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Pathological study on occurrence of various lesions on organs of gastro-intestinal tract of broilers in Jammu

Navrose Sangha and Shilpa Sood

Abstract
Nearly all of the necropsied birds had digestive tract abnormalities. Numerous lesions were discovered in the oesophagus, proventriculus, gizzard, liver, pancreas and colon. These lesions included congestion, haemorrhages, edema, degeneration, necrosis and acute and chronic inflammation. In 80.22% of cases, the intestines were affected. Liver was affected with one or the other lesion in 71.83% birds while, gizzard was affected in 8.86% cases and pancreas in 2.84% of cases. Oesophagus was found to be affected in 3% of the necropsied birds. It is advised to use improved management techniques to reduce the likelihood of sickness and the resulting financial losses from morbidity and mortality in grill chickens. The data produced by this study should be used as a baseline for subsequent research to ascertain the molecular prevalence of several diseases in Jammu's broiler population.

Keywords: Broilers, gastrointestinal tract, lesions, organs

Introduction
The digestive process, assimilation and absorption of nutrients are critical tasks carried out by the gastrointestinal tract (GIT). Any anomaly in the GIT's functioning causes problems with nutrients' absorption and assimilation, which lowers growth, the feed conversion ratio and production losses. These problems have a serious negative impact on the health of the broiler farming industry (Ficken and Wages, 1997) [4]. Gastrointestinal lesions of variable severity are caused by common infectious and non-infectious disorders in chicken.
It is common everywhere throughout the world, even in India (Tonu *et al*., 2011) [20]. The lesions include perihepatitis, congestion, haemorrhages and excess mucus in the lumen of several GIT organs. Necrotic enteritis is another new problem for the broiler industry that can cause reduced weight gain and production losses because of poor digestion and absorption (Kaldhusdal *et al*., 2001) [6]. Essentially, it results in widespread villous necrosis and pseudo-membrane formation, giving the small intestine's mucosa a characterized as Turkish towel appearance. Hepatomegaly, cholangio-hepatitis and hepatic necrosis are other possible consequences. Few reports exist regarding the incidence of broiler illness in Jammu. As of yet, no comprehensive research has been done on the GIT lesions that broilers in Jammu experience.

Materials and Methods
Gross pathology
Necropsies were performed on representative carcasses from various flocks. A post-mortem analysis was conducted on 632 birds in total. 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 pathological alterations were recorded.

Histopathology
Following a comprehensive gross examination, representative sections of the oesophagus, proventriculus, gizzard, pancreas, liver and intestine each measuring less than 5 mm in thickness, were taken and placed in a 10% neutral buffered formalin solution. Following three to four days of fixation, the tissues were cleaned in xylene, dehydrated in increasing grades of ethyl alcohol, and finally embedded with melted paraffin wax. All of this was done while the tap water was running. After the paraffin blocks were ready, slices were cut using a rotating microtome at a thickness of 4-5 microns. Subsequently, the paraffin embedded sections underwent a series of procedures including deparaffinization in xylene, rehydration by a series

of descending ethyl alcohol grades and running tap water, and staining using standard haematoxylin and eosin stain (Luna, 1968) [11].

Occurrence of gross and microscopic lesions was calculated as follows:

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

Result and Discussion

Detailed study of pathological 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. In the majority of the birds, intestines were found to be affected.Occurrence of pathological lesions in intestine, liver, proventriculus, gizzard, pancreas and oesophagus were indicated in Table1, with percentage involvement respectively. Various pathological lesions observed in this investigation, along with their corresponding occurrences are detailed below.

Intestines

Of the 632 necropsied birds, 80.22% (507/632) had intestinal lesions. Gross lesions were observed in 40.43% (205/507), 21.10% (107/507), 12.42% (63/507), and 68.44% (347/507) cases, respectively. The microscopic pathological lesions, which primarily consisted of congestion, haemorrhage, edema, degeneration, necrosis, acute, subacute 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. Similary, Daryoush *et al*. (2011) [1] 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. Whereas, Lee *et al*. (2002) [21] also found that broiler chickens suffered more than others from enteritis.

In the present study, gross examination revealed thickening of intestinal wall, congestion, petechial haemorrhages on the mucosa and mucoid exudate in the lumen (Fig 1). These similar lesions of enteritis with along with ecchymotic haemorrhages were also reported by El-Sayed *et al.* (2017) [3].

