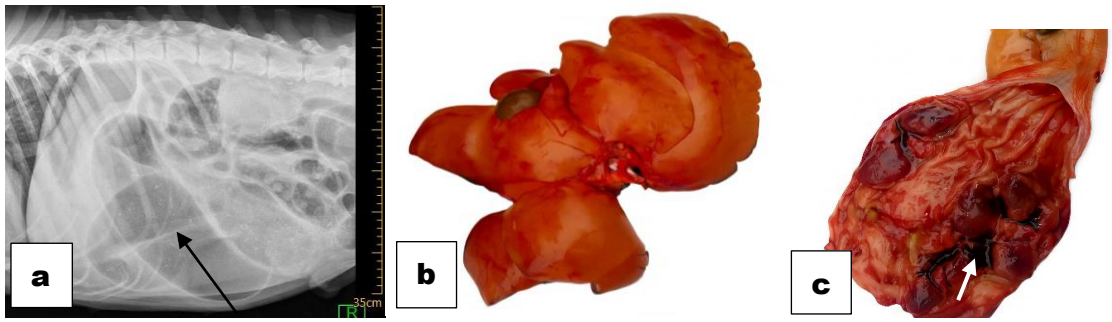

Figure 1. Radiograph imaging and post-mortem findings of a 5-year-old male brindle Boerboel dog. a: lateral view of the abdominal x-ray from the radiograph imaging of the abdomen showing impaction on the lower gastrointestinal tract, black arrow. b: An enlarged and diffusely yellow liver was taken during post-mortem examination. c: The swollen and haemorrhagic rectal tonsils were taken during post-mortem examination, white arrow. Source of the images: Authors of the present study.

2.6. Histopathology

Samples from liver, kidney, and intestine were collected and fixed in buffered formalin 10% for histology. Tissue from each organ was trimmed, placed in a tissue cassette, and properly labelled before the histological processing and H&E satining[10]. Histopathological evaluation of the collected tissue samples provided valuable insights into the underlying pathological changes using hematoxylin and eosin stain and viewed under a microscope (Olympus, Japan). The observations of tissue samples under a light microscope showed the following results. The liver sections demonstrated moderate dissociation of hepatocytes from their arrangement in parenchymal cords (Figure 2a). Hepatocytes with cytoplasmic lipid vacuoles of varying sizes exhibited mild to moderate liver damage and lipidosis (Figure 2b). Multifocal periportal areas were infiltrated by mixed leukocytes, predominantly neutrophils, lymphocytes, and plasma cells. Increased mitotic activity was noted, consistent with a regenerative response, and multifocal cholestasis was observed with distended bile canaliculi. The kidney exhibited lymphoplasmacytic interstitial nephritis characterized by dense infiltration of lymphocytes and plasma cells (Figure 2c). Degenerative changes in the renal tubular epithelium included cytoplasmic swelling, vacuolation, and loss of brush border (Figure 2d). In chronic cases, thickening of Bowman's capsules and partial encasement of glomeruli in fibrous connective tissue were noted, consistent with chronic glomerular fibrosis. Significant gut-associated lymphoid tissue (GALT) reactivity was observed in the intestinal mucosa, along with hypertrophy of Peyer's patches and isolated lymphoid follicles. The lamina propria and submucosa were infiltrated by small lymphocytes and plasma cells, while the overlying mucosal epithelium exhibited mild flattening and focal intraepithelial lymphocytic infiltration.

