STUDIES ON PATHOMORPHOLOGICAL LESIONS ASSOCIATED WITH COMMON HELMINTH PARASITIC DISEASES IN SLAUGHTER PIGS

By

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Certificate

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This is to certify that the thesis entitled ***"STUDIES ON PATHOMORPHOLOGICAL LESIONS ASSOCIATED WITH COMMON HELMINTH PARASITIC DISEASES IN SLAUGHTER PIGS"*** *submitted in partial fulfillment of the requirements for the degree of* ***"MASTER OF VETERINARY SCIENCE"*** *of the Sri Venkateswara Veterinary University, Tirupati, is a record of the bonafide research work carried out by* ***Ms. P.SUNITHA*** *under our guidance and supervision. The subject of the thesis has been approved by the Student's Advisory Committee.*

No part of the thesis has been submitted for any other degree or diploma. The published part has been fully acknowledged. All assistance and help received during the course of investigations have been duly acknowledged by the author of the thesis.

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I ***Ms. P.SUNITHA*** *hereby declare that the thesis entitled* ***"STUDIES ON PATHOMORPHOLOGICAL LESIONS ASSOCIATED WITH COMMON HELMINTH PARASITIC DISEASES IN SLAUGHTER PIGS"*** *submitted to Sri Venkateswara Veterinary University, Tirupati for the degree of MASTER OF VETERINARY SCIENCE is the result of original research work done by me. I also declare that the materials contained in this thesis have not been published earlier.*

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LIST OF CONTENTS

LIST OF TABLES

LIST OF ILLUSTRATIONS

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ABSTRACT

In India, of late many commercial piggery farms are being setup in large scale, in addition to traditional pig farming in rural areas. Parasitic diseases caused by different helminths are one of the major causes of economic losses in pig farming. Losses due to parasitic diseases are incurred annually as mortality, morbidity, condemnation of organs or of entire carcasses, cost of treatments and decreased rates of weight gain, feed conversion efficiency and carcass quality. Only a few reports are available on the incidence of helminthic diseases and the associated gross and microscopical lesions in the infested organs in the slaughtered pigs. In view of scanty literature particularly related to Andhra Pradesh, the present investigation was carried out to study the incidence and to describe the pathomorphological lesions of common helminth parasitic diseases in the slaughtered pigs. By understanding the pathomorphological lesions in common helminth parasitic diseases in pigs, proper preventive and control methods can be undertaken to reduce the economic losses.

In the present study, a total of 320 pigs of either sex and of different age groups from various slaughter houses, field mortalities, private piggery farms located in and around Gannavaram, Vishakapatnam and Tirupati towns and from postmortem examinations conducted at the NTR College of Veterinary Science, Gannavaram were subjected to detailed examination and different organs showing

lesions of parasitic diseases or organs showing parasitic infestation were collected. All the parasites collected during the study were identified on the basis of their morphological characters.

Out of 320 pigs examined in the present study, 216 (67.5%) animals revealed definite lesions of helminth parasitic diseases on gross and microscopical examination of tissues. Various helminth parasitic diseases that were observed in pigs were broadly grouped as the diseases caused by trematodes (3.13%), larvae of cestodes (8.75%) and nematodes (55.63%). Overall, the diseases caused by nematodes had highest percentage (55.63%) of incidence than others.

Two diseases viz. schistosomosis and opisthorchiosis caused by trematodes were noticed in the present study. The incidence of schistosomosis in livers was 2.81% of total animals. Grossly, the affected livers were slightly enlarged and had a granular irregular surface. Microscopically, perioval granulomas or pseudotubercles with extensive fibrocellular reaction in the interlobular septa and in the portal triads were noticed. The granuloma contained ova of *schistosoma* surrounded by eosinophils, macrophages, epithelioid cells, giant cells and lymphocytes along with extensive portal and septal fibrosis resulting in thickening of interlobular septa. No adult parasites were found in the sections. In addition, congestion, degenerative changes in the hepatic cells, bile duct hyperplasia, thickening and destruction of blood vessels, phlebitis, formation of angiomatoids with additional vascular channels, proliferation of fibrous tissue and infiltration by eosinophils and mononuclear cells in the interlobular septum were also found. In some areas, the hepatic lobules were separated and individualized by extensive infiltration and fibrous tissue proliferation and lymphoid nodule formation was also evident.

The incidence of opisthorchiosis was 0.32% of the total animals examined. Grossly, the liver was slightly enlarged and showed moderate increase of interlobular connective tissue. A whitish raised structure was noticed on the surface that on opening revealed thickened, cystic bile duct containing small flukes. They were identified as *Opisthorchis tenuicollis*. Microscopically, the affected part of the liver revealed the presence of cross section of fluke in the bile duct, that was extensively thickened, dilated and fibrosed. There was hyperplasia of bile duct epithelium, desquamation and necrosis of epithelial lining, proliferation of goblet cells and glands in the submucosa and infiltration of lymphocytes, plasma cells and macrophages in the mucosa and submucosa.

The larvae of cestodes caused two diseases in the present study. Of these, the incidence of hydatidosis was 1.25%. Grossly, a single cyst was noticed on the ventral surface of liver and on the dorsum of lung and spleen. It contained clear watery fluid and distinct whitish, thick cyst wall consisting of laminated layer of variable thickness. Microscopically, the cyst consisted of hyaline cyst wall surrounded by fibrous connective tissue capsule infiltrated with large numbers of eosinophils, mononuclear cells and plasma cells. The protoscolices were also noticed attaching to the cyst wall.

Cysticercosis was noticed in 7.5% of the animals. The metacestodes of *T.solium* were present in various organs like tongue, heart, liver, skeletal muscles and brain. They were typically spherical or oval, white or yellow vesicle with a translucent bladder wall through which the scolex was seen like a small solid, eccentric granule. In some areas, degenerated cysts were also seen. Microscopically, the cross sections of cysticerci were evident with or without invaginated scolex in different organs, surrounded by fibrocellular reaction consisting of lymphocytes, plasma cells and eosinophils .

In the present study, various diseases caused by nematodes noticed were ascariosis, stephanurosis, metastrongylosis and parasitic gastritis caused by stomach worms. The incidence of ascariosis was 34.06% which was highest among all the parasitic conditions observed. Grossly, milk spots were present on liver which were star like in shape and grayish white in colour or of pearl like nodules scattered throughout the liver. Microscopically, the liver sections revealed areas of extensive haemorrhage into the lobule, distortion of hepatic architecture, eosinophilic granulomas, presence of ascarid larvae, thickening of interlobular septa and lymphoid follicle formation. Adult worms were present in the intestines of some animals apart from the milk spots on the livers. The intestinal mucosa revealed desquamation of the epithelium and infiltration by lymphocytes, plasma cells and macrophages.

The incidence of stephanurosis was 8.75% of the total animals examined. Grossly, the liver had whitish nodules and revealed the presence of immature parasites. In some animals, the worms were also found in the perirenal fat in the free state. Microscopically in liver, congestion of sinusoids, haemorrhages, cut sections of immature worms surrounded by extensive eosinophilic infiltration, thickened interlobular septa, hyperplasia of bile ducts and thickening of blood vessels were noticed. Cut sections of worms were noticed in the perirenal fat along with infiltration of eosinophils. In the kidney, degenerative changes, necrosis of tubular epithelium, cystic dilatation of tubules, atrophy of few glomeruli and infiltration with mononuclear cells in the interstitium were noticed.

Metastrongylosis was noticed in 11.88% of animals out of 320 animals examined. Grossly, small gray nodules were noticed on the caudoventral borders of diaphragmatic lobes. Large numbers of male and female lung worms of *Metastrongylus apri and M. pudendotectus* were noticed in the bronchi and bronchioles in the ventral borders of the diaphragmatic lobes. Microscopically, several sections of the parasites in different stages of development were present in the lumen of the bronchioles, surrounded by mucus and inflammatory cells. The bronchial epithelium was desquamated and eosinophils, lymphocytes and neutrophils infiltrated the epithelium and lamina propria. Hyperplasia of bronchial, bronchiolar and alveolar epithelium and bronchial associated lymphoid tissue was also noticed.

Stomach worms (*Physocephalus sexalatus*) were noticed in 0.94% of total animals causing parasitic gastritis. Grossly, small red worms were present in the

stomach. They appeared as small red colour streaks on the mucosa of the stomach. Areas of slight hyperemia of the mucosa or formation of eroded areas or ulcers were evident. Microscopically, the mucosa of stomach revealed changes of chronic catarrhal gastritis. In the fundic region of stomach, necrosis and desquamation of the epithelium and infiltration of mononuclear cells, a few eosinophils and macrophages were noticed. There was atrophy of glandular epithelium or cystic dilatation of glands and infiltration by mononuclear cells and a few eosinophils in the cardiac region.

The present study revealed that helminth parasitic diseases are the important diseases in pig population in the area of study with an incidence of 67.5%. The pathomorphological lesions indicated that the parasitic diseases in pigs can lead to condemnations during meat inspection resulting in severe economic losses, apart from having public health importance in some diseases.

CHAPTER – I
INTRODUCTION

In India, pig rearing is very popular amongst the tribal, backward and weaker sections of the society and it plays an important role in improving the socio-economic status of the people. The pigs, with high prolificacy, efficient mothering ability, rapid growth, most economic feed conversion efficiency, shorter gestation period and high dressing percentage are rated as one of the best meat producing animals in the world (Anubrata Das and Bujarbaruah, 2005). Having realized the income generation potential of pig rearing, many commercial farms are being setup in large scale in coastal Andhra Pradesh.

Government of India has also launched a project to further the development of pig farming. It provides subsidy to pig rearing farmers, cooperative units and corporations through NABARD.

Pork is the second largest nutritious food in the world. Low cost of pork in comparison to other commonly consumed meat and changing food faddism of the people have enhanced its demand as a delicious and nutritive source of animal protein. Global production and consumption of pork has increased substantially in recent decades. World pork consumption has increased by 27% from 1997 to 2005 as per FAO (2005). India, with an estimated pig population of 14 million (FAO, 2008), ranks 11^{th} and has around 1.41% of the world's pig population. Pork production in the country stands at 0.5 million tonnes, as against the world production level of 115.45 million tonnes (FAO, 2008).

Pigs can convert inedible feeds, forages, certain grain byproducts obtained from mills, meat byproducts, damaged feeds and garbage into valuable nutritious meat and most of these feeds are not edible or not very palatable to human beings. In 4th Five-year-plan (1969-74), Indian Council of Agricultural Research (ICAR) has established All India Coordinated Research Project (AICRP) on pigs, with an objective of evaluation of the performance of the exotic breeds under optimum managerial conditions at Izatnagar, Jabalpur, Tirupati and Khanapara. The current goal of pig industry is to produce high quality lean meat at minimum cost which inturn will play an important role in improving the socio-economic status of pig rearing farmers (Anubrata Das and Bujarbaruah, 2005). Inspite of many developmental programmes in five year plans, major problems like poor management and disease occurrence are crippling the pig farming.

Swine rearing in our country is mainly in the hands of weaker sections of the society. Pigs by these people are reared in open or free range system, so usually they thrive as scavengers. The scavenging habit of pigs makes them susceptible to various parasitic diseases. One of the most important aspects of efficient swine production programme is to control parasitic diseases because they not only cause direct damage but also lowers the immunity and predispose the host to wide array of diseases. Losses due to parasitic diseases are incurred annually as mortality, morbidity, condemnation of organs, or of entire carcasses, cost of treatments and decreased rate of weight gain, feed conversion efficiency and carcass quality. The effects of internal parasites on daily weight gain and feed efficiency of pigs may be the most insidious economic loss to swine producers

and in many instances, helminth parasitic diseases are the major causes of losses or condemnations. The information on gross and histopathological lesions caused by helminthic parasites in the organs in which they reside in the body of infested pigs is very scanty. There are only a few published reports from Andhra Pradesh on the incidence and pathology of helminth parasitic diseases in slaughtered pigs. Lesions of the alimentary tract, liver, lungs and other organs are responsible for condemnation of carcasses at slaughter. Hence, by studying the incidence and understanding the pathomorphological lesions in common helminth parasitic diseases in pigs, proper preventive and control methods can be undertaken to reduce the economic losses.

Keeping this in view, the present study was planned with the following objectives:

1. To know the incidence of common helminth parasitic diseases in slaughter pigs.
2. To describe the gross and histopathological lesions in some common helminth parasitic diseases in slaughter pigs.

CHAPTER - III

MATERIALS AND METHODS

3.1 SOURCE

The materials for the present study consisted of various tissue samples of pigs having suspected parasitic conditions, collected from various slaughter houses, field mortalities and from private piggery farms located in and around Gannavaram, Visakhapatnam and Tirupati towns and from postmortem examinations carried out at NTR College of Veterinary Science, Gannavaram. The duration of study lasted from June 2010 to July 2011. A total of 320 pigs of either sex and of different age groups were examined in detail and the tissue samples of various organs were collected for the study.

3.2 MODE OF COLLECTION OF SAMPLES

Detailed examination of pigs was carried out during slaughter for the presence of any helminthic parasites in various organs and digestive tracts and for any gross lesions that were suggestive of parasitic diseases. Tissue pieces from representative portions of various organs showing suspected parasitic lesions or heavily infested by parasites were collected and fixed in 10 per cent neutral

buffered formalin for histopathological examination. The parasites found in different organs were collected in normal saline for further studies.

3.3 METHODS EMPLOYED

3.3.1 Collection of parasites

The helminths infesting different visceral organs of pigs were collected during slaughter and were processed as per the standard procedures (Soulsby, 1982) for identification in possible cases.

3.3.2 Histopathology

Tissue pieces from different organs showing parasitic lesions or infested by parasites were collected in 10% neutral buffered formalin for histopathological examination. The samples were processed by routine paraffin embedding technique and sections of 4 to 5 micron thickness were cut and stained by Harris Haematoxylin and Eosin method (Luna, 1968). Special staining procedures like Van-Gieson's staining technique and Azan trichrome method for collagen fibres (Mc Manus and Mowry, 1964) and Oil Red O staining technique for fat (Singh and Sulochana, 1996) were employed.

CHAPTER – II
REVIEW OF LITERATURE

The common helminthic parasitic diseases in pigs include various diseases caused by trematodes *(Fasciolopsis buski, Gastrodiscoides hominis, Artyfechinostomum sufrartyfex, Opisthorchis tenuicollis, Fasciola spp. and Dicrocoelium dendriticum*), nematodes (*Metastrongylus apri, Ascaris suum, Stephanurus dentatus, Trichuris suis, Oesophagostomum spp., Ascarops strongylina, Physocephalus sexalatus, Schistosoma spp. and Trichinella spp.)* and cestodes (larvae of *Taenia solium and Echinococcus granulosus*).

The available literature on the pathology of common helminth parasitic diseases in pigs is reviewed here under.

2.1 Incidence / Prevalence

Out of one hundred specimens of livers obtained from 946 indigenous pigs slaughtered, 14 cases of hydatid cyst infection and 2 cases of bile duct ascariasis were noticed (Chauhan and Rao, 1971).

Chauhan and Rao (1972) described the pathology of some common parasitic diseases in indigenous pigs. Out of 946 carcasses examined, 695 were found affected with kidney worm (*Stephanurus dentatus*) and 31 with lung worm (*Metastrongylus apri).* In 22 cases, hydatid cysts were found in lungs.

A survey conducted by Batte *et al*. (1975) in the southern USA showed that 95.0% of condemned pig livers had lesions associated with kidney worm and / or ascarid infections.

Economoic losses attributable to parasites of swine in Chile were recorded by Gonzalez and Plaza (1975) during 1959-1973 in an abattoir. There were 0.055% of condemnations for trichinosis, 0.474% for cysticercosis, 1.94% for distomatosis, and for hydatidosis 0.004% in muscle, 6.6% in liver and 6.5% in lung.

Henne *et al*. (1978) conducted studies on helminths occurrence in European wild pigs by helminthological dissection and coprological methods in the north-eastern part of Germany from November 1972 to March 1974 in which about 10 species of helminths were found in a total of 102 killed wild boars.

Mandal (1978) studied the parasitic pathology of liver in pigs and reported that on examination of 1102 adult pigs at slaughter in India, 60 pigs had livers with gross evidence of disease and in 12 of these, parasites were considered responsible. Schistosomal granulomata in six, angiomatoid formation due to schistosomiasis in two, *opisthorchis* in one, *ascaris* in one and unidentified parasites in two livers were noticed.

A two-week study on causes of condemnation of carcass and organs in Shah Alam abattoir was conducted by Tham and Sheikh-Omar (1981). Out of 100 organs of each study, 37 lungs and 10 livers were condemned due to lung worms (*Metastrongylus salmi*) and milk spots respectively.

Varma (1982) reported the incidence of helminth parasites of domestic pigs by examining a total of 332 pigs at Gurgaon during 1973 to 1976. Overall, 15 species of helminths (trematodes 5, cestodes 4 and nematodes 6) were

identified with multiple infections of various species like *F. gigantica, F. buski, C. tenuicollis and A. suum*. The incidence of hydatid cysts was 1.80% in lungs and 0.90% in livers.

An investigation was carried out by Pattanayak (1984) to find out the incidence, types and morphology of the lung affections and to study the etiopathology of various types of lung lesions in pigs. A total of 1440 pigs of both sexes, ranging in age from one month to two years were examined and histopathologial examination of tissue pieces was conducted in 351 pigs. Seventeen (4.84%) cases of echinococcosis, 14 (3.90%) cases of verminous pneumonia and 1 (0.28%) case of blood fluke (Schistosomiasis) infestation were identified.

Out of 142 pigs examined at autopsy by Dev Sarma and Gogoi (1986) in a study on helminths and histopathology of some common trematodes of local pigs in Assam, 80 animals revealed the presence of different types of helminths and lesions showing an overall incidence of 56.34%.

Gibbens *et al*. (1989) recorded the prevalence of gastrointestinal helminths and *S. dentatus* in pigs in Belize city, Central America. Out of 137 pigs examined in an abattoir, *H. rubidus*, *P. sexalatus* , *Globocephalus sp.* and *Trichostrongylus colubriformis (T. colubriformis)* were recorded for the first time and *Oesophagostomum spp.* (45.0%) *and S. dentatus* (42.0%) were found as most prevalent parasites.

Gill *et al*. (1991) conducted a survey for one year on prevalence of gastro-intestinal nematodes of pigs in Punjab state. On post mortem examination of 50 intestinal tracts, six species of nematodes viz. *A. suum, O. dentatum, A. strongylina, P. sexalatus, Strongyloides sp*. and *T. suis* were recorded. Maximum number of pigs (85.92%) were found positive for nematode infections during July to September and minimum number (59.78%) during January to March .

In a total of 352 pigs examined by Varma (1993) at slaughter, 305 (86.64%) animals were infected by different helminth parasites in Bareilly. Of these, 47 (13.34%) animals were infected by trematodes, 52 (14.75%) animals were infected by cestodes and 206 (58.49%) animals were infected by nematodes.

The incidence of different gastrointestinal parasites in pigs in Meghalaya was studied by Rajkhowa (1996). Faecal samples as well as slaughter house materials from 421 animals were examined and 202 were found positive for various parasites viz., *A. suum* (23.99%), *Oesophagostomum spp*. (19.48%), *Strongyloides sp*. (18.29%), *T. suis* (4.75%), *Metastrongylus sp*. (4.51%), *M. hirudinaceus* (2.61%) and *F. buski* (1.19%).

The overall incidence of spontaneously occurring parasitic conditions was 34.3% in 242 livers of pigs that were slaughtered for pork. Out of these infected livers, 34 showed stephanuriasis, 24 ascariasis, 20 schistosomiasis, 4 hydatidosis, one cysticercosis and a mixed infection with *Opisthorchis* and *Schistosoma* was found in one animal (Rao and Paliwal, 1996).

Pence *et al*. (2002) studied the visceral helminth communities of an insular population of feral swine in Cumberland Island, Georgia. Nine species of nematodes were recovered from viscera of 48 feral swine and there was an apparent increase in the prevalence and abundance of three species of helminths viz., *S. dentatus*, *Metastrongylus apri (M. apri)* and *Metastrongylus pudendotectus (M. pudendotectus).*

Solaymani-Mohammadi *et al.* (2003) reported the helminthic parasites of wild boars (*Sus scrofa*) in Luristan province, western Iran. Seven helminths, including the cestode larvae, *Cysticercus tenuicollis (*25.0%), *Cysticercus cellulosae* (8.3%) and the nematodes *M. apri (*41.6%), *M. pudendotectus (*16.6%), *M. salmi* (8.3%), *T. suis (*8.3%) and acanthocephalan *M. hirudinaceus* (41.6%) were obtained from 12 wild boars during a survey from 2000 to 2001 and no trematodes were found in the study.

Datta *et al.* (2004) conducted a survey on the prevalence of gastrointestinal parasites with special reference to pathology of *F. buski* infection in local pigs of West Bengal. A total of 1074 faecal samples and 93 stomachs and intestines of pigs were examined and the incidence of encountered trematodes viz, *F. buski, G. hominis* and *S. suis* was 27.9, 20.67 and 25.04 % respectively. The nematodes noticed were *Strongyle sp*. (14.89%), *A. suum* (28.58%), *T. suis* (13.22%), *Metastrongylus spp*. (20.11%), *Strongyloides sp.* (14.89%), *P. sexalatus* (8.19%) and *A. strongylina* (6.33%).

Dimarco *et al.* (2004) observed lesions due to parasitism in 32.0% of the liver and 70.9% of stomach samples out of 155 slaughtered wild nebrodi black pigs, aged 8 months to 4 years. Accidental parasitosis, like fascioliasis and dicrocoeliosis were also observed.

Out of 210 pig carcasses examined at an abattoir for parasites by Borthakur *et al*. (2007), 41 (19.52%) showed the presence of parasitic infection. The infection was prevalent throughout the year with highest incidence (44.44%) in post-monsoon season. *Ascaris* showed the highest prevalence in animals below 6 months of age group. The other parasites recorded were *S. ransomi, Oesophagostomum spp., A. strongylina, T. suis, P. sexalatus* and *A. surfurtyfex.*

Nissen *et al*. (2007) studied the prevalence of gastrointestinal nematodes in growing pigs in Kabale district in Uganda. The post-mortem examinations revealed that 93% pigs were infected with *Oesophagostomum spp*., 73% with *A. suum*, 67% with *T. suis* and 20% with *H. rubidus* .

The prevalence and pathology of helminth infections in pigs slaughtered at Tangail and Mymensingh districts of Bangladesh were studied by Islam *et al.* (2008). From 30 viscera collected, six species of helminths were identified, 2 of them were trematodes namely *F. buski* and *G. hominis* and 4 species were nematodes viz., *A. suum, M. elongatus, S. dentatus* and *P. sexalatus*.

Nganga *et al.* (2008) reported the prevalence of gastrointestinal helminth infections in pigs in Kenya. A total of 115 gastrointestinal tracts from 61 growers and 54 adult pigs were examined between February 2005 and January 2006.

Seventy eight samples (67.8%) had one or more helminth parasites, of which thirty six (31.3%) were of mixed infection. Ten types of helminth parasites were encountered viz, *O. dentatum* (39.1%), *T. suis* (32.2%), *A. suum* (28.7%), *O. quadrispinulatum* (14.8%), *T. colubriformis* (10.4%), *T. axei* (4.3%), *S. ransomi* (4.3%), H. *rubidus* (1.7%), *A. strongylina* (1.7%) and *P. sexalatus* (0.9%). *O.dentatum* was more prevalent in adult pigs (51.9%) and *T.suis* in growers (44.3%).