In the present study, histopathological examination in cases of acute enteritis revealed thickening of intestinal wall, serosal congestion and presence of heterophils (Fig 2) along with mucosal epithelium necrosis, blunting-stunting and fusion of villi. Acute catarrhal enteritis was characterised by necrosis of epithelium, hyperplasia of goblet cells, presence of edema and inflammatory cells along with severe congestion and haemorrhages in lamina-propria. However, in necrotic enteritis, severe denudation and necrosis of intestinal epithelium extending upto the crypts along with fusion of adjacent villi was also noticed. These findings are in concurrence with Kumari *et al.*, 2013 [8]; El-Sayed *et al.*, 2017 [3]. Beside, Daryoush *et al*. (2011) [1] observed degeneration and necrosis of the villous apex, fibrin and infiltration of the inflammatory in necrotic enteritis. In the present study, less frequently chronic enteritis was found with necrosis of epithelial cells of villi, hyperplasia of goblet cells and proliferation of mononuclear cells (MNC's) and fibrous connective tissue in lamina propria. Sharma *et al*. (2014) [19] found identical lesions.

Liver

A lesion on the liver was present in 71.83% (454/632) of the birds. Grossly, congestion, haemorrhages, necrosis, fatty changes and perihepatitis were seen in45.15% (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. Detailed systematic studies exclusively on spontaneous liver lesions in broilers have been undertaken by other workers also (Parimala, 2003 and El-Sayed *et al.*, 2017) [17, 3].

In the present study, upon grossly, liver was enlarged, round-edged and dark crimson in hue. In few cases, liver was seen to have a pale and mottled appearance. Haemorrhages could be petechial, dispersed over the surface of the liver lobes (Fig 3), or localised to specific lobe sections. Multifocal necrotic areas appeared as whitish circumscribed nodular areas scattered randomly over surface of liver. These findings were agreement with Nazir *et al*., 2012 [14]; Kumari *et al.*, 2013 [8]; Muna *et al.*, 2016 [12] and El-Sayed *et al.*, 2017 [3].

In the present study, microscopically, congested liver sections had dilated and engorged central veins and sinusoids along with varying degree of degenerative changes and necrosis of hepatocytes. Necrotic hepatocytes had deep eosinophilic cytoplasm. Nuclear changes varied from pyknosis to karyorrhexis and karyolysis. 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. 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 (Fig. 4). Similar to the changes observed by Hafeeji *et al.* (2000) [5], Parimala (2003) [17], Muna *et al.* (2016) [12] and El-Sayed *et al*. (2017) [3]. In present study, chronic hepatitis was characterized 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.

Gizzard and Proventriculus

Proventriculus was impacted in 135 out of 632 cases, or 21.86%. Grossly, congestion, haemorrhages, edema and necrosis was seen as shown in Table 2. The lesions under the microscope included congestion, haemorrhages, edema, degeneration, necrosis and proventriculitis that were presented in Table 3.

Grossly, congestion in the proventriculus manifested as a dark red discoloration of the serosal or mucosal surface and bloated with thick walls. Thick mucoid exudate that adhered to the underlying congested mucosa was frequently seen in the proventricular lumen. Some birds had a build-up of thick,

persistent, white, slimy exudate in their proventriculus that adhered to the walls (Fig 5). Dormitorio *et al.* (2007) [2] observed similar lesions in experimently in broilers. Similar lesions were reported by Lenz *et al. (*1998) [9], associated with reovirus infection.

Microscopically, lamina propria haemorrhages and congestion were prevalent lesions. In several cases, accumulation of edematous fluid in sub mucosa and inter-glandular area along with proliferation of small arterioles, capillaries and infiltration of heterophils and few lymphocytes was observed (Fig 6). 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. Similar proventriculus lesions have been described only in relation to certain specific poultry diseases by several authors. Kumari *et al.* (2013) [8] saw congestion, mucosal degeneration with infiltration of heterophils and lymphocytes in mucosa extending up to serosal layer in cases of salmonellosis. Noiva *et al.* (2015) [15] 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.

