ETIO-PATHOMORPHOLOGICAL STUDIES ON GASTROINTESTINAL TRACT OF BROILER CHICKENS IN JAMMU

By

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Thesis submitted to Faculty of Postgraduate Studies
in partial fulfillment of requirements
for the degree of

MASTER OF VETERINARY SCIENCE

IN

VETERINARY PATHOLOGY

Division of Veterinary Pathology

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

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2017

CERTIFICATE-I

This is to certify that the thesis entitled **"Etio-pathomorphological Studies on Gastrointestinal Tract of Broiler Chickens in Jammu"** submitted in partial fulfillment of the requirements for the degree of **Master of Veterinary Science** in the subject of **Veterinary Pathology** to the Faculty of Post-Graduate Studies, Sher-e-Kashmir University of Agricultural Sciences and Technology of Jammu is a record of bonafide research carried out by **Miss. Navrose Sangha,** Regd. No. **J-15-MV-452,** under my supervision and guidance. No part of the thesis has been submitted for any other degree or diploma. It is further certified that such help and assistance received during the course of investigation have been duly acknowledged.

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ACKNOWLEDGEMENT

*The Almighty **Lord**, The Most Compassionate and Most Merciful had blessed me for undertaking this endeavor. I got inspiration during my research work from **Bani of Guru Granth Sahib Ji.***

*I don't have words to express my gratitude for my venerated and esteemed major advisor **Dr. Shilpa Sood**, Assistant Professor, Division of Veterinary Pathology for her expert guidance, keen interest and constant encouragement during my entire degree programme. I simply feel myself blessed being provided with an academic advisor like her.*

*It is my sole prerogative to place on record my indebtedness and everlasting gratitude to the members of my Advisory Committee, **Dr. Shagufta Azmi**, Ex-Professor and Head, Division of Veterinary Pathology, **Dr. Nawab Nashiruddullah**, Assoc. Professor & Head, Division of Veterinary Pathology, **Dr. Anil Taku**, Professor & Head, Division of Veterinary Microbiology and **Dr. Mudasir Sultana**, Professor & Head, Division of Veterinary Pharmacology & Toxicology for their help, innovative guidance and invaluable suggestions during the course of my study.*

*I do extend my respectful thanks and warm regards to **Dr. Rajesh Katoch**, (Professor & Head, Division of Vety. Parasitology), **Dr. Altaf Bhat**, (Professor, Division of Vety. Microbiology) and **Dr. Shafiqur Rahman,** (Assistant Professor, Division of Vety. Pathology)*

*I am thankful to **Hon'ble Vice Chancellor** of SKUAST-Jammu for allowing me to undertake the study and for providing necessary facilities. It is rearest to thank **Director Education, Dean** (FVSc. & A.H) and **Associate Dean** (FVSc. & A.H) for their courteous & indulgent moral, extraordinary help, technical and timely academic support throughout the course of the study.*

My sincere thanks are due to all technical and non-technical staff of Department of Veterinary Pathology, especially to Kiran Bala, Mr. Joginder, Mr. Mohinder and Mr. Bodraj.

I extend my sincere thanks to my colleagues and seniors particularly Sachin Dogra, Burhan Nabi, Bhavesh Sharma, Satuti Sharma, Sankalp Sharma, Sanak Sharma, Riya and Mehroon for always being around me with a smile and helping hands.

All the words in the lexicon will be futile and meaningless, if I fail to divulge my extreme sense of regards to my best friends, Dr. Mir Mudasir, Dr. Bilal Ahmed Malik, Dr. Malkeet Singh, Dr. Yassir Chauhan, Dr. Deepshikha Bali, Dr. Jasmeen Kaur, Dr. Rakshanda Bhagat and Dr. Mehak Shafiq for their worthy advices and for being always there at the times of need.

My endeavour could not have been realized without the love, support and encouragement from my fiance, Dr. Barinder Singh Aulakh.

With heartist reverence to my family, I admire the confidence bestowed on me by my parents, S. Surjit Singh and Sdn. Sukhpreet Kaur; brother, Dr. Jatandeep Singh, my indebtedness to my parents is beyond expression, as next to Lord I owe everything of my life to them and without their blessing it would have been an impossible task to complete this study.

I would also like to thank Dr. Shayaib Kamil, Dr. Suresh Kohli, Dr. Fayaz Ahmed and Dr. Raman Dar for their intense endeavour and for giving final shape to my thesis.

Needless to say, all omissions and errors are mine.

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ABSTRACT

Title of Thesis	**:**	**Etio-pathomorphology of Gastrointestinal Tract of Broiler Chickens in Various Parts of Jammu.**
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Year of Award of Degree	**:**	2017
Name of the University	**:**	Sher-e-Kashmir University of Agricultural Sciences & Technology of Jammu (J&K).

The present study was carried out to study etio-pathomorphology of gastro-intestinal tract of broiler chickens where disease occurrence was reported. 200 flocks were surveyed in different areas in and around Jammu. Occurrence of colibacillosis (24.16%) was maximum followed by that of infectious bursal disease (IBD) (7.13%), omphalitis (6.79%), salmonellosis (5.36%), non specific enteritis (4.29%), coccidiosis (2.22%), haemorrhages in proventriculus (1.67%), gout (1.44%), nephro-hepatotoxicity (1.42%), ascites (1.14), fatty liver syndrome (1.03%), caseous nodules in lungs/ suspected brooder pneumonia (0.75%) and non specific liver ailments (0.50%). The maximum mortality was caused by colibacillosis (8.33%). Characteristic lesions in colibacillosis were fibrinous pericarditis, airsacculitis and perihepatitis. Isolates of *E.coli* belonged to serogroup O1, O22, O37, O114, O118 and O149. Congestion, necrotic foci and bronze discolouration in liver were seen in birds affected with salmonellosis. IBD cases had paint brush haemorrhages on thigh and breast muscles along with enlarged, haemorrhagic, edematous bursa with necrosis of follicles and infiltration of heterophils in interfollicular areas. In suspected cases of Newcastle disease or avian influenza, haemorrhages in proventriculus were found. In cases of coccidiosis, haemorrhages in the small intestine and caecal tonsil were found. Further, degenerating and necrotic intestinal

epithelium harboring coccidian life-cycle stages were observed. Nephro-hepatotoxicity, degenerative and necrotic changes were seen in kidney and liver.

Out of 632 necropsied birds, intestines were found to be affected in maximum number of birds (80.22%) followed by liver (71.83%), proventriculus (21.36%), gizzard (8.86%), pancreas (8.38%) and oesophagus (3.00%). Lesions in intestine were consistent with presence of different forms of enteritis viz., acute which was mostly catarrhal, necrotic and chronic. In liver, congestion, degeneration and necrosis of hepatocytes, hyperplasia of bile duct epithelium, proliferation of fibrous connective tissue and infiltration of inflammatory cells in portal triad areas were recorded. Hepatitis was either acute, fibrinous, suppurative or chronic type. Proventriculus revealed congestion, presence of thick mucoid exudate over mucosa, haemorrhages, necrotic mucosa and infiltration of inflammatory cells mainly heterophils and few MNCs. In gizzard, haemorrhages, ulcerations, ventriculitis and necrosis of smooth muscle fibers with infiltration of lymphocytes were observed. Pancreas revealed congestion, haemorrhages, hyperplasia of pancreatic ducts, degeneration and necrosis of acinar cells. Acute pancreatitis characterised by degeneration, necrosis and infiltration of heterophilis was also noticed. Oesophagus revealed congestion, oedema along with infiltration of heterophils in sub mucosa around the oesophageal glands.

Key words: Infectious Bursal Disease, Mononuclear cells.

Signature of Major Advisor

Signature of Student

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ABBREVIATION

et al.	And other people
^{0}C	Degree Celsius
E.coli	*Escherichia coli*
EMB	Eosin methylene blue agar
ELISA	Enzyme Linked Immunosorbent Assay
FCR	Feed conversion ratio
GIT	Gastrointestinal tract
GPPW	Glucose phosphate peptone water
H&E	Hematoxylin and eosin
IBD	Infectious Bursal Disease
ILT	Infectious Laryngotracheitis
KOH	Potassium hydroxide
MLA	MacConkey's Lactose Agar
MNCs	Mononuclear cells
NCD	Newcastle disease
no.	Number
%	Percent
PF	Poultry farm
±	Plus or minus
RSS	Runting and stunting syndrome
RVs	Rotaviruses
Spp.	Species
viz.,	Videlicet (namely)

Chapter-1

Introduction

CHAPTER 1

INTRODUCTION

Poultry production is an important agro-based enterprise of our country. India has witnessed a rapid development of poultry industry and the poultry population has increased from 307.07 million in 1992 to 729.2 million in 2012 (Livestock census, 2012). India ranks third in broiler production in the world. Jammu and Kashmir (J&K) being a large meat consuming state a rapidly developing broiler industry provides an excellent means for supplying quality meat and a good source of livelihood to the un-employed youth (Mehta *et al*., 2003).

The J&K state occupies 17th place in poultry production in the country with the percentage share of 1.13. The total poultry population of J&K state is 8.3 million. According to the population census 2010-11, fowl population in Jammu region is 2.2 million. Morbidity and mortality due to various poultry diseases cause great economic losses to the farmers all over the world even though vaccination, proper nutrition and other preventive measures are routinely followed (Tabler *et al*., 2004).

Changing geo-climatic conditions, improper management practices and lack of awareness regarding biosecurity measures serve as major constraints for broiler industry and generally the aetiology of outbreak of diseases remains undiagnosed.

The gastrointestinal tract (GIT) performs important functions of digestion, assimilation and absorption of food. Any abnormality in functioning of GIT leads to disturbances with absorption and assimilation of nutrients resulting in decrease in growth, decrease in feed conversion ratio (FCR) and production losses, thereby severely affecting the health status of broiler farming (Ficken and Wages, 1997). Common infectious and non-infectious diseases of poultry cause gastro-intestinal lesions of varying severity.

Important infectious diseases of broilers which target GIT are salmonellosis, colibacillosis, mycoplasmosis, coccidiosis, inclusion body hepatitis, Newcastle disease and avian influenza among others (Saif, 2008). Salmonellosis is among the commonest

gastro-enteric diseases, causing serious losses to the poultry industry in terms of mortality and reduced growth rate (Gupta *et al*., 1990). Important pathological lesions in the GIT associated with Salmonella infection include congestion, hepatomegaly, bronze discolouration of liver, perihepatitis, necrotic foci on liver, nodular lesions in the gizzard and typhlocolitis with caseous caecal core (Swayne *et al.*, 2013).

Colibacillosis is another economically important disease affecting broiler chicken at very young age inflicting severe damage to the GIT. It is prevalent in all over the world including India (Tonu *et al.*, 2011). The lesions include congestion and haemorrhage with excess mucus in the lumen of various organs of GIT along with perihepatitis.

Another emerging issue for the broiler industry is necrotic enteritis caused by *Clostridium perferingens* (Swayne *et al*., 2013) leading to production losses due to poor digestion and absorption resulting in reduced weight gain (Elwinger *et al*., 1992; Kaldhusdal *et al*., 2001). Grossly, it produces a characteristic turkish towel appearance of the mucosa of small intestine as a result of extensive villous necrosis and pseudo-membrane formation. In addition, it may cause cholangio-hepatitis, hepatomegaly and hepatic necrosis.

Newcastle disease, commonly known as Ranikhet disease in India caused by avian paramyxovirus type 1 (Narayanan *et al*., 2010) It is a devastating disease of poultry leading to almost 100% morbidity and mortality in a short period of time with huge losses and producing characteristic haemorrhagic and necrotic lesions throughout the GIT. There are pin point haemorrhages in the proventriculus and intestinal mucosa, ulceration of ceacal tonsils and intestines (Swayne *et al*., 2013). Similar lesions may be seen in avian influenza caused by orthomyxovirus.

Adenoviruses have been associated with gastro-intestinal pathology in very young broilers. Lesions in chickens infected include marked gizzard erosions, necrotizing pancreatitis, ecchymotic haemorrhages, proventriculitis, focal hepatic necrosis with intra-nuclear inclusion bodies in the hepatocytes (Itakura *et al*., 1974; Lenz *et al*., 1998; Swayne *et al*., 2013).

IBD is an immunesuppressive disease in which haemorrhages at the junction of gizzard and proventriculus are often seen in birds (Swayne *et al*. 2013).

Runting-stunting syndrome, a multifactoral disease condition in chicken is characterized by denuding along with stunting and blunting of villi resulting in mal-absorption syndrome (Swayne *et al*., 2013).

Coccidiosis, an important protozoal disease causes severe morbidity and mortality in broiler chicken. The affected birds are anaemic due to bloody diarrhoea and have an impaired growth rate and reduction in body weight gain (Lillehoj *et al*., 2004). Post-mortem examination usually reveals petechial haemorrhages, oedema, necrosis and sloughing of intestinal epithelium (Soomro *et al*., 2001).

Ascaridia galli is a common nematode in broiler chicken causing weight loss, decrease FCR and catarrhal enteritis.

So, it is of tremendous importance to study the various diseases of broilers, prevalent in a region which reduce the growth rate as well as feed conversion efficiency. Moreover, knowledge of occurrence of various diseases during the first few weeks of bird's life will permit quick diagnosis of diseases and allow adequate corrective measures to be adopted to minimize losses. Furthermore, an understanding of GIT lesions will serve as guideline in future research and improved management practices.

There are only few reports on occurrence of disease of broilers in Jammu. No systematic study has been conducted so far regarding and pathomorphlogy of GIT lesions occurring in broilers in Jammu. Keeping in view the above facts, the present study was undertaken to achieve the following objectives:

1) To study gross & histopathology of gastro-intestinal tract lesions of broilers in Jammu.

2) Attempts to identify/demonstrate causative organisms in these lesions.

Chapter-2

Review of Literature

CHAPTER 2

REVIEW OF LITERATURE

A large number of pathological conditions and diseases affect the broilers. These include bacterial, viral, protozoan infection, metabolic diseases and non specific disease conditions. In the present study the literature has been reviewed regarding information on the occurrence of the different pathological conditions in broiler flocks.

2.1 Occurrence and mortality pattern

Abroad

Reddy and Reddy (1991) studied the mortality pattern in broilers. Commonest cause of death was coccidiosis (21.51%) followed by Ranikhet disease (11.8%) and enteritis (1.95%).

Islam *et al*. (2003) recorded the occurrence of poultry diseases in 1352 birds of Sylhet region, Bangladesh. The occurrence of diseases were IBD (24.26%), Newcastle disease (6.73%), Salmonellosis (6.73%), colibacillosis (5.17%), omphalitis (2.81%), necrotic enteritis (0.44%), aspergillosis (17.53%) and coccidiosis (9.46%). Highest cases were recorded in the age of 8-21 days (42.60%) followed by 22-35 days age (26.62%) and 0-7 days age (26.10%). Study revealed that poultry diseases occur mostly in rainy season (56.36%) followed by summer (28.11%) and the least in winter season (15.53%).

Ahmed *et al.* (2009) studied the occurrence of infectious diseases in broiler chickens at Kapasia in Gazipur district in Bangladesh. Diseases recorded were colibacillosis 52.26%, salmonellosis 1.01%, omphalitis 11.56%, coccidiosis 4.52%, IBD 11.06% and mixed infection of Gumboro & Coccidiosis 1.51%.

Ahmed *et al.* (2012) reported IBD as most prevalent (29.37%) in broilers followed by colibacillosis (18.61%), coccidiosis (17.38%) and Chronic respiratory disease (CRD) (17.27%) in district of Poonch Azad J&K. On the basis of altitude, the occurrence of various diseases were recorded as IBD (29.22%), colibacillosis (18.53%), CRD (17.89%) and coccidiosis (17.00%) above 4000 feet height, while IBD (32.01%),

colibacillosis (18.78%), coccidiosis (18.28%) and CRD (15.82%) below 4000 feet were found.

Hasan *et al*. (2010a) reported that a total 47 broilers were collected from 4 different poultry farms of Mymensingh and Gazipur districts by. In clinical diagnosis, colibacillosis was found in 34% and salmonellosis in 23.40% birds. In laboratory, 25.53% cases had colibacillosis and 14.89% had salmonellosis.

El-Sayed *et al*. (2017) observed the pathological lesions in intestine and liver of different breeds of 100 broiler chickens at Alexandria province, Egypt. *E.coli* and *Salmonella gallinarum* bacteria were isolated from intestinal samples and hepatic tissues. In parasitological examination only coccidiosis was found. Hepatic necrosis and necrotic enteritis was 29.41% and 67.80% respectively was recorded in broilers.

India

Chakraborty *et al.* (1982) investigated causes of broiler chick mortality in and around Calcutta. Various conditions such as infectious agents (40.68%) and nutritional deficiencies (21.19%) were found to be responsible for mortality.

Sheriff and Kumaran (1987) studied the mortality pattern in broilers in and around Pudukkottai district of Tamil Nadu during the year 1985. Colibacillosis, yolk sac infection, enteritis and intestinal coccidiosis were recorded. Maximum mortality was in 0-3 weeks of life. During 4-6 weeks of age, coccidiosis and heat stroke were the main problems.

Lalrintlunga and Baruah (1993) observed that highest mortality was due to Ranikhet disease (25.9%), coccidiosis (15.7%), colisepticaemia (14.6%), aflatoxicosis (10.5%) and omphalitis (5.8%) in broiler chicken in Assam. The highest mortality in broilers was recorded in the month of November to January and again in the month of April.

Mahajan *et al.* (1994) studied the major broiler diseases in Haryana and reported the morbidity rate varied from 8.2% to 10.8%. Maximum mortality was caused by fowl typhoid (10.54%) followed by colibacillosis (8.88%), brooder pneumonia (7.69%), coccidiosis (6.85%), Ranikhet disease (6.03%) and other miscellaneous diseases (8.05%). Incidence of diseases was more in monsoon and winter than in summer.

Anjaneyulu *et al.* (1998) while studying broiler mortality in Prakasam district of Andhra Pardesh reported that colisepticaemia (13.4%) followed by coryza (9.6%), gumboro disease (9.1%), respiratory mycoplasmosis (8.4%), coccidiosis (7.0%) and ascites (6.78%) were the major causes of mortality in broilers.

Singh *et al*. (1998) studied the etiopathology of chick mortality in Punjab and recorded the average mortality of 5.69% from 0 to 6 weeks of age. Maximum mortality (1.29%) was caused by omphalitis followed by IBD (0.86%), coccidiosis (0.56%), colibacillosis (0.32%) and fowl typhoid (0.23%). Starvation syndrome, ascites, aspergillosis, hepatitis and gout were other causes of mortality.

Mahajan *et al*. (2002) investigated the outbreak of aflatoxicosis and IBD in two adjoining organized poultry farms in Palampur. Morbidity and mortality was 80-90% among broiler chicks in the 4 to 6 week age group. Samples of vital organs comprising liver, kidney, bursa of Fabricious and intestines were processed microbiologically. Morbid materials yielded *E.coli* and *Proteus* spp. *Aspergillus flavus* was isolated from feed samples collected from feed stocks.

Singh *et al.* (2003) recorded the prevalence and mortality pattern in broilers at selected farms of Punjab, India. They recorded maximum (0.14%) prevalence of omphalitis followed by aflatoxicosis (0.41%), colibacillosis (0.39%), coccidiosis (0.16%), aspergillosis (0.14%), IBD (0.08%) and gout (0.06%). Maximum mortality was recorded in 1-2 weeks age groups and minimum in 6-7 weeks old birds.

Goyal (2004) recorded 26.03 % prevalence of hepatic lesions associated with various diseases of poultry. Diseases affecting the liver were colibacillosis, IBD, non-specific hepatitis, omphalitis, fatty liver and liver rupture and infarction, gout and ascites syndrome. In ascites syndrome, chronic perihepatitis, fibrosis and pseudolobulation of

liver were seen. Visceral form of gout was recorded in all age groups affecting mostly the surface of visceral organs.

Pugashetti and Shivakumar (2007) observed that maximum deaths in broiler birds in an organised poultry farm of Karnataka were caused by pneumonia (29.60%), enteritis (27.73%), ascites (7.22%) and coccidiosis (2.51%).

Balasubramaniam *et al.* (2009) analyzed the influence of season on the mortality pattern of poultry in Namakkal, Tamil Nadu. The disease diagnosis was based on history, gross and microscopic lesions, isolation and other diagnostic tests. Results revealed that the occurrence of Newcastle disease (15.07%, 14.22% and 17.98%) was higher in winter and rainy seasons than other diseases. Incidence of colibacillosis was high among bacterial disease without any influence of season on the occurrence while necrotic enteritis was high in winter (2.53%) and rainy seasons (1.84%).

Buragohain and Kalita (2010) observed the mortality pattern of broiler reared under intensive system (deep litter) in Mizoram. From necropsy of 312 broilers, recorded that ascites syndrome (34.3%) was main cause of mortality followed by colibacillosis (19.23%), omphalitis (12.18%) and caecal coccidiosis (8.33%).

Itoo *et al.* (2013) observed 186 broiler flocks, with a total of 488942 birds in and around Srinagar and reported the occurrence of colibacillosis in 26 flocks, Newcastle disease in 2 flocks, aspergillosis in 8 flocks, gout in 4 flocks and ascites in 12 flocks, with respective mortalities of 6.4%, 13.7%, 7.0%, 8.7% and 9.3%.

Bhutia and Singh (2016) conducted a survey on the prevalence of viral diseases of poultry in Mizoram. A total of 476 birds were collected from different organized and unorganized poultry farms and 208 cases (43.69%) were diagnosed as viral diseases. IBD was found in 15.13% followed by Newcastle disease in 8.40% cases. Incidence of diseases was found to be highest in winter season in 3-6 week age group.

Borah *et al.* (2017) recorded the occurrence of various infectious diseases in broiler chicken in Kamrup and Kamrup (Metro) districts of Assam. A total of 567 birds were examined from 100 different farms. Highest incidence recorded was of omphalitis

(13.40%) followed by colibacillosis (11.11%), IBD (10.58%), necrotic enteritis (6.35%), bacillary white diarrhoea (5.11%), Newcastle disease (4.59%) and brooder pneumonia (3.70%). Incidence of omphalitis, colibacillosis and bacillary white diarrhoea were found throughout the year.

2.2 Bacterial diseases

2.2.1 Colibacillosis

Abroad

Nakamura *et al.* (1985) studied pathology of spontaneous colibacillosis in a broiler flock. They observed fibrinous thrombi in sinusoids of the liver, necrosis of hepatic cells and fibrinopurulent inflammation with granulomatous changes in the serosa of liver.

Chowdhury *et al*. (2009) examined a total 4372 broiler and layer birds to identify the different forms of colibacillosis in commercial broiler and layer birds in Chittagong region of Bangladesh. Among them, 1893 (70.87%) broiler birds were diagnosed as affected with colibacillosis. The most frequent form of colibacillosis was omphalitis, airsacculitis, pericarditis, perihepatitis and peritonitis. Out of total in 30.48% birds, different forms of colibacillosis were recorded.

Omer *et al*. (2010) recorded the outbreak of colibacillosis in broilers in Kassala State, Eastern Sudan. Overall 6.8 % mortality was recorded in broiler flocks. Diagnosis was made on the basis of case history, clinical signs, postmortem findings and laboratory examination.

Tonu *et al*. (2011) studied the pathogenicity of *E.coli* in birds. Gross examination showed congestion, haemorrhages with excess mucus on the luminal surface of duodenum. Microscopically, the duodenum showed severe infiltration of leukocytes, heterophils, lymphocytes and macrophages in the sub-mucosa of its wall.

Samah and Ahmed (2013) collected the 105 carcasses of broilers from farms showing the high mortality in Sharkia Governorate. *E.coli* was isolated from 84 (80%)

cases. Total 11 different serotypes were identified, of which O114:K90 was the most detected with 17.9% occurrence. While O125:K70, O55:K59, O111:K58 and O26:K60 were identified in 14.3%, 14.3%, 10.7% and 10.7% birds respectively. Other serotypes (O145, O25:K11, O44:K74, O126:K71, O118) had the same percentage (3.6%) share of occurrence.

Abdeltawab *et al.* (2015) studied incidence of *E.coli* infection in broiler chickens in winter and summer season. A total of 205 chicken samples were collected from Menofyiea government in provinces of Egypt. Incidence of *E.coli* infection in healthy broiler chickens, diseased broiler chickens and freshly dead ones was 15.7 %, 37.1 % and 55 % respectively in winter season while in summer season the incidence was 15.8 % in healthy, 17.5 % in diseased and 18.7 % in dead birds. Serogroups of *E.coli* involved were O1, O2, O78, O55, O111, O114, O124, O128 and O142.

Ali and Ali (2015) collected 50 broiler birds from Basra Province which were showing lesions of fibrinous perihepatitis, fibrinous pericarditis and airsacculitis. Bacterological examination revealed presence of *E.coli* infection in 46% birds. The isolates belonged to the serogroup O78: K80.

Matin *et al*. (2017) recorded that the overall prevalence of colibacillosis in broilers in poultry farms of Mymen Singh and Tangail districts of Bangladesh was 0.84%. Age wise prevalence was 1.0% in 25-30 days old and 0.5 % in 31-35 days old broilers. *E.coli* infection was confirmed by sugar fermentation, biochemical tests and polymerase chain reaction.

India

Balani (1983) conducted postmortem examination of poultry birds brought to the Rohtak Disease Investigation Laboratory and diagnosed colibacillosis in 38.4% of cases.

Ghosh (1987) recorded 37% of mortality in broilers at organized farms in Nagaland and found that *E.coli* serogroups O61, O143, O147, O91 and O119 were most prevalent.

Javed *et al.* (1991) observed colibacillosis in 11.74% birds of a poultry flock in one year and the prevalence was found to be higher in broilers (13.13%) compared with layers (9.40%).

Kaul *et al.* (1992) reported an outbreak of colibacillosis in broiler chicks in North Gujrat in which 16.25% of broilers died at 3-7 weeks of age. *E.coli* was isolated in pure culture from various organs of dead birds.

Mukhopadhyaya and Mishra (1992) isolated and identified *E.coli* from suspected cases of colibacillosis in West Bengal. 304 strains of *E.coli* from 508 morbid chicks (59.8%) were isolated which were serotyped as O1, O2, O55, O78, O120, O158 and O162.

Baliarsingh *et al.* (1993) induced experimental colibacillosis in chicks. Histopathological studies revealed severe congestion in liver and small intestine. Necrosis with infiltration of heterophils and a few lymphoid cells was also seen.

Mukherjee and Khanapurkar (1994) observed the presence of cheesy yellow pseudomembrane covering the viscera accompanied by perihepatitis and pericarditis in cases affecting with colibacillosis. Histopathological examination revealed congestion, degeneration and infiltration of inflammatory cells in hepatic parenchyma.

Pourbakhsh *et al.* (1997) observed airsacculitis, pericarditis and perihepatitis in *E.coli* infection in chickens. Microscopically, infiltration of inflammatory cells, serous to fibrinous exudates and cellular debris on the serosal surfaces were present in the liver, spleen and air sacs.

Sharada *et al.* (1999) isolated different serotypes of *E.coli* of poultry affected with perihepatitis, pericarditis, enteritis, airsacculitis, yolk sac infection and pneumonitis. 44.61% of cases with perihepatitis and 33.85% of cases with enteritis had *E.coli* infection.

Shankar *et al.* (2010) collected 162 *E.coli* isolates from infected cases in Hisar that belonged to 37 different 'O' serogroups and 6 were rough. Serotypes O78 (46), untypable (33), O75 (14), O2 (10), O6 (10) and O111 (10) were recorded.

Sahoo *et al.* (2012) collected samples of livers, heart bloods, pericardial fluids, yolk sacs and intestine from different poultry farms of Odisha from suspected cases of colibacillosis and processed them for confirmatory diagnosis. *E.coli* was isolated from 52.6% yolk sac and 38.4% heart blood samples in 0-4 week birds. In (4-7week) older birds *E.coli* isolation was done from 35.8% pericardial fluid samples followed by 33.4% heart blood samples. Occurrence of O9 strain was highest (16.7%) followed by O1, O33 & O51 (13.3%), O23 & O119 (10%), O103 & 79 (6.7%) and serotype O90 (3.3%) respectively.

Bhalerao *et al.* (2013) observed that the maximum mortality in 3-4 week old birds occurred due to *E.coli* infection in Hisar. Gross pathological examination revealed congestion in various organs, accumulation of fibrin on the liver and heart. Microscopically, there was fibrinous pericarditis, myocarditis, fibrinous perihepatitis, fatty changes in hepatocytes, interstitial pneumonia, necrosis and depletion of lymphocytes in spleen and enteritis

Sarker *et al.* (2013) screened 162 samples from different poultry farms of West Bengal, India and 109 (67.3%) were found to be positive for the *E.coli.* 72.6% and 61.1% samples from intestines and liver tissues were positive for *E.coli*. Biochemical characteristics of the isolates were indole positive, methyl red postive, nitrate negative and non-reactive to voges proskauer, citrate & urease test. In serotyping revealed presence of O2, O8, O9, O19, O37, O47, O55, O69, O86, O101, O103, O109, O133, O151 and O173 serotypes.

2.2.2 Omphalitis

Abroad

Nasrin *et al*. (2012) identified *E.coli*, *Salmonella* sp. and *Staphylococci* sp. in yolk sac contents from cases of omphalitis in chicks in Bangladesh.

India

Gross (1964) described the histopathological lesions associated with retained caseous yolk sacs due to *E.coli* infection. The walls of the yolk sacs were edematous,

macrophages and giant cells formed a thin layer next to the yolk material. Heterophils and plasma cells were also present.

Bhatia *et al.* (1972) studied the pathological lesions associated with yolk sac infection in young chicks. Pathological examination of dead birds revealed congestion, edema and degeneration of various internal organs most often liver.

Srivastava (1990) recorded 23.7% mortality due to omphalitis in 0-4 week old broiler chickens and *E.coli* was isolated from 45% cases.

Ghodasara *et al*. (1992) observed 26.23% mortality in broilers out of which 31.45% mortality was attributed to omphalitis caused by *E.coli.*

2.2.3 Salmonellosis

Abroad

Barbour *et al.* (1999) found that mortality percentage in broiler chicks of 8-15 days of age due to *Salmonella enteritidis* was 4%. The clinical signs included somnolence, profuse diarrhea followed by dehydration, pasting vents, drooping wings and shivering.

India

Bhattacharyya *et al.* (1984) investigated broiler chick mortality pattern in West Bengal. Mortality mostly occurred due to pullorum disease and brooder pneumonia. In chicks affected with *Salmonella pullorum* infection, mortality started on day one and lasted until the 3rd week of life, killing 43.52-54.07% of chicks. Most affected organ was liver.

Jindal *et al*. (1999) reported *Salmonella gallinarum* and *Salmonella enteritidis* infection in poultry from Haryana. Mortality due to fowl typhoid was 4.82 % during 1994-‘95 and had gone up to 12.12 % during the year 1996-‘97. These infections accounted for 5% of total poultry disease out breaks that were recorded during the period.

Aetiopathological investigation of *Salmonella gallinarum* infection in broilers was done by Hafeeji *et al.* (2000). Average mortality due to fowl typhoid was 8.29 %. Liver showed marked enlargement, congestion, white grey necrotic foci and necrotic patches that were distributed uniformly on their surfaces. In some cases typhlitis was seen. Histopathologically, mild to moderate congestion and haemorrhages, focal to diffuse areas of coagulative necrosis and MNC infiltration in parenchyma were noticed. Occasionally, septic emboli were observed in hepatic parenchyma.

Kumar *et al*. (2010) studied the epidemiological status of fowl typhoid in broilers in Haryana. A total of 227 outbreaks of fowl typhoid were recorded in chickens during the period from 2005 to 2008. The maximum number of outbreaks was recorded in the age group of 7-9 days while maximum mortality and case fatality rate were found in 1-2 week old birds.

Nazir *et al.* (2012) encountered presence of salmonellosis in different commercial broiler farms of Srinagar district and adjoining areas. Clinical signs included weakness, droopy wings with ruffled feathers, anorexia, increased thirst, reluctance to move, watery to mucoid greenish yellow diarrhea, reduced growth rate and rarely lameness. Grossly, hepatomegaly, bronze discoloration of liver, congestion and necrotic foci on liver was noticed. Histopathological lesions in liver comprised of congestion, haemorrhages, areas of necrosis, reticular endothelial hyperplasia along with infiltration of MNCs and heterophils. Intestinal changes comprised of congestion of mucosal vessels along with marked hyperplasia of goblet cells and infiltration of heterophils and mononuclear cells in the lamina propria of villi.

Detailed patho-microbiological studies were done by Kumari *et al.* (2013) on *Salmonella gallinarum* infection in broiler chickens in Haryana. Mortality pattern of 134 birds revealed that maximum mortality occurred in 1-2 week aged birds. 23 Salmonella isolates were identified, out of which 19 samples were identified as *S. gallinarum* and 4 samples as *S. enteritidis*. Pathological lesions observed included bronze discoloration of liver and necrotic foci on liver. Microscopically, liver revealed aggregation of heterophils, lymphocytes and macrophages. Necrotic enteritis was also seen.

Arora *et al*. (2015) recorded that 309 outbreaks of fowl typhoid occurred in broilers in Haryana. They observed 9.45%, 6.77% and 71.55% of morbidity, mortality and case-fatality rate respectively in broilers. The yearly observations were divided into quarters A (January-March), B (April-June), C (July-September) and D (October-December). Maximum number of outbreaks 106 (34.3%) were recorded in quarter D followed by quarters B -84 (27.3%), C - 64 (20.7%) and A - 55 (17.7%). Typical morphology and colony characters of *Salmonella* on MacConkeys lactose agar and brilliant green agar, biochemical reactions and serogroups of isolates were recorded.

2.3 Viral diseases

2.3.1 Infectious Bursal disease (IBD)

India

Chauhan *et al.* (1980) reported the outbreaks of IBD at three poultry farms of Ranchi (Bihar). The disease affected birds of 2-7 weeks of age. Mortality was 17.89% in one poultry farm and 19.8% on the other farms.

Gill *et al.* (1988) observed outbreaks of IBD in broiler chickens in Punjab. Affected birds exhibited characteristic clinical symptoms of anorexia, ruffled feathers, depression and diarrhoea. A total of 38.46 % mortality was recorded.

Rajashewar *et al.* (1992) reported a mortality rate of 23.2 % while studying the incidence of IBD in chickens in Kanyakumari district of Tamil Nadu.

Farooq *et al.* (2003) investigated the outbreaks of IBD in Mirpur and Kotli districts of Kashmir from 50 broiler farms. Higher losses were found due to IBD in flocks experiencing a concurrent coccidiosis problem. Average mortality due to IBD was 15.31±1.04% with a co-efficient of variation of 48.04%. Higher losses were found in winter than spring season. Significantly, higher losses were found at the age above 32 days than at 19-23 days of age.

Jindal *et al*. (2004) collected the epidemiological data related to IBD outbreaks in 795 broiler flocks in Hisar. Disease affected 8.89% flocks during the nine year period

with morbidity, cumulative mortality and case fatality rates of 5.9, 3.63 and 61.43% respectively. Affected birds were dull, depressed and had ruffled feather and yellow white diarrhoea. Postmortem changes were recorded in the bursa of Fabricius followed by changes in thigh and breast muscles.

Mor *et al.* (2010) analysed the epidemiological data on IBD obtained from 483 broiler chicken flocks in Haryana. Overall morbidity, cumulative mortality and case fatality rate were recorded as 4.54 %, 2.34 % and 51.69 %, respectively. Clinically, affected birds were dull, depressed, had ruffled feathers and suffered from diarrhoea. At necropsy, the gross lesions were observed mainly in bursa of Fabricius and in thigh and breast muscles. Maximum cases (52.80%) were observed in birds of 21-30 days of age followed by 33.13% cases in the age group of 31-40 days.

Choudhary *et al*. (2012) recorded that overall incidence of IBD was 33.90 % in and around Ranchi. Commercial broiler chicks showed higher sero-prevalence (37.97%) than local breeds (29.74%). Incidence was also found to be highest in the age group on 4-7 weeks of chickens (43.18%) with higher rate of infection in male chickens (35.12%). Higher rate of infection was also recorded in the monsoon (36.73%) than the winter season (30.83%).

Rathore *et al*. (2013) recorded 723 cases of IBD in flock of 9000 birds at Verma poultry farm in Panipat, Haryana. Clinically affected birds were dull, depressed, reluctance to move, had ruffled feathers and white diarrhoea with vent pasting. On postmortem examination, haemorrhages were found on leg muscles and bursa along with severe pus accumulation in bursal lumen.

Singh *et al*. (2015) included gross, histopathological and immunopathological approaches in their study for the diagnosis of IBD. A total of 33 samples were collected from the six different poultry farms of Ludhiana. Macroscopic changes were seen in bursa included as swelling, hemorrhages and atrophy. Microscopically, bursa showed prominent fibrotic and atrophic changes, infiltration of MNCs along with chronic cystic changes.

2.3.2 Newcastle Disease (NCD)

Abroad

Rahman *et al.* (2012) determined the prevalence of Newcastle disease virus using rapid antigen detection kit from field samples of poultry in Bangladesh. The cloacal swabs were collected from 10 randomly selected birds which included broilers, layers, native chickens and ducks in four different districts or areas. A total of 160 field samples were successfully tested. They recorded that prevalence of NDV in broiler bird was 12.5% respectively.

Babaca (2015) surveyed 17 farms in and around Erbil City (Iraq) with a history of outbreaks of NCD during late spring, summer and winter of 2008. Birds were vaccinated with Lasota strain. They found that fowls 1-8 weeks age were susceptible to ND and that the mortality rate was 8.1% in broilers

Kumar *et al.* (2016) reported that prevalence of Newcastle disease in commercial broiler farms at Bochaganj Upazila of Dinajpur district was 5.35%. Grossly, severe haemorrhages in caecal tonsils and on surface near junction of proventriculus and gizzard were observed. Mortality in non-vaccinated and vaccinated broiler flocks was 20.76% and 4.6% respectively.

India

Brar *et al*. (2017) reported the severe enteric form of Newcastle disease virus infections in backyard poultry birds. Grossly, ulcers in intestine, haemorrhages in proventriculus and caecal tonsils, congestion and haemorrhages in liver were noticed. Microscopically, severe haemorrhages in the intestine, degeneration, necrosis of the intestinal villi and fatty changes in hepatocytes were observed. On immunohistochemistry, Newcastle disease viral antigens were found to be localized in the necrotic cells of epithelium of proventriculus, gizzard, liver and intestine.

2.3.3 Avian influenza

Abroad

Rahman *et al.* (2012) determined the prevalence of avian influenza virus using rapid antigen detection kit from field samples of poultry in Bangladesh. The cloacal swabs were collected from 10 randomly selected birds which included broilers, layers, native chickens and ducks in four different districts or areas. A total of 160 field samples were successfully tested. They recorded that prevalence of AIV in broiler bird was 32.5% respectively.

Arif *et al.* (2015) collected serum samples from 570 broilers from 52 poultry farms of Quetta to determine the seroprevalence of avian influenza virus by Enzyme Linked Immunosorbent Assay (ELISA). The sero-positivity of avian influenza virus was recorded to be 14.03% in broilers. All the positive sera of boilers determined by ELISA were further tested by using H5, H7 and H9 specific strains antigen through haemagglutination inhibition and haemagglutination tests. Only H9 was recognized from the sera of broilers.

2.4 Protozoan disease

2.4.1 Coccidiosis

Abroad

Long *et al.* (1974) collected 124 intestines of broilers and examined them for presence of necrotic enteritis. Lesions of necrotic enteritis were recorded in one or more areas of the intestine in all but six of 94. Coccidia were found in small numbers in birds. Brown and Brenn stained sections showed Gram-positive bacilli associated with early necrotic lesions on the tips of villi. Tissue sections from the intestines show the lesions starting at the tips of villi.

Soomro *et al.* (2001) observed the symptoms exhibited by birds suffering from coccidiosis included loss of appetite, unthriftiness and greenish or reddish diarrhea.

Postmortem revealed intestinal ballooning, petechial haemorrhages, edematous walls, necrosis and sloughing of intestinal and caecal epithelium.

Nematollahi *et al.* (2009) recorded an overall prevalence of 55.96% of *Eimeria* spp. among 218 broiler farms in Tabriz northwest of Iran.

Gharekhani *et al*. (2014) reported that prevalence of coccidiosis in broiler birds in Hamedan province, Western Iran was 31.8%.

