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Case Report

Bilateral Valvular Endocarditis with Multiple Renal Infarcts in an Adult Pit-Bull: A Case Report

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ABSTRACT

Introduction: Vegetative valvular endocarditis is an uncommon but potentially fatal condition in dogs, often diagnosed post-mortem due to nonspecific clinical presentation and rapid progression. The present case report aimed to document the post-mortem and histopathological findings specific to bilateral valvular endocarditis with septic embolism-induced renal infarcts in a 9-year-old male Pit Bull.

Case report: A 9-year-old male Pitbull presented with anorexia, exercise intolerance, recumbency, and a haemorrhagic scrotal mass. Clinical evaluation revealed hyperthermia, tachycardia, laboured respiration, and mitral regurgitation. The dog died despite intervention and was necropsied. Tissue samples taken from the heart, kidney, lungs, and liver were fixed in 10% buffered formalin, sectioned at 5 µm, and stained with haematoxylin and eosin. Endocardial samples were cultured on MacConkey and Blood agar. Necropsy revealed vegetative thrombotic endocarditis (Mitral valve), bronchopneumonia, renal infarctions, splenomegaly, and hindlimb myositis. Histology examinations confirmed fibrinous thrombi with bacterial colonies, pulmonary oedema, and hepatic chord atrophy, Kupffer cellular hyperplasia, and renal tubular thromboembolism and necrosis. *Staphylococcus aureus* and *Klebsiella* spp. were isolated from the endocardial samples and confirmed by Gram stain and biochemical tests such as catalase, indole, citrate, and oxidase tests.

Conclusion: The present case demonstrated the systemic implications of canine valvular endocarditis, particularly its association with renal infarction and multi-organ pathology. Given the rarity of the condition, with an estimated prevalence of only 0.11% in dogs, the present report contributes to the expanding knowledge on understanding and managing similar cases in veterinary practice.

1. Introduction

Endocarditis is a life-threatening condition characterized by the formation of thrombotic vegetations on heart valves, often leading to systemic embolization[1]. Valvular endocarditis is an inflammatory disease affecting the heart valves, often linked to high morbidity and mortality. In dogs, several studies indicated mortality rates for infective endocarditis can reach 50-70%, especially when the kidneys are involved present[1,2]. In veterinary medicine, endocarditis is a condition most often diagnosed post-mortem in dogs, typically associated with cardiovascular compromise and systemic embolic events[3]. The pathogenesis of endocarditis typically involves bacterial colonization of heart valves, often following bacteremia due to dental disease, skin infections, or other

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sources of infection[4,5]. The resulting vegetative lesions can cause valve dysfunction, leading to heart failure and embolic complications, including renal infarcts[4,5].

Renal infarcts occur when blood flow to a portion of the kidney is obstructed, commonly due to emboli originating from the heart[6]. These infarcts can result in acute kidney injury and chronic renal disease[2,3]. In dogs, the clinical presentation of renal infarcts can differ, ranging from asymptomatic findings on routine examination to severe clinical signs[7-9]. Renal infarcts are common, with studies indicating that 38 out of 46 dogs with endocarditis experienced kidney involvement caused by embolic events[1].

Valvular endocarditis in dogs typically presents with a range of clinical signs, including lethargy, anorexia, fever, heart murmurs, and signs of renal failure such as hematuria and azotemia. The duration of the clinical disease may be either acute or chronic and results in a high mortality rate if untreated[8,10,11]. The primary aim of therapy for infective endocarditis is to manage congestive heart failure, resolve arrhythmias, sterilize vegetative lesions, and prevent further spread of infection[9,12]. Management of infective endocarditis should start with blood cultures to determine the causative organism and conduct sensitivity tests to inform effective antibiotic therapy selection. Initial management includes 1 to 2 weeks of parenteral broad-spectrum bactericidal antibiotics, such as Enrofloxacin (5-10 mg/kg) and Ceftriaxone (25-30 mg/kg), often most effective when combined with Gentamicin, all administered intravenously, followed by a 6-8-week course of oral antibiotics, such as Doxycycline (10 mg/kg) or Amoxicillin-Clavulanate (12.5-25 mg/kg), with either given orally twice daily[12]. Empirical therapy demonstrated improved outcomes with combinations that include gentamicin[12]. Renal function should be monitored due to the nephrotoxic potential of certain medicines such as gentamicin[11].

