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CASE REPORT

Recrudescence of a Suspected Case of Contagious Bovine Pleuropneumonia in a Friesian Bull: Clinicopathological Report

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INTRODUCTION

Contagious bovine pleuropneumonia (CBPP) is an endemic disease in Nigeria (Cadmus and Adesokan, 2009; Alawa *et al*., 2011; Musa *et al*., 2016; Francis *et al*., 2018), caused by the small colony variant of *Mycoplasma mycoides* subsp. *mycoides* (*Mmm Sc*) mainly affecting cattle (Musa *et al*., 2016). It is characterized clinically by respiratory distress, and pathologically by fibrinous pneumonia and pleurisy (Swai *et al*., 2013). Outbreaks of CBPP in Nigeria, especially Abuja, have been underreported despite the huge economic losses incurred by owners of affected cattle (Hajara, M., personal communication, December, 2019), and the disease being among the reportable diseases of cattle as listed by OIE (2020). This clinicopathological report therefore describes a case of recrudescence CBPP in a Friesian bull from a cattle ranch in Giri, Abuja, Nigeria.

CASE DESCRIPTION

Clinical History

On 27th January, 2020, the services of the Necropsy Unit of the Department of Veterinary Pathology, University of Abuja, was requested by the manager of a cattle ranch in Giri, Abuja, to carry out a postmortem examination on a carcass of an adult Friesian bull. The bull was reportedly brought into the farm in July, 2019 for the purpose of breeding.

The bull reportedly lagged behind during grazing. Inappetence and diarrhoea were reportedly observed in the morning, a day prior to death (26th January, 2020), which necessitated treatment with Amoxicillin LA (6.7mg/kg; IM) and Sulphadimidine (10mg/kg; IM). The appetite improved later in the evening of the same day but the diarrhoea persisted. The bull was found dead in the early hours of the following day (27th January, 2020).

Further history revealed that there was a suspected outbreak of CBPP on the farm in the third quarter of 2019, which was diagnosed based on clinical signs (respiratory distress) and postmortem findings (acute pleuropneumonia). Mortality in the White Fulani breed was 16.9% (102/605) and 11.1% (5/45) in the Friesian breed, whereas 98 cattle that were severely affected by the disease were culled at very low prices. The remaining cattle recovered from the disease following treatment with Tylosin (10mg/kg; IM, 5/7). The herd had no history of

vaccination against CBPP or any other endemic diseases of cattle.

Postmortem Evaluation

A detailed postmortem examination was carried out on the carcass following standard protocol (Maxie and Miller, 2016). Gross lesions observed were described and recorded appropriately, while the pictures of the lesions were taken using a digital camera. Tissues from organs with gross lesions were fixed in 10% neutral buffered formalin and processed for histopathology using standard technique (Maxie and Miller, 2016).

The carcass was moderately fleshed, while the ocular and oral mucous membranes were moderately pale.

The subcutaneous fat was gelatinous. The thorax contained moderate amount (93 ml) of fibrinous exudate. The lumena of the trachea and bronchi contained large amount of froth, while the parenchyma of the lungs had grey and red areas of hepatization. There was straw coloured gelatinous (fibrinous) exudate on the ventral aspect of the parietal pleura of the left lung. There was adhesion of the left diaphragmatic lobe of the lung to the ventral aspect of the thoracic wall (Plates IA and IB). The pleurae of the lungs were cloudy and the interlobular septa were thickened, giving the lungs a marble appearance (Plate IC). The ventral aspect of the cranial lobe of the left lung had a focal, cream-coloured multi-layered, caseous nodule (sequester) of about 4 cm in length and 2 cm in diameter.

There was atrophy of the coronary fat. The pericardium adhered to the left cranial lobe of the lung and the diaphragm. The pericardial sac contained small amount (27 ml) of serosanguinous exudate, while the epicardium and endocardium had multifocal petechial haemorrhages (Plate ID).

Histopathological Findings

The lung was markedly congested, with oedematous and thickened vascular walls. The alveolar walls (Plate IIA), interlobular septa, and pleura were markedly thickened. There were fibrin strands and fibrous connective tissue constricting the atrophied alveoli, which contained necrotic debris and inflammatory cells, consisting predominantly mononuclear cells (Plate IIB).

The cardiomyocytes were fragmented and undergoing degeneration and necrosis (Plate IIC). There was haemorrhage, fibrin, and infiltration of mononuclear cells within the interstitium which replaced the necrosed cardiomyocytes (Plate IID).

Diagnosis

The diagnosis of CBPP was arrived at based on clinical history of previous outbreak of the disease on the farm, as well as the pathological findings of fibrinous pleuropneumonia and fibrinous myocarditis.

