

**PATHOLOGICAL REPORT OF ACUTE TRAUMATIC BOVINE RETICULO-PERICARDITIS IN A 4½-YEAR-OLD BUNAJI (WHITE FULANI) COW****Saleh, A<sup>1\*</sup>; Fatihu, M. Y<sup>1</sup>; Sani, N. A<sup>2</sup>; and Abalaka, S. E<sup>2</sup>.**

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**ABSTRACT**

Traumatic reticulo-pericarditis (hardware disease) is produced by a sharp object such as a piece of wire or a nail that pierces through the wall of the reticulum, and diaphragm, and gains access to the heart. A carcass of 4½-year-old White-Fulani was presented to the Necropsy unit of the Department of Veterinary Pathology, Ahmadu Bello University, Zaria for post-mortem examination. Gross findings revealed a shaft of an 18-gauge x 1½inch hypodermic needle within fibrinous tissue extending from the pericardial sac. Other findings include oedema in the jowl and brisket region, ascites, haemothorax, yellow and turbid fluid within the pericardial sac with fibrin flake showing a typical 'bread and butter' appearance. Histopathological observation of affected organs showed fibrinous pericarditis with severe fibrin deposition and fibrous tissue proliferation between the cardiac muscle fibres along with infiltration of neutrophils, lymphocytes, plasma cells and hepatic centrilobular necrotic changes. The result of the post-mortem examination revealed that the cow died of traumatic pericarditis. Acute traumatic pericarditis was diagnosed with cardiac tamponade as the possible cause of death. Cattle herders should be familiarized with the causes and symptoms of hardware disease and prompt action to save lives and avoidance of huge economic losses.

**Keywords:** Cow, Needle Shaft, Traumatic Reticulo-Pericarditis.

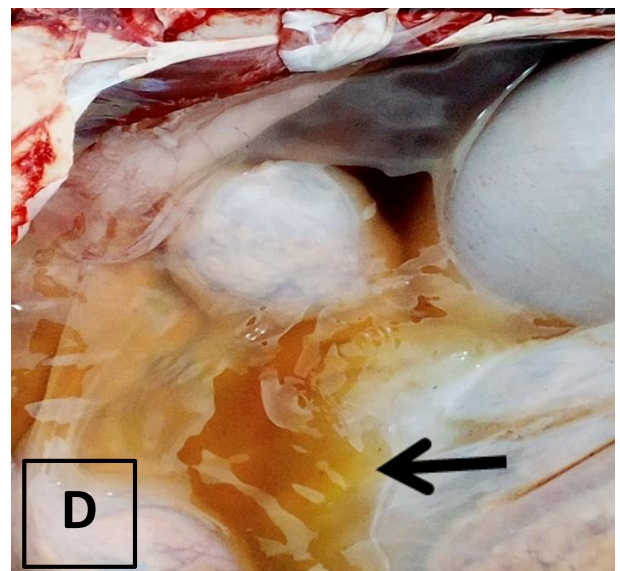
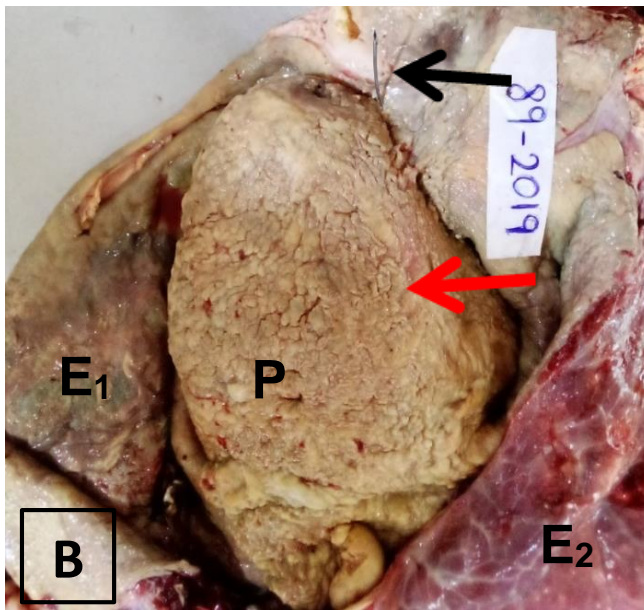
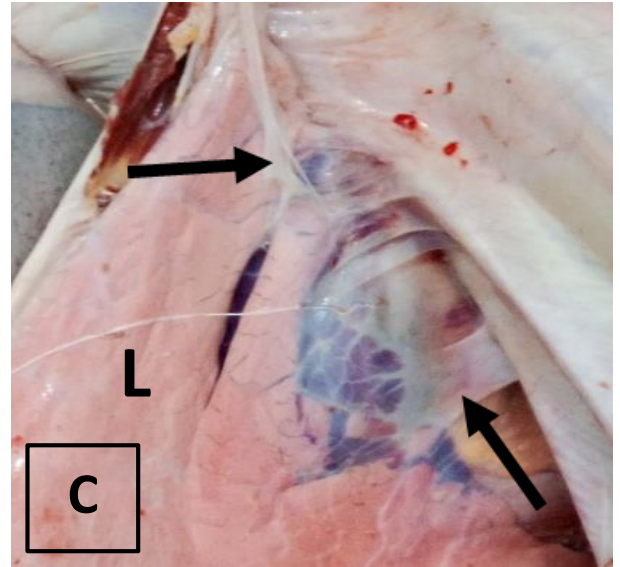
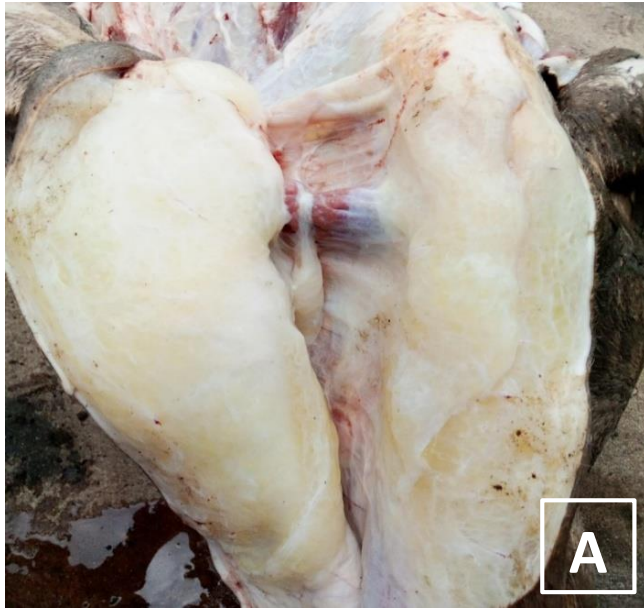
## INTRODUCTION

