PATHOLOGY OF THE RESPIRATORY SYSTEM IN RABBITS

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Thesis submitted in partial fulfilment of the requirement for the degree of

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University, Thrissur

2003

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DECLARATION

I hereby declare that the thesis entitled **"PATHOLOGY OF THE RESPIRATORY SYSTEM IN RABBITS"** is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

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Certified that the thesis entitled **"PATHOLOGY OF THE RESPIRATORY SYSTEM IN RABBITS"** is a record of research work done independently by **Dr. Rekha, S.,** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to her.

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ACKNOWLEDGEMENTS

I thank the Almighty for selecting me as one of the luckiest persons who could work under the meticulous guidance of my outstanding mentor, ***Dr. C. R. Lalithakunjamma****, Associate Professor, Centre of Excellence in Pathology and Chairperson of the Advisory Committee. I am in short of words for expressing the gratitude and thankfulness for the motherly love and care offered during the entire period of this study without which this task would not have been completed. I would also like to express my heartfelt thanks towards her family for all the moral support.*

I express my profound gratitude to ***Dr. K. V. Valsala****, Professor and Head, Centre of Excellence in Pathology for her incessant affection, expert advice, generous support, timely help and also for providing me the facilities required for the conduct of the research work.*

I, with absolute pleasure put on a record my deep sense of gratitude and respect to ***Dr. N. Divakaran Nair****, Associate Professor, Centre of Excellence in Pathology for his expert advices throughout the period of this work.*

I would like to express my heartfelt thanks to ***Dr. M. Mini****, Assistant Professor, Department of Microbiology, for her whole- hearted co-operation and the prompt correction of the thesis*

I wish to express my sincere thanks to ***Dr. T. Sreekumaran****, Professor, Centre of Excellence in Pathology for the valuable help provided. Special note of thanks to* ***Dr. Koshy Varghese****, and* ***Dr. N. Vijayan****, Associate Professors, Centre of Excellence in Pathology, for the encouragement, timely help and the valuable advices provided. I am also thankful to* ***Dr. Mammen. J. Abraham,*** *Associate Professor, Centre of Excellence in Pathology.*

I place on record my reverence and gratefulness to ***Dr. A. Rajan*** *(Dean, Retd.).*

*I am grateful to **Dr.E. Nanu**, Dean i/c, College of Veterinary and Animal Sciences, Mannuthy for providing me the facilities needed for this work.*

*I am in short of words to express my deep sense of gratefulness to my colleagues **Dr. Pradeep, Dr. Sivakumar and Dr. Smitha** for being always with me in all my thicks and thins with encouraging words, incessant help and friendly atmosphere.*

*I am very much grateful to **Dr. Antony** and **Dr. Manju**, Department of Microbiology for the valuable help rendered for this study.*

*Special note of thanks to **Dr. Sajitha** for the sisterly affection, pleasant support and unforgettable help given during the study.*

*I remember with gratitude the co-operation and the warm friendship showered by **Dr. Suraj, Dr. Balasubramanian, Dr. Kalaiselvan, Dr. Bissy, Dr. Dhanya Menon, Dr. Chitra, Dr. Deepa Jacob, Dr. Seena, Dr. Bindu Mathew, Dr. Suprabha and Dr. Seema.***

Thanks are due to all the non-teaching staff members of department of Pathology for the pleasant co-operation.

*My sincere thanks are due to **Mr. O. K. Ravindran**, C/o Peagles for his well timed assistance in the preparation of the thesis.*

*With immense pleasure I would like to acknowledge the moral support and sustained encouragement extended my beloved father, sister and brother in law. I would like to express my sincere gratitude to my soul mate **Umesh** for all his prayers.*

*Above all, I bow before the **Almighty God** and my late **Mother** for all the blessing showered on me which helped my small boat to reach the shore safely.*

REKHA, S.

CONTENTS

LIST OF FIGURES

Introduction

1. INTRODUCTION

Rabbit (*Oryctolagus cuniculus*) farming is a rapidly growing industry in India. They are raised for a variety of purposes like meat, fur and skin production besides their use in the laboratory. As efficient converters of vegetable protein in to high quality animal protein broiler rabbit has gained much popularity among people and their rearing has become a common occupation. Angora rabbit farming for fur and wool production has emerged as a lucrative enterprise in India, since average wool yield per rabbit is four hundred to five hundred grams per annum with an average market value of rupees 1000.00 to 1500.00 per kilogram. It is mainly concentrated in the foot hills of the Himalayas, particularly in the states like Himachal Pradesh, Jammu and Kashmir and hilly areas of Uttar Pradesh.

Rabbit farming has potential to serve all sections of the society providing work for woman, children, aged or handicapped people. Since rabbits are amenable to backyard farming, semi-intensive and intensive systems of management rabbit rearing can be a profitable operation for both landed and landless small farmers.

They are easy to handle, highly prolific with short generation interval and they have good capacity to utilise fodder and agricultural by-products. Further, rabbits require less concentrate feed which curtails the cost of feeding. Hence, many farmers maintain rabbits for a supplementary occupation to increase income from the farm operations.

Rabbit meat is a good source of high quality protein, low in fat content and is sentimentally accepted by the non-vegetarians of all religions. Rabbit rearing for meat has significant potential to improve food security and nutrition in developing countries. India is producing 5000 to 19000 tonnes of rabbit per year.

Broiler rabbit farming has been accepted as a source of income among the educated people in the Kerala too.

The production of rabbit meat on an industrial scale has been very slow to develop. Excessive mortality among the growing broiler rabbit has hindered the tradition to mass production. Therefore the success of rabbit farming at all levels depends upon the identification and control of the diseases common to the rabbits and the effective management practices. The introduction of giant varieties of rabbits has brought disease problems among the rabbits in many of the rabbitries.

Diseases are responsible for considerable economic loss in the rabbit farms since they lead to lowered productivity and increased mortality. Many epizootics appear in the colonies maintained by the research institutions often following the stress of experimentation or the introduction of new rabbits. Diseases occurring in research colonies adversely affect the progression of the research.

One of the disease conditions severely affecting the rabbits causing increased mortality among the flock is the diseases of the respiratory system. Chemicals and infectious agents can enter into the respiratory tract through the inspired air which get in contact with the mucous membrane of the nares, paranasal sinuses, pharynx, larynx, trachea and bronchi and lead to disease conditions. Respiratory system is also susceptible to the disturbances of circulation and parasitic diseases. Hence, data on the spectrum of etiological agents as well as pathological changes leading to respiratory diseases will be very much helpful in practicing suitable prophylactic measures including vaccination.

In this context, the present study was conducted to gather information on the disease conditions commonly occurring in the respiratory system of rabbits, so that appropriate control measures can be taken and thus mortality can be prevented to a high extent. The objectives of the present investigation include the study of the various disease conditions affecting the upper respiratory tract,

the lungs and the associated lymphnodes, their gross and histopathological features and isolation of the bacterial organisms associated with the respiratory infections in rabbits.

Review of Literature

2. REVIEW OF LITERATURE

2.1 ETIOLOGY OF RESPIRATORY TRACT DISEASES

Jones and Hunt (1983) reported that disturbances of circulation, viral, bacterial and fungal infections, infection due to mycoplasma, rickettsia, spirochetes, protozoal diseases, parasitic helminths and arthropods and extraneous poisons have profound effect on the respiratory system.

The location of larynx and trachea is such that frequently they become inflamed as part of inflammatory disease of either the upper or lower part of the respiratory tract (Dungworth, 1993).

2.1.1 Bacteria

Pasteurellosis was the most common and important bacterial infectious disease complex in rabbits (Flatt , 1974).

Stookey and Moe (1978) reported that Pneumococci was one of the bacterial organisms causing pneumonia in rabbits.

Suppurative pneumonia in rabbits was reported by Sudharma and Sulochana (1985) and they isolated *Moraxella nonliquifaciens* from the lungs.

Nair *et al.* (1987) recorded haemorrhagic tracheitis in rabbits. Gross examination revealed hyperaemic and oedematous trachea, enlarged dark brown and firm lungs and hyperaemic bronchial lymphnodes. Histolopathology of the trachea showed engorgement and rupture of the capillaries and extensive destruction of the mucosa and infiltration of inflammatory cells. Necrotic debris were found in the bronchioles with cuff of inflammatory cells around the bronchi. Erythrocytes, neutrophils and lymphocytic accumulations in the alveoli and rupture and fusion of the alveoli were seen in the lungs. Congestion and reticular

hyperplasia in the lymphnodes were the other lesions noticed. Gram-negative bipolar *Pasteurella sp* organisms were found in the tracheal smears.

Lebban *et al.* (1989) studied lung lesions of rabbits immunized against pasteurellosis and challenged with virulent *Pasteurella multocida* and observed bronchopneumonia and pleuropneumonia in rabbits vaccinated intravenously. Absence of lung lesions were also noticed in rabbits vaccinated by aerosol or conjunctival route.

Shah *et al.* (1989) observed congestion, emphysema and petechial haemorrhages in the diaphragmatic lobes of the lungs along with enteric lesions in rabbits affected with colisepticemia. Histologically they found varying degree of congestion, haemorrhage, moderate alveolitis and alveolar oedema in the lungs. They have isolated *Escherichia coli* from the lungs.

Devi *et al.* (1990) conducted a study on rabbit mortality for a period of four years and concluded that 84.11 % of the rabbits died due to pneumonia caused by *Pasteurella sp.*

Glavits and Magyar (1990) experimentally inoculated *Pasteurella multocida* serotypes A:3, A:4 and A:12 in rabbits and observed serous rhinitis, tracheitis, atrophy of lymphoid tissue and pleuritis with accumulation of fibrinopurulent exudate in the thoracic cavity. They also noticed acute serous rhinitis and acute catarrhal fibrinopurulent pneumonia in some rabbits from which they isolated *Bordetella bronchiseptca.*

Redondo *et al.* (1990) inoculated *Pasteurella multocida* type:E intra tracheally in to rabbits and observed colonisation of alveoli and bronchi by the bacteria. They stated that the destruction of the bacteria by neutrophils and mononuclear phagocytes lead to the release of bacterial endotoxins causing vascular lesions and hypoxia leading to necrosis of alveolar and bronchiolar epithelial cells.

Frymus *et al.* (1991) observed atrophic rhinitis in rabbits and isolated toxigenic *P. multocida* from the nasal cavity.

Pneumonia and pleuritis in rabbits inoculated with purified protein toxin from *P. multocida* strain D were reported by Chrisp and Foged (1991).

Dillehay *et al.* (1991) recorded fibrino-suppurative pleuritis, pyothorax and pneumonia in rabbits died in an outbreak . *Pasteurella multocida* A:3 was reported as the etiologic agent.

Turbinate atrophy in rabbits infected with *Pasteurella multocida* serotype A:12 was reported by Di-Giacomo *et al.* (1993).

Hastie *et al.* (1993) stated that the Cilia- Assosiated Respiratory (CAR) bacilli (Gram-negative bacteria closely related to *Helicobacter sp.*) found in the rabbit respiratory tract lead to pathological changes in the ciliated cells by decreasing the number of cilia in the tracheal epithelium .

Koshimizu and Sato (1994) isolated P. multocida serotype : A from cases of snuffles in rabbits.

Abscesses were recorded on the surface of rabbit lungs by Rai *et al.* (1995). *Staphylococcus aureus* was reported to be the etiology. They also observed bronchopneumonia, haemorrhagic pneumonia and interstitial pneumonia and isolated bacteria like *Bordetella bronchiseptica* (21.2 % cases), *Escherichia coli*(16 % cases), *Streptococcus pneumoniae* (3.4 % cases), *Proteus sp.* (3.4 % cases) and *Salmonella sp* (2.5 % cases) from the respiratory system of rabbits died of pneumonia.

Cilia Associated Respiratory Bacillus was demonstrated in the surface of the bronchial epithelial lining using Warthen Starry staining by Oros *et al.* (1997).

Caniatti *et al.* (1998) observed inflammatory lesions in the trachea and lungs of rabbits and demonstrated Cilia Associated Respiratory bacillus in the lesions.

Pawaiya *et al.* (1998) conducted a study on rabbit mortality and recorded deep congestion to purplish red consolidation with or without fibrinous adhesion in the lungs of rabbits died of pneumonia. Microscopically they observed highly engorged blood vessels with massive haemorrhage in the alveoli and interalveolar tissue and inflammatory reaction including macrophage reaction in the lungs. *Pasteurella sp, Klebsiella sp*, Pneumococci and *E.coli* were isolated from the lungs. .

Sinusitis, bronchitis and pneumonia were reported in rabbits by Berglof *et al.* (2000) and they found colonisation of *Bordetella bronchiseptica* in the respiratory system.

Ultra structural pathology of the nasal and tracheal mucosa of the rabbits experimentally infected with *Pasteurella multocida* serotype D:1 was described by Al-Haddawi *et al.* (2001). They found that the changes on the nasal mucosa were degeneration of the surface epithelium with inflammatory response and that of the trachea were degeneration and sloughing of the surface epithelium.