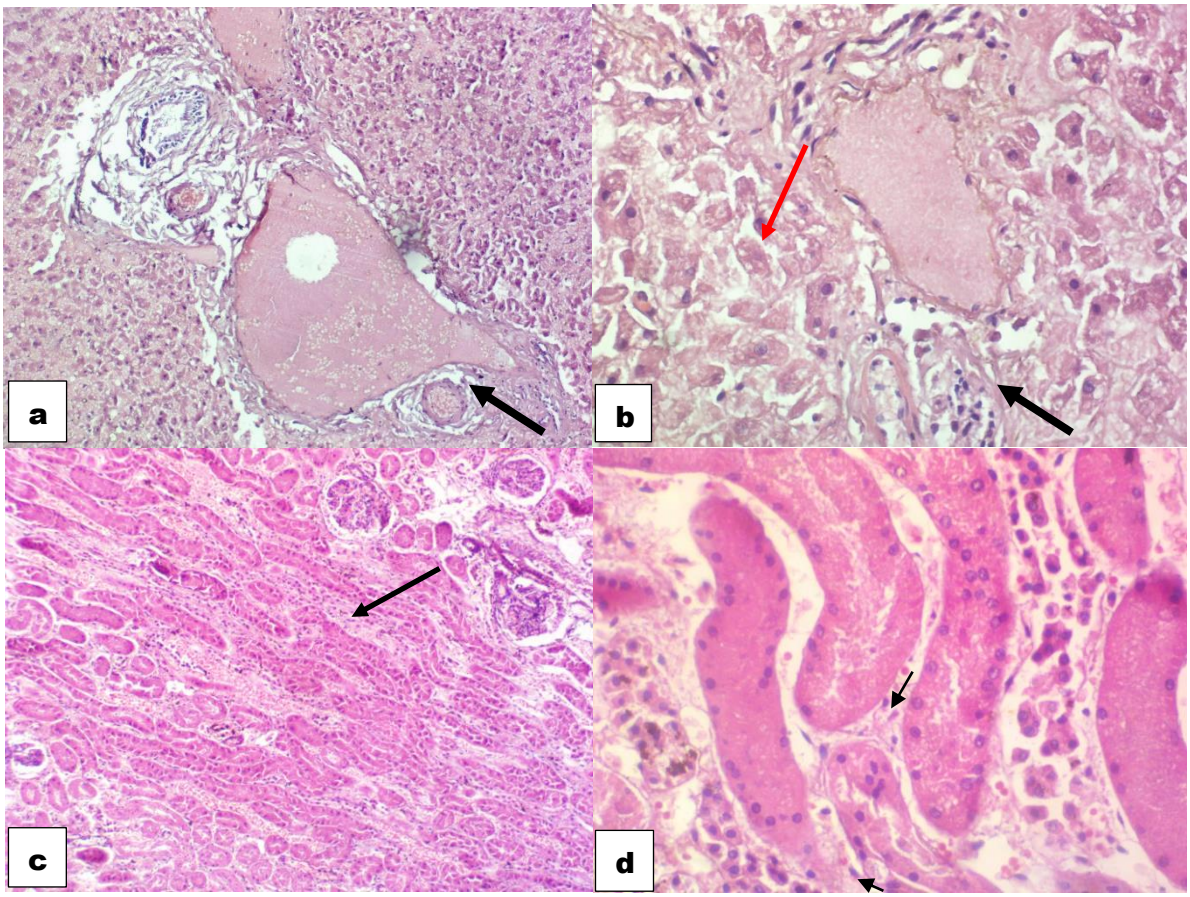

Figure 2. Histopathological results of different tissues in a 5-year-old male brindle Boerboel dog. a: Liver showing cholestasis, black arrow (H&E, 100x). b: Liver showing mild steatosis (red arrow), dissociation of hepatic cords, and periportal infiltration by leukocytes, black arrow (H&E, 400x). c: Kidney showing dense interstitial infiltration of leukocytes, black arrow (H&E, 100x). d: Kidney showing renal tubular epithelial degeneration and infiltration of the interstitium by lymphocytes and plasma cells, black arrows (H&E, 400x).

2.7. Polymerase chain reaction

To further investigate the etiology of the dog's condition, three frozen samples of the liver, intestine, and spleen were submitted for PCR antigen detection using pathogen-specific *Leptospira* spp. primer. The primer targets a portion of the *16S* ribosomal RNA gene, including LEP-16Sf - 5'- CAT GCA AGT CAA GCG GAG TA -3'[11] and LEP-163R – 5'- CTT AAC TGC TGC CTC CCG TA -3'[12]. The DNA extraction, PCR amplification, and analysis were performed according to the methods described by Alaka et al.[13]. As shown in Figure 3, only the liver sample tested positive for the pathogenic *Leptospira* spp. antigen at

300 bp, confirming a molecular diagnosis of acute canine leptospirosis.

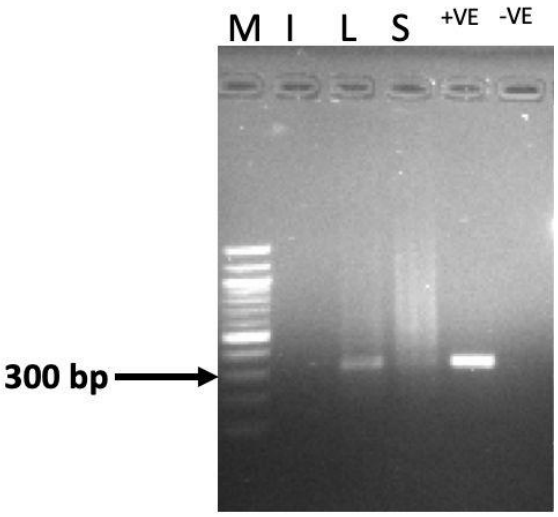

Figure 3. Detection of *Leptospira* spp. in a 5-year-old Boerboel dog via PCR. Positive bands are seen on the liver at 300 bp. M: 100 bp ladder, I: Intestine sample, L: Liver sample, S: Spleen, +ve: Positive control, -ve: Negative control.