Hardware disease in bovine is a matter of concern in ruminant veterinary practices all over the world (Aref and Abdel-Hakim, 2013; Sileshi *et al.*, 2013; Abu-Seida and Al-Abbadi, 2014). The major clinical signs of pericarditis include asynchronous abnormal heart sounds (e.g., muffled heart sounds), tachycardia, jugular vein distension and submandibular, brisket and ventral abdominal oedema (Braun, 2009). Reports have shown a disease prevalence of approximately 2–12% in cattle (Starke and Rehage, 2000; Cramers *et al.*, 2005). A prevalence of 25% was recorded in examined buffaloes in Egypt (Aref and Abdel-Hakim, 2013) while 93% of buffaloes were over 2 years of age and 87% of dairy buffaloes in India (Sharma *et al.*, 1994). This disease is of high economic importance due to the severe reduction in milk and meat yield, costs of treatment, fetal losses and potential fatalities in affected pregnant animals (Radostits *et al.*, 2007; Tesfaye and Chanie, 2012; Sileshi *et al.*, 2013). The condition may prove lethal due to bacterial and protozoan contamination of the body cavity resulting in peritonitis. The diaphragm and heart may be punctured causing respiratory and cardiac failure. The incidence of hardware disease is high in all developing countries though poorly reported especially in Nigeria, resulting in economic losses.