Klebsiella pneumonia was isolated from samples of lung collected from rabbits by Coletti *et al.* (2001).

Mir *et al.* (2001) described the gross and histopatholgical lesions in the respiratory system of rabbits intra nasally inoculated with *P. multocida* 12:A . They recorded congestion and emphysema in the lungs. Microscopically the trachea revealed engorgement of blood vessels and degeneration and sloughing of the mucosa. Mild hemorrhages, thickening of interalveolar septa, areas of emphysema, bronchitis and desqamation of bronchial epithelium could be seen in the lungs.

2.1.2 Virus

Rai *et al.* (1985) reported pneumoenteritis in rabbits. They observed congestion and consolidation of the lungs along with enteric lesions. Microscopically oedema, compensatory emphysema, congestion, mononuclear cell infiltration, perivascular and peribronchial lymphocytic infiltration, and degeneration and desqamation of bronchial epithelium were seen. Calici like virus was reported to be the etiology.

Sundaram *et al.* (1991) first reported an outbreak of Viral Haemorrhagic Disease of rabbits in India. The clinical signs included nasal discharge and dyspnoea resulting in death.

Electron microscopic investigation of Rabbit Haemorrhagic Disease was conducted by Alexandrov *et al.* (1993) and Calici virus was reported to be the etiology.

Chasey *et al.* (1994) observed that the rabbits died of outbreaks of Viral Haemorrhagic Disease revealed petechial haemorrhages in the tracheal tissue.

Kpodekon and Alogninouwa (1998) reported that in rabbits died of Viral Haemorrhagic Disease, blood was found around the nostrils with extensive haemorrhage in the larynx and trachea along with haemorrhage in other visceral organs including lungs.

Motha and Kittelberger (1998) stated that Rabbit Haemorrhagic Disease is an acute fatal disease of wild and domesticated rabbits caused by highly contagious Calici virus.

Myxoma virus was isolated from 10 % of the rabbits died of pulmonary lesions by Marlier *et al.* (2000). They also demonstrated serological evidence of Myxoma virus infection in 44 % of rabbits.

2.1.3 Mycoplasma

Villa *et al.* (2001) observed red to grey areas of consolidation in the apical and medial lobes of lungs and abundant serous non purulent exudate in the lungs of 45 day old rabbits.They detected *Mycoplasma pulmonis* from the lungs by microbiological and immunocytochemical analysis. Forty five percent of the cases of rabbit pneumonia were positive for Mycoplasma.

2.1.4 Chlamydia

Flatt and Dungworth (1971b) experimentally produced pneumonia in rabbits by inoculation of Chlamydial organisms.

Chlamydial pneumonia in Angora rabbits was reported by Krishna and Gupta (1989)

Villa *et al.* (2001) reported that 7% of the total rabbits with respiratory problems carried *Chlamydia sp* in their lungs.

2.1.5 Fungus

Pulmonary aspergillosis in rabbits was reported by Patton (1973).

Viable *Aspergillus fumigatus* and *Penicillium sp* were detected in the lung tissues of rabbits two to three weeks after experimental inoculation with the fungal spores by Thurston *et al.* (1979).

Matsui *et al.* (1985) reported pulmonary aspergillosis in apparently healthy young rabbits. Gross lesions they observed were white nodules of one to three mille metre size on the surface of all lobes of lungs. Microscopically, they observed the hyphae of the fungus and granulomatous inflammation. *Aspergillus fumigatus, Aspergillus flavus* and *Rhizopus sp* were isolated from the lesion.

Chattopadhyay *et al.* (1994) recorded congestion and focal caeseated nodules in the trachea and multiple caeseated nodules and suppurative foci in

the lungs of rabbits experimentally inoculated with *Aspergillus fumigatus.* Histopathological lesions observed were tracheitis with mycotic granuloma in the trachea and acute inflammation with necrosis and suppuration in the lungs.

2.1.6 Parasites

Krishna and Vaid (1987) recorded patchy areas of emphysema and engorgement of the blood vessels along with presence of oedema fluid in the alveoli in the lungs of rabbits died in an out break of intestinal coccidiosis.

Sato *et al.* (1994) studied the histopathology of the lungs of rabbits experimentally infected with *Dirofilaria immitis* and observed arteritis and periarteritis.

Chandra *et al.* (1999) experimentally produced trypanosomiasis in rabbits with *Trypanosoma evansi* and observed congestion and consolidation in lungs. Most conspicuous histological lesions in the lungs were septal thickening, serous exudation,leucocytic infiltration and emphysema.

Toxocara vitullorum infection in rabbits was induced by Pramanik *et al.* (1994) and recorded that the lungs contained multiple foci of haemorrhage, consolidation and greyish white spots. Histologically granulomatous reaction was seen around the parasitic larva.

Villa *et al* (2001) reported *Toxoplasma gondii* in 4 % of the rabbit carcasses with respiratory problems.

2.1.7 Vitamin deficiency

Sravanthy *et al.* (1996) experimentally induced vitamin A deficiency in rabbits and found squamous metaplasia of the tracheal epithelium . Congestion, focal pneumonia and emphysema in the lungs and mononuclear cell aggregation in the alveoli around bronchioles and blood vessels were the other changes.

2.1. 8 Toxins

Haemmorhage and oedema in the lungs were seen in experimentally induced aflatoxicosis in rabbits by Singh (1997).

Singh *et al.* (1993) recorded lesions in the lungs as mild hyperplasia and desquamation of the bronchiolar epithelium, peribronchial mononuclear cell aggregation; moderate thickening of bronchial wall and alveolar septa along with congestion of the capillaries in rabbits died in an outbreak of aflatoxicosis in Himachal Pradesh.

Titame *et al.* (1996) experimentally fed rabbits with diet containing Molybdenum at 0.3% level and observed haemosiderin laden phagocytes in the perivascular and peribronchiolar regions.

Toxicity with Ochratoxin-A in rabbits was studied by Mir *et al.* (1999) and they found degenerative changes, intense congestion, extensive interstitial and alveolar haemorrhage and compensatory emphysema in the lungs.

2.1.9 Aspiration

Radostitis *et al.* (1995) reported that aspiration of foreign materials into the respiratory tract occur due to faulty drenching and also due to regurgitation of stomach contents and leads to serious disease conditions.

2.2. PATHOLOGY

2.2.1 Vascular changes

Uzal *et al.* (1991) noticed severe endothelial changes and intra vascular deposition of collagen in the septal capillaries of clinically asymptomatic rabbits.

A variety of circulatory disturbances such as congestion, haemorrhage, embolism, thrombus, infarction, and hypertension in the pulmonary artery were recorded by Dungworth (1993).

2.2.1.1 Congestion

Krishna and Vaid (1987) observed congestion in the lungs of rabbits died in an outbreak of intestinal coccidiosis. Engorgement of the capillaries was seen in the lungs.

Congestion of the capillaries in the trachea and lungs of rabbits died due to pneumonia caused by *Pasteurella sp* was recorded by Nair *et al.* (1987). They also observed congestion in the lymphnodes.

Pulmonary congestion was reported in colisepticemia in rabbits by Shah *et al.* (1989).

Mir *et al.* (1999) recorded intense congestion in the lungs of rabbits died due to Ochratoxin-A.

2.2.1.2 Thrombus

D' suze *et al.* (1999) reported abundant microthrombi in the rabbit lungs exposed to scorpion venom and stated that the clotting alterations are fundamental to produce pulmonary oedema.

2.2.1.3 Haemorrhage

Haemorrhagic tracheitis was reported to be the pathognomonic lesion of pneumonia due to *Pasteurella sp* in rabbits (Nair *et al.*, 1987). Haemorrhage was also observed in the alveoli.

Rai *et al.* (1995) recorded brick red coloured lungs in rabbits died due to hemorrhagic pneumonia. Microscopically the lungs revealed diffuse accumulation of erythrocytes in the alveoli, interstitial tissues and in a few bronchioles.

Kpodekon and Alogninouwa (1998) reported extensive haemorrhage in the trachea and lungs of rabbits died due to Viral Haemorrhgic Disease.

Pramanik *et al.* (1994) observed multiple foci of haemorrhage in the lungs of the rabbits died due to experimentally induced *Toxocara vitullorum* infection.

2.2.1.4 Oedema

Rai *et al.* (1985) observed oedema in the lungs of rabbits died due to pneumoenteritis. Lungs on microscopical examination revealed uniform pink coloured fluid in the alveoli.

The presence of oedema fluid in the lungs of rabbits affected with colisepticaemia was recorded by Shah *et al.* (1989).

Villa *et al.* (2001) noticed abundant non purulent exudate in the lungs of rabbits died of Mycoplasmal pneumonia.

2.2.2 Degeneration

Rai *et al.* (1995) observed degeneration of the bronchial epithelium in bronchopneumonia in rabbits

Degeneration of the nasal and tracheal epithelium in rabbits was reported by Al-Haddawi *et al.* (2001).

Mir *et al.* (2001) reported degeneration and sloughing of the tracheal mucosa in rabbits experimentally inoculated with *Pasteurella sp* organisms.

2.2.3 Inflammation

Pneumonia is the inflammation of gas exchanging parts of the lungs including respiratory bronchiole and alveoli (Kirk *et al.*, 1977).

Flatt and Dungworth (1971a) conducted postmortem examination of 3967 pairs of lungs from 8 to 10 week old apparently healthy rabbits slaughtered for human consumption and recorded that pneumonia was present in 20 % of cases.

Nair *et al.* (1987) conducted a study on 106 rabbit carcasses and found that 78.3 % of the total mortality was due to pneumonia.

2.2.3.1 Haemorrhagic pneumonia

Rai *et al.* (1995) reported haemorrhagic pneumonia in rabbits and observed that the affected lungs were brick red in colour and consolidated. Microscopically they noticed the diffuse accumulation of erythrocytes in the alveoli, interstitial tissues and in a few bronchioles.

Hemorrhagic pneumonia in rabbits was also reported by Sharma *et al.* (1995)

Marlier *et al.* (2000) conducted a postmortem study on rabbit carcasses and recorded haemorrhagic pneumonia in 38 % of the cases and fibrino-haemorrhagic pneumonia with fibrinous pleuritis in 27 % of the cases.

2.2.3.2 Broncho pneumonia

Rai *et al.* (1995) reported that bronchopneumonia was the most frequent type of pneumonia in rabbits. The gross lesions were congestion, oedema and consolidation in the lungs with variable amount of frothy exudate in the bronchi. Histopathologically they observed degeneration and desquamation of the bronchial epithelium forming exudate in the lumen admixed with inflammatory cells. Vascular congestion and infiltration of inflammatory cells were seen throughout the lung parenchyma.

Bronchopneumonia was observed in 47 % of the rabbit carcasses by Sharma *et al.* (1995).

2.2.3.3 Suppurative pneumonia

Rai *et al.* (1995) recorded suppurative pneumonia in rabbits. They observed that the lungs were consolidated and grey in colour. On incision of the lung purulent exudate was seen. Large abscesses were also recorded in some

cases. Microscopically, they observed thick acidophilic exudate in the alveoli with inflammatory cells.

Sharma *et al.* (1995) conducted postmortem study in 118 rabbits and observed suppurative pneumonia in 22 % of the cases.

Marlier *et al.* (2000) recorded acute suppurative pneumonia in 35 % of the rabbit carcasses examined.

2.2.3.4 Interstitial pneumonia

Ultrasound study of spontaneous chronic lung lesions in asymptomatic rabbits was conducted by Uzal *et al.* (1991) and reported focal chronic interstitial pneumonia .

Rai *et al.* (1995) reported interstitial pneumonia in rabbits and the lungs on microscopical examination revealed, infiltration and hyperplasia of mononuclear cells in the inter lobular septa, peribronchial and peribronchiolar regions and around blood vessels.

2.2.5 Neoplasms

Pulmonary adenomatosis in the laboratory animals was reported by Stookey and Moe (1978). Metaplastic transformation, hypertrophy and hyperplasia of the lining cells of the alveoli into cuboidal or columnar cells were seen.

Dungworth (1993) reported that bronchial papilloma, bronchial gland adenoma, adenocarcinoma, bronchioalveolar adenoma and carcinoma were the primary epithelial tumuors of lungs.

Sharma *et al.* (1995) reported lymphosarcoma in rabbits with lesions in the lungs and lymphnodes.

Materials and Methods

3. MATERIALS AND METHODS

The present study was conducted at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy to investigate the presence of various disorders in the respiratory system of rabbits.

3.1 SAMPLE COLLECTION

The respiratory system especially the trachea, lungs and bronchial lymphnodes of the rabbit carcasses brought for postmortem examination at the Centre of Excellence in Pathology were subjected to detailed gross and histopathological examination. Fifty rabbit carcasses presented for autopsy, during the period 2002 – 2003 were examined.

