دراسة على التهاب الرئوى فى الاغنام وخصوصا الحالات المرتبطة بعدوى الميكوبلازما

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لقد تم فحص ٨٧ رئة لأغنام مصابة بالتهاب رئوى . ومن هذا العدد تم زرع ٥٣ حالة للميكوبلازما ، وقد وجد ٢٢ حالة من الميكوبلازما أرجينى بالاضافة الى ال٤ عترات للميكوبلازما مخمرة الجلوكوز .

وقد تم عمل فحص عينى وميكرسكوبى دقيق . لوحظ بالفحص العينى لحالات التهاب الرئوى الميكوبلازما احتقان وتلون الرئة بلون رمادى داكن . وكان مقطع الرئة داكن ومتصلب الملمس . وكانت الفصوص الأمامية أكثر عرضة للاصابة من غيرها وعزى هذا الى حدوث العدوى عن طريق استنشاق الهواء . وبالفحص الميكرسكوبى ظهر الالتهاب الرئوى البين خلوى الانقسامى مع اتساع انتشار الخلايا الليمفاوية الانقسامية .

ومن هذا يتضح أن عدوى الميكوبلازما فى الأغنام تبدأ بالتهاب رئوى بين خلوى انقسامى مع زيادة من عدد الخلايا الليمفاوية الانقسامية ، التى تبدأ فى الجزء الأمامى والجانب البطنى ثم تستمر فى توزيع فصيصى الى أن تشمل كل الفصوص الرئوية .

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STUDY ON PNEUMONIA IN SHEEP WITH PARTICULAR REFERENCE TO THOSE CASES ASSOCIATED WITH MYCOPLASMA SPECIES.

(with one table and 10 fignres)

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SUMMARY

87 ovine pneumonic lungs were examined. Out of these cases 52 cases were subjected for mycoplasma cultivation 22, were found to recover Mycoplsama arginini in addition to 4 strains of glucose fermenter mycoplsama were obtained.

Gross and detailed micoscopic investigations were performed. It was concluded from this work that the gross appearence in ovine mycoplasmal pneumonia consists mainly of congestion to dark greyish colour. Cut section have dark and of firm texture. Anterior lobes were found to be the most liable parts to harbour the infection thus means an air-born infection. Microscopically the main lesion was proliferative interstitial pneumonia with excessive lymphoid infiltration. From such cases it could be concluded that mycoplasma infection in sheep initially induces proliferative interastitial pneumonia with lymphoid hyperplasia which begins in the cranial part and ventral side then proceed to involve the whole lebes of the lung in a lobular distribution.

INTRODUCTION

Respiratory infections represent one of the main outbreaks among sheep, either alone or as a complex of pneuomonia enteritis disease condition. Only few published reports describing the praticulate type of pneumonia known as interstitial virus pneumonia, pneumonitis or atypical pneumonia. Chronic interstitial pneumonia characterized by progressive develompment most commonly ending in death has been reported in Egypt as well as many other countries.

This work was done to find a (pathological) pathognomonic lesion in the lung of atypical pneumonia in sheep. MITCHELL (1915) was the first worker that gave a discription for Atypical pueumonia known as Jaagsiekte's disease. GORODRY (1925) and Davis (1960), gave the discription for the mycoplasmal pneumonia in sheep. This condition was givin by these authors to consists mainly of an incidious form of progressive pneumonia preceeded by thickneing of interalveaolar septa with hyperaemia and accu mulation of macrkophages, and lymphocytes especially at the peribronchial and perivascular tissues.

A condition of adenomatosis of pulmonary tissue as an epithelization of the lung was the main feature of Caprine mycoplasmal pneumonia. This was given by Moulton (1960), Stamp and Nisbet (1863), ADLER (1965), WANDERA (1968), and WANDERA (1970). Pavlov (1969) classified pneumonia of neorikettsial cause according to the type of cellular response into, Acute interstitial pneumonia, histiocytic interstitial pneumonia, lymphohistiocytic interstitial pneumonia and lymphocytic pneumonia. In most of these conditions it was found by these authers, that initially lymphatic vessels showed necrobiotic and necrotic changes and giving the name necrotic interstictial pneumenia (PAVLOVE 1969) or pneumonitis. JUBB AND KENNEDY (1970) mentioned that the gross appearence of atypical pneumonia consisted of enlargement, heavinesss, light grey or purplish colour with a widespread distribution of greyish patches.