Khaier *et al*. (2015) identified *Eimeria tenella* infection in broiler chickens. They recorded 19.63 length and 17.02 width of oocyst. Microscopically examination showed different parasitic stages in chickens mucosa and glandular region

Shamim *et al.* (2015) recorded that overall prevalence of coccidiosis in broilers in Mirpur, Azad Kashmir, Pakistan was 9.59%. Age-wise highest prevalence (10.88%) was seen in 0 to 3 week old birds. Prevalence of coccidiosis observed in spring season was 12.49% as compared with 6.60% in summer season. *Eimeria tenella* was more prevalent than *Eimeria maxima*.

India

Panda *et al*. (1997) reported the incidence of coccidiosis in broiler birds from Orissa. Mortality due to coccidiosis was found to be 11.57 % and mortality due to caecal form (7.03%) was more than that from the intestinal form (4.72%). Mortality rate was higher during summer and rainy months. Higher incidence was found in birds of 3-6 weeks of age followed by below 3 weeks and above 6 weeks old birds.

Jithendran (2001) studied the prevalence of outbreaks of coccidiosis over a period of ten year (1986-1990 and 1994-1998) interval in Himachal Pradesh and found that the disease was more prevalent in rainy and winter season with a slight recession during summer months.

Kumar *et al*. (2008) conducted the study on the outbreak of coccidiosis in broiler farms in Ladakh. The outbreak eventually involved a total of 6,754 birds of 11 different

batches from different age groups. The mortality was 11.75 % and majority of birds involved in the outbreak were aged of 35 days and above..

Sood *et al.* (2009) studied the prevalence of coccidiosis in poultry birds in R.S. Pura, Jammu. 117 faecal samples were examined, 39 were positive for coccidial oocysts, 24 positive for *Ascaridia galli* and 12 positive for *Heterakis gallinarum*. Blood tinged mucous exudates clinging to mucosa of intestine and ulceration of mucosa with haemorrhages in different parts of intestine were observed in birds affected with coccidiosis. Histopathologically, intestinal sections were severely inflamed with increased thickness of intestinal wall and infiltration of MNCs. Gamonts and schizonts were numerous in the enteric epithelial cells as well as lumen along with exudates. Multifocal areas of epithelial cell denudation, goblet cell hyperplasia with congestion, haemorrhage and edema in submucosal area was also seen.

Patra *et al.* (2010) observed the *Eimeria tenella* infection in broiler chicken in Mizoram. Post-mortem examination revealed the distended caeca filled with bloody faeces and mucoid debris with haemorrhages on the mucosa. Histopathological revealed haemorrhages, edema, necrosis and sloughing of caecal epithelium.

Datta *et al.* (2013) studied the epidemiology of enteritis in broiler chickens in Hisar. A total of 481 cases of enteritis were recorded in rainy and winter seasons. Maximum 146 (30.35%) cases were recorded in broiler birds of 29-35 days of age which was associated with coccidiosis.

Naphade (2013) recorded the (4.79%) highest incidence of the coccidiosis in the small poultry farms followed by medium poultry farms (3.04%) and lowest (1.35%) in the largest farms in and around Aurangabad city. Highest incidence was recorded in rainy season followed by winter and summer season.

Kala *et al.* (2013) conduct the survey on 556 poultry birds maintained at Central Poultry Breeding Farm, Patna and in local poultry farms of Patna, Bihar. The incidence of coccidiosis in the study area was found to be 16.54%. It was higher in broilers (21.38%) than in layers (11.27%) and greater in young (22.81%) than in adult (9.3%).

Incidence was highest in rainy season (32.14%) than other three seasons. *Eimeria tenella* was the predominant species with higher prevalence.

Sharma *et al*. (2013) recorded coccidiosis with an overall occurrence of 39.58% in both organized and unorganized farms of Jammu region. Maximum cases of coccidiosis were found in monsoon season and least in summer season. Coccidiosis was found to be most prevalent in 31–45 day old birds.

Ahad *et al*. (2015) found that the prevalence of coccidiosis in broilers in Kashmir valley was 29.87%. Microscopic examination revealed the presence of severe enteritis and presence of coccidial oocysts in intestinal epithelium. Coccidiosis was most prevalent in autumn followed by summer, spring and winter season.

2.5 Metabolic diseases

2.5.1 Ascites

Abroad

Ascites syndrome was noticed by Coello *et.al.* (1985) as a major cause of mortality for broiler flocks in Mexico with losses averaging 15 %.

Anjum *et al*. (1998) conducted studies on ascites syndrome in and around Faisalabad, during winter season 27 broiler farms were included in the study and recorded an overall mortality was 4.46 %. Affected birds showed clinical signs including dullness, depression, slow movements, ruffled feathers, difficult breathing and distended abdomens. Postmortem examination revealed swelling of liver with smooth or dimpled surfaces, swollen and congested kidneys. Microscopically, liver revealed necrosis and inflammatory changes, kidney showed congestion and degenerative changes in tubular epithelium.

Habib-ur-Rehman *et al.* (1999) made clinical, gross and histopathological observations on spontaneous cases of ascites syndrome in broiler chickens reared at low altitude in Japan. Gross lesions included accumulation of straw yellow colored fluid in the abdominal cavity, hydropericardium and fibrosis of liver. Histopathology showed

congestion and necrosis with proliferation of connective tissue in sinusoidal space along with infiltration of heterophils and MNCs.

Tafti and Karima (2000) observed ascites syndrome in 34 commercial broiler chickens of breeder strain in Shiraz area, Iran. Gross changes included dark breast muscle, marked abdominal distention and presence of clear yellow fluid with fibrin clots in the abdominal cavity. Congestion in liver, kidneys and intestines was also noticed. Histopathologically, dilatation of sinusoids, atrophy and degeneration of hepatocytes, marked thickening of capsule and fatty change of liver were observed. Congestion of glomeruli and urate deposits in the lumen of collecting tubules were noticed in the kidneys.

India

Davis *et al.* (2012) studied hepato-renal pathology associated with nutritional and metabolic diseases in chickens. Ascites syndrome was observed in 13 chickens with accumulation of clear yellowish fluid in the abdominal cavity. Microscopically, liver revealed multifocal areas of necrosis and disorganization of hepatic cords. Intertubular hemorrhages and degeneration was seen in kidney sections.

2.5.2 Gout

India

Nayak *et al.* (1988) reported an outbreak of gout in broilers in West Bengal. Total mortality was 60.33 %. Mortality in age groups 0-7 days, 8-14 days, 15-21 days and 21-29 days was 0%, 14%, 20% and 26.33% respectively. Histologically the liver showed multiple necrotic foci throughout the parenchyma and massive deposition of urate crystals in the sinusoids and central veins in the form of fine network of meshes. Kidney also showed complete destruction of both glomeruli and tubular structures with abundant deposition of urate crystals in the form of spongy balls of variable sizes.

Rao *et al.* (1993) investigated an outbreak of gout in broiler birds in organized poultry farm in East Godavari district of Andhra Pradesh and recorded 17.90 % mortality in the flock of 2000 broilers in a span of 5 days.

Mir *et al.* (2005) investigated an outbreak of gout in a flock of Kashmir favorella maintained under intensive managemental system, causing a mortality of 18.76% birds over a period of six months. Severe changes with presence of urate crystals were found in kidneys. Heart, liver and serosal surfaces of the intestines along with proventriculus and gizzard had whitish frosty appearance. Histopathologically, acute to chronic nephritis with hepatitis were predominant lesions.

Kumar *et al.* (2008) reported that mortality due to gout among 40 flocks varied from 2.04 to 25.00 % with an average of 8.85 % and majority of the flocks showed mortality ranging from 2.04 to 8.00 %.

Jana *et al.* (2009) examined a total of 33,713 broiler birds during 2004 and 2005 in West Bengal. Overall incidence of gout recorded was 7.03%.

Davis *et al.* (2012) studied hepato-renal pathology associated with nutrional and metabolic diseases in chickens. Gout was observed in 18 chickens showing deposition of white chalky crystals on the visceral organs and joints. Microscopically the liver and kidneys showed urate crystal deposition.

Singh *et al.* (2013) recorded that the prevalence of visceral gout in different commercial broiler farms of Chhattisgarh state was around 21.47%.

2.6 Fatty Liver

India

Fatty liver haemorrhagic syndrome in poultry from Tamil Nadu was reported by Parthasarathy *et al*. (1979). Grossly, massive blood clots covering the liver and abdominal cavity were seen. Liver revealed rupture of the capsule and was pale yellowish in colour and extremely friable in consistency. In others there were haematomas of varying sizes in the parenchyma and/or ecchymoses over the capsule. Histopathologically, varying degrees of fatty changes, focal parenchymal and/or subcapsular haemorrhages, disruption and disappearance of reticulum fibres along with the sinusoids were noticeable features.

Lonkar and Prasad (1988) reported fatty liver syndrome in chickens. Grossly liver was enlarged and friable with rounded edges with mild to moderate congestion. Histopathologically, hepatocytes contained fat vacuoles in different cords pushing aside the nucleus. In severely affected cases the normal architecture of liver was disrupted. The cords were distorted and disrupted.

Joshi and Bhagwat (1995) recorded incidence of fatty liver syndrome in poultry. Mortality data of 10 years (1979-1989) from AICRP on poultry for meat and of 6 years (1983-‘89) from the poultry research station, Akola, was analyzed to find out the incidence of fatty liver syndrome in layers and broilers. The incidence was significantly higher in broilers (8.83%) than layer strain (1.27%). Grossly, liver was enlarged, greasy to touch, pale, fragile with rounded edges and showed capillary haemorrhages and hematoma. Histopathological examination revealed mild to severe fatty changes. Parenchymatous cells showed large fatty vacuoles causing complete distortion of the architecture of hepatic cells and hepatic lobules. Hepatic lobules also revealed haemorrhage.

Davis *et al.* (2012) studied hepato-renal pathology associated with nutrinonal and metabolic diseases in chickens. Fatty liver syndrome was observed in 41 chickens. Histopathologically, liver showed enlargement with yellowish brown discolouration and hepatocytes were distended with fat vacuoles.

2.7 Fungal disease

2.7.1 Aspergillosis

Abroad

Sultana *et al.* (2015) studied the pathological lesions of aspergillosis in commercial broiler chickens at Chittagong district and recorded overall 6.14 % incidence of aspergillosis. Highest incidence (8.22%) was observed in rainy season and lowest (3.16%) in winter but moderate (5.16%) in summer season. Occurrence of disease was higher (8.27%) in age between 6-10 days and lower (4.11%) in age between 0-5 days.

India

Chaudhury and Kwatra (1977) examined seventy cases of respiratory mycosis in chickens in Assam. Incidence of infection was found to be highest (34.25%) in chickens between 10 days to 1 month old and was comparatively low in adults (3.07%). Study also revealed higher incidence of this condition in winter.

Sharma *et al.* (1979) reported three outbreaks of acute aspergillosis, which occurred concurrently in three separate flocks. A mortality rate up to 75 % was reported in 3-4 days old chicks.

Rao *et al.* (1982) reported an acute outbreak of aspergillosis in chicks at private poultry farm in Hyderabad with a mortality rate of 26 %.

Dhaliwal (1989) recorded seventeen outbreak of respiratory mycosis. Mortality in different outbreaks varied from 0.2 to 31.4 % and birds between 5 to 20 days of age were found to be more susceptible.

2.8 Nephro-hepatotoxicity

Abroad

Lalrintluanga and Baruah (1997) noticed aflatoxicosis in broiler chickens. Liver showed enlargement, congestion and pale appearance with extreme distension of gall bladder with bile. The hepatic surface appeared glistening and fatty with small white nodular foci. Histologically, degenerated, necrosed areas were replaced by newly formed hepatocytes. The biliary epithelium showed active proliferation with hyperplastic changes in hepatocytes in portal region. MNC infiltration in the interlobular connective tissue was also present.

Outbreaks of aflatoxicosis in broilers were reported by Al-Sadi *et al*. (2000) in Iraq. Liver showed swelling and yellowish discoloration. Microscopically, severe fatty degeneration, cytoplasmic vacuolization of hepatocytes and proliferation of bile duct epithelium were detected in periportal zone. In some cases, nodular hyperplasia of liver parenchyma was also seen.

India

Raina (1989) reported the overall 6.22% prevalence of naturally occurring mycotoxicosis in Punjab. Out of 148 outbreaks of mycotoxicosis, 96 were in broilers.

Asim *et al.* (1990) described occurrence of aflatoxins in poultry liver and associated pathological changes. Histopathological changes included fatty change, cellular dissociation, necrosis, cellular infiltration, fibrosis and bile duct hyperplasia.

Prevalence and pathology of mycotoxicosis in poultry was recorded by Singh *et al*. (1994). Liver was pale yellow, mottled, moderate to severely enlarged with subcapsular haemorrhages. Microscopically, congestion, haemorrhages, degeneration and necrosis of hepatocytes, bile duct proliferation with infiltration of MNCs and heterophils and individualization of hepatocytes were observed.

2.9 Intestine

Abroad

Lenz *et al.* (1998) isolated adenoviruses and reoviruses from proventriculus and intestinal tract of broiler chickens which were evaluated for gastrointestinal pathogenicity in SPF Leghorn chickens in Alabama. Chickens in all infected groups developed wet and unformed fecal droppings. Lesions found in reovirus-inoculated chickens were hyperplasia of lymphocyte aggregates in various organs and mild gizzard erosions. Adenovirus infected chickens exhibited more severe lesions in the digestive tract which consisted of marked gizzard erosions, necrotizing pancreatitis and mild proventriculitis. .

Otto *et al.* (2006) studied the intestinal tract etio-pathology associated with runting and stunting syndrome (RSS) in Northern Germany with a special focus on rotaviruses (RVs). Severe villous atrophy was seen in chicks with RSS. Lesions were distributed in the middle-to-distal small intestine. In addition, RV particles were observed in intestinal contents of flocks with RSS.

In 2011, Daryoush *et al.* performed a study to find out the presence of different pathological lesions occurring in the intestines of dead fowls suffering with enteritis at

Tabriz poultry clinics in Iran. Prevalence of the various kinds of enteritis and their association with age, strain and sex was evaluated. Highest rate of occurrence (78.57%) was found in broilers. Also, a significant co-relation of occurrence of enteritis with sex and age was seen. Most severe cases were seen in males 4-6 weeks of age and in the females 1-3 weeks of age. Histopathological, enteritis could be categorized as catarrhal, hemorrhagic and necrotic enteritis.

Nunez *et al.* (2016) recorded the signs of enteric disorders in broiler chickens. Grossly, curving of duodenal loop and intestines filled with liquid and gaseous content was observed. Histopathologically, pancreatic atrophy and enteritis characterised by fusion of intestinal villi, hyperplasia of lymphoid follicles, hemorrhage in the lamina propria and infiltration of lymphocytes and plasma cells was seen.

India

Grossly, accumulation of clear or amber-colored watery or jelly-like fluid in the pericardial sac and discolored, swollen, reticulated and friable liver with focal necrosis were observed in Hydropericardium hepatitis syndrome of broilers by Ganesh and Raghavan (2000). Microscopically, small multifocal areas of coagulative necrosis, mononuclear cell infiltration and the presence of intranuclear basophilic inclusions in the hepatocytes were observed.

2.10 Proventriculus and Gizzard erosions

Abroad

Nakamura *et al.* (2002) investigated histopathological lesions in hydropericardium syndrome with pancreatic necrosis and gizzard erosions in 19-day-old broilers. Macroscopically, hydropericardium, pinpoint white foci in the pancreas and ventricular erosions was absorbed. Histopathologically, chicken had multifocal hepatic necrosis with intranuclear inclusions in hepatocytes, multifocal necrosis of pancreatic acinar cells with intranuclear inclusions, focal necrosis of the ventricular koilin layer and degeneration of the ventricular glandular epithelium with intranuclear inclusions was seen.

In Poland, a study was carried out by Dolka *et al*. (2012) to estimate the prevalence of histopathological lesions in the different organs of commercial chickens. Out of a total of 189 cases, 66.7% of the affected cases were broiler chickens. The gastrointestinal tract especially liver was found to be the most frequently affected site with regards to the presence of histopathological lesions. In 29% of the cases of hepatic injury, pathognomonic lesions associated with inclusion body hepatitis were found. Also, proventriculitis and gizzard lesions were seen in many cases. Inclusions in the epithelial cells within the proventriculus were also noticed in many cases.

Noiva *et al.* (2015) detected proventricular necrosis virus from outbreak of transmissible viral proventriculitis associated with runting stunting syndrome in 25-28 day old broiler chickens. At necropsy, enlarged proventriculus with diffusely pale serosa and thickened walls were seen. Microscopically, degeneration and necrosis of the epithelium of the proventricular glands, glandular hyperplasia and formation of lymphoid nodules within the glandular parenchyma was noticed.

Chapter-3

Materials and Methods

Chapter-4

Results

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CHAPTER 3

MATERIALS AND METHODS

The present investigation was carried out to study the etiology, gross and histopathology of gastrointestinal tract lesions of broilers.

3.1 Study area:

To achieve the envisaged objectives of the research, a survey of 200 different poultry flocks in and around Jammu was conducted during the period July 2016 to June 2017. The study period was divided into four seasons as per Meteorological Department of India, Pune, viz., monsoon (July to September), post monsoon (October to November), winter (December to February) and summer (March to June) (Table 3.1 to 3.4). Age wise and season wise disease occurrence and mortality pattern at the field level was determined. Occurrence of disease/pathological conditions and mortality pattern was determined as per the method described by Thrusfield (1995). Disease occurrence was calculated as per the formula given below:

$$\text{Occurrence of disease} = \frac{\text{Total no. of morbidity + Total no. of mortality}}{\text{Total no. of birds in the flock}} \times 100$$

Mortality percentage was calculated as per the formula given below:

$$\text{Mortality (\%)} = \frac{\text{Total no. of dead birds}}{\text{Total no. of sick birds in the flock}} \times 100$$

3.2 Pathomorphological studies

3.2.1 Gross pathology

Representative carcasses were necropsied from the mortality in different flocks. Post-mortem examination of a total 632 birds was done. Systemic examination was carried out for the presence of any lesions in the GIT. Oesophagus, proventriculus, gizzard, pancreas, liver and intestine of dead birds were thoroughly examined and visible pathomorphological alterations were recorded.

3.2.2 Histopathology

After thorough gross examination, representative pieces of less than 5mm thickness from respective visceral organs, viz., oesophagus, proventriculus, gizzard, liver, intestine and pancreas were collected in 10% neutral buffered formalin solution. After 3-4 days of fixation, the tissues were washed in running tap water overnight, dehydrated in ascending grades of ethyl alcohol, cleared in xylene and ultimately embedded with melted paraffin wax. The paraffin blocks were prepared and sections were cut at 4-5 micron thickness with rotary type microtome. The paraffin embedded sections were then passed through sequential steps of deparaffinisation in xylene, rehydration by passing through descending grades of ethyl alcohol to running tap water and stained by routine haematoxylin and eosin stain (Luna, 1968).

Occurrence of gross and microscopic lesions was calculated as follows:

$$\text{Occurrence of lesions in an organ} = \frac{\text{Total no. of carcasses showing one or the other lesion in that organ}}{\text{Total no. of birds necropsied}} \times 100$$

3.3 Bacteriological studies

3.3.1 Collection of tissue samples for bacterial isolation

Liver and heart blood swabs were collected from representative dead birds aseptically into sterilized petri-plates. All the samples collected were processed on the same day.

3.3.2 Isolation and identification of bacteria

The organisms were identified on the basis of their morphological, cultural and biochemical characteristics. The procedure for isolation and identification of bacterial culture adopted for the present work was as per Cruickshank *et al.* (1975). The samples were inoculated in nutrient broth and kept at 37°C. After 24 hours of incubation, the culture was transferred to MacConkey's lactose agar (MLA) kept at 37°C. After 24 hours of incubation the pure colonies were stained with Gram's stain for microscopic examination. Respective cultures were identified on the basis of standard criteria.

Organisms giving pink coloured colonies on MLA were also cultured on eosin methylene blue agar (EMB). Cultures giving pale-yellow coloured colonies on MLA were also identified. All cultures were subjected to various biochemical tests.

3.3.3 Biochemical tests

E.coli and *Salmonella* were characterized on the basis of biochemical tests according to standard procedure described by Carter *et al*. (1994). *E.coli* and *Salmonella* isolates were subjected to Indole test, Methyl red test, Voges Proskauer test, Citrate utilization test (IMViC pattern).

3.3.3.1 Indole test

A few drops of xylene were added into the two-day-old growth of the isolate in 2ml of tryptone water and mixed thoroughly to dissolve indole and 2 drops of Kovac's reagent was added. Pink layer at the top surface of broth was considered as positive.

3.3.3.2 Methyl red test

2-4 drops of methyl red reagent were added to 2 day old growth of the isolate in 5ml of glucose phosphate peptone water (GPPW). Development of pink or bright red colour was considered to be positive and development of yellow colour was considered as negative.

3.4.3.3 Voges-Proskauer test (Barrett's method)

3ml of 5% solution of naphthol in absolute ethanol and 1 ml of 40% KOH were added to 2 day growth of the isolate in 5 ml of GPPW. Development of pink colour in the mixture was indicative of a positive test.

3.3.3.4 Citrate utilization

Slant of Simmon's citrate agar was inoculated with the culture and incubated at 37 °C for 2 days. Growth with a development of blue colour of the medium was considered as a positive reaction.

3.3.4 Maintenance of cultures

On the basis of biochemical tests, the purified cultures were inoculated on maintenance medium (nutrient agar slant) in duplicate and incubated at 37 °C for 24 hours. These were sealed with paraffin wax and slants were stored at 4°C in the refrigerator for further identification/conformation. Culture was revived after each month to keep the bacteria alive.

3.3.5 Serotyping of the *E.coli* and *Salmonella* isolates

After complete biochemical characterization, all the isolates of *E.coli* and *Salmonella* were sent for serotyping to the National *Salmonella* and *Escherichia* centre, CRI, Kasauli (HP), India.

3.4 Parasitological examination

Scrapings were taken from mucosa of intestine from suspected cases and examined by direct wet mount smear.

Table 3.1: Farm location, total strength of flocks, age, number of sick and dead birds with pathological conditions in monsoon season.

S. No.	Location of poultry farm	Strength of flock	Birds age (weeks)	Sick birds number	Number of dead birds	Pathological conditions diagnosed
1.	R.S. Pura Venod poultry farm (PF)	4000	0-1	2400	181	Omphalitis
2.	R.S. Pura Pankaj PF	1000	3-4	600	35	Coccidiosis
3.	R.S.pura Balwinder PF	2000	1-2	1500	125	Colibacillosis
4.	R.S.pura Nirmal PF	1400	2-3	1200	74	Colibacillosis
5.	Sujadpur	700	3-4	560	18	Coccidiosis
6.	Chatha	3200	3-4	3020	129	IBD suspected
7.	Chatha	4000	1-2	1500	44	Salmonellosis
8.	Domana Trishla PF	6000	4-5	5000	266	Colibacillosis

9.	Domana Raj Kumar PF	3500	2-3	2100	105	Colibacillosis
10.	Simbal	2000	1-2	1400	160	Colibacillosis
11.	Simbal	10000	3-4	1950	08	Fatty liver syndrome
12.	Samba	2500	1-2	1500	46	Salmonellosis
13.	Samba Gaya PF	4000	3-4	3370	210	IBD suspected
14.	Samba Sonu PF	3000	2-3	1500	156	Colibacillosis
15.	Palawala	9900	3-4	5600	59	Coccidiosis
16.	Bari Brahman	2000	1-2	1400	142	Colibacillosis
17.	Bari Brahmana Versha PF	1000	2-3	700	69	Colibacillosis
18.	Satwari Bigani PF	2000	4-5	1690	69	IBD suspected
19.	Satwari Bittu PF	14000	2-3	1350	07	Fatty liver syndrome
20.	Satwari Yash PF	1500	3-4	1000	44	Non specific enteritis
21.	Satwari Arshdeep PF	4000	4-5	2400	67	Non specific enteritis, Proventriculitis
22.	Satwari	3000	4-5	2450	61	IBD suspected
23.	Bakore	16000	2-3	1500	03	Fatty liver syndrome
24.	Akhnoor Ashwari PF	4500	2-3	1000	06	Fatty liver syndrome
25.	Akhnoor Amar PF	4000	2-3	2600	55	Non specific enteritis
26.	Akhnoor Roshan PF	1000	2-3	700	99	Colibacillosis
27.	Marh	4400	0-1	2900	198	Omphalitis
28.	Marh	5000	2-3	4000	239	Colibacillosis
29.	Raipur Khalsa PF	4000	0-1	2400	165	Omphalitis
30.	Chawala Sanjeev PF	2200	0-1	1360	120	Omphalitis
31.	Kud	1000	3-4	900	134	IBD suspected
32.	Udampur	1400	2-3	940	19	Non specific enteritis

33.	Raya	3000	2-3	1600	46	Non specific enteritis and proventriculitis
34.	Raya Sawaran PF	3000	4-5	1600	202	Colibacillosis
35.	Raya	1000	0-1	700	44	Salmonellosis
38	Satwari Yash PF	5500	3-4	3200	46	Coccidiosis
36.	Manda Munir PF	700	3-4	520	87	Colibacillosis
37.	Marh Soma PF	3000	3-4	2100	255	Colibacillosis
38.	Marh Radhe Sham PF	1500	3-4	1250	56	IBD suspected
39.	Jakh	3000	2-3	2200	230	Colibacillosis
40.	Jakh Taw PF	2000	1-2	1300	128	Colibacillosis
41.	Jakh Uttam PF	3000	3-4	1800	265	Colibacillosis
42.	Bishna Prem PF	2500	4-5	1900	158	Colibacillosis
43.	Bishna GS PF	15000	0-1	13,300	162	Omphalitis
44.	Kotbalwal Subash PF	1500	3-4	950	102	Colibacillosis
45.	Kotbalwal	5000	4-5	3800	266	Colibacillosis
46.	Ghagwal Bhullar PF	2400	2-3	1400	52	Salmonellosis
47.	Ghagwal	1500	2-3	900	123	Colibacillosis
48.	Rehasi	2500	1-2	1500	58	Omphalitis
49.	Rehasi	3200	3-4	2300	179	Colibacillosis
50.	Amphalle Kala PF	2500	1-2	1000	155	Colibacillosis
51.	Amphalle	1000	3-4	750	70	IBD suspected

Table 3.2: Farm location, total strength of flocks, age, number of sick and dead birds with pathological conditions in autumn (post-monsoon) season.

S. No.	Location of poultry farm	Strength of flock	Birds age (weeks)	Sick birds number	Number of dead birds	Pathological condition diagnosis
1.	Manda	6500	3-4	3800	65	Hepatotoxicity
2.	Chatha	1000	4-5	615	78	Colibacillosis
3.	Chatha Khalsa PF	2000	0-1	1080	81	Omphalitis
4.	R.S.Pura	2000	0-1	1130	46	Omphalitis
5.	R.S. Pura Parveen PF	2500	0-1	1460	71	Omphalitis
6.	R.S. Pura VK PF	3000	2-3	1780	87	Salmonellosis
7.	Marh	3000	3-4	1600	42	Coccidiosis
8.	Satwari	800	1-2	440	23	Omphalitis
9.	Satwari	2500	1-2	1270	135	Omphalitis
10.	Ghagwal	1800	3-4	1170	125	Colibacillosis
11.	Ghagwal	1500	4-5	1150	109	Colibacillosis
12.	Dhansar Indepal PF	2000	2-3	1200	44	Salmonellosis
13.	Dhansar	1000	2-3	800	72	Colibacillosis
14.	Dhansar	4000	1-2	3000	180	Colibacillosis
15.	Akhnoor Rajesh PF	4400	0-1	2600	125	Omphalitis
16.	Akhnoor	400	1-2	230	13	Salmonellosis
17.	Akhnoor	1500	3-4	810	149	Colibacillosis
18.	Akhnoor Deol PF	3000	2-3	1700	310	Colibacillosis
19.	Raya morh	3500	1-2	2000	49	Non specific enteritis
20.	Raya morh	4000	3-4	3000	172	IBD suspected
21.	Nanak nagar Darshan PF	2500	1-2	1510	201	Colibacillosis
22.	Vijaypur	5000	0-1	2600	89	Salmonellosis
23.	Hira nagar	1500	3-4	820	10	Coccidiosis
24.	Vijaypur	2000	2-3	1300	128	Colibacillosis
25.	Hira nagar	3000	2-3	1540	78	Non specific enteritis
26.	Bilawar	1300	4-5	1100	12	IBD suspected
27.	Dansar	1500	3-4	950	77	Colibacillosis
28.	Chatha guzra	3000	2-3	1800	112	Colibacillosis

29.	Chatha guzra	3300	1-2	1880	105	Colibacillosis
30.	Simbal	500	1-2	300	49	Colibacillosis
31.	Simbal	1200	3-4	800	79	Colibacillosis
32.	Talab Tilo	800	3-4	600	63	Colibacillosis
33.	Talab Tilo	1500	1-2	880	28	Salmonellosis
34.	Smaylpur	4000	3-4	1200	22	Ascites
35.	Bishna	1000	2-3	680	65	Colibacillosis
36.	Satrayian	2000	3-4	1545	62	Non specific-enteritis and liver ailments
37.	Satrayian	1700	3-4	1225	22	Non specific-enteritis and liver ailments
38.	Marh	1400	2-3	970	18	Non specific enteritis
39.	Gajnso	2000	3-4	1400	52	Non specific-enteritis and liver ailments
40.	Gajnso	700	2-3	350	03	Ascites
41.	Ghagwal	200	2-3	115	35	Colibacillosis
42.	Channi JK PF	2000	2-3	1270	96	Colibacillosis
43.	Channi Rishu Saini PF	4000	2-3	2200	04	Gout
44.	Sujadpur	1200	1-2	650	41	Salmonellosis
45.	Sujadpur	7000	3-4	4500	186	Colibacillosis
46.	Digyana	1100	3-4	740	29	Hepatotoxicity
47.	Digyana	1400	1-2	800	145	Colibacillosis
48.	Flyai madal	1200	2-3	580	08	Ascites
49.	Flyai madal Chaudry PF	800	4-5	500	88	Colibacillosis
50.	Majalta	3000	2-3	1800	18	Ascites
51.	Samba	1000	3-4	540	140	Colibacillosis
52.	Samba	1200	2-3	680	105	Colibacillosis
53.	Ghagwal	4400	2-3	3000	144	Colibacillosis
54.	Gatala	2600	2-3	2025	75	Non specific enteritis and gizzard erosions

Table 3.3: Farm location, total strength of flocks, age, number of sick and dead birds with pathological conditions in winter season

S. No.	Location of poultry farm	Strength of flock	Birds age (days)	Sick birds number	Number of dead birds	Pathological condition diagnosis
1.	Kotli	2000	0-1	1580	55	Salmonellosis
2.	Kotli	1800	2-3	1300	113	Colibacillosis
3.	Samba	1500	2-3	1000	106	Colibacillosis
4.	Samba	1500	1-2	1200	122	Colibacillosis
5.	Satrayian Parveen PF	4000	2-3	3120	165	NCD/Avian influenza suspected
6.	Satrayian Manhas PF	5000	2-3	3910	192	NCD/Avian influenza suspected
7.	Akhnoor	1500	3-4	980	136	Colibacillosis
8.	Amphalla	2000	1-2	1600	139	Colibacillosis
9.	Amphalla	1000	1-2	540	59	Salmonellosis
10.	R.S.Pura	1500	2-3	1160	57	Colibacillosis
11.	R.S.Pura	12500	2-3	-	01	Internal haemarrhages
12.	Chakmar	1500	4-5	1310	160	IBD suspected
13.	Kud	3000	1-2	800	20	Brooder pneumonia suspected
14.	Kud	2000	4-5	1770	78	IBD suspected
15.	Satwari	500	4-5	290	12	Hepatotoxicity
16.	Satwari	4000	3-4	3500	107	Colibacillosis
17.	Manda Ramesh PF	4903	3-4	3300	132	Colibacillosis
18.	Digyana	2000	1-2	1150	92	Omphalitis
19.	Smaylpur	5800	3-4	1500	10	Ascites
20.	Heera Nagar Kala PF	1400	3-4	1200	96	IBD suspected
21.	Bajalta Vicky PF	2000	1-2	1500	77	Omphalitis
22.	Ghagwal	2800	3-4	2370	87	IBD suspected
23.	Ghagwal	1500	3-4	1250	80	Colibacillosis
24.	Ghagwal	2500	2-3	1950	139	NCD/Avian influenza suspected
25.	Deoli	1200	4-5	920	68	Colibacillosis
26.	Deoli	1400	3-4	840	47	Hepatotoxicity
27.	Deoli	1800	1-2	1450	73	Colibacillosis
28.	Samba Manjoor PF	1000	1-2	780	56	Colibacillosis

29.	Palowala	3000	4-5	2750	61	IBD suspected
30.	Palowal	3500	3-4	3150	155	IBD suspected
31.	Rehasi	1000	1-2	640	29	Salmonellosis
32.	Rehasi	1200	4-5	1000	54	IBD suspected
33.	Vjaypur	4000	3-4	3500	172	Colibacillosis
34.	Vijaypur	1000	4-5	780	92	IBD suspected
35.	Udampur	2500	3-4	1900	98	Colibacillosis
36.	Udampur	4000	1-2	1400	36	Brooder pneumonia suspected
37.	Udampur	1000	1-2	680	60	Colibacillosis
38.	Simbal	3000	3-4	2580	85	IBD suspected
39.	Simbal	2500	3-4	2100	86	Colibacillosis
40.	Channi	600	0-1	345	28	Omphalitis
41.	Bajalta	1000	0-1	570	18	Salmonellosis
42.	Bishna Prem PF	2500	1-2	1350	49	Salmonellosis
43.	Chawala	1000	0-1	660	34	Salmonellosis
44.	Marh	1000	2-3	640	85	Colibacillosis
45.	Marh Sanjeev PF	4000	1-2	2000	28	Brooder pneumonia suspected
46.	Parkhu	3200	3-5	2300	192	Colibacillosis
47.	Sujadpur	1000	2-3	480	04	Ascites

Table 3.4: Farm location, total strength of flocks, age, number of sick and dead birds with pathological conditions in summer season.

S. No.	Location of poultry farm	Strength of flock	Birds age (weeks)	Sick birds number	Number of dead birds	Pathological condition diagnosis
1.	R.S. Pura	2000	3-4	1860	110	Colibacillosis
2.	R.S. Pura	1400	4-5	1000	38	Nephro-hepatotoxicity
3.	R.S. Pura	1200	3-4	1000	74	IBD suspected
4.	Mishri Wala	500	1-2	350	96	Colibacillosis
5.	Ghagwal	1000	0-1	600	59	Omphalitis
6.	Bishna	1800	3-4	1500	142	IBD suspected
7.	Satwari	1000	2-3	480	01	Ascitis
8.	Raipur Satwari	1400	2-3	1200	103	Colibacillosis
9.	Sujadpur	5000	1-2	1400	25	Gout
10.	Chatha	400	4-5	260	36	IBD suspected
11.	Chatha	2000	3-4	1760	119	Colibacillosis
12.	Chatha Gujra	2700	1-2	1460	54	Salmonellosis
13.	Samba	3000	1-2	2800	138	Colibacillosis
14.	Samba	1700	2-3	1520	105	Colibacillosis
15.	Simbal	2000	0-1	1180	44	Salmonellosis

16.	Simbal	4000	2-3	3650	145	Colibacillosis
17.	Hira Nagar	4000	3-4	3400	122	Colibacillosis
18.	Hira Nagar	2100	3-4	1200	40	Hepatotoxicity
19.	Jakh	1600	3-4	1400	125	Colibacillosis
20.	Jakh	4000	1-2	2340	50s	Salmonellosis
21.	Kotli	1000	3-4	780	103	IBD suspected
22.	Majra Radhesham PF	6000	1-2	1900	12	Gout
23.	Datriyal	2000	1-2	1140	37	Salmonellosis
24.	Datriyal	1000	2-3	900	94	Colibacillosis
25.	Akhnoor	2000	0-1	1250	45	Omphalitis
26.	Akhnoor	2000	2-3	1020	24	Salmonellosis
27.	Akhnoor	2000	1-2	1800	116	Colibacillosis
28.	Digyana	3000	3-4	2850	135	Colibacillosis
29.	Digyana	2000	2-3	1800	164	Colibacillosis
30.	Udampur	10400	2-3	-	02	Internal haemorrhages
31.	Rehasi	6000	2-3	2200	08	Gout
32.	Rehasi	1500	3-4	490	07	Ascites
33.	Kud	2500	1-2	1800	61	Salmonellosis
34.	Kud	4000	1-2	1400	07	Gout
35.	Marh	3500	2-3	1200	11	Gout
36.	Vijaypur	13500	2-3	-	02	Internal haemorrhages
37.	Nanak Nagar Nirmal PF	2500	0-1	1570	38	Salmonellosis
38.	Nanak Nagar	2000	3-4	1900	146	Colibacillosis
39.	Amphalla	2000	4-5	1780	91	Colibacillosis
40.	Amphalla	1500	2-3	1360	123	Colibacillosis
41.	Barnai	2000	2-3	1110	43	Non specific enteritis
42.	Gatala	3500	1-2	2300	62	Non specific enteritis
43.	Sujadpur	2700	1-2	2550	162	Colibacillosis
44.	Sujadpur	1400	2-3	1000	39	Non specific enteritis
45.	Palawal	550	4-5	320	98	Colibacillosis
46.	Deoli	1400	2-3	940	26	Salmonellosis
47.	Deoli	1600	1-2	1430	60	Non specific enteritis

Chapter-4

Results

CHAPTER 4

RESULTS

Occurrence of diseases

The present study was carried to study etio-pathomorphology of gastrointestinal tract of broiler chickens in various parts of Jammu. Data was collected from 200 flocks in different areas in and around Jammu. The cumulative strength of birds in the farms was 564,753. Necropsy was conducted on representative carcasses of 632 birds. Occurrence and mortality pattern of diseases at different farms is given in Table 4.1. Disease conditions recorded in different age groups is given in Table 4.2.

Different diseases/pathological conditions affecting broiler population in and around Jammu were colibacillosis, omphalitis, salmonellosis, IBD, coccidiosis, haemorrhages in proventriculus, caseous nodules in lungs, hepatotoxicity, gout, ascites, internal haemorrhage, fatty liver syndrome, non specific enteritis and non specific liver ailments. In cases where haemorrhages in proventriculus were seen, occurrence of Newcastle disease/avian influenza was suspected. Likewise in cases where caseous nodules were seen, brooder pneumonia was suspected.

Table 4.1: Occurrence (%) and Mortality pattern of diseases at different farms in Jammu.

S. No.	Confirmed and suspected pathological conditions	Total no. of flocks	Total number of birds	Total number of morbid birds (app.)	Total number of dead birds	Occurrence (%)	Mortality (%)
1.	Colibacillosis	82	180753	126000	10500	24.16	8.33
2.	Omphalitis	17	53900	36685	1666	6.79	4.54
3.	Salmonellosis	24	50600	29250	1071	5.36	3.66
4.	IBD	22	44800	38000	2272	7.13	5.97
5.	Coccidiosis	06	21600	12380	210	2.22	1.69
6.	Haemorrhages in proventriculus	03	11500	8980	496	1.67	5.52

7.	Caseous nodules in lungs	03	11000	4200	84	0.75	2.00
8.	Hepatotoxicity	06	13000	7830	231	1.42	2.95
9.	Gout	05	24500	8100	63	1.44	0.77
10.	Ascites	07	17200	6400	72	1.14	1.12
11.	Non specific enteritis	16	35600	23485	791	4.29	3.36
12.	Fatty liver syndrome	04	42500	5800	24	1.03	0.41
13.	Non specific liver ailments	02	4700	2765	84	0.50	3.03
14.	Internal haemorrhage	03	36400	-	05	-	-
	Total	20	564753	309875	17569	57.98	5.66

Occurrence of colibacillosis (24.16%) was maximum followed by that of IBD (7.13%), omphalitis (6.79%), salmonellosis (5.36%), non specific enteritis (4.29%), coccidiosis (2.22%), haemorrhages in proventriculus (1.67%), gout (1.44%), hepatotoxicity (1.42%), ascites (1.14%), fatty liver syndrome (1.03%), caseous nodules in lungs (0.75%) and non specific liver ailments (0.50%).

The maximum mortality was caused by colibacillosis (8.33%) followed by IBD (5.97%), haemorrhages in proventriculus (5.52%), omphalitis (4.54%) followed salmonellosis (3.66%), non specific enteritis (3.36%), non specific liver ailments (3.03%), hepatotoxicity (2.95%), caseous nodules in lungs (2.00%), coccidiosis (1.69%), ascites (1.12%), gout (0.77%) and fatty liver syndrome (0.41%).

