PATHOLOGY OF GASTRO - INTESTINAL DISORDERS IN PIGLETS

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DECLARATION

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

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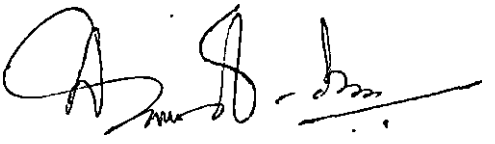
Certified that the thesis, entitled **"PATHOLOGY OF GASTRO-INTESTINAL DISORDERS IN PIGLETS"** is a record of research work done independently by **Dr. S. Smitha,** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to her.



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Introduction

1. INTRODUCTION

The ability of pigs to convert plant material and animal by-products into high quality palatable human food is widely recognized and as a result pig farming has gained much importance. It's prolific breeding capacity, fast growth rate, better feed conversion efficiency and omnivorus habits are adapted for economical production and for these reasons pig raising is adopted as a remunerative adjunct to the farming of other livestock and is becoming popular among small farmers in Kerala.

Profitability in pig farming is largely dependent on the survivability of piglets upto marketing age. A major problem encountered by the farmers engaged in pig farming is the loss of pigs very early in life as a result of various diseases. The mortality of piglings should be considered as a serious problem since it adversely affects the success of any piggery enterprise. Although, several disorders affect piglets, gastro-intestinal disorders are extremely important as they not only cause mortality but also lead to poor growth efficiency.

Eventhough a variety of symptoms can be caused by gastro-intestinal disorders, diarrhoea is the hall mark of intestinal dysfunction. Diarrhoea and poor growth in the period immediately after weaning continue to be important causes of reduced performance in the pig industry. Neonatal diarrhoea accounts for increased mortality and reduced growth rates. Helminths, protozoa, inflammatory disorders, gastrointestinal irritants and a variety of pathogenic bacterial and viral organisms have been incriminated as the cause.

Many workers in India and abroad have reported heavy mortality of piglets and have given considerable importance for the infectious causes in relation to piglet mortality. The infectious agents that affect piglets in the age group of 0 to 56 days as reported by Glock (1981) were mainly *Escherichia coli* (1 day – post weaning), *Clostridium perfringens* type C (1-14 days), *Treponema*

hyodysenteriae (7 days – adult), Transmissible gastroenteritis virus (1 day – adult), Rotavirus (1 day – post weaning) and *Isospora suis* (5 – 15 days).

A preliminary survey of records in the Pig Breeding Centre, Mannuthy, indicated that the highest mortality among pigs was in piglets (92.78 per cent) in the age group of 0 to 2 months, than in the adult pigs (7.22 per cent).

Survey of records of piglet autopsies done at the Centre of Excellence in Pathology for a period of five years from 1997-2001 indicated that gastro-enteritis was the major cause of mortality (78.9 per cent), followed by pneumonia (13.04 per cent).

Comprehensive studies attempting to explore the different aspects of the various conditions causing gastro-intestinal disorders and mortality in piglets have not been undertaken so far in Kerala. Therefore an attempt has been made to investigate the pathological disorders affecting the gastro-intestinal system in piglets with the following objectives.

1. Identify the common gastrointestinal disorders that are responsible for the mortality among piglets.
2. Identify the lesions in the gastrointestinal tract.
3. Identify the bacterial and parasitic pathogens.
4. Provide awareness among clinicians on the common gastrointestinal disorders in piglets.
5. Suggest suitable preventive and curative measures to reduce piglet mortality.

Review of Literature

2. REVIEW OF LITERATURE

2.1 PREVALENCE

The mortality rate in piglets upto eight weeks of age in 2,581 litters in England, surveyed during a period of two years, was 25.9 per cent (Anon, 1959). Eighty per cent of the losses occurred before the end of the first week of life. Mortality in pigs over two months was estimated to be less than 2 per cent.

Two thousand nine hundred and thirty six dead piglets examined by Svendsen *et al.* (1975) revealed that about 17 per cent incidence was due to gastrointestinal disease.

Postmortem examination carried out in 536 piglets, which died between birth and weaning, revealed that non-infectious conditions such as trauma, starvation and suffocation were the most common causes (Glastonbury, 1977). Bacterial septicaemia and infection with enterotoxic strains of *E. coli* were the most prevalent infectious conditions.

Vrbanac *et al.* (1980) identified that gastro-intestinal disease was responsible for the death of 18 per cent of 6,439 piglets which had died before weaning.

Halgaard (1981) studied the host factors involved in the development of diarrhoea. He observed higher rate of diarrhoea in litters born to gilts than litters born to sows.

Urcelay *et al.* (1984) studied the effect of parity of sow on the incidence of diarrhoea. They observed piglets of young sows (parity ≤ 2) were 1.7 times more likely to develop diarrhoea before weaning than litters born to older sows (parity ≥ 3).

In the 2,426 piglets autopsied, birth to weaning mortality was 11.3 per cent. Out of this, death due to diarrhoea was 1.7 per cent (Spicer *et al.*, 1986).

2.2 PATHOLOGICAL CONDITIONS

2.2.1 Developmental Anomalies

Spicer *et al.* (1986) reported that *atresia ani* was the most common congenital condition affecting the gastro-intestinal tract in piglets.

Common congenital defects in piglets reported by Barker *et al.* (1993) were segmental anomalies of the intestine, *atresia coli, atresia ilei* and *atresia ani* of the lower gastro-intestinal tract. Very rarely, persistence of Meckel's diverticulum was also noticed in piglets.

2.2.2 Degeneration and Necrosis

Wilcock (1979) described lesions of salmonellosis in piglets. These included haemorrhagic necrosis of the mucosa extending to the muscularis mucosa in the small and large intestine, necrosis in the mesenteric lymphnodes and diffuse fatty change of the hepatocytes with widely scattered microgranuloma associated with focal hepatic necrosis. Very rarely, there was fibrinoid necrosis of the small vessels at the tip of the eroded villi.

Ultrastructural observations in the Porcine Epidemic Diarrhoea Virus (PEDV) infection revealed considerable degeneration of the villus enterocytes (Ducatelle *et al.*, 1981; Pospischil *et al.*, 1981; Sueyoshi *et al.*, 1995).

In *Escherichia coli* infections, Penrith *et al.*, (1995) described extensive necrosis of the ileal and colonic mucosa with fusion of the villi and loss of crypts of the ileum and necrosis of the gut associated lymphoid tissue of the ileum and colon. There was occasional single cell necrosis of the hepatocytes in the liver.

Manjunatha *et al.* (1995) described the gross and microscopic lesions in colibacillosis. This consisted of occasional degeneration and desquamation of the lining epithelial cells and atrophy of the villi, characterized by short and stumpy villi with a broad base, in the ileal mucosa. Rarely gastroenteritis of haemorrhagic or necrotic type were noted.

Lesions in *Salmonella cholerae suis* infection reported by Manjunatha *et al.* (1995) were severe congestion, multifocal areas of ulcers or necrosis along with diphtheritic deposits in the ileum and haemorrhage in the colon.

Kim and Chae (2000) studied piglet diarrhoea caused by PEDV and observed microscopic lesions like moderate to severe multifocal to diffuse villus atrophy, in the distal portion of the jejunum and proximal portion of the ileum. Villi were often fused and covered with degenerated or regenerated flattened epithelium. Rarely villi lacked an epithelial lining. Vacuolated enterocytes were seen on the tip of the villi or spread over the entire villi in the jejunum. Moderate number of exfoliated enterocytes were seen in the scattered villi. The caecum and colon showed vacuolation of the superficial enterocytes.

Poutahidis *et al.* (2001) conducted studies on piglets infected with *Helicobacter pylori*. The infected piglets exhibited severe congestion, microerosive lesions and haemorrhage of the gastric glandular mucosa.

2.2.3 Inflammation

Wilcock (1979) studied salmonellosis in piglets and observed infiltration of neutrophilic leukocytes at 60 hours and later mononuclear leukocytic infiltration in the lamina propria, but not in the submucosa. In the liver, widely scattered microgranulomas associated with focal hepatic necrosis were observed. Mesenteric lymphnodes were twice the normal size in salmonellosis infected piglets.

Lomax and Glock (1982) described lesions characteristic of proliferative enteritis such as reduced number of goblet cells, hypertrophy and hyperplasia of the crypt epithelial cells with infiltration of inflammatory cells and presence of necrotic debris in the lumen of the glandular crypts.

Electron microscopic study of swine dysentery by Albassam *et al.* (1985) revealed superficial vascular congestion and dialatation, oedema of the lamina propria and intercellular separation of the epithelial cells at the crypt shoulders. The intercellular fluid had the same electron density as the fluid in the lamina propria and the plasma of the subepithelial blood vessels.

Extensive crypt hyperplasia with infiltration of inflammatory cells was reported by Mapother *et al.* (1987) and Gebhart *et al.* (1994) in porcine proliferative enteritis.

Catarrhal gastroenteritis was the predominant lesion observed by Manjunatha *et al.* (1995) in colibacillosis, Klebsiella and Enterobacter infections.

2.3 INFECTIOUS CONDITIONS

2.3.1 Bacterial Etiology

Stevens *et al.* (1972) reported that piglets were extremely susceptible to *E. coli* enterotoxin, while on milk diet.

Sporadic outbreaks of diarrhoea in piglets have been ascribed to *Clostridium perfringens* type C (Barnes and Moon, 1964; Moon and Bergeland, 1965) or swine dysentery (Harris and Glock, 1973).

Salmonellosis was a major disease of weaned pigs (Moorehead, 1972; Barnes and Sorensen, 1975) but was considered rare in suckling pigs (Barnes and Sorensen, 1975; Wilcock *et al.*, 1979).

Callinan and Russell (1975) studied the etiology and pathogenesis of swine dysentery. The local effects of swine dysentery on the large intestinal

mucosa included congestion of mucosal vessels, followed by fluid exudation into lamina propria, detachment of degenerated epithelial cells and hyperactivity of the goblet cells.

Proliferation of *Treponema hyodysenteriae* within the large intestine of affected pigs caused mucohaemorrhagic diarrhoea in swine dysentery (Harris *et al.*, 1978, Whip *et al.*, 1979).

Klebsiella pneumoniae was isolated from the jejunum of a week old piglet with diarrhoea by Wilcock (1979) and the organism produced heat labile and heat stable enterotoxins in piglets. Grossly the small and large intestine were filled with abnormal amount of fluid and foamy intestinal content. No microscopic lesions were noticed in Klebsiella infection.

Lomax and Glock (1982) isolated *Campylobacter sputorum* subspecies *mucosalis* from the intestinal mucosa of proliferative enteritis affected pigs. The lesions produced by this organism were the proliferation of crypt epithelial cells, crypt elongation, dialatation of lacteals and shortening of the villi. The epithelial cells covering the mucosal surface had an immature appearance, characterized by basophilic cytoplasm and large oval-to-oblong, basally oriented, hyperchromatic nuclei. The epithelial cells lacked a prominent microvillus border.