In a total of 29 Japanese wild boars collected during hunting seasons of 2005 and 2006, 17 nematodes and one cestoda species were identified by Sato *et al*. (2008).

Gaurat and Gatne (2009) described the gross and histopathological lesions associated with helminthic parasites in a survey based on examination of 501 pigs slaughtered at Deonar abattoir during May 2002 to August 2002.

Senlik *et al.* (2010) reported the status of helminth infections in wild boars during 2007-2008. Individual samples of tongue and diaphragm from 27 necropsied wild boars and an additional 22 tongue samples provided by hunters were examined for *Trichinella spp.* larvae. Twenty animals (74.0%) were identified as being infected with atleast one helminth species and in total twelve species of helminths were detected viz., *M. apri* (59.0%), *M. salmi* (52.0%), *M. pudendotectus* (52.0%), *Dicrocoelium dendriticum (D. dendriticum)* (33.0%), *Globocephalus urosubulatus* (22.0%), *M. hirudinaceus* (19.0%), *Gongylonema*

*pulchrum (*11.0%), *P. sexalatus* (7.0%), *T. suis (*7.0%), *A. strongylina* (4.0%), *H. rubidus (*4.0%) and *Taenia hydatigena (T. hydatigena)* larvae (4.0%).

2.2 Pathomorphological lesions of common helminth parasitic diseases in pigs

2.2.1 Diseases caused by Trematodes

2.2.1.1 Schistosomosis

The distribution of lesions from stomach to intestine in experimental *Schistosoma incognitum* (*S. incognitum*) infection in pigs was discussed by Ahluwalia (1972). In the liver, a granulomatous reaction developed along portal sheaths by 2 months, by 4 months there were pseudotubercles which tended to fibrosis, by 9 months the lesions were fibrosed, and by 12 months there was cirrhosis, periphlebitis and periportal fibrosis.

In a survey on natural heterologous schistosome infection in domestic animals carried out at Jabalpur by Agarwal and Sahasrabudhe (1982), 16 (80.0%) were positive for *S. incognitum* infection, out of 20 pigs slaughtered.

Clinical and pathologic features of experimental *Schistosoma japonicum* (*S. japonicum*) infection in pigs were studied by Carmencita *et al*. (1984). Nine piglets, 2-5 months old and weighing 12-15 kg were infected percutaneously with 5000 to 6000 cercariae of *S. japonicum*. Infected animals were necropsied and the pathological changes induced by *S. japonicum* included erythematous papules on the site of inoculation, petechial hemorrhages in the lungs, catarrhal to hemorrhagic enteritis, bluish-gray discoloration of the liver and egg granulomas in the liver, lungs, spleen, intestines, pancreas and mesenteric lymph nodes.

Endophlebitis with intimal hyperplasia was occasionally observed in veins harboring adult schistosomes.

In schistosomiosis of pigs, the affected liver grossly showed patchy grayish areas with prominently thickened interlobular septa over a wide surface (Rao and Paliwal, 1996), In certain cases, multiple pin point dark grayish brown foci with pale interlobular septa were discernable. Histologically, the inter and perilobular septa consisted of multiple focal pseudotuberculous lesions characterized by the presence of schistosome egg and infiltration of epithelioid cells, a few histiocytes, numerous lymphocytes and occasional foreign body giant cells with fibrocellular reaction. At places, massive lymphoid reaction was also noticed around degenerated ovum. The portal veins were dilated and contained cross sections of adult schistosoma.

Congenital *S. japonicum* infection in pigs was studied by Willingham *et al.* (1998). Eggs were recovered from the livers of all piglets and hepatic granulomas appeared consistently in portal areas, displaying a prominence of epithelioid macrophages and intermingled surrounding lymphocytes that were consistent with T cell dependent reaction.

The histopathologic study of different stages of single low or high-dose experimental *S. japonicum* infections in the pig was carried out by Hurst *et al*. (2000). Tissue responses of 48 pigs infected intramuscularly with either 100 or 2000 *S. japonicum* cercaria were examined at necropsy 4, 7, 11 and 24 weeks post infection. Egg granulomas were present in the liver, intestine and

occasionally in the lungs from 11 weeks post infection (PI). There were also free eggs and early exudative reactions to eggs, with acute inflammatory foci in the intestine. At 11 weeks PI, pigs in the higher dose group showed marked periportal and septal fibrosis with minimal parenchymal destruction. Thereafter, the lesions regressed spontaneously as the pig underwent self-cure phenomenon. The lower dose group showed only mild lesions throughout the study. The degree of hepatic fibrosis correlated with the density of eggs and granulomas in the liver tissue.

S. japonicum infection in the pig as a model for human schistosomiasis was studied by Johansen *et al*. (2000). Like in humans, *S. japonicum* established mainly in the large intestinal veins, with high faecal egg counts during the acute phase of infection. Hepatomegaly, increased portal diameter, periportal fibrosis and ascites in chronic infections were the common findings in both humans and pigs. Low protein diet aggravated the disease in pigs by increasing the establishment rates, the faecal egg excretion and the morbidity.

Agarwal *et al*. (2001) noticed sudden death of piglets when they were infected with cercariae of *S. incognitum* by polythene tail method. No gross and histopathological changes were observed in the liver and intestine of the piglets that died within 24 hrs, whereas piglets that died on 18^{th} and 21^{st} DPI showed emphysematous changes. Histopathologically, the intestines were normal and liver showed cellular swelling and mild fatty change.

Hurst *et al.* (2002) studied the immunohistology of different stages of *S. japonicum* egg granuloma in the liver sections of experimentally infected pigs.

Histologically, perioval granulomas were located in portal triads and interlobular septa, frequently obstructing portal and distributing veins. Affected vessels often showed phlebitis and periphlebitis. Portal and septal fibrosis and diffuse inflammatory cell infiltration to variable degree were present in all infected pigs. Most granulomas were dominated by epithelioid cells mixed with variable numbers of multinucleated giant cells, infiltrates of eosinophils and small mononuclear cells, mainly lymphocytes and some plasma cells and contained a single egg. Lymphoid nodules were detected in approximately one quarter of all granulomas. Immunohistochemistry results indicated that all stages of hepatic *S. japonicum* egg granuloma in the pig manifests MHC class II dependant CD4 T cell activity concomitant with infiltration of CD8 T cells.

Baddamwar *et al.* (2004) studied hepatic fibrosis in experimental *S. japonicum* infection in pigs. The major histopathological lesions were variable number of perioval granulomas, diffuse infiltration of inflammatory cells, mainly eosinophils and small mononuclear cells and fibrosis of portal areas and interlobular septa. Granulomatous obstruction of portal veins with destruction of vein wall was also frequently observed.

Birck *et al*. (2006) studied distribution of mast cells in relation to *S. japonicum* induced lesions in pigs and noticed perioval granulomas along with multifocal or diffuse septal and portal fibrosis in the liver. Granulomas were mainly of the mature productive types and primarily located in portal triads and interlobular septa and granuloma associated lymphoid follicles were eccentrically located at the border of a few granulomas.

A study on the evolution of the liver fibrosis during long term experimental *S. japonicum* infection in pigs was carried out by Ngwa (2006). The liver lesions were characterized by perioval granulomas, diffuse inflammatory cell infiltration and portal and septal fibrosis. The degree of fibrosis was assessed in Masson's trichrome stained liver sections, using both semi-quantitative histopathological scoring and quantitative area measurement by image analysis.

Iburg *et al*. (2007) studied the hepatic changes in congenital *S. japonicum* infection in pigs. Inflammatory lesions in the liver consisted mainly of granulomas in portal areas, often obliterating the portal veins and frequently with central eggs or egg remnants. The granuloma reaction consisted of epithelioid cells and occasional giant cells surrounded by layers of lymphocytes, eosinophils, plasma cells and various amounts of collagen and fibroblasts. Mild to moderate infiltration of portal and septal connective tissue with eosinophils and lymphocytes was common.

2.2.1.2 Infestation of liver flukes

Ohshima *et al*. (1971) reported tissue reactions against immature liver flukes invaded in pigs. Grossly, the lesions of liver were as large as a soyabean to a thumb size. Cavities measuring up to 10.0 mm in diameter were recognized in the central part of each lesion. Histopathologically, accumulation of eosinophilic leucocytes with or without flukes was noted in the cavity at the center of each lesion. Vacuolar fibroblastic histiocytes were found proliferated on the inner surface of the cavities and a thin fibroblastic cell layer was present.

Lymphofollicular structures around the cavities and the broadened periportal connective tissue were frequently observed in the liver.

Comparitive studies were made between groups of pigs infected with *Fasciola hepatica* (*F. hepatica*) metacercariae at the age of less than 1 and 8 weeks (Nansen *et al*., 1972). There was increased resistance with age that coincided with development of the ability to mount a marked fibroblastic reaction in response to the infection and the fibrous tissue might act as a mechanical barrier to migration of parasite in the liver parenchyma.

A three year survey was conducted by Roy and Tandon (1992) on helminths in animals of food value in north-east India and *Opisthorchis noverca* (*O. noverca)* was recovered from the liver of pigs on several occasions.

Pathomorphological changes in the liver in experimental superinvasive opisthorchiasis in 30 piglets were studied by Tsybina *et al.* (1992). Histologic examinations of the liver revealed diffuse persistent hepatitis in nine animals and aggressive hepatitis in four that was carried out in 2.5 months after the first infestation.

Bile duct showed extensive hyperplasia, cross section of *opisthorchis spp.* of parasite and infiltration of eosinophils, polymorphs, lymphocytes, macrophages with fibrocellular reaction in one case during the study of pathology of parasitic conditions affecting slaughtered pigs by Rao and Paliwal (1996).

A massive infestation of *Opisthorchis tenuicollis* (*O. tenuicollis)* in a pig slaughtered at Mumbai abattoir was described in relation to gross and histopathological changes in liver by Gatne *et al.* (2008). Grossly, the liver was enlarged and showed extensive fibrosis all over as well as in the parenchyma. A balloon like structure was present on the surface that contained small flukes measuring 3.0 and 8.0 mm which was found to be dilation of the bile duct. Microscopically, the liver revealed biliary hyperplasia with cirrhotic changes in the adjacent hepatic parenchyma that was divided into number of irregular lobules due to extensive fibrosis, thickened bile duct and the hepatocytes showed degenerative changes and necrosis. Mild mononuclear infiltration was evident in the portal triads.

Cappucchio *et al*. (2009) reported the results of a retrospective study of trematode infestation carried out on Nebrodi Black male and female pigs aged from 8 months to 4 years. *F. hepatica* and *Dicrocoelium dendriticum* (*D. dendriticum)* flukes were detected in 4.37% of the livers and the predominant histological features were multifocal to diffuse chronic hepatitis with fibrosis, severely thickened walls of the bile ducts and chronic parietal sometimes nodular inflammation.

F. hepatica infection in the livers of wild boars slaughtered in the UK was identified by Thompson *et al*. (2009). Out of 3021 livers examined, six were regarded as having massive infection, with 11 to 18 adult flukes in the bile ducts. Grossly, multiple white button-like cysts either on or just below the surface of the liver were noticed. They have a fibrous capsule and a yellow-brown caseous core.

The bile duct mucosa was thrown into thickened white folds but was not calcified.

2.2.2 Diseases caused by larvae of cestodes (Metacestodes)

2.2.2.1 Hydatidosis

Szarek (1975) noticed a very large number of hydatid cysts in a 14 month old pig liver that showed a five fold enlargement and marked parenchymal atrophy. The morphology of hydatid cysts (*Echinococcus granulosus*) in livers of pigs was studied by Vanek (1975) and a new classification of cysts was proposed defining 3 forms of unilocular cysts viz. cystic, cystic with cavities and multilobular.

Tissue reaction to spherical and lobular hydatid cysts in pigs was described by Slais and Vanek (1980). Typical initial host tissue reaction to a growing hydatid cyst in the lungs and livers was a local allergic inflammation characterized by heavy eosinophil infiltration. In infections with lobular and multilobular cysts, the early type of tissue reaction persisted around the pouches of hydatid wall and was characterized by an epithelioid rim, new production of connective tissue and an inflammatory infiltration. In old large fertile cysts, the acute inflammatory reaction disappeared from the inner side of the connective tissue sheath and the wall of the hydatid remained in direct contact with a thick layer of hyalinely degenerated fibrous connective tissue.

Slais (1981) described the capsule formation and shape of young hydatids of *E. granulosus* in the liver in a spontaneous infection of pigs. The hydatids were surrounded by a thick capsule that had an inner layer consisting of epithelioid histiocytes, multinuclear giant cells and cellular spongy connective tissue, a middle layer of richly vascularised, loose connective tissue and an outer layer of maturing fibrous connective tissue that merged with the liver tissue.

Ninety four of 58567 pigs slaughtered at meat inspection centre, Japan had nodular foci in the liver, 34 of which were diagnosed as multilocular echinococcosis (Sakui *et al.,* 1984). The nodules were less than 100 mm in diameter with a central area, but neither brood capsules nor protoscolices were observed.

Hydatid cysts were noticed in six lungs out of 190 pig lungs collected at slaughter by Baruah *et al*. (1985). Grossly, cystic structures were observed on the caudal end of the diaphragmatic lobe. Microscopically, the cysts showed an eosinophilic mass surrounded by connective tissue capsule with cellular infiltrations and peribronchiolar cuffing with mononuclear cells.

An epidemiological survey on distribution of echinococcosis was carried out in 88 slaughter houses from 1979 to 1983 in Italy by Lorenzini and Ruggieri (1987). Out of 1,909,911 pigs observed, 4200 pigs were found to be positive.

The incidence of hydatid cysts in domestic animals was reported as 1.42% in pigs with 90% organ involvement by Varma and Malviya (1988) in Bareilly, India from May 1983 to December 1984 .

Sahu *et al.* (1993) described the spontaneous pulmonary hydatidosis in pigs and out of 1402 pigs examined, 14 (0.99%) had pulmonary hydatidosis. Grossly, the lungs contained cysts on the surface as well as deep in parenchyma. The number and size of cysts were varied and they were smooth, white colored and contained distinct cell wall which was thick due to varying degrees of encapsulation. Histopathologically, the cyst consisted of a dense laminated eosinophilic cyst wall without a germinated centre and was encircled by a thick connective tissue capsule outside that was massively infiltrated with large numbers of eosinophils, mononuclear cells and plasma cells.

An abattoir survey carried out by Vargas-rivera *et al.* (1995) revealed 0.27% prevalence of hydatidosis in approximately 40,000 pigs slaughtered in Los Reyes, Lapaz.

In a case of hydatidosis of pig, the liver had 1-4 cysts ranging from 1.0 to 6.0 cm in diameter lodged on the surface and deep into the parenchyma and the cysts were sterile (Rao and Paliwal, 1996).

Deka and Gaur (1998) investigated the prevalence of hydatidosis in different abattoirs of Western Uttar Pradesh and reported that the overall infection rate was 0.73% out of 2980 pigs examined. Multiple nature of cysts in lungs or liver was observed with the liver harbouring more fertile cysts (92.0%) than lungs (70.0%).

Ashok Kumar *et al.* (2000) reported the pathology of parasitic pneumonia in a study conducted in 200 slaughtered pigs. One hydatid cyst was recorded in

this study in an indigenous male pig. The cyst was solitary, firm, globular with smooth glistening surface measuring 2.5cm in diameter located deep on anterio-ventral border of left cardiac lobe and the cystic components revealed it as fertile cyst. Microscopically, the cyst consisted of both parasitic and host capsules. The inner parasitic capsule appeared homogeneously pinkish like membrane, which was separated from host capsule at few places. The outer host capsule consisted of fibroblastic proliferation with infiltration of lymphocytes and inner fusiform layers. The alveoli adjacent to the cyst were atelectatic with focal area of suppuration.

The pathological alterations in spleens of pigs of 1-3 years of age and either sex were observed by Singh (2000) at Bareilly and Aligarh abattoirs. Out of 305 pigs examined, tissue pieces from 50 pigs collected for histopathological examination revealed hydatidosis in 7 cases. The cysts were well distended having gray opaque walls that were laminated and the surrounding splenic substance was compressed. Microscopic examination revealed laminated cyst wall surrounded by giant cell granulomatous reaction. There were eosinophilic infiltrations around the laminations of the cyst wall, connective tissue capsule, trabaculae and in the red pulp of the spleen.

The prevalence of hydatid cysts was investigated by Dev sarma *et al.* (2000) in pigs slaughtered at different abattoirs in Greater Guwahati. Out of 279 pigs, 5 (1.79%) were positive for hydatidosis.

Of 1,540 pigs examined by Colavita *et al.* (2001) between 1996 and 1999, 122 pigs (7.9%) showed hydatidosis mainly in the liver (95.0%), in the liver and lungs (4.1%) and in only one case (0.8%) in the lungs. Out of 236 pig carcasses examined at different local slaughter houses in and around Ludhiana city, 11 animals (4.66%) had hydatidosis and the hydatid cysts were mainly observed in the liver and lungs (Sharma *et al*., 2004).

A survey on hepatic hydatidosis in domestic animals in Haiti conducted by Blaise and Raccurt (2007) showed a high prevalence (5.2%) in pigs than in other animals.

Bruzinskaite *et al.* (2009) carried out post slaughter examination of 684 pig livers in Lithuania that revealed significantly higher number of *E. granulosus* infection in animals from family farms as compared to industrial farms. The prevalence was also reported to be significantly higher in pigs older than one year than younger ones. The prevalence of cystic echinococcosis along with evaluation of size, fertility and number of hydatid cysts in pigs from Slovakia was studied by Turcekova *et al.* (2009). Out of 103 pig livers collected from abattoirs, 63 were positive for cystic echinococcosis, whereas cysticercosis was diagnosed in 40 livers.

Borua *et al.* (2010) assessed the prevalence of hydatid cysts in lung, liver and spleen of slaughtered food animals in Guwahati during February to August, 2008. Out of 130 pigs examined, the overall incidence of hydatid cysts was 7.68%.

2.2.2.2 Cysticercosis

The epidemiology of *Taenia solium* in certain parts of Uttar Pradesh, India was studied by Pathak (1984). Out of 2000 pigs slaughtered during December 1980 to December 1982, 340 were found to be positive, giving an overall incidence of 17.0%.

Dingeldein (1985) noticed *C. tenuicollis* infection in two pigs that was characterized by an unusual heavy parasitization of the liver with numerous parasitic canals and cysts. The liver and lymphnodes were swollen.

Deka *et al*. (1985) studied the incidence of cysticercosis in domestic animals in north east region of India during September 1981 to March 1984. Out of 106 pigs examined at slaughter, *C. cellulosae* was found in 20.8% of animals.

Occurrence of *C. cellulosae* in slaughtered pigs in Calcutta was studied by Pramanik *et al*. (1985). Out of 11,237 pigs examined, 477 (4.24%) were positive for *C. cellulosae* and the cysts were detected in different muscles viz. neck (54.51%), masseter (32.29%), thigh and gluteal (30.15%), heart (29.98%), diaphragm (19.50%), axial muscle (12.16%), and eye, liver and brain (2.94%).

de Aluja and Vargas (1988) reported the histopathology of porcine cysticercosis in the samples obtained from 28 infected pigs of different ages and provenance. A total of 296 larvae were observed, of which 58 were degenerated, causing a severe granulomatous reaction in host tissue and finally fibrosis and twenty eight showed no inflammatory response.

Varma and Ahluwalia (1989) reported the incidence of *C. cellulosae* in slaughtered pigs in western and central Uttar Pradesh. Out of 757 pig carcasses examined, 41 were found infected with *C. cellulosae* with an overall incidence of 5.35%, while Shinde (1991) observed 6.02% incidence of *C. cellulosae* infection in the survey of 400 pigs in Maharastra.

A total of 12798 cysticerci were recovered from four small ear miniature pigs in an experimental infection with *Taenia saginata* (*T. saginata*) by Fan *et al.* (1992). All the cysticerci were found only in the livers, more in parenchyma (89%) than on the surface (11%). Only 11 cysticerci in two pigs were mature and the rest were either immature, degenerated or calcified.

In cysticercosis of pig, the liver contained multiple cysts of 0.5 to 1.0 cm in diameter lodged on the surface and were usually attached to the Glisson's capsule. Histologically, the liver had typical chronic fibrocellular reaction around the cyst (Rao and Paliwal, 1996).

C. tenuicollis and hydatid cysts were recorded to the extent of 5.16% in the livers of slaughtered pigs in Poland by Michalski (1996).

de Aluja *et al.* (1998) reported *T. solium* cysticercosis in young pigs in which metacestodes were found in liver in four months aged piglets and the cysts were surrounded by marked inflammatory reaction. In five months age group, granulomatous lesions with structures that suggested destroyed metacestodes were present in the liver.

D'Souza and Hafeez (1998) studied the prevalence of *C. cellulosae* in pigs in an organized abattoir in Andhra Pradesh over a period of 2 years from April 1993 to March 1995 and out of 5,828 pigs, 263 (4.5%) were found infected. Economic losses due to condemnation was amounted to 4.22% of total value of pigs slaughtered while, Munde (1999) recorded 1.78% of *C. cellulosae* infection in pigs in Deonar abattoir.

Singh and Parihar (1999) reported certain parasitic conditions in brain in Indian pigs aged 2-3 years. Out of 90 cases, the gross lesions due to *C. cellulosae* were diagnosed in 9 cases. The cysts were 1 to 20 in number measuring about 1 cm diameter and were found on cerebrum and cerebellum on the surface of sulci and gyri and in lateral ventricles. Similar cysts were also found on skeletal and cardiac muscles. Microscopically, cysts with or without invaginated scolices in the bladder wall were encapsulated by connective tissue capsule. The cyst wall was uniformly thick and deeply eosinophilic. There was mild to moderate infiltration with mononuclear cells and eosinophils particularly around the blood vessels in the connective tissue.

The occurrence of *C. cellulosae* in and around private abattoirs in Ludhiana city was observed by Avapal *et al.* (2003). Out of 229 pigs, 4 (1.75%) were found infected and the cysts were almost a centimeter in length having white grey and translucent appearance in skeletal muscles of thigh, tongue, neck, masseter and diaphragmatic muscles being common sites.

Hafeez *et al.* (2004) investigated the prevalence of porcine cysticercosis during meat inspection in South India, from July 2000 to August 2003. Out of 1813 pigs examined in four states of south India, *C. cellulosae* was observed in 82 pigs and the overall percentage of infection was 4.52, with highest incidence in Karnataka (5.73%), followed by Tamilnadu (5.50%), Kerala (5.38%) and Andhra Pradesh (3.52%).

Rajnish Sharma *et al.* (2004) carried out a study to record the prevalence of swine cysticercosis in pigs of Northern Punjab. Out of 236 swine carcasses examined at different meat shops, 15 were found positive for the presence of *C. cellulosae* showing an overall prevalence of 6.35%. Grossly, the cysts were visible and present on or embedded inside different tissues and organs of the carcasses. Smaller cysts were white in color while larger ones were either pink or yellowish containing younger and older larvae respectively. All cysts were cellulose type having thin collagenous capsule and were typically spherical or oval, white or yellow vesicle with translucent bladder wall, through which the scolex could be seen like small solid eccentric granule.

Phiri *et al.* (2006) assessed the value of tongue examination and meat inspection as diagnostic tools for porcine cysticercosis in 65 Zambian village pigs by comparing the results with carcass dissections. Five pigs (7.7%) were positive on tongue examination, while routine meat inspection showed 12 (18.5%) positives. However, carcass dissections detected cysticerci in 31 (47.7%) pigs demonstrating the serious shortcomings of routine detection methods for porcine cysticercosis.

