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Pathological studies of enteritis in chickens of Odisha

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Abstract
The pathological study of enteritis in chicken was carried out in two organised farms of Odisha over a period of one and half years. Dead birds with history of diarrhoea, vent soiling, increased mortality was thoroughly necropsied. The changes in the entire length of intestine were studied in detail. The tissues were processed, sectioned and stained with routine Haematoxylin-Eosin stain for studying histopathological changes. The major clinical signs observed during the physical visit to the farms and recorded from the owner's complaints were dullness, depression, drowsiness, watery to mucoid yellowish diarrhoea, and soiled vent. At necropsy, the carcasses were dry, dehydrated with atrophied muscles with a predominant involvement of anaemia and debility. In gross examination of intestine, there was yellowish catarrhal content and undigested food materials in the duodenum. The most common microscopic lesion in intestines were desquamation, presence of necrotic debris & inflammatory cells with congestion of the mucosa.

Keywords: Chicken, enteritis, necropsy, histopathology

Introduction
In the Indian scenario poultry farming is one of the optimum livelihood options for rural farmers especially women and unemployed youth. As far as the poultry health management is concerned there are numerous infectious and non-infectious diseases affecting poultry which is a major hindrance in taking up this venture. Out of these hindrances and health deteriorating factors one of the important but neglected problem is enteritis. Enteritis is the inflammation of intestine in particular the small intestine. A remarkable and major portion of the poultry farming accounts is expended in feeding of poultry. And if the feed is not absorbed properly by the birds due to any disturbances in the gastro intestinal tract, it will undoubtedly reduce the overall performance of the birds with regards to egg and meat production, body weight, feed conversion rate and immunocompetence.

Owing to extensive scientific and technical transformation in poultry production, birds are becoming susceptible to various digestive disorders. Among the various digestive disorders in chickens, enteritis is a major problem which not only affects the health of chickens but also affects the production and economy of the poultry farming. A wide range of factors associated with diet and infectious disease agents can negatively affect the delicate balance among the components of the chicken gut and, as a result, affect health status and production performance of birds in commercial poultry operations as suggested by Yegani *et al.* (2008) [17].

According to Korver *et al.* (2006) [10] the digestive tract of the chicken is a major site of potential exposure to pathogens. The GI tract must selectively allow the nutrients to cross the intestinal wall into the body while preventing the deleterious components of the diet from crossing the intestinal barrier Diagnosis of enteritis in chickens in the field is done basing on the flock history and physical examination of sick birds. However, a definitive diagnosis is missing in most of the cases. Dead and moribund birds need to be submitted to a pathological laboratory where more specific tests are done. In enteritis that involves other organ systems; routine examination and diagnosis in the field usually overlook the intestinal tract. In fact, an intestine that is normal to the naked eye may have inflammatory lesions that can only be detected by light microscopic evaluation. Therefore, gross and histological examination of the intestine is essential for determining the extent of intestinal damage, whereas bacterial culture may be quite useful to reach a confirmatory diagnosis [13]. Subsequently diagnosis of enteritis should be done at early stage in order to minimize the heavy loss to the farmers involved with poultry production.

Materials and Methods

The present study involved an attempt to study the pathological alterations of enteritis in chicken from two organized farms of Odisha over a period of one and half years from January 2016 to July 2017. Government farms like Poultry Breeding Farm of AICRP on poultry improvement, Intensive Poultry Development Project, Laxmisagar, Bhubaneswar and others birds presented from private poultry farms to the department of Veterinary pathology were included in the study. Clinical signs were noted before the death of the birds and in some cases, it was observed by the owners of private poultry farms. Thorough post-mortem was conducted on the dead birds which were presented in the college. The dead birds presented to Department of Veterinary Pathology, College of Veterinary Science and Animal Husbandry, OUAT with history of enteritis were also thoroughly necropsied. The gross lesions in different organs were noted. The pathological lesions found in various internal organs were noted. We studied the changes in entire length of intestine in detail because our focus was on enteritis. Each part of intestine i.e., duodenum, jejunum, ileum, caecum, colon, and rectum were separated and lesions were noted. After noting the gross pathological changes in the intestinal tract and internal organs, the representative tissue samples were collected immediately in small pieces in appropriate size and placed in 10% formal saline (fixative) of 20 times the volume of the tissues. Representative morbid materials were collected and preserved in 10% formal saline. The paraffin block was adjusted to the rotary microtome. The sharp knife was fixed in its proper position and the indicator regulator was adjusted to the thickness of the sections (5-6 micrometers). The tissues were processed, sectioned and stained with routine Haematoxylin-Eosin stain and studied in detailed for histopathological changes.