## CASE PRESENTATION

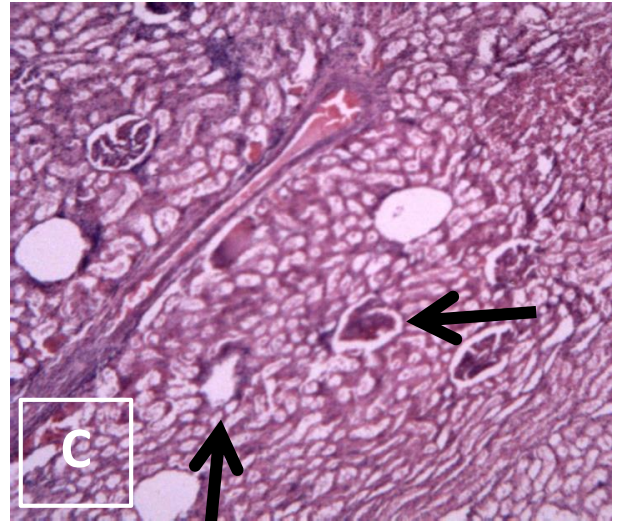
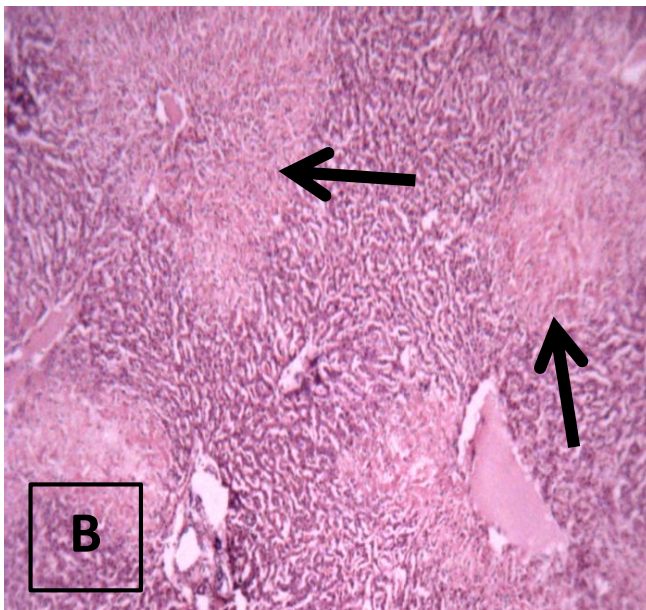
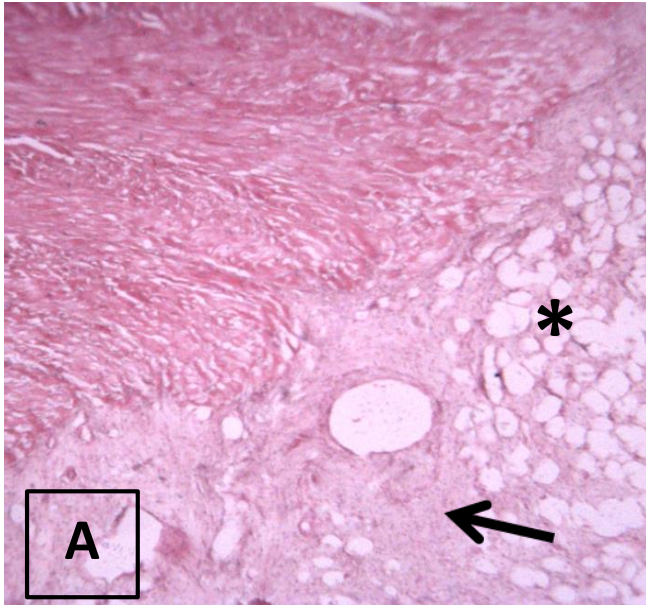
The carcass of a 4½-year-old White-Fulani cow weighing 320 kg was presented to Necropsy unit of the Department of Veterinary Pathology, Ahmadu Bello University (A.B.U.) Zaria, on the 30<sup>th</sup> September, 2019. History revealed that the cow had shown decreased appetite, lagged behind during grazing, arching of back, abducted elbow

and recumbent for about 8 hours prior to death. There were 205 heads of cattle on the farm managed under a semi-intensive system with current vaccinations and routine deworming records. A standard head-to-tail necropsy procedure was conducted to determine the cause of death (King *et al.*, 2014). Gross pathology revealed oedema in submandibular, jowl, brisket (Plate IA), hock joints and a shaft of 18-gauge x 1½-inch hypodermic needle within the fibrinous tissue extending from the pericardial sac and myocardium causing acute traumatic fibrinous pericarditis. Thickened epicardium and pericardium, perforation into the heart, with the pericardial sac containing yellow, turbid fluid with fibrin flake showing typical ‘bread and butter’ appearance (Plate IB), cardiac tamponade, with visceral pleural adhesions to thoracic wall (Plate IC) and pneumonia. Other prominent findings were haemothorax (8 L) and ascites (25 L) (Plate ID), oedematous and anaemic intestinal serosa, enlarged and congested liver, and enlarged kidney. Histopathology revealed severe fibrin deposition and fibrous tissue proliferation between the cardiac muscle fibres along infiltration of inflammatory cells (Plate IIA), acute fibrinous interstitial pneumonia, hepatocellular necrosis (Plate IIB), enteritis, atrophy and dissolution of the glomerular tuft (Plate IIC). Diagnosis of Acute Traumatic Bovine Reticulo-Pericarditis was reached.



