# PATHOLOGY OF PNEUMO-ENTERIC LESIONS IN GOATS

HAMZA PALEKKODAN

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Centre of Excellence in Pathology
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY, THRISSUR-680651
KERALA, INDIA

## DECLARATION

I hereby declare that the thesis entitled **"PATHOLOGY OF PNEUMO-ENTERIC LESIONS IN GOATS"** is a bonafide record of research work done by me during the course of research and that this 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.

Mannuthy

**HAMZA PALEKKODAN**

## CERTIFICATE

Certified that this thesis, entitled **"PATHOLOGY OF PNEUMO-ENTERIC LESIONS IN GOATS"** is a record of research work done independently by **Dr. Hamza Palekkodan,** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, associateship or fellowship to him.

**Dr. N.Vijayan**
(Chairman, Advisory Committee)
Professor,
Centre of Excellence in Pathology,
College of Veterinary and Animal Sciences,
Mannuthy.

Mannuthy

## CERTIFICATE

We, the undersigned members of the Advisory Committee of **Dr. Hamza Palekkodan**, a candidate for the degree of **Master of Veterinary Science in Pathology**, agree that the thesis entitled **"Pathology of pneumo-enteric lesions in goats"** may be submitted by **Dr. Hamza Palekkodan,** in partial fulfilment of the requirement for the degree.

**Dr. N. Vijayan**
(Chairman, Advisory Committee)
Professor,
Centre of Excellence in Pathology,
College of Veterinary and Animal Sciences,
Mannuthy.

**Dr. C. R. Lalithakunjamma**
Professor and Head,
Centre of Excellence in Pathology,
College of Veterinary and Animal Sciences,
Mannuthy.
(Member)

**Dr. N. Divakaran Nair**
Associate Professor,
Centre of Excellence in Pathology,
College of Veterinary and Animal Sciences,
Mannuthy.
(Member)

**Dr. G. Krishnan Nair**
Professor and Head,
Department of Microbiology,
College of Veterinary and Animal Sciences,
Mannuthy.
(Member)

**EXTERNAL EXAMINER**

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# *Introduction*

# 1. INTRODUCTION

Goat as poor man's cow plays a significant role in the economy and nutrition of landless, small and marginal farmers in the country. Goats are kept as a source of additional income and as insurance against disaster. Goat farming is generally reported as successfully integrated with traditional agricultural production system and have the advantage that there are no cultural restrictions to goat keeping. They are the source of income for women who take primary responsibility for their care.

From the unorganised backyard system of rearing, which was predominant in Kerala, goat rearing is now gradually shifting to organised goat farming on commercial basis. As part of the intensive rearing, new germplasm is being introduced to our herds. Along with introduction of vigorous management practices to increase the profit, disease conditions which were not previously reported are also emerging. There are increased reports of mortality due to diseases such as Peste des petitis ruminants (PPR) and Bluetongue in the neighbouring states from where large number of sheep and goats are being brought to Kerala.

Among the various conditions in goats and kids, pneumonia and enteritis accounts for increased mortality rate causing high economic loss to farmers. Even though there are specific pathogens which are responsible for producing enteritis and pneumonia, these diseases are of multifactorial etiology. The conditions such as intensive husbandry practice, physical stress such as over crowding, exposure to inclement weather, unhygienic stable conditions, starvation and parasitism, reduce the natural resistance and make the animal more susceptible to infections. Since the respiratory and gastro-intestinal systems are in continuous exposure to the external environment through inspired air and ingested feed, any reduction in natural resistance will predispose these systems to serious affections leading to morbidity and mortality in animals. Under these circumstances pathogens present

in upper respiratory tract and digestive tract which does not cause diseases under normal condition, may flare up and produce severe effects.

Considering the importance of these two systems as the seat of many diseases, a detailed investigation on the common disease conditions affecting these systems and a systemic correlation of concurrent, co-existent or otherwise of the lesions is inevitable. Hence a detailed investigation is warranted to elucidate the pathology of pneumo-enteric conditions seen in goat population so as to reschedule various prophylactic measures in commercial goat production from time to time and to recommend proper control measures, thus making goat farming more viable and profitable in the coming years. Therefore the present study has been undertaken with the following objectives.

1. Study the mortality pattern and prevalence of pneumo- enteric lesions.
2. Evaluate the gross and histopathological lesions encountered in the pulmonary and digestive systems and their correlation.

# 2. REVIEW OF LITERATURE

## 2.1 INCIDENCE

Manomohan (1980) in a study conducted to evaluate the pathoanatomical features of the important diseases causing mortality in kids, reported gastroenteritis (73.3 per cent) as the most important cause of postnatal mortality in kids. Pneumonia caused mortality in seventeen per cent cases.

Nair (1982) observed 53.45 per cent incidence of pneumonic lesions in goats and classified the lesions as acute suppurative bronchopneumonia, chronic bronchopneumonia, fibrinous pneumonia, acute catarrhal bronchopneumonia, haemorrhagic pneumonia and aspiration pneumonia.

Sriraman *et al.* (1982) in a study on the causes of mortality in 554 goats observed pneumonia and enteritis as the most frequent causes in kids and adults. Mortality was highest among females compared to males. Highest mortality was recorded during first two quarters of the year.

Mondal *et al.* (1995) in an epizootiological study in an outbreak of Pestes des petits ruminants (PPR) observed that the disease outbreak occurred during late winter and summer months, due to high humidity that favours the thriving and multiplication of the virus. Morbidity rate was 45.29 per cent and mortality rates varied from 23.8 to 38.2 per cent.

Singh *et al.* (1995 a) reported lesions of schistosomiasis in the lungs of 19 sheep and 23 goats, out of 351 sheep and 249 goat lungs having pneumonic lesions examined.

Sasani *et al.* (1998) collected 73 pneumonic lungs of sheep and classified the lesions as lymphoproliferative interstitial pneumonia, purulent broncho pneumonia, chronic interstitial pneumonia, pulmonary adenamatosis, chronic pneumonia and fibrosis, broncho interstitial pneumonia, chronic pleurisy, serous

bronchopneumonia with PI-3 virus, nonpurulent broncho pneumonia and fibrous broncho pneumonia.

An outbreak of PPR amongst migratory Gaddi sheep and goats in Sirmaur district of Himachal Pradesh has been reported. The morbidity rate was 25.84 per cent and mortality rate was 3.43 per cent (Katoch *et al.*, 1999).

A detailed study on PPR disease outbreaks was made during the two years period from February 1997 to January 1999, involving 18 districts of Andhra Pradesh. It was observed that the percentage of infection was more in young stock, (63.58 per cent) compared to adults (45.92 per cent) in both sheep and goats (Rajeswari *et al.*, 2000).

Srinivasan *et al.* (2003) reported 24 per cent mortality due to pneumonia among 243 sheep, from April 2000 to March 2001, at Sheep breeding research station Sandynallah.

Radfar *et al.* (2005) in an abattoir study observed that 12.87 per cent sheep were infected with *Cysticercus tenuicollis* cyst while in goats the incidence rate was18.04 per cent.

Ravishankar *et al.* (2005) confirmed presence of bluetongue virus antibody in sheep and goats of Kerala by conducting dot Enzyme-Linked Immunosorbent Assay (dot ELISA) and the over all prevalence observed was 5.1± 1.9 per cent.

In a study conducted by Sharif *et al.* (2005) in North Jordan about kid mortality, observed diarrhoea as the cause for 59.75 per cent mortality while pneumonia was present in 13.3 per cent cases.

Shome *et al.* (2005) reported six outbreaks of contagious ecthyma in goats in Meghalaya within a period of 2 months (May-June, 2003) from six goat farms

comprising of 203 goats. The mortality and morbidity recorded was 4.4 per cent and 64.04 per cent respectively.

Illango (2006) reported outbreak of bluetongue in Tamil Nadu during the monsoon season of 1997-1998 which caused death of 30,000 sheep and goats.

Karunanithi *et al.* (2006) reported an outbreak of goat pox in an organized farm in Tamil Nadu. The mortality and morbidity in goats were found to be 16.36 per cent and 57.57 per cent respectively.

Soundararajan *et al.* (2006) studied mortality pattern in Tellichery goats under semi-intensive system from April 1992 to March 2002. The overall mortality rate was 6.14 per cent. The female (56.85 per cent) had higher mortality rate than the males (43.15 per cent). The highest mortality was recorded in young (45.64 per cent) followed by kids (30.29 per cent) and adult animals (24.07 per cent). Enteritis (50.68 per cent) was the major problem followed by pneumonia (36.99 per cent).

## 2.2 PULMONARY LESIONS

### 2.2.1 Etiology of pulmonary lesions

#### *2.2.1.1 Bacteria*

Samuel *et al.* (1975) isolated *Corynebacterium pyogenes* and *Pasteurella multocida* from a mountain goat kid showing numerous pulmonary abscesses.

*Corynebacterium pseudotuberculosis was* isolated from enlarged upper cervical and mediastinal lymphnodes of a goat which contained small pockets of yellowish green caseous material (Guthery and Beasom, 1979).

Davis *et al.* (1999) isolated *Rhodococcus equi* from lung of two goats showing raised nodular lesion in pulmonary parenchyma.

*Corynebacterium pseudotuberculosis* was isolated from abscesses in the mandibular, mediastinal and mesenteric lymphnodes of an eighteen month goat by Karimi *et al.* (2003).

Srinivasan *et al.* (2003) isolated *Klebsiella* sp., *Pasteurella* sp., *Staphylococcus* sp., *Streptococcus* sp., *Escherichia* sp. and *Proteous* sp. from pneumonic lung of sheep collected from slaughter houses.

Kapoor *et al.* (2004) conducted microbiological examination of pneumonic lung samples from goats and isolated bacteria such as *Pasteurella multocida, Escherichia coli, Actinomyces pyogenes, Streptococcus pneumoniae, Staphylococcus* sp., *Citrobactor* sp., *Pseudomonas* sp., *Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus, Bordetella bronchiseptica, Klebsiella* sp., *Bacillus cereus* and *Streptococcus pyogenes* from the samples.

*2.2.1.2 Virus*

Neutralization antibodies to Respiratory syncitial virus (RSV) were detected in 29 (42%) serum samples out of 69 goat samples examined. This virus is potential predisposing agent of pasteurellosis and other respiratory diseases in animals. (Dunbar and Foreyt, 1986).

Rajeswari *et al.* (2000) confirmed PPR outbreak by conducting Immunocapture Elisa test on lung samples collected from goats died of pneumonia and diarrhoea.

Sharp and Nettleton (2000) detected acidophilic intracytoplasmic inclusion bodies in the bronchiolar epithelium of the lung in sheep affected with Parainfluenza viral infection.

Shakya *et al.* (2004) isolated and characterised Capri pox virus from field outbreaks in goats showing pock lesion on the skin, respiratory and gastro-intestinal mucosa.

Shome *et al.* (2005) by conducting Agar Gel Immuno Diffusion confirmed an out break of Contagious ecthyma in goats showing papular lesions on buccal cavity, on the muzzle and nose, along with signs of respiratory distress and mucopurulent discharge.

Karunakaran *et al.* (2006) detected PPR viral antigen in clinical samples and postmortem samples such as lung, mesenteric lymphnodes, spleen, and liver collected from goats showing symptoms of respiratory distress by conducting sandwich ELISA.

#### *2.2.1.3 Fungus*

Sharmah and Dwivedi (1977) reported pulmonary Blastomycosis caused by *Blastomyces braziliensis* in sheep and goat. The lung showed calcified mycotic granulomas in the parenchyma.

Singh *et al.* (1995c) observed nodular lesion of pulmonary Aspergillosis in sheep and goats with consolidation of lung. Nodules were more prominent in diaphragmatic lobes and when cut revealed thick discoloured pus.

Baro *et al.* (1998) isolated yeast like fungus *Cryptococcus neoformans* from lung samples of goat with chronic pneumonia.

#### *2.2.1.4 Mycoplasma*

Kinde *et al.* (1994) isolated *Mycoplasma agalactiae* and *Mycoplasma mycoides* from the lungs of the goats with lesions of fibrinous pleuritis, emphysema, and peritonitis.

Bolske *et al.* (1996) identified the presence of *Mycoplasma capricolum* subsp. *capripneumonia* by Polymerase Chain Reaction (PCR) in clinical samples collected from goats and sheep with Contageous caprine pleuropneumonia (CCPP).

Hernandez *et al.* (2006) isolated *Mycoplasma capricolum* subsp. *capripneumoniae* from an outbreak of respiratory disease occurred in goats in Mexico.

*Mycoplasma capricolum* and *Mannheimia haemolytica* were isolated from lung lesion and thoracic fluid of goats affected with fibrinous pneumonia. (Shiferaw *et al.*, 2006).

### *2.2.1.5 Parasites*

*Muellerius* sp. were isolated from different cases of goats with various lesions such as chronic pneumonia and interstitial pneumonia (Dermartini and Davies, 1977; Nimmo, 1979).

Mandial *et al.* (1999) detected presence of *Protostrogylus rufescens* in the lung of goats died after showing the symptoms of chronic cough.

Kumari *et al.* (2000) observed hydatid cyst in the lung of goats, characterised by presence of elevated cyst surrounded by thin transparent membrane on the lung parenchyma.

### *2.2.1.6 Aspiration*

Nair (1982) experimentally produced aspiration pneumonia in kids using rumen liquor. The lesions varied from mild bronchopneumonia on the first day to extensive acute suppurative bronchopneumonia on the third day. *Escherichia coli (E.coli)* and *Klebsiella pneumoniae* were isolated from the infected lung.

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

### 2.2.2 Pathology of pulmonary lesions

#### *2.2.2.1 Vascular changes.*

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

Mondal *et al.*, (1995) observed haemorrhages in trachea, bronchi and lung parenchyma in goats died of PPR infection.

Batra *et al.* (2000) noticed congestion of pulmonary capillaries, oedema, congestion of alveolar wall and thickening of interlobular septa in lung of lambs experimentally infected with *Mycoplasma mycoides* subsp. *mycoides.*

According to Sastry (2001) congestion in the lungs could occur due to change in the vascular tone causing redistribution of blood from systemic to pulmonary circulation.

Batra *et al.* (2002) observed haemorrhagic patches in apical and diaphragmatic lobes of lung of the lambs experimentally inoculated with *Mycoplasma mycoides* subsp. *mycoides* and *Pasteurella hemolytica* simultaneously. Histologically there was marked vascular changes and presence of eosinophilic exudate in alveoli where as grossly trachea exhibited congestion and blood mixed frothy exudates along with haemorrhagic streaks in the cartilaginous rings.

Sivaseelan (2003) observed haemorrhage at the base of pulmonary artery in a spotted deer died due to bluetongue disease and described it as the most distinctive lesion.

Sharma *et al.* (2003) in an experimental study by orally administering *E. coli* (strain O26) observed petechial haemorrhage in lung parenchyma. There was congestion of septal wall leading to thickening of alveolar septa.

Pawaiya *et al.* (2004) observed linear haemorrhage throughout the mucosa of trachea and bronchi with frothy exudates in goats naturally infected with PPR virus. Apical and cardiac lobes were prominently involved with congestion and purple red discolouration. Mediastinal and bronchial lymphnodes were swollen and congested.

### *2.2.2.2 Inflammation*

Abundance of fibrin, involvement of inter lobular connective tissue, vasculitis and perivasculitis were predominant features of infection due to *Pasteurella* sp. in sheep and goat pneumonia (Ramachandran and Sharma, 1969).

Bhagwan and singh (1972) classified pneumonia in sheep and goat as exudative and non-.exudative. Exudative type included acute suppurative pneumonia and haemorrhagic pneumonia, while non-.exudative pneumonia included interstitial pneumonia, pneumonitis, verminous pneumonia and proliferative pneumonia.