In birds 0-1 week of age the pathological conditions recorded were omphalitis and salmonellosis. Colibacillosis, omphalitis, salmonellosis, caseous nodules in lungs, gout and non specific enteritis were seen in birds of 1-2 weeks of age. However, colibacillosis, salmonellosis, haemorrhages in proventriculus, ascites, gout, internal haemorrhage, fatty liver syndrome, proventriculitis, gizzard erosions and non specific enteritis were major conditions observed in 2-3 weeks old birds. Birds in 3-4 weeks of age were found to be affected with ascites, colibacillosis, coccidiosis, IBD, hepatotoxicity, fatty liver syndrome, non specific liver ailments, non specific enteritis whereas 4-5 week old birds

were found to be affected with colibacillosis, IBD, hepatotoxicity and non specific enteritis (Table 4.2).

Table 4.2: Age wise distribution of disease conditions

Sr. No.	Age of birds (weeks)	Pathological conditions
1.	0-1	Omphalitis (*E.coli and Salmonella*) and salmonellosis
2.	1-2	Colibacillosis, omphalitis, salmonellosis, caseous nodules in lungs, gout and non specific enteritis.
3.	2-3	Colibacillosis, salmonellosis, haemorrhages in proventriculus, ascites, gout, internal haemorrhage, fatty liver syndrome, non specific proventriculitis and enteritis.
4.	3-4	Ascites, colibacillosis, IBD, coccidiosis, Hepatotoxicity, fatty liver syndrome, non specific liver ailments and non specific enteritis.
5.	4-5	Colibacillosis, IBD, Hepatotoxicity and non specific enteritis.

The following are the details of diseases/pathological conditions recorded in different farms.

4.1 Bacterial diseases

4.1.1 Colibacillosis

Occurrence of colibacillosis was observed in 82 flocks. It was the major cause of mortality in all age groups with occurrence of 24.16% and mortality of 8.33%. Infection of *E.coli* was observed throughout the year. Clinically, birds were dull, depressed, listless and huddled together. Birds were anorectic with droopy and ruffled feathers. Some birds were showing signs of gasping and labored breathing.

Gross lesions

The characteristic lesions recorded were fibrinous pericarditis and perihepatitis (Plate 1). The pericardial sac was opaque, whitish and adherent to the epicardium. The liver was moderately enlarged with whitish thin fibrinous layer on its surface. The air sacs were opaque and thickened. Proventriculus showed presence of mucoid exudates adhering to the underlying mucosa (Plate2). In addition, focal haemorrhagic lesions in

wall of proventriculus were seen in some birds. Congestion and haemorrhages in intestinal tract was commonly seen especially in duodenum (Plate3).

Microscopic lesions

Perihepatitis was seen in almost all the cases (Plate 4), where fibrin strands admixed with heterophils formed separate layer over the underlying liver parenchyma which showed varying degrees of degeneration and necrosis. Liver sections were frequently congested. Multifocal areas of degeneration, necrosis, with infiltration of heterophils were commonly seen (Plate 5). Proventriculus revealed necrosis of epithelium of mucosal folds, edema, congestion and severe infiltration of inflammatory cells mainly heterophils in lamina propria and underlying sub mucosa (Plate 6). In some cases tunica muscularis and serosal layers were severely congested. Tunica serosa had large amounts of fibrinous exudate comprising of fibrin strands and heterophilic infiltration (Plate 7). Intestinal sections revealed enteritis characterized by congestion, necrosis of villi and infiltration of heterophils in lamina propria or sub mucosa and occasionally serosal layer was thickened due to severe congestion and deposition of fibrinous exudate (Plate 8).

E.coli was isolated from liver and heart of dead birds. Morphologically, *E.coli* isolates formed pink colonies when cultured on MLA (Plate 9), green metallic sheen on EMB agar (Plate 10) and organisms were Gram –ve (Plate 11). Biochemically, all the isolates were positive for indole, methyl red and negative for voges-proskauer and catalase test (Plate12). Isolates of *E.coli* belonged to serogroup O1, O22, O37, O114, O118 and O149.

4.1.2 Omphalitis

Omphalitis was recorded in 17 flocks affecting birds 0-2 weeks of age with an overall occurrence of 6.79% and mortality of 4.54%. Clinically, affected birds were depressed and huddled together near the sources of heat. Abdomen of affected chicks was enlarged and flabby.

Gross lesions

Naval was inflamed and greenish red in colour. Unabsorbed and haemorrhagic yolk sac was found to be attached to intestine through a stalk (Plate 13). The contents of the unabsorbed yolk sac were yellow in colour and foul smelling. In some chicks, the yolk content was caseous and inspissated. Liver was enlarged, congested or pale yellow in colour. Intestine showed congestion and catarrhal exudate in the lumen.

Microscopic lesions

The yolk sac wall appeared edematous along with an inflammatory zone comprising of heterophils and macrophages. Liver revealed degeneration, atrophy and necrosis of hepatocytes and consequent dilation of sinusoids (Plate 14). Enteritis was characterised by necrosis of villi, desquamation of epithelium inside the lumen of vill, presence of inflammatory cells such as lymphocytes and heterophils in lumen of lamina propria (Plate 15).

Salmonella sp. and *E.coli* was isolated from the chicks affected with omphalitis.

4.1.3 Salmonellosis

Salmonellosis was reported in chicks up to 3 weeks of age. 24 flocks were found to be affected with an overall occurrence of 5.36% and mortality of 3.66%. Clinically, diseased birds were dull, depressed with closed eyes, emaciated and diarrheic. Diarrhoea varied from being thin and watery to mucoid greenish yellow in appearance.

Gross lesions

Bronze discolouration of liver was the characteristic lesion seen in affected birds (Plate 16). Moreover, swelling, mottling and presence of multiple necrotic foci were frequently observed in the affected birds (Plate 17). In some cases liver revealed severe enlargement and haemorrhagic foci on its surface. Lungs were congested. Proventriculus showed haemorrhages and congestion on serosa (Plate 18). Serosal congestion and haemorrhages throughout the intestinal tract were commonly seen (Plate 19).

Histopathology

Histopathological lesions affecting liver were mild to severe congestion of sinusoids and central veins (Plate 20), haemorrhages, vacuolar degeneration and multifocal areas of coagulative necrosis of hepatocytes. Multifocal areas of suppurative hepatitis characterised by presence of islands of eosinophilic necrotic hepatocytes floating in pool of heterophils was the typical lesion found (Plate 21). Heterophilic infiltration in portal triads along with pyknotic and karyorrhectic nuclei of necrotic hepatocytes was also observed. Thickening of mucosal folds of proventriculus due to edema, presence of necrotic material, infiltration of heterophils and congestion in lamina propria was observed (Plate 22). Intestinal sections revealed necrotic villous epithelium along with severe congestion in lamina propria of villi and infiltration of heterophils (Plate 23).

Morphologically, *Salmonella* sp. isolates were Gram –ve rods (Plate 24) and appeared as yellow colonies when cultured on MLA (Plate 25). Biochemically, all isolates exhibited IMViC pattern characteristic of *Salmonella* spp. (I-M+Vi-C+) (Plate 26).

4.2 Viral diseases

4.2.1 Infectious bursal disease (IBD)

IBD was recorded in a total of 22 flocks with occurrence and mortality of 7.13% and 5.97%, respectively. Disease was recorded throughout the year in 3-5 week old birds. Affected birds appeared dull, depressed, anorectic had ruffled feathers and were suffering from yellowish white or greenish yellow diarrhoea. Dehydration and prostration was observed in the severely affected birds.

Gross lesions

Bursa of Fabricius was observed to be enlarged, edematous and had haemorrhages on its serosal and mucosal surfaces (Plate 27). Accumulation of creamy exudate in the lumen was present (Plate 28). Darkening and discolouration of thigh and breast muscles along with presence of paint brush like haemorrhages was noticed in the affected birds

(Plate 29). Kidneys were congested and swollen (Plate 27). Intestines showed severe enteritis characterised by mild congestion and edema especially in duodenum in many birds.

Microscopic lesions

Bursa of Fabricius revealed congestion, edema and infiltration of inflammatory cells mainly heterophils in interfollicular areas (Plate 30, 31). Degeneration, necrosis of lymphocytes and presence of esoniphilic debris and cystic cavities in bursal follicles was observed. In addition serofibrinous exudate which was an admixture of fibrin, edema and heterophils was present in the interfollicular area and also adhering to the external bursal surface (Plate 32). Kidney showed congestion, haemorrhages and desquamation of degenerating tubular epithelial cells in the lumen (Plate 33). Liver of many birds showed moderate congestion, degeneration, coagulative necrosis and infiltration of MNCs. Enteritis was seen in most of the cases characterised by congestion, haemorrhages and necrosis of villi with infiltration of inflammatory cells mainly lymphocytes and a few heterophils in lamina propria (Plate 34).

4.2.2 Haemorrhages in proventriculus (Suspected cases of Newcastle disease/Avian influenza)

Haemorrhages in proventriculus in 2-3 week old chickens were recorded with 1.67% occurrence and 5.52% mortality. These cases were recorded during the winter season only. Clinically, severely affected birds exhibited signs of dullness, depression, sneezing, nasal discharge, prostration and greenish watery diarrhoea.

Gross lesions

Hemorrhages in proventriculus were the most prominent and commonly seen lesion (Plate 35). Severe congestion and haemorrhages were seen involving different parts of intestine particularly caecum. Trachea revealed mild haemorrhages. Liver had mottled appearance in most of the cases (Plate 36).

Microscopic lesions

In the intestine, the lesions consisted of severe congestion, presence of MNCs mainly lymphocytes and few macrophages (Plate 37). Proventriculus revealed congestion, haemorrhage, edema in lamina propria and sub mucosa along with necrosis of mucosal papillae (Plate 38). Degeneration of hepatocytes, severe congestion in sinusoids and blood vessels in portal triad areas and mild infiltration of heterophils in periportal areas was seen in liver (Plate 39).

4.3 Protozoan diseases

4.3.1 Coccidiosis

Coccidiosis was observed in 3-4 week old birds in six flocks with occurrence of 2.22% and mortality of 1.69%. Clinically, the affected birds voided bloody feces. Comb and wattles of affected birds were pale and anemic.

On necropsy, haemorrhagic caecal walls with severe petechial haemorrhages on mucosal surface were seen (Plate 40). Occasionally caeca fully distended with blood were also found (Plate 41).

Histopathological examination revealed severe necrosis and exfoliation of intestinal layers exposing tunica muscularis and severe infiltration of inflammatory cells mainly heterophils in tunica mucosa and sub mucosa (Plate 42). Intestinal section typically showed severe congestion, haemorrhage and edema. Necrosis and degeneration of villi and sub-mucosal glandular epithelium; harboring coccidian life-cycle stages was characteristically present (Plate 43). Presence of developing schizonts and oocysts in villi crypts and sub mucosal glandular epithelium caused severe degeneration and necrosis of the epithelium (Plate 44, 45). Scrapings taken from mucosa of intestine from suspected cases followed by direct examination of wet mount smears under microscope, revealed presence of myriad *Eimeria* oocysts with each oocyst containing four sporocysts and each sporocyst having two sporozoites (Plate 46).

4.4 Metabolic diseases

4.4.1 Ascites

Ascites was observed in 2-4 week old birds with total occurrence and mortality of 1.13% with 0.14%, respectively in seven flocks. Clinically, abdominal distension and increase in water consumption was observed in affected birds. Grossly, abdomen was found to be filled with clear straw coloured fluid (Plate 47). Liver was enlarged, pale and had irregular edges. Kidneys were also pale and swollen. Microscopically changes in liver varied from vacuolar degeneration, fatty degeneration to severe coagulative type of necrosis of hepatocytes (Plate 48). Additionally, heterophils were found to have infiltrated in areas undergoing degeneration and necrosis.

4.5 Nephro-hepatotoxicity

This was recorded in six different farms affecting 3-6 week old chickens with 1.42% occurrence and causing 2.95% mortality.

In most of the cases, kidneys were pale and swollen (Plate 49). Some kidneys had urate crystal deposition. In many cases pin point haemorrhages on the surface of the kidneys were seen (Plate 50). In some cases kidneys appeared dark red due to congestion (Plate 51). In addition liver was pale, mottled (Plate 52, 53) and occasionally also had necrotic foci on its surface.

Histopathologically, in kidney sections interstitial capillaries were dilated and engorged with blood. Severe haemorrhages and edematous fluid accumulation was also noticed in intertubular areas. Also degeneration and desquamation of tubular epithelial cells into the lumen was seen (Plate 54). In some cases degeneration and coagulative necrosis of tubular epithelium was seen. The necrotic tubular cells were hypereosinophilic. Such necrotic areas were also infiltrated focally by heterophils in low to moderate numbers (Plate 55). In liver major histopathological changes were degeneration, necrosis and infiltration of inflammatory cells chiefly heterophils and macrophages and lymphocytes in low numbers. Portal triad areas were also infiltrated with inflammatory cells predominantly heterophils (Plate 56).

4.6 Fatty Liver Syndrome

Fatty liver syndrome was seen in 4 flocks accounting for an overall occurrence and mortality of 1.03% and 0.41%, respectively. Broilers in age group 2-4 weeks were mostly affected.

Grossly, liver appeared friable and pale yellow in colour (Plate 57). Microscopically, fatty degeneration which was characterised by the presence of vacuoles in the cytoplasm pushing the nucleus to the periphery giving signet ring appearance was seen. Hepatocytes became swollen and rounded due to accumulation of fat vacuoles in the cytoplasm (Plate 58). In addition, sinusoids were dilated and congested. Necrosis of hepatocytes was observed at multifocal areas with mild heterophilic infiltration.

4.7 Internal Haemorrhages

Internal haemorrhages were recorded in a total of 5 birds belonging to three different farms. Grossly**,** big clots of blood were seen in abdominal cavity (Plate 59). In two such cases, liver rupture was found to be the cause of internal bleeding. Light pale, anemic liver and kidneys were seen. In one case large hematoma on thigh region was found which correlated with clinical history of lameness in that bird.

4.8 Occurrence and pathology of various lesions of GIT of broilers in Jammu

Detailed analysis of pathomorphological lesions occurring in GIT of birds was performed. Out of the 632 birds necropsied, almost all the birds had presence of one or the other lesion in their GIT. Intestines were found to be affected in maximum number of birds. In all, 507 birds had intestinal lesions with a percentage involvement of 80.22%. Liver, proventriculus, gizzard, pancreas and oesophagus were seen to be affected in 454, 135, 56, 53 and 19 cases with percentage involvement of 71.83%, 21.36%, 8.86%, 8.38% and 3.00%, respectively (Table 4.3). The different pathological lesions found in present study with their respective occurrences are described below.

4.8.1 Intestines

Intestines were affected in 80.22% (507/632) cases. Grossly, the lesions consisted of congestion, haemorrhages, necrosis and enteritis which were seen in 40.43%

(205/507), 21.10% (107/507), 12.42% (63/507) and 68.44% (347/507) cases, respectively as shown in Table 4.4. Microscopic pathological lesions comprising mainly of congestion, haemorrhage, edema, degeneration, necrosis, acute, sub acute and chronic enteritis; were observed in 76.72% (389/507), 57.79% (293/507), 52.66% (267/507), 90.33% (458/507), 76.72% (389/507), 69.03% (350/507), 2.36% (12/507) and 3.35% (17/507) cases, respectively as depicted in Table 4.5

Upon gross examination, congested intestines appeared red whereas, haemorrhages were either petechial or echymotic and were present both on serosa and mucosa. Enteritis was characterised by thickened intestinal wall, presence of congestion, petechial haemorrhages on the mucosa and mucoid exudate in lumen (Plate 60). Congestion and haemorrhages were frequently seen involving only the serosa of intestine (Plate 61). In such cases catarrhal exudate coating the surface of intestine as well as thickened intestinal wall was frequently noticed.

Histopathological examination of intestinal sections from cases of acute enteritis revealed necrosis of mucosal epithelium, blunting-stunting, fusion of villi and thickening because of infiltration of macrophages, heterophils and plasma cells in lamina propria and sub mucosa. Thickening of intestinal wall also due to serosal congestion and presence of heterophils was seen (Plate 62). Acute catarrhal enteritis was characterised by necrosis of epithelium, hyperplasia of goblet cells, presence of edema and inflammatory cells mainly heterophils along with severe congestion and haemorrhages in lamina propria. Necrotic enteritis characterised by severe denudation and necrosis of intestinal epithelium extending upto the crypts along with fusion of adjacent villi also noticed. Severe inflammatory reaction consisting of an admixture of necrotic epithelial cells, fibrin and inflammatory cells mainly heterophils overlying degenerating and necrotic intestinal villi was seen (Plate 63). Sub acute enteritis was observed in many sections characterised by thickened mucosal wall due to infiltration of heterophils, lymphocytes and plasma cells in the lamina propria, sub mucosa and occasionally serosa along with mild to moderate congestion and haemorrhage (Plate 64). Less frequently chronic enteritis was found which was characterised by infiltration of MNCs predominantly lymphocytes, macrophages and few plasma cells in sub mucosa. In addition severe

necrosis of epithelial cells of villi, hyperplasia of goblet cells and proliferation of MNCs and fibrous connective tissue in lamina propria in some cases was seen (Plate 65). Regeneration characterised by hyperplasia of crypt epithelium as evidenced by the presence of numerous mitotic figures was a common change associated with enteritis (Plate 66).

4.8.2 Liver

Liver was affected with one or the other lesion in 71.83% (454/632) of the birds. Grossly, congestion, haemorrhages, necrosis, fatty changes and perihepatitis were seen in 45.15% (205/454), 23.56% (107/454), 13.87% (63/454), 39.42% (179/454) and 68.94% (313/454) cases, respectively as depicted in Table 4.6. Microscopically, lesions consisted of congestion, haemorrhage, edema, vacuolar degeneration, necrosis, fatty changes, acute and chronic hepatitis which were seen in 74.22% (337/454), 29.07% (132/454), 58.81% (267/454), 69.82% (317/454), 60.79% (276/454), 39.42% (179/454), 77.09% (350/454) and 3.74% (17/454) cases, respectively as shown in Table 4.7

Grossly, liver was dark red in colour, enlarged and had rounded edges (Plate 67). In some cases, pale and mottled appearance of liver was also noticed (Plate 68). Haemorrhages appeared either as petechial, scattered throughout the liver lobe surface or were present on particular areas of lobes (Plate 69). Bronze discolouration of liver was seen in cases of Salmonellosis. Multifocal necrotic areas appeared as whitish circumscribed nodular areas scattered randomly over surface of liver. In some cases these areas seemed to be coalescing (Plate 70).

Microscopically, congested liver sections had dilated and engorged central veins and sinusoids (Plate 71). Hepatocytes surrounding the congested central vein and sinusoids showed varying degree of degenerative changes and necrosis. Many liver sections revealed dissociation and individualization of hepatocytes and coagulative necrosis (Plate 72). Necrotic hepatocytes had deep eosinophilic cytoplasm. Nuclear changes varied from pyknosis to karyorrhexis and karyolysis. Necrosis of hepatocytes beneath the capsule was occasionally seen (Plate 73). Haemorrhages were often focal with few RBC’s accumulating in the parenchyma or rarely diffused where RBC’s were

present in large numbers and the latter was mostly seen in sub capsular areas. Edema was mostly seen as accumulation of pink staining fluid in and around central vein, sinusoids or portal tract (Plate 74). In cases of acute hepatitis congestion of central vein, dilatation of sinusoids, focal areas of degeneration and necrosis of hepatocytes with infiltration of inflammatory cells mainly heterophils was seen. (Plate 75, 76). Suppurative hepatitis was characterised by congestion, multifocal areas of vacuolar degeneration, necrosis of hepatocytes and focal infiltration of large number of heterophils in these areas (Plate 77). In case of colibacillosis, fibrinous perihepatitis was seen; characterised by presence of pink strands of fibrin and enterprised between the fibrin were present heterophils in large number (Plate 78, 79). The underlying hepatic capsule was adhering loosely with the overlying fibrin and necrotic debris which formed a pseudo membrane. The hepatocytes adjacent to the capsule mostly exhibited degenerative changes but at some areas the hepatocytes were necrotic. Beneath the capsule, dilatation of blood capillaries was seen (Plate 78). In cases of chronic hepatitis, degeneration, necrosis of hepatocytes along with presence of mononuclear cells mostly lymphocytes, macrophages and few plasma cells was noticed. In addition, infiltration of MNCs chiefly lymphocytes and proliferation of fibrous connective tissue in portal triad areas was also observed. Hepatocholangitis was also encountered concurrently with necrosis and inflammation of liver parenchyma; degeneration, necrosis and hyperplasia of epithelium of bile duct with presence of inflammatory cells consisting of an admixture of heterophils and lymphocytes in portal triad areas (Plate 80).

4.8.3 Gizzard and Proventriculus

Proventriculus was affected in 21.86% (135/632) cases. Grossly, congestion, haemorrhages, edema and necrosis was seen in 36.29% (49/135), 51.11% (69/135), 85.92% (116/135) and 12.59% (17/135) cases, respectively as given in Table 4.8. Microscopically, lesions consisted of congestion, haemorrhages, edema, degeneration, necrosis and proventriculitis visualized in 56.29% (76/135), 42.22% (57/135), 97.77% (132/135), 77.77% (105/135), 77.03% (104/135) and 95.55% (129/135) cases, respectively as presented in Table 4.9.

Congestion in proventriculus appeared as dark red discolouration of serosal or mucosal surface (Plate 81) while edematous proventriculus appeared swollen with thick walls. In many cases proventricular lumen had presence of thick mucoid exudate adhering to underlying congested mucosa (Plate 82). Swollen and edematous proventricular glands were a common finding (Plate 83). Accumulation of thick tenacious whitish slimy exudate in proventriculus adherent to walls was seen in some birds (Plate 84).

Microscopically, congestion and haemorrhages of lamina propria was a common lesion. Degeneration and necrosis of epithelium of mucosal folds and infiltration of heterophils in lamina propria and sub mucosa was also very commonly seen (Plate 85). Likewise, degeneration and necrosis of sub mucosal proventricular glands was also frequently observed (Plate 86). Accumulation of edematous fluid in sub mucosa and interglandular area along with proliferation of small arterioles and capillaries and infiltration of inflammatory cells predominantly heterophils and few lymphocytes was observed in some cases (Plate 87). Proliferation and hyalinisation of connective tissue in the interglandular area was seen in rare cases (Plate 88). Fibrinous proventriculitis with involvement of tunica serosa characterized by presence of congested blood vessels, necrotic debris, fibrin and varying number of inflammatory cells mainly heterophils were observed in some cases (Plate 89). In sub acute infections, thickened sub-mucosa, edema, necrosis of mucosal folds and infiltration of MNCs mainly lymphocytes and few heterophils were seen in lamina propria and sub mucosa. In chronic cases, degeneration and necrosis of glandular epithelium, necrosed, blunted, fused mucosal folds along with inflammatory cells mainly MNC's and fibrous connective tissue proliferation was seen in lamina propria and sub mucosa (Plate 90).

Gizzard was affected in 8.86% (56/632) cases. Grossly, haemorrhages and necrosis were seen in 28.57% (16/56) and 57.14% (4/56) cases, respectively as shown in Table 4.8. Microscopically, lesions consisted of congestion, haemorrhages, edema, degeneration, necrosis and ventriculitis as observed in 41.07% (23/56), 33.92% (19/56), 60.71% (34/56), 62.5% (35/56), 76.78% (43/56) and 64.28% (36/56) cases as presented in Table 4.9. Gross lesions in gizzard mostly consisted of haemorrhages and ulcerations.

Erosions developed as a result of necrosis of overlying koilin layer revealing the underlying mucosa and sub mucosa (Plate 82, 91). Microscopically, these lesions were characterised by necrosis of lining of epithelium with accumulation of inflammatory cells mainly lymphocytes and few heterophils in lamina propria. Fragmentation, degeneration and necrosis of smooth muscle fibers along with infiltration of inflammatory cells mainly heterophils in these areas was observed in two cases (Plate 92). Additionally, focal areas of accumulation of MNCs mostly macrophages, lymphocytes and few heterophils among degenerating and necrotic muscle bundles of gizzard muscular layer was sometimes observed (Plate 93). In serosa, proliferation of several congested blood vessels, edema, presence of fibrin and infiltration of heterophils and this fibrinous exudate encroaching the muscle layer was seen in many cases (Plate 94).

4.8.4 Pancreas

Pancreas was affected in 2.84% (18/632) cases. Grossly, congestion, haemorrhages and necrosis was seen in 50% (9/18), 27.77% (5/18) and 22.22% (4/18) cases, respectively as depicted in Table 4.8. Microscopically, lesions consisted of congestion, haemorrhages, edema, degeneration, necrosis and pancreatitis which were seen in 33.33% (6/18), 16.66% (3/18), 38.88% (7/18), 83.33% (15/18), 61.11% (11/18) and 44.44% (8/18) cases, respectively as given in Table 4.9

Gross lesions affecting pancreas mostly consisted of congestion where pancreas were dark red in colour (Plate 95). Haemorrhagic and necrotic lesions were seen in few cases and appeared as dark depressed sharply circumscribed erosions (Plate 96). Microscopically, lesions consisted of congestion, haemorrhage (Plate 97), degeneration and necrosis of pancreatic acinar cells and pancreatitis (Plate 98). Focal pancreatitis was occasionally seen which was characterised by necrosis of acini and presence of heterophilic infiltration (Plate 99). Severe pancreatitis was seen in two birds which was characterised by presence of hyperplasia of pancreatic ducts, congested blood vessels in interlobular areas along with degeneration, necrosis of acinar cells and infiltration of large number of heterophilis (Plate 100). Additionally, the inflammatory lesion involved the adjacent adipose tissue causing severe steatitis characterised by proliferation of blood vessels, edema and infiltration of heterophils (Plate 100).

4.8.5 Oesophagus

Oesophagus was found to be affected in 3% (19/632) of the necropsied birds. No gross lesions could be appreciated. Microscopic lesions in esophagus included congestion 63.15% (12/19), haemorrhage 21.05% (4/19), edema 42.10% (8/19), degeneration 78.94% (15/19), necrosis 84.21% (16/19) and oesophagitis 26.31% (5/19) cases, respectively as depicted in Table 4.9

Microscopically, changes appreciated included congestion, edema and inflammation in sub mucosa and serosa (Plate 101). Suppurative oesophagitis characterised by infiltration of inflammatory cells mostly heterophils in sub mucosa around the oesophageal glands was recorded (Plate 102, 103). Sometimes expanding micro-abscesses were found squeezing the oesophageal glands (Plate 104).

Table 4.3 Occurrence of pathological lesions of various organs of GIT of broilers

Sr. No.	Organ	Total affected	Percentage
1.	Intestine	507	80.22
2.	Liver	454	71.83
3.	Proventriculus	135	21.36
4.	Gizzard	56	8.86
5.	Pancreas	53	8.38
6.	Oesophagus	19	3.00

Table 4.4: Occurrence of various gross lesions in intestine of broilers.

Organs	Congestion		Haemorrhages		Necrosis		Enteritis	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Intestine	205	40.43	107	21.10	63	12.42	347	68.44

Table 4.5: Occurrence of various microscopic lesions in intestine of broilers

Organs	Congestion		Haemorrhages		Edema		Degeneration		Necrosis		Inflammation					
	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	Acute		Sub acute		Chronic	
											No.	%	No.	%	No.	%
Intestine	389	76.72	293	57.79	267	52.66	458	90.33	389	76.72	350	69.03	12	2.36	17	3.35

Table 4.6: Occurrence of various gross lesions in liver of broilers

Organ	Congestion		Haemorrhages		Enlargement		Necrosis		Fatty changes		Perihepatitis	
	No.	(%)	No.	(%)	No		No.	(%)	No.	(%)	No.	(%)
Liver	205	45.15	107	23.56	267	58.81	63	13.87	179	39.42	313	68.94

Table 4.7: Occurrence of various microscopic lesions in liver of broilers

Organ	Congestion		Haemorrhages		Edema		Vacuolar degeneration		Necrosis		Fatty changes		Inflammation			
	No.	(%)	No.	(%)	No.	%	No.	(%)	No.	%	No.	(%)	Acute		Chronic	
													No.	%	No.	%
Liver	337	74.22	132	29.07	267	58.81	317	69.82	276	60.79	179	39.42	350	77.09	17	3.74

Table 4.8: Occurrence of various gross lesions in various organs of GIT of broilers

S. No.	Organs	Congestion		Haemorrhages		Edema		Necrosis	
		No.	(%)	No.	(%)	No.	(%)	No.	(%)
1.	Oesophagus	-	-	-	-	-	-	-	-
2.	Proventriculus	49	36.29	69	51.11	116	85.92	17	12.59
3.	Gizzard	-	-	16	28.57	-	-	32	57.14
4.	Pancreas	09	50.00	05	27.77	-	-	04	22.22

Table 4.9: Occurrence of various microscopic lesions in various organs of GIT of broilers

S. No	Organs	Congestion		Haemorrhages		Edema		Degeneration		Necrosis		Inflammation	
		No.	(%)	No.	(%)	No.	(%)	No	(%)	No.	(%)	No.	(%)
1.	Oesophagus	12	63.15	04	21.05	08	42.10	15	78.94	16	84.21	05	26.31
2.	Proventriculus	76	56.29	57	42.22	132	97.77	105	77.77	104	77.03	129	95.55
3.	Gizzard	23	41.07	19	33.92	34	60.71	35	62.5	43	76.78	36	64.28
4.	Pancreas	06	33.33	03	16.66	07	38.88	15	83.33	11	61.11	08	44.44

Plate 1: Liver showed a thin layer and grayish white material on its surface.

Plate 2: Proventriculus from a case of colibacillosis showing presence of mucoid exudate adhering to the underlying mucosa and focal haemorrhagic lesions (arrow) in the proventricular wall.

Plate 3: Duodenum from a case of colibacillosis showing congestion.

Plate 4: Perihepatitis in case of colibacillosis showing fibrin strands admixed of heterophils forming separate layer over the underlying liver parenchyma. H&E X40

Plate 5: Congestion, degeneration, necrosis and infiltration of heterophils in a liver section from a case of colibacillosis. H&E X400

Plate 6: Section of proventriculus from a case of colibacillosis showing necrosis of epithelium of mucosal folds, edema, congestion and severe infiltration of heterophils in lamina propria and underlying sub mucosa. H&E X40

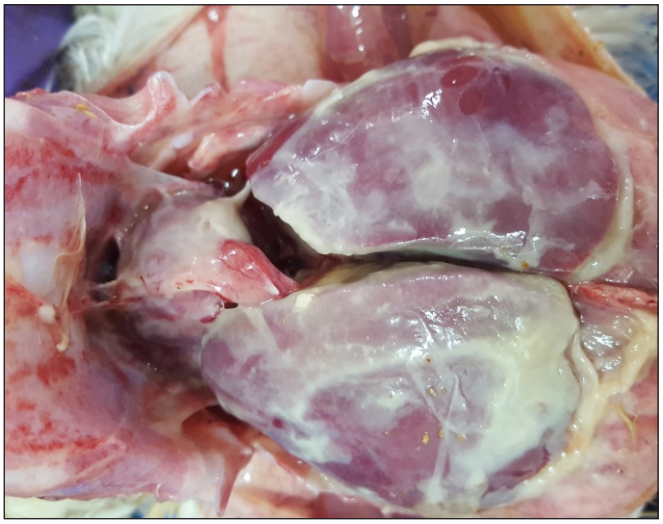


Plate 1

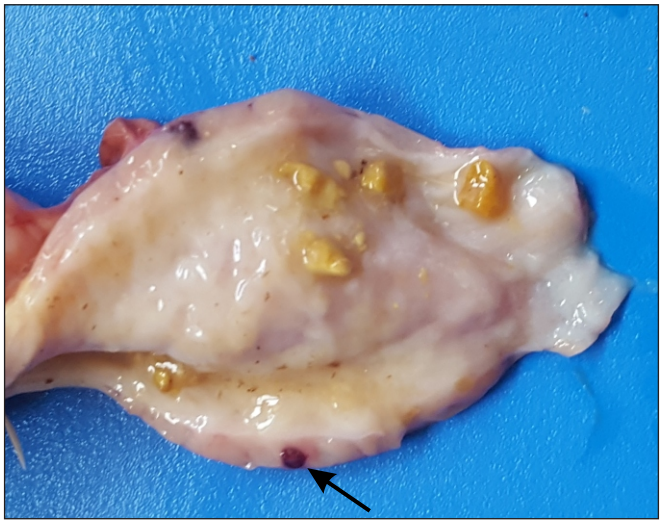
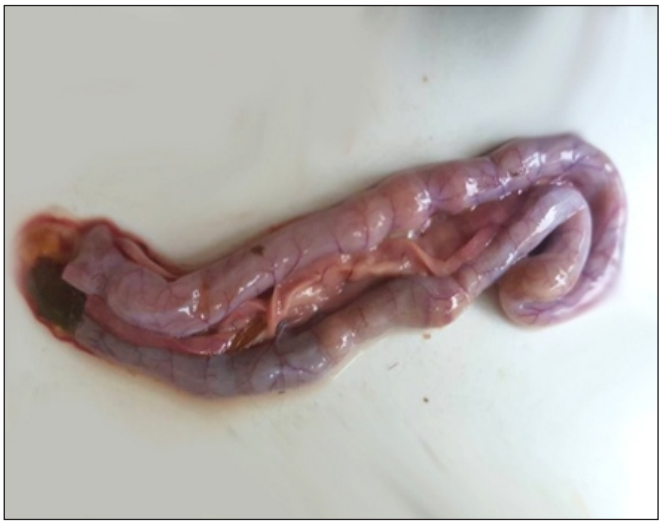


Plate 2



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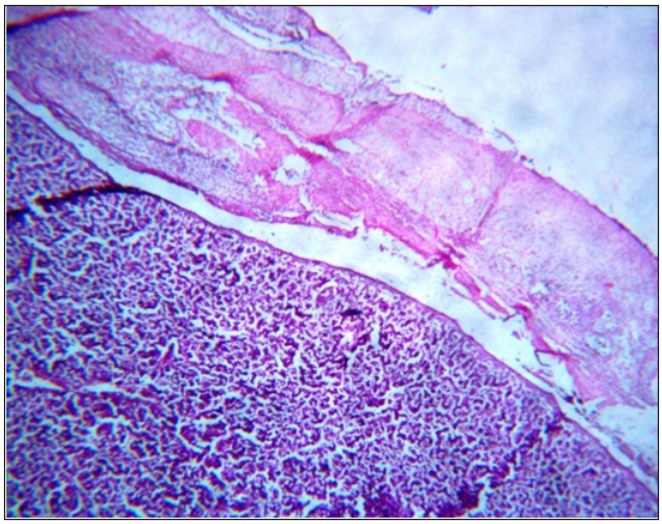
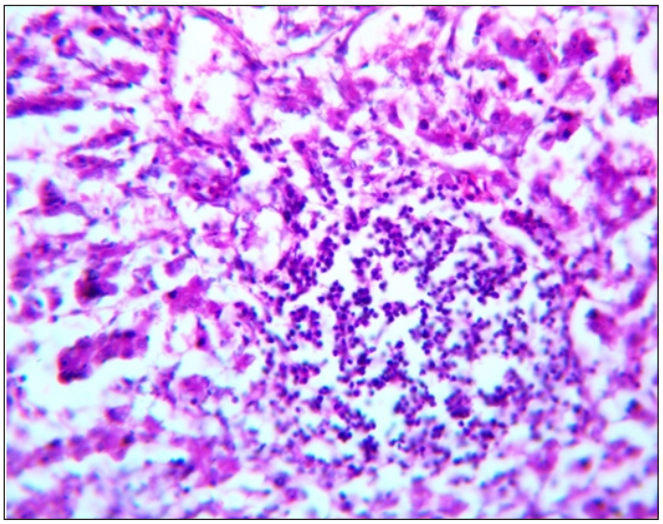


Plate 4



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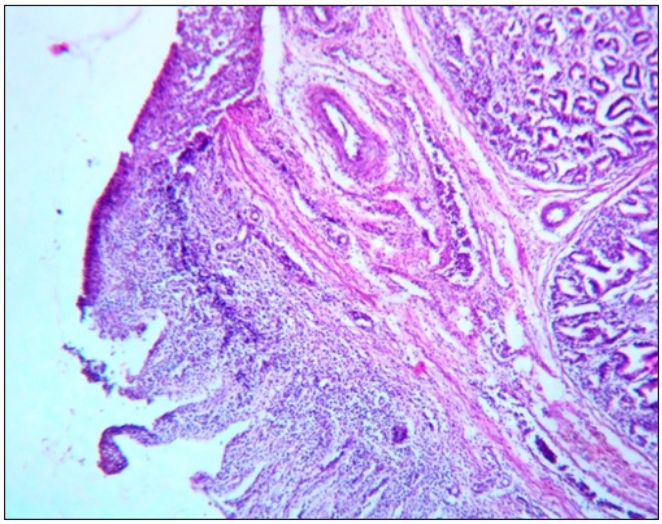


Plate 6

Plate 7: Section of proventriculus from a case of colibacillosis showing congestion in tunica muscularis and serosa and fibrinous exudate in serosa. H&E X400

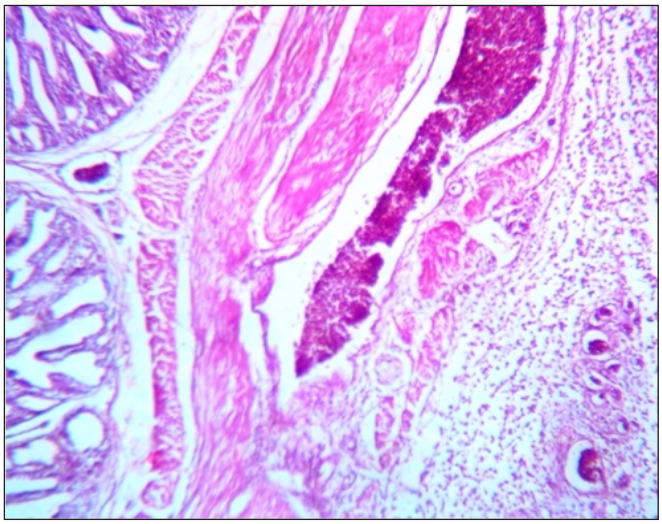
Plate 8: Section of intestine from a case of colibacillosis showing congestion, necrosis of villi and infiltration of heterophils in lamina propria or sub mucosa and deposition of fibrinous exudate in serosal layer. H&E X40

Plate 9: *E.coli* isolates forming pink colored colonies on MacConkey lactose agar.

Plate 10: *E.coli* showing characteristics greenish metallic sheen on EMB agar.

Plate 11: Gram's staining revealing pink color, cocco bacilli (Modified Gram's stain, X1000).