Prakash *et al*. (2007) reported neurocysticercosis in free roaming pigs and observed the pathomorphological lesions in affected brains. A total of 200 brains collected between August 2005 to March 2006, showed 3.0% occurrence on gross and histopathological examination. Grossly, the brains revealed 1-5, small transparent cystic structures filled with clear fluid and a white spot, with adjacent parenchyma showing slight peripheral congestion. Microscopically, cut sections of metacestodes were evident in the large cystic structures, and adjacent brain parenchyma revealed compression. In some cases, the cystic cavity was devoid of any parasite. The surrounding brain parenchyma revealed inflammatory changes characterized by infiltration of mononuclear cells, eosinophils and plasma cells.

Sikasunge *et al.* (2008) studied the prevalence of porcine cysticercosis in eastern, southern and western provinces of Zambia. A total of 1691 pigs were examined, out of which 183 (10.8%) were positive on tongue examination.

Mandakhalikar *et al.* (2009) studied the prevalence of cysticercosis in pigs and estimated the economic losses due to condemnation of pork in an organized slaughter house in Mumbai. Prevalence of cysticercosis was found to be 0.89% of 896 carcasses examined.

Sikasunge *et al.* (2009) used immunohistochemistry to examine the immuno-pathological changes and the extent of neuronal damage caused by either viable or dead *T. solium* cysticerci during porcine neurocysticercosis. Thirty pig brains with cerebral cysticercosis and 5 brains from *T. solium* free pigs were used for this study and the results revealed extensive astrogliosis, neuronal and mostly

axonal damage in early and late lesions as evidenced by an increased expression of glial fibrillary acidic protein (GFAP) and neurofilament protein (NFP).

2.2.3 Diseases caused by Nematodes

2.2.3.1 Ascariosis (White spots / Milk spots)

Lesions caused by migrating *ascaris* larvae in experimentally infected piglets were studied by Zendulka (1960). Eight hours after feeding, larvae passed through the mucous membranes of digestive system, producing minute hemorrhages and in the case of massive doses, oedema. Within 5 days focal lesions become apparent in the liver, characterized by whitish spots of varying size.

Roneus (1966) studied the etiology and pathogenesis of white spots in the liver of pigs. Morphologically, they were divided into two main groups viz., white spots of the granulation tissue type and white spots of the lymphonodular type.

Kim and Lim (1970) recorded the pathological changes in the hepatic ascariasis of swine and detected *ascaris* infection in 475 livers out of 1,500 pig livers examined.

Postmortem examination of piglets that were infected at 2 days of age with *A. suum* eggs showed no milk spots on liver, while varying numbers were seen in infected older pigs. Liver showed interstitial fibrosis and cellular infiltration (Bindseil, 1972).

Kozlowicz (1972) described histopathological changes in the liver of pigs naturally infected with *ascaris* and reported that the commonest feature was reactive inflammation of the liver that was found in 16 cases out of 50 animals.

Batte *et al*. (1975) described the two morphologically distinct types of hepatic white spots induced by *A. suum* infection in pigs. The granulation tissue type was built around a central tissue lesion, intralobularly situated consisting of a small granuloma containing larval remains surrounded by a strongly eosinophilic mass of degenerate eosinophilic leukocytes with heavily thickened interlobular septa. Cells of lymphonodular types were mainly lymphocytes which tended to form follicles. The white spots were demarcated by a connective tissue capsule. The eosinophilic leukocytes were sparse but there were larval remains.

Qureshi *et al. (*1978) reported the esophagogastric ulcers associated with *A. suum* infestation in pigs. Two pigs that were necropsied had large esophagogastric ulcers, hepatic fibrosis with milk spots and swollen edematous lungs. The ulcers involved the full thickness of the gastric mucosa with pronounced eosinophilic infiltration and perivascular cuffing of submucosal vessels. There was an acute interstitial and granulomatous pneumonia with inflammatory exudates composed of many eosinophils and *ascarid* larvae were recovered from the lungs.

Flesja and Ulvesaeter (1979) recorded 1.03 to 1.9% (average 1.5%) of pathological lesions as white spots in the liver, caused by *A. suum* out of 10051 slaughtered sows.

Prophylactic medication with pyrantel to prevent liver condemnation due to *ascaris* damage was evaluated by Kennedy *et al.* (1980). No liver from the medicated animal was condemned, whereas livers from 101 (21.0%) of 479 unmedicated pigs were condemned due to extensive hepatic scarring.

Polley and Mostert (1980) carried out an abattoir survey on the prevalence and intensity of infection of *A. suum* in pigs in Saskatchewan. Out of 2500 pigs examined in two surveys (A and B), 46.0 and 44.0% of livers had milk spot lesions respectively.

A series of experiments conducted in pigs with *A. suum* eggs indicated that the incidence of white spots on liver coincided with the migration of larvae from intestine and the white spots regressed in 3 to 4 weeks. It was suggested that the presence of white spots on the liver at slaughter was an indication of recent massive *A. suum* egg infection (Eriksen *et al*., 1981).

Antia and Alonge (1982) conducted a survey of abattoir data in south Nizeria. Data from 9 abattoirs collected from 1976 -1979 showed that abscess and *ascarid* infestations of liver were the main causes of condemnations in swine.

Conway and Young-Sun Hong (1983) carried out a survey of ascariasis in pigs and examined livers from 960 pigs slaughtered at three South Korean abattoirs and noticed mild lesions characteristic of *A. suum* migrating larvae in 44.0%, moderate lesions in 13.0% and severe lesions in 0.7% of livers.

White spots of the liver were studied by Yoshihara *et al.* (1983) in three groups of pigs that were inoculated orally with embryonated *A. suum* eggs. At autopsy, mesh worked white spots and lymphonodular lesions were observed on the surface of the liver in all the pigs.

Mehl *et al*. (1983) reported ascarid larvae as a cause of liver lesions (milk spots) in swine. Piglets infected experimentally with eggs of *Toxocara canis* developed milk spots in the liver 8 days after infection which disappeared after 85 days while *Toxocara* larvae were recovered from the liver only during 20 days after infection. In contrast piglets infected with *A. suum* developed milk spots only upon reinfection, 34 days after the first and lesions had healed 45 days after the second infection.

Martin *et al. (*1984) studied the mucosal surface lesions in young protein-deficient pigs infected with *A. suum.* The tissue samples collected at post mortem examination from pigs after 58 days contained *A. suum* showing varying degrees of villous atrophy and fusion.

Tahir and Sheikh-Omar (1985) conducted a five week survey on the pathology of condemned swine livers in Shah Alam abattoir in Malaysia. Out of 8,558 pigs slaughtered, 36 (0.12%) livers showed milk spots. Grossly, they were small stellate white spots, 0.1 to 1.5 cm in diameter. In a few, the bigger spots tended to have a nodular cystic centre of 0.1 cm diameter. Microscopically, there were mild to marked focal fibrosis, biliary hyperplasia and cellular infiltration with predominantly eosinophils and lymphocytes.

Nakagawa *et al*. (1985) made pathological observations on white spots in the liver of adult breeding pigs and identified four types of lesions macroscopically consisting of minute, mesh- work, lymphonodular and compact types. Histologically, there were three types viz., eosinophilic interstitial hepatitis with intralobular haemorrhage and necrosis and degeneration of interlobular arterioles, interstitial connective tissue proliferation with occasional mild lymphocyte and eosinophil infiltration and lymphonodular hyperplasia. They opined that the first and third histological types seem to have been caused by *A. suum*, but the second type was not necessarily associated with it.

Bernardo and Dohoo (1988) examined 386 pigs from 15 herds on Prince Edward Island. The study showed that 34.6% pigs had intestinal ascarids, 82.4% had milk spot lesions on the liver and in 51.3% of the pigs milk spots occurred in the absence of intestinal ascarids.

Sanford and Josephson (1991) reported severe parasitic hepatits in pigs of five farms caused by ascarid larval migration. Grossly, the lesions consisted of large number of milk spots up to 2.0 cm in diameter scattered throughout the liver. Haemorrhages and necrosis, accompanied by large numbers of eosinophils filling entire lobules were seen microscopically. The interlobular septa were expanded 5-10 times their normal width by exudates consisting of fibrin, large number of eosinophils and a few mononuclear cells and mild fibroplasia. Less severe lesions had fewer eosinophils, more mononuclears, more prominent fibrosis and no interlobular haemorrhages. Microgranulomas also occurred in interlobular septa in livers of pigs from one farm.

Thorough evaluation of the gross appearance of liver lesions, sometimes combined with histopathology and bacteriological examination, resulted in condemnation of 2651 livers (11.0%) because of ascariasis (milk spots) out of 23,000 pigs examined by local meat inspection authority in Haugesund, Norway (Alfredsen, 1992).

Hoshino *et al.* (1995) reported the lymphonodular lesions in the swine lung due to migration of *A. suum* larvae. Lymphonodular white spots were detected in the lung of slaughtered pigs with pulmonary petechiae and multiple milk spots in the liver. These nodules mainly consisted of follicular hyperplasia of lymphoid cells, including a few sections of third stage larvae of *A. suum* at the center of the foci in some cases.

Rajkhowa (1996) studied the incidence of *A. suum* infection in pigs in faecal samples as well as slaughter house materials in Meghalaya. Out of 421 pigs examined, the percentage of animals affected were 23.99. *A. suum* showed highest rate of infection (69.30%) in 0-3 months age pigs and 8.91% infection in above 6 months age pigs.

Rao and Paliwal (1996) studied the pathology of ascariasis affecting pig liver. The liver showed diffuse fibrotic lesions with numerous spherical white spots ranging from 0.2-0.5 cm in diameter. Histologically, the white spots revealed massive focal lymphofollicular aggregates encircled by connective tissue replacing liver parenchyma and multiple necrotic granuloma having caseonecrotic mass in the centre and infiltration of histiocytes, epithelioid cells,

eosinophils and lymphocytes with fibrous tissue encapsulation. Cross section of *ascaris* nematode larvae was seen in these lesions.

Two surveys examining the prevalence and intensity of *A. suum* were conducted by Wanger and Polley (1997) at an abattoir in Canada and out of 2,500 livers examined in the first survey, 44.0% displayed some degree of scarring due to *ascaris* migration and 8.0% were severely damaged. In the second survey, out of 500 livers examined, 50.0% had lesions typical of ascarid migration and 53.0% had some evidence of ascarid infection.

Schuh *et al*. (2000) observed milk spots in the liver in 47.5% of slaughtered pigs. Some new aspects of pathology, pathogenesis and etiology of disseminated lung lesions in slaughtered pigs were studied by Liljegren *et al*. (2003). During the study lungs, spleen, liver and kidneys were subjected to an extended macroscopic and microscopic examination from 40 pigs rejected for human consumption due to an apparent presence of pyemic lung lesions. The livers contained multiple areas of chronic interstitial fibrosis related to migration of *A. suum* larvae and such hepatic lesions were significantly related to the simultaneous occurrence of disseminated pulmonary ecchymosis with a central area of fibroplasia .

A survey on *A. suum* infection in north-western Italy was carried out by Rambozzi *et al*. (2006). A total of 223,339 pigs from 270 different farms were slaughtered and *A. suum* was found in 202 (74.8%) farms and in 4,403 (19.7%) pigs.

Gaurat and Gatne (2009) described the gross and histopathological lesions associated with helminthic infestations in pigs. In *A. suum* infection, large number of opaque spots measuring 2.0-7.0 mm in diameter were observed on the surface of liver. Microscopically, areas of focal necrosis and calcification surrounded by macrophages, mononuclear cells and fibroblasts were observed.

Miclaus *et al*. (2009) studied the vascular changes induced by the migration of *A. suum* larvae through pig liver. The structural changes in affected blood vessels seemed to have a multistage evolution. At first stage, endothelium was affected with vacuolar degeneration and detached from basement membrane and endothelial cell hyperplasia was noticed in other vessels. Proliferating connective tissue from mean vessel wall region became dominant in detriment of muscular tissue. In this manner, muscular cells were dislocated and anarchically oriented. In some situations, some small vessels had complete fibrosis.

2.2.3.2 Stephanurosis

Ashizawa *et al*. (1972) described the pathological findings in *Stephanurus dentatus (S. dentatus)* infection in pigs. The kidneys and ureters from 13 infected pigs were examined and four of which were infected bilaterally showing tissue destruction with haemorrhages and necrosis by migrating worms and cyst formation. The cysts were found in the renal calyces and pelvis, the ureteral wall and peri-ureteral tissues. The cysts opened directly into urinary tract or by some fistulous passages. The ureteral lesions tended to be more severe than the renal lesions.

The incidence of stephanuriasis in the Ghanaian dwarf pigs was studied by Sapong (1972) by examining the internal organs of animals slaughtered, at the Accra abattoir. The results of 3 years investigation showed an infection rate of 33.0%. The effect of the infection and the body's reaction to it was studied by infecting worm-free pigs with the larvae of *S. dentatus*. Results indicated that the disease caused emaciation in the affected animals, due to the extensive damage done to the internal organs.

Swine parasites causing liver condemnations were studied by Batte *et al.* (1975). Extensive damage in the liver due to the migration of larvae of the kidney worm was evident from extremely widespread fibroblastic reaction. Numerous parasitic infiltrations and cellular reaction were seen throughout the hepatic lobules. In more advanced cases, there was atrophy of the liver lobule which was replaced by fibrous tissue.

Dykova (1977) studied the detailed pathology of natural *S. dentatus* infection in 261 pigs. More extensive damage was noticed in the liver than other organs, both in acute phase of larval migration and later in the course of the disease, due to extensive capillary damage and thrombus and granuloma formation with considerable loss of parenchymatous tissue. Young adult worms were found in the kidney parenchyma that was also showing extensive interstitial nephritis and granulomas often egg contained in various stages of development.

Harwell *et al.* (1977) experimentally infected one male pig with *S. dentatus* infective larvae. Necropsy at 170 days post infection showed pale, contracted,

diffusely fibrotic liver that contained live and dead larvae of *S. dentatus*. The portal vein contained an occluding thrombus, which extended into the liver that was composed almost entirely of *S. dentatus* larvae. They were also present in the caudal vena cava immediately cranial to the liver and in the perirenal tissue and renal medulla.

S. dentatus was found in 7 of 11 feral swine and in two domestic sows of a herd maintained on an Island near Yarmouth, Canada by Smith and Hawkes (1978). Worms were recovered by dissection of perirenal fat, kidneys, ureters and adjacent organs and tissues. In one instance, no adults were found but *Stephanurus* larvae were identified histologically in the liver.

Rao (1980) observed the renal pathology in 60 kidneys collected from 38 pigs at municipal abattoir in Bombay and found kidneys of two animals to be infected with *S. dentatus* .

Hutchinson and Wanduragala (1981) conducted serodiagnosis of *S. dentatus* infection in pigs. At post mortem examination, infected pigs had fibrotic lesions in livers and portal thrombi containing *S. dentatus* even after 270 days.

Ashizawa *et al.* (1988) reported the hepatic lesions in pigs infected with *S. dentatus* which consisted of haemorrhaging, degeneration and necrosis accompanying invasion, penetration or migration of worms, cellular infiltration and aggregation, abscess formation and proliferation of encapsulated connective tissue. Vigorous proliferation of connective tissue in the area of the lesion lead to atrophy of liver parenchyma in the peripheral lesion.

Macroscopic investigation was conducted on 33 specimens of urinary organs of pigs with kidney worm disease by Ashizawa and Moritomo (1990a). The lesions caused by parasitism of swine kidney worms were seen in 30.3% of the renal capsules, 45.5% of the proximal areas of the renal sinus, 60.6% of the walls of the renal pelvis and renal calix, 24.2% of the renal parenchyma and 75.8% of the ureteral walls and periureteral regions. The lesions seen in the ureteral wall and renal pelvis were able to be roughly classified into those caused by migrating worms and the cysts. The migration of the worms caused destruction of the tissue, haemorrhage, necrosis and abscess formation, whereas the cyst was a fibrotic pouch containing the worms and pus component and was communicating through a fistula with the pelvic cavity and ureteral cavity.

Ashizawa and Moritomo (1990b) described the histopathological findings in specimens of the urinary organs derived from 19 pigs parasitized with *S. dentatus*. When swine kidney worms penetrated the wall of ureter or the wall of renal pelvis and calix to parasitize, formation of tunnels or cysts were observed macroscopically. Microscopically, the innermost layer of the coastal tissue of these tunnels and cysts consisted of thin degenerative and necrotic zone, while its outside consisted of granulation tissue layer, which was further surrounded by connective tissue layer. Specific large cells which seemed to be modified type of large mononuclear cells appeared in large number in the inner zone of the granulation tissue layer, while a lot of small round cells and a few eosinophils were observed infiltrating in the outer zone. Cell infiltration in the connective

tissue was mild. Around cysts, tunnels and abscesses, hyperplasia of connective *tissue was noticed.*

Rao and Paliwal (1996) noticed stephanuriasis in 34 (40.96%) livers when 242 pigs were examined. Grossly, the liver was enlarged, hard and nodular in appearance. On cutting, the nodules revealed pulpy parenchyma with presence of parasite. Histologically, disruption of hepatic architecture, perilobular fibrous tissue proliferation and infiltration of eosinophils, a few polymorphs and mononuclear cells mainly lymphocytes, plasma cells and macrophages were the conspicuous features and the nodules revealed necropurulent exudates and cross section of adult *S. dentatus* parasite.

Das (1997) studied the prevalence of *S. dentatus* in indigenous pigs in and around Pantnagar during January 1991 to December 1994. Out of 282 slaughtered pigs examined, 27 (9.57%) were found to be infected with *S. dentatus*. The parasites were found in cortex, medulla and peritoneal fat either in free state in haemorrhagic zone or in the cyst of 2-4 cm diameter filled with serosangunious fluid in the renal calyces and pelvis. The wall of ureter was haemorrhagic, inflammed and oedematous. The infected kidneys were swollen with petechial haemorrhage in the cortex and in 3 cases whitish spots were seen on the surface.

Ashok Gupta *et al*. (2000) studied the pathomorphological lesions of stephanuriasis in Andaman and Nicobar islands. During the postmortem examination of 9-12 months old pig, liver was found to be pale, enlarged and firm with uneven consistency. Numerous cysts were found in perirenal fat. Both

ureters were enlarged, thickened and cystic and revealed the presence of worms in each cyst. Microscopically, the liver showed the proliferation of fibrous tissue in perilobular locations and at some places, there was extensive portal fibrosis with infiltration of eosinophils and other inflammatory cells. Renal lymphnodes revealed reticular hyperplasia and large amount of hemosiderin. In ureter, the wall was thickened and at some places the epithelial lining was desquamated. Sub-epithelial lining was oedematous with cellular infiltration and fibroplasia.

Gaurat and Gatne (2009) encountered kidney worms in the pelvic and medullary regions of the kidney in slaughtered pigs showing fibrosis of adjacent parenchyma. Histopathological picture revealed extensive fibrosis and moderate infiltration of mononuclear cells in cortical portion near the hylus and the tubules and glomeruli in the adjacent tissue showed pressure atrophy with degenerative changes.

2.2.3.3 Metastrongylosis

Gross and histopathological changes noticed in the lungs in pigs with *Metastrongylus salmi (M. salmi)* infection were atelectasis, mottling on the surface of the affected parts, frothy serosanguinous exudation, innumerable lungworms in the bronchi and bronchioles along the ventral borders of the diaphragmatic lobes, presence of haemorrhages in the alveoli and infiltration of eosinophils (Bhattacharya *et al*., 1971).

Ramachandran and Sivadas (1971) described the lesions in *Metastrongylus apri* (*M. apri*) infection in pigs as emphysematous bullae separated by dark red

depressed areas of collapse in the caudal portions of diaphragmatic lobes of lungs and thick yellow inspissated pus plugging the bronchi with numerous parasites.

Chauhan and Rao (1972) reported 28.57% of verminous pneumonia cases caused by *M. apri* of all pulmonary affections of the pigs. They noticed male and female worms in the bronchioles surrounded by excess of mucous, ova with coiled larva inside, hyperplasia of bronchiolar epithelium, mucus metaplasia and lymphocytic hyperplasia in the walls of the bronchioles. Alveoli contained characteristic ova surrounded by macrophages, giant cells and desquamated epithelial cells along with the thickening of alveolar walls due to lymphocytic infiltration, proliferation of macrophages and varying degrees of fibrosis.

Giant cell pneumonia associated with lung worm infection was observed by Chaterjee *et al.* (1975) in three country bred piglets. Stockdale (1976) described the lesions of pneumonitis caused by *Metastrongylus elongatus (M. elongatus*) infection in swine as focal interstitial pneumonitis, production of granulomata, atelectasis, fibromuscular hyperplasia of alveoli, alveolar epithelialization or foetalization, presence of mononuclear cells, macrophages, neutrophils, eosinophils, giant cells and possibly fibrin in the lumina of affected alveoli.

Baruah *et al*. (1985) recorded the lung lesions associated with pulmonary metastrongylosis. Out of 190 pigs, fifteen lungs showed changes associated with *M. apri* infection. In one lung, sclerosis of a blood vessel was noticed in the peribronchiolar area.

Ashok Kumar *et al. (*2000) studied the pathology of parasitic pneumonia in pigs. Out of 200 pigs examined, 21 had parasitic lesions, which included *M. apri* infection in 20 and hydatidosis in a solitary case. The gross lesions in *M. apri* infection were in the diaphragmatic lobes particularly in the caudal portion consisting of patchy emphysematous bullae alternating with focal areas of collapse. In addition, irregularly distributed islands of whitish and firm pulmonary tissues were also seen. Microscopically, cross section of adult parasites and their ova were observed in the lumen of bronchi and bronchioles. The bronchial epithelium was hyperplastic with severe goblet cell activity and submucosal glands showed hyperplasia. The adjacent parenchyma revealed well-demarcated haemorrhagic tracts and moderate to severe infiltration of eosinophils and lymphocytes and multinucleated giant cells. Peribronchial cuffing of mononuclear cells and sclerosis of blood vessels around the bronchi were also observed.

Ashok Gupta *et al*. (2002) studied the prevalence of metastrongylosis infection associated with swine fever in pigs in Andaman and Nicobar islands. During an outbreak of swine fever, many post-mortems were performed and in almost all cases adult lung worms were observed in the bronchi and bronchioles of the affected animals. The lungs revealed areas of focal consolidation and trachea, bronchi and bronchioles were filled with frothy exudates along with the adult worms.

Prevalence of lung worms belonging to the genus *Metastrongylus* was surveyed in 42 Japanese wild boars during April 2000 to April 2001 in the western region of Tokyo, Japan by Morita *et al.* (2007). The number of parasites

was highest in the caudal lung lobes. Four species, *M. elongatus, M. salmi, Metastrongylus asymmetricus (M. asymmetricus)* and *M. pudendotectus* were identified. All the boars were infected with 2 or more species and 64.3% of boars had all four species.

Gaurat and Gatne (2009) studied the gross and histopathological lesions associated with helminthic infestations in pigs. Grossly, the affected lobe of lung with *M. elongatus* revealed peripheral emphysema, patchy congestion and focal consolidation with excessive mucus in parasitized bronchi. Histopathological analysis showed cross section of worms with leucocytic infiltration in the surrounding mucosa, emphysema in the associated alveoli and aggregation of lymphocytes with proliferation of fibrous tissue in the interlobular septae.

Nosal *et al.* (2009) surveyed the presence of lung nematodes of the genus *Metastrongylus* in wild boars in hunting area of southern Poland during consecutive winters of 2008. A total of 17 wild boars were necropsied and their lungs were removed, divided into smaller parts and examined along the bronchial lobes. The prevalence of lungworms reached 76.5% and all of the 13 infected animals had two or more parasite species. Out of 250 identified lung worms, 48.8% were *M. pudendotectus* and 32.8% were *M. elongatus* and 14.0% belonged to *M. salmi,* whereas 4.4% were *M. asymmetricus,* which were recorded for first time in Poland.

