

PATHOLOGICAL LESIONS AND INCIDENCE OF FIBRINOUS PLEUROPNEUMONIA IN IMPORTED BEEF CATTLE SLAUGHTERED AT ABU-SIMBEL CITY – ASWAN GOVERNORATE

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ABSTRACT

The current study was conducted on 110 lung samples from 2 - 3 years old imported beef cattle slaughtered at the Middle East abattoirs of Abu-Simbel city –Aswan Governorate- Egypt, in the period from July 2017 to March 2018. The aim of the present work was to characterize and detect the incidence rate of the pulmonary affections. During the postmortem examination, the pulmonary affections were recorded grossly and specimens were taken for histopathological examinations. The histopathological examinations revealed that 36 samples (32.7%) showed fibrinous pleuropneumonia. According to the characteristic features of lesions in the microscopical studies, we grouped them into (a) Acute fibrinous pleuropneumonia in 31 cases (28.1 %) and (b) Organized fibrinous pleuropneumonia in 5 cases (4.5 %). The causes of these affection and their importance were discussed.

Key words: Lung, Fibrinous, Pleuropneumonia, Bronchopneumonia.

INTRODUCTION

Pneumonia is defined as the inflammation of pulmonary parenchyma and it is usually accompanied by inflammation of the bronchioles (Bronchopneumonia) and/or by pleurisy (pleuropneumonia) (Blood *et al.*, 1983). Cattle with fibrinous pleuropneumonia had grossly bilateral fibrinous pleuritis with either removable sheets of fibrin or firmly attached strands and networks of fibrin on the visceral and parietal surfaces. On cut section the most prominent characteristic feature of the fibrinous pleuropneumonia is the marbled appearance which resulted from the varying degrees of red to gray hepatization with distension of the interlobular septa and interstitium surrounding the vessels and airways with a yellowish serofibrinous exudate. (Kumar *et al.*, 2006 and Lopez, 2012).

Fibrinous pleuropneumonia is characterized by congestion and consolidation of lungs and is usually accompanied by pleuritis. (Chauhan, 2010). The pulmonary lesions is formed in a similar way

regardless of the type of etiological agent. It include various stages of congestion, red hepatization, grey hepatization then resolution. (Chauhan, 2010). Fibrinous pleuropneumonias was classified previously into acute fibrinous pleuropneumonia and organized fibrinous pleuropneumonia. (Schiefer *et al.*, 1978 and Haridy, 2003). The latter stages of fibrinous pleuropneumonia representing the organized fibrinous pleuropneumonia type (Jensen *et al.*, 1976). Moreover Gibbs *et al.* (1983) stated that, the pleural surface was bulged and thus produced mild nodulation. Later on, areas of fibrinous pleuropneumonia were often confined by fibrous tissue forming nodules.

In cattle, fibrinous pneumonia is characterized by the accumulation of exudates in the bronchiolar and alveolar spaces. Organization of this material is one of the most distinguished phenomenon of the pneumonic lesion in cattle especially when died during severe *M. haemolytica infection*. (Lopez, 2012 and Azizi *et al.*, 2013).

Concerning the fibrinous pleuritis in cases of fibrinous pleuropneumonia, Siqueira *et al.* (2018) stated that the pleura had marked edema, fibrin deposition and moderately severe infiltration of neutrophils and macrophages, Trichard *et al.* (1989)

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mentioned that the sequestra were the most characteristic feature of contagious bovine pleuropneumonia (CBPP). The sequestra varied in size from 15 mm to very large, involving more than one lobe of a lung. The sequestra were often surrounded by a thin irregular fibrous capsule and contained reddish –pink to yellowish –gray necrotic tissue. Histologically, part of a lobule or a lobule or multiple lobules and their intervening septa became necrotic. Vasculitis and thrombosis of the intralobular and interlobular arteries were noticed in some of the necrotic areas. Trichard *et al.* (1989).