Diarrhoea in piglets under one week of age was often reported to be caused by *Escherichia coli* (Alexander, 1981; Bergeland and Henry, 1982; Morin *et al.*, 1983).

Mapother *et al.* (1987) experimentally reproduced porcine proliferative enteritis in pigs. *Campylobacter hyointestinalis* and *Campylobacter coli* were isolated from the mucosal scrapping of pigs. Gross lesions noted in the affected pigs included thickening and rigidity of the small intestine with transverse and, or, longitudinal folds running throughout the mucosa and reticulation of serosa. The lesions in the small intestine were loss of villous architecture, moderate

hyperplasia of the epithelial cells lining the crypts, accumulation of crypt debris and absence of goblet cells.

Fairbrother *et al.* (1988) isolated ninetynine nonclassical serogroups of *Escherichia coli* from the newborn pigs with neonatal diarrhoea. They were tested for fimbrial antigens F4(K88), F5(K99), F6(987P), F41 and F165 and for heat-labile enterotoxin, heat-stable enterotoxin a (STa), heat stable enterotoxin b, verotoxin and cytolethal distending toxin. Thirty two strains, belonging to serogroups O64: K "V142", O9:K103 and O20:K101, were F5 positive and usually produced STa.

Driesen *et al.* (1993) reported that the *E. coli* associated with piglet diarrhoea in 30 days age group were the same 'O' serotype as those that caused neonatal diarrhoea.

Manjunatha *et al.* (1995) isolated *Treponema* organism from the colon of diarrhoeic piglets. The section of the colon stained by Warthin-starry silver stain showed jet black coloured spirochaetes present in the crypt lumen and glandular component.

Cooper and Gebhart (1998) observed lesions produced by *Lawsonia intracellularis* in piglets. This included segmental thickening of the intestine, mucosal hyperplasia, oedema and necrosis in affected piglets.

Poutahidis *et al.* (2001) demonstrated that *Helicobacter pylori* induced a severe lymphocytic gastritis in piglets.

Segales *et al.* (2001) studied the proliferative enteropathy complex of pigs caused by *Lawsonia intracellularis* and observed marked proliferation of the ileal and caecal crypts, with intense epithelial hyperplasia and complete absence of goblet cells in the affected pig. Most of the intestinal crypts were dialated and contained mucus, inflammatory cells (mostly neutrophils) and cellular debris. Peyers patches showed mild to moderate depletion of lymphocytes. The

superficial mucosa showed necrosis, haemorrhage and fibrin exudation. Degenerating inflammatory cells were present in the ileum and caecum.

2.3.2 Parasitic Etiology

Ten day old piglets experimentally infected with coccidial oocysts showed symptoms like profuse yellowish diarrhoea, dehydration, weight loss and death (Stuart *et al.*, 1980). Gross lesions were characterized by a fibrinous necrotic membrane in the mucosa of the jejunum and ileum. Histologically there was villous atrophy and variable erosions with an adhered necrotic membrane. Atrophic villus in the duodenum was characterized by the presence of male and female gamonts within villous epithelium. Merozoites and meronts were present in the villus epithelium of the jejunum.

Morin *et al.* (1983) reported that coccidiosis was most often observed in piglets in the age group of 5-15 days.

Henriksen and Christensen (1992) identified oocysts of *Isospora suis* in more than 35 per cent of faecal samples or postmortem material from 856 piglets of the age group 7 to 14 days. Affected piglets showed diarrhoea which had a pasty consistency.

Driesen *et al.* (1993) reported that *Isospora suis* was the most common enteropathogen affecting piglets (7 to 14 days) in 35.8 per cent of single and 18 per cent of mixed infections.

2.3.3 Viral Etiology

In Porcine Epidemic Diarrhoea Virus (PEDV) infected cases, morbidity and mortality among neonatal piglets approached 100 per cent as a result of severe diarrhoea and dehydration. Mortality among infected piglets older than 10 days was less than 10 per cent (Chasey and Cartwright, 1978; Pensaert and Debouck, 1978; Jimenez *et al.*, 1985).

Faecal samples of five piglets with diarrhoea revealed particles with ultrastructural features typical of calci, astro and rotaviruses (Bridger, 1980).

Dea *et al.* (1985) isolated numerous parvovirus like particles from the faeces of diarrhoeic piglets from six Quebec pigherd.

Garwes (1988) identified transmissible gastroenteritis virus in the epithelium covering the tip of the villi in the jejunum and ileum of diarrhoeic piglets. The infected cells were shed and replaced by cells that migrated up from the crypts. This process was accompanied by shortening of the villi and lengthening of the crypts.

Driesen *et al.* (1993) examined faecal samples from 2,380 diarrhoeic piglets, 5 to 30 days of age and reported that 16.9 per cent were infected with rotavirus.

Concurrent infection with porcine circovius-2 was reported to occur along with other viral, chlamydial or protozoan pathogens (Carrasco *et al.*, 2000).

Kim and Chae (2000) reported that Porcine Epidemic Diarrhoea Virus caused acute enteritis in swine of all ages and that it was often fatal for neonatal piglets.

Hirai *et al.* (2001) noted that neonatal piglets in 420 piggeries developed diarrhoea and died within two or three days after birth. Porcine Epidemic Diarrhoea Virus, TGE and porcine rotavirus were identified histochemically from these cases, using sections of the small intestine, and procine circovirus using sections of the mesenteric lymphnodes.

2.3.4 Chlamydial Etiology

Macroscopic lesions in 4-5 week old piglets experimentally inoculated with *Chlamydia* were seen in the ileum and caecum (Pospischil and Wood,

1987). The ileal wall showed extensive thickening along with focally prominent fibrinous typhlitis.

2.4 NEOPLASM

Glock (1981) reported that congenital neoplasia was rare among piglets and accounted for a minor percentage of findings in surveys of neonatal mortality.

2.5 MISCELLANEOUS

Wilson (1974) observed that conventionally the unweaned piglets were protected by specific and nonspecific inhibitors present in the sow's milk, which exerted a protective effect locally in the small intestine.

A field trial was conducted by Ward and Bigland (1976) to evaluate the effectiveness of formalin-treated live *Escherichia coli* vaccine in the prevention of neonatal diarrhoea in piglets. They observed that in the non-vaccinated groups piglet mortality was 2.15/sow and in the vaccinated group it was 0.93/sow.

Urcelay *et al.* (1984) demonstrated the association of diarrhoea with other variables like dam, breed, sire, gestation groups, gestation length, size of litter, number of mummies, still birth per litter, farrowing barn, or day of the week farrowed was weak or non-existent.

Spicer *et al.* (1986) showed in their study that there was no significant difference observable in the number of scouring litters while comparing first and older parity sows.

Mezoff *et al.* (1991) observed that the binding capacity of the enterocytes of three week old weaned pigs for *E.coli* with heat stable toxin a (STa) was nearly three times greater than that of the unweaned pigs. Guanylate cyclase activation was greater in weaned pigs than unweaned pigs.

Experimental inoculation of shiga like toxin I from *Escherichia coli* into two to seven days old piglets resulted in widespread endothelial cell swelling and necrosis in the submucosa, the muscular layer of the large intestine and the mesocolon (Dykstra *et al.*, 1993). Arterial endothelial swelling was noticed in the fundic region of the stomach.

The study conducted in pre-weaning piglets by Driesen *et al.* (1993) revealed no seasonal variations in the incidence of piglet diarrhoea.

Gastric venous infarction was a common lesion in pigs affected by salmonellosis and *Escherichia coli* septicaemia. It was related to endothelial damage and thrombosis in the venules (Barker *et al.*, 1993).

Nabuurs *et al.* (1994) reported that in all weaned and unweaned piglets infected with enterotoxic *E. coli*, the net absorption of fluid and electrolytes was significantly less in the proximal part than in the distal part of small intestine.

Steenhard *et al.* (2002) demonstrated interaction between intestinal nematodes and *Salmonella typhimurium* in pigs. They observed that pigs with dual infection excreted significantly higher amount of *S. typhimurium* in faeces, compared with nematode free pigs. Dual infected pigs also excreted *S. typhimurium* on more days than nematode free pigs.

Materials and Methods

3. MATERIALS AND METHODS

The present study was conducted at the Centre of Excellence in Pathology and Pig Breeding Centre, College of Veterinary and Animal Sciences, Mannuthy, to investigate the prevalence and pathology of the various disorders of the gastrointestinal system of piglets in the age group of 1 to 56 days.

3.1 MATERIALS

3.1.1 Data Collection

Data regarding the prevalence of mortality among pigs for a period of five years from January 1997 to December 2001 were obtained from the records available in the Pig Breeding Centre, College of Veterinary and Animal Sciences. The prevalence of the gastro-intestinal disorders in piglets before the weaning age was also obtained during this period from the post mortem records kept in the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy.

The details regarding the carcasses of piglets brought for autopsy to the Centre of Excellence in Pathology during the period of the study (December 2001 to May 2003) were documented in the proforma prepared (Appendix-1).

3.1.2 Sample Collection

A total of fifty carcasses of piglets brought for autopsy to 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 lymphnodes were collected for gross and histopathological investigation.

Faecal samples were screened for any parasitic ova.

Materials for bacteriological studies were collected from sow's milk, liver, heart blood and intestinal contents of piglets in appropriate cases.

3.2 METHODS

3.2.1 Analysis of the Data

A preliminary survey of records in the Pig Breeding Centre, Mannuthy was conducted for a period of five years to evaluate the mortality pattern in pigs. Age-wise and sex-wise classifications were done.

Post mortem records kept at the Centre of Excellence in Pathology were studied for a period of five years. Age-wise and sex-wise classifications were also done.

Age-wise, sex-wise and breed wise details of carcasses (Appendix-I) brought for autopsy to Centre of Excellence in Pathology during the period of the study were obtained and recorded in the proforma prepared. The lesions were classified based on the age and sex.

3.2.2 Gross Examination

A detailed systematic postmortem examination of the piglet carcasses brought for autopsy was conducted. The cases were in the form of fresh carcasses. The liver and mesenteric lymphnodes were carefully studied for gross lesions like change in size, shape, colour and consistency and presence of cysts, abscesses or tumours. Gross lesions of the stomach, small intestine and large intestine were also examined. Representative samples were collected for histopathological and microbiological examination.

3.2.3 Histopathological Examination

Representative samples of the liver, mesenteric lymphnodes, stomach, small and large intestines obtained from the carcasses were fixed in 10 per cent neutral buffered formalin. They were then processed and paraffin embedded as described by Sheehan and Hrapchak (1980). The sections were stained with Haematoxylin and Eosin as per the technique followed by Bancroft and Cook (1984). Special staining techniques like Periodic Acid Schiff's (PAS) for the demonstration of glycogen, Perl's technique for haemosiderin, Van Gieson's for collagen fibres and Oilred O for fat were done as and when required, as per the methods described by Luna (1968). The sections were examined in detail under light microscope and lesions were classified.

