PATHOLOGY OF GASTROINTESTINAL DISORDERS OF RABBITS

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DECLARATION

I hereby declare that the thesis, entitled **"PATHOLOGY OF GASTROINTESTINAL DISORDERS OF RABBITS"** 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, fellowship or associateship or other similar title, of any other University or Society.

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DEVI, S.S

CONTENTS

LIST OF TABLES

LIST OF FIGURES

9.1	DUODENUM Congestion of submucosa Haemorrhage in the villi Infiltration of inflammatory cells	29&30
9.2	JEJUNUM Congestion of submucosa- H&Ex100 Infiltration of inflammatory cells	29&30
9.3	ILEUM Congestion of submucosa	29&30
10.1	DUODENUM Streaks of haemorrhages in the mucosa	29&30
10.2	JEJUNUM Patchy areas of haemorrhages in the mucosa	29&30
11.1	DUODENUM Degeneration of Brunner's glands Desquamation of villous epithelium	29&30
11.2	JEJUNUM Denudation of degenerated villi tips appearing as clumps in the lumen	29&30
11.3	JEJUNUM Break in the villi tips along with haemorrhage	29&30
11.4	ILEUM Fusion of the villi tips	29&30

11.5	ILEUM Degeneration and necrosis of lymphoid follicles in the Peyer's Patches	29&30
11.6	ILEUM Washed out appearance of Peyer's Patches	29&30
12	JEJUNUM Discontinuity of villous epithelium Congestion Focal inflammation	29&30
13	CATARRHAL ENTERITIS	29&30
14.1	DUODENUM Hemorrhagic inflammation	29&30
14.2	JEJUNUM Hemorrhagic inflammation	29&30
15.1	ILEUM Necrosis and degeneration of mucosa, Necrotic remnants covering the mucosal surface	29&30
15.2	ILEUM Necrotic inflammation	29&30
16	DUODENUM Schizonts of coccidia in the villous epithelial cells	29&30

17	JEJUNUM Larva of pinworm in the submucosa	29&30
18	JEJUNUM Necrosis of the epithelium Fibrin covering the mucosal surface	29&30
19	JEJUNUM Adenoma Proliferative growth of villi Hyperplasia of goblet cells	29&30
20.1	MESENTERIC LYMPH NODE Congestion, Lymphocytic depletion	29&30
20.2	MESENTERIC LYMPH NODE Haemorrhage	29&30
20.3	MESENTERIC LYMPH NODE Necrosis of germinal centre	29&30
21	LIVER Congestion Haemorrhage	29&30
22	LIVER Fatty change-H&Ex400 Fat globules appearing as vacuoles	29&30
23	LIVER Necrosis Cytoplasm of hepatocytes appearing pink and homogenous	29&30

24	LIVER Focal infiltration of inflammatory cells	29&30
25.1	LIVER Hepatic coccidiosis Gray white nodules on the surface	29&30
25.2	LIVER Hepatic coccidiosis-H&Ex400 Developing forms of coccidia seen within the bile duct	29&30
26	LIVER Fibrous tissue proliferation	29&30
27	LIVER Hepatic cyst Multiple cysts containing proteinaceous material inside	29&30

Introduction

INTRODUCTION

Rabbit farming is a burgeoning industry in our country. Rabbits are excellent animals to be raised for meat, fur and skin. They are also very good model animals for laboratory experimentations. As efficient converters of vegetable proteins into high quality animal protein, broiler rabbits have become highly popular among people and their rearing has become a remunerative entrepreneurial area. Rabbit meat is highly delicious and rich in protein. It is extremely lean making it perfect for cholesterol reducing diets. Rabbits produce white meat that is finely grained and rabbit carcasses are only 20 per cent bone.

Rabbits are hardy animals that can be raised in a very small area and they have remarkable ability to utilize fodder and agricultural by products. They are highly prolific with short generation interval. Rabbits require only less concentrate feed thus curtailing their rearing cost. Rabbits are also raised for non-food purposes. High quality rabbit skin and fur are used in garment industry. Medical and veterinary researches also require a large number of rabbits each year. Many people raise rabbits for show or as pets. Thus raising rabbits can provide nutritious and wholesome meat to the family table, educational experiences for the young, enjoyable activity for family members of all ages, rich manure for gardening and potential extra income.

In spite of all these advantages, rabbit farming on an industrial scale has been slow to develop. Excessive mortality has hindered the tradition to mass production. Diseases are responsible for considerable economic loss in rabbit farms on account of high mortality, lowered productivity and decreased performance. Diseases occurring in research colonies adversely affect the progression of research.

Heavy mortality among rabbits due to digestive disorders has been reported by many workers in different parts of the world. Pawaiya *et al.* (1998)

identified that diseases of digestive system were responsible for the death of more than 62 per cent of the cases they studied. While these conditions can largely be prevented and treated, they often go undiagnosed to the point where serious physical injury occurs.

Diarrhoea and poor growth are the main manifestations of gastrointestinal dysfunction. Parasites, toxins, irritants and a variety of bacterial and viral pathogens have been incriminated as the causes. Hence comprehensive studies aimed at exploring the spectrum of etiological agents and the resulting pathological changes responsible for digestive disorders will facilitate the choice of suitable preventive and curative measures to reduce rabbit mortality. Therefore an attempt has been made in the present study to investigate the pathological disorders affecting the gastrointestinal system of rabbits with the threefold objective of identifying the common digestive disorders, studying the gross and microscopic lesions and isolating the associated parasitic and bacterial pathogens.

Review of literature

2. REVIEW OF LITERATURE

2.1 PREVALENCE

Hinton (1980) opined that an increased prevalence of gastric ulcers in female rabbits could be due to pregnancy, labour and post parturient complications.

An investigation conducted by Devi *et al.* (1990) on the mortality pattern of rabbits revealed an increased incidence of intestinal coccidiosis during summer months.

Srilatha *et al.* (1993) reported a prevalence of digestive disorders in 20 out of 150 rabbit carcasses, they autopsied. Conditions like overgrown incisors, hairballs, ulcers, haemorrhagic enteritis, mucoid enteritis and coccidiosis were encountered.

Out of 102 Angora rabbits autopsied by Sharma *et al.* (1995), enteritis was the cause of death in 14 animals while hepatitis accounted for death in eleven.

Pawaiya *et al.* (1998) observed that the diseases of digestive system were responsible for the death of 62.01 per cent of 853 rabbits they examined. They also reported a higher mortality in weanlings.

In a survey conducted by Mondal *et al.* (2007) in Himachal Pradesh, 20.13 per cent of the mortality observed among Angora rabbits was due to hairball occlusion and 17.5 per cent due to enteritis.

An investigation undertaken by Das *et al.* (2007) to know the mortality pattern of rabbits under hot-arid conditions revealed an equal mortality rate in all the seasons. They also reported pneumonia and enteritis as the main causes of mortality in kits up to fifteen days.

2.2 ETIOLOGY

2.2.1 Bacterial Etiology

Savage and Sheldon (1973) conducted pathological and bacteriological examination in a group of rabbits with spontaneously occurring diarrhoea. The lesions included cecocolitis with oedema of the gut wall, necrosis of the mucosa and hemorrhages of the serosa in most of the cases. High numbers of *E. coli* were isolated from the cecum of such rabbits.

According to Peeters *et al.* (1978) proliferation of enteropathogenic *E. coli* was associated with a high carbohydrate and low fibre diet.

Targowski and Targowski (1979) isolated, characterized and identified *Haemophilus paracuniculus* from the gastrointestinal tracts of rabbits with mucoid enteritis.

Borriello and Carman (1983) isolated *Clostridia spiroforme* from the cecal contents of rabbits with spontaneous diarrhoea. They also isolated the organism from animals with clindamycin associated colitis.

Yuill and Hanson (1985) observed that the presence of large numbers of *E. coli* throughout the intestine of rabbits was associated with mortality only when the animals are exposed to low temperatures. They also opined that juvenile rabbits experienced increased susceptibility to *E. coli* as compared with adults.

Hampson (1986) reported that Tyzzer's disease caused by *Bacillus piliformis* produced acute watery diarrhoea in weanlings causing acute mortality.

Intestinal lesions resembling paratuberculosis was reported by Angus (1990) in a feral rabbit which showed thickened intestinal mucosa with a rugose appearance.

Srilatha *et al.* (1993) isolated *Salmonella* and *E. coli* from the intestinal contents of rabbits with haemorrhagic enteritis.

Songer (1996) opined that destabilization of cecal microflora was the important initiation factor for enterotoxemia of rabbits caused by *Clostridium spiroforme*.

E. coli and *Salmonella* were isolated by Pawaiya *et al.* (1998) from rabbits with gastroenteritis.

Schauer *et al.* (1998) found that dual infection with enteropathogenic *E. coli* and *Lawsonia intracellularis* in rabbits produced unusually high mortality due to proliferative enterocolitis.

Duhamel *et al.* (1998) opined that intracellular organisms causing proliferative enteropathy associated with infection of villous and crypt enterocytes were related to *Lawsonia intracellularis* of various other animal species.

The first description of Listeriosis was made by Sabocanec *et al.* (2000) in farm Chinchillas of Croatia. They observed whitish gray nodules along the capsule of the liver, serosa of the colon, and in the mesenteric lymph nodes.

The most common enteropathogens isolated by Sharma *et al.* (2006) using rectal swabs from 65 rabbits were *E. coli* and *Klebsiella.*

2.2.2 Viral Etiology

An ultra structural investigation of rabbit haemorrhagic disease was conducted by Alexandrov *et al.* (1993) and Calici virus was reported to be the etiology

The pathogenesis of infection with rinderpest virus was investigated in rabbits by Okita *et al.* (1995) and found that lymphoid tissue of the gut showed marked virus growth with the most severe lesion being in Peyer's Patches of ileum.

Motha and Kittelberger (1998) stated that rabbit haemorrhagic disease was an acute fatal disease of wild and domesticated rabbits caused by a highly contagious Calici virus.

Rabbit haemorrhagic disease caused by calici virus infection could kill most adult rabbits within 24 to 72 hours after viral inoculation (Ferreire *et al.*, 2006).

2.2.3 Parasitic Etiology

Jacobson *et al.* (1974) reported that infection with intestinal flukes *Hasstilesia triclor* could cause fatal consequences in rabbits. The lesions observed in such cases were enteritis associated with extreme villi destruction in the small intestine.

According to Catchpole and Norton (1979), intestinal coccidiosis in rabbits resulted in high morbidity and mortality.

Hampson (1986) observed that hepatic coccidiosis produced severe emaciation and jaundice in affected rabbits.

Devi *et al.* (1990) conducted an analysis of the mortality pattern in broiler rabbits and reported intestinal coccidiosis as one of the major causes of death.

Hoste and Mallet (1990) identified that heavy infection with *Trichostrongylus colubriformis* in rabbits could produce marked crypt hyperplasia and lesions of the villi.

The prevalence of coccidiosis was more among rabbits penned in loose housing system (Srilatha *et al.*, 1993).

The main species of coccidia, identified by Pillai and Subramanian (1993) from fecal samples of rabbits were *Eimeria magna, E. perforans, E. media* and *E. intestinalis.*

The helminth parasite *Passalurus ambiguus* produced decreased weight gain, constipation, impaction and intussusception in rabbits (Gentz *et al.*, 1995).

Gustafsson *et al.* (1997) observed that the hare, unlike the rabbit was unable to limit the multiplication and dissemination of *Toxoplasma gondi* and hence developed severe tissue necrosis of the small intestine, mesenteric lymph nodes and the liver.

Singla *et al.* (1998) conducted investigations in New Zealand white rabbits by feeding the metacercariae of *Paramphistomum cervi* and rabbits were found to be only abnormal hosts.

Unexpected deaths in young New Zealand white rabbits were attributed to hepatic coccidiosis wherein the livers of all the animals showed multiple military abscesses in all the lobes (Wilkinson *et al.*, 2001).

Pazhanivel *et al.* (2007) observed congestion of lungs and trachea in hepatic coccidiosis of rabbits.

2.2.4 Fungal Etiology

Rabbits experimentally infected with avian isolates of *Candida albicans* showed lesions like necrotic foci and grayish white nodular abscesses in the liver, gall bladder, and stomach (Lakkawar *et al.*, 2003). Histologically, there was congestion, hemorrhages, and degenerative changes along with an inflammatory reaction.