Sparker and Collins (1986) observed suppurative bronchopneumonia in lamb, with severe consolidation of lung in respiratory syncitial virus infection.

Lopez (1995) used the term bronchointersitial pneumonia to describe the pulmonary lesions showing histological features of both bronchopneumonia and interstitial pneumonia.

Singh *et al.* (1995a) observed nodular lesions in lungs of goats affected with schistosomiasis. Microscopically the nodular structure revealed few to multiple granulomata surrounding the schistosome ova. There was large number

of macrophages, giant cells and lymphocytes, surrounded by proliferating fibroblasts.

Singh *et al.* (1995 b) observed caseopurulent to caseocalcified nodules embedded in various lobes of lungs in pulmonary pseudotuberculosis. The bronchial and mediastinal lymph nodes were enlarged and had caseopurulent/ caseocalcified lesions.

Storset *et al.* (1997) described the lung lesion in caprine arthritis encephalitis infection in goats which was charecterised by perivascular lymphocytic cell infiltration along with septal fibrosis.

Sasani *et al.* (1998) observed that in most of the cases pneumonic lesions were present on the cranio-ventral aspect of apical and cardiac lobe and about 50 per cent mediastinal lymphnodes were swollen.

Concomitant infection of *Cryptococcus neoformans* and *Mycobacterium bovis* causing pneumonia in goat was reported by Gutierrez and Marin (1999). Grossly lung parenchyma revealed nodules and caseous foci of 0.5 - 2 centimeter diameter. Microscopically nodules revealed yeast like organisms extracellularly and remnants of alveolar septa with minimal inflammatory response. Caseous foci appeared with a necrotic center, surrounded by a thin layer of epitheliod and multinucleated giant cell with infiltration of lymphocytes and plasma cells.

Batra *et al.* (2000) observed serofibrinous exudates containing mononuclear cells and neutrophils in the alveolar lumen of lung of lamb experimentally infected with *Mycoplasma* sp.

Lehmkuhl *et al.* (2001) observed bronchopneumonia in adeno viral infection in kids.

Shivarudrappa *et al.* (2004) classified suppurative pneumonia into mucopurulent pneumonia, fibrinopurulent pneumonia, acute suppurative

bronchopneumonia, necrotising suppurative bronchopneumonia and Chronic suppurative bronchopneumonia depending upon either severity and the type of exudates.

Lung showed fibrinohaemorhagic pneumonia with accumulation of fibrin, erythrocytes and neutrophils in alveolar lumen and large areas of coagulation necrosis in kids died due to adeno viral infection (Olson *et al.*, 2004).

Wesonga *et al.* (2004) observed fibrinous pleuropneumonia charecterised by mucopurulent to fibrinopurulent exudates in dilated hyperplastic bronchi in lungs of goats experimentally infected with *Mycoplasma* sp. The alveolar septum and peribronchial region exhibited various degrees of fibrotic changes.

Pawaiya *et al.* (2004) reported characteristic broncho-intersitial pneumonia in goats naturally infected with PPR virus. Bronchial epithelium was hyperplastic and the lumen of bronchi was obliterated with inflammatory exudates and desquamated epithelial cells which fused to form multinucleated syncitia containing single to multiple intracytoplasmic inclusions.

Abraham *et al.* (2005) reported severe consolidation of lungs in goats died during a PPR outbreak. Microscopically lungs showed multifocal suppuration, perivascular infiltration, diffuse proliferation of intersitium and mononuclear cell infiltration.

Samanta *et al.* (2006) observed fibrinous pneumonia in lambs and *Plesiomans shigelloides* was isolated from the postmortem samples.

### *2.2.2.3 Neoplasms*

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

Pawaiya and Bhagwan (2000) reported a case of primary bronchiolo-alveolar carcinoma in a lamb with grey white bulging nodules of varying sizes in lung parenchyma. Histopathological examination revealed cuboidal epithelial cell clumps exfoliating into the alveolar lumen with hyper chromatic dividing nuclei.

## 2.3 LESIONS OF INTESTINAL TRACT

### 2.3.1 Etiology of intestinal lesions

#### *2.3.1.1 Bacteria*

Dimitracopoulos *et al.* (1976) isolated *Staphylococcus aureus* in 96 per cent of the rectal swabs from 133 sheep and in 80 per cent of the swabs from 125 goats.

Saxegaard (1985) collected parts of jejunum, ileum and associated lymphnodes from 1501 goats and inoculated in selective Dubos medium, out of which 18 samples were positive for *Mycobacterium paratuberculosis.*

Roy *et al.* (1986) collected rectal swabs from 103 kids showing the symptoms of enteritis and got *Escherichia coli* (66.24 per cent), *Proteus mirabilis* (18.9 per cent), *Citrobacter* (2.4 per cent) and *Klebsiella* (2.4 per cent) as isolates.

Olson *et al.* (2004) isolated *E.coli, Streptococcus* sp., *Proteus* sp. and *Clostridium perfringens* from intestine of kids infected with adeno virus.

Entero-toxigenic strains of *E. coli* were isolated from faecal and mucosal scraping of the large intestine of lambs showing the signs of haemorrhagic diarrhoea (Elfaki, 2000; Novatna *et al.,* 2005).

### *2.3.1.2 Virus*

Eligulashvili *et al.* (1999) detected PPR viral antigen in the cytoplasm of epithelial cells of intestinal crypts by Immuno histochemical method in goat died with natural PPR infection. Intracytoplasmic inclusion bodies were also observed in epithelial cells of intestinal crypts.

Lehmkuhl *et al.* (2001) isolated adeno virus from the small intestine of a three week old kid died of diarrhoea.

Ahmed *et al.* (*2003*) detected rota virus in 16 samples out of total 259 faecal samples collected from diarrhoeic goat kids. The highest rate of rota virus infection occurred in one week to one month old kids.

Olson *et al.* (2004) isolated an adeno virus and an adeno - associated virus from the intestinal contents of kids died with diarrhoea and dyspnoea.

### *2.3.1.3 Parasites*

Manomohan (1980) observed one case of Taeniasis in a kid with lesions of catarrhal enteritis in small intestine.

Out of 1208 goats and 542 sheep examined, 297 goats and 120 sheep showed lesions of helminthiasis in intestinal tract and lesions of coccidiosis were observed in 15 goats and 5 sheep (Sharma *et al.*, 1997a).

Deger *et al.* (2003) collected faecal samples from the rectum of 242 goats and subjected to microscopical examination and *Eimeria* oocysts were detected in 73.6 per cent of the samples.

Mandonnet *et al.* (2003) observed *Haemonchus contortus* and *Trichostrongylus colubriformis* as major strongyles responsible for mortality in kids. The probability of death following gastro intestinal infection was more than three times greater in males than females.

Das *et al.* (2005) reported higher percentage mortality in kids born from goats infected with gastro intestinal nematodes, compared to kids born from nonparasitized pregnant goats.

Dhollander *et al.* (2005) observed 20 per cent mortality in kids at preweaning stage and the microscopical examination of intestinal content revealed high number of coccidial oocysts.

Sevinc *et al.* (2005) reported massive outbreak of watery diarrhoea in kids aged 5 to 15 days in a goat herd in Turkey, caused by *Cryptosporidium parvum*, which resulted death of 70 kids.

Out of 106 faecal samples of goats examined, 20 samples revealed presence of *Crytptosporidium* sp. with a prevalence rate of 18.8 per cent (Goz *et al.*, 2006).

***2.3.1.4 Chlamydia.***

Krishna and Rajya (1999) in an investigation on perinatal mortality in kids observed homogenous eosinophilic mass, indicative of degenerated forms of chlamydial organism, in mononuclear cell infiltrate in mucosa and submucosa of intestine of kids. They were able to demonstrate the chlamydial group specific antigen through fluorescent antibody technique.

**2.3.2 Pathology of intestinal tract.**

***2.3.2.1 Degeneration and necrosis.***

Sharma *et al.* (1966) observed local papillary like of elevation of the mucosa as a result of focal hyperplasia, due to large number of parasiting gametocyte stages and oocyst, in small intestine of kids naturally infected with *Eimeria arloingi.*

Bundza *et al.* (1988) observed eosinophilic intranuclear inclusion in the epithelial cells of intestine in sheep and goats experimentally infected with PPR. Small intestine showed degeneration of villi, cellular casts in the crypts, and depletion of the lymphoid cells in the Peyer`s patches.

Gelberg (1995) reported acellular polypoid projection with faded stroma in mucosa of small intestine due to toxin of *Clostridium perfringens.*

Pathak and Parihar (1995) noticed occasional degeneration and desquamation of the surface epithelium along with congestion of lamina propria and submucosa in both small intestine and large intestine of sheep after inoculating epsilon toxin of *Clostridium perferingens* through intravenous route.

Uzal and Kelly (1998) observed necrotising pseudomembranous formation in colon of kids after intraduodenal inoculation of *Clostridium perfringens* culture and culture products.

DeBey *et al.* (2001) reported villous atrophy in the small intestine of lambs died with adeno viral infection.

Lehmkuhl *et al.* (2001) observed crypt necrosis in small intestine and colon of kids in adeno viral infection.

Barlow *et al.* (2004) demonstrated attaching and effacing lesions caused by *Escherichia coli* in the intestine of an adult goat with the symptoms of diarrhoea, by immunoperoxidase method. Grossly the mucosa appeared normal with moderate amount of fluid in the lumen. Ileum showed severe stunting and fusing of the villi, leading to flattening of the mucosa along with adherence of bacteria on mucosal surface.

Devi *et al.* (2004) observed small grayish white foci of 1-2 mm diameter, scattered through out the mucosa of small intestine of sheep infected with

*Eimeria arloingi*. Microscopically this area revealed severe necrosis of the intestinal villi.

Housawi *et al.* (2004) noticed mucosal congestion, haemorrhage and small erosion in the duodenum of sheep and goats naturally infected with PPR. The jejunum was congested and Peyer's patches appeared shallow with hyperaemic rims. The ileum was congested while colon and rectum had congested mucosa with linear haemorrhages.

Olson *et al.* (2004) noted cellular debris in crypt of ileum along with basophilic to amphophilic intranuclear inclusion bodies, filling the nucleus in adeno viral infection.

Abraham *et al.* (2005) reported necrosis, desquamation and denudation of the villi in intestine of goat infected with PPR.

**2.3.2.2 *Inflammation.***

Lees *et al.* (1991) observed mucosal congestion along with severe mononuclear infiltration in the gut wall of lambs infected with border disease virus. Distal third of intestinal villi showed necrotic changes.

Duhamel *et al.* (1992) reported multifocal coalescing necrosis of the superficial mucosa associated with suppurative inflammation in the lamina propria of large intestine of a kid died with enteric colibacillosis.

Rae (1994) noticed lymphocytic enteritis in seven sheep with lesion involving the entire length of small intestine. The lamina propria was filled with diffuse infiltrate of lymphocytes. Small number of plasma cells and polymorphonuclear cells were also present.

Oros *et al.* (1996) observed catarrhal enteritis in a two month old kid infected with adeno virus, characterised mainly by neutrophilic granulocyte

together with a small number of lymphocytes, plasma cells and macrophages. Many of the epithelial cell nuclei were enlarged, round and displaced to the apical portion of the cells. These nuclei showed peripheral margination of chromatin and acidophilic intra nuclear inclusion bodies with a peripheral halo.

An intense inflammatory reaction consisting mainly of lymphocyte and some eosinophils in the lamina propria of small intestine of a goat against natural infection of *Schistosoma* sp. was reported by Chac *et al.* (1996).Tunica muscularis showed symmetrical hyperplasia of both smooth muscle layers.

Sharma *et al.* (1997b) reported inflammatory fibroid polyp in jejunum of a goat, which appeared as focal annular thickening in which lumen was almost obliterated. Histopathologicaly there was a large mass of fibrous connective tissue in the submocosa elevating the superficial mucosal layer. Mucosa showed mild to moderate infiltration of mononuclear cells and a few neutrophils.

Sharma *et al.* (1997a) observed varying number of *Trichuris* worms in caecum and colon of sheep and goat and grossly the intestine remained apparently normal except for focal mucosal thickening in severe infection. Sections of the worms were discernible in superficial tunnel in the mucosa. Areas in the immediate vicinity revealed compression atrophy of epithelial tissue and mononuclear cell infiltration.

Sharma *et al.* (1998) conducted histopathological examination of grossly affected alimentary tract from 462 goats and 147 sheep and observed inflammatory lesion unassociated with any viral or parasitic infection in 70 goats and 30 sheep. He classified the lesions as acute enteritis, subacute enteritis and chronic enteritis.

Foreyt *et al.* (2001) described the lesion of salmonellosis as severe diffused necro suppurative entero colitis. Intestinal villi were blunted and usually

denuded of epithelium. Large areas of necrosis through out the lamina propria extending deep into the submucosa were characteristic.

Catton (2002) reported paucobacillary type of paratuberculosis in a five-year old Saanen doe which was characterised by extensive lymphocytic infiltration of the ileal lamina propria. Gross postmortem findings were thickened corrugated ileum and oedematous mesenteric lymph nodes.

Pawaiya *et al.* (2004) noted inflammatory exudation with mononuclear cells in the lamina propria along with degenerative, necrotic and oedematous changes in PPR infection of goats.

Ragione *et al.* (2005) observed moderate infiltration of mixed lymphoid cell and polymorphonuclear cells in lamina propria of kids experimentally infected with toxigenic strain of *E.coli.*

# *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 pathology of pneumo-enteric lesions in goats as well as to study the mortality pattern of goats.

## 3.1 PREVALENCE STUDY.

A retrospective study for a period of five years from 2002 to 2006 was conducted to find out the mortality pattern as well as the prevalence of pulmonary and intestinal lesions. The data for this study were collected from the postmortem records maintained at the centre. The data obtained were analysed to study the influence of age, sex and season on the mortality. To study the age-wise incidence goats were divided into three age groups *viz.,* kids (up to six month old), young ones (six month to one year old) and adults (above one year old). To study the seasonal influence on mortality, year was divided into first quarter (January to March), second quarter (April to June), third quarter (July to September) and fourth quarter (October to December). The available data pertaining to the gross pathological lesions of lung and intestine were collected and analysed.

## 3.2 PATHOLOGY OF LUNG AND INTESTINE.

### 3.2.1 Sample Collection

Carcasses of goats brought for autopsy were utilised for the study. Samples of lung, small intestine, large intestine and associated lymphnodes were collected and subjected to detailed gross and histopathological examination. Animals of all age groups were utilised for the study.

### 3.2.2 Gross Examination

Detailed postmortem examination was conducted on goats brought for postmortem examination. Respiratory and digestive systems were carefully dissected and examined systematically for any appreciable gross lesions.

The thoracic cavity was examined for the presence of any exudates. The pleural surface and the borders of the lungs were examined for change in lusture, colour, consistency and presence of any adhesion. The size, colour and consistency of the bronchial and mediastinal lymphnodes were noted. The trachea and bronchi were opened and the contents of the lumen were examined for exudates. Each lobe of the lung was examined separately for changes on the surface and parenchyma and the lobes were palpated to detect the changes in consistency.

The intestinal tract was separated from rest of the carcass and examined for any gross lesion. All parts of small intestine and large intestine were thoroughly examined. The intestine was incised for examining the mucosal surface, nature of contents and for the presence of any parasites. Mesenteric lymphnodes were examined along with intestine for any change in colour, size and consistency.

### 3.2.3 Histopathology

The representative parts of lung, small intestine, large intestine and associated lymphnodes showing gross pathological changes were collected and preserved in 10 per cent neutral buffered formaldehyde solution for histopathological examination. All the tissue samples collected were processed by paraffin embedding technique (Sheehans and Hrapachak, 1980). The paraffin embedded tissues were cut at four micron thickness and stained with Haematoxylin and Eosin stain (H & E stain) as described by Bancroft and Cook

(1995). The stained sections were subjected to detailed examination under the light microscope and the lesions were classified.