DISCUSSION

The clinical signs and pathological features of CBPP are highly suggestive of the disease, and are readily recognised by trained professionals (Noah *et al*., 2015). Fibrinous pleuropneumonia, characterized by red and gray hepatization, marble appearance of the lung, hydrothorax, and sequestrum, as observed in this case have also been reported by several authors (Nawathe, 1992; Swai *et al*., 2013; Musa *et al*., 2016).

Although control of CBPP through stamping-out and restriction of cattle movement achieved desired results in some countries, mass vaccination and treatment of affected animals using antimicrobials are the realistic and widely applied control measures in Nigeria, due majorly to logistical difficulties and cost implication of eradication measures (Alhaji *et al*., 2020). However, treatment of animals diagnosed to have CBPP may lead to the sequestration of *Mmm* in

necrotic encapsulated nodules in the lungs, and the agent eventually released when the animal is stressed leading to recrudescence. That was why the client was advised to screen the remaining herd for CBPP, which will serve as basis to either cull the animals and replace with healthy stock, or vaccinate if found to have fully recovered from the disease.

The source of the infection in the herd could not be ascertained. However, there is a nearby river, which serves as watering point for many herds of cattle from different parts of Nigeria with no know vaccination or disease status. Moreover, outbreaks of CBPP was reported in many states of northern Nigeria, including Abuja, between the last quarter of 2019 and the first quarter of 2020 (AR, 2019), which coincided with the period the outbreak occurred on the farm. Suleiman *et al*. (2015) enumerated factors that are likely to predispose susceptible animals to CBPP to include: unvaccinated herds; sharing of water points with herd of unknown CBPP status; large sized herds especially above 40; and the introduction of new animals with unknown health status into herd. All these predisposing factors were observed on the farm in this case.

In conclusion, CBPP causes huge economic losses to farmers. Therefore, efforts should be made to protect cattle from the disease through yearly mass vaccination programmes.

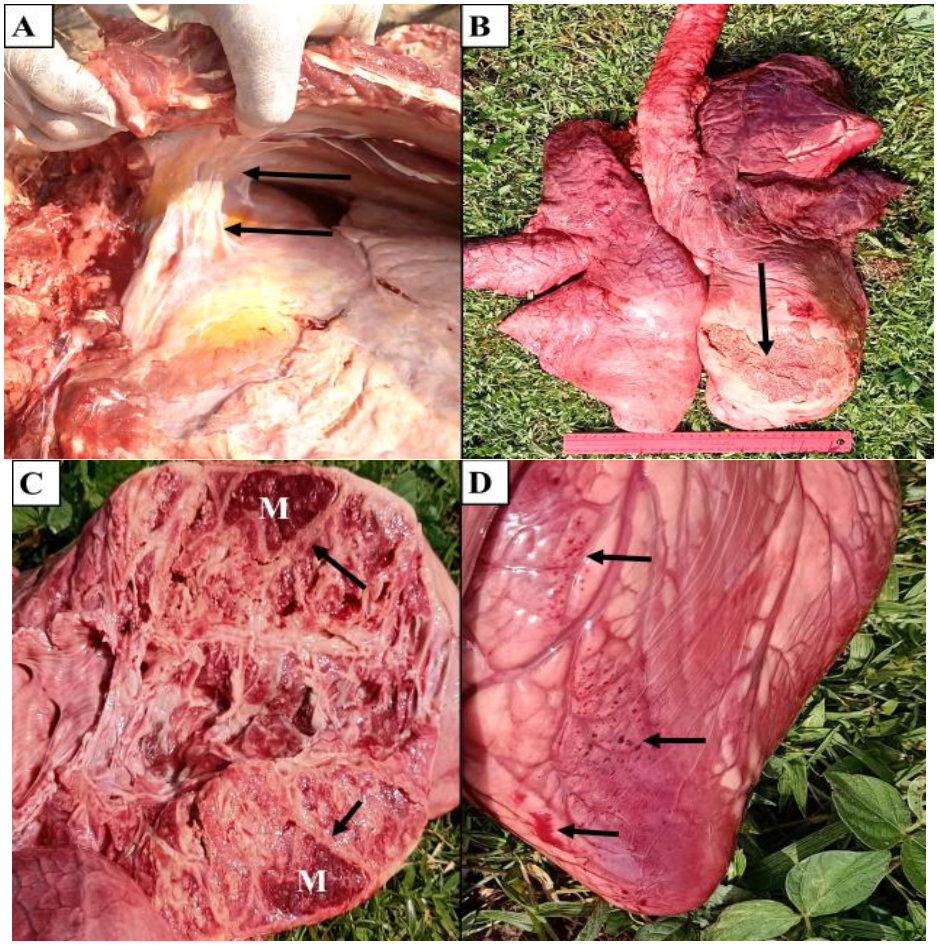

Plate I: Gross lesions in the lung and heart of carcass of the Friesian bull suspected to have died of CBPP. A. Note the adhesion of the pleura to the parietal surface of the thoracic cage (arrow). B. Note the enlarged caudal lobe of the left lung. There is also a large ulcerated area indicating the point of attachment of the pleura to the rib cage (arrow). C. Note the marble appearance of the cut surface of the caudal lobe of the left lung (M) and thickened interlobular septa (arrows). D. Note the haemorrhages on the epicardium (arrows).