WOOLF ET. AL. (1970), ST. GEORGE ET. AL. (1971) considered that the proliferation of septal cells was of lobular nature while affected lobules were irregularly distributed throughout the lung consolidation varying from hepatization of apical, cardiac, and anterior part of diaphragmatic lobes to total involvement of the lungs. The same was discribed by FOGGIE (1972) who added that in experimentally infected animals with Mycoplasma agrinini, there were no macroscopic changes of any kind in the thoracic or abdominal organs. The right apical lobe were found to show localized proliferative interstitial pneumonia.

ST. GEORGE (1972) in his advanced investigation mentioned that there was extravasted erythrocytes into the alveolar spaces which were mixed with cellular debris, mucous and polymorph nuclear cells, While there were oedematious and hypercellular septa.

MATERIALS AND METHODS

Samples from 80 congested lung from slaughtered sheep were taken, 10 apparently normal lungs were taken as a control lungs. 7 cases were obtained from necropsied animals at P.M. room of the pathology dept. of Faculty of Vet. Med. Cairo University. Preliminary gross examination was performed to show the extent and distribution of lesion among the different lung lobes. Part from affected lung tissues was fixed in 10% formalin then procceded as routine method of parffin embeding method, Harris Haematoxylin and Eosin was of choice, Van Gieson's, Mallory P.T.A.H., PAS and Weigert's resorcinfuchin stain were performed (Culling 1963). In Collaboration with the Mycoplasma division, Dokki research institute all Mycoplasmal studies were made (Al-Zeftawi 1973).

RESULTS

Gross examination revealed the results tabulated as follows :

TBALE 1 **Shows distirdution of lesions in different lobes of examined sheep,s lung.**

Lobes affected	R	L	Both lungs	Total Samples	Incidence %
All lobes of both sides			10	10	11 .50
All lobes of one side	3	14	—	17	19 .53
Anterior lobes	15	38	—	53	60 .92
Diaphragmatic lobes	1	6	—	7	8 .05
Total	19	58	10	87	100.00

From this table it could be concluded that the anterior lobes were the most frequently affected parts in the lung.

Microscopicaiiy : The main pathological changes were proliferative interstitial pneumonia with excessive lymphoid hyperplasia. From such cases Mycoplasma arginini were recovered from 22 out of 52 cultivated cases, in addition to 4 strains of glucose fermenter Mycoplasma. These microscopical findings cuold be collected as :

1. Thickening of the alveolar wall (interalveolar septa) this thickening was found in the form of either extensive proliferation of the alveolar lining. or in other cases there was congestion and infiltration of lymphocytes and macrophages into the area. Many cases showed combination of proliferation of the alveolar wall and an infiltration of cellular components. Fig. 1.

This combined lesions were sometimes so extensive as to be finger like projection casing into or across the alveolar space. The thickening might be uniform thus causing a variable degree of narrowing of the alveolar lumen.

2. Lymphoid infiltration in the peribronchial, peribronchiolar and perivascular tissues : There was an expansion of the peribronchial and peribronchiolar tissue due to lymphocytic cellular infiltration. This was observed around the blood vessels too, Fig. 4. Monocytes, histiocytes, plasma cells and neutrophils were added in some of the lymphocytic foci. The degree of lymphohistiocytic infiltration varies widely in different cases and in different lobules of the same affected lung. peribronchial cuffs were found in most lesions (Fig. 2., Fig. 3.).

3. Proliferation, desquamation and degeneration of bronchial and bronchiolar epithelium :

Hyperplasia of bronchial epithelium was observed in 23 cases out of 87 cases examined, while 20 cases showed degeneration and desquamation. Accompaining hyperplasia with degeneration and desquamation was found in only 8 cases examined. Hyperplasia was found in the form of focal thickening of the bronchial M.M. due to its hyperplastic epithelium.