3.2 GROSS EXAMINATION

The nostrils and the nasal cavity of the rabbit carcasses were examined carefully for the presence of any discharge or lesions. The thoracic cavity was examined for the presence of any exudates. The pleura was observed for any lesions especially congestion or inflammatory change. The trachea, lungs and the bronchial lymphnodes were subjected to detailed gross examination. The trachea was dissected along the whole length. The course of the bronchioles were traced to examine for the presence of blood or froth.

Smears were prepared from the blood and the exudates present in the trachea. Impression smears were also prepared from the trachea and the lungs. The smears were stained with Wright's stain and examined for the presence of bacterial organisms.

3.3 HISTOPATHOLOGY

Representative samples of tissues from the trachea, lungs and associated lymphnodes were collected and preserved in 10% Neutral Buffered Formalin.

The tissues were processed by routine paraffin embedding techniques (Sheehan and Hrapchak, 1980). Sections were cut at 4 micron thickness and stained with routine Haematoxylin and Eosin stain (Bancroft and Cook, 1995) for histopathological studies. Special staining was performed with Mallory's Phospho Tungstic Acid-Haematoxylin. (Luna, 1968) whenever required. The stained sections were subjected to detailed examination under the light microscope and the lesions were classified.

3.4 MICROBIOLOGICAL STUDIES

The bacterial cultures were made by streaking aseptically harvested deep tracheal and pulmonary tissues from the fresh rabbit carcasses on bovine blood agar plates and Trypticase soya Agar plates and the plates were incubated both aerobically and anaerobically at 37°C for 48hours. The colonies were identified by cultural, morphological and biochemical characters (Barrow and Filtham, 1993). Biochemical tests for the identification of the Gram negative bacteria were performed using the biochemical kit, KB-002 (HI-MEDIA). Identification of the Gram positive bacteria was performed by following the tests described by Barrow and Filtham (1993).

3.5 PATHOGENICITY STUDY

Four-week old Swiss Albino mice were used for the pathogenicity study. The pathogenicity trial was conducted by randomly selecting two samples of eighteen hour old broth culture of the *Escherichia coli* isolated from the trachea and lungs of the rabbits. Intraperitoneal injection of 0.2 ml of the inoculum containing 100 cfu in 0.1 ml of the medium was given to the mice and observed for any lesions or death occurred during forty eight hours.

Results

4. RESULTS

4.1 GROSS PATHOLOGY

Fifty rabbit carcasses were studied. On gross examination of the respiratory tract there was presence of foamy exudate in the nostrils of two rabbit carcasses. Blood was found in the nasal cavity in one case. Moderate amount of sero-sanguinous fluid was present in the thoracic cavity in eight cases along with lesions like congestion and haemorrhage in the respiratory system and other visceral organs. Lesion in the lymphnode was present in two cases.

Tracheal mucosa was found to be hyperaemic in some cases and highly congested in many of the cases. There were focal areas of haemorrhage in the submucosa of the trachea (Fig.1). Copious amount of blood was present in the tracheal lumen in five cases. In many of the carcasses tracheal lumen was filled with frothy exudate and on tracing the airways the bronchi and the bronchioles were also found to be filled with froth. In some of the cases the froth in the tracheal lumen was blood tinged.

Examination of the smears prepared from the blood and the exudate in the trachea revealed the presence of bipolar bacteria indicative of *Pasteurella sp.* organisms in five cases.

The pleural membrane covering the lungs was congested in some cases. Sub-pleural haemorrhages evidenced by patchy reddish areas were seen in two cases (Fig. 2). The lungs appeared to be congested in most of the cases. Congested lobes appeared dark red in colour. Congestion was uniformly present in all the lobes in thirteen cases. Apical and cardiac lobes appeared to be congested in twelve cases. The congestion was more prominent in the diaphragmatic lobes in ten cases. Mild congestion was seen in the bronchial lymphnodes in two cases.

Gross examination of the lungs revealed oedema in many cases. Areas of oedema appeared wet and heavy and fluid oozed out from the cut surfaces on incision. There was frothy exudate in the lumen of the trachea and bronchi.

On palpation of the various lobes of the lungs, areas of consolidation of varying degree could be detected in twelve cases. The consolidated lesions were most often located in the cranioventral regions of the lungs. Stages of red and grey hepatization were seen in different lobes of the lungs. Lobes showing red hepatization were brownish red in colour and hard in consistency. Areas of grey hepatization were pale red in colour. The lesions varied in size from 1 to 2 cm to involvement of the entire lobes. The affected parenchyma was firm and fleshy and the major airways contained frothy exudate. A distinct line of demarcation separated consolidated areas from the adjacent parenchyma, which was frequently mildly emphysematous.

Purple coloured depressed areas of collapse could be seen in the lungs in five cases. These lesions were seen distributed over the diaphragmatic lobes of the lungs. On incision the areas of collapse were seen extending from the pleural surface deep into the parenchyma.

Emphysema was seen in ten cases. Emphysematous lungs appeared to be puffed up with air and inflated. In one case the lung was highly enlarged and had focal emphysematous area and areas with froth fluid. The trachea and the bronchioles were also completely filled with frothy exudate (Fig. 3).

A case of tumour of lung was seen in one case. Lungs were consolidated and there were multiple nodules in the parenchyma of both the lungs (Fig. 4). On incision, a large number of greyish white nodules varying in size from five to twenty mille metres. The neoplastic tissue was diffusely distributed in the parenchyma of all the lobes. Metastasis of the lesion was seen in the mesentery and the ovary.

4.2 HISTOPATOLOGY

4.2.1 Vascular changes

4.2.1.1 Congestion

Congestion of the capillaries in the tracheal wall was noticed in 62 per cent of the cases. The capillaries were found to be engorged with erythrocytes (Fig.5).

Vessels in the sub pleural area were congested in four per cent of the cases (Fig. 6).

Pulmonary congestion was noticed in 70 per cent of the cases. Pulmonary artery, arterioles, pulmonary vein, peribronchial capillaries and alveolar capillaries were found to be filled with blood (Fig. 7).

Mild congestion was noticed in the bronchial lymphnodes in five per cent of the cases (Fig. 8).

4.2.1.2 Haemorrhage

Haemorrhagic tracheitis was noticed in 12 per cent of the cases. The extravasated erythrocytes were seen scattered in the tracheal mucosa, submucosal area and in the lumen of the trachea. Polymorphs and mononuclear cells were seen distributed throughout the submucosa.

Haemorrhage in the lung parenchyma was seen in 22 per cent of the cases. Free erythrocytes were present in the lumen of the alveoli as well as in the interstitial space (Fig. 9).

4.2.1.3 Thrombosis

Arterial thrombi (Fig. 10) were present in five per cent of the cases.

Microthrombi were present in the pulmonary capillaries in 20 per cent of the cases.

4.2.1.4 Embolism

Bacterial emboli were seen in the tracheal wall in two per cent of the cases. The capillaries on the tracheal wall were found to be filled with neutrophils and bacteria (Fig . 11).

4.2.1.5 Oedema

Pulmonary oedema was seen in 62 per cent of the lungs examined. The alveoli and interstitial space were filled with uniform pink coloured fluid (Fig. 12). The capillaries in the surrounding area were engorged. Rupture of the wall of the alveoli and fusion with the neighbouring alveoli indicating compensatory emphysema was also found along with oedema in 13 per cent of the cases.

4.2.1.6 Cystic dilatation of the capillary

Cystic dilatation of the capillaries was seen in the tracheal wall in 13 per cent of the cases. The lumen of the capillaries on the tracheal wall was dilated.

4.2.1.7 Blood vessel wall thickening

Thickening of the wall of the pulmonary arteries were noted in ten per cent of the cases (Fig. 13). Non inflammatory thickening of the tunica media of the blood vessel wall could be seen on special staining with Mallory's Phospho Tungstic Acid stain.

4.2.2 Emphysema

Emphysema of the alveoli was seen in 19.5 per cent of the lungs examined. .Abnormal distention of the alveoli with air accompanied by

destruction of the alveolar walls and coalescence with the neighbouring ones and formation of bullae could be seen (Fig . 14).

4.2.3 Pulmonary collapse

Focal areas of collapse of the alveoli were noticed in 13 per cent of the cases along with other lesions like congestion, oedema and emphysema. The alveolar walls were found to be in close apposition in these areas and the alveolar lumen were greatly reduced in size (Fig. 14).

4.2.4 Inflammation

Inflammatory changes were seen in the trachea in 20 per cent of the cases. Infiltration with polymorphonuclear leucocytes, macrophages and plasma cells were seen in the tracheal mucosa and submucosa along with focal desquamation of the tracheal mucosa (Fig. 15).

Inflammatory reaction in the bronchi characterised by presence of exudate in the lumen of the bronchi with varying number of neutrophils and mononuclear cells. Similar reactions were seen in the alveolar lumen adjacent to the bronchioles also in 13 per cent of the cases (Fig.16). Pribronchial lymphocytic infiltration was present in 19 per cent of the cases.

Inflammatory changes in the lung parenchyma characterised by infiltration with neutrophils, lymphocytes and macrophages were seen in 30 per cent of the cases (Fig. 17). Perivascular accumulation of lymphocytes was present in two per cent of the cases. Infiltration of mononuclear inflammatory cells and subsequent moderate thickening of the alveolar septum were seen in four per cent of the cases.

In all these cases inflammatory changes were accompanied by other changes like congestion and oedema. In one case the stage of resolution of pneumonia characterised by presence of macrophages and regeneration of alveolar epithelium was seen on microscopical examination (Fig. 18).

4.2.5 Collapse of the bronchiole

Collapse of the bronchioles were noticed in 30 per cent of the cases. The epithelial lining of the bronchioles were found to be collapsed.

4.2.6 Proliferation of bronchiolar epithelium

Proliferation of the epithelium lining the bronchial lumen was recorded in ten per cent of the cases along with the presence of exudate in the lumen in some cases. Multifocal adenomatous proliferation of the bronchiolar epithelium was present in one case. The lining epithelial cells were thrown into small papillary projections into the lumen (Fig 19).

4.2.7 Degeneration of Respiratory Epithelium

Focal degeneration and desquamation of the epithelium of the trachea was seen in 17 per cent of the cases. There was infiltration with neutrophils and mononuclear cells in the submucosal area (Fig .20).

Degeneration and desquamation of the epithelium of the bronchioles were seen in 25 per cent of the lungs (Fig. 21).

4.2.9 Necrosis

A focal necrotic mass was observed in the sub-pleural region of lungs in two per cent of the cases (Fig. 22).

4. 2 10 Alveolar carcinoma

One or more layer of cuboidal, columnar, polygonal or bizzare cells lined the pulmonary airsacs. Ample eosinophilic cytoplasm and basally situated nuclei were seen. Some of the proliferating alveolar cells appeared as processes which projected into the alveolar lumen.

In certain alveoli spherical groups of neoplastic cells were found free within the lumen. Areas of cellular pleomorphism, occasionally were seen. Spindle cells and multinucleated giant cells were observed. Mitotic figures were also seen but were a few in number. In certain areas the alveolar walls were found to be well preserved and were slightly or markedly thickened to support the neoplastic cells. Areas of emphysema, oedema, congestion and haemorrhage could also be observed in the adjacent areasclose to the neoplasm (Fig. 23).

4.2.8 Lymphocyte depletion

Depletion of the lymphocytes and loose appearance of the parenchyma was present in two per cent of the lymphnodes (Fig. 8).

4.3 MICROBIOLOGICAL STUDIES

A total of 20 bacterial isolates were obtained from animals which had inflammatory changes in the trachea and lungs. The bacterial isolates obtained were *Escherichia coli* (16), *Staphylococcus sp.* (3) *Proteus sp.* (1). *Pasteurella sp.* could not be isolated from cases of haemorrhagic tracheitis and pneumonia.

The mice used for the biological inoculation of the eighteen hour old broth culture of the *Escherichia coli* isolated from the rabbit died 29 hours post inoculation. Autopsy of the carcasses of the mice revealed sero-sanguinous fluid in the thoracic cavity, pulmonary congestion, hepatic congestion and catarrhal enteritis. *Escherichia coli* was isolated from the lungs of the mice (Fig.24), proving that the *E.coli* isolated from the rabbit is pathogenic.

