Pathological, Histochemical and Haemato-biochemical Studies on Gastrointestinal Parasitosis in Domestic Fowl (*Gallus Domesticus*)

Dr. Riyaz Ahmad Khan
(2007-V-70-M)

Division of Veterinary Pathology

Faculty of Postgraduate Studies

Sher-e-Kashmir University of Agricultural Sciences & Technology of Kashmir

2011

Pathological, Histochemical and Haemato-biochemical Studies on Gastrointestinal Parasitosis in Domestic Fowl (*Gallus Domesticus*)

Dr. Riyaz Ahmad Khan
(2007-V-70-M)

Thesis

Submitted to

The Faculty of Postgraduate Studies
Sher-e-Kashmir University of Agricultural Sciences &
Technology of Kashmir
in partial fulfilment of requirement for the award of the degree of

Master of Veterinary Sciences
(Veterinary Pathology)

2011

This work is dedicated
to my
Family-Mummy, Daddy
& Ruhi…

Sher-e-Kashmir
University of Agricultural Sciences & Technology of Kashmir
Division of Veterinary Pathology, Shuhama Campus Srinagar– 190 006
-::o::-

Certificate – I

This is to certify that the thesis entitled, **"Pathological, Histochemical and Haemato-biochemical Studies on Gastrointestinal Parasitosis in Domestic Fowl (*Gallus domesticus*)"** submitted in partial fulfilment of the requirements for the award of the degree of **Master of Veterinary Sciences (Veterinary Pathology)**, to the **Faculty of Postgraduate Studies, Sher-e-Kashmir University of Agricultural Sciences & Technology of Kashmir** is a record of bonafide research work carried out by **Dr. Riyaz Ahmad Khan (Regd. No. 2007-V-70-M)** under my supervision and guidance. No part of the thesis has been submitted for any other degree or diploma.

It is further certified that information received during the course of investigation has duly been acknowledged.

(Prof. M.M. Darzi)
Chairman
Advisory Committee

Endorsed

Professor & Head,
Division of Veterinary Pathology,
FVSc & AH., Shuhama

Sher-e-Kashmir
University of Agricultural Sciences & Technology of Kashmir
Division of Veterinary Pathology, Shuhama Campus Srinagar
-::o::-

Certificate – II

We, the members of the Advisory Committee of **Dr. Riyaz Ahmad Khan (Regd. No. 2007-V-70-M)**, a candidate for the degree of **Master of Veterinary Sciences (Veterinary Pathology)** have gone through the manuscript of the thesis entitled, **"Pathological, Histochemical and Haemato-biochemical Studies on Gastrointestinal Parasitosis in Domestic Fowl (*Gallus domesticus*)"** and recommend that it may be submitted by the student in partial fulfilment of the requirements for the award of the degree.

ADVISORY COMMITTEE

Chairman

Prof. M.M. Darzi
Head, Division of Veterinary Pathology,
FVSc & AH, Shuhama

Members

Dr. M. S. Mir,
Associate Professor, Division of Veterinary Pathology, FVSc & AH., Shuhama

Prof. R. A. Shahardar,
Professor and Head, Division of Veterinary Parasitology, FVSc & AH., Shuhama

Mr. M.I. Bhat,
Associate Professor (Agri-Statistics) & Incharge Computer Lab, FVSc & AH., Shuhama

Dean PG Nominee

Dr. Abdul Shakoor Bhat,
Associate Professor,
Division of Veterinary Pharmacology and Toxicology, FVSc & AH., Shuhama

Sher-e-Kashmir
University of Agricultural Sciences & Technology of Kashmir
Shalimar Campus Srinagar– 191 121
-::o::-

Certificate – III

This is to certify that the thesis entitled, **"Pathological, Histochemical and Haemato-biochemical Studies on Gastrointestinal Parasitosis in Domestic Fowl (*Gallus domesticus*)"** submitted by **Dr. Riyaz Ahmad Khan (Regd. No. 2007-V-70-M)** to the **Faculty of Postgraduate Studies, Sher-e-Kashmir University of Agricultural Sciences & Technology of Kashmir** in partial fulfilment of the requirements for the award of the degree of **Master of Veterinary Sciences (Veterinary Pathology)** was examined and approved by the Advisory Committee and External Examiner on …….

Chairman
Advisory Committee

External Examiner

Professor & Head,
Division of Veterinary Pathology,

Director Resident Instruction-cum-Dean
Postgraduate Studies, SKUAST-K

Sher-e-Kashmir
University of Agricultural Sciences & Technology of Kashmir
Division of Veterinary Pathology, Shalimar – 191 121

-::o::-

Name of the student	:	Dr. Riyaz Ahmad Khan
Registration No.	:	2007-V-70-M
Major subject	:	Veterinary Pathology
Minor subjects	:	Veterinary Parasitology
Major advisor	:	Prof. M.M. Darzi, Prof. & Head, Division of Veterinary Pathology
Title of the Thesis	:	**"Pathological, Histochemical and Haemato-biochemical Studies on Gastrointestinal Parasitosis in Domestic Fowl (*Gallus domesticus*)"**

ABSTRACT

The present study was conducted to investigate the prevalence and pathology of various gastrointestinal parasites of domestic fowl in Ganderbal District of Kashmir valley. A total of 100 birds were screened. The overall prevalence of gastrointestinal parasites was 94%. Eleven different helminth species were recorded which included five species of nematodes and cestodes each and a single species of trematode. The prevalence of each species was: *Hetrakis gallinarum* (63%), *Ascaridia galli* (41%), *Capillaria annulata* (35%), *Acuaria hamulosa* (6%), *Dysphrynx spiralis* (1%), *Raillietina cesticillus* (21%), *Hymenolepis carioca* (19%), *Raillietina tetragona* (10%), *Amoebotaenia sphenoides* (8%), *Choanotaenia infundibulum* (7%) *and Prosthogonymus pellucidus* (5%). 100% prevalence of parasitic infections was observed in birds scored as low, followed by medium (94.36%) and good (80%). Growers (100%) were more frequently infected than adults (90.32%). The histopathological lesions observed include goblet cell hyperplasia, compression and disruption of villi,

desquamation of epithelium, broadening and clubbing of the villi, congestion, haemorrhages, localised edema, focal necrosis and inflammatory reactions characterized by infiltration of mononuclear and polymorphonuclear cells. Mast cell hyperplasia was characteristically observed in some parasitic infections indicating local type-1 hypersensitivity reaction. Qualitative increase of mucopolysaccharides especially of acidic type was observed in all infected organs. Haemoglobin and total leukocyte counts revealed significant decrease and increase, respectively, in case of mixed nematode and cestode infections. Packed cell volume showed a significant decrease in pure nematode and mixed infections. Total Erythrocyte count and Erythrocyte sedimentation rate showed no significant change. Lymphocyte count observed significant increase in pure cestode and mixed infections of nematodes and cestodes while as monocyte counts revealed significant increase in pure infections of nematodes and cestodes. Eosinophil counts showed significant increase in case of pure nematode and mixed infections. Serum biochemical values revealed significant decrease in total protein and albumin contents in case of mixed infections.

Key words: Domestic fowl, Ganderbal District, Haemato-biochemical, Histochemistry, Parasites, Pathology, Prevalence.

Signature of Student

Dated: _____

Signature of Major Advisor

Dated: _____

ACKNOWLEDGEMENT

After School and College life, I have learned one thing – I could never have done any of this, particularly the research and writing that went into this dissertation, without the support and encouragement of a lot of people.

First, I would like to thank my advisor, ***Dr. Mohammad Maqbool Darzi,*** *Professor-cum-chief scientist, Division of Veterinary Pathology. I owe you so much. You've been my mentor, my confidant, and a never-ending fount of moral support. You have given so much of yourself to help me succeed. If I do take the academic path, I only hope that I can be half the advisor that you have been to me. Whatever path I do take, I will be prepared because of you. You have given me the courage to make the next transitions in my life. For all of this, I thank you sir.*

If there were to be any more well wishers, who were as keen as my guide, in my progress they were ***Dr. Masood Salim Mir,*** *Associate Profssor-cum-Sr. Scientist, Division of Veterinary Pathology, member Advisory committee, who guided me in the research work done here. His interest in my welfare contributed a lot in bringing this work to shape. I will forever be indebted to him. You're the first person I turn to in good times and in bad during my stay at FVSc & A. H. as M.V.Sc. scholar.*

I would also like to thank my advisory committee members ***Dr. Rafiq A. Shahardar,*** *Professor-cum-chief scientist, Division of Veterinary Parasitology and* ***Mr. M. I. Bhat,*** *Associate Profssor, Agri. Statistics, who provided me with invaluable advice and comments on both my research and my future research career plans. I would also like to thank* ***Dr. Abdul Shakoor Bhat,*** *Associate Professor, Division of Veterinary Pharmacology and Toxicology for providing apt help and valuable suggestions.*

This work would not have been possible without the support of ***Dr. Shoab A. Kamil,*** *Associate Professor-cum-Sr. Scientist, Division of Veterinary Pathology. You're always there for me, when I need help with my research and when I need moral support. You were instrumental in helping me find my dissertation topic and in helping me get past all the self-doubting that inevitably crops up in the course*

of M.V.Sc. You're the first person I turn to in good times and in bad during my stay at FVSc & A. H. as M.V.Sc. scholar. You have given me the courage to make the next transitions in my life. For all of this, I thank you.

I express my sincere thanks to ***Dr. Pankaj Goswami,*** *Associate Professor Division of Veterinary Pathology for his valuable suggestions during the course of my studies. I especially express my infinite indebtedness* ***Dr. Showkat A. Shah****, Assistant Professor, Division of Veterinary Pathology for his friendly aptitude, ardent humanistic help, appreciation and guidance. Thank You Sir.*

I am extremely thankful to staff members of Division of veterinary pathology, ***Mr A. R. Rather, Mr. M. S. Wadoo, Mrs. Nighat, Mr. M. S. Bhat, Mr. Ali Mohammad, , Mr. Ghulam Mohammad and Mr Fayaz Ahmad*** *for their timely help and consistent cooperation.*

I sincerely acknowledge the support and unconditional timely help provided by ***Dr. T. A. S. Ganaie****, Professor and Head, Division of Animal Breeding and Genetics, for his able, kind, dedicated and concerted guidance, deep personal interest, valuable suggestions.*

I am also highly thankful to my all teachers who have taught me from my school life to this point of life.

I am also grateful to my seniors ***Dr(s); Imran Hamid, Feroz Itoo,*** *and my colleagues* ***Dr(s); Shabir Ahmad , Tariq Ahmad, and Umar*** *for their cooperation and help during my work. I take this opportunity to thank all my juniors for their keen interest and help.*

I would remember the pleasurable company of my batch mates and friends ***Dr(s); Altaf , Mudasir , Shahid, Manzar, Bilal , Zahoor Khanday, Maqsood, Khursheed, Zahoor wani, Rafeh, and Zubair,*** *throughout.*

There are absolutely no words to describe the awe-inspiring company of my wife ***Rabiya****. She has been a revelation for me, a shoulder to cry on, a friend to rely upon and what not, during all these years. Always remember, this work would' not have been possible without your overwhelming prop up.*

As one grows older, one feels increasingly conscious of the debt owed to my best friends with whom one has lived, shared and enjoyed

life. It is my pleasure to acknowledge the larger than life company of my best friends ***Mushtaq, Riyaz and Sajad.***

I am happy to acknowledge the pleasant and everlasting company and encouragement rendered by my cousins: ***Feroz, John, Amir, and Adil*** *and the three wonder kids* ***Uzair, Tahura and Ibraheem.***

I am highly thankful to ***Dr. Bashir Ahmad War (Aba-G)*** *for his continuous encouragement and support.*

Where the emotions are involved, words cease to mean, there are no words to pay regards to my reverend parents who took pains, pains and pains to bring me to this stage. No words shall be adequate to prove how indebted I am to my Parents: ***Mr. Mohmad Afzal Khan and Mrs. Zaiba Khan*** *Without your unending support and love from childhood to now, I never would have made it through this process or any of the tough times in my life. Thank you. I am indebted to my Inlaws* ***Mummy***, ***Papa***, ***Sabi*** *and* ***Umar*** *who helped me morally and have firmlys stood by me in my endeavours, in word and action. Your immeasurable love and benevolent patronage is beyond the bounds of acknowledgement.*

My thanks are due to my divine brother ***Fayaz Ahmad Khan*** *for being a joy in my life. Always remember that you are absolutely unique!*

Faith remains as the surest and strongest hope during darkest days and the Almighty Allah leads us to light. Faith in Allah has been the prime support that leads me to this position in life.

Riyaz Ahmad

Place :

Date :

CONTENTS

LIST OF TABLES

LIST OF FIGURES

86	Section of fowl intestine infected with *Raillietina tetragona* revealing destruction of the villi and desquamation of epithelium. HE. X250.	40
87	Section of fowl duodenum infected with *Raillietina tetragona* revealing duodenitis. HE. X280.	40
88	Section of fowl jejunum infected with *Raillietina tetragona* revealing jejunitis. HE. X280.	40
89	Section of fowl intestine infected with *Raillietina tetragona* revealing necrosis of the villi. HE. X300.	40
90	Section of fowl intestine infected with *Raillietina tetragona* revealing infiltration of the inflammatory cells. HE. X350.	40
91	Intestine of Fowl infected with *Amoebotaenia sphenoides* revealing fluidy intestinal contents.	40
92	Section of fowl duodenum infected with *Amoebotaenia sphenoides* revealing scolex burrowed deep into the mucosa. HE. X300.	40
93	Section of fowl intestine infected with *Amoebotaenia sphenoides* revealing desquamation of epithelium. HE. X250.	40
94	Section of fowl intestine infected with *Amoebotaenia sphenoides* revealing the infiltration of inflammatory cells into the core of villi. HE. X330.	40
95	Intestine of Fowl revealing multiple *Choanotaenia infundibulum* worms.	40
96	Section of fowl intestine infected with *Choanotaenia infundibulum* revealing mild duodenitis. HE. X350.	40
97	Section of fowl intestine infected with *Choanotaenia infundibulum* revealing necrotic villi. HE. X400.	40
98	Section of fowl intestine infected with *Choanotaenia infundibulum* revealing desquamation of the epithelium. HE. X300.	40

CHAPTER – 1

INTRODUCTION

Poultry is one of the fastest growing segments of the agricultural sector in India today. While the production of agricultural crops has been increasing from 1.5 to 2 percent per annum that of eggs and broilers has been rising at a rate of 8 to 10 percent per annum (Mehta *et al.,* 2003). As a result, India is now the world's fifth largest egg producer and the eighth largest producer of broilers. Poultry industry occupies an important position in the provision of animal protein (meat and egg) to man and generally plays a vital role in the national economy as a revenue provider. Poultry is one of the most intensively reared of the domesticated species and one of the most developed and profitable animal production enterprises (Obiora, 1992). Its importance in national economies of developing countries and its role in improving the nutritional status and income of many small and marginal farmers have been recognized by various scholars and rural development agencies (FAO, 1987; Creevey, 1991; Kitalyi, 1998).

Poultry includes chicken, ducks, geese, quails, pigeons etc. Among these the most important is the domestic fowl which nested the human backyards since the civilization of man. Poultry production in Africa and parts of Asia is still distinctively divided into commercialized and village enterprise sub sectors, each with its peculiarities.

In the twentieth century, the fowl rearing came up as a commercial sector at the global level and its fast growth rate invited capital investments. In the second half of the century, fowl rearing emerged as an industrial sector. Currently the commercial poultry industry constitutes an important component of the economy of almost all the developed and developing countries. The commercial farming remains a venture in the urban and suburban areas involving improved, high producing specialized breeds. The village enterprise however, consists of indigenous domestic fowls (*Gallus domesticus*) variously referred to as local or

rural chickens, backyard poultry or village chickens, or free range chickens. These refer to breeds, strains and ecotypes with no improvement history (Njue *et al*., 2001) and chickens indigenous to the particular locality they are found. These constitute a rich genetic resource base for any future genetic improvement and production of strains adaptable to the particular ecotypes (Horst, 1988).

Poultry raising, as a livestock enterprise, is available to all farming families including the poorest (Bell, 1992). The management of the backyard poultry is largely the responsibility of women and children (Losada *et al*., 1997; Martins, 1995). Domestic fowl (*Gallus domesticus*), therefore, forms the backbone of our rural economy providing petty cash to the poor farmers especially to the village women who are having basically no job related to income generation. Over 90% of rural households rear chickens in small flocks of about 20 birds. These flocks are of mixed age and mostly unhoused or poorly housed. Most village chicken production systems are based mainly on native, unimproved domestic species which require very low levels of inputs (Sayila, 1999) leading to low output, hence the term 'low input/low output system' has been coined for backyard poultry.

The local native chicken population of Jammu and Kashmir State is 7.3 lacs out of which 63.50 per cent is in Kashmir Division alone (17^{th} Indian Livestock Census, 2003), being reared as backyard in scavengering system of farming. These birds thrive on kitchen waste, damaged cereals, leftover human foods, insects, green vegetables, leaves or occasionally provided rice, paddy or maize as a supplementary feed.

In India backyard poultry production has increased only by 16 per cent as compared to 150 times increase in broiler production (Ravi Kumar *et al*., 2002). With the advent of a concept of sustainable and ecofriendly agriculture, it is being realized to explore the local indigenous genetic potential to conserve indigenous livestock genetic resources and evolve different types of birds suitable for their own agroecological conditions and production systems (Sharma *et al*., 2002). A

new research focus on village chicken has developed in many developing African and Asian countries and in China village chicken production system has been included in mainstream agriculture. Household poultry has been included in the FAO special programme for food security (FAO, 1997).

Production of domestic fowl is constrained by many extrinsic factors among which malnutrition, poor management and the absence of biosecurity are outstanding. Losses have also been attributed to limited housing and veterinary care services. Furthermore, poor genetic potential due to lack of selection and predation are also potential threats to productivity (Calnek *et al*., 1997)

Among the various diseases, parasitism ranks high among factors that threaten the domestic fowl production (Adene and Dipeolu, 1975). Parasitism is an extreme form of interspecific relationships in which one organism (parasite) is metabolically dependent on the other organism (host), hence the association is an obligatory one for the parasite throughout or during a particular stage of life cycle. Parasites have the potential to harm their hosts (Marquardt and Demaree, 1985; Zelmer, 1998) and may even cause premature death of the hosts (Soulsby, 1975) but it is their ability to evade the host immune response that distinguishes them from other symbionts (Zelmer, 1998). Parasitism in birds is a great concern causing heavy losses and may affect hosts at the individual, population and community level. At the individual level parasites can cause disease and death of the host. The effects are usually density dependent and heavy infections are often encountered in dying and dead individuals. Postmortem examinations have shown that the free range chickens with high worm loads tend to have poor body conditions (He *et al*., 1990; Permin *et al*., 1997b; Mukaratirwa *et al.,* 2001). Helminths exert their effects on the host by different ways such as blood sucking, tissue destruction during larval migration, feeding, mechanical or chemical irritation of contact surfaces, liberation of toxic metabolites and obstruction of excretory ducts, air passages or blood vessels (Nielson, 1976; Kassai, 1999). The infected organs vary considerably in their ability to compensate for the loss of

functional tissue cells, for example the gastrointestinal tract (GIT) can tolerate a substantial number of helminth parasites than does the trachea (Soulsby, 1976). However, heavy worm counts have been implicated to cause reduced growth and egg production, emaciation, and anaemia (Whitmarsh, 1997; Permin and Hansen, 1998; Ruff, 1998; Irungu *et al*., 2004; Kaufman *et al*., 2007) and in severe conditions they are reported to cause deaths in poultry (Flatt and Nelson, 1969; De Rosa and Shivaprasad, 1999). In addition, the roles of poultry worms such as *Heterakis gallinarum* has been associated with the transmission of *Histomonas meleagridis* in turkeys and chicks (Denmark and Cromroy, 2006). Moreover, it has been reported that parasitic infection or their concurrent infections result in immunosuppression, especially in response to vaccines against some poultry diseases.

Owing to the physical functions of the blood and its intimate relationship with all body tissues, it is to be expected that in many, if not in all, parasitic infections slight to extensive alterations may occur in either the formed or unformed elements of the tissue. Various reports indicate that parasitic infections cause leucocytosis, heterophilia, eosinophilia, erythrocytopaenia, decreased packed cell volume, low haemoglobin value, hypoglycemia and hypoproteinaemia (Sharma *et al*., 1984; Samad *et al*., 1986; Sekar *et al*., 1986; Verma *et al*., 1993; Phukan, 2006; Deka and Borah 2008; Shiekh, 2009).

While there is some knowledge regarding the presence of helminth parasites in the domestic fowl, there is a paucity of information on the epidemiology, clinicopathology and level of helminth infections in domestic fowl of Kashmir valley. There is also the need to constantly assess the status of village chicken production constraints and the dynamic of their interactions. In addition, as cofactors in other poultry diseases, the knowledge of their occurrence and prevalence is essential in understanding the epidemiology of such diseases and the design of their appropriate control measures. The current study was, therefore, designed to investigate the prevalence, pathological, histochemical and haemato-

biochemical changes of gastrointestinal parasitosis in domestic fowl of Ganderbal district of Kashmir valley with the following objectives :

1. To determine the prevalence of various gastrointestinal parasites of domestic fowls in Ganderbal district of Kashmir valley.
2. To study gross and histopathological alterations in spontaneous cases of gastrointestinal parasitosis.
3. To study the histochemical alterations associated with natural parasitism.
4. To compare haematological values viz. Total Erythrocyte Count (TEC), Total Leukocyte Count (TLC), Differential Leukocyte Count (DLC), Haemoglobin Concentration (Hb), Packed Cell Volume (PCV) and Erythrocyte Sedimentation Rate (ESR) of infected birds with those of normal ones.
5. To compare blood biochemical parameters like Total Protein (TP), Serum Albumin (A), Serum Globulin (G) and A/G ratio with that of normal ones.

CHAPTER – 2

REVIEW OF LITERATURE

2.1 Prevalence

Joshi and Kamalapur (1971) studied the incidence of *Heterakis gallinarum* in domestic fowls to be 0.2 per cent of the 900 fowls examined in Madhya Pradesh.

Mutafova (1976) studied the biology of *Heterakis gallinae* (Schrank, 1780) and provided the evidence of greater influence of female sex on survival rate of *Heterakis gallinae* than that of male sex in natural female chicken populations in Bulgaria.

Mishra *et al*. (1980) in Patna, India found that 78.6 per cent of 1415 fowls were infected with *Ascaridia galli.*

Ssenyonga (1982) during his studies on the prevalence of helminth parasites of both broiler and local poultry in Uganda found that the nematodes like *Ascaridia galli* and *Heterakis* spp. and cestodes like *Raillietina* spp. were most prevalent in both the groups of birds.

Fotedar and Khateeb (1986) studied the incidence and seasonal variation of helminth parasites of domestic fowl (*Gallus gallus domesticus*) in Kashmir (India) and concluded that the percentage of infection and worm burden increased at optimum temperature and during rainy season.

Pandit *et al*. (1991) studied the prevalence of helminth parasites in Desi fowls and investigation showed average rate of infection as high as 80.00 per cent. The prevalence of helminth parasites recorded was *Ascaridia galli* (28%), *Heterakis gallinarum* (22%), *Heterakis indica* (8%), *Syngamus trachea* (9%), *Echinuria uncinata* (1%), *Capillaria annulata* (4%), *Capillaria caudinflata* (2%), *Cotugnia diagonopora* (10%) and *Echinostoma revolutum* (1%).

Yadav and Tandon (1991) after their thorough study on the helminth parasitism of domestic fowl (*Gallus domesticus*) in a subtropical high-rainfall area of India stated that *A. galli* was the most prevalent nematode in domestic fowl in India with a prevalence of 60 per cent.

Morgenstern and Lobsiger (1993) found that in Switzerland the prevalence of *Ascaridia galli* was 24.3 per cent on free range, 8.5 per cent on deep-litter and 0 per cent in battery cages. For *Capillaria* spp prevalence was 29.5 per cent on free range, 1.7 per cent on deep-litter and 0 per cent in battery cages.

Khan *et al.* (1994) examined Chickens in Pakistan and proved 12 per cent to be infected with *Ascaridia galli*.

Jacobs *et al.* (1997) described some of the important nematode parasites of Poultry like *Ascaridia galli, Heterakis gallinae, Capillaria annulata, Oxyspirura mansoni, Syngamus trachea* and *Cheilospirura hamulosa*. They also explained their location in the gastrointestinal tract.

Permin *et al.* (1999) carried out cross sectional prevalence study of gastrointestinal helminthes in Danish poultry production and confirmed the high risk of helminth infections in free-range and backyard systems but prevalence may also be high in deep litter systems.

Eshetu *et al.* (2001) studied the gastro-intestinal helminths of scavenging chickens in four rural districts of Amhara region of Ethiopia and found that 91.01 per cent of chicks were infected with helminths with their prevalence as *Heterakis gallinarum* (17.28%), *Subulura brumpti* (17.60%), *Ascaridia galli* (35.58%), *Cheilospirura hamulosa* (0.75%), *Dispharynx spiralis* (2.62%), *Raillietina echinobothrida* (25.84%), *Raillietina tetragona* (45.69%), *Raillietina cesticillus* (5.62%), *Amoebotaenia sphenoides* (40.45%), *Davainea proglottina* (1.12%) and *Choanotaenia infundibulum* (4.49%).

Mond *et al*. (2001) studied the maturity status and seasonality of *Raillietina permista* infesting poultry at Allahabad, U.P., India and found the

infection prevalence of the cestode to be 52.63 per cent with mean worm burden to be 46.0. They also found that the tapeworms attained the maturity during breeding period in female and during pre-breeding period in male poultry.

Shah *et al*. (2001) made comparative studies on the prevalence of cestode parasites in indigenous and exotic layers at Faisalabad, Pakistan and found that prevalence was higher (59.4%) in indigenous layers than in exotic layers (16.0%).

Magwisha *et al*. (2002) compared the prevalence and burdens of helminth infections in growers and adult free-range chickens and found that there were significantly higher worm burdens in growers than in adults for *Ascaridia galli, Capillaria caudinflata, Raillietina tetragona, Syngamous trachea, Tetrameres americana, Tetrameres fissispina* and *Trichostrongylus tenuis*. Conversely *Allodapa suctoria* and *Capillaria annulata* showed significantly higher worm burdens. The sex of the chickens influenced the burdens of *Heterakis brevispiculum*.

Nithiuthai *et al*. (2003) examined about two thousand gastrointestinal tracts from native chicken sold in the avian market of Klongtoey, Prakanong, Bangkok and recovered three groups of worms viz. intestinal flukes, tapeworms and nematodes. Intestinal flukes comprised of *Echinostoma* spp (1.0%), *Prosthogonimus* spp (0.1%) and *Notocotylus* spp (0.1%). Tapeworms were *Amoebotaenia sphenoides* (0.5%), *Cotugnia digonopora* (2.1%), *Diorchis* spp (0.1%), *Hymenolepis* spp (0.9%) and *Raillietina* spp (14.5%). The nematodes were of five genera viz. *Ascaridia galli* (7.2%), *Capillaria* spp (1.0%), *Gongylonema ingluvicola* (4.1%), *Heterakis* spp (9.4%), *Strongyloides* spp (0.4%) and *Tetrameres americans* (1.1%)

Ashenafi and Eshetu (2004) conducted a survey on gastrointestinal helminthes of local chickens in Central Ethopia and recovered six nematode and six cestode species. The major cestode species recovered include *Raillietina echinobothrida* (65.3%), *Hymenolopis containana* (53.7%), *Amoebotaenia* spp

(37.4%), *Raillietina tetragona* (35.8%), *Raillietina cesticillus* (19.0%) and *Choanotaenia infundibulum* (3.2%). The major nematode species recovered were *Ascaridia galli* (55.3%), *Heterakis gallinarum* (32.6%), *Subulura* spp. (27.4%), *Cheilospurura* spp. (2.1%), *Dispharynx* spp. (2.1%) *and Capillaria* spp. (1.6%).

Hassouni and Belghyti (2006) studied the distribution of gastrointestinal helminths in chicken farms in the Gharb region, Morocco and reported the various helminth species with their prevalence as: *Notocotylus gallinarum* (0.7%), *Hymenolepis carioca* (3.7%), *Raillietina echinobothrida* (5.7%), *Hymenolepis contaniana* (7%), *Raillietina tetragona* (9.3%), *Raillietina cesticillus* (12%), *Capillaria obsignata* (6%), *Subulura brumpti* (15.3%), *Heterakis gallinarum* (10%), *Cheilospirura hamulosa* (2.7%), *Dispharynx nasuta* (5.3%), *Ascaridia galli* (9%) and *Tetrameres* spp. (3.3%). The prevalence and mean intensity of helminth infections did not differ significantly between male and female chickens.

Dehlawi (2007) studied the occurrence of nematodes in the intestine of local (Baladi) chicken (*Gallus gallus domesticus*) in Jeddah province, Saudi Arabia and got three nematodes with their occurrence as: *Ascaridia galli* (34.4), *Sublura brumpti* (12.5) and *Capillaria caudinflata* (12.5).

Luka and Ndams (2007) investigated upon the gastrointestinal parasites of domestic chicken *Gallus gallus domesticus* in Samura, Zaria Nigeria and found that out of the 92 birds examined about 62 per cent were infected with various species of gastrointestinal parasites with species specific rates as: *Raillietina tetragona* (23.9%), *Raillietina echinobothrida* (13.0%), *Raillietina cesticillus* (9.8%), *Choanotaenia infundibulum* (10.9%), *Hymenolepis carioca* (25.0%), *Ascaridia galli* (43.8%) and *Heterakis gallinarum* (22%).

Abdelqader *et al*. (2008) found the prevalence and burden of gastrointestinal helminthes among local chickens, in northern Jordan as: *Ascaridia galli* in female 28 per cent, in male 43 per cent; *Capillaria obsignata* 0.5 per cent; *Heterakis gallinarum* 33 per cent; *Amoebotaenia cuneata* 4.3 per cent;

Choanotaenia infundibulum female 23 per cent, male 13 per cent; *Davainea proglottina* 1.4 per cent; *Hymenolepis cantaniana* 11 per cent; *Hymenolepis carioca* female 35 per cent, male 24 per cent; *Raillietina cesticillius* female 5 per cent, male 11 per cent; *Raillietina echinobothrida* 16 per cent; and *Raillietina tetragona* 18 per cent. The prevalences of *A. galli* and *Raillietina cesticillus* were higher in male than female hosts while those of *Choanotaenia infundibulum* and *Hymenolepis carioca* were higher in females.

Salam *et al*. (2009a) studied the prevalence of *Cheilospirura (Acuaria) hamulosa* in the indigenous chicken of Kashmir valley, India during the period of two years and got an overall prevalence of 3.5 per cent.

Salam *et al*. (2009b) carried out a survey for a period of two years on the prevalence of cestode *Amoebotaenia sphenoides* in a sample size of 478 birds collected from different localities of Kashmir valley, India. The overall prevalence rate of the cestode was found to be 6.69 per cent (32/478) and annual occurrence rates of 6.8 per cent (16/233) and 6.5 per cent (16/245) were respectively reported for the 1st and 2nd years of study.

Nnadi and George (2010) in a cross sectional study recovered eight species of endoparasites from village chicken of Nigeria with their prevalence as: *Ascaridia galli* (17.2%),*Heterakis gallinarum* (12.6%), *Capillaria.* spp (5.7%), *Raillietina* spp (5.7%), *Syngamus treachea* (4.6%), *Davainea proglottina* (3.45%), *Sublura brumpti* (2.3%), *Amoebataenia* spp (1.15%).

2.2 Pathology

2.2.1 *Ascaridia galli*

Ackert and Herrick (1928) studied the effects of the nematode *Ascaridia lineata* (Schneider) on growing chickens and concluded that infections with this nematode may cause reductions in the growth and weight loss which may be related to the damage of the intestinal mucosa, leading to loss of blood and probably secondary infections.

Kadziolka (1960) described the histopathology of ascaridiasis in chicken and observed microscopical changes in the wall of intestine.

Ikeme (1971) studied the pathogenicity and pathology of *Ascaridia galli* and found hemorrhagic lesions, and destruction and erosion of the glandular epithelium by the migrating larvae.

Matta (1980) carried out studies on *Ascaridia galli* infection of chick with special reference to its histopathology and found that after 7 to 15 days of infection with *Ascaridia galli*, there were gross petechial lesions and generalized edema in the intestine and after 90 days of infection, the lesions diminished and ultimately there was only a thick layer of mucin.

Mishra *et al*. (1980) worked on pathogenicity of *Ascaridia galli* in poultry in Patna, India and found that fowls infected with *Ascaridia galli,* showed pronounced sloughing of the intestinal epithelia, infiltration of lymphocytes, macrophages and histiocytes in lamina propria and proliferation of fibroblasts in the submucosa.

Soulsby (1982) reported that in ascaridiasis intestinal mucosa reveals inflammatory lesions and focal hemorrhages caused by the burrowing of parasites.

Dahl *et al.* (2002) studied the effect of concurrent infections with *Pasteurella multocida* and *Ascaridia galli* on free range chickens and found that *A. galli* infection followed by a secondary infection with *P. multocida* resulted in more birds with pathological lesions and continued *P. multocida* excretion.

Arunachalam *et al*. (2003) carried out an experiment to study the histopathological changes in broiler chicken with infective stages of *Ascaridia galli* eggs. Histopathological changes revealed mild to moderate goblet cell hyperplasia, disruption of villi, desquamation of epithelium, infiltration with mononuclear cells, focal necrosis, congestion and hemorrhage in the mucosa, submucosal edema, squamous metaplasia of lining epithelium cells and cystic changes of mucosal glands.

Permin *et al.* (2006) worked on the consequences of concurrent *Ascaridia galli* and *Escherichia coli* infections in chickens and concluded that the infections are characterized by airsacculitis, peritonitis and/or polyserositis.

Adang *et al*. (2010) studied histopathology of *Ascaridia galli* infection on the liver, lungs, intestines, heart, and kidneys of experimentally infected domestic pigeons in Zaria, Nigeria ad observed hemorrhagic lesions in intestines. The infected pigeons had necrosis of the intestines that involved the villi, intestinal glands and the muscularis mucosa. There were mononuclear and polymorphonuclear cells in the necrotized areas.

2.2.2 *Heterakis gallinarum*

Soulsby (1968) stated that the direct effects of *Heterakis gallinarum* are slight and only in heavy infection there may be thickening of the caecal mucosa with a number of petechial hemorrhages on the surface.

Kaushik and Deorani (1969) worked on the tissue responses in primary and subsequent infections with *Heterakis gallinae* in chickens and on the process of formation of caecal nodules and reported that the causes for the appearance of granulomatous nodules would be continuous reinfections with *H. gallinarum* provoking a tissue phase for the parasite.

Joshi and Kamalapur (1971) studied gross and histopathology of the organs infected with *Heterakis gallinarum* which revealed prominent erosion of the epithelial lining, atrophy of the villi and preponderance of the lymphocytes in the mucous membrane near the worm.

Menezes *et al.* (2003) studied the histopathology associated with the *Heterakis gallinarum* and *Heterakis isolonche* in Pheasants. The histological sections revealed necrotic areas with cholesterol clefts in the submucosa, giant cell granuloma in the submucosa and serosa was centralized by necrosis and worm sections and nodules in the muscular and submucosa.

Brener *et al.* (2006) studied the pathology of the nematode *Heterakis*

gallinarum, in the turkey, and reported that the histological findings in the caeca were represented by the presence of *Heterakis gallinarum* worms, intense chronic diffuse inflammatory processes with mononuclear and polymorphonuclear leucocyte (heterophils) infiltrations.

Rabbi *et al*. (2006) investigated gastrointestinal helminths infection in different types of poultry and found that pathological lesions included tiny, white, circumscribed nodules of about 2-3 mm of diameter in the caecal mucosa in case of *Heterakis gallinarum* infection.

2.2.3 *Capillaria annulata*

Cram (1936) made certain studies on the species of *Capillaria* parasite in the upper digestive tract of birds. He reported marked necrosis and sloughing of the epithelium of the oesophagus and crop in bobwhite (*Colinus virginianus*) infected with *Capillaria contorta* and also found that a thin connective tissue capsule surrounded adult worms. Lymphocyte and mononuclear infiltration was seen everywhere.