## 3.3 MICROBIOLOGICAL STUDIES

Pieces of pulmonary tissue and intestine ligated at both ends were collected aseptically from the fresh postmortem cases showing gross lesions for isolation studies. Surface of pulmonary tissue as well as intestine was singed using a hot spatula. On the singed surface a deep cut was made using a flamed pointed scissors. Sterilised bacterial loop was introduced deep into cut surface of pulmonary tissue and then streaked on Trypticase Soya Agar (TSA). Intestinal contents were streaked on both Mac-conkey and TSA agar. The plates were incubated at 37°C for 48 hours. The colonies were identified by cultural, morphological and biochemical characters.(Barrow and FIltham, 1993)

# *Results*

# 4. RESULTS

## 4.1. RETROSPECTIVE STUDY

### 4.1.1. Mortality pattern

As per the records maintained in the centre a total of 448 goat carcasses were autopsied during the period of 2002-2006.The Highest mortality occurred in the year 2006 with death of 131 goats and the lowest mortality in the year 2003 (Table 1). During the above period mortality was highest among kids (54 per cent) followed by adults (37 per cent) and least in young ones (9 per cent). The mortality rate of females was 62 per cent while that of males was 38 per cent. The mortality showed its peak in $4^{th}$ quarter (35 per cent), followed by $3^{rd}$ quarter (32 per cent), and least mortality occurred in $1^{st}$ quarter (12 per cent); (Table 2). The main lesion observed was enteritis (62 per cent), followed by pneumonia (29 per cent).

### 4.1.2. Pulmonary lesion

Out of 448 cases pulmonary lesions of varying intensity were present in 63 per cent cases. Pneumonia was observed in 29 per cent cases. In 16 per cent cases pulmonary congestion was noticed. In 11 per cent cases oedema was seen as gross lesion, while oedema along with congestion were observed in five per cent cases. Pulmonary emphysema and pulmonary collapse were observed in one percent cases each.

### 4.1.3. Lesions of Intestinal Tract

Seventy five percent cases showed various pathological lesions in intestinal tract. Among the conditions affecting intestinal tract the major lesion observed was catarrhal enteritis which was present in 62 per cent of the cases. Chronic enteritis characteristic of Johne's disease was observed in five cases. Among the parasitic conditions coccidiosis was seen in 31 cases. In 12 cases

there were lesions of pimply gut. Cestodiasis was present in eight cases. Intussuception of intestinal tract was noted in one case.

## 4.2. PATHOLOGY OF THE LUNG AND INTESTINE

In the present study total 111 cases were examined which included 51 males and 60 females. Among the total cases there were 80 kids, 11 young ones and 20 adults. Eighty five cases were from organised farms while twenty six cases were from homestead farms. The postmortem examination revealed lesions of varying intensity and nature in the lungs and intestine. The lesions were classified based on age (Table 3) and sex (Table 4). The number of cases showing pulmonary and intestinal lesions was found to be more among kids.

### 4.2.1. Pulmonary Lesions

The pulmonary lesions of varying nature were evident in 89 (80.18 per cent) cases.

#### *4.2.1 1. Pulmonary congestion*

Pulmonary congestion varying from mild to severe intensity was present in 72 per cent of cases. In some cases congestion was present along with pulmonary oedema (Fig. 1). Congested lung appeared red to dark red in colour. Microscopically pulmonary artery, arterioles, pulmonary vein, peribronchial capillaries and alveolar capillaries were found to be engorged with blood (Fig. 2).

#### *4.2.1.2. Pulmonary haemorrhage*

Haemorrhage in the lung parenchyma was seen in three per cent cases. One case showed petechiae on the apical lobe which appeared as bright red spots on the sub-pleural surface. In other two cases the haemorrhages appeared as patchy reddish areas with irregular outlines. Microscopically there was

extravasation of erythrocytes into the alveolar space and intersitium as well as in to the sub-pleural tissue.

#### *4.2.1.3. Pulmonary oedema*

Pulmonary oedema was seen in thirty seven per cent cases and grossly the lung appeared wet and heavy. Fluid oozed out from the cut surface. On incision, trachea, bronchi and all the airways revealed frothy exudates in the lumen. Histologically oedema appeared as homogenous pink staining fluid in the alveoli as well as in the interstitium and the bronchioles (Fig. 3). The colour of the oedema varied from pale to dark pink.

#### *4.2.1.4. Pulmonary atelectasis*

Seven percent cases revealed pulmonary collapse which appeared as patchy depressed areas with dark colour and firm consistency. On incision the area was seen extending from the pleural surface deep into the parenchyma. Microscopically the alveolar walls were found to be in close apposition, giving a cleft-like appearance. Alveolar lumen was found to be greatly reduced in these areas (Fig. 4).

#### *4.2.1.5 Pulmonary emphysema*

Thirty two per cent of cases showed lesion of emphysema with other changes such as pneumonia and congestion. The emphysematous area appeared pale and distended (Fig. 5). Histologically emphysema was characterised by formation of macro alveoli. Thinning of inter-alveolar septa were observed along with widening of the lumen and flattening of lining epithelial cells (Fig. 6). The bronchi were dilated in some cases.

### *4.2.1.6. Pneumonia*

Twenty cases revealed various pneumonic changes in lung parenchyma. These lesions were generally confined to the apical and cardiac lobes. Two cases showed pneumonic lesions in diaphragmatic lobe. The type of pneumonic lesion observed could be classified into suppurative pneumonia six cases (30%), interstitial pneumonia five cases (25%), fibrinous pneumonia five cases (25%), haemorrhagic pneumonia and aspiratory pneumonia two cases(10%) each.

#### *4.2.1.6.1. Suppurative pneumonia*

Suppurative pneumonia was observed in six cases. Grossly the affected part exhibited various degrees of consolidation (Fig. 7). Both red and grey hepatisation was noted in the same lung occasionally. The cut surface was coarsely granular and bronchi contained small quantity of mucopurulent exudate. Microscopically suppurative type of lesion was charecterised by bronchiolitis and alveolitis along with exudates containing neutrophils (Fig. 8). In advanced cases there was destruction of alveolar stroma. Two cases revealed darkly stained areas of dystrophic calcification in lung parenchyma. One goat with Caseous lymphadenitis showed micro abscesses in the lung parenchyma (Fig. 9). One case revealed cartilagenous metaplastic changes in the lung parenchyma (Fig.10).

#### *4.2.1.6.2. Fibrinous pneumonia*

Fibrinous pneumonia was noticed in five cases. The affected portion of the lung showed a characteristic marbled appearance due to accumulation of fibrinous exudates in the interlobular septa. The consolidated area was firm and dark brown in colour. Two cases showed fibrinous coagulum on the surface of lung along with fibrin deposit on the pleural surface (Fig. 11). Microscopically the alveoli, bronchioles and smaller bronchi were filled with homogenous pink staining exudates rich in neutrophils and fibrin. Vessels were severely engorged. Some capillaries showed fibrin plug in the lumen. Infiltration of leukocytes

caused thickening of interlobular septa (Fig. 12). In two cases there was marked fibrous tissue proliferation in the peribronchial area as well as in interlobular septa. Peribronchial accumulations of lymphocytes were observed in three cases.

*4.2.1.6.3. Interstitial pneumonia*

Five lungs showed the lesions of interstitial pneumonia. Grossly the lungs were voluminous, rubbery in consistency and bluish red in colour and frothy exudate was observed on squeezing (Fig. 13). Microscopically there was thickening of alveolar septa due to infiltration and hyperplasia of mononuclear cells and proliferation of type II pneumocyte in the alveolar septa along with engorgment of alveolar capillaries (Fig. 14). Mononuclear cell infiltration was also observed in perivascular and peribronchial region. In two cases there were lesions of bronchopneumonia along with interstitial changes. There were focal proliferation of the bronchial epithelium and presence of inflammatory exudates in the bronchial lumen.

*4.2.1.6.4. Haemorrhagic pneumonia*

Two cases showed haemorrhagic pneumonia with focal areas of brick red consolidation along with petechial haemorrhage on subpleural surface (Fig. 15). The lesion was present on cardiac lobe in both the cases. The bronchial lumen contained large quantity of blood tinged froth. Microscopically the lung alveoli were filled with erythrocytes and few neutrophils along with oedma fluid (Fig.16).

*4.2.1.6.5 Aspiration pneumonia*

Aspiration pneumonia was noticed in two cases. Grossly congestion and consolidation of apical, cardiac and anterior portion of diaphragmatic lobes were observed. The unaffected part of the lungs showed emphysematous changes (Fig. 17). Trachea and bronchi were filled with catarrhal exudates mixed with ruminal contents. Microscopically bronchi and alveoli showed oedema along with

neutrophils. In one case there was cross section of plant ingesta in the lumen of large bronchi (Fig. 18).

### *4.2.1.7. Lesions of lymphnode*

The bronchial and mediastinal lymphnodes of pneumonic animal exhibited various degrees of enlargement along with congestion. Microscopically four cases revealed depletion of lymphocytes along with necrotic changes in the cortical area (Fig. 19).The lymphnode of the goats with haemorrhagic pneumonia showed haemorrhagic lesion in the cortex (Fig. 20). One case revealed lesion of caseous lymphadenitis in mediastinal lymphnode, which was filled with semisolid pus (Fig. 21) and *Corynebacterium* sp. were isolated from the pus material collected. Microscopically multiple caseous areas along with polymorphonuclear cell infiltration were present in the lymphnode (Fig. 22).

### *4.2.1.8 Lesions of bronchi and bronchioles.*

The bronchi and bronchioles revealed mild to severe pathological alteration. In three cases there was desquamation of epithelial cells into the lumen along with hyperplasia of epithelial lining. In two cases the lumen of bronchi were found to be almost obliterated with desquamated cells, inflammatory cells and exudate (Fig. 23). In one case proliferation of the bronchial epithelium appeared as papillary projection in to the lumen (Fig. 24). Three cases showed peribronchial accumulation of lymphoid cells (Fig. 25). Suppurative pneumonic cases revealed mild cellular infiltration to frank suppuration in bronchial and bronchiolar structure. In one case lumen of bronchi showed hyaline formation adhering to the lining epithelium (Fig. 26).

### *4.2.1.9 Lesions of pleura and thorax*

In three percent of the cases there was hydrothorax characterised by accumulation of moderate amount of clear fluid in the thoracic cavity. One case showed adhesion of the lung to the dorsal thoracic wall. Two cases showed

congestion and thickening of pleural membrane. Histologically pleura were thickened due to fibrin deposition along with inflammatory cell infiltration (Fig. 27). The inflammatory changes extended into the parenchyma with thickening of interalveolar septa due to inflammatory cell infiltration and fibrin deposition. The bronchi also revealed inflammatory exudates with desquamated epithelial cells.

### 4.2.2. Lesions of Intestinal Tract

Out of 111 cases 80 (72%) cases showed various pathological lesions in intestinal tract. The lesions were mainly located in small intestine, especially duodenum and jejunum.

#### *4.2.2.1. Small Intestine*

##### *4.2.2.1.1. Congestion*

Fifty six per cent showed congestion of varying degrees in mucosa, serosa as well as mesenteric vessels. Congestion was more prominent in duodenum and jejunum. Grossly mucosa appeared red (Fig. 28). Microscopically blood vessels in the mucosa, submucosa and serosa were engorged with erythrocytes (Fig. 29).

##### *4.2.2.1.2. Degeneration and Necrosis*

Sixty seven per cent of cases showed degeneration and necrotic changes of the surface epithelium of small intestine. Duodenum showed degenerative and necrotic changes mainly affecting the surface epithelium of mucosa. In the mucosa there was degeneration of epithelium lining the villi as well as the cells of crypts of Lieberkuhn. The degenerated epitheliums were denuded into the lumen (Fig. 30). In some area the villi appeared flattened, stunted and fused. In some cases Brunner's gland showed degenerative changes in the epithelium lining the lumen. Few cases revealed moderate oedema of submucosa. Five cases revealed villous atrophy along with fusion, leading to reduction of villous height

to crypt depth ratio. Two cases showed presence of intranuclear inclusion bodies in the epithelium at the tip of the villi along with lytic changes (Fig. 31).

In jejunum the microscopic lesion varied from simple vacuolar degeneration of the villous epithelium to complete loss and necrosis of the enterocytes of the villi (Fig. 32). Some cases showed degenerative and necrotic changes in crypts of lieberkuhn along with cystic dilatation of crypts with loss of lining cells (Fig. 33). In two cases the intestinal villi were acellular with faded stroma and at places it revealed polypoid projection into the lumen.

In ileum there was shedding of the epithelium covering the villi and degeneration of the cells of crypts of Lieberkuhn. Twenty percent cases revealed depletion of lymphocytes in the Peyer's patches (Fig. 34). In certain other cases the follicles in Peyer's patches had degenerative and necrotic changes (Fig. 35).

*4.2.2.1.3. Ulcers*

One case showed multiple ulcers in the jejunum with raised borders. Microscopically there was complete loss of mucosal and submucosal layer exposing the muscular layer. Villi in the surrounding area showed degenerative and necrotic changes along with inflammatory cell infiltration (Fig. 36).

*4.2.2.1.4. Catarrhal Enteritis*

Catarrhal enteritis in small intestine was seen in thirty two per cent cases. Grossly rough tenacious mucus was found adhered to the mucosa and in some cases intestinal contents were mixed with mucus. The mucosa was hyperaemic and oedematous which occurred as diffuse or as patchy zones (Fig. 37). Microscopically there were desquamation of epithelium along with hyperaemia and oedema of lamina propria. There were hydropic degeneration and increased goblet cell activity. Neutrophils were the characteristic infiltrate along with small number of mononuclear cells (Fig. 38). The crypt cells in some cases appeared

degenerated and desquamated. Some cases revealed neutrophilic infiltration in periglandular area (Fig. 39).

*4.2.2.1.5. Subacute Enteritis*

Subacute enteritis was seen in twenty three per cent cases. Grossly there were hypraemic changes in the mucosa. Microscopically there was degeneration and desquamation of lining epithelium of villi and crypts. Mucosal blood vessels were engorged. Various degrees of mononuclear cell infiltration were evident in lamina propria and villous epithelium (Fig. 40). In few cases the infiltrating cells were predominated by eosinophils along with small number of lymphocytes and plasma cells (Fig. 41).

*4.2.2.1.6. Haemorrhagic enteritis*

Two cases showed lesion of haemorrhagic enteritis. The mucous membrane grossly appeared swollen with haemorrhagic foci. Microscopically it was characterised by exudates containing very few leukocytes and erythrocytes in lamina propria (Fig. 42).

*4.2.2.1.7. Parasitic enteritis*

Eleven kids showed lesions of coccidial enteritis. The distribution of lesion was more prominent in the jejunum and ileum, characterised by small pin head to millet sized nodules diffusely distributed in the mucosa (Fig. 43). Histologically the nodular area corresponded to the hyperplasic areas of the intestinal epithelium. Hyperplastic changes were observed both in glandular as well as epithelial lining. The epithelial cells and crypt cells were found to contain schizonts along with focal denudation of intestinal epithelium (Fig. 44).

Three animals showed lesions of pimply gut in small intestine. The outer surface exhibited nodules of 3-4 millimeter diameter, which revealed creamy caseous material enclosed in fibrous covering.

Two animals revealed numerous tape worms in the intestine. There was moderate degree of catarrhal enteritis and the tape worm was identified as *Moniezia* sp. The carcasses were emaciated and showed moderate amount of clear fluid in the abdominal cavity.

### *4.2.2.2. Lesions of large intestine*

#### *4.2.2.2.1. Congestion*

Nine per cent cases showed congestion of the mucosa of large intestine, especially caecum and colon. Microscopically the vessels were engorged with erythrocytes.