**Plate I: A, Brisket region of the cow showing pale oedematous brisket muscle. B, Heart of the cow showing fibrinous pericarditis (red arrow). Note the perforation and the presence of shaft of 18-gauge needle x 1½ inch (black arrow), and fibrin flake on pericardium ('bread and butter') appearance (P). The epicardium is thickened and visceral surface covered by a thick, yellow layer of a fibrinopurulent exudate (E1) while the parietal surface appeared congested (E2). C, Thoracic region of a cow showing pale lungs (L) and area of adhesions**

between the lungs and parietal pleura (arrows).  
**D, Peritoneal cavity of the cow showing ascitic fluid (arrow).**



**Plate II: A, Photomicrograph of the heart of the cow showing fibrinous pericarditis characterized by marked oedema and severe vacuolation in cardiac muscle fibres (asterisk), myocardial fibre degeneration and necrosis with loss of cross striations (black arrow). H&E stain, x100. B, Photomicrograph of the liver of the cow showing necrotic changes around the central vein (black arrows). H&E stain, x 100. C, Photomicrograph of the kidney of a cow showing atrophy and dissolution of the glomerular tuft (black arrows). H&E stain, x100.**

**DISCUSSION**

Management of cattle on semi-intensive or extensive systems predisposes them to traumatic pericarditis as they can pick up metallic and non-metallic foreign bodies during grazing. This condition is of utmost importance as it leads to economic losses to livestock owners. In addition, the condition is difficult to diagnose only on the basis of clinical symptoms and treatment is usually not rewarding (Athar *et al.*, 2012). Accidental ingestion and lodgment of strange objects while feeding is more common in cattle than in other ruminants due to

their indiscriminate feeding habits (El-Sebaie, 1994; Ehsan *et al.*, 2011). More so, industrialization and mechanization of agriculture have further increased the incidence of foreign objects in the feeds and pastures of these animals. According to Radostitis *et al.* (2003); Sharma and Kumar (2006), the penetrating foreign objects first lodged in the reticulum, cause irritation and then forced by ruminal contractions into the reticular wall causing several complications such as traumatic reticulitis, reticular abscess, traumatic reticulo-peritonitis, hepatic abscess, splenic abscess, rupture of left gastro-epiploic artery, diaphragmatic hernia, vagal indigestion, pleurisy, traumatic pneumonia and mediastinal abscess, traumatic pericarditis and tamponade some of which were observed in the current case report. Adhesions between the epicardium and pericardium due to the accumulation of fibrinous exudates and fluid accumulation in the pericardial sac might cause severe constriction of the ventricles and regional arteries affecting basic cardiac function might have contributed to the death of the cow. The 'bread and butter' appearance occurs as a result of the organization of fibrin which leads to adhesions. The condition can also result in pneumonia, pleurisy and pulmonary, diaphragmatic, hepatic or splenic, abscesses (Radostitis *et al.*, 2007; Vanvleet and Ferrans, 2007). The macroscopic and microscopic findings such as fibrinous pericarditis, pulmonary oedema and centrilobular necrotic changes are in agreement with earlier reports (Radostitis *et al.*, 2007; Baydar *et al.*, 2016). This portion of the hepatic lobules receives the least oxygenated blood and is thereby rendered more susceptible to hypoxia, and it has the greatest enzymatic activity (mixed function oxidases) capable of activating compounds into toxic forms (Zachary, 2017).

## CONCLUSION

Based on the necropsy findings, the diagnosis of Acute Traumatic Bovine Reticulo-Pericarditis was established. Exposure to foreign objects in cattle usually occurs during grazing or less commonly in feed. Therefore, appropriate management practices such as physically screening feeds and fodder with the aid of appropriate sieves and magnets are important to prevent traumatic reticulo-pericarditis. Also, herds should not be grazed in a polluted area or around a construction site. Oral administration of Magnets could be an effective prophylaxis for hardware disease.

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## DISCLOSURE STATEMENT

The authors declared no conflicts of interest with respect to the report, authorship and/or publication of this article.

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