Pizarro *et al*. (2000) studied histopathology of the upper digestive system in *Capillaria contorta* parasitism in red-legged partridge under farm conditions in Spain and concluded the fundamental epithelial lesion consisted of spongiosis of the oesophagus and crop. In the individual with proventricular parasitism, epidermal metaplasia was also seen. The inflammatory reaction observed in the lamina propria was discrete and diffuse, except in those cases in which epithelial necrosis and bacterial colonies were detected.

Pito *et al*. (2008) studied the pathology of Capillarid nematodes *Baruscapillaria obsignata* and *Eucoleus annulatus* in Brazilian turkeys and reported that lesions due to *Baruscapillaria obsignata* mainly consisted of the thickening of intestinal villi with a mild mixed inflammatory infiltrate with the presence of mononuclear cells and heterophils. The lesions induced by *Eucoleus annulatus* were represented by foci of inflammatory infiltrate with heterophils in

the crop epithelium and esophagus of a single infected female.

2.2.4 *Acquaria hamulosa*

Padhi *et al.* (1987) studied the pathology caused by nematode helminthiasis in Desi fowls and concluded that the gross pathological changes consisted of nodule formation in the musculature of the caudal lobe of the gizzard in *Acuaria hamulosa* infection. The microscopic changes caused by *A. hamulosa* infection consisted of chronic inflammatory changes with marked infiltration with large number of reactive cells in the proventriculus and gizzard.

Menezes *et al.* (2003) worked on the pathology and frequency of *Cheilospirura hamulosa* (Nematoda, Acuarioidea) in Galliform hosts from backyard flocks and reported that the infections of this parasite was associated with severe lesions in the gizzard such as haemorrhages, ulcers and thickening of the mucosa and cuticle and single yellowish nodules on the caudoventral muscle. The microscopic lesions were characterized by chronic diffuse inflammatory processes and ulcers in the mucosa and granulomas in the muscular, submucosa and serosa layers of this organ.

Brener *et al*. (2006) studied the occurrence and pathology of *Cheilospirura hamulosa* (Diesing, 1851) (Nematoda, Acuariodea) in turkeys in Brazil and concluded that the microscopic lesions of the gizzard were severe and characterized by intense chronic diffuse inflammatory mixed granulocyte infiltrations, extending to the muscular layers.

Salam *et al*. (2009a) showed that lesions associated with *Cheilospirura* (*Acuaria*) *hamulosa* include discrete and coalescing nodules in the infected gizzard with cellular reactions characterized by large number of lymphocytes, monocytes, plasma cells, heterophils and in some sections severe eosinophilic reaction.

2.2.5 *Dysphrynx spiralis*

Soulsby (1982) stated that in severe infections of proventriculus with

Dysphrynx spiralis deep ulcers are seen, in which the anterior extremities of the worms are embedded. There is an extensive destruction of the glands and marked cellular infiltration of the underlying tissues.

Ramaswamy and Sundaram (1984) studied the histopathological changes in the proventriculus of fowls given experimental monospecific infection with *Acuaria spiralis* and observed an acute inflammation due to the migration of 3rd and 4th stage larvae in the initial stages of infection. They reported that there was a severe non-keratinizing squamous cell metaplasia of the lining epithelium with pronounced granulocytic infiltration at 4 to 8 days post infection. As the disease progressed there was extensive fibroplasia in the organ. They also found that by 50 to 100 days post infection pedunculated fibroadenamatoid growths developed in the mucosa obliterating the entire lumen of the organ.

Bawe *et al*. (2005) studied the pathology of *Dyspharynx spiralis* in guinea fowls and found that the parasites were embedded in the mucosa of the proventriculus. Lesions included discolouration, ulceration and petechial haemorrhages. Histopathology revealed degeneration of the submucosa and necrosis of the tissues of the proventricular gland.

2.2.6 *Raillietina* spp.

Gray (1976) studied the cellular response of the fowl small intestine to primary and secondary infections of the cestode *Raillietina cesticillus* and observed the mononuclear leukocyte infiltration in the tissues around the worm scolices.

Jha *et al.* (1981) studied the histopathology of poultry intestine in cestodiasis and revealed degeneration of epithelial cells, infiltration of macrophages and lymphocytes and proliferation of connective tissue of the intestine infected with *Raillietina tetragona* and *Raillietina cesticillus*.

Bawe *et al.* (2005) observed lesions associated with gastrointestinal parasites of Guinea fowls (*Numida meleagris galeata*) in Zaria, Nigeria and

reported that *Raillietina* species were attached to the mucosa of the ileum. Gross lesions observed were haemorrhages, thinning of the mucosa and oedema of the serosa. Histopathology included swelling and flattening of the intestinal villi as well as cellular (eosinophillic) infiltration of the superficial mucosae.

Anwar *et al*. (2000) studied the pathology of *Raillietina tetragona* infection in layers and the lesions observed were pin point haemorrhages, catarrhal enteritis, desquamation of villi and submucosal glands, congestion, cellular infiltration and granulomas.

Salam *et al*. (2010) worked on the pathology of *Raillietina cesticillus* in indigenous chicken (*Gallus gallus domesticus*) in the temperate Himalayan region of Kashmir and found that lesions in the intestines were characterized by varying degrees of degenerative changes to sloughing of mucosa in heavy and multiple infestations. In cases with higher parasitic load, partial villous atrophy with broadened surface and increased vascularity was observed in the duodenum and jejunum. At the site of parasitic attachment, the epithelium and glands were disintegrated. The inflammatory reaction was characterized by predominant heterophil presence especially in the areas of mechanical damage by scolices. Sparse infiltration of mononuclear cells, chiefly lymphocytes, and eosinophils was observed throughout the mucosa, especially in the lamina propria. Infiltration was not observed in muscularis or serosa.

2.2.7 *Choanotaenia infundibulum*

Mcorist *et al*. (1984) studied *Choanotaenia* spp infestation of Australian finches (Estrildidae) and found marked distension of the duodenum caused by 20-40 parasites and fluid filled distal intestines. Histological examination of the duodenum revealed inflammation.

Shiekh (2009) studied the pathology of intestines infected with *Choanotaenia infundibulum* and reported the presence of catarrhal exudates in the intestinal lumen and the anterior ends of the parasites penetrating into the mucosa.

The necrotic foci were evident at the point of attachment. Histopathological sections revealed atrophy of the villi. The lining epithelium revealed degenerative changes. Necrosis of the mucosa was observed at the site of penetration of the cestode.

2.2.8 *Amoebataenia sphenoids*

Anwar *et al*. (2000) studied the pathology of cestode infection in indigenous and exotic layers and observed that mucosa of duodenum appeared rough and pale in *Amoebataenia cuneata* infection while as histopathological lesions observed were desquamation of villi and submucosal glands.

Rabbi *et al*. (2006) investigated gastrointestinal helminth infection in different types of poultry and pathological lesions were found only in backyard poultry. Pathological changes were detected in case of *Amoebotaenia sphenoides* and *Heterakis gallinarum* infection. In *Amoebotaenia sphenoides* infection petechial hemorrhages were observed in the mucosa of the duodenum.

Salam *et al*. (2009b) showed that histopathological sections of the domestic fowl infected with *Amoebotaenia sphenoides* revealed the scolices burrowing deep into the mucosa. Disintegration of epithelium and glands, and infiltration of mononuclear cell was prominent.

2.2.9 *Hymenolepis carioca*

Pinto *et al*. (2008) studied the pathology of *Hymenolepis contaniana* in Brazilian turkeys. The lesions consisted of mild mixed inflammatory reaction with the presence of mononuclear cells and heterophils or severe transmural inflammatory processes characterized by the presence of mononuclear cells along the muscular and serosa layers of intestines.

2.2.10 *Prosthogonymus* spp

Soulsby (1965) reported marked exudative, desquamative and eosinophilic inflammation of the oviduct in *Prosthogonymus* spp. infection. Histologically,

marked oedema with infiltration of cells into the interstitial tissue of mucous membrane of the oviduct, especially in the albumin secreting region (pars albuminifera) was seen. Further, reduction in the glandular elements was reported. Similar changes were reported in the isthmus along with atrophy of the uterine glands.

Leok *et al.* (2002) studied the morphology of the oviduct fluke, *Prosthogonimus ovatus,* isolated from Indonesian native chickens and also studied the histopathological changes in the infected chickens which revealed polypous elevations, degeneration and exfoliation of the mucous epithelium of *Bursa of Fabricius* in addition to the stratification of the mucous epithelium and interstitial cell infiltration.

2.3 Histochemical Studies

Matta (1980) studied some histochemical changes in the small intestine of chicks experimentally infected with *Ascaridia galli* and *Echinoparyphium* sp. He found that in chickens infected with *Ascaridia galli*, there was little change in the amount of acid phosphatase in the intestine but the alkaline phosphatase was increased around the parasites and in the damaged tissues. It was reduced when the parasites matured. In *Echinoparyphium* infection the alkaline phosphatase was increased at the sites of attachment but the acid phosphatase was unchanged.

Lee *et al*. (1986) propounded that mast cells are major effector cells in the immune response to infection with helminths.

Tuli *et al*. (1992) made histochemical studies of intestines in cestodiasis of poultry wherein Alcian blue periodic acid Schiff (AB PAS) stained sections showed increased alcian blue positive material near the parasitic infestation in the mucosa indicating degenerative and necrotic changes. Bromophenol blue stained sections showed depletion of basic proteins in the cytoplasm of the epithelial cells.

Grencis (1997) proved that expulsion of intestinal parasites is temporally associated with an increase in the number of mast cells in the intestine and

secretion of mast cell proteases and leucotrienes into the tissues and serum.

Dezfuli (2010) studied the response of intestinal mucous cells to the presence of enteric helminthes and their distribution in fish. He found that the number of mucous cells close to the site of parasite attachment within the intestine was significantly higher than the number detected in uninfected fishes and in infected individuals at sites 1 cm or greater from the point of parasite attachment. There were no significant differences between the numbers of mucous cells found at the latter two sites. It was observed that alcian blue and periodic acid-Schiff's staining of representative histological sections revealed a significant increase in the number of mucous cells staining positively for acid glycoconjugates compared to the number of cells found in the intestines of uninfected *Salmo trutta*.

2.4 Haemato-biochemical studies

Ackert and Wissseman (1946) performed certain experiments involving testing of parasitized and control fowls to test their tolerance to moderate infections of ascarids and tapeworms. The criteria for judging the tolerance included growth, blood sugar level and haemoglobin content of parasitized fowls comparable to those of the control chickens. The criteria for the tapeworm tests included also differential blood counts. They concluded that chickens of 23 days age were able to tolerate infections of from one to 46 *Ascaridia galli* worms without manifesting definite harm from the worms in four weeks. Similarly growing chickens parasitized at 40 days of age were not significantly harmed by infections of from two to 172 *Raillietina cesticillus* tapeworms during a period of eight weeks.

Ramaorao and Cohly (1953) carried out serial studies on chicks infected with *Plasmodium gallinaceum* and showed a fall in total protein concentration. A loss of albumin together with no increase in total globulin contents was demonstrated.

Berghen (1966) studied the influences of *Capillaria obsignata* infection

on serum constitution of two species of birds viz. chicks and pigeons and revealed that infected pigeons showed a marked decrease in total proteins and albumin content, while globulins evidenced small changes (not significant). In contrast infected chicks showed elevated total protein levels. While albumin fractions were relatively constant, total globulins was considerably higher than in control animals.

Bhowmik *et al*. (1982) conducted studies on the pathobiology of chicks experimentally infected with *Raillietina cesticillus*, and noted a significant increases in heterophills and eosinophils at the early stage followed by progressive decrease of heterophills and a concomitant increase of lymphocytes at the latter stage of infection. They also recorded a significant hypoglycemia and hypoproteinaemia in the infected birds.

Sharma *et al*. (1984) carried out the blood cellular and biochemical studies in chicken experimentally infected with *Toxocara canis*. They reported decreased Hb, PCV, corpuscular volume, corpuscular hemoglobin concentration, total serum protein, heterophills, serum albumin and albumin: globulin ratio; increased total leukocyte count, erythrocytic sedimentation rate, esonophills, monocytes, lymphocytes, serum globulin and mean corpuscular haemoglobulin.

Samad *et al*. (1986) found that blood profile of the domestic fowls infected with *Raillietina echinobothrida* revealed anemia with a significant increase of total leukocyte counts and decrease of total serum protein.

Sekhar *et al*. (1986) evaluated blood hemoglobin levels in chickens naturally infected with *Raillietina* spp*, Choanotaenia infundibulum, Hymenolepis carioca, Cotugnia digonopora and Ascaridia galli*. They observed that in cockerels, single infection with *Raillietina echinobothrida*, *Raillietina cesticillus* and *Choanotaenia infundibulum* significantly decreased Hb levels whereas Hb levels were significantly increased by *Raillietina tetragona* and *Hymenolepis carioca*. They also reported that multiple infections tended to decrease Hb levels.

In infected pullets, in general, infection was associated with reduced Hb levels except in case of *Raillietina cesticillus* infection.

Ramadan *et al.* (1991) studied pathological and biochemical alterations following experimental infection with *Ascaridia galli* in chickens and observed a decrease in the level of glycogen and protein in muscles. The author related the observation to the parasite's capacity to survive on host metabolites.

Verma *et al*. (1993) studied the biochemical changes in chicken experimentally infected with *Ascaridia galli* and found that Hb, PCV, TEC, DLC, serum proteins, albumin, globulin and albumin:globulin showed no significant change. However TLC, eosinophils and monocyte counts and levels of alkaline phosphates increased in the infected and untreated birds. The values, however, returned to normal after treatment.

Phukan (2006) reported that the chicken infected with *Heterakis gallinarum* revealed decrease in Total Erythrocyte Count, Haemoglobin Concentration, Packed Cell Volume and Erythrocyte Sedimentation Rate with an increased Total Leukocyte Count, Lymphocyte and Eosinophil counts.

Deka and Borah (2008) studied haemato-biochemical changes in Japanese quails and chickens due to *Ascaridia galli* infection and found that PCV, TEC and Hb showed a significant decrease while as TLC showed a significant increase in the infected groups of quails and chickens. Biochemical study showed that total serum protein and serum albumin (A) levels decreased significantly in all the infected groups. Serum globulin (G) and A/G ratio failed to show any significant difference between control and infected groups of quails and chickens.

CHAPTER – 3

MATERIALS AND METHODS

3.1 Study area and Sampling

The bird population under study comprised of free domestic fowls in Ganderbal District of Kashmir valley. Ganderbal is a newly formed district of the state of Jammu and Kashmir. It is located at 34°14′N 74°47′E / 34.23°N 74.78°E. It has an average elevation of 1,619 metres (5,312 ft.) above mean sea level. The district was divided into 3 strata representing only 3 Tehsils namely Ganderbal, Lar and Kangan of the said District. The sampling included randomly selected birds from different villages of all the 3 strata (stratified random sampling). For sampling purposes the birds were categorized into growers aged 12-24 weeks and adults aged 32 weeks or above. Growers were those chickens that were old enough to fend for themselves but had not started reproducing, whereas adults included cocks that were mating and hens that had had at least one clutch of chicks.

All birds in the study area were housed in the backyard. The feed (cereals) was supplemented in the morning and in the evening. The remaining time of the day the chickens were allowed to scavenge freely at the homestead and the area around where they picked up feed like insects, arthropods, earthworms, different larvae, grasses, leaves, various grains etc. Besides all farmers provided, household wastes like table and kitchen remains to their birds. No preventive treatment was recorded to be done by the investigated farmers.

3.2 Clinical examination

Thorough clinical examination of the birds included in the study was performed before slaughter in order to detect any clinical signs of disease. The volume of the pectoral muscle was determined on palpation and body condition of the birds was scored as low, medium and good (Fig. 1).

1	Low body condition	**(-)**	Highly prominent keel bone with wasting of breast muscle mass.
2	Medium body condition	**(+)**	Prominent keel bone with reduced breast muscle mass.
3	Good body condition	**(++)**	Low palpable keel bone with full breast muscle mass.

3.3 Clinical samples

Blood samples were collected by veinupuncture directly from the alar vein (wing vein), with the help of disposable syringe using 22 gauge needles in sterile vials containing sodium salt of ethylinediaminetetraaceticacid (EDTA) (@2 mg/ml blood). 2 and ½ ml of blood was collected. One ml of blood was preserved for haematology in vials whereas one and a half ml of blood was allowed to coagulate for separation of serum to study serum chemistry. Sera was harvested by keeping the test tubes containing fresh blood in slanting position over night at room temperature and then stored at -20^{o}C until further use.

3.4 Haematobiochemical studies

All the blood samples were analysed for hemoglobin (Hb), Packed Cell Volume (PCV), Erythrocyte Sedimentation Rate (ESR), Total Erythrocyte Count (TEC), Total Leukocyte Count (TLC) and Differential Leukocyte Count (DLC). Hb estimation was done by Sahli's acid haematin method, PCV and ESR by wintrobe tube method (Bernard *et al*., 2000). TEC and TLC were done using Neubaur's hemocytometer and Toluidine blue (0.015%) saline as diluent (Brar *et al*., 2002). The blood films were stained with Wright's stain (Benjamin, 1985) for DLC.

Serum samples were analysed for total protein (TP), albumin (A), globulin (G) and A/G ratio.

Total protein content of the plasma of birds was estimated by the Biuret

method (Commercially available kit from Aspen Laboratories). The reagents were pipetted into dry clean test tubes labeled as blank (B), standard (S) and test (T) as under :

Reagent	Blank	Standard	Test
Total protein reagent	3.0 ml	3.0 ml	3.0 ml
Distilled water	50 µl	-	-
Total protein standard (6 mg/dl)	-	50 µl	-
Sample Plasma	-	-	50 µl

The tubes were incubated at 30°C for 10 minutes and the optical density of all the tubes was measured at 540nm against blank adjusted to zero and total protein value of the sample was calculated by

$$\text{Total proteins} = \frac{\text{O.D Test}}{\text{O.D Std.}} \times 6.0 mg / dl$$

The plasma albumin was estimated by the BCG method. Three test tubes were selected and labeled as blank, standard and test, then the reagents were pipetted into the test tubes as shown under

Reagents	Blank	Standard	Test
Albumin reagent	4 ml	4 ml	4 ml
Distilled water	0.02 ml	-	-
Albumin Std. (4 mg/dl)	-	0.02 ml	-
Sample Plasma	-	-	0.02 ml

The tubes were mixed well and allowed to stand for 5 minutes at room temperature. The absorbance of all the tubes was measured at 630 nm against blank. The total albumin content of the sample was given by

$$\text{Plasma albumin} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard.}} \times 4 mg/dl$$

Globulin was simply estimated by subtracting total protein value with the total albumin value.

3.5 Post mortem and parasitological examination

Post-mortem examination was performed according to Fowler (1996). After slaughtering the birds by halal method, the trachea, abdominal and thoracic cavities were opened, followed by systematic necropsy examination which included oesophagus to the gizzard, the small intestine (duodenum, jejunum and ileum), the caeca, the ileocaeca-colic junction to the cloaca and the oviduct. Each section of the alimentary tract was then cut and placed in a separate labeled Petri-dish, slit open and the contents washed gently into a Petri-dish. The mucosal scrapings were also added to the contents of respective petridishes to collect the helminthes embedded in the mucosal layer. The serosal surface of the proventriculi was examined carefully for the presence of embedded *Tetrameres* spp. The keratinised layer of the gizzard was removed for detecting the presence of *Acuaria hamulosa*. The parasites were collected from petri dishes by repeatedly washing the contents with water and decanting the supernatant after sedimentation. The collected parasites were kept in saline till they were fixed. All the helminths were counted before being fixed in 70% ethanol for further identification (Soulsby, 1982).

3.5.1 Fixation and preservation of parasites

Nematodes, Cestodes and trematodes were fixed in warm 30% alcohol and then preserved in 70% alcohol.

3.5.2 Processing and mounting of parasites

Temporary mounts were made in case of nematodes. The nematodes were dehydrated in ascending grades of alcohol (70%, 90% and absolute). After

dehydration parasites were cleared in lactophenol and mounted in Canada balsum or Dextrin Plasticized Xylene (DPX).

Permanent mounts were made in case of trematode and cestodes. In case of cestodes scolex, few immature, mature and gravid segments were used for making permanent mounts.

For making the permanent mounts of the cestodes and trematodes, the specimens preserved in 70% alcohol were first hydrated by passing them through the descending series of alcoholic grades viz. 50 and 30% and were then washed in distilled water. After washing, the specimens were stained with Bullough's acetoalum carmine stain (Gray, 1954). The specimens were kept overnight in dilute stain (1 part of acetoalum carmine in 10 parts of distilled water) to get the specimens slightly over stained, which helped in controlled differentiation by destaining them in acid water (1%) to clear out the various organs. Destaining was stopped when the boundaries of internal organs were seen. After destaining, the specimens were again washed in the distilled water and then passed through the ascending series of alcohols i.e. 30, 50, 70, 90 and 100%. After dehydration the specimens were transferred to clove oil for clearing. The cleared specimens were then mounted in Canada balsum or DPX. A few parasites were also stained with Borax carmine by the same method as described above.

All parasites were identified using the helminthological keys of Chabaud (1978), Anderson and Bain (1982), Soulsby (1982), Calnek *et al.* (1991), Anderson (1992) or Khalil *et al*. (1994).

3.6 Pathology

3.6.1 Gross pathology

The affected parts of the gastrointestinal tract were examined for any gross alterations associated with the parasitism. Gross lesions, if any, were recorded.

3.6.2 Histopathology

For studying the pathological effects of the parasites on the host tissue, the infected tissues (oesophagus, crop, proventriculus, gizzard, duodenum, jejunum, ileum, caecum, cloaca, bursa and oviduct) with *in-situ* parasites or with parasitic lesions were collected and preserved in 10% formalin. The tissue after fixation were cut in pieces of 2-3 mm thickness and washed for several hours before subjecting to dehydration in ascending grades of alcohol. Clearing of tissues was performed in benzene, embedding was done in paraffin. Sections of 5µm thickness were cut and stained with routine Harri's Haematoxilin and eosin method (Luna, 1968).

3.7 Histochemistry

Parallel tissue sections were specially stained for following histochemical observations

3.7.1 Demonstration of Iron by pearls stain (Luna, 1968)

Sections were immersed in 10% potassium ferrocyanide solution for 5 minutes and then in 10% postassium ferrocyanide and 10% HCL (7:3 ratio) for 20 minutes. Slides were rinsed in distilled water and counterstained with saturated solution of picric acid for 15 minutes. Dehydrated slides were cleaned in xylene (2 changes) and mounted in DPX. The results of the staining were recorded as iron-detectable and only siderocytes detectable.

3.7.2 Demonstration of Acid and Neutral Mucin by Combined Alcian Blue PAS stain (Bancroft and Gamble, 2002)

After dewaxing, sections were brought to water and put in alcian blue for 5 minutes. Slides were washed in water and then distilled water and then put in aqueous periodic acid for 5 minutes. After rinsing well in distilled water slides were put in Schiff's reagent for 15 minutes and then washed in running tap water for 5-10 minutes. Nuclei were lightly stained with usual Haematoxylin solution, differentiated as appropriate, blued and washed in water. Washed slides were

rinsed in absolute alcohol, cleared in xylene and mounted in DPX. Results of staining were recorded as acid mucin if blue coloured, neutral mucin if magenta coloured or mixture of these two.

3.7.3 Demonstration of mast cells by Toluidine blue (Bancroft and Gamble, 2002)

Paraffin sections, prepared from tissue fixed for 3 hrs. in Carnoy's fluid (and floated out on alcohol), were taken to alcohol, blotted dry and stained with toluidine blue solution for 10 seconds only. Stained sections were blotted dry, dehydrated, cleared and mounted in a DPX. Staining reactions of mast cells were recorded as purple.

3.8 Statistical analysis

The prevalence of the individual helminth species was calculated as a percentage of the host population that was infected with a specific parasite at a point in time (Thrushfield, 1995). The data collected was statistically analysed using standard statistical procedures (Snedecor and Cochran, 1967).ecor and Cochran (1968).

CHAPTER – 4

EXPERIMENTAL FINDINGS

4.1 Parasitofauna recovered

Gastrointestinal helminth parasites recovered from the domestic fowl included five species of nematodes, five species of cestodes and a single species of trematode.

4.1.1 Nematodes

The nematodes recovered belonged to four different generas and were identified as

4.1.1.1 *Heterakis gallinarum*

This parasite occurred in the caeca of the fowl. These were small white worms 7-13 mm in length in male and 10-17 mm in length in female (Fig. 2). Mouth was surrounded by three small lips (Fig. 3), oesophagus ended in a well developed posterior bulb (Fig. 4). In the male the tail was straight, preanal sucker well developed and spicules were dissimilar (Fig. 5). Tail end of the female was long, narrow and pointed (Fig. 6).

4.1.1.2 *Ascaridia galli*

It was recovered from the small intestine. These were whitish yellow worms, males measured 5-7.5 cm in length while as females measured 7-11.5 cm (Fig. 7). Anteriorly there were three large lips around the mouth and oesophagus had no posterior bulb (Fig. 8). Tail end of the male had weakly developed caudal alae and anterior to the cloaca there was a precloacal sucker with a thick cuticular rim and the spicules were subequal (Fig. 9).

4.1.1.3 *Capillaria annulata*

This species was recovered from crop, oesophagus and proventriculus. Males were 15-25 mm and females 30-80 mm long. A characteristic swelling was

Fig.1 Dressed domestic fowl showing good, medium and low body conditions of domestic fowls.

Fig. 2 *Heterakis gallinarum* worms recovered from the caecum of domestic fowl.

Fig. 3 *Heterakis gallinarum* revealing three anterior lips. X620.

Fig. 1

Fig. 2

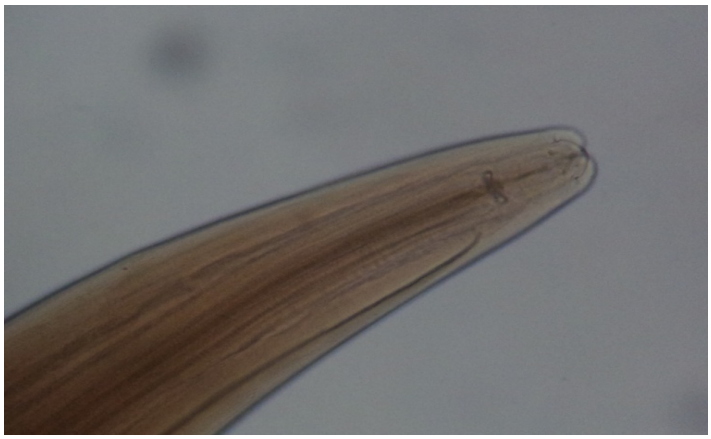

Fig. 3

Fig. 4 ***Heterakis gallinarum*** **revealing characteristic posterior oesophageal bulb. X350.**

Fig. 5 Tail end of the male ***Heterakis gallinarum*****. X350.**

Fig. 6 Narrow pointed tail end of the female ***Heterakis gallinarum*****. X350.**

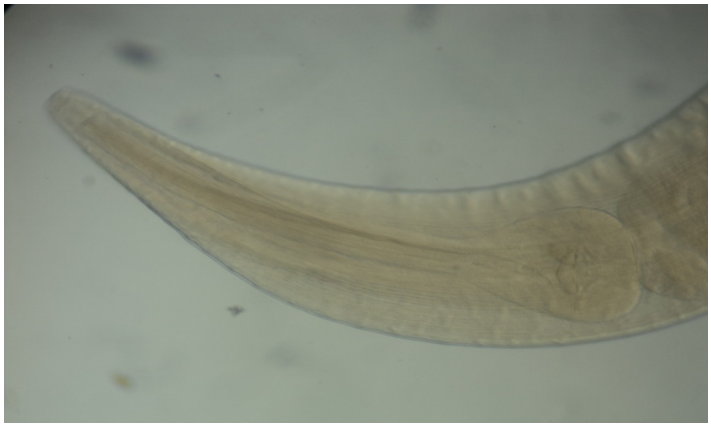

Fig. 4

Fig. 5

Fig. 6

Fig. 7 ***Ascaridia galli*** **recovered from domestic fowl.**

Fig. 8 ***Ascaridia galli*** **revealing three large anterior lips and absence of oesophageal bulb. X350.**

Fig. 9 Tail end of the male ***Ascaridia galli*** **revealing precloacal sucker and subequal spicules. X350**

Fig. 7

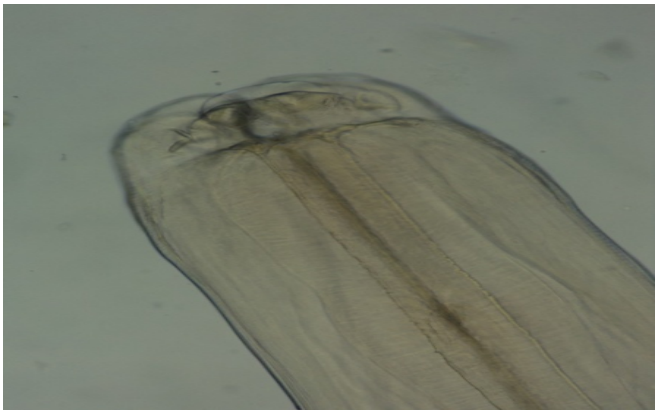

Fig. 8

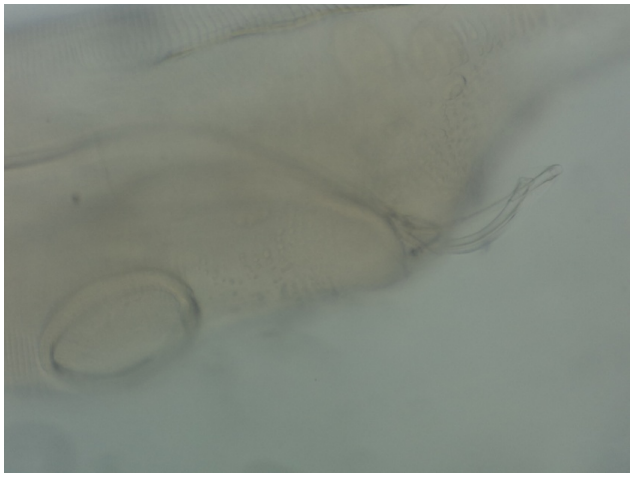

Fig. 9

present behind the head that was formed by the cuticle of the anterior end (Fig. 10). The eggs measured 60-65 by 25-30 micrometers with two protruding polar plugs one at either end (Fig. 11).

4.1.1.4 *Acuaria hamulosa*

Reddish worms were found embedded in the musculature of gizzard forming soft yellowish nodules (Fig. 12). Males were 12-14mm and females 16-25mm in length. This parasite had four cuticular cordons which were non-recurrent and non- anastamosing, double irregularly wavy and extended to the posterior end of the body (Fig. 13). Posterior end was pointed (Fig. 14).

4.1.1.5 *Dysphrynx (Acuaria) spiralis*

A spirurid was found to inhabit the wall of the proventriculus. Males measured 7-8 mm and females measured 9-10mm in length. The four cuticular cordons had a sinous course, were recurrent and did not anastamose (Fig. 15 and 16).

4.1.2 Cestodes

The cestodes recovered from the domestic fowl belonged to four different genera and were identified as

4.1.2.1 *Raillietina cesticillu*s

The parasite was found in the duodenum and jejunum. Length of the parasite was up to 12cm (Fig. 17). Suckers were unarmed and the tapeworm was readily recognized by the broad rostellum armed with large number of minute hooks (Fig. 18 and 19). Genital organs were found to alternate irregularly (Fig. 20) and eggs were present singly in egg capsules (Fig. 21).

4.1.2.2 *Hymenolepis carioca*

Large number of these worms was present especially in the duodenum part of the intestine. Parasites were 3-8cm in length and 0.5-1mm in width (Fig. 22) and possessed unarmed rostellum (Fig. 23). Gravid uterus lied transversely (Fig.

Fig. 10 ***Capillaria annulata*** **revealing a characteristic swelling behind the head. X2400.**

Fig. 11 Eggs of ***Capillaria annulata*** **. X2400.**

Fig. 12 ***Acuaria hamulosa*** **worms recovered from the nodules of gizzard**

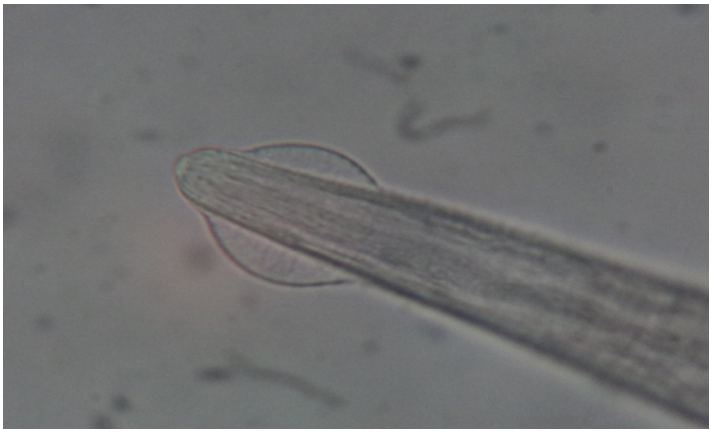

Fig. 10

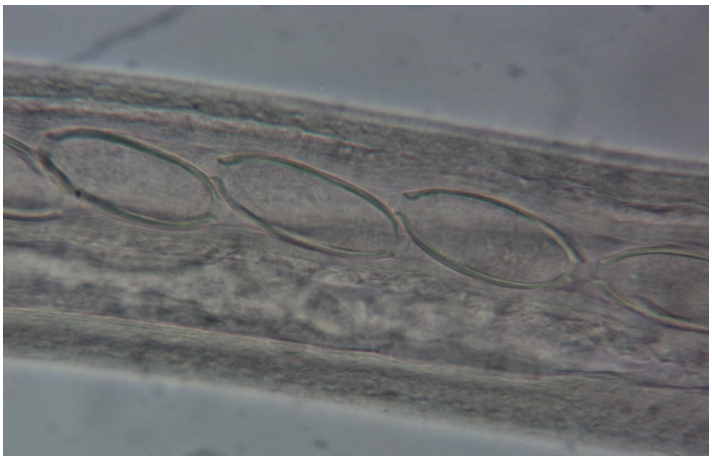

Fig. 11

Fig. 12

Fig. 13 Anterior end of *Acuaria hamulosa* revealing cuticular cordons. X140.

Fig. 14 Posterior end of *Acuaria hamulosa*. X140.

Fig. 15 Anterior end of *Dysphrynx (Acuaria) spiralis* revealing cuticular cordons. X350.

Fig. 13

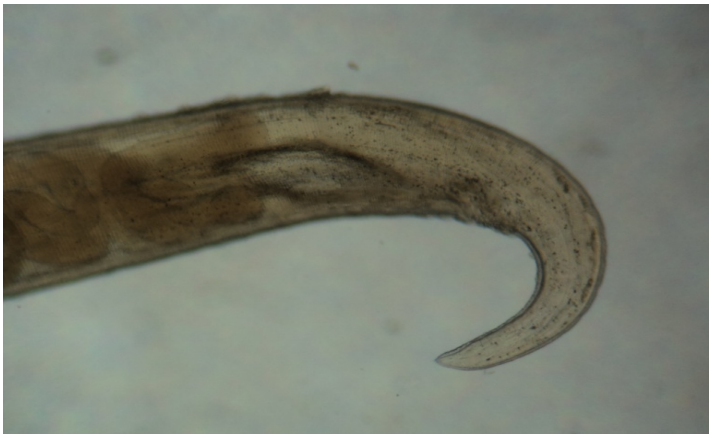

Fig. 14

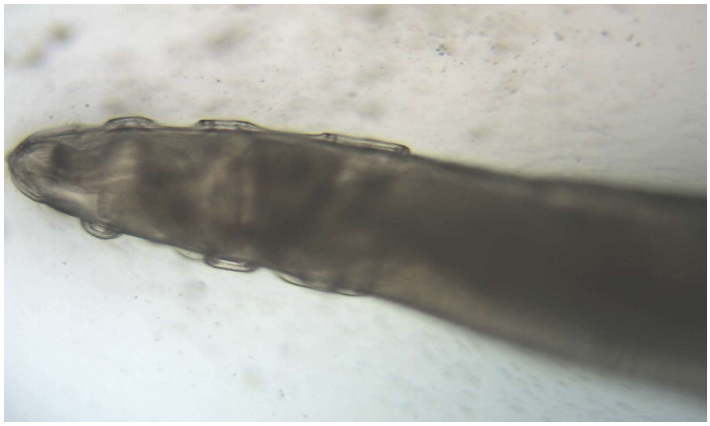

Fig. 15

Fig. 16 Anterior end of *Dysphrynx (Acuaria) spiralis* revealing recurrent cuticular cordons. X350.