Gizzard was affected in 8.86% (56/632) cases. According to Table 2, grossly, haemorrhages and necrosis were seen. Microscopically, the lesions included congestion, haemorrhages, edema, degeneration, necrosis and ventriculitis as shown in Table 3. The majority of the gross lesions in the gizzard were ulcerations and haemorrhages. Erosions appeared as a result of the koilin layer's necrosis, exposing the mucosa and submucosa underneath (Fig 7). Under the microscope, in serosal layer, proliferation of several congested blood vessels, edema, presence of fibrin and infiltration of heterophils was seen. In many casessmooth muscle fibre fragmentation, degeneration and necrosis coupled with the infiltration of inflammatory cells, primarily heterophilswas observed (Fig 8).Similarly, Ono *et al.*, 2003 [16] were observed similar lesions with presence of intranuclear inclusions in experimentally produced adenovirus infection. Besides, Lim *et al.* (2012) [10] observed similar lesions in experimentally produced adenovirus infection in layers.

Pancreas

Pancreas was affected in 2.84% (18/632) cases. Grossly, haemorrhages, necrosis and congestion were observed as presented in Table 2. Whereas, Table 3 indicates that the lesions under microscopy included congestion, haemorrhages, edema, degeneration, necrosis and pancreatitis, respectively. However, Kumari*et al.* (2013) [8] noticed similar lesions in pancreas of birds affected with salmonellosis.

The majority of gross lesions affecting the pancreas were congestions and few cases of hemorrhagic and necrotic lesions; as they manifested as dark, depressed erosions with sharp borders (Fig 9).

Microscopically, lesions consisted of severe pancreatitis, which was marked by hyperplasia of the pancreatic ducts, clogged blood vessels in the interlobular spaces, acinar cell necrosis, degeneration and a high number of heterophilis infiltrating the area (Fig 10). Khan *et al.* (1995) [7] observed similar lesions in experimentally produced runting and stunting syndrome. In contrast, Nakamura *et al.* (2002) [13] noticed pinpoint white foci in thepancreas and multifocal necrosis of pancreatic acinar cells with intranuclear inclusions in adenovirus infection.

Oesophagus

Oesophagus was found to be affected in 3% (19/632) of the necropsied birds. No gross lesions could be appreciated. Microscopic lesions as follows: congestion, haemorrhage, edema, degeneration, necrosis and oesophagitis were depicted in Table 3.

Changes observed under a microscope included suppurative oesophagitis characterised by infiltration of heterophils in sub mucosa around the oesophageal glands. Expanding micro-abscesses have occasionally been observed compressing the oesophageal glands (Fig 11). Sary *et al.* (2017) [18] observedsevere erosive and necrotic oesophagitis in cases of infectious laryngotracheitis.

Table 1: 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 2: 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 3: 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

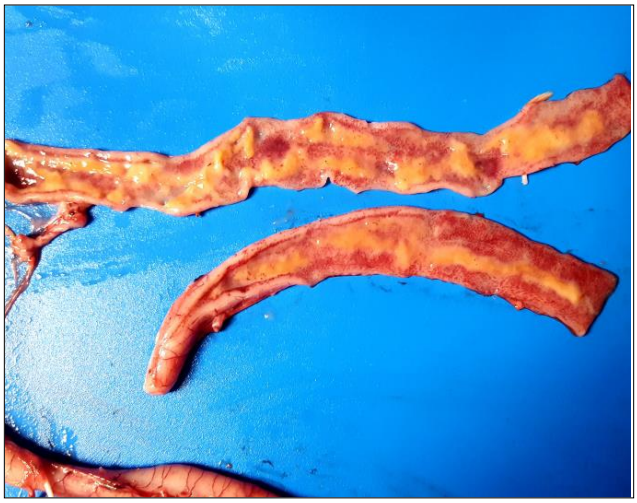

Fig 1: Presence of congestion, petechial haemorrhages with mucoid exudate in lumen of intestine