2.2.3.4 Trichinellosis

A total of 22,451 swine diaphragms were examined by Zimmermann and Zinter (1971) from all geographic regions of United States for the presence of *Trichinella spiralis (T. spiralis)* from November 1966 through October 1970. Twentyfive of 20,003 farm raised butcher swine, none of 1,858 farm raised breeder swine and three of 590 garbage fed swine were positive for trichinae and the prevalence (0.33%) obtained during 1970 was the highest yearly rate of the decade.

Henriksen and Clausen (1977) demonstrated *T. spiralis* larvae in muscle samples of 4 animals, out of total 137 wild boars examined during the period from 1972 to 1977.

Jarzebski (1979) examined 370 and 317 cases of pig trichinellosis in 1975 and 1976 respectively by using trichinoscopy in Poland and found 0.0019% *T. spiralis* infection in two years.

The distribution of *T. spiralis* larvae in selected muscles and organs was studied by Kotula *et al.* (1984) in thirty two Hampshire-Yorkshire pigs (6 to 8 wk old) by inoculating with the Beltsville strain at a level of 880 larvae/kg body weight. At 100 kg, the pigs were slaughtered and 10 g samples were digested in pepsin-HCl and were examined microscopically for *T. spiralis* larvae. The mean number recovered/gram was, 452 in tongue, 391 in diaphragm, 130 in obliqous abdominis internus, 116 in serratus ventralis, 105 in psoas major, 100 in triceps brachii, 83 in biceps femoris, 74 in semitendinosus, 60 in intercostals, 58 in semimembranosus and 37 in longissimus dorsi. Larvae were found in one sample

each of the blood, brain and kidney, in two samples of the heart and four samples of lymph tissue.

The diaphragms of slaughter pigs were examined by Schad *et al.* (1985) from Newzealand states for the presence of *T. spiralis* infection from June to December, 1983. Out of 5,315 pigs examined, 39 (0.73%) were found infected and the infections were light, none exceeding 30 larvae/g of tissue.

A small survey of pigs slaughtered in an abattoir was undertaken by Bjorland *et al.* (1993) to study *T. spiralis* infection in Bolivian Altiplano during September 1991. In a group of 100 pigs slaughtered consecutively on a single day, two (25%) of eight pools were found positive when tested by the pooled digestion method.

Pozio and Kapel (1999) found *Trichinella nativa (T.nativa)* infection in two wild boars and *T. spiralis* infection in 15 domestic pigs, out of total 17 *Trichinella* isolates from Estonia.

Nine farmed wild boars from a single farm were condemned at meat inspection out of 25 slaughtered animals, because of trichinellosis (Oivanen *et al.*, 2000).

The epidemiology of swine trichinellosis has been reported in 26 provinces of China by Wang and Cui (2001b). The prevalence ranged from 0.12% to 34.2% in 5 provinces and from 0.01% to 0.0001% in other provinces. There was a high infection rate of *Trichinella* in pigs slaughtered at abattoirs in autumn and winter.

Larrieu (2004) investigated the porcine infection with *Trichinella* in Argentina and 300 pigs slaughtered for consumption between 2000 and 2002 were checked by artificial digestion of a muscle sample. Four (7.3%) of the 55 checked in 2000, five (4.8%) of the 105 investigated in 2001, and three (2.1%) of the 140 investigated in 2002 were found infected.

Gamble *et al.* (2005) conducted routine inspection in wild boars intended for human consumption in December 2001 that revealed the presence of *Trichinella* spp. larvae. Biological, morphological and genetic analysis demonstrated the parasite to be *Trichinella pseudospiralis (T. pseudospiralis)*.

Pozio (2007) studied the world wide distribution of *Trichinella spp.* infections in animals and humans. In Poland, the prevalence in wild boars was reported to be 0.25% and the yearly incidence of *T. spiralis* in domestic pigs ranged from 0.0032 to 0.00036%. *T. pseudospiralis* was detected during routine examination at the slaughter house in four swine from a farm of eastern Slovakia. In Romania, the incidence of trichinellosis was 0.15% and 1.3% in domestic pigs and wild boars respectively.

Gamito-Santos *et al.* (2009) reported the histopathology of trichinellosis in wild boars and compared the lesions of early infection loads with those of more prolonged loads for two species of *T. spiralis* and *Trichinella britovi (T. britovi).* In *T. spiralis,* recently infected wild boars showed larvae with lymphocytes and plasma cells and slight to moderate inflammation was evidenced in *T. britovi* infection. In prolonged infections with *T. spiralis*, massive infection with

moderate interstitial reaction formed by lymphocytes was noticed and in case of *T. britovi,* plasma cells and few larvae with slight inflammatory reaction along with calcifications were evident.

The first occurrence of *Trichinella murrelli* (*T. murrelli*) in a wild boar in Iran was studied by Kia *et al*. (2009). By direct microscopic examination, *Trichinella* larvae were visible inside thick collagen capsules. In the histological sections of the muscle, encapsulated *Trichinella* larvae were observed, surrounded by an inflammatory reaction.

2.2.4 Miscellaneous diseases

Davenport and Stockdale (1967) recorded a case of hyostrongylosis in a four year old sow in Ontario. At necropsy, the gastric mucosa was covered with clear tenacious mucus. Numerous red, thread-like worms 5 to 10 mm long were seen beneath the mucus. Microscopically, the lumina of the pits of the gastric mucosa were distended in many areas to produce cyst-like structures. These were lined by cuboidal epithelial cells and many contained cellular debris. It appeared that mucus metaplasia of gastric glands had occurred. The lamina propria was extensively infiltrated by lymphocytes and eosinophils.

Rajan *et al*. (1975) studied the incidence and pathology of necrotic enteritis in pigs. Out of 462 animals, 196 piglings of age group of 1 to 4 months died showing lesions of necrotic enteritis. At autopsy, striking lesion was seen in the colon which appeared as prominent thickened spirals. From the serosa dense

opaque grayish white patches were evident coating the mucosa. In certain instances the portion of the colon was devoid of any ingesta but contained only cheesy granular necrotic flakes. The distribution of necrotic patches were either in the form of isolated grayish patches or diffuse irregular bands. During the study, in the colon of some animals, *Oesophagostomum spp.* were encountered. Microscopically, large cystic spaces containing granular necrotic materials were seen opening into the lumen of the intestine in the sections of colon that harboured the parasite.

Out of 190 pig lungs collected by Baruah *et al*. (1985), three lungs showed the lesions associated with *paragonimus* infection. Grossly, the lungs were enlarged and pink red in colour. In all the cases, brownish circular raised cysts of about 2cm in diameter were observed on the dorsal surface of the caudal end of the diaphragmatic lobe. On incision, the cyst revealed a pair of brownish flukes of about 1 cm in length having tapering end. Microscopically, the lung parenchyma showed the cross section of ova, embryonated ova and immature stages of the parasite within the alveoli and bronchioles. In some areas, the ova were seen in clusters, some of which were calcified and surrounded by cellular infiltration of mononuclear cells, macrophages and eosinophils with dense fibrous tissue encapsulation around forming granuloma and on many occasions with giant cells engulfing the ova. Bronchial and bronchiolar epithelia showed mild hyperplastic changes and inter alveolar septa were very much thickened.

Dev Sarma and Gogoi (1986) described the lesions of *F. buski* in intestines of pigs. Grossly, catarrhal enteritis and congestion in mucosal surface

of the small intestine were seen. Microscopically, the intestines revealed thickening of mucosa and submucosa with the infiltrations of lymphocytes, plasma cells and eosinophils. The glands of submucosa were found to be hypertrophied. In certain areas, there was formation of necrotic membrane over the mucous surface.

Histopathological studies of the lesions of caecum caused by *G. hominis* in pigs were carried out by Dev Sarma and Gogoi (1986). Grossly, circular papilla-like areas were observed in the mucosa of caecum. Microscopically, the areas revealed necrosis of the cells of mucosa and submucosa with some replacement by fibrinous exudate. The fibrinous exudates mostly consisted of eosinophils, plasma cells, lymphocytes, neutrophils and other cellular damage. In some, hyperplastic changes in the epithelium of mucous membrane and nodule formation in the transverse muscular layer were observed. In addition, they also described the lesions in infection of *A. sufrartyfex* in pigs. Grossly, catarrhal enteritis and congestion in the mucosal surface of the small intestine and microscopically, desquamation of the surface epithelium and infiltration of lymphocytes and eosinophils were found.

The histopathology related to *F. buski* infection reported by Datta *et al.* (2004) revealed haemorrhage, ulceration and denudation of epithelial layers with infiltration of mononuclear cells like lymphocytes, plasma cells, macrophages and eosinophils in the intestinal tissues of the pigs.

Gross and histopathological lesions produced by *A. strongylina* were studied by Gaurat and Gatne (2009) during May 2002 to August 2002. The gastric mucosa of fundic region was swollen and thrown into characteristic folds. Numerous reddish nematodes were found embedded in the thick mucous layer forming clusters at few places. Histopathological findings revealed extensive hyperplasia of goblet cells, eosinophilic infiltration of lamina propria and denudation of epithelium of the villi. Further, they also described the histopathological lesions pertaining to *F. buski* and *A. sufrartyfex* infection in pigs. Denudation of epithelial lining of the intestinal villi and diffuse infiltration of numerous mononuclear cells and few eosinophils in lamina propria were noticed in *F. buski* infection and the small intestine showed hydropic degeneration of villous epithelium and extensive eosinophilic infiltration of lamina propria in *A. sufrartyfex* infection.

CHAPTER IV

RESULTS

4.1 INCIDENCE

In the present investigation, a total of 320 pigs of either sex and of different age groups from various slaughter houses, field mortalities, private piggery farms located in and around Gannavaram, Vishakapatnam and Tirupati towns and from postmortem examinations conducted at the NTR College of Veterinary Science, Gannavaram, were subjected to detailed postmortem examination and different organs showing lesions suggestive of parasitic diseases and organs having parasitic infestation were collected to carry out further studies (Table. 1). Majority of the tissue samples collected were heavily infested by a single species of a parasite and in some animals, more than one organ was infested by the parasite. All the collected parasites were identified morphologically. On gross and histopathological examination, 216 (67.5%) animals revealed definitive lesions of helminth parasitic diseases (Table. 2).

In the present study, various helminth parasitic diseases that were observed in pigs were broadly grouped as the diseases caused by trematodes, larvae of cestodes and nematodes and accordingly the pathomorphological lesions were described. The details of different diseases noticed and the number of animals affected were presented in the Table 3 and Chart 1 and 2.

Table 1: Collection of samples

S. No	Sources for Sample collection	No. of samples
01	Slaughter houses and piggery farms located in and around Gannavaram	169
02	Slaughter houses and piggery farms located in and around Visakhapatnam	88
03	Slaughter houses and piggery farms located in and around Tirupati	30
04	Postmortems conducted at Department of Veterinary Pathology, NTR College of Veterinary Science, Gannnavaram	13
05	Field mortality cases	20
TOTAL		320

Table 2: Incidence of lesions of helminth parasitic diseases in pigs

S. No	No. of pigs examined	No. of pigs with definitive parasitic lesions (%)
1	320	216 (67.5%)

Table 3: Incidence of different types of helminth parasitic diseases in pigs

S.No.	Type of disease	Number of animals affected	No. (%)
I	**Diseases caused by trematodes**	**10**	**3.13**
	Schistosomosis	9	2.81
	Opisthorchiosis	1	0.32
II	**Diseases caused by larvae of cestodes**	**28**	**8.75**
	Hydatidosis	4	1.25
	Cysticercosis	24	7.5
III	**Diseases caused by nematodes**	**178**	**55.63**
	Ascariosis	109	34.06
	Stephanurosis	28	8.75
	Metastrongylosis	38	11.88
	Parasitic gastritis (Stomach worms)	3	0.94
	Total	**216**	**67.51**

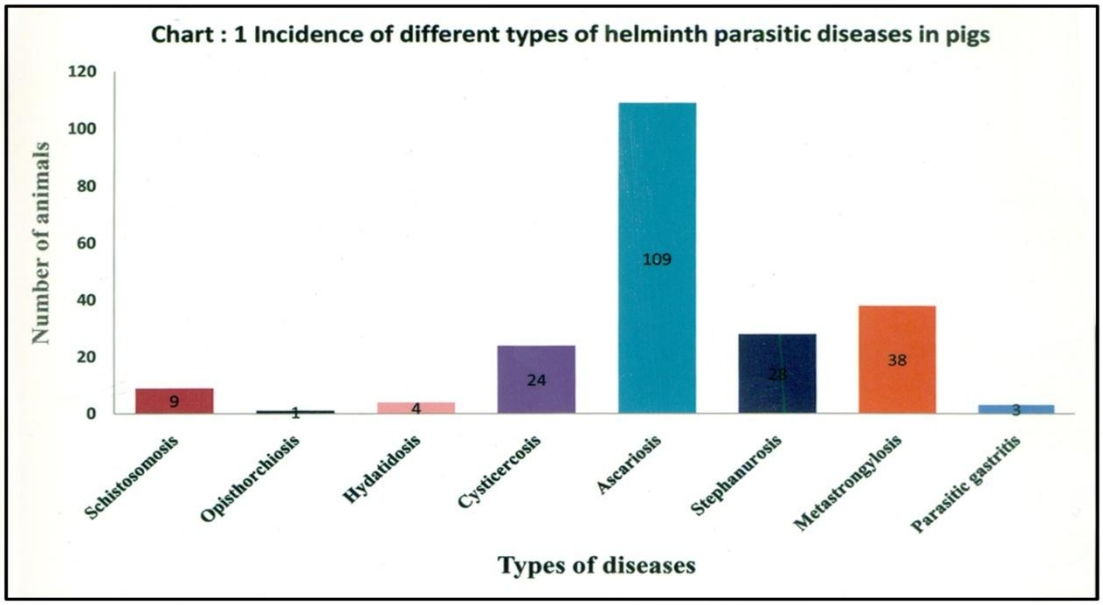


Chart : 1 Incidence of different types of helminth parasitic diseases in pigs
Number of animals
120
100
80
60
40
20
0
9
1
4
24
109
28
38
3
Schistosomosis
Opisthorchiosis
Hydatidosis
Cysticercosis
Ascariosis
Stephanurosis
Metastrongylosis
Parasitic gastritis
Types of diseases

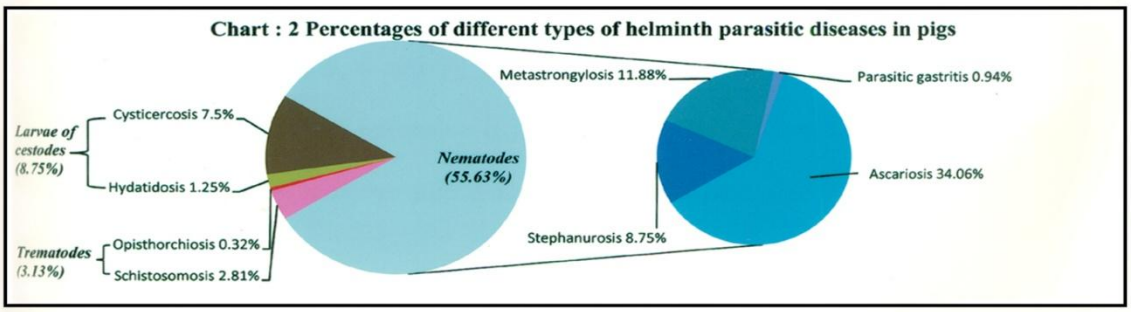


Chart : 2 Percentages of different types of helminth parasitic diseases in pigs
Larvae of cestodes (8.75%)
Cysticercosis 7.5%
Hydatidosis 1.25%
Trematodes (3.13%)
Opisthorchiosis 0.32%
Schistosomosis 2.81%
Nematodes (55.63%)
Metastrongylosis 11.88%
Parasitic gastritis 0.94%
Ascariosis 34.06%
Stephanurosis 8.75%

4.2 Helminth parasitic diseases

4.2.1 Diseases caused by trematodes

4.2.1.1 Schistosomosis

The incidence of schistosomosis was 2.81% out of total 320 animals examined.

Grossly, the affected livers were slightly enlarged and had a granular surface (Fig. 1).

Microscopically, the sections of liver revealed perioval granulomas or pseudotubercles with extensive fibrocellular reaction in the interlobular septa and in the portal triads (Fig. 2 and 3). Adult parasites were not found in the sections. In addition, congestion, degenerative changes in the hepatic cells, bileduct hyperplasia, thickening and destruction of blood vessels, phlebitis, formation of angiomatoids with additional vascular channels, proliferation of fibrous tissue and infiltration by eosinophils and mononuclear cells in the interlobular septum were also found (Fig. 4, 5, 6 and 7). In some areas, the hepatic lobules were separated and individualized by extensive infiltration and fibrous tissue proliferation (Fig. 8 and 9). At few places, lymphoid nodule formation was also evident (Fig. 10).

Figure 1 Schistosomosis : Enlarged liver with a granular surface.

Figure 2 Schistosomosis : Liver - Perioval granuloma in the portal area H & E x 100.

Figure 3 Schistosomosis: Liver - Early stages of perioval granuloma formation in the lumen of portal vein and phlebitis H &E x 400.

Figure 4 Schistosomosis: Liver - Section showing thrombophlebitis H & E x 400.

Figure 5 Schistosomosis: Liver - Thickening of interlobular septa with infiltration H& E x 100.

Figure 6 Schistosomosis: Liver - Section showing fibrosis and infiltration in the interlobular septa H & E x 100.

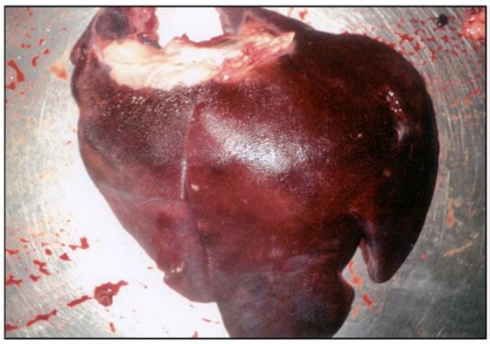


Figure 1

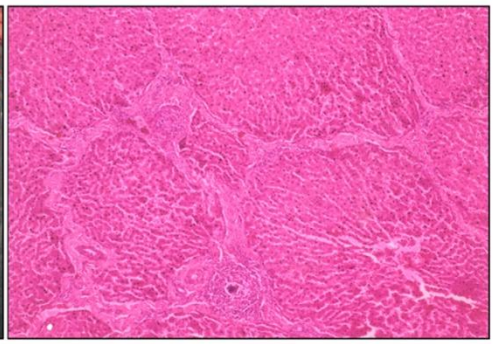


Figure 2

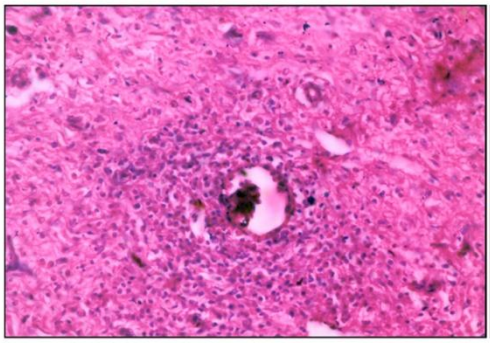


Figure 3

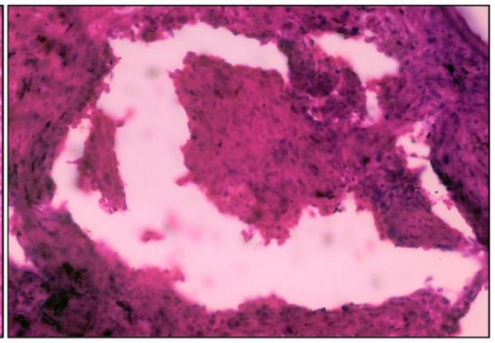


Figure 4

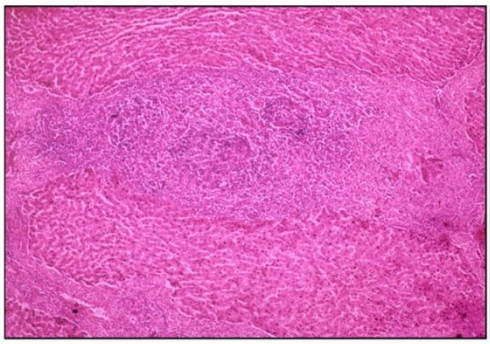


Figure 5

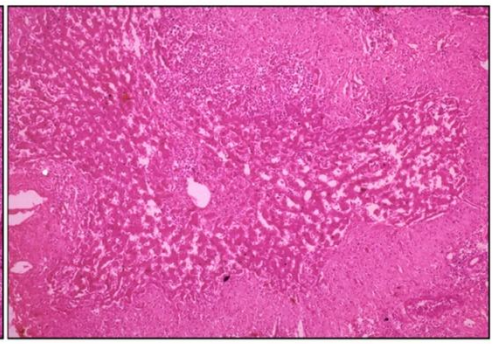


Figure 6

Figure 7 Schistosomosis: Liver - Note the presence of giant cells along with otherinflammatory cells in the interlobular septa H & E x 100.

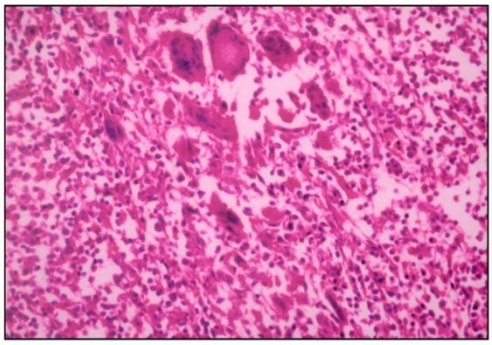
Figure8 Schistosomosis: Liver - Note widening of interlobular septa with fibrosisand infiltration and individualization of hepatic lobules
H & E x 40.

Figure 9 Schistosomosis: Liver - Note the proliferation of fibrous tissue in the
interlobular septa VanGieson x 100.

Figure 10 Schistosomosis: Liver - Lymphoid nodules near perioval granuloma
H & E x 100.

Figure 11 Schistosomosis: Liver - Perioval granuloma in the interlobular septa
H & E x 400.

Figure 12 *Opisthorchistenuicollis* (adult).