Results and Discussions

Clinical picture

The major clinical signs observed during the physical visit to the farms and recorded from the owner's complaints were dullness, depression, drowsiness, watery to mucoid yellowish diarrhea, soiled vent (Fig.1), increased water intake, inappetence, reluctance to move, sitting with closed eyes, tendency to huddle, ruffled feathers, dropped wings, respiratory distress. Many birds were debilitated, weak, stunted and decreased body weight. These observations have been corresponding to the reports by previous workers [1, 4, 6, 7, 9].

Postmortem examination

On necropsy, the carcasses were dry, dehydrated with atrophied muscles (Fig. 2). In most of the carcass, anemia and debilitated condition (Fig. 3) was predominantly involved. There was deposition of fibrinous exudates in form of layers on the outer surface of heart and liver. Liver showed enlargement, congestion, (Fig. 4) necrotic areas and perihepatitis. Spleen was congested and enlarged. Heart showed congestion (Fig. 5), pericarditis with deposition of fibrinous exudates on the surface. Lungs showed enlargement, congestion and edema (Fig. 6). Kidneys were found congested. Intestine showed congestion of the serosal vessels, segmental dilatation and ballooning (Fig. 7). On opening the intestine, there was yellowish catarrhal content and undigested food materials in the duodenum (Fig. 8). Some carcass had thick slimy mucous inside the lumen of intestine. In few cases there was hemorrhagic enteritis and the hemorrhages were more apparent on the mucosa of jejunum and ileum of the intestine (Fig. 9 & 10). These observations were in congruence to those reported in previous research works [1, 2, 4, 5, 7, 9, 11, 12, 14, 16].

Histopathological studies

Histopathological examination of all the enteritis cases associated with other system dysfunctions was made. The changes observed in heart were congestion along with disruption of myocardial fibrils. In addition to congestion and disruption there was also infiltration in between myocardial fibers of the heart (Fig. 11). Epicardial congestion was also distinctive in some of the affected cases (Fig. 12). Pericarditis with thickened pericardium and edema was also seen. These changes may be due to accumulation of severe fibrinous exudate in pericardial sac as reported by Bhalerao *et al*. (2011) [2] in histopathological lesions like fibrinous pericarditis, myocarditis, fibrinous perihepatitis, hepatitis and fatty changes in hepatocytes, interstitial pneumonia, necrosis and depletion of lymphocytes in spleen and enteritis.

Sections of liver showed congestion in between hepatic lobules (Fig. 13). There was replacement of hepatic parenchyma with focal infiltration of inflammatory cells with disorganization of hepatic lobules & vacuolar degeneration of hepatocytes which was quite evident. Few cases showed mild congestion & fatty changes of the hepatocytes in almost all the hepatic lobules of the liver (Fig. 14). There was disorganization of hepatic lobules with congestion, degeneration & necrosis of hepatocytes with infiltration of inflammatory cells was also found in some of the affected liver samples. Bacterial toxigenic injury in the liver may be induced by overgrowth of gut bacteria and release of exotoxins or bacteria translocation to the liver and release of endotoxins after bacterial killing by reticuloendothelial cells. These findings were similar to Ito MNK *et al*. (2004) [8] study of lesions in the parenchyma like increased leucocytic response in the sinusoidal space, vacuolar degeneration of hepatocytes or focal necrosis in the perilobular region, increased size of number of lymphoid nests in the perilobular region, the perivascular space and surrounding the billiary duct and increased extramedular granulocytopoiesis. Heavy infiltration of inflammatory cells in the parabronchial space mostly by macrophages & mononuclear cells were found in the lungs (Fig. 15). Some sections of lungs revealed marked congestion (Fig. 16) and oedema of parabronchial space. These types of lesions, could be categorized into enteroinvasive form of colibacillosis as investigated by Tonu *et al.* 2011. [16]

Kidney sections revealed marked inter tubular congestion and degeneration of tubular epithelial lining (Fig. 17 & 18). Kumari *et al*. (2013) [11] observed lesions in liver and spleen and found aggregation of heterophils, lymphocytes and macrophages, non-suppurative myocarditis, fibrinous pericarditis, interstitial nephritis, necrotic enteritis and serofibrinous pneumonia.