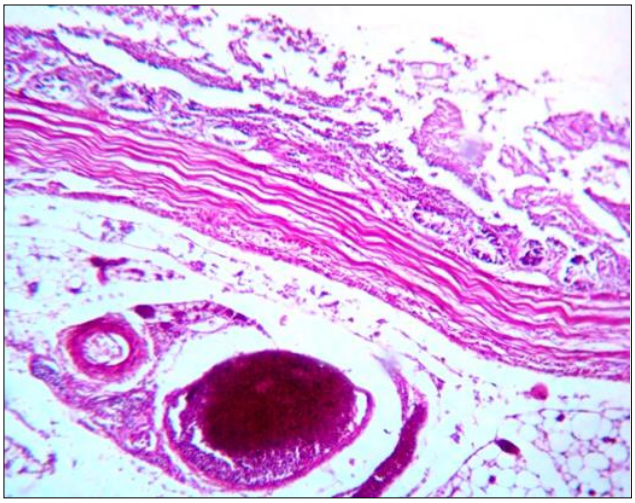

Fig 2: Severe necrosis in intestinal villi and thickening of intestinal wall due to serosal congestion. H&E X400

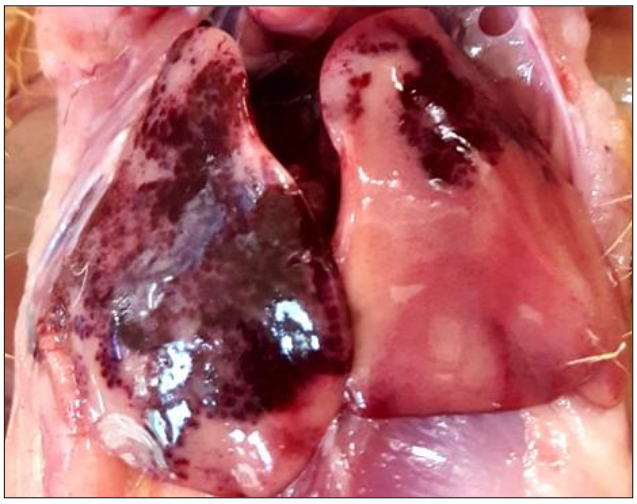

Fig 3: Liver: Petechial haemorrhages scattered throughout the surface of right lobe and involving only upper half of right left lobe

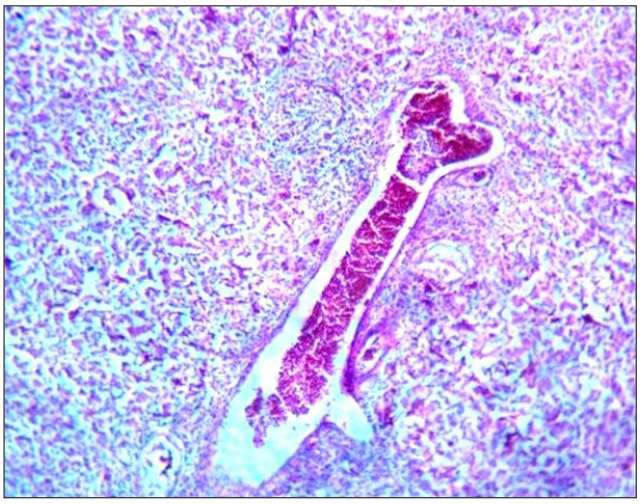

Fig 4: Liver showing oedema and inflammatory cells in portal tract. H&E X100.

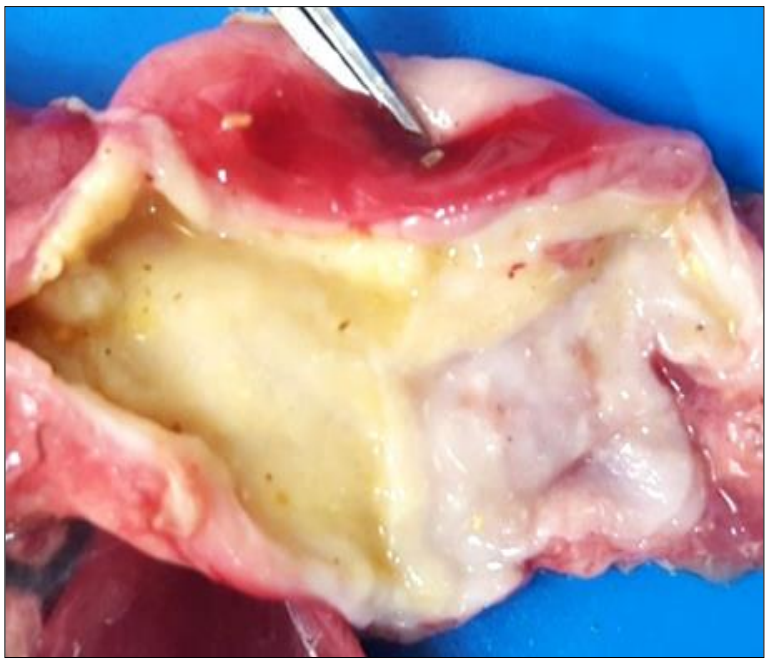

Fig 5: Accumulation of thick tenacious slimy exudate adherent to walls of proventriculus