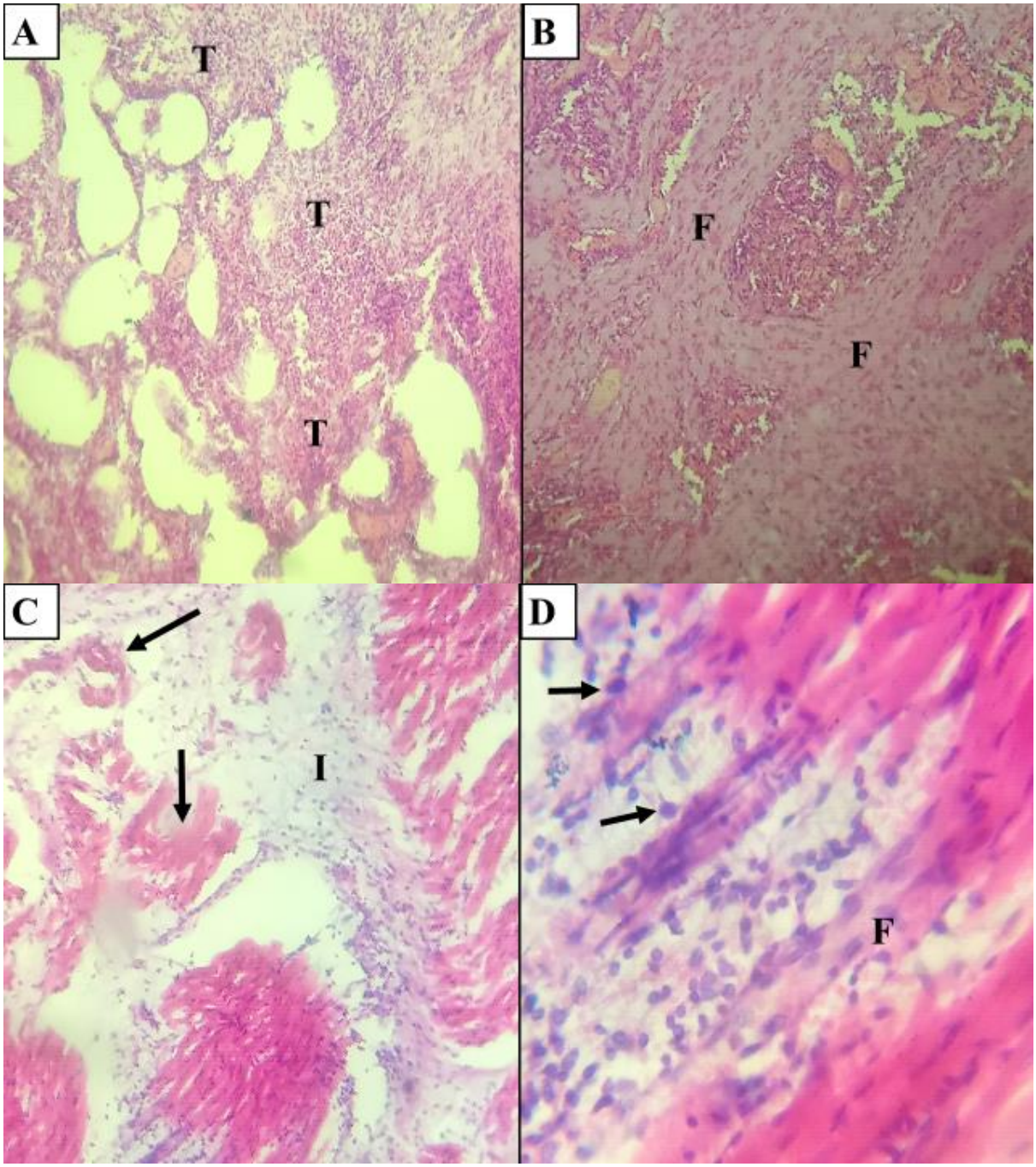

Plate II: Photomicrographs of histopathological changes observed in lung and heart of the carcass of a Friesian bull suspected to have died of CBPP (H and E stain). A. Note the thickened alveolar walls (T) (X100). B. Note the fibrous connective tissue (F) surrounding necrotic areas (X100). C. Note the necrosis of the cardiomyocytes (arrows) and fibrin strands (I) within the interstitium (X100). D. Note the fragmented cardiomyocytes (F) with mononuclear cellular infiltration (arrows) (X400).

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