The changes in intestines were infiltration of inflammatory cells in the mucosal & submucosal area. The most common lesion was desquamation, presence of necrotic debris & inflammatory cells with congestion of the mucosa (Fig.19 & 20). There was infiltration of inflammatory cells & edematous fluid in the intervillous space (Fig.21). Congestion of muscular layer of intestine as well as inner muscular layer of the intestinal wall (Fig.22) was also evident in most of the

findings. In addition, there was focal infiltration of inflammatory cells in villi, desquamation of mucosal villi & congestion. Sloughing of the mucosa with edema of the submucosa & infiltration was also found. These are characteristic features of enteritis as reported by Das *et al*. (2008) [3] who investigated necrotic enteritis and revealed massive necrosis and complete destruction of the intestinal villi within the intestinal mucosa. Sawale *et al*. (2010) [14] observed microscopic lesions like diffuse coagulative necrosis of mucosa reaching deep up to muscularis in the intestine. Ahmad *et al*. (2013) [1] pathologically investigated necrotic enteritis in desi chickens. During the histopathological study they found necrosis of enteroepithelial cells with marked desquamation, increased cellular infiltration in lamina propria of intestinal mucosa. Malmarugan *et al*. (2013) [12] demonstrated degeneration, necrosis of enterocytes, microcyst formation and mononuclear cell infiltration within villi and haemorrhages between villi in intestine. Sivaseelan *et al*. (2013) [15] microscopically observed that intestinal damage characterized by destruction of crypts cell as well as villi enterocytes.