Endocarditis is relatively rare in dogs, with a prevalence of approximately 0.11% in a study of 51,655 cases, and males are more frequently affected, particularly in the 7-9 years age group[12]. Affected valves often include the mitral and aortic valves, with the mitral valve being the most commonly involved in significant cases leading to systemic embolization[2-4].

Although endocarditis in dogs is uncommon, the seriousness of the disease and the difficulties in diagnosing and treating it highlight the need for increased veterinary awareness and studies in this area. The present study aimed to describe a fatal presentation of vegetative valvular endocarditis with associated pulmonary, renal, and muscular complications in a 9-year-old Pitbull.

2. Case report

A 9-year-old male Pitbull weighing 32 kg was brought to the Small Animal Clinic at the Veterinary Teaching Hospital, University of Ibadan, Nigeria, on November 14, 2024. The dog had a history of progressive loss of appetite and activity, accompanied by a solitary, haemorrhagic and encrusted mass on the surface of the scrotum. The scrotal mass was reportedly observed three weeks prior to the current examination. At the time of presentation, the dog was recumbent yet exhibited adequate body condition. No ectoparasites were observed, and the mucous membranes were slightly pale but moist.

There was moderate swelling of the right hind limb, and the owner reported that the limb had been partially lame three days before the dog became recumbent. The swollen limb was notably cold to the touch, whereas all other limbs were warm. The owner further reported that the dog had a reduced appetite and exhibited exercise intolerance for the past three weeks. On clinical examination, the rectal temperature was 40.7°C (Hyperthermia), with a pulse rate of 160 beats per minute and a respiratory rate of 68 breaths per minute. Laboured breathing and coarse rales were detected upon auscultation. Electrocardiogram (ECG) revealed ventricular premature contractions. The animal's condition deteriorated during clinical management, and it died the same day. A post-mortem examination was subsequently performed as requested by the client.

2.1. Post-mortem findings

The carcass has a body condition score within normal limits, but the ocular and oral mucous membranes were severely pale, indicating anaemia. A firm, haemorrhagic, encrusted mass measuring approximately 2.5 cm in diameter was observed on the scrotum (Figure 1A). The mass was a healing traumatic wound. The spleen was markedly enlarged, dark, and exuded blood on the cut surface, indicative of severe congestive splenomegaly (Figure 1B). Ecchymotic haemorrhages were on the epicardium (Figure 1C). The fundic region of the stomach showed multiple healing ulcers and ecchymosis (Figure 1D). There was a localized swelling on the right hind limb, and upon excision of the affected muscles, there was evidence of oedema, hyperemia, and purulent discharge consistent with suppurative myositis.

The lungs were bilaterally firm with cranioventral consolidation affecting approximately 40% of the lung parenchyma. The affected lobes of the lungs, including most of the cranial, middle, and caudal lobes, were dark, hyperaemic, and firm, suggestive of bronchopneumonia (Figure 2A). On the endocardium, large, raised, red thrombotic masses measuring between 1 and 3 cm were attached to the cusps of the mitral and tricuspid valves, indicating vegetative valvular endocarditis (Figure 2B and C). Multifocal haemorrhagic and pale infarcts measuring between 5 mm and 2 cm were observed in the kidneys (Figure 3 A to C).