These foci of epithelial hyperplasia occured as tongue like projections into the lumen. Fig. 7.

In case of degenerated and or desquamated epithelial lining it was found to be accompanied with cellular debres and inflamatory cells within the exudate in the bronchial and bronchiolar lumen, as Fig. 8. Cases which posessed both hyperplasia, degenerating and desquamating process, it was observed that there is plug of pinkish exudate mixed with degenerated cells of epithelial and inflammatory origin Fig. 9.

4. Exudative changes :

Exudation as manifested by oedematous fluid was found inside the alveoli with or without the presence of inflammatory cells and large macrophages. Sometimes this exudate was noticed in and around the bronchiole and bronchi. Subpleural oedema and inflammatory process was also a prominent feature Fig. 10. This exudeate was serous, pink or in few cases was fibrinous.

It could be deduced from the 87 cases examined that one or more of the preceding main changes was met in the animals. Some cases showed combination of alveolar wall thickening, peribronchial and peribronchiolar hyperplasia, degenerative and desquamative. epithelial changes as well as exudative changes.

Other group of cases showed alveolar thickening, lymphiod hyperplasia with degenerative and desquamation of epithelial lining. Few cases had thickened alveolar wall with lymphiod hyperplasia only.

Mycoplasma arginini was obtained from 22 out of 52 cultivated, in addition 4 strains of glucose fermenter Mycoplsama were obtained.

DISCUSSION

Naked eye examination of the total cases collected revealed bilateral affection in 10 cases. Such finding agreed wih WANDERA 1970, Stamp and Nisbet (1963) and WOOLF ET. AL. (1970) who concluded that affection of all lobes resulted from extension of the lesion from one part to another begining from the anterior lobes.

Anterior lobe involvement was found in 60. 92% a result that coincided with those given by STAMP and NISBERT (1968), WOOLF Et. AL. (1970) and FOGGIE and ANGUS (1972).

Diaphragmatic lobes were noticed to show limited lesions without involvement of anterior lobes in 7 cases of this investigation, a finding that disagrees that mentioned by Stamp (1963) and WOOLF ET. AL. (1970) while most of the examined lungs in the present study were light to dark red in colour, as described by FOGGIE and ANGUS (1972). However JUBB and KENNEDY (1970) mentioned that the lesion took mostly greyish yellow colour.

The consistency of most of examined cases was slightly firm however it was mentioned that the lungs were tough or meaty DAVIES (1960) and Stamp and NISBERT (1963). On the other side JUBB and KENNEDY said that the lesioms of Mycoplasma pneumonia were rubbery or like rubber sponge but in this work, only few cases showed firm consistancy. This could be explained as a result of excessive alveolar wall proliferation and lymphoid hyperplasia. These cases having slight proliferation and hyperplassia were slightly firm in consistancy.

Histopathological results obtained from examined pneumonic lungs proved + ve to Mycoplasma infection, gave the characteristic alveolar wall thickening due to proliferation of the lining epithelium.

This observation agreed well with GOWDRY a1925, MOULTON 1961 and St. GEORGE 1972.

Additional infiltration with monomeuclear cells observed in this study is nearly a uniform finding among those givin by STAMP amd NISBET, 1963 and JUBB and KENNEDY (1970). Also the thickening of the alveolar walls due to congestion, oedema and monocytic infiltration was noticed and comes in agreement with an observation of WANDERA (1968) and (1970). Alveolar walls were greatly thickened to a point that a scarcely and alveolar opening remained, a finding which was detected in 13 cases of this work DAVIES (1960), mentioned that thickening of the alveolar walls was due to conenective tissue. However, during this study the application of specific stains for c.T. (Van Giesson's and Mallory's P.T.A.H. stain) revealed that this increased interalveolar septa was due to epithelial proliferation. The cellular contents inside the alveolar lumen were found to consist of macrophages, monocytes and lymphocytes, and observation that coincided with those given by GOWDRY 1925, DAVIES 1960 and WANDERA 1970. Meanwhile, desquamated alveolar macrophages and lymphocytes were observed in this study, a finiding that was given by JUBB and KENNEDY (1970).