Plate 12: IMViC pattern characteristic of *E.coli.*



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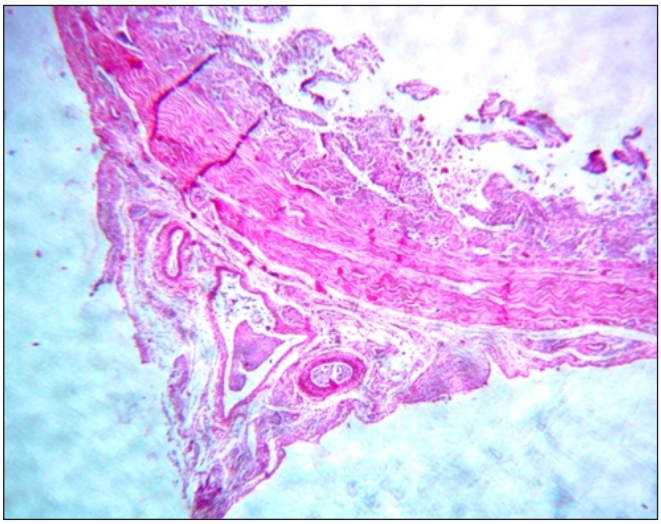

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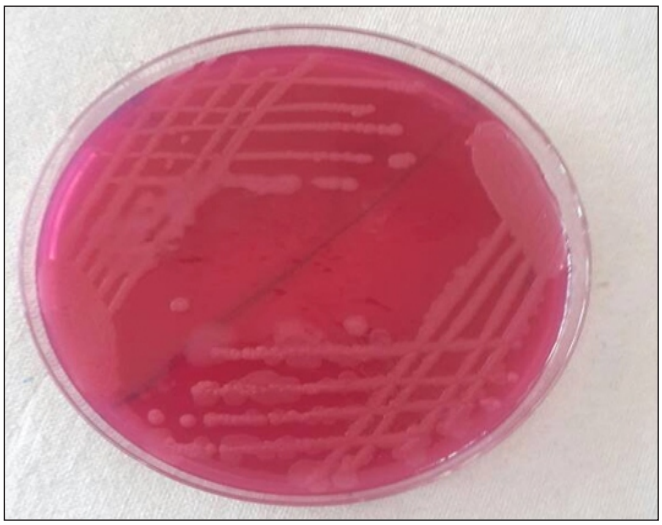

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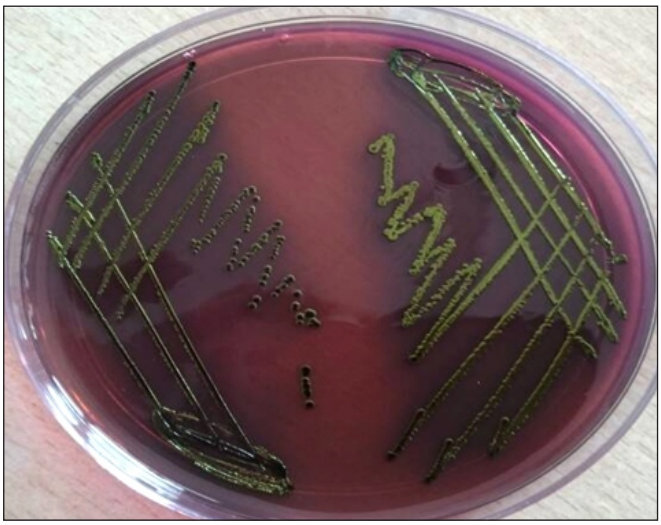

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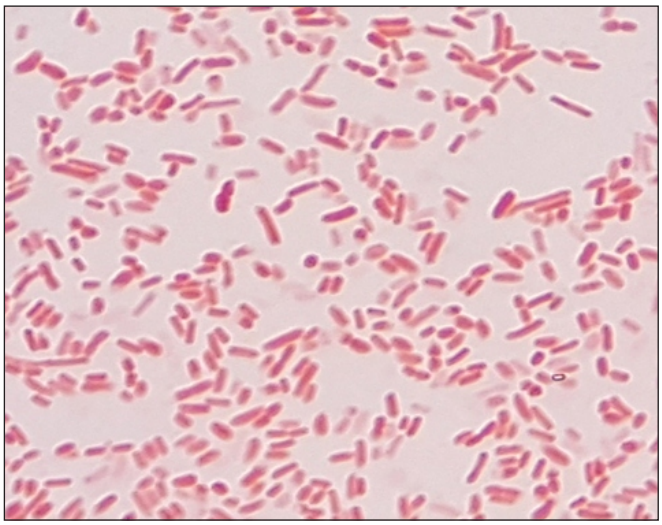

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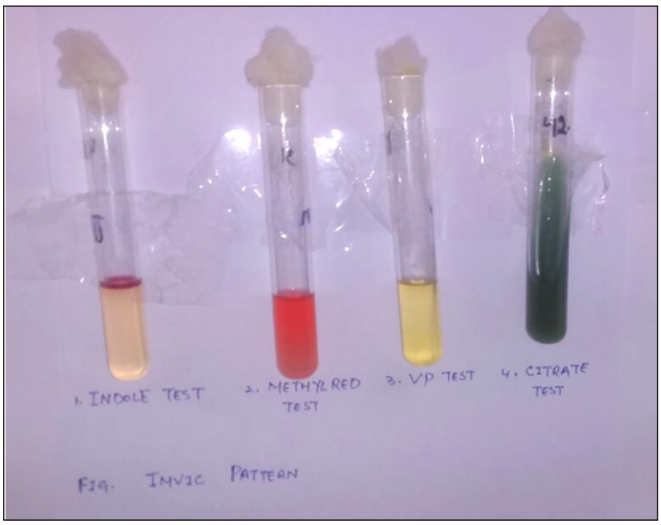


Plate 12

Plate 13: Unabsorbed and haemorrhagic yolk sac in cases of omphalitis.

Plate 14: Liver section from a case of omphalitis showing degeneration, atrophy and necrosis of hepatocytes with dilation of sinusoids. H&E X400

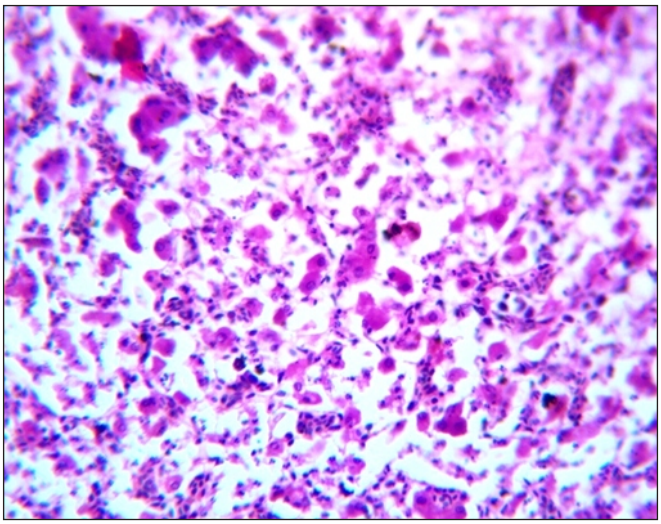
Plate 15: Enteritis in a case of omphalitis revealing desquamation of epithelium and presence of inflammatory cells in lumen of lamina propria. H&E X40

Plate 16: Greenish-bronze discolouration of liver from a case of salmonellosis.

Plate 17: Swelling, mottling and presence of multiple necrotic foci on the surface of liver in a case of salmonellosis.

Plate 18: Haemorrhages and congestion localized on serosa of proventriculus in a case affected with salmonellosis.

Plate 13



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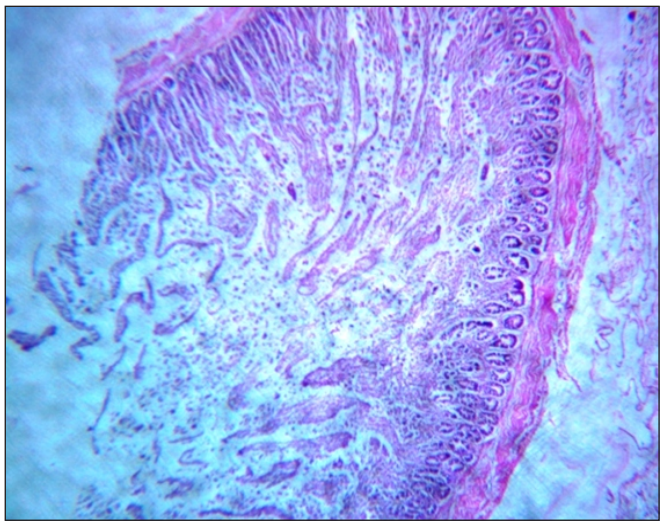

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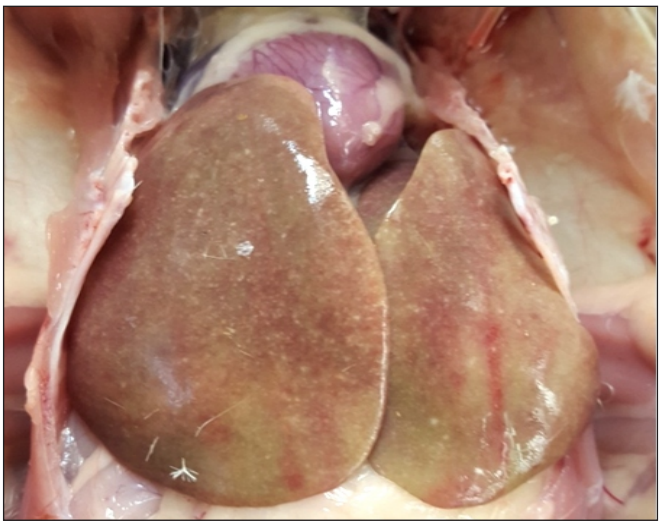

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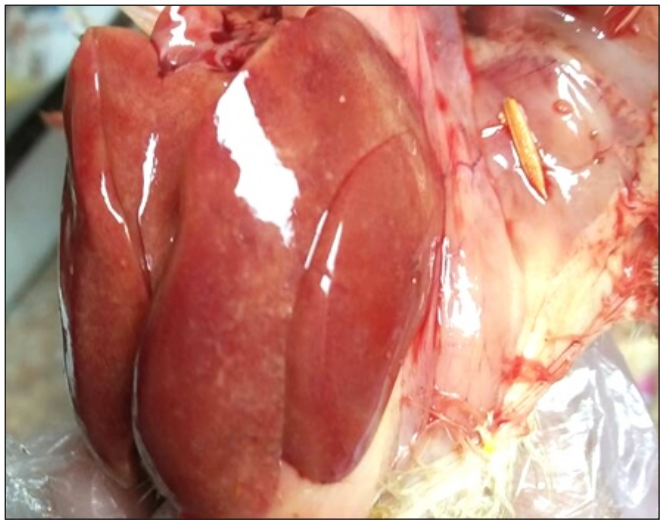

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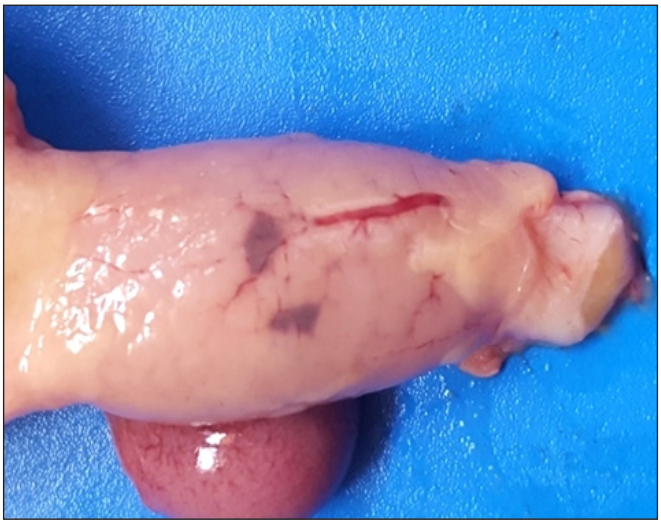


Plate 18

Plate 19: Serosal congestion and haemorrhages in intestinal tract in a bird affected with Salmonellosis.

Plate 20: Liver section revealing severe congested sinusoids and central veins in a case of salmonellosis. H&E X40

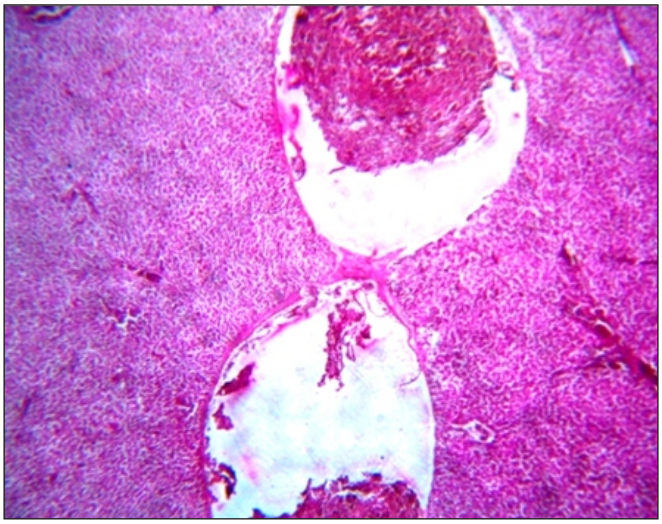
Plate 21: Liver section from a *Salmonella* affected bird showing necrotic hepatocytes floating in pool of heterophils. H&E X100

Plate 22: Proventriculus showing thickening of mucosal folds, edema, presence of necrotic material, infiltration of heterophils and congestion in lamina propria. H&E X100

Plate 23: Intestinal section from a case of Salmonellosis revealing necrotic villi with severe congestion in lamina propria. H&E X100

Plate 24: Gram's staining rods of *Salmonella* sp. showing negative, pale yellow color, cocco bacilli organisms (Modified Gram's stain, X 1000)

Plate 19



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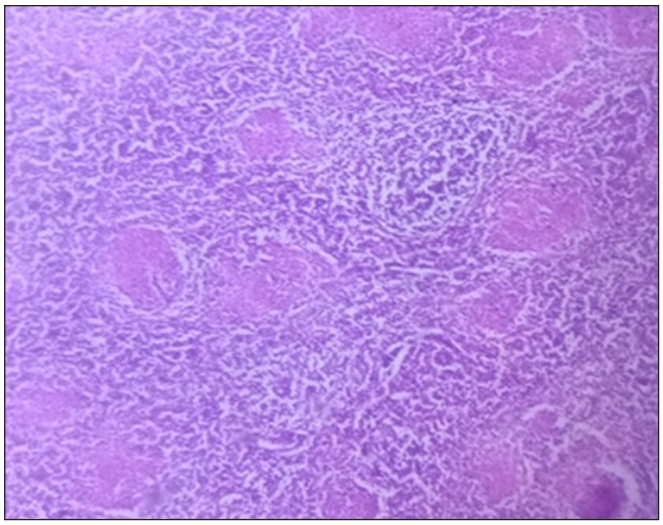

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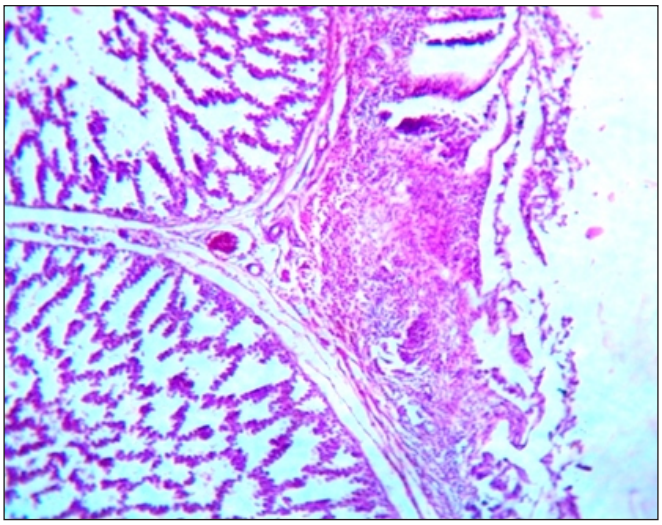

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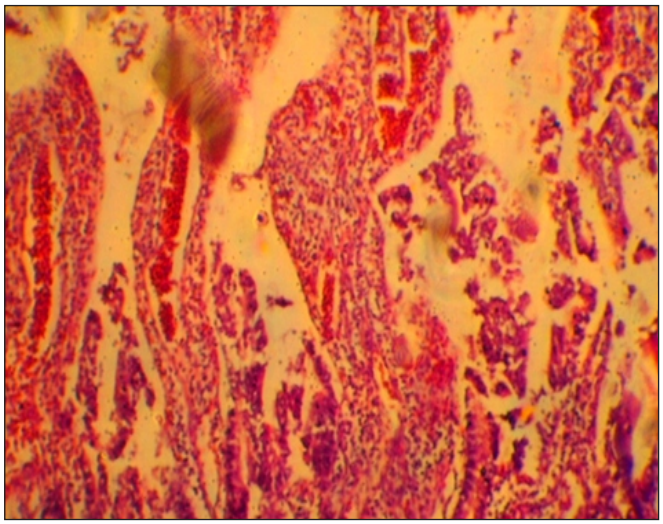

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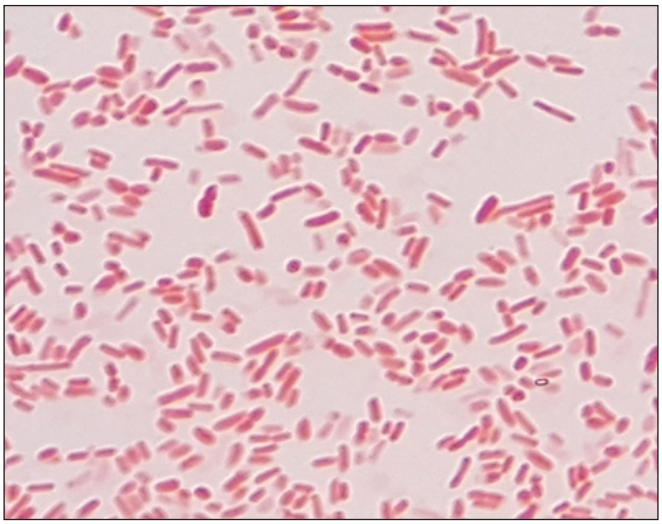

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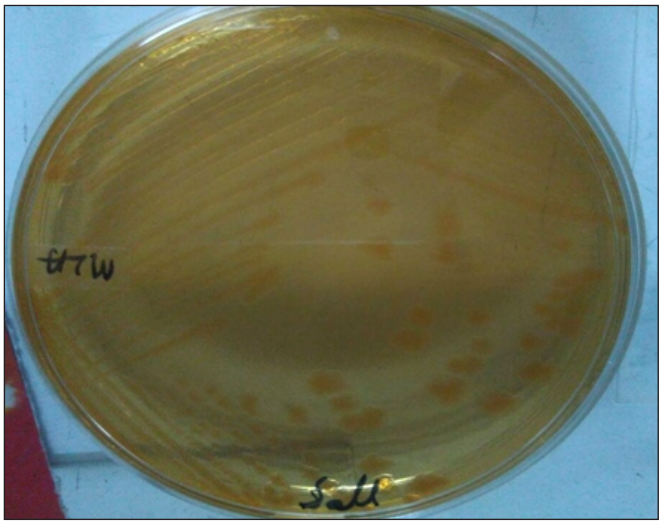

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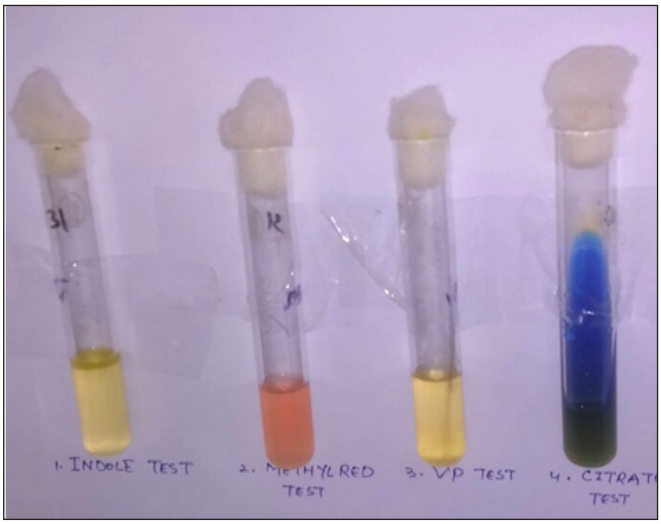

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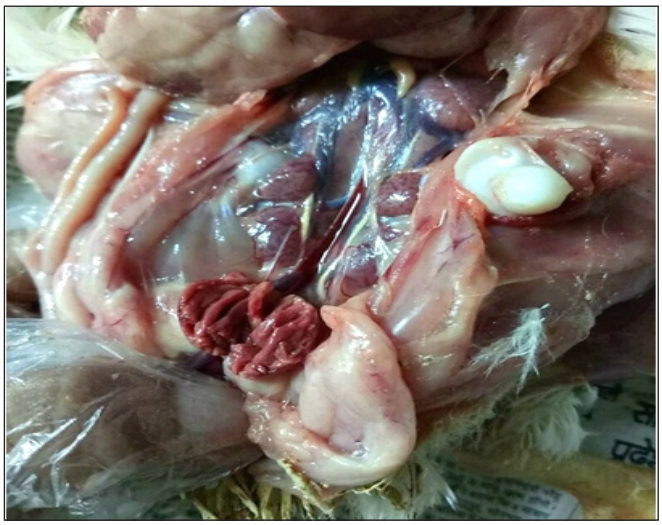

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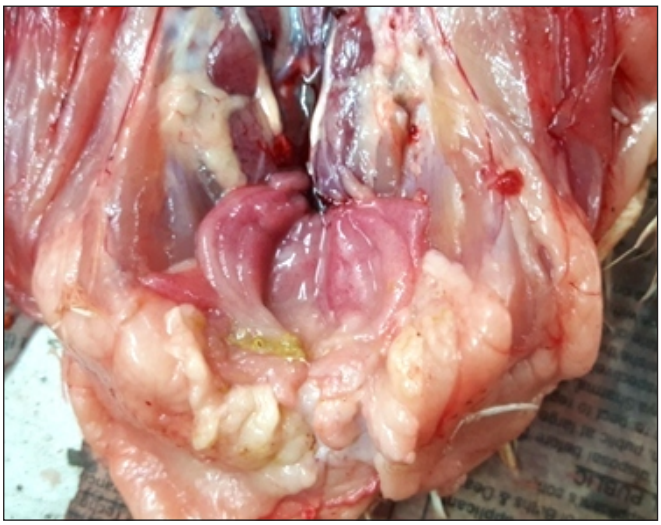

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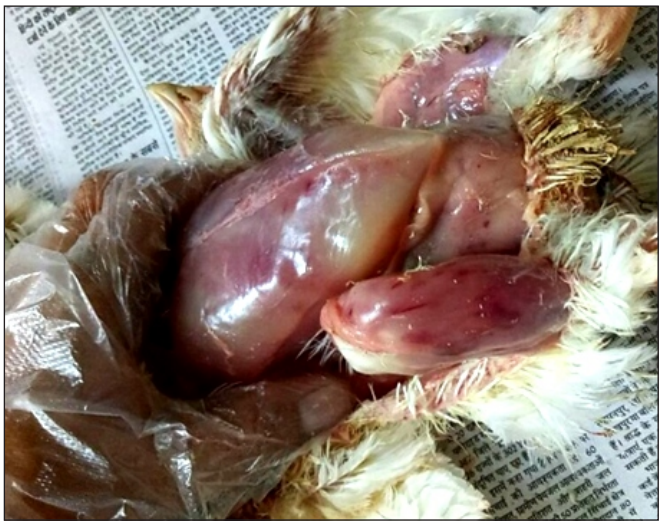

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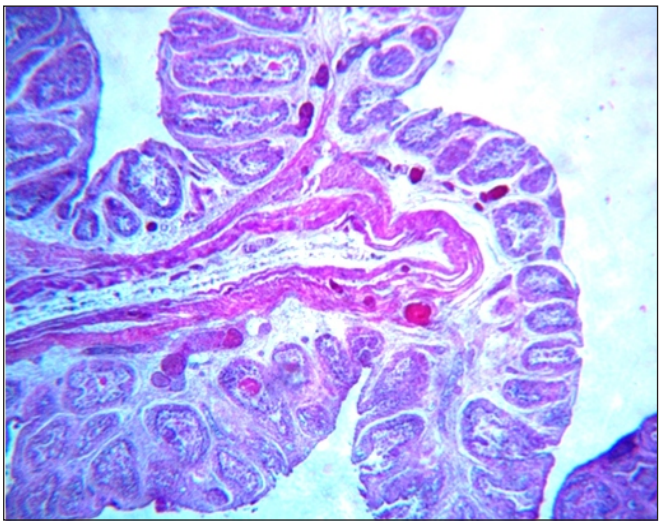


Plate 30

Plate 31: Necrosis of bursal follicles, edema and severe infiltration of heterophils in interfollicular areas. H&E X400

Plate 32: Presence of serofibrinous exudate in the interfollicular area and on the external bursal surface. H&E X100

Plate 33: Kidney section showing congestion, haemorrhages, desquamation of degenerating and necrotic tubular epithelial cells in the lumen in a case of IBD. H&E X100

Plate 34: Intestinal section from a case of IBD showing congestion, haemorrhages, necrosis of villi and infiltration of inflammatory cells in lamina propria. H&E X100

Plate 35: Hemorrhages in proventriculus from a case suspected of NCD or Avian influenza.

Plate 36: Mottled appearance of liver from a case suspected of NCD or Avian influenza.

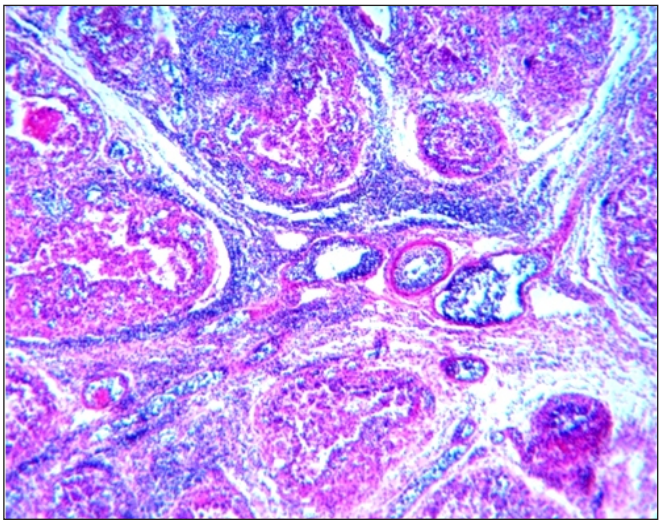
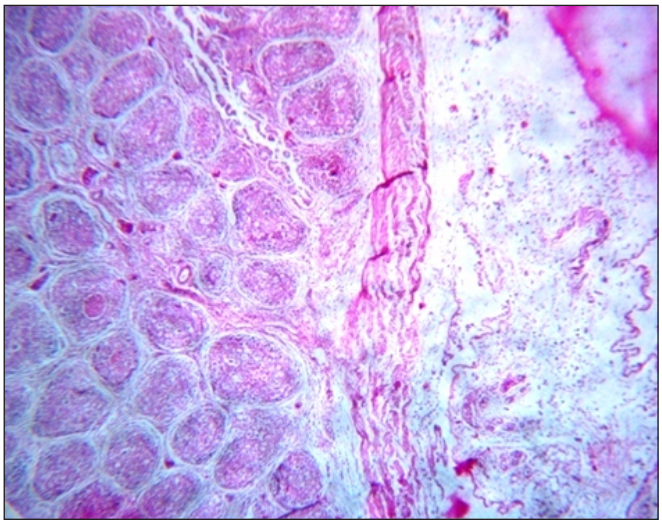


Plate 31



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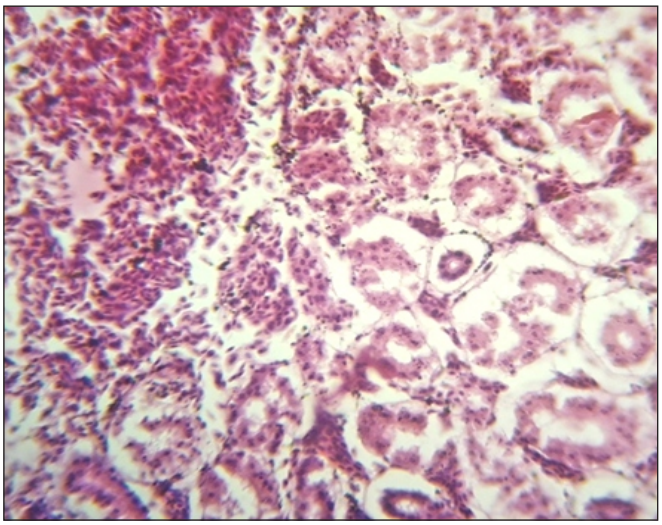


Plate 33

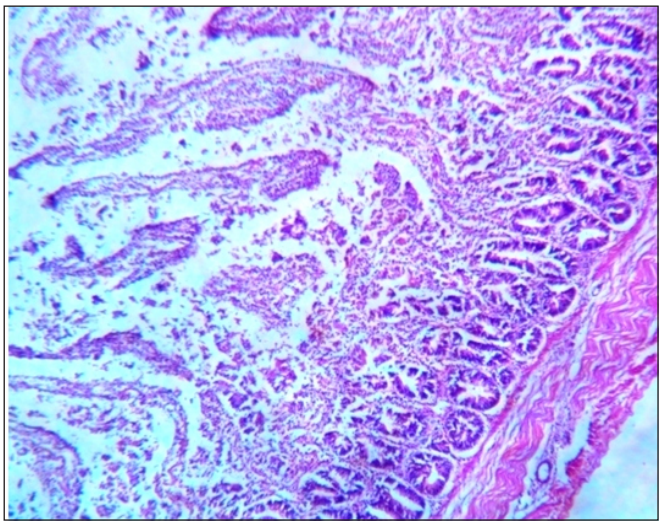


Plate 34

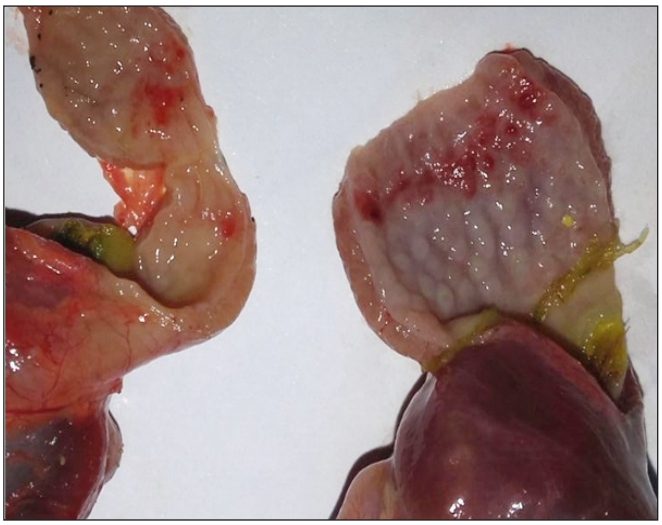


Plate 35

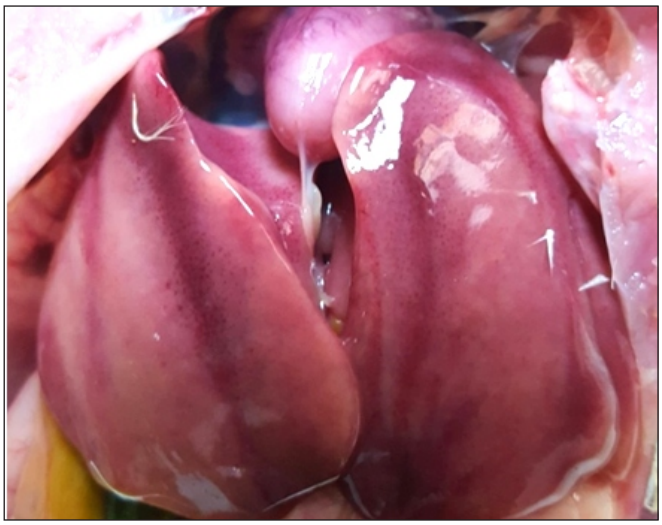


Plate 36

Plate 37: Severe congestion and presence of MNCs mainly lymphocytes in a section of intestine from cases suspected to be NCD or Avian influenza. H&E X100

Plate 38: Section of proventriculus revealing congestion, haemorrhage, edema in lamina propria and sub mucosa along with necrosis of mucosal papillae. H&E X100

Plate 39: Degeneration of hepatocytes, severe congestion along with infiltration of heterophils in portal triad areas from a suspected cases of NCD or Avian influenza. H&E X40

Plate 40: Severe petechial haemorrhages on mucosal surface of caeca in a case of coccidiosis.

Plate 41: Caeca distended with blood clot.

Plate 42: Intestinal section from a case of coccidiosis showing severe necrosis and exfoliation of intestinal layers along with infiltration of inflammatory cells mainly heterophils in tunica mucosa and sub mucosa. H&E X100

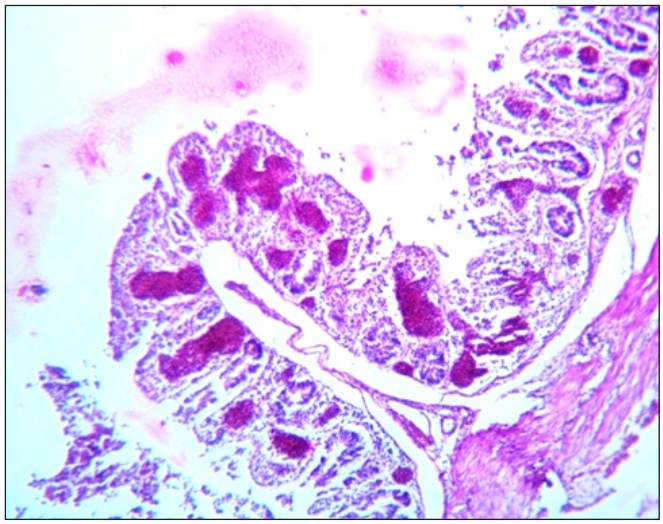
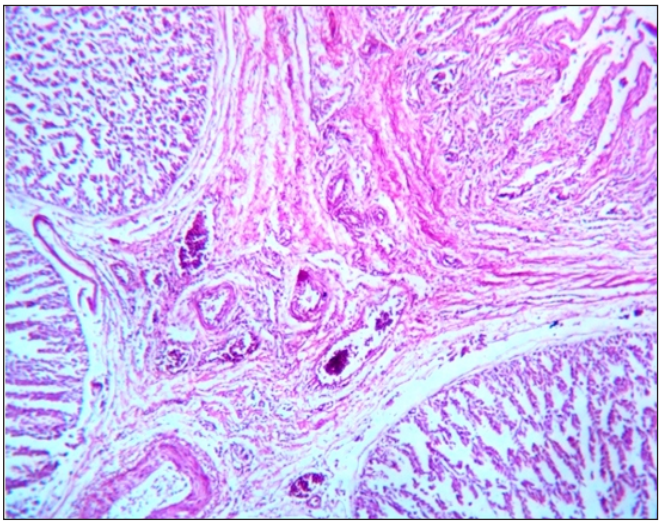


Plate 37



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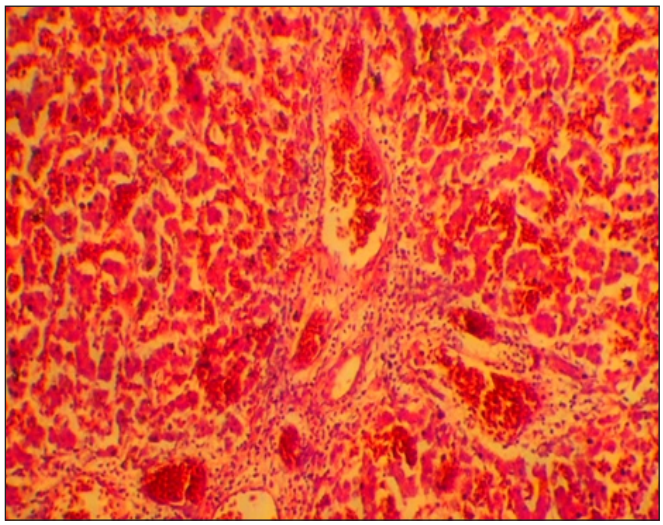


Plate 39

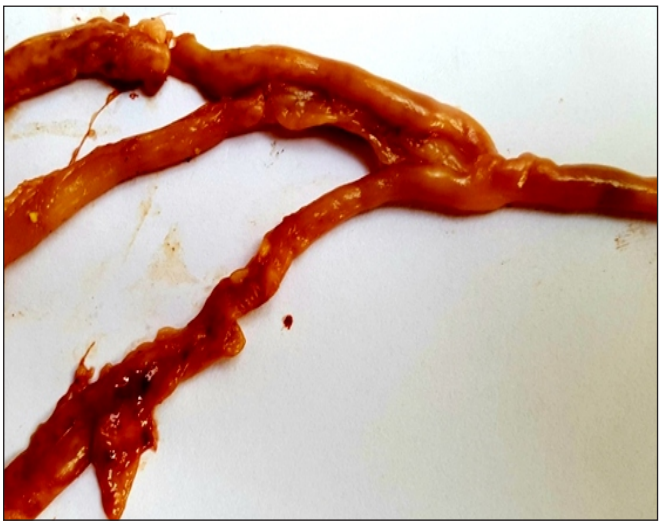


Plate 40

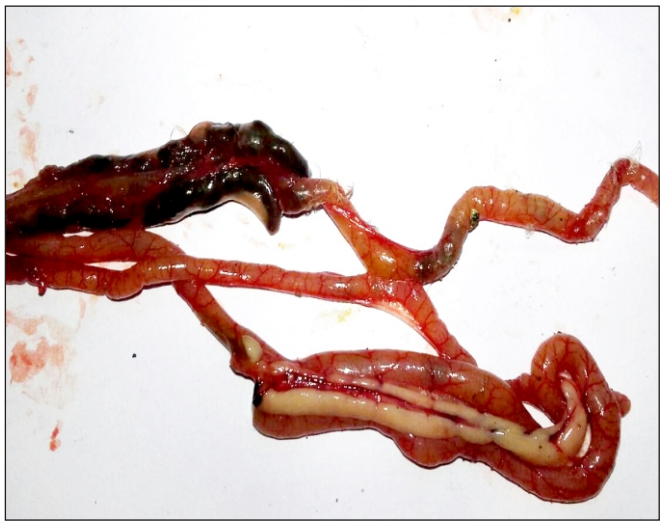


Plate 41

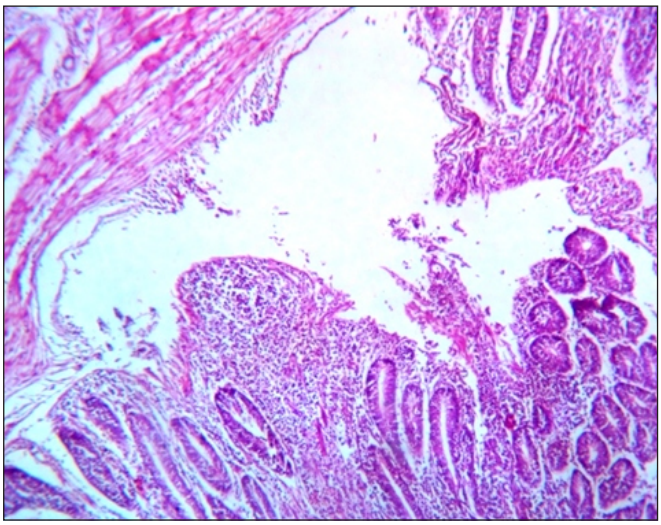


Plate 42

Plate 43: Congestion, haemorrhage, edema, infiltration of inflammatory cells and presence of coccidian life-cycle stages in degenerating and necrotic intestinal epithelium. H&E X100

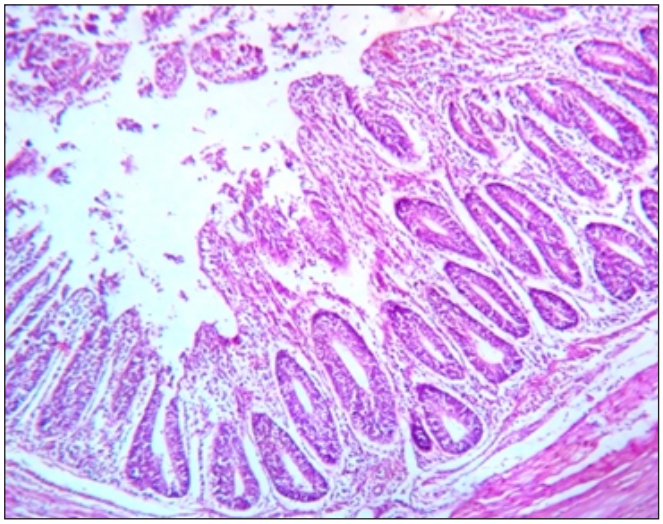
Plate 44: Presence of developing coccidian parasites (arrow) in degeneration and necrotic epithelium of crypts and sub mucosal glands. H&E X400

Plate 45: Presence of developing coccidian stages in epithelium of villi. H&E X400

Plate 46: Wet mount smear prepared from intestinal scrapings showing oocysts of *Eimeria spp.* X400

Plate 47: Accumulation of straw colored fluid in peritoneal cavity in a case affected with ascites syndrome.