The lesions were scored under six primary markers viz., vascular disturbances, degenerative and necrotic changes, inflammatory changes, proliferative changes, neoplastic changes and other disorders.

3.2.4 Microbiological Studies

Bacterial isolation was attempted from the sow's milk, liver, heart blood and intestine from fresh carcasses of piglets. Identification of the organism was done using microbiological kit (HiIMViC Biochemical Test Kit KB001).

3.2.5 Parasitological Examination

Intestinal contents were collected and screened by direct smear examination for any parasitic ova.

Results

4. RESULTS

4.1 INCIDENCE

Analysis of the data on the mortality pattern among pigs in the Pig Breeding Centre, Mannuthy indicated that the incidence of gastrointestinal disorders in piglets was about 78 per cent. Out of the 2,241 cases studied during the period from 1997 to 2001, highest mortality was during the first week (35.4 per cent). In older piglets (above 2 months of age) it was only 7.22 per cent.

Data on the incidence of gastro-intestinal disorders collected from post mortem reports maintained at the Centre of Excellence in Pathology, showed that out of 1,455 cases studied during the period 1997 to 2001, 86.4 per cent revealed gross lesions in the gastro-intestinal tract and 21.8 per cent in both the liver and the gastro-intestinal tract.

Samples of stomach, small intestine, large intestine, mesenteric lymphnodes and liver from 50 cases of piglets autopsied at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy, between December 2001 to May 2003 were examined. Out of these, 76 per cent showed pathological changes in the stomachs, 88 per cent in the intestine, 60 per cent in the liver and 56 per cent in the mesenteric lymphnodes. The lesions were classified based on the age (Table I) and sex (Table 2). The percentage of incidence of GI tract lesions was found to be more in the age group of 10-20 days and females showed more lesions in the GI tract as compared to the male.

Table 1. Age wise distribution of lesions in the stomach, large/small intestine, liver mesenteric lymphnodes

Group	Total	No. with stomach lesions		No. with liver lesions		No. with intestine lesions		No. with mesenteric lymphnode lesions	
		No.	%	No.	%	No.	%	No.	%
1-4 days	11	7	63	7	63	11	100	5	46
5-10 days	7	5	71.4	3	43	5	71	4	57
10-20 days	18	17	94	13	72	18	100	13	72
20-30 days	6	5	83	2	33	4	66	3	50
30-56 days	8	4	50	5	63	6	75	3	38
	50	38	76	30	60	44	88	28	56

Table 2. Sex wise distribution of lesions in the stomach, liver, large/small intestine and mesenteric lymphnodes (CHL. N)

Sex	Total	No. with Stomach lesions		No. with Liver lesions		No. with Intestine lesions		No. with M.L.N. lesions	
		No.	%	No.	%	No.	%	No.	%
Male	22	15	68	13	59	19	86	13	59
Female	28	23	82	17	61	25	89	15	54

4.2 CLASSIFICATION OF LESIONS

4.2.1 Stomach

Seventy six per cent of cases revealed lesions in the stomach. The lesions in most cases occurred in combination with the other lesions.

4.2.1.1 Vascular Changes

4.2.1.1.1 Congestion

Eighteen out of 38 cases with stomach lesions (47 per cent) showed congestion. Grossly, the mucosal and serosal vessesls were prominent. The mucosa appeared in varying shades of red. In majority of the cases the congestion was confined to the fundic region. Histopathologically, mucosal, submucosal and serosal blood vessels were severely engorged and packed with erythrocytes.

4.2.1.1.2 Haemorrhage

Two out of 38 cases (15 per cent) affected, revealed haemorrhage. Grossly, the organ exhibited patchy reddish areas in the mucosa. Microscopically, there was extravasation of blood in the *foveolae gastricae* and in the *lamina propria*.

4.2.1.2 Degeneration and Necrosis

Seven out of 38 cases (18 per cent) showed degeneration and necrosis of mucosa. Grossly, there was peeling of the mucus membrane at some region and moderate congestion of the mucosa.

Microscopically, the gastric mucosa showed a variety of changes of varying intensities. In the milder form, there was degeneration of the surface epithelium and multiple epithelial cell death deep in the glands. Increase in the depth of foveolae gastricae, fusion of foveolae, loss of surface epithelium, cystic

dilatation of the upper tunica mucosa and glandular atrophy were seen in few cases (Fig. 1). In two cases, there was complete degeneration and necrosis of the glandular epithelium and mild oedema of the lamina propria, loss of architecture, moderate dilatation of glandular crypts, necrosis of the gastric mucus cells, chief cells and parietal cells, presence of necrotic remnants within the crypts (Fig.2). Collection of macrophages and few lymphocytic aggregations in the lamina propria were the other lesions observed. Severe necrosis of the glandular structures with cystic dilatation of the glandular crypts containing cell debri as well as protenaceous material (Fig. 3) was seen in one case. In all these cases, the histological lesions were confined to the tunica mucosa. Dilatation of the lymphatics, congestion of the vessels of the lamina propria, foveolae gastricae and tunica muscularis mucosa were often seen.

4.2.1.3 Ulcer

Out of the 38 cases having stomach lesions, five (13 per cent) showed erosions and ulcers of varying size (Fig. 4). The size of the ulcers varied from 0.5 to 1 cm in diameter. Erosions and ulcers were diffuse, but predominant in the corpus and antrum of the stomach. The mucosa of the unaffected region appeared thickened. Erosions appeared very small involving the superficial mucosa. Ulcers appeared circumscribed, extending deep into the mucosa and dirty brown or grey with raised and hyperaemic borders. Histopathologically, there was discontinuity of the gastric foveolae, loss of epithelium, congestion, glandular damage and disappearance (Fig. 5). The area was covered with an exudate consisting of mucus, moderate amount of fibrin, degenerated and destroyed glandular epithelium and few inflammatory cells. The ulcers appeared extending deep involving the entire tunica mucosa.

4.2.1.4 Inflammation

Six out of 38 cases (16 per cent) revealed catarrhal gastritis and one each superficial necrotic gastritis and chronic gastritis.

4.2.1.4.1 Catarrhal Gastritis

The stomach was distended with mucus mixed contents. The mucosa was hyperaemic which appeared reddish brown and swollen. This occurred as a diffuse condition or as patchy zones. Microscopically, there was epithelial desquamation, hyperaemia, oedema and thrombosis of the capillaries of lamina propria, widening of the foveolae gastricae and their fusion and crowding of cells in certain region and glandular epithelial damage. The glands appeared shrunken and diminished in number and degenerated. Mononuclear cell infiltration was observed in the lamina propria, within gastric crypts and in the tunica muscularis (Fig. 6 and 7). Hyperplasia of the goblet cells (Fig. 8) and intense submucosal hyperaemia was also noticed.

4.2.1.4.2 Superficial necrotic gastritis

This was observed in one case. Grossly, there was congestion and accumulation of dirty material focally in the mucosa. Severe degeneration, necrosis and loss of superficial epithelium, gastric glandular cells, fusion of foveolae, infiltration of inflammatory cells in the superficial region of the tunica mucosa (Fig. 9), dilatation of lymphatics of lamina propria and mild oedema of the lamina propria were the characteristic microscopic lesions.

4.2.1.4.3 Chronic gastritis

Chronic gastritis was seen in one case. Grossly, there was moderate thickening of the gastric mucosa along with moderate congestion. Microscopically, there was complete loss of superficial epithelium and glandular structures which was replaced by proliferated fibrous tissue of the lamina propria (Fig. 10). There was thickening of the tunica muscularis mucosa and mononuclear cell infiltration. Remnants of glandular structures appearing as dilated spaces without lining or containing cellular debri were observed amidst the fibrous tissue.

4.2.2. Small Intestine

Eighty eight per cent of the cases showed intestinal lesions. Region wise evaluation of the lesions was done and were classified as vascular changes, degeneration and necrosis and inflammatory changes.

4.2.2.1 Duodenum

4.2.2.1.1. Vascular changes

4.2.2.1.1.1 Congestion

Twenty nine out of 44 cases (66 per cent) showed congestion of varying degrees. Grossly, duodenal mucosa appeared red. Microscopically, blood vessels in the mucosa, submucosa and serosa were engorged with erythrocytes.

4.2.2.1.2 Degeneration and necrosis

There was degeneration and necrosis of the surface epithelium in 27 cases (63 per cent). The degenerative and necrotic changes were seen both in the tunica mucosa and in the submucosa. In the mucosa, it was observed in the epithelium of the plicae circulare and cells of the crypts of Lieberkuhn (Fig. 11). The degenerated epithelium denuded into the lumen. In some areas the villi appeared flattened, stunted and fused. Atrophy of the villi characterized by short stumpy villi with a broad base was frequently observed (Fig. 13). The Brunners gland was intact in many cases, but in few cases the glands appeared degenerated. Degeneration and desquamation of the epithelium lining the glands, cystic dilatation of glands with or without homogenous pink staining materials within was also observed (Fig. 14). Congestion of the vessels, dilatation of lymphatics along with moderate oedema of the submucosa were also seen.

4.2.2.1.3 Ulcer

Ulcer in the duodenum was noticed in two cases. Only a single ulcer was noticed in the duodenal region in both cases. It was about 0.2 – 0.3 cm in

diameter with yellowish necrotic borders. Microscopically, there was complete loss of mucosa (Fig. 15), exposing the lamina muscularis mucosae. Villi in the surrounding area appeared completely flattened and fused. Moderate degeneration of the Brunners gland close to this lesion and congestion of the submucosa and muscular layers were also observed.

4.2.2.1.4 Catarrhal duodenitis

Twenty seven cases (47 per cent) showed catarrhal type of inflammation. Grossly the duodenum was flaccid and dilated. The mucosa was hyperaemic and lumen contained excess mucus. Epithelial desquamation, mucosal infarcts, haemorrhage, hyperaemia, oedema of the lamina propria, infiltration of mononuclear cells in the mucosa and submucosa, goblet cell hyperplasia both in the tip of the villi and crypts of Lieberkuhn, fusion of the villi and moderate degeneration of the Brunners glands were the characteristic microscopic lesions (Fig. 16). In some cases the glands were shorter and diminished in number. The crypts appeared dilated and cystic.

4.2.2.2 Jejunum

4.2.2.2.1 Vascular changes

Forty one out of 44 cases (93 per cent) showed congestion of blood vessles in mucosal and submucosal layer. Severe congestion of blood vessels was noticed in four cases (Fig. 17).

4.2.2.2.2 Degeneration and necrosis

Thirty one cases showed degeneration. The gross lesion observed was moderate congestion of the mucosa. The microscopic lesion varied from simple vacuolar degeneration of the villus epithelium in a few cases to complete loss and necrosis of the enterocytes of villi (Fig. 18). In few other cases, there was denudation of the necrotic villi which appeared as clumps within the lumen. Fusion of the necrotic villi with necrosis of the crypts of Lieberkuhn, cystic

dilatation of crypts with loss of lining cells were observed in few other cases (Fig. 19). The submucosal vessels were congested and there was dilatation of lymphatics.

