

Penetrating Trauma and Bronchopneumonia in a Ram - A Case Report

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Abstract

This case report documents the gross and histopathological findings in the lungs of a ram that died after a period of prolonged sternal recumbency and dyspnoea. The carcass of a nine months old Yankasa ram was presented to the necropsy unit after three weeks of dysnoea, prolonged sternal recumbency and treatment with tylosine, penicillin-streptomycin and griseofulvin prior to death. An area of necrotic dermatitis on the sternum and penetrating wound area with haemorrhages on the left lateral thoracic wall between 8th-10th intercostal spaces were observed. On gross examination, hydrothorax, hydropericardium and hepatization of the lungs were observed. On histopathology, fluid was accumulated in the alveoli, bronchioles and bronchi coupled with thickening of the interalveolar walls. On microbial culture and isolation, Staphylococcus aureus was isolated and identified. Based on these findings, a diagnosis of penetrating trauma and bronchopneumonia were made. This is the first report of bronchopneumonia in Zaria, Nigeria.

Keywords: Dyspnoea, penetrating trauma, hepatization, Staphylococcus aureus, bronchopneumonia

INTRODUCTION

Pneumonia refers to the inflammation of the pulmonary parenchyma usually accompanied by the inflammation of bronchioles and alveoli (Hoare and Lim, 2006). It occurs when viruses, bacteria or fungi cause inflammation and infection in the alveoli in the lungs. Pneumonias are the most common cause of death among infectious diseases (Shah et al., 2017). Bronchopneumonia (lobular pneumonia) is an acute or chronic inflammation of the lungs, in which the alveoli and / or interstitial are affected and characterized by foci of consolidation surrounded by normal parenchyma (Cotran et al., 2005). It is caused by bacteria such as *staphylococcus*, *streptococcus*, *proteus*, *Escherichia coli* and *Haemophilus influenza* (Hoare and Lim, 2006). Clinical signs include fever, moist painful cough, dyspnoea, anorexia and depression (Cotran et al., 2005). This report is aimed at documenting the gross and hisopathological findings in the lungs associated with penetrating wound trauma and bronchopneumonia in a ram.

CASE REPORT

The carcass of a nine months old Yankasa ram was presented to the necropsy unit of the Ahmadu Bello University Teaching Hospital, Nigeria. The ram had no vaccination record, was fed on grass and wheat offal and administered tylosine, penicillin-streptomycin and griseofulvin prior to death.

The clinical signs observed antemortem included bilateral pale ocular mucus membranes, prolonged sternal recumbency, lameness of the left forelimb and severe dyspnoea.

On postmortem examination, the gross pathological findings were area of necrotic dermatitis on the sternum (23-28cm in diameter) (fig. 1), penetrating wound area with haemorrhages on the left lateral thoracic wall btw 8th-10th intercostal spaces (fig. 2), hydrothorax (3 litres of straw coloured fluid) (fig. 3), hydropericardium (15 mls of fluid), congested and haemorrhagic trachea with frothy exudate, grey hepatization of the whole left lung (fig. 4) with fibrinous adhesion to the heart and thoracic wall,

congestion and red hepatization of the right lung (fig. 6) with frothy exudates in the bronchi, pericarditis with fibrinous adhesion to the left lung (fig. 5), cardiomegaly (fig. 7) and endocardialhaemorrhage (fig. 8), enlarged and congested liver (fig. 9) with fibrinous adhesion to the diaphragm, rumen and spleen and enlarged spleen (fig. 10) with fibrinous adhesion to the liver and rumen.



Fig 1. Area of necrotic dermatitis on the sternum (arrows).



Fig 2. Penetrating wound area with haemorrhages on the left lateral thoracic wall btw 8th-10th intercostal spaces. (arrows).