Plate 48: Severe vacuolar degeneration and necrosis of hepatocytes in a case of ascites. H&E X100



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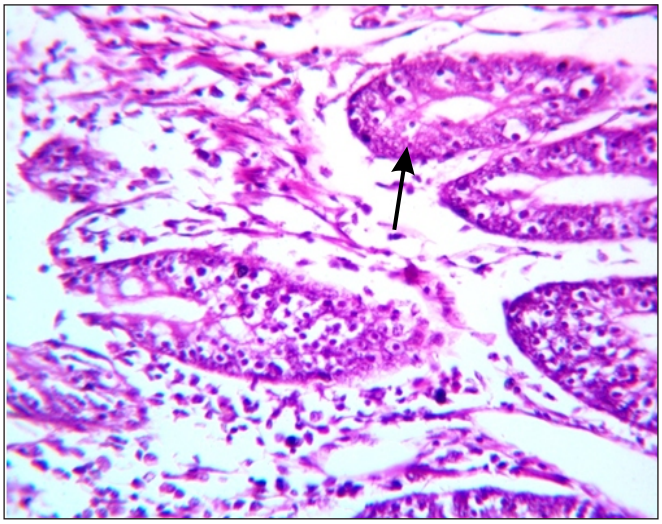

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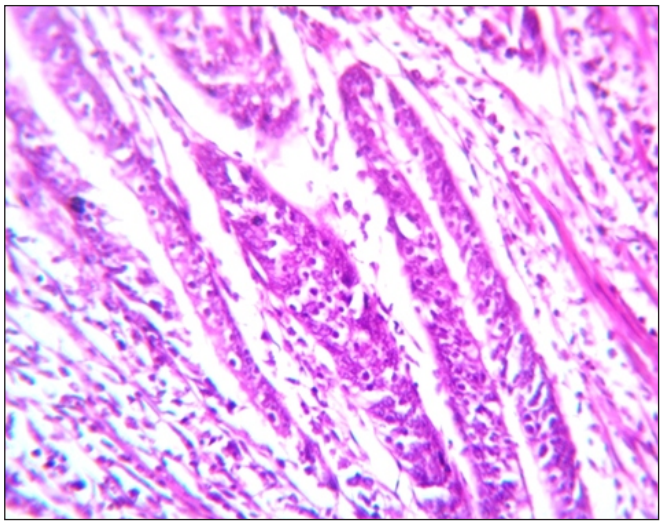

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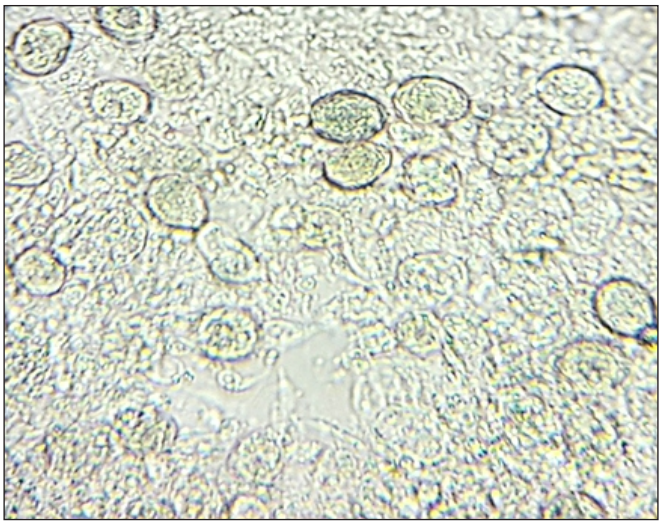

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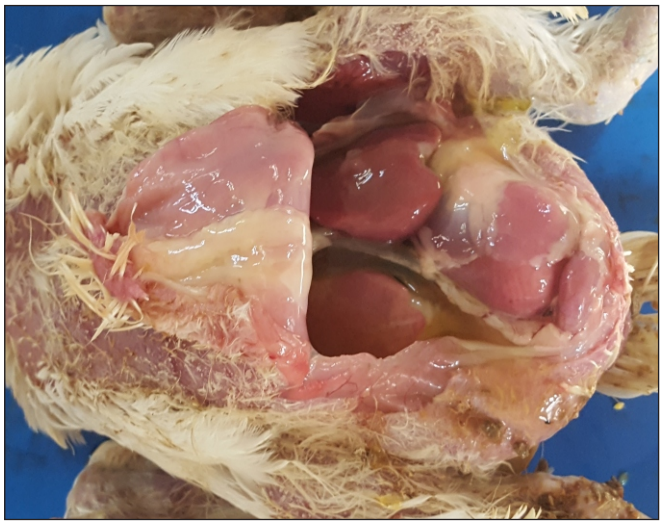

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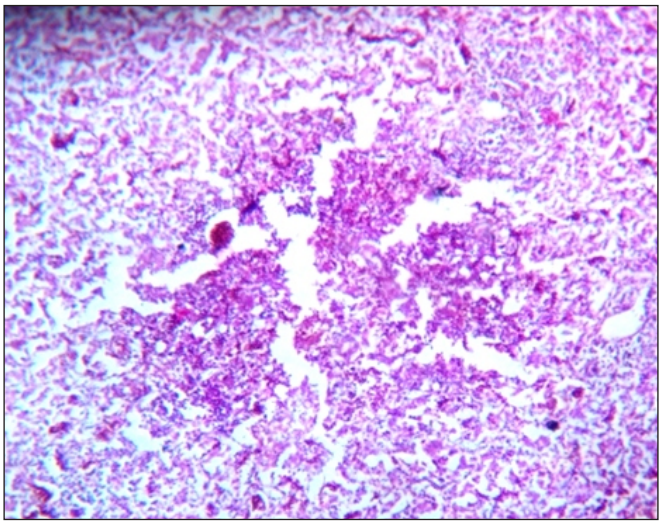


Plate 48

Plate 49: Pale and swollen kidneys in a case suffering from nephro-hepatotoxicity.

Plate 50: Edema and petechial haemorrhages on the surface of kidneys.

Plate 51: Edematous and congested kidneys.

Plate 52: Mottled liver from a case of nephro-hepatotoxicity.

Plate 53: Enlarged, pale and mottled liver from a case of nephro-hepatotoxicity.

Plate 54: Kidney section revealing congestion, haemorrhage, sloughing of tubular epithelial cells into the lumen in case of nephro-hepatotoxicity. H&E X100

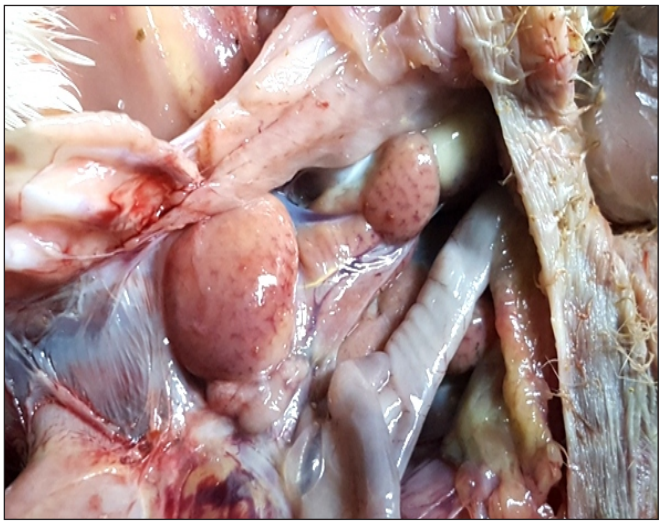
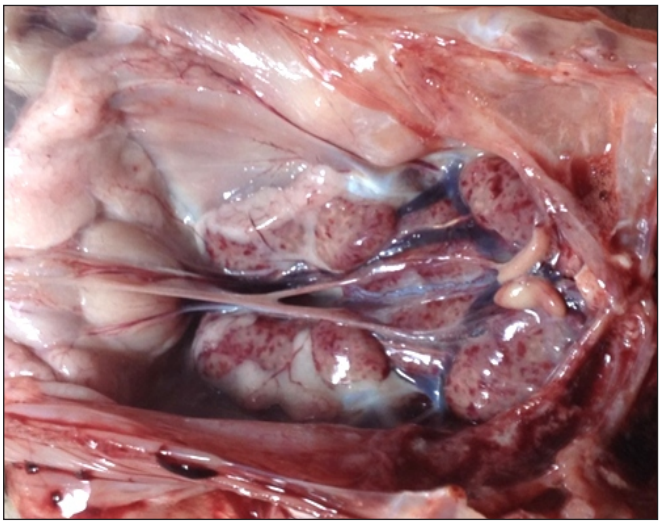


Plate 49



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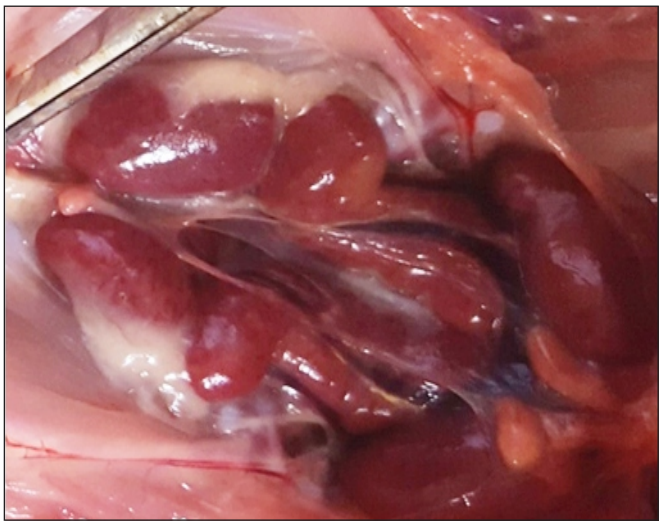

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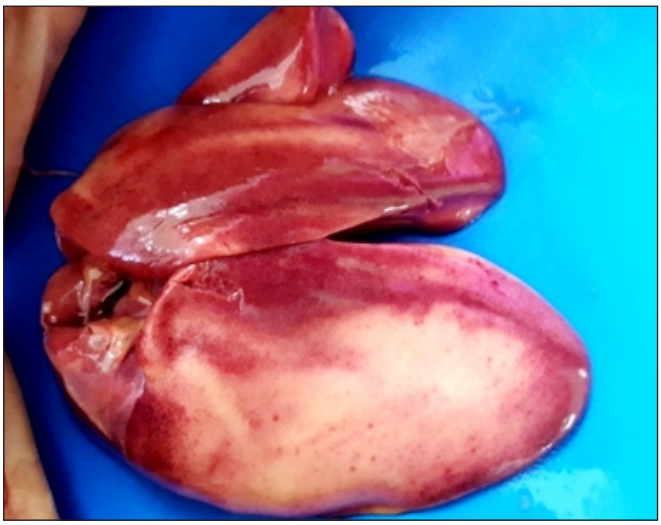

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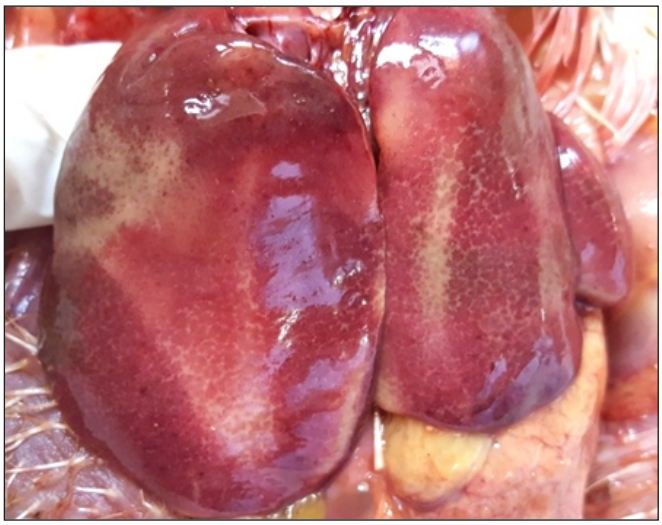


Plate 53

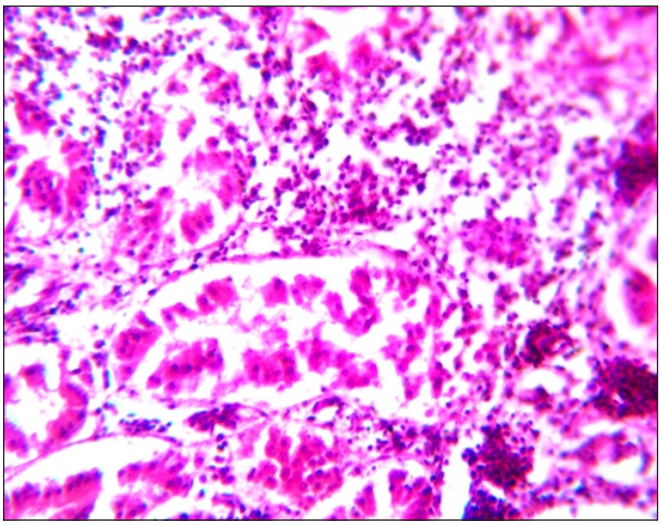


Plate 54

Plate 55: Nephrotoxicity. Kidney: Degeneration, desquamation, necrosis and presence of heterophils in intertubular areas. H&E X40

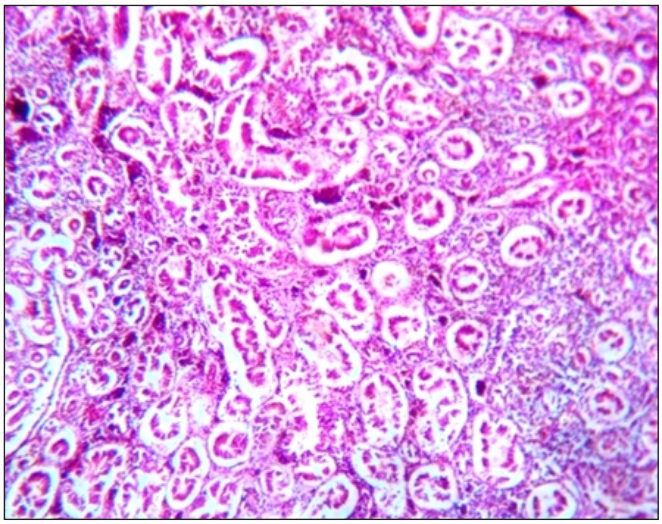
Plate 56: Liver showing degeneration, necrosis of hepatocytes and infiltration of inflammatory cells mainly heterophils in portal triad areas in case of nephro-hepatotoxicity. H&E X100

Plate 57: Fatty liver syndrome: Enlargedt, friable and pale liver.

Plate 58: Fatty liver syndrome: Fatty degeneration and characteristic signet ring appearance of hepatocytes (arrow). H&E X100

Plate 59: Liver showing rupture and presence of clotted blood in the abdominal cavity.

Plate 60: Presence of congestion, petechial haemorrhages on the yellowish mucosa with mucoid exudate in lumen of intestine.



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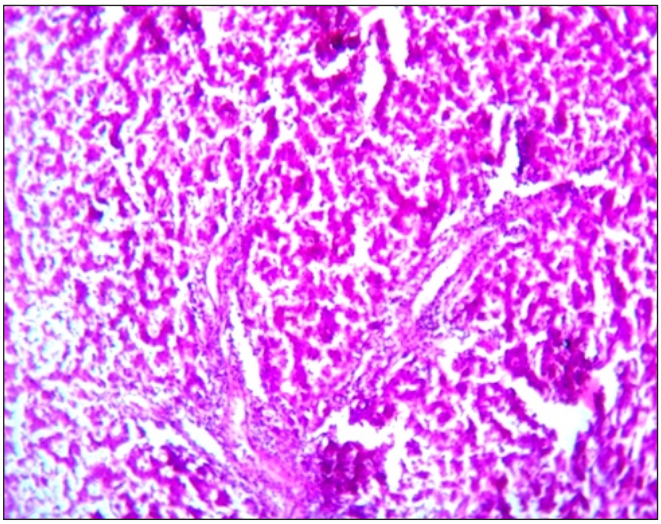

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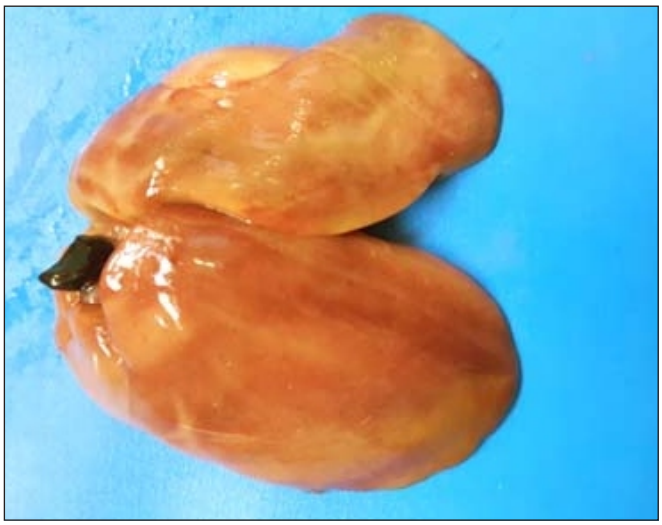

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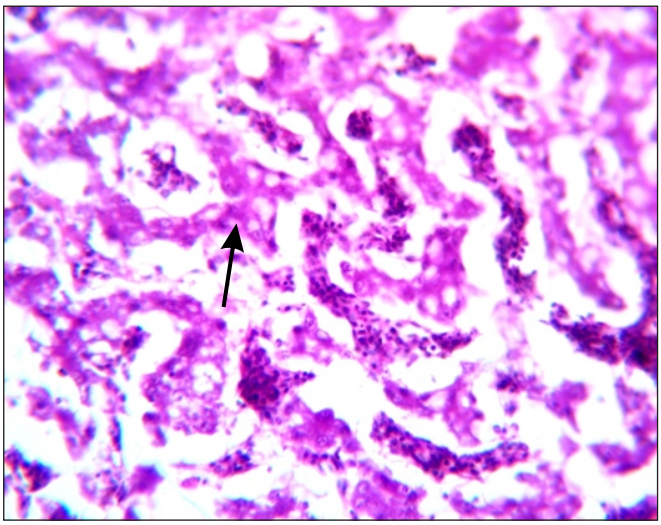

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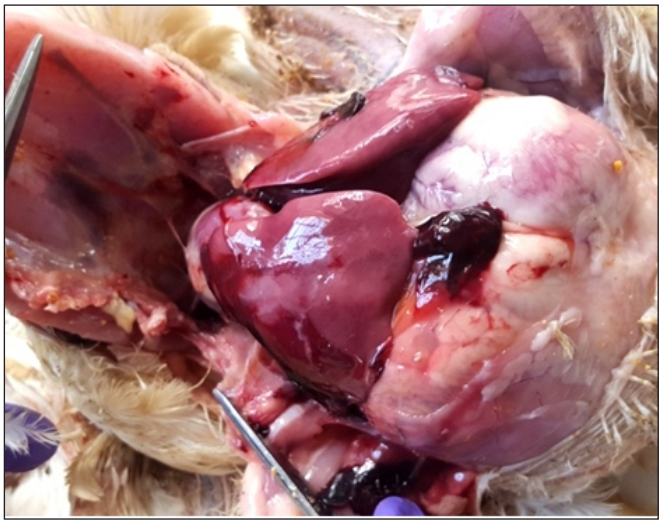

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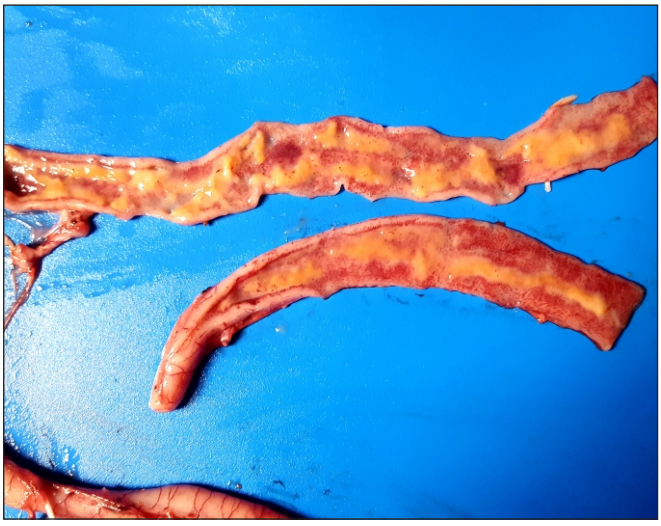


Plate 60

Plate 61: Congestion and haemorrhages on serosa of intestine.

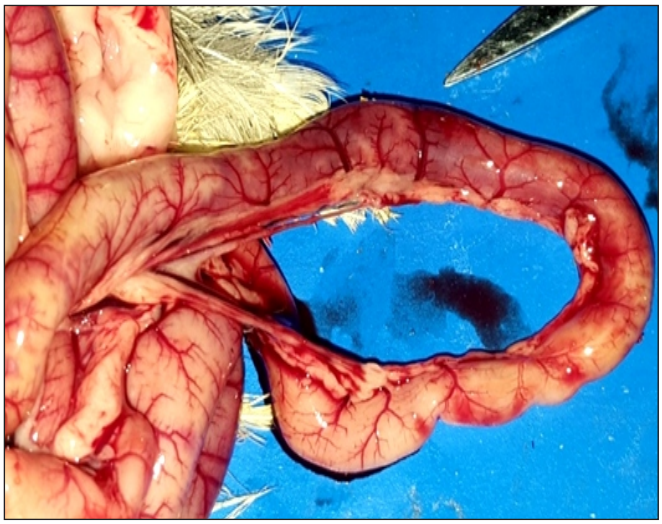
Plate 62: Photomicrograph of intestine revealing severe necrosis in intestinal villi and thickening of intestinal wall due to serosal congestion. H&E X400

Plate 63: Necrotic and thickened intestinal wall due to infiltration of lymphocytes and plasma cells in the lamina propria and sub mucosa. H&E X100

Plate 64: Necrotic enteritis characterised by presence of an admixture of necrotic material, fibrin and inflammatory cells mainly heterophils overlying degenerating and necrotic villous epithelium. H&E X40

Plate 65: Intestine: Necrosis of villi, hyperplasia of goblet cells and proliferation of MNC's in lamina propria and sub mucosa. H&E X400

Plate 66: Hyperplasia of crypts epithelium and presence of mitotic figures. H&E X100



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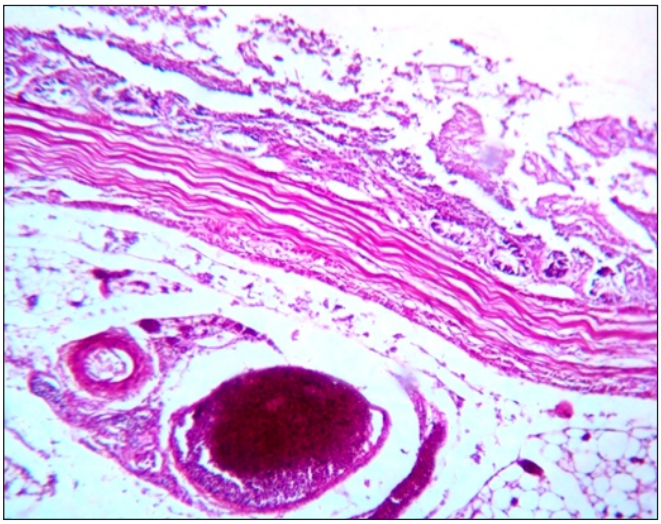

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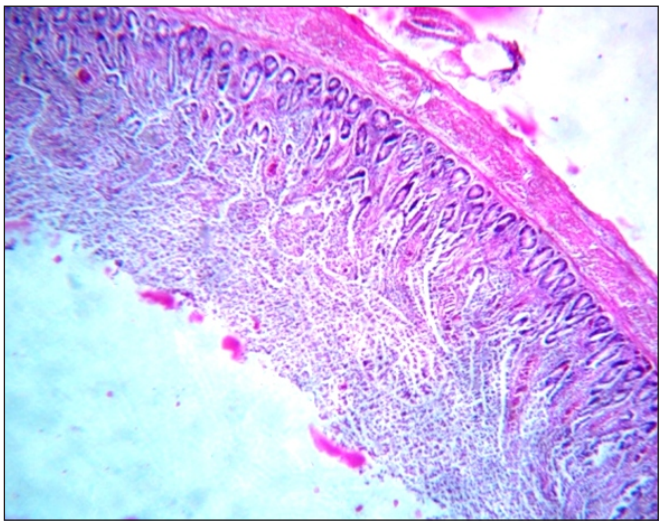

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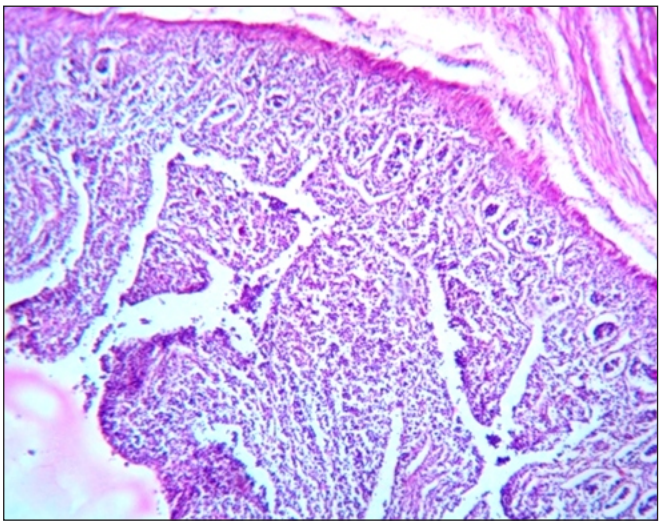

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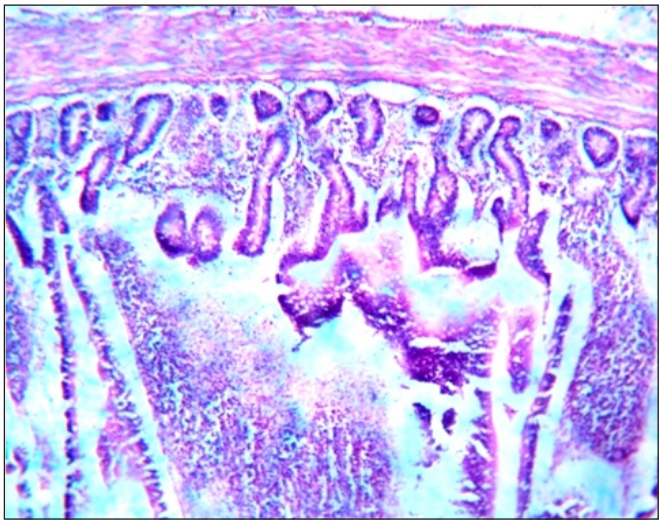

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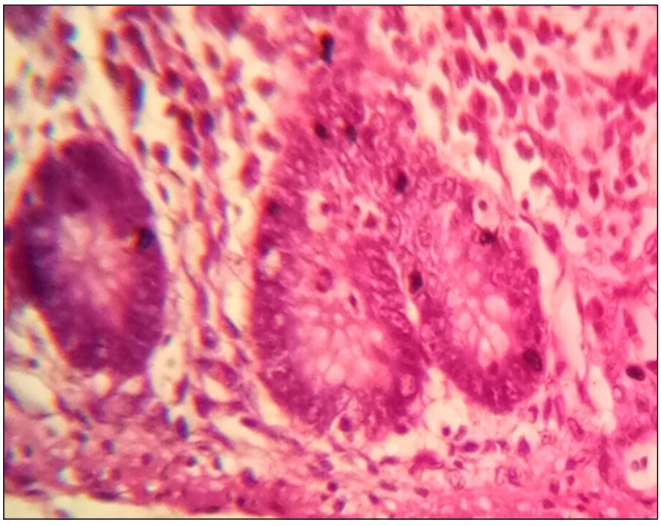


Plate 66

Plate 67: Liver showing diffuse severe congestion and enlargement.

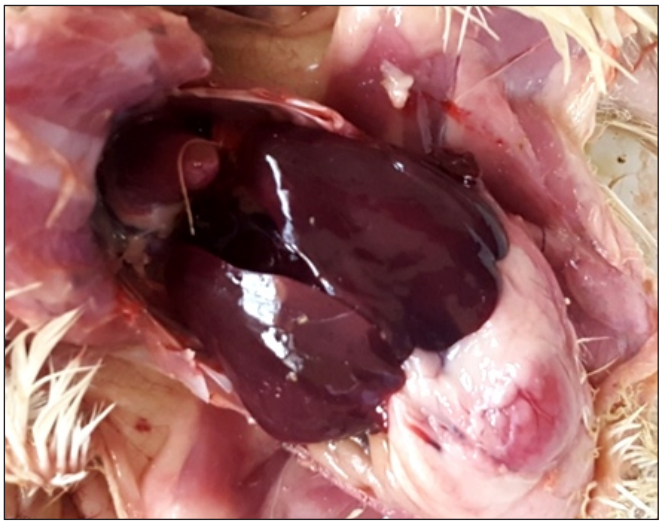
Plate 68: Liver showing fatty changes.

Plate 69: Liver: Petechial haemorrhages scattered throughout the surface of right lobe and involving only upper half of right left lobe.

Plate 70: Liver: Presence of coalescing necrotic foci on surface.

Plate 71: Liver showing congestion and dilatation of central veins along with degeneration and necrosis of hepatocytes. H&E X100

Plate 72: Liver showing necrosis, dissociation and individualization of hepatocytes. H&E X100



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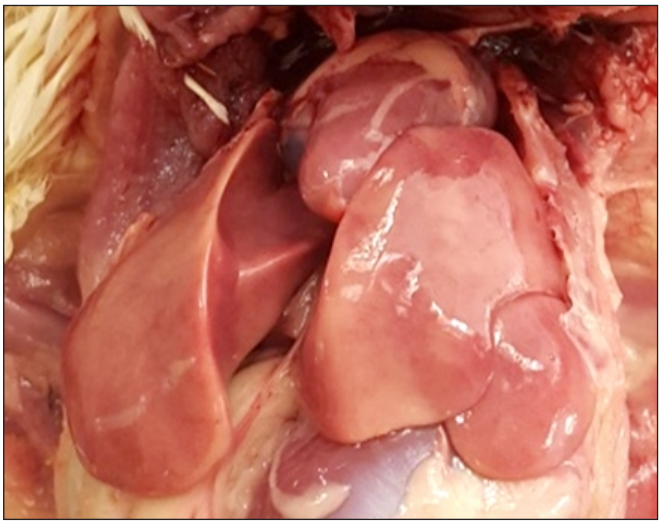

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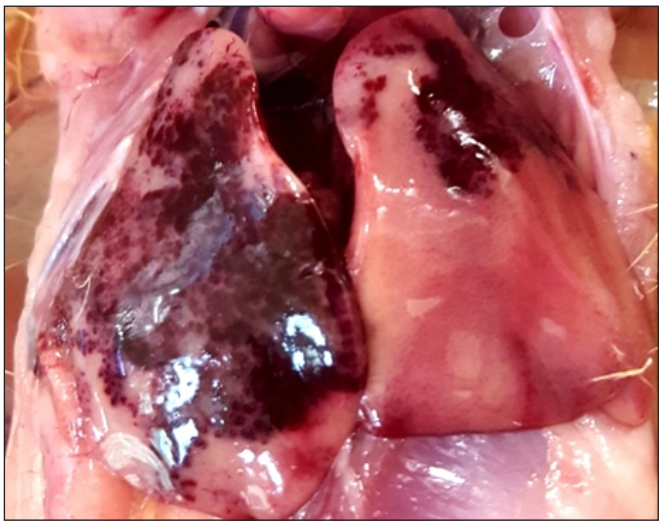

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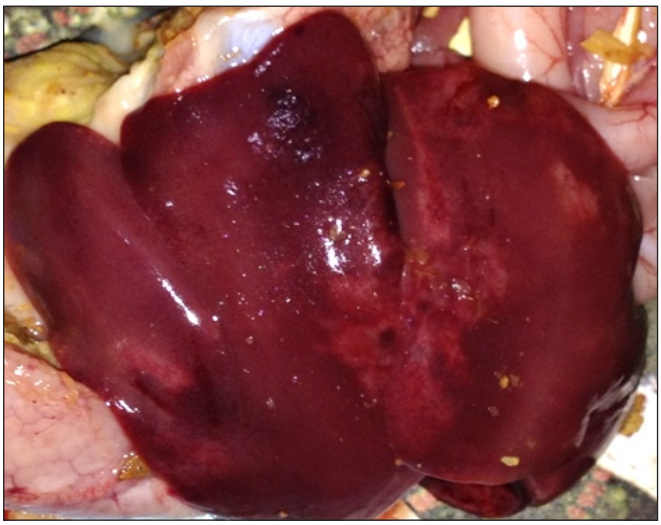

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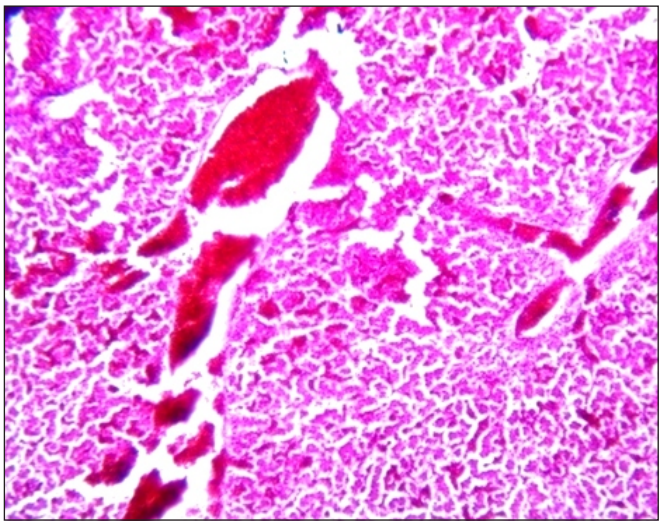

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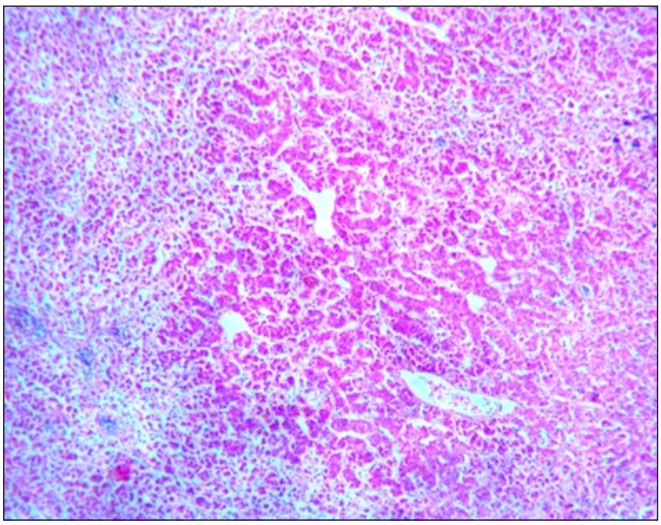


Plate 72

Plate 73: Liver showing necrosis of hepatocytes beneath the capsule. H&E X100

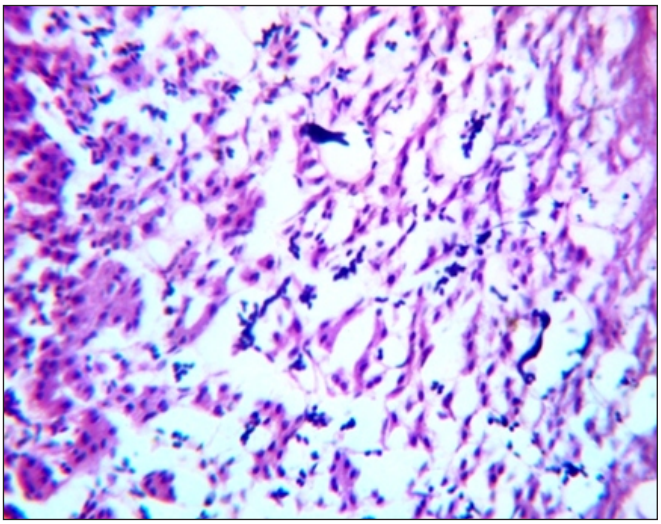
Plate 74: Liver showing oedema and inflammatory cells in portal tract. H&E X100

Plate 75: Liver showing congestion of central vein, dilatation of sinusoids, degeneration and necrosis of surrounding hepatocytes long with infiltration of heterophils. H&E X100

Plate 76: Liver showing congestion of central vein, dilatation of sinusoids, degeneration and necrosis of surrounding hepatocytes long with infiltration of heterophils. H&E X100

Plate 77: Liver showing congestion, multifocal areas of hepatic necrosis and infiltration of large number of heterophils (arrow). H&E X400

Plate 78: Liver showing presence of pseudomembrane with fibrin and infiltrating heterophils overlying a congestion and degenerating liver parenchyma. H&E X100



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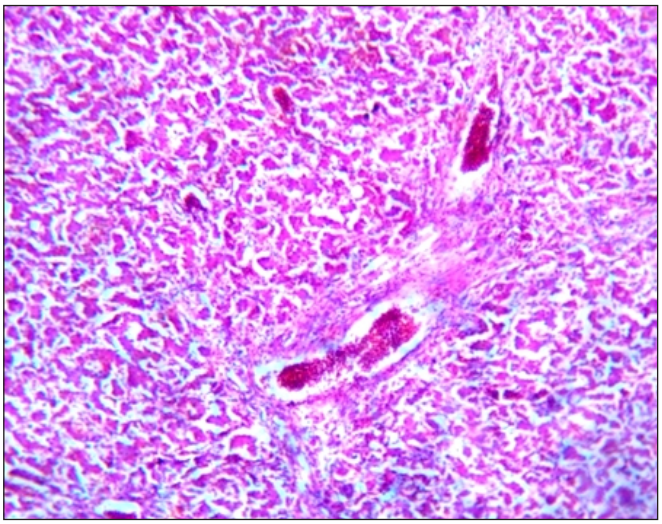
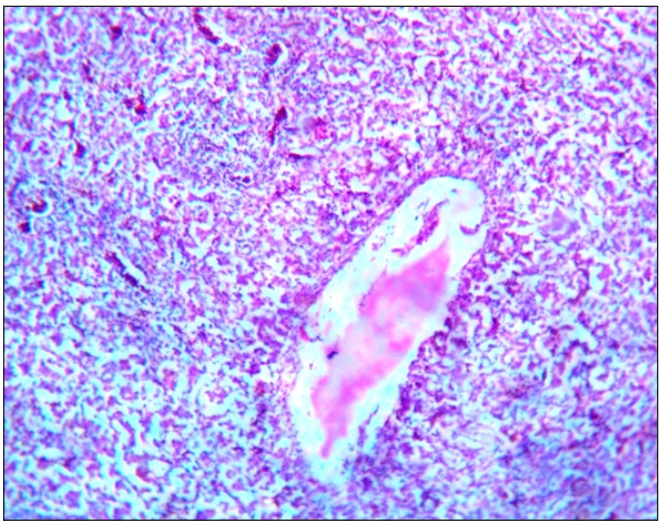


Plate 74

Plate 75



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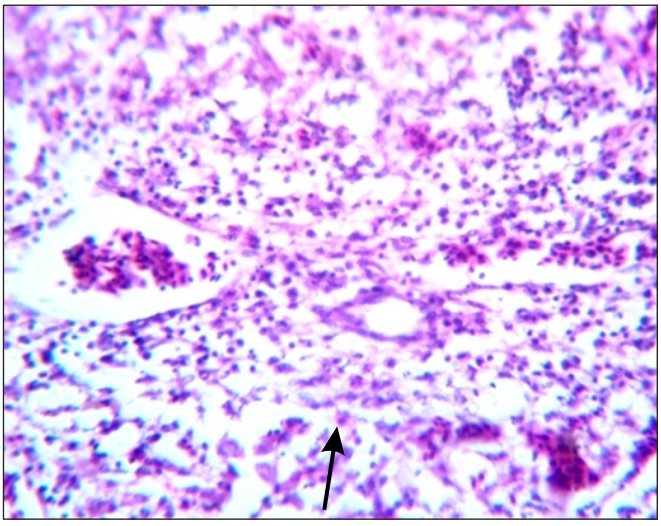

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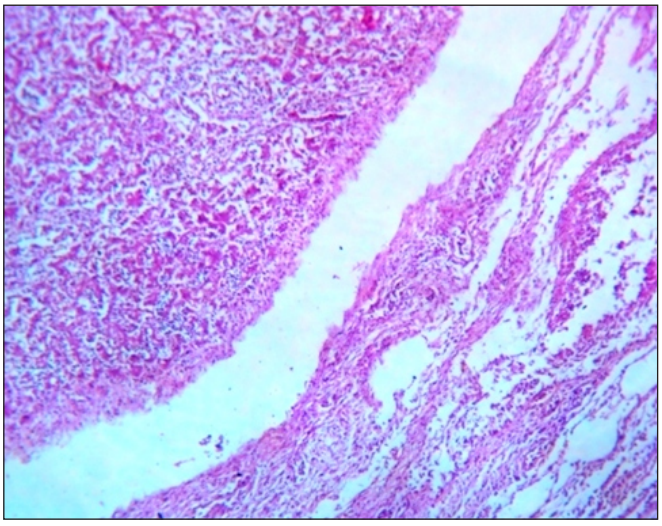


Plate 78

Plate 79: Liver showing presence of fibrin and infiltration of heterophils in large number. H&E X400

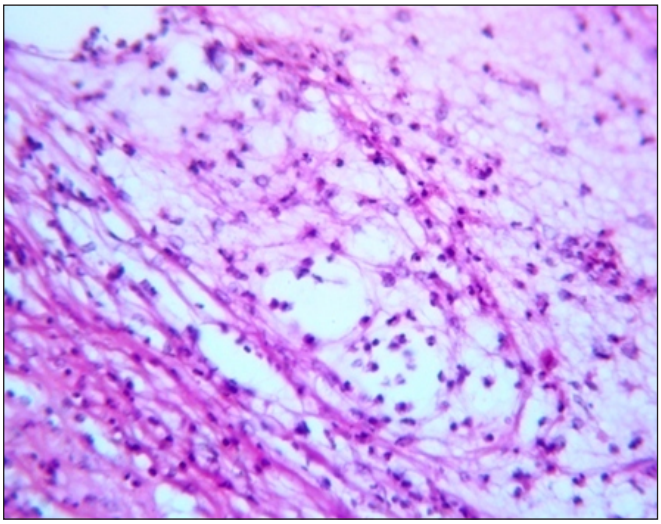
Plate 80: Liver showing degeneration and hyperplasia of epithelium of bile duct. H&E X100

Plate 81: Serosal surface of proventriculus showing diffuse severe congestion.