4.2.2.2.3 Ulcer

Ulcer was observed in the jejunum in one case. The ulcers were multiple and deep with raised hyperaemic borders (Fig. 20). In some cases necrotic debri was seen attached to the ulcers. Microscopically, in some ulcers there was complete loss of mucosal and submucosal layer exposing the muscular layer, where as in others there was irregular loss of surface epithelium and extensive necrosis of the villi and crypts appearing as fused homogenous pink staining debri containing fibrinous materials (Fig. 21). The necrosis extended deep into the submucosa. In these cases, the crypts were exposed and few of the vessels of the lamina propria and submucosa, there was fibrinoid degeneration. The surviving glands appeared shrunken and atrophic. Moderate infiltration of inflammatory cells were seen in the lesion. *Salmonella cholerae suis* was isolated from these lesions.

4.2.2.2.3 Jejunitis

Thirty three out of 44 cases showed inflammatory changes. Grossly, the entire mucosa was highly congested and the lumen contained excess mucus. The mucosa showed irregular thickening. Microscopic lesions varied from congestion and haemorrhage of the lamina propria and crypts, goblet cell hyperplasia and moderate mononuclear cell infiltration in the lamina propria (Fig. 22) to very extensive cystic dilatation of crypts of Lieberkuhn, pericapillary and periglandular mononuclear cell infiltration (Fig. 23). The endothelial cells in certain regions appeared swollen and necrotic. The dilated glandular crypt contained cellular debri and mucin. Denudation and desquamation of villus epithelium, fusion of the villi and infiltration of mononuclear cells were also

observed (Fig. 24). Thrombosis of the capillaries of the villi, dilatation of the lymphatics, crypts and fat emboli were seen in some cases.

4.2.2.3 Ileum

4.2.2.3.1 Vascular changes

4.2.2.3.1.1 Congestion

Out of 44 cases 38 (86 per cent) showed congestion of mucosal and submucosal blood vessels. Microscopically, the vessels were engorged with erythrocytes.

4.2.2.3.2 Degeneration and necrosis

Forty one out of 44 cases (93 per cent) showed degeneration and desquamation of the villi epithelium. In most of the cases, there was complete shedding of the epithelium covering the villi, fusion of the villi and necrosis of the cells of crypts of Lieberkuhn (Fig. 25). Thirty two out of 44 cases (72 per cent) revealed depletion of lymphocytes from the Peyer's patches. In some of the cases with submucosal oedema and congestion, the follicles in the Peyers patches had a washed out appearance (Fig. 26). In certain other cases there was diffuse depletion of lymphoid follicles followed by necrosis and caseation (Fig. 27).

4.2.2.3.3 Ulcer

Ulcer was noticed in one case. Multiple ulcers were noticed in the mucosa (Fig. 20). The ulcer was about 0.3 – 0.4 cm in diameter with yellowish necrotic border. Microscopically, there was complete loss of mucosal and submucosal layer exposing the muscular layer. Villi in the surrounding area were fused completely and appeared as necrotic eosinophilic remnants (Fig. 28). Congestion of the vessels of the crypts, lamina propria and necrosis of the crypts of Lieberkuhn were observed. No frank inflammation was seen.

4.2.2.3.4 Ileitis

Thirty three cases (75 per cent) showed catarrhal type of inflammation. Grossly, the mucosa appeared diffusely congested and the content was watery and mixed with mucus. There was scattered petechial haemorrhages. The microscopic lesion in some cases revealed fusion of the villi, shedding of the villus epithelium, congestion of the vessels of lamina propria and crypts, goblet cell hyperplasia and inflammatory cell infiltration (Fig. 29). In some other cases there was destruction and necrosis of the villi, necrosis of crypts of Lieberkuhn and extensive infiltration of mononuclear cell in the mucosa. Lymphoid depletion, dissolution of the lymphoid architecture of Peyer's patches and caseation of the follicles were also observed (Fig.27). In one case there was sinus histiocytosis.

4.2.2.4 Large intestine

4.2.2.4.1 Vascular changes

4.2.2.4.1.1 Congestion

Twenty three out of 44 cases (52 per cent) showed congestion of blood vessels in the mucosa and submucosa. In most of the cases it was observed in the colon.

4.2.2.4.2 Degeneration and necrosis

There was degeneration and desquamation of surface epithelium in 19 cases (43 per cent). Microscopic examination revealed degeneration and shedding of the epithelium covering the glands in certain areas. Necrosis and desquamation of the superficial epithelium which appeared as aggregates were seen in few cases.

4.2.2.4.3 Inflammation

4.2.2.4.3.1 Catarrhal colitis

Catarrhal type of inflammation was noticed in 22 cases. Grossly, the large intestine was filled with copious of mucus. Goblet cell hyperplasia, congestion of the vessels of lamina propria and sparse mononuclear infiltration were the microscopic lesions.

4.2.2.4.3.2 Necrotic colitis

Necrotic colitis was noticed only in one case. Grossly, erosions and ulcers were seen in the mucosa of the colon. Microscopically, there was complete destruction of the glandular epithelium and the glandular surface appeared covered by eosinophilic necrotic mass (Fig. 30). Inflammatory cell infiltration in the mucosa was extensive.

4.2.2.4.4 *Atresia ani*

Atresia ani was noticed only in one case. Grossly, the whole intestine was filled with blood (Fig. 31). Microscopically, the villi in small intestine was stunted and fused. There was sparse number of mononuclear cells infiltrating the mucosa of deodenum, jejunum and ileum. There was degeneration and desquamation of surface epithelium in large intestine.

4.2.3 Liver

Sixty per cent of the cases revealed lesions in the liver. The lesions in most cases occurred in combination with the other lesions observed in the gastrointestinal tract.

4.2.3.1 Vascular changes

Vascular changes were classified as congestion and haemorrhage.

4.2.3.1.1 Congestion

Out of the 30 cases with lesions in the liver 17 (56 per cent) had congestion of varying degrees. Grossly, the organ appeared red and the cut surface revealed nut-meg appearance in some cases. In severe cases there was rounding of the edges of the lobes and the organ was firm. Histopathologically, the sinusoids, central veins and portal vessels were dilated and filled with blood. In the severe form, the sinusoidal dilatation and filling with blood were so extensive that there was thinning of hepatic cords.

4.2.3.1.2 Haemorrhage

Three (10 per cent) of the 30 cases revealed haemorrhages of varying degrees. Grossly, the organ exhibited pin point areas of petechiae or patchy reddish areas. Microscopically, the erythrocytes were seen free within the parenchyma either as diffuse collections or aggregates displacing the parenchyma. This was accompanied by fatty changes of hepatocytes also (Fig. 32).

4.2.3.2 Degenerative changes

Grossly, the areas of degeneration appeared as diffuse, pale to yellowish streaks extending into the parenchyma. The cases with severe fatty changes, there was hepatomegaly and the cut surface bulged out.

4.2.3.2.1 Cloudy swelling

This was recorded in 13 cases (43 per cent), out of the 30 cases with lesion. Microscopically, the cytoplasm of the hepatocytes was more eosinophilic and granular. There was hepatomegaly with loss of architecture and obliteration of the sinusoids. Congestion of the sinusoids along with vacuolar degenerative changes were also seen along with cloudy swelling in some of the cases.

4.2.3.2.2 Fatty changes

Three (10 per cent) out of 30 cases of affected liver had fatty change. The hepatic parenchymal cells contained fat globules either as a single large globule or as multiple small globules, displacing the cytoplasm and compressing the nucleus (Fig. 32) which most often appeared distorted. The globules stained red with oil-red-O confirming that they were fat globules. In one of the cases bile duct hyperplasia was observed.

4.2.3.3 Necrosis

Hepatic necrosis was seen in two cases. Grossly, the necrotic areas appeared as greyish white spots or patches extending into the parenchyma. The cut surface appeared mottled, having red and grey zones. Microscopically, both cytoplasmic and nuclear changes were seen. The lesions appeared focal in one and in another case, it was diffused. The nuclei of necrotic hepatocytes were in varying stages of pyknosis, karyorrhexis or karyolysis. The cytoplasm appeared homogenous and pink stained. The normal sharp contour of the cells could not be seen and the cell out lines had disappeared. Necrosis was mostly accompanied by vascular disturbances like petechiae, focal haemorrhages and hydropic degenerative changes.

4.2.3.4 Hepatic cyst

Hepatic cyst was observed in one of the cases with liver lesions. Grossly, the lesion revealed no recognizable changes. On microscopical examination, multiple cysts of varying size with proteinaceous material inside (Fig.33) were seen. The surrounding hepatocytes showed severe fatty change and atrophy.

4.2.3.5 Inflammatory changes - Hepatitis

Seven out of the 30 cases (23 per cent) with liver lesions revealed hepatitis of varying degrees.

4.2.3.5.1 Focal hepatitis

This was observed in three cases. Grossly, the liver had areas of focal yellowish to greyish discolouration extending into the parenchyma. Microscopically, focal collection of mononuclear cells could be seen distributed throughout the parenchyma (Fig. 34).

4.2.3.5.2 Suppurative hepatitis

Suppurative hepatitis was seen in one case. Grossly, the liver was enlarged. Pinpoint whitish areas of developing abscesses distributed diffusely in the parenchyma were seen. Two of the lobes showed extensive lesions of suppuration. The lesion was embedded within the parenchyma. The cut surface revealed granular necrotic inspissated material (Fig. 35). Loss of architecture of the parenchyma, extensive necrosis of hepatocytes, individualization, fragmentation and lysis of hepatic cords along with intense infiltration of neutrophils were observed microscopically (Fig. 36). *Salmonella cholerae suis* was isolated from this case.

4.2.4 Pathology of mesenteric lymphnodes

Twenty eight out of 50 cases examined for gastro-intestinal disorders revealed microscopic lesions in the mesenteric lymphnodes. Congestion of vessels with variable degree of lymphocytic depletion was the most frequent lesion (Fig. 37). This was observed in 20 cases (40 per cent). Degeneration and necrosis of lymphocytes in the germinal centres (Fig. 38), diffuse degeneration of lymphocytes of the medullary areas and moderate sinus histiocytosis were observed in two cases. In one case there was moderate lymphoid hyperplasia of some of the cortical follicles. Thickening of the capsule, congestion of capsular

vessels (Fig. 39), trabecular thickening and diffuse degeneration of lymphocytes were seen in the other cases.

4.3 MICROBIOLOGICAL STUDIES

Heart blood, liver, intestine and sow's milk were taken from animals for bacterial isolation. A total of 17 bacterial isolations were obtained from 11 animals.

Age	Number of cases	Materials used	Organisms isolated	Number of isolates
Day old	1	Heart blood, liver and sow's milk	*Escherichia coli*	3
Day old	1	Heart blood, intestine and sow's milk	*Escherichia coli*	3
3 weeks	1	Heart blood and liver	*Klebsiella pneumoniae*	2
45 days	1	Liver and intestine	*Salmonella cholerae suis*	2
21-28 days	5	Intestine	*Escherichia coli*	5
36 days	1	Intestine	*Salmonella cholerae suis*	1
43 days	1	Intestine	*Klebsiella pneumoniae*	1

4.5 PARASITOLOGICAL EXAMINATION

Intestinal contents from the fifty cases were screened by direct smear examination. No ova of any parasites could be detected in any of the samples.