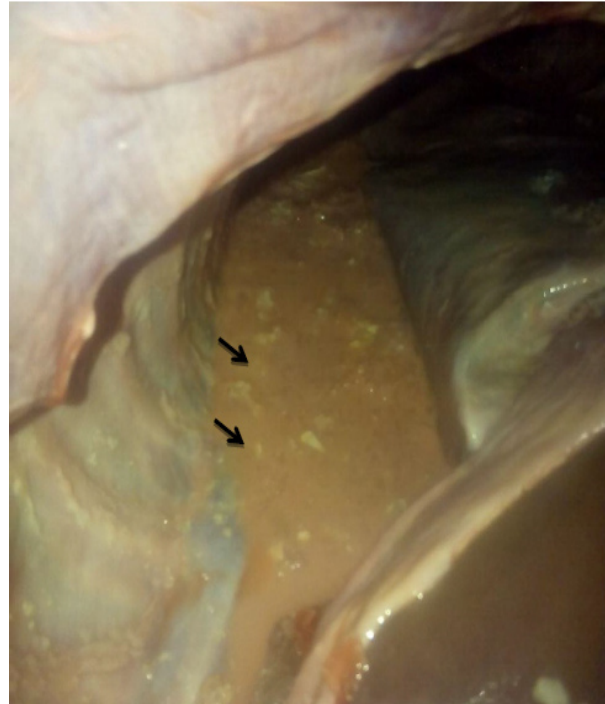


Fig 3. Thoracic cavity. Note hydrothorax (3 litres of straw coloured fluid) (arrows)



Fig 4. Lung. Note grey hepatization of the whole left lung

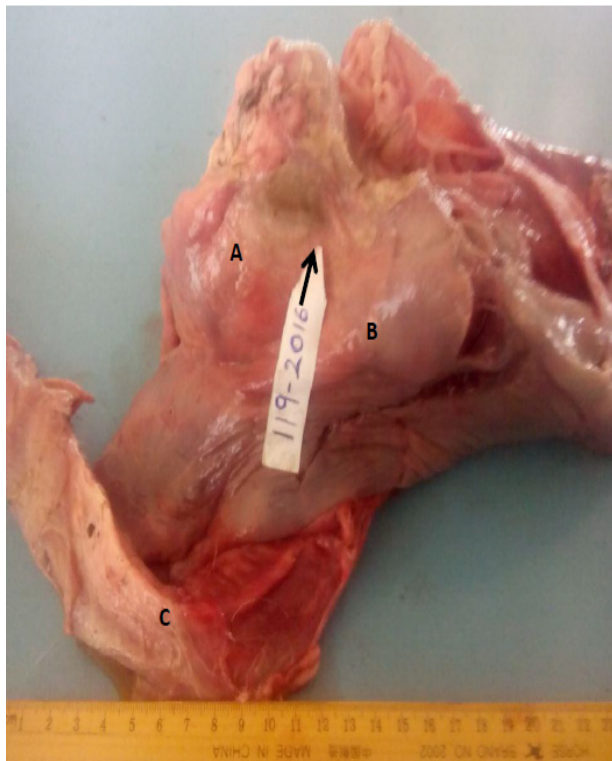


Fig 5. Heart (A), lung (B) and trachea (C). Note fibrinous adhesion of the left lung to the heart (arrow)