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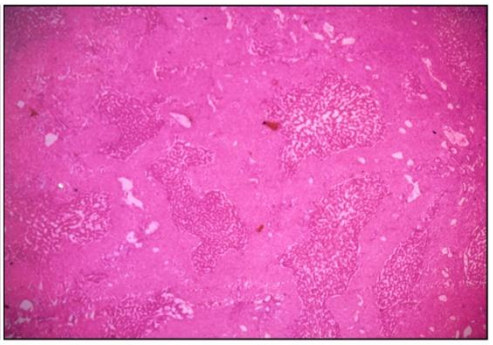

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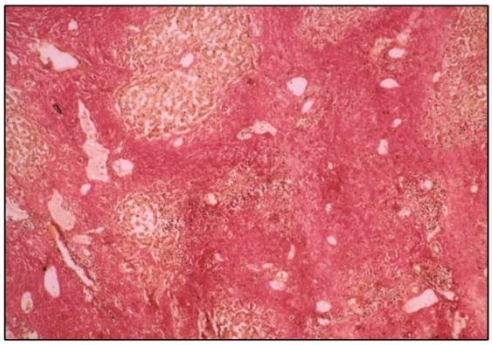

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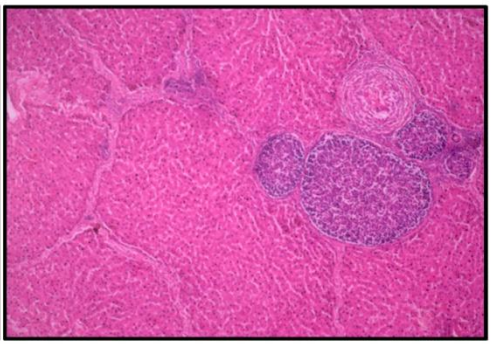


Figure 10

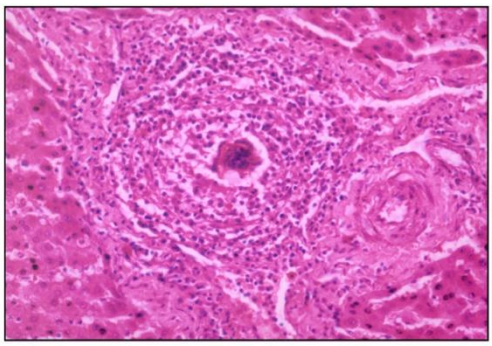


Figure 11

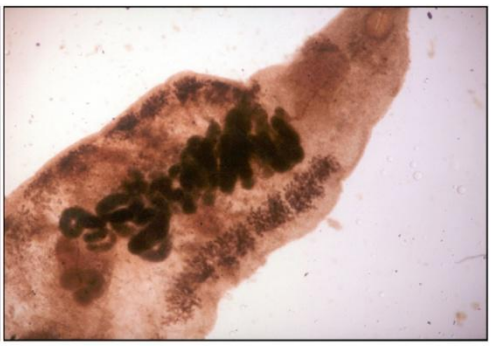


Figure 12

The granuloma contained ova of *schistosoma* and surrounded by eosinophils, macrophages, epithelioid cells, giant cells and lymphocytes along with extensive portal and septal fibrosis resulting in thickening of interlobular septa (Fig. 11).

4.2.1.2 Opisthorchiosis

The incidence of liver fluke infestation was 0.32% of total animals examined.

Grossly, the liver was slightly enlarged and showed moderate increase of interlobular connective tissue. A whitish raised structure was present on the surface of the liver and on opening, thickened, cystic bile duct was noticed containing small flukes measuring 4-10 mm. The flukes were also present in some other branches of the bile ducts, which were identified as *Opisthorchis tenuicollis* (Fig. 12).

Microscopically, the affected part of the liver revealed the presence of cross section of fluke in the bile duct (Fig. 13 and 14) that was extensively thickened, dilated and fibrosed. There was hyperplasia of bile duct epithelium, desquamation and necrosis of epithelial lining, proliferation of goblet cells and glands in the submucosa and infiltration of lymphocytes, plasma cells and macrophages in the mucosa and submucosa (Fig. 15 and 16).

Figure 13 Opisthorchiosis : Liver - Section showing cross section of fluke in the bileductH & E x 40.

Figure 14 Opisthorchiosis : Liver - Section showing cross section of fluke in the bile duct H & E x 100.

Figure 15 Opisthorchiosis : Liver - Note hyperplasia of the bile duct epithelium along with desquamation and necrosis H & E x 400.

Figure 16 Opisthorchiosis: Liver - Proliferation of glands in the submucosa of the bile duct along with infiltration H & E x 400.

Figure 17 Hydatidosis: Note the presence of hydatid cyst on the ventral surface ofliver.

Figure 18 Hydatidosis: Note the presence of hydatid cyst on the dorsal surface of lung.

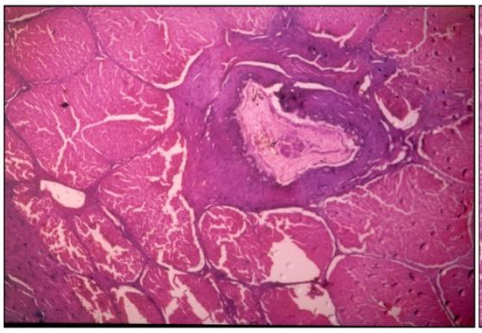
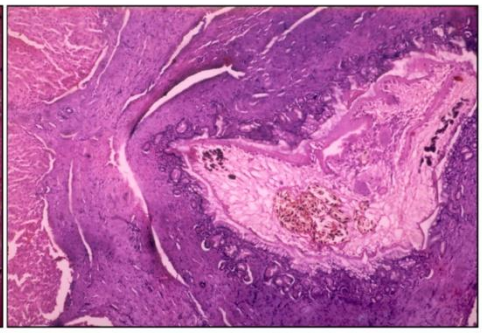


Figure 13



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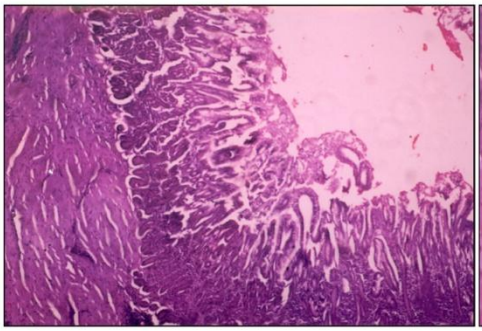

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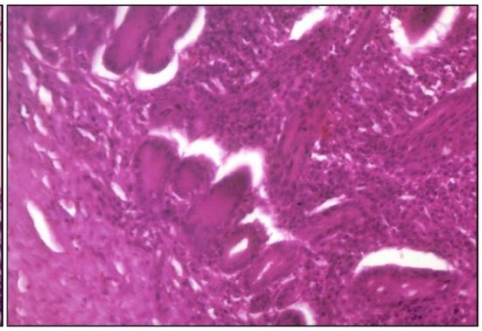


Figure 16

Figure 17

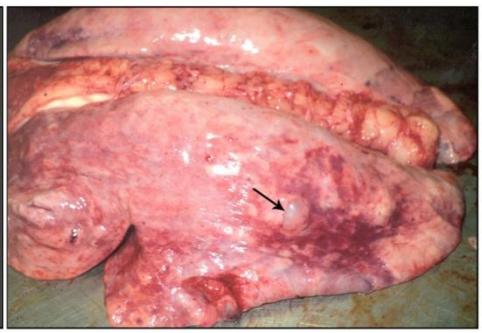


Figure 18

Small lymphoid nodules were also noticed around the bileducts. At few places in the vicinity of bile duct, hepatocytes showed degenerative changes and necrosis. Mild mononuclear infiltration was evident in the portal triads and in the interlobular septa.

4.2.2 Diseases caused by larvae of cestodes (Metacestodes)

4.2.2.1 Hydatidosis

Out of 320 pigs examined, 1.25% of animals showed the presence of hydatid cysts.

Grossly, a single cyst measuring 2.0 - 6.0 cm in diameter was noticed on the ventral surface of liver, and on the dorsum of lung and spleen (Fig. 17, 18 and 19). It contained clear watery fluid and distinct whitish, thick cyst wall. The cyst wall consisted of laminated layer of variable thickness.

Microscopically, the cyst consisted of hyaline cyst wall surrounded by fibrous connective tissue capsule infiltrated with large numbers of eosinophils, mononuclear cells and plasma cells (Fig. 20, 21 and 22). The protoscolices were also noticed attaching to the cyst wall (Fig. 23). In liver, the surrounding hepatic parenchyma revealed vacuolar degeneration, mild bile duct hyperplasia, infiltration of few mononuclear cells and eosinophils and mild proliferation of

Figure 19 Hydatidosis: Note the presence of hydatid cyst on the dorsal surface of spleen.

Figure 20 Hydatidosis: Liver - Section showing fibrous tissue capsule and hyaline cyst wall H & E x 100.

Figure 21 Hydatidosis: Lung - Section showing fibrous tissue capsule and hyaline cyst wall H & E x 40.

Figure 22 Hydatidosis: Lung - Section showing fibrous tissue capsule and hyaline cyst wall H & E x 100.

Figure 23 Hydatidosis: Liver - Presence of protoscolex attached to the cyst wall H& E x 400.

Figure 24 Hydatidosis: Spleen showing some degree of sacculation in the cyst H & E x 100.

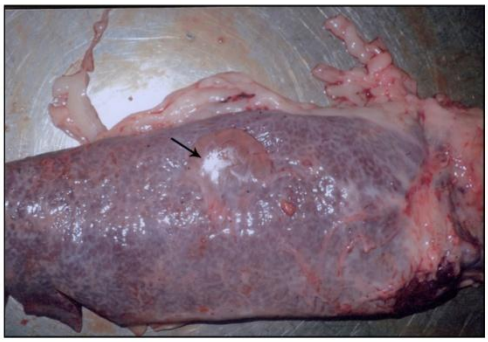


Figure 19

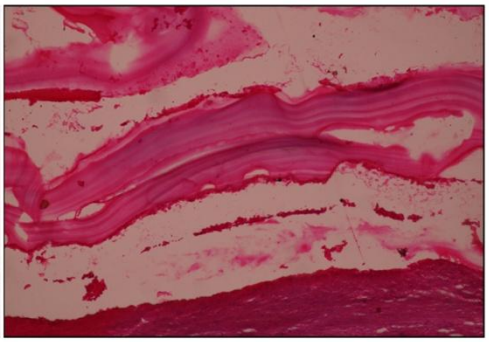


Figure 20

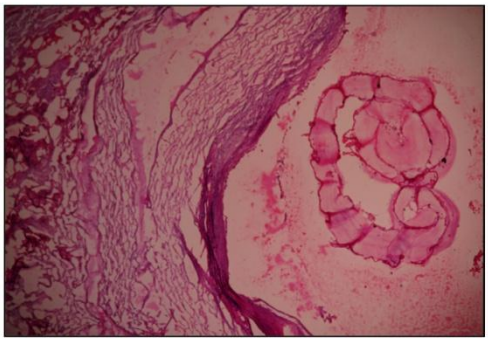


Figure 21

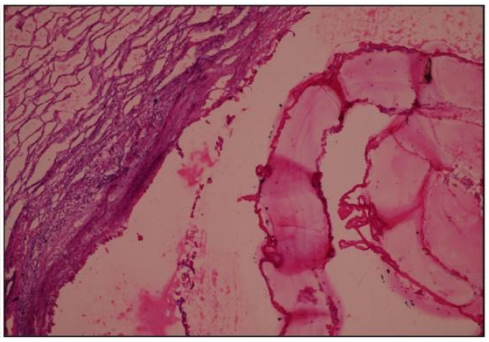
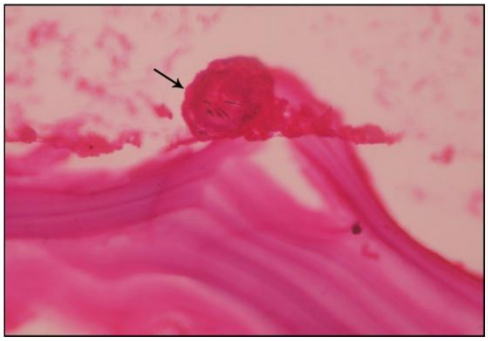


Figure 22



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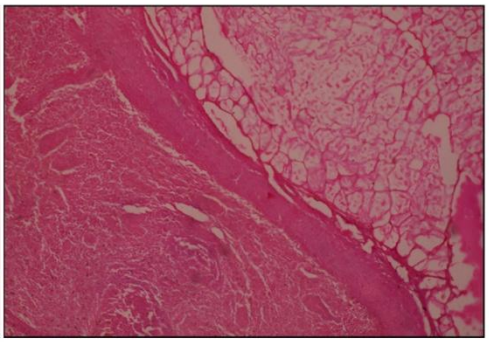


Figure 24

fibrous tissue. In the spleen, the hydatid cyst showed some degree of sacculation (Fig. 24 and 25).

4.2.2.2 Cysticercosis

Cysticercosis was noticed in 7.5% of animals out of 320 animals examined.

The metacestodes of *T. solium* were present in various organs like tongue, heart, liver, skeletal muscles and brain with a diameter of about 0.5 – 1.0cm (Fig. 26, 27, 28, 29, 30 and 31). The cysts were visible and present on or embedded inside different tissues and organs of the carcasses. They were typically spherical or oval, white or yellow vesicle with a translucent bladder wall through which the scolex was seen like a small solid, eccentric granule. In some areas, degenerated cysts were also seen.

Microscopically, the cross sections of cysticerci were evident with or without invaginated scolex in different organs, surrounded by fibrocellular reaction consisting of lymphocytes, plasma cells and eosinophils (Fig. 32, 33, 34, 35, 36, 37 and 38).

Figure 25 Hydatidosis: Spleen showing some degree of sacculation along with presence of protoscolex H & E x 100.

Figure 26 Cysticercosis : Note the presence of cysts on the tongue.

Figure 27 Cysticercosis : Note the presence of cyst on the heart.

Figure 28 Cysticercosis : Presence of cyst in the heart.

Figure 29 Cysticercosis : Note the presence of a cyst on the liver.

Figure 30 Cysticercosis : Note the presence of multiple cysts in the skeletal muscle.

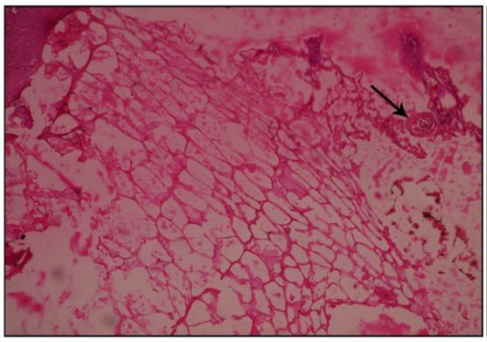


Figure 25

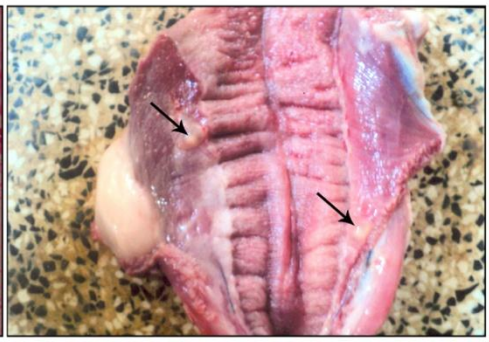


Figure 26

Figure 27

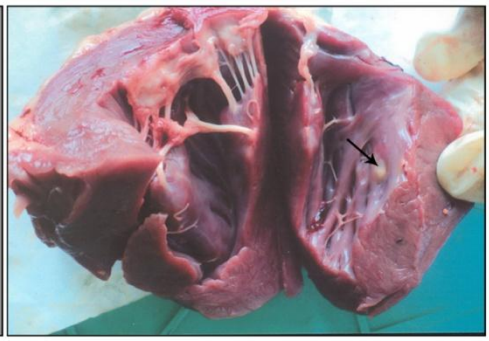


Figure 28

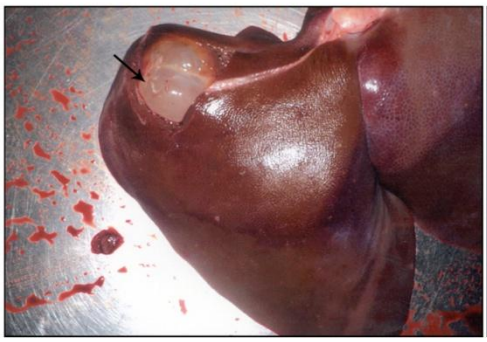


Figure 29

Figure 30

Figure 31 *Cysticercuscellulosae* cysts on the cerebrum.

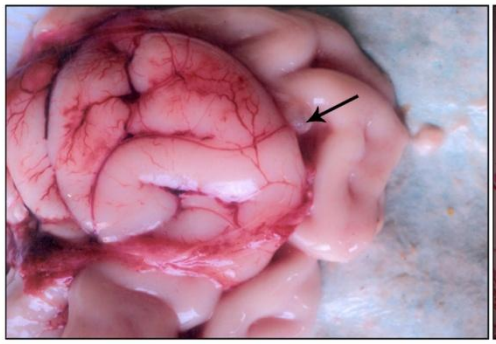
Figure 32 Cysticercosis: Tongue - Note cross section of cysticercus surrounded by fibrocellular reaction H & E x 40.

Figure 33 Cysticercosis: Tongue - Note cross section of cysticercus surrounded by fibrocellular reaction H & E x 100.

Figure 34 Cysticercosis: Heart - Showing cross section of cysticercus H & E x 100.

Figure 35 Cysticercosis: Heart - Showing cross section of cysticercus H & E x 400.

Figure 36 Cysticercosis: Liver - Note cross section of cysticercusH & E x 400.



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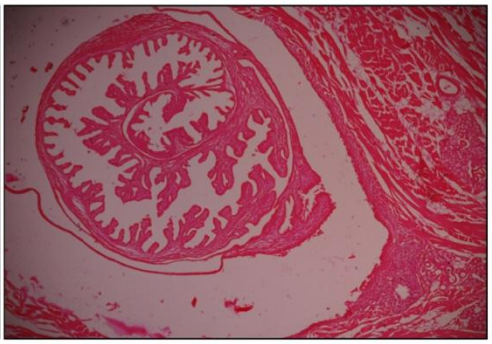

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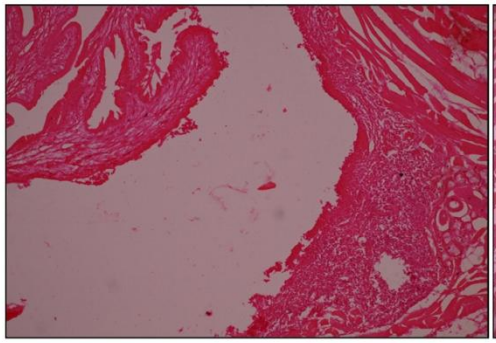

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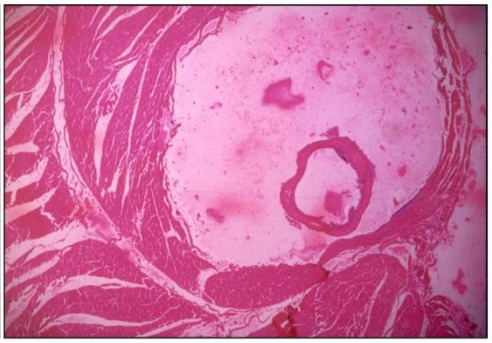

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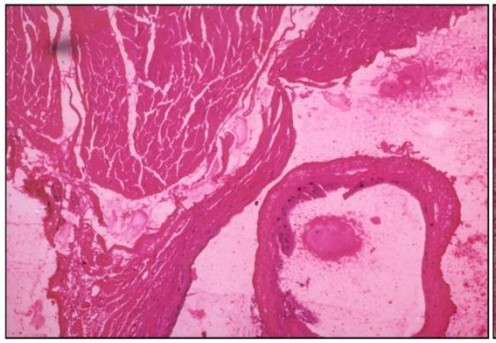

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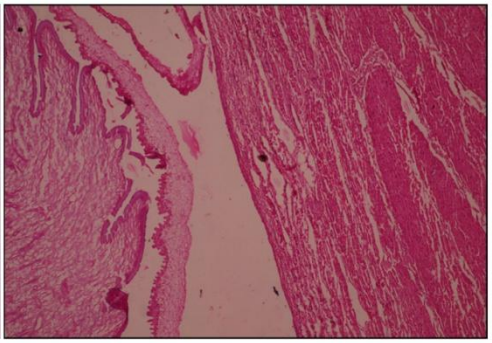

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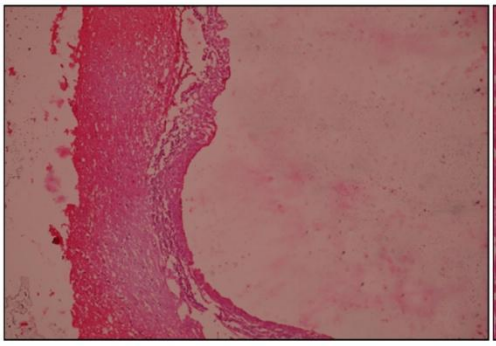

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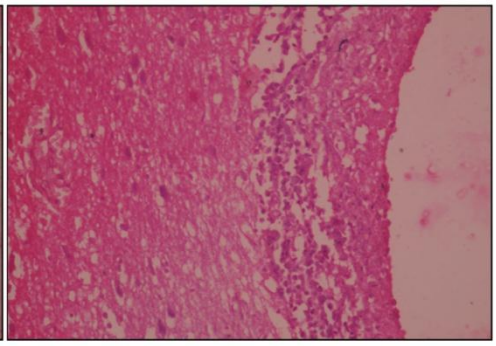

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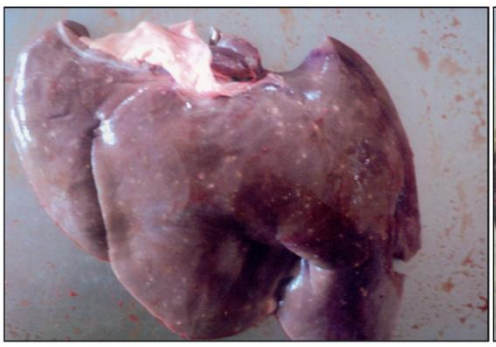


Figure 39

Figure 40

Figure 41

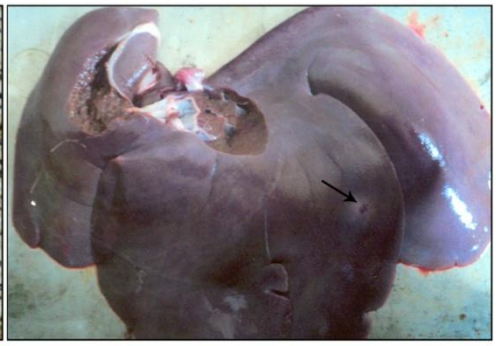


Figure 42

4.2.3 Diseases caused by nematodes

4.2.3.1 Ascariosis (Milk spots / White spots)

The lesions of ascariosis were observed in 34.06% of a total of 320 animals examined. In many animals, livers showed milk spots without the presence of adult worms in the intestines, whereas in some, adult worms were also present in the intestines.

Grossly, the milk spots were star-like in shape and grayish white in colour or of pearl like nodules scattered throughout the liver (Fig. 39, 40 and 41). They were of different sizes and were found simultaneously in the same liver. The diameter of these spots ranged from 0.5-2.5cm. Some spots had haemorrhagic centres (Fig. 42).

Microscopically, the liver sections revealed areas of extensive haemorrhage into the lobule and some times the entire lobule was necro haemorrhagic except for a thin marginal zone of liver cells at the periphery (Fig. 43 and 44). There was distortion of hepatic architecture. In some white spots there was a central lesion in the form of a nodular granulomatous infiltration with massive number of eosinophils and with cut section of larvae (Fig. 45 and 46). These eosinophilic granulomas also revealed areas of necrosis surrounded by cells showing pyknosis and karyorrhexis (Fig. 47). Areas of calcification were also noticed in some of the

Figure 43 Ascariosis: Liver - Note extensive intralobularhaemorrhages and infiltration in the interlobular septa H & E x 100.

Figure 44 Ascariosis: Liver - Note extensive intralobularhaemorrhages and infiltration in the interlobular septa H & E x 400.

Figure 45 Ascariosis: Liver - Eosinophilic granuloma with areas of necrosis and section of ascarid larvae and surrounded by lymphoid follicles H & E x 40.

Figure 46 Ascariosis: Liver - Eosinophilic granuloma with areas of necrosis andsection of ascarid larvae and surrounded by lymphoid follicles H & E x 100.

