

## PATHO-ANATOMICAL STUDIES ON POULTRY MORTALITY WITH SPECIAL REFERENCE TO GASTRO-INTESTINAL TRACT DISORDERS

ASHISH HOODA, S. K. MISHRA\*, VIKAS NEHRA and DEEPIKA LATHER

Department of Veterinary Pathology, College of Veterinary Sciences  
Lala Lajpat Rai University of Veterinary & Animal Sciences, Hisar-125 004

### ABSTRACT

Patho-anatomical studies on poultry mortality during the period from July 2008 to March, 2009 were undertaken with special reference to gastrointestinal tract disorders. The affections of digestive system accounted for higher mortality (30.88%) followed by 10.97% due to respiratory tract disorders and the remaining by affections of other systems. Representative tissue pieces from sixty five chickens revealing changes in gastrointestinal tract were collected in 10% buffered formalin for histopathological examination. Enteritis, hepatitis, fatty liver and enlargement of liver were the main gastrointestinal tract conditions\disorders encountered during postmortem examination of the poultry carcasses in this study.

**Key words:** Poultry, pathology, gastro-intestinal tract disorders

During the last few decades, methods of poultry husbandry have changed considerably due to an emphasis on intensive rearing which has predisposed birds to various infectious diseases, thereby causing economic losses to the farmers. Among infectious diseases, the conditions such as colibacillosis, salmonellosis, coccidiosis, Ranikhet disease, necrotic enteritis etc affecting gastro-intestinal tract are quite common. These infections lead to heavy morbidity, mortality and lowered egg or meat production. Importance of these diseases can be judged from the fact that the incidence of coccidiosis (15.50%) was the highest followed by *Escherichia coli* infections (14.0%), hepatopathy (7.0%), fatty liver kidney syndrome (3.3%) and Ranikhet disease (2.7%) (Suresh *et al.*, 1990). Keeping in view the above facts, patho-anatomical studies on poultry mortality was undertaken with special reference to gastrointestinal tract disorders.

### MATERIALS AND METHODS

**Pathological Studies:** Detailed postmortem examination of all 441 carcasses of poultry brought to the Department of Veterinary Pathology was conducted during the period of July, 2008 to March, 2009. The representative pieces of tissues from each carcass revealing lesions in gastrointestinal tract were collected in sterile petridish for

bacteriological examination.

**Isolation and identification of bacteria:** Isolation of bacteria from liver and lung tissue samples was conducted by inoculation of the samples on to blood agar and Mc-Conkey's lactose agar (MLA) plates as described by Cruickshank *et al.* (1965).

**Gross Changes:** All the organs and tissues of the dead birds were examined critically to detect gross changes during postmortem examination and lesions so observed, were recorded.

**Histopathological Examination:** Representative tissue pieces from organs revealing lesions in gastrointestinal tract were collected in 10% buffered formalin for detailed histopathological examination. The formalin fixed tissues were washed in running tap water, dehydrated in acetone, cleared in benzene and embedded in paraffin wax (melting point 60-62°C). Paraffin sections were cut at the thickness of 4-5 µ and stained with routine haematoxylin and eosin stain using Lilly Mayer's haematoxylin and 2% water soluble eosin (Luna, 1968).

### RESULTS AND DISCUSSION

During the course of postmortem examination, representative tissue pieces from 65 chickens revealing changes in gastrointestinal tract were collected for detailed histopathological examination. The affections

\*Corresponding author: skmishra@hau.ernet.in

of digestive system accounted for higher mortality (30.88%) followed by respiratory tract disorders (10.97%) and the remaining (58.15 %) by affections of other systems. Higher mortality due to lesions in digestive system may be due to involvement of large number of organs of gastrointestinal tract in the pathogenesis of various bacterial diseases. Enteritis, hepatitis, fatty liver and enlargement of liver were the main gastrointestinal tract conditions in this study. Pathological lesions associated with bacterial or parasitic infections are as under:

#### **PATHOLOGICAL LESIONS ASSOCIATED WITH BACTERIAL INFECTIONS**

**Gross Changes:** Most of the birds infected with *E. coli* revealed the presence of a fibrin layer on all the visceral organs particularly on heart and liver. The carcasses were dark coloured, septicæmic and dehydrated. Liver and spleen were found to be congested and haemorrhagic in most cases. In some cases, necrotic foci were present on the liver. In the cases of ascites syndrome, there was accumulation of excessive fluid in the pericardial sac and peritoneal cavity with distended abdomen. Enteritis was another common observation in *E. coli* infected birds. In some cases, catarrhal exudate was present in the intestines. Peritonitis with haemorrhages in the liver, spleen and intestines was noticed in such cases. These findings were in accordance with reports of Vandekerchove *et al.* (2004) and Gangane *et al.* (2006). In young birds, *E. coli* infection was mainly associated with omphalitis. Peritonitis was also observed in such cases. This finding was similar to earlier reports (Vegad, 2004; Goyal *et al.*, 2004).

Carcasses of birds infected with *Salmonella* spp. were discoloured or jaundiced in appearance. There was enlargement of liver and spleen in most cases along with presence of small, grayish white necrotic foci. In few cases, liver was swollen and at surface there was bronze discolouration. Catarrhal enteritis having bile stained material was also present in some cases. Similar findings have already been reported by many workers (Goswami *et al.*, 2003; Msoffe *et al.*, 2006).

The carcasses infected with *Proteus* spp. were found emaciated and dehydrated in all the cases. In few cases, fibrin layer along with necrotic foci was present on the liver. Enteritis was also observed in all the affected cases.

All three cases infected with *Citrobacter* spp. showed haemorrhages on the intestines. Liver was pale and mottled in one case. In two cases, fibrin layer was also present on liver. Catarrhal exudate was present in the intestine in two cases while in the remaining one, slight enteritis was revealed on postmortem examination. There is not enough literature available regarding role of *Citrobacter* spp. in poultry mortality. Fales *et al.* (1978) and Venkanagouda *et al.* (1996) have described role of this organism as opportunistic pathogen in birds with respiratory diseases.

In one case, *Klebsiella pneumoniae* was isolated from the bird affected with ascites syndrome. Liver was fatty in consistency and mild enteritis was also observed.

Carcasses infected with *Clostridium* spp. were dehydrated and septicæmic with presence of haemorrhages on all the visceral organs. Necrotic foci were present on the surface of liver. Haemorrhagic enteritis was also evident in the intestines. However, lesions were more apparent in jejunum and ileum. Similar lesions have already been recorded by Katoch *et al.* (2004) and Gholamiandehkord *et al.* (2007). Necrotic enteritis was observed as main cause of poultry mortality by Work *et al.* (1998).

The carcass of *Corynebacterium pyogenes* infected bird was weak and emaciated with pale musculature. Liver was mottled with presence of necrotic foci. Spleen was found to be congested. Slight enteritis was also noticed with swollen and congested mucosa. This bacterium is not commonly isolated from poultry, however, Awan and Matsumoto (1998) isolated *Corynebacterium* along with several other unusual bacteria from birds.

#### **Microscopic Changes**

##### ***E. coli* Infection:**

**Intestine:** Changes in intestines included congestion, haemorrhages, desquamation of mucosal epithelium, massive infiltration of leukocytes and erythrocytes, goblet cell hyperplasia, focal necrosis of villi and mucosal folds and inflammatory and vascular changes extending upto serosal layer (Fig. 1). There was atrophy of the glandular tissue due to exudate alongwith severe infiltration of leucocytes. In one case, fibroblasts were observed in the lamina propria indicating that infection may be of chronic type.