Fig 1: Soiled vent

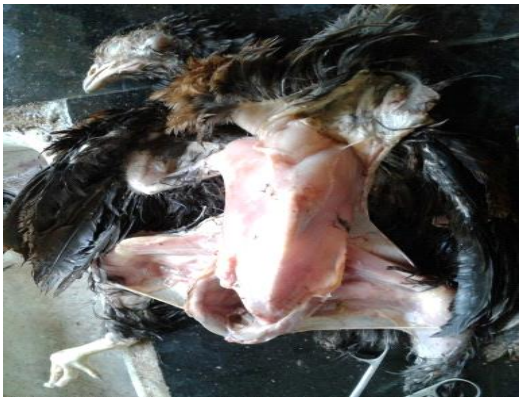

Fig 2: Atrophied muscle

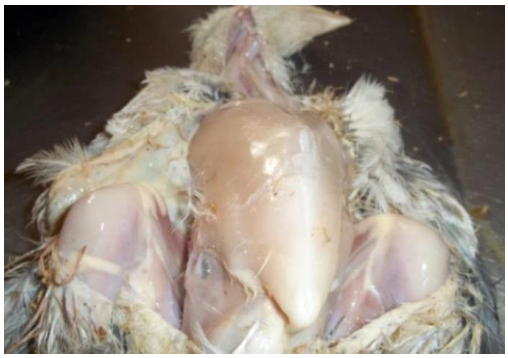

Fig 3: Anaemic condition

Fig 4: Enlarged liver with congestion

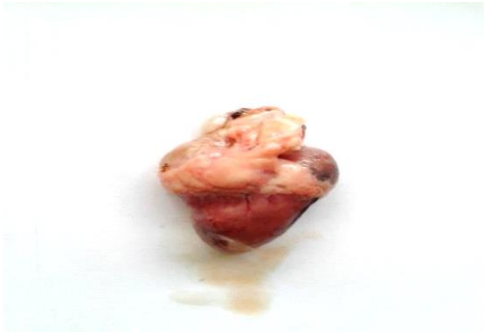

Fig 5: Congestion of heart

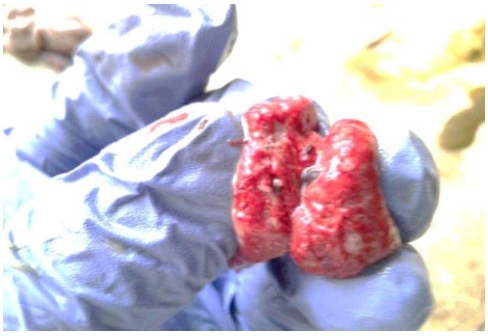

Fig 6: Congestion and edema of lungs

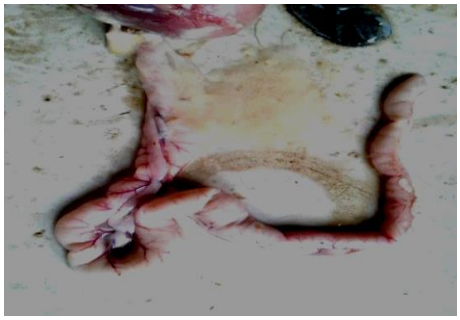

Fig 7: Intestinal dilatation

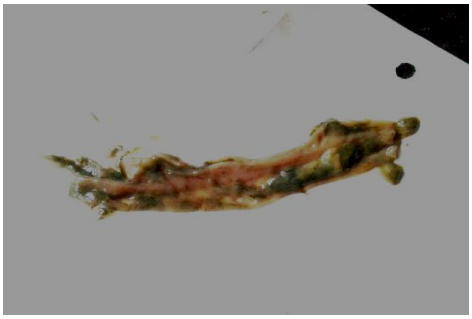

Fig 8: Catarrhal contents in intestinal lumen

Fig 9: Intestinal hemorrhage

Fig 10: Mucosal hemorrhage & catarrhal exudates in intestine

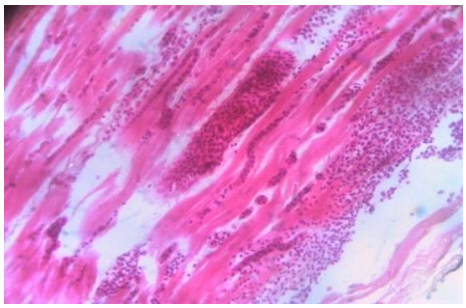

Fig 11: Heart showing congestion, disruption & infiltration of myocardial fibres. (H & E x400)

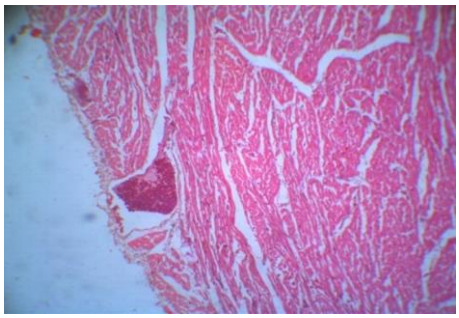

Fig 12: Epicardial Congestion of heart. (H & E x100)

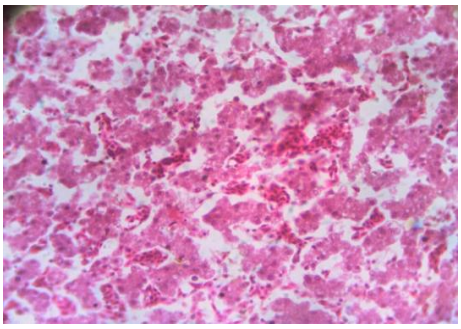

Fig 13: Liver showing congestion in between hepatic lobules. (H & E x400)

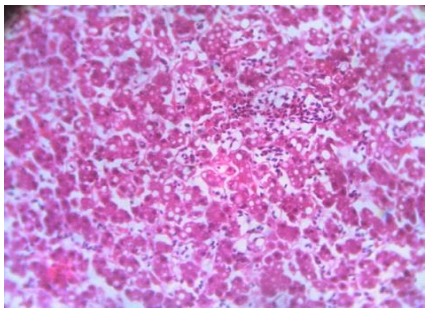

Fig 14: Fatty changes of the hepatocytes in hepatic lobules of the liver (H & E x400)

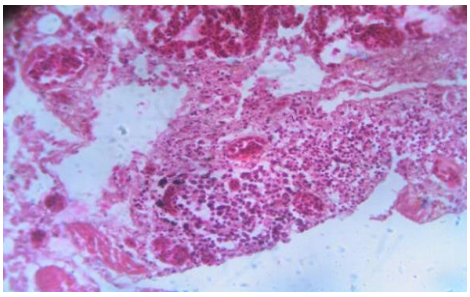

Fig 15: Lungs showing heavy infiltration of inflammatory cells in the parabronchial space mostly macrophages & mononuclear cells. (H & E x400)