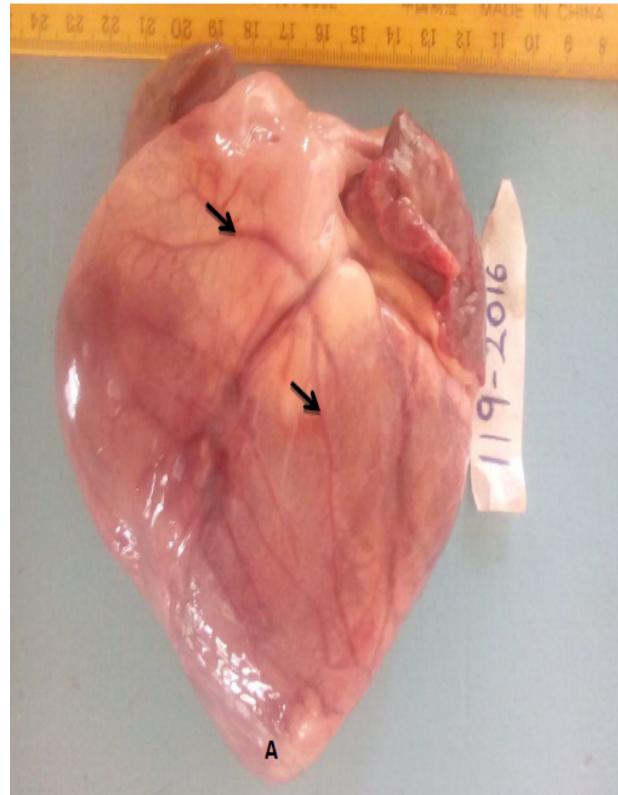


Fig 7. Heart. Note cardiomegaly (A) and congestion (arrows) of the coronary vessels

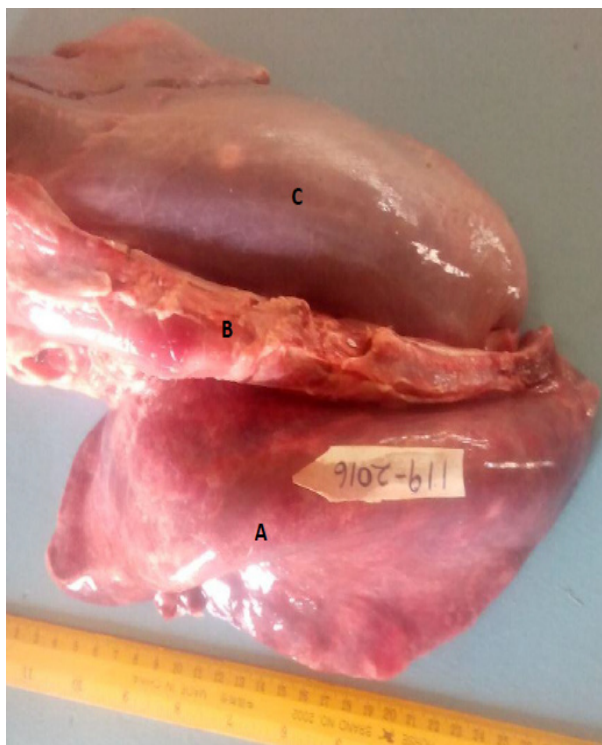


Fig 6. Right lung, trachea and left lung. Note congestion and red hepatization of the right lung (A), congestion of the trachea (B) and grey hepatization of the left lung (C)

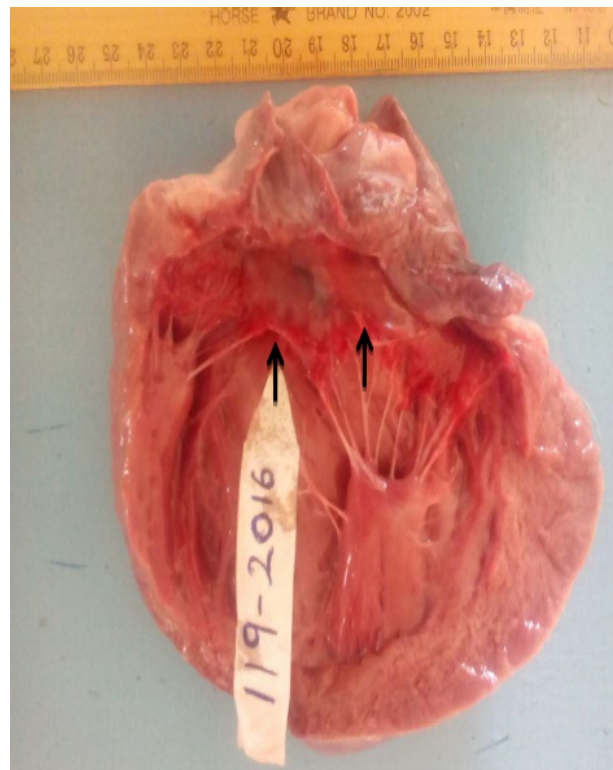


Fig 8. Heart. Note endocardial haemorrhages (arrows)



Fig 9. Liver. Note hepatomegaly, hepatic congestion (A) and fibrin deposits (B)