St. GEORGE (1972) was the olny one who mentioned that there is extravasation of blood into the alveolar lumen beside cellular debris, mucous and polymorphs, these findings agreed with the findings obtained from this work.

The bronchi and bronchioles were found to be surrounded by considerable amount of lymphoid hyperplasia which compressed the air passages and the alveolar spaces too. This observation was also given by MITCHELL (1915) , WANDERA (1968), and PAVLOV (1969). Other small bronchi and bronchioles were idented from one side by lymphoid accumulation, a result which was also mentioned by Jubb and KENNEDY (1970) and FOGGLE and ANGUS (1972).

Perivascular lymphoid condensation was observed in some cases, a finding that agrees with those given by MITCHELL, 1915, STAMP and MISHET, 1963, JUBB and KENNEDY, 1970 and WANDERA, 1970. Observations of this study revealed hyperplasia of the bronchial and bronchiolar epithelium such finding was also described by MOULTON (1950) .

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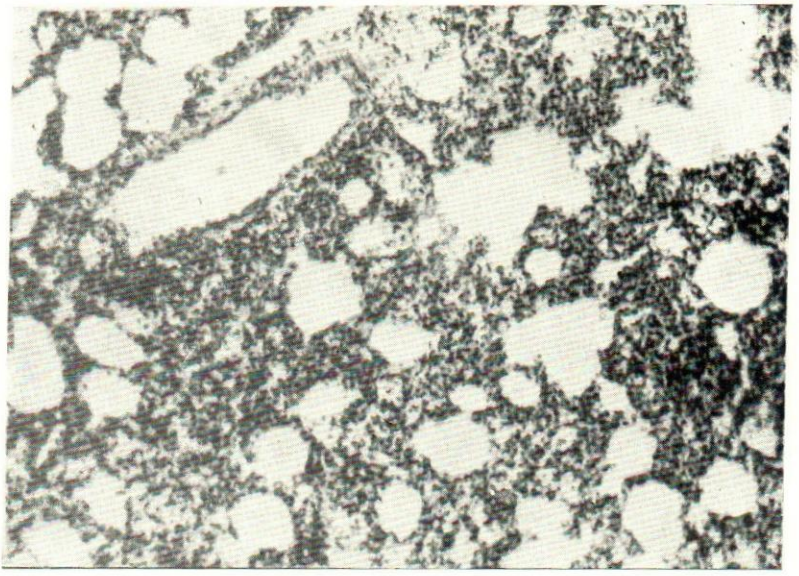


Fig. 1— Thickening of the alveolar walls due to cellular proliferation. (x100)

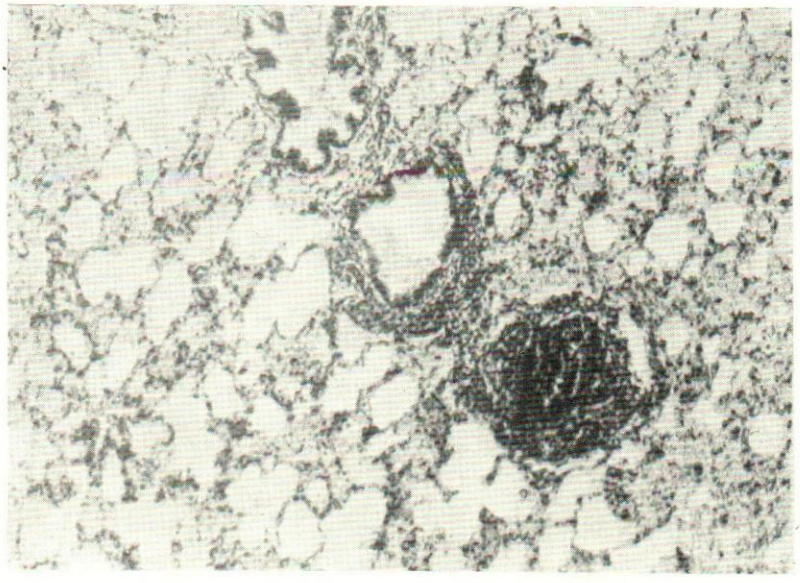


Fig. 2— Peribronchial cuffing and isolated lymphoid nodules. (x100)