Atasever *et al.* (2004) reported that experimental aspergillosis in rabbits could produce grayish white nodules in the liver and hemorrhagic ulcers in the stomach.

2.2.5 Chlamydial Etiology

Sharma *et al.* (1996) reported a case of chlamydial enteritis in a male broiler rabbit of 6-12 weeks. The characteristic lesions they observed were inflamed small intestine distended with gas and hemorrhagic exudates along with typical paintbrush type of hemorrhage that was conspicuous on the cecal serosa.

2.3 NEOPLASMS

Jardine *et al.* (2004) reported malignant mesenchymal tumors involving liver and mesentery in two white tailed jack rabbits.

A well differentiated hepatocellular carcinoma was reported by Narayanan *et al.* (2004) in a white giant rabbit. They observed multiple foci of neoplastic cells forming nodules of varying sizes.

2.4 TOXICITY

2.4.1 Heavy Metal Toxicity

In a study conducted in rabbits by Titame *et al.* (1997) to assess the toxicity of molybdenum, they observed deposition of iron pigments in macrophages of small intestine and liver.

Cadmium toxicity in rabbits produced degenerative changes, multiple focal necrosis, periportal fibrosis and infiltration of inflammatory cells in liver (Pawaiya *et al.*, 1998).

2.4.2 Insecticide Toxicity

Toxicity studies in rabbits using chlorpyrifos, a broad spectrum and systemic insecticide, revealed degenerative and inflammatory changes in the liver and small intestine (Bansal *et al.*, 1995).

Bansal *et al.* (2006) described the pathomorphological changes in digestive organs due to experimental cypermethrin toxicity consisting of desquamation of epithelial cells of esophagus, stomach and intestine together with infiltration by mononuclear cells.

2.4.3 Mycotoxins

Ochratoxicosis in rabbits could produce lesions ranging from mild degenerative changes to severe necrosis on various vital organs like kidney, liver, gastrointestinal tract and mesenteric lymph nodes (Mir *et al.*, 1999).

Reddy *et al.* (2000) conducted histopathological studies of vital organs in young ones of dams fed with aflatoxin B1 and observed degenerative changes and formation of cystic spaces in the liver.

Experimental studies undertaken by Mir and Dwivedi (2000) on subacute ochratoxicosis in rabbits showed that the characteristic clinical syndrome included diarrhoea, anorexia, swollen hyperemic rectum and decreased body weight gain.

2.5 PATHOLOGICAL CONDITIONS

2.5.1 Degeneration and Necrosis

Immunosuppressive effect of T-2 mycotoxin was studied by Niyo *et al.* (1988) in rabbits and they found mild to severe lymphocytic necrosis in the appendix, sacculus rotundus, Peyer's Patches and mesenteric lymph nodes.

Kagawa (1990) reported that the most characteristic finding in acute corrosive gastritis in rabbits was a brown discoloration of the mucosa, which histologically corresponded to gangrene of the gastric wall.

Park *et al.* (1992) studied the changes in hepatocytes of rabbits infected with rabbit hemorrhagic disease virus and found that the earliest change was hydropic degeneration and focal necrosis. Necrotic hepatocytes showed shrunken cell body and karyolysis.

Experimental studies with *Polystichum squarrosum* fern in rabbits revealed ulceration of gastric mucosa, degeneration and desquamation of intestinal epithelium and atrophy of hepatocytes (Somvanshi and Gounalan., 1996).

Mir *et al.* (1999) conducted studies on rabbits fed with ochratoxin A. They observed degeneration and necrosis of gastrointestinal mucosa with desquamation of epithelial cells into the luminal spaces. Mesenteric lymph nodes revealed necrosis of the germinal centres in the lymphoid follicles.

Chandra *et al.* (1999) described the lesions of experimental *Trypanosoma evansi* infection in rabbits. In liver, vacuolation of hepatocytes, sinusoidal congestion and focal necrotic changes were observed.

Toxicity testing of *Senna occidentalis* seeds in rabbits produced extensive vacuolar degeneration of the hepatocytes (Tasaka *et al.*, 2000).

Histopathological examination of the liver of pregnant rabbits with induced aflatoxicosis showed hepatocytomegaly, degeneration of liver cells, and bile duct proliferation (Reddy and Rao, 2001).

Chakrabarti *et al.* (2004) opined that induced *Staphylococcus aureus* infection in rabbits could produce necrotic hepatitis.

Kimpe *et al.* (2004) first reported a case of *Leptospira ivanoii* septicemia in a chinchilla. The lesions described were vacuolar degeneration, multifocal necrosis of liver and catarrhal enteritis.

2.5.2 Congestion and Haemorrhage

Gastric ulcers in rabbits observed by George and Somvanshi (1987) were characterised by capillary haemorrhages in lamina propria. Extravasated blood in the ulcerated areas imparted dark red or brown colour to the lesions.

Srilatha *et al.* (1993) described the lesions of intestinal coccidiosis in rabbits. This consisted of ballooning of intestines with thickened wall, petechial haemorrhages on the mucous membrane of small intestine and blood mixed intestinal contents. Congestion, haemorrhages, epithelial desquamation and hyperplastic glands were observed microscopically.

Prominent hyperemia and haemorrhage in liver with sinusoids engorged with edema fluid and erythrocytes were reported by Somvanshi *et al.* (1997) in rabbits fed with *Dryopteris juxtaposita* fern.

Pawaiya *et al.* (1998) isolated Clostridium and Pasteurella from septicemic rabbits that revealed widespread petechial to ecchymotic haemorrhages and congestion of intestines and other visceral organs.

Infection of rabbits with lapinised rinderpest virus produced congestion of the liver and mesenteric lymph nodes. The sacculus rotundus, cecal tonsils, and appendix were edematous and Peyer's Patches had follicular hyperplasia (Renukaradhya *et al.*, 1999).

Srilatha *et al.* (2001) described lesions of Tyzzer's disease in New Zealand white rabbits which consisted of paint brush streaks of haemorrhages at

the junction of the cecum and colon. Liver showed severe congestion with necrotic spots.

2.5.3 Inflammation

Experimental *Yersinia enterocolitica* enteritis in rabbits was characterised by pronounced histopathological alterations in the ilea. The consistent observation was the presence of crypt abscesses of the intestinal glands composed of a bacterial nidus admixed with and enveloped by inflammatory cells (Pai *et al.*, 1980).

Barshstein *et al.* (1982) observed that infection with opportunistic agents like *Proteus* and *Klebsiella* produced catarrhal enteritis with predominant involvement of epithelial sheet (epithelitis).

Songer (1996) described the lesions of iota enterotoxemia in rabbits which was grossly characterized by an immensely dilated cecum with watery contents. Microscopically there was necrosis of surface epithelium accompanied by a prominent inflammatory infiltration of lamina propria.

Schauer *et al.* (1998) described the lesions of proliferative enteropathy in rabbits. Upon gross examination, the affected animal had thickening of both the ileum and colon along with mesenteric lymphadenopathy. Histological examination revealed intestinal epithelial cell hyperplasia and a moderate inflammatory cell infiltration.

Gomez *et al.* (2002) reported that typical histological changes in rabbits infected by *Shigella flexneri* included villous blunting with sloughing of epithelial cells, submucosal edema, infiltration of leukocytes, venous congestion and haemorrhages. They also observed that lactoferrin protected rabbits from development of the above lesions.

Beltz *et al.* (2005) conducted histological and ultrastuctural examinations in commercial bred rabbits with severe diarrhoea. Their histological findings consisted of mucoid neutrophilic or lympho-plasmacytic enteritis with atrophy and fusion of the villi, hyperplasia of goblet cells and serosal and submucosal oedema in small intestine. Ultrastructurally, there was a loss of microvilli.

2.6 MISCELLANEOUS CONDITIONS

Lazarus and Volk (1964) observed fatty metamorphosis and fibrosis of the liver in rabbits with protein malnutrition.

Kivilaaksa *et al.* (1978) suggested that an important factor that rendered the rabbits susceptible to gastric ulcers was the nature of the stomach mucosa, which responded to stress very rapidly.

Targowski and Targowski (1979) identified that hyperpasia of goblet cells in the duodenum, jejunum and ileum was the characteristic lesion in mucoid enteritis complex in rabbits.

George and Somvanshi (1987) observed that gastric ulcers were more in rabbits with coccidiosis and Encephaltizoon cuniculi infection.

An experimental study undertaken by Vijayan and Rajan (1994) to assess the toxicological effect of an anticoccidial preparation containing nitrofurazone and furazolidone in rabbits revealed centrilobular fatty change, distended gall bladder and engorged central vein.

Gentz *et al.* (1995) reported that one of the primary causes for anorexia in rabbits was gastric obstruction by trichobezoars. According to them, the inherent absence of myoelectrical peristalsis in the stomach was the cause for the common occurrence of gastric trichobezoars.

Mir and Dwivedi (1998) observed the presence of a bifid gall bladder filled with thin bile in a rabbit of 12 weeks of age.

Studies on young New Zealand white rabbits maintained on ochratoxin A diets revealed diarrhoea as the first clinical sign (Mir and Dwivedi., 2000).

A severe digestive syndrome causing heavy mortality in rabbit farms was named as Epizootic Rabbit Enteropathy (ERE) (Licois *et al.*, 2005). The disease was characterised by an absence of inflammatory or congestive lesions in the gut or on other organs. Stomach and intestines appeared dilated with liquid and gas and there were no specific histological lesions.

Kesavan *et al.* (2005) observed an extensive lymphocytic depletion from lymph nodes, Peyer's Patches and appendix in rabbits, which had undergone dexamethasone treatment. Similar depletion of lymphocytes from peripheral lymphoid organs was also noticed by them in protein malnutrition.

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 prevalence and pathology of various disorders of the gastrointestinal system of rabbits.

3.1 SAMPLE COLLECTION

A total of fifty carcasses of rabbits brought for autopsy in the Centre of Excellence in Pathology during the period of study were examined and samples of tissues from the liver, stomach, small and large intestines and the mesenteric lymph nodes were collected for gross and histopathological investigations. Intestinal contents were screened for ova of parasites.

3.2 GROSS EXAMINATION

A detailed and systematic examination of rabbit carcasses brought for autopsy was made. The liver and mesenteric lymph nodes were carefully examined for gross lesions like changes in size, shape, colour, consistency, presence of cysts, tumors, abscesses etc. Gross lesions of the stomach, small intestine and large intestine were also studied. Representative samples were collected for histopathological and microbiological examination.

3.3 HISTOPATHOLOGICAL EXAMINATION

Representative samples of the liver, mesenteric lymph nodes, stomach, small and large intestines obtained from the carcasses were collected and preserved in 10 per cent neutral buffered formalin. They were then processed and paraffin embedded as described by Sheehan and Hrapchak (1980). Sections were cut at four microns thickness and stained with routine Hematoxylin and Eosin as per the technique followed by Bancroft and Cook (1995). Special staining

techniques like Oil red O for fat and PAS for glycogen were done as and when required as per the methods of Luna (1968). The lesions were examined in detail under light microscope and were classified.

3.4 MICROBIOLOGICAL STUDIES

Bacterial isolation was attempted from the liver, heart blood and intestine of fresh carcasses. Colonies were identified by cultural and morphological characters. Specific biochemical tests helpful in identifying the organisms were also carried out.

3.5 PARASITOLOGICAL EXAMINATION

Intestinal contents were collected and screened for the presence of ova of parasites by direct smear method.

Results

4. RESULTS

4.1 PREVALENCE

Postmortem examination of rabbit carcasses conducted at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy, between March 2006 and May 2007, revealed that 52 per cent of the deaths were due to gastrointestinal disorders.

Samples of stomach, small intestine, large intestine, mesenteric lymph node and liver from fifty rabbit carcasses were examined and the lesions were classified based on the age, sex and pathological findings. Out of these fifty cases, 42 per cent showed lesions in the stomach, 74 per cent in the intestine, 58 per cent in the liver and 30 per cent in the mesenteric lymph nodes. Of the twenty nine cases with lesions in the liver, eighteen had concurrent lesions of gastrointestinal tract also.

The study revealed a high prevalence of gastrointestinal disorders among weanlings. In addition, it was found that gastrointestinal lesions were more in female rabbits as compared with males.