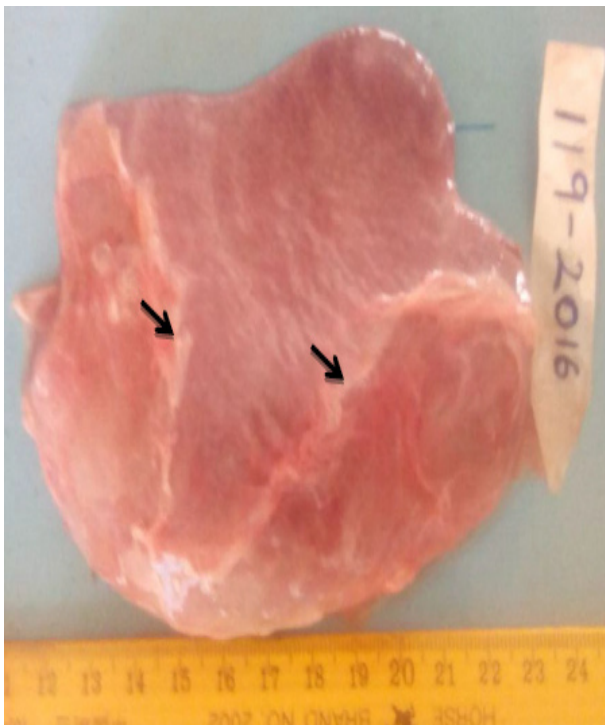


Fig 10. Spleen. Note enlarged spleen with fibrin deposits (arrows).

The histopathological findings included fluid accumulation in the bronchioles and alveoli (fig. 11), thickening of the interalveolar walls and massive inflammatory cellular infiltrations (fig. 12).

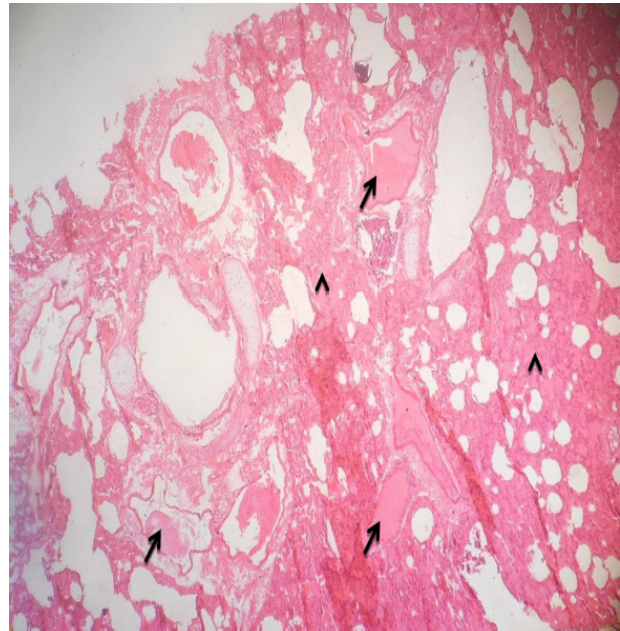


Fig 11. Photomicrograph of section of the lung. Note fluid accumulation (arrows) in bronchioles and alveoli and thickening of the interalveolar walls (arrow heads). H&E x100.