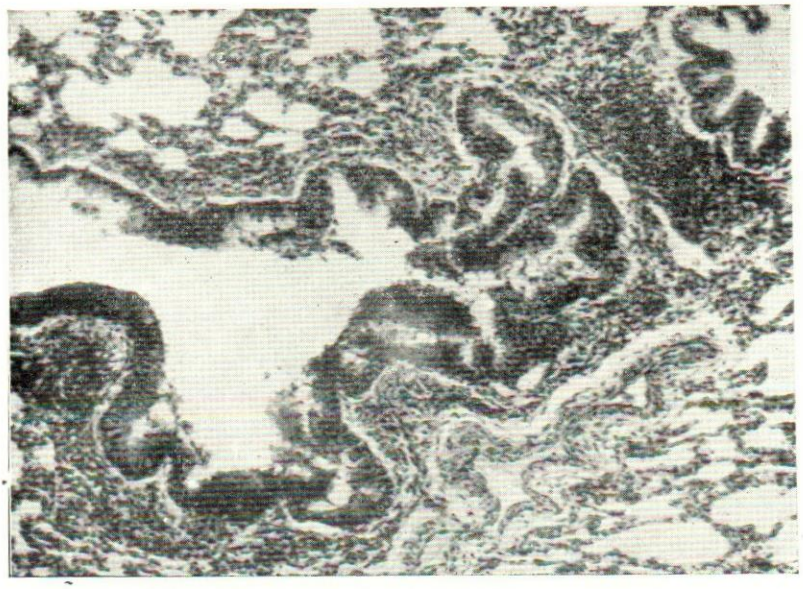


Fig. 3- Showing peribronchial lymphoid aggregation. (x100)

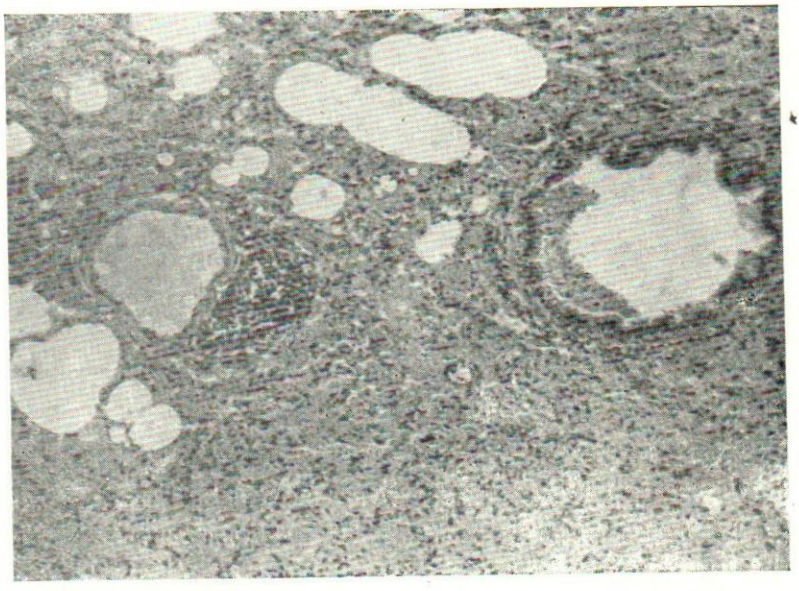
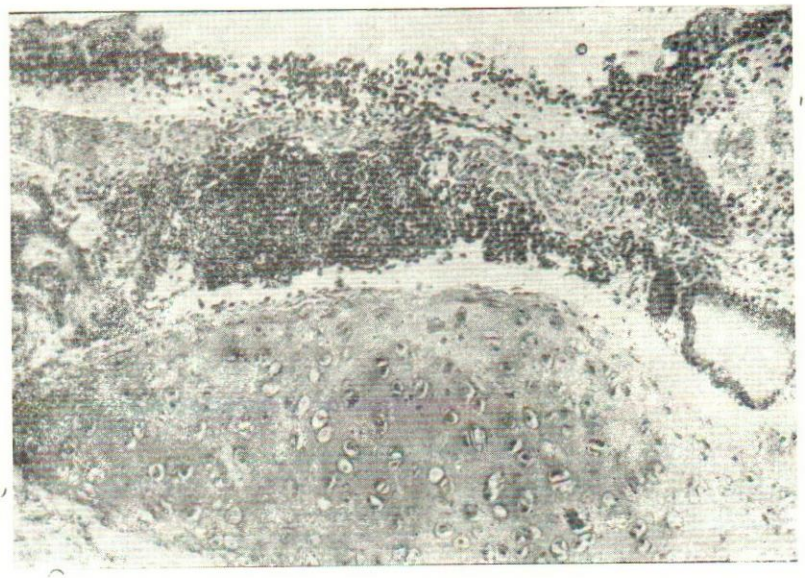