Table 1. Age wise distribution of lesions in the stomach, liver, intestine and mesenteric lymph nodes

Age	Total No.	Stomach Lesions		Liver Lesions		Intestinal Lesions		Lymph Node Lesions	
		No.	%	No.	%	No.	%	No.	%
Day old	14	4	29	6	43	10	71	2	14
Weanlings	17	9	53	13	76	16	84	6	35
Adults	19	8	42	10	53	11	65	7	37

Table 2. Sex wise distribution of lesions in the stomach, liver, intestine and mesenteric lymph nodes

Sex	Total No.	Stomach Lesions		Liver Lesions		Intestinal Lesions		Lymph Node Lesions	
		No.	%	No.	%	No.	%	No.	%
Male	21	7	33	12	57	15	71	6	29
Female	29	14	48	17	59	22	75	9	31

4.2. CLASSIFICATION OF LESIONS

4.2.1 Stomach

Stomach lesions were seen in 42 per cent of the fifty cases. Majority of the lesions occurred in combination with those in other organs.

4.2.1.1 Vascular changes

4.2.1.1.1 Congestion

Congestion of varying degrees was observed in six (29 per cent) out of the twenty one cases with stomach lesions. Grossly, the mucosa appeared red and dark. The blood vessels of mucosa and serosa appeared prominently injected with blood. Histopathologically, mucosal, submucosal and serosal blood vessels were severely dilated and packed with erythrocytes (Fig.1).

4.2.1.1.2 Haemorrhage

Two (ten percent) out of twenty one affected cases showed various degrees of haemorrhages. Grossly, there were patchy red areas in the mucosa.

Microscopically, there was extravasation of erythrocytes in the gastric foveolae and in the lamina propria.

4.2.1.2 Degeneration and Necrosiss

Six (29 per cent) out of twenty one cases with stomach lesions showed degeneration and necrosis of gastric tunics. Grossly, there was focal disappearance of mucous membrane and the mucosa appeared congested. Microscopically, the lesions varied from mild degenerative changes to extreme necrosis. In milder forms there was degeneration of epithelial cells of gastric mucosa, together with desquamation of surface epithelium. In certain cases there were fusion of the gastric foveolae, cystic dilatation of upper mucosa and atrophy of glandular epithelium. Extreme necrosis of the glands and necrotic remnants within crypts was seen in one of the cases (Fig.2 and Fig.3). There was complete necrosis of the gastric mucus cells, chief cells and parietal cells, mild oedema and congestion of vessels of lamina propria and dilatation of lymphatics.

4.2.1.3 Ulcers and Erosions

Out of the twenty one cases with stomach lesions, four had ulcers and erosions of varying sizes. The size varied from a few mms to 0.5 cm in diameter. Ulcers appeared as circumscribed areas with hyperaemic and raised borders sometimes extended deep into the submucosa (Fig.4). Erosions were shallow and produced only a superficial loss of mucosa. Histopathologically, there was a break or discontinuity in the gastric mucosa and submucosa exposing the underlying muscular layer (Fig.5). The area was covered with mucus exudate, fibrin, degenerated epithelium and a few inflammatory cells.

4.2.1.4 Inflammation

Inflammations of various types were present in six out of twenty one cases (29 per cent) with gastric lesions.

4.2.1.4.1 Catarrhal Gastritis

Stomach appeared distended with mucus mixed exudate. The mucosa was congested and dark in colour (Fig.6). The mucus appeared as glistening slimy clear material. Microscopically, there was proliferation of goblet cells of gastric foveolae, desquamation of epithelial cells into the lumen, infiltration of mononuclear cells in the lamina propria, gastric crypts and muscularis mucosa (Fig.7). There was also engorgement of submucosal and serosal blood vessels.

4.2.1.4.2 Necrotic Gastritis

Necrotic gastritis was observed in one case. Grossly, there was congestion and focal accumulation of a cheesy material on the mucosal surface. Severe degeneration and necrosis of superficial epithelium and gastric glandular cells, infiltration of inflammatory cells in the mucosa, congestion of blood vessels in sub mucosa and oedema of lamina propria were the histopathological findings. (Fig.8).

4.2.2 Intestine

Intestinal lesions were seen in thirty seven (74 per cent) cases. The lesions were classified as vascular changes, degeneration and necrosis, inflammatory changes and neoplasm. Region wise evaluation of the lesions was also carried out.

4.2.2.1 Vascular changes

4.2.2.1.1 Congestion

Grossly, the mucosa was swollen and appeared red. The serosal and mucosal blood vessels were engorged and prominent. Microscopically, blood vessels in the mucosa, submucosa and serosa were packed with erythrocytes. Nine animals showed congestion of duodenum (Fig. 9.1), whereas jejunal

congestion (Fig.9.2) was present in seventeen cases. Congestion of ileum was noticed in twelve rabbits (Fig.9.3). In large intestine it was mostly the colon that showed congestion. Congestion of colon was seen in six out of the thirty seven affected cases.

4.2.2.1.2 Haemorrhage

Five (14 per cent) out of thirty seven cases with intestinal lesions showed haemorrhages of varying degrees. Grossly, haemorrhages appeared as pinpoint, ecchymotic or patchy areas on the intestinal mucosa. The intestinal contents were mixed with blood. Microscopically, there was extravasation of erythrocytes into the villi and lamina propria along with severe congestion of blood vessels in sub mucosa and serosa. There were two cases with duodenal haemorrhage, one of which showed streaks of haemorrhages of considerable length (Fig.10.1). Three cases showed jejunal haemorrhages (Fig.10.2). Haemorrhage was almost absent in the lower intestinal tract.

4.2.2.2 Degeneration and Necrosis

Degeneration and necrosis of varying intensities was observed in nineteen cases (51 per cent). It varied from a focal mild degeneration of surface epithelium to extreme necrosis of the entire epithelium with necrotic material adhering to the surface. The gross lesion observed was moderate congestion of the mucosa along with a necrotic mass adhering to the mucosal surface. Eight cases had degeneration and necrosis of duodenal mucosa. Here the changes were seen in both mucosa and submucosa. Plicae circulare and crypts of Lieberkuhn showed degeneration of epithelial cells. In some areas the villi appeared fused whereas certain other areas showed a homogenous pink staining necrotic mass adhering to the surface of mucosa. Degeneration of Brunner's glands and desquamation of villous epithelium were also observed in a few cases (Fig.11.1). Congestion, oedema of sub mucosa and dilated lymphatics were the other findings. Degenerative and necrotic lesions in the jejunum were seen in twelve

carcasses. Here the microscopic lesion varied from a mild focal vacuolar degeneration of the villous epithelium in a few cases to severe necrosis of the enterocytes of villi and crypts. In some cases, there were denudation of the degenerated villi tips, which appeared as clumps in the lumen (Fig.11.2) whereas some other cases revealed break in the villi tips along with haemorrhage and glandular destruction (Fig.11.3). Ten cases showed degenerative changes in the ileum. The lesions consisted of desquamation of villous epithelial cells, degeneration of glandular crypts, and fusion of villi tips in a few cases (Fig. 11.4). Two of the carcasses showed lesions in the Peyer's Patches. In one case there was extreme degeneration of lymphoid follicles in the Peyer's Patches with necrosis of the germinal centres (Fig.11.5). In the other case there was depletion of lymphocytes from lymphoid follicles giving a washed out appearance to the Peyer's Patches (Fig.11.6).

4.2.2.3 Ulcer

Intestinal ulcers were noticed only in two (five per cent) cases. Solitary ulcers were noticed in the jejunal part of small intestine. They were circumscribed with raised hyperaemic borders. The wall of the intestine was thin, flaccid and discoloured with a greyish tinge. Histopathologically, there was discontinuity of villous epithelium, congestion, and focal infiltration of inflammatory cells (Fig.12). Muscularis mucosa showed fragmentation of muscle fibres. Sub mucosal oedema and congestion were also characteristic. Moderate degeneration of the villi was noticed in the neighbouring areas also. There was a focal infiltration of inflammatory cells in the vicinity.

4.2.2.4. Inflammation

Thirty two out of thirty seven cases with intestinal lesions showed inflammatory changes of various types. The morphologic and histopathological details of the inflammatory lesions of the intestine showed great diversity.

Changes of intestinal mucosa with hyperaemia, haemorrhage, and increased secretory activity were noticed.

4.2.2.4.1 Catarrhal Enteritis

Catarrhal enteritis more often involved the entire length of small intestine. Grossly the mucosa was swollen and hyperaemic. Intestinal lumen contained mucus mixed slimy contents (Fig.13). Histological changes corresponding to catarrhal enteritis were destruction of villi tips and denudation of epithelium at focal areas. There was mild to intense lymphocytic infiltration of the mucosa. Some cases showed capillary engorgement of mucosa and sub mucosa. About twelve animals showed catarrhal inflammation of duodenum (Fig.9.1), fifteen had catarrhal inflammation of jejunum (Fig.9.2), nine had catarrhal inflammation of ileum and five had catarrhal inflammation of colon.

4.2.2.4.2 Haemorrhagic Enteritis

Haemorrhagic inflammation of the intestine was noticed in twelve cases. Grossly, the intestinal contents were mixed with blood and mucus and had a pasty consistency. The mucous membrane was hyperaemic with haemorrhagic patches. Serosa showed congestion. Haemorrhagic inflammation was more prominent in the small intestine. Histopathologically, there were pronounced capillary engorgement, extravasation of erythrocytes in the tips of villi and infiltration of inflammatory cells. In extreme cases necrosis and desquamation of villous epithelium dominated the haemorrhagic lesions. There was concurrent destruction of tubular glands and diffused infiltration of lymphoid cells. Haemorrhages and villous destruction of glands produced dilated cystic spaces in the basal layers of mucosa in some cases. In one of the cases, the cystic spaces were seen occupied by pink staining necrotic material. Haemorrhagic inflammation of duodenum (Fig.14.1) was observed in five cases while jejunum showed haemorrhagic inflammation (Fig.14.2) in four cases and haemorrhagic

ileitis was a finding in three animals. Haemorrhagic inflammation of the large bowel was not observed in any of the animals.

4.2.2.4.3. Necrotic Enteritis

Necrotic inflammation of ileal mucosa was seen in two cases. Grossly, the intestinal mucosa showed a gray discolouration and the lumen contained turbid fluid with gas bubbles. Histologically, there was severe degeneration and necrosis of the mucosa. Necrotic tissue of lining mucosa could be seen as a homogenous mass adhering to lamina propria and sub mucosa (Fig.15.1 and Fig.15.2). The superficial villi were lost in these areas. Muscularis mucosa also showed degeneration of muscle fibres.

4.2.2.4.4. Parasitic Enteritis

4.2.2.4.4.1. Coccidiosis

Intestinal coccidiosis was seen in thirteen cases. Grossly, there were petechiae and multiple small nodules in the intestinal mucosa. The intestinal contents were copious and slimy with a brown tinge. Histologically, there was superficial and deep capillary haemorrhages and mononuclear infiltration in the villi and lamina propria (Fig.16). Coccidial infestation was more in the villi tips. Developing forms of coccidia predominantly schizonts, were seen distributed in the epithelium of the villi tips. Out of the thirteen cases, seven showed developing forms of coccidia in the duodenal mucosa whereas in the remaining six cases they were observed in the jejunal region.

4.2.2.4.4.2. Verminous Enteritis

Pinworm infestation was observed in one of the rabbits. Grossly, the intestine showed features of catarrhal enteritis with slightly blood tinged contents. Histopathologically, larva was seen penetrating the sub mucosa (Fig.17). It produced mononuclear infiltration of the submucosa and muscularis mucosa

along with a moderate degree of congestion. The worm, *Trichuris* was observed in the intestinal contents of one hare.

4.2.2.4.5. Diphtheritic Enteritis

Diphtheritic inflammation of the jejunum was seen in one case. Grossly, the mucosa of the affected part was seen covered by a membrane imparting a dirty yellowish red colour. Histopathologically, there was coagulative necrosis of the surface epithelium and pink staining fibrin was seen covering the entire mucosal surface (Fig.18).

4.2.2.5. Neoplasm

4.2.2.5.1. Adenoma

Adenoma of jejunum was noticed in one of the cases (three per cent). Grossly, there was an increase in the secretory activity of mucosa and intestinal wall was thickened. Histopathologically, there was proliferative growth of intestinal villi obliterating the lumen. Goblet cells showed high degree of hyperplasia (Fig.19). The basic architecture of the tissue was preserved even if there was a moderate degree of fibrosis.