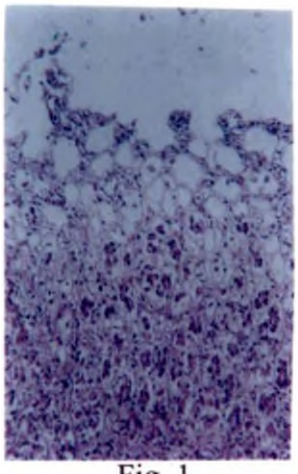


Fig. 1

Fig. 2

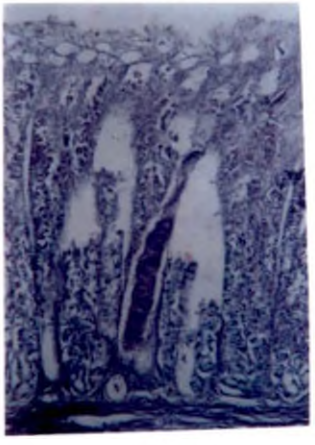


Fig. 3

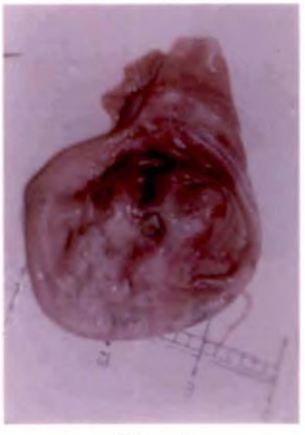


Fig. 4

Figure - 1. Stomach.
Fusion of foveolae, loss of surface epithelium, cystic dilatation of the upper tunica mucosa and glandular atrophy - H & E x 250

Figure - 2. Stomach.
Degeneration and necrosis of the glandular epithelium, dilatation of the glandular crypts and necrosis of the gastric mucus cells - H & E x 250.

Figure - 3. Stomach.
Necrosis, cystic dilatation of the glandular crypts - H & E x 250.

Figure - 4. Stomach.
Erosions and ulcers of varying sizes .

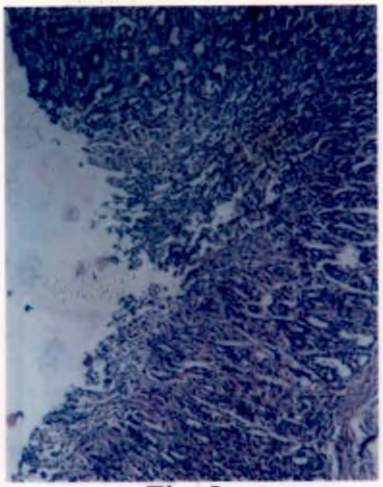


Fig. 5

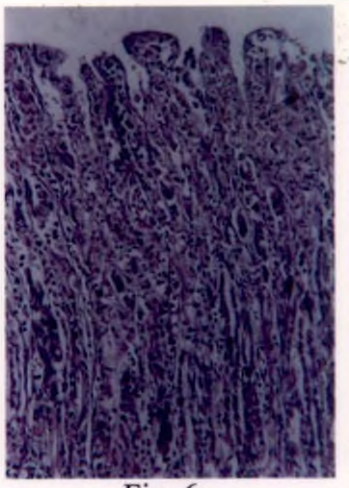


Fig. 6

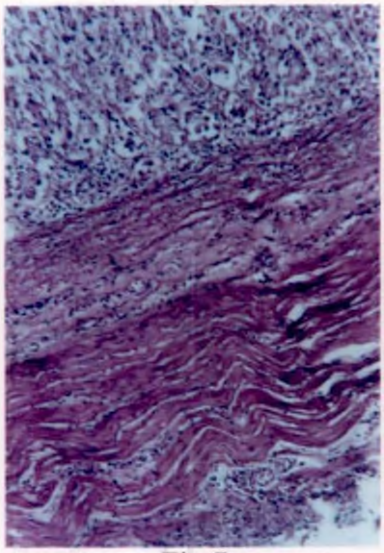


Fig.7

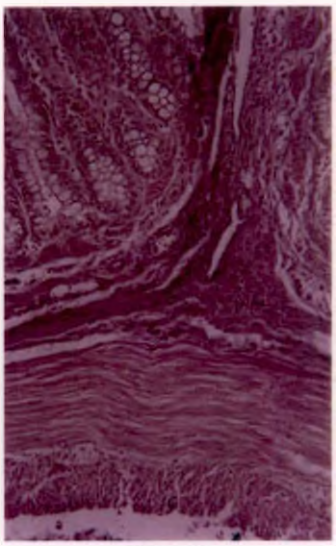


Fig. 8

Figure - 5. Stomach.

Discontinuity of the gastric foveolae, loss of epithelium, glandular damage and disappearance - H & E x 160.

Figure - 6. Stomach.

Mononuclear cell infiltration in lamina propria, within gastric crypts and glandular degeneration of superficial mucosa - H & E x 250.

Figure - 7. Stomach.

Mononuclear cell infiltration in the tunica muscularis - H & E x 250.

Figure - 8. Stomach.

Hyperplasia of the goblet cells - H & E x 250.

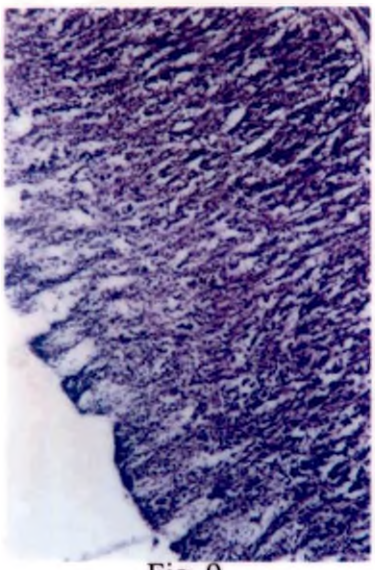


Fig. 9

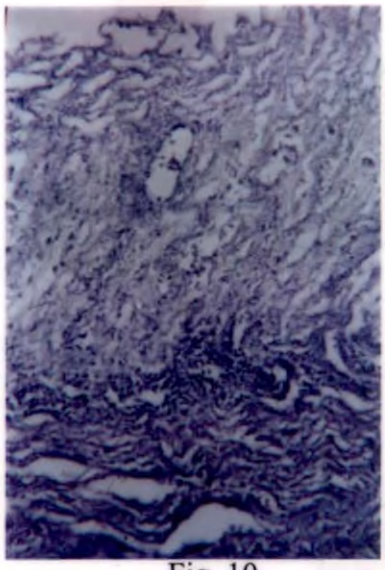
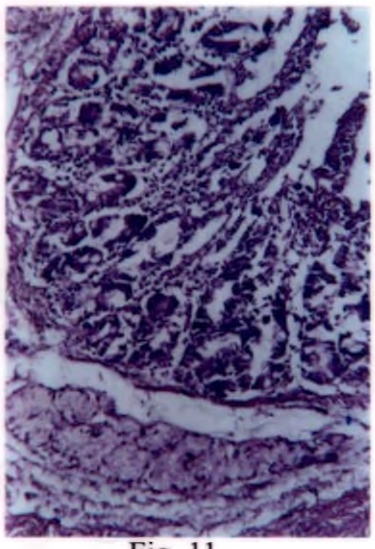


Fig. 10



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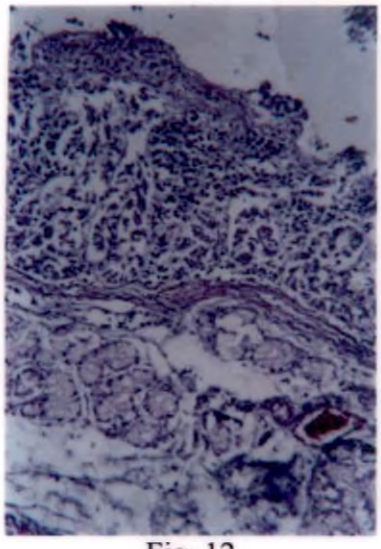

Fig. 12

Figure - 9. Superficial necrotic gastritis.
Necrosis and loss of superficial epithelium, gastric glandular cells, fusion of foveolae and inflitration of inflammatory cells - H & E x 160.

Figure - 10. Chronic gastritis.
Loss of superficial epithelium and glandular structures replaced by proliferated fibrous tissue of the lamina propria - H & E x 250.

Figure - 11. Duodenum.
Degenerative and necrotic changes in the epithelium of plicae circulare and cells of the crypts of Lieberkuhn - H & E x 250.

Figure - 12. Duodenum.
Flattened, stunted and fused villi - H & E x 250.

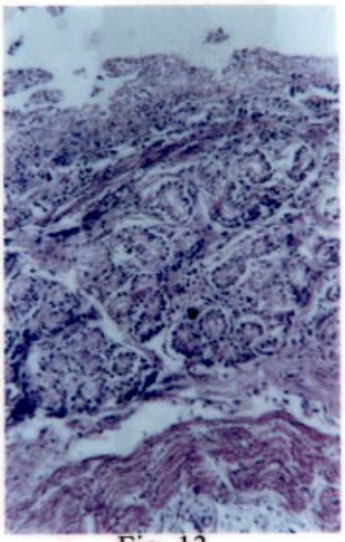

Fig. 13

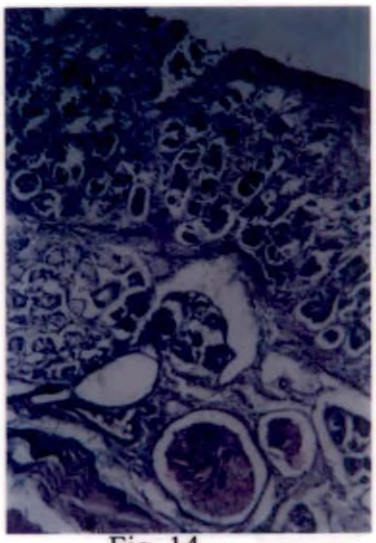

Fig. 14

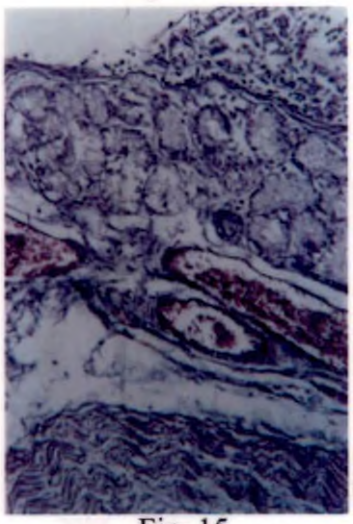

Fig. 15

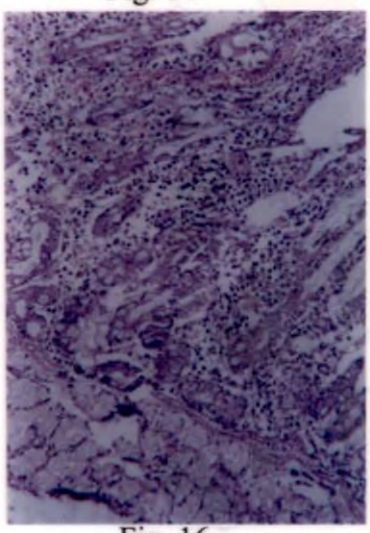

Fig. 16

Figure - 13. Duodenum.
Atrophy of the villi, short stumpy villi with a broad base - H&E x 250.