Fig. 4— Showing lymphoid hyperplasia in a perivascular nodule formation. (x100)



Fih 5— Showing the bronchial wall with denuded epilhelium and lympoid aggregation between the muscularis layer and the cartilagenous disk (x100)

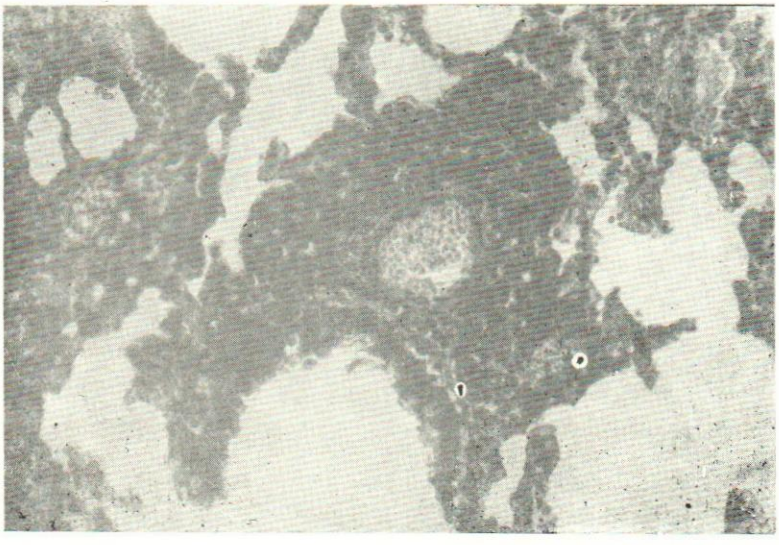


Fig 6— Showing Perivascular Cuffing (X250

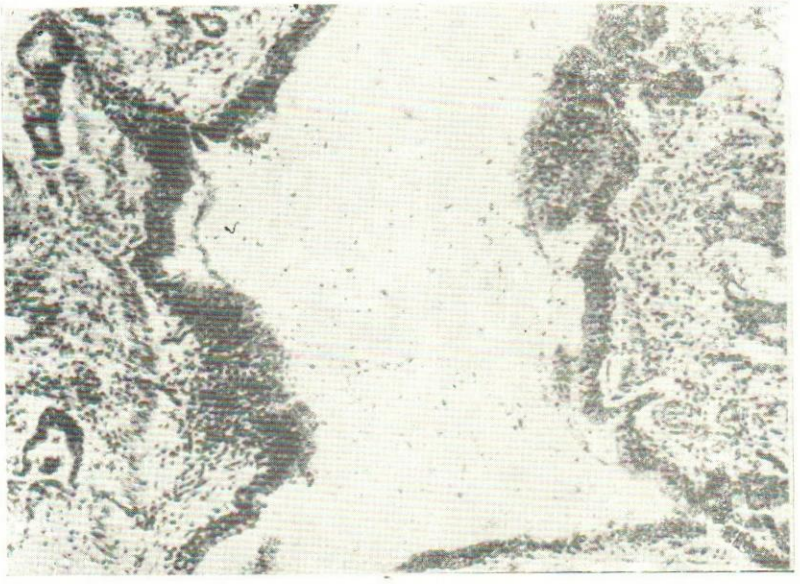


Fig. 7— Showing focal hyperplasia of the bronohial epitnelium. (X100) H. E.