Fig. 17 *Raillietina cesticillu*s recovered from the intestine domestic fowl.

Fig. 18 Anterior end of *Raillietina cesticillu*s revealing scolex with inconspicuous unarmed suckers and armed rostellum. X350.

Fig. 16

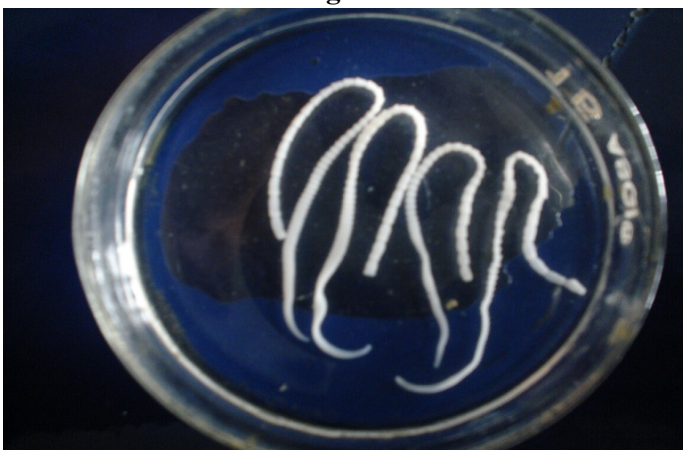

Fig. 17

Fig. 18

Fig. 19 **Anterior end of *Raillietina cesticillu*s revealing scolex with inconspicuous unarmed suckers and armed rostellum. Borax carmine. X350.**

Fig. 20 ***Raillietina cesticillu*s revealing mature segments containing irregularly alternating genital organs. Borax caramine. X350.**

Fig. 21 ***Raillietina cesticillu*s revealing gravid segments containing egg capsules. Borax carmine. X350.**

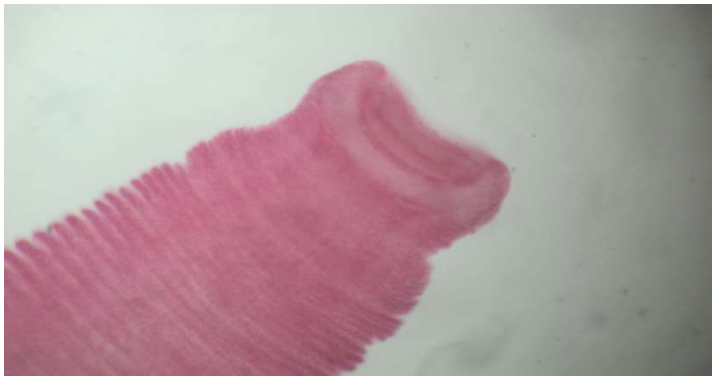

Fig. 19

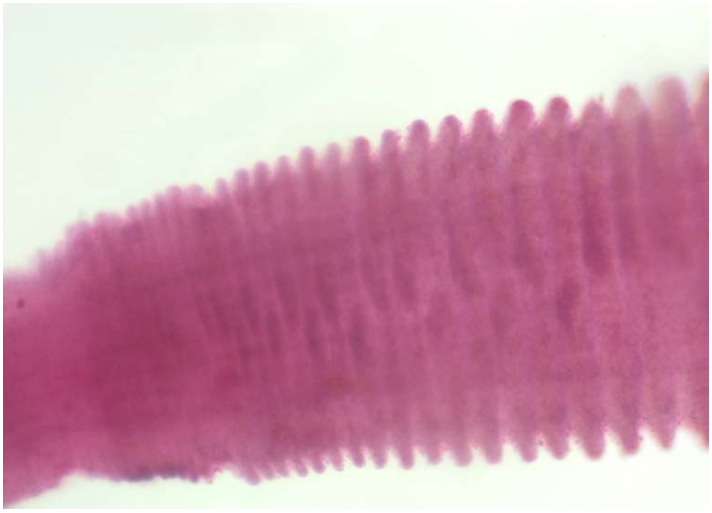

Fig. 20

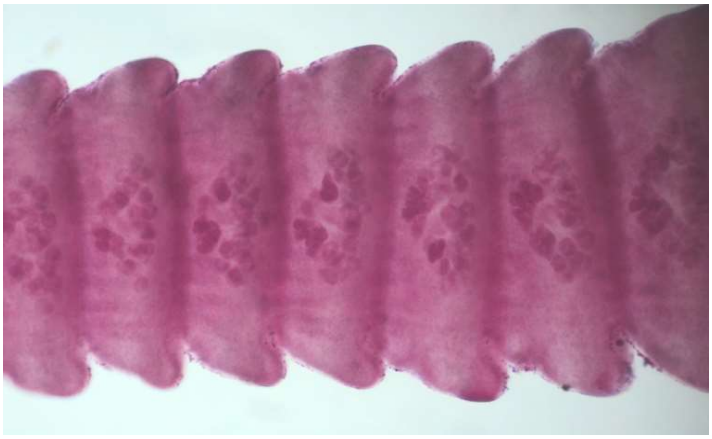

Fig. 21

Fig. 22 ***Hymenolepis carioca*** **worms recovered from the intestine domestic fowl.**

Fig. 23 **Scolex of** ***Hymenolepis carioca*** **revealing unarmed rostellum. Aceto carmine. X350**

Fig. 24 **Segments of** ***Hymenolepis carioca*** **revealing transversely lying uterus. Alcoholic Borax caramine. X350.**

Fig. 22

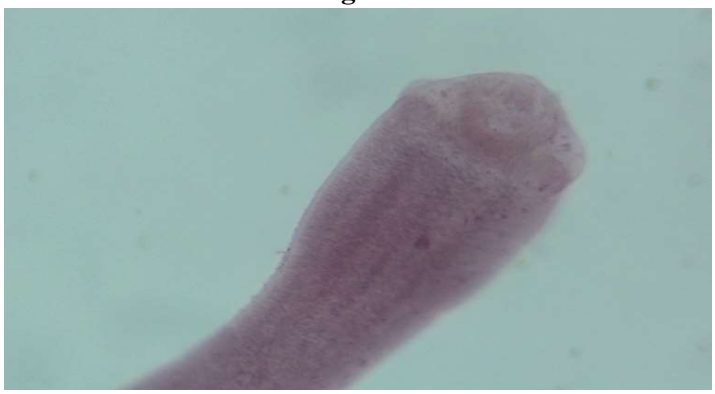

Fig. 23

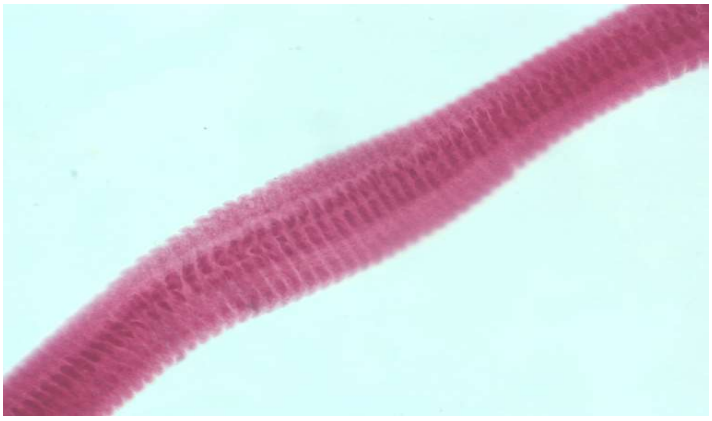

Fig. 24

24) and eggs were enclosed in three membranes.

4.1.2.3 *Raillietina tetragona*

This parasite measured up to 25cm in length (Fig. 25). The scolex had a distinct neck and both rostellum and suckers were armed with small hooks (Fig. 26). The genital pores were usually unilateral (Fig. 27) and gravid segments contained 6-12 eggs.

4.1.2.4 *Amoebotaenia sphenoides*

It was isolated mostly from the duodenum. The size of this parasite was very small about 3mm in length and only one mm in width. It was roughly triangular in shape and possessed about twenty segments; the mature and gravid ones were much broader than the more anterior proglottids. Rostellum was armed with hooks and suckers were large and unarmed (Fig. 28 and 29).

4.1.2.5 *Choanotaenia infundibulum*

It was much larger worm up to 20cm in length (Fig. 30). The suckers were unarmed and rostellum armed (Fig. 31). The segments were wider posteriorly giving parasite a serrated appearance (Fig. 32).

4.1.3 Trematodes

Only one trematode species was recovered from the domestic fowl which was identified as

4.1.3.1 *Prosthogonymus pellucidus*

It was recovered from *Bursa of Fabricius* and posterior intestine of the domestic fowl. It was upto 9 mm in length and 4mm broad (Fig. 33). Cuticle was spiny near the mid body. Ventral sucker was larger than the oral sucker (Fig. 34). Testes were oval lying horizontally, ovary was deeply lobed lying anterior to testes and vitellaria were present in the lateral fields (Fig. 35).

Fig. 25 ***Raillietina tetragona*** **worms recovered from the intestine domestic fowl.**

Fig. 26 ***Raillietina tetragona*** **revealing armed suckers and rostellum . Acetocarmine. X350.**

Fig. 27 Mature segments of ***Raillietina tetragona*** **revealing unilateral genital organs. Alcoholic Borax caramine. X350**

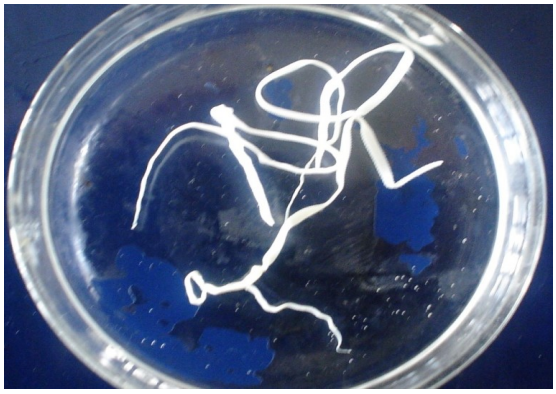

Fig. 25

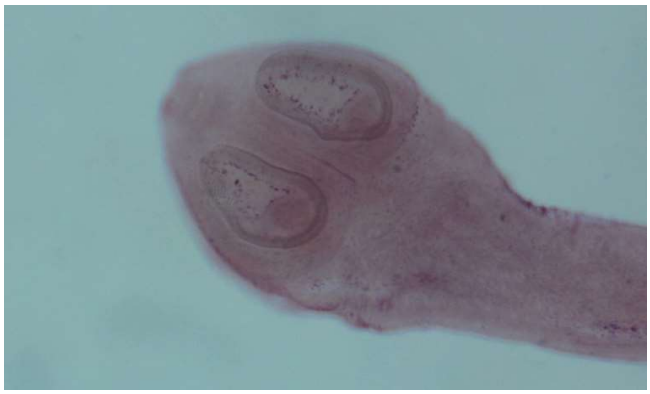

Fig. 26

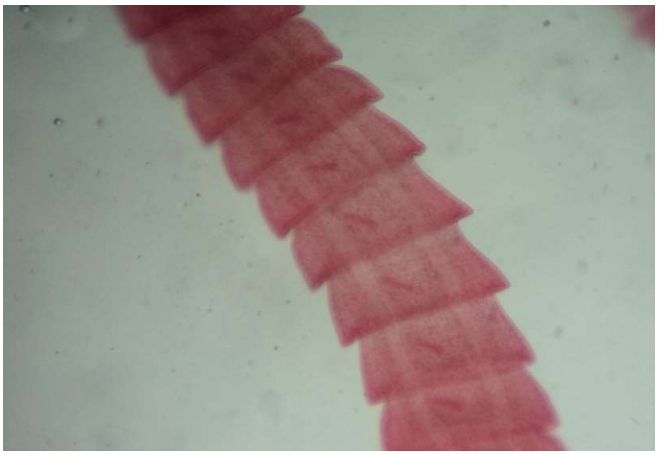

Fig. 27

Fig. 28 ***Amoebotaenia sphenoides*** **revealing armed rostellum and large unarmed hooks . X350.**

Fig. 29 ***Amoebotaenia sphenoides*** **revealing armed rostellum. Borax carmine. X350.**

Fig. 30 ***Choanotaenia infundibulum*** **recovered from the intestine of domestic fowl.**

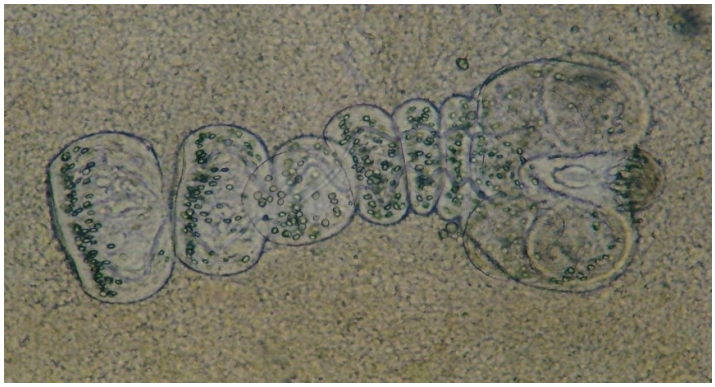

Fig. 28

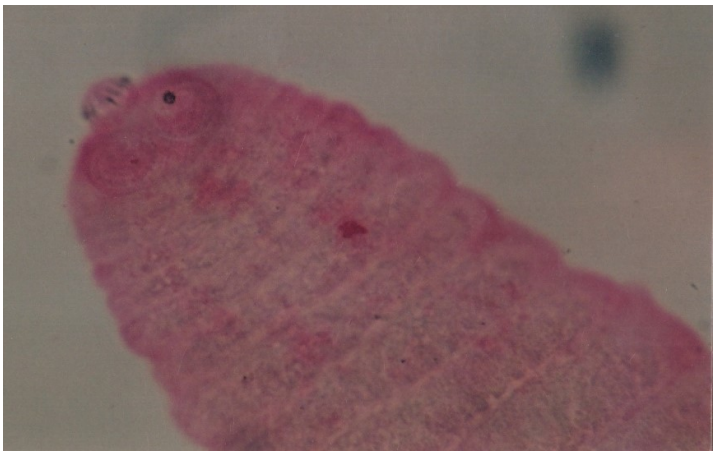

Fig. 29

Fig. 30

Fig. 31 ***Choanotaenia infundibulum*** **revealing scolex. Borax carmine. X350.**

Fig. 32 Posterior broad segments of ***Choanotaenia infundibulum*** **revealing serrated appearence. Borax carmine. X350.**

Fig. 31

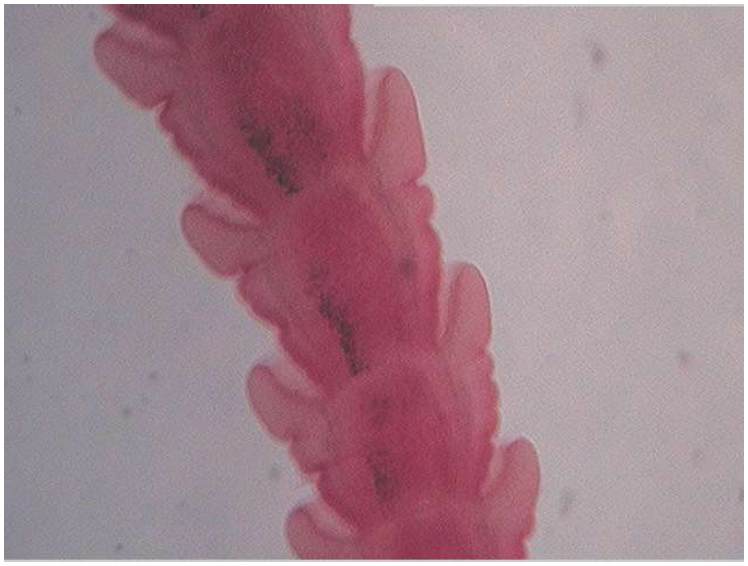

Fig. 32

Fig.33 ***Prosthogonymus pellucidus*** **recovered from the bursa of domestic fowl.**

Fig.34 ***Prosthogonymus pellucidus*** **revealing oral and ventral suckers. Borax carmine. X140.**

Fig. 35 ***Prosthogonymus pellucidus*** **revealing testes, ovary and vitellaria. Borax carmine. X140.**

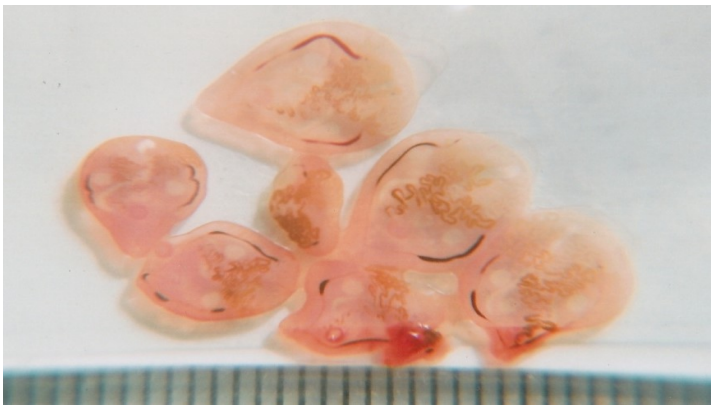

Fig. 33

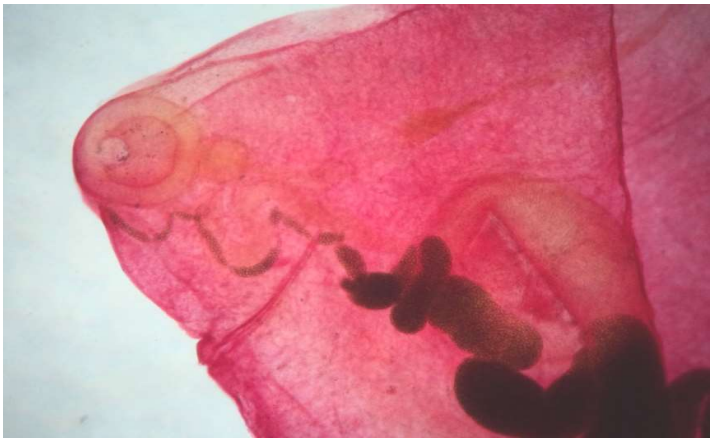

Fig. 34

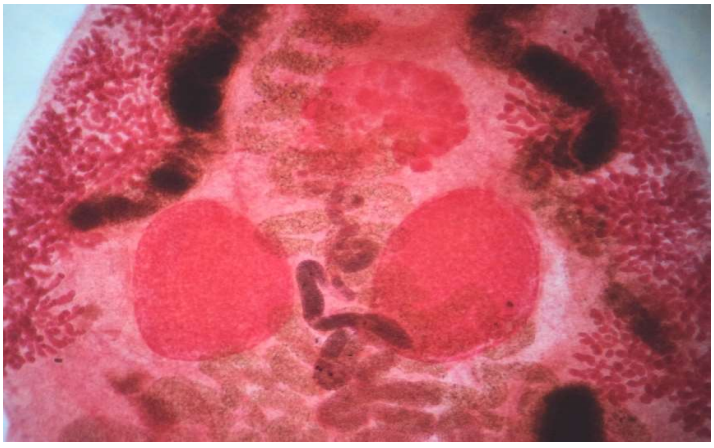

Fig. 35

4.2 Average worm burdens and pattern of helminthosis

The average worm burden per chicken was 68.4. Most of the birds were infected with multiple helminth species with a mean of 2.17 and a range of 0 to 5. The pattern of helminth infections observed in the domestic fowl is presented in Table 1.

Table 1: Pattern of helminth infections in domestic fowl of Ganderbal district

Name of the helminths	Total examined	Number positive	Percentage Parasitic infections
Nematode	100	40	40^{A}
Nematode + Cestode	100	38	38^{A}
Cestode	100	11	11^{AB}
Uninfected	100	6	6^{B}
Nematode + Trematode	100	3	3^{B}
Nematode +Cestode + Trematode	100	2	2^{B}
Trematode	100	0	0^{B}
Cestode + Trematode	100	0	0^{B}

(The number of positive bearing similar superscripts did not differ significantly)

In general pure infection of nematodes was 40 per cent followed by mixed infection of nematodes and cestodes (38%) and pure infection of cestodes (11%).

These three patterns of infection were non significantly varying among themselves but were varying significantly from other patterns of infection except in the case of pure infection of cestodes which falls in the middle of the patterns of infection.

4.3 Prevalence studies

Table-2 summarizes the prevalence of various gastrointestinal helminth parasites recovered from the domestic fowl in Ganderbal district of the Kashmir valley. In general out of a total of 100 birds screened, 94 per cent were found to be infected out of which 68 per cent were infected with more than one species of helminths.

Among the nematodes recovered from the fowl, *Heterakis gallinarum* was found to be the most prevalent nematode. Its prevalence of 63 per cent was found to be much higher than other four nematode parasites. After *Heterakis gallinarum,* next most prevalent nematode was found to be *Ascaridia galli* having an occurrence rate of 41 per cent. This was in turn followed by *Capillaria annulata, Acuaria hamulosa and Dysphrynx spiralis* whose respective prevalence rates were found to be 35, 6 and 1 per cent.

Among cestodes, *Raillietina cesticillus* was found to be most prevalent with a prevalence of 21 per cent. The next most prevalent cestode was found to be *Hymenolepsis carioca* (19%). *Raillietina tetragona* and *Amoebotaenia sphenoides* were found 10 and 8 per cent, respectively. However *Choanotaenia infundibulum* was found to be least prevalent (7%).

The prevalence of only trematode parasite *Prosthogoymus pellucidus* was found to be 5 per cent.

In general the prevalence of *Heterakis gallinarum* was found to vary non-significantly from *Ascaridia galli* and *Capillaria annulata* but significantly from all other parasites.

Table 2: Prevalence and intensity of gastrointestinal helminth infections in domestic fowls in Ganderbal district of the Kashmir valley (n=100)

Helminth species	No. of infected birds	Infection (%)	Parasite count (Range)	Mean
Heterakis gallinarum	63	63^{A}	4-312	67.34
Ascaridia galli	41	41^{AB}	1-26	10
Capillaria annulata	35	35^{ABC}	3-24	9.66
Raillietina cesticillus	21	21^{BCD}	1-80	22.80
Hymenolepis carioca	19	19^{BCD}	1000-3000	2000
Raillietina tetragona	10	10^{CD}	3-29	9.76
Amoebataenia sphenoides	8	8^{CD}	4-37	17.73
Choanotaenia infundibulum	7	7^{CD}	7-180	34
Acquaria hamulosa	6	6^{D}	1-7	3
Prosthogonymus pellucidus	5	5^{D}	5-9	6
Dyspharynx spiralis	1	1^{D}	5	5

4.4 Association of gastrointestinal helminthosis with body condition scoring

Out of a total of 100 birds screened, 19 were having low, 71 medium, and 10 good body condition. Assessment of different parasitic infections in association with body condition of the birds revealed that the low body condition of the domestic fowls was associated with more frequent and more severe parasitic

infections (Table 3). The occurrence of grossly visible abnormities of gastrointestinal tract was 43 per cent in low, followed by 26 per cent in medium and 20 per cent in good body condition birds.

In general all the three body conditions in domestic fowl were not significantly different with respect to the occurrence of the parasitic infections.

Table 3: Relationship between body condition score and parasitic infection in domestic fowl in Ganderbal district of Kashmir valley

Body condition		Total examined	Number positive	Percentage Parasitic infections
Low	(-)	19	19	100
Medium	(+)	71	67	94.36
Good	(++)	10	8	80
Total		100	94	94

(The mean difference is significant at the 0.05 level)

4.5 Age and sex-wise distribution of parasitism

Age and sex-wise distribution of parasitism is given in Table 4. The occurrence of parasitism was found to be 100 per cent in growers (12-24 weeks) and 90.32 per cent in adults (32 weeks or above). Furthermore, prevalence of some of the parasites was significantly higher in growers compared to adults. Females showed an occurrence of 94.5 per cent while as in males occurrence of 93.3 per cent was recorded which did not differ significantly.

Table 4: Age and sex-wise distribution of parasitism

	Age		Sex	
	Growers	**Adults**	**Male**	**Female**
No. screened	38	62	45	55
No. positive	38	56	42	52
%age	100 [A]	90.32 [A]	93.3 [a]	94.5 [a]

(The mean difference is significant at the 0.05 level)

4.6 Pathology

Majority of the birds were infected with more than one type of parasite. Thus the overall effect observed in birds could be attributed to the multiple parasitisms. However, on the basis of boring site predilection of the parasites, predominant lesions in a particular area could be associated to a given parasite. The various pathological changes caused by the trematodes, cestodes and the nematodes in the domestic fowl are presented.

4.6.1 Pathology of Nematodes

4.6.1.1 *Ascaridia galli*

Ascaridia galli were found to inhabit lumen of the intestine (Fig. 36). Gross changes observed depended on the parasite load. In most of the cases, low load of worms was observed and was not associated with any grossly observable lesions. Moderate infection was associated with mucous enteritis. The intestinal wall appeared to be thickened with mucosa giving a velvety appearance. Lumen contained thick white pasty mucous.

Histologically sections of the *Ascaridia galli* were found in the lumen of the intestine (Fig. 37). The histopathological changes revealed mild to moderate goblet cell hyperplasia (Fig. 38), compression of villi (Fig. 39), disruption of villi and desquamation of its epithelium (Fig. 40), broadening and clubbing of the villi (Fig. 41), focal necrosis, submucosal edema. Mild inflammatory reaction (Fig. 42)

Fig. 36 Intestine of Fowl revealing multiple *Ascaridia galli* worms.

Fig. 37 Section of *Ascaridia galli* in the lumen of intestine of domestic fowl. H&E. X280.

Fig. 38 Section of fowl intestine infected with *Ascaridia galli* revealing goblet cell hyperplasia. HE. X400.

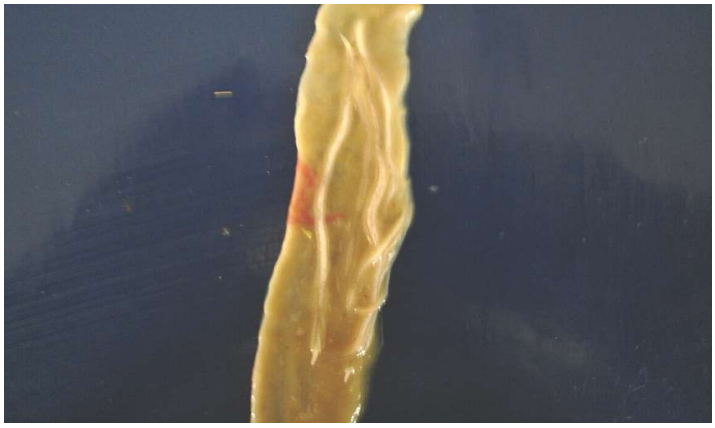

Fig. 36

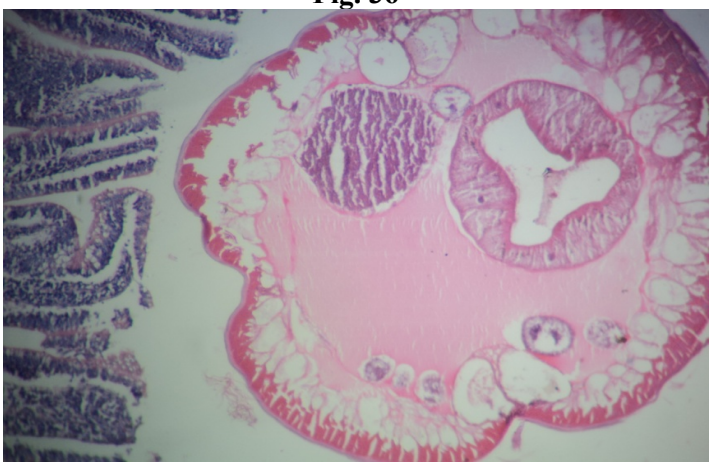

Fig. 37

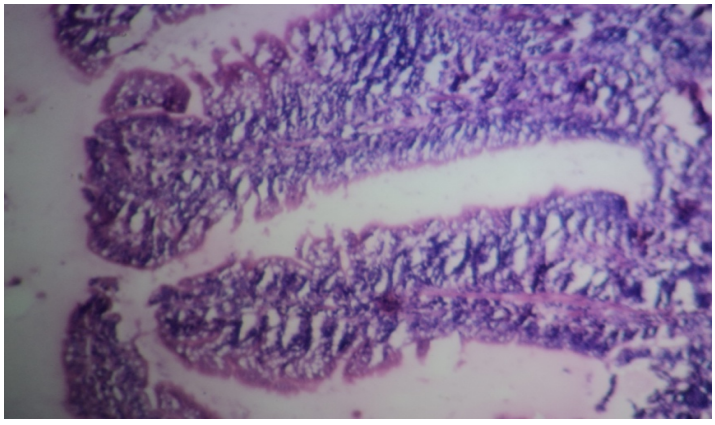

Fig. 38

Fig. 39 **Section of fowl intestine revealing *Ascaridia galli* causing compression of villi. HE. X330.**

Fig. 40 **Section of fowl intestine infected with *Ascaridia galli* revealing disruption of villi, desquamation of its epithelium and mild inflammatory reaction with infiltration of mononuclear cells. HE. X350.**

Fig. 41 **Section of fowl intestine infected with *Ascaridia galli* revealing broadening and clubbing of the villi. HE. X350.**

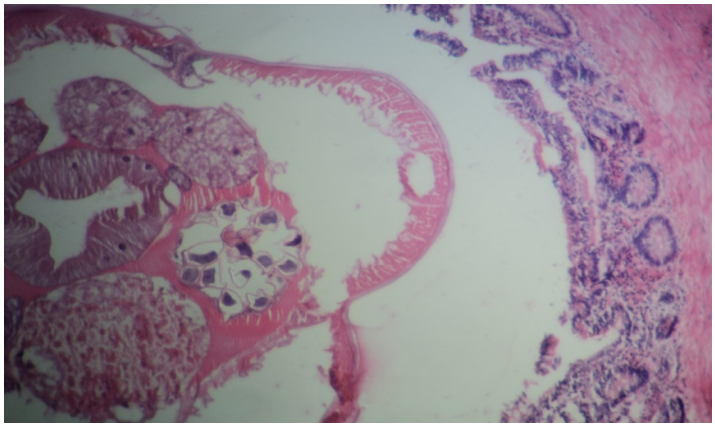

Fig. 39

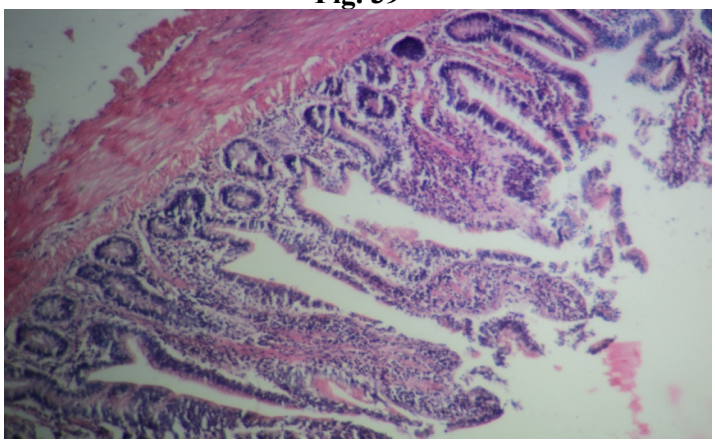

Fig. 40

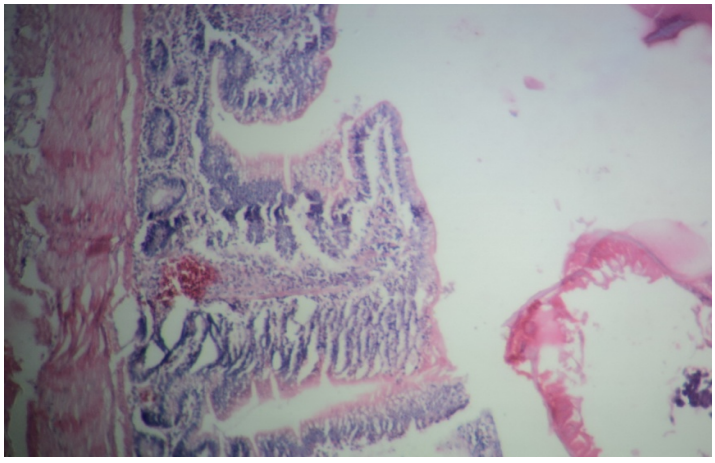

Fig. 41

was characterized mainly by infiltration of mononuclear cells and a few polymorphonuclear cells including eosinophils. Sections of the parasites with degenerated cuticles were also noticed in the lumen (Fig. 43).

4.6.1.2 *Heterakis gallinarum*

Heterakis gallinarum occurred in the lumen of caecum especially at the blind end (Fig. 44). The caecal wall was thickened and revealed petechial haemorrhages.

Histological findings in the caeca were represented by the presence of of *Heterakis gallinarum* worms in the lumen as well as in the wall of mucous membrane (Fig. 45), intense chronic diffuse typhlitis with mononuclear and polymorphonuclear (heterophils) leucocyte infiltrations (Fig. 46). Some of the stained sections showed extensive damage of the mucosal lining (Fig. 47). There were prominent erosions of the epithelial lining. Besides goblet cell hyperplasia (Fig. 48), edema of the caecal wall (Fig. 49), necrosis (Fig. 50) and preponderance of necrotic material in the caecal lumen (Fig. 51) were common findings.

4.6.1.3 *Capillaria annulata*

Capillaria annulata was found in the oesophagus and crop. In light infections the wall of the crop and oesophagus was slightly thickened and inflamed, but this extended to a marked thickening, inflammation with mucopurulent exudates in the lumen in severe infections. Large number of parasites was found in the mucosa and necrotic membranes.

Histological sections of the *Capillaria annulata* were found in the squamous epithelial layer of oesophagus and crop (Fig. 52 and 53). Histopathology revealed oesophagitis (Fig. 54) and ingluvitis (Fig. 55), sloughing of the mucosa containing parasitic ovas (Fig. 56), necrotic changes in squamous epithelium characterized by pyknotic nuclei (Fig. 57). A thin connective tissue capsule surrounded the adult worms and every where there were infiltrates of lymphocytes and mononuclears. Oesophageal glands were inflammed (Fig. 58)

Fig. 42 Section of fowl intestine infected with *Ascaridia galli* revealing mild inflammatory reaction. HE. X280.

Fig. 43 Section of fowl intestine revealing degenerated cuticle of *Ascaridia galli*. HE. X250.

Fig. 44 Caecum of fowl revealing multiple *Heterakis gallinarum* worms.