Plate 82: Proventriculus: Presence of thick mucoid exudate adhering to underlying congestion of mucosa and erotic mucosa of gizzard.

Plate 83: Proventriculus showing swelling and edematous glands.

Plate 84: Accumulation of thick tenacious slimy exudate adherent to walls of proventriculus.



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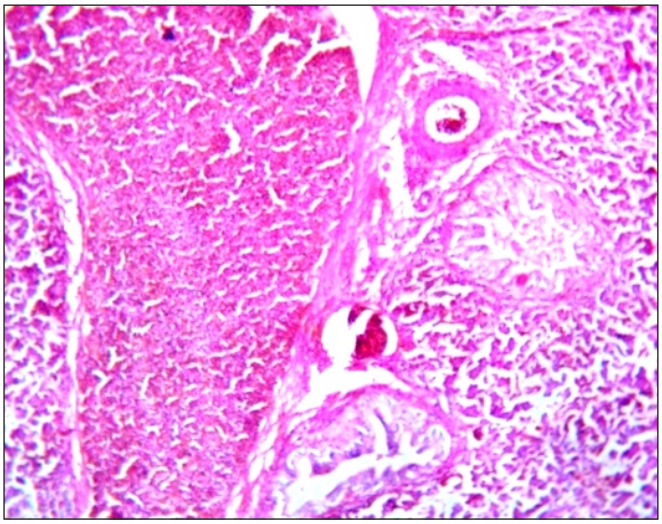

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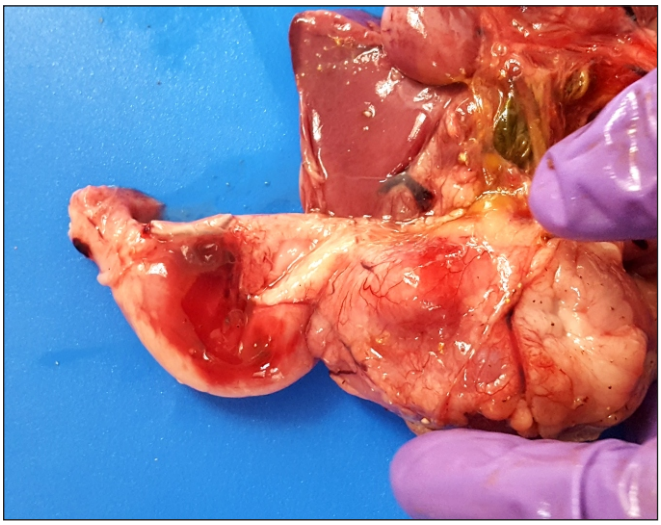

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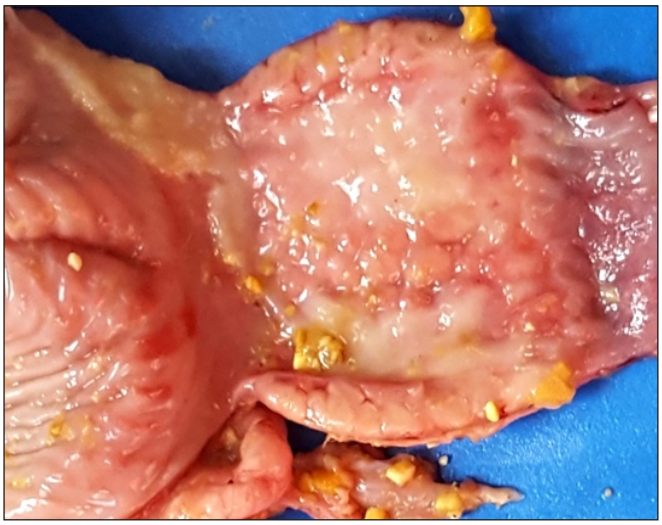

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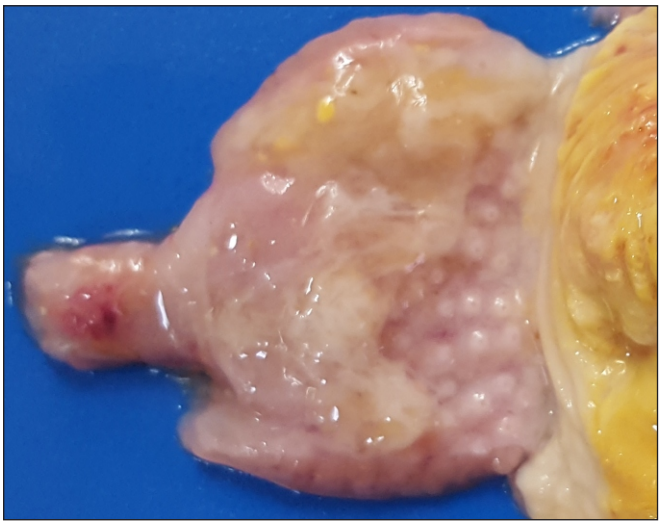

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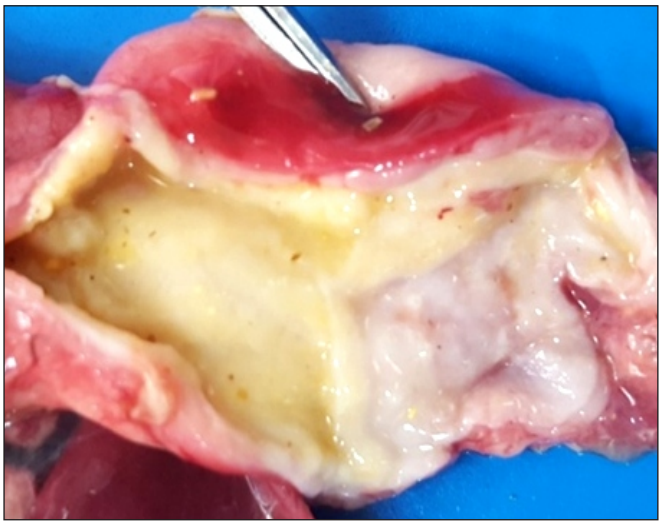


Plate 84

Plate 85: Proventriculus: Degeneration and necrosis of epithelium of mucosal folds and infiltration of heterophils in lamina propria and sub mucosa. H&E X100

Plate 86: Degeneration and necrosis of glands of proventriculus. H&E X100

Plate 87: Proventriculus: Accumulation of edematous fluid in sub mucosa, proliferation of small arterioles and capillaries and infiltration of inflammatory cells. H&E X100

Plate 88: Proventriculus: Proliferation and hyalinisation of connective tissue in the interglandular area. H&E X100

Plate 89: Fibrinous proventriculitis: Congestion of blood vessels, necrotic debris, fibrin and infiltration of inflammatory cells mainly heterophils in serosal layer of proventriculus. H&E X100

Plate 90: Proventriculus showing necrosis, blunted, fused mucosal folds along with inflammatory cells and fibrous connective tissue proliferation in lamina propria and sub mucosa H&E X100

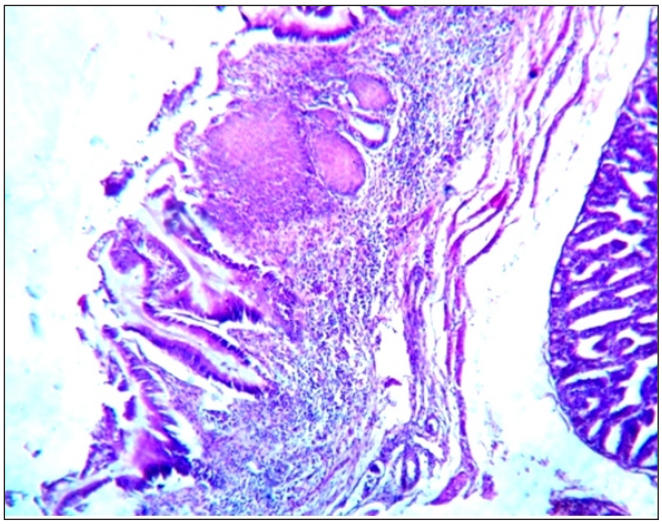


Plate 85

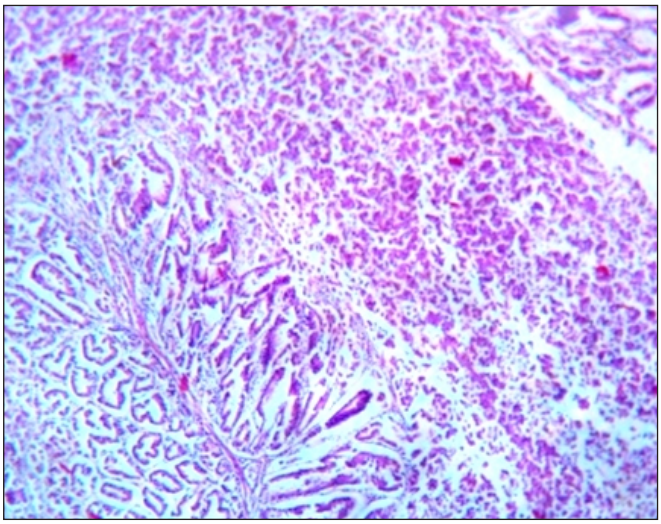
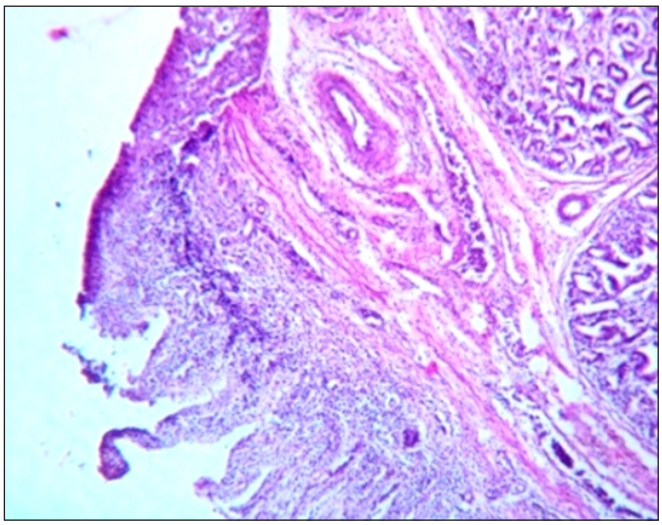


Plate 86



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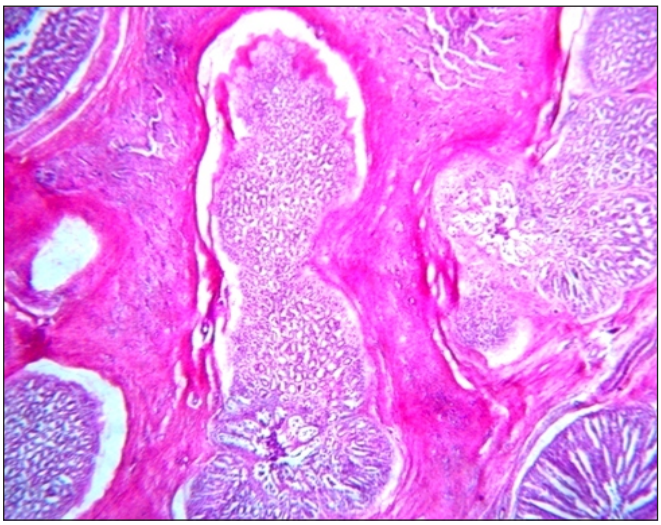

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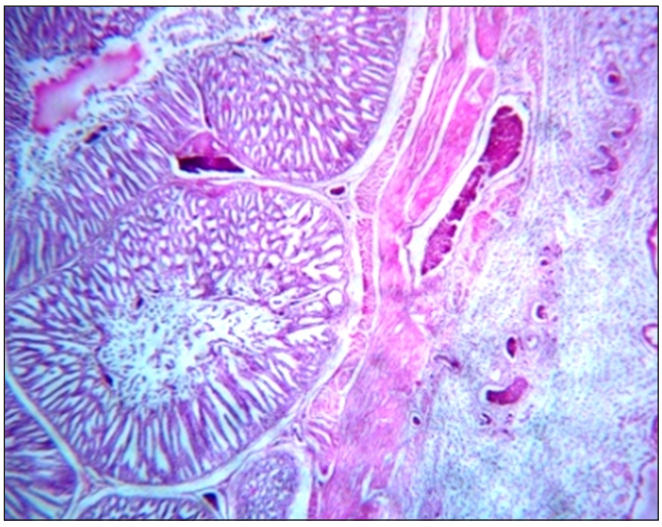

Plate 89

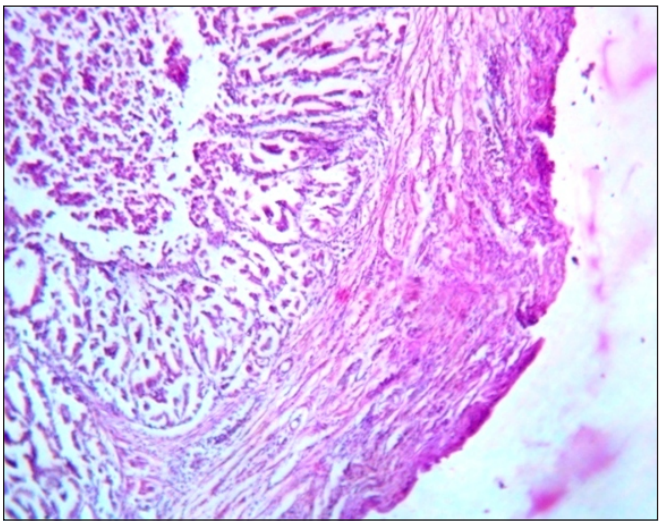

Plate 90

Plate 91: Gizzard showing necrosis of overlying keratinized layer revealing the underlying mucosa and sub mucosa.

Plate 92: Gizzard showing fragmentation, degeneration and necrosis of smooth muscle fibers along with infiltration of inflammatory cells mostly heterophils. H&E X400

Plate 93: Gizzard showing degeneration and necrosis of smooth muscle fibers along with infiltration of inflammatory cells mainly lymphocytes. H&E X400

Plate 94: Gizzard showing proliferation of several congested blood vessel, edema, presence of fibrin in serosa and infiltration of heterophils.

Plate 95: Congestion in pancreas.

Plate 96: Presence of focal ulceration in pancreas.

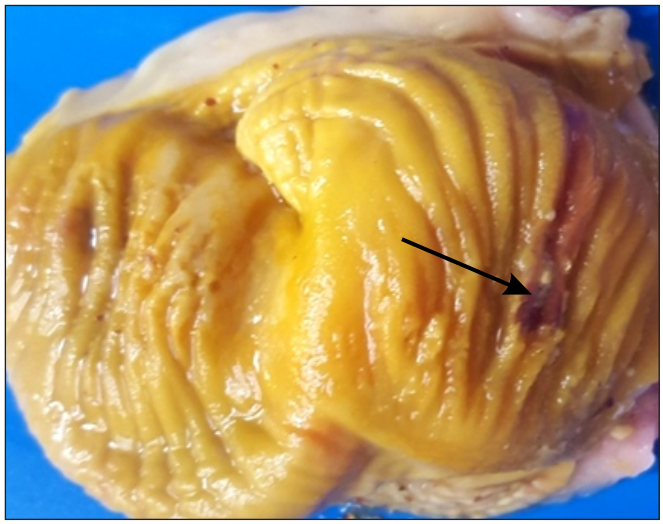

Plate 91

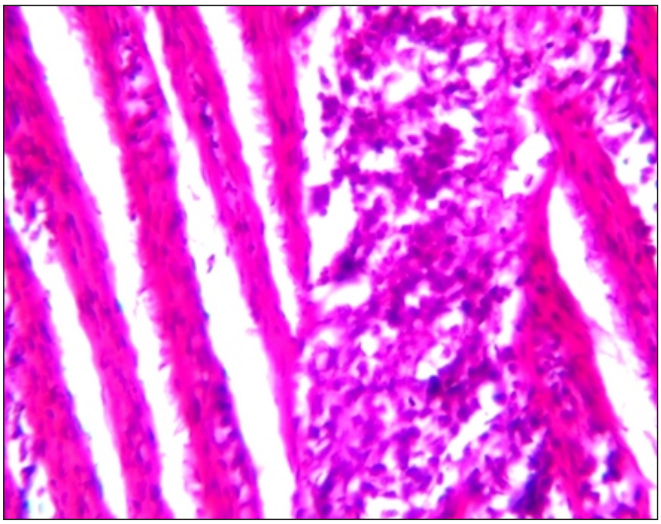

Plate 92

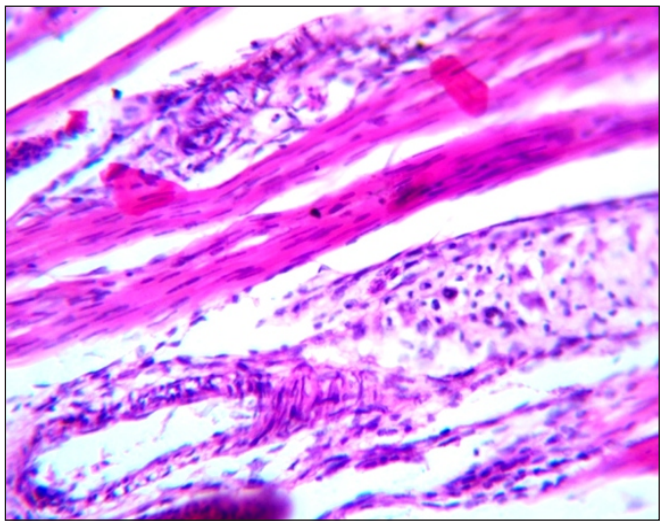

Plate 93

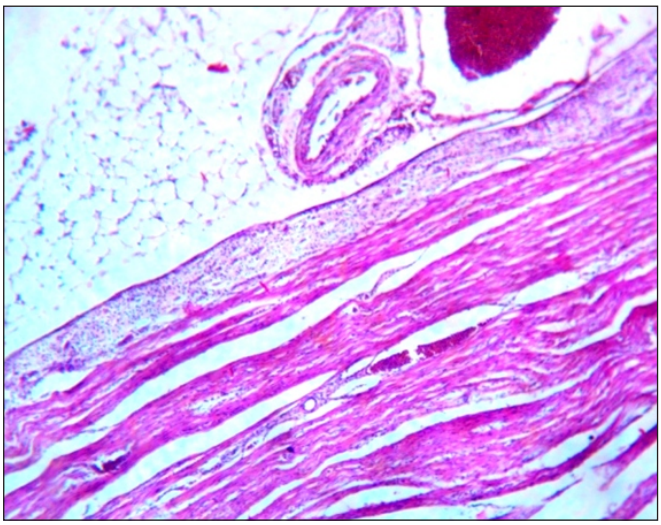

Plate 94

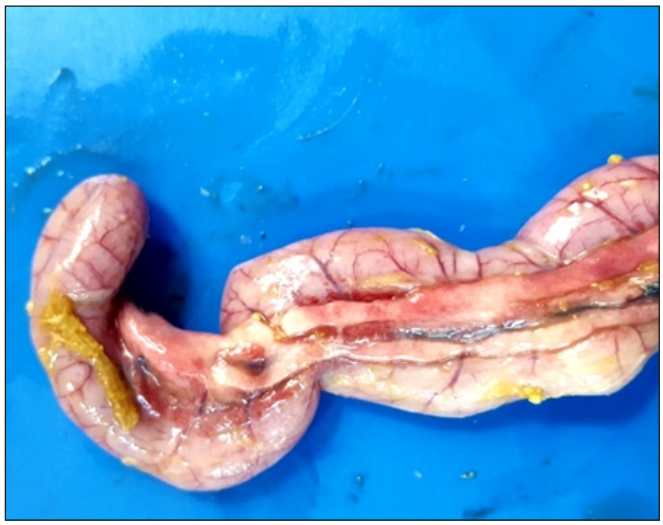

Plate 95

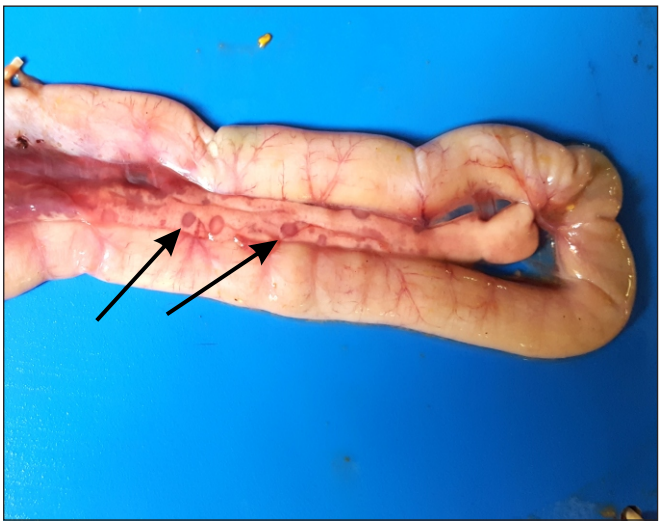

Plate 96

Plate 97: Pancreas showing congestion and haemorrhages. H&E X100

Plate 98: Pancreas: Degeneration and necrosis of acinar cells and heterophilic infiltration. H&E X100

Plate 99: Pancreas: Necrosis of acini and infiltration of heterophils. H&E X400

Plate 100: Pancreas showing hyperplasia of pancreatic ducts, congestion of blood vessels in interlobular areas, degeneration, necrosis of acinar cells and heterophilic infiltration. H&E X100

Plate 101: Oesophagus showing congestion, edema and inflammation in sub mucosa and serosa. H&E X40

Plate 102: Oesophagus: Presence of inflammatory cells in sub mucosa around the oesophageal glands. H&E X100

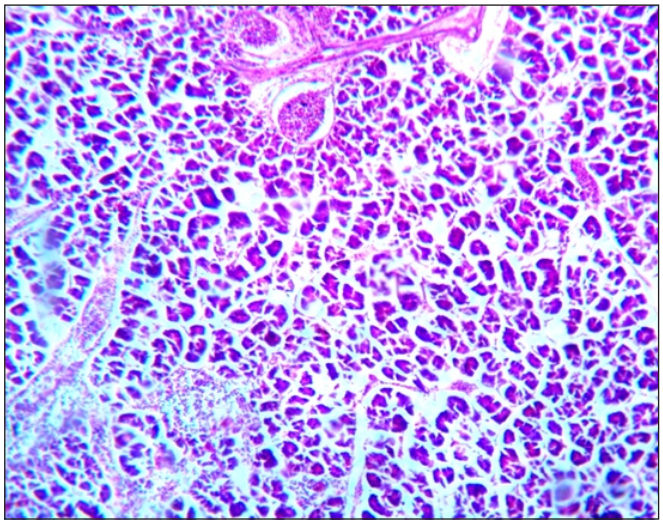

Plate 97

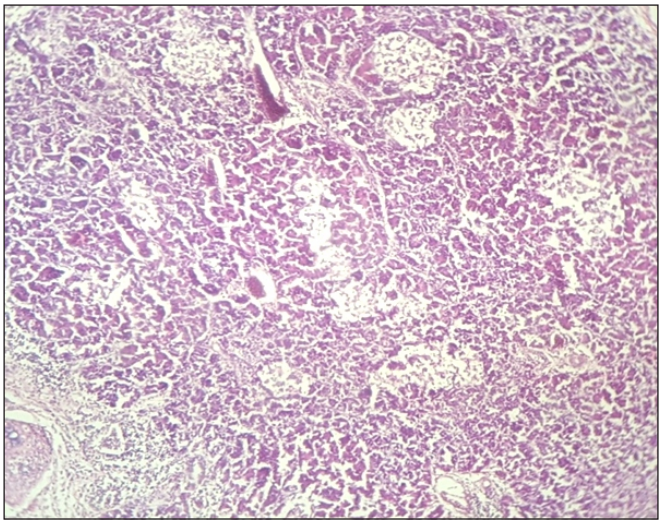

Plate 98

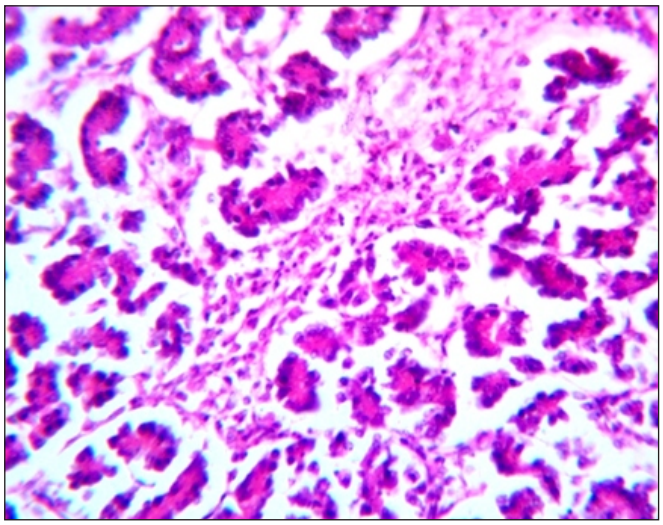

Plate 99

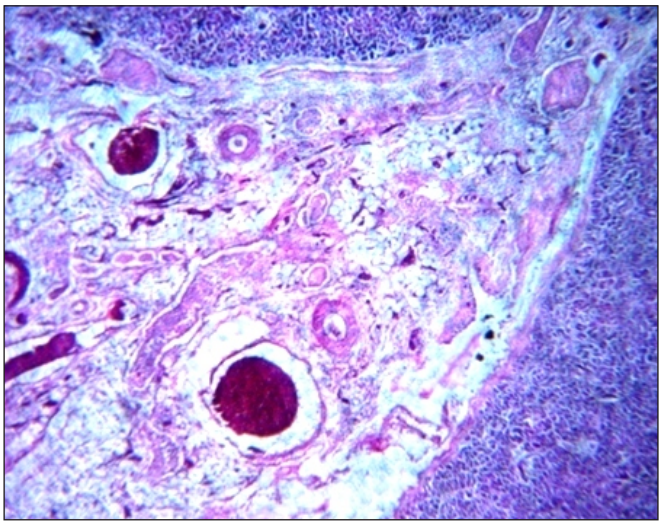

Plate 100

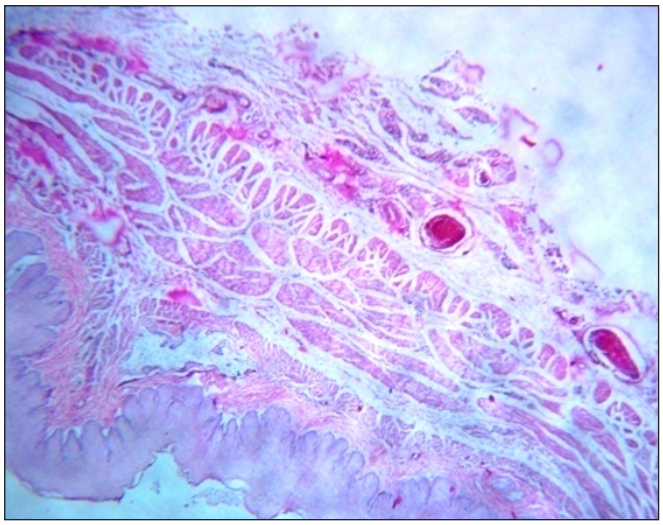

Plate 101

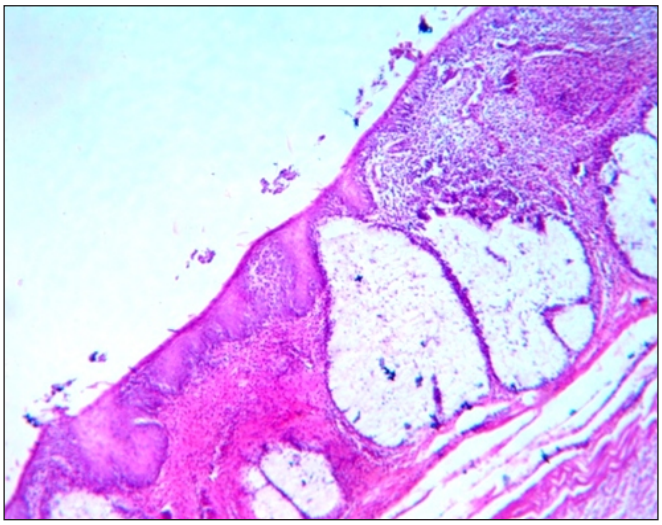

Plate 102

Plate 103: Oesophagus: infiltration of inflammatory cells mostly heterophils in sub mucosa around the oesophageal glands. H&E X400

Plate 104: Oesophagus: Expanding micro-abscesses squeezing the oesophageal glands. H&E X100

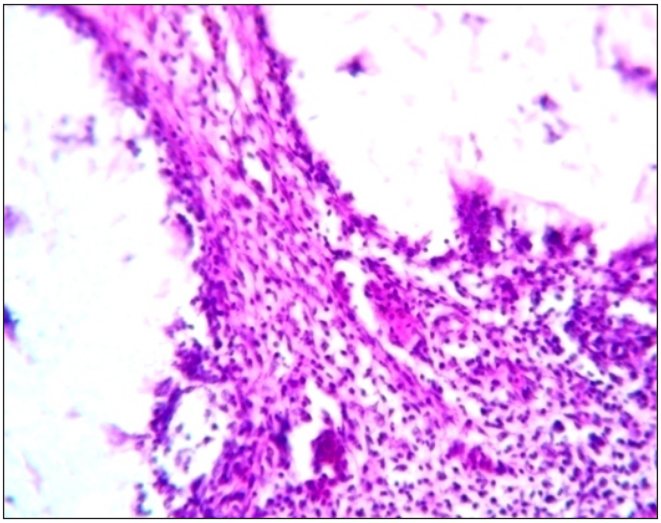

Plate 103

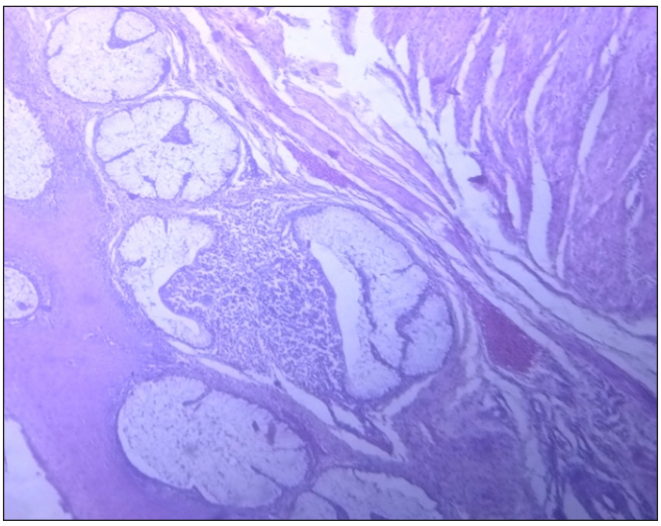

Plate 104

Chapter-5

Discussion

CHAPTER 5

DISCUSSION

The present study was planned to determine etio-pathomorphology of GIT lesions occurring in broilers in Jammu. In addition, a survey of 200 flocks from different parts of Jammu, where morbidity and mortality was reported, was carried out to ascertain occurrence of various diseases affecting the broiler population of Jammu and their mortality pattern.

Occurrence of diseases

During the present study, occurrence of colibacillosis (24.16%) was found to be the highest followed by that of IBD (7.13%), omphalitis (6.79%), salmonellosis (5.36%), enteritis due to nonspecific reasons (4.29%), coccidiosis (2.22%), conditions with haemorrhages in proventriculus (NCD/Avian Influenza suspected) (1.67%), gout (1.44%), hepatotoxicity (1.42%), ascites (1.14%), fatty liver syndrome (1.03%), caseous nodules in lungs (0.75%) and non-specific liver ailments (0.50%). However, Singh *et al.* (2003) found the occurrence of omphalitis, aflatoxicosis, colibacillosis, coccidiosis, aspergillosis, IBD and gout in Punjab to be much lower at 0.44%, 0.41%, 0.39%, 0.16%, 0.14%, 0.08% and 0.06%. Borah *et al*. (2017) recorded incidence of omphalitis, colibacillosis, IBD, necrotic enteritis, Newcastle disease and brooder pneumonia as 13.40%, 11.11%, 10.58%, 6.35%, 4.59% and 3.70%, respectively from birds in Assam. Ahmed *et al.* (2009) however, found the occurrence of colibacillosis IBD, omphalitis, and coccidiosis in Bangladesh to be much higher at 52.26%, 11.06%, 11.56% and 4.52%, respectively. Similarly, Islam *et al*. (2003) in Sylhet region of Bangladesh recorded a higher occurrence of all the above mentioned diseases except that of colibacillosis which at 5% was found to be less than present study. Ahmed *et al*. (2012) reported IBD 29.37% to be the most prevalent in broilers followed by colibacillosis 18.61% and coccidiosis 17.38% in district Poonch Azad J&K.

In the present study, the maximum mortality was seen in cases of colibacillosis (8.33%) followed by IBD (5.97%), conditions with haemorrhages in proventriculus

where Newcastle disease/avian influenza was suspected (5.52%), omphalitis (4.54%) followed salmonellosis (3.66%), nonspecific enteritis (3.36%), nonspecific liver ailments (3.03%), nephro-hepatotoxicity (2.95%), cases with caseous nodules in lungs (2.00%), coccidiosis (1.69%), ascites (1.12%), gout (0.77%) and fatty liver syndrome (0.41%). Similarly, Itoo *et al.* (2013) recorded occurrence of colibacillosis, Newcastle disease, aspergillosis, gout and ascites with respective mortalities of 6.4%, 13.7%, 7.0%, 8.7% and 9.3% in and around Srinagar. However, Singh *et al*. (1998) reported that maximum mortality was caused by omphalitis (1.29%) followed by IBD (0.86%), coccidiosis (0.56%), colibacillosis (0.32%) and fowl typhoid (0.23%) in Punjab. Mahajan *et al*. (1994) in Hisar, observed that the maximum mortality was caused by fowl typhoid 10.54% followed by colibacillosis 8.88%, miscellaneous diseases like gout, ascites (8.05%), brooder pneumonia (7.69%), coccidiosis (6.85%) and Newcastle disease (6.03%). Buragohain and Kalita (2010) observed that ascites syndrome (34.3%) was main cause of mortality followed by colibacillosis (19.23%), omphalitis (12.18%) and caecal coccidiosis (8.33%) in Mizoram. Lalrintlunga and Baruah (1993) from Assam reported that highest mortality was due to Newcastle disease (25.9%), coccidiosis (15.7%), colisepticaemia (14.6%), aflatoxicosis (10.5%) and omphalitis (5.8%). Anjaneyulu *et al*. (1998) reported that colisepticaemia (13.4%) followed by IBD (9.1%), coccidiosis (7.0%) and ascites (6.78%) were the major causes of mortality in broilers in Prakasam district of Andhra Pardesh. Pugashetti and Shivakumar (2007) observed that maximum deaths in broiler birds in an organised poultry farm of Karnataka were caused by pneumonia (29.60%), enteritis (27.73%), ascites (7.22%) and coccidiosis (2.51%). Reddy and Reddy (1991) observed the mortality pattern in broilers was maximum due to coccidiosis (21.51%) followed by Newcastle disease (11.8%) and enteritis (1.95%). However, Ahmed *et al.* (2009) reported that highest mortality was due to colibacillosis (52.26%) followed by omphalitis (11.56%), IBD (11.06%), coccidiosis (4.52%), mixed infection of IBD & coccidiosis (1.51%) and salmonellosis (1.01%) at Kapasia in Gazipur district.

5.1 Bacterial diseases

5.1.1 Colibacillosis

In the present study, colibacillosis was major cause of mortality in all age groups with occurrence of 24.16% and mortality of 8.33%. Infection of *E.coli* was observed throughout the year. Similar, Sultana *et al.* (2012) recorded 21.36% occurrence in 220 flocks throughout year in Lahore, Pakistan. Matin *et al*. (2017) recorded 0.84% prevalence of colibacillosis in broilers in Mymensingh district of Bangladesh. During earlier study, Kaul *et al.* (1992) reported an outbreak of colibacillosis in 3-7 weeks broiler chicks in North Gujrat where 16.25% mortality was observed.

Bhalerao *et al.* (2013) studied the maximum mortality was reported in birds of 3-4 weeks of age due to *E.coli* infection in Haryana state. Sheriff and Kumaran (1987) observed 14.1% mortality in broilers due to colibacillosis in and around Pudukkottai district, Tamil Nadu. Yunus *et al.* (2009) found *E.coli* infection occurs throughout the year in Pakistan. Ahmed *et al.* (2009) reported that colibacillosis is a major problem for broiler production in Bangladesh. High incidence of *E. coli* infection is observed when unhygienic conditions are present at poultry farm premises or due to supply of contaminated water (Sultana *et al*., 2012). Also, *E.coli* is an opportunist and colibacillosis mostly occurs as a secondary bacterial infection when host defences are lowered due to any other viral infection (Saif, 2008).This problem can be overcome by improving sanitary conditions and adopting effective biosecurity conditions at the farm.

The prominent lesions recorded in the present study were enlarged, friable or congested livers, fibrinous pericarditis, airsacculitis, multifocal areas of degeneration and necrosis of hepatocytes with infiltration of heterophils and perihepatitis. Proventriculitis was characterised by necrosis of epithelium of mucosal folds, edema, congestion, presence of fibrinous exudate and severe infiltration of inflammatory cells mainly heterophils in lamina propria, sub mucosa or serosa was seen. Enteritis characterized by congestion, necrosis of villi, deposition of fibrinous exudate and infiltration of heterophils in lamina propria or sub mucosa was also seen. Similar lesions have been described to be associated with colibacillosis by workers all over the world (Nakamura *et*

al., 1985; Mukherjee and Khanapurkar, 1994; Pourbakhsh *et al*., 1997; Sharada *et al.,* 1999; Islam *et al.,* 2008; Gosh *et al*., 2006; Ahmed *et al.,* 2009; Chanie *et al.,* 2009; Chowdhury *et al*., 2009; Omer *et al*., 2010; Hasan *et al.,* 2010a; Tonu *et al*., 2011; Bhalerao *et al.,* 2013 and Ali and Ali *(*2015). Besides, Baliarsingh *et al.* (1993) observed similar lesions in experimentally produced *E.coli* infection in chicks.

In the present study, isolates of *E. coli* belonged to serogroup O1, O22, O37, O114, O118 and O149. Different serotypes were isolated by different workers. Shankar *et al.* (2010) were recorded O78, O75, O2, O6 and O111 serogroups in Hisar whereas Sahoo *et al.* (2012) recorded O1, O9, O23, O33, O103 & O119 serotypes in different farms of Odisha. Sarker *et al.* (2013) revealed O2, O8, O9, O19, O37, O86, O101, O133, O151 and O173 in West Bengal.

5.1.2 Omphalitis

In the present study, omphalitis was recorded in birds 0-2 weeks of age with an overall occurrence of 6.79% and mortality of 4.54%. Ahmed *et al.* (2009) recorded 11.56% occurrence in Gazipur district of Bangladesh. Buragohain and Kalita (2010) recorded 12.18% mortality in Mizoram. Sheriff and Kumaran (1987) reported 22.23% mortality due to yolk sac infection in broilers in and around Pudukkottai district of Tamil Nadu. Srivastava (1990) recorded 23.7% mortality due to omphalitis in 0-4 week old broiler chickens. Ghodasara *et al*. (1992) recorded 26.53% mortality in chicks with yolk sacs infection. In the present study, yolk sac infection might signify the poor quality of day old chick available in Jammu.