3. Discussion

Leptospirosis is a zoonotic disease of major global importance. Detecting canine leptospirosis is critical for human public health, as infected dogs can contaminate the environment and serve as sources of *Leptospira* sp. that infects humans, underscoring the necessity of a One Health approach for surveillance and control[1,3]. The diagnostic challenge highlighted in the present study reflected a well-documented global challenge in canine leptospirosis, as supported by Costa et al.[4]. Leptospirosis is prevalent among dog populations worldwide, especially *L. Canicola* and *L. Icterohaemorrhagiae* being the most frequently detected species[4].

The haematology results indicated a severe systemic inflammatory process, with tissue damage; these changes were consistent with what has been reported by van de Maele et al.[3] in dogs with acute leptospirosis[3]. Thrombocytopenia observed in the present study was a typical finding and may have resulted from vasculitis, endothelial damage, and disseminated intravascular coagulation, which was consistent with the findings of Barthélemy et al.[14].

Disruption of the clotting system is another essential sign of leptospirosis[14]. Many infected dogs illustrated prolonged clotting times and platelet dysfunction, reflecting the combined effects of systemic vasculitis and activation of the coagulation cascade[13]. Although coagulation assays were not performed in the present study, the presence of thrombocytopenia and widespread haemorrhage at necropsy strongly suggested a haemostatic imbalance, similar to the findings of Alaka et al.[13].

The biochemical profile further supported systemic infection across multiple organs. The post-mortem changes observed in the liver and kidneys, including hepatocellular lipidosis, cholestasis, lymphoplasmacytic tubulo-interstitial nephritis, and renal tubular degeneration, were suggestive of renal disease and hepatic damage. These findings are consistent with those of Barthélemy et al.[14], who reported co-infection with leptospirosis and aflatoxicosis, in which significant hepatic lesions, such as bile ductular hyperplasia and extensive necrosis, were documented. The involvement of these two organs (liver and kidney) reflected the well-known ability of different *leptospira* serovars to preferentially target specific tissues[5,15].

Ultimately, molecular analysis confirmed the diagnosis, with PCR detecting *Leptospira* spp. DNA from the liver that was analysed during the present study. The high prevalence of leptospirosis reported by Ajayi et al.[16], who employed multiple methods including culture, serology, and immunohistochemistry, highlighted the technical complexity and practical limitations of culturing, which PCR effectively overcomes in clinical settings. Culturing method remains the globally accepted method, but it is not practicable due to its technical demands and slow turnaround as reported by Ahmed et al.[6] and Ajayi et al.[16]. The negative results from the spleen and intestines further confirmed that the pathogen prefers renal and hepatic organs, making PCR a crucial method for quick and accurate detection[3].

4. Conclusion

The present study underscored the importance of considering leptospirosis as a differential diagnosis in dogs with severe systemic disease. Careful interpretation of haematology, pathology, and PCR was essential to reaching an accurate diagnosis. Clinicians should always have leptospirosis as a differential diagnosis when dogs present with hepatic and renal disorders, especially in endemic regions such as Nigeria. The present study highlighted that canine leptospirosis is a significant health concern in Nigeria. Timely diagnosis not only improves clinical outcomes for dogs but also reduces the risk of environmental contamination and human exposure, reinforcing the importance of a One-Health approach to tackling the neglected zoonosis. Further immunological studies should be conducted to evaluate the impact of

vaccination on the progression, incidence, and prognosis of leptospirosis in dog populations.

Declarations

Ethical considerations

The authors declared that this original case report has not been published or submitted elsewhere. The manuscript underwent plagiarism screening using standard software. AI tools were not used in preparing and writing the present study.

Competing interests

There was no conflict of interest to declare in the present study.

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Availability of data and materials

Data supporting the present study are included within the article.

Authors' contributions

The concept of the manuscript was developed by Olawale Olatunde Modupeoluwa and Billy Quadri Omoniyi, the literature search was done by Taiwo Oluwatosin and Titilope Oluwatoyin Olagbegi. Bello Olaoluwa Isaac and Margaret Omotayo Owolabi performed the post-mortem examination. The original draft was written by Abdulrauf Adekunle Usman and Olanrewaju Samuel Olaifa, and reviewed by Onyinye Ikejiofor and Tolulope Akin-Fawole. Olanrewaju Samuel Olaifa wrote the post-mortem findings. The clinical management was done by Bello-Ibiyemi Abdulrahman Oluwatosin. Olawale Olatunde Modupeoluwa and Abdulrauf Adekunle Usman performed the molecular diagnosis. All authors have read and confirmed the final edition of the manuscript.

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