4.2.3 Mesenteric Lymph nodes

Fifteen (30 per cent) out of fifty cases examined for gastrointestinal disorders revealed pathological changes in mesenteric lymphnode. Vascular changes predominated in five cases. Microscopically, the blood vessels were engorged with erythrocytes (Fig.20.1). In haemorrhages of lymph node, extravasation of erythrocytes into the tissue spaces was seen (Fig.20.2). Depletion of lymphocytes from cortical nodules and medulla was evident in a few cases. In one case, there was focal degeneration and necrosis of germinal centres (Fig.20.3).

4.2.4 Liver

Liver lesions were seen in 58 per cent of the cases. The lesions in 50 per cent of the cases occurred in combination with the other lesions observed in the gastrointestinal tract.

4.2.4.1 Vascular Changes

Vascular changes were classified as congestion and haemorrhage.

4.2.4.1.1 Congestion

Out of twenty nine cases, fourteen (48 per cent) had congestion of varying degrees. Grossly, the organ appeared larger with a dark brown colour. The edges were round. On section, large amounts of blood exuded and the cut surface had a peculiar nutmeg like appearance. Histopathologically, the central veins, portal vessels and sinusoids were dilated and filled with erythrocytes. In extreme cases, there was necrosis and atrophy of hepatocytes.

4.2.4.1.2 Haemorrhage

Six cases (21 per cent) with liver lesions showed haemorrhage in the hepatic parenchyma. Grossly, there were areas of petechiae or ecchymosis on the liver surface. Histopathologically, the erythrocytes were seen infiltrating into the tissue spaces displacing the hepatocytes (Fig.21). In most of the cases, the vascular lesions were accompanied by degenerative and necrotic changes.

4.2.4.2 Degeneration

Grossly, the areas of degeneration appeared as pale yellow streaks. Degenerative changes were classified as cloudy swelling and fatty degeneration.

4.2.4.2.1. Cloudy Swelling

Cloudy swelling was observed in eight (28 per cent) out of twenty nine cases with liver lesions. Grossly, the affected organ was swollen and had a parboiled appearance. On section, the cut surface bulged out. Histopathologically the cytoplasm of the hepatocytes was eosinophilic and granular. Sinusoidal congestion was also a finding in such cases.

4.2.4.2.2 Fatty Change

Five out of twenty nine cases (17 per cent) of animals with liver lesion had fatty change. Grossly, the liver was enlarged, soft and pale with rounded borders. Fat globules appeared as vacuoles in the cytoplasm of hepatocytes compressing the nucleus (Fig.22). Fat took a red colour on staining with oil red O.

4.2.4.3 Necrosis

Necrosis was observed in the livers of three rabbit carcasses (ten per cent). Grossly, there were areas of grayish white spots or streaks extending into the parenchyma. The cut surface of the affected livers appeared mottled. Histopathologically, cytoplasm appeared pink and homogenous (Fig.23). The nuclei were in varying stages of pyknosis, karyorrhexis and karyolysis. Cell boundaries were not clearly demarcated and there were accompanying vascular changes.

4.2.4.4 Hepatitis

Inflammation of the liver was observed in twelve cases (41 per cent). Grossly, the affected livers had focal yellowish gray spots extending to the hepatic parenchyma. Microscopically, the lesions were characterised by degeneration of hepatocytes, together with infiltration of lymphocytes. The distribution of inflammatory cells varied in different cases In a few of the cases,

the infiltrating cells were seen concentrated focally (Fig-24) whereas in other cases they were seen scattered through out the hepatic parenchyma.

4.2.4.5 Hepatic Coccidiosis

Various developing forms of coccidia were observed in liver sections of four rabbits (14 per cent). Grossly, the affected livers showed focal gray white nodules on the surface. Microscopically, different developmental stages of coccidia, mainly oocysts and schizonts were present within the bile ducts (Fig.25), where they induced hyperplasia of the bile duct epithelium.

4.2.4.6 Fibrosis

Hepatic fibrosis (Fig.26) was noticed in two cases (six per cent). The livers were hard and firm with an uneven surface. There was an increase in fibrous tissue within and around the lobules. Central veins were absent in some lobules or were placed eccentrically. Hepatocytes showed various stages of degeneration to frank necrosis.

4.2.4.7 Hepatic Cyst

Hepatic cysts were observed in three of the cases (ten per cent). Histopathologically, single or multiple cysts of varying sizes with proteinaceous material inside were observed in hepatic parenchyma (Fig.27). The surrounding cells showed degeneration and necrosis.

4.3 PARASITOLOGICAL EXAMINATION

Intestinal contents from the fifty cases were screened by direct smear examination. Oocysts of *Eimeria* were observed on examination of thirteen cases.

4.4 MICROBIOLOGICAL EXAMINATION

Heart blood, intestine and liver were taken from animals for bacterial isolation. Tissues from nine carcasses were used for microbiological studies. Of these, *E. coli* was isolated from the intestines of five animals. Two of the cases yielded *Pasteurella* from liver. One case had *Salmonella* from intestine and one showed a mixed intestinal infection by *E. coli* and *Salmonella*.

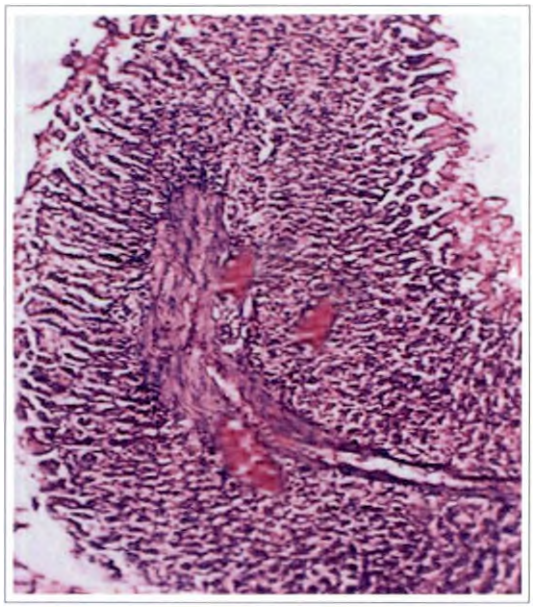

Fig - 1

STOMACH
Congestion of mucosa-H&Ex100

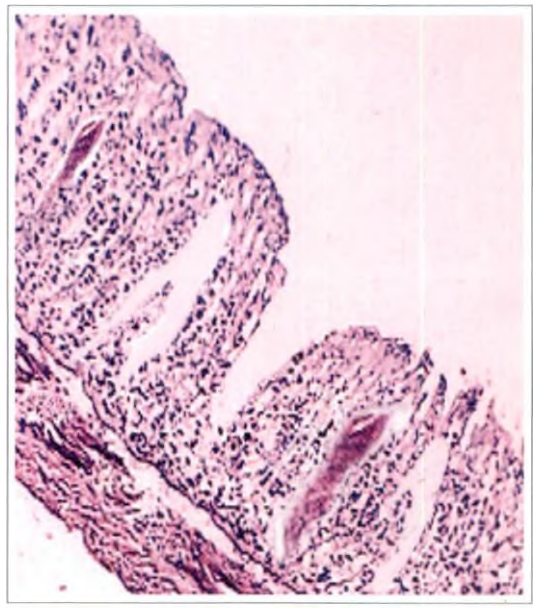

Fig - 2

STOMACH
Cystic dilatation of upper mucosa -H&Ex100
Necrotic remnant within the cystic spaces.

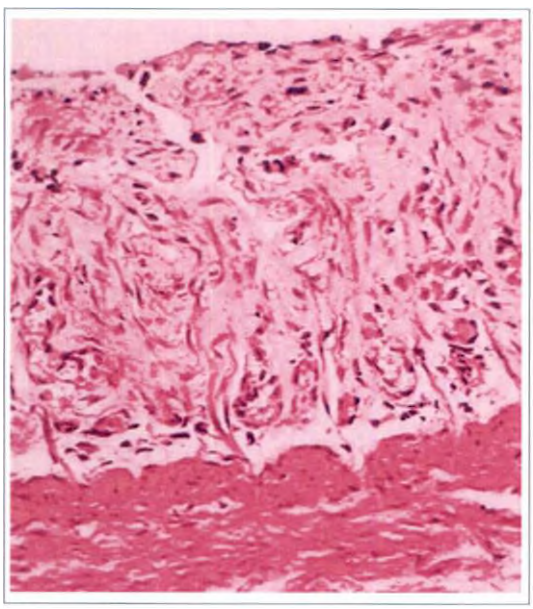

Fig - 3

STOMACH
Fusion of the gastric foveolae- H&Ex400
Degeneration of glandular epithelium

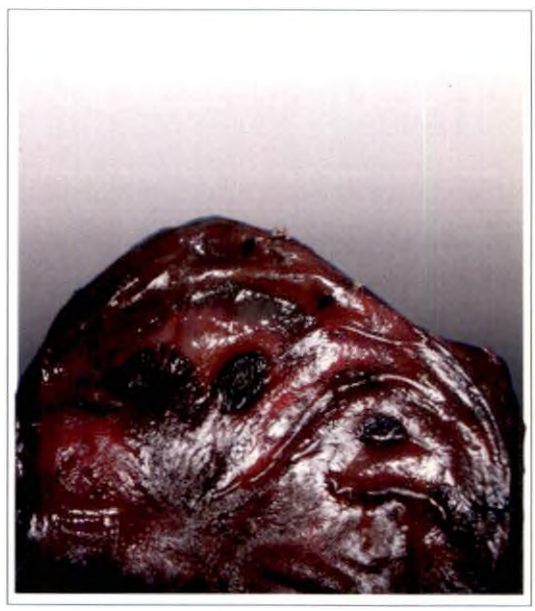

Fig - 4

STOMACH
Ulcers

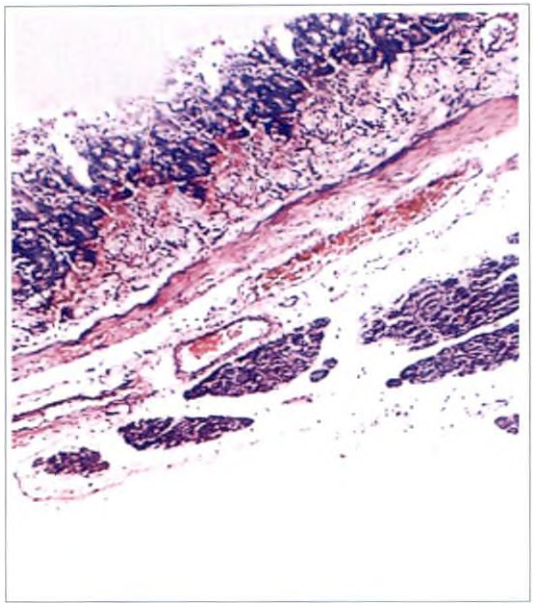

Fig - 9.1

DUODENUM
Congestion of submucosa -H&Ex100
Haemorrhage in the villi
Infiltration of inflammatory cells