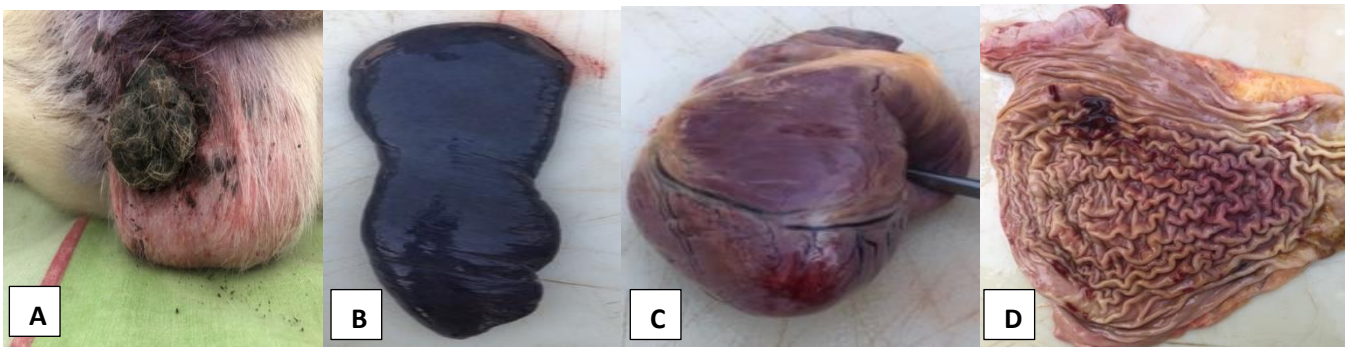

Figure 1. Gross lesions associated with bacterial endocarditis in a 9-year-old male Pit Bull. A: An encrusted scrotum, B: The spleen is markedly congested, suggestive of vascular stasis or systemic infection, C: The heart displays epicardial haemorrhages, D: The gastric mucosa reveals haemorrhagic ulcers.

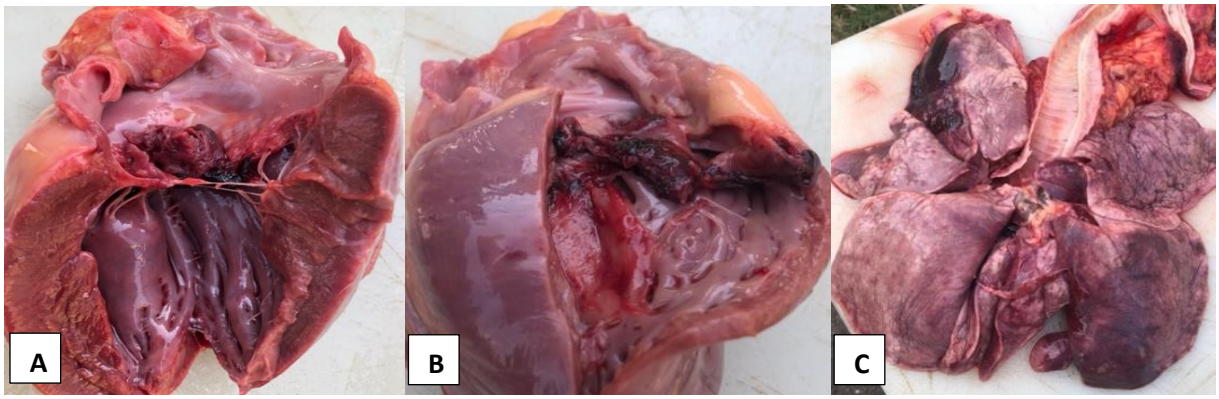

Figure 2. Heart and lung lesions associated with bacterial endocarditis in a 9-year-old male Pit Bull. A and B: Cut sections of the heart reveal vegetative growths on the atrioventricular valves, consistent with endocarditis, C: The lungs exhibit cranioventral consolidation, characterized by firm, dark red lobes suggestive of bronchopneumonia.