Fig. 1. **Submucosal haemorrhage in the tracheal wall**

Fig. 2. **Pulmonary congestion and sub pleural haemorrhage**

Fig. 3. **Pulmonary oedema and emphysema**

Fig. 4. **Tumour in the lung**

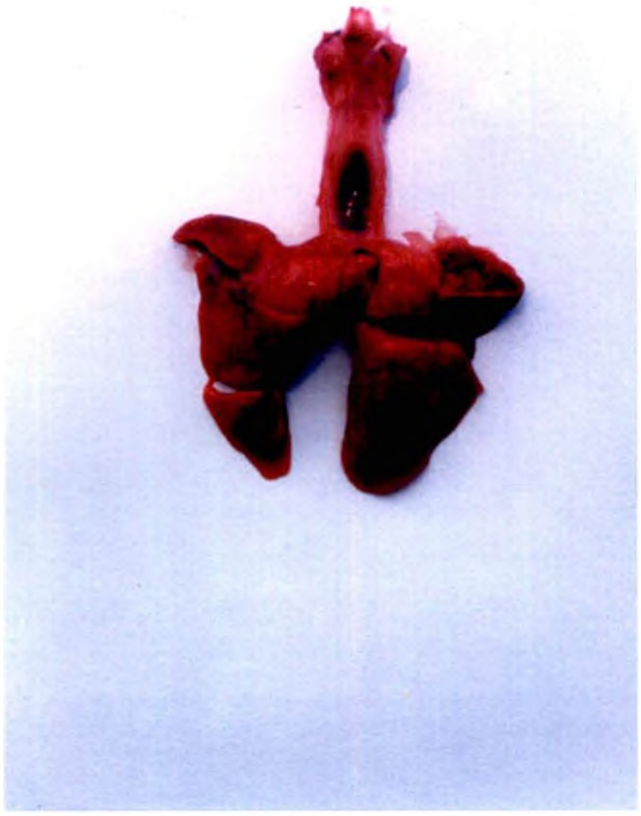


Fig - 1

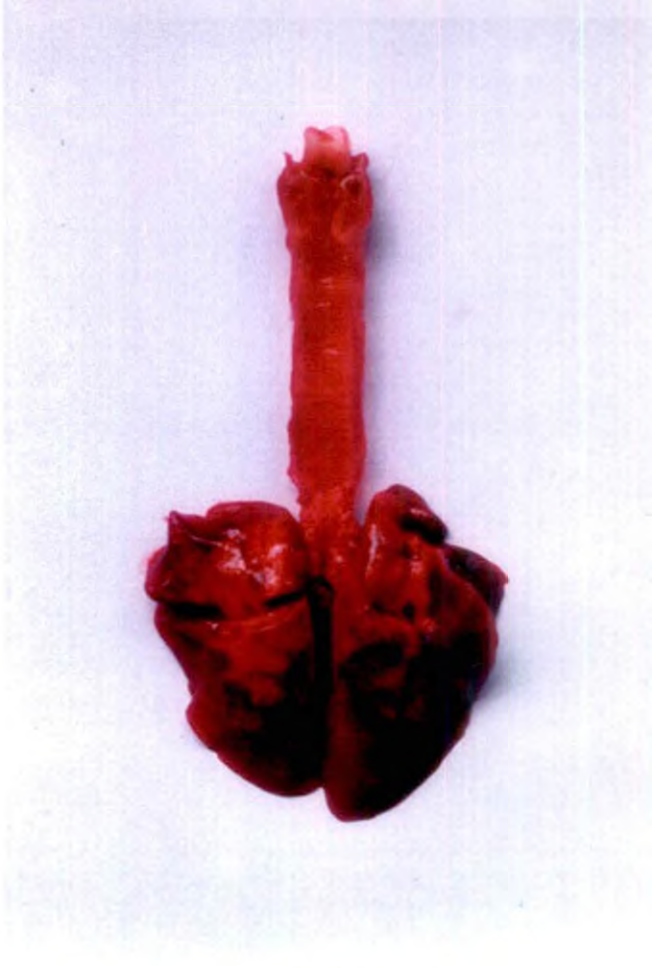
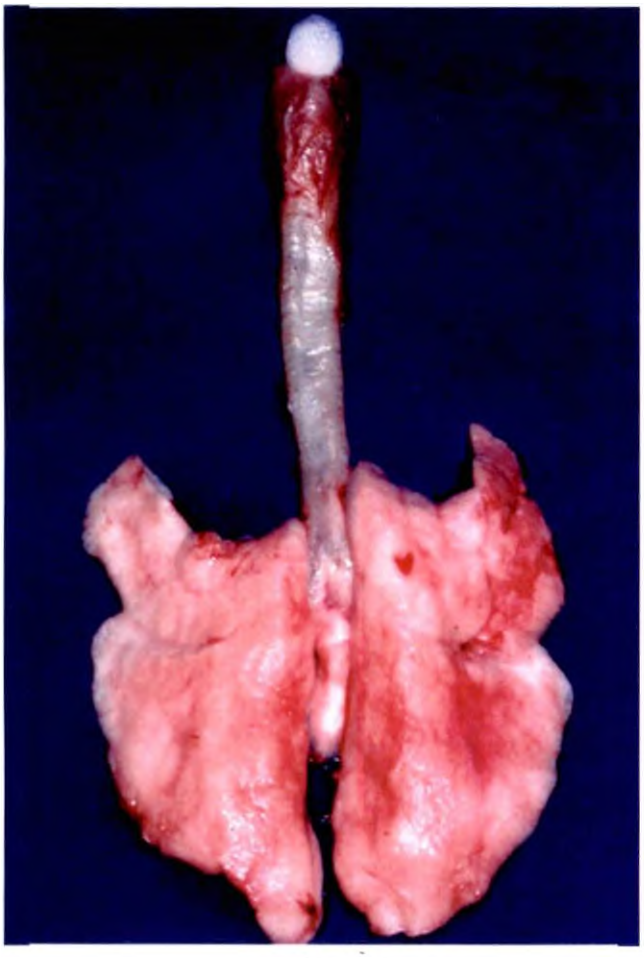


Fig - 2



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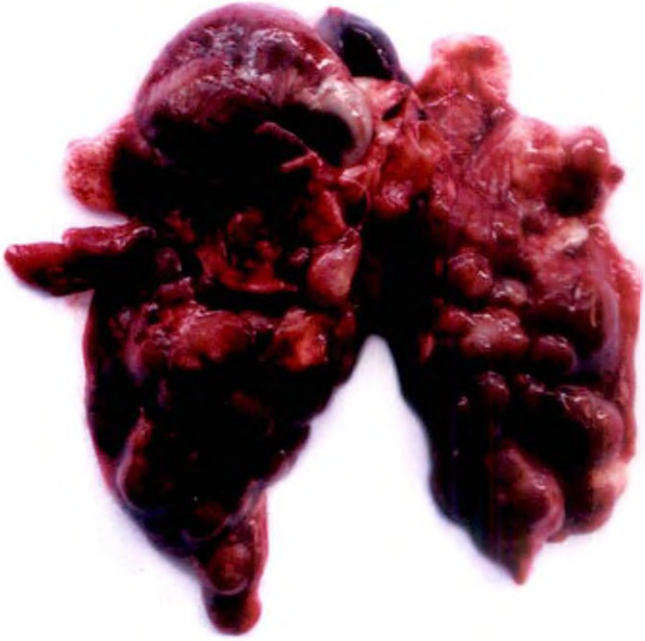


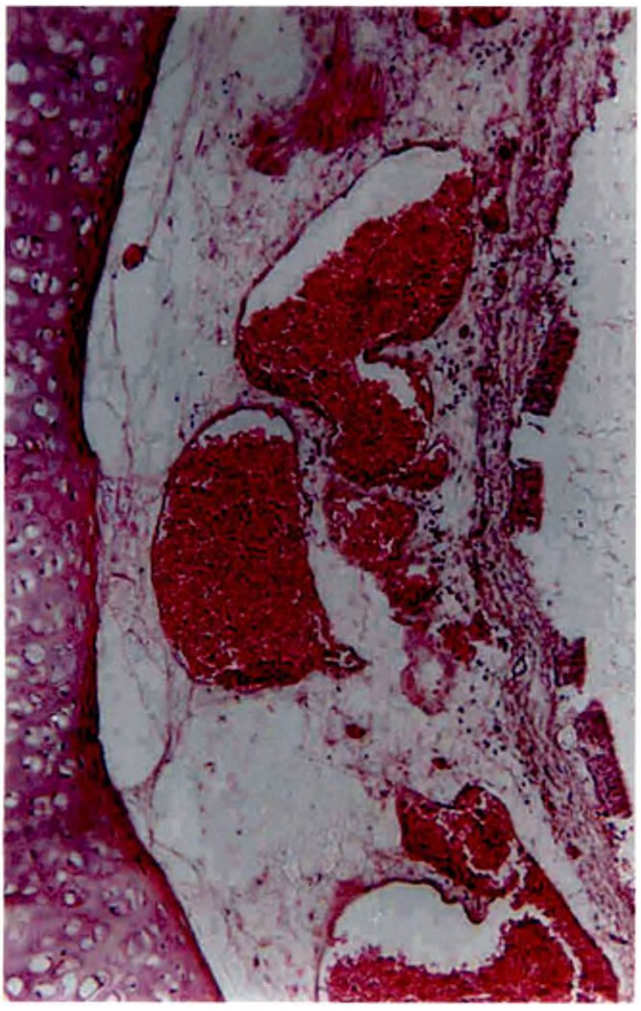
Fig - 4

Fig. 5. **Congestion of tracheal capillary. H&E x 100**

Fig. 6. **Sub pleural congestion. H&E x 100**

Fig. 7. **Pulmonary congestion. H&E x 160**

Fig. 8. **Bronchial lymph node: congestion and lymphocytic depletion. H&E x 100**



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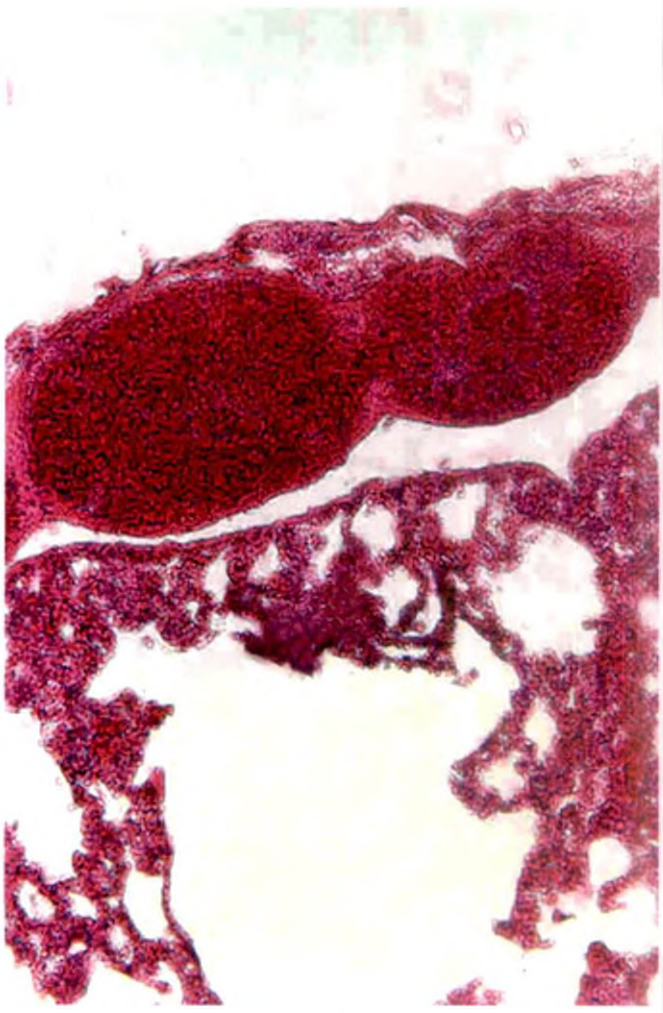

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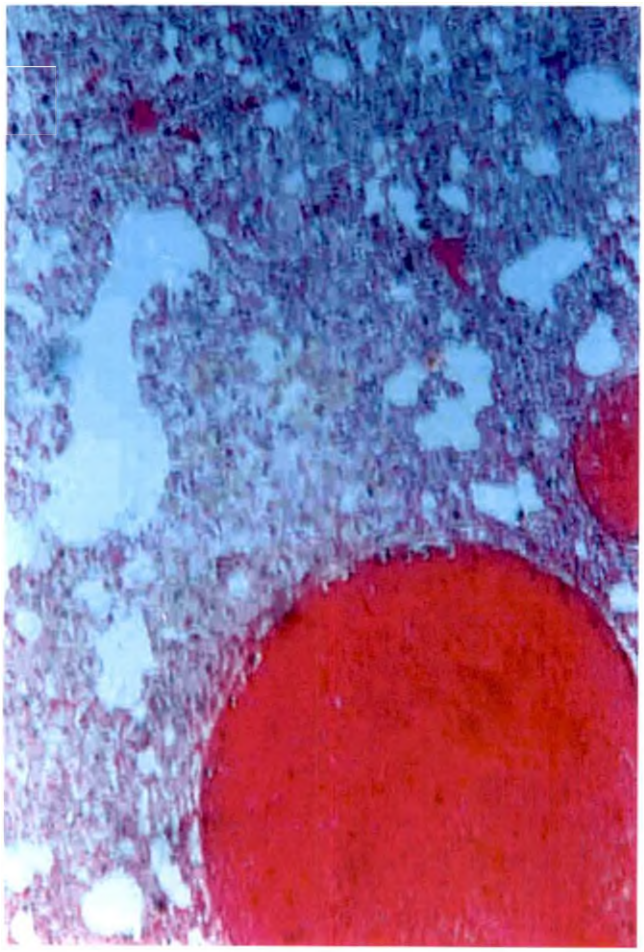