#### *4.2.2.2.2. Degeneration and necrosis*

Degeneration and desquamation of surface epithelium of large intestine were observed in fourteen per cent cases. Microscopically there were degeneration and shedding of the epithelium covering the gland in certain areas. Some cases showed aggregation of desquamated epithelium in the lumen.

#### *4.2.2.2.3. Catarrhal colitis*

Eight per cent cases revealed catarrhal colitis characterised by moderate amount of mucus in the lumen. Microscopically increased goblet cell activity, congestion of the vessels of lamina propria and mild mononuclear cell infiltration were observed.

#### *4.2.2.2.4. Necrotic colitis*

Necrotic colitis was noticed in one case. There was complete destruction of glandular epithelium, along with extensive inflammatory cell infiltration in the mucosa. The mucosa revealed presence of eosinophilic necrotic masses on the surface (Fig. 45).

*4.2.2.2.5. Caecal impaction*

Sixteen kids revealed distended caecum with partially digested milk which appeared as pasty creamy material filling the lumen. The abomasum of these kids revealed curdled and clotted milk adhering to the abomasal mucosa. There was severe congestion of mesenteric vessels. *Escherichia coli* were isolated from lungs, liver and intestine of those kids. Microscopically there was no specific lesion except hyperactive goblet cells in the intestinal mucosa.

*4.2.2.2.6. Lesions of mesenteric lymphnodes*

Mesenteric lymphnode in some animals with enteritis were swollen and odematous. Varying degrees of lymphocytic depletion was the most frequent lesion observed. Oedema and congestion were observed in most of the cases. Three cases revealed hyperplasia of lymphoid cells in cortical area (Fig. 46). Diffuse degeneration and necrosis of lymphocytes in the cortical region along with thickening of capsule were observed in nine cases (Fig. 47). Two cases revealed infiltration of eosinophils into the medullary region (Fig. 48).

**4.2.3. Pneumo-enteric lesion**

Twenty cases with pneumonia showed various pathological lesions in intestine also.

Five cases with interstitial pneumonia showed mononuclear cell infiltration in the lamina propria of small intestine, along with degenerative and necrotic changes in mucosal epithelium. Peyer's patches showed mild to moderate degree of lymphoid cell depletion. Large intestine showed moderate degenerative changes in mucosal layer. The crypt glandular epithelium showed lesions ranging from degeneration to necrosis. Bronchial epithelium was hyperplastic and lumen was obliterated with inflammatory exudates including desquamated epithelial cells. Interalveolar septa showed thickening due to septal cell proliferation and inflammatory cell infiltration. Bronchial and mesenteric

lymphnode revealed lymphoid cell depletion and necrotic changes in cortical region. Two cases revealed lesion of broncho-interstitial pneumonia.

Two cases with suppurative lesion in lung showed infiltration of neutrophils in the lamina propria along with small number of mononuclear cells in the small intestine. Degenerative and necrotic changes were observed in mucosal layer of small intestine as well as large intestine.

Three cases of suppurative pneumonia showed moderate degree of mononuclear cell infiltration in the lamina propria along with degenerative changes in the mucosa of small intestine and large intestine.

One case with lesion of fibrinous pneumonia revealed presence of intranuclear inclusion body in the mucosal epithelium of the duodenum undergoing lytic changes.

In nine lungs with various pneumonic lesions, the inflammatory changes in lung were accompanied by lesions such as congestion, degeneration and necrosis in small intestine and large intestine.

## 4.3. MICROBIOLOGICAL STUDIES

Samples for microbiological examination were collected aseptically from lung and intestine of fresh postmortem cases showing lesions. The isolates obtained from cases having lung lesions were *Escherichia coli* (12), *Klebsiella* sp. (3), and *Corynebacterium* sp. (1). The isolates obtained from goats showing enteritis were *Escherichia coli* (11) and *Klebsiella* sp (2).

**Table 1. Year-wise incidence of mortality**

| Year | Total |
|---|---|
| 2002 | 77 |
| 2003 | 49 |
| 2004 | 73 |
| 2005 | 118 |
| 2006 | 131 |
| **Total** | **448** |

**Table 2. Mortality pattern 2002-2006**

| Quarter | Adults | | | Young ones | | | Kids | | | Grand Total | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Male | Female | Total | Male | Female | Total | Male | Female | Total | Male | Female | Total |
| I Quarter (Jan-Mar) | 5 | 16 | 21 | 2 | 3 | 5 | 10 | 17 | 27 | 17 | 36 | 53 (12%) |
| II Quarter (Apr-Jun) | 4 | 42 | 46 | 3 | 6 | 9 | 22 | 16 | 38 | 29 | 64 | 93 (21%) |
| III Quarter (Jul-Sep) | 9 | 53 | 62 | 1 | 15 | 16 | 40 | 27 | 67 | 50 | 95 | 145 (32%) |
| IV Quarter (Oct-Dec) | 4 | 33 | 37 | 5 | 7 | 12 | 63 | 45 | 108 | 72 | 85 | 157 (35%) |
| Total | 22 13% | 144 87% | 166 37% | 11 26% | 31 74% | 42 9% | 135 56% | 105 44% | 240 54% | 168 38% | 280 62% | 448 |

**Table 3. Age wise distribution of lesions in the lung and intestine.**

| Group | Total | No. With Lung Lesion | | No. with Intestine Lesions | |
|---|---|---|---|---|---|
| | | No. | % | No. | % |
| **Kid (1-6 months)** | 80 | 60 | 75 | 61 | 76 |
| **Young ones (6 months - 1 year)** | 11 | 10 | 90.9 | 9 | 81.8 |
| **Adult (above 1 year)** | 20 | 19 | 95 | 10 | 50 |
| **Total** | **111** | **89** | **80** | **80** | **72** |

**Table 4. Sex wise distribution of lesions in the lung and intestine**

| Sex | Total | No. With Lung Lesions | | No. With Intestine Lesions | |
|---|---|---|---|---|---|
| | | No | % | No | % |
| **Male** | 51 | 40 | 78.4 | 42 | 82 |
| **Female** | 60 | 49 | 81.7 | 38 | 63 |
| **Total** | **111** | **89** | **80** | **80** | **72** |

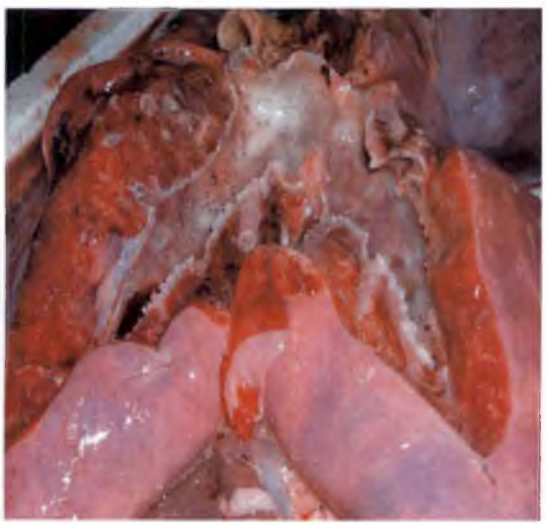


Fig. 1

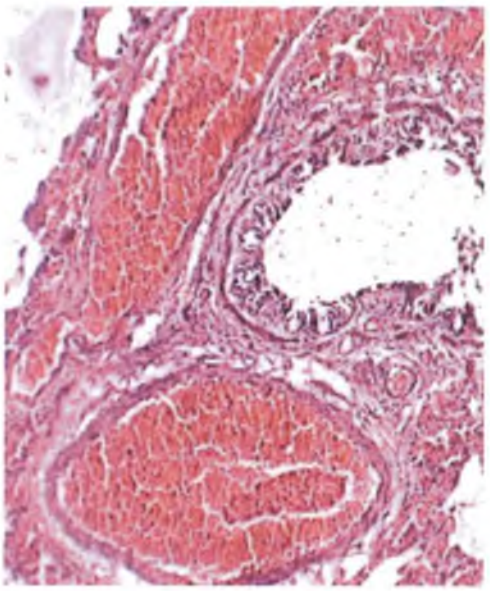


Fig. 2

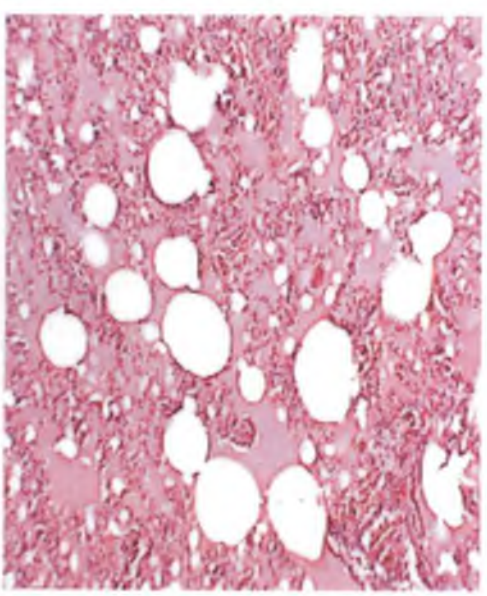


Fig. 3

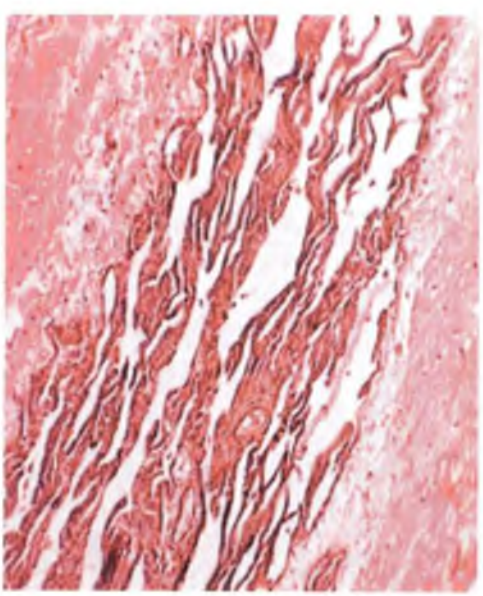


Fig. 4

Figure 1: Pulmonary oedema and congestion

Figure 2: Congestion of peribronchial vessels - H & E x 100

Figure 3: Pulmonary oedema - H & E x 100

Figure 4: Pulmonary atelectasis - H & E x 40

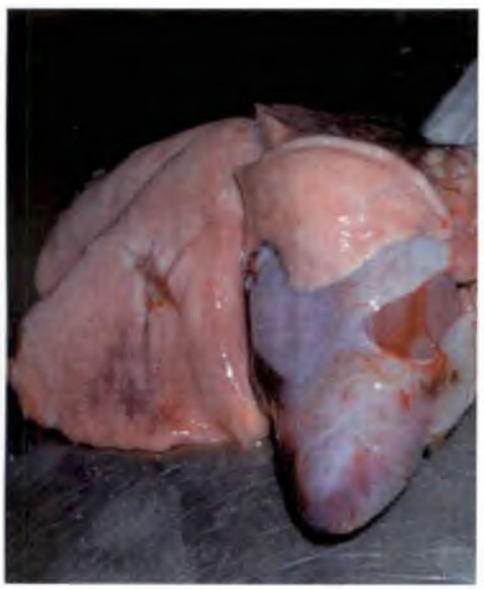
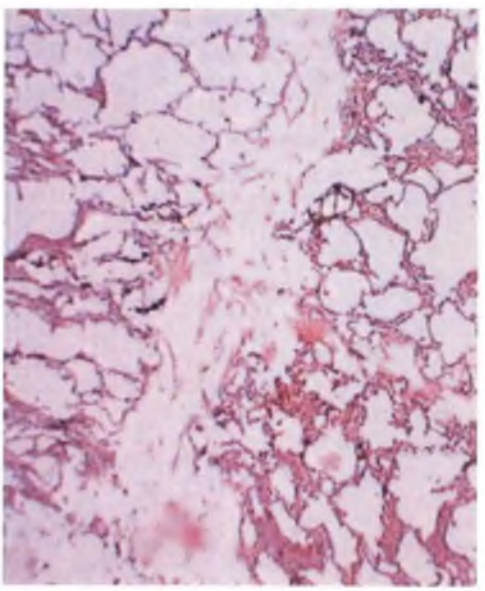


Fig. 5



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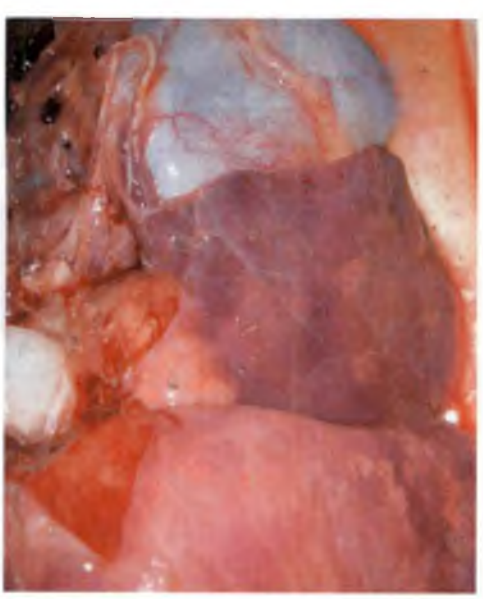

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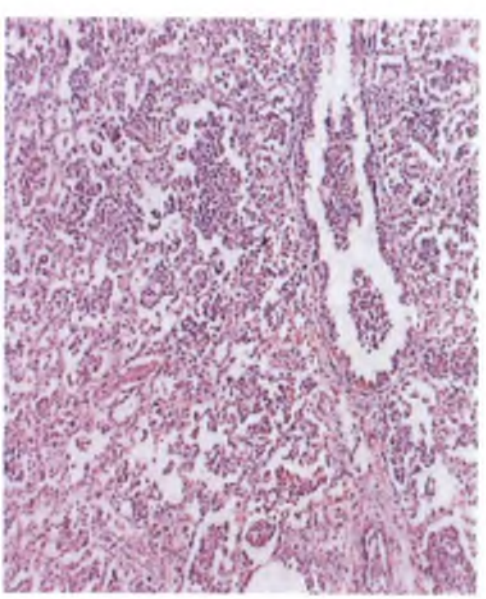


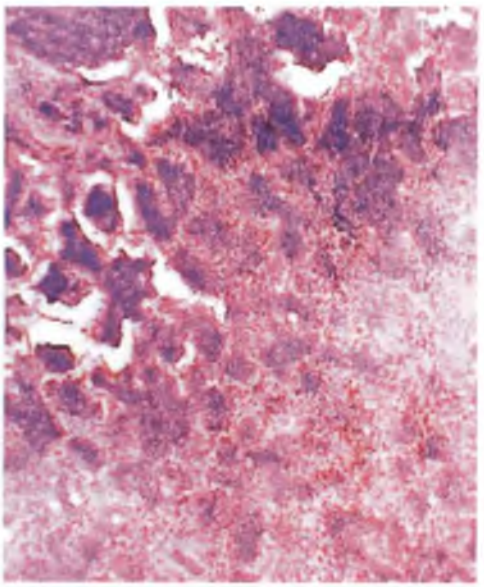
Fig. 8

Figure 5: Pulmonary emphysema

Figure 6: Pulmonary emphysema - Thinning of inter-alveolar septa and widening of lumen - H & E x 100

Figure 7: Lung - Consolidation of cardiac lobe

Figure 8: Suppurative pneumonia with exudate containing neutrophils in bronchiolar and alveolar lumen - H & E x 100