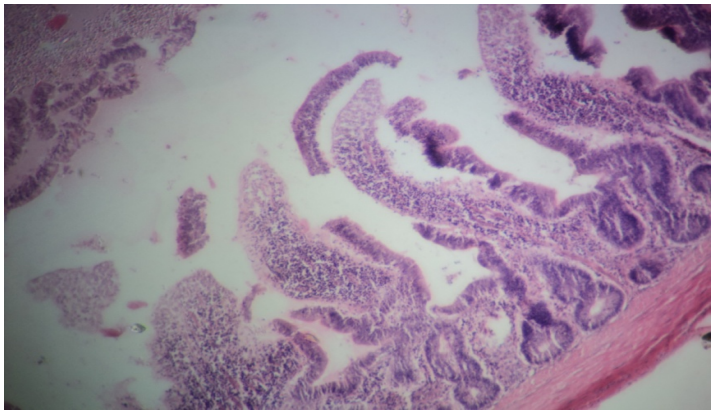

Fig. 42

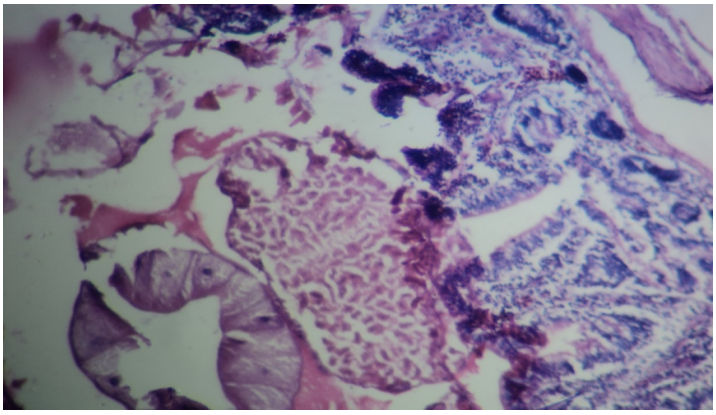

Fig. 43

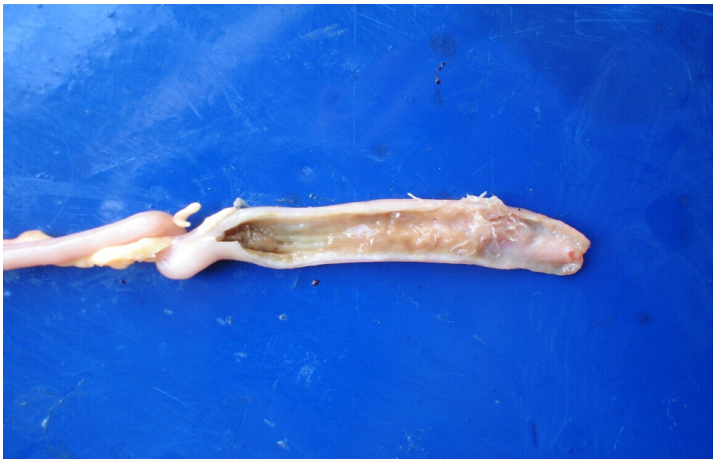

Fig. 44

Fig. 45 **Sections of *Heterakis gallinarum* in the caecum of domestic fowl. HE. X280.**

Fig. 46 **Section of fowl caecum infected with *Heterakis gallinarum* revealing typhlitis. HE. X2500.**

Fig. 47 **Section of fowl caecum infected with *Heterakis gallinarum* revealing desquamation of the mucous membrane. HE. X350.**

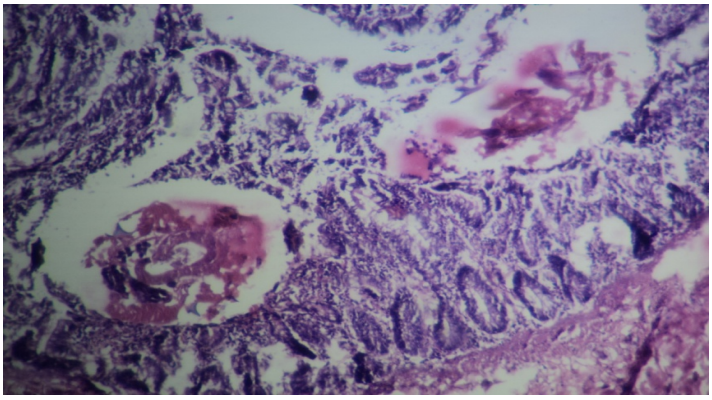

Fig. 45

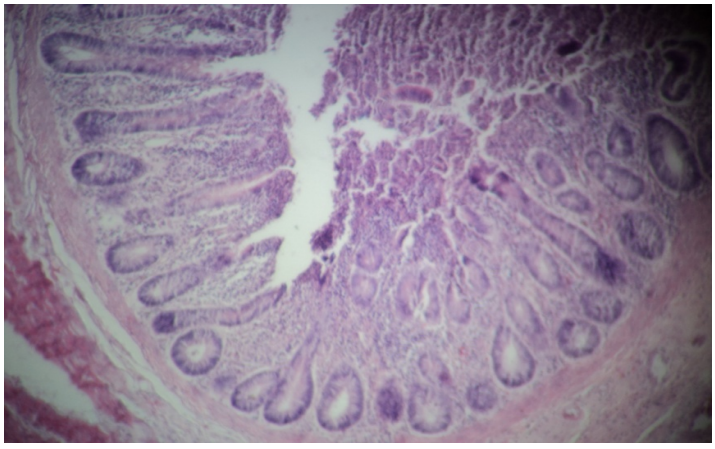

Fig. 46

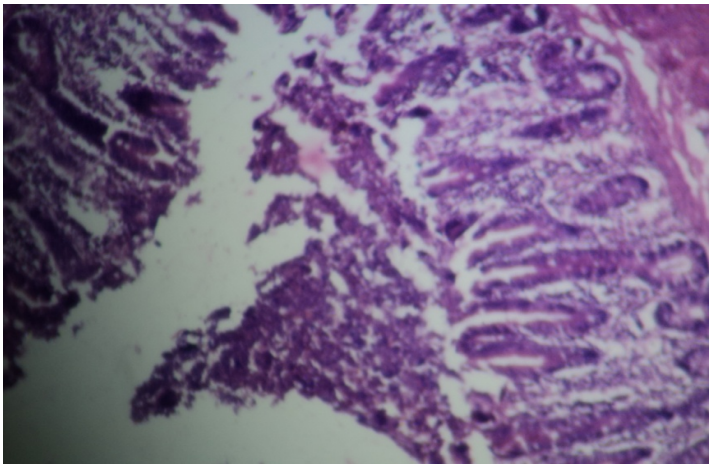

Fig. 47

Fig. 48 Section of fowl caecum infected with *Heterakis gallinarum* revealing goblet cell hyperplasia of the mucous membrane. HE. X350.

Fig. 49 Section of fowl caecum infected with *Heterakis gallinarum* revealing oedema of the caecal wall. HE. X400.

Fig. 50 Section of fowl caecum showing necrosis induced by *Heterakis gallinarum*. HE. X300

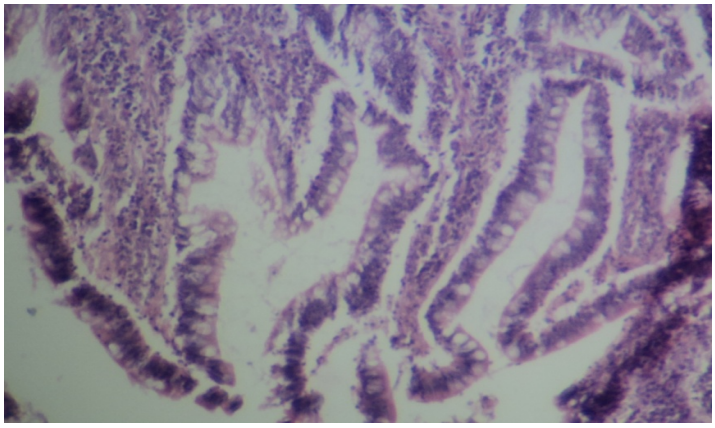

Fig. 48

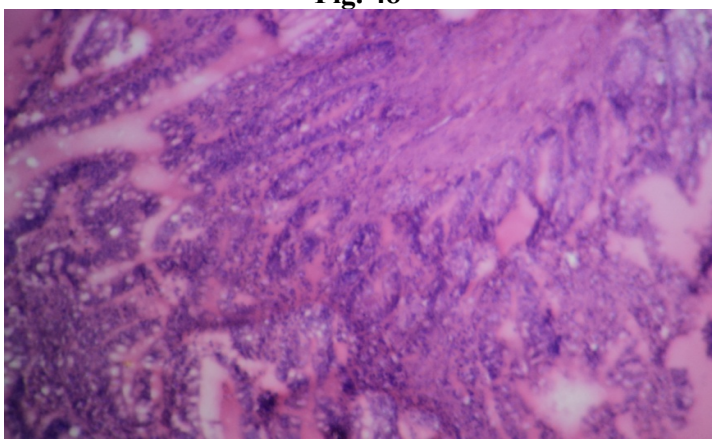

Fig. 49

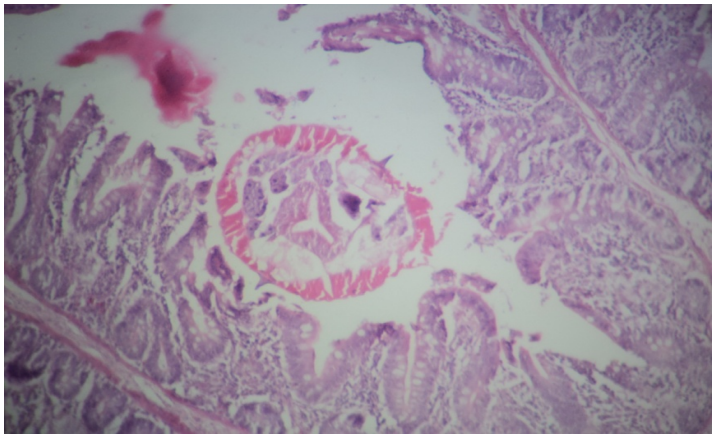

Fig. 50

Fig. 51 Section of fowl caecum infected with *Heterakis gallinarum* revealing necrotic material in the caecal lumen. HE. X400.

Fig. 52 Sections of *Capillaria annulata* in the oesophagus of domestic fowl. HE. X360.

Fig. 53 Sections of *Capillaria annulata* in the crop of domestic fowl. HE. X360.

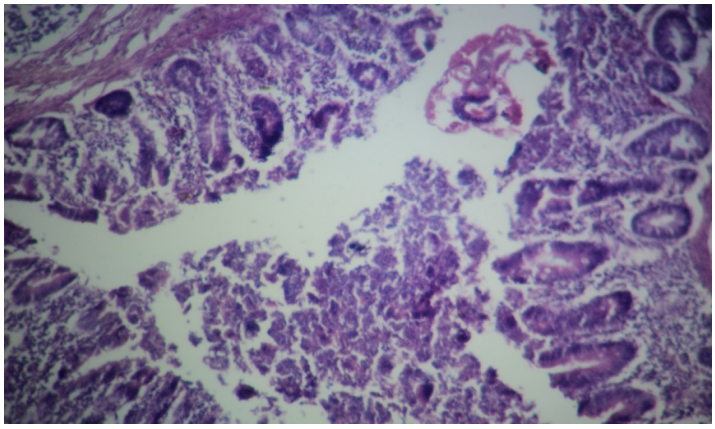

Fig. 51

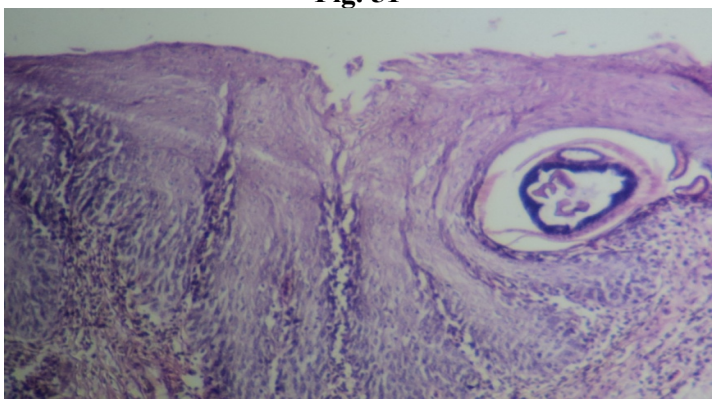

Fig. 52

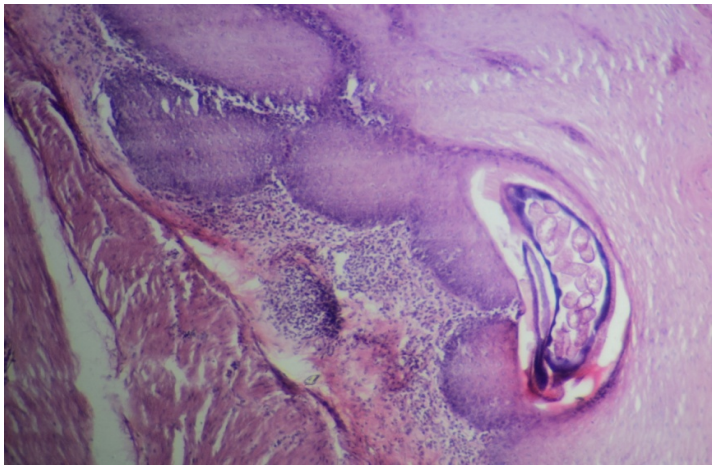

Fig. 53

Fig. 54 Section of fowl oesophagus infected with *Capillaria annulata* revealing oesophigitis. HE. X360.

Fig. 55 Section of fowl crop infected with *Capillaria annulata* revealing inflammatory changes in the submucosa (proctitis). HE. X300.

Fig. 56 Section of fowl crop infected with *Capillaria annulata* revealing desquamated material containing parasitic ovas. HE. X300.

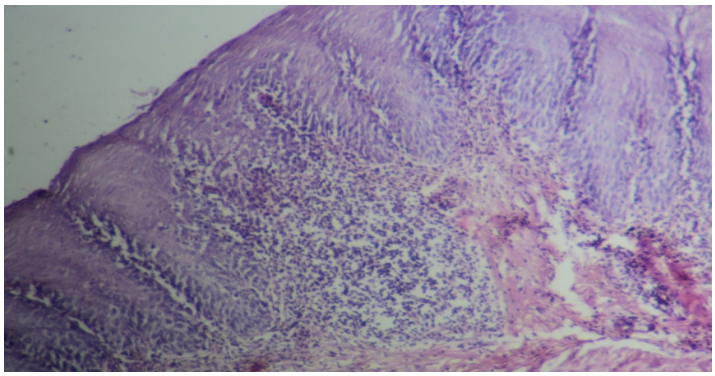

Fig. 54

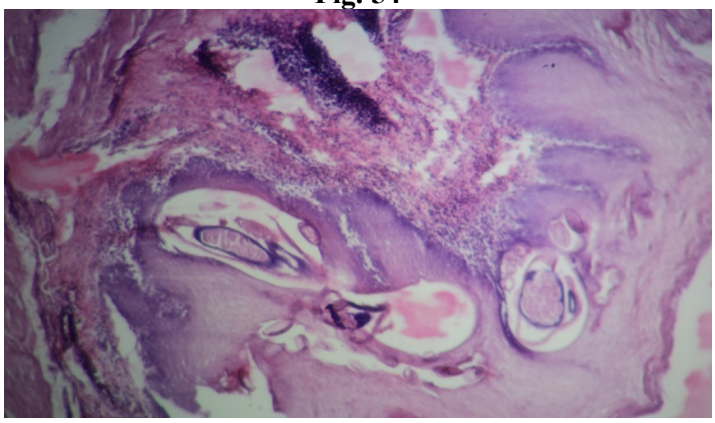

Fig. 55

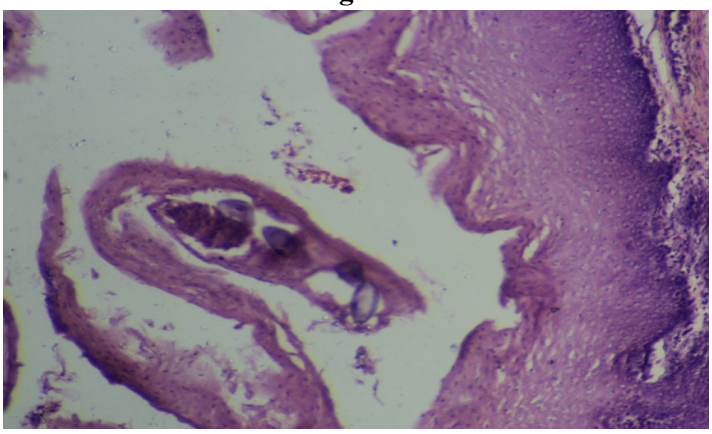

Fig. 56

and sometimes cystic (Fig. 59). In some sections acanthotic changes (Fig. 60) and enlargement of lymphoid follicles (Fig. 61) was observed. Proventricular capillariasis was characterized by proventriculitis (Fig. 62), desquamation of the epithelial cells into the common cavities of the lobules (Fig. 63) and cystic proventricular glands (Fig. 64).

4.6.1.4 *Acuaria hamulosa*

Acuaria hamulosa red coloured worms were found emerging from the gizzard muscular wall. The parasites could be detected after the removal of the horny layer koilin of the gizzard and cutting through the musculature (Fig. 65). Grossly the musculature of the caudal lobe of the gizzard revealed soft yellowish nodules. Lesions were found extending into the muscular portion of the organ.

Histopathology revealed discrete nodular lesions in the musculature which contained sections of the parasite (Fig. 66). Within the musculature compression of the muscle (Fig. 67), severe myositis, haemorrhages and necrosis were seen (Fig. 68). Mucosa showed severe inflammation and there was marked destruction of glands (Fig. 69). A chronic inflammatory reaction of granulomatous type (Fig. 70) characterized by presence of mononuclear cells and giant cells was observed. Fibroplasia and hyperplasia of peripheral lymphoid tissues (Fig. 71) was observed. Cellular reaction in the lesions was characterized by large number of lymphocytes, monocytes, plasma cells, and heterophils. Chronic inflammatory lesions around the parasites were heavily infiltrated by eosinophils (Fig. 72).

4.6.1.5 *Acuaria (Dysphrynx) spiralis*

Dysphrynx spiralis worms were found with their heads buried deeply in the mucosa of the proventriculus where they had formed ulcers. The mucous membrane showed catarrhal inflammation and the wall of the organ showed hypertrophy (Fig. 73). There was marked prominence of the proventricular glands and the mucosa showed petechial hemorrhages (Fig. 73).

Histopathological examination revealed desquamation of the epithelium

Fig. 57 Section of fowl crop infected with *Capillaria annulata* revealing necrotic changes in squamous epithelium characterized by pyknotic nuclei. HE. X300.

Fig. 58 Section of fowl crop infected with *Capillaria annulata* revealing inflammation of oesophageal gland. HE. X300.

Fig. 59 Section of fowl oesophahus infected with *Capillaria annulata* revealing cystic gland. HE. X300.

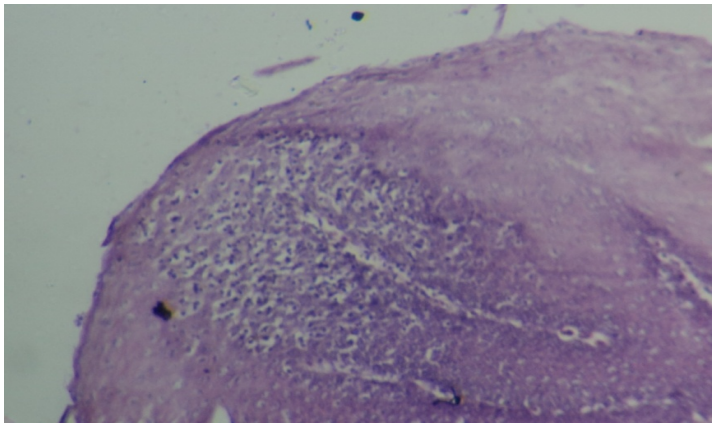

Fig. 57

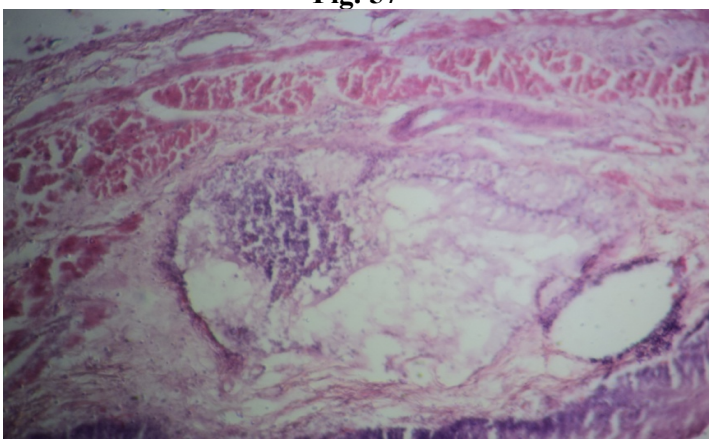

Fig. 58

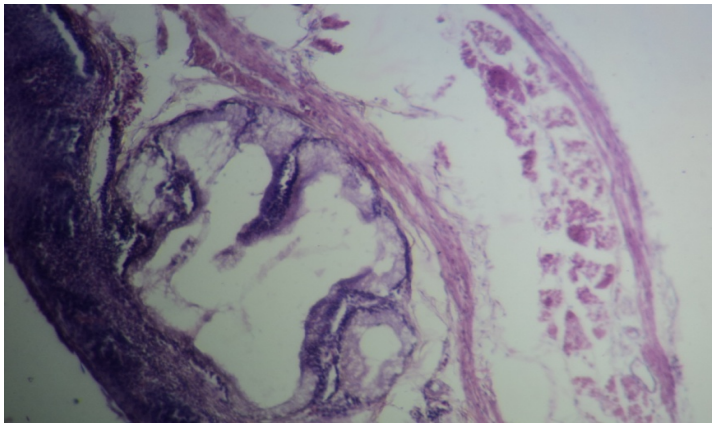

Fig. 59

Fig. 60 Section of fowl crop infected with *Capillaria annulata* revealing acanthotic changes. HE. X350.

Fig. 61 Section of fowl crop infected with *Capillaria annulata* revealing enlargement of lymphoid follicles in squamous epithelium. HE. X350.

Fig. 62 Section of fowl proventriculus infected with *Capillaria annulata* revealing proventriculitis. HE. X330.

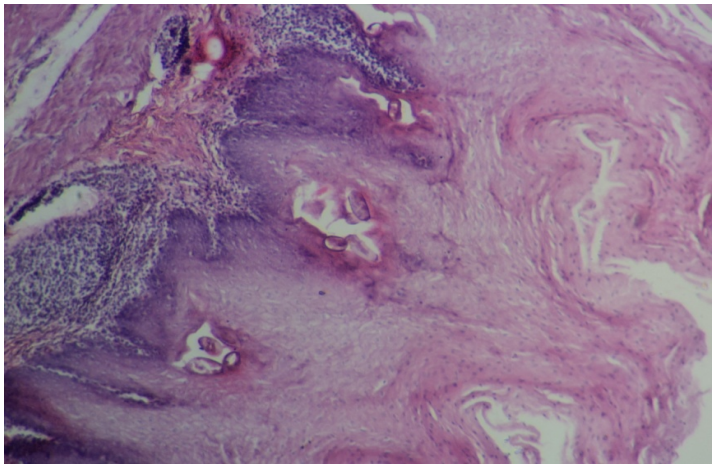

Fig. 60

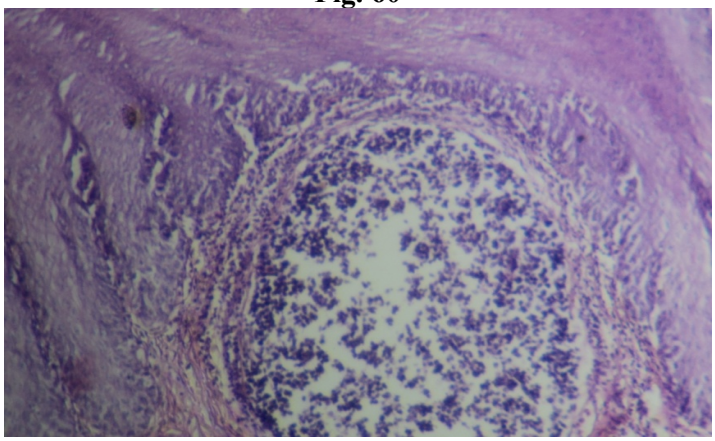

Fig. 61

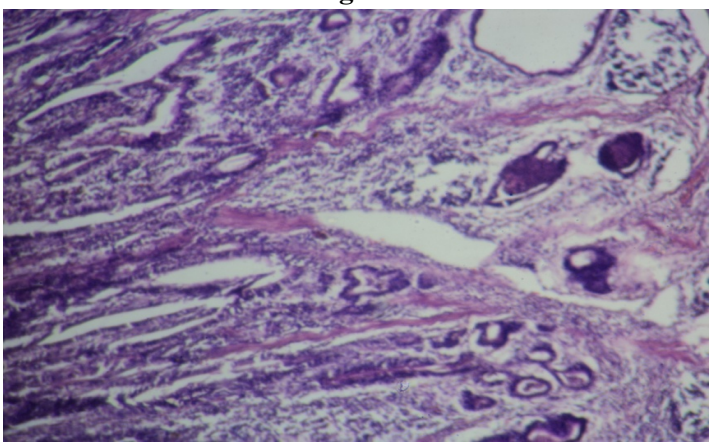

Fig. 62

Fig. 63 **Section of fowl proventriculus infected with *Capillaria annulata* revealing desquamation of epithelial cells into the common cavity of lobule. HE. X250.**

Fig. 64 **Section of fowl proventriculus infected with *Capillaria annulata* revealing cystic proventricular glands. HE. X330.**

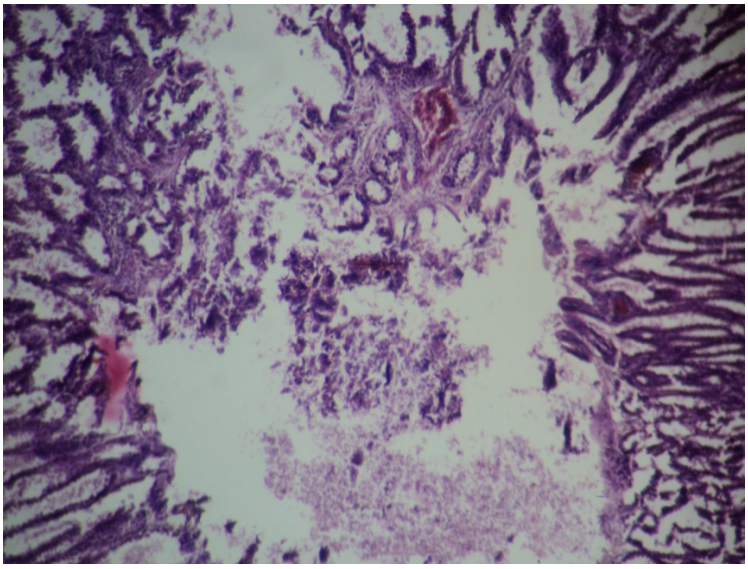

Fig. 63

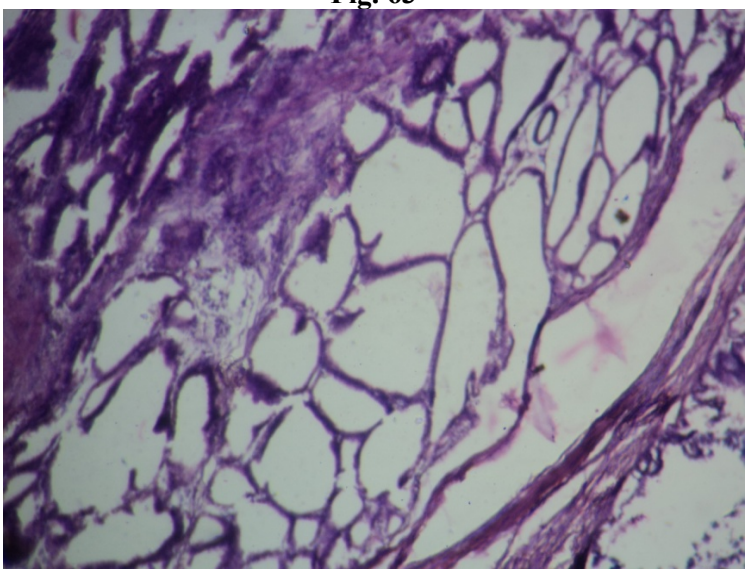

Fig. 64

Fig.65 Gizzard of domestic fowl showig *Acuaria (Cheilospirura) hamulosa* worm emerging from the musculature (arrow).

Fig.66 Sections of *Acuaria hamulosa* in the in the musculature of gizzard of domestic fowl. H&E X280.

Fig. 67 Section of fowl gizzard revealing compression of the muscle wall caused by *Acuaria hamulosa.* HE. X530.

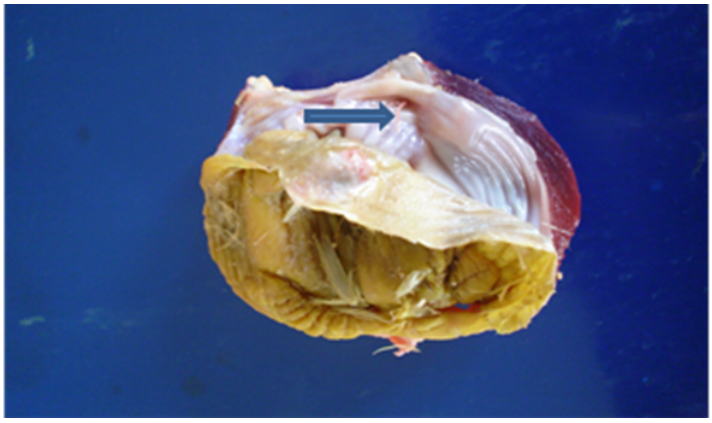

Fig. 65

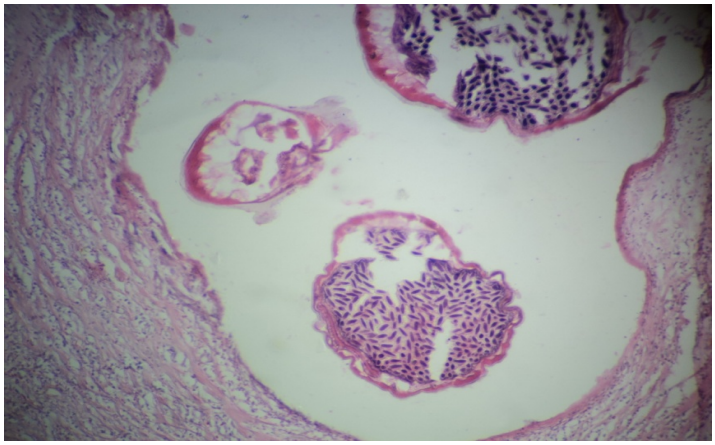

Fig. 66

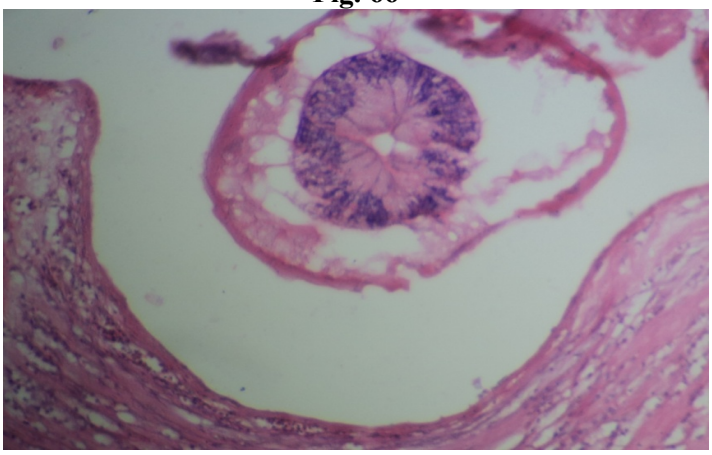

Fig. 67

Fig. 68 **Section of fowl gizzard infected with *Acuaria hamulosa* revealing haemorrhage, severe myositis and necrosis. HE. X350.**

Fig. 69 **Section of fowl gizzard infected with *Acuaria hamulosa* revealing inflammation of the mucous membrane. HE. X350.**

Fig. 70 **Section of fowl gizzard infected with *Acuaria hamulosa* revealing granulomatous inflammation. HE. X350.**

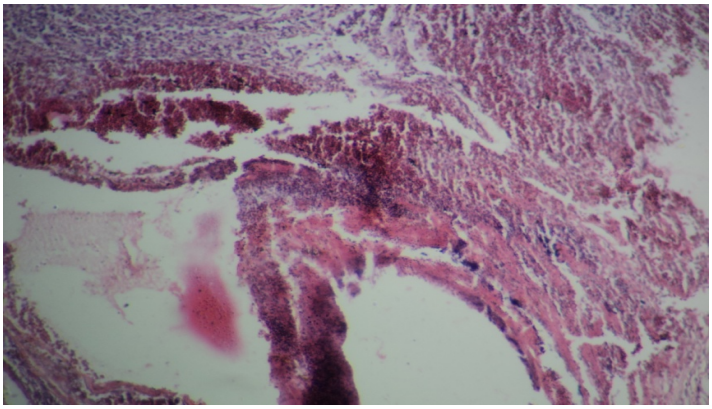

Fig. 68

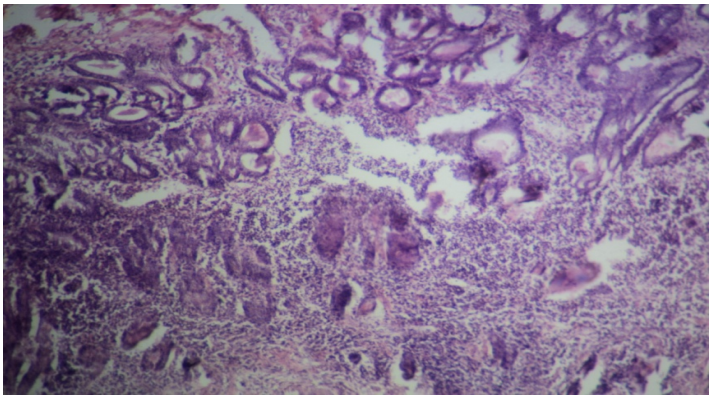

Fig. 69

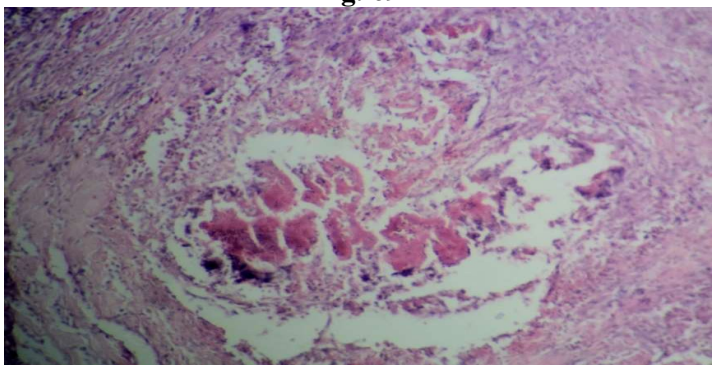

Fig. 70

Fig.71 Section of fowl gizzard infected with *Acuaria hamulosa* revealing hyperplasia of lymphoid tissue. HE. X350.

Fig.72 Section of fowl gizzard infected with *Acuaria hamulosa* revealing severe eosinophilic reaction. HE. X580.

Fig. 73 Proventriculus of fowl infected with *Dysphrynx spiralis* revealing catarrhal inflammation, petechial haemorrhages, hypertrophy of the wall of the organ.

