Animal Health Research Institute
Assiut Regional Laboratory

STUDIES ON PNEUMONIC LUNGS OF SLAUGHTERED BUFFALO-CALVE AT ASSIUT GOVERNORATE:- 1-ATYPICAL BRONCHO-INTERSTITIAL PNEUMONIA

(With 8 Figures)

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(Received at 13/11/1999)

دراسات على الإلتهاب الرئوي في عجول الجاموس المذبوحة في محافظة أسيوط الإلتهاب الشعبي – النسيج البيني الغير نموذجي

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أمكن تشخيص ثلاث حالات (١٢%) التهاب شعبي – نسيج بيني غير نموذجي في عدد خمسة وعشرين حالة التهاب في عجول الجاموس المذبوحة في مجزر محافظة أسيوط. وكانت التغيرات الباثولوجية على هيئة اجزاء صغيرة ومتعددة حمراء ارجواني اللون في كل من الفص القلبي والفص الرئوي بجوار الحجاب الحاجز وقد اختلفت درجة الاصابة في الرئات الثلاث . وكانت أهم التغيرات الميكروسكوبية هي زيادة في عدد الخلايا المبطنة للحويصلات الهوائية والخلايا المبطنة للشعب والشعيبات الهوائية وكذلك الخلايا الموجودة في جدار الحويصلات الهوائية مع وجود خلايا عديدة الأنوية. بالاضافة إلى وجود أجسام ضمنية حمراء اللون في الخلايا المبطنة للحويصلات الهوائية والشعيبات الهوائية. كما لوحظ رشح خلوي في الحويصلات الهوائية. نوقشت النتائج واتضح أن هذه الصورة مقاربة للآفات الباثولوجية الناتجة عن الاصابة بفيروس البارا انفلونزا.

SUMMARY

In a number of 25 pneumonic lungs, a typical broncho-interstitial pneumonia could be diagnosed in 3 cases. Gross findings revealed multiple scattered consolidated red purplish areas especially in both cardiac and diaphragmatic lobes. The degree of lung involvement varied in the three cases but reached to confluent consolidation in one case. The consistent microscopic lesions included proliferative hyperplastic changes of alveolar, septal and bronchiolar epithelium. In addition

multinucleated giant cells were prominent. Most characteristically was the presence of intracytoplasmic inclusion bodies in both the bronchiolar and alveolar epithelium. Cellular exudative changes were also seen. These microscopic lesions are basically similar to those induced by influenza viruses.

Keywords: *Para influenza group viruses-Lung-Buffalo-Broncho-Interstitial pneumonia.*

INTRODUCTION

Respiratory diseases are a major source of economic losses to the cattle, buffalo and sheep industry in many parts of the world Babiuk et al. (1987), Wohlgemuth and Herrick (1987) and Soroor (1999).

The causes and forms of pneumonia in domestic animals are neumorus and were classified into several categories. The infectious pneumonias have many causes, bacterial, viral, mycotic, parasitic or mixed infections, Darbyshire and Roberts, (1968), Gourlay et al. (1970), Jubb et al. (1985) and Mohamed et al. (1999). The authors added that the extrastresses of harsh climatic condition and husbandry practices and mycoplasmas probably activate respiratory viruses.

Many viruses were included in the aetiology of bronchointerstitial pneumonia in cattle, reovirus, adenovirus, infectious bovine rhinotracheitis, bovine respiratory syncytial virus, bovine virus diahrea, Herpsvirus, Rota virus, Parvovirus and parainfluenza, (Darbyshire, 1968 and Gourlay et al., 1970, Morein and Dinter 1975, Vanden Ingh et al. (1982), Ultenthal et al., 1996, Potgieter 1997 and Attia et al., 1999).

Para influenza group of viruses play an important role in initiation of broncho-interstitial pneumonia in domestic animals. The para influenza group of viruses include type A influenza virus which causes swine influenza and equine influenza, Parainfluenza-2 which have been recovered from humans, monkeys and dogs and Parainfluenza type-3. Viral Pneumonia in Cattle.