Previous studies recorded a strong correlation between bacteriological and histopathological findings in bovine fibrinous pneumonia (Schiefer *et al.*, 1978), as Bovine respiratory disease (BRD) including shipping fever, which is indicated by fibrinous pneumonia or pleuritis is usually caused by *Mannheimia haemolytica* (Singh *et al.*, 2011) and *Pasteurella multocida* (Welsh *et al.*, 2004 and Caswell and Williams, 2016), the above mentioned micro-organisms are the most common bacteria isolating from animals with BRD (Singh *et al.*, 2011). In addition, *M. haemolytica* as well as *Mycoplasma mycoides*, are the micro-organisms frequently related to fibrinous pathological condition (Jubb *et al.*, 2007). *Hemophilus somnus* was recorded with either *P. hemolytica* or *P. multocida* in association or with other bacterial microbe like *Bacillus* spp, *staph. aureus* or *E.coli* as well (Schiefer *et al.*, 1978).

MATERIALS AND METHODS

Animals

Beef cattle of sudanese-origin aged from 2 – 3 years were kept in quarantine for 21 days under observation and supervision of Egyptian Quarantine Veterinarians in the Sudanese city of Wadi Half (bordering Egypt). Afterwards, cattle are shipped down the Nile to the city of Abu Simbel, Aswan Governorate where they are slaughtered.

Specimen collection

Regular visits were done to the Middle East Slaughterhouse during the period from July 2017 to March 2018. Routine postmortem examination of 1450 slaughtered beef cattle was carried out with particular attention to the pulmonary affections. Tissue specimens from lungs of 110 animals grossly showing pathological changes were collected and all lesions are imaged by a digital camera.

Histopathological examinations

Specimens from different lesions of lungs were fixed in 10 % neutral buffered formalin. Preparation of tissue for histopathological examination was carried out according to the method described by (Bancroft *et al.*, 1996).

RESULTS

Pathological examination:

Out of 110 pneumonic lung cases, pleuropneumonia was observed in 36 (32.7 %) cases. Histopathologically, these lesions were grouped them into: (a) Acute fibrinous pleuropneumonia in 31 (28.1 %) cases and (b) Organized fibrinous pleuropneumonia in 5 (4.5 %) cases.

a. Acute fibrinous pleuropneumonia:

The number of cases, which showed acute fibrinous pleuropneumonia, was 31 cases.

Gross examination

Our gross examination during lungs necropsy revealed marked lung enlargement, non-collapsed, firm, and covered diffusely with large amounts of a yellowish fibrillary material (fibrin). The parietal and visceral pleura were thickened. There were large amounts of fibrinous exudate above the pleura led to adhesions between the visceral and parietal pleural layers (Fig 1, a) as well as adherence to the chest wall. The thoracic cavity contained a yellowish serous fluid with a small amount of fibrin casts. In the cut sections we observed the interlobular septa are widened with serofibrinous exudate, lymphatic vessels were dilated and there were multiple foci of hepatization (red or gray) according to the age of the lesion, giving the lung marbling or mosaic appearance (Fig 1, b).

Microscopical examination

The histopathological examination recorded that, the pleura was extended by a diffuse inflammatory infiltrate constituted from degenerate neutrophils combined with a deposition of a fibrillary eosinophilic material (fibrin) (Fig 2, a). The interlobular septa were markedly expanded with fibrinous exudate, which formed by fibrin network, macrophages, neutrophils, and eosinophilic faint pink serous exudate (Fig 2, b). The bronchiolar epithelium was showed degenerative changes and the bronchiolar lumen was filled with fibrinous exudate (Fig 2, c). The alveolar lumens were dilated and filled with fibrinous exudate (Fig 2, d). The fibrinous exudate was composed of fibrin network, macrophages, some neutrophils and erythrocytes.

b. Organized fibrinous pleuropneumonia

The number of cases which showed organized fibrinous pleuropneumonia, were 5 cases.