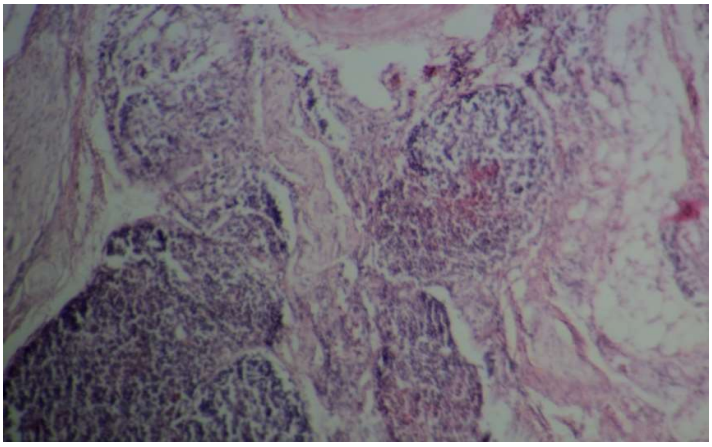

Fig. 71

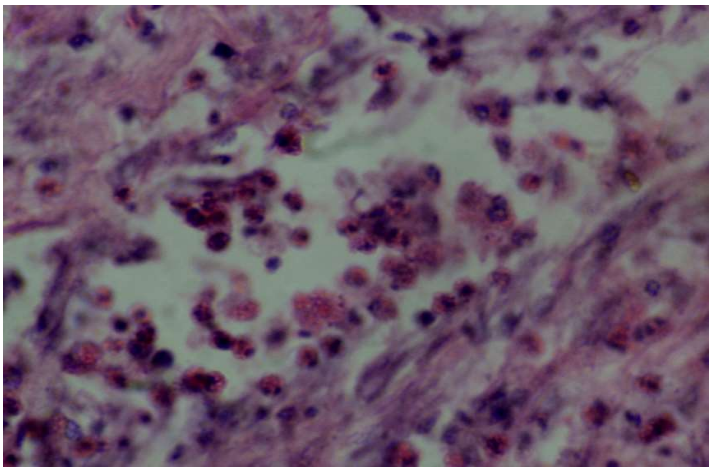

Fig. 72

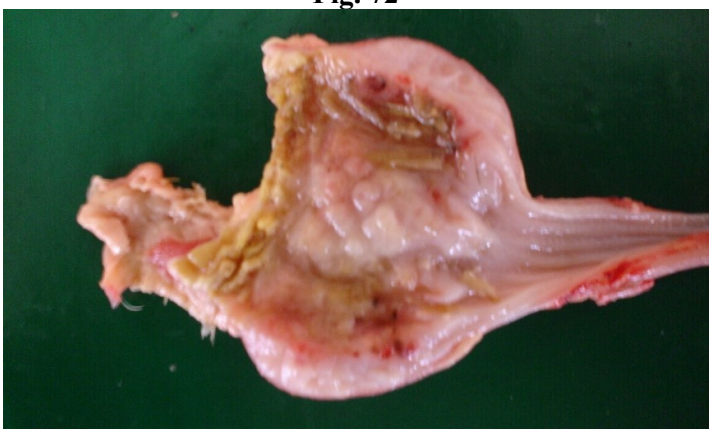

Fig. 73

into the glandular lumens (Fig. 74) and proventriculitis (Fig. 75) characterized by an intense cellular, especially eosinophilic, infiltration into the mucosa.

4.6.2 Pathology of Cestodes

Cestodes were found especially inhabiting duodenum and jejunum parts of the intestine. In general parasites were found attached to the mucosa with the help of rostellum. Intestinal lumen was full of pasty mucous containing proglottid segments.

4.6.2.1 *Raillietina cesticillus*

Infection with *Raillietina cesticillus* was characterized by the presence of robust cestodes anchored to the mucosa with the help of scolices (Fig. 76).

Histological study revealed the sections of the cestode in the lumen (Fig. 77). The histopathological lesions were desquamation of epithelium and degeneration (Fig. 78) and broadening of intestinal villi (Fig. 79). There was mild to moderate enteritis (Fig. 80) and glands at the site of infection were disrupted. The inflammatory reaction was characterized by predominant heterophils, especially in the areas of mechanical damage by scolices. Also a few lymphocytes and eosinophis were seen, especially in the lamina propria.

4.6.2.2 *Hymenolepis carioca*

Though this parasite was present in large numbers, no gross lesions were seen on the guts infected with *Hymenolepis carioca.*

Histopathological examination revealed desquamation of epithelial cells (Fig. 81) with infiltration of inflammatory cells into the core of the villi (Fig. 82).

4.6.2.3 *Raillietina tetragona*

Raillietina tetragona infected intestines revealed catarrhal enteritis, slight thickening of the wall and the lumen was full of thick pasty mucous seeded with long robust worms (Fig. 83). Some of the infected intestines revealed petechial haemorrhages and haemorrhagic contents (Fig. 84).

Fig. 74 **Section of fowl proventriculus infected with *Dysphrynx spiralis* revealing desquamation of the epithelium into the glandular lumen. HE. X330.**

Fig. 75 **Section of fowl proventriculus infected with *Dysphrynx spiralis* revealing inflammatory changes. HE. X330.**

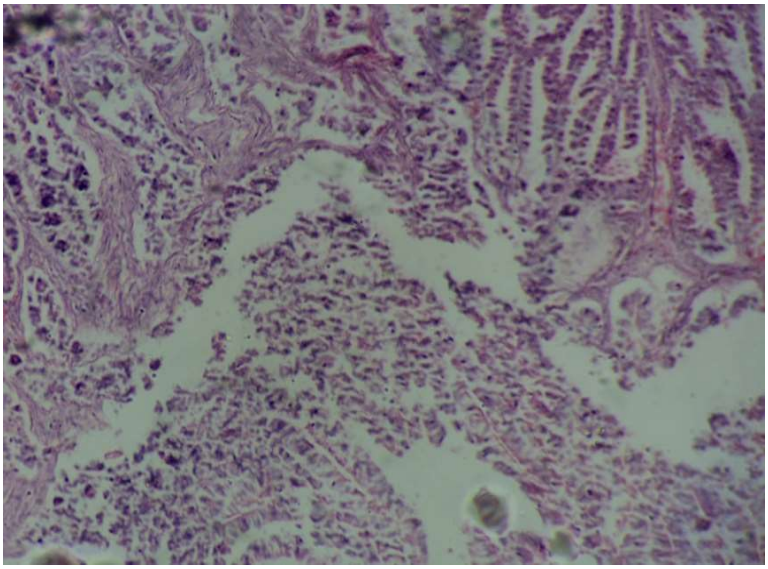

Fig. 74

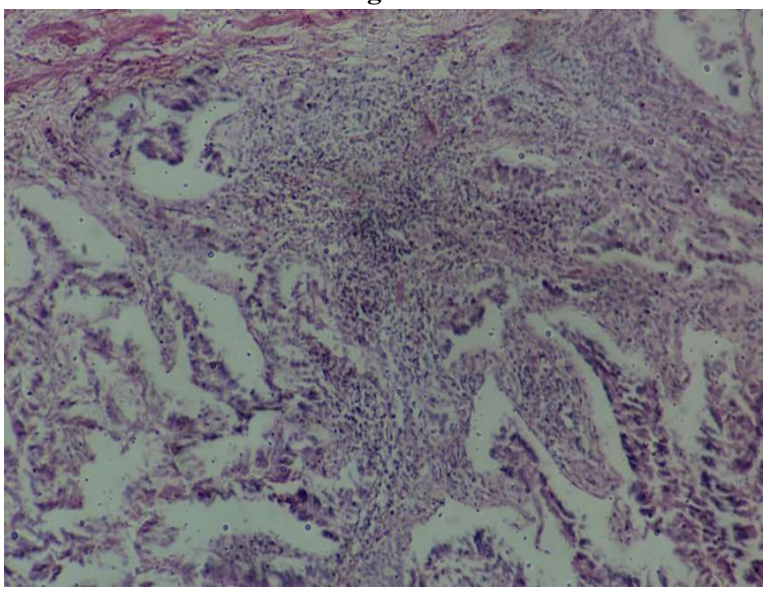

Fig. 75

Fig.76 Intestine of Fowl revealing multiple multiple *Raillietina cesticillus* worms attached to mucosa.

Fig. 77 Section of *Raillietina cesticillus* in the intestine of fowl. HE. X280.

Fig. 78 Section of fowl intestine infected with *Raillietina cesticillus* revealing desquamation of the epithelium and degeneration of villi. HE. X300.

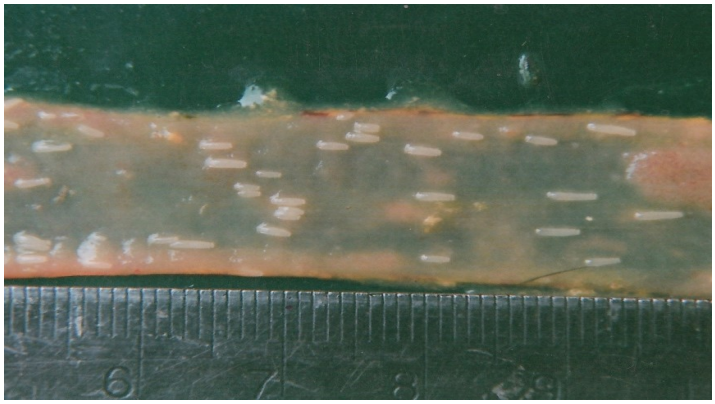

Fig. 76

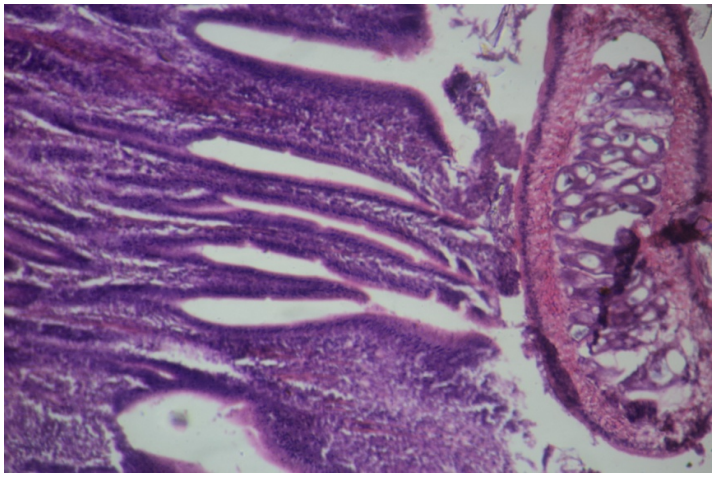

Fig. 77

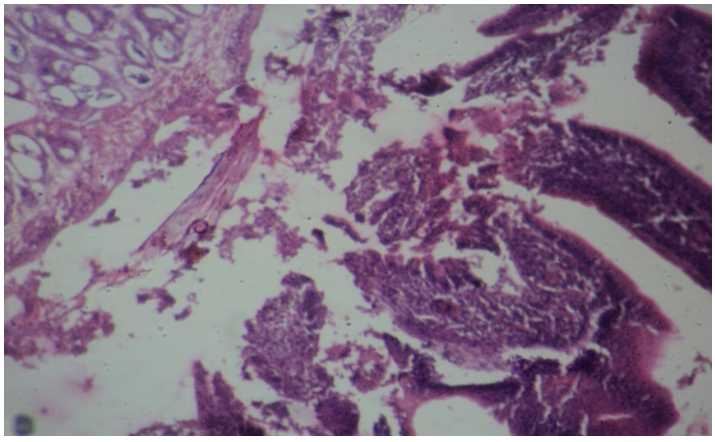

Fig. 78

Fig. 79 Section of fowl intestine infected with *Raillietina cesticillus* revealing broadening of intestinal villi. HE. X330.

Fig. 80 Section of fowl intestine infected with *Raillietina cesticillus* revealing enteritis. HE. X280.

Fig. 81 Section of fowl intestine infected with *Hymenolepis carioca* revealing desquamation of epithelial cells. HE. X330.

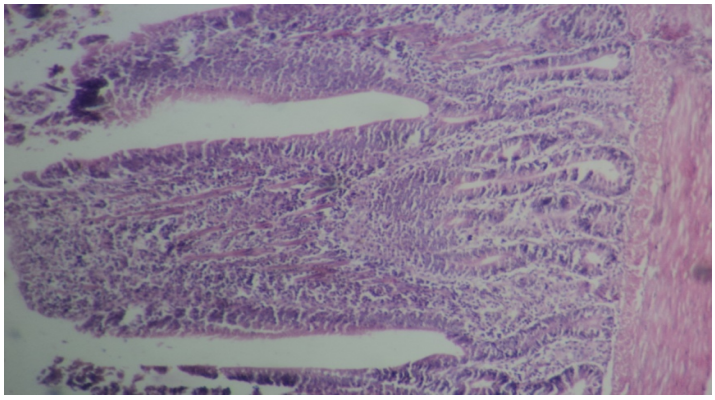

Fig. 79

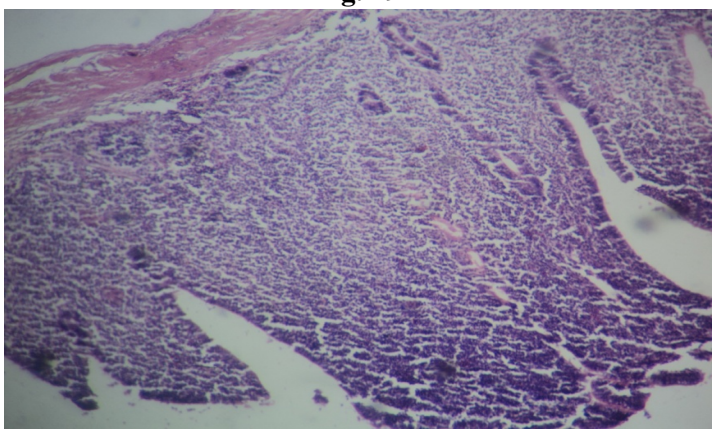

Fig. 80

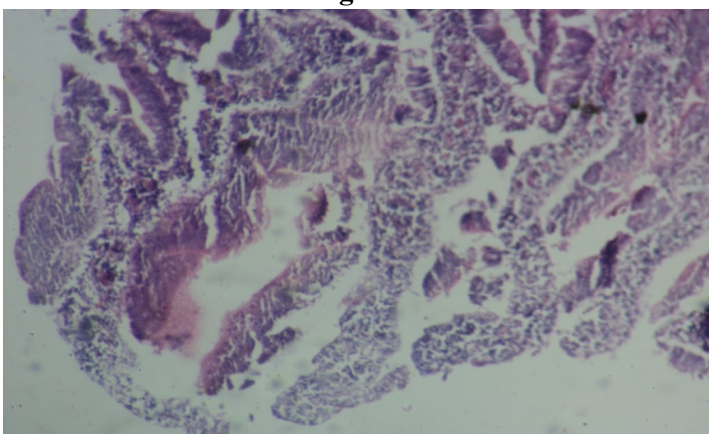

Fig. 81

Fig.82 Section of fowl intestine infected with *Hymenolepis carioca* revealing infiltration of the inflammatory cells. HE. X250.

Fig. 83 Intestine of Fowl revealing multiple *Raillietina tetragona* worms attached to mucosa.

Fig. 84 Intestine of Fowl revealing *Raillietina tetragona* worms and haemorrhagic intestinal contents.

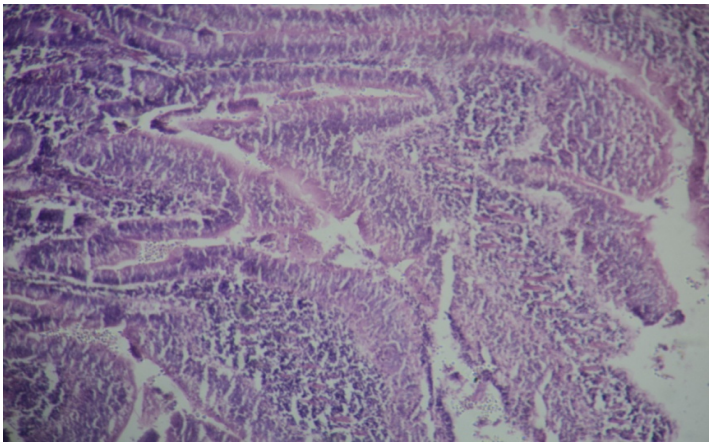

Fig. 82

Fig. 83

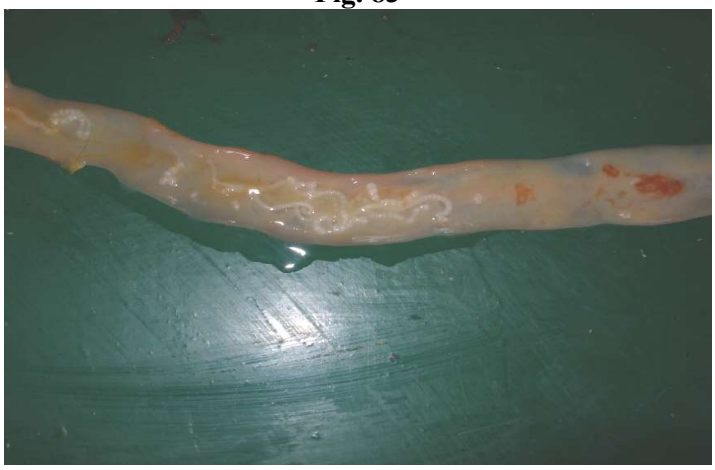

Fig. 84

Histopathological examination revealed sections of the parasites in the lumen (Fig. 85) with destruction of the villi and desquamation of the epithelial cells (Fig. 86). All the sections of duodenum and jejunum examined revealed duodenitis (Fig. 87) and jejunitis (Fig. 88) respectively besides necrosis of the villi (Fig. 89) and congestion. Inflammatory reaction around the lesions was characterized by lymphocytes and a few heterophils (Fig. 90).

4.6.2.4 *Amoebotaenia sphenoides*

In the fowl intestines infected with *Amoebotaenia sphenoides*, mucosa of duodenum appeared rough and pale. Intestial lumen was full of fluidy contents (Fig. 91). Parasites were found attached to mucosa.

Histopathological sections revealed the scolices burrowing deep into the mucosa (Fig. 92). In general the lesions were desquamation of epithelium and mild inflammation (Fig. 93). Villi appeared to be atrophied with rounding of tips. At the site of infection, glands were disintegrated. The inflammatory reaction was characterized by mononuclear cell infiltration which was more severe at the site of infection (Fig. 94).

4.6.2.5 *Choanotaenia infundibulum*

The intestines infected with *Choanotaenia infundibulum* revealed presence of catarrhal exudates in the lumen and the anterior end of the parasite penetrating into the mucosa (Fig. 95).

Histopathological sections of the infected intestines revealed mild inflammatory reaction (Fig. 96), necrosis of the villi (Fig. 97) and desquamation of the epithelium (Fig. 98). Tips of the villi represented hyperplastic changes (Fig. 99). A characteristic finding was hypertrophy and hyperplasia of smooth muscles and muscularis mucosa (Fig. 100). There was a heavy infiltration of mononuclear cells especially lymphocytes in the tips of the villi (Fig. 101).

Fig. 85 Section of the *Raillietina tetragona* in the lumen of intestine. HE. X250.

Fig.86 Section of fowl intestine infected with *Raillietina tetragona* revealing destruction of the villi and desquamation of epithelium. HE. X250.

Fig.87 Section of fowl duodenum infected with *Raillietina tetragona* revealing duodenitis. HE. X280.

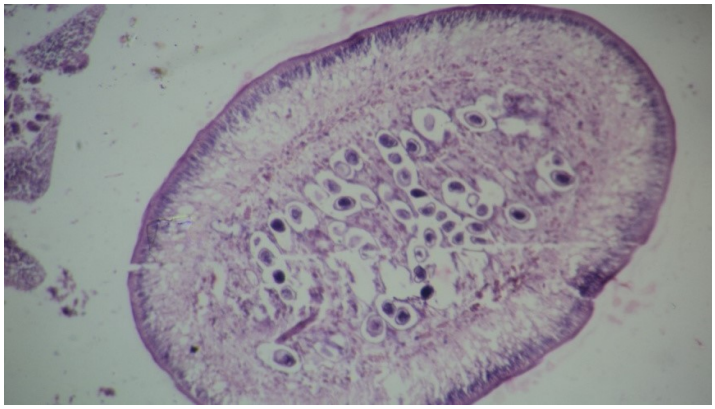

Fig. 85

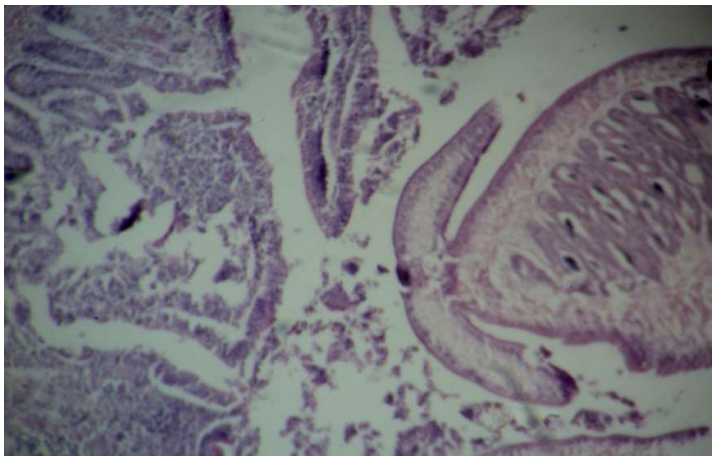

Fig. 86

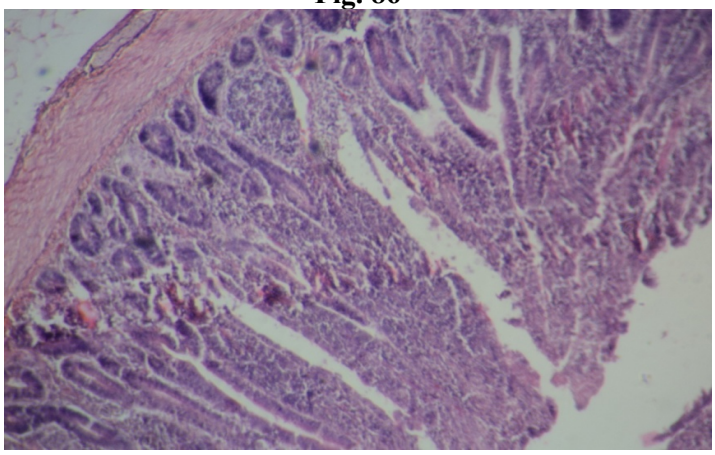

Fig. 87

Fig.88 Section of fowl jejunum infected with *Raillietina tetragona* revealing jejunitis. HE. X280.

Fig.89 Section of fowl intestine infected with *Raillietina tetragona* revealing necrosis of the villi. HE. X300.

Fig. 90 Section of fowl intestine infected with *Raillietina tetragona* revealing infiltration of the inflammatory cells. HE. X350.

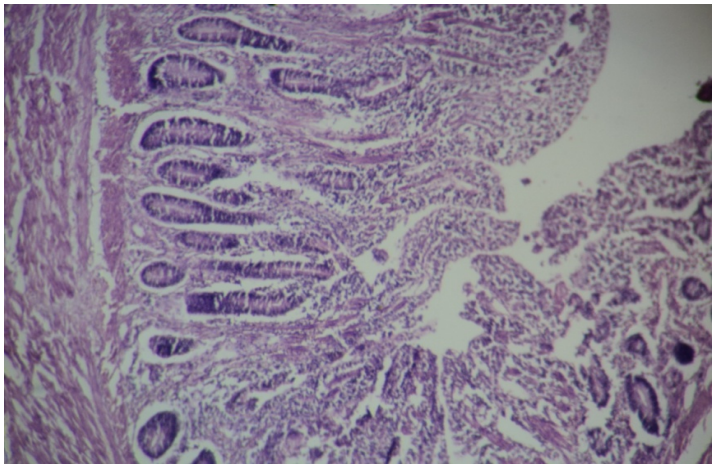

Fig. 88

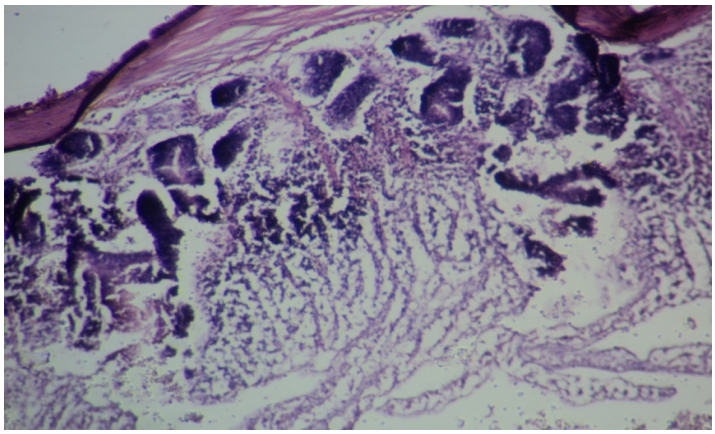

Fig. 89

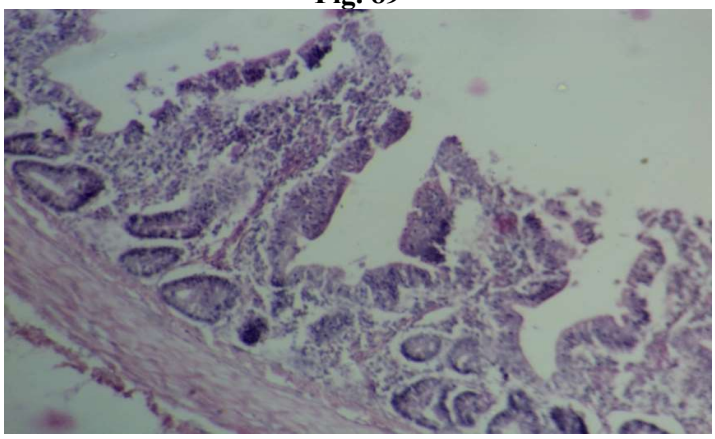

Fig. 90

Fig. 91 Intestine of Fowl infected with *Amoebotaenia sphenoides* revealing fluidy intestinal contents.

Fig. 92 Section of fowl duodenum infected with *Amoebotaenia sphenoides* revealing scolex burrowed deep into the mucosa. HE. X300.

Fig. 93 Section of fowl intestine infected with *Amoebotaenia sphenoides* revealing desquamation of epithelium. HE. X250.

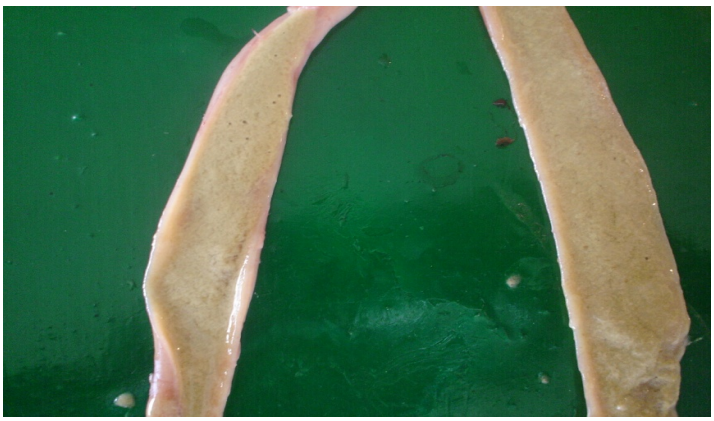

Fig. 91

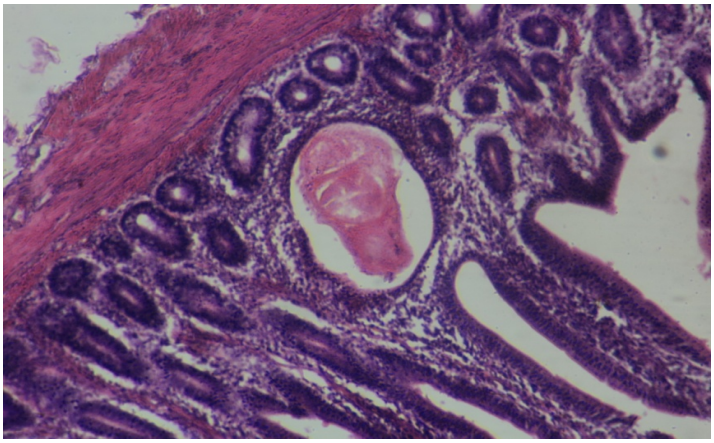

Fig. 92

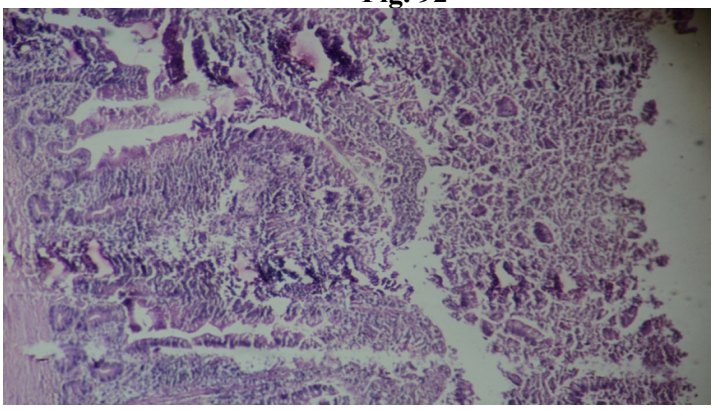

Fig. 93

Fig. 94 Section of fowl intestine infected with *Amoebotaenia sphenoides* revealing the infiltration of inflammatory cells into the core of villi. HE. X330.

Fig. 95 Intestine of Fowl revealing multiple *Choanotaenia infundibulum* worms.

Fig. 96 Section of fowl intestine infected with *Choanotaenia infundibulum* revealing mild duodenitis. HE. X350.

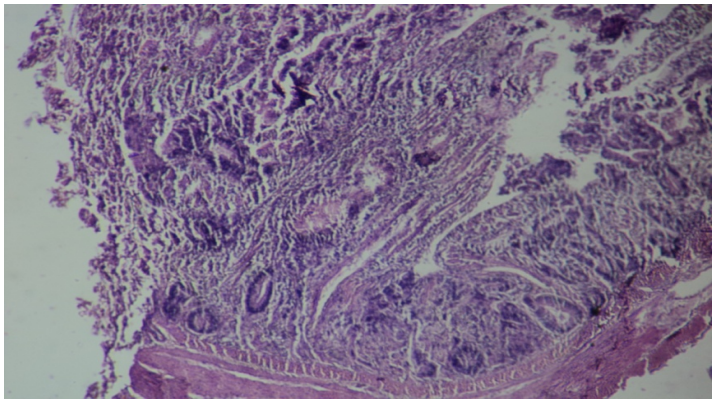

Fig.94

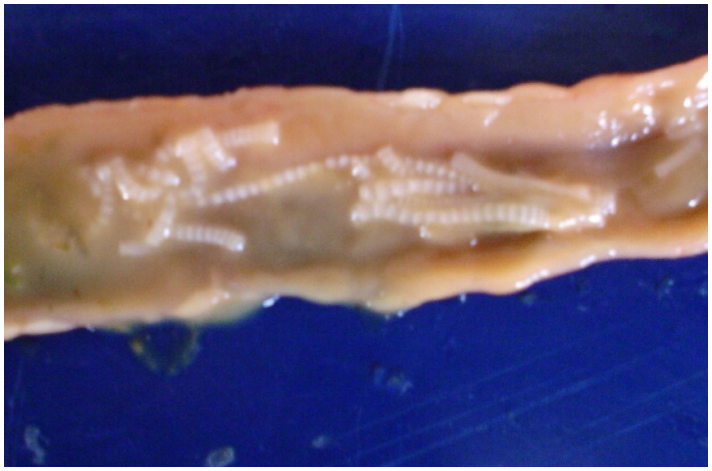

Fig. 95

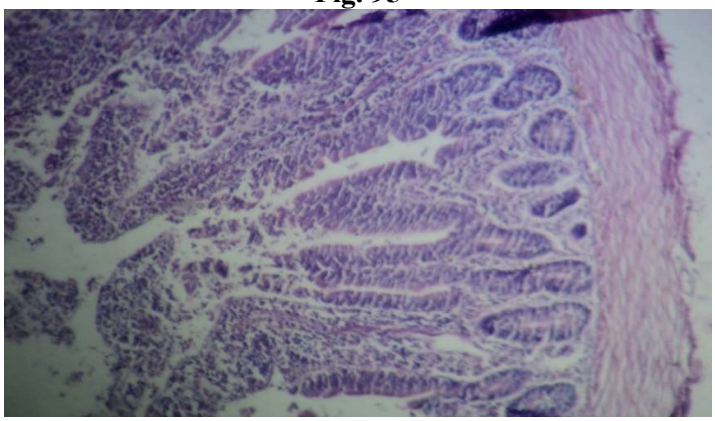

Fig. 96

Fig. 97 Section of fowl intestine infected with *Choanotaenia infundibulum* revealing necrotic villi. HE. X400.

Fig. 98 Section of fowl intestine infected with *Choanotaenia infundibulum* revealing desquamation of the epithelium. HE. X300.

Fig. 99 Section of fowl intestine infected with *Choanotaenia infundibulum* revealing hyperplastic changes in the tips of the villi. HE. X300.

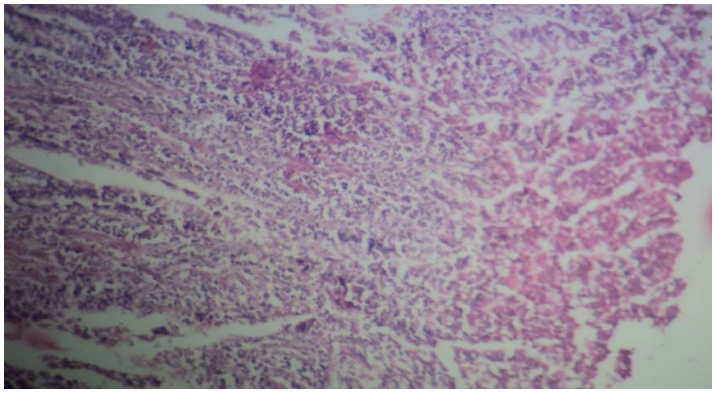

Fig. 97

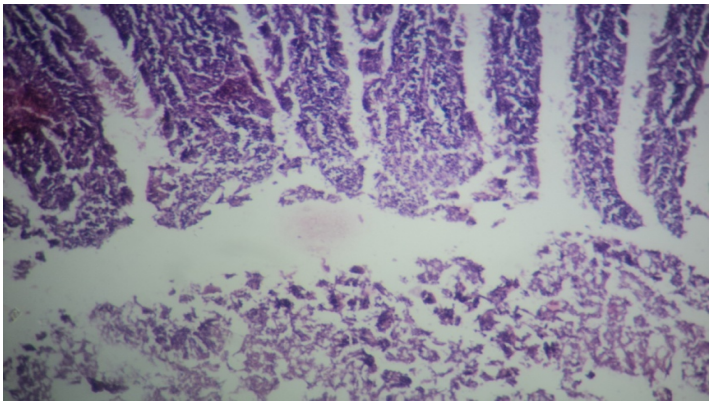

Fig. 98

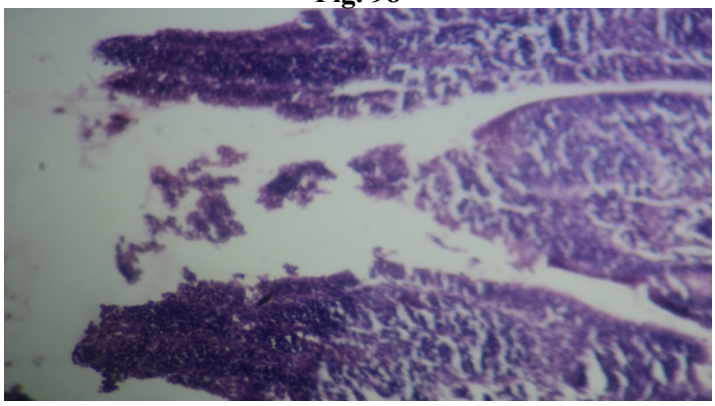

Fig. 99

Fig.100 Section of fowl duodenum infected with *Choanotaenia infundibulum* revealing hypertrophy and hyperplasia in the smooth muscles. HE. X350.

Fig.101 Section of fowl duodenum infected with *Choanotaenia infundibulum* revealing infiltration of the mononuclear cells in the tips of the villi. HE. X1400.