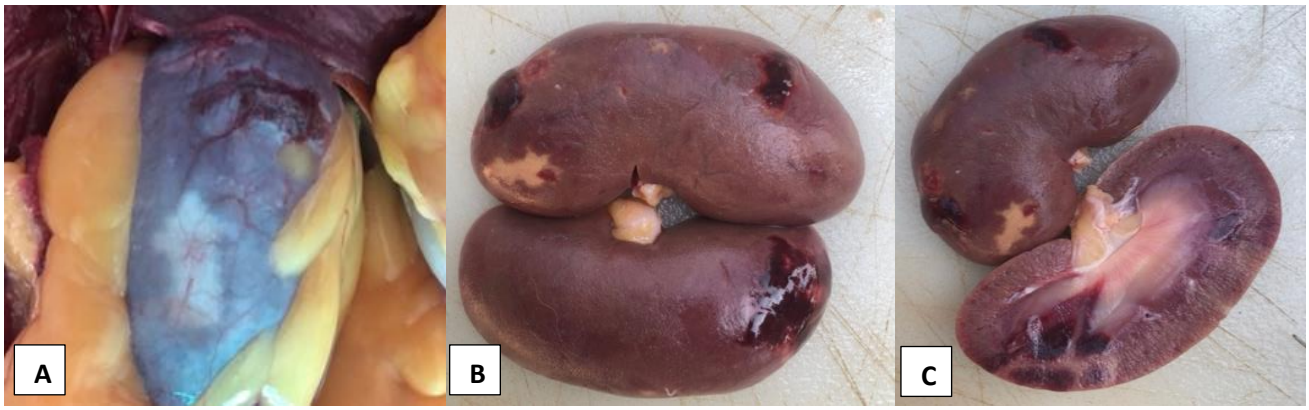

Figure 3. Kidney lesion associated with bacterial endocarditis in a 9-year-old male Pit Bull, showing red and pale infarcts. A: In-situ view of the kidney, B: Cortical surface of the kidney, and C: Cut surface of the kidneys reveal multifocal red infarcts indicative of hemorrhagic necrosis and multifocal pale infarcts, suggesting areas of arterial occlusion and tissue ischemia.

Following post-mortem examination, an endocardial vegetations sample from the heart was aseptically collected and inoculated onto MacConkey agar (Merck KGaA, Germany) and Blood agar plates (Becton Dickinson, BD Difco™ Blood Agar, United States of America) and incubated aerobically at 37°C for 24 hours[13-15]. Colonies suggestive of *Klebsiella* spp. appeared on MacConkey agar as large, pink, mucoid colonies, indicative of lactose fermentation. On Blood agar, cream-colored colonies were observed, which were presumptively identified as *Staphylococcus* (*S.*) spp.

Gram staining was performed on representative colonies. *Klebsiella* spp. appeared as Gram-negative rods, while *Staphylococcus* spp. appeared as Gram-positive cocci arranged in clusters. Presumptive isolates were further characterized using conventional biochemical tests; *Klebsiella* spp. were catalase-positive, indole-negative, citrate-positive, oxidase-negative, and non-motile. The *S. aureus* was identified by positive catalase and coagulase reactions[16].

The morphological, staining, and biochemical characteristics confirmed the presence of *Klebsiella* spp. and *S. aureus* in the processed samples.

Following the processing method detailed by Al-Sabawy et al.[17], tissue samples taken during post-mortem examination were preserved in 10% neutral buffered formalin. These samples were reconstituted from Formaldehyde 40% W/Vol (Brentag NV, Deerlijk, Belgium), then dehydrated through increasing alcohol concentrations,

cleared in xylene, and finally embedded in paraffin wax. Sections were cut at a thickness of 5 µm from the paraffin blocks, affixed to glass slides, and stained using haematoxylin and eosin (H&E) for microscopic examination. The stained sections were examined under a light microscope (Olympus CX21, Japan) at 100 and 400 magnifications. Photomicrographs were captured using a DinoEye eyepiece digital camera (AnMo Electronics Corporation, version 2016Q3 IDCP BV, Taiwan).