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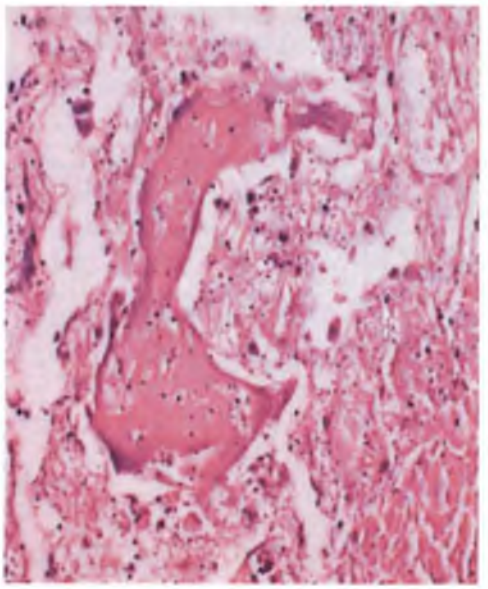

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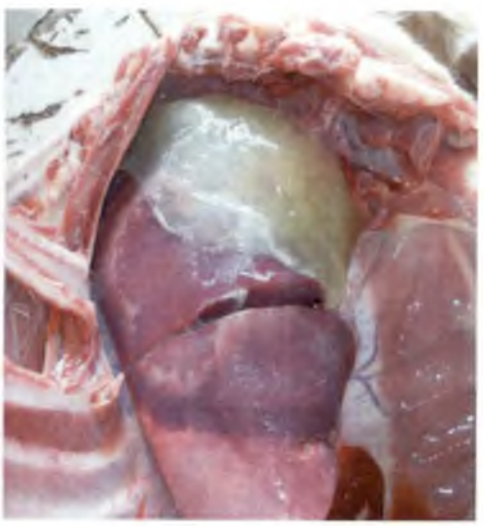

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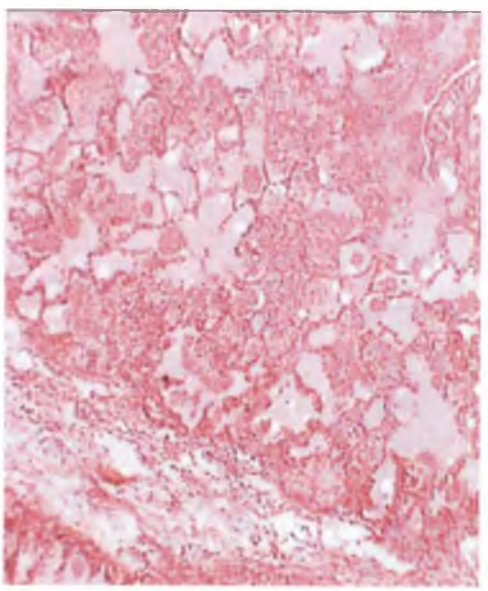


Fig. 12

Figure 9 : Microabscesses in lung parenchyma - H & E x 100

Figure 10: Cartilagenous metaplastic changes - H & E x 400

Figure 11: Fibrinous coagulum on the pleural surface of lung

Figure 12: Fibrinous pneumonia - Fibrinous exudate in the alveoli along with mononuclear and polymorphonuclear cell infiltration - H & E x 100

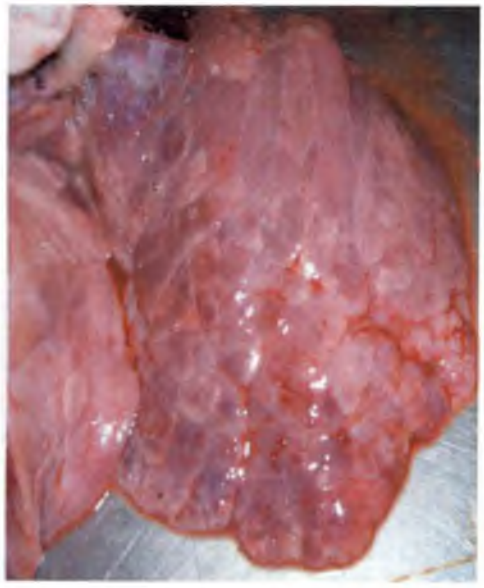
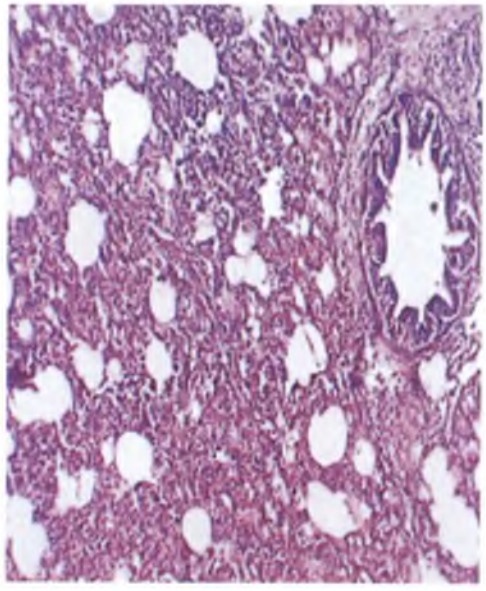


Fig. 13



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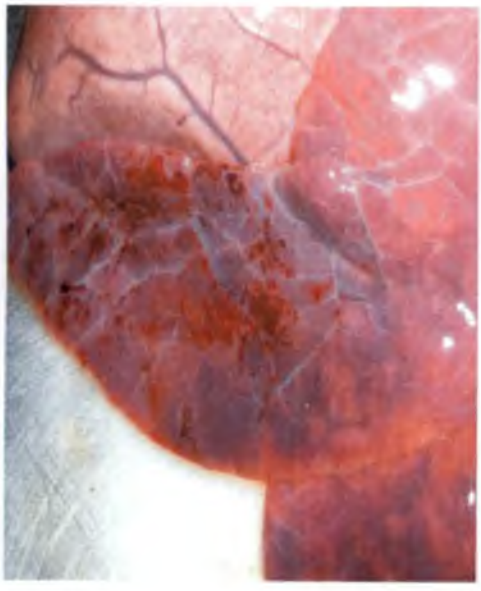

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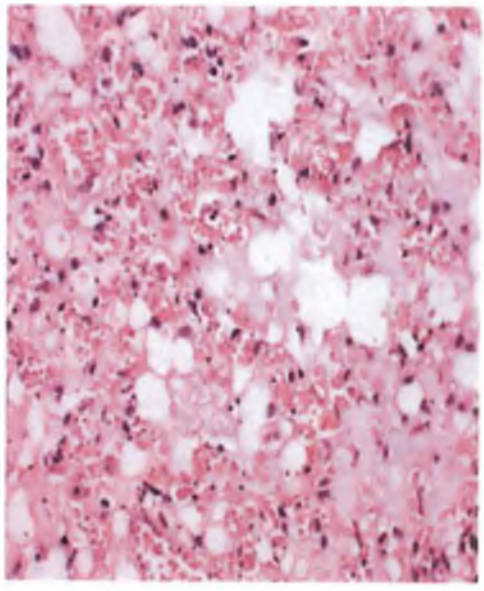


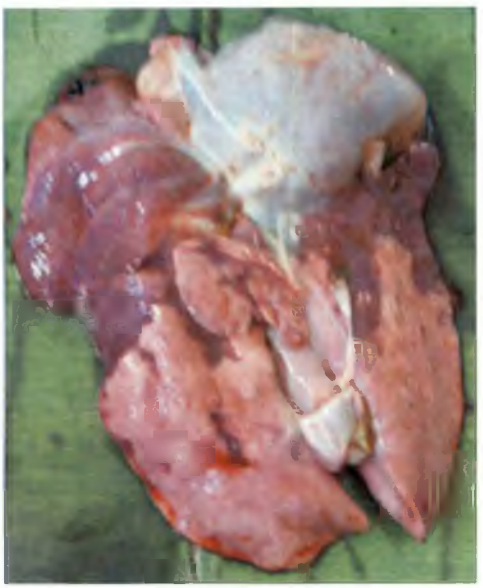
Fig. 16

Figure 13: Interstitial pneumonia - Rubbery lung with bluish red colour

Figure 14: Interstitial pneumonia - Thickening of alveolar septa and engorgement of alveolar capillaries - H & E x 100

Figure 15: Haemorrhagic pneumonia - Brick red consolidation of lung

Figure 16: Haemorrhagic pneumonia - Alveoli filled with erythrocytes, neutrophils and oedema fluid - H & E x 400



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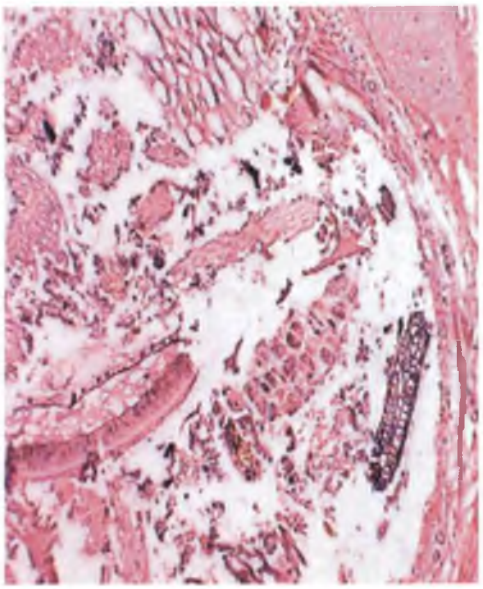

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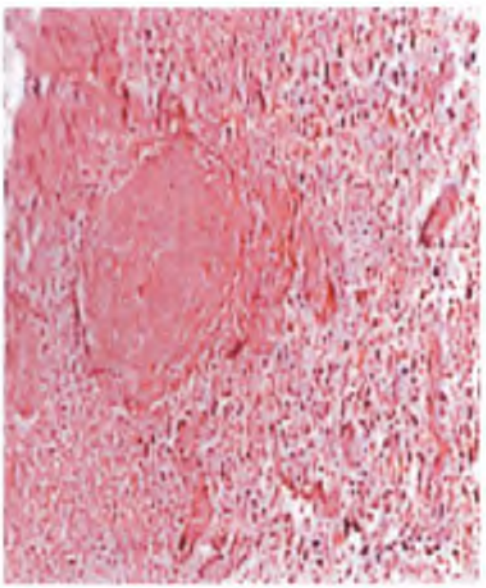

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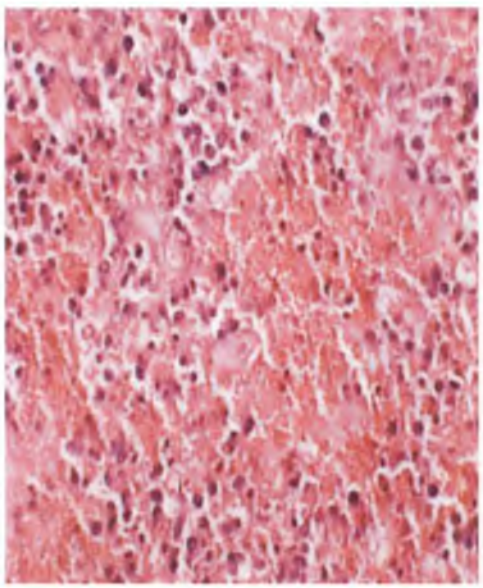


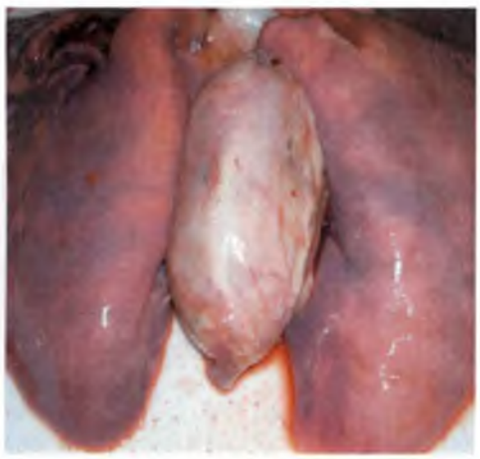
Fig. 20

Figure 17: Aspiration pneumonia - Lung showing consolidation in the apical, cardiac and anterior portion of the diaphragmatic lobes

Figure 18: Aspiration pneumonia - Cross section of plant ingesta in the lumen of large bronchi - H & E x 100

Figure 19: Bronchial lymph node - Depletion of lymphocytes and necrotic change in the cortical area - H & E x 100

Figure 20: Bronchial lymph node - Haemorrhage in cortical area - H & E x 400



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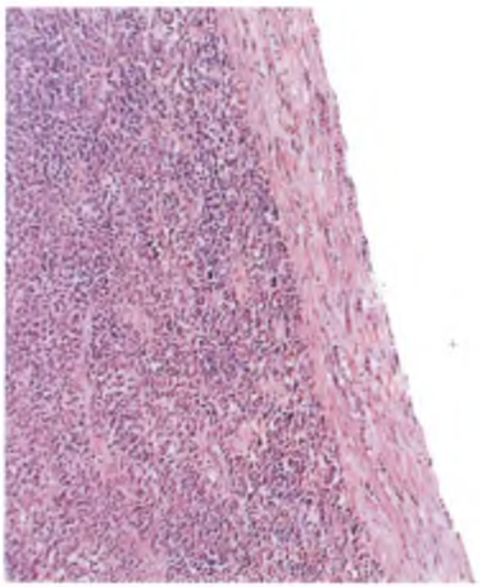

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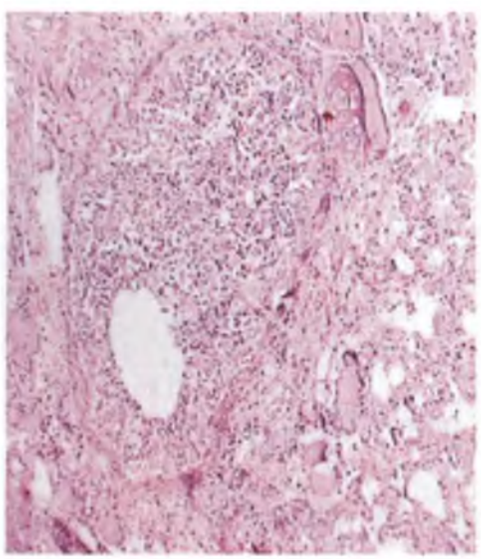


Fig. 23

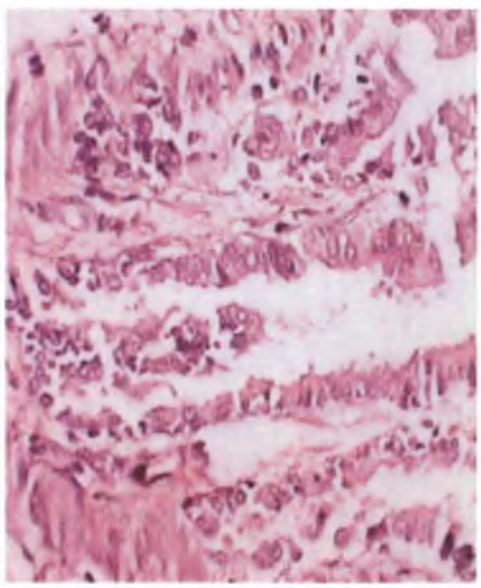


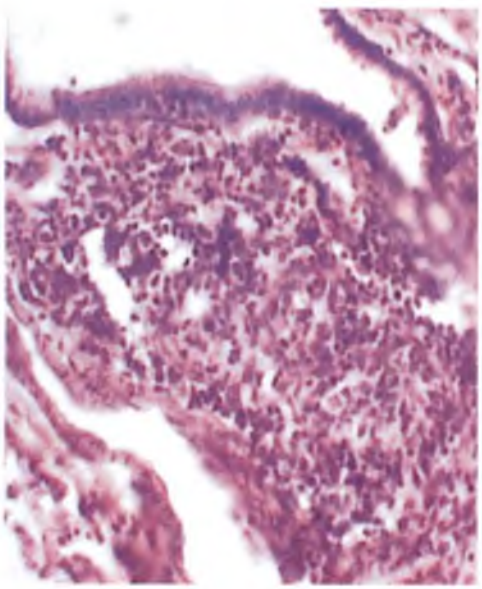
Fig. 24

Figure 21: Mediastinal lymphnode filled with semisolid pus - Caseous lymphadenitis