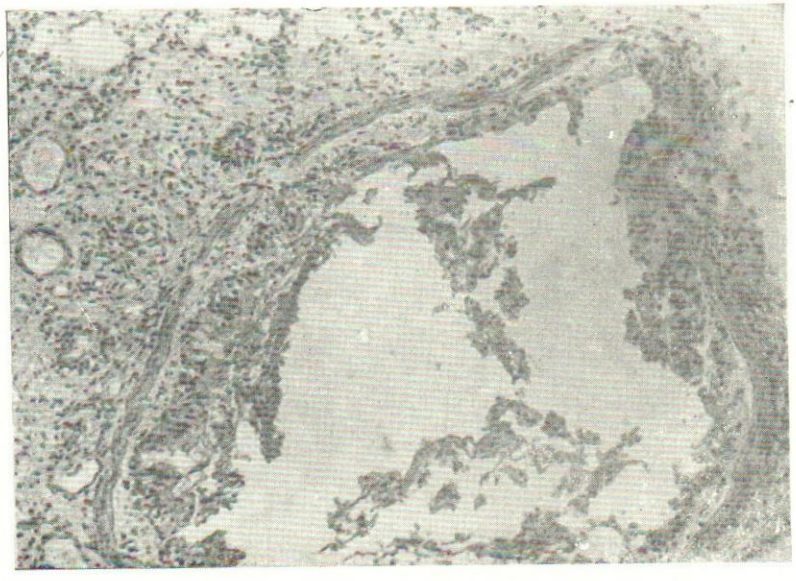


Fig. 8 —Showing a bronchiole with denuded epithelium and celluular debris in the lemen. (X 100)

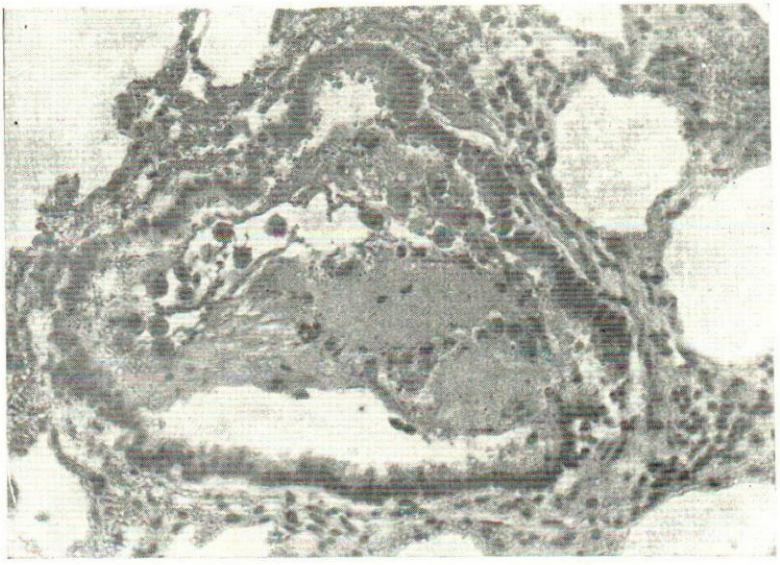


Fig. 9—Showing a bronchiole containing an exudate together with large macro phages. (X 250) H. E.

Fig. 10—Thickening of the interstitial septa with connective tissue formation. (X100) H. E.