Gross observation

Marked thickening in the pleural tissue. Thickening is due to organizations of fibrinous exudate, which turned into fibro-vascular tissue. Organization was manifested by presence of fibrous bands connecting the parietal and visceral pleural layers (Fig 3, a). In some chronic cases there were large area of necrosis which surrounded by fibrous capsule occupying

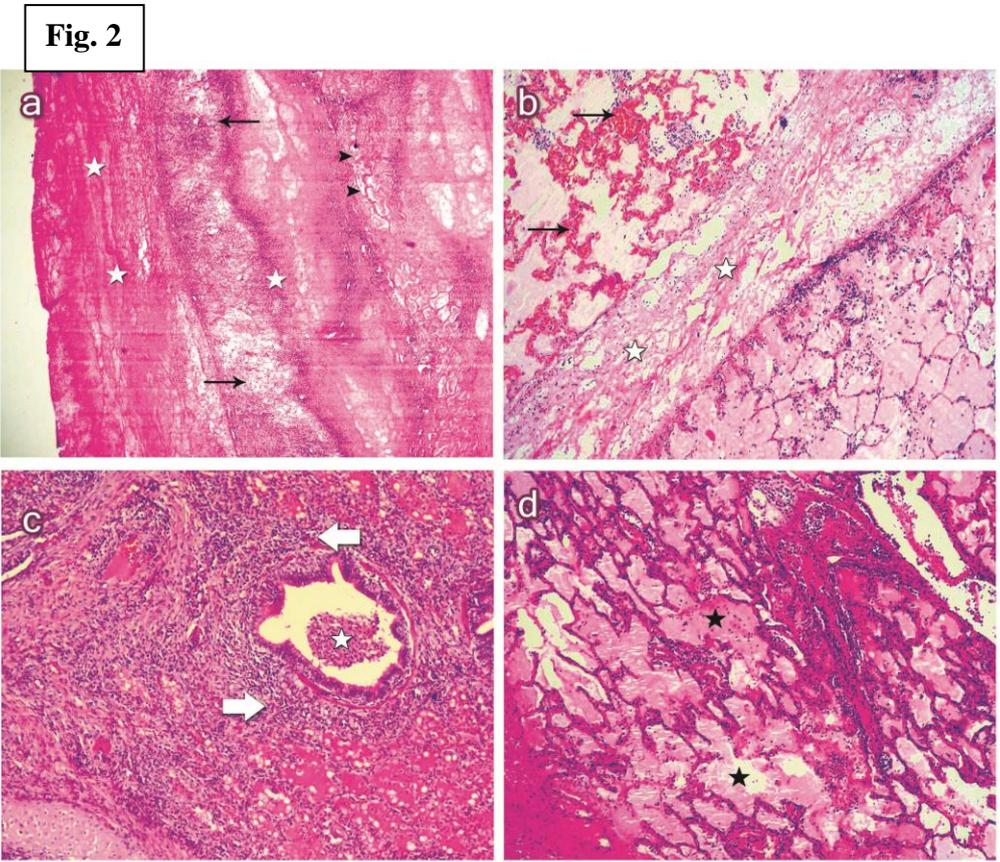
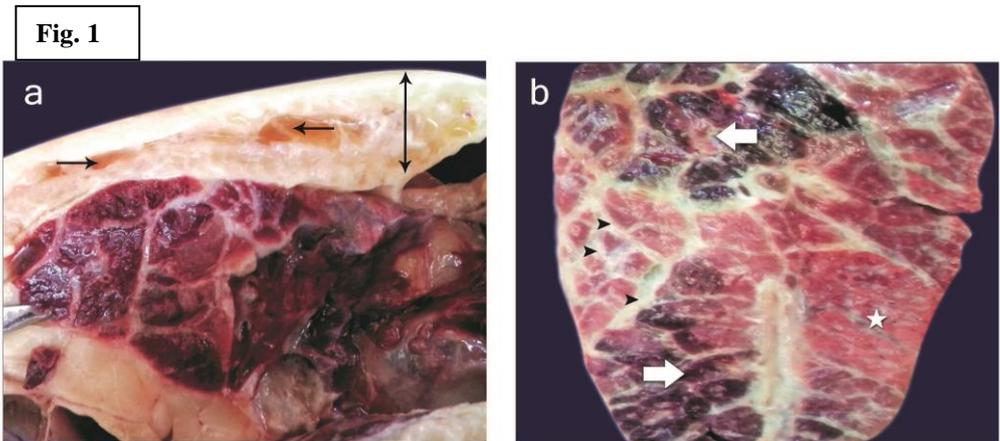
large area of lung lobules (Fig 3, b) (sequestrum), in cut section alternating areas of red, gray hepatization, coagulative necrosis and thickening of interlobular septa were observed.