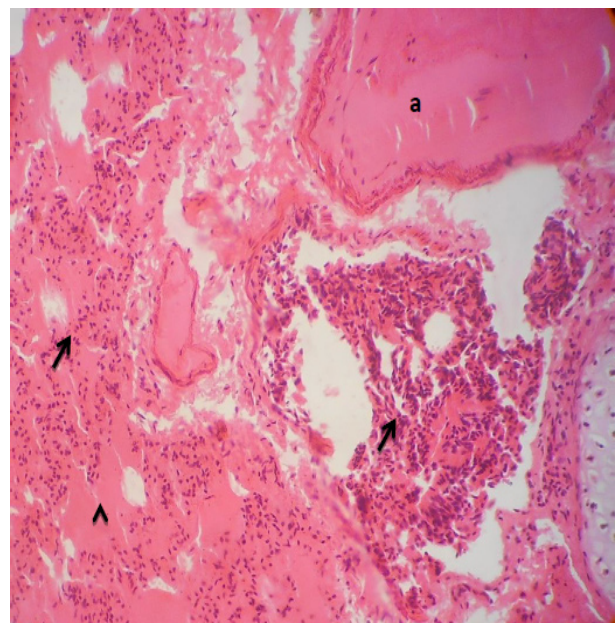


Fig 12. Photomicrograph of section of the lung. Note massive inflammatory cellular infiltrations (arrows), fluid accumulation (a) in bronchiole and thickening of the interalveolar walls (arrow head). H&E x200.

DISCUSSION

Penetrating trauma results when an object pierces the skin and enters a tissue of the body thus creating an open wound (Stewart, 2005). However in blunt or non-penetrating trauma, an impact may be present, but the skin is not necessarily broken. The penetrating object may remain in the tissues, come back out the way it entered, or pass through the tissues and exit from another area (Blank-Reid, 2006). Penetrating trauma can be serious because it can damage internal organs and present a risk of infection (Prentice and Ahrens, 1994).

The areas of necrotic dermatitis on the sternum (fig. 1) resulted from prolonged recumbency due to severe pain and respiratory distress. In this position, there was pressure on the skin overlying the sternum, thus occluding the blood supply to the skin in the region. Hence, ischemic necrosis was the outcome (Tasliyurt et al., 2012). The area of haemorrhages on the left thoracic wall (fig. 2) indicated a penetrating trauma although the skin manifestation of the trauma was not grossly evidenced (Smith and Ball, 1998).

The fibrinous deposits on the liver (fig. 9) and spleen (fig. 10) suggested an internal haemorrhage in the abdominal cavity resulting in peritonitis and fibrinous adhesions of the organs. This was part of the inflammatory responses but the mechanism is not fully understood.

The fluid accumulation in the thoracic cavity (fig. 3) and pericardiac sac might have resulted from decrease in plasma protein (Vikra, et al., 2011) due to blood loss. The hydrothorax contained straw coloured fluid suggesting internal haemorrhage in the thoracic cavity. This indicated that the trauma might have caused damages to blood vessels within the thoracic cavity. However, this blood loss caused a decrease in blood volume leading to hypotension and increased stress on the heart (Musher et al., 2007), hence cardiac hypertrophy as evidenced by the enlarged heart (fig. 7).

Also, the grey hepatization stage of the left lung (fig. 4) suggested that the penetrating object introduced infectious agent into the thoracic cavity while the red hepatization of the right lung (fig. 6) indicated an ongoing infectious process similar to that of the left lung. This hepatization stage of both lungs was evidenced by the thickened interalveolar

walls, massive inflammatory cellular infiltrations and fluid accumulation in the alveoli, bronchioles and bronchi (fig. 11 and 12). This led to decreased lung capacity and increased pulmonary arterial resistance. The decrease in lung capacity led to difficulty in breathing (dyspnoea), hypoxia, hypoxemia, increased cardiac stress and arrhythmias (Shah et al., 2017).

The presence of infectious agents was also evidenced by the isolation and identification of *Staphylococcus aureus*. These hepatizations of both lungs and decreased blood volume due to haemorrhages contributed to the enlargement of the heart (hypertrophy) (fig. 7). The congestion of the coronary vessels (fig. 7) and endocardial haemorrhages (fig. 8) might have resulted from the infection and the mechanism was not fully understood. The adhesion of the heart to the lungs (fig. 5) resulted in increase in cardiac resistance leading to cardiac tamponade hence death. Consequently, death resulted from multiple effects of decreased pulmonary capacity, hypoxia, hypoxaemia and cardiac failure. It is therefore recommended that further studies should be carried out to provide a better understanding of the sequence of events leading to bronchopneumonia.

This is the first published attempt to explain the sequence of events resulting in death of a ram that died after prolonged sternal recumbency and dyspnoea following unnoticed penetrating trauma to the thoracic wall in Zaria, Nigeria.

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