The isolation of the parainfluenza-3 virus from cases of shipping fever, other forms of respiratory illness or even uncomplicated cases has been made repeatedly in many parts of the world. (Allan et al., 1978, Bryson et al., 1979, Marcato et al., 1987 and Virakul et al., 1997).

Viral-bacterial synergism in respiratory diseases in cattle was reported by many authors, Jakab (1982), Yates (1982).

In the present investigation a gross and hitopathological findings for three cases of proliferative broncho-interstitial pneumonia in calves was carried out. On the basis of the histomorphologic alterations, atrial to relate the alterations to their etiologic agent was carried out and discussed.

MATERIALS and METHODS

The present investigation is conducted on twenty five pneumonic buffaloe calf lungs. They were obtained from Assiut Governorate abbatoir. Lungs were examined grossly and representative samples of lung tissues were fixed in 10% neutral buffered formalin. Paraffin sections were prepared and stained with haematoxylin and eosin, Bancroft (1982).

In the present paper the pathological findings in three calves with proliferative broncho-interstitial pneumonia were fully described, illustrated and discussed.

Results

Gross findings:

Although the pulmonary lesions in the three cases were basically the same, they differ only in the extent of lung involvement. The lesions involved both right and left lungs but consistently present in cardiac and diaphragmatic lobes espicially in the cranioventral regions. Varying-sized, multiple and red purplish areas of consolidation were prominent. In one calf the areas of consolidation were coalesced and appeared confluent.

Microscopic Findings:

The grossly consolidated areas appeared microscopically as areas of proliferative pneumonia. The septal cells were hypertrophied and hyperplastic inducing varying degrees of septal thickening (Fig, 1). The alveolar air spaces appeared either atelectatic, compressed narrow slit like openings, or appeared as emphysematous over distended alveoli.

In many areas proliferative alveolitis was prominent. In the alveolar lumina, mononuclear macrophages were recognized. Some of these macrophages were binucleated and even multinucleated, (Fig.2). Some of these cells revealed rounded eosinophilic intracytoplasmic inclusions (Fig.3). The latter are mostly surrounded by a clear hallow zone. Most of the alveolar spaces are packed with cellular exudate. The cellular exudate consists of proliferated alveolar epithelium, mononuclear macrophages and few neutrophils (Fig. 4). Some alveoli

revealed fibrinous like exudate infiltrated with a less number of neutrophilis. Some alveoli are partially or completely repithelialized. Regarding the intrapulmonary air conducting system, the alterations are present in both bronchi and bronchioles but mostly prominent in the bronchioles. The epithelial lining showed proliferative hyperplasia with intraluminal papillary ingrowths (Fig. 5). Many of bronchiolar epithelial cells showed intracytoplasmic acidophilic inclusions (Fig 6). They are nearly of moderate size and each is surrounded by a hallow zone. Some bronchioles revealed intraluminal neutrophilic cellular exudate (Fig.7). Sometimes the exudate contains necrotic and desquamated epithelial cells. In some bronchioles the inflammatory process involves the whole bronchiolar wall either acutly or chronically. Some areas show chronic bronchiolitis in which the bronchioles appeared with dustructed wall and obliterated lumen, Fig. 8) Many bronchioles revealed peribronchiolar lymphoid hyperplastic changes. The peribronchial lymphoid hyperplasia appeared as peribronchial lymphoid nodules.

In one case, the pulmonary vascular ramifications showed features of vasculitis with proliferative and alterative changes in their intimal endothelium, medial cells. Adventitial inflammatory cell infiltrations was also noticed.

DISCUSSION

Bronchointerstitial pneumonia is seen in many viral infections. Carlton and Mc Gavin (1995), mentioned that the principal lesions are air way epithelial damage and prolifiration of pneumocyte type II. Marcato et al. (1987), and Rubin and Farber (1994) reported that viral infections of the pulmonary parenchyma produce interstitial, rather than alveolar pneumonia and diffuse alveolar damage.

In the present work septal thickening with cellular infiltrations is a prominent feature. In addition alveolar cell as well as bronchial epithelial damage were seen in all of the examined cases. The proliferative hyperplastic alterations in both of the alveolar and air way conducting system was noticed also by Bryson et al. (1983) and Viuff et al. (1996).