Figure 22: Mediastinal lymphnode - Multiple caseous area along with polymorphonuclear cell infiltration - Caseous lymphadenitis - H & E x 100

Figure 23: Lumen of bronchi obliterated with desquamated cells and inflammatory cell infiltrates - H & E x 100

Figure 24: Papillary projections into lumen of bronchi - H & E x 400



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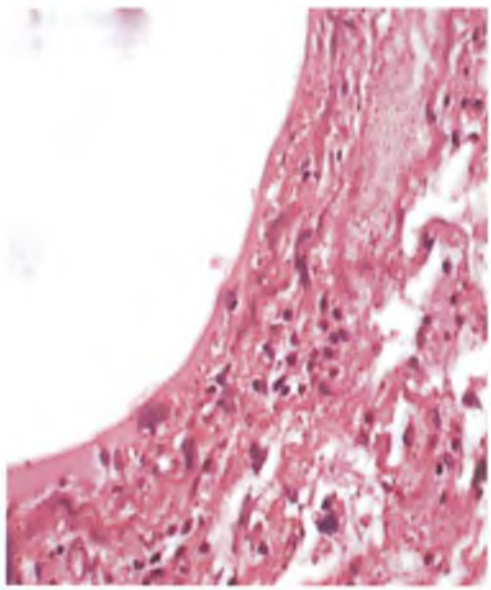

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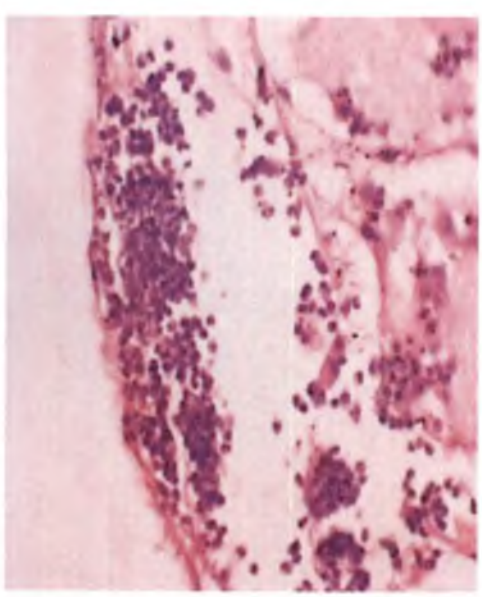

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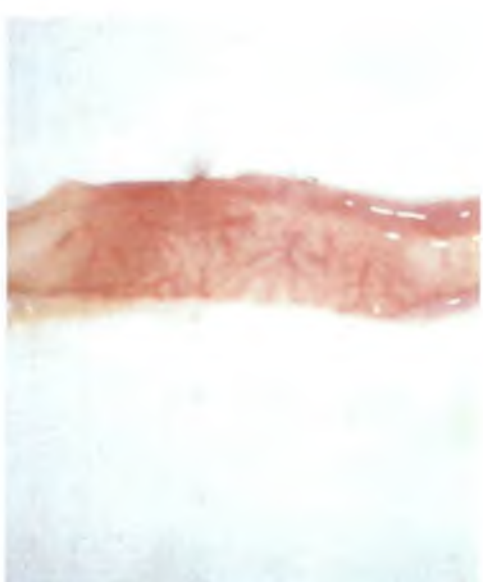


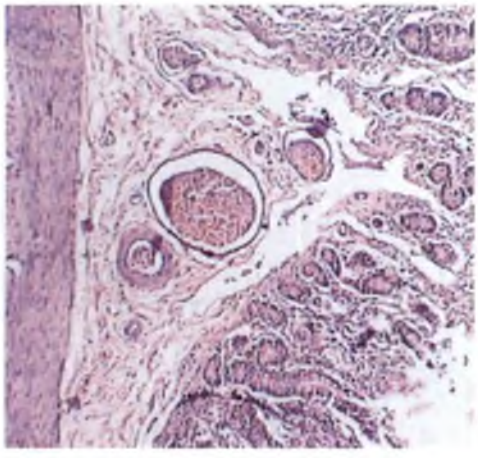
Fig. 28

Figure 25: Peribronchial accumulation of lymphoid cells - H & E x 400

Figure 26: Bronchi showing hyaline formation adhering to lining epithelium - H & E x 400

Figure 27: Mononuclear cell infiltration and fibrin deposition on pleural membrane - H & E x 400

Figure 28: Jejunum - Congestion with reddening of mucosa



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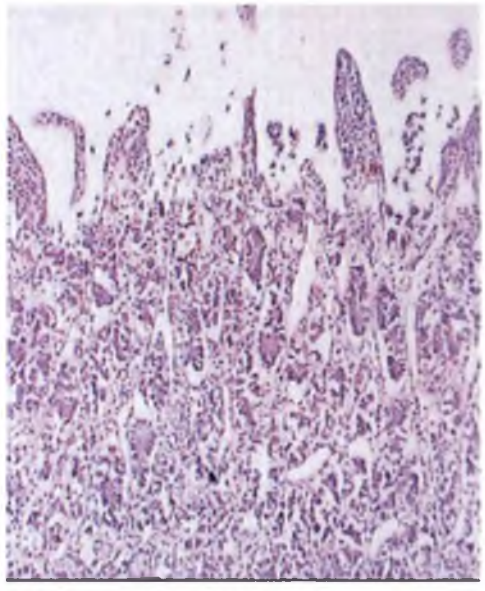

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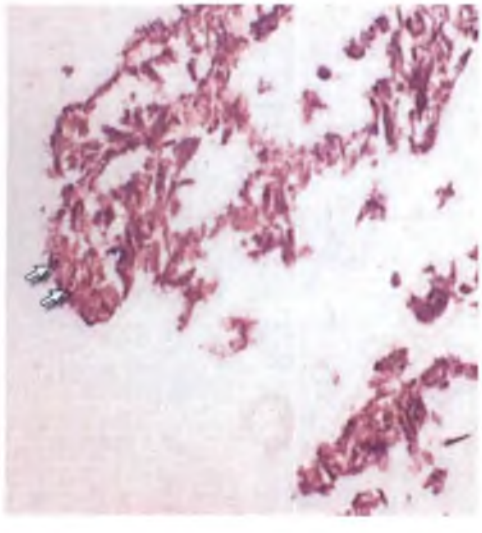

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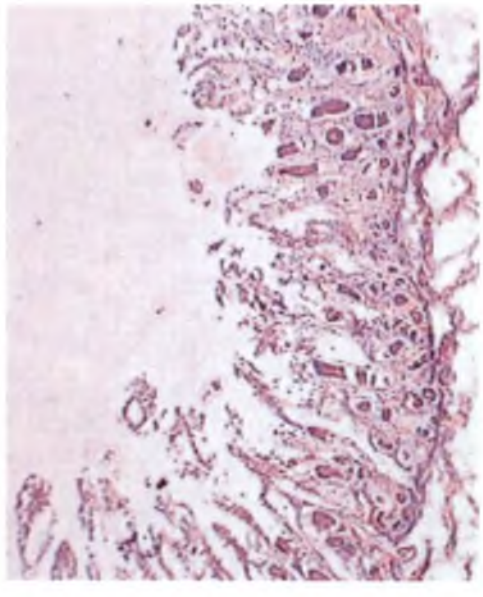


Fig. 32

Figure 29: Jejunum - Congestion of submucosal blood vessels - H & E x 100

Figure 30: Duodenum - Degenerative and necrotic changes in the epithelial lining and cells of crypts of Lieberkuhn - H & E x 100

Figure 31: Duodenum - Intranuclear inclusion bodies in the degenerating epithelial cells at the tip of villi - H & E x 400

Figure 32: Jejunum - Complete loss and necrosis of enterocytes of the villi - H & E x 100

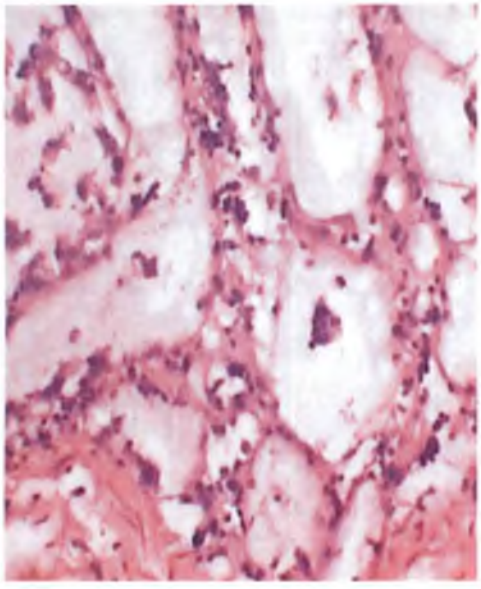
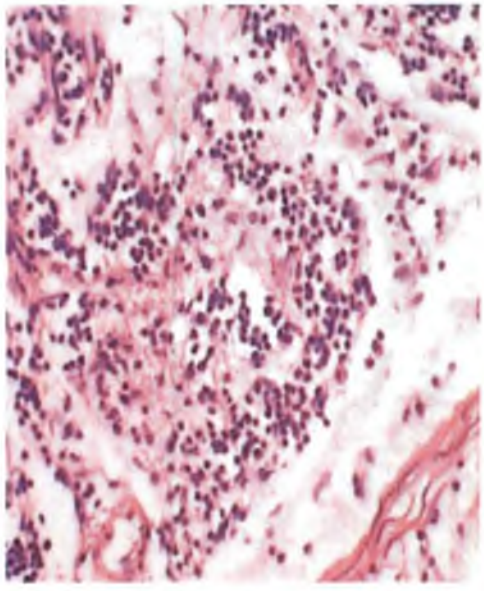


Fig. 33



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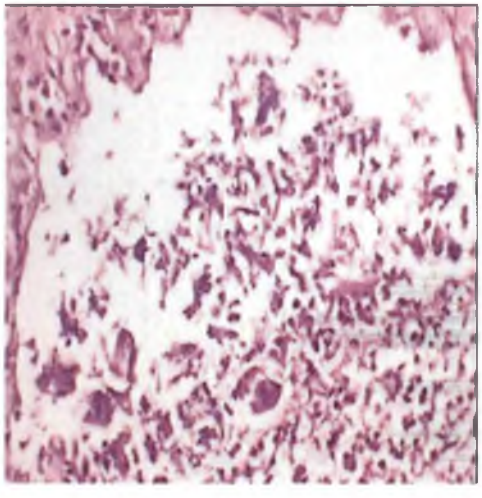


Fig. 35

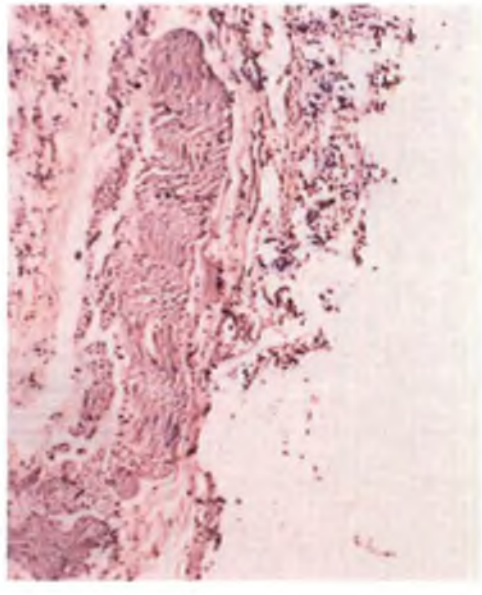


Fig. 36

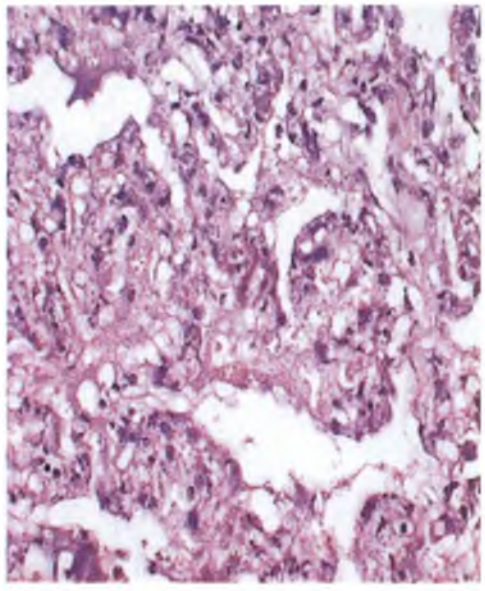
Figure 33: Jejunum - Cystic dilatation of glandular crypts with loss of lining cells - H & E x 400

Figure 34: Ileum - Depletion of lymphoid cells in the Peyer's patches - H & E x 400

Figure 35: Ileum - Degenerative and necrotic changes in Peyer's patches - H & E x 400

Figure 36: Jejunum - Ulcer : Complete loss of mucosal and submucosal layer exposing muscular layer - H & E x 100

Fig. 37



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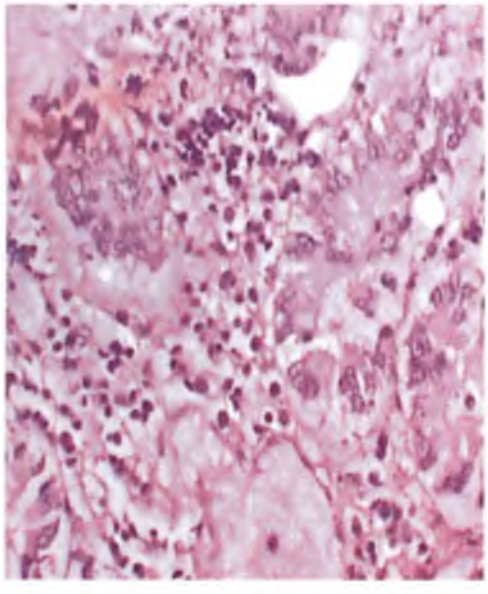

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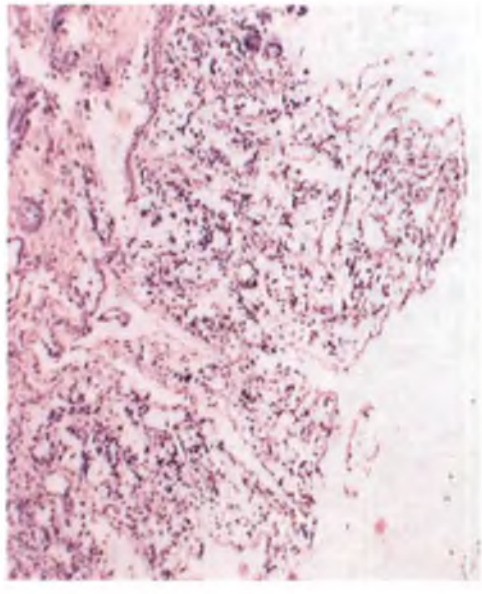


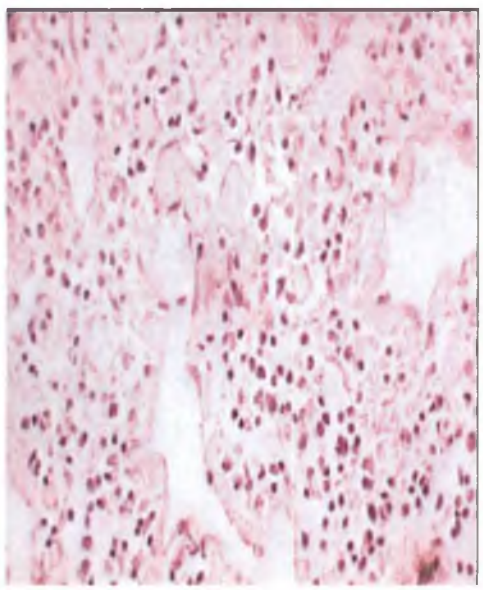
Fig. 40

Figure 37: Duodenum - Catarrhal enteritis : Exudate mixed with intestinal contents along with hyperaemia of mucous membrane