Figure - 14. Duodenum.
Degeneration and desquamation of epithelial cells lining the glands, cystic dilatation of glands with homogenous pink staining material within - H & E x 250.

Figure - 15. Duodenum - Ulcer
Complete loss of mucosa - H & E x 250.

Figure - 16. Duodenum.
Goblet cell hyperplasia, fusion of the villi and moderate degeneration of the Brunners glands - H & E x 250.

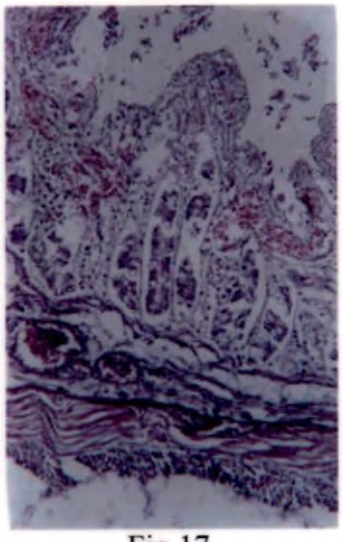

Fig.17

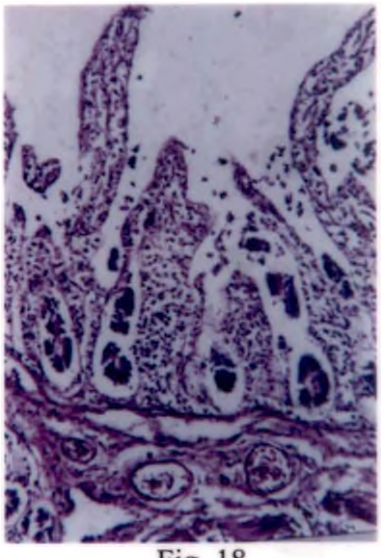

Fig. 18

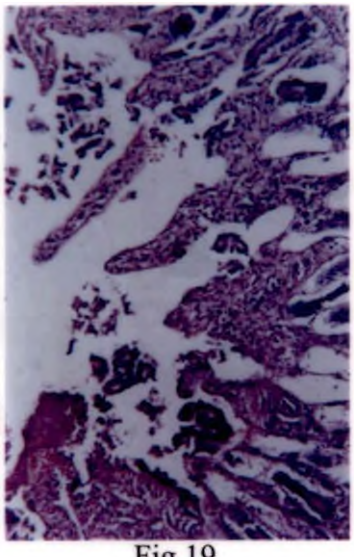

Fig.19

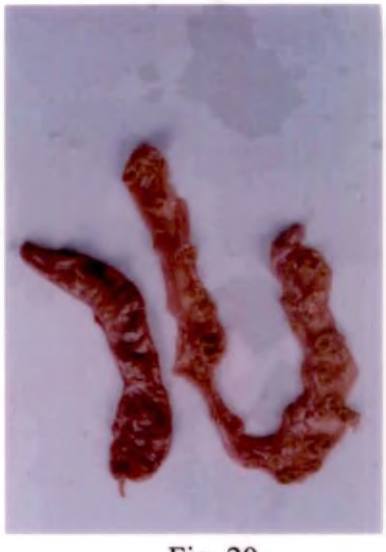

Fig. 20

Figure - 17. Jejunum.
Congestion of blood vessels - H & E x 250.

Figure - 18. Jejunum.
Complete loss and necrosis of the enterocytes of villi - H & E x 250.

Figure 19. Jejunum.
Fusion of the necrotic villi with necrosis of the crypts of Lieberkuhn, cystic dilatation of crypts with loss of lining cells - H & E x 250.

Figure - 20. Jejunum.
Multiple and deep ulcers with raised hyperaemic borders.

Fig. 21

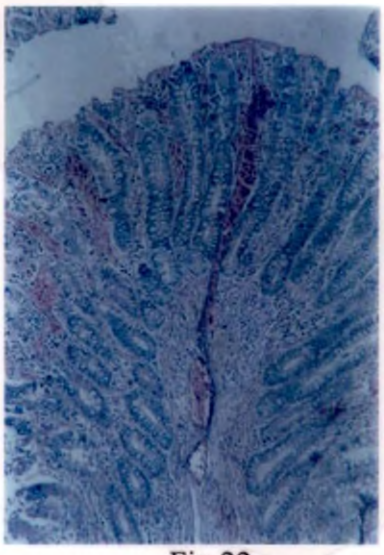

Fig.22

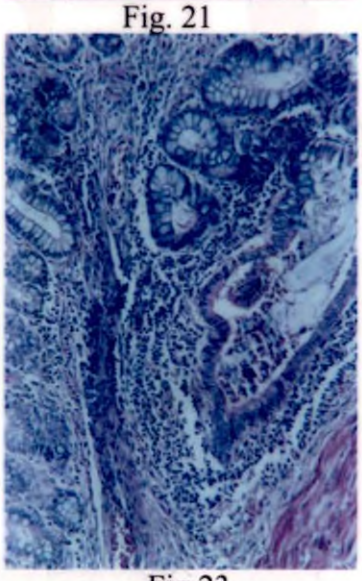

Fig.23

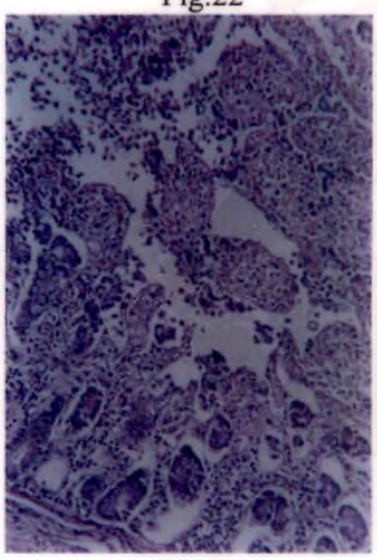

Fig.24

Figure - 21. Jejunum.
Extensive necrosis of the villi and crypts appearing as fused homogenous pink staining debri containing fibrinous materials - H & E x 160.

Figure - 22. Jejunitis.
Congestion and haemorrhage of the lamina propria and crypts, goblet cell hyperplasia and moderate mononuclear infiltration - H & E x 160.

Figure - 23. Jejunitis.
Extensive cystic dilatation of crypts of Lieberkuhn, pericapillary and periglandular mononuclear inflitration - H & E x 250.

Figure - 24. Jejunum.
Denudation and desquamation of villus epithelium, fusion of villi and infiltration of mononuclear cells - H & E x 250.

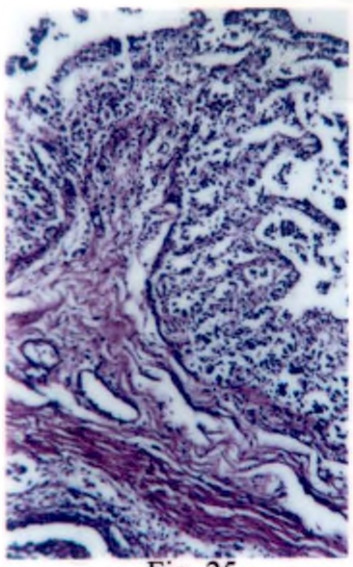

Fig. 25

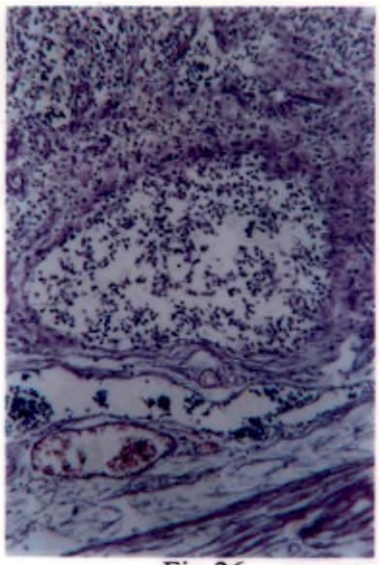

Fig.26

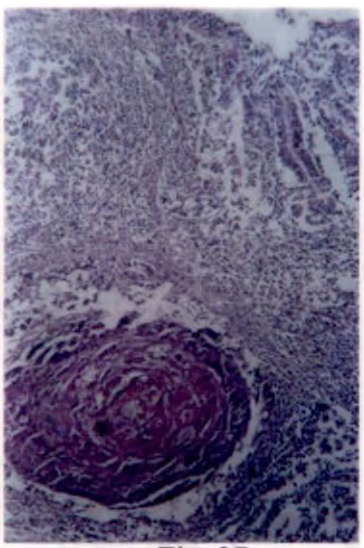

Fig. 27

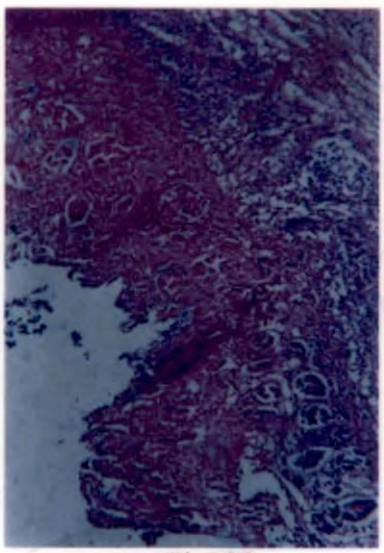

Fig. 28

Figure - 25. Ileum.
Complete shedding of the epithelium covering the villi, fusion of the villi and necrosis of the cells of crypts of Lieberkuhn - H &E x 250.

Figure - 26. Ileum.
Submucosal oedema and congestion, the follicles in the peyer's patches has a washed out appearance - H & E x 250.

Figure - 27. Ileum.
Diffuse depletion of lymphoid follicles followed by necrosis and caseation - H & E x 250.

Figure - 28. Ileum - Ulcer.
Villi in the surrounding area appears fused completely and appears as necrotic eosinophilic remnents - H & E x 160.