Figure 47 Ascariosis: Liver - Eosinophilic granuloma with areas of necrosis H & E x 100.

Figure 48 Ascariosis: Liver - Eosinophilic granuloma with areas of necrosis, calcification and lymphoid follicles H & E x 40.

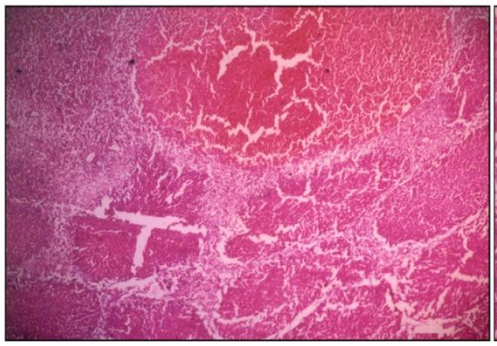
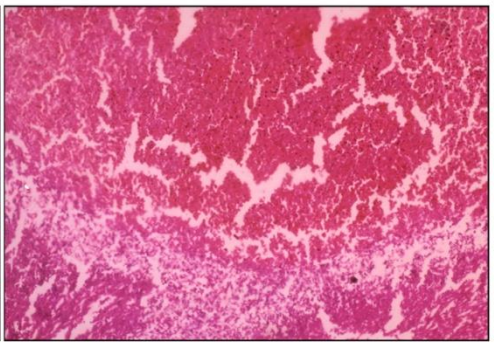


Figure 43



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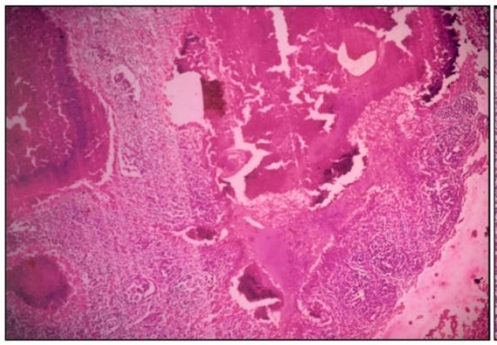

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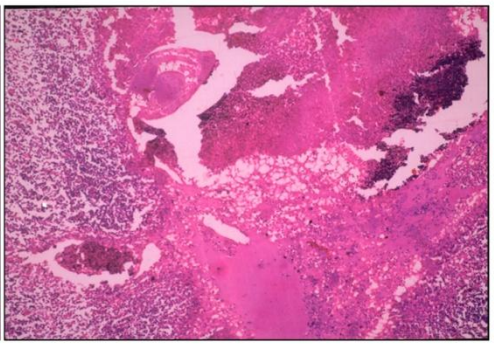

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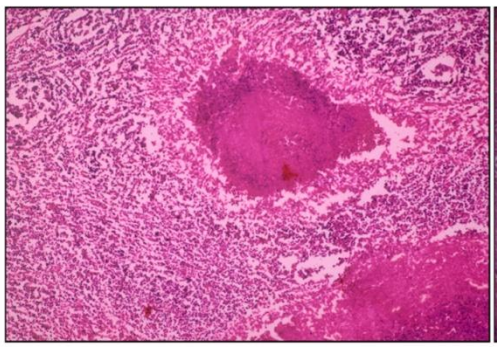


Figure 47

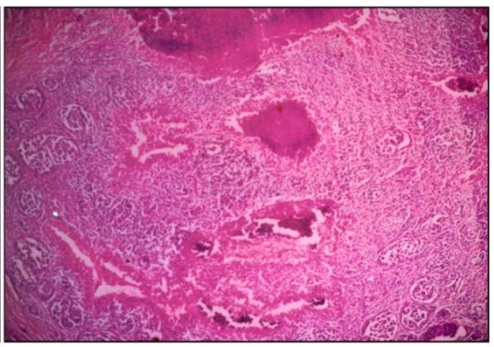


Figure 48

necrotic eosinophilic granulomas and they were surrounded by lymphoid accumulations in a few cases (Fig. 48 and 49). The interlobular septa were markedly thickened with infiltration of eosinophils and a few mononuclear cells and proliferation of fibrous tissue (Fig. 50 and 51). In some livers, extensive thickening of interlobular septa caused reduction in the size of hepatic lobules (Fig. 52 and 53). In more advanced cases, there was atrophy of the hepatic lobules that were replaced by fibrous tissue. Proliferation of bileducts and thickening of walls of blood vessels were also evident (Fig. 54).

The livers that showed sharply defined pearl like nodules grossly, revealed lymphoid follicle formation microscopically. Larval remnants were found in the center in a few of the follicles (Fig. 55). Strands of connective tissue separated the lymphoid follicles from their surroundings or from each other. No intra lobular haemorrhages were seen in this lymphonodular type of white spots.

Adult worms were present in the intestines of some animals (Fig. 56). The intestinal mucosa revealed desquamation of epithelium and infiltration by lymphocytes, plasma cells and macrophages (Fig. 57, 58 and 59).

4.2.3.2 Stephanurosis

The incidence of stephanurosis was 8.75 % in the total of 320 animals examined .

Figure 49 Ascariosis: Liver - Eosinophilic granuloma with areas of necrosis, calcification and lymphoid follicles H & E x 100.

Figure 50 Ascariosis: Liver - Infiltration in the interlobular septa H& E x 100.

Figure 51 Ascariosis: Liver - Section showing infiltration of eosinophils and a few fibroblasts in the interlobular septa H & E x 40.

Figure 52 Ascariosis: Liver - Note extensive thickening of interlobular septa and reduction in the size of hepatic lobule H & E x 100.

Figure 53 Ascariosis: Liver - Infiltration and individualization of hepatic cells H & E x 400.

Figure 54 Ascariosis: Liver - Note bile duct proliferation H & E x 100.

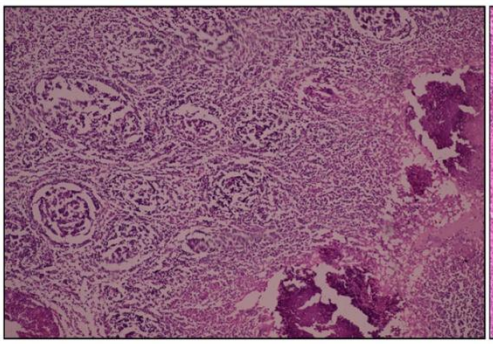


Figure 49

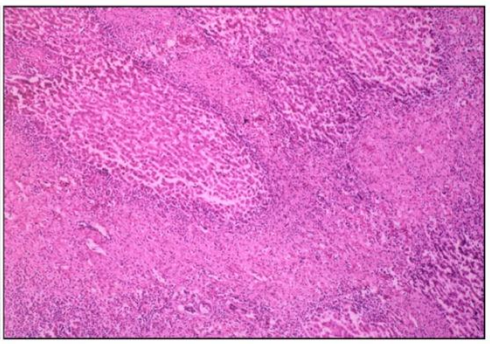


Figure 50

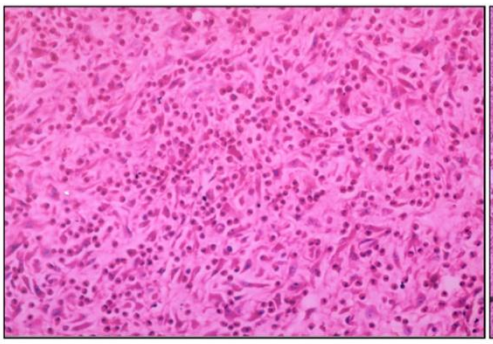


Figure 51

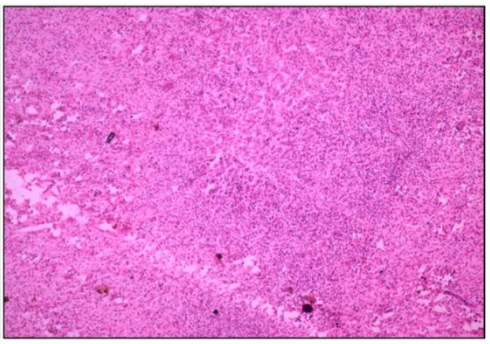


Figure 52

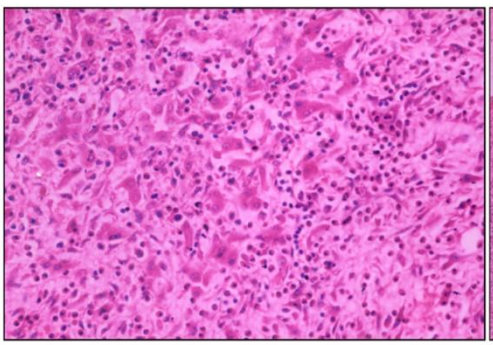


Figure 53

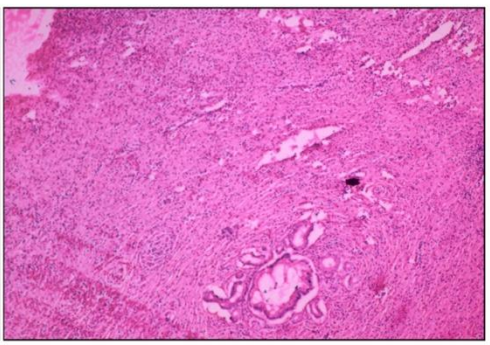


Figure 54

Figure 55 Ascariosis: Liver - Section showing larval remnants within the lymphoid follicles H & E x 100.

Figure 56 Ascariosis: Note the presence of adult worm in the intestine.

Figure 57 Ascariosis: Intestine - Section showing desquamation of epithelium and infiltration H & E x 100.

Figure 58 Ascariosis: Intestine - Section showing desquamation of epithelium and infiltration H & E x 400.

Figure 59 Ascariosis: Intestine - Showing infiltration H & E x 400.

Figure 60 Stephanurosis: Note enlarged liver with multiple whitish nodules.

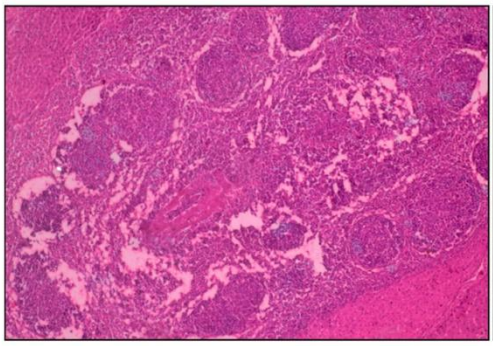
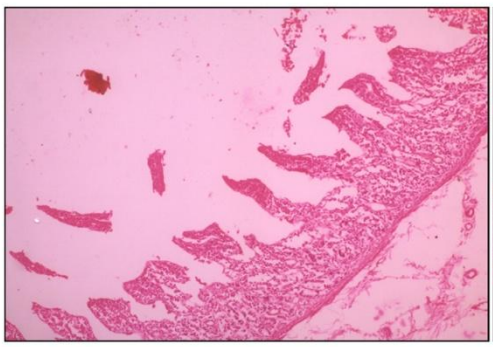


Figure 55

Figure 56



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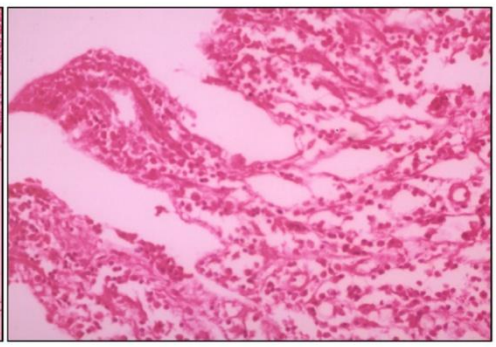

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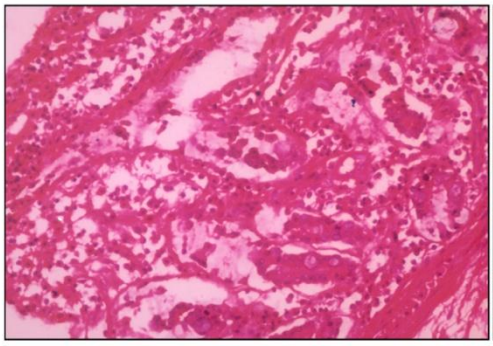


Figure 59

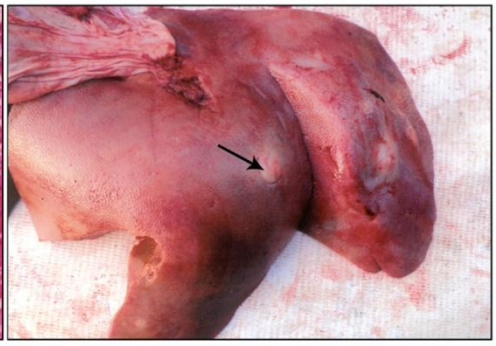


Figure 60

Grossly, the liver was enlarged and had multiple whitish nodules with a diameter of 1.5-2.0 cm (Fig. 60, 61 and 62). In few livers, presence of immature parasites was noticed on cutting and they were identified morphologically as *S. dentatus*. In some animals, the worms were also found in the perirenal fat in the free state (Fig. 63 and 64).

Microscopically, there was extensive damage in the liver due to migration of larvae. The sinusoids were congested and dilated and areas of haemorrhages into the hepatic lobule were noticed (Fig. 65 and 66). The cut sections of immature worms were surrounded by extensive eosinophilic infiltration forming granulomas (Fig. 67, 68, 69 and 70). A few giant cells were also present at the periphery of the granuloma. In addition, the hepatic cells revealed necrotic changes and the interlobular septa were thickened due to infiltration by eosinophils, mononuclear cells and fibrous tissue (Fig. 71). These changes were so extensive leading to atrophy of the hepatic cells and reduction in the size of hepatic lobule. There was also hyperplasia of bileducts and thickening of blood vessels and some vessels showed thrombus formation and endoangitis (Fig. 72, 73, 74 and 75). A few macrophages present in the interlobular septum also contained hemosiderin pigment (Fig. 76). Cut sections of worms were noticed in the perirenal fat along with infiltration of eosinophils (Fig. 77, 78 and 79). In the kidney, degenerative changes, necrosis of tubular epithelium, cystic dilatation of tubules, atrophy of few

Figure 61 Stephanurosis: Note enlarged liver with multiple whitish nodules.

Figure 62 Stephanurosis: Note enlarged liver with multiple whitish nodules.

Figure 63 Stephanurosis: Presence of worms in the perirenal fat.

Figure 64 Stephanurosis: Presence of worms in the perirenal fat.

Figure 65 Stephanurosis: Liver - Showing intralobular haemorrhages
H & E x 100.

Figure 66 Stephanurosis: Liver- Showing intralobular haemorrhages
H & E x 400.

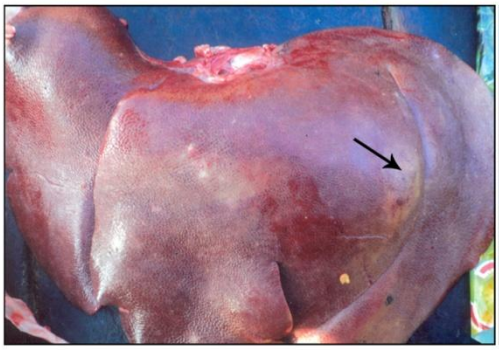
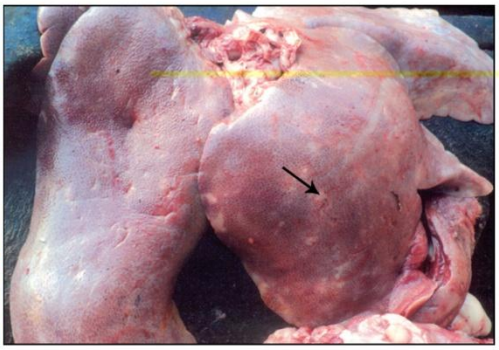


Figure 61



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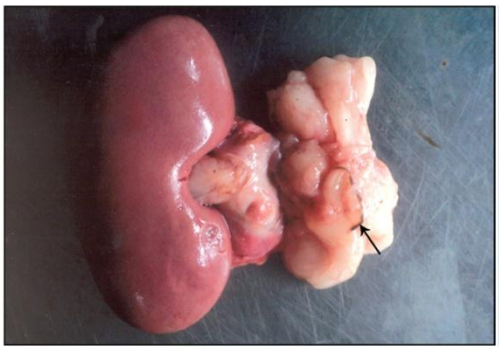
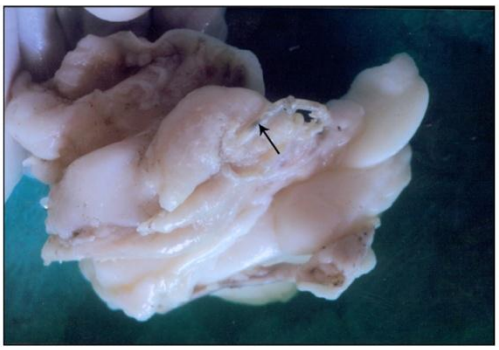


Figure 63



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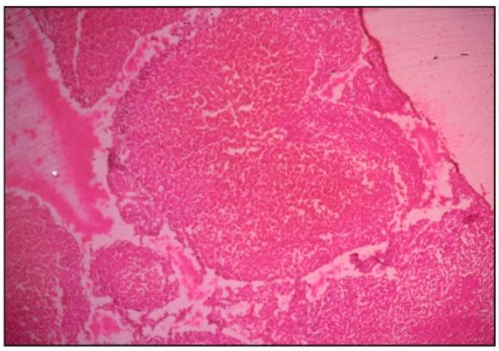


Figure 65

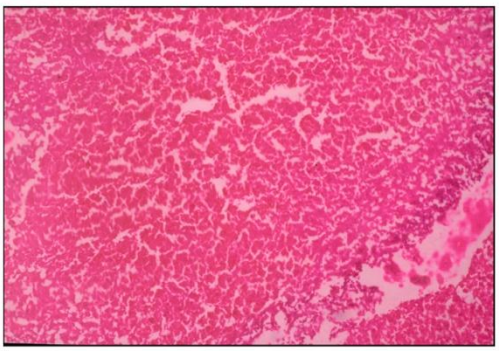


Figure 66

Figure 67 Stephanurosis: Liver - Note cut section of immature worms surrounded by extensive eosinophilic infiltration H & E x 40.

Figure 68 Stephanurosis: Liver - Note cut section of immature worms surrounded by extensive eosinophilicinfiltration H& E x 100.

Figure 69 Stephanurosis: Liver - Note cut section of immature worms surrounded by extensive eosinophilic infiltration H & E x 100.

Figure 70 Stephanurosis: Liver - Note extensive eosinophilic infiltration H & E x 100.

Figure 71 Stephanurosis: Liver - Section showing extensive fibrosis and infiltration H & E x 100.

Figure 72 Stephanurosis: Liver - Note hyperplasia of bile ducts and thickening of interlobular septum H & E x 100.

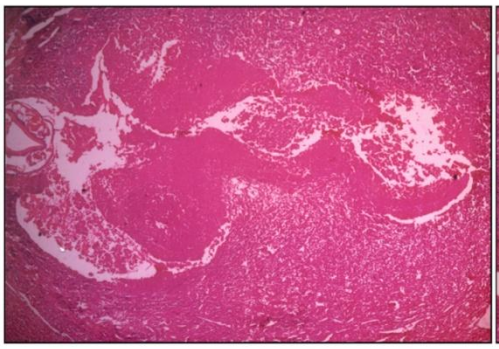
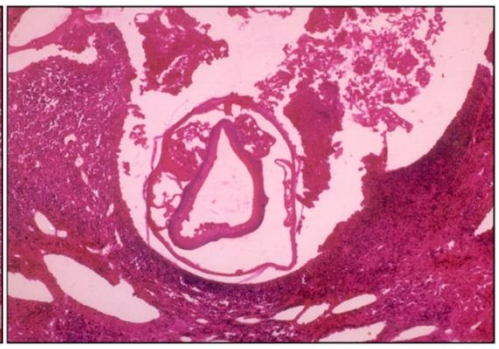


Figure 67



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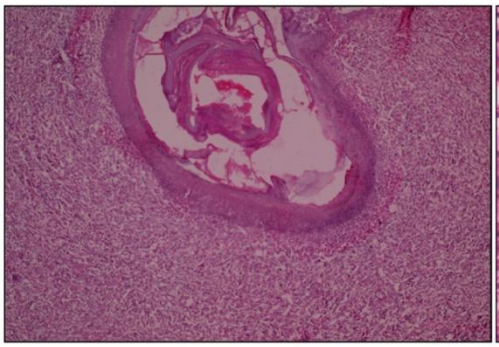

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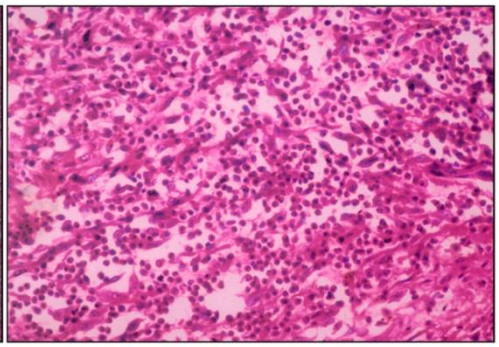

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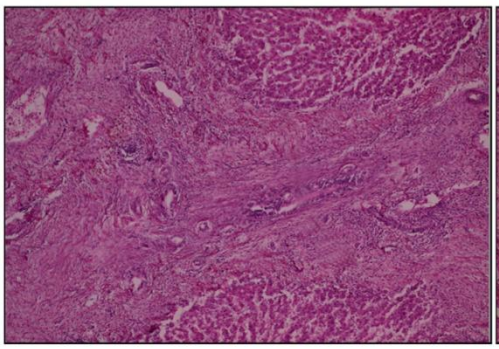

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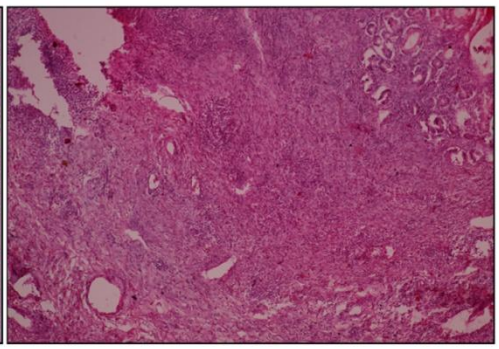


Figure 72

Figure 73 Stephanurosis: Liver - Section showing thickened blood vessels, fibrosis and thrombus formation H & E x 100.

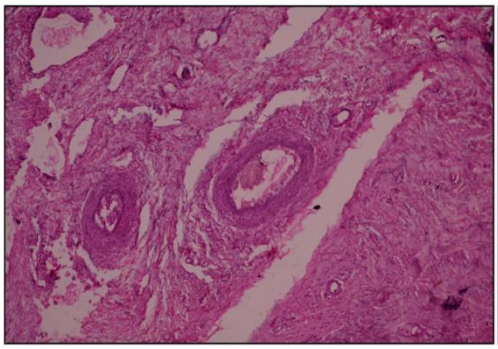
Figure 74 Stephanurosis:Liver -Showing infiltration,bile duct proliferation, hemosiderin deposition and endoangitis H & E x 400.

Figure 75 Stephanurosis: Liver-Note the blood vessel showing endoangitis H & E x 400.

Figure 76 Stephanurosis:Liver-Note macrophages containing hemosiderin pigment in the interlobular septum H & E x 400.

Figure 77 Stephanurosis: Perirenal fat- Note presence of cut sections of worms along with infiltration H & E x 100.

Figure 78 Stephanurosis: Perirenal fat- Note infiltration of eosinophils H & E x 100.