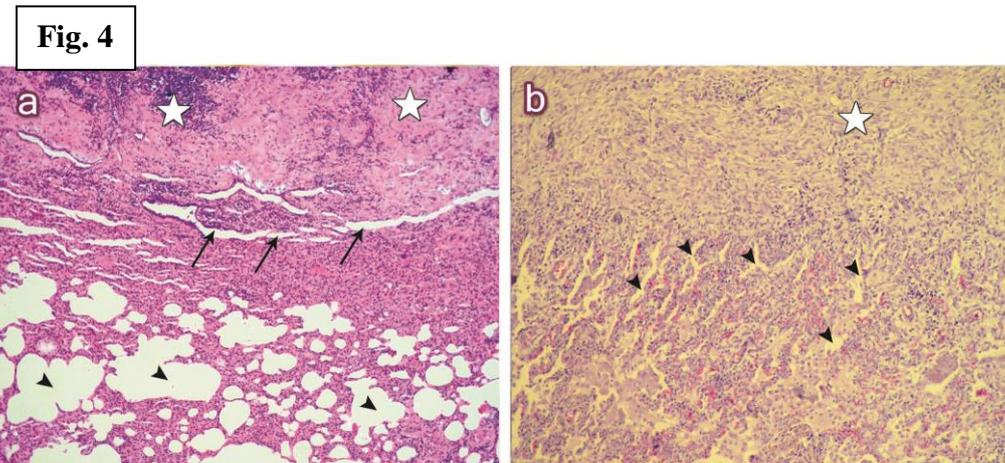
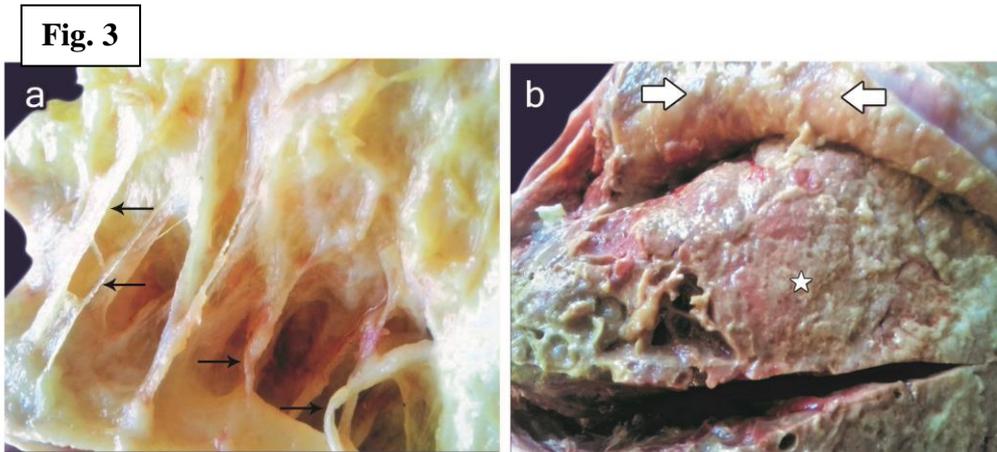
Microscopical examination

The main lung changes were in the presence of proliferating connective tissue replacing the destructed alveolar tissue besides existence of remnant of fibrinous exudation (Fig 4, a). The

alveolar walls were thickened due to development of dense fibrous connective tissue. The thickened alveolar walls were distorted giving rise to atelectasis of the some alveoli (slit-like appearance) (Fig 4, b). The interlobular septa were greatly thickened due to presence of granulation tissue. The granulation tissue was formed of collagen fibrils, fibroblasts, lymphocytes, macrophages and newly formed blood vessels, some blood vessels showed fibrinoid degeneration of thier wall.