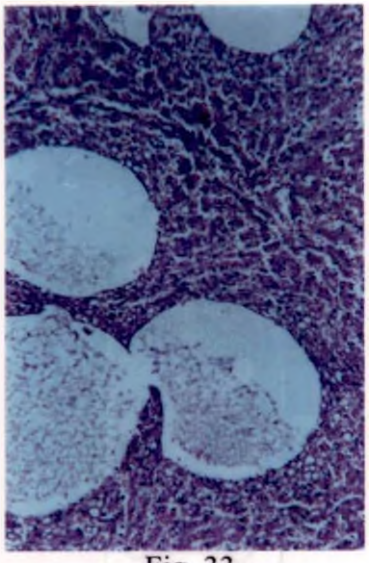

Fig. 33

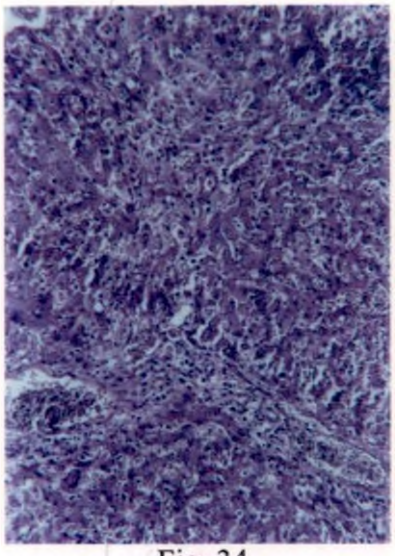

Fig. 34

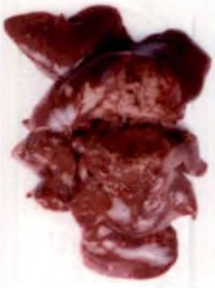

Fig. 35

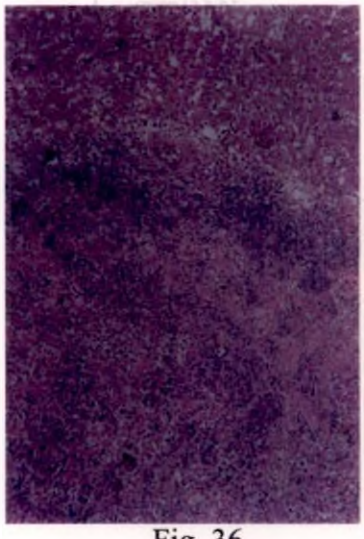

Fig. 36

Figure - 33. Liver.
Multiple cysts with proteinaceous material inside - H & E x 250.

Figure - 34. Liver - Focal hepatitis.
Focal collection of mononuclear cells distributed through out parenchyma - H & E x 250.

Figure - 35. Liver - suppuration - micro abscesses.
Granular necrotic inspissated material in the cut abscess.

Figure - 36. Liver - Suppurative hepatitis.
Loss of architecture of the parenchyma, extensive necrosis of hepatocytes, fragmentation and lysis of hepatic cords with intense infiltration of neutrophils - H & E x 160.

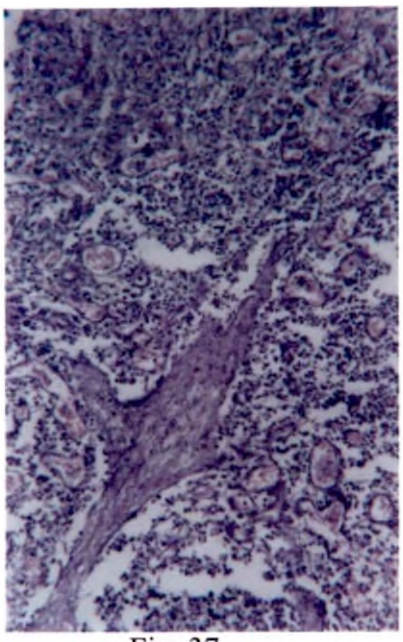

Fig. 37

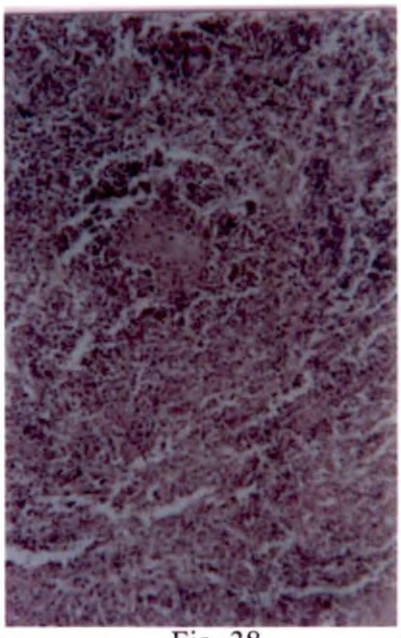

Fig. 38

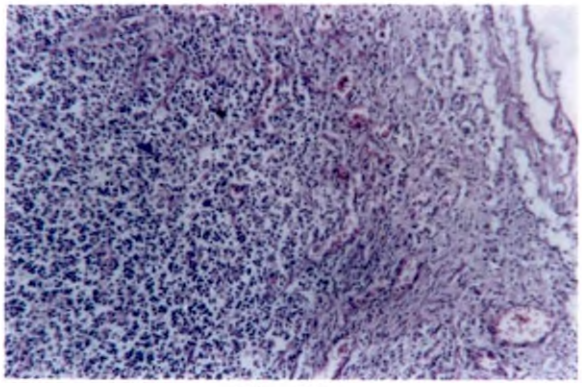

Fig. 39

Figure - 37. Mesenteric lymphnode.
Lymphocytic depletion - H & E x 250.

Figure - 38. Mesenteric lymphnode.
Degeneration and necrosis of lymphocytes in germinal centres - H & E x 250.

Figure - 39. Mesenteric lymphnode.
Thickening of the capsule and congestion of capsular vessels - H & E x 250.

Discussion

5. DISCUSSION

The present study was undertaken to evaluate the prevalence and nature of pathological disorders of gastrointestinal system in piglets. The consistent mortality of piglets born in the farm attached to the College of Veterinary and Animal Sciences, Mannuthy, also necessitated the investigation. The information gathered from this investigation enabled categorization of gastro-intestinal tract disorders and has made it absolutely clear that disorders of gastrointestinal tract of piglets were more than expected.

Analysis of the records maintained at the Pig Breeding Centre and Centre of Excellence in Pathology for the past five years showed 78 per cent and 86 per cent of gastro intestinal lesions respectively. A systematic gross and histopathological study carried out in fifty carcasses of piglets brought for autopsy during the period of study also revealed the presence of pathological lesions in the gastrointestinal tract to the magnitude of 93 per cent, suggesting a similar pattern over these years.

Among the piglets, the incidence of gastrointestinal disorders was high between the age group of 10-20 days. It was followed by animals between the age group of 1-4 days. Urcelay *et al.* (1984) reported similar observations as that of the findings of the present study. According to Driesen *et al.* (1993) gastroenteritis occurred more frequently in piglets between 7 and 14 days of age with a peak age of onset at 10 days, contradictory to the findings of Spicer *et al.* (1986) who stated that maximum death due to gastro-enteritis was more in 2-4 days. The fact that mortality was in the very young groups suggests the possibility of defective immune status of the animals. Neonatal immunity is mainly conferred by the parent and passive immunity acquired through colostrum is lost by second or third week. The critical period is after this stage till the immune system is endowed with the capability to build up it's antibody profiles

and hence the increased incidence. The large number of litters born and the competition between them for sucking milk leaves some piglings immunodeficient and ultimately they die of gastro-enteritis. The gastrointestinal lesions and the lesions in the liver and mesenteric lymphnodes observed in 50 piglings were categorized based on the gross and histopathological changes. Degenerative and necrotic lesions predominated, followed by vascular and inflammatory changes which could be associated with hypoxic injuries, infections and toxicities. In all cases, the degenerative and necrotic lesions were predominant in the tunica mucosa involving the epithelium and the glands. Congestion, haemorrhages and thrombosis observed accounted for these degenerative and necrotic changes.

Erosions and ulcers were seen in the stomach in five cases. Ulcers either single or multiple were also observed in the duodenum, jejunum and ileum of a few cases. Congestion, focal haemorrhages, disturbed nutrition of the epithelium leading to their necrosis and digestion by the gastric juice may be attributed to the development of ulcers. Gastric ulcers occurs as a result of imbalance between the erosive capability of the low gastric pH and protective mechanisms of the gastric mucosa. Preservation of adequate mucosal blood flow and the presence of an intact bicarbonate rich layer of mucous over the epithelium are essential in maintaining the resistance of the epithelium and digestion of gastric acid and pepsin which in part are dependent on the normal prostaglandin E concentration in the mucosa. Factors that inhibits prostaglandin E production such as ischemia contribute to the development of ulcers (Radostitis *et al.*, 2000). The necrotic and fibrinoid type of material seen at the erosive lesion in the present cases indicate that whatever may be the primary factors causing mucosal damage, there is secondary mucolysis by acid and pepsin. Excessive gastric acid production, depletion of the gastric buffering system resulting in prolonged activation of pepsinogens and changes in the mucus composition are factors related to gastric ulcer in swine (Poutahidis *et al.*, 2001). According to Barbosa *et al.* (1995) *Gastropirillum suis* that inhabit the stomach should be considered as possible

factor correlated with etiopathogens of gastric ulcers. Poutahidis *et al.* (2001) observed gastic ulcer in *Helicobacter pylori* infection. In one case multiple ulcers were noticed throughout the intestinal tract from duodenum to colon. It could be attributed to *Salmonella cholerae suis* infection as the organism was isolated from one case. Wilcock (1979) observed similar ulcerative lesions and inflammation in Salmonellosis. The inflammatory reaction seen in the stomach mostly was catarrhal gastritis in which hyperplasia of goblet cells were frequent. This may be attributed to metaplastic transformation of gastric epithelial cells due to chronic irritation or as a reaction of stomach to inflammation. Inflammation leads to increase in the secretion of mucus which protects the mucosa to some extent, but it delays digestion and may allow putrefactive breakdown of ingesta. The abnormal digestion may cause further inflammation favouring spread and persistence of infection (Radostitis *et al.*, 2000). The superficial necrotic gastritis and chronic gastritis seen in one case each can be attributed to the above factors.