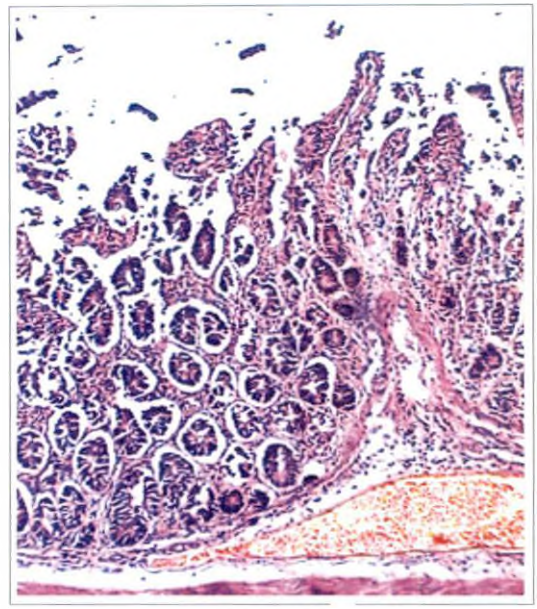

Fig - 9.2

JEJUNUM
Congestion of submucosa- H&Ex100
Infiltration of inflammatory cells

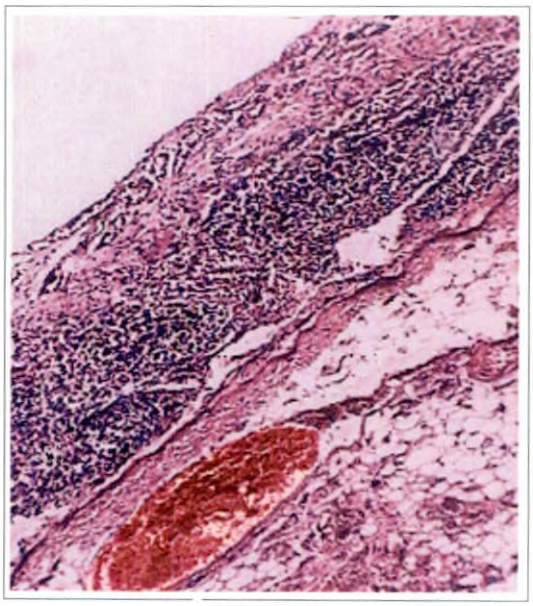

Fig - 9.3

ILEUM
Congestion of submucosa-H&Ex100

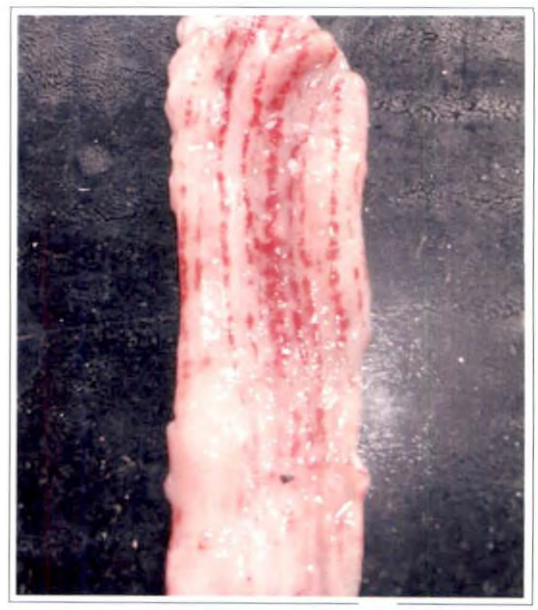

Fig - 10.1

DUODENUM
Streaks of haemorrhages in the mucosa

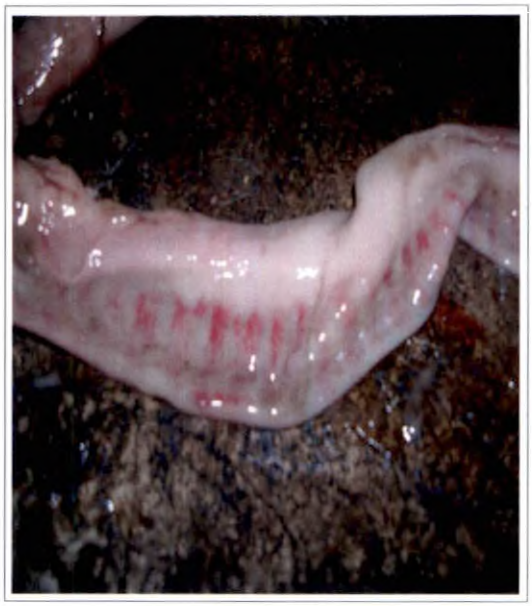

Fig - 10.2

JEJUNUM
Patchy areas of haemorrhages in the mucosa

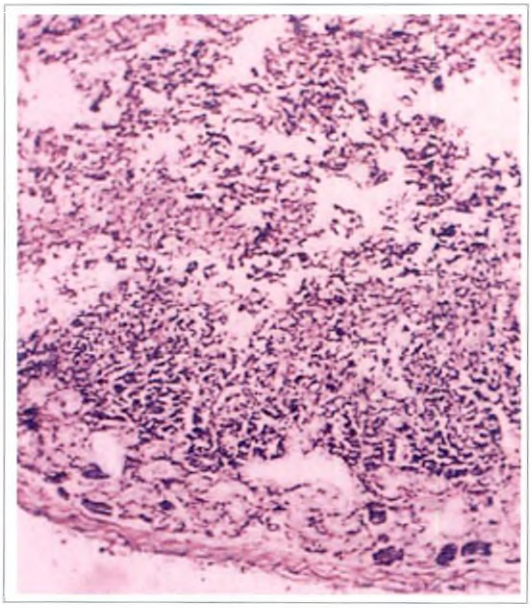

Fig - 11.1

DUODENUM
Degeneration of Brunner's glands-H&Ex100
Desquamation of villous epithelium

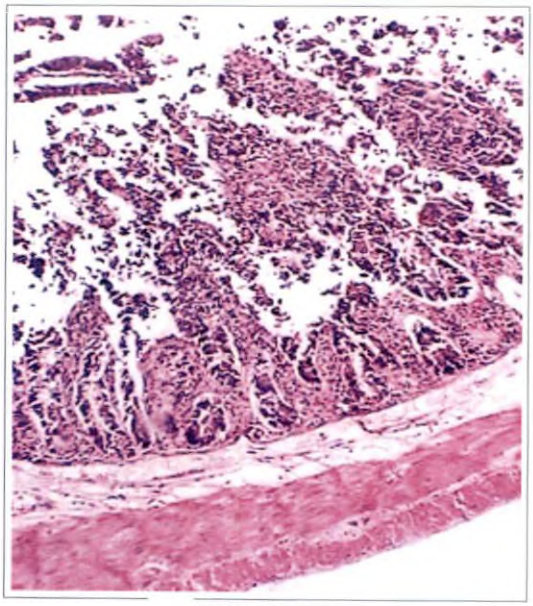

Fig - 11.2

JEJUNUM
Denudation of degenerated villi tips appearing as clumps in the lumen-H&Ex100

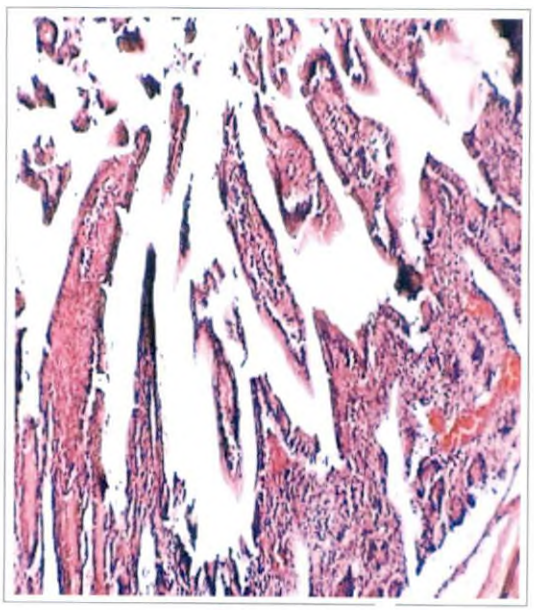

Fig - 11.3

JEJUNUM
Break in the villi tips along with haemorrhage-H&Ex100

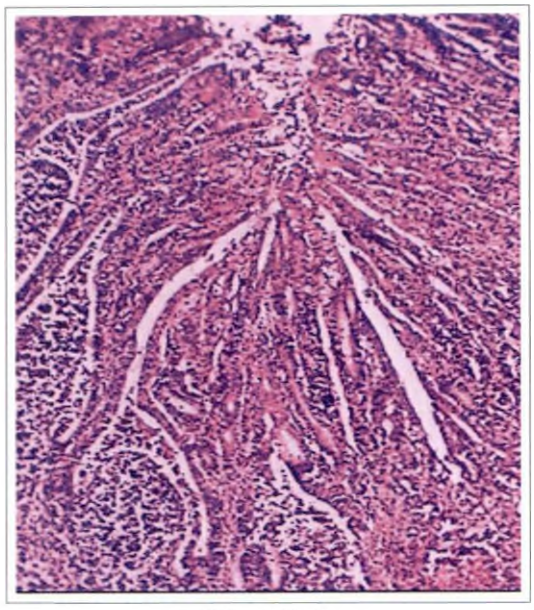

Fig - 11.4
ILEUM
Fusion of the villi tips-H&Ex100

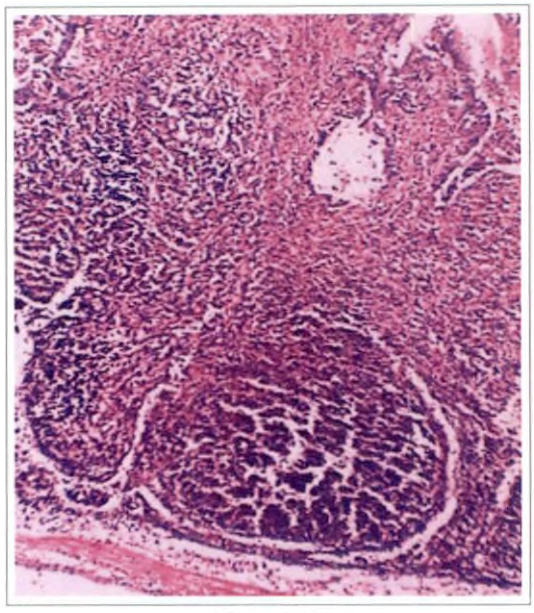

Fig - 11.5
ILEUM
Degeneration and necrosis of lymphoid follicles in the Peyer's Patches- H&Ex100

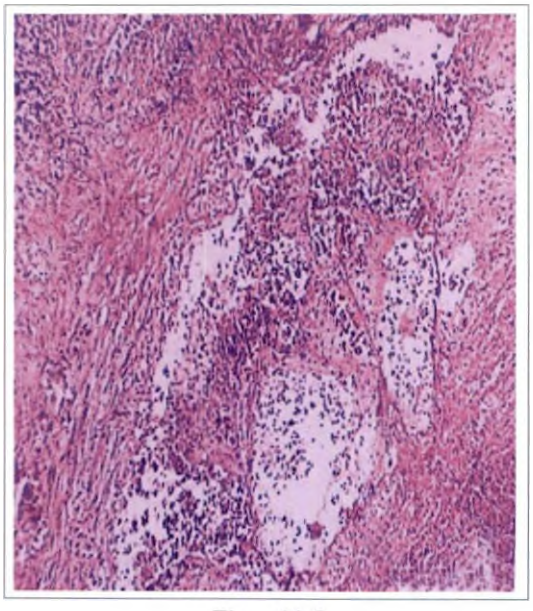

Fig - 11.6
ILEUM
Washed out appearance of Peyer's Patches- H&Ex100

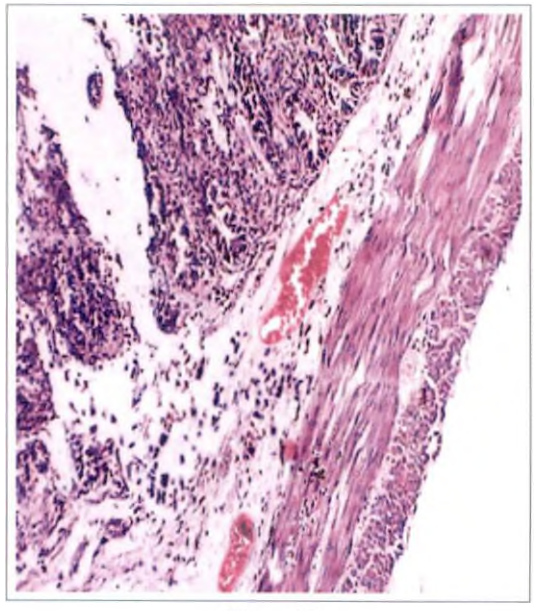

Fig - 12
JEJUNUM
Discontinuity of villous epithelium Congestion, Focal inflammation- H&Ex100