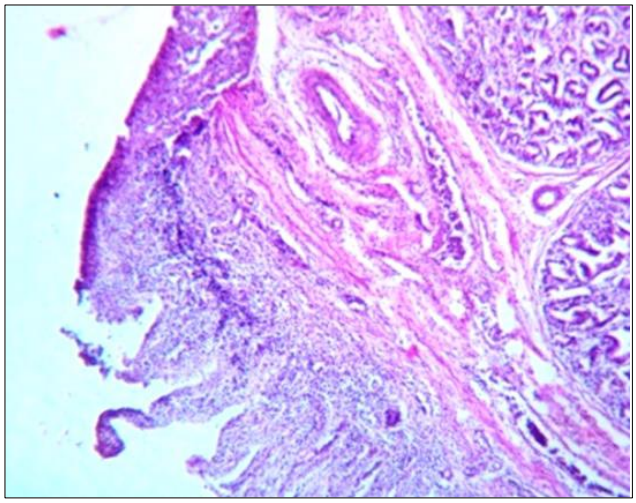

Fig 6: Proventriculus: Accumulation of edematous fluid in sub mucosa, proliferation of small arterioles and capillaries and with infiltration of inflammatory cells. H&E X100

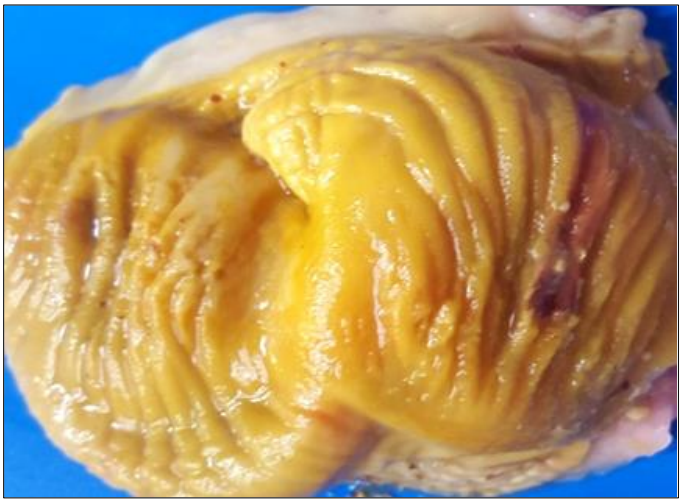

Fig 7: Gizzard showing necrosis of overlying keratinized layer revealing the underlying mucosa and sub mucosa

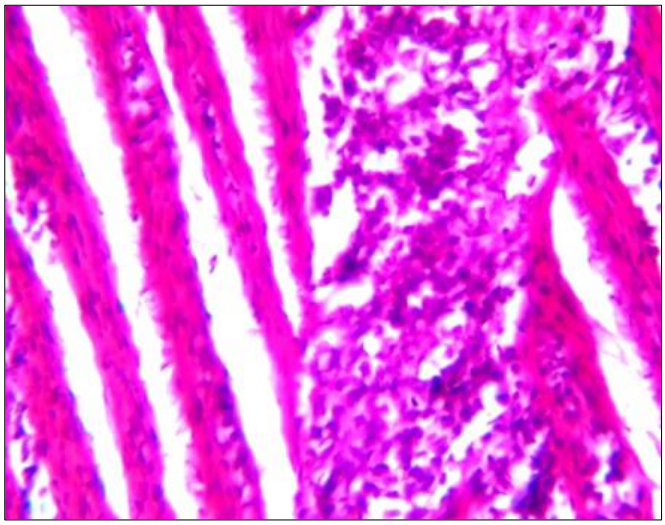

Fig 8: Gizzard showing fragmentation, degeneration and necrosis of smooth muscle fibers along with infiltration of inflammatory cells mostly heterophils. H&E X400