The disorders of duodenum, jejunum, ileum and large intestine were studied and the lesions were classified. The lesions included congestion, haemorrhage, degeneration, necrosis and inflammatory changes. Among the inflammatory changes, catarrhal inflammation predominated in all the segments. In the present study, attempts were made to isolate the intestinal species associated with enteric disorders. Eventhough *E. coli,* Klebsiella and *Salmonella cholerae suis* were isolated, it could not be verified by experimental studies as whether these organisms which were isolated had any specific role in the causation of disease which resulted in the death of piglets. There is much difference in the opinion regarding *E. coli* and Klebsiella in causing enteritis, since they are natural inhabitance of the alimentary tract. It has to be attributed that artificial rearing conditions have caused a changed mileu in which *E. coli* or similar organsims gain an upper hand over the defence potential of an animal. Once a strain of *E. coli* of sufficient virulence has established in herd, predisposing factors become unnecessary and young animals may become affected and die before such factors have time to operate (Barker *et al.*, 1993). In

this context, it is worth mentioning that in the animals with enteritis of different types, wherein *E. coli* and Salmonella sp. were isolated, the 'Peyer's patches of the ileum and the lymphoid follicles of the mesenteric lymphnodes revealed depletion of lymphocytes and washed out appearance of germinal centres. This indicated a suppression of the defence potential which favoured flaring up of infection of the natural inhabitants of the gastro-intestinal tract. Seagles *et al.* (2001) reported depletion of lymphocytes from Peyer's patches in proliferative enteropathy. Wilcock (1979) observed necrosis of lymphocytes and atrophy of Peyer's patches in Salmonellosis. In most of the catarrhal type of inflammation, the prominent cell type was mononuclear cells with a prominence of lymphocytes. The predominant histopathological changes were villous atrophy, their fusion, denudation of villi, coagulation necrosis of the villus epithelium, damage of glands and inflammatory cell infiltration. In some cases of degeneration and necrosis also similar villus changes predominated. Similar lesions were observed by Manjunatha *et al.* (1995) and Penrith *et al.* (1995) in *E. coli* induced gastroenteritis in swine. The possibility of virus infection in these conditions has also to be considered when considering the etiological factors of gastro-enteritis. Kim and Chae (2000) observed similar histopathological changes in transmissible gastroenteritis and porcine epidemic diarrhoea viral infection. Stuart *et al.* (1980) described similar lesions in Porcine coccidiosis. In this case, coccidiosis was ruled out as the faecal samples did not show any oocysts. Most of the animals brought for autopsy were very weak and emaciated and the other lesions observed were pulmonary congestion, oedema and cardiac dialatation. This was the result of poor absorption of water, electrolytes and nutrients in the absence of an intact villous epithelial cells as revealed in the histopathological studies.

Mortality in gastroenteritis is not a single problem with one specific cause, but is a result of many factors. Infection in the first few weeks of life is a common cause of losses. Milk borne infection has a significant role in causing mortality. In two of the cases with enteritis, the cause could be attributed to milk

borne infection as *E. coli* was isolated from the sow's milk and the sow was suffering from mastitis. The organisms were also isolated from the intestine, liver and heart blood of the piglings.

Hepatic lesions of varying type were seen in association with gastrointestinal disorders. The lesions varied from haemorrhage, cloudy swelling fatty change and necrosis to hepatitis. In some cases bile duct hyperplasia was also observed with fatty change. It is quite likely that all these lesions could be the result of injury due to toxins. In case of suppurative hepatitis, *Salmonella cholerae suis* was isolated. Necrotic ulcers were observed throughout the intestinal tract in such case, indicating septicaemic spread. Wilcock (1979) made similar observations in salmonellosis. Hepatic cyst was noticed in one case. Grossly, there was no observable lesions in the liver. The cyst appeared circumscribed, containing proteinaceous material which gave an impression of dilated ducts. The possibility of hepatocyte rupture due to coalesion of fat globules and subsequent formation of cyst cannot be ruled out as fatty change was extensive surrounding the cyst.

Mesenteric lymphnodes showed histopathological lesions such as lymphoid depletion, washed out appearance of germinal centers, lymphoid necrosis and caseation and extensive congestion of vessels. This can be attributed to hypoxia following stasis of blood and extension of infection of gastro intestinal tract. Wilcock (1979) observed similar histopathological lesions in salmonellosis. In few cases, there was sinus histiocytosis and moderate follicular hyperplasia which could be the result of inflammation.

Eventhough, direct smear examination of faecal sample was conducted, no possible ova could be detected. This may be because of timely deworming and cleanliness followed in the farm.

The present study has indicated the necessity for in depth study on the role of factors like immune reaction of the animals and co-pathogens in the causation of gastro-enteric diseases in piglings.

The findings of the study highlighted the importance of gastro-intestinal pathogens and immunodeficiency as the causes of neonatal mortality in piglets. The need for monitoring the common bacterial pathogens especially *E. coli* and Salmonella and their timely treatment definitely would reduce piglet mortality. Boosting up of the immune status of piglets and improving the health status of the dam are also important in controlling the gastro-intestinal problems and piglet mortality.

Summary

6. SUMMARY

An investigation was undertaken to assess the prevalence and pathology of the gastro-intestinal disorders in piglets. The prevalence of gastro-intestinal disorders for the past five years based on the analysis of the data collected from the autopsy records maintained at the Pig Breeding Centre, Mannuthy as well as Centre of Excellence in Pathology was 78 per cent and 86 per cent respectively.

Fifty piglets brought from the Pig Breeding Centre, Mannuthy as well as from the carcasses brought for autopsy were subjected to detailed systematic gross and histopathological examination. Samples for bacteriological examination were collected from the heart blood, liver, intestine and sow's milk in appropriate cases. The disorders encountered in the gastro-intestinal system were recorded. Age wise and sex wise comparison of the lesions encountered were made and it was seen that piglets in the age groups of 10-20 days were more susceptible to gastro-intestinal disorders. A loss of passive immunity acquired through colostrum, lowering of resistance and subsequent infections and toxicities have been highlighted as the causes. The incidence of gastro-intestinal disorders was more in females.

Out of the 50 cases examined, 76 per cent showed pathological changes in the stomach, 88 per cent in the intestine, 60 per cent in the liver and 50 per cent in the mesenteric lymph nodes.

The lesions encountred in the stomach were congestion (47 per cent), haemorrhage (15 per cent), degeneration and necrosis (18 per cent), ulcer (13 per cent) and inflammation (16 per cent). The inflammatory conditions noticed were catarrhal gastritis, superficial necrotic gastritis and chronic gastritis.

The lesions in the duodenum were congestion (66 per cent), degeneration and necrosis (65 per cent), ulcer (5 per cent) and catarrhal duodenitis (47 per

cent). The jejunum showed 93 per cent congestion, 70 per cent degeneration and necrosis, 75 per cent jejunitis and 3 per cent ulcers. The lesions observed in the ileum were congestion (86 per cent), degeneration and necrosis (93 per cent), ileitis (75 per cent) and ulcer (5 per cent). In the ileum, the inflammatory changes were associated with depletion, degeneration, necrosis and even caseation of the lymphoid cells of the Peyer's patches in most of the cases.

Lesions in the large intestine especially colon, were congestion (52 per cent), degeneration and necrosis (43 per cent), catarrhal colitis (2 per cent) and *atresia ani* (3 per cent).

Coincident with gastro-intestinal disorders, the liver and mesenteric lymphnodes also revealed varying degrees of changes. The liver lesions encountered were congestion (56 per cent), haemorrhage (10 per cent), cloudy swelling (43 per cent), fatty change (10 per cent), necrosis (7 per cent), hepatic cyst (3 per cent) and 23 per cent hepatitis which included both focal hepatitis and suppurative hepatitis. *Salmonella cholerae suis* was isolated from the case of suppurative hepatitis, which indicated septicaemic spread as necrotic ulcers were observed throughout the intestinal tract.

In some cases bile duct hyperplasia was also observed with fatty change. All these lesion could have been the result of toxic injuries.

Mesenteric lymph node changes included congestion (40 per cent), degeneration and necrosis (5 per cent) and lymphoid hyperplasia and sinus histiocytosis in 3 per cent cases. In this regard, the role of immunosuppression in the spread of infection has been discussed.

Microbiological examination of the samples collected revealed pathogens and a total of 17 bacterial isolations were obtained from 11 animals. The isolates were *E. coli, Klebsiella pneumoniae* and *Salmonella cholerae suis*. The role of these pathogens in the causation of gastro-intestinal disorders have been explained.

Among the lesions in the gastro-intestinal system, vascular, degenerative and necrotic changes predominated, followed by inflammation which could be associated with hypoxic injuries, infections and toxicities. The catarrhal inflammation was frequent in the stomach and in the various segments of the intestinal tract. Villous epithelial damage of varying degrees, glandular damage in the lamina propria and mononuclear infiltration with a predominance of lymphocytes were the characteristic histopathological features. Since *E. coli* and *Klebsiella* were isolated from the lesions, a bacteriological etiology has been suggested and further the possibility of virus infection in these condition has also been considered. Most of the animals brought were very weak and emaciated and the other lesions observed were pulmonary congestion, oedema, cardiac dilatation. This was the result of poor absorption of water, electrolytes and nutrients in the absence of intact villus epithelial cells as revealed in the histopathology.

The systematic investigation undertaken has helped to focus attention on the prevalence of various disorders of the gastro-intestinal system. As pig farming is becoming popular among the rural farmers, the mortality of piglings should be considered as a serious problem and the results of this study will be of much help to the veterinarian in formulating strategies for control and treatment of diseases of piglings. The results of the investigation make it imperative that studies elucidating the role of factors like immune reaction of the animals and co-pathogens in the causation of gastro-enteric diseases in piglings must be undertaken.

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Appendix

PROFORMA

Sl. No. Date of death:

Date of Birth : Sex: Breed:

Weight of the animal :

Weight of the organ :

Clinical signs before death :

Gross lesions :

Other organs :

Cultural examination :

Faecal examination :

Others :

Remarks :

PATHOLOGY OF GASTRO - INTESTINAL DISORDERS IN PIGLETS

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ABSTRACT

The present study was undertaken to assess the prevalence and pathological disorders of the gastro-intestinal system of piglets. The results of the present investigation and evaluation of the data from the records revealed a high incidence of gastro-intestinal disorders to the level of eighty eight per cent. A detailed systematic examination of fifty piglet carcasses brought for autopsy during the period of investigation was conducted and the gross and histopathological lesions were studied in detail and were classified based on age and sex. Higher incidence of gastro-intestinal lesions was recorded in piglets aged 10-20 days and their possible causes are described. Vascular and degenerative changes were the predominant lesions, followed by inflammation. Among the inflammatory changes, catarrhal gastro-enteritis was the most prevalent lesions observed. *Escherichia coli* and Klebsiella were isolated from such cases and are suspected as the possible pathogenesis has been described. Viral etiology was also suspected in certain cases as the infiltrating cells appeared predominantly lymphocytes. Besides these ulcers were recorded in the stomach and the intestinal tract. *Salmonella cholerae suis* was isolated from the intestinal ulcers. Bacterial isolations were obtained in the intestine, liver, heart blood and sow's milk. A total of 17 bacterial isolations were obtained from 11 animals. Their role in the causation of gastro-intestinal disorders have been explained. Coincident with various gastro-intestinal disorders, liver of 30 cases and mesenteric lymph nodes of 28 cases revealed variety of vascular, degenerative and inflammatory lesions. In one case with suppurative hepatitis, *Salmonella cholerae suis* was isolated. The variable degrees of lymphoid depletion, degeneration and necrosis of lymphoid follicles of mesenteric lymph nodes and the lymphocytes of Peyer's patches indicated immunosuppression. The vascular and degenerative lesions, without any evidence for infections etiology observed in the gastro-intestinal tract and liver suggested the involvement of some toxic factors. Further the higher incidence of the disorders in the young ones, the isolation mostly of the resident flora of the intestinal tract and the lymph node changes indicated the necessity for an in depth study on the role of factors like immune status of the animals and co-pathogens in the causation of gastro-intestinal disorders in piglings.