Fig - 13
CATARRHAL ENTERITIS

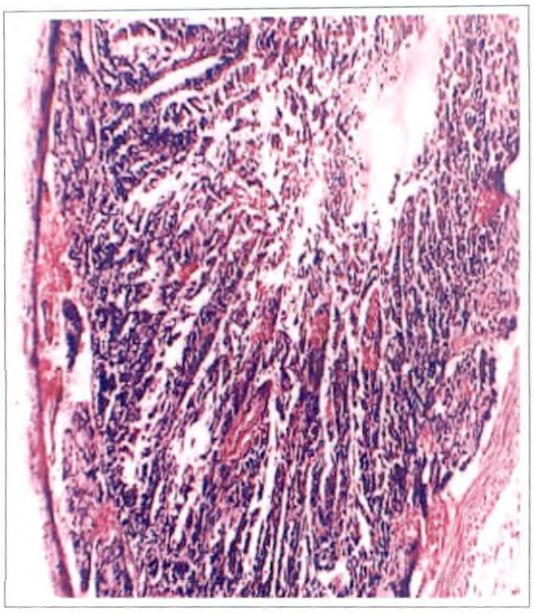

Fig - 14.1
DUODENUM
Hemorrhagic inflammation- H&Ex100

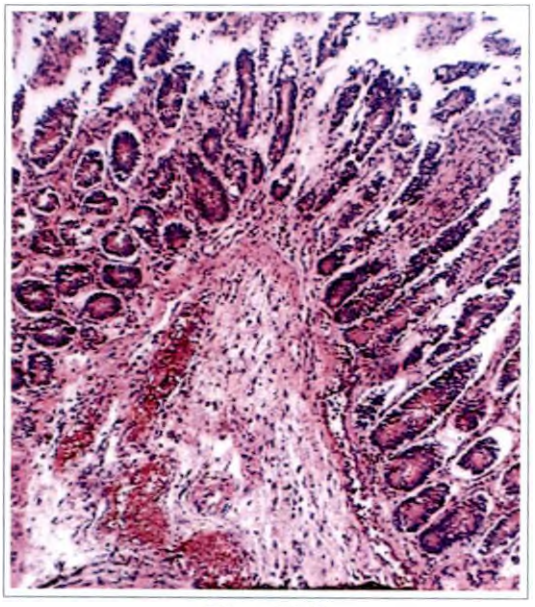

Fig - 14.2
JEJUNUM
Hemorrhagic inflammation- H&Ex100

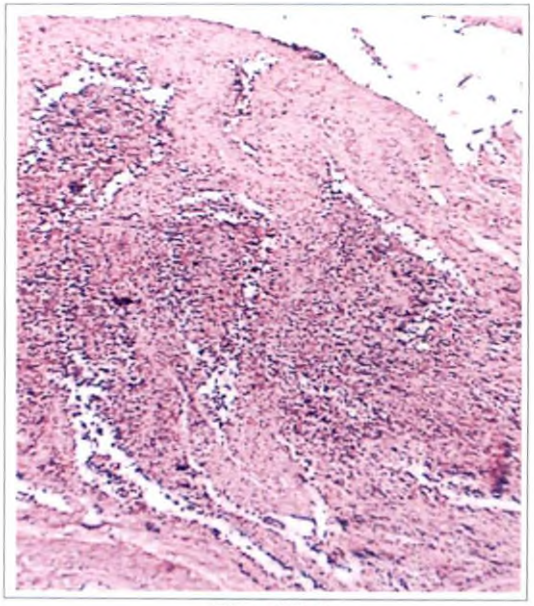

Fig - 15.1
ILEUM
Necrosis and degeneration of mucosa
Necrotic remnants covering the mucosal surface- H&Ex100

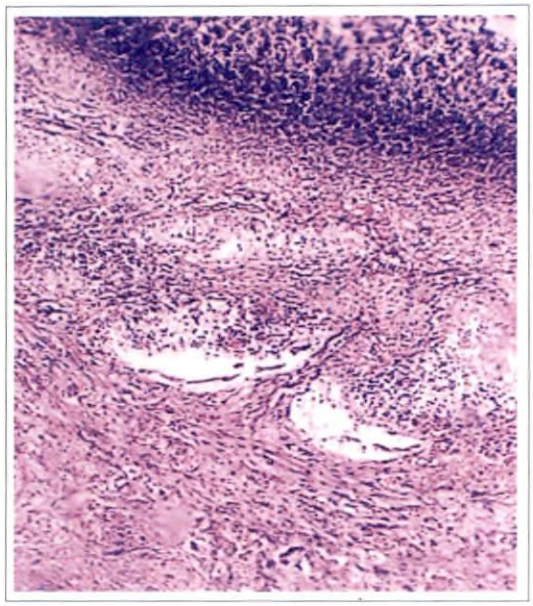

Fig - 15.2
ILEUM
Necrotic inflammation- H&Ex100

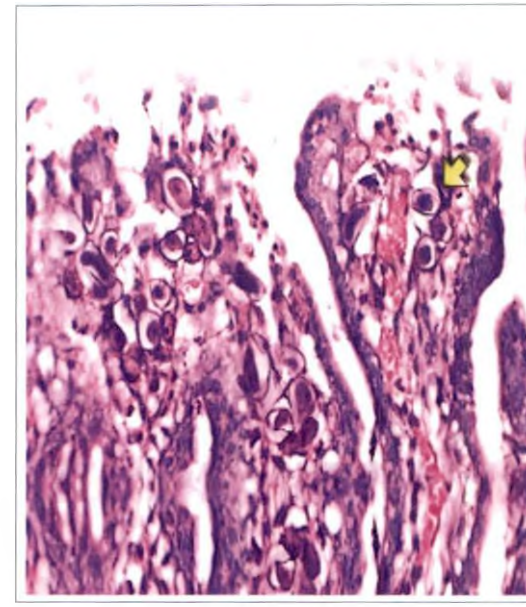

Fig - 16
DUODENUM
Schizonts of coccidia in the
villous epithelial cells- H&Ex400

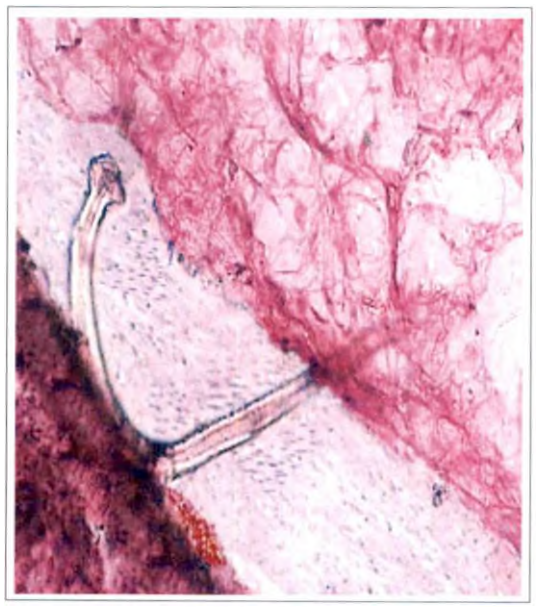

Fig - 17
JEJUNUM
Larva of pinworm in the submucosa-
H&Ex400

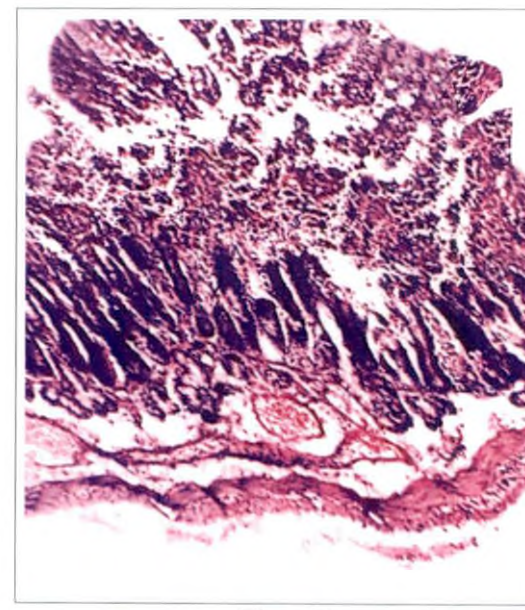

Fig - 18
JEJUNUM
Necrosis of the epithelium
Fibrin covering the mucosal surface-
H&Ex100

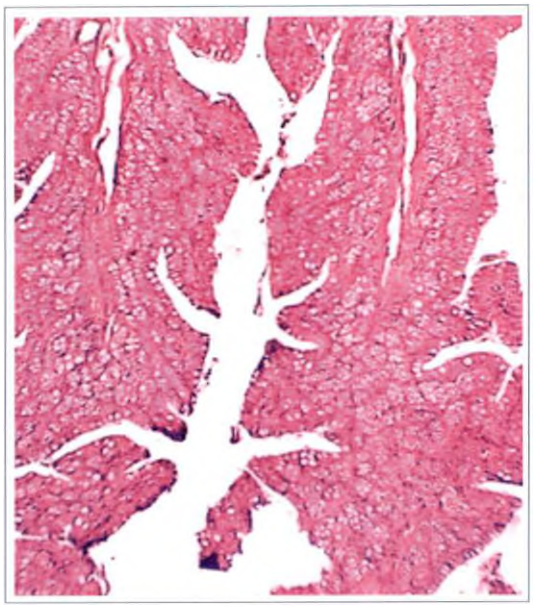

Fig - 19
JEJUNUM
Adenoma
Proliferative growth of villi
Hyperplasia of goblet cells- H&Ex100