Other lung lesions were recorded in the imported beef cattle and diagnosed and will be published in the further work.





LEGEND OF FIGURES

Figure 1: (a) Lung showing large amounts of fibrinous exudate above the pleura (arrows), adhesions of the thickened visceral and parietal pleura (double arrows) and thickening of interlobular septa. (b) Cut section showing a small portion of the healthy lung (star), while the remaining surface showed red to gray hepatization of lobules (white arrows) and thickening of interlobular septa (arrows head).

Figure 2: (a) Lung showing thickened pleura with deposited fibrin (stars) and leucocytic cellular infiltration (arrows). The alveoli as well contained fibrin threads mixed with red blood cells and leucocytes (arrows head) (Hx & E.X 4). (b): The interlobular septa showing expansion (Stars) with fibrin network, macrophages as well as neutrophils and congestion of inter alveolar capillaries (arrows) (Hx&E, X10), (c): Lung showing moderate degenerative changes of the bronchiolar epithelium, fibrinopurulent exudate in the lumen (star) and peribronchiolar leucocytic infiltrations (arrows) (Hx&E, X10). (d): Lung showing fibrino-cellular exudate in the alveolar luminae (stars) (Hx&E, X10).

Figure 3: (a) the parietal and visceral pleura were thickened and organizations of fibrinous exudate into fibrous bands (arrows) led to adhesions connect the thickened visceral and parietal pleura (b): Pneumonic lung showing large sequestrum consisting of necrotic parenchyma (star) surrounded by a thick fibrotic capsule (arrows).

Figure 4: (a) Pneumonic lung showing organized fibrinous pleuritis (star), atelectasis (arrows) and some compensatory alveolar emphysema (arrows head) (Hx&E, X10). (b): Pleura showing great thickening due to organization with collagenous connective tissue (star) and marked neovascularization and fibroblasts proliferation (arrows head) (Hx&E X10).

DISCUSSION

In the present study, 36 cases out of 110 lungs from 1450 slaughtered imported beef cattle showed fibrinous pleuropneumonia. According to the microscopic characteristic features of this lesion two categories were observed: (a) Acute fibrinous pleuropneumonia in 31 cases (28.1 %) and (b) organized fibrinous pleuropneumonia in 5 cases (4.5 %). This was nearly similar to the incidence recorded by (Schiefer *et al.*, 1978 and Haridy, 2003).

Our data revealed that, acute fibrinous pleuropneumonia in which their gross appearance surfaced great thickened parietal and visceral pleura in addition it was covered with abundant amounts of a yellowish fibrillary material (fibrin) diffusely. such copious fibrinous exudate above the pleura led to adhesion between visceral and parietal pleura as well as its adherence to the chest wall. The lungs were enlarged, non-collapsed, firm, hepatized and exhibited interlobular septa that were thickened with a gelatinous material (oedema). This picture was previously described by (Schiefer *et al.*, 1978; Kumar *et al.*, 2006 and Lopez, 2012).

Here in the present study, widening of interlobular septa with serofibrinous exudate, dilatation of lymphatic vessels and presence of multiple foci of hepatizations were observed. According to the age of the lesion, the alternating appearance of red and gray hepatizations gave the marbling or mosaic appearance. These observations are consistent with others observations (Schiefer *et al.*, 1978; Kumar *et al.*, 2006 and Lopez, 2012).