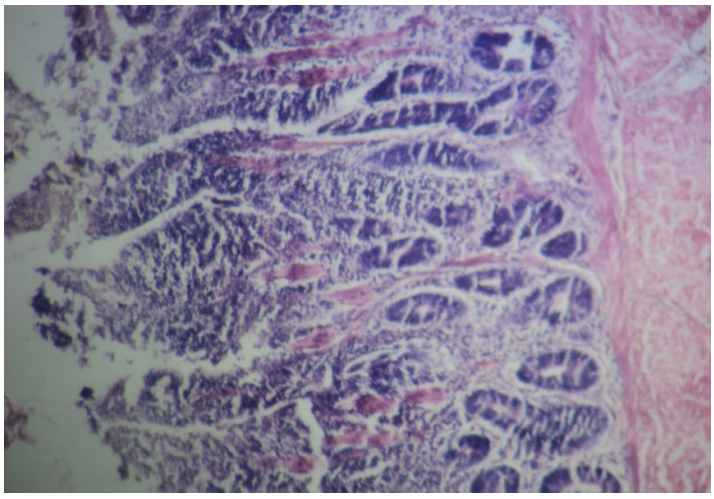

Fig. 100

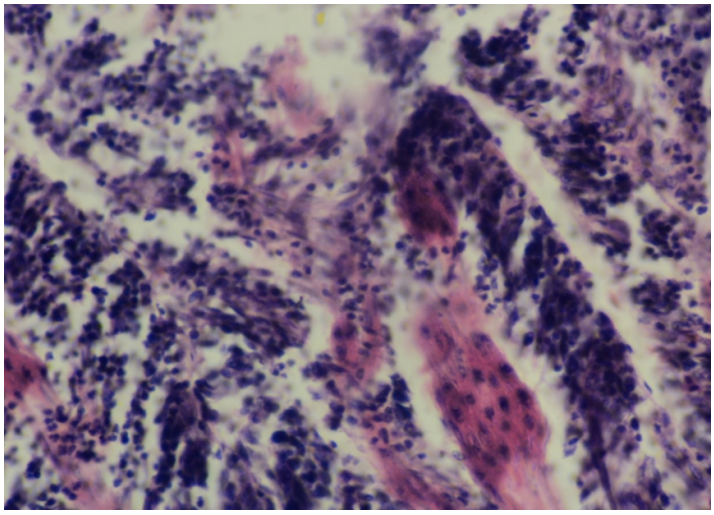

Fig. 101

4.6.3 Pathology of Trematode

4.6.3.1 *Prosthogonymus pellucidus*

The trematode was found to inhabit bursa of fabricius and coloaca (Fig. 102). On postmortem examination, the parasites were found attached to lie within to intestinal mucosa.

Histopathological examination revealed degeneration and exfoliation of the mucous epithelium of bursa of fabricius (Fig. 103) in addition to the stratification of the mucous epithelium and interstitial cell infiltration. Moderate degree of lymphoid hyperplasia (Fig. 104) and congestion (Fig. 105) was also seen. Cloaca observed desquamation of the mucous membrane of the cloacal folds (Fig. 106) and congestion (Fig. 107). However no changes were observed in oviduct.

4.7 Histochemistry

4.7.1 *Ascaridia galli*

Staining of affected tissues by Combined Alcian Blue-PAS technique revealed varying degrees of acid and neutral mucopolysaccharide reaction in different types of lesions. Goblet cell hyperplasia was positive for acid mucopolysaccharides with basement membrane positive for neutral mucopolysaccharides (Fig. 108). Positivity for acid mucopolysaccharide substances was observed both in infiltrating cells (Fig. 109) as well as desquamated material (Fig. 110).

No mast cell reaction was detectable in any of the sections when stained by Toluidine blue method.

4.7.2 *Heterakis gallinarum*

Caecum revealed acid mucopolysaccharide reaction in the glandular epithelium and goblet cells (Fig. 111 and 112).

Fig. 102 Bursa of the fowl revealing *Prosthogonymus ovatus worms*.

Fig. 103 Section of fowl bursa infected with *Prosthogonymus ovatus* revealing degeneration and exfoliation of the mucous epithelium. HE. X250.

Fig.104 Section of fowl bursa infected with *Prosthogonymus ovatus* revealing lymphoid hyperplasia. HE. X280.

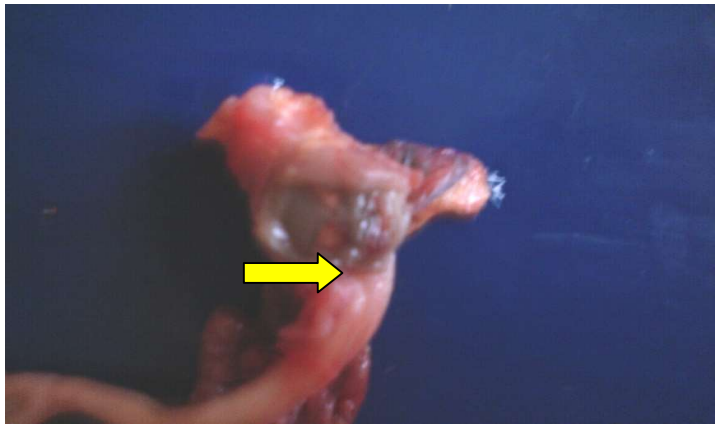

Fig. 102

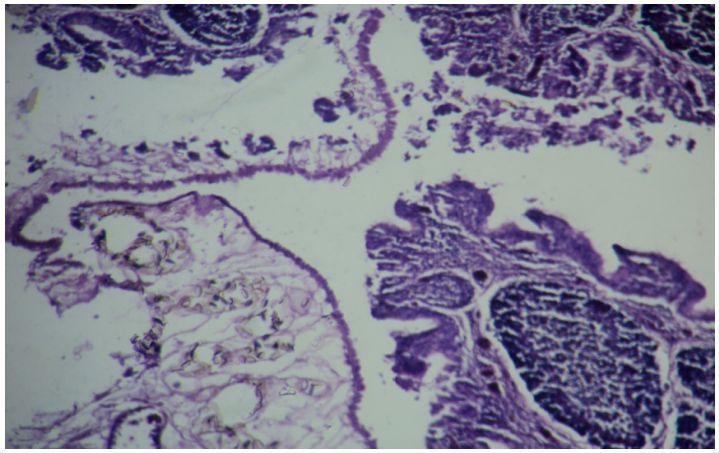

Fig. 103

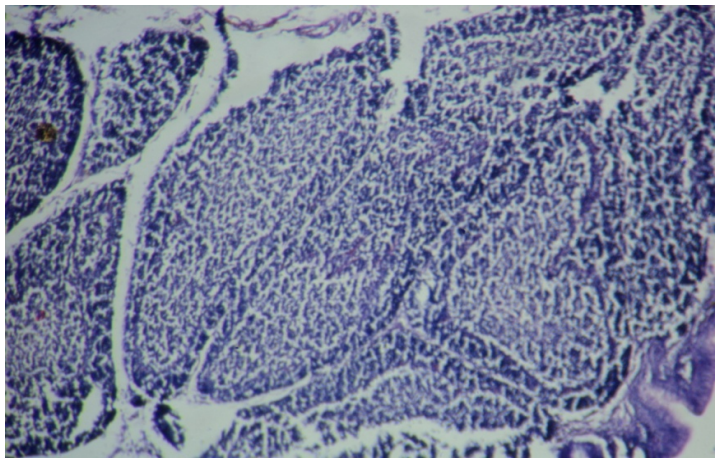

Fig. 104

Fig.105 Section of fowl bursa infected with *Prosthogonymus ovatus* revealing congestion. HE. X400.

Fig. 106 Section of fowl cloaca infected with *Prosthogonymus ovatus* revealing desquamation of the mucous membrane of folds. HE. X280.

Fig.107 Section of fowl cloaca infected with *Prosthogonymus ovatus* revealing congestion. HE. X250.

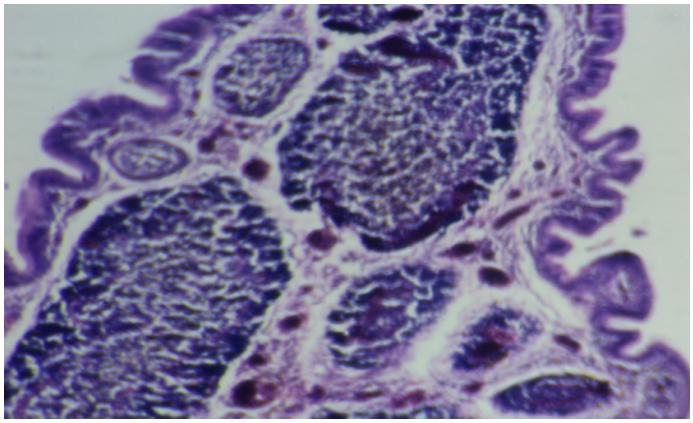

Fig. 105

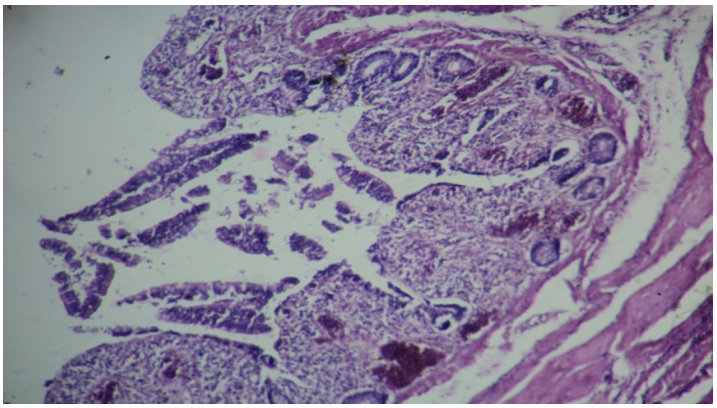

Fig. 106

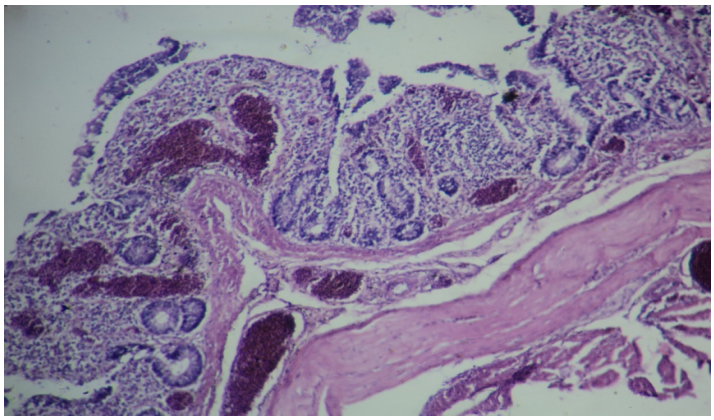

Fig. 107

Fig. 108 **Section of fowl intestine infected with *Ascaridia galli* revealing goblet cell hyperplasia positive for acid mucopolysaccharides with basement membrane positive for neutral mucopolysaccharides (arrow). Combined Alcian blue PAS. X650.**

Fig. 109 **Section of fowl intestine infected with *Ascaridia galli* revealing infiltrating cells positive for acid mucopolysaccharides. Combined Alcian blue PAS. X650.**

Fig. 110 **Section of fowl intestine infected with *Ascaridia galli* revealing desquamated material positive for acid mucopolysaccharides. Combined Alcian blue PAS. X650.**

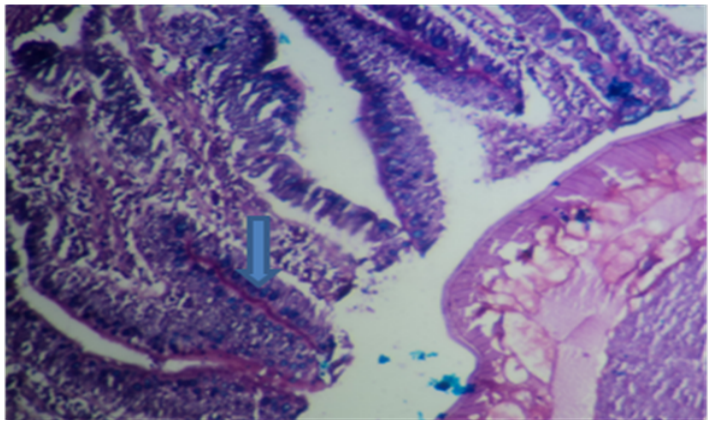

Fig. 108

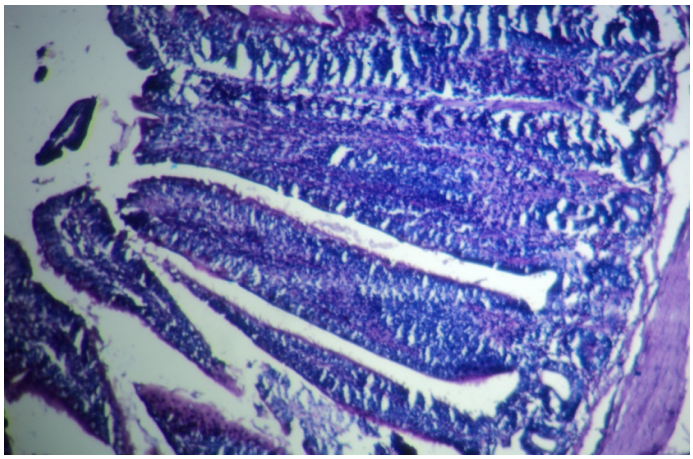

Fig. 109

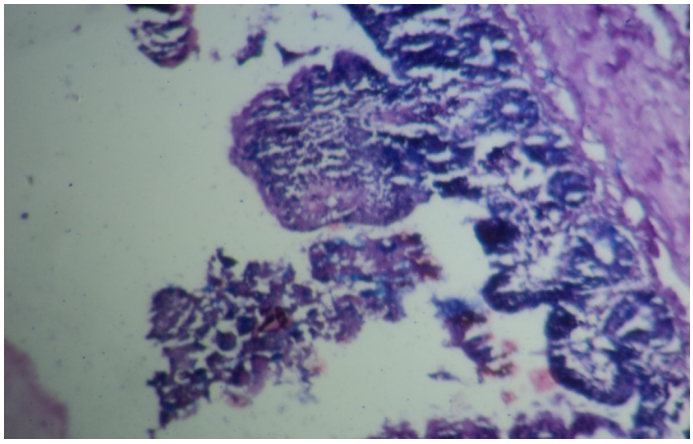

Fig. 110

Toluidine blue stained sections revealed metrachromatic mucosal mast cells reaction (Fig. 113).

4.7.3 *Capillaria annulata*

Oeosophagus revealed a strong positive reaction for acid mucopolysaccharides in the oesophageal glands (Fig. 114 and 115) and within the same glands intervening stroma was positive for neutral mucopolysaccharide (Fig. 116). Adventitial layer of oesophagus and desquamated epithelium were positive for acid mucopolysaccharide substances (Fig. 117 and 118). Crop revealed negative reaction for acid mucopolysaccharides. However a positive reaction for acid mucopolysaccharide substances in the epithelium of proventricular glands was observed (Fig. 119).

Toluidine blue stained sections of crop revealed mast cell reaction in the submucosal layer (Fig. 120).

4.7.4 *Acuaria hamulosa*

Gizzard revealed moderate increase in acid mucopolysaccharide in glands (Fig. 121) while as glandular secretions were positive for neutral mucopolysaccharides (Fig.122).

No mast cell reaction was detectable in any of the sections when stained by Toluidine blue method.

4.7.5 *Dysphrynx spiralis*

Proventriculs revealed a strong positive reaction for acid mucopolysaccharides in the glandular epithelium (Fig. 123) and mucous membrane (Fig. 124).

No mast cell reaction was detectable in any of the sections when stained by Toluidine blue method.

Fig.111 Section of fowl caecum infected with *Heterakis gallinarum* revealing positive reaction for acid mucopolysaccharides in the glandular epithelium. Combined Alcian blue PAS. X1200.

Fig.112 Section of fowl caecum infected with *Heterakis gallinarum* revealing positive reaction for acid mucopolysaccharides in the goblet cells. Combined Alcian blue PAS. X650.

Fig.113 Section of fowl caecum infected with *Heterakis gallinarum* revealing mast cell reaction in the mucosa (arrow). Toluidine blue. X1400

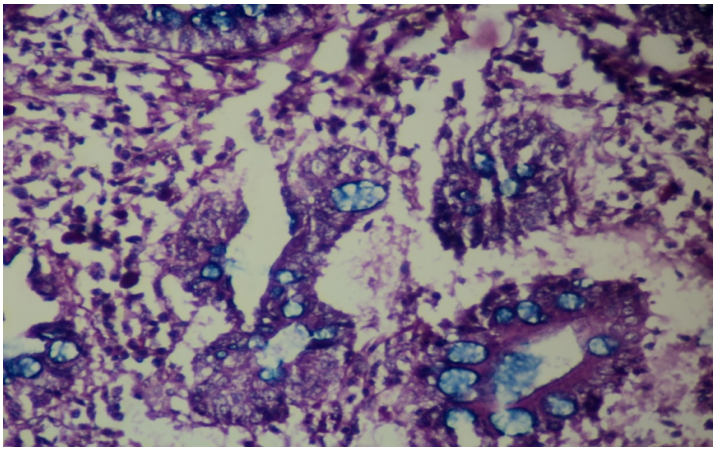

Fig. 111

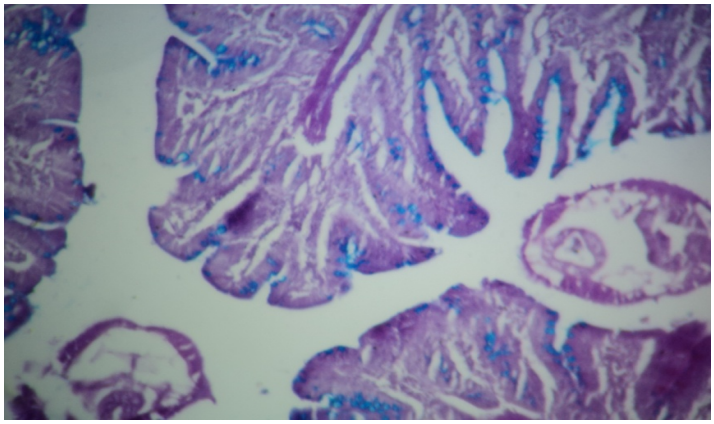

Fig. 112

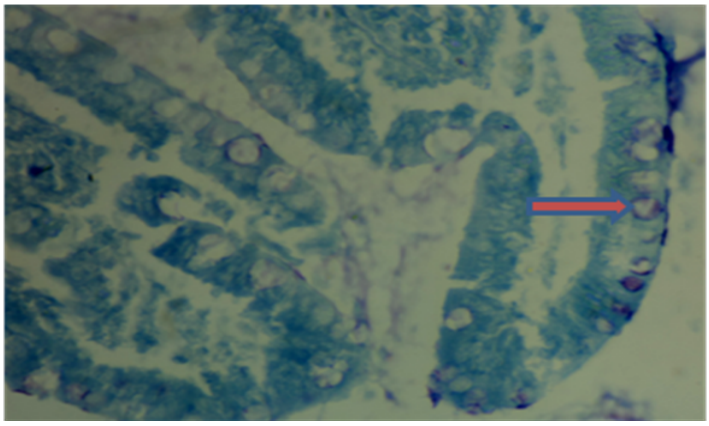

Fig. 113

Fig.114 Section of fowl oesophagus infected with *Capillaria annulata* revealing positive reaction for acid mucopolysaccharides in the oesophageal glands. Combined Alcian blue PAS. X250.

Fig.115 Section of fowl oesophagus infected with *Capillaria annulata* revealing positive reaction for acid mucopolysaccharides in the oesophageal glands. Combined Alcian blue PAS. X280.

Fig.116 Section of fowl oesophagus infected with *Capillaria annulata* revealing positive reaction for neutral mucopolysaccharides in the interveining stroma of oesophageal glands. Combined Alcian blue PAS. X1120.

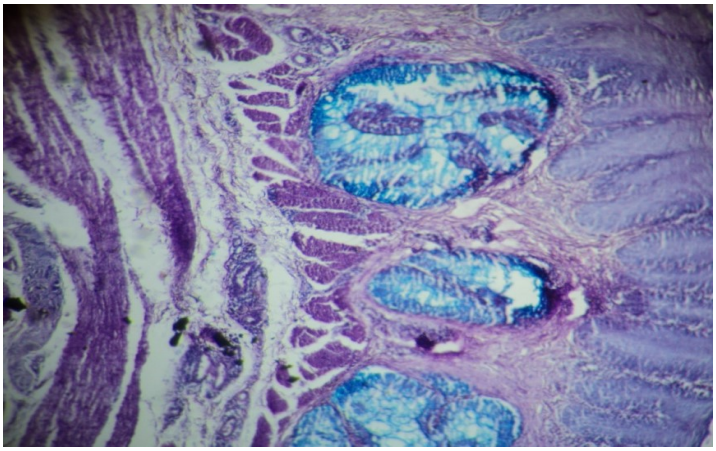

Fig. 114

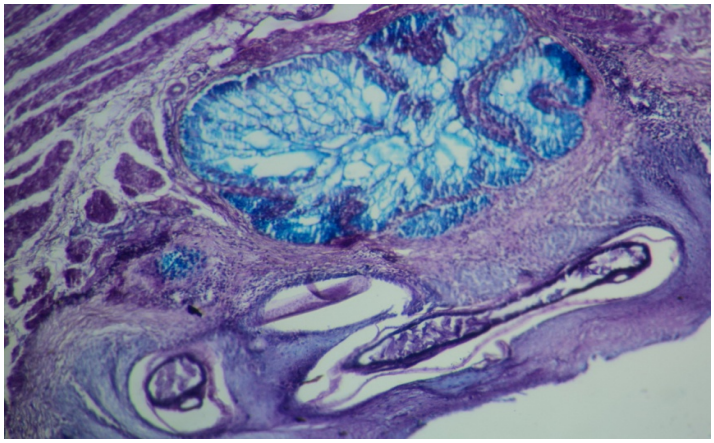

Fig. 115

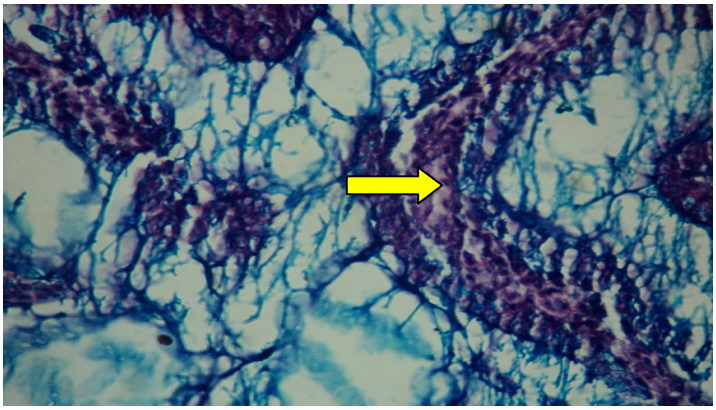

Fig. 116

Fig.117 Section of fowl oesophagus infected with *Capillaria annulata* revealing positive reaction for acid mucopolysaccharides in the sub of oesophagus. Combined Alcian blue PAS. X300.

Fig.118 Section of fowl oesophagus infected with *Capillaria annulata* revealing positive reaction for acid mucopolysaccharide substaces in the desquamated epithelium of oesophagus. Combined Alcian blue PAS. X300.`

Fig.119 Section of fowl proventriculus infected with *Capillaria annulata* revealing positive reaction for acid mucopolysaccharide in the glandular epithelium. Combined Alcian blue PAS. X560.

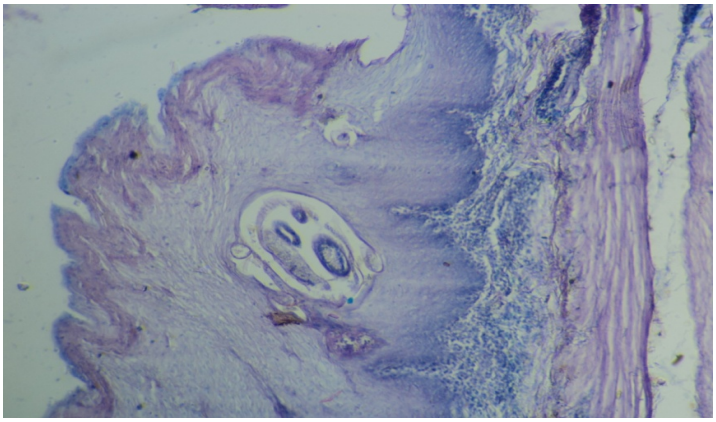

Fig. 117

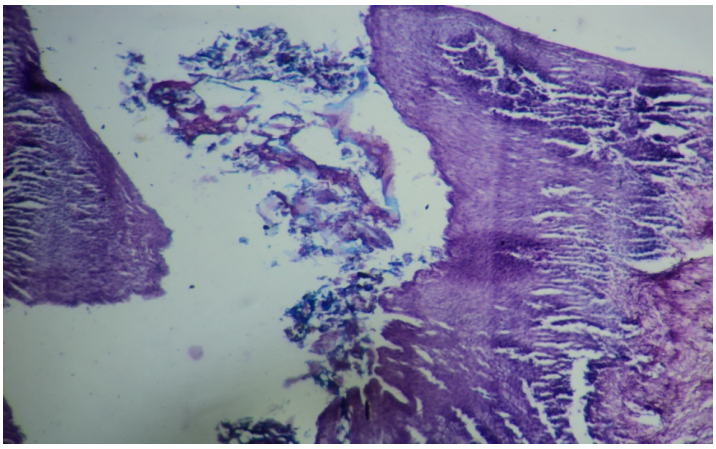

Fig. 118

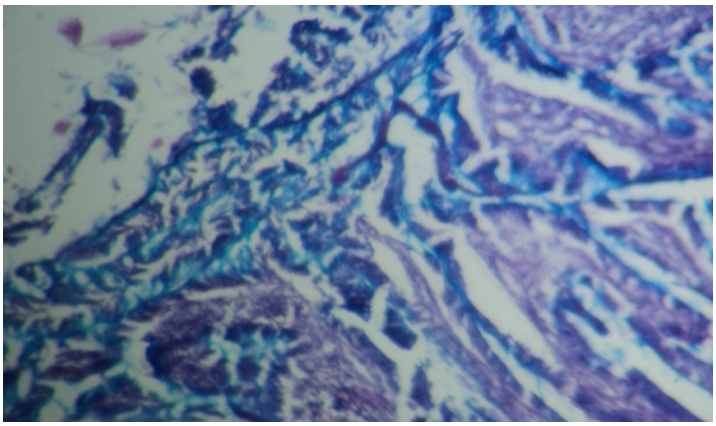

Fig. 119

Fig. 120 **Section of fowl crop infected with *Capillaria annulata* revealing mast cell reaction in the submucosa. Toluidine blue. X560.**

Fig. 121 **Section of fowl gizzard infected with *Acuaria hamulosa* revealing positivity for acid mucopolysaccharide substaces in the glandular epithelium. Combined Alcian blue PAS. X350.**

Fig. 122 **Section of fowl gizzard infected with *Acuaria hamulosa* showing glandular secretions positive for neutral mucopolysaccharide substaces. Combined Alcian blue PAS. X330.**

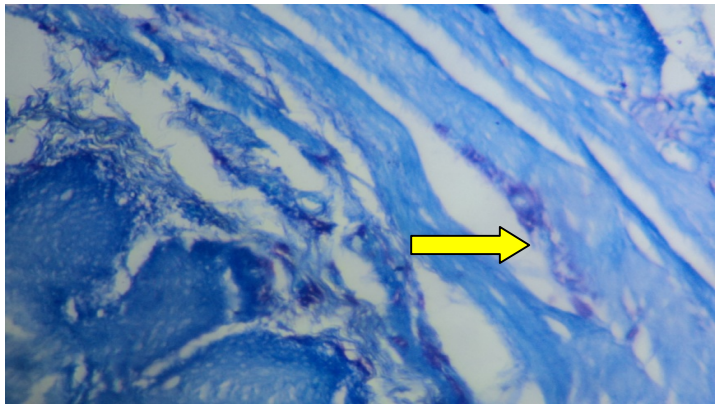

Fig. 120

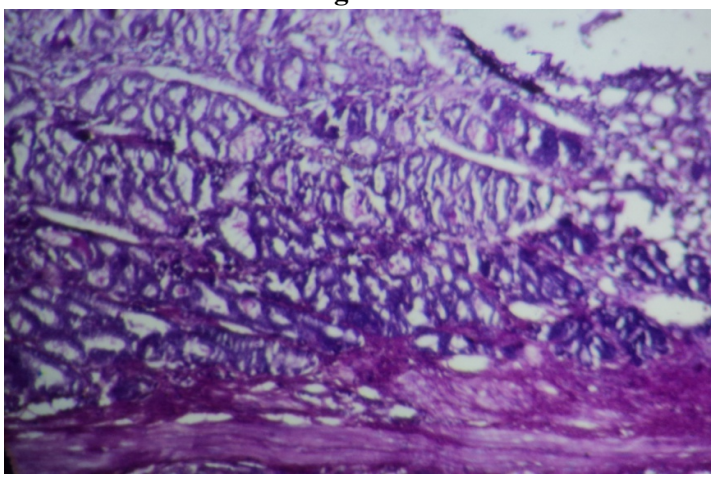

Fig. 121

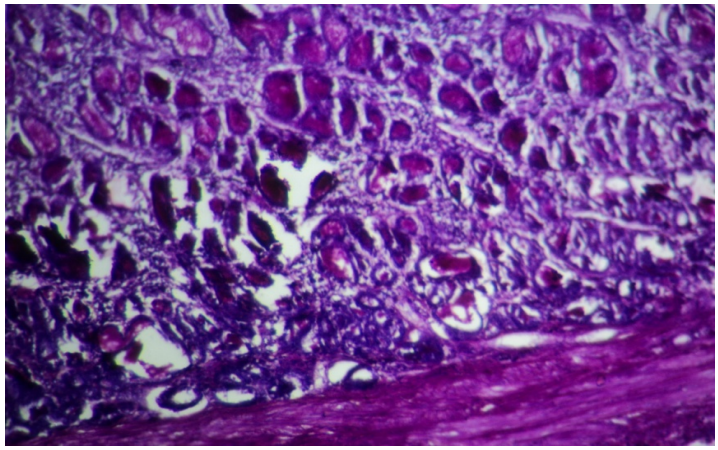

Fig. 122

Fig.123 Section of fowl proventriculus infected with *Dysphrynx spiralis* revealing increased alcian blue positive material in the glandular epithelium. Combined Alcian blue PAS. X330.

Fig.124 Section of fowl proventriculus infected with *Dysphrynx spiralis* revealing increased alcian blue positive material in the mucous membrane. Combined Alcian blue PAS. X330.

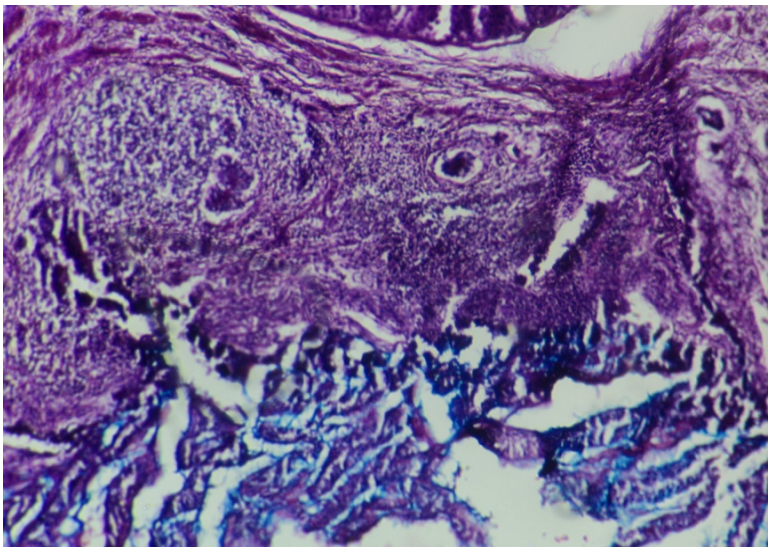

Fig. 123

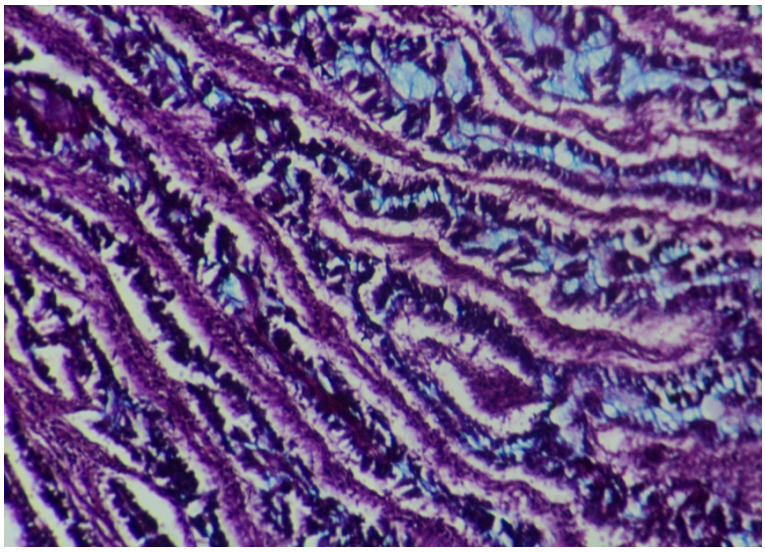

Fig. 124

4.7.6 *Raillietina cesticillus*

Small intestines revealed increased acid mucopolysaccharide content in the glandular epithelium and desquamated material (Figs. 125 and 126).

Toluidine blue stained sections of intestines revealed positive reaction for metachromasia in the glandular epithelium (Fig. 127).

4.7.7 *Hymenolepis carioca*

Staining of duplicate sections from infected portions of the intestine by Combined Alcian Blue-PAS technique revealed goblet cell hyperplasia strongly positive for acid mucopolysaccharide substances (Fig. 128 and 129).

No mast cell reaction was detectable in any of the sections when stained by Toluidine blue method.

4.7.8 *Raillietina tetragona*

Increased acid mucopolysaccharide content was observed in the intestinal glands and glandular epithelium (Fig. 130 and 131).

No mast cell reaction was detectable in any of the sections when stained by Toluidine blue method.

4.7.9 *Amoebotaenia sphenoides*

Staining of duplicate sections from infected intestines by Combined Alcian Blue-PAS technique revealed a strong positive reaction for acid mucopolysaccharides in the mucous membrane (Fig. 132).

No mast cell reaction was discernable in any of the sections when stained by Toluidine blue method.

4.7.10 *Choanotaenia infundibulum*

Parallel sections upon staining by Combined Alcian Blue-PAS method revealed increased acid mucopolysaccharide content in the glandular epithelium

Fig.125 Section of fowl intestine infected with *Raillietina cesticillus* revealing increased alcian blue positive material in the glandular epithelium. Combined Alcian blue PAS. X280.

Fig.126 Section of fowl intestine infected with *Raillietina cesticillus* revealing increased alcian blue positive material in the glandular epithelium and desquamated material. Combined Alcian blue PAS. X280.

Fig. 127 Section of fowl intestine infected with *Raillietina cesticillus* revealing a positive reaction for metachromasia in the glandular epithelium. Toluidine blue. X1400.