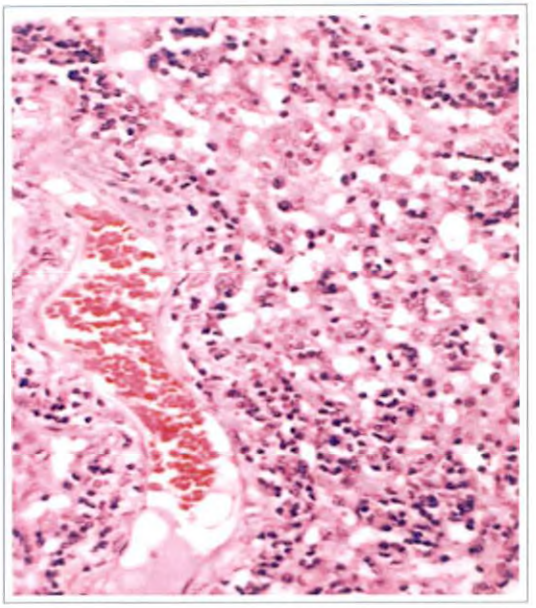

Fig - 20.1
MESENTERIC LYMPH NODE
Congestion-H&Ex400
Lymphocytic depletion

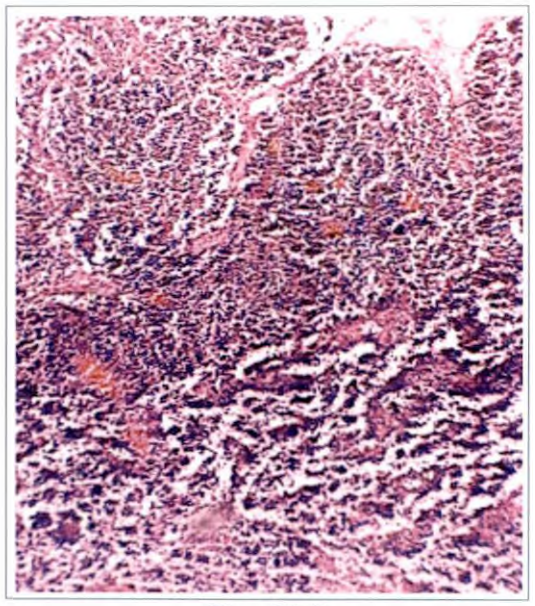

Fig - 20.2
MESENTERIC LYMPH NODE
Haemorrhage-H&Ex100

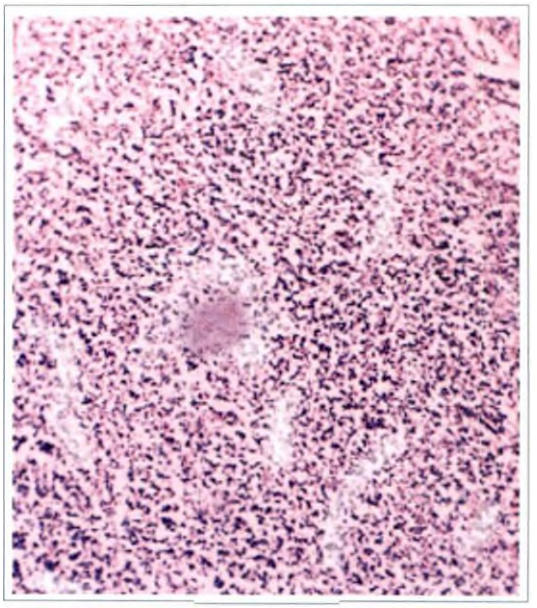

Fig - 20.3
MESENTERIC LYMPH NODE
Necrosis of germinal centre-H&Ex100

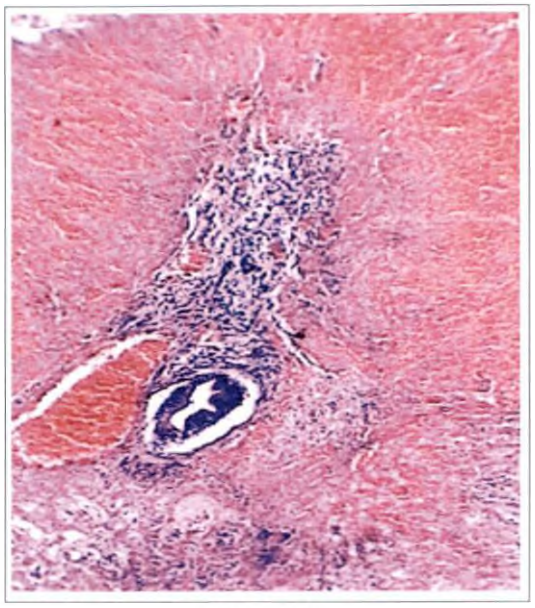

Fig - 21
LIVER
Congestion-H&Ex100
Haemorrhage

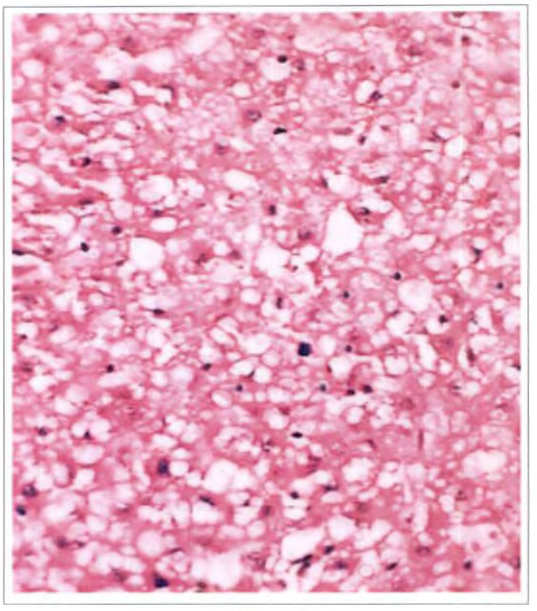

Fig - 22
LIVER
Fatty change-H&Ex400
Fat globules appearing as vacuoles

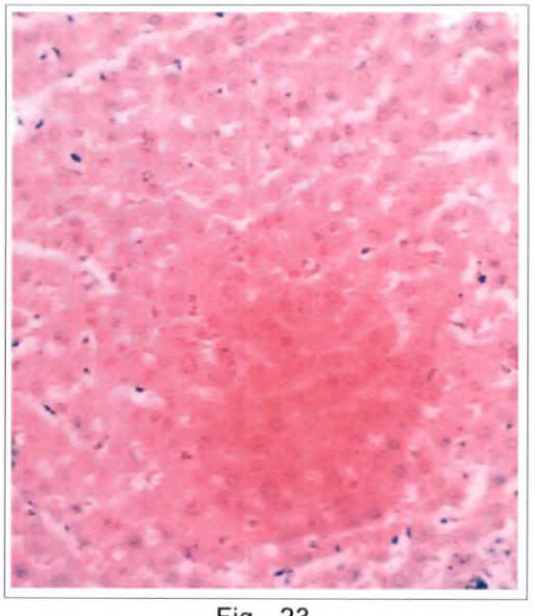

Fig - 23
LIVER
Necrosis-H&Ex400
Cytoplasm of hepatocytes
appearing pink and homogenous

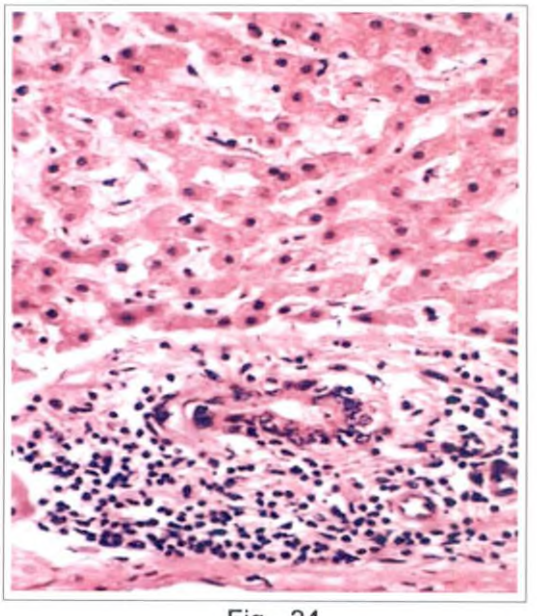

Fig - 24
LIVER
Focal infiltration of
inflammatory cells-H&Ex400

Fig - 25.1
LIVER
Hepatic coccidiosis
Gray white nodules on the surface

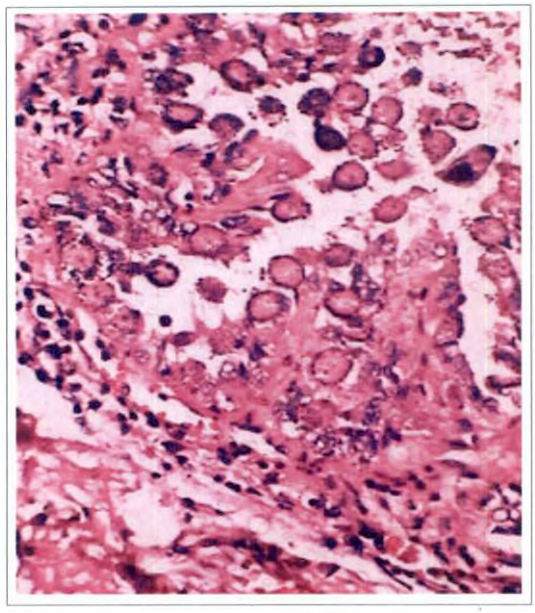

Fig - 25.2
LIVER
Hepatic coccidiosis-H&Ex400
Developing forms of coccidia seen within the bile duct

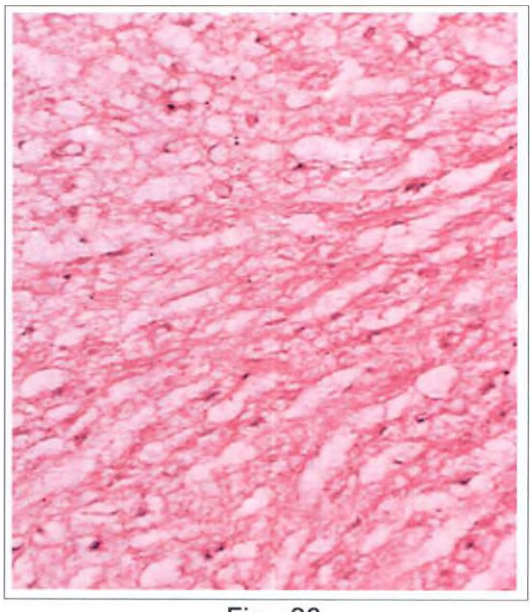

Fig - 26
LIVER
Fibrous tissue proliferation-H&Ex400

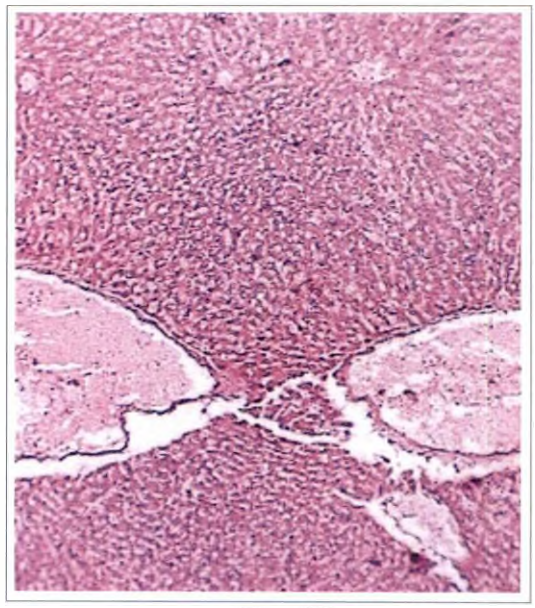

Fig - 27
LIVER
Hepatic cyst-H&Ex100
Multiple cysts containing proteinaceous material inside

Discussion

5. DISCUSSION

The present study was undertaken to assess the prevalence and pathology of gastrointestinal disorders in rabbits. The study not only enabled the categorization of gastrointestinal disorders but also incriminated gastrointestinal disorders as the major cause of mortality among rabbits. Based on the gross and histopathological changes the lesions observed in the gastrointestinal tract, liver and mesenteric lymph nodes observed in fifty rabbits were classified into vascular, degenerative and inflammatory changes.

The predominant vascular changes in stomach included congestion and hemorrhage, which could be due to the stasis in the systemic circulation. Congestion and hemorrhages in the gastric mucosa were seen accompanying other changes like erosions, ulcers and gastritis. Pawaiya *et al.* (1998) reported ecchymoses on the stomach wall in cases of stomach impaction in rabbits. Traumatic injuries that occur when the animals are put on coarse roughages could also be one of the reasons for gastric hemorrhages. Sastry (2001) opined that injuries due to corrosive chemicals, which could damage the capillary endothelium might be an important cause for hemorrhages.

Erosions and ulcers were observed in the stomach of four rabbits, of which three animals showed ulcers in the fundic mucosa. Hinton (1980) also reported a higher incidence of fundic ulcers as compared with pyloric ulcers. Histologically, the lesions were characterized by coagulative necrosis of epithelial layer and capillary hemorrhages in lamina propria. The occurrence of gastric ulcers could be attributed to stress factors, which could induce exaggerated vagal activity, histamine production and hyperacidity. Excessive vagal stimulation could result in vasoconstriction and local ischemia and degeneration of surface mucus cells. The gastric mucus that provides protection against acid digestion of mucosa could get depleted which makes the stressed

rabbits more vulnerable to ulceration. Another factor that renders rabbits more susceptible to ulcers is the nature of stomach mucosa, which responds to stress very rapidly. The rabbit stomach has a relatively more permeable membrane, which is prone to leak even when concentration of intra luminal acid is within the normal range (Kivilaaksa *et al.,* 1978). In the present study, females were found to be more susceptible to gastric ulcers. The higher prevalence of gastric ulcers in female animals could be due to pregnancy, labour, and post parturient complications (Hinton, 1980). In one of the cases, gastric ulcer was seen in association with intestinal coccidiosis. George and Somvanshi (1987) observed a higher incidence of gastric ulcers in female rabbits and animals with coccidiosis and *Encephaltizoon cuniculi* infection.

Inflammatory reactions seen in the stomach was mostly of catarrhal type. The gastric contents in such cases were slimy and mucosa was congested. Microscopically, there was proliferation of goblet cells, which could be due to the metaplastic transformation of gastric epithelial cells in response to chronic irritation. This would inturn lead to increased secretion of mucus, which protects the mucosa from the irritant to some degree. But the increased mucus secretion produces a delay in digestion allowing the putrefactive break down of the ingesta. The abnormal digestion may cause further inflammation and favours spread and persistence of infection (Radostitis *et al.*, 2000). Stress, spoiled and coarse food materials, faulty dentition that prevents mastication can all predispose the animal to gastritis. The necrotic enteritis that was seen in one of the cases could also be due to the exaggerated and prolonged action of the above factors. In the present study, though attempts were made, no pathogenic agent could be isolated from cases with gastritis. Pawaiya *et al.* (1998) reported the isolation of *E. coli* and *Klebsiella* from rabbits with gastroenteritis.