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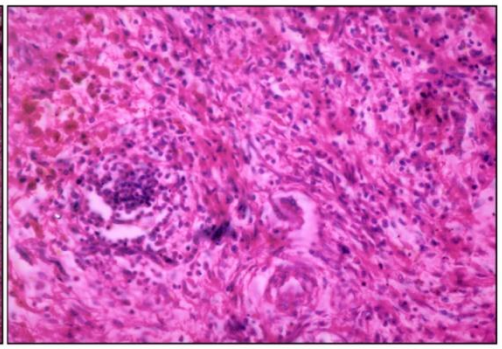

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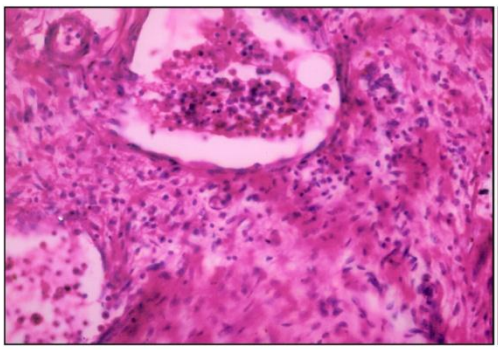

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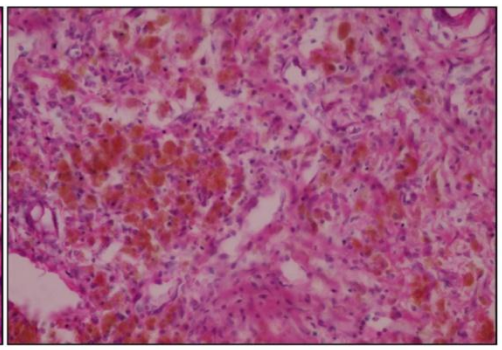

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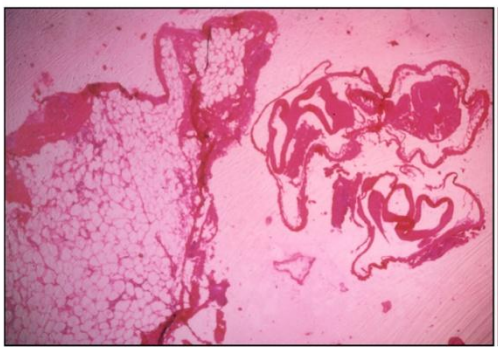

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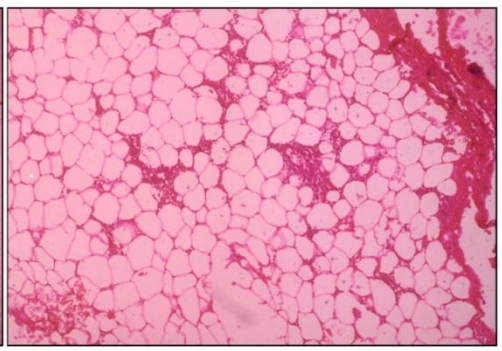


Figure 78

glomeruli and infiltration of mononuclear cells in the interstitium were noticed (Fig. 80, 81 and 82).

4.2.3.3 Metastrongylosis

Metastrongylosis was noticed in 11.88% of animals out of 320 animals examined.

Grossly, small gray nodules were noticed on the caudoventral borders of diaphragmatic lobes (Fig. 83 and 84). Emphysematous bullae and dark red depressed areas of collapse were also noticed. Large numbers of male and female lung worms of *Metastrongylus apri and M. pudendotectus* were noticed in the bronchi and bronchioles in the ventral borders of the diaphragmatic lobes (Fig. 85 and 86). The adult worms were white, thread like and surrounded by mucous exudates. The bronchial lymphnodes were slightly enlarged.

Microscopically, several sections of the parasites in different stages of development were present in the lumen of the bronchioles, surrounded by mucus and inflammatory cells (Fig. 87 and 88).

The bronchial epithelium was desquamated and eosinophils, lymphocytes and neutrophils infiltrated the epithelium and lamina propria (Fig. 89 and 90). Hyperplasia of bronchial, bronchiolar and alveolar epithelium and bronchial associated lymphoid tissue was also noticed. The lining epithelium of some of the

Figure 79 Stephanurosis: Perirenal fat- Note infiltration of eosinophils H & E x 400.

Figure 80 Stephanurosis: Kidney-Section showing degenerative changes, cystic tubules and glomerular atrophy H& E x 100.

Figure 81 Stephanurosis: Kidney- Showing mononuclear cell infiltration in theinterstitiumH& E x 100.

Figure 82 Stephanurosis: Kidney- Showing mononuclear cell infiltration in the interstitium H & E x 400.

Figure 83 Metastrongylosis: Lung showing gray nodules on the caudoventral borders of diaphragmatic lobes.

Figure84 Metastrongylosis: Lung showing gray nodules and emphysematous bullae and dark red depressed areas of collapse.

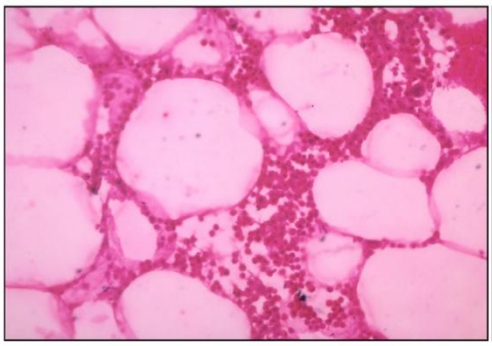
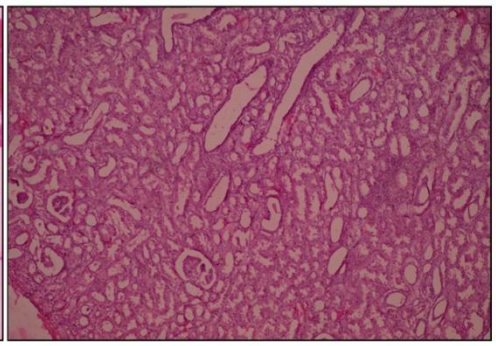


Figure 79



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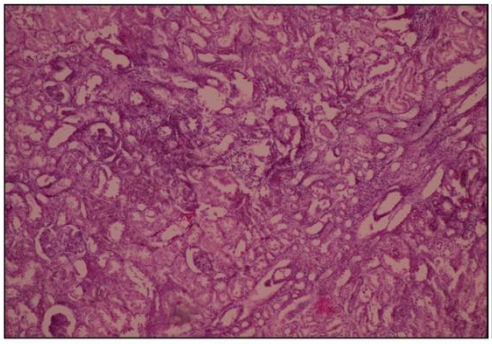

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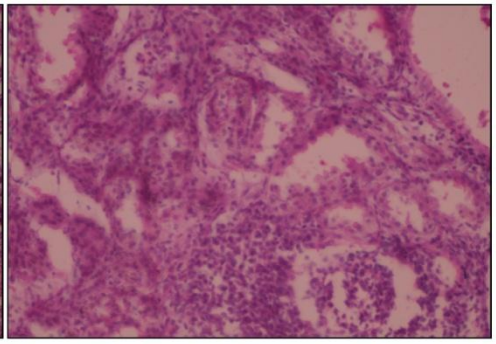

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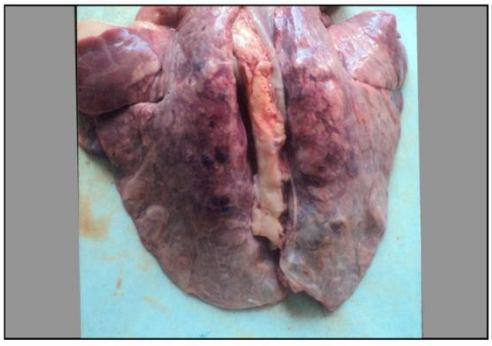


Figure 83

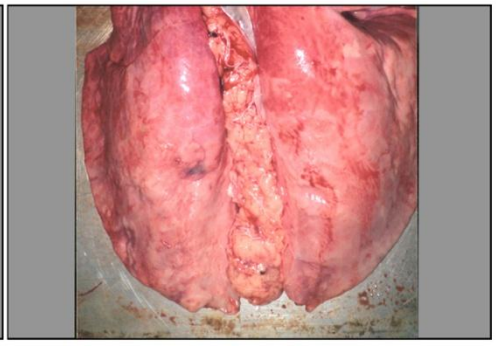


Figure 84

Figure 85 Metastrongylosis: Lung- Presence of lung worms in the bronchus.

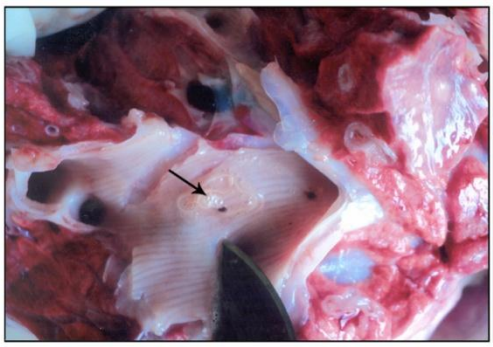
Figure 86 Metastrongylosis: Lung-Showing presence of lung worms in the bronchi.

Figure 87 Metastrongylosis: Lung-Sections of parasites in the bronchus, surrounded by mucous and inflammatory cells H & E x 40.

Figure 88 Metastrongylosis: Lung-Sections of parasites in the bronchus, surrounded by mucous and inflammatory cells H & E x 100.

Figure 89 Metastrongylosis: Lung-Sections of parasites in the bronchus, surrounded by mucous and inflammatory cells H & E x 400.

Figure 90 Metastrongylosis: Lung-Presence of inflammatory cells in the wall of the bronchus H & E x 400.



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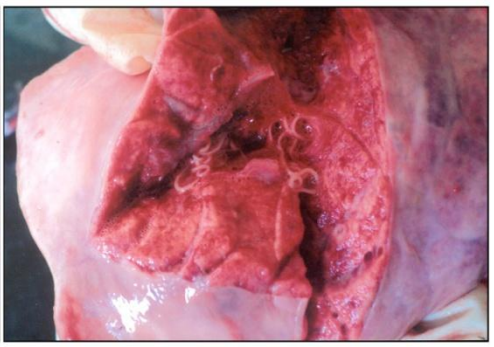

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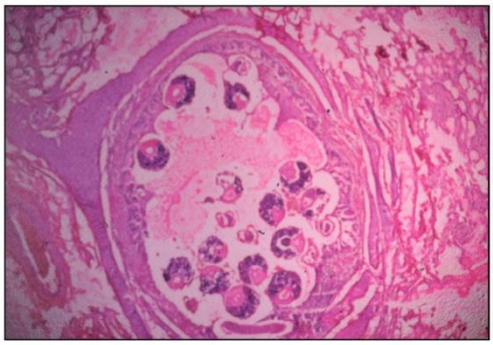

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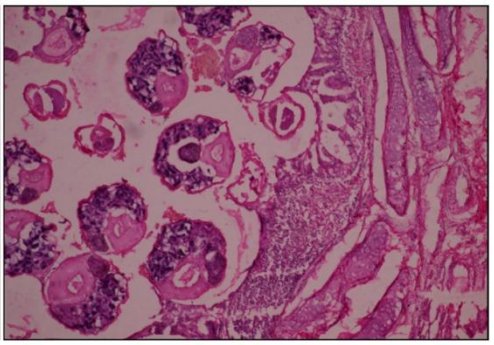

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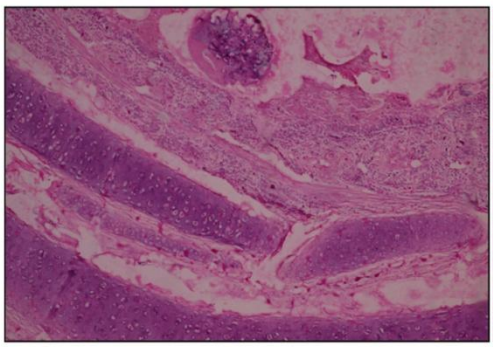

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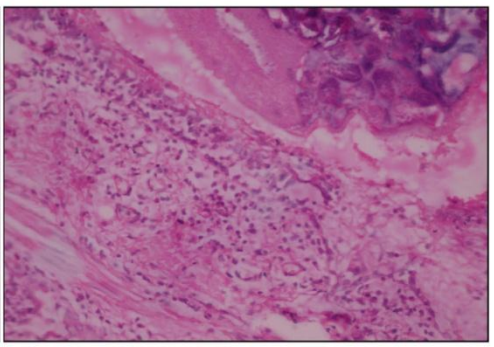


Figure 90

Figure 91 Parasitic gastritis : Hyperemia and haemorrhages on the mucosa of stomach.

Figure 92 Parasitic gastritis : Areas of hyperemia and formation of ulcers in the mucosa of stomach.

Figure 93 Parasitic gastritis : Stomach-Fundic region - Showing areas of necrosis, desquamation of epithelium and infiltration H & E x 100.

Figure 94 Parasitic gastritis : Stomach-Fundic region - Showing areas of necrosis,desquamation of epithelium and infiltration H & E x 400.

Figure 95 Parasitic gastritis : Stomach-Cardiac region- Note atrophy or cystic dilatation of glands H & E x 100.

Figure 96 Parasitic gastritis : Stomach-Cardiac region-Note degenerative changesin the glandular epithelium and infiltration H & E x 400.

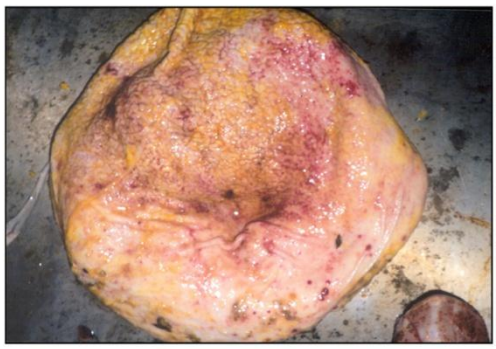
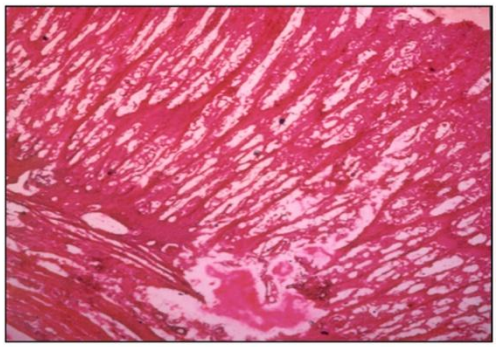


Figure 91

Figure 92



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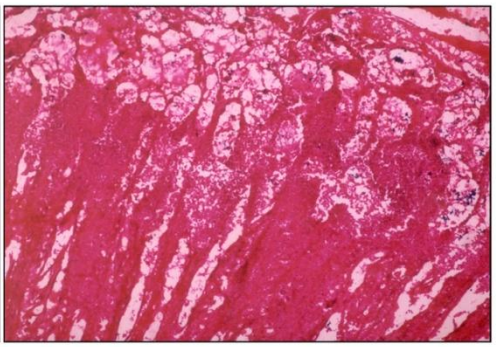

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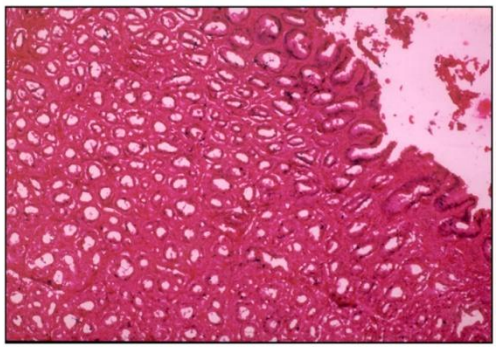

Figure 95

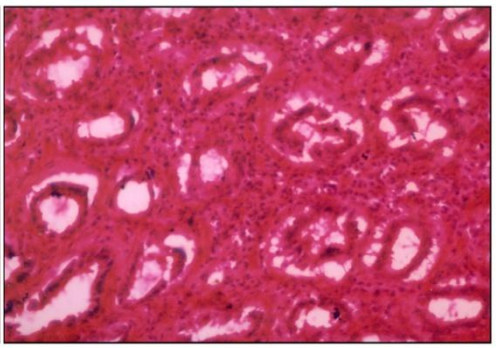

Figure 96

bronchioles was vacuolated. Capillaries were engorged and the alveoli were atelectatic in the surrounding parenchyma. The bronchial lymph nodes showed mild lymphoid hyperplasia.

4.2.3.4 Parasitic gastritis (Stomach worms)

Stomach worms (*Physocephalus sexalatus*) were noticed in 0.94% of animals out of 320 animals examined.

Grossly, small red worms were present in the stomach. They appeared as small red colour streaks on the mucosa of the stomach. Areas of slight hyperemia and petechial haemorrhages in the mucosa (Fig. 91) or formation of eroded areas or ulcers were evident on the mucosa of stomach (Fig. 92).

Microscopically, the mucosa of stomach revealed changes of chronic catarrhal gastritis. In the fundic region of stomach, necrosis and desquamation of the epithelium and infiltration of mononuclear cells, a few eosinophils and macrophages were noticed (Fig. 93 and 94). There was atrophy of glandular epithelium or cystic dilatation of glands and infiltration by mononuclear cells and a few eosinophils in the cardiac region (Fig. 95 and 96). In the superficial mucosa, mild congestion and few haemorrhages in two cases and ulceration in one case were also evident.

CHAPTER V
DISCUSSION

Parasitic diseases caused by different helminths in pigs are one of the major causes of economic losses in pig farming. They are the main causes for the condemnation of organs and even entire carcasses leading to severe economic losses. Only a few reports are available on the incidence of helminthic diseases and the associated gross and microscopical lesions in the infested organs in the slaughtered pigs (Dev Sarma and Gogoi, 1986 and Gaurat and Gatne, 2009). In view of scanty literature, the present investigation was carried out to study the incidence and to describe the gross and histopathological lesions of some common helminth parasitic diseases in the slaughtered pigs.

In the present investigation, a total of 320 pigs of either sex and of different age groups from various slaughter houses, field mortalities, private piggery farms located in and around Gannavaram, Vishakapatnam and Tirupati towns and from postmortem examinations conducted at the NTR College of Veterinary Science, Gannavaram, were subjected to detailed postmortem examination and different organs showing lesions suggestive of parasitic diseases or organs having parasitic infestation were collected. All the parasites collected during the study were identified on the basis of their morphological characters.

Out of 320 pigs examined, 216 (67.5%) animals revealed definite lesions of helminth parasitic diseases on gross and microscopical examination of tissues. Various types of helminth parasitic diseases noticed in 216 pigs, out of total 320 pigs examined accounted for an overall incidence of 67.5%. This high incidence indicated that a significant number of animals were affected by parasitic diseases that lead to economic losses due to condemnations .

Out of 142 local pigs examined by Dev Sarma and Gogoi (1986) at autopsy, 80 animals revealed the presence of different types of helminths and lesions having an overall incidence of 56.34%. Varma (1993) examined 352 pigs for helminth parasites at slaughter and found 86.64% of infected animals whereas, 41(19.52%) pig carcasses showed the presence of helminth parasites in a study conducted in 210 pigs by Borthakur e*t al*. (2007). The incidence of helminth parasitic diseases in the present study was slightly higher than that reported by Dev Sarma and Gogoi (1986) and Borthakur e*t al*. (2007) and lower than that reported by Varma (1993). The difference might be due to variations in location, climatic conditions, availability of the intermediate host, managemental practices, local conditions etc. Moreover, most of the available reports on the incidence or prevalence of parasitic infestations were based on the faecal examination (Roepstorff *et al*., 1989; Sanjeev Kumar *et al*., 2002 and Katoch *et al*., 2008). Though a few reports are available based on carcass examination during slaughter, they did not describe the associated lesions in the infested organs and only the species of helminths were identified and their incidences were presented (Dev Sarma and Gogoi, 1986, Solaymani-Mohammadi *et al.,* 2003 and Islam *et al*., 2008). In some studies, the incidence and lesions described were limited to one parasitic disease (Kim and Lim, 1970; Das, 1997 and Avapal *et al*., 2003).

In the present study, a total of 320 pigs were examined and the various helminth parasitic diseases observed were grouped as diseases caused by trematodes, that accounted for 3.13%, diseases caused by larvae of cestodes that accounted for 8.75% and diseases caused by nematodes that accounted for

55.63%. Diseases caused by various nematode parasites amounted to the highest percentage (55.63%) when compared to other parasitic diseases. Similarly, out of 352 pigs examined for helminth parasites at slaughter by Varma (1993) the number of animals infected by nematodes (58.49%) were more than trematodes (13.34%) and cestodes (14.75%). This finding indicated that the nematode parasites are the major pathogens causing losses in pig farming.

In the present investigation, schistosomosis and opisthorchiosis were the trematodal diseases noticed. The incidence of schistosomosis in the liver was 2.81% of the total animals examined. Rao and Paliwal (1996) reported the incidence of schistosomosis as 20.09% in swine livers, which was higher than that observed in the present study. In a survey carried out in domestic animals by Agarwal and Sahasrabudhe (1982), 16 (80%) were positive for *S. incognitum* infection out of 20 pigs slaughtered.

Grossly, the affected livers in schistosomosis were slightly enlarged and had a granular surface. Microscopically, the sections of the liver revealed perioval granulomas with extensive fibrocellular reaction in the interlobular septa and in the portal triads. The granuloma contained ova of the *schistosoma* surrounded by eosinophils, macrophages, epithelioid cells, giant cells and lymphocytes. In addition, congestion, degenerative changes, bile duct hyperplasia, thickening and destruction of blood vessels, phlebitis, individualization of hepatic lobules and extensive portal and septal fibrosis were also evident. At few places, lymphoid nodule formation was also seen. The gross and microscopic lesions in schistosomosis were similar to that reported by Rao and Paliwal (1996). They also

noticed multiple focal pseudotuberculous lesions characterized by the presence of schistosome egg and infiltration of epithelioid cells, a few histiocytes, numerous lymphocytes and foreign body giant cells with fibrocellular reaction. The distribution of lesions from stomach to intestine in experimental *S. incognitum* infection in pigs was discussed by Ahluwalia (1972). Agarwal *et al*. (2001) noticed sudden death of piglets when they were experimentally infected with cercaria of *S. incognitum*. Histopathologically, the intestines were normal and livers showed cellular swelling and mild fatty change.

The literature on schistosomosis revealed various reports of *S. japonicum* infection in pigs (Baddamwar *et al.*, 2004 and Ngwa, 2006). Pig is a natural host for *S. japonicum* and it is associated with clinical disease, marked pathological lesions, including fibrosis of liver and reduced growth. Among the schistosomes pathogenic to man *S. japonicum* is the only one of zoonotic importance.

In the present study, opisthorchiosis was observed in one case (0.32%) of the total animals examined. Rao and Paliwal (1996) recorded one case of opisthorchiosis out of 242 livers examined in slaughtered pigs. *O. tenuicollis* was noticed in 3 (2.11%) pigs out of 142 examined by Dev Sarma and Gogoi (1986) and Gatne *et al*. (2008) noticed a massive infestation of *O. tenuicollis* in a pig slaughtered at Mumbai abattoir. Grossly, the liver was slightly enlarged with a raised structure on the surface and on opening, thickened cystic bile duct was noticed containing small flukes that were identified as *Opisthorchis tenuicollis.* Microscopically, the affected part of liver revealed cross section of the fluke in the bile duct that was thickened, dilated and fibrosed with hyperplasia of bile duct

epithelium. In addition, there was desquamation and necrosis of epithelial lining of the bile duct, proliferation of goblet cells and glands in the sub mucosa and infiltration of lymphocytes, plasma cells and macrophages and small lymphoid nodules were also noticed around the bile ducts. The gross and microscopic lesions were akin to the findings of Ohshima *et al*. (1971), Rao and Paliwal (1996) and Gatne *et al.* (2008). Bile duct showed extensive hyperplasia, cross section of *Opisthorchis sp*. and infiltration of eosinophils, polymorphs, lymphocytes and macrophages with fibrocellular reaction in a case of opisthorchiosis (Rao and Paliwal, 1996). Grossly, a balloon like structure which was found to be dilatation of the bile duct containing small flukes and biliary hyperplasia with cirrhotic changes and thickened bile duct microscopically were noticed by Gatne *et al*. (2008) in a massive infestation of *O. tenuicollis* in a pig.