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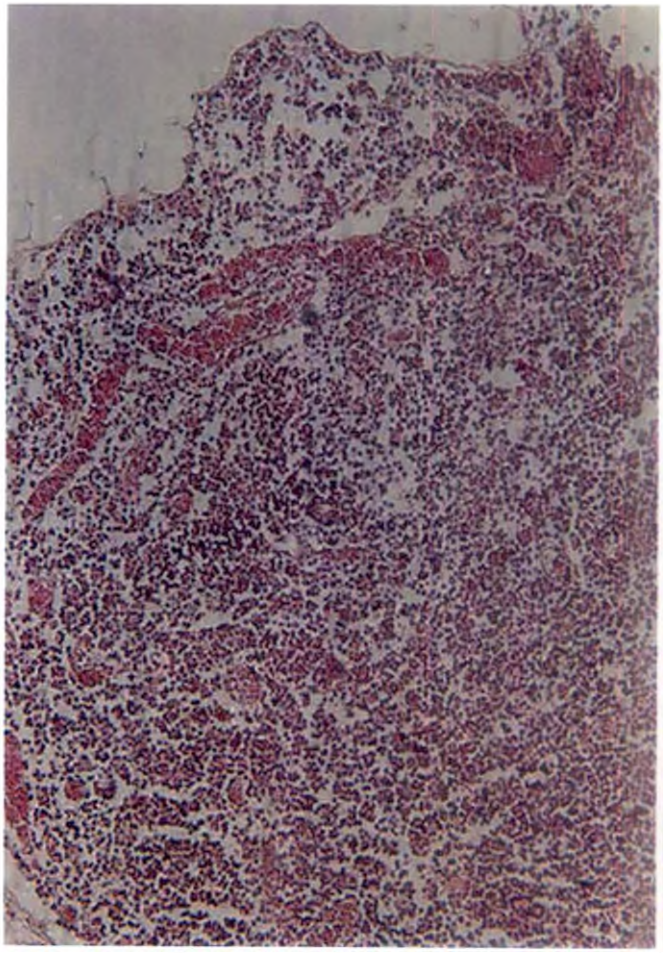


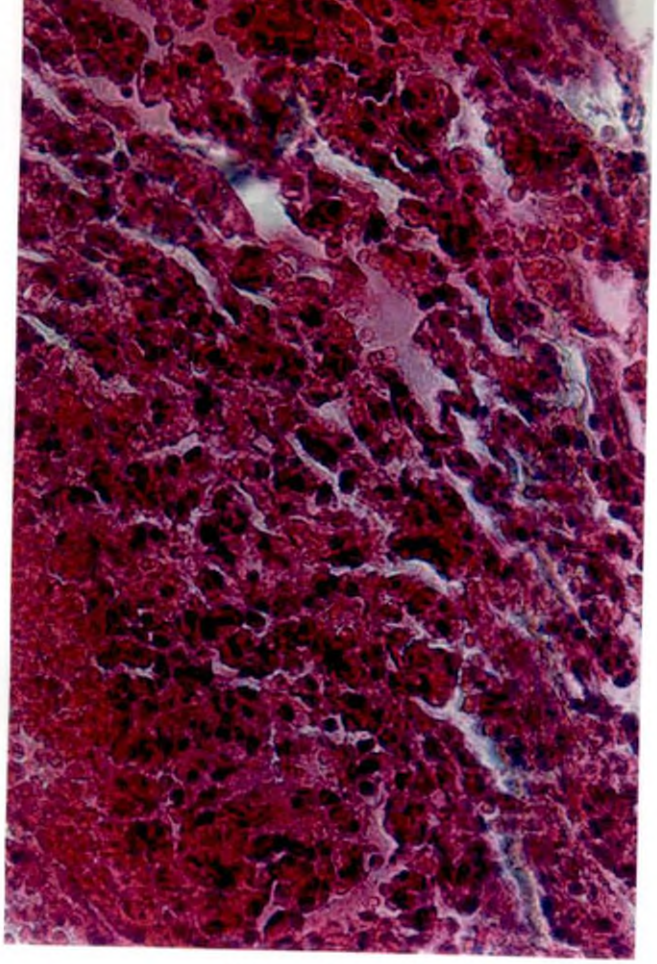
Fig - 8

Fig. 9. **Haemorrhage in the lung parenchyma. H&E x 400**

Fig. 10. **Lung: thrombus. H&E x 100**

Fig. 11. **Bacterial emboli in the tracheal capillary. H&E x 100**

Fig. 12. **Pulmonary oedema. H&E x 400**



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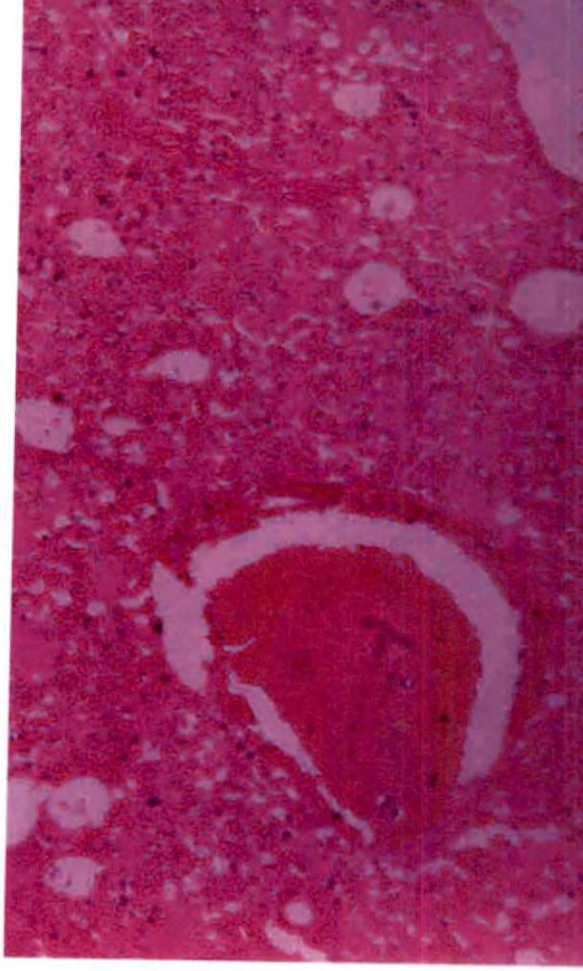

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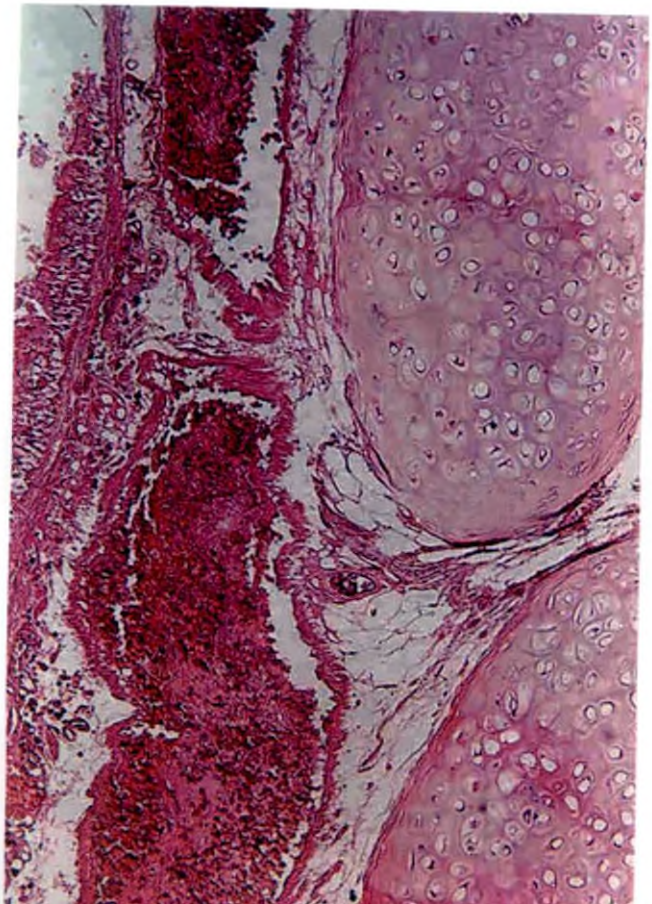


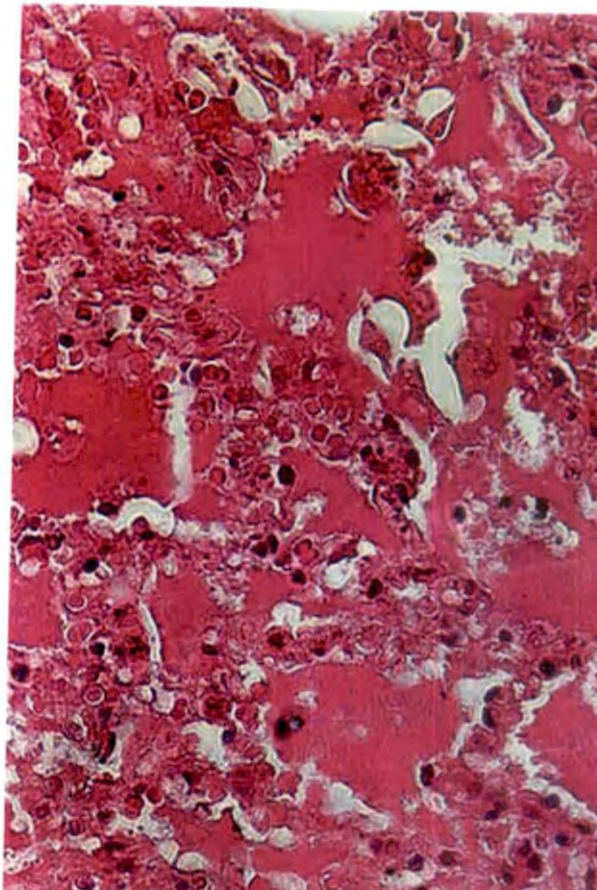
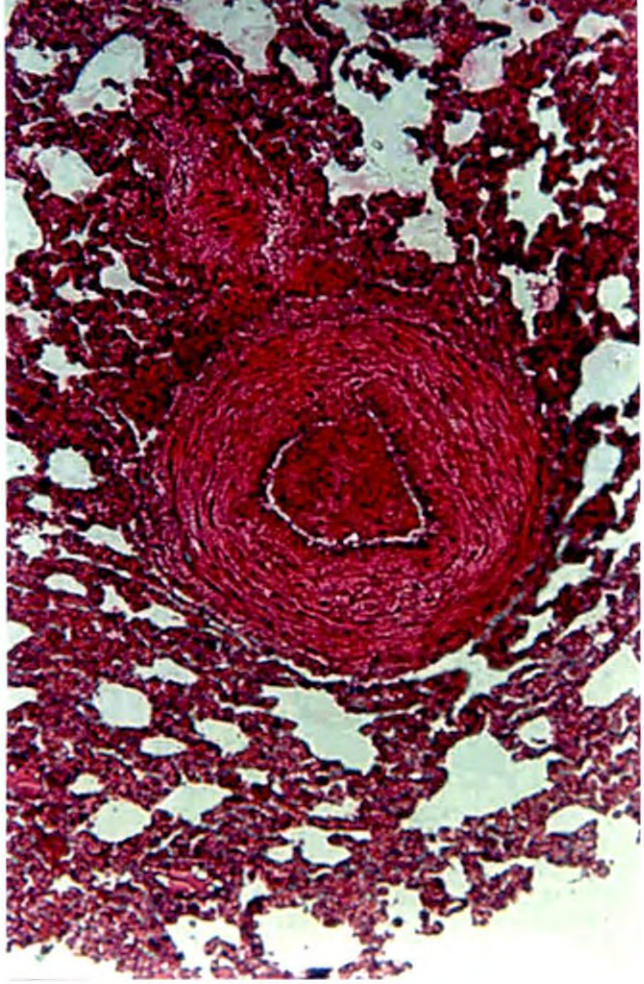