Figure 38: Duodenum - Catarrhal enteritis : Infiltration of neutrophils and hyperactive goblet cells in the villi - H & E x 400

Figure 39: Jejunum - Periglandular infiltration of neutrophils - H & E x 400

Figure 40: Jejunum - Subacute enteritis : Moderate mononuclear cell infiltration in lamina propria and villous epithelium - H & E x 100



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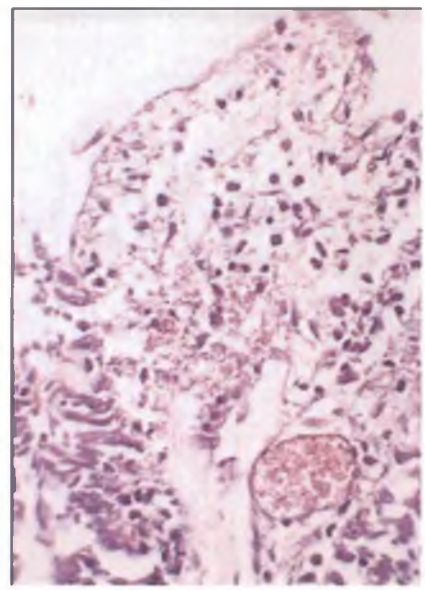


Fig. 42

Fig. 43

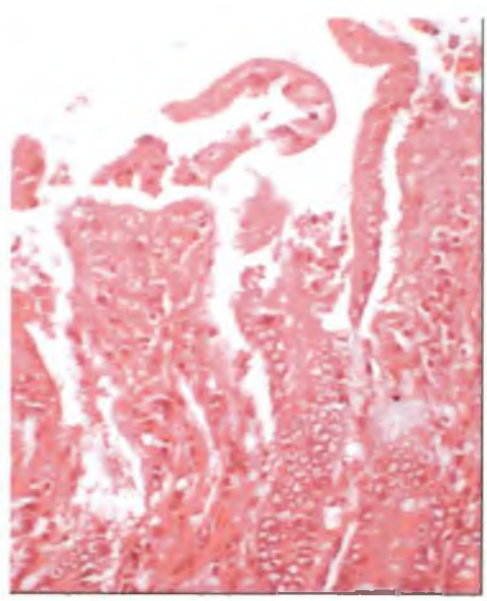


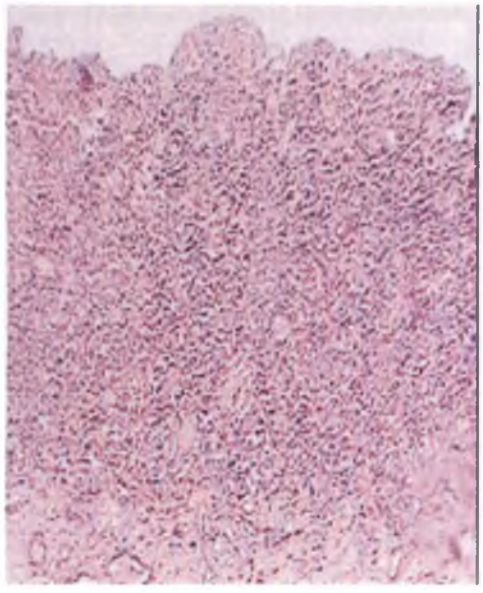
Fig. 44

Figure 41: Ileum - Infiltration of eosinophils and small number of plasma cells - H & E x 400

Figure 42: Jejunum - Haemorrhagic enteritis : Exudate containing few leukocytes and erythrocytes in lamina propria - H & E x 400

Figure 43: Ileum - Coccidial nodules on the mucosal surface

Figure 44: Ileum - Schizonts in the lining epithelial cells - H & E x 400



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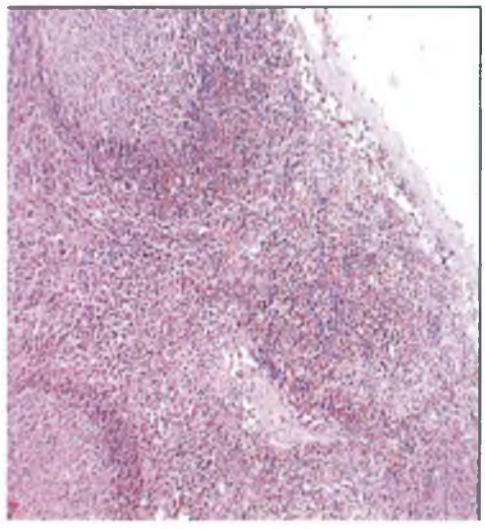

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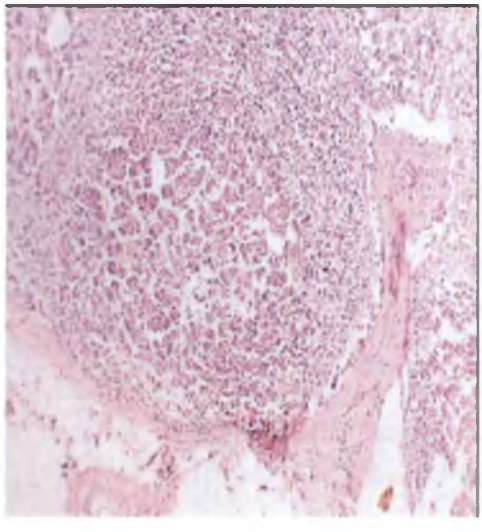

Fig. 47

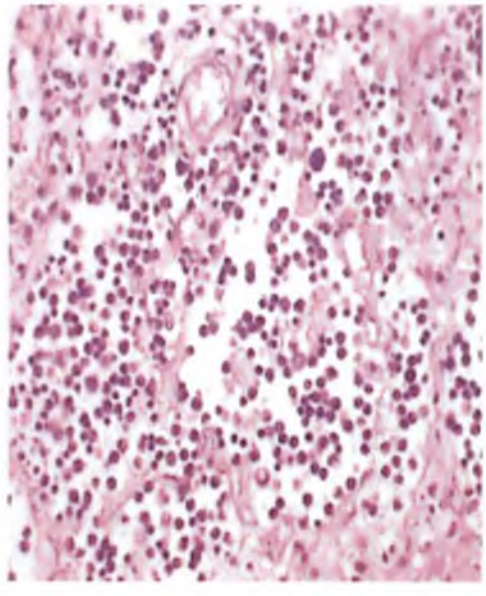

Fig. 48

Figure 45: Colon - Necrotic colitis : Destruction of glandular epithelium and extensive inflammatory cell infiltration into mucosa H & E x 100

Figure 46: Mesenteric lymphnode - Hyperplasia of lymphoid cells in cortical area - H & E x 100

Figure 47: Mesenteric lymphnode - Degeneration and necrosis of lymphoid cells in cortical region - H & E x 100

Figure 48: Mesenteric lymphnode - Infiltration of eosinophils in the medullary region - H & E x 400

# *Discussion*

# 5. DISCUSSION

The present study was under taken with the objective of investigating the pathological conditions affecting the respiratory and digestive system of goats and their mortality pattern. A retrospective analysis of the cases affecting lungs and intestine during the last five years was also been undertaken to study the prevalence.

During the five years of study a total number of 448 cases of goat mortality were recorded. The mortality among kids was higher (53.57%) than adults (37.4%). This observation is in agreement with the findings of Soundararajan *et al.* (2006). But Sriraman *et al.* (1982) observed almost similar mortality rate in kids and adults. The higher mortality rate observed among kids in the present study may be attributed to the low level of immunoglobin in the body of kids due to deprived colostrum feeding, predisposing them to conditions such as colisepticaemia which is prevalent in the farm. Females showed high mortality (62%) rather than males (38%).This observation is in agreement with the finding of Soundararajan *et al.* (2006). They reported higher mortality rate in females compared to males. Season wise incidence of mortality revealed the highest mortality rate during the fourth quarter. Sriraman *et al.* (1982) reported highest mortality in first quarter while Soundararajan *et al.* (2006) observed highest mortality in second quarter. Even though there is no well defined winter season in Kerala, during the period from November to December it is very windy and atmosphere is very dusty in and around Trichur. Added to that the difference between day and night temperature is much higher than other seasons. So it could be considered that these factors act as the main extrinsic component and predispose the goats, especially kids, to infections of various nature. The major lesions observed were enteritis (62.28%) and pneumonia (28.79%). This observation is in agreement with the observations made by Sriraman *et al.* (1982) and Soundararajan *et al.* (2006). Analysis of the data for the five year period collected from the autopsy records revealed 63.17 per cent occurrence of various

pulmonary lesions and 75 per cent of various intestinal lesions. On the other hand the present investigation revealed 80 per cent incidence of pulmonary lesions and 72 per cent incidence of intestinal lesion which clearly indicates the increase in the occurrence of pneumo-enteric disorders compared to the past.

The various pulmonary and intestinal lesions observed in the present study were classified based on the gross and histopathological observations. The frequently encountered lesions of lungs in present study were pneumonia, congestion, oedema, haemorrhage, emphysema, and collapse. The lung lesions were more prominent in apical and cardiac lobes.

Congestion of mild to moderate degree was present in 72 per cent cases. 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. Pulmonary congestion in goats were recorded in various conditions like, *Mycoplasma* infection (Batra *et al.*, 2000), combined infection of *Mycoplasma* and *Pasteurella* (Batra *et al.*, 2002), *E. coli* infection (Sharma *et al.*, 2003) and PPR (Pawaiya *et al.*, 2004).

Three per cent cases revealed hemorrhages in the lung parenchyma. Pulmonary hemorrhage was reported in various conditions like combined infections of *Mycoplasma* and *Pasteurella* (Batra *et al.*, 2002), in bluetongue (Sivaseelan, 2003), *E. coli* infection (Sharma *et al.*, 2003) and PPR (Pawaiya *et al.*, 2004).

Several cases revealed the presence of frothy exudates in trachea, bronchi and bronchioles indicating pulmonary oedema. Dungworth (1993) reported that foam mixed with fluid would be often present in the trachea and intrapulmonary airways in severe cases of pulmonary oedema which was frequent complication of many diseases. Pulmonary oedema was reported for conditions such as *Mycoplasma* infection (Batra *et al.*, 2000) and PPR (Pawaiya *et al.*, 2004). The oedema was noticed as pink staining fluid in the alveoli. The intensity of the

staining was deeper in cases where evidence of inflammatory changes was present.

Twenty cases showed pneumonic lesions in the lung parenchyma and were prominent in apical and cardiac lobes. This is in consonance with the observations made by Manomohan (1980) in kids and Sasani *et al.* (1998) in sheep.

Suppurative pneumonia was observed in six cases. Grossly there was patchy area of consolidation. Microscopically consolidated area showed bronchiolitis, alveolitis along with exudates containing neutrophils. The gross and histopathological findings agree with observations made by earlier workers (Bhagwan and Singh, 1972; Manomohan, 1980; Nair, 1982; and Shivarudrappa *et al.,* 2004). The massive suppurative lesions suggest bacterial etiology. Although suppurative pneumonia can be seen as secondary infection to viral pneumonia, there was no indication of viral induced pneumonic changes in any of these cases examined.

Fibrinous pneumonia was reported in five cases. Grossly lung showed marbled appearance. Microscopically fibrinopurulent exudates were present in lumen of alveoli and bronchi. Similar observation was made by Nair (1982) and Shivarudrappa *et al.* (2004). Ramachandran and Sharma (1969) observed fibrinous pneumonia due to *Pasteurella* species in sheep and goats. Wesonga *et al.* (2004) observed fibrinous pleuropneumonia with septal and peribronchial fibrosis in goat infected with contagious caprine pleuropneumonia. Samanta *et al.* (2006) observed fibrinous pneumonia in *Plesiomonas shigelloides* infection in lambs. Pulmonary fibrosis has also been observed in few cases, where the fibrous tissue proliferation was marked in the peribronchial and the interlobular septa. Though *Mycoplasma* organisms could not be isolated, the involvement of *Mycoplasma* cannot be ruled out without more effort on isolation. Pulmonary fibrosis may also suggest role of unknown immunological reactions against

altered self antigen triggering inflammatory change leading to thickening of alveolar septa.

Five cases showed lesions of interstitial pneumonia. Grossly, lungs were voluminous and microscopically it was characterised by thickening of alveolar septa due to mononuclear cell infiltration, proliferation of type II pneumocytes and engorgement of alveolar capillaries. The bronchial epithelium showed focal to diffuse proliferative changes. The histological changes were mainly of proliferative nature in both alveolar septa and bronchi. The proliferative type of lesions observed were in consonance with the reports of Sharp and Nettleton (2000), Pawaiya *et al.* (2004) and Abraham *et al.* (2005) in viral type of pneumonia. The proliferative changes also suggest a chronic irritation of the air passages by environmental pollutants and allergens. The findings of lymphocytic reaction and proliferation of type II pneumocytes may be due to acute viral pneumonitis. However definite inclusion bodies could not be detected in alveolar or bronchial epithelium. Though virus could be the etiological factor it may not be possible to detect inclusion bodies if the examination is not done at the acute stage of the diseases or sometimes the secondary infection by bacteria may mask the primary lesion. In two cases there were lesions of bronchopneumonia along with interstitial changes. The presence of histological features of both bronchopneumonia and interstitial pneumonia suggest the classification of lesion into broncho interstitial pneumonia where a combined effect of virus and the secondary bacterial infection is suspected (Lopez, 1995).

Haemorrhagic pneumonia was present in two cases. Focal areas of brick red consolidation were observed grossly. Histologically the lung alveoli were filled with erythrocytes and neutrophils. Similar observation was also made by Bhagwan and Singh (1972). Nair (1982) observed the similar pattern of gross and microscopical features and opined these lesions as pathological change caused by *Pastuerella* species in goats.

Aspiration pneumonia was present in two cases and the gross and histopathological lesions observed closely resembled those described by Nair (1982). The distribution of the lesion as well as presence of foreign material in the lung tissue was confirmatory for the diagnosis of aspiration pneumonia.

Three per cent cases revealed moderate amount of clear fluid in thoracic cavity. Sastry (2001) documented that presence of fluid in the thoracic cavity can occur in case of systemic infection due to increased permeability of the capillary endothelium. Wesonga *et al.* (2004) and Shiferaw *et al.* (2006) reported marked accumulation of serous fluid in pleural cavities along with fibrinous pleural pneumonia in CCPP. Sasani *et al.* (1998) observed few cases of pneumonia in sheep accompanied by chronic inflammatory changes in pleura. The lesions need to be studied along with effort to isolate the organism, especially with emphasis on *Mycoplasma* organism.

Lesions like congestion, oedema, haemorrhage, lymphoid cell depletion and necrotic changes were observed in bronchial and mediastinal lymphnodes. Sasani *et al.* (1998) reported swelling of mediastinal lymphnodes in 50 per cent of pneumonic cases in sheep. Nair (1982) observed congestion, oedema, haemorrhage, lymphoid cell depletion and hyperplastic reaction in bronchial lymphnode of pneumonic goats. Lymphoid congestion and depletion suggested that the immune system of those animals was weak. One case showed lesion of caseous lymphadenitis in mediastinal lymphnode filled with semisolid pus, indicating the infection by *Corynebacterium* sp. which agrees with several earlier findings (Guthery and Beasom, 1979; Singh *et al.*, 1995b; Karimi *et al.*, 2003).