These proliferative changes could be related either directly to the insult or even partially to cover the epithelial cleft left by necrosis of the cells caused by the etiologic agent.

In our results many alveolar spaces were packed with cellular exudate which consists of proliferated alveolar epithelum, mononuclear,

macrophages, few neutrophils and desquamated epithelial cells. The alveolar exudate could be probably attributed to the damage of the alveolocytes which disrupted their light junction and permitted the exudation from the interstitium into the alveolar spaces. In the present paper the presence of neutrophils in the alveolar exudate is either an expression for the acute process intiated by the necrotic debris or could be related to secondary bacterial infection. Although in most bacterial infections the intralveolar exudate predominates and the intersitium is incidentally involved.

In some alveoli partial or complete repithelialization was noticed. This is also related to the regenerative hypertrophic proliferation of pneumocyte type II cells.

The most important viruses which cause bronchointerstitial pneumonia in cattle were infectious bovine rhinotracheitis, bovine respiratory syncytial virus, Herpes virus and par-influenza viruses, Marcato et al. (1987) Virakul et al. (1997) and Rusval and Fodor (1998). Necrotizing bronchiolitis necrosis of type I pneumocytes with hyperplasia of type II pneumocytes and mild interstitial reaction was also described by Carlton and McGavin (1995). In the present paper atelectasis and emphysematous alveoli were seen. Atelectasis could be related to the obstruction of the Conducting air ways by cellular proliferation and necrotic exudate. In addition pressure of the alveoli by the interstitial reaction can not be neglected. The overinflation of some alveoli is a compensatory mechanism. The prominent lesions in bovine Syncytial virus infection are acute bronchiolitis and syncytial giant cell formation, Kimman et al. (1989), Viuff et al. (1996). The proliferating bronchiolar epithelial cells may contain acidophilic intracytoplasmic inclusion bodies, (Jacobs and Edington, 1975; Thamas et al., 1982, Trigo et al., 1984, Baker et al., 1986 and Redondo et al., 1994 discussed the interaction between bovine respiratory syncytial virus and pasteurella infection in the ovine lung. The authors described suppurtive bronchopneumonia, columnar, cuboidal, hemispheric or squamous, syncytial giant cells with intracytoplasmic inclusions. Syncytial giant cells could not be observed in our results.

In case of bovine rhinotrachitis virus infection the viral replication, the epithelial damage and the characterstic inclusions are mainly in the upper respiratory tract in natural infections (Jubb et al. 1985). Although we have dealed with the lung parenchyma, no history for other forms of rhinotracheitis viral infection was reported.

In the present paper, the histomorphological differences probably exclude this type of infection. In our investigation the alveolar and bronchiolar reactions as well as the interstitial pneumonia with the presence of the acidophilic intracytoplasmic inclusions give resemblance to the histomorphological picture described in parainfluenza virus (Dawson et al., 1965, Bryson et al., 1978, 1979; Allan et al., 1978, Jubb et al., 1985 and Carlton and McGavin 1995). Omar et al., (1966) described also similar picture in colostrum-deprived calves. In the present investigation have seen that the exudations into the alveolar spaces are more copious and extensive. Darbyshire et al. (1966) omar et al. (1966) and Coele (1971) stated that more alveolar exudation in parainfluenza viral pneumonia than in adeno-and Reo-viral pneumonia was present. The later authors reported also that the proliferative bronchiolitis is of greater exuberance than that in reoviral infection. The bronchio-alveolar and interstitial involvement and the presence of inclusion bodies in bronchiolar and alveolar epithelium in the present investigation may indicate parainfluenza type three infection.