Fig. 13. **Thickening of the wall of the pulmonary artery. H&E x 100**

Fig. 14. **Focal areas of collapse of the alveoli and emphysema. H&E x 100**

Fig. 15. **Inflammatory cells in the wall of the trachea. H&E x 1000**

Fig. 16. **Presence of inflammatory cells in the bronchial lumen. H&E x 400**



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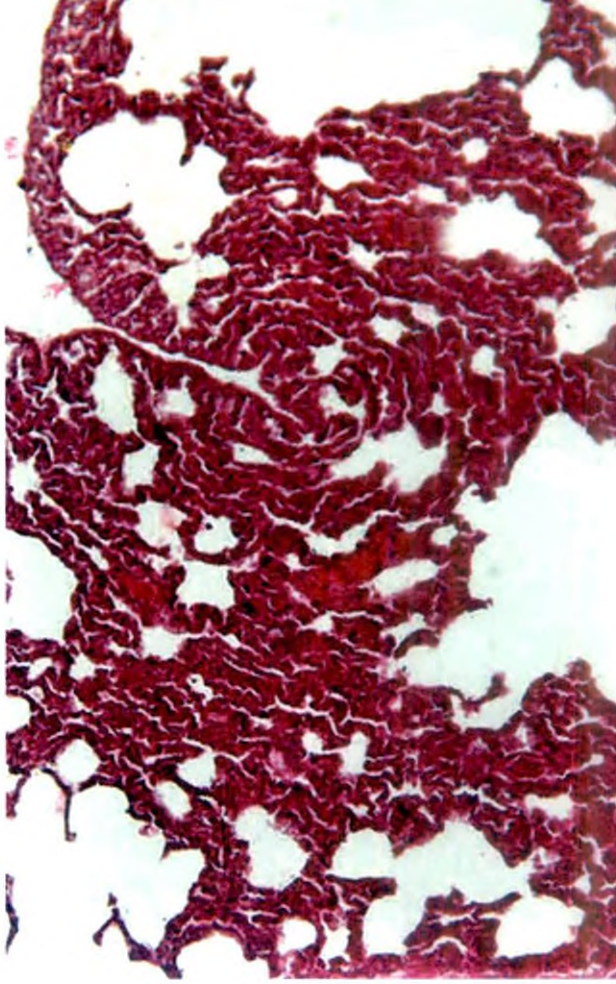

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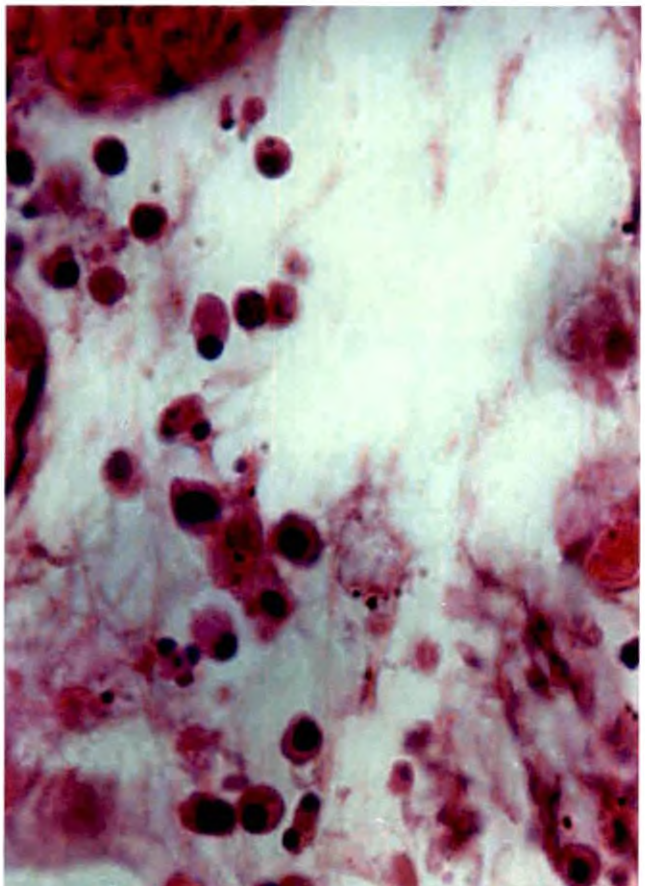


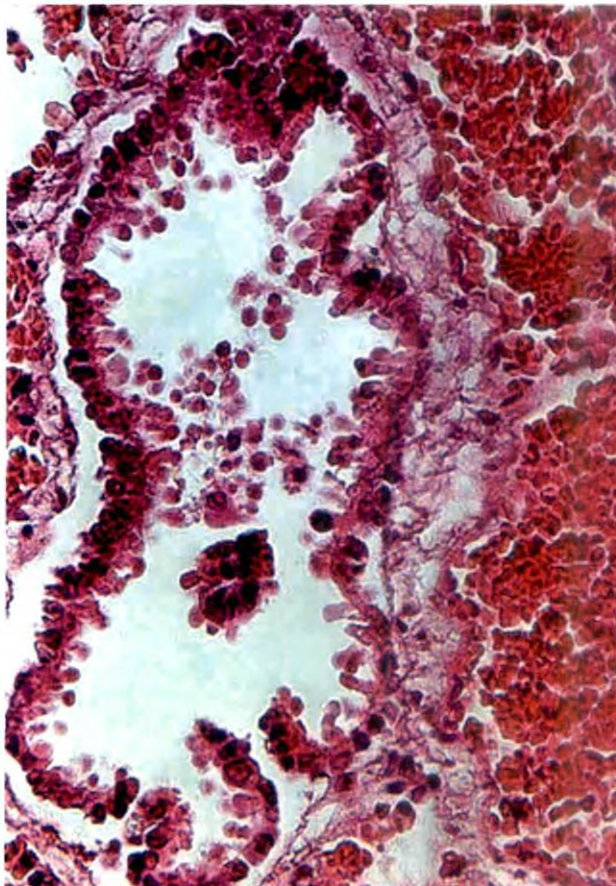
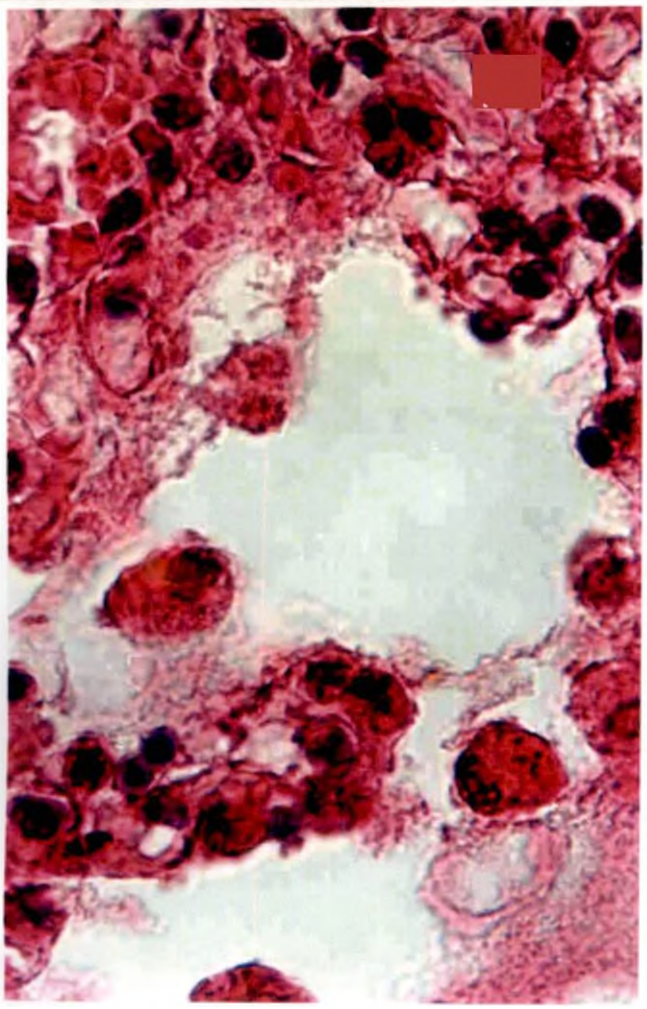


Fig. 17. **Inflammatory cells in the lung. H&E x 1000**

Fig. 18. **Stage of resolution of pneumonia. H&E x 400**

Fig. 19. **Adenomatous proliferation of bronchiolar epithelium. H&E x 100**

Fig. 20. **Desquamation of the tracheal epithelium and presence of inflammatory cells in the tracheal wall. H&E x 400**



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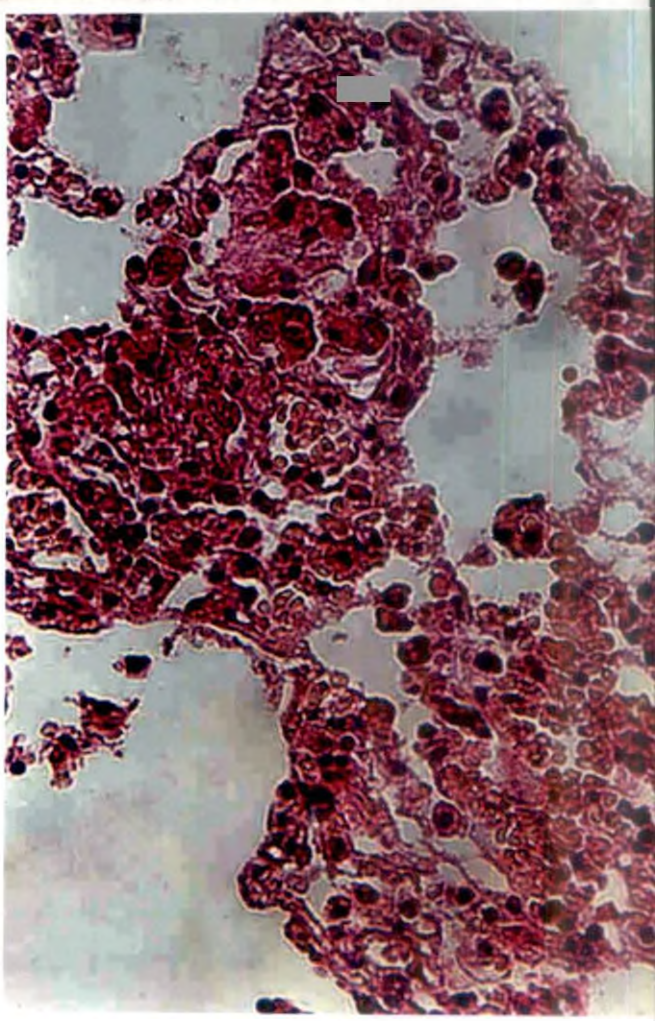

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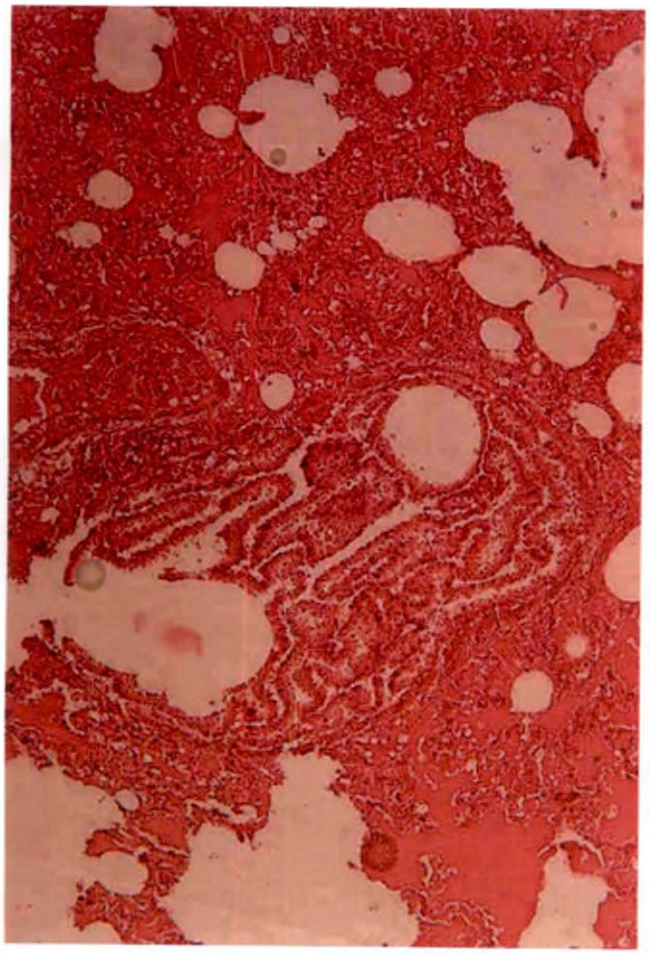