In the present study, 28 (8.75%) slaughtered pigs were infected with larvae of cestodes. Of these, hydatid cysts were found in 4 (1.25%) animals and cysticercosis was noticed in 24 (7.5%) animals.

Out of 320 pigs examined in the present study, 1.25% of animals showed hydatid cysts. The incidence of hydatid cysts in pigs was found as 0.73%, 1.79% and 7.68% by Deka and Gaur (1998), Dev Sarma *et al. (*2000) and Borua *et al*. (2010) respectively. Rao and Paliwal (1996) noticed 4.81% of hydatid affected swine livers in a total of 242 animals. Grossly, a single cyst measuring 2.0-6.0 cm in diameter was noticed on the ventral surface of the liver and on the dorsum of lung and spleen. The cyst contained clear watery fluid with whitish thick cyst wall consisting of laminated layer of variable thickness. Microscopically, the cyst

consisted of hyaline wall surrounded by fibrous connective tissue capsule infiltrated by large number of eosinophils, mononuclear cells and plasma cells. The protoscolices were also noticed attached to the cyst wall. In addition, the surrounding hepatic parenchyma revealed vacuolar degeneration, mild bile duct hyperplasia, infiltration of few mononuclear cells and eosinophils and mild proliferation of fibrous tissue. These findings were in conformity with the previous reports of Slais and Vanek (1980), Slais (1981), Sahu *et al*. (1993), Rao and Paliwal (1996) and Singh (2000). Slais (1981) described the capsule formation and shape of young hydatids in liver in a spontaneous infection of pigs. In a case of hydatidosis of pig, Rao and Paliwal (1996) observed that the liver had 1-4 cysts ranging from 1.0-6.0 cm in diameter lodged on the surface and deep into the parenchyma and the cysts were sterile. Sahu *et al.* (1993) reported 0.99% of pulmonary hydatidosis in pigs. Histopathologically, the cyst consisted of a dense laminated eosinophilic cyst wall and was encircled by a thick connective tissue capsule that was infiltrated by a large number of eosinophils, mononuclear cells and plasma cells. Singh (2000) observed hydatidosis in spleen in 7 cases during a study conducted on the pathological alterations in spleens of pigs.

The incidence of cysticercosis (7.5%) noticed in the present study was slightly higher than the previous reports (Varma and Ahluwalia, 1989; Shinde, 1991; D'souza and Hafeez, 1998 and Hafeez *et al.,* 2004). Varma and Ahluwalia (1989) reported that out of 757 pig carcasses examined, 41 were found infected with *C. cellulosae* with an overall incidence of 5.35% while Shinde (1991) observed 6.02% incidence of *C. cellulosae* infection in a survey of 400 pigs in

Maharastra. During the period of two years, D'Souza and Hafeez (1998) found cysticercosis in 263 (4.5%) pigs, out of 5,828 pigs examined in an organized abattoir in Andhra Pradesh. Hafeez *et al*. (2004) investigated the prevalence of porcine cysticercosis during meat inspection in South India from July 2000 to August 2003. Out of 1,813 pigs examined in four states of South India, *C. cellulosae* was observed in 82 pigs and the overall percentage of infection was 4.53%, with highest incidence in Karnataka (5.73%), followed by Tamilnadu (5.50%), Kerala (5.38%) and Andhra Pradesh (3.52%). Out of 229 pigs examined by Avapal *et al*. (2003) in and around private abattoirs in Ludhiana, 4 (0.0175%) were found infected with *C. cellulosae*. The cysticerci were found in the organs like tongue, heart, liver, skeletal muscles and brain in the present study. D'Souza and Hafeez (1998) observed cysticerci in the skeletal muscles of shoulder, thigh, undercut, tongue, heart, liver, kidney and brain. Cysticerci were noticed in the skeletal muscles of thigh, tongue, liver and brian by Avapal *et al*. (2003) in pigs. Cysticercosis in liver was described by Rao and Paliwal (1996). The pathomorphological lesions of cysticerci in the pig brains were studied by Singh and Parihar (1999) and Prakash *et al*. (2007). Grossly, the cysticerci were typically spherical or oval, white or yellow vesicle with a translucent bladder wall, through which the scolex was seen like a small solid, eccentric granule. In some cases, degenerated cysts were also seen. Microscopically, the cross sections of cysticerci were evident with or without invaginated scolex in different organs, surrounded by fibrocellular reaction consisting of lymphocytes, plasma cells and eosinophils. These findings were similar to the observations of Rao and Paliwal

(1996), de Aluja *et al*. (1998), Singh and Parihar (1999), Rajnish Sharma (2004) and Prakash *et al*. (2007) .

Cysticercosis, a zoonotic disease caused by metacestode of pork tape worm (*T. solium*) is an infection of pigs and human that is associated with poor sanitation and hygiene. In human and pigs apart from other sites, the cysticerci are also located in the central nervous system, causing neurocysticercosis and are the main cause of late-onset epilepsy in tropical countries in human (Klotz *et al.*, 2006). In the present study, the cysticerci were also noticed in the pig brains.

The diseases caused by different nematodes were noticed in 55.63% of the total pigs examined. Of these, 34.06% was affected by ascariosis, 8.75% by stephanurosis, 11.88% by metastrongylosis and 0.94% by stomach worms.

The incidence of ascariosis (34.06%) observed in the present study amounted to highest percentage of all the parasitic diseases. Yap *et al.* (1983) reported that the majority (71.4%) of livers of swine were condemned due to milk spots. Tahir and Sheikh-Omar (1985) noticed milk spots as the most common lesions with an incidence of 36.0% in condemned pig livers. A study on 386 pigs from Prince Edward Island by Bernardo and Dohoo (1988) showed that 34.6% pigs had intestinal ascarids, 82.4% had milk spots on the liver and in 51.3% of the pigs, milk spots occurred in the absence of intestinal ascarids. Rao and Paliwal (1996) noticed ascariosis in 24 cases (28.19%) out of 242 pig livers examined. Schuh *et al.* (2000) observed milk spots in the liver in 47.5% of slaughtered pigs. The wide variation in the incidence of ascariosis in pigs in the previous reports

might be due to variation in breed, location and managemental practices. Barbara *et al*. (2006) opined that the prevalence of parasitic infections varies considerably between units.

Grossly, the milk spots were star-like in shape and grayish white in colour or of pearl like nodules scattered throughout the liver. They were of different sizes and were found simultaneously in the same liver. The diameter of these spots ranged from 0.5-2.5 cm. Some spots had haemorrhagic centres. The gross lesions were in accordance with the reports of Roneus (1966), Yoshihara *et al.* (1983) and Tahir and Sheikh-Omar (1985). Morphologically, Roneus (1966) described the white spots in pig livers as white spots of the granulation tissue type and white spots of the lymphonodular type. Eriksen *et al.* (1981) opined that the presence of white spots on the liver at slaughter was an indication of recent massive *A. suum* egg infection. At autopsy, mesh worked white spots, some compact and some lymphonodular lesions were observed on the surface of the liver by Yoshihara *et al.* (1983). Nakagawa *et al*. (1985) identified four types of lesions macroscopically consisting of minute, mesh-work, lymphonodular and compact types in ascariosis of adult breeding pigs. Grossly, the milk spots were small, stellate white spots and 0.1 to 1.5 cm in diameter and the bigger spots tended to have a nodular cystic centre of 0.1 cm diameter in few livers (Tahir and Sheikh-Omar, 1985). Sanford and Josephson (1991) observed the lesions that grossly consisted of large numbers of milk spots up to 2.0 cm in diameter scattered through out the liver. In ascariosis, the liver showed diffuse fibrotic lesions with numerous spherical white spots ranging from 0.2 to 0.5 cm in diameter (Rao and

Paliwal, 1996). Gaurat and Gatne (2009) described large numbers of opaque spots measuring 2.0 to 7.0 mm in diameter on the surface of pig liver in *A. suum* infection.

Microscopically, the liver sections revealed areas of extensive haemorrhage into the lobule, distortion of hepatic architecture, eosinophilic granulomas with areas of necrosis and calcification and presence of ascarid larvae, thickening of interlobular septa with infiltration of eosinophils and few mononuclear cells, proliferation of fibrous tissue, atrophy of the hepatic lobules, proliferation of bile ducts and thickening of walls of blood vessels. Those livers that showed pearl like nodules grossly revealed lymphoid follicle formation microscopically. Larval remnants were found in the center in some of the follicles. The microscopic appearance of white spots / milk spots in the livers of pigs was akin to the previous reports of ascariosis (Roneus, 1966; Nakagawa *et al*., 1985; Sanford and Josephson, 1991; Rao and Paliwal, 1996 and Gaurat and Gatne, 2009).

The view most commonly held to day is that white spots are caused by *A. suum* (Roneus, 1966). It is generally agreed in the literature that the lesions in nature of white spots or hepatitis interstitial parasiteria multiplex are frequently seen in the liver of slaughtered pigs (Roneus, 1966). Batte *et al*. (1975) also stated that the cause of white spots or milk spots which are the basis for condemnation is generally thought to be migrating larvae of *A. suum*.

The first detailed and fundamental description of the morphological changes in this liver lesion in pigs was given by Joest and Felber (1908). They named the

lesions hepatitis interstitialis chronica multiplex and later, the description was changed into hepatitis interstitialis parasitaria multiplex (eosinophilica). Jelinek (1956) described a special type of nodular lesion in pig livers that was termed as lymphadenopathia nodularis mulptiplex hepatis suum and was characterized by lymphoid follicular structures.

Roneus (1966) stated that the histological studies of different white spots revealed a central tissue injury and a surrounding distant reaction. The central tissue injury was situated intra lobularly, as a direct consequence of ascarid larval migration. The distant reaction consisted of proliferative and infiltrative lesions localized to the interlobular connective tissue septa of the affected lobules. Similar findings were noticed in the present study.

Nakagawa *et al*. (1985) identified three types of lesions histologically viz. eosinophilic interstitial hepatitis, interstitial connective tissue proliferation and lymphonodular hyperplasia in ascariosis of adult breeding pigs. Hoshino *et al.* (1995) noticed lymphonodular white spots lesions in the swine lung with pulmonary petechiae and multiple milk spots in the liver. These nodules mainly consisted of follicular hyperplasia of lymphoid cells, including a few sections of third stage larvae of *A. suum* at the center of the foci in some cases. Histologically, the white spots revealed massive focal lymphofollicular aggregates encircled by connective tissue replacing liver parenchyma and multiple necrotic granuloma having caseonecrotic mass in the centre and infiltration of histiocytes, epithelioid cells, eosinophils and lymphocytes with fibrous tissue encapsulation and with cross section of *ascaris* nematode larvae (Rao and Paliwal, 1996). As

observed in the present study, Gaurat and Gatne (2009) noticed areas of focal necrosis and calcification surrounded by macrophages, mononuclear cells and fibroblasts in the pig liver in *A. suum* infection.

Adult worms were also noticed in the intestines of some animals apart from milk spots on the liver. The intestinal mucosa revealed desquamation of epithelium and infiltration by lymphocytes, plasma cells and macrophages. Islam *et al*. (2008) reported that in *A. suum* infection, the intestinal wall was infiltrated with plasma cells, lymphocytes and eosinophils. Gaurat and Gatne (2009) noticed adults of *A. suum* in the intestines and on microscopic examination the intestines revealed denudation of epithelial cells, leucocytic infiltration in lamina propria and villous atrophy at few places.

The incidence of stephanurosis was 8.75% of the total animals examined in the present study. Sapong (1972) reported the incidence of stephanurosis in Ghanian dwarf pigs as 33.0% and Rao and Paliwal (1996) noticed stephanurosis in 40.96% of livers out of 242 pigs examined. Both these incidences are higher than that observed in the present study. Das (1997) studied the prevalence of *S. dentatus* in indigenous pigs in and around Pant Nagar and reported that out of 282 slaughtered pigs examined, 9.57% were found to be infected.

Grossly, the liver was enlarged and had multiple whitish nodules with a diameter of 1.5-2.0 cm. In few livers, the presence of immature parasites was noticed on cutting and they were identified morphologically as *S. dentatus*. In some animals, the worms were found in the perirenal fat in the free state. Rao and

Paliwal (1996) reported that the liver was enlarged, hard and nodular in appearance. On cutting, the nodules revealed pulpy parenchyma with presence of parasite. Das (1997) noticed the parasites in the cortex, medulla and peritoneal fat either in free state in haemorrhagic zone or in the cyst of 2-4 cm diameter filled with serosanguinous fluid in the renal calyces and pelvis. The infected kidneys were swollen with petechial haemorrhage in the cortex and white spots were also seen on the surface.

Microscopically, congestion of sinusoids, haemorrhages into the lobule, cut sections of immature worms surrounded by extensive eosinophilic infiltration, thickened interlobular septa, atrophy of the hepatic cells and hepatic lobules, hyperplasia of bile ducts, thickening of blood vessels, thrombus formation and endoangiitis were noticed. There was extensive damage in the liver due to migration of larvae. The sinusoids were congested and dilated and areas of haemorrhages into the hepatic lobule were noticed. The cut sections of immature worms were surrounded by extensive eosinophilic infiltration forming granulomas. A few giant cells were also present at the periphery of the granuloma. In addition, the hepatic cells revealed necrotic changes and the interlobular septa were thickened due to infiltration by eosinophils, mononuclear cells and fibrous tissue. These changes were so extensive leading to atrophy of the hepatic cells and reduction in the size of hepatic lobule. Cut sections of worms were also noticed in the perirenal fat along with infiltration of eosinophils. In the kidney, degeneration, necrosis, cystic dilatation, glomerular atrophy and infiltration by mononuclear cells were noticed. The gross and microscopic lesions were in accordance with the

findings of Dykova (1977); Smith and Hawkes (1978); Ashizawa *et al*. (1988); Ashizawa and Moritomo (1990 a and b); Rao and Paliwal (1996); Ashok Kumar *et al*. (2000) and Gaurat and Gatne (2009). Atrophy of hepatic lobules observed in the present study was also described by Batte *et al*. (1975) in advanced cases of stephanurosis. Extensive damage in the liver from migration of the larvae of the kidney worm was evidenced by extremely wide spread fibroblastic reaction (Batte *et al*., 1975). Ashizawa *et al*. (1988) classified the hepatic lesions in pigs in stephanurosis into two groups viz., lesions in the blood vessel system and in the hepatic tissue including parenchyma and interstitial tissue. In the present study, similar type of hepatic lesions were noticed. Barbara *et al.* (2006) stated that the extensive and massive involvement of the liver makes kidney worm infection easily differentiated from that caused by ascarid migration. In kidney worm infestation in pigs, Gaurat and Gatne (2009) noticed extensive fibrosis and moderate infiltration of mononuclear cells in the cortical portion near hylus and the tubules and glomeruli in the adjacent tissue showed pressure atrophy with degenerative changes.

In the present study, the incidence of metastrongylosis was 11.88% out of 320 animals examined. Chauhan and Rao (1972) reported 28.57% of verminous pneumonia cases caused by *M. apri* of all pulmonary affections of the pigs. Baruah *et al.* (1985) recorded the lung lesions associated with pulmonary metastrongylosis. Out of 190 pigs examined, 15 lungs (7.9%) showed changes associated with *M. apri* infection. Morita *et al*. (2007) identified four species of

worms belonging to the genus *Metastrongylus* viz. *M. elongatus*, *M. salmi*, *M. asymmetricus* and *M. pudendotectus* in Japanese wild boars.

Grossly, small gray nodules were noticed on the caudoventral borders of the diaphragmatic lobes. Large number of male and female lung worms of *M. apri* and *M. pudendotectus* were noticed in the bronchi and bronchioles in the ventral borders of the diaphragmatic lobes. Microscopically, the major findings were presence of several parasitic sections in the lumen of bronchioles surrounded by mucous and inflammatory cells and desquamation of bronchial epithelium along with infiltration of eosinophils, lymphocytes and neutrophils into the epithelium and lamina propria. These findings were in conformity with the lesions explained previously by Bhattacharya *et al*. (1971), Ramachandran and Sivadas (1971), Chauhan and Rao (1972), Chatterjee *et al.* (1975), Ashok Kumar *et al.* (2000), Ashok Gupta *et al.* (2002) and Gaurat and Gatne (2009). Ashok Kumar *et al*. (2000) noticed gross lesions in the diaphragmatic lobes particularly in the caudal portion in *M. apri* infection and also found the cross sections of the adult parasites and their ova in the lumen of bronchi and bronchioles.

The present study revealed stomach worms (*Physocephalus sexalatus*) in 0.94% of animals. Dev Sarma and Gogoi (1986) reported that out of 142 pigs examined, 25 (17.60%) animals were found infected with *P. sexalatus*. Grossly, small red worms were present in the stomach, that appeared as small red colour streaks on the mucosa of the stomach. Areas of slight hyperemia of the mucosa or formation of eroded areas or ulcers were also evident. Microscopically, the mucosa of stomach revealed changes of chronic catarrhal gastritis. In the fundic

region of stomach, necrosis and desquamation of the epithelium and infiltration of mononuclear cells, a few eosinophils and macrophages were noticed. There was atrophy of glandular epithelium or cystic dilatation of glands and infiltration by mononuclear cells and a few eosinophils in the cardiac region. The gross and microscopic lesions were similar to that described by Soulsby (1982) in *P. sexalatus* infestation and *Leman et al.* (1986) in *A. strongylina* and *P. sexalatus* infection. A few reports are available on the lesions caused by other stomach worms in pigs. Davenport and Stockdale (1967) reported the gross and microscopic lesions in a case of hyostrongylosis in a four year old sow. Gaurat and Gatne (2009) described the gross and histopathological lesions associated with *A. strongylina* infection in pigs. The gastric mucosa of fundic region was swollen and thrown into characteristic folds and numerous reddish nematodes were found embedded in thick mucous layer forming clusters at few places. Histopathological findings revealed extensive hyperplasia of goblet cells, eosinophilic infiltration of lamina propria and denudation of epithelium of the villi.

The present study revealed that considerable number of parasitic diseases were noticed in pigs with an incidence of 67.5%. It can be concluded from the results noticed in the present study that the helminth parasitic diseases are common in the area of study in pigs and can cause severe economic losses due to condemnation of organs or entire carcasses during meat inspection.

CHAPTER VI

SUMMARY

The present investigation was carried out to study the incidence and the associated pathomorphogical lesions in various helminth parasitic diseases in slaughtered pigs.

Out of 320 animals examined in the present study, 216 showed definitive lesions of different parasitic diseases on gross and microscopic examination accounting to an incidence of 67.5%.

In the present study, various helminth parasitic diseases that were observed in pigs were broadly grouped as the diseases caused by trematodes (3.13%), larvae of cestodes (8.75%) and nematodes (55.63%) and accordingly the pathomorphological lesions were described. Of these, trematodes caused schistosomosis (2.81%) and opisthorchiosis (0.32%), larvae of cestodes caused

hydatidosis (1.25%) and cysticercosis (7.5%) and the nematodes caused ascariosis (34.06%), stephanurosis (8.75%), metastrongylosis (11.88%) and parasitic gastritis resulting from stomach worm infestation (0.94%). Overall, the diseases caused by nematodes had highest percentage (55.63%) of incidence than others.

The incidence of schistosomosis in livers was 2.81% of total animals. Grossly, the affected livers were slightly enlarged and had a granular irregular surface. Microscopically, perioval granulomas or pseudotubercles with extensive fibrocellular reaction in the interlobular septa and in the portal triads were noticed. The granuloma contained ova of *schistosoma* and surrounded by eosinophils, macrophages, epithelioid cells, giant cells and lymphocytes along with extensive portal and septal fibrosis resulting in thickening of interlobular septa. No adult parasites were found in the sections.

The incidence of opisthorchiosis was 0.32% of the total animals examined. Grossly, thickened, cystic bile duct was noticed containing small flukes which were identified as *Opisthorchis tenuicollis*. Microscopical examination revealed the presence of cross section of fluke in the bile duct, that was extensively thickened, dilated and fibrosed along with hyperplasia of the bile duct epithelium.

The incidence of hydatidosis was 1.25%. Grossly, the hydatid cyst contained clear watery fluid and distinct whitish, thick cyst wall consisting of laminated layer of variable thickness. Microscopically, the cyst consisted of hyaline cyst wall surrounded by fibrous connective tissue capsule infiltrated with

large numbers of eosinophils, mononuclear cells and plasma cells. The protoscolices were also noticed attaching to the cyst wall.

Cysticercosis was noticed in 7.5% of the animals. The metacestodes of *T. solium* were present in various organs like tongue, heart, liver, skeletal muscles and brain. The cross sections of cysticerci were evident with or without invaginated scolex in different organs, surrounded by fibrocellular reaction consisting of lymphocytes, plasma cells and eosinophils.

The incidence of ascariosis was 34.06% which was highest among all the parasitic diseases observed. Grossly, the milk spots were found on the liver that were star like in shape and grayish white in colour or of pearl like nodules scattered throughout the liver. Microscopically, the liver sections revealed areas of extensive haemorrhage into the lobule, distortion of hepatic architecture, eosinophilic granulomas, presence of ascarid larvae, thickening of interlobular septa and lymphoid follicle formation. Adult worms were present in the intestines of some animals apart from the milk spots on the livers. The intestinal mucosa revealed desquamation of epithelium and infiltration by lymphocytes, plasma cells and macrophages.

The incidence of stephanurosis was 8.75%. The liver in these animals had whitish nodules and revealed the presence of immature parasites. In some animals, the worms were also found in the perirenal fat in the free state. Microscopically, congestion of sinusoids, haemorrhages, cut sections of immature worms surrounded by extensive eosinophilic infiltration, thickened interlobular

septa, hyperplasia of bile ducts and thickening of blood vessels were noticed. Cut sections of worms were noticed in the perirenal fat along with infiltration of eosinophils. In the kidney, degenerative changes, necrosis of tubular epithelium, cystic dilatation of tubules, atrophy of few glomeruli and infiltration with mononuclear cells in the interstitium were noticed.

Metastrongylosis was noticed in 11.88% of animals out of 320 animals examined. Grossly, small gray nodules were noticed on the caudoventral borders of diaphragmatic lobes. Large numbers of male and female lung worms of *M. apri and M. pudendotectus* were noticed in the bronchi and bronchioles in the ventral borders of the diaphragmatic lobes. Microscopically, several sections of the parasites in different stages of development were present in the lumen of the bronchioles, surrounded by mucus and inflammatory cells. The bronchial epithelium was desquamated and eosinophils, lymphocytes and neutrophils infiltrated the epithelium and lamina propria.

The incidence of stomach worms (*P. sexalatus*) was 0.94% out of 320 pigs. Grossly, small red worms were present in the stomach. Microscopically, the mucosa of stomach revealed changes of chronic catarrhal gastritis.

From the results recorded in the present study, it was evident that the helminth parasitic diseases are the major diseases in pigs with an overall incidence of 67.5%. The pathomorphological lesions noticed in different organs indicated the extent of damage caused by the helminth parasites.

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