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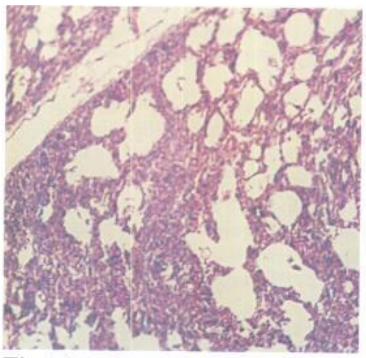
LIST OF FIGURES



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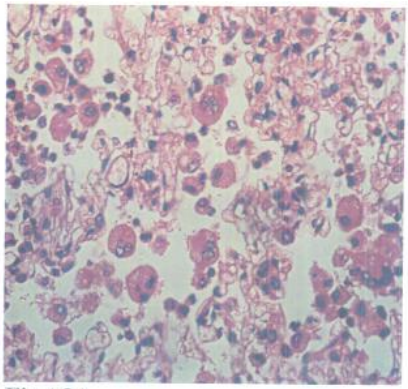

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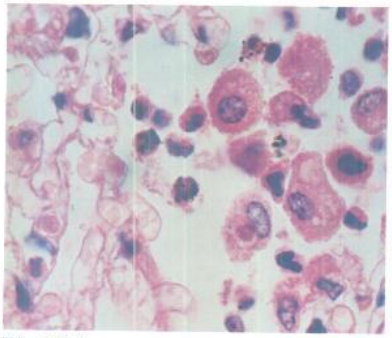

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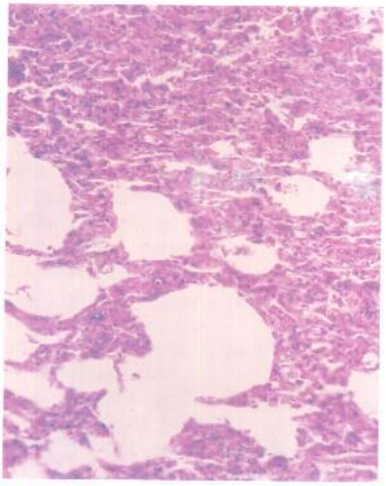

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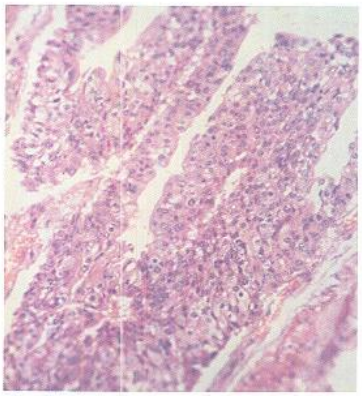

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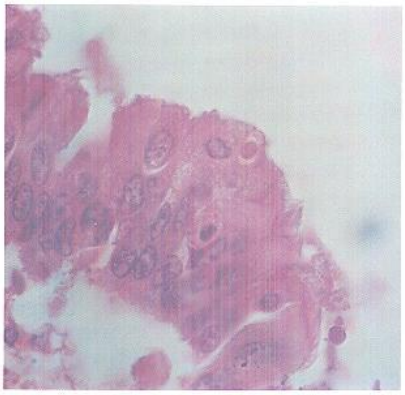

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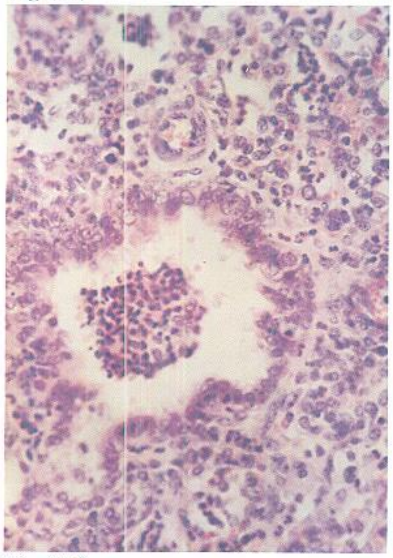

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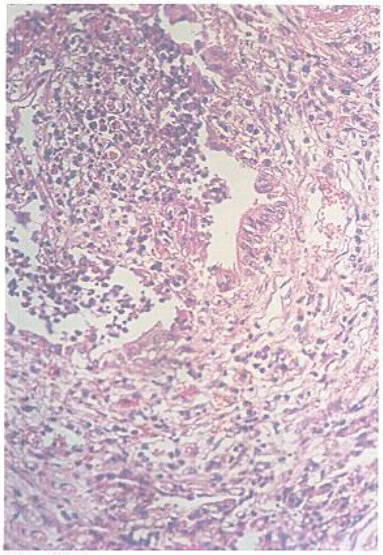

Fig (8)