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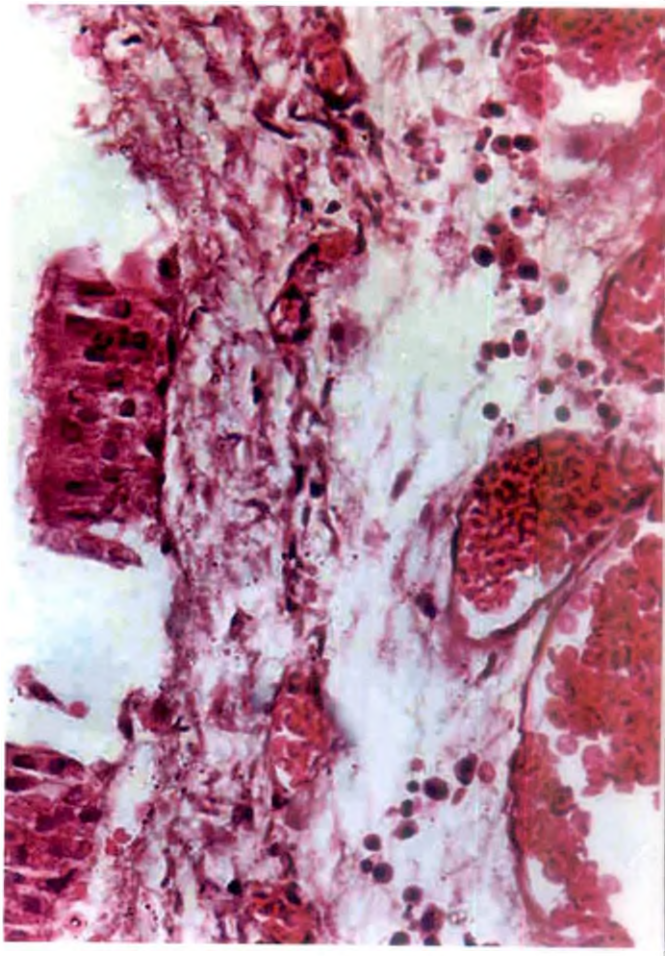


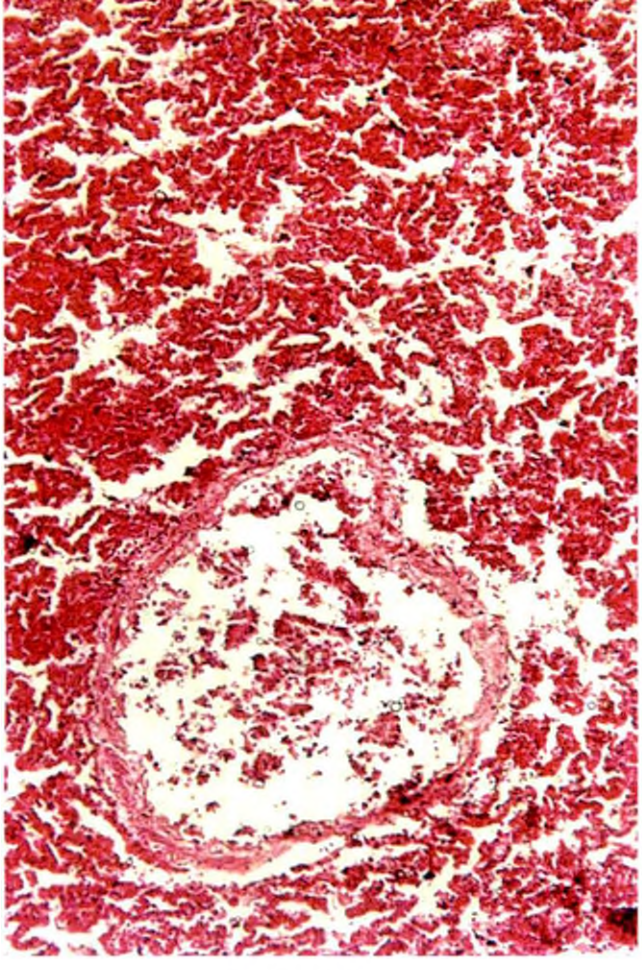
Fig - 20

Fig. 21. **Degeneration and desquamation of the bronchiolar epithelium. H&E x 100**

Fig. 22. **Necrotic mass in the sub pleura. H&E x 1000**

Fig. 23. **Alveolar carcinoma. H&E x 63**

Fig. 24. ***Escherichia coli*** **isolates on Mac Conkey's Agar**



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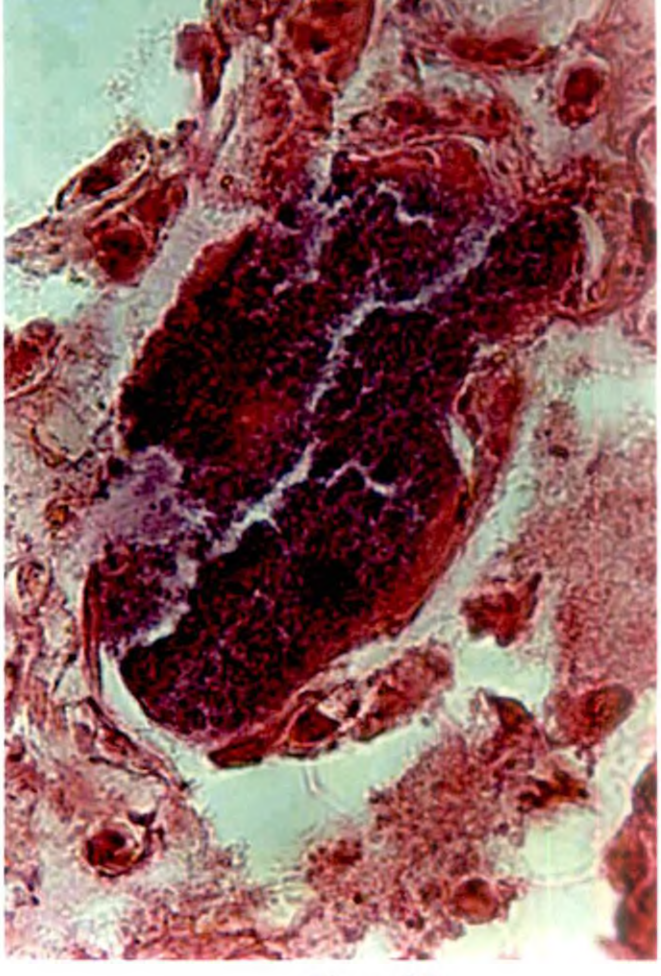

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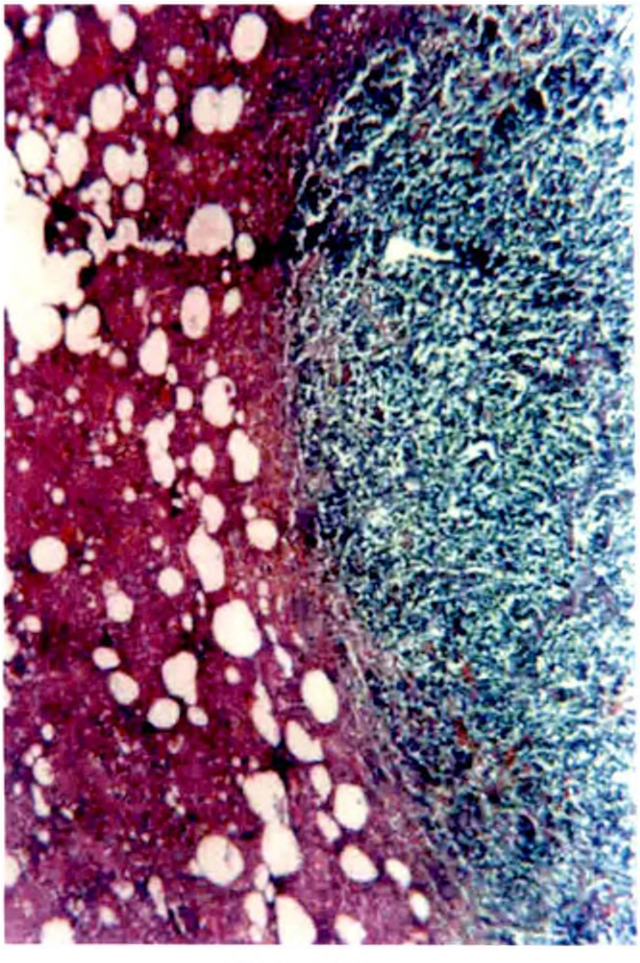

Fig - 23

Fig - 24

Discussion

5. DISCUSSION

The present study was undertaken to investigate the pathology of the respiratory system in rabbits. Samples of the trachea, lungs and bronchial lymphnodes collected from the necropsy cases were studied in detail and the various respiratory disorders were categorised based on the gross and histopathological features. Isolation of the bacteria was attempted from the trachea and lungs of fresh carcasses and the pathogenecity of the isolated bacteria was tested by biological inoculation into Swiss Albino mice.

Various gross lesions were observed on autopsy and one such lesion was foamy exudate in the nostrils. On tracing the course of the respiratory tract, the trachea, bronchi and the bronchioles were also found to be filled with foamy exudate indicative of pulmonary oedema. This is in agreement with the findings of Dungworth (1993). He reported that foam was discharged from the nostrils and foam mixed with fluid would be often present in the trachea and intra pulmonary airways in severe cases of pulmonary oedema which was a frequent complication of many diseases.

Blood was seen in the nasal cavity in one case. Presence of blood in the nasal cavity could occur due to accidental rupture of the capillaries in the nasal cavity and also in the case of systemic infections due to bacteria producing endotoxins which damage the endothelium of the blood vessels resulting in haemorrhage. Viral infections also could lead to haemorrhage in the nostrils in rabbits. Kpodekon and Alogninouwa (1998) reported that blood was found in the nostrils with extensive haemorrhage in the larynx and trachea in the rabbits died of Viral Haemorrhagic Disease. But isolation of virus was not attempted in the present study.

Moderate amount of sero-sanguinous fluid was present in the thoracic cavity in eight cases. The carcass revealed pulmonary congestion, hepatic congestion and catarrhal enteritis indicating septicaemia. Sastry (2001) reported

that presence of fluid in the thoracic cavity can occur in the case of systemic infections due to increased permeability of the capillary endothelium injured by anoxia.

Gross examination of the trachea revealed hyperaemia and congestion of the tracheal mucosa in many of the cases. It could be due to irritants, systemic infections or inflammation. Boyd (1970) reported that in the case of acute bronchitis mucous membrane of the trachea and the large bronchi was red, swollen and covered with tenacious exudate.

On histopathological examination of the trachea, 62 percent cases revealed engorged capillaries. Similar findings were reported by Nair *et al.* (1987) and Mir *et al.* (2001) in the tracheal mucosa of rabbits died of pneumonia caused by *Pasteurella multocida.* Mild congestion in the trachea was recorded in experimental hypovitaminosis- A in rabbits by Sravanty *et al.* (1996).

Copious amount of blood was found in the tracheal lumen in five cases and haemorrhage was found in the submucosa in some cases. Variety of factors like bacterial and chemical toxins, hypoxia and passive congestion have been indicated to cause damage to the endothelium resulting in haemorrhage. Nair *et al.* (1987) reported diffuse hyperaemia and foci of blood clots in the trachea and froth in the larger bronchi in rabbits died of pneumonia due to *P. multocida.* Petechial haemorrhages were reported in the tracheal mucosa in the rabbits died in outbreaks of Viral Haemorrhagic Disease by Chasey *et al.* (1994).

In many of the carcasses the tracheal lumen was found to be filled with frothy exudate and on tracing the course of the airways the bronchi and the bronchioles were also found to be filled with froth. Variety of factors such as pulmonary oedema, acute or chronic bronchitis and aspiration might have led to the frothy exudate in the airways. Sastry (2001) reported that frothy exudate in the trachea and bronchi was due to churning action of the tracheal air in the protein containing fluid. In one case the animal was having the history of aspiration of glucose fluid.

Examination of the smears prepared from the blood and the exudate in the trachea revealed bipolar organisms suggestive of *Pasteurella multocida* in five cases. Nair *et al.* (1987) also reported similar findings and they could isolate the bacteria from the trachea in such cases. However *P. multocida* could not be isolated in the present study. Most of the cases were having the history of antibiotic treatment for respiratory infection and this may be one of the reasons why the bacteria could not be isolated.

Microscopical examination revealed haemorrhagic tracheitis in 12 percent of the cases. This finding is in agreement with the findings of Nair *et al.* (1987). They noticed rupture of the capillaries in the tracheal mucosa resulting in haemorrhage and stated that haemorrhagic tracheitis was the pathognomonic lesion in pneumonia due to *P. multocida* in rabbits. But in the present study *P. multocida* could not be isolated.

Inflammatory changes alone without haemorrhage were noticed in the tracheal mucosa in 20 percent of the cases. Infiltration with neutrophils, lymphocytes, macrophages and plasma cells were seen. Lymphocytes and macrophages were found to be predominant indicative of chronic nature. Vegad (1995) reported that lymphocytes, and plasma cells constitute the small round cells of chronic inflammation and the macrophages play an important role in healing and repair.

Focal degeneration and desquamation of the tracheal epithelium was seen in 17 per cent of the cases. Extensive destruction of the tracheal mucosa and erosions and ulcerations in the trachea in the rabbits died of pneumonia due to *P. multocida* was reported by Nair *et al.* (1987). Similar findings were also reported by Al- Haddawi *et al.* (2001). They recorded degeneration and sloughing of the surface epithelium in the trachea of rabbits infected with *P. multocida* serotype D:1.