The major changes observed in bronchi and bronchioles were degeneration and desquamation of lining epithelial cells along with hyperplasia of neighbouring cells. In one case the hyperplastic changes led to the formation of papillary projections into the lumen. Inflammatory cells composed of neutrophils and lymphoid cells were also present along with the desquamated and degenerated cells. Similar findings were reported in bronchopneumonia by

Dungworth (1993) and Manomohan (1980) in kids. Pawaiya *et al.* (2004) observed similar changes in bronchi and bronchioles of goat infected with PPR virus. Sharp and Nettleton (2000) observed hyperplastic change in the bronchiolar epithelium of sheep lung affected with parainfluenza viral infection. Sivarudrappa *et al.* (2004) reported mucopurulent exudates in the bronchi along with proliferative changes of bronchiolar epithelium in suppurative bronchopneumonia in goats. The regenerative process to replace the desquamated epithelium might be the cause for the hyperplastic changes observed in the present cases.

Out of 111 cases 80 showed various pathological lesions in intestinal tract. The lesions were mainly located in small intestine especially duodenum and jejnum. The number of cases with intestinal lesions was found more among kids. The distribution of lesion was almost equal in males (42 cases) and females (38 cases). Degeneration and necrotic changes were predominated, followed by vascular and inflammatory changes which could be associated with hypoxic injuries, infection and toxicities. In all cases the degenerative and necrotic changes were predominant in tunica mucosa involving the epithelium and glands.

The lesion in small intestine included congestion, degeneration and necrosis, ulceration and inflammatory changes. Among the inflammatory changes catarrhal inflammation predominated in all the segments, followed by subacute enteritis. Most of the cases showed degeneration and necrotic changes in surface epithelium of small intestine. Degenerated and necrotic cells were desquamated into the lumen in some cases. Some cases revealed villous atrophy and their fusion. Degenerative and necrotic changes in intestine were recorded in various conditions like PPR (Bundza *et al.*, 1988 and Abraham *et al.*, 2005), adeno viral infection (Olson *et al.*, 2004 and Lehmkuhl *et al.*, 2001), *E.coli* infection (Elfaki, 2000), due to toxin of *Clostridium perefringens* (Pathak *et al.*, 1995 and Uzal, 1998) and parasitic conditions (Devi *et al.*, 2004 and Sharma *et al.*, 1997b). Sharma *et al.* (1997a) reported compression atrophy of villi in caecum and colon

of sheep and goat in *Trichuris* infection. The villous atrophy and fusion reduces the absorptive surface of the mucosa and results in malabsorption. The degenerative and necrotic changes observed thus indicate an insult to the gastro intestinal system, associated with hypoxic injuries, infection and toxicities. Two cases revealed intra nuclear inclusion bodies in epithelial lining cell undergoing lytic changes. Intra nuclear inclusion bodies were observed in adenoviral infection in kids by Olson *et al.* (2004) and Oros *et al.* (1996) and in PPR by Bundza *et al.* (1988). Eligulashvili *et al.* (1999) detected intracytoplasmic inclusion bodies in epithelial cells of intestinal crypts in PPR infection. The detection of inclusion body in these two cases thus suggests a possible viral etiology. The acellular and faded stroma with polypoid projection into the lumen observed in two cases could be due to the exotoxin of *Clostridium perfringens,* as described by Gelberg (1995).

Among the inflammatory changes of intestine catarrhal inflammation predominated in all the segments, followed by subacute type of enteritis. The histopathological changes observed were villous atrophy, their fusion, denudation of villi, necrosis of villous epithelium, damage of glands and inflammatory cell infiltrates. In catarrhal type, infiltration with neutrophils was noticed along with small number of mononuclear cells. The predominant cell types noticed were lymphocytes, macrophage and plasma cells in case of subacute enteritis. This agrees with observation made by Sharma *et al.* (1998) in enteritis in sheep and goat. The lesion observed in catarrhal type agrees with observation made by Manomohan (1982) in kids. Oros *et al.* (1996) observed catarrhal enteritis in kids due to adeno viral infection. Mononuclear cells as predominant infiltrating cells in intestinal mucosa was reported in several conditions such as border disease (Lees *et al.*, 1991), Lymphocytic enteritis (Rae, 1994), *Trichuris* infestation (Sharma *et al.*, 1997b) and in PPR (Pawaiya *et al.*, 2004). The nature of inflammatory cells in subacute enteritis thus suggests either viral or parasitic etiology or chronic nature of the condition. The presence of eosinophils in some cases indicates the reaction against the parasitic infection, though section of

parasite could not be traced. Similar observation was made by Chac *et al.* (1996) in *Schistosoma* infection in goats.

It was observed that the number of enteritic cases was more among kids than other age groups. Even though some species of *Enterobacter* were isolated, it could not be verified by experimental studies whether these organisms had any specific role in the causation of disease. There is much difference of opinion regarding the significance of *E. coli* in causing enteritis since they are natural inhabitants of alimentary tract. It has to be admitted that any predisposing factors, which causes a reduction in defense potential of an animal results in a situation in which *E. coli* or similar organism gain an upper hand producing diseases. Once a strain of *E. coli* with sufficient virulence has established in a herd, predisposing factors become unnecessary and young animals may become affected and die before such factors have time to operate (Barker *et al.*,1993). The fact that mortality due to enteritis was very high in kids suggests the possibility of a defective immune status of animals. The nonreactivity or depletion of the cortical and paracortical areas in those kids reflects the poor immune status or response of the animals. Immunodeficiency during the first few days may be due to the insufficiency of colostrum which may be qualitative or quantitative.

Sixteen kids showed distended caecum filled with creamy pasty material in the lumen. The abomassum of these kids showed curdled and clotted milk adhering to the mucosa. Manomohan (1982) made similar observation in kids died due to colibacillosis and isolated *E. coli* from these animals. It has been established that over feeding may result colibacillosis in kids.

Eleven kids showed nodular lesion of coccidial enteritis. Microscopically there was necrosis of the villi along with hyperplastic areas in the intestinal epithelium. The epithelial cells were found to contain schizonts. Similar observation was made by Devi *et al.* (2004) and Sharma *et al.* (1966). Two animals showed numerous tapeworms in the intestine. Lesion of pimply gut was observed in three animals. The goats presented for postmortem are mainly from

organised farms and are regularly dewormed and that might be the reason for the lesser incidence of parasitic condition.

Mesenteric lymphnodes showed lesions such as lymphoid cell depletion, oedema, thickening of capsule, congestion, trabecular thickening and diffuse degeneration and necrosis of lymphocyte in cortical region. Manomohan (1982) made similar observation in enteritis in kids and opined that these changes may be due to a defect in immune status of animal. Lymphoid follicles of Peyer's patches also showed depletion of lymphoid cell along with degenerative and necrotic change. Bundza *et al,* (1988) observed lymphoid cell depletion in lymphnodes and Peyer's patches in goats infected with PPR. The lesions observed thus suggest either a viral infection or an immunosuppressive condition. Some of the lymphnodes revealed proliferation of lymphoid cells in cortical area which could be due to immune stimulation by the pathogenic organisms. One case revealed infiltration of eosinophils in medullary region. Presense of eosinophils in the intestinal mucosa of the same animal indicates a parasitic or allergic condition.

In the present investigation 20 cases with pneumonic changes was accompanied by various degrees of pathological involvement in the intestine also. Five cases showed interstitial pneumonic changes in the lungs. The small intestine of these cases revealed infiltration of mononuclear cells into lamina propria, along with degenerative and necrotic changes in mucosal layer. Lymphoid cell depletion was observed in Peyer's patches. Degenerative changes were evident in the large intestine also. Bronchi showed hyperplasia and desquamation of epithelial cells into the lumen. Similar observation was made by Pawaiya *et al.* (2004) and Abraham *et al.* (2005) in PPR infection. Eventhough there was no typical gross lesion suggesting PPR the possibility cannot be ruled out without using modern techniques such as immunohistochemical studies. One case showed fibrinous pneumonia along with mild mononuclear cell infiltration in lamina propria of small intestine. Intranuclear inclusion body was observed in

degenerating epithelial cells of intestinal mucosa. The lesion observed in this case agrees with observation made by Olson *et al.* (2004) in adeno viral infection. In two cases the infiltrating cells in both lungs and intestine were mainly neutrophilic in nature, indicating a systemic bacterial infection in those cases. Three cases revealed suppurative lesion in lung along with mononuclear cell infiltration in small intestine. The lesion in the intestine in these cases denotes a chronic condition which might have an immunosuppressive effect, resulting in flaring up of bacterial infection in lung, leading to suppurative changes. Nine cases with pneumonic lesion in lung showed various degrees of degenerative and necrotic changes in the intestinal mucosa. Inflammatory reactions were not evident in intestine in those cases.

The present study revealed lesions of various nature and intensity both in lung and intestinal tract, and highlighted the pathological importance of these two systems as seat of many disease conditions. The findings suggests the necessity of assessing the lesions affecting both the system together rather than considering them separately, since a common etiological factor may be involved. Though serological prevalence of diseases like PPR and bluetongue has been reported, the result of this study did not reveal any specific gross and histopathological lesions which could be attributed to PPR or bluetongue, at least in Trichur area. Since a viral etiology could be suspected in many cases, an in depth study is indicated to evaluate the combined effect of virus, secondary bacterial infection, along with toxicological and immunological injuries.

# *Summary*

# 6. SUMMARY

An investigation was carried out to study the mortality pattern, prevalence and pathology of pulmonary and intestinal tract disorders in goat. Analysis of records maintained in the Center of Excellence in Pathology for the past five years showed a total number of 448 goat mortality cases. Higher mortality rate was observed among kids (54 per cent). Higher mortality was recorded in female (62 per cent) than males (38 per cent). Mortality was higher in the fourth quarter of the year. Enteritis (62 per cent) and pneumonia (29 per cent) were found to be most important lesions observed. The prevalence of pulmonary and intestinal tract disorders was found to be 63 and 75 per cent respectively.

A total of 111 cases were subjected to detailed investigation which included 80 kids, 11 young ones and 20 adults. The samples collected were subjected to detailed gross and histopathological examination. The lesions observed were classified and recorded and the lesions were explicated giving possible etiopathogenesis.

The study revealed 80 percentage incidence of pulmonary lesions. Pulmonary lesions observed were congestion (72 per cent), pulmonary haemorrhage (3 per cent), pulmonary oedema (37 per cent), atelectasis (7 per cent) pulmonary emphysema (32 per cent) and pneumonia (22 per cent). The pneumonic lesions encountered were classified based upon gross and histopathological features as suppurative pneumonia (6 cases) fibrinous pneumonia (5 cases) interstitial pneumonia (5 cases) haemorrhagic pneumonia (2 cases) and aspiration pneumonia (2 cases). The gross and histopathological features of each type of pneumonia have been described and possible etiological factors have been indicated. *Escherichia coli, Klebsiella* sp. and *Corynebacterium* sp. were isolated from the pneumonic lungs. The lesions in the bronchial and mediastinal lymph nodes were also studied. The lesions observed were congestion, oedema, haemorrhage, depletion of lymphocytes and necrotic changes in the cortical area. Bronchi and bronchioles revealed inflammatory and

proliferative lesions resulting in desquamation of epithelium into the lumen. Some cases showed peribronchial accumulation of lymphoid cells. Hydrothorax was observed in 3 per cent of cases. Two cases showed congestion and thickening of pleural membrane.

Seventy two per cent of cases showed various pathological lesions in intestinal tract. The major lesions encountered in small intestine were congestion (56 per cent), degeneration and necrosis (67 per cent), catarrhal enteritis (32 per cent) and subacute enteritis (23 per cent). Two cases revealed lesions of haemorrhagic enteritis while in one case there was ulceration in the small intestinal tract. Intranuclear inclusion bodies were detected in two cases, suggesting a viral etiology. Eleven kids showed lesions of coccidial enteritis involving jejunum and ileum. In three animals there were lesions of pimply gut, while two animals showed lesions of cestodiasis caused by *Monezia* sp. Lesions in the large intestine were congestion (9 per cent), degeneration and necrosis (14 per cent) and catarrhal colitis (8 per cent), while one case showed necrotic colitis. Sixteen kids showed impacted caecum with pasty creamy material filling the lumen. Mesenteric lymph node changes included oedema, congestion, degenerative and necrotic changes. Three cases revealed hyperplasia of lymphoid cells in cortical area. Two cases showed infiltration of eosinophils in the medullary region.

*Escherichia coli* and *Klebsiella* sp. were isolated from intestinal contents and the possible role of these organisms in the causation of enteritis had been discussed. Though bacterial organisms were isolated the possibility of viral infection as revealed by intranuclear inclusion bodies and parasitic condition observed indicate a multiple etiology.

An attempt was made to correlate the lesions in respiratory and intestinal tract. In twenty cases with pneumonic lesion various pathological changes were observed in intestinal tract also. Even though there were no typical gross lesions

suggesting PPR, the possibility cannot be ruled out without using modern techniques such as immuno histochemical studies.

The systematic investigation undertaken has helped to focus attention on the prevalence of various respiratory and intestinal tract disorders in goats. As organised goat farming on commercial basis is becoming popular in Kerala, the mortality should be considered as a serious problem and the result of this study will be of much help to control the mortality and treatment of diseases among kids and goats.

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* Originals not seen.

# PATHOLOGY OF PNEUMO-ENTERIC LESIONS IN GOATS

**HAMZA PALEKKODAN**

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**Centre of Excellence in Pathology**
**COLLEGE OF VETERINARY AND ANIMAL SCIENCES**
MANNUTHY, THRISSUR-680651
KERALA, INDIA

## ABSTRACT

The present study was undertaken to assess the mortality pattern, prevalence and pathology of pulmonary and intestinal tract disorders in goat. The result of the present investigation and evaluation of data from the records revealed a high incidence of pulmonary and intestinal tract disorders in goat. A total of 111 cases were subjected to detailed investigation which included 80 kids, 11 young ones and 20 adults. The samples collected were subjected to detailed gross and histopathological examination. The lesions observed were classified and recorded and the lesions were explicated giving possible etiopathogenesis. The study revealed eighty percentage incidence of pulmonary lesion. The pulmonary lesions observed were congestion, oedema, atelectasis, emphysema, pneumonia and pulmonary haemorrhage. The pneumonic lesions encountered were classified as suppurative pneumonia, fibrinous pneumonia, interstitial pneumonia, haemorrhagic pneumonia and aspiration pneumonia. The gross and histopathological features of each type of pneumonia have been described and possible etiological factors had been indicated. *Escherichia coli, klebsiella* sp. and *Corynebacterium* sp. were isolated from the pneumonic lungs. The lesions observed in bronchial lymph nodes were congestion, oedema, depletion of lymphocytes and necrotic changes in the cortical area. Bronchi and bronchioles revealed proliferative and inflammatory lesions suggesting a viral etiology with secondary bacterial infection or exposure to chronic air pollutant which act as irritant. Seventy two per cent of cases showed various pathological lesions in intestinal tract. The major lesions encountered in small intestine were congestion, degeneration and necrosis, catarrhal enteritis, subacute enteritis, haemorrhagic enteritis and ulceration. The parasitic lesions observed in intestinal tract were coccidiosis, pimply gut and cestodiasis. Lesions in the large intestine were congestion, degeneration and necrosis, catarrhal colitis and necrotic colitis. Mesenteric lymph node changes included oedema, congestion, degenerative and necrotic changes. *Escherichia coli* and *Klebsiella sp.* could be isolated from intestinal contents and the possible role of these organisms in the causation of enteritis had

been discussed. An attempt was made to correlate the lesions in respiratory and intestinal tract. Even though there were no typical gross lesions suggesting PPR, the possibility cannot be ruled out without using modern techniques such as immuno histochemical studies.