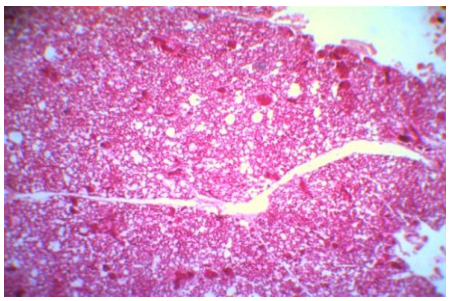

Fig 16: Lungs with marked congestion of parabronchial wall of lungs. (H & E x100)

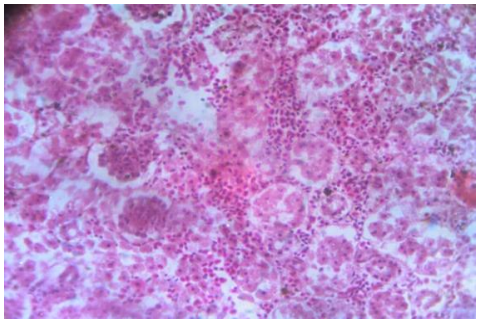

Fig 17: Marked inter tubular congestion of kidney (H & E x400)

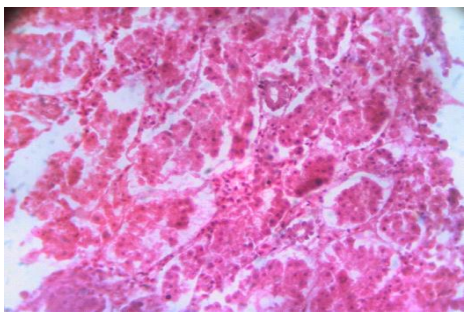

Fig 18: Degeneration of tubular epithelial lining in kidney. (H & E x400)

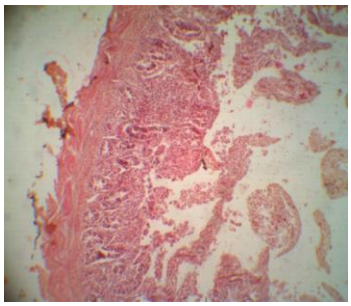

Fig 19: Intestine showing desquamation of the mucosal villi. (H & E x100)

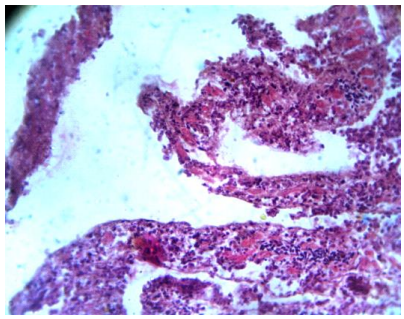

Fig 20: Desquamation of mucosal villi & congestion of intestine. (H & E x400)

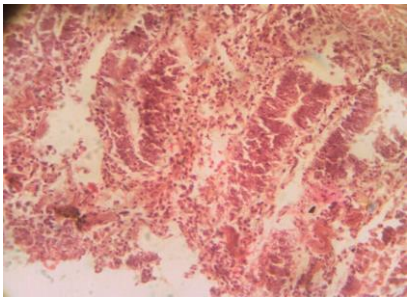

Fig 21: Intestine showing intervillous space infiltrated with inflammatory cells & edematous fluid. (H & E x400)

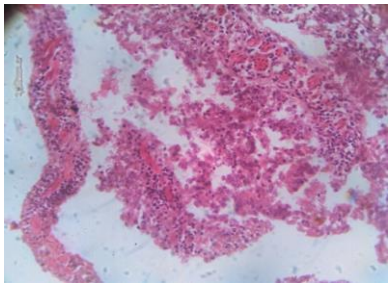

Fig 22: Congestion of inner muscular layer of the intestinal wall. (H & E x400)

Conclusion

As regards to other inflammatory conditions in poultry, enteritis is given less importance while diagnosing the disease condition but the epidemiological history, detailed study of the signs of enteritis along with meticulous inspection of gross and histopathological changes can reveal the real cause of mortality of birds with proper diagnosis and control of enteritis thereby reducing the economic loss of farmers. Study of epidemiological history, clinical signs, gross and histopathological changes will be very much helpful in proper diagnosis of enteritis.

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