Microscopic examination of the lungs exhibited alveolar spaces filled with homogeneous eosinophilic material, indicating pulmonary oedema (Figure 4A). There was evidence of bacterial colonies scattered within the pulmonary parenchyma (Figure 4B). Thrombi were observed within several blood vessels, and rupture of inter-alveolar septa along with haemorrhage were noted in a few alveoli (Figure 4C). The pulmonary interstitium and perivascular areas exhibited polymorphonuclear cellular infiltration, and hemosiderin-laden macrophages were present throughout the parenchyma. There was evidence of bacterial colonies scattered within the pulmonary parenchyma (Figure 4B). The liver had diffused hepatic cord atrophy, Kupffer cell hyperplasia with hemosiderosis, and severe lymphocytic infiltration of the hepatic parenchyma (Figure 5A). Focal areas of fibrosis, hepatocyte loss, hepatic cord disruption, and individualization of hepatocytes were observed (Figure 5B). Sections of the endocardial growths (Figure 6A) on the mitral and tricuspid valves revealed fibrin strands interspersed with red blood cells and numerous clusters of round-shaped bacterial colonies (Figure 6B), confirming the presence of vegetative valvular endocarditis. The kidneys exhibited widespread coagulative necrosis of the renal tubular epithelium (Figure 7). Additionally, homogeneous eosinophilic material was observed within Bowman's spaces, forming crescentic structures, accompanied by thrombi in the interstitium (Figure 7A), with dense neutrophilic infiltration in the renal interstitium and occasional mononuclear cells (Figure 7B), and the presence of bacterial colonies (Figure 7C).

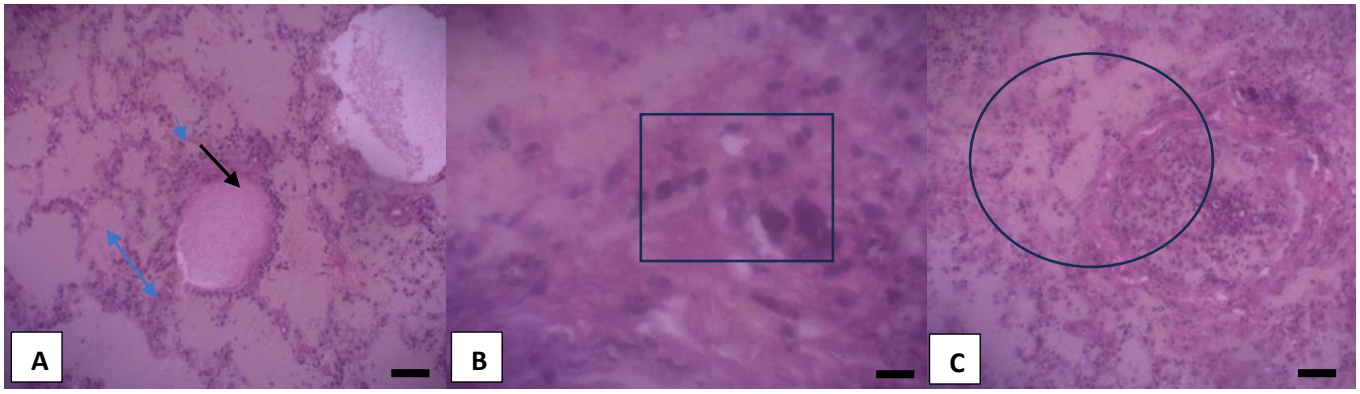

Figure 4. Lung lesions associated with bacterial endocarditis in a 9-year-old male Pit Bull. A: Pulmonary capillaries are occluded by amorphous eosinophilic thrombi (Black arrow), accompanied by perivascular inflammation (Perivasculitis, blue arrow) and severe pulmonary edema (Blue double arrowheads, 100x), B: Focal neutrophilic infiltration is observed surrounding a bacterial colony (Box), indicative of bacterial pneumonia (400x), C: Additional capillary thrombi are evident (100x), associated with both cellular and fluid exudates (Circled), reflecting mixed inflammatory and vascular involvement. H&E staining, Scale bar = 0.5 µm.