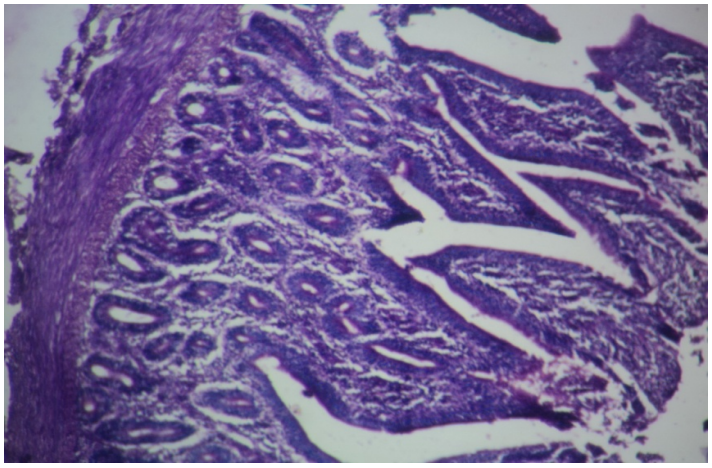

Fig. 125

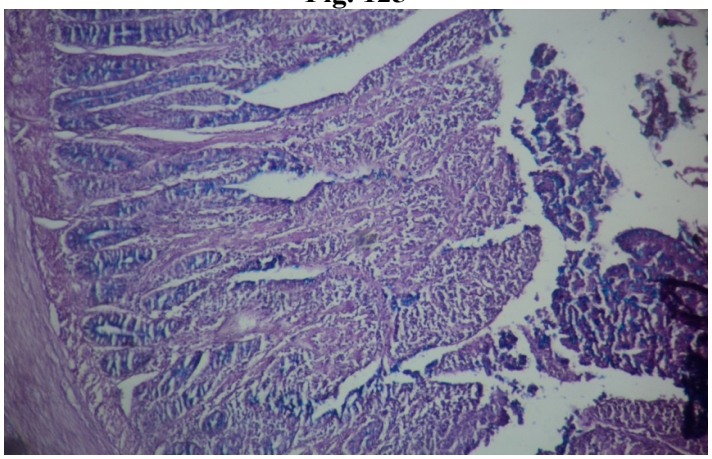

Fig. 126

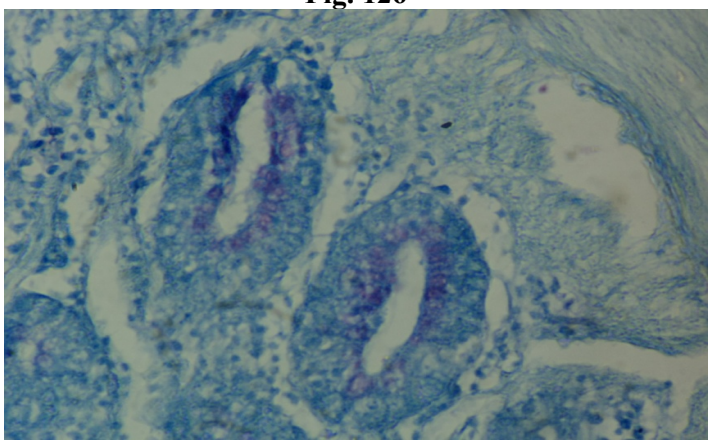

Fig. 127

Fig. 128 **Section of the fowl intestine infected with *Hymenolepis carioca* revealing goblet cell hyperplasia strongly positive for acid mucopolysaccharide substances. Combined Alcian blue PAS. X350.**

Fig. 129 **Section of the fowl intestine infected with *Hymenolepis carioca* revealing goblet cell hyperplasia strongly positive for acid mucopolysaccharide substances. Combined Alcian blue PAS. X1200.**

Fig.130 **Section of fowl intestine infected with *Raillietina tetragoa* revealing increased alcian blue positive material in the intestinal glands. Combined Alcian blue PAS. X330.**

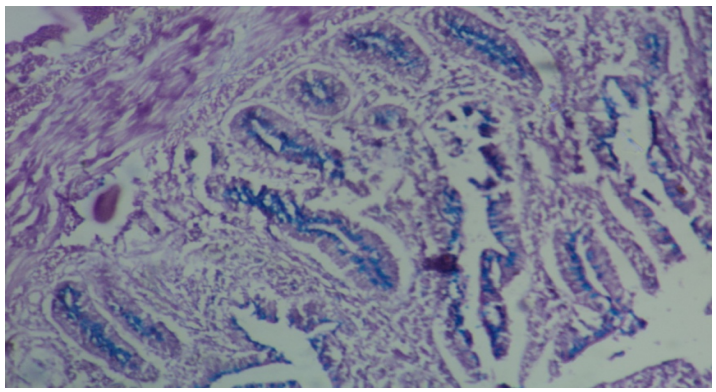

Fig. 128

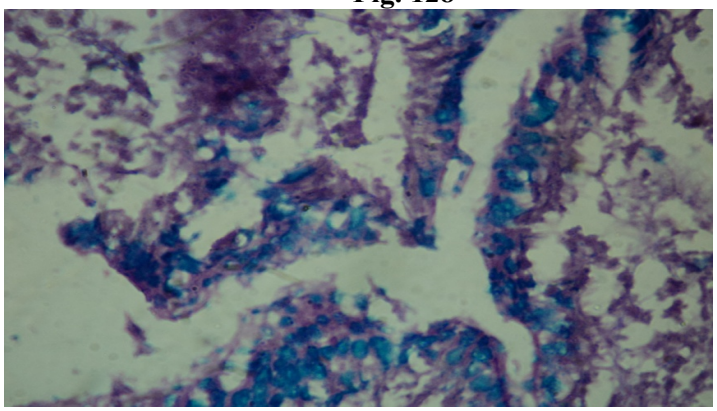

Fig. 129

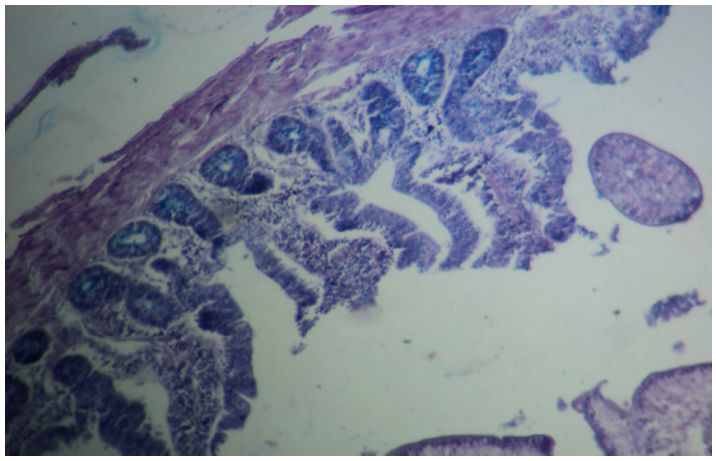

Fig. 130

Fig.131 **Section of fowl intestine infected with *Raillietina tetragoa* revealing increased alcian blue positive material in the glandular epithelium. Combined Alcian blue PAS. X500.**

Fig.132 **Section of fowl intestine infected with *Amoebotaenia sphenoides* revealing positive reaction for acid mucopolysaccharides in the mucous membrane. Combined Alcian blue PAS. X280.**

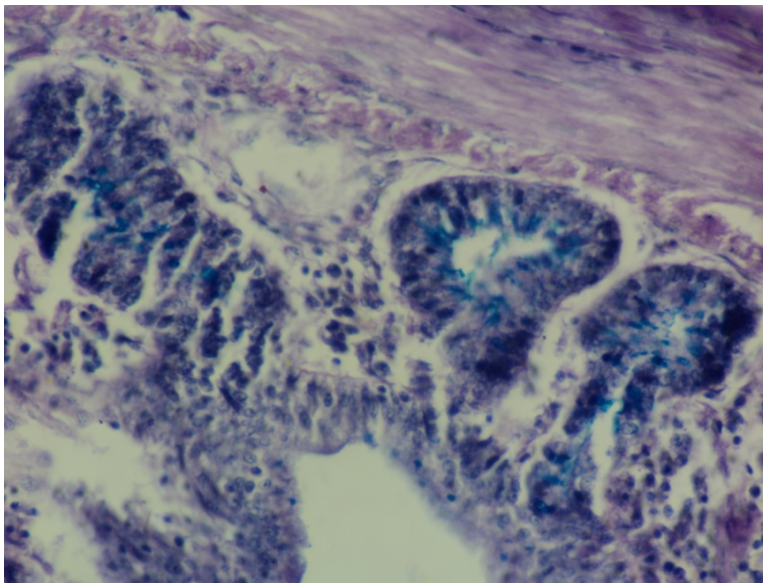

Fig. 131

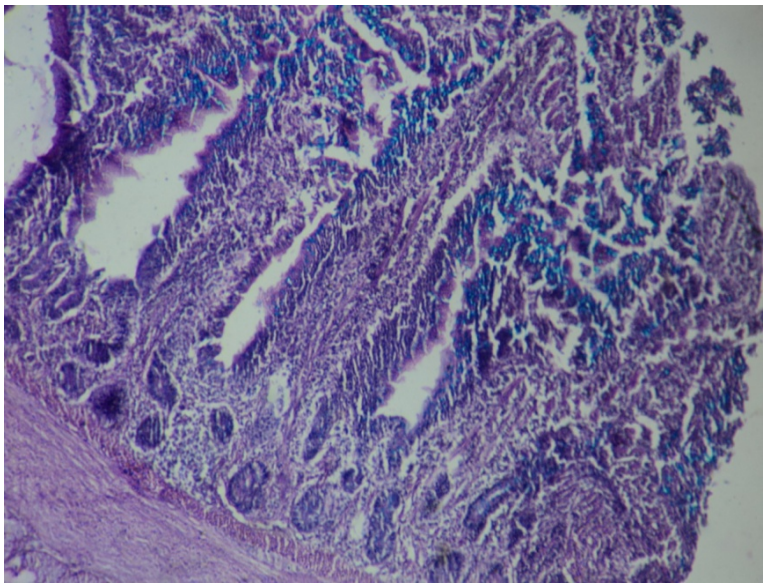

Fig. 132

(Fig. 133). Infiltrating mononuclears also revealed a positive reaction for acid mucopolysaccharides.

No mast cell reaction was discernable in any of the sections when stained by Toluidine blue method.

4.7.11 *Prosthogonymus ovatus*

Staining of duplicate sections from bursa by Combined Alcian Blue-PAS technique revealed a positive reaction for acid mucopolysaccharides in the bursal epithelium (Fig. 134). Sections from cloaca showed positivity for acid mucopolysaccharide substances in the cloacal glands and desquamated material (Fig. 135). The epithelium of infundibulum also reacted positively for increased acid mucopolysaccharides (Fig. 136).

A mucosal mast cell reaction was occasionally observed in the infundibulum of the infected birds (Fig. 137).

4.8 Haemato-biochemical analysis

The haematological and biochemical values of the infected visa-a-vis non-infected birds are given in table 5. Haemoglobin and total leukocyte counts revealed significant decrease and increase, respectively, in case of mixed nematode and cestode infections. Packed cell volume showed a significant decrease in pure nematode and mixed infections. Total Erythrocyte count and Erythrocyte sedimentation rate showed no significant change. Lymphocyte count observed significant increase in pure cestode and mixed infections of nematodes and cestodes while as monocyte counts revealed significant increase in pure infections of nematodes and cestodes. Eosinophil counts on the other hand showed significant increase in case of pure nematode and mixed infections. Heterophil and basophil counts showed no significant difference statistically in comparison to control.

Serum biochemical values revealed no significant differences in total protein, albumin, globulin and A/G ratio in case of pure nematode and pure

Fig.133 **Section of fowl intestine infected with *Choanotaenia infundibulum* revealing positive reaction for acid mucopolysaccharides in the glandular epithelium. Combined Alcian blue PAS. X530.**

Fig.134 **Section of fowl bursa infected with *Prosthogonymus pellucidus* revealing increased alcian blue positive material in the bursal epithelium. Combined Alcian blue PAS. X1000.**

Fig.135 **Section of fowl cloaca infected with *Prosthogonymus pellucidus* revealing positivity for acid mucopolysaccharide substances in the cloacal glands and desquamated material. Combined Alcian blue PAS. X250.**

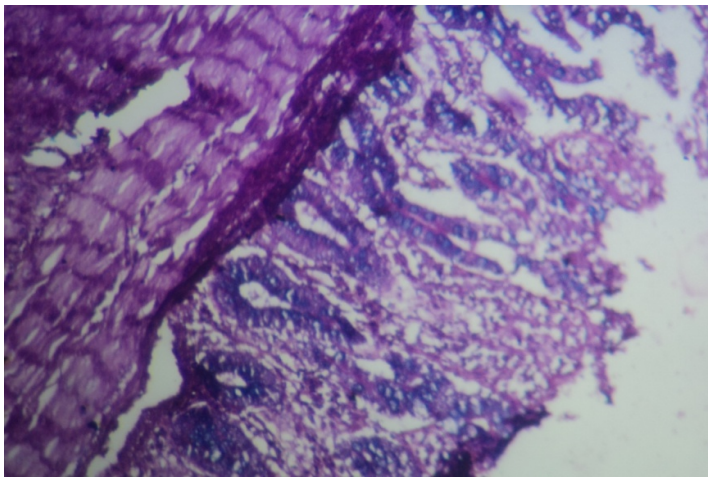

Fig. 133

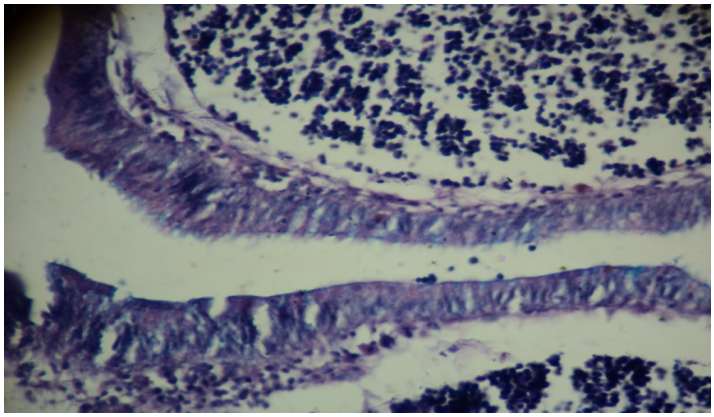

Fig. 134

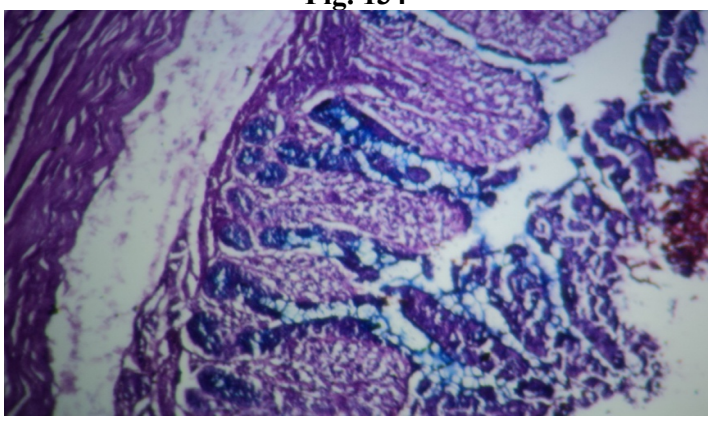

Fig. 135

Fig.136 **Section of fowl infundibulum (oviduct) infected with *Prosthogonymus pellucidus* revealing positivity for acid mucopolysaccharide substances in the epithelium. Combined Alcian blue PAS. X280.**

Fig.137 **Section of fowl infundibulum (oviduct) infected with *Prosthogonymus pellucidus* revealing a mucosal mast cell reaction. Toluidine blue. X300.**

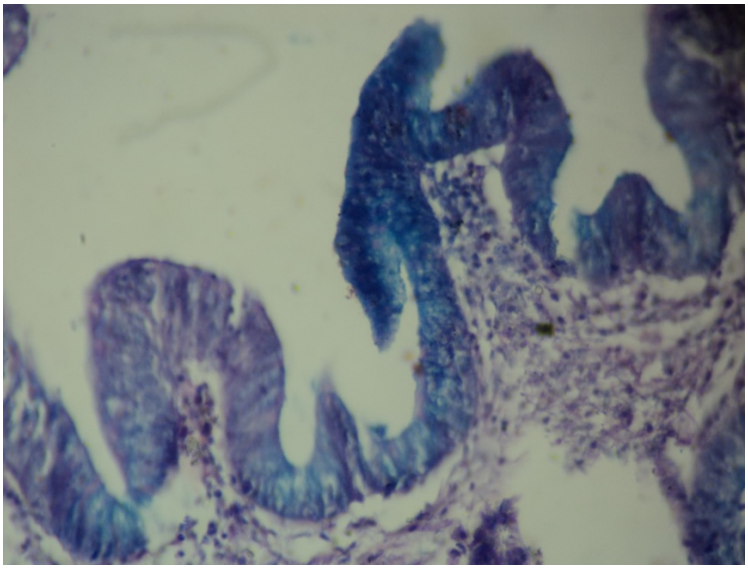

Fig. 136

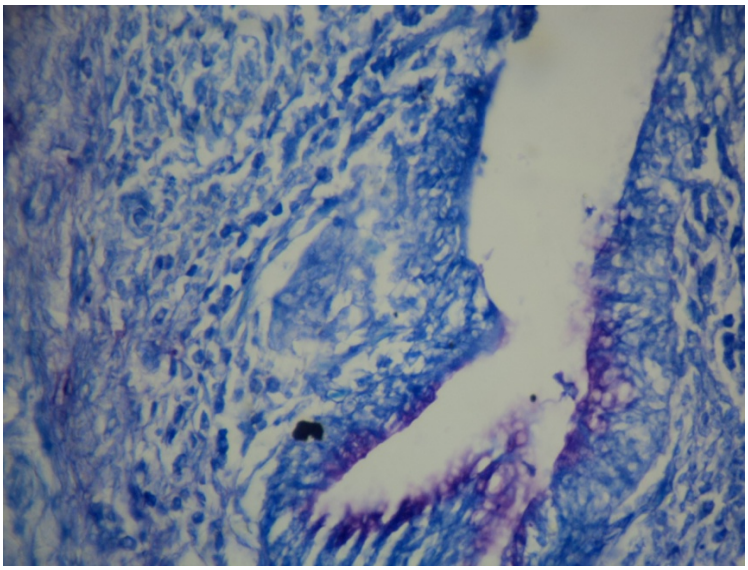

Fig. 137

cestode infections. However significant decrease in total protein and albumin contents was recorded in case of mixed infections.

Table 5: Haemato-bioiochemical analysis of blood/serum from domestic fowls (mean±S.E)

Biochemical parameters	Uninfected (n=6)	Nematode infection (n=40)	Cestode infection (n=11)	Mixed Infection (n=43)
Haemoglobin (g%)	11.08±0.486	10.06±0.280	11.96±0.598	9.66±0.222
Packed cell volume (%)	38.67±0.843	34.30±0.626	36.18±0.932	32.62±0.566
Total Erythrocyte count (million/cumm)	3.32±0.278	2.90±0.137	3.70±0.187	3.16±0.139
Total leukocyte count (thousand/cumm)	32.67±2.679	34.40±3.047	30.73±4.300	44.36±2.964
Erythrocyte sedimentation rate (mm)	1.33±0.211	1.10±0.090	1.05±0.196	1.39±0.111
Heterophil (%)	31.33±0.333	30.10±0.151	31.09±0.251	30.09±0.104
Lymphocyte (%)	49.77±0.167	49.88±0.141	50.82±0.263	50.81±0.064
Monocyte (%)	10.67±0.333	11.52±0.098	11.64±0.152	10.03±0.074
Eosinophil (%)	5.83±0.167	7.02±0.107	4.36±0.152	7.09±0.064
Basophil (%)	2.40±0.159	1.78±0.067	2.09±0.251	2.14±0.152
Total protein (g/dl)	4.85±0.338	4.32±0.070	4.23±0.207	4.22±0.124*
Albumin (g/dl)	1.69±0.177	1.39±0.032	1.57±0.213	1.36±0.050
Globulin (g/dl)	3.16±0.356	2.90±0.078	2.65±0.248	2.87±0.124
Albumin:Globulin ratio	0.58±0.106	0.48±0.019	0.64±0.148	0.55±0.061

(The mean difference is significant at the 0.05 level.)

CHAPTER – 5

DISCUSSION

5.1 Prevalence

For evaluating the prevalence of gastrointestinal helminth parasites in the domestic fowl, sample size of 100 birds was screened for different types of parasites over a period of six months. The various types of parasites recovered include nematodes viz. *Ascaridia galli*, *Heterakis gallinarum, Capillaria annulata, Acuaria hamulosa* and *Dysphrynx spiralis*, cestodes viz. *Raillietina cesticillus, Raillietina tetragona, Hymenolepis carioca, Amoebotaenia sphenoides* and *Choanotaenia infundibulum,* and a single species of trematode viz. *Prosthogonymus pellucidus*.

The present study revealed that 94 per cent of the domestic fowl examined were found to be infected with the gastrointestinal helminth parasites. Similar observations have been made by earlier workers in backyard poultry in various parts of the world (95.7% by Fatihu *et al*., 1991 in Zaria, Nigeria; 95% by Msanga and Tungaraza, 1985 in Mwanza, Tanzania; 94.5% by Hassan, 1966 in Pakistan; 94.3% by Ayeni, 1973 in Nigeria; 93.7% by Mpoame and Agbede, 1995 in Western Cameroon; 96% by Eslami *et al*., 2009 in Iran). However, comparatively lower prevalence has been reported by various workers in India (90.9% by Yadav and Tandon, 1991 in India; 79.9% by Bali and Kalra, 1975 in Punjab, India; 76.5% by Virk *et al*., 1987 in Chandigarh, India; 74.3% by Gogoi, 1975 in Assam, India; 65.7% by Matta and Ahluwalia, 1981 in Uttar Pradesh; 74% by Fotedar and Khateeb, 1986 in Kasmir, India).

The high prevalence of helminth infections indicates that virtually almost all free range chickens are subclinically infected. According to Muchadeyi *et al*. (2004) and Mwale and Masika (2009) village chickens are raised mainly under the free-range (scavenging) production system, with partial or no housing and this predisposes the chickens to diseases and parasites especially helminths (Swatson

et al., 2003). Losses due to this high morbidity might be considered of greater economic importance than the high counts that cause mortality in a few birds.

Different workers from India and abroad have reported varying rates of prevalence of individual parasites. In the present study *Heterakis gallinarum* was found to be most prevalent parasite as seen by some earlier workers (Permin *et al*., 1997; Mwale and Masika, 2010) however lesser prevalence has been observed by various researchers (Pandit, *et al*., 1991; Eshetu, *et al.,* 2001; Nithiuthai *et al*., 2003; Ashenafi and Eshetu, 2004; Hassouni and Belghyti, 2006; Abdelqader *et al*., 2008). The high prevalence observed in free range chickens could be attributed to compounding effects of management, chicken breeds and agroecological zone and, therefore, could be explained after conducting seasonal prevalence study. A prevalence of 41 per cent *Ascaridia galli* infection, observed in present study was in congruence with observations made earlier by Luka and Ndams, (2007), Huq (1986) and Samad *et al.*, (1985), who recorded a prevalence of 43, 45 and 45 per cent respectively. Compatible prevalence has been found for *Capillaria annulata* by Islam and Shaikh (1967) and Huq (1986), who reported prevalence 30 and 32 per cent respectively. Lower prevalence for *Acuaria hamulosa* and *Dysphrynx spiralis* were in agreement with the findings of Eshetu *et al.,* 2001; Ashenafi and Eshetu, 2004; Hassouni and Belghyti, 2006; Salam *et al*., 2009.

The prevalence of *Raillietina cesticillus* (21%), observed presently, is comparable to 23 and 19 per cent observed by Salam *et al*., (2010) and Ashenafi and Eshetu (2004) respectively, in Kashmir, India. With regard to *Raillietina tetragona* higher prevalence has been reported by Eshetu *et al.* (2001), Ashenafi and Eshetu (2004) and Luka, and Ndams (2007) who observed prevalence of 45.69 and 35.8 and 23.9 per cent, respectively. The variation observed in the present study might be attributed to seasonal variation in number and activity of the arthropod intermediate hosts. Similar causes may explain the higher prevalence observed for *Hymenolepis carioca* than earlier reports (Nithiuthai *et*

al., 2003; Hassouni and Belghyti, 2006). Prevalence of 6.69 per cent has been observed for *Amoebotaenia sphenoides* by Salam *et al.* (2009) in Kashmir, India which is comparable to prevalence observed in the present investigation. However, prevalence of 40.45 and 37.4 per cent has been observed by Eshetu *et al.* (2001) and Ashenafi and Eshetu (2004). The fairly low prevalence finding for *Choanotaenia infundibulum* is concurrent with the findings by Eshetu *et al.* (2001); Ashenafi and Eshetu (2004); Luka and Ndams (2007).

In the present study, only one trematode species *Prosthogonymus pellucidus* with a prevalence of 5 per cent was seen. Similar observation was recorded by previous workers (Ssenyonga, 1982; Jenkins, 2007; Abdelgader *et al*., 2008) who noted a few or no trematodes in local chickens. As reported earlier that in most cases of trematode infections the prevailing environmental conditions might not be conducive for the perpetuation of their intermediate hosts (Junker and Boomker, 2007; Abdelgader *et al*., 2008). Thus, the life cycle of the parasites is rarely completed. In addition, trematodes are more important parasites of wild water-fowl, domesticated ducks, geese and not chickens (University of Reading, 2007).

5.2 Association of gastrointestinal helminthosis with body condition scoring

In present study birds with lesser occurrence and intensity of helminth infections had good body condition score when compared with birds having medium and lower body condition scores who had moderate to severe infection. Definitely a trend was observed that the intensity of parasitic infections increased in birds fom good to low body conditions. Similar observations have been reported by earlier workers who opined that the reduced body condition is probably a reflection of the weight loss due to helminth infections (Reid and Carmon, 1958; Ikeme, 1971; Ackert and Herrick, 1928). They explained that in moderate to severe infection the parasite may cause retarded performance and growth, reduced quality of meat as well as live weight loss. Association of higher

worm burden with lower body condition score may also be explained in terms of immunocompetance of animals. Lowering of body condition due to any stress factor may compromise the immune system of the animal which when infected favours development of higher number of worms. However, elucidation of cause-effect mechanism warrants further investigations. However with regard to prevalence of parasitic infections in birds having different body condition no significant differences were observed.

5.3 Age and sex-wise distribution of parasitism

The higher prevalence (100%) of helminth infections in growers than in adult birds (90%) might be due to susceptibility of young and resistance of adult birds. Previous studies have strongly suggested that prevalence is heavily influenced by age (Magwisha *et al*., 2002; Ackert *et al.*, 1935a). This study also showed higher worm burdens of pathogenic helminths in growers as reported earlier (Soulsby, 1982). The presence of these species in moderate numbers might not be harmful, but in large numbers may be detrimental to the host.

The absence of an association between the prevalence of helminth infections and the sex of the host is largely in agreement with reports by Mpoame and Agbede (1995) and Poulsen *et al*. (2000), indicating that there is not usually a natural affinity of helminth species to either sex of the host.

5.4 Average worm burdens pattern of helminthosis

In the present study the average worm burdens for some of the helminths were significantly higher and majority of birds (68%) harboured multiple helminth species, which was similar to the findings of Shamsul-Islam (1985), Fakae *et al.* (1991), Yadav and Tandon (1991), Mpoame and Agbede (1995), Permin *et al*. (1997), Poulsen *et al*. (2000), and Magwisha *et al*. (2002). Multiple helminth infections in rural chickens indicate that the prevailing environmental conditions and the management systems in the free range are favourable for the

simultaneous development of different helminth species (Kabatange and Katule, 1990; Pandey *et al.*, 1992)

5.5 Pathology

5.5.1 Pathology of nematodes

5.5.1.1 *Ascaridia galli*

Ascariasis caused by *Ascaridia galli* in domestic fowl has been studied by various workers (Padhi *et al.,* 1987; Verma *et al.*, 1993; Dahl *et al.*, 2002; Arunachalam *et al.*, 2003; Permin *et al.*, 2006). As observed in the present study, the parasites are mostly recovered from the lumen of small intestine without causing much gross pathology. However, heavy infections have been reported to cause severe changes. The pathomorphological changes are in agreement with the earlier observations with comparable parasite loads (Padhi *et al.,* 1987; Arunachalam *et al.*, 2003).

5.5.1.2 *Heterakis gallinarum*

Heterakis gallinarum has been considered of low pathogenicity in single infections (Lund and Chute, 1973). However more severe pathology has been associated with the parasite in domestic galliformes, causing nodular typhilitis, with the formation of inflammatory or granulomatous caecal nodules (Kaushik and Deorani 1969; Riddel and Gajadhar, 1988; Khan *et al.*, 1994). Although in the present study, gross nodular lesions were not evident, histopathology revealed nodular aggregation of mononuclear cells as well as parasitic sections in the mucosa. Erosive lesions as seen in the present study have also been recorded by Nath (1963) who opined that eroding habit of *Heterakis gallinarum* imparts greater pathogenic significance to this nematode.

5.5.1.3 *Capillaria annulata*

In light infections the wall of the crop and oesophagus was slightly thickened and inflamed, but this extended to a marked thickening, inflammation

with mucopurulent exudates in the lumen. Similar findings have been reported by Soulsby (1982).

Histopathological lesions were presence of sections of the *Capillaria annulata* in the squamous epithelial layer of oesophagus and crop (Hung, 1926), sloughing of the mucosa and necrotic changes in squamous epithelium (Cram, 1936).

5.5.1.4 *Acuaria hamulosa*

Acuaria hamulosa has been reported worldwide in various galliformes (Freitas and Hipolito, 1949; Grisi and Carvalho, 1974; Padhi *et al.,* 1987; Menezes *et al*., 2003), there are scanty reports of pathological studies (Mohammed and Sokkar, 1979; Padhi *et al.*, 1987; Fatihu *et al.*, 1991). The pathological changes observed in the current study were very severe and in accordance with the earlier pathological descriptions by Srivastava (1939), Whur (1966), Nath and Pande (1963) and Padhi *et al.* (1987) who recorded the formation of soft nodules inside the musculature of the caudal lobe of the gizzard and destruction of the Koilin layer in heavy infection. However, in the present study, the mononuclear infiltration was more severe and formed follicular aggregates. Severe eosinophilic reaction and mucosal changes were additionally recognized.

5.5.1.5 *Dysphrynx spiralis*

The pathological changes in the proventriculus of fowl due to *Dysphrynx spiralis* infection showed that the parasite is pathogenic and causes catarrhal inflammation, hypertrophy of wall of the organ, petechial haemorrhages and desquamation of the epithelium, edema, slight congestion and proventriculitis. The pathological lesions, as recorded in the present investigation are consistent with the findings of previous researchers (Soulsby, 1965; Joshi and Kamalapur, 1971). Adenomatous proliferative changes in the proventriculus as reported by Ramaswamy and Sundaram, (1985) in fowls given monospecific experimental

infection with *Acuaria spiralis* were not observed in the present study. Desi birds may be more resistant to the infection than non native breeds of the birds (Joshi and Kamalapur, 1971).

5.5.2 Pathology of cestodes

5.5.2.1 *Raillietina cesticillus*

Raillietina cesticillus infection as observed in fowls in the present study was characterized by desquamation of epithelium, degeneration and broadening of intestinal villi, increased vascularity and glandular disintegration of varying degree. Similar changes but of varying degree have been reported by other workers (Biester and Schwarte, 1957; Nath and Pande, 1963; Jha *et al.,* 1981). The mucosal changes have been attributed to the mechanical damage. The infiltration of neutrophils around the mucosal lesions, as observed in the present study had also been reported by Nath and Pande (1963). Some authors reported mononuclear infiltration of lamina propria also and opined that heterophilic infiltration was not a primary reaction but only secondary to bacterial invasion of the mucosal lesions (Gray, 1975; Jha *et al.,* 1981, Bhowmik *et al.,* 1982).

5.5.2.2 *Raillietina tetragona*

The pathological changes due to *Raillietina tetragona* observed in current study were in accordance with earlier reports but were less severe in nature. In the present study, the scolices were found penetrating only superficial mucosa. Jha *et al.* (1981) reported suckers reaching up to muscular layer. Also various authors have reported much severe lesions including nodule formation, closely resembling to tuberculous lesions (Ackert and Case, 1938). However Schwartz (1957) opined that such lesions were rarely associated with *Raillietina tetragona* and are more characteristic of *Raillietina echinobothrida*. Jha *et al.* (1981) could not find such lesions in poultry. Nath and Pande (1963) also reported that scolices of *Raillietina tetragona* were not found to burrow deeper beyond the mucosa. The observed discrepancies in the severity of lesions could be attributed to low parasite load as

recorded in the present study. The lesions observed in the present study were indicative of mechanical damage due to sucker armature. It has been reported that sucker hooks, inserted on the mucosal tissue, exercised a prominent point in affecting a strong hold on the lining than the rostellar hooks which seemed to have a secondary role in attachment (Nath and Pande, 1963). The cellular reaction observed in the present study, although was mild but is in accord with earlier reports (Nath and Pande, 1963; Jha *et al.*, 1981).

5.5.2.3 *Hymenolepis carioca*

Absence of gross lesions in *Hymenolepis carioca* infection as observed in the present investigation is highly consistent with the findings of previous researchers (Padhi *et al*., 1986 and Bybee, 1996).

Histopathological observations were partially in line with the observations of the earlier studies by various workers ((Padhi *et al*., 1987; Bhowmik and Sinha, 1983). Some variation in the pathological lesions could be attributed to the degree of resistance of different breeds of poultry to cestode infection.

5.5.2.4 *Amoebotaenia sphenoides*

The pathological changes observed with *Amoebotaenia sphenoides* infection in fowl were in agreement with earlier reports (Nath and Pande, 1963; Jha *et al.,* 1981). The parasites have been frequently reported free in the lumen and also attached to the lining. The mucosal damage has been attributed to dragging of epithelial tissue by the parasite sucker.

5.5.2.5 *Choanotaenia infundibulum*

Infection with *Choanotaenia infundibulum* was associated with mild degree of catarrhal enteritis which has also been observed by Salam (2009).

Histoathological examination revealed desquamation of epithelium, enteritis, necrosis of villi as reported earlier (Anwar *et al*., 2000 and Salam, 2009). One of the histopathogical finding was hypertrophy and hyperplasia of smooth

muscles of the intestines which can be attributed to presence of relatively large intensity of worms.

5.5.3 Pathology of trematode

5.5.3.1 *Prosthogonymus pellucidus*

Histopathological examination revealed degeneration and exfoliation of the mucous epithelium of *Bursa of Fabricius* in addition to the stratification of the mucous epithelium and interstitial cell infiltration. Similar findings have been reported by Leok *et al.* (2002) in *Prosthogonimus ovatus* infection in Indonesian native chickens. Moderate degree of lymphoid hyperplasia was observed which could be attributed to highly lymphoid nature of the bursa and spiny cuticle of the parasite leading to mechanical irritation and consequent hyperplasia.

5.6 Histochemistry

Histochemical investigations revealed positive reaction for neutral mucopolysaccharide the basement membranes while infiltrating cells, glandular epithelium, goblet cells, glandular secretions and hyperplastic cells were positive for acid mucopolysaccharides. These findings were in consonance with that of Tuli *et al*. (1992) who reported increased alcian blue positive material near the parasitic infestation in the mucosa indicating degenerative and necrotic changes. Various authors have opined that hypersecretion of mucopolsaccharides in and around the lesions may be attributed to prolonged irritative action of insults. Qualitative increase in both acid and neutral mucopolysaccharides has been attributed to inflammatory process (Darzi *et al.,* 2003; Shah, 2009). Darzi *et al.* (2003) reported that the inflammatory exudates revealed qualitative increase in acid mucopolysaccharides and the basement membrane positive for neutral mucopolysaccharide in spontaneous hepatic coccidiosis in rabbits and opined that it constitutes a component of local defense reaction.

Mucopolysaccharides especially acidic types were found to be increased in all helminth infections of entire gastrointestinal tract. This could be attributed to

increase in the number of mucous cells close to the site of parasite attachment. Therefore, Combined Alcian blue and periodic acid-Schiff's staining of representative histological sections revealed a significant increase in the number of mucous cells staining positively for acid glycoconjugates compared to the number of cells found in the gastrointestinal tracts of uninfected birds. Similar results have been obtained by Dezfuli *et al*. (2010) who found number of mucous cells close to the site of parasite attachment within the intestine significantly higher than the number detected in uninfected individuals and in infected individuals at sites 1cm or greater from the point of parasite attachment staining positively for acid glycoconjugates. Hyperplasia of Paneth and intermediate cells is a recently described component of the response of the small intestine of mice to infection with the nematode Trichinella spiralis (Kamal *et al*., 2002).

Increased production of mucopolysaccharides might also be attributed to their probable role in the inflammation (Darzi *et al.*, 2003; Shah, 2009) and prolonged irritative action of different insults, which are believed to determine hypersecretion of these substances (Lupu, *et al*., 1959).

Toludine blue stained sections revealed metrachomsia, and the mast cells were prominent in the submucosa and mucosa in *Heterakis gallinarum, Capillaria annulata, Raillietina cesticillus and Prosthogonymus ovatus* infections. Mast cells have been suggested to be major effector cells in the immune response to infection with helminths (Lee *et al.,* 2003). Occurrence of mast cell reaction as demonstrated by toluidine blue staining is an indication of local type-1 hypersensitivity and has also been frequently observed in parasitic infections (Darzi *et al.,* 2003). Expulsion of intestinal parasites is temporally associated with an increase in the number of mast cells in the intestine and secretion of mast cell proteases and leucotrienes into the tissues and serum (Grencis, 1997). This mastocytosis is dependent on both T cell-derived cytokines (IL-3, IL-4, IL-9 and IL-10) and T cell-independent factors stem cell factor (SCF).