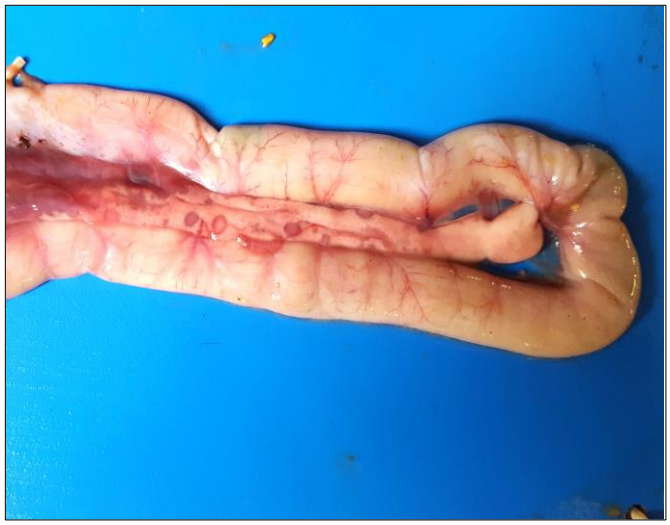

Fig 9: Presence of focal ulceration in pancreas

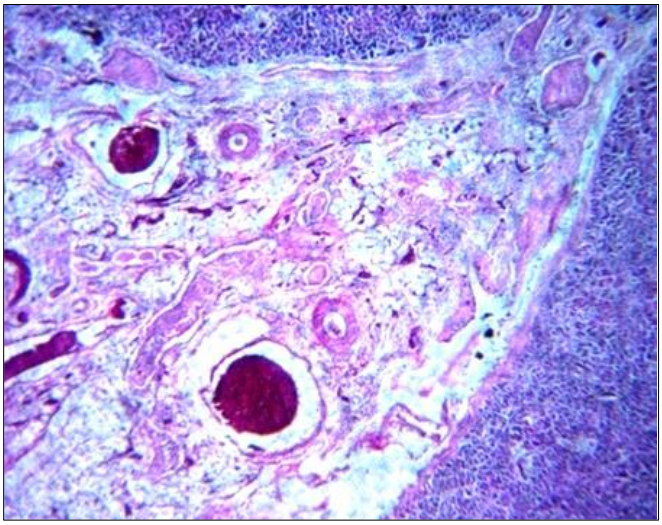

Fig 10: Pancreas showing hyperplasia of pancreatic ducts, congestion of blood vessels in interlobular areas, degeneration, necrosis of acinar cells and heterophilic infiltration. H&E X100

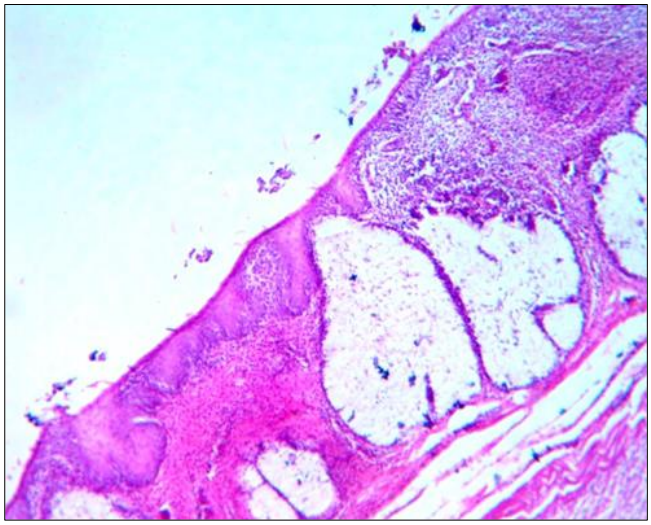

Fig 11: Oesophagus: Presence of inflammatory cells in sub mucosa around the oesophageal glands. H&E X100

Conclusion

Gastrointestinal tract was found to be affected in almost all the necropsied birds. A variety of lesions including congestion, haemorrhages, edema, degeneration, necrosis, acute and chronic inflammation were found in intestine, liver, proventriculus, gizzard, pancreas and oesophagus. 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.