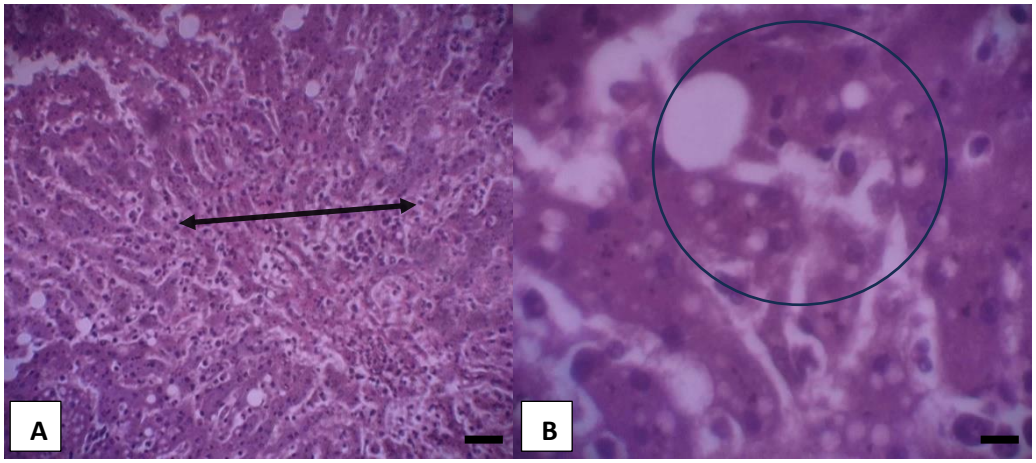

Figure 5. Liver lesions associated with bacterial endocarditis in a 9-year-old male Pit Bull. A: The liver shows diffuse hepatic cord atrophy (Double-edged arrows), indicative of parenchymal degeneration (100x). There is marked Kupffer cell hyperplasia accompanied by hemosiderosis, suggesting increased erythrophagocytosis. Moderate lymphocytic infiltration is observed within the hepatic parenchyma, B: Focal fatty infiltration is evident (Circled), consistent with hepatocellular injury (400X). H&E staining, Scale bar = 0.5 µm.

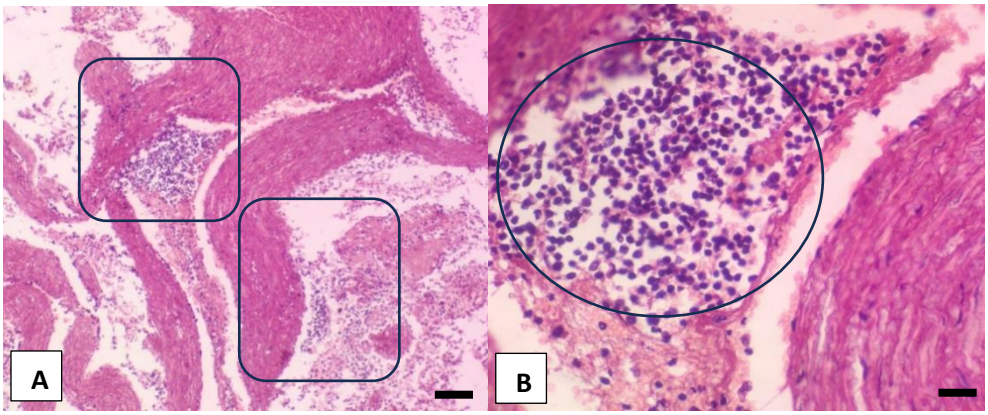

Figure 6. Heart valve lesions associated with bacterial endocarditis in a 9-year-old male Pit Bull. A: The valve surface reveals irregular fibrin strands admixed with red blood cells and numerous round-shaped bacterial colonies arranged in clusters, interspersed with inflammatory cells (Square, 100x). B: Vegetative lesions on the endocardial surface show fibrin interwoven with mononuclear inflammatory cells and clustered bacterial colonies (Circled), consistent with infective endocarditis (400x). H&E staining, Scale bar = 0.5 µm.