Histopathologically, the parietal and visceral pleura were expanded by a diffuse inflammatory infiltrate composed of degenerate neutrophils together with deposition of a fibrillary eosinophilic material (fibrin). The alveolar luminae were dilated and filled with fibrinous exudate which appeared as a fibrin network engaged with macrophages, neutrophils and erythrocytes. The interlobular septa was markedly expanded with fibrinous exudate, which formed of fibrin network, macrophages, neutrophils and eosinophilic faint pink serous exudate. This picture was previously described by Haridy (2003); Jubb *et al.* (2007); Lopez (2012) and Azizi *et al.* (2013). In the present study, organized fibrinous pleuropneumonia was recorded in 5 cases with an incidence of 4.5 %. This was grossly, manifested by presence of fibrous bands in the lobular pattern which somewhat compressed lung alveoli. In addition, the pleura was highly thickened as a result of organizations of fibrinous exudate which converted into fibro-vascular tissue. In more chronic cases, massive necrosis occupying large area of lung lobules (sequestrum) and surrounded by fibrous capsule was observed. Moreover variable alternation in the lung tissue in the form of red, gray hepatization, coagulative necrosis and thickening of interlobular septa had been detected. The same descriptions were reported by Schiefer *et al.* (1978); Gibbs *et al.* (1983); Trichard *et al.* (1989); and Lopez (2012). The microscopical finding of organized fibrinous pleuropneumonia in our results revealed that the alveolar walls were thickened due to presence of dense fibrous connective tissue. The thickened alveolar walls were distorted giving rise to atelectasis of the some alveoli (slit like appearance). The pleura and interlobular septa were greatly

thickened due to presence of granulation tissue formed of collagen fibrils, fibroblasts, lymphocytes, macrophages and newly formed blood vessels. This picture was previously described by Schiefer *et al.* (1978) and Jubb *et al.* (2007). Many authors in the previous studies mentioned that the etiological prevalence of the fibrinous pleuropneumonia was attributable to *Mannheimia hemolytica* Schiefer *et al.* (1978); Singh *et al.* (2011); and Jubb *et al.* (2007), *Mycoplasma mycoides subsp mycoides* Scheneider *et al.* (1994); Schiefer *et al.* (1978) and Jubb *et al.* (2007), *Pasteurella multocida* Schiefer *et al.* (1978); Radostits *et al.* (2000); Welsh *et al.* (2004) and Siqueira *et al.* (2018).

CONCLUSION

High incidence of fibrinous pleuropneumonia was recorded during slaughtering imported beef cattle at Middle East abattoirs in Abu-Simbel city, Aswan Governorate- Egypt. The etiological agents of the fibrinous pleuropneumonia could be attributable to *Mannheimia hemolytica*, *Mycoplasma mycoides subsp mycoides* or *Pasteurella multocida* infections. The importations of animals in a live state increase the possibility of infections in our animals, so we advise to perform strict quarantine measures to these animals, however slaughtering it in the original country is better to decrease the possibility of diseases transmission.

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الأفات الباثولوجية للالتهاب الرئوي البللوري الفيبريني ومعدل حدوثه في عجول التسمين المستورده والتي يتم ذبحها في مدينه ابوسمبل - محافظة اسوان

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أجريت هذه الدراسة على ١١٠ عينة من رئات عجول التسمين المستوردة عمرها من ٢ - ٣ سنة والتي يتم ذبحها في مجزر الشرق الأوسط بمدينه ابوسمبل - محافظة اسوان في الفترة من يوليو ٢٠١٧ الى مارس ٢٠١٨ بعد الذبح تم فحص رئات عجول التسمين وتسجيل الاصابات الظاهرية واخذ عينات للفحص الهستوباثولوجى وبعد الفحص وجد ان ٣٦ (٣٢,٧%) حاله تعانى من التهاب رئوى بللورى فيبرينى بمعدل عدد ٣١ (٢٨,١%) حاله التهاب رئوى بلورى فيبرينى حاد وعدد ٥ (٤,٥%) حاله التهاب رئوى بلورى فيبرينى مزمن . وقد نوقشت مسببات المرض واهميتها.