The pathological lesions in different segments of intestine were also studied and classified. Inflammatory lesions dominated, followed by vascular and degenerative changes. Among inflammatory changes, catarrhal enteritis was

the most prominent change. Diarrhoea was the hallmark of almost all the affected cases. One of the major causes could be intestinal parasitism. In thirteen cases, various developing forms of coccidia were observed in the intestinal villous epithelium. According to Catchpole and Norton (1979) intestinal coccidiosis in rabbits was much frequent and resulted in acute morbidity and mortality. As per Soulsby (1982) coccidiosis in rabbits was responsible for severe ill health and mortality especially in young ones. Macroscopically, the intestines of the affected rabbits were edematous and hyperemic. A few of the cases showed gray white foci on the intestinal mucosa. Histopathological examination revealed hyperplastic intestinal glands, desquamation of the epithelium along with hemorrhage and congestion of capillaries and infiltration of inflammatory cells. Srilatha *et al.* (1993) observed similar lesions in intestinal coccidiosis of rabbits. Pillai and Subramanian (1993) identified *Eimeria magna* as the causative agent in 89 per cent of the cases they studied. Devi *et al.* (1990) reported that the incidence of intestinal coccidiosis had a seasonal influence, with more cases occuring during summer months. Coccidiosis is an important disease as it makes the animal susceptible to other infections. Moreover it can exist sub clinically and hence may go unnoticed. Sharma *et al.* (2006) also reported the incidence of subclinical coccidiosis among rabbits up to a magnitude of 41 percent.

In the present study attempts were made to isolate the pathogenic organisms associated with enteritis and agents like *E. coli* and *Salmonella* were isolated. It was mostly the weanlings that yielded *E. coli* on cultural isolation. Lack of passive immunity through colostrum and weaning stress could be the possible reasons. This was in agreement with the observation of Yuill and Hanson (1985) who reported an increased susceptibility for juvenile rabbits to coliform enteritis when compared with adults. The histological changes noticed in such cases included hyperplasia of goblet cells, infiltration of inflammatory cells and submucosal or serosal edema of the small intestine. Since *E. coli* and *Salmonella* are the natural inhabitants of alimentary tract, their roles in the

causation of disease are highly controversial. The rabbit's defenses are apparently sufficient to control these organisms under all but extremely unfavourable circumstances. Beltz *et al.* (2005) isolated *E. coli* from the rabbits showing lesions of mucoid enteritis complex and they concluded that diarrhoea in rabbits had a multifactorial etiology, probably due to change in feed, bacterial proliferation and co-infection with enteric virus. Peeters *et al.* (1978) opined that proliferation of enteropathogenic *E. coli* was associated with a high carbohydrate and low fibre diet. The sudden change in diet may result in destabilization of the cecal microflora and proliferation of enteropathogenic organisms. Songer (1996) observed that destabilization of cecal microflora could be an important initiation factor for enterotoxemia of rabbits caused by *Clostridium spiroformi.* All these necessitate careful selection of feed for rabbits and routine monitoring for enteropathogens.

Hemorrhagic enteritis was observed in twelve cases. Histopathologically, haemorrhages, necrosis of the villi and infiltration of mononuclear cells were noticed. *Salmonella* and *E. coli* were isolated from two cases with hemorrhagic enteritis. Srilatha *et al.* (1993) also reported the isolation of. *E coli,* from the luminal contents of rabbits with hemorrhagic enteritis.

Verminous enteritis was observed in one of the cases where the larva of pinworm was found to have penetrated the submucosa of the jejunum. Gentz *et al.* (1995) identified that the pinworm, *Passalurus ambiguus* could cause constipation, impaction and intussusception in rabbits.

Mesenteric lymph nodes showed histopathological lesions like lymphoid depletion, washed out appearance of germinal centres, lymphoid necrosis and extreme congestion of vessels. Most of these lesions occurred in combination with other gastrointestinal pathologies. Savage and Sheldon (1973) reported necrosis of mesenteric lymph nodes of weanling rabbits with acute diarrhoea. Kesavan *et al.* (2005) observed lymphocytic depletion from Peyer's Patches, mesenteric lymph nodes and other primary lymphoid organs in rabbits that had

undergone corticosteroid therapy and in animals with severe protein malnutrition. Lymphocytic depletion was suggestive of immunodeficiency, which further increased the susceptibility of affected animals to infection. Lymph node lesions could also be due to stress, parasitic infestations and antibiotic therapy.

Hepatic lesions were seen in 58 per cent cases, out of which 50 per cent occurred in combination with other gastrointestinal lesions. The lesions were classified under degenerative, vascular and inflammatory changes. Degenerative changes varied from a mild cloudy swelling to extreme necrosis. Degeneration and necrosis was present in 55 per cent of the cases with liver lesions. The degenerative changes in liver could be associated with toxicities and poisoning wherein the etiological agent could directly disrupt the functional anatomy and physiology of the hepatocytes. The probable cause could be moldy feed considering the fact that rabbits are highly susceptible to even traces of aflatoxin in the feed. Reddy and Rao (2001) observed degeneration, hepatocytomegaly and bile duct proliferation of rabbit livers in aflatoxicosis. They also observed malformation in developing fetuses of affected dams. This warrants screening of rabbit feed for aflatoxins periodically.

Inflammatory changes included focal and diffused hepatitis. Fibrosis of the liver seen in two cases could be attributed to some chronic irritation and the livers in such cases were firmer and harder in consistency. Lazarus and Volk (1964) reported fibrous tissue proliferation in rabbits with protein malnutrition. According to Sastry (2001) fibrosis could also occur in animals with long standing enteric toxemia. *Pasteurella* organism isolated from the liver tissue in two of the cases indicated a septicemic spread. Hepatic cysts containing proteinaceous material were noticed in three cases. One among them revealed the presence of multiple cysts accompanied by other changes like fatty degeneration of surrounding hepatocytes and mild congestion of blood vessels. According to Reddy *et al.* (2000) formation of cystic spaces was the characteristic lesion in livers of rabbits in aflatoxicosis.

Hepatic form of coccidiosis was observed in four cases. Grossly, there was hepatomegaly and numerous yellow white spots on the liver surface. According to Gentz *et al.* (1995) the focal gray nodules on the liver surface of rabbits with hepatic coccidiosis corresponded to hyperplastic bile ducts. The carcasses of rabbits with hepatic coccidiosis were emaciated and jaundiced similar to the observations made by Hampson (1986). Histopathologically, the various developing forms of coccidia like oocysts, gametocytes and schizonts were observed in the hepatic parenchyma. Degenerative and vascular lesions predominated in the surrounding parenchyma. The affected livers showed focal fatty degeneration and necrosis. The developing coccidial forms were seen within the bile ducts where they induced hyperplastic changes. The occlusion of bile ducts caused by the hyperplasia of bile duct epithelium produced stasis of bile, which could account for the gross discoloration of the liver in affected cases. The irritation produced by the organism within the hepatic tissue induced fibrous tissue proliferation.

The present study has thus highlighted the necessity for an in depth investigation into the roles of a host of factors involved in the etiology of gastrointestinal disorders of rabbits. The findings of the study have vividly pointed out the relative roles of parasites, bacterial agents and other stress factors in inducing gastrointestinal disturbances. Selection of feed for rabbits also deserves special attention. The feed should be well balanced with adequate concentrate to supplement the sufficient crude protein and adequate roughage to provide the necessary crude fibre. An imbalance between these two constituents is capable of bringing about cecal destabilization, which could further exacerbate enteropathogenic bacterial proliferation. The need for timely deworming should be well appreciated and considered for successful rabbit rearing. Careful and regular monitoring for common bacterial pathogens like *E.coli, Salmonella* and *Pasteurella* would definitely reduce rabbit mortality to a considerable extent. Role of stress elements in inducing immunosuppression and subsequent infections should be clearly identified and strategies aimed at ruling them out

need to be adopted. Sudden change in climate and environment, improper housing and lack of hygiene can all predispose the animal to diseases. Hence all these factors need to be taken into account in order to ensure profitable rabbit rearing.

Summary

6. SUMMARY

An investigation was undertaken to assess the prevalence and pathology of gastrointestinal disorders in rabbits. The study was based on a detailed gross and histopathological examination of fifty rabbit carcasses brought for autopsy at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy. Microbiological and parasitological examinations were conducted in suitable cases.

Autopsy reports between March 2006 and May 2007 revealed a prevalence of gastrointestinal disorders in rabbits to an extent of 52 per cent making it abundantly clear that disorders of gastrointestinal system in rabbits are more than what is generally expected.

Age wise and sex wise comparison of the lesions were made and it was found that weanlings were more susceptible to gastrointestinal disorders. A loss of passive immunity acquired through colostrum, weaning stress, lowered immune status and further infections have been incriminated as the causes. In the present study, a greater incidence of gastrointestinal disorders was observed in female rabbits as compared with males.

Out of the fifty cases examined, 42 per cent showed pathological changes in the stomach, 74 per cent in the intestine, 30 per cent in the mesenteric lymph nodes and 58 per cent in the liver. The lesions encountered in the stomach were congestion (29 per cent), hemorrhage (10 per cent), degeneration and necrosis (29 per cent), ulcers and erosions (14 per cent) and inflammation (28 per cent). The inflammatory conditions observed were catarrhal gastritis and necrotic gastritis.

The lesions observed in different segments of the intestine were also studied and classified. The lesions in the duodenum were congestion (24 per cent), haemorrhage (five per cent), degeneration and necrosis (22 per cent), catarrhal duodenitis (32 per cent), hemorrhagic duodenitis (14 per cent) and

coccidiosis (19 per cent). Jejunum showed 46 per cent congestion, eight per cent hemorrhage, 81 per cent jejunitis and five per cent ulcer. In the ileum, the inflammatory changes were associated with depletion, degeneration and necrosis of Peyer's Patches. Lesions in the large intestine especially colon, were congestion, degeneration and necrosis, and catarrhal colitis.

Varying degrees of changes in liver and mesenteric lymph nodes were seen in association with gastrointestinal disorders. The liver lesions encountered were congestion (48 per cent), haemorrhage (21 per cent), cloudy swelling (28 per cent), fatty change (17 per cent), hepatitis (41 per cent), coccidiosis (14 per cent), fibrosis (six per cent) and hepatic cyst (10 per cent). In some cases bile duct hyperplasia was also seen along with fatty change. All these lesions could have been the result of toxic injuries.

Thirty per cent of the cases showed changes in the mesenteric lymph nodes. The lesions included congestion, hemorrhage, depletion, degeneration and necrosis of germinal centres. Lesions in the lymph node were suggestive of immune deficiency and in this regard the role of immunosuppression in the spread of disease has been discussed.

Microbological examination was attempted in nine cases. The isolates obtained were *E. coli, Salmonella and Pasteurella* and the roles of these pathogens in the etiology of gastrointestinal disorders have been explained. Stress, immunosuppression and subsequent infections have been highlighted as the causes.

The present investigation has thus helped to focus attention on the various disorders that occur commonly in rabbits. The results of the present study highlights the need for further investigations, into the relative roles of the immune status, stress factors and pathogens in the causation of diseases in rabbits.

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PATHOLOGY OF GASTROINTESTINAL DISORDERS OF RABBITS

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ABSTRACT

The present study was undertaken to assess the prevalence and pathology of gastrointestinal disorders in rabbits. The results of the study revealed a high incidence of gastrointestinal disorders in rabbits. The study was based on a detailed postmortem examination of fifty rabbit carcasses. The gross and histopathological lesions were closely observed and classified based on age and nature of changes. Vascular lesions were predominant in the stomach whereas inflammatory conditions outnumbered vascular and degenerative changes in the intestine. In the stomach the most conspicuous vascular change was congestion while catarrhal gastritis was the prominent inflammatory lesion. Catarrhal enteritis was the commonly observed pathology in the intestine. Parasitic as well as bacterial agents were found to be associated with the lesions. The most important bacterial pathogens isolated were *E. coli* and *Salmonella*. Intestinal coccidiosis was also observed in a considerable number of cases. Various developing forms of *Eimeria spps* were identified in the sections of duodenum and jejunum in affected cases. Verminous enteritis was observed in one case where the larvae of pinworm was seen penetrating the submucosa of the jejunum. Mesenteric lymph nodes of fifteen cases revealed vascular, degenerative and necrotic changes of varying degrees. Depletion of lymphocytes associated with degeneration and necrosis of germinal centers in lymphoid follicles of mesenteric lymph nodes were all indicative of immune suppression. Lymphoid depletion was also evident in the Peyer's Patches of ileum. Liver samples of thirty animals showed pathological changes, some in combination with gastrointestinal lesions. *Pasteurella* was isolated from the liver in two cases with hemorrhagic tracheitis. Hepatic coccidiosis was observed in certain cases, which were histologically characterized by bile duct hyperplasia, fatty degeneration and vascular changes. The higher incidence of gastrointestinal disorders, the role of immune suppression and isolation of different pathogens indicated the necessity for an indepth study on the multi factorial etiology associated with gastrointestinal disorders in rabbits.