Cystic dilatation of the capillaries on the tracheal wall was seen in 13 percent of the cases. Aneurysm and dilatation of the blood vessel could occur due to some localised weakness of the vessel wall (Crawford, 1976). Bacterial embolus was seen in the tracheal capillary in one case. Vegad (1995) reported that bacterial emboli were frequently observed in many diseases and occurred as single cells or clumps of bacteria blocking in the capillaries and could establish new foci of infection where ever they were lodged.

The pleural membrane covering the lungs was congested in some cases and sub pleural haemorrhage was also evident in two cases. One of the rabbits with the sub pleural haemorrhage was having the history of an accident. Wolf (1997) reported that haemorrhage into the pleural space is usually the result of trauma to the lung or great vessels in the thorax. Haemorrhage occurred frequently in the lungs and beneath the pleura in haemorrhagic diathesis, septicaemia, disseminated Intravascular coagulation and severe congestion or trauma or infarction (Dungworth, 1993).

On gross examination, the lungs appeared to be congested in most of the cases. And on microscopical examination, congestion was the most common lesion (70 per cent) in the lungs. As reported by Sastry (2001) congestion in the lungs could occur due to changes in the vascular tone causing redistribution of blood from systemic to pulmonary circulation. Various conditions such as toxic conditions, vitamin deficiencies, parasitic diseases and systemic infection can lead to pulmonary congestion. Pulmonary congestion in rabbits was recorded in various conditions like, pneumoenteritis due to Calici like virus (Rai *et al.*,1985), pneumonia due to *P. multocida* (Nair *et al.*, 1987), colisepticaemia (Shah *et al.*,1989), aflatoxicosis (Singh *et al.*, 1993), vit-A deficiency (Sravanthy *et al.*, 1996), intestinal coccidiosis (Krishna and Vaid,1987), *Trypanosoma evansi* infection (Chandra *et al.*,1999) and ochratoxin-A toxicity (Mir *et al.*, 1999).

In 22 per cent of the cases haemorrhage was seen in the lung parenchyma. Pulmonary haemorrhage was reported in various conditions like pneumonia

caused by *P. multocida* (Nair *et al.*, 1987), colisepticaemia (Shah *et al.*, 1989), aflatoxicosis (Singh, 1997), haemorrhagic pneumonia (Rai *et al.*, 1995), Viral Haemorrhgic Disease (Kpodekon and Alogninouwa, 1998) and *Toxocara vitullorum* infection (*Pramanik et al.*, 1994).

The lungs were oedematous in many cases and histopathological examination revealed oedema fluid in lung parenchyma in 62 per cent cases. It may be due to the increased capillary permeability due to the toxins produced by the bacterial organisms as stated by Sastry (2001) and usually preceded pneumonia. Pulmonary oedema was reported in aspiration (Spencer, 1968), pneumoenteritis (Rai *et al.*, 1985), colisepticaemia (Shah *et al.*, 1989), bronchopneumonia (Rai *et al.*, 1995) and mycoplasmal infection (Villa *et al.*, 2001).

Emphysema of the alveoli were seen in 19.5 per cent of the cases. In one case the lung was found to be focally emphysematous and with areas filled with froth and the animal was having the history of aspiration of glucose fluid. On microscopical examination, infiltration of mononuclear cells in and around the bronchioles was seen. Dungworth (1993) reported that in aspiration pneumonia the lungs remain inflated and small amount of exudate could be expressed from the small airways and histologically acute bronchitis with varying degree of acute alveolar emphysema.

The various lobes of the lungs were dark red in colour and consolidated in many cases. Rai *et al.*, (1995) reported similar lesions in haemorrhagic pneumonia in rabbits. Consolidation of the cranio-ventral lobes was seen in many cases. Consolidation in the cranio-ventral lobe was indicative of bronchopneumonia. (Dungworth, 1993). Stages of red and grey hepatization indicative of pneumonia were seen. Purplish red consolidation was observed in the lungs of rabbits died due to pneumonia by Pawaiya *et al.*, (1998) and they isolated bacterial organisms *Pasteurella sp.*, *Klebsiella sp.*, pneumococci and *E. coli* . Consolidation in the lungs of rabbits infected with *Trypanosoma evansi*

was reported by Chandra *et al.* (1999). Red to grey consolidation was reported in the lungs in pneumonia due to *Mycoplasma pulmonis* in rabbits by Villa *et al.*, (2001). In the present study the bacteria isolated from the consolidated lungs were *Escherichia coli, Proteus sp.* and *Staphylococcus sp.*

Inflammatory changes indicative of pneumonia were seen in the lung parenchyma in 30 per cent of the cases. Stage of resolution of pneumonia with predominant macrophage reaction was seen in one case. In one case the macrophage reaction was the most prominent reaction indicating the stage of repair. Peribronchial lymphocyte infiltration was noticed in 19 percent of the cases and peri vascular accumulation of lymphocytes was present in one case. Similar findings were reported in enzootic pneumonia caused by septicaemia due to *E.coli* and *Proteus sp.* in rabbits by Flatt *et al.* (1971a) and in pneumoenteritis due to Calici like virus by Rai *et al.* (1985). Rai *et al.* (1985) also reported infiltration of mononuclear inflammatory cells and subsequent moderate thickening of the alveolar septum in the case of interstitial pneumonia in rabbits. Similar type of inflammatory reaction was seen in four percent cases of the present study.

Proliferation of the bronchial epithelium was recorded in 10 per cent of the cases and adenomatous proliferation of the epithelium was seen in one case. Degeneration and desquamation of the epithelium of the bronchiolar epithelium were seen in 25 per cent of the lungs. Similar findings were reported in bronchopneumonia by Dungworth (1993) and aflatoxicosis by Singh *et al.* (1993). Rai *et al.* (1995) observed degeneration and desquamation of the bronchial epithelium forming exudate in the lumen admixed with inflammatory cells in bronchopneumonia in rabbits.

Thrombus in the pulmonary artery was noticed in five percent of the cases and microthrombi were seen in 20 per cent of the cases. Vegad (1995) reported that the capillary thrombi are mostly associated with inflammation as the aetiological agents easily injure the vascular endothelium. Capillary thrombi

were reported by D'suze *et al.* (1999). They observed abundant microthrombi in the rabbit lungs exposed to scorpion venom. Sastry (2001) reported that thrombi often occur in the case of pneumonia due to extension of infection to the blood vessels and thrombosis was frequently observed in septicaemic diseases like Pasteurellosis.

Thickening of the wall of the pulmonary artery was noticed in 10 percent of the cases. In one case thrombus was seen in the hypertrophied artery. Robinson and Maxie (1985) reported that hypoxia induced vasoconstriction lead to work hypertrophy of the muscular tunica media of the pulmonary arteries and hence hypertension leading to endothelial damage and thrombosis.

Though there are no much reports on tumour in the rabbits, in the present study a case of alveolar carcinoma was observed with metastatic foci in other areas including the ovary indicating that tumours are not uncommon in rabbits.

In the present study, the lesions in the lymphnodes were limited in number. Congestion, lymphocyte depletion and loose appearance of parenchyma were present in the bronchial lymphnodes in two cases. This is in contrast to the findings of Nair *et al.* (1987). They observed active germinal centres and reticular hyperplasia along with congestion in the bronchial lymphnodes of the rabbits died of pneumonia due to *Pasteurella multocida.* Glavits and Magyar (1990) reported atrophy of the lymphoid tissues in the rabbits died due to inoculation of *P. multocida* and *Bordetella bronchiseptica* in the respiratory system.

Bacterial isolations were obtained from twenty out of the fifty cases (40 per cent). The bacteria were isolated from the lungs and trachea with vascular and inflammatory lesions. This indicate that most cases of the vascular and inflammatory conditions were associated with some infectious cause. However, *Pasteurella multocida* could not be isolated from cases of haemorrhagic tracheitis. Though pathogenecity study in the mice revealed that the *Escherichia coli* isolated from the rabbit was pathogenic to mice but such study was not

conducted in rabbits and hence confirmatory results were not obtained regarding the pathogenecity of *E.coli* in rabbits.

The respiratory system is always in direct contact with the outside environment and is susceptible to various changes occurring in the environment. The systemic investigation undertaken on the pathology of the respiratory system has helped to focus attention on the prevalence of various respiratory disorders in the rabbits. The study has been very fruitful in the classification of different types of histopathological lesions and also finding out the microbial etiology of the respiratory disorders to some extent so that appropriate line of prophylaxis and treatment can be suggested against the respiratory infections.

Summary

6. SUMMARY

An investigation was undertaken to study the pathology of the respiratory system in rabbits.

Samples from the respiratory system especially the trachea, lungs and bronchial lymphnodes collected from fifty rabbit carcasses brought for autopsy at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy were used for the study. The samples collected were subjected to detailed gross, histopathological as well as microbiological examination. The conditions encountered were classified, the pathological features were recorded and each lesions were explicated giving possible etiopathogenesis.

The study revealed a high prevalence (80 per cent) of respiratory disorders in rabbits. The lesions seen in the trachea were congestion (62 per cent), haemorrhagic tracheitis (12 per cent), Emboli (two per cent), cystic dilatation of the capillary (13 per cent), inflammation (20 per cent) and degeneration and desquamation of the epithelium (17 per cent).

The pleura revealed congestion (four per cent) and sub pleural haemorrhage (4 per cent) and focal necrotic area in one case. The lesions in the lungs were congestion (70 per cent), haemorrhage (22 per cent), arterial thrombi (five per cent), capillary thrombi (20 per cent), oedema (62 per cent), blood vessel wall thickening (10 per cent), emphysema (19.5 per cent), pulmonary collapse (13 per cent), inflammation (30 per cent), peribronchial lymphocytic infiltration (19 per cent), perivascular lymphocytic accumulation (two per cent), thickening of alveolar septum (four per cent), collapse of the bronchioles (30 per cent), proliferation of bronchiolar epithelium (ten per cent) and degeneration and desquamation of bronchiolar epithelium (25 per cent).

Vascular lesions were predominant among the various histopathological lesions encountered in the trachea, lungs and the bronchial lymphnodes. The vascular changes included congestion, haemorrhage, oedema, thrombus, embolus, cystic dilatation of capillaries and thickening of the blood vessel wall. The involvement of an acute infectious condition was suspected in many of the cases. The prevalence of congestion in histopathological observations could be attributed to the pneumonic changes, which formed majority of the pathological conditions.

Inflammatory reactions in the trachea were indicative of a picture of chronic inflammation with infiltration of lymphocytes, plasma cells and macrophages. Degenerative changes were also seen in the trachea along with desquamation of the epithelium. *Escherichia coli* was isolated from such cases which was found to be pathogenic to mice.

Pneumonia claimed 30 per cent of the rabbit mortality. Bronchopneumonia was found to be the most prevalent type of pneumonia. Infiltration of inflammatory cells, degeneration and desquamation of the bronchiolar epithelium were also found along with other pneumonic changes. Cases of haemorrhagic pneumonia were also seen. *E.coli*, *Proteus sp.* and *Staphylococcus sp.* were isolated from the pneumonic lungs.

Congestion and lymphocytic depletion were noticed in the bronchial lymphnodes occasionally.

The investigation proved beyond doubt that the respiratory pathology in the rabbits was more common than expected. This will create an awareness among the clinicians on the common respiratory disorders leading to mortality and aid them in choosing the suitable preventive and curative measures. The respiratory system manifests various pathological disorders and there is need to have well drawn out research project covering a large population of animals in order to asses the magnitude of the problem.

The history of the carcasses pointed that the respiratory disorders were more common in the rabbits in extremes of the climate and may be due to the flaring up of the microbial organisms which are the normal inhabitants of the respiratory tract in the rabbits. Hence, it is possible that a detailed investigation will help to bring to light the high prevalence rate of many of the known conditions and certain unknown conditions.

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PATHOLOGY OF THE RESPIRATORY SYSTEM IN RABBITS

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ABSTRACT OF A THESIS

Submitted in partial fulfilment of the requirement
for the degree of

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University, Thrissur

2003

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ABSTRACT

The present study was undertaken to assess the pathology of the respiratory system in rabbits. The results of the study revealed a high incidence (80 per cent) of respiratory disorders. A detailed systematic examination of fifty cases of rabbit carcasses brought for autopsy during the period of investigation was conducted and the gross and histopathological lesions were studied in detail. Vascular and inflammatory lesions were predominant in the trachea and the lungs. Degenerative changes were seen in the trachea, pleura and the lungs. Bronchial lymph nodes revealed mild congestion and depletion of lymphocytes occasionally. A case of alveolar carcinoma was encountered in the present study. Adenomatous proliferation of the bronchial epithelium was seen in one case. Bacterial isolations were obtained from the trachea and the lungs. *Escherichia coli* (16 cases), *Staphylococcus sp.* (3 cases) and *Proteus sp.* (1 case) were obtained. The need and the scope of investigation into the pathological disorders in the respiratory system of the rabbits were highlighted.