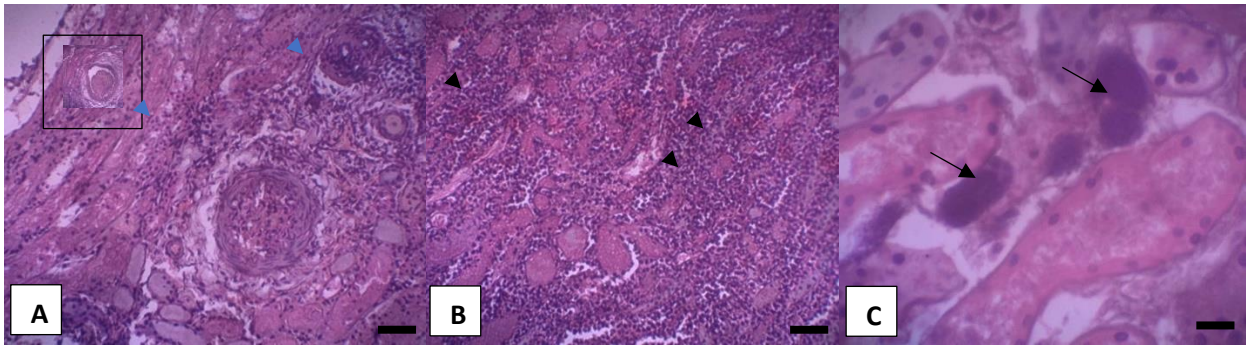

Figure 7. Kidney Lesions associated with bacterial endocarditis in a 9-Year-Old male Pit Bull**.** A: The renal cortex shows diffuse tubular necrosis with intraluminal deposition of eosinophilic protein casts. A small arteriole exhibits thromboembolic occlusion (Square, 100x), and mild perivascular mononuclear cell infiltration is observed (Blue arrowhead), B: The interstitium reveals severe inflammatory cellular infiltration (Black arrowheads, 100x), indicating tubulointerstitial nephritis, C: Clusters of bacterial colonies are visible within renal tissue (Black arrows, 400x), suggesting septic embolic nephritis. H&E staining, Scale bar = 0.5 µm.

3. Discussion

The post-mortem and histopathological findings confirmed a diagnosis of vegetative valvular endocarditis with systemic thromboembolism, leading to renal infarcts, pulmonary oedema, and splenomegaly. The presence of bacterial colonies in the heart valves and lungs suggested a bacterial septicaemia, likely originating from the scrotal mass or an unidentified infection site. The hindlimb ischemia and suppurative myositis further support systemic thromboembolic disease[1, 3,4,12].

The development of bronchopneumonia and pulmonary oedema likely contributed to respiratory distress, ultimately leading to death[3,12]. The presence of ventricular premature contractions on ECG suggested myocardial compromise, possibly due to septic emboli affecting coronary circulation[3,18].

Bilateral valvular endocarditis is a significant cardiovascular condition in dogs, characterized by inflammation of the heart valves due to bacterial infection[3, 12]. The present case described a 9-year-old male Pit-Bull diagnosed with bilateral valvular endocarditis and multiple renal infarcts. The complexity and systemic effects of this condition enhanced understanding of its clinical and pathological significance for veterinarians.

Valvular endocarditis usually occurs after bacteria colonize the heart valves, often due to bacteraemia from dental issues, skin infections, or other sources sources[16]. In the current case, the dog presented with clinical signs such as lethargy, anorexia, and hyperthermia, which are consistent with those reported in similar cases[5,8]. The occurrence of renal infarcts as a complication of endocarditis has been documented, with studies indicating that up to 38 out of 46 dogs with endocarditis exhibited kidney involvement due to embolic events[3,17]. As supported by Wojda et al.[19], vegetative endocarditis as a condition is systemic and often associated with significant renal compromise.