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References

1. Daryoush M, Yousef D, Mehrdad N. Histopathological study on poultry enteritis in Azerbaijan Province of Iran. International Journal of Poultry Science. 2011;10(11):886-890.
2. Dormitorio TV, Giambrone JJ, Hoerr FJ. Transmissible proventriculitis in broilers. Avian Pathology. 2007;36(2):87-91.
3. El-Sayed NM, Oda SS, Tohamy HG, El-Manakhly MS. Pathologic study on the enterohepatic affections in chickens at Alexandria Province, Egypt. Advances in Animal and Veterinary Sciences. 2017;5(1):30-38.
4. Ficken MD, Wages DP. Necrotic enteritis. In: Diseases of Poultry, 10th (B.W. Calnek, Ed.). Iowa State University Press, Ames, Iowa; c1997. p. 261-264.
5. Hafeeji YA, Joshi BP, Prajapati KS, Dave CJ, Ghodasara DJ, Roy A. Aetiopathological investigation of *Salmonella gallinarum* infection in broilers. Indian Journal of Veterinary Pathology. 2000;24:119-120.
6. Kaldhusdal M, Schneitz C, Hofshagen M, Skjerve E. Reduced incidence of *Clostridium perfringens* associated lesions and improved performance in broiler chickens treated with normal intestinal bacteria from adult fowl. Avian Diseases. 2001;45:149-156.
7. Khan SA, Mustafa G, Chaudhry RA, Iqbal M, Khan ML. Infectious stunting syndrome of broiler chicks clinical signs and pathological lesions. 1995;8(1):1-6.
8. Kumari D, Mishra SK, Lather D. Pathomicrobial studies on *Salmonella gallinarum* infection in broiler chickens. Veterinary World. 2013;6(10):725-729.
9. Lenz SD, Hoerr FJ, Ellis AC, Toivio-Kinnucan MA, Yu M. Gastrointestinal pathogenicity of adenoviruses and reoviruses isolated from broiler chickens in Alabama. Journal of Veterinary Diagnostic Investigation. 1998;10:145-151.
10. Lim TH, Kim BY, Kim MS, Jang JH, Lee DH, Kwon YK, *et al*. Outbreak of gizzard erosions with fowl adenovirus infections in Korea. Poultry Science. 2012;91:1113-1117.
11. Luna LG. Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology, (ed. 3rd). McGraw Hill Book; c1968.
12. Muna EA, Salih MH, Zakia AM, Halima MO, Abeer AM, Ameera MM, *et al*. 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. 2016;10(1):1-8.
13. Nakamura K, Ohyama T, Yamada M, Abe T, Tanaka H, Mase M. Experimental gizzard erosions in specific pathogen-free chicks by serotype 1 group 1 adenovirus from broilers. American Association of Avian Pathologists. 2002;46(4):893-900.
14. Nazir S, Kamil AS, Darzi MM, Mir SM, Nazir K, Amare A. Pathology of spontaneously occurring salmonellosis in commercial broiler chickens of Kashmir Valley. Journal of World's Poultry Research. 2012;2(4):63-69.
15. Noiva R, Guy JS, Hauck R, Shivaprasad HL. Runting stunting syndrome associated with transmissible viral proventriculitis in broiler chickens. Avian Diseases. 2015;59(3):384-387.
16. Ono M, Okuda Y, Yazawa S, Imai Y, Shibata I, Sato S, *et al*. Adenoviral gizzard erosion in commercial broiler chickens. Veterinary Pathology. 2003;40:294-303.
17. Parimala DM. Study on the pathology of liver lesions in chicken. M.V.Sc. Thesis. Acharya N.G. Ranga Agricultural University, Rajendranagar, Hyderabad, India; c2003.
18. Sary K, Chenier S, Gagnon CA, Shivaprasad HL, Sylvestre D, Boulianne M. Esophagitis and pharyngitis associated with avian infectious laryngotracheitis in backyard chickens: Two cases. Avian Diseases. 2017;61:255-260.
19. Sharma S, Asrani RK, Singh G, Gulati BR, Patil PK, Gupta VK. Outbreak of hydropericardium syndrome associated with ascites and liver rupture in caged broilers. Veterinary Research International. 2014;2(2):33-45.
20. Tonu NS, Sufian MA, Sarker S, Kamal MM, Rahman MH, Hossain MM. Pathological study on colibacillosis in chickens and detection of Escherichia coli by PCR. Bangladesh Journal of Veterinary Medicine. 2011;9(1):17-25.
21. Lee HL. Aligning supply chain strategies with product uncertainties. California management review. 2002 Apr;44(3):105-19.