5.7 Haemato-biochemical analysis

Parasites are a source of physiological stress for their hosts. Effects of parasites on their host have been frequently reported and a vast literature exists on host parasite interactions in birds (Loye and Zuk, 1991; Clayton and Moore, 1997). In any case parasites impose undoubtedly a drain of resources upon their hosts thereby forcing them to evolve abilities to reduce these costs by mounting effective immune and/or stress responses. Such responses may include changes in white blood cell profiles (Graczyk *et al*., 1994; Ots and Horak, 1998), total serum proteins (Delope *et al*., 1998; Ots and Horak, 1998) and antibody titers in serum (Ots and Horak, 1998). A significant decrease in haemoglobin values and packed cell volume with a significant increase in total leukocyte counts in infected birds have been reported by various workers (Sharma *et al*., 1984; Samad *et al*., 1986; Sekar *et al*., 1986; Verma, *et al*., 1993; Phukan, 2006; Deka and Borah, 2008). Increase in lymphocyte, monocyte and eosinophil counts are the hallmarks of parasitic infections. (Sharma *et al*., 1984; Verma *et al*., 1993 and Phukan, 2006).

Results obtained in the plasma biochemical study of the domestic fowls clearly revealed that on comparing the biochemical values of uninfected birds with the infected ones, no significant variations were recorded in the total protein, albumin (A), globulin (G) and A/G ratio in case of pure nematode or cestode infections and this was in accordance with the findings of Verma *et al*. (1993) who found no statistical variation between the control and *Ascaridia galli* infected birds. However, significant decreases in the total protein and albumin contents were recorded in case of mixed nematode and cestode infections coinciding with the findings of Sheikh (2009). The reason behind the significant decrease in the total plasma proteins and albumin in mixed parasitic infections might be affecting the absorption and assimilation of small molecules such as amino acids on one hand and also by compound effect on absorption by multiple parasites from the hosts as noted earlier.

CHAPTER – 6

SUMMARY AND CONCLUSION

The present was study aimed to investigate the prevalence, pathology and haemato-biochemical changes caused by the parasitic infections in domestic fowl in Ganderbal district of Kashmir valley. A total of 100 randomly selected domestic fowl were screened for the purpose. For sampling purposes the birds were categorized into growers aged 12-24 weeks and adults aged 32 weeks or above. The study revealed an overall prevalence of 94 per cent. The prevalences of pure nematode, pure cestode and mixed infections were 40, 11 and 43 per cent, respectively.

11 species of parasitofauna recovered from the domestic fowl with their prevalences include *Heterakis gallinarum* (63%), *Ascaridia galli* (41%), *Capillaria annulata* (35%), *Raillietina cesticillus* (21%), *Raillietina tetragona* (10%), *Hymenolepsis carioca* (19%), *Amoebotaenia sphenoides* (8%), *Choanotaenia infundibulum* (7%), *Acuaria hamulosa* (6%), *Prosthogonymus pellucidus* (5%) *and Dysphrynx spiralis* (1%). Prevalence of *Heterakis gallinarum Ascaridia galli* and *Capillaria annulata* did not vary significantly but significant variations of these parasites was observed with regard to other species of parasites.

Assessment of different parasitic infections in association with body condition of the birds revealed that the intensity of parasitic infections were more frequent and more severe in birds whose general body condition was low. However prevalence of parasitic infections did not vary significantly with the body condition of the birds. Higher prevalence (100%) of helminth infections was observed in growers than in adult birds (90%) nevertheless this difference was statistically non-significant. There was no association between the prevalence of helminth infections and the sex of the host.

The studies on histopathology clearly revealed that there were marked

pathological changes caused by the parasites in the domestic fowl. Among nematode infections, the infection of *Ascaridia galli* was associated with mucous enteritis. The intestinal wall appeared to be thickened with mucosa giving a velvety appearance. Histopathological changes were moderate goblet cell hyperplasia, compression of villi, disruption of villi and desquamation of epithelium, broadening and clubbing of the villi, focal necrosis and submucosal edema. Mild inflammatory reaction was characterized mainly by infiltration of mononuclear cells and a few polymorphonuclear cells including eosinophils. Sections of the degenerated parasites were also noticed in the lumen. The infection of *Heterakis gallinarum* was associated with the thickened caecal wall. Histological findings in the caeca were represented by the presence of eosinophilic sections of worms in the lumen as well as in the wall of mucous membrane, intense chronic diffuse typhlitis with mononuclear and polymorphonuclear (heterophils) leucocyte infiltrations. Some of the stained sections showed extensive damage of the mucosal lining with erosions, goblet cell hyperplasia, oedema of the caecal wall, necrosis and preponderance of necrotic material in the caecal lumen. *Capillaria annulata* was found in the oesophagus and crop. In light infections the wall of the crop and oesophagus was slightly thickened and inflamed, but this extended to a marked thickening, inflammation with mucopurulent exudates in the lumen. Histological sections of the *Capillaria annulata* were found in the squamous epithelial layer of oesophagus and crop. Histopathology revealed oesophagitis and ingluvitis, sloughing of the mucosa containing parasitic ova, necrotic changes in squamous epithelium characterized by pyknotic nuclei. A thin connective tissue capsule surrounded the adult worms and everywhere there were infiltrates of lymphocytes and mononuclears. Oesophageal glands were inflamed and sometimes cystic. In some sections acanthotic changes and enlargement of lymphoid follicles was observed. Proventricular capillariasis was characterized by proventriculitis, desquamation of the epithelial cells into the common cavities of the lobules and cystic proventricular glands. *Acuaria hamulosa* worms were found deeply embedded in

the musculature of the caudal lobe of the gizzard. The musculature of the caudal lobe of the gizzard revealed soft yellowish nodules. Histopathology revealed discrete nodular lesions in the musculature. Cellular reaction in the lesions was characterized by large number of lymphocytes, monocytes, plasma cells, heterophils and few eosinophils. The mucosa and submucosa showed marked thickened and diffuse mononuclear infiltration.

Cestodes were found especially inhabiting duodenum and jejunum parts of the intestine. In general cestodes were found attached to the mucosa with the help of rostellum. Intestinal lumen was full of pasty mucous containing proglottid segments. The mucosa appeared to be rough. Histopathological examination revealed broadening of villi which at places were fused to give much broader appearance. Scolices of the cestodes were found embedded in the superficial mucosa. The mucosa revealed mechanical disintegration of the lining epithelium and glands in the vicinity of the scolices. Inflammatory reaction around the lesions was characterized by lymphocytes and heterophils. The pathological changes in the proventriculus of fowl due to *Dysphrynx spiralis* were hypertrophy wall of the organ, petechial haemorrhages, desquamation of the epithelium, edema, slight congestion and proventriculitis.

Prosthogonymus pellucidus was found to inhabit bursa of fabricius and coloaca .Histopathological examination revealed degeneration and exfoliation of the mucous epithelium of *Bursa of Fabricius*, interstitial cell infiltration, moderate degree of lymphoid hyperplasia and congestion.

Histochemical investigations revealed positive reaction for acid mucopolysaccharide in infiltrating cells, glandular epithelium, goblet cells, glandular secretions and hyperplastic cells. Toluidine blue stained sections revealed metrachomsia, and mast cells were prominent in the submucosa and mucosa in *Heterakis gallinarum, Capillaria annulata, Raillietina cesticillus and Prosthogonymus ovatus* infections. Haematologiical studies revealed a significant decrease in haemoglobin values and packed cell volume with a

significant increase in total leukocyte, lymphocyte, monocyte and eosinophil counts in infected birds. Biochemical analysis revealed significant decreases in the total protein and albumin contents in case of mixed nematode and cestode infections.

Conclusions:

- The present study revealed high prevalence of gastrointestinal helminth parasites which is indicative of the conducive environmental conditions for helminthosis in Kashmir valley.
- *Heterakis gallinarum* (63%), *Ascaridia galli* (41%) and *Capillaria annulata* (35%) are the three most prevalent helminth species of domestic fowl in Ganderbal district of Kashmir valley.
- Backyard poultry is in the high risk of helminth infections which are associated with the development of pathological changes. Therefore, they have a serious economic impact on the production of domestic fowl.

FUTURE SUGGESTIONS

- Extensive systematic studies on parasitofauna of various avian species and migratory birds need to be conducted.
- Morphological characterization of avian parasitofauna prevalent in the area need to be carried out on large scale.
- Rigorous monitoring and surveillance of helminthosis in backyard poultry.
- Long term cohort studies are needed to investigate the impact of these helminth infections on productivity and mortality in commercial systems as well as in backyard systems.
- Regular deworming is essential in backyard poultry birds to obtain better production from them.

- Elucidation of descriptive pathogenesis following experimental studies in definitive hosts and intermediate hosts.
- Antigenic characterization of the various developmental stages of the parasites.
- Cohort studies evaluating the genetic susceptibility/ resistance to the infection or disease development.

LITERATURE CITED

Abdelqader, A., Gauly, M., Wollny, C.B. and Abo-Shehada, M.N. 2008. Prevalence and burden of gastrointestinal helminthes among local chickens, in northern Jordan. *Preventive Veterinary Medicine* **85**(1-2):17-22.

Ackert J.E. and Herrick C.A. 1928. Effects of the nematode *Ascaridia lineata* (Schneider) on growing chickens. *The Journal of Parasitology* **15**:1-15.

Ackert J.E., Porter D.A. and Beach T.D. (1935a). Age resistance of chickens to the nematode *Ascaridia lineata* (Schneider). *Journal of Parasitology* **21**: 205-213.

Ackert, J.E. and Case, A.A. 1938. Effects of tapeworms. *Raillietina cesticillus* (Molin) on growing chicken. *Journal of Parasitology* **24**:14-19.

Adang, K. L., Abdu, P.A., Ajanusi, J.O., Oniye, S.J. and A.U. Ezealor, A.U. 2010. Histopathology of *Ascaridia galli* Infection on the Liver, Lungs, Intestines, Heart, and Kidneys of Experimentally Infected Domestic Pigeons (*C. l. domestica*) in Zaria, Nigeria. *Iranian Journal of Parasitology* **13** (3):27-32.

Adene, D.F. and Dipeolu, O.O. 1975. Survey of blood and ectoparasites of domestic fowls in Ibadan, Western State if Nigeria. *Bulletin of Animal Health and Production in Africa* **23**: 333-335.

Anwar A. H., Rana S.H., Shah A.H., Khan M.N. and Akhtar M.Z. 2000. Pathology of cestode infection in indigenous and exotic layers. *Pakistan Journal of Agricultural Scinces* **37**(1-2): 93-95.

Arunachalam, K., George, C.V. and Manu Mohan, B. 2003. Histopathological changes in broiler chicken experimentally infected with *Ascaridia galli, Journal of Veteirnary Parasitology* **17**(2): 155-157.

Ashenafi, H. and Eshetu, Y. 2004. Study on gastrointestinal helminthes of local chickens in Central Ethiopia. *Revue Med. Vet.* **155**(10): 504-507.

Ayeni, A.O. 1973. Internal parasites of livestock in the Western State of Nigeria. Occurrence of helminth parasites in local chickens. *Nigeria Agriculture Journal* **10**: 46-47.

Bali, H.S. and Kalra, I.S. 1975. Studies on the incidence of helminths in domestic and wild birds in Punjab State. *Journal for Research in Punjab Agricultural University* **12**(3): 313-316.

Bancroft, J.D. 1975. Histochemical techniques. *2nd ed*. Butterworths London.

Bawe, N. M., Joseph, A.O., Idris, A. R. and Esievo, K.A.N. 2005. Observation of lesions associated with gastrointestinal parasites of guinea fowls (*Numida meleagris*) in Zaria Nigeria. ISAH-Warsaw, Poland, **2**:511-513.

Berghen, P. 1966. Serum protein changes in *Capillaria obsignata* infections. *Expt. Parasitol*, **19**: 34-41.

Bernard, F.F., Joseph, G.Z. and Jain, N.C. 2000. Schalm's Veterinary Hematology. 5th Ed. Lippincott Williems and Wilkins, Philadelphia.

Benjamin, M.M. 1985. Outline of veterinary clinical pathology. 3rd Ed. The Iowa State Uni. Press, Ames.

Bhowmik, M.K., Sinha, P.K. and Chkraborty, A.K. 1982. Studies on the pathobiology of chicks experimentally infected with *Raillietina cesticillus* (cestode). *Indian Journal of Poultry Science* **17**: 207-213.

Bhowmik, M.K. and Sinha, P.K. 1983. Studies on the pathology of taeniasis in domestic fowl. *Indian Veterinary Journal* **60**:6-8.

Biester, H.E. and Schwarte, L.H. 1965. Diseases of Poultry, Iowa State College Press, Ames Iowa, pp. 1382.

Brar, R.S., H.S. Sandhu, and Singh A. 2002. Veterinary clinical diagnosis by laboratory methods. 1st Ed. Kalyani Publishers, New Delhi.

Bybee, A. 1996. Nematodes (roundworms) and cestodes (tapeworms) in poultry. *Pluimvee Poultry Bulletein* **7** : 350-351.

Calnek, B.N, Barnes, H.J, Beard, C.W, McDougald L.R. and Saif, Y.M. 1997. Diseases of Poultry. Ames, Iowa, USA: Iowa State University Press.

Clayton, D.H. and Moore, J. 1997. Host parasite evolution. General principles and avian models. Oxford University Press, Oxford.

Cram, E.B. 1936. Species of *Capillaria* parasite in the upper digestive tract of birds. U.S.D.A. Bull. No. **516**, Washington, D.C.

Creevey, L.E. 1991. Supporting small-scale enterprises for women farmers in the Sahel. *Journal of International Development* **3**(4):355-386.

Dahl, C., Permin, A., Christinsen, J.P., Bisgaard, M., Muhairwa, A.P., Petersen, K.M.D., Poulsen, J.S.D. and Jensen, A.L. 2002. The effect of concurrent infections with *Pasteurella multocida* and *Ascaridia galli* on free range chickens. *Veterinary Microbiology* **86**: 313-324.

Darzi, M.M., Mir, M.S. and Khan, M. 2003. Concurrent anthracosis and parasitic pneumonia in a sheep. *SKUAST Journal of Research* **5**:213-216.

Dehlawi, M.S. 2007. The Occurrence of Nematodes in the Intestine of Local (Baladi) Chicken (*Gallus gallus domesticus*) in Jeddah Province-Saudi Arabia. *Scientific Journal of King Faisal University* **8**(2): 61-71.

Deka, K. and Borah, J. 2008. Haematological and biochemical changes in Japanese quails and chickens due to Ascaridia galli infection. *International Journal of Poultry Science* **7**(7): 704-710.

Dellope, F., Moller A.P. and de la cruz, C. 1998. Parasitism, immune response and reproductive success in the house martin. *Oecologia* **114**: 188-193.

Denmark, H.A. and Cromroy, H.L. 2006. Tropical Fowl Mites, Ornithonyssus Bursa (Berlese) Gainesville, Fla, USA: Institute of food and Agricultural Sciences, University of Florida.

Dezfuli, B.S., Pironi, F. and Campisi1, A.P. 2010. The response of intestinal mucous cells to the presence of enteric helminths: their distribution, histochemistry and fine structure. *J. of Fish Diseases*. **33**(6): 481–488.

Eslami, A., Ghaem, P. and Rahbar, S. 2009. Parasitic Infections of Free –Range Chickens from Golestan Province, Iran. *Iranian Journal of Parasitology* **4**(3):10-14.

Eshetu, Y., Mulualem E., Ibrahim, H., Berhanu, A. and Aberra, K. 2001. Study of gastro-intestinal helminths of scavenging chickens in four rural districts of Amhara region, Ethiopia. *Rev. Sci. Tech. Off. Int. Epiz.* **20**(3): 791-796.

Fakae B.B., Umeorizu J.M. and Orajaka L.J.E. 1991. Gastrointestinal helminth infections of the domestic fowl (*Gallus gallus domesticus*) during the dry season in Eastern Nigeria. *Journal of African Zoology* **105**: 503-508.

FAO, (Food and Agricultural Organization of the United Nations) Tech. Rep. 274415. Rome, Italy: FAO; March (1987). Report on the expert consultation on rural poultry development in Asia, Dhaka, Bangladesh.

FAO, 1997. Special programme for food security. Diversification component. Draft report, Rome.

Fatihu, M.Y., Ogbogu, V.C., Njoku, C.O. and Saror, D.I. 1991. Comparative studies of gastrointestinal helminths of poultry in Zaria, Nigeria. *Revue D'Elevage et de Medecin Veterinaire des pays Tropicaux* **44**(2): 175-177.

Fotedar, D.N. and Khateeb, N.G. 1986. Occurrence and seasonal variation of helminth parasites of domestic fowl in Kashmir. *Indian Journal of Helminthology* **38**: 49-54.

Frantovo, D. 2000. Some parasitic nematodes (Nematoda) of birds (Aves) in the Czech Republic. *Acta Societatis Zoologicae Bohemicae* **66**(1):13-28.

Freitas, M.G and Hipolito, O. 1949. Notas de helmintologia de *Gallus gallus domesticus* em Minas Gerais. *Arquivos da Escola Superior de Veterinaria* **2**: 51-58.

Fatihu, M.Y. Ogbogu, V. C. Njoku, C. V. and Saror, D. I. 1991. Comparative studies of gastrointestinal helminths of poultry in Zaria. Revue d' E'Levage Medicine *Veterinaire pour pays Tropicaux* **44**(2):175-177.

Gogoi, A.R. 1975. Occurrence of nematodes and trematodes in local fowls in Assam. *Kerala Journal of Veterinary Science* **5**(2): 131- 134.

Graczyk, T.K., Cranfield, M.R., Shah, M.L. and Beall, F. 1994. Haematological characteristics of avian malaria cases in Afarican black footed penguins during first outdoor exposure season. *Journal of Parasitology* **80**: 302-308.

Grencis, R.K. 1997. Th2-mediated host protective immunity to intestinal nematode infections. Philosophical Transactions of the Royal Society of London. Series B: *Bio. Sci*. **352**: 1377–1384.

Grisi, L. and Carvelho, L.P. 1974. Prevalencia de helmintos parasitos de *Gallus gallus domesticus* L., no Estado do Rio de Janeiro. *Revista Brasileira de Biologia*. **34**: 115-118.

Hassan, Z. 1966. Investigation into the incidence of helminthic infestation in country fowls. *Pakistan Journal of Science* **18**(1&2): 7-9.

Hassouni, T. and Belghyti, D. 2006. Distribution of gastrointestinal helminths in chicken farms in the Gharb region-Morocco. *Parasitology Research* **99**: 181-183.

Horst, P. 1988. Native fowl as reservoir for genomes and major genes with direct and indirect effect on production adaptability. **In** : *Proceedings of the 18th World Poultry Congress*; Nagoya, Japan :156-160.

Huq, M.S. 1986. Studies on the helminth infections of poultry under rural conditions of Bangladesh. *Bangladesh Veterinary Journal* **20**(3-4): 55-60.

Ikeme, M.M. 1971. Weight changes in chickens placed on different levels of nutrition and varying degrees of repeated dosage with *Ascaridia galli* eggs. *Parasitol.* **63:** 251-260.

Islam, A.W.S. and Shaikh, H. 1967. A survey of helminth infections in the gastrointestinal tract of domestic fowl of Mymensingh district, East Pakistan. *Ceylon Veterinary Journal* **15**(3): 107-109.

Irungu, L.W., Kimani, R.N., Kisia, S.M. 2004. Helminth parasites in the intestinal tract of indigenous poultry in parts of Kenya. *Journal of the South African Veterinary Association* **75**(1):58-59.

Jenkins, M. 2007. Curbing Coccidiosis in Chickens: A Fine-tuned Approach. *Agriculture Research and Management* **55** : 2.

Jha, A.N. and Sahai, B.N. 1981. On the histopathology and histochemistry of the intestine in common poultry cestodiasis, with a note on the incidence of parasites in Patna (Bihar). *Indian Journal of Animal Science* **51**(6): 655-660.

Joshi, S.C. and Kamalapur, S.K. 1971. Incidence of *Heterakis gallinarum* (Schrank, 1788) in domestic fowls in Madhya Pradesh and observation on its histopathology. *The Indian Poultry Gazette* **55**(2): 40-43.

Junker, K. and Boomker, J. 2007. Helminths of guineafowls in Limpopo Province, South Africa. *Journal of Veterinary Reserch* **74**: 265-280.

Kabatange, M.A. and Katule, A.M. 1990. Rural poultry production systems in Tanzania.pp. 171-176. **In**: *Proceedings of an international workshop on rural poultry in Africa* (Ed. Sonaiya E.B). African Network on Rural Poultry Development, Nigeria.

Kamal, M., Dehlawi, M., Rosabrunet, L. and Wakelin, D. 2002. Paneth and intermediate cell hyperplasia induced in mice by helminth infections. *Parasitology* **125**(3): 275-81.

Kaufman, P.E., Koehler, P.G. and Butler, J.F. 2007. External Parasites of Poultry. Gainesville, Fla, USA: University of Florida institute of Food And Agricultural Sciences.

Kaushik, R. K. and Deorani, V. P. S. 1969. Studies on tissue responses in primary and subsequent infections with *Heterakis gallinae* in chickens and on the process of formation of caecal nodules. *Journal of Helminthology* **43**: 69-78.

Kekeocha, C.C. 1984. Pfizer poultry production handbook. First Edn.Pfizer Corporation, Nairobi. In association with Macmillian Publishers Limited, London and Basingstoke.

Khan, S.A., Iqbal, M. and Ashraf, S.K. 1994. Occurrence and pathology of cecal granuloma in guinea fowl (*Numida meleagris*) associated with *Heterakis gallinarum* infection. *International Journal of Animal Science* **9**: 143-145.

Kitalyi, A.J. 1998. Village chicken Production Systems in Rural Africa. Household Food Security and Gender Issues. Animal Production and Health Paper. Rome, Italy: Food and Agriculture Organisation of the United Nation.

Lee, T.D.G., Swietera, M. and Befusa, A.D. 2003. Mast cell responses to helminth infection. *Parasitology Today* **2**(7): 186-191.

Leok, C.S., Inoue, I., Sato, T., Haritani, M., Tanimura, N. And Okada, K. (2002). Morphology of the oviduct fluke, *Prosthogonimus ovatus*, isolated from Indonesian native chickens and histopathological observations of the infected chickens. *Journal of Veteirnary and Medical Science* **64**(12):1129-1131.

Loye, J.E. and Zuke, M. 1991. Bird parasite interactions. Ecology, evolution and behaviour. Oxford University Press, Oxford.

Luka, S.A. and Ndams, I.S. 2007. Gastrointestinal parasites of domestic chicken Gallus gallus domesticus Linnaeus 1758 in Samura, Zaria Nigeria. *Science World Journal* **2**(1): 27-29.

Luna, L.G. 1968. Manual of histologic staining methods of the Armed forces, Institute of Pathology, 3rd edition New York, McGraw Hill Book Company.

Lund, E.E. and Chute, A.M. 1973. Means of acquisition of *Histomonas meleagridis* by eggs of *Heterakis gallinarum*. *Parasitology* **66**: 335-342.

Lupu, N.G., Velican, D., Velican, C. and Olinescu, V. 1959. The action exerted by pneumoconiotic factors upon the acid mucopolysaccharide contents of pulmonary macrophages. *British Journal of Industrial Medicine* **16**:244.

Magwisha, H.B., Kassuka, A.A., Kyvsgaard, N.C. and Permin, A. 2002. A comparison of the prevalence and burdens of helminth infections in growers and adult free-range chickens. *Tropical Animal Health Proudction* **34**(3): 205-214.

Matta, S.C. and Ahluwalia, S.S. 1981. Note on the survey of gastrointestinal helminths of domestic fowls in Uttar Pradesh. *Indian Journal of Animal Science* **15**(10): 1013-1015.

McOrista, N., Bartona, N. J. and Jones, A. 1984. Choanotaenia spp. infestation of Australian finches (Estrildidae). *Avian Pathology*, **13** (3):479-485.

Mehta, R., Nambiar, R.G., Delgado, C. and Subramanyam, S. 2003. Annex II: Livestock Industrialization Project: Phase II-Policy, Technical, and Environmental Determinants and Implications of the Scaling-Up of Broiler and Egg Production in India. IFPRI-FAO project on Livestock Industrialization, Trade and Social-Health-Environment Impacts in Developing Countries.

Menezes, R.C., Tortelly, R., Gomes, D.C. and Pinto, R.M. 2003. Pathology and frequency of *Cheilospirura hamulosa* (Nematoda, Acuarioidea) in Gallifores hosts from backyard flocks. *Avian Pathology* **32**: 151-156.

Mohammed, M.A. and Sokkar, S.M. 1979. New observation in chickens naturally infected with *Acuaria hamulosa*. *Assiut Veterinary Medical Journal* **6**: 115-118.

Mpoame, M. and Agbede, G. 1995. The gastrointestinal helminth infections of domestic fowl in Daschang, West Cameroon. *Revue D'Elevage et de Medecin Veterinaire des pays Tropicaux* **48**(2): 147-151.

Msanga, J.F. and Tungaraza, R. 1985. The incidence of external and internal parasites of indigenous poultry in Mwanza Municipality, Tanzania. *Tanzania Veterinary Bulletin* **7**(3): 11-14.

Muchadeyi, F.C., Sibanda, S., Kusina, N.T., Kusina, J., Makuza, S. 2004. The village chicken production system in Rushinga District of Zimbabwe. *Livestock Research and Rural Development* **16**(6): 194-199.

Mwale, M., Masika, P.J. 2009. Ethno-veterinary control of parasites, management and role of village chickens in rural households of Centane district in the Eastern Cape, South Africa. *Tropical Animal Health Production* **41**: 1685-1693.

Nath, D. and Pande, B.P. 1963. A histological study of the lesions in tapeworm infestations in domestic fowl. *The Indian Journal of Veterinary Science and Animal Husbandry* **33**: 1-7.

Nithiuthai, S., Chungpivat, S. and Sukumavasi, 2003. The study of gastro-intestinal helminthes in native chicken and the efficacy of Mebendazole against the helminth parasites. *Thai Journal of Veteinrary Medicine* **33**(3): 65-72.

Njue, S.W., Ksiiti, J.L, Machria, J.M., Gucheru, S.G. and Mbugua, H.W.C. 2001. A survey of the diseases status of village chicken in Kenya. Livestock,

community and environmen. **In**: *Proceedings of the 10th Conference of the Association of Institutions of Tropical Veterinary Medicine*; Copenhagen, Demark.

Obiora, F.C. 1992. A Guide to Poultry Production in the Tropics. 1st edition. Enugu, Nigeria: Acena Publishers.

Ots, I. and Horak, P. 1998. Health impact of blood parasites in breeding great tits. *Oecologia* **116**: 441-448.

Padhi, B.C., S.C. Misra, and Panda, D.N. 1987. Pathology of helminthiasis in Desi fowls. 1. Cestode infection. *Indian Journal of Animal Health* **25** :127-131.

Pampori, Z.A. and Iqbal S. 2007. Haematology, Serum Chemistry and Electrocardiographic Evaluation in Native Chicken of Kashmir. *International Journal of Poultry Science* **6**(8): 578-582.

Pandey, V.S., Demey, F. and Verhulst, A. 1992. Parasitic diseases: A neglected problem in village poultry in Sub-Shaharan Africa pp. 136-141. **In**: *Village Poultry Production in Africa* (Eds. Pandey, V.S. and Demey F.). Rabat, Morocco.

Pandit, B. A., Mir, A. S., Banday, M.A.A and Shahardar, R.A. 1991. Prevalence of helminth parasites in Indigenous fowls of Kashmir Valley. *Poultry Adviser* Vol. **XXIV** Issue X.

Permin, A., Bojesen, M., Nansen, P., Bisgaard, M., Frandsen, F. and Pearman, M. 1997. *Ascaridia galli* productions in chickens following single infections with different dose levels. *Parasitology Research* **83**: 614-617.

Permin, A. and Hansen, J.W. 1998. Epidemiology, Diagnosis and Disease Control of Poultry Parasites. Rome, Italy: FAO.

Phukan, S.C. and Das, M. 2006. Clinico-haematological changes in Heterakis gallinarum infection in chicken. *Journal of Veterinary Parasitology* **20**(2): 159-161.

Poulsen, J., Permin, A., Hindsbo, O., Yelifari, L., Nansen, P. and Bloch, P. 2000. Prevalence and distribution of gastrointestinal helminths and haemoparasites in young scavenging chickens in upper eastern region of Ghana, West Africa. *Preventive Veterinary Medicine* **45**(3-4): 237-245.

Pinto, R.M., Breaner, B., Tortelly, R., Menezes, R. C. and Muniz-Pereira, L. C. 2008. Capillariid nematodes in Brazilian turkeys, *Meleagris gallopavo* (Galliformes, Phasianidae): pathology induced by *Baruscapillaria obsignata* and *Eucoleus annulatus* (Trichinelloidea, Capillariidae). *Mem Inst Oswaldo Cruz*, *Rio de Janeiro* **103**(3): 295-297.

Pinto, R.M., Breaner, B., Tortelly, R. and Menezes, R. C. 2008. Two cestode species in Brazilian turkeys, *Meleagris gallopavo* (Galliformes, Phasianidae): pathology induced by *Hymenolepis cantaniana* and occurrence of *Raillietina tetragona. Parasitol. Latinoam*. **63**: 81-84.

Pizarro, M., Villegas, P., Rodriguez, A., Gonzalez, M. and Flores, J.M. 2000. Capillaria contorta parasitism in red-legged partridge under farm conditions in Spain histopathology of the upper digestive system. *World's Poultry Science Journal* **58**(2): 159-166.

Qureshi, H.S. 1950. Incidence of helminth infections in fowls in Uttar Pradesh. *Indian Journal for Helminthology* **2**(1): 57-62.

Rabbi, A., Islam, S.M., Anisuzzaman, A. and Rahman, M.H. 2006. Gastrointestinal helminths infection in different types of poultry. *Bangladesh Journal of Veterinary Medicine* **4**(1): 13-18.

Ramarao, P., Sriraman, P.K and Gopal Naidu, N.R. 1980. Lamb mortaility in Andra Pradesh. *Indian Veterinary Journal* **57**(6):491-495.

Ramaswamy, K and Sundaram, R.K. 1984. Histopathological changes in the proventriculus of fowl given experimental monospecific infection with *Acuaria spiralis. Parasitology* **17**(4): 309-317.

Ravi Kumar, M., T. Bothra and K. Ashok, 2002. Backyard poultry for socio-nutritional security of rural and tribal masses. *Poultry Planner* **4**: 10-11.

Reid, W.M. and Carmon, J.L. (1958). Effects of numbers of *Ascaridia galli* in depressing weight gains in chicks. *Journal of Parasitology* **44**: 183-186.

Riddell, C. and Gajadhar, A. 1988. Caecal and hepatic granulomas in chickens associated with *Heterakis gallinarum* infection. *Avian Diseases* **32**: 836-838.

Ruff, M.D. 1999. Important parasites in poultry production systems. *Veterinary Parasitology* **84**(3-4):337-347.

Salam, S.T., Mir, M.S., Shahnaz, S. and Khan, R.A. 2009a. Prevalence and the associated lesions of *Cheilospirura (Acuaria) hamulosa* in the indigenous chicken of Kashmir valley, India. *Journal of Parasitology* **95** : 6.

Salam, S.T., Khan, A.R., Mir, M.S. 2009b. Prevalence and Pathology of Amoebotaenia sphenoides in free ranging chicken of Kashmir Valley. *The Internet Journal of Parasitic Diseases* **4** : 1.

Salam, S.T. 2009. Histopathological and biochemical studies of parasitic infections in pigeons and domestic fowl of Kashmir valley. Ph.D thesis submitted to P.G. Department of Zoology, the University of Kashmir.

Salam, S.T., Mir, M.S. and Khan, A.R. 2010. The prevalence and pathology of *Raillietina cesticillus* in indigenous chicken (*Gallus gallus domesticus*) in the temperate Himalayan region of Kashmir. *Veterinarski Arhiv.* **80**(2): 323-328.

Samad, M.A., Alam, M.M. and Rahman, A. 1985. Incidence of gastrointestinal parasitic infection in domestic fowls of Bangladesh. *Poultry Adviser* **18**(10): 33-38.

Sekhar, P.C., Mohan, U.C. and Sinha S.S. 1986. The effect of helminthiasis on total blood hemoglobin levels of domestic fowl. *Indian Journal of Poultry Science* **21**(3) : 243-246.

Shah, A.H., Anwar, A.U., Khan, M.N., Iqbal, Z. and Qudoos, A. 2001. Comparative studies on the prevalence of cestode parasites in indigenous and exotic layers at Faisalabad. *International Journal of Agriculture and Biology* **1**(4): 277-279.

Shah, I.H. 2009. Pathological and histochemical studies on paratuberculosis in goats. MVSc thesis submitted to Faculty of PG studies, SKUAST-Kashmir.

Shamsul-Islam, A.W.M. 1985. Prevalence of helminth parasites of domestic fowls in Zambia. *Poultry Adviser* **18**: 47-51.

Sharma, R.K., Banerjee, A.K. and Singh, K.S. 2002. Indigenous chicken genetic resources of India, their utility and relevance in poultry production. *Poultry Punch*. **18** : 50-51.

Snedecor, G.W. and Cochran W.G. 1967. Statistical Methods. 6th Ed. Oxford and IBH Publishing Co., New Delhi.

Soulsby, E.J.L. 1965. Text Book of Veterinary Clinical Parasitology. Vol. I. Helminths. 1st edn. Blackwell, Oxford.

Soulsby, E.J.L. 1982. Helminths, arthropods and protozoa of domesticated animals. Bailliere Tindall, London.

Srivastava, H.D. 1939. The important helminth parasites of poultry, their treatment and control. *Indian Journal of Veteirnary Science* **9**(4): 393-409.

Ssenyonga, G.S.Z. 1982. Prevalence of helminth parasites of domestic fowl (*Gallus gallus domesticus*) in Uganda. *Tropical Animal Health and Production* **14**(4): 201-204.

Swatson, H.K., Tshovhote, J., Nesamvumi, E., Ranwedzi, N.E. and Fourie, C. 2003. Characterization of indigenous free-ranging poultry production systems under traditional management conditions in the Vhembe district of the Limpopo Province, South Africa. Available at: http://www.ilri.org

Thrusfield, M. 1995. *Veterinary Epidemiology*. 2nd edition. 479 pp. Blackwell Science Ltd., London, UK.

Tuli, J.S., Bali, H.S. and Gupta, P.P. 1992. Histopathological and Histochemical studies of intestines in cestodiasis of poultry. *Indian Journal of Comparative Microbiology, Immunology and Infectious Diseases* **13**(3&4): 100-4.

University of Reading, 2007. Index of Poultry Diseases: Helminths. Available at: http://www.organicvet. reading.ac.uk/poultryweb/disease/helm/helm.htm.

Verma, N., Bhatnagar, P.K. and Banerjee, D.P. 1993. Pathological and biochemical changes in chicks experimentally infected with *Ascaridia galli*. *Indian Journal of Animal Science* **63**(4): 415-418.

Virk, K.J., Jain, M. and Prasad, R.N. 1987. Qualitative and quantitative analysis of helminth fauna in *Gallus domesticus*. *Zeitschrift Angeoand'te Zoologie* **74**: 329-336.

Whitmarsh, S. 1997. Parasitic Diseases (Internal). Poultry Science. Starkville, Miss, USA: Mississippi State University.

Whur, P. 1966. Relationship of globule leucocytes to gastrointestinal nematodes in the sheep and *Nippostrongylus brasiliensis* and *Hymenolepis nana* infection in rats. *Journal of Comparative Pathology* **76**: 57-65.

Yadav, A.K. and Tandon, V. 1991. Helminth parasitism of domestic fowl (*Gallus domesticus* L.) in a subtropical high-rainfall area of India. Beitr. *Tropical Landwiirtsch. Veterinary Medicine* **29**: 97-104.

Sher-e-Kashmir
University of Agricultural Sciences & Technology of Kashmir
Division of Veterinary Pathology,
Shuhama Campus Srinagar– 190 006
-::o::-

CERTIFICATE

Certified that all the corrections/amendments as suggested by External Examiner **Prof. Vipan Kumar Gupta, Head, Deptt. Of Veterinary Pathology, COVAS, CSK HPKV, Palampur (H.P)** have been incorporated in the manuscript entitled **"Pathological, Histochemical and Haemato-biochemical Studies on Gastrointestinal Parasitosis in Domestic Fowl (*Gallus domesticus*)"** submitted by **Dr. Riyaz Ahmad (Regd. No. 2007-V-70-M)**.

Dr. M.M. Darzi
Chairman
Advisory Committee