Gross post-mortem examination revealed severe anaemia, which is common in cases of endocarditis due to chronic disease and potential blood loss from embolic events[19,20]. The presence of a crusted mass on the scrotum and the localized swelling and purulent discharge in the right hind limb further suggested a systemic inflammatory response, indicative of the severe clinical presentation of the dog's condition.

Histopathological examination of the renal tissue exhibited evidence of ischemic necrosis and inflammation due to embolic occlusion of renal blood vessels, and the liver showed coagulative necrosis. Microbiological investigations are crucial in confirming the aetiology of valvular endocarditis[13]. Bacterial cultures from the vegetative lesion

in the cardiac tissue yielded heavy growths of *Klebsiella* sp. and *S. aureus* as the causative organisms of the bacteraemia.

The characterization of *Klebsiella* spp. through conventional biochemical tests revealed distinct traits that aid in their identification[21-24]. Diagnosing endocarditis is challenging because of its nonspecific signs, which often result in delays detection[3,21,25]. Treatment is further complicated by the presence of biofilms on heart valves, which increase bacterial resistance to antibiotics[21,26]. The prognosis for dogs with endocarditis is generally poor, particularly in cases involving specific bacteria such as *Streptococcus canis*[21] and *Actinomyces neuii*[22]. successful treatment outcomes are rare, highlighting the importance of early diagnosis and personalized treatment approaches.

The current case explained the One Health significance of valvular endocarditis in dogs. The isolation of *S. aureus* and *Klebsiella* spp., both of which are potentially zoonotic pathogens, raises concerns in environments where humans and animals interact closely, such as rural households and animal shelters[12]. *Staphylococcus aureus and Klebsiella spp.* are essential contributors to antimicrobial resistance, which has profound implications for public health[23]. Furthermore, the features of canine endocarditis, especially systemic embolism, resemble those seen in human medicine, thereby strengthening the translational importance of such cases in improving the understanding of infective endocarditis across species[4,26-28]. The present findings supported the need for integrated clinical evaluation and diagnostic strategies that acknowledge the interconnectedness of animal and human health.

4. Conclusion

The present report on valvular endocarditis in a 9-year-old male Pit Bull enhanced understanding of its clinical signs, underlying mechanisms, and possible complications in dogs. This knowledge will facilitate the enhancement of clinical outcomes for affected animals and enable clinicians to adopt a more informed approach. Further studies are recommended for the molecular characterization of causative bacterial agents and their antimicrobial resistance profiles to enhance diagnostic precision and treatment approaches.

Declarations

Competing interests

All authors declared no conflict of interest.

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Availability of data and materials

Data supporting the present study are included within the article and are available from the corresponding author upon reasonable request.

Authors' contributions

The concept of the present manuscript was from Olawale Olawumi Ola, and the literature search was done by Olawale Olawumi Ola, Habeeb Shakir, Olanrewaju Samuel Olaifa, and Jarikre Theophilus Aghogho. Tijani Monsuru Oladunjoye, Habeeb Shakiru, Usman Abdulrauf Adekunle, Tola Felicia Ajani, Olumuyiwa Abiola Adejumobi, and Ajayi John Olurotimi performed the post-mortem examination. The original draft was written by Olawale Olawumi Ola and Habeeb Shakiru, and reviewed by Olutayo Temidayo Omobowale, Tijani Monsuru Oladunjoye, and Ajayi John Olurotimi. Olutayo Temidayo Omobowale and Olumuyiwa Abiola Adejumobi supervised the post-mortem findings. All authors have read and confirmed the final edition of the manuscript.

Ethical considerations

The authors declared that this original case report has not been published or submitted elsewhere. The manuscript underwent plagiarism screening using a standard software.

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