Gross lesions in the present study included unabsorbed and haemorrhagic yolk. Liver was enlarged, congested or pale yellow in colour. Microscopically, liver revealed degeneration, atrophy and necrosis of hepatocytes and consequent dilation of sinusoids. Intestine showed congestion and catarrhal exudate in the lumen. Enteritis characterised by necrosis of villi, desquamation of epithelium inside the lumen of vill, invasion of inflammatory cells such as lymphocytes and heterophils in lumen of lamina propria. Almost similar type of lesions were earlier described by Gross (1964), Bhatia *et al.* (1972), Khan *et al.* (2004), Ahmed *et al.* (2009) Tonu *et al*. (2011) and Jalob *et al.*

(2015). Khan *et al.* (2002) observed similar lesions in experimentally produced in broilers.

5.1.3 Salmonellosis

In the present study, salmonellosis was reported in chicks up to 3 weeks of age with an overall occurrence of 5.36% and mortality of 3.66%. Ahmed *et al.* (2009) recorded 1.01% prevalence in broilers in Gazipur district. Jindal *et al*. (1999) reported 4.82 % mortality due to fowl typhoid during 1994-95 and had gone up to 12.12 % during the year 1996-97 in Haryana. Kumari *et al*. (2013) observed high mortality in young chicks in Haryana. Barbour *et al.* (1999) recorded high average mortality of 4% in 11 broiler flocks between 8 to 15 days of age in Lebanon.

In the present study, grossly bronze discoloration, presence of haemorrhagic and necrotic foci was observed. Histopathologically, liver showed mild to severe vacuolar degeneration and multifocal areas of coagulative necrosis along with congestion and haemorrhages. Multifocal areas of necrosis infiltrated by heterophils were seen. Proventriculitis characterized by congestion and infiltration of heterophils in lamina propria and enteritis characterized by congestion, necrosis and infiltration of heterophils was observed. These findings are in concurrence with those of Bhattacharyya *et al.* (1984), Chisti *et al.* (1985), Porter and Long (1998), Hafeeji *et al.* (2000), Ahmed *et al.*(2009), Hasan *et al.* (2010a), Nazir *et al.* (2012) Kumari *et al*. (2013) and Muna *et al.* (2016). Besides, Majid *et al.* (2000) and Dutta *et al.* (2015) observed similar lesions in layers. Singh (1998) observed similar lesions in experimentally produced in broilers

5.2 Viral diseases

5.2.1 Infectious bursal disease (IBD)

In the present study, IBD had occurrence and mortality of 7.13% and 5.97% respectively. Disease was recorded throughout the year. Choudhary *et al*. (2012) round the overall incidence of IBD to be 33.90 % in and around Ranchi (Bihar) whereas Bhutia and Singh (2016) recorded the 15.13% incidence of disease in Mizoram. Higher mortality (48.04%) as compared to our findings was recorded by Farooq *et al*. (2003) in Mirpur

and Kotli districts of Kashmir. Gill *et al.* (1988) recorded 38.46% mortality in Punjab. Chauhan *et al.* (1980) recorded the 17.89 % mortality in one poultry farm and 19.8 % on the other farm of Ranchi. Rajashewar *et al.* (1992) reported mortality rate of 23.2 % in Kanyakumari district of Tamil Nadu.

Lesions observed in the present study were enlarged, edematous bursa with presence of haemorrhages and creamy exudate in the lumen, Degeneration and necrosis of follicular lymphocytes followed by formation of cystic cavities, infiltration of heterophils in interfollicular haemorrhages on thigh and breast muscles. Congested kidneys, nephritis and enteritis characterised by congestion, haemorrhages and necrosis of villi with infiltration of inflammatory cells in lamina propria were also seen. The findings of present study were strongly agreement with the study of Mahajan *et al*. (2002), Gurel *et al.* (2003), Jindal *et al*. (2004), Ghosh *et al.* (2006), Singh, (2008), Chanie *et al.* (2009), Ahmed *et al.* (2009), Mor *et al.* (2010), Hasan *et al.* (2010b), Sellaoui *et al.* (2012), Singh *et al*. (2015) and Bhutia and Singh (2016).

5.2.2 Haemorrhages in proventriculus (Suspected cases of Newcastle disease/Avian influenza)

In the present investigation, haemorrhages in proventriculus were observed in 2-3 week old chickens with 1.67% occurrence and 5.52% mortality. These cases were recorded during the winter season as similarly described by Bhutia and Singh (2016) in Mizoram. Kumar *et al.* (2016) reported the 5.35% prevalence and 20.76% mortality in non-vaccinated and 4.6% mortality in vaccinated broiler flocks in commercial broiler farms at Bochaganj Upazila of Dinajpur district.

In the present study, haemorrhages were found in proventriculus and cases were suspected for NCD/Avian Influenza which was earlier described by Singh (2008), Datta *et al.* (2013), Bhutia and Singh (2016) and Kumar *et al.* (2016).

In the present study grossly, hemorrhages near the junction of proventriculus and gizzard, congestion and haemorrhages in intestine. Microscopically, proventriculus revealed presence of congestion, haemorrhage, edema in lamina propria and sub mucosa along with necrosis of mucosal papillae. Intestine section showed severe congestion and

infiltration of MNCs. Almost similar findings were noticed by Singh (2008), Hasan *et al.* (2010b), Hadipour *et al*. (2011), Khan *et al.* (2011), Bhutia and Singh (2016), Kumar *et al.* (2016) and Brar *et al.* (2017). Khorajiya *et al.* (2015) saw multifocal to diffuse hemorrhages around proventricular glands, necrotic (diphtheritic) haemorrhagic ulcers throughout the intestine in layers. Capue *et al.* (2002) observed haemorrhagic lesions in mucosa of proventriculus in outbreak of Newcastle disease in Italy.

5.3 Protozoan diseases

5.3.1 Coccidiosis

In the present study, coccidiosis was observed with overall occurrence of 2.22% and mortality of 1.69% respectively in monsoon and post-monsoon season only. In contrast, in a study conducted by Sharma *et al*. (2015) in Jammu, 39.58% occurrence was found in organized poultry farms and backyard poultry in monsoon season. 29.08% the samples were found to be positive for coccidiosis in Aurangabad district of Maharashtra by Jadhav *et al.* (2011). Kala *et al*. (2013) reported the 21.38% incidence of coccidiosis in broilers at Patna, Bihar. Shamim *et al.* (2015) recorded the overall 9.59 % prevalence of coccidiosis in broilers in Mirpur, Azad Kashmir, Pakistan. Ahad *et al*. (2015) reported that prevalence of disease was low in summer and high in autumn season in Kashmir valley. Panda *et al.* (1997) found 11.57% mortality in Orissa due to coccidiosis. Datta *et al.* (2013) observed that 28.27% cases of enteritis reported in Hisar suffered from coccidiosis. Variation in occurrence of coccidiosis might be due to difference in geographical location, agro-climatic conditions and inclusion of layers or backyard poultry in the investigations. Higher prevalence in monsoon period seen in our study as in other previous studies could be attributed to high humidity and drop in temperature in this season. These weather conditions are generally conducive for sporolution of oocysts, Sharma *et al*. (2015). Also, high stocking density, poor handling, short resting period, failure to disinfect feeding or watering equipments and rearing of birds in deep litter system are predisposing factors for occurrence of coccidiosis at a poultry farm (Lobago *et al*. 2005; Razmi and Kalideri, 2000 and Shirzad *et al.*, 2011).

In the present study, lesions included congestion, haemorrhage necrosis of villi and presence of developing meronts, schizonts and oocysts in epithelium of villi and crypts. Similar lesions have been recorded by Long *et al*. (1974), Dhillon, (1992), Jithendran, (2001), Soomro *et al*. (2001), Ito *et al.* (2004), Lobago *et al.* (2005), Kumar *et al*. (2008), Ahmed *et al.*(2009), Chanie *et al.* (2009), Sood *et al.* (2009), Patra *et al.* (2009), Patra *et al*. (2010), Daryoush *et al.* (2011), Dolka *et al.* (2012), Khaier *et al*. (2015) and El-Sayed *et al.* (2017). Besides, Kadhim, (2014) observed similar lesions in experimentally produced coccidial infection in chicks.

5.4 Metabolic diseases

5.4.1 Ascites

In the present study, ascites was observed in 2-4 week old birds with total occurrence and mortality of 1.14% with 1.12% respectively. Datta *et al.* (2013) found 13.92% cases of ascites in Hisar. Buragohain and Kalita (2010) recorded 34.3% mortality in broilers due to ascites in Mizoram. Pugashetti and Shivakumar (2007) observed 7.22% mortality due to ascites in broiler birds in an organised poultry farm of Karnataka. Parimala (2003) 0.58% chickens in Hyderabad were found to be affected with ascites syndrome. Anjum *et al.* (1998) recorded 4.46% mortality in and around Faisalabad. Nakamura *et al.* (1999) recorded 55.8% mortality in broilers affected with ascites syndrome. Among many predisposing factors leading to ascites syndrome; pulmonary hypertension, higher metabolic oxygen requirement, heat stress and improper management practices.

In the present study, grossly, abdomen was filled with straw yellow colored fluid and liver was enlarged pale and had irregular edges. Kidneys were also pale and swollen. Microscopically, changes in liver varied from vacuolar degeneration, fatty degeneration to severe coagulative type of necrosis of hepatocytes with infiltration of heterophils. Anjum *et al*. (1998), Habib-ur-Rehman *et al*. (1999), Nakamura *et al.* (1999), Tafti and Karima (2000), Parimala (2003), Davis *et al.* (2012), Franciosini *et al*. (2012) and Bhalerao *et al*. (2013) reported identical changes in chickens suffering from ascites syndrome. Likewise, Biswas *et al.* (1995) observed hepatic congestion and necrosis along

with fibrosis in sinusoidal space. Davis *et al.* (2012) observed accumulation of clear yellow fluid in the abdominal cavity and multifocal areas of necrosis and disorganization of hepatic cords in liver. Anjum *et al*. (1998) also saw congested kidneys and flabby heart in chickens suffering from ascites syndrome. Granulomas in kidneys were found in broliers with ascites syndrome by Parimala (2003). Other workers have found lesions associated with ascites which were not found in present study like Wilson *et al*. (1988) observed hyperplasia and capsular fibrosis of liver in birds with ascites and Zafra *et al*. (2008) saw granulomatous pneumonia in cases of ascites.

5.5 Hepatotoxicity

In the present study, this condition was recorded with an overall occurrence of 1.42% and 2.95% mortality. However, Raina (1989) reported slightly higher prevalence (6.22%) of naturally occurring mycotoxicosis in Punjab.

In the present study, pale and swollen kidneys with deposition of urate crystal were seen. Additionally, congestion and pin point haemorrhages on the surface of the kidneys were seen. Liver was pale, mottled and had necrotic foci on its surfaces. Microscopically, congestion, haemorrhages, degeneration and coagulative necrosis and infiltration of inflammatory cells chiefly heterophils in portal triad areas were also present. Similar types of microscopic lesions have been observed by Singh *et al*. (1994) in cases of mycotoxicosis. Lalrintluanga and Baruah (1997) and Al-Sadi *et al*. (2000) noticed similar morphological features in liver in cases of aflatoxicosis. Rao *et al.* (1985) reported degenerative and necrotic changes in an outbreak of aflatoxicosis. Moorthy *et al. (*1985) also observed similar lesions in cases of experimental aflatoxicosis. Bakshi *et al.* (1995) reported enlargement of liver with petechial haemaorrhages, degenerative and necrotic changes in pathomorphological studies on experimental aflatoxicosis in commercial broilers. Kumar and Balachandran (2009) and Ramdas *et al.* (2013) noticed similar morphological features in liver in cases of experimentally produced aflatoxicosis in broilers.

5.6 Fatty liver syndrome

In the present study, overall 1.03% occurrence and 0.41% mortality was recorded in cases of fatty liver syndrome. Higher (8.83%) and lower (0.58%) occurrences of fatty liver syndrome have been reported by Joshi and Bhagwat (1995) and Parimala (2003) respectively. Grossly, liver appeared friable and pale yellow in color. Fatty degeneration was characterised by the presence of vacuoles in the cytoplasm of hepatocytes. Sinusoids were dilated and congested, necrosis of hepatocytes was observed at multifocal areas with mild heterophilic infiltration. Similar types of lesions were observed by Lonkar and Prasad (1988); Madan Joshi and Bhagwat (1995), Parimala (2003) and Davis *et al.* (2012).

5.7 Internal haemorrhages

Grossly**,** in the present study big clots of blood were found in abdominal cavity and liver rupture was found to be the cause of internal bleeding. Light pale, anemic liver and kidneys were also seen. Parthasarathy *et al*. (1979) and Parimala (2003) observed similar lesions in fatty liver haemorrhagic syndrome.

5.8 Occurrence and pathology of various lesions of GIT of broilers in Jammu:

5.8.1 Intestines

In the present study, intestines were affected in 80.22% cases. Grossly, lesions consisted of congestion, haemorrhages, necrosis and enteritis which were seen in 40.43%, 21.10%, 12.42% and 68.44% cases respectively. Microscopic pathological lesions comprised mainly of congestion, haemorrhage, edema, degeneration, necrosis, acute, subacute and chronic enteritis which were encountered in 76.72%, 57.79%, 52.66%, 90.33%, 76.72%, 69.03%, 2.36% and 3.35% cases respectively. Similarly, Datta *et al.* (2013) noticed that 30.35% of the necropsied birds in the age group of 29-35 days had lesions consistent with enteritis in Haryana. The specific diseases in which enteritis were observed alongside included coccidiosis, ascites, mycotoxicosis, *E.coli* infections, IBD, necrotic enteritis, Newcastle disease and vitamin deficiencies. El-Sayed *et al.* (2017) did a pathological study on enteric affections in chickens and observed that occurrences of

necrotic enteritis, catarrhal enteritis and parasitic enteritis in broilers were 67.80%, 11.86% and 8.47% respectively. Long (1973) diagnosed 7.7% cases of necrotic enteritis in Ontario. Hermans and Morgan (2007) reported 12.3% occurrence of necrotic enteritis in broilers. However, Pugashetti and Shivakumar (2007) observed 27.73% mortality due to enteritis in broiler birds in an organised poultry farm of Karnatak. Daryoush *et al.* (2011) recorded that 78.57% of the dead birds had enteritis. Catarrhal enteritis, haemorrhagic enteritis and necrotic enteritis was seen in 51.43%, 17.14% and 15.71% mortality cases. Islam *et al.* (2003) recorded necrotic enteritis in 0.44% broiler birds in Bangladesh. Daryoush *et al*. (2011) conducted a histopathological study on poultry enteritis in Iran and found that the highest rate of occurrence of enteritis (78.57%) was seen in broilers. Similarly, Lee *et al*. (2002) also found that broiler chickens suffered more than others from enteritis.

Pathological lesions in intestinal tract consisted of catarrhal exudate coating the surface of intestine and thickened intestinal walls, desquamation, severe necrosis of mucosal epithelium, blunting-stunting, fusion of villi and thickening because of infiltration of macrophages, heterophils and plasma cells in lamina propria and sub mucosa. Similar lesions were noticed by Talha *et al.* (2001), Islam *et al.* (2003) and Ghosh *et al.* (2006). Nunez *et al.* (2016) observed similar lesions associated with parvovirus infection in broilers, while Khan *et al*. (1995) and Goodwin *et al.* (1993) observed that birds with runting-stunting syndrome had similar gross and histopathological changes in the intestinal tract. Tonu *et al*. (2011) and Soomro *et al.* (2001) recorded similar lesions while studying colibacillosis and coccidiosis, respectively. Acute catarrhal enteritis in the present study was characterised by necrosis of epithelium, hyperplasia of goblet cells, presence of edema and inflammatory cells mainly heterophils along with severe congestion and haemorrhages in lamina propria. Similar type of lesions was observed by (Talha *et al*., 2001; Islam *et al*., 2003; Ghosh *et al.*, 2006; Ahmed *et al.*, 2009; Daryoush *et al.,* 2011; Kumari *et al.,* 2013 and El-Sayed *et al.,* 2017). Necrotic enteritis was characterised by severe denudation and necrosis of intestinal epithelium along with fusion of adjacent villi also noticed. These findings are in concurrence with (Daryoush *et al.,* 2011; Nazir *et al*., 2012; Kumari *et al.,* 2013; Muna *et al.,* 2016 and El-Sayed *et al.,* 2017). Long *et al*. (1974) also did a pathological study on

cases of necrotic enteritis in broilers and most of the intestines examined in their study had presence of diphtheritic membrane over the affected intestinal surface which comprised of degenerated epithelial cells, erythrocytes, inflammatory cells viz., heterophils and MNCs. They also demonstrated gram positive bacteria closely associated with villous lesions. Beside, Daryoush *et al.* (2011) observed degeneration and necrosis of the villous apex, fibrin and infiltration of the inflammatory in necrotic enteritis. In present study, sub acute enteritis was characterised by infiltration of heterophils, lymphocytes and plasma cells in the lamina propria, sub mucosa while congestion and haemorrhages in serosa. These lesions were also reported by Hafeeji *et al.* (2000) and El-Sayed *et al.* (2017). Chronic enteritis was characterised by necrosis of epithelial cells of villi with hyperplasia of goblet cells, infiltration of MNCs predominantly lymphocytes, macrophages and few plasma cells in sub mucosa. Nazir *et al*. (2012) and Sharma *et al*. (2014) found identical lesions. Regeneration in intestinal sections was seen in our cases characterized by hyperplasia of epithelium of crypts as evidenced by presence of numerous mitotic figures. Similar findings were also seen by Long *et al*. (1974) while examining cases of necrotic enteritis. Our findings were strongly in agreement with study of Nunez *et al.* (2016).

5.8.2 Liver

In the present study, liver was affected with one or the other lesion in 71.83% (454/632) of the birds. Grossly, congestion, haemorrhages, necrosis, fatty changes and perihepatitis were seen in 45.15% (205/454), 23.56% (107/454), 13.87% (63/454), 39.42% (179/454) and 68.94% (313/454) cases, respectively. Microscopically, lesions consisted of congestion, haemorrhage, edema, vacuolar degeneration, necrosis, fatty changes, acute and chronic hepatitis which were seen in 74.22% (337/454), 29.07% (132/454), 58.81% (267/454), 69.82% (317/454), 60.79% (276/454), 39.42% (179/454), 77.09% (350/454) and 3.74% (17/454) cases, respectively. These findings were lower than the findings of Parimala (2003) who recorded congestion, haemorrhage, degenerative changes, necrosis, fatty changes, acute and chronic hepatitis in 4.68% (16/342), 2.34% (08/342), 19.32% (66/342), 9.94% (34/342), 4.10% (14/342), 4.10% (14/342) and 3.80% (13/342) cases, respectively El-Sayed *et al.* (2017) recorded

congestion, necrosis and hepatitis in 25.88%, 29.41%, and 20% cases of broilers. Detailed systematic studies exclusively on spontaneous liver lesions in broilers have been undertaken by other workers also (Rahamathulla, 1972; Randall *et al*., 1986; Hutchison and Riddell *et al*., 1990; Parimala, 2003 and El-Sayed *et al.*, 2017). Liver lesions have been frequently described in relation to specific poultry diseases by numerous authors. Nakamura *et al.* (1985), Pourbakhsh *et al.* (1997), Mukherjee and Khanapurkar (1994), Parimala (2003), Omer *et al*. (2010) and Bhalerao *et al.* (2013) observed similar lesions associated with colibacillosis. Nazir *et al*. (2012), Hafeeji *et al.* (2000), Kumari *et al.* (2013) and Muna *et al.* (2016) reported identical changes in chickens suffering from salmonellosis.

Grossly, congested liver was dark red in colour, enlarged and had rounded edges. In some cases, pale and mottled appearance of liver was also noticed. Haemorrhages appeared either as petechial scattered throughout the liver lobe surface or were present on particular lobes. Necrotic foci appeared as whitish circumscribed nodular areas scattered randomly over surface of liver. These findings were agreement with Chisti *et al*., 1985; Ahmed *et al*., 2009; Omer *et al*., 2010; Nazir *et al*., 2012; Kumari *et al.*, 2013; Muna *et al.*, 2016 and El-Sayed *et al.*, 2017.

In the present study, acute hepatitis characterised by congestion of central vein, dilated sinusoids, focal areas of degeneration and necrosis of hepatocytes with infiltration of heterophils. Similar to the changes observed by Hafeeji *et al.* (2000), Parimala (2003), Muna *et al.* (2016) and El-Sayed *et al*. (2017). In the present study cases of colibacillosis, fibrinous perihepatitis as described by early workers (Rahamathulla, 1972; Nakamura *et al.*, 1985; Mukherjee and Khanapurkar, 1994; Pourbakhsh *et al.*, 1997; Ito *et al.*, 2004; Bhalerao *et al.*, 2013 and El-Sayed *et al.*, 2017). Nazir *et al*. (2012) saw similar lesions associated with salmonellosis. Khan *et al*. (1995) encountered similar findings in liver in experimentally induced runting and stunting syndrome. Chronic hepatitis was characterised by degeneration, necrosis of hepatocytes and proliferation of fibrous connective tissue with predominant infiltration of MNCs. Degeneration or hyperplasia of bile duct epithelium, presence of inflammatory cells in portal triad areas was often seen.

Accordance with the findings of Rahamathulla (1972), Parimala (2003) and Nazir *et al*. (2012).

5.8.3 Gizzard and Proventriculus

In present study, proventriculus was affected in 21.86% cases. Pathological lesions in proventriculus consisted of congestion, presence of thick mucoid exudate over mucosa, haemorrhages, necrotic mucosa and infiltration of heterophils in lamina propria. Similar proventriculus lesions have been described only in relation to certain specific poultry diseases by several authors. Kumari *et al.* (2013) saw congestion, mucosal degeneration with infiltration of heterophils and lymphocytes in mucosa extending up to serosal layer in cases of salmonellosis. Nazir *et al.* (2012) observed focal infiltration of MNCs in mucosa in salmonellosis. Muna *et al*. (2016) observed sloughing of epithelial layers and necrosis in birds affected with *Salmonella* infection. Mor *et al.* (2010) recorded haemorrhages in proventriculus in birds suffering from IBD. Bhutia and Singh (2016) recorded similar findings associated with Newcastle disease. Noiva *et al.* (2015) observed degeneration and necrosis of the epithelium of the proventricular glands, glandular hyperplasia and formation of lymphoid nodules within the glandular parenchyma in transmissible viral proventriculitis associated with runting and stunting syndrome. Kutkat *et al*. (2010) and Dormitorio *et al.* (2007) observed similar lesions in experimently in broilers. Lenz *et al. (*1998) associated with reovirus infection.

In the present study, gizzard was affected in 8.86% cases. Lesions consisted of haemorrhages, ulcerations and erosions revealing the underlying mucosa and sub mucosa. Microscopically lesions were characterised by fragmentation, degeneration and necrosis of smooth muscle fibers along with infiltration of inflammatory cells mainly lymphocytes. In serosa proliferation of several congested blood vessel, edema, presence of fibrin and infiltration of heterophils and severely encroaching muscle layer was seen. Bayyari *et al.* (1995) observed gizzard erosions in experimental reproduction of proventriculitis using homogenates of proventricular tissue. Similarly, Lenz *et al*. (1998) associated with reovirus infection in broilers. Nakamura *et al*., 1999; Nakamura *et al*., 2002; Ono *et al.,* 2003 were observed similar lesions with presence of intranuclear inclusions in experimentally produced adenovirus infection. Besides, Lim *et al.* (2012)

observed similar lesions in experimentally produced adenovirus infection in layers. Itakura *et al*. (1982) observed similar lesions in experimentally fed fish diet, histamine and copper. Furthermore, there are many other conditions that can produce gizzard erosions, such as mycotoxins and vitamin deficiencies, which can result in lesions that are indistinguishable from our findings Okazaki *et al.* (1983).

5.8.4 Pancreas

In the present study, pancreas was affected in 2.84% cases. Lesions consisted of congestion, haemorrhages and sharply circumscribed erosions. Microscopically, focal pancreatitis was characterised by presence of hyperplasia of pancreatic ducts, congested blood vessels in interlobular areas along with degeneration, necrosis of acinar cells and infiltration of heterophilis. Reece *et al.* (1984) recorded similar pancreatic lesions in spontaneously occurring cases of runting and stunting syndrome and Khan *et al*. (1995) observed similar lesions in experimentally produced runting and stunting syndrome. However, Kumari *et al.* (2013) noticed similar lesions in pancreas of birds affected with salmonellosis. Lenz *et al. (*1998) noted occurrence of necrotic pancreatitis in cases of reoviral infection. In contrast, Nakamura *et al.* (2002) noticed pinpoint white foci in the pancreas and multifocal necrosis of pancreatic acinar cells with intranuclear inclusions in adenovirus infection.

4.8.5 Oesophagus

Oesophagus was found to be affected in 3% of the necropsied birds. Lesions included congestion, edema and suppurative oesophagitis. Sary *et al.* (2017) observed severe erosive and necrotic oesophagitis in cases of ILT.

Chapter-6

Summary and Conclusion

CHAPTER 6

SUMMARY AND CONCLUSION

The current study was carried out to determine etio-pathomorphology of gastrointestinal tract of broiler chickens in Jammu. For this, occurrence of gross and histopathological lesions of GIT lesions of broilers was studied. Attempts were made to identify causative organisms from these lesions. Additionally, data was collected from 200 flocks in and around Jammu for studying occurrence and mortality pattern due to various diseases and pathological conditions. Necropsy was done on representative carcasses in a total of 632 birds.

Occurrence of colibacillosis (24.16%) was maximum followed by that of IBD (7.13%), omphalitis (6.79%), salmonellosis (5.36%), non specific enteritis (4.29%), coccidiosis (2.22%), haemorrhages in proventriculus (1.67%), gout (1.44%), hepatotoxicity (1.42%), ascites (1.14), fatty liver syndrome (1.03%), caseous nodules in lungs (0.75%) and non specific liver ailments (0.50%).

The maximum mortality was caused by colibacillosis (8.33%) followed by IBD (5.97%), conditions with haemorrhages in proventriculus where Ranikhet disease or avian influenza was suspected (5.52%), omphalitis (4.54%) followed salmonellosis (3.66%), non specific enteritis (3.36%), non specific liver ailments (3.03%), hepatotoxicity (2.95%), caseous nodules in lungs (2.00%), coccidiosis (1.69%), ascites (1.12%), gout (0.77%) and fatty liver syndrome (0.41%).

In colibacillosis grossly, fibrinous pericarditis, airsacculitis and perihepatitis was present. Microscopic examination revealed degeneration, necrosis of hepatocytes and presence of fibrin strands admixed with heterophils. Enteritis was characterised by congestion, necrosis of villi and infiltration of heterophils in lamina propria and sub mucosa. Occasionally serosal layer also had deposition of fibrinous exudate. *E.coli* isolates were Gram –ve, formed pink colonies when cultured on MLA and green metallic sheen on EMB agar. Biochemically, all *E.coli* isolates were Indole+, MR+, VP-, Citrate- and belonged to serogroups O1, O22, O37, O114, O118 and O149.

In omphalitis, affected birds had flabby and pendulous abdomen. Necropsy revealed presence of cheesy discoloured yolk material in the abdomen. Other prominent changes included congestion, haemorrhages, degeneration, and necrosis of hepatocytes and acute enteritis characterised by presence of inflammatory cells such as lymphocytes and heterophils in lamina propria.

Salmonella sp. affected birds were dull, depressed and diarrhoreic. Grossly, congestion, bronze discoloration and multiple small necrotic foci were observed in liver. Intestine was severely congested. Histopathologically, characteristic changes were mild to severe vacuolar degeneration and multifocal areas of coagulative necrosis along with congestion and haemorrhages. Morphologically, *Salmonella* isolates formed yellow colonies on MLA. Biochemically, all *Salmonella* isolates were Indole-, MR+, VP-, Citrate+.

The suspected cases of IBD, had paint brush haemorrhages on thigh and breast muscles along with enlarged, haemorrhagic and edematous bursa. In addition, there was degeneration, necrosis of lymphocytes, presence of cystic cavities in bursal follicles and infiltration of inflammatory cells in interfollicular areas. Congestion, haemorrhages and degenerative changes were also evident in kidneys and liver.

In cases of coccidiosis, haemorrhages in the small intestine and caecum were found. Additionally, degenerating and necrotic intestinal epithelium harboring coccidian life-cycle stages along with infiltration of heterophils were observed up on microscopic examination. Examination of wet mount smears prepared from intestinal scrapings revealed presence of *Eimeria* oocysts.

Birds suffering from ascites had clear straw colored fluid in the abdomen. Liver was enlarged and pale in colour. Microscopically, vacuolar degeneration, fatty changes, necrosis of hepatocytes and infiltration of heterophils were observed in liver section.

In suspected cases of nephro-hepatotoxicity, kidneys were pale, swollen, congested and had urate crystal deposition. Severe haemorrhages and edema in intertubular areas, degeneration and coagulative necrosis of tubular epithelium were seen.

Liver was pale, mottled or congested and microscopically revealed degeneration, necrosis and infiltration of inflammatory cells chiefly heterophils.

Cases of fatty liver syndrome revealed pale and friable liver. Microscopically, hepatocytes showed presence of fat vacuoles in the cytoplasm giving signet ring appearance. Individualization, disassociation and necrosis of hepatocytes was observed at multifocal areas with mild heterophilic infiltration. Some cases had haemorhages in abdominal cavity associated with liver rupture.

Detailed analysis of pathomorphological lesions occurring in GIT of birds was done. Intestine was affected in 80.22% cases. Grossly the lesions consisted of congestion, haemorrhages, necrosis and enteritis in 40.43%, 21.10%, 12.42% and 68.44% cases respectively. Microscopically congestion, haemorrhage, edema, degeneration, necrosis, acute, sub acute, chronic enteritis occurred in 76.72%, 57.79%, 52.66%, 90.33%, 76.72%, 69.03%, 2.36% and 3.35% cases respectively.

Examination of intestinal sections revealed congestion, haemorrhages in lamina propria, necrosis of epithelium, hyperplasia of goblet cells, edema and inflammatory cells mainly heterophils in acute catarrhal enteritis. Necrotic enteritis characterised by severe necrosis of villous epithelium, presence of fibrino-necrotic exudate and infiltration of heterophils in large numbers was seen. Sub acute enteritis was characterised by thickened mucosal wall due to infiltration of heterophils, lymphocytes and plasma cells in the lamina propria and sub mucosa. Chronic enteritis was characterised by infiltration of MNC's predominantly lymphocytes, macrophages and few plasma cells in sub mucosa. Regeneration characterised by hyperplasia of crypt epithelium and presence of numerous mitotic figures was also seen.

Liver was affected with one or the other lesion in 71.83% birds. Grossly, congestion, haemorrhages, necrosis, fatty changes and perihepatitis were seen in 45.15%, 23.56%, 13.87%, 39.42% and 68.94% cases respectively. Microscopically, lesions consisted of congestion, haemorrhage, edema, vacuolar degeneration, necrosis, fatty degeneration, acute and chronic hepatitis which were seen in 74.22%, 29.07%, 58.81%, 69.82%, 60.79%, 39.42%, 77.09% and 3.74% cases respectively.

Congestion of central veins and sinusoids; dissociation, individualization and coagulative necrosis of hepatocytes with heterophilic infiltration was seen in acute hepatitis. Suppurative hepatitis was characterised by infiltration of large number of heterophils in the hepatic parenchyma. In cases of chronic hepatitis, degeneration, necrosis of hepatocytes, proliferation of fibrous connective tissue in the portal triad areas was observed. Hepatocholangitis with necrosis and inflammation of liver parenchyma; degeneration, necrosis and hyperplasia of epithelium of bile duct with presence of heterophils and lymphocytes in portal triad areas were seen.

Proventriculus was affected in 21.86% cases. Grossly, congestion, haemorrhages, edema and necrosis was seen in 36.29%, 51.11%, 85.92% and 12.59% cases respectively. Microscopically, lesions consisted of congestion, haemorrhages, edema, degeneration, necrosis and proventriculitis visualized in 56.29%, 42.22%, 97.77%, 77.77%, 77.03% and 95.55% cases. Detailed examination revealed degeneration and necrosis of epithelium of mucosal folds and sub mucosal proventricular glands, congestion, haemorrhages and infiltration of heterophils in lamina propria and sub mucosa. In chronic cases, blunted, fused mucosal folds, proliferation of fibrous connective tissue along with infiltration of MNCs was seen.

Gizzard was affected in 8.86% cases. Grossly, haemorrhages and necrosis were seen in 28.57% and 57.14% cases respectively. Microscopically, lesions consisted of congestion, haemorrhages, edema, degeneration, necrosis and ventriculitis as observed in 41.07%, 33.92%, 60.71%, 62.5%, 76.78% and 64.28% carcasses. Other changes were degeneration, fragmentation and necrosis of smooth muscle fibers and infiltration of inflammatory cells. In serosa, proliferation of congested blood vessels, edema, presence of fibrinous exudate and infiltration of heterophils was seen in some cases.

Pancreas was affected in 2.84% cases. Grossly, congestion, haemorrhages and necrosis were seen in 50%, 27.77% and 22.22% cases respectively. Microscopically, congestion, haemorrhages, edema, degeneration, necrosis and pancreatitis was seen in 33.33%, 16.66%, 38.88%, 83.33%, 61.11% and 44.44% cases respectively.

Oesophagus was found to be affected in 3% of the necropsied birds. Lesions in oesophagus included congestion, haemorrhage, edema, degeneration, necrosis and oesophagitis in 63.15%, 21.05%, 42.10%, 78.94%, 84.21% and 26.31% birds respectively.

From present investigation it is concluded that:-

1. Colibacillosis was most common disease in broiler chickens affecting all age groups and prevalent in all seasons with an occurrence of 24.16% and caused 8.33% mortality.

2. Other diseases or pathological conditions affecting the broilers were IBD, omphalitis, salmonellosis, non specific enteritis, coccidiosis, haemorrhages in proventriculus (NCD/Avian influenza suspected), gout, nephro-hepatotoxicity, ascites, fatty liver syndrome, presence of caseous nodules in lungs and non specific liver ailments.

3. Gastrointestinal tract was found to be affected in almost all the necropsied birds.

4. A variety of lesions including congestion, haemorrhages, edema, degeneration, necrosis, acute and chronic inflammation were found in intestine, liver, proventriculus, gizzard, pancreas and oesophagus.

5. Implementations of better managemental practices are recommended to minimize disease occurrence and the consequent economic losses due to morbidity and mortality in broiler birds. The information generated in present study should serve as base line data for future studies to determine molecular prevalence of various diseases in broilers of Jammu.

References

REFERENCES

Abdeltawab, A.A., Abdelaal, S.A., Mazied, E.M. and El-Morsy, D.A. 2015. Prevalence of *E.coli* in broiler chickens in winter and summer seasons by application of PCR with its antibiogram pattern. *Benha Veterinary Medical Journal*, **29(**2): 253-261.

Ahad, S., Syed, T. and Malik, T.A. 2015. Seasonal impact on the prevalence of coccidian infection in broiler chicks across poultry farms in the Kashmir valley. *Journal of Parasitic Disease*s, **39**(4): 736-740.

Ahmed, I., Anjum, M.S. and Hanif, M. 2012. Prevalence of poultry diseases at high altitudes of district Poonch Azad Jammu & Kashmir. *Pakistan Journal of Science*, **64**(4): 334.

Ahmed, M.S., Sarker, A. and Rahman, M.M. 2009. Prevalence of infectious diseases of broiler chickens in Gazipur district. *Bangladesh Journal of Veterinary Medicine*, **7(**2): 326-331.

Ali, A.Z.R. and Ali, A.S.AL-Mayah. 2015. Isolation of pathogenic *Escherichia coli* O78:K80 serotypes from broiler chicks with spontaneous pathological conditions in Basra Province. *Kufa Journal for Veterinary Medical Sciences*, **6**(1): 109-117.

Al-sadi, H.I., Shareef, A.M. and Al-Attar, M.Y. 2000. Outbreaks of aflatoxicosis in broilers. *Iraqi Journal of Veterinary Sciences*, **13**(1): 93-106.

Anjaneyulu, Y., Babu, N.S. and James, R.M. 1998. Mortality pattern in broilers in Prakasam district (Andhra Pradesh). *Indian Journal of Veterinary Pathology*, **22**(1): 44-46.

Anjum, R., Javed, M.T. and Khan, A. 1998. Pathophysiology of ascites syndrome in broiler chickens during winter under local conditions. *Pakistan Veterinary Journal*, **18**(2): 68-73.

Arif, M., Rind, R.U., Shah, M.G., Nisha, A.R., Umer, M., Kaka, U., Zaman, A., Tariq, M., Reehman, S.A., Hasan, S.M. and Khan, M.S. 2015. Seroprevalence of avian

influenza in broilers of District Quetta, Balochistan, Pakistan. *Journal of chemical and Pharmaceutical Research*, **7(**4): 1378-1384.

Arora, D., Kumar, S., Jindal, N., Narang, G., Kapoor, P.K. and Mahajan, N.K. 2015. Prevalence and epidemiology of *Salmonella* enteric serovar Gallinarum from poultry in some parts of Haryana, India. *Veterinary World*, **8(**11): 1300-1304.

Asim, A., Khan, K.N.M, Cheema, A.H., Mir, F.A. and Afzal, M. 1990. Occurrence of aflatoxins in poultry liver and associated pathological changes. *Pakistan Veterinary Journal*, **10**:51-54.

Babaca, Z.A.J. 2015. Outbreak prevalence of Newcastle disease in Erbil and surrounding areas (Iraq). *Veterinary Science Development*, **5**: 7-9.

Bakshi, C.S., Sikdar, A. and Chattopadhyay, S.K. 1995. Experimental aflatoxicosis in commercial broilers: Pathomorphological studies. *Indian Journal of Veterinary Pathology*, **19**:112-115.

Balani, D.K. 1983. Incidence of various poultry diseases in Rhotak and Sonepat districts (Haryana). *Poultry Advisor*, **26(**5): 21-23.

Balasubramaniam, A. and Dorairajan, N. 2009. Influence of season on occurrence of poultry diseases in Namakkal region of South India, *Indian Journal of Field Veterinarians*, **4**(4): 27-29.

Baliarsingh, S.K., Rao, A.G. and Mishra, P.R. 1993. Pathology of experimental colibacillosis in chicks. *Indian Veterinary Journal*, **70:** 808-812.

Barbour , E.K., Jurdi, L.H., T alhouk, R., Q atanani, M., Eid, A., Sakr, W., Bouljihad, M. and Pasojevicr, S. 1999. Emergence of *Salmonella* Enteritidis outbreaks in broiler chickens in the Lebanon: epidemiological markers and competitive exclusion control. *Revue scientifique Technique (International Office of Epizootics)*,**18** (3): 710-718.

Bayyari, G.R., Huff, W.E., Balog, J.M., Rath, N.C. and Beasley, J.N. 1995. Experimental reproduction of proventriculitis using homogenates of proventricular tissue. *Poultry Science*, **74**:1799-1809.

Bhalerao, A.K.D., Gupta, R.P. and Kumari, M. 2013. Pathological studies on natural cases on avian colibacillosis in Haryana state. *Haryana Veterinarian,* **52:** 118-120.

Bhatia, K.C., Sharma, V.K. and Singh, N. 1972. Studies on persistant yolk sac condition in chicks. *Indian Journal of Animal Health,* **11**: 173-176.

Bhattacharyya, H.M., Chakraborty, G.C., Chakraborty, D., Battacharyya, D., Goswami, U.N. and Chatterjee, A. 1984. Broiler chick mortality due to pullorum disease and brooder pneumonia in West Bengal. *Indian Journal of Animal Health,* **23**(1): 85-88.

Bhutia, L.D. and Singh, Y.D. 2016. Pathological studies on viral diseases of poultry in Mizoram, India. *Veterinary Sciences*, **6(**9): 606-610.

Biswas, N.K., Dalapati, M.R. and Bhowmik, M.K. 1995. Ascites syndrome in broiler chicken: Observations on certain biochemical and pathological changes. *Indian Journal of Animal Sciences*, **65:** 1068-1072.

Borah, M.K., Islam, R., Sarma, M., Mahanta, J.D. and Kalita, N. 2017. Prevalence and seasonal variation of certain microbial diseases in Kamrup and Kamrup (Metro) Districts of Assam. *International Journal of Chemical Studies,* **5**(3): 724-726.

Brar, R.S., Leishangthem, G.D., Gadhave, P.D., Singh, N.D., Banga, H.S., Mahajan, V. and Sodhi, S. 2017. Diagnosis of Newcastle disease in broiler by histopathology and immunohistochemistry. *Indian Journal of Veterinary Pathology*, **41**(1): 60-62.

Buragohain, R. and Kalita, G. 2010. Assessment of mortality pattern of broiler under intensive system of management in Mizoram. *Tamilnadu Journal of Veterinary & Animal Sciences,* **6**(5): 239-241.

Capua, I., Pozza, D.M., Mulinelli, F., Marangon, S. and Terregino, C. 2002. Newcastle disease outbreaks in Italy during 2000. *Veterinary Record*, **150**:565-568.

Carter, G.R., Quinn, P.J., Carter, M.E. and Markey, B. 1994. *Clinical Veterinary Microbiology*. pp. 209-236.

Chakraborty, D., Pramanik, A.K. and Naudi, S.N. 1982. Investigation of brooder chick mortality in and around Calcutta. *Indian Journal of Animal Health*, **21**(1): 65-67.

Chanie, M., Negash, T. and Tilahun, S.B. 2009. Occurrence of concurrent infectious diseases in broiler chickens is a threat to commercial poultry farms in Central Ethiopia. *Tropical Animal Health Production*, **41:** 1309-1317.

Chaudhury, B. and Kwatra, M.S. 1977. Studies on the prevalence and pathology of respiratory mycosis in chickens in Assam. *Indian Veterinary Journal*, **54**(10): 791-794.

Chauhan, H.V.S., Jha, G.J., Singh, M.P. and Thakur, H.N. 1980. Outbreaks of infectious bursal disease (Gumboro disease) in poultry. *Indian Journal of Poultry Science*, **15**: 253-258.

Chishti, M.A., Khan, M.Z. and Irfan, M. 1985. Pathology of liver and spleen in avian salmonellosis. *Pakistan Veterinary Journal*, **5** (4): 157-160.

Choudhary, U.K., Tiwary, B.K., Prasad, A. and Ganguly, S. 2012. Study on incidence of infectious bursal disease in and around Ranchi. *Indian Journal of Animal Research*, **46** (2): 156 – 159.

Chowdhury, S., Masuduzzaman, M. and Shatu, S.N. 2009. A pathological investigation to identify different forms of colibacillosis in commercial broiler and layer birds in Chittagong region. *Eco-friendly agricultural Journal*, **2**(1): 368-373.

Coello, C.L., Odem, T.W. and Wideman, R.F. 1985. Ascites major cause of mortality in broilers. *Poultry Digest*, **44**:284-286.

Cruickshank, R., Duguid, J.P., Marsion, B., Pand Swain, R.H.A. 1975. In: Medical-Microbiology Vol II 12th ed. IBM, New Delhi.

Daryoush, M., Yousef, D. and Mehrdad, N. 2011. Histopathological study on poultry enteritis in Azerbaijan Province of Iran. *International Journal of Poultry Science*, **10**(11): 886-890.

Datta, S., Rakha, N.K., Narang, G. and Mahajan, N.K. 2013. Epidemiology of enteritis in broiler chickens in the state of Haryana and its association with different meterological parameters. *Haryana Veterinarian*, **52:** 22-25.

Davis, D.C., Abraham, M.J., Lalithakunjamma, C.R., Nair, N.D. and Vijayan, N. 2012. Nutritional and metabolic diseases associated with hepato-renal pathology in chicken. *Indian Journal of Animal Research*, **46**(4): 397-400.

Dhaliwal, A.S. 1989. Clinico-pathological studies of respiratory system of domestic fowl. M.V.Sc. thesis, submitted to Punjab Agricultural University, Ludhiana, India.

Dhillon, N.K. 1992. Therapy of *Eimeria tenella* in poultry. M.V.Sc. thesis submitted to Punjab Agricultural University, Ludhiana.

Dolka., Sapierzyński, R., Bielecki, W., Malicka, E., Żbikowski, A. and Szeleszczuk, P. 2012. Histopathology in diagnosis of broiler chicken and layer diseases – review of cases 1999-2010. *Journal of Veterinary Sciences*, **15**(4): 773-779.

Dormitorio, T.V., Giambrone, J.J. and Hoerr, F.J. 2007. Transmissible proventriculitis in broilers. *Avian Pathology*, **36(**2): 87-91.

Dutta, P., Borah, M.K., Gangil, R. and Singathia, R. 2015. Gross/histopathological impact of *Salmonella Gallinarum* isolated from layer chickens in Jaipur and their antibiogram assay. *International Journal of Advanced Veterinary Science and Technology*, **4**(1): 153-159.

El-Sayed, N.M., Oda, S.S., Tohamy, H.G. and El- Manakhly, M.E.S. 2017. Pathologic study on the enterohepatic affections in chickens at Alexandria Province, Egypt. *Advances in Animal and Veterinary Sciences,* **5**(1):30-38.

Elwinger, K., Schneitz, C., Berndtson, E., Fossum, O., Teglof, B. and Engstrom, B. 1992. Factors affecting the incidence of necrotic enteritis, caecal carriage of *Clostridium perfringens* and bird performance in broiler chicks. *Acta Veterinaria Scandinavica,* **33**: 369-378.

Farooq, M., Durrani, F.R., Imran, N., Durrani, Z. and Chand, N. 2003. Prevalence and economic losses due to infectious bursal disease in broilers in Mirpur and Kotli districts of Kashmir. *International Journal of Poultry Sciences,* **2(**4): 267-270.

Ficken, M.D. and Wages, D.P. 1997. Necrotic enteritis. In: *Diseases of Poultry*, 10th. (B.W. Calnek, Ed). Iowa State University Press, Ames, Iowa, pp: 261-264.

Franciosini, M.P., Tacconi, G. and Leonardi, L. 2012. Ascites syndrome in broiler chickens. *Veterinary Science Research,* **3**(1): 60-66.

Ganesh, K. and Raghavan, R. 2000. Hydropericardium hepatitis syndrome of broiler poultry: current status of research. *Research in Veterinary Science,* **68**: 201-206.

Gharekhani, J., Sadeghi-Dehkordi, Z. and Bahrami, M. 2014. Prevalence of coccidiosis in broiler chicken farms in Western Iran. *Journal of Veterinary Mediciene,* 1-4.

Ghodasara, D. J., Joshi, B. P., Jani, B., Purnima, Gangopadhya, R. M. and Prajapati, K. S. 1992. Pattern of mortality in chickens. *Indian Veterinary Journal,* **6**(9): 888-890.

Ghosh, R.C., Hirpurkar, D.D. and Suryawnshi, P.R. 2006. Concurrent colibacillosis and infectious bursal disease in broiler chicks. *Indian Veterinary Journal,* **83**: 1019-1020.

Ghosh, S.S. 1987. Occurrence of *E.coli* serotypes in chicks in Nagaland. *Indian Veterinary Journal*, **64**: 464-466.

Gill, B.S., Mangat, A.P.S. and Kwatra, M.S. 1988. An outbreak of infectious bursal disease in Punjab. *Poultry Guide,* **7**: 73-75.

Goodwin, M.A., Davis, J.F., McNulty, M.S., Brown, J. And Player, E.C. 1993. Enteritis (so called runting stunting syndrome) in Georgia broiler chicks. *Avian Diseases*, **37**: 451-458.

Goyal, D. 2004. Prevalence and pathology of hepatic lesions associated with diseases of poultry and quail. M.V.Sc. Thesis. Punjab Agricultural University, Ludhiana, India.

Gross, W.B. 1964. Retained caseous yolk sacs caused by *Escherichia coli*. *Avian disease*, **8**: 438-441.

Gupta, S., Choudhury, M.A., Yadav, J.N.S., Srivastava, V. and Tandan, J.S. 1990. Anti-diarrhoeal activity of diterpenes of Andrographispaniculata (Kalmegh) against *Escherichia coli* enterotoxin in in vivo models. *International Journal of Crude Drug Research,* **28:** 273-283.

Gurel, A., Kuscu, B., Arun,S.S. and Yesildere,T. 2003. Studies on diagnosis of spontaneous Gumboro Disease (IBD) by histopathological and immunoperoxidase methods. *Arch Geflugelk*, **67**(3): 131 – 137.

Habib-ur-Rehman, Khan, A. and Khan, M.Z. 1999. Clinical, gross and histopathological observation in spontaneous cases of ascites syndrome in broiler chickens reared at low altitude. *Pakistan Veterinary Journal*, **19**(3): 115-118.

Hadipour, M.M., Habibi, G.H., Golchin, P., Hadipourfard, M.R. and Shayanpour, N. 2011. The Role of Avian Influenza, Newcastle Disease and Infectious Bronchitis Viruses during the Respiratory Disease Outbreak in Commercial Broiler Farms of Iran. *International Journal of Animal Veterinary Advances,* **3(**2): 69-72.

Hafeeji, Y.A., Joshi, B.P., Prajapati, K.S., Dave, C.J., Ghodasara, D.J. and Roy, A. 2000. Aetiopathological investigation of *Salmonella gallinarum* infection in broilers. *Indian Journal of Veterinary Pathology*, **24**: 119-120.

Hasan, R.A.K.M., Ali, M.H., Siddique, M.P., Rahman, M.M. and Islam, M.A. 2010a. Clinical and laboratory diagnosis of common bacterial diseases of broiler and layer chickens. *Bangladesh Journal of Veterinary Mediciene*, **8**(2): 107-115.

Hasan, R.A.K.M., Ali, M.H., Siddique, M.P., Rahman, M.M. and Islam, M.A. 2010b. Clinical and laboratory diagnosis of Newcastle and Infectious Bursal Diseases of chickens. *Bangladesh Journal of Veterinary Medicine*, **8**(2): 131-140.

Hermans, P. G. and Morgan, K. L. 2007. Prevalence and associated risk factors of necrotic enteritis on broiler farms in the United Kingdom; A cross-sectional survey. *Avian Pathology*, **36**(1): 43-51.

Hutchison, T.W.S. and Riddell, C. 1990. A study of hepatic lesions in broiler chickens at processing plants in Saskatchewan. *Canadian Veterinary Journal*, **31**:20-25.

Islam, M.N., Rashid, S.M.H., Hoque, M.F., Juli, M.S.B. and Khatun, M. 2008. Pathogenicity of IBDV related to outbreaks in the vaccinated flocks and the causes of vaccination failure. *Journal of Innovation and Development Strategy*, **2**: 22-30.

Islam, M.R., Das, B.C., Hossain, K., Lucky, N.S. and Mostafa, M.G. 2003. A study on the occurrence of poultry diseases in Sylhet region of Bangladesh. *International Journal of Poultry Science*, **2**(5): 354-356.

Itakura, C., Hakotani, Y. and Goto, M. 1982. Comparative pathology of gizzard lesions in broiler chicks fed fish meal, histamine and copper. *Avian Pathology*, **11:** 487–502.

Itakura, C., Yasuba, M. and Goto, M. 1974. Histopathological studies on inclusion body hepatitis in broiler chickens. *Japanese Journal Veterinary Science*, **36**(4): 329–340.

Ito, N.M.K., Miyaji, C.I., Lima, E.A., Okabayashi, S., Claure, R.A. and Graca, E.O. 2004. Entero-hepatic pathobiology: Histopathology and semi-quantitative bacteriology of the duodenum. *Brazilian Journal of Poultry Science,* **6**:31-40.

Itoo, F.A., Kamil, S.A., Mir, M.S., Baba, O.K., Dar, T.A. and Darzi, M.M. 2013. Occurence and pathology of diseases with associated respiratory tract affections in commercial broiler chickens reared in Kashmir. *Journal of Research,* **15(**1): 23-34.

Jadhav, B.N., Nikam, S.V., Bhamre, S.N. and Jaid, E.L. 2011. Study of *Eimeria necatrix* in broiler chicken from Aurangabad district of Maharashtra state India. *International Multidisciplinary Research Journal,* **1(**11): 11-12.

Jalob, Z.K., Farhan, W.H., Ibrahiem, Z.Y. and Jumaa, B.N. 2015. Bacteriological and pathological study of Omphalitis in broiler chicks. *Kufa Journal for Veterinary Medical Sciences,* **6(**2): 17-26.

Jana, S., Mukhopadhayay, S.K., Niyogi, D., Damodar, Y.S. and Thiyagaseelan, C. 2009. Epidemio-pathological studies of gout in broiler birds in West Bengal. *Indian Journal of Veterinary Pathology,* **33(**2): 222-224.

Javed, M. T., Anjum, R., Khan, M. Z. and Khan, A. 1991. Studies on isolation, pathogenicity and sensitivity of *E.coli* in layers and broilers. *Pakistan Veterinary Journal*, **11**(4): 187-190.

Jindal, N., Mahajan, N.K., Mittal, D., Gupta, S.L. and Khokhar, R.S. 2004. Some epidemiological studies on infectious bursal disease in broiler chickens in parts of Haryana, India. *International Journal of Poultry Science*, **3**(7): 478-482.

Jindal, N., Raja, N., Kumar, S., Narang, G. and Mahajan, N.K. 1999. *Salmonella gallinarum* and *Salmonella enteritidis* infection in poultry in some parts of Haryana. *Indian Veterinary Journal,* **76**:563-564.

Jithendran, K.P. 2001. Coccidiosis an important disease among poultry in Himachal Pradesh. *ENVIS Bulletin: Himalayan Ecology and Development*, **9**(2): 1-3.

Joshi, M. and Bhagwat, S. 1995. Incidence of fatty liver syndrome in poultry. *Indian Veterinary Journal*, **72**:1259-1261.

Kadhim, L.I. 2014. Histopathological changes of broilers immunized with sonicated oocysts against *Eimeria Tenella*. *International Journal of Advanced Biotechnology Research*, **4**(1): 31-35.

Kala, S., Gattani, A., Kumar, A. and Samantary, S., 2013. Infection of different species of *Eimerian* chicken. *Animal Science Reporter*, **7**(4): 139-145.

Kaldhusdal, M., Schneitz, C., Hofshagen, M. and Skjerve, E. 2001. Reduced incidence of *Clostridium perfringens* associated lesions and improved performance in broiler chickens treated with normal intestinal bacteria from adult fowl. *Avian Diseases*, **45**: 149-156.

Kaul, L., Kau, P.L. and Shah, N.M. 1992. An outbreak of colibacillosis in broiler chicks at an organized poultry farm under semi-arid zone of North Gujrat. *Indian Veterinary Journal*, **69**(4): 373-374.

Khaier, M.A.M., Abdelhalim, A.I., and Abukashawa, S.M.A. 2015 Isolation and Morphological identification of *Eimerica Tenella* (Family: Eimeriiddae) from Khartoum State (Sudan), *Journal of Applied and Industrial Sciences*, **3**(5): 177-181.

Khan, K.A, Khan, S.A., Aslam, A., Rabbani, M.R. and Tipu, M.A. 2004: Factors contributing to yolk retention in poultry: a review.

Khan, K.A., Khan, S.A., Hamid, S., Aslam, A. and Rabbani, M. 2002. A study on the pathogenesis of yolk retention in broiler chicks. *Pakistan Veterinary Journal*, **22(**4): 175-180.

Khan, M.Y., Arshad, M., Mahmood, M.S. and Hussain, I. 2011. Epidemiology of Newcastle disease in rural poultry in Faisalabad, Pakistan. *International Journal of Agriculture & Biology*, **13**: 491–497.

Khan, S.A., Mustafa, G., Chaudhry, R.A., Iqbal, M. and Khan, M.L. 1995. Infectious stunting syndrome of broiler chicks clinical sings and pathological lesions. **8**(1): 1-6.

Khorajiya, H.J., Pandey, S., Ghodasara, P.D., Joshi, B.P., Prajapati, K.S., Ghodasara, D.J. and Mathakiya, R.A. 2015. Patho-epidemiological study on genotype-XIII Newcastle disease virus infection in commercial vaccinated layer farms. *Veterinary World,* **8**(3): 372-381.

Kumar, A., Gabhane, G., Gogoi, D. and Raut, B. 2008. Coccidiosis in broilers: An outbreak in cold desert region. *Journal of Veterinary Parasitology*, **22**(1): 61-62.

Kumar, P., Harun-ur-Rashid, S.M., Ali, Md.H., Mobarak, H., Islam, Md. A. and Haydar, R. 2016. Prevalence and pathology of Newcastle disease in broiler at Bochaganj Upazila of Dinajpur, Bangladesh. *Asian Journal of Medical and Biological Research,* **2**(2): 352-356.

Kumar, R. and Balachandran, C. 2009. Histopathological changes in broiler chickens fed aflatoxin and cyclopiazonic acid. *Veterinarski Arhiv,* **79**(1): 31-40.

Kumar, T., Mahajan, N.K. and Rakha, N.K. 2010. Epidemiology of fowl typhoid in Haryana, India. *World Poultry Science*, **66**(3): 503-510.

Kumar, T., Prathap, S.M., Satyanarayana, M.L., Mallikarjuna, A.R. and Shivakumar, R., 2008. Serum biochemistry and epidemiology of gout in broiler chicks. *Indian Veterinary Journal*, **85**(4): 452-453.

Kumari, D., Mishra, S.K. and Lather, D. 2013. Pathomicrobial studies on *Salmonella Gallinarum* infection in broiler chickens. *Veterinary World,* **6**(10): 725-729.

Kutkat, M.A., Ahmed, H.M., Khalil, S.A., Abd El-Fatah, M. and Torky, H.A. 2010. Studies on proventriculitis in broilers with molecular characterization to its viral causes. *Journal of American Sciences,* **6**(9): 582-592.

Lalrintluanga, C. and Baruah, G.H. 1993. Mortality pattern in broilers in Assam. *Indian Journal of Veterinary Pathology*, **17**(2): 126-128.

Lalrintluanga, C. and Baruah, G.K. 1997. Aflatoxicosis in broiler chickens. *Indian Journal of Veterinary Pathology*, **21**: 155-156.

Lee, M.D., J. Lu, U. Idris, B. Harmon, C. Hofacre and J. Maure, 2002. Microbial dynamic of the broiler intestinal tract. The Elan co Global Enteritis symposium, pp: A1-A7.

Lenz, S.D., Hoerr, F.J., Ellis, A.C., Toivio-Kinnucan, M.A. and Yu, M. 1998. Gastrointestinal pathogenicity of adenoviruses and reoviruses isolated from broiler chickens in Alabama. *Journal of veterinary Diagnostic Investigation*, **10**: 145–151.

Lillehoj, H.S., Min, W. and Dalloul, R.A. 2004. Recent progress on the cytokine regulation of intestinal immune responses to *Eimeria. Poultry Science,* **83**: 611–662.

Lim, T.H., Kim, B.Y., Kim, M.S., Jang, J.H., Lee, D.H., Kwon, Y.K., Lee, J.B., Park, S.Y., Choi, I.S. and Song, C.S. 2012. Outbreak of gizzard erosions with fowl adenovirus infections in Korea. *Poultry Science*, **91**: 1113-1117.

Livestock census-2012. All India Report. Ministry of Agricultural Department of Animal Husbandry, Dairying and Fisheries. Krishi Bhawan, New Delhi.

Lobago, F., Worku, N. and Wossene, A. 2005. Study on Coccidiosis in Kombolcha poultry farm, Ethiopia. *Tropical Animal Health and Production,* **37**: 245-251.

Long, J.R. 1973. Necrotic enteritis in broiler chickens: A review of the literature and the prevalence of the disease in Ontario. *Canadian Journal of Comparative Medicine,* **37**: 302-308.

Long, J.R., Pettit, J.R. and Barnum D.A. 1974. Necrotic enteritis in broiler chickens pathology and proposed pathogenesis. *Candian Journal of Comparative medicine,* **38**: 467-474.

Lonkar, P. S. and Prasad, M.C. 1988. Fatty liver syndrome in chickens. *Indian Journal of Veterinary Pathology*, **12**: 66-68.

Luna, L.G. 1968. Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology, (ed. 3rd). McGraw Hill Book Company, New York.

Mahajan, A., Katoch, R.C., Chahota, R., Verma, S. and Manuja, S. 2002. Concurrent outbreak of infectious bursal disease (IBD), aflatoxicosis and secondary microbial infections in broiler chicks. *Veterinarski Arhiv*, **72(**2): 81-90.

Mahajan, N.K., Jindal, N. and Kulshreshtha, R.C. 1994. Major broiler diseases in some parts of Haryana. *Indian Journal of Animal Sciences,* **64(**11): 1118-1122.

Majid, A., Siddique, M. and Khan, A. 2000. Avian Salmonellosis: Gross and histopathological lesions. *Pakistan Veterinary*, **20**(4): 183-186.

Matin, M.A., Islam, M.A. and Khatun, M.M. 2017. Prevalence of colibacillosis in chickens in greater Mymensingh district of Bangladesh. *Veterinary World,* **10**(1): 29-33.

Mehta, R., Nambiar, R.G., Delgado, C.L. and Subramanyam, S. 2003. Annex II: Livestock industrialization project: Phase II-policy, technical and environment determinants and implications of the scaling-up of broiler and egg production in india. IFPRI-FAO Project, Food and Agricultural Organization of the United Nation, Rome, Italy, July24, 2003.

Mir, M.S., Darzi, M.M., Khan, A.A., Ganaie, N.A., Gupta, S., Nashiruddullah, N. and Kamil, S.A. 2005. Investigation of an outbreak of gout in Kashmir Favorella poultry. *Indian Journal of Veterinary Pathology*, **29**(1): 35-37.

Moorthy, A.S., Mahander, M. and Rao, P.R. 1985. Hepatopathology in experimental aflatoxicosis in chickens. *Indian Journal of Animal Science,* **55**: 629-632.

Mor, S.K., Narang, G., Jindal, N., Mahajan, N.K., Sharma, P.C. and Rakha, N. K. 2010. Epidemiological studies on infectious bursal disease in broiler chickens in Haryana, India. *International Journal of Poultry Science,* **9**(4): 395-400.

Mukherjee, S.R. and Khanapurkar, S.R. 1994. Occurrence of *Escherichia coli*, Newcastle disease virus and infectious bursal disease virus in broilers. *Indian Veterinary Journal,* **71**: 533-538.

Mukhopadhyaya, B.N. and Mishra, S.K. 1992. Incidence of colibacillosis in chicks in some poultry pockets of West Bengal. *Indian Journal of Poultry Science*, **27:** 103-107.

Muna, E.A., Salih, M.H., Zakia, A.M., Halima, M.O., Abeer, A.M., Ameera, M.M., Ali, H.A. and Idris, S.B. 2016. Pathology of broiler chicks naturally infected with *Salmonella enteridis* (*S. Enteridis*) & Salmonella typhimurium (*S. Typhimurium*) during an outbreak in Sudan. *Journal of Scientific Research & Report*, **10**(1): 1-8.

Nakamura, K., Ibaraki, Y., Mitarai, Z. and Shibahara, T. 1999. Comparative pathology of heart and liver lesions of broiler chickens that died of ascites, heart failure, and others. *Avian Diseases*, **43**(3): 526-32.

Nakamura, K., Maeda, M., Imada, Y., Imada, T. and Sato, K. 1985. Pathology of spontaneous colibacillosis in a broiler flock. *Veterinary pathology*, **22**: 592-597.

Nakamura, K., Mase, M., Yamaguchi, S., Shibahara, T. and Yuasa, N. 1999. Pathologic study of specific-pathogen-free chicks and hens inoculated with adenovirus isolated from hydropericardium syndrome. *Avian Diseases*, **43:** 414–423.

Nakamura, K., Ohyama, T., Yamada, M., Abe, T., Tanaka, H. And Mase, M. 2002. Experimental gizzard erosions in specific pathogen free chicks by serotype 1 group 1 adenovirus from broilers. *American Association of Avian Pathologists*, **46**(4): 893-900.

Nakamura, K., Tanaka, H., Mase, M., Imada, T. and Yamada, M. 2002. Pancreatic necrosis and ventricular erosion in adenovirus-associated hydropericardium syndrome of broilers. *Veterinary Pathology*, **39**:403-406.

Naphade, S.T. 2013. Studies on the incidence of infection of the disease coccidiosis in broiler chickens from in and around Aurangabad city. *Indian Journal of Science Research and Technology*, **1**(3):39-43.

Narayanan, M.S., Parthiban, M., Sathiya, P. and Kumanan, K. 2010. Molecular detection of Newcastle disease virus using Flinders Tehnology Associates–PCR Tehnology. *Journal Veterinarski Arhiv,* **80**(1): 51-60.

Nasrin, S., Islam, M.A., Khatun, M., Akhter, L. and Sultana, S. 2012. Characterization of bacteria associated with omphalitis in chicks. The *Bangladesh Veterinarian*, **29**(2): 63-68.

Nayak, N.C., Chakraborti, T. and Chakraborti, A. 1988. An outbreak of gout in poultry in West Bengal. *Indian Veterinary Journal,* **65**(12): 1080-1081.

Nazir, S., Kamil, A.S., Darzi, M.M., Mir, S.M., Nazir, K. and Amare, A. 2012. Pathology of spontaneously occuring salmonellosis in commercial broiler chickens of Kashmir Valley. *Journal of World's Poultry Research,* **2**(4): 63-69.

Nematollahi, A., Moghaddam, G.H. and Pourabad, R.F. 2009. Prevalence of *Eimeria* species among broiler chicks in Tabriz (Northwest of Iran). *Munis Entomology and Zoology Journal*, **4**(1): 53.

Noiva, R., Guy, J.S., Hauck, R. and Shivaprasad, H.L. 2015. Runting stunting syndrome associated with transmissible viral proventriculitis in broiler chickens. *Avian Diseases,* **59**(3): 384-387.

Nunez, L.F.N., Sa, L.R.M., Parra, S.H.S., Astolfi-Ferreira, C.S., Carranza, C. and Ferreira, A.J.P. 2016. Molecular detection of chicken parvovirus in broilers with enteric disorders presenting curving of duodenal loop, pancreatic atrophy and mesenteritis. *Poultry Science*, 00: 1-9.

Okazaki, T., Noguchi, T., Igarashi, K., Sakagami, Y., Seto, H., Mori, K., Naito, H., Masumura, T. and Sugahara, M. 1983. Gizzard erosion, a new toxic substance in fish meal causes severe gizzard erosion in chicks. *Agricultural and Biological Chem*istry, **47:**2949–2952.

Omer, M.M., Abusalab, S.M., Gumma, M.M., Mulla, S.A., Omer, E.A., Jeddah, I.E., AL-Hassan, A.M., Hussein, M.A. and Ahmed, A.M. 2010. Outbreak of colibacillosis

among broiler and layer flocks in intensive and semi intensive poultry farms in Kassala State, Eatern Sudan. *Asian Journal of Poultry Science*, **4**(4): 173-181.

Ono, M., Okuda, Y., Yazawa, S., Imai, Y., Shibata, I., Sato, S. and Okada, K. 2003. Adenoviral gizzard erosion in commercial broiler chickens. *Veterinary Pathology*, **40**: 294-303.

Otto, P., Liebler-Tenorio, E.M., Elschner, M., Reetz, J., Lohren, U. and Diller, R. 2006. Detection of rotaviruses and intestinal lesions in broiler chicks from flocks with runting and stunting syndrome (RSS). *Avian Disease*, **50**(3): 411-418.

Panda, D.N., Mishra, A., Mishra, S.C. and Panda, B.K. 1997. Incidence of coccidiosis of broiler birds in and around Bhubaneswar, Orissa. *Indian Veterinary Journal*, **74**(5): 430-431.

Parimala, D.M. 2003. Study on the pathology of liver lesions in chiken. M.V.Sc. Thesis. Acharya N.G. Ranga Agricultural University, Rajindranagar, Hyderabad, India.

Parthasarathy, K.R., Shakir, S.A. and Ramakrishnan, R. 1979. Liver haemorrhagic syndrome in poultry in Tamil Nadu. *Indian Journal of Veterinary Pathology*, **3**: 1-5.

Patra ,G., Rajkhowa, T.K., Ali, M.A., Tiwary, J.G. and Sailo, L. 2009. Studies on clinical, gross, histopathological and biochemical parameters in broiler birds suffered from *Eimeria necatrix* infection in Aizawl district of Mizoram, India. *International Journal of Poultry Science,* **8**(11): 1104-1106.

Patra, G., Ali, M.A., Victoria, K.H., Jonathan, L., Joy, L.K., Prava, M., Ravindran, R., Das, G. and Devi, L.I. 2010. PCR based diagnosis of *Eimeria tenella* infection in broiler chicken. *International Journal of Poultry Science*, **9**(8): 813-818.

Porter, R.E. and Long, J.R. 1998. Bacterial Enteritides of Poultry. *Poultry Science*, **77:** 1159-1165.

Pourbakhsh, S.A., Boulianne, M., Martineau-Doize-B, Dozois, C.M., Desautels, C. and Fairbrother, J.M. 1997. Dynamics of *Escherichia coli* infection in experimentally inoculated chickens. *Avian Diseases*, **41**: 221-233.

Pugashetti, B.K. and Shivakumar, M.C. 2007. Mortality incidence of various causative agents in an organised poultry farm. *Karnataka Journal of Agricultural Sciences,* **20**(1): 187-188.

Rahamathulla, P.M. 1972. Study of pathology of liver and kidney lesions in poultry. M.V.Sc., Thesis, Submitted to University of Agricultural Sciences, Hebbal, Banglore.

Rahman, M.S., Rabbani, M.G., Uddin, M.J., Chakrabartty, A. and Her, M. 2012. Prevalence of Avian Influenza and Newcastle Disease viruses in poultry in selected areas of Bangladesh using rapid antigen detection kit. *Archives of Clinical Microbiology*, **3**(1:3): 1-8.

Raina, J.S. (1989). Prevalence and pathology of mycotoxicosis in Punjab. M.V.Sc. thesis, Punjab Agricultural University, Ludhiana, India.

Rajashewar, J.J., Chandramohan, C.P., Dinakaran, A.M. and Avadayappan, T. 1992. Recent incidence of intercurrent infections of IBD and other diseases among broilers in Kanyakumari district. *Indian Veterinary Journal*, **72**(6): 650-652.

Ramdas, R.P., Balkrishna, K.G. and Govind, G. 2013. Pathological effect of low grade aflatoxicity in broilers. *The Bioscan,* **8**(3): 1115-1118.

Randall, C.J., Krikpatrick, K.S. and Pearson, D.B. 1986. Liver abnormality in broilers. *Veterinary Record*, 11(9):576.

Rao, A.G., Dehuri, P.K., Chand, S.K., Mishra, S.C., Mishra, P.K. and Das, B.C. 1985. Aflatoxicosis in broiler chickens. *Indian Journal of Poultry Science*, **20**(4): 240-244.

Rao, M.R.K.M., Chaudary, C.H. and Khan, D.I. 1982. An outbreak of acute Aspergillosis in chicks. *Indian Veterinary Journal*, **59**(4): 341-342.

Rao, T. B., Das, J. H. and Sharma, D. R., 1993. An outbreak of gout in poultry in East Godavari district, Andhra Pradesh. *Poultry Advisor*, **26**(4): 43-45.

Rathore, S., Ashok, K., Pramod, T., Nirmala, K., Mamta, M. and Prapti, N. 2013. Pathological study of infectious bursal disease in broilers of Haryana. *Journal of Immunology and Immunopathology*, **15**(1): 62.

Razmi, G.R, and Kalideri, G.A. 2000. Prevalence of subclinical coccidiosis in broiler chicken farms in the municipality of Marshhad, Khorasan, Iran. *Preventive Veterinary Medicine,* **44**(3): 247-253.

Reddy, P.R. and Reddy, A.R.M. 1991. Study on mortality pattern in broilers. *Poultry Advisor,* **24**(5): 21-24.

Reece, R.L., Hooper, P.T., Tate, F.H., Beddome, V.D., Forsyth, W.M., Scott, P.C. and Barr, D.A. 1984. Field, clinical and pathological observation of runting stunting syndrome in broilers. *Veterinary Record,* **11**(65): 483-485.

Sahoo, T.K., Sahoo, L., Sarangi, L.N., Panda, S.K. and Panda, H.K. 2012. Prevalence, isolation, characterization and antibiogram study of pathogenic Escherichia coli from different poultry farms of Odisha. *Journal Advanced Veterinary Research*, **2**: 169-172.

Saif, Y.M. 2008. Disease of Poultry. 12th Edition, Blackwell Publishing Ltd, Iowa.

Samah, E.A.S. and Ahmed, M.E. 2013. Characterization of *E.coli* associated with high mortality in Poultry flocks. *Assiut Veterinary Medical Journal,* **59**(139): 51-61.

Sarker, M., Roy, J.P. and Batabyal, K. 2013. Characterization and antibiogram of enteropathogenic *Escherichia coli* isolated from poultry. *Exploratory Animal and Medical Research*, **3**(2): 165-168.

Sary, K., Chenier, S., Gagnon, C.A., Shivaprasad, H.L., Sylvestre, D. and Boulianne, M. 2017. Esophagitis and pharyngitis associated with avian infectious laryngotracheitis in backyard chickens: Two cases. *Avian Diseases*, **61**:255–260.

Sellaoui, S., Alloui, N., Mehenaoui, S. and Djaaba, S. 2012. Evaluation of size and lesion scores of bursa cloacae in broiler flocks in Algeria. *Journal of World's Poultry Research*, **2**(2): 37-39.

Shamim, A., Ul-Hassan, M., Yousaf, A., Iqbal, Md.F., Zafar, Md.A., Siddique, R.Md. and Abubakar, M. 2015. Occurence and identification of Eimeria species in broiler rearing under traditional system. *Journal of Animal Sciences and Technology*, **57**: 41.

Shankar, T.V.S., Sharma, A. and Grover, Y.P. 2010. Studies on different virulence factors of avian pathogenic *Escherichia coli*. *Haryana Veterinarian*, **49**: 45-47.

Sharada, R., Krishnappa, G., Raghavan, R., Gowda, R.N.S. and Upendra, H.A. 1999. Isolation and serotyping of *Escherichia col*i from different pathological conditions in poultry. *Indian Journal of Poultry Science*, **34**(3): 366-369.

Sharma, S., Asrani, R.K., Singh, G., Gulati, B.R., Patil, P.K. and Gupta,V. K. 2014. Outbreak of hydropericardium syndrome associated with ascites and liver rupture in caged broilers. *Veterinary Research International*, **2** (2): 33-45.

Sharma, S., Iqbal, A., Azmi, S. and Shah, H.A. 2013. Study of poultry coccidiosis in organized and backyard farms of Jammu region. *Veterinary World*, **6**(8): 467-469.

Sharma, S., Iqbal, A., Azmi, S., Mushtaq, I., Wani, Z.A. and Ahmad, S. 2015. Prevalence of poultry coccidiosis in Jammu region of Jammu & Kashmir state. *Journal of Parasitic Diseases*, **39**(1): 85-89.

Sharma, V.D., Sehi, M.S. and Joshi, H.C. 1979. Acute Aspergillosis in chicks. *Indian Veterinary Journal*, **56**(2): 151-152.

Sheriff, F.R. and Kumaran, K. 1987. A study of mortality pattern in broilers in and around Pudukkottai district of Tamil Nadu. *Poultry Guide*, **24**(3): 43-49.

Shirzad, M. R., Seifi, S., Gheisari, H. R., Hachesoo, B. A., Habibi, H. and Bujmehrani, H. 2011. Prevalence and risk for subclinical coccidiosis in broiler chicken farms in Mazandaran province, Iran. *Tropical Animal Health Production*, **43**: 1601-1604.

Singh KS. 2008. Studies on some common diseases of birds with special reference to Infectious bursal disease, Ranikhet disease, Colibacillosis and Gangrenous dermatitis. Ph.D. Thesis, submitted to West Bengal University of Animal and Fishery Science, 37, 38, KB Sarani, Kolkata 00037.

Singh, G., Sharma, N.S., Jand, S.K. and Brar, R.S. 2003. Mortality pattern in broilers at selected farms in Punjab. *Journal of Research,* **40**(3): 452-455.

Singh, H., Sandhu, B.S. and Singh, B. 1994. Prevalence and pathology of mycotoxicosis in poultry. *Indian Journal of Poultry Science*, **29**: 101-103.

Singh, J., Banga, H.S., Brar, R.S., Singh, N.D., Sodhi, S. and Leishangthem, G.D. 2015. Histopathological and immunohistochemical diagnosis of infectious bursal disease in poultry birds. *Veterinary World,* **8**(11):1331-1339.

Singh, N., Ghosh, R.C. and Singh, A., 2013. Prevalence and haemato-biochemical studies on naturally occurring gout in Chhattisgarh. *Advances in Animal and Veterinary Sciences*, **1**(38): 9-11

Singh, R.P. 1998. Aetiopathological studies of chick mortality with particular reference to bacterial infections. M.V.Sc. thesis, Punjab Agricultural University, Ludhiana, India.

Sood, S., Yadav, A., Vohra, S., Katoch, R., Ahamad, D.B. and Borkatari, S. 2009. Prevalence of coccidiosis in poultry birds in R.S.Pura region, Jammu. *Veterinary Practitioner*, **10**(1): 69-70.

Soomro, N.M., Rind R., Arijo, A.G. and Soomro, S.A. 2001. Clinical, gross and histopathological studies of coccidial infection in chicken. *International Journal of Agriculture and Biology*, **3**(4): 426-427.

Srivastava, P. K. 1990. Mortality in chicks due to bacterial, viral, parasitic and neoplastic diseases- A pathomorphological study. *Indian Veterinary Journal,* **67**(12): 1095-1098.

Sultana, R., Siddique, B., Ali, R., Chaudhary, S.I. and Maqbool, A. 2012. A study on the prevalence of respiratory diseases in broiler and layer flocks in and around Lahore district. *Punjab University journal of Zoology*, **27(**1): 13-17.

Sultana, S., Rashid, S.M.H., Islam, M.N., Ali, M.H., Islam, M.M. and Azam, M.G. 2015. Pathological investigation of avian aspergillosis in commercial broiler chicken at Chittagong district. *International Journal of Innovation and Appplied Studies*, **10**(1): 366-376.

Swayne, D.E., Glisson, J.R., McDougald, L.R., Nolan, L.K., Suarez, D.L. and Nair, V. 2013. Diseases of Poultry, (Edn. 13th). A John Wiley and Sons, Inc., Publication.

Tabler, G.T., Berry, I.L. and Mendenhall, A.M. 2004. Mortality patterns associated with commercial broiler production. *Spring*, **6**: 1.

Tafti, A.K. and Karima, M.R. 2000. Morphological studies on natural ascites syndrome in broiler chickens. *Veterinarski Arhiv,* **70**(5): 239-250.

Talha, A.F.S.M., Hossain, M.M., Chowdhury, E.H., Bari, A.S.M., Islam, M.R. and Das, P.M. 2001. Poultry diseases occurring in Mymensingh district of Bangladesh. *Bangladesh Veterinary Journal*, **18**: 20-30.

Thrusfield, M. 2005. Veterinary Epidemiology, Third edition, Blackwell Science Ltd., 53–65.

Tonu, N.S., Sufian, M.A., Sarker, S., Kamal, M.M., Rahman, M.H. and Hossain, M.M. 2011. Pathological study on colibacillosis in chickens and detection of Esc*herichia coli* by PCR. *Bangladesh journal of Veterinary Medicine,* **9**(1): 17-25.

Wilson, J.B., Julian, R.J. and Barker, I.K. 1988. Lesions of right heart failure and ascites in broiler chickens. *Avian Diseases,* **32**: 246-261.

Yunus, A.W., Nasir, M.K., Aziz, T. and Bohm, J. 2009. Prevalence of poultry diseases and their interaction with mycotoxicosis in district Chakwal: 2. Effect of season and feed. *The Journal of Animal and Plant Sciences*, **19**(1): 1-5.

Zafra, R., Perez, J., Parez- Ecija, R.A., Borge, C., Bustamante, R., Carbonero, A. and Tarradas, C. 2008. Concurrent aspergillosis and ascites with high mortality in a farm of growing broiler chickens. *Avian Diseases*, **52**(4): 711-713.

CERTIFICATE–IV

Certified that all the necessary corrections as suggested by the external examiner/evaluator and the Advisory Committee have been duly incorporated in the thesis entitled **"Etio-pathomorphological studies on gastrointestinal tract of broiler chickens in Jammu"** submitted by **Miss. Navrose Sangha,** Regd. No. **J- 15- MV- 452.**

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