**Liver:** There was dilatation of hepatic sinusoids with

presence of RBCs in the sinusoids in few cases. Vacuolation and degeneration of the hepatocytes, congestion and haemorrhages, and hyperplasia of Kupffer cells was observed in many cases. Necrotic areas were evident with presence of macrophages, lymphoid cells, plasma cells and other inflammatory cells. In some cases, severe inflammatory changes were observed near the blood vessels. Fibrinous perihepatitis was also evidenced in many cases which is the characteristic feature of colibacillosis. These findings were in agreement with Peighambari *et al.* (2000), Goyal *et al.* (2004) and Gangane *et al.* (2006). In cases of ascites syndrome too, *E. coli* was isolated indicating the role of this organism in pathogenesis of the disease as studied earlier by many workers (Tottori *et al.*, 1997; Yamaguchi *et al.*, 2000).

#### **Salmonella Infection:**

**Intestine:** Desquamation of mucosal epithelium resulting into denuded villi was observed and cellular debris was present in the lumen. Secretory glands were atrophied at some places because of severe infiltration and presence of exudate. Congestion and haemorrhages were common in the lamina propria that extended upto the muscularis or serosa in few cases. Goblet cell hyperplasia was also present along with leukocytic infiltration in the lamina propria.

**Liver:** Multiple necrotic foci, Kupffer cell hyperplasia, extensive fatty changes with eccentric nuclei in hepatic cells, focal aggregation of macrophages, plasma cells and lymphoid cells around necrosed areas (Fig. 2), fibrinous perihepatitis and congested/thrombosed capillaries were noticed. Continuation of cords was missing indicating towards the chronic lesion in few cases. Hydropic changes showing vacuolation in cytoplasm of hepatocytes were observed in some cases. In two cases, nodule formation was also present with separation of hepatocytes by leukocytes, Kupffer cells and plasma cells.

**Pancreas:** Areas of mild congestion and haemorrhages, mild degenerative changes and necrotic areas were present. All these findings were in accordance with those reported by Kokosharov *et al.* (1997) and Deshmukh *et al.* (2007).

#### **Proteus Infection**

**Intestines:** Mucosal epithelium was desquamated in most of the cases. Lamina propria revealed infiltration

of leukocytes along with fibrous connective tissue. At places, glandular tissue was atrophied.

**Liver:** Degenerative changes in the hepatocytes including hydropic vacuolation, fatty changes and Kupffer cell hyperplasia were noticed. At places, infiltration of leukocytes was there in and around areas of necrosis and around the blood vessels.

The lesions noticed in different organs were more or less similar to those described by Lin *et al.* (1993) and Ye *et al.* (1995).

#### **Citrobacter Infection**

**Intestines:** There was mild congestion, degeneration, desquamation of the mucosal epithelium and goblet cell hyperplasia in most of the cases. Chronic catarrhal enteritis was present in two cases.

**Liver:** Hepatic sinusoids were dilated and filled with erythrocytes at many places. Focal areas of necrosis, leukocytic infiltration and hyperplasia of Kupffer cells were noticed. Fibrinous perihepatitis was also observed in two cases.

#### **Klebsiella Infection**

**Intestines:** Degeneration and desquamation of epithelial cells was observed. Vascular changes were evident in mucosa which extended upto serosa in some cases. Cellular debris along with RBCs and leukocytes were present in the lumen of the intestine.

**Liver:** Fatty changes were evident in hepatocytes in two cases. Haemorrhages and congestion, Kupffer cell hyperplasia and bile duct hyperplasia were also present in all cases.

#### **Clostridium Infection**

**Intestines:** Atrophied mucosal glandular tissue, congestion and haemorrhages in mucosa which extended upto muscularis and serosa, desquamation of epithelial cells which accumulated in the lumen along with leukocytes and RBCs, goblet cell hyperplasia, multiple areas of necrosis and fibroblasts in lamina propria were the major findings in intestines.

**Liver:** There were moderate congestion and haemorrhages alongwith extensive fatty changes and leukocytic infiltration. At places, evidence of necrotic areas was present.

**Pancreas:** Mild degenerative changes were observed in the pancreas.

The observations of Work *et al.* (1998) and Gholamiandehkordi *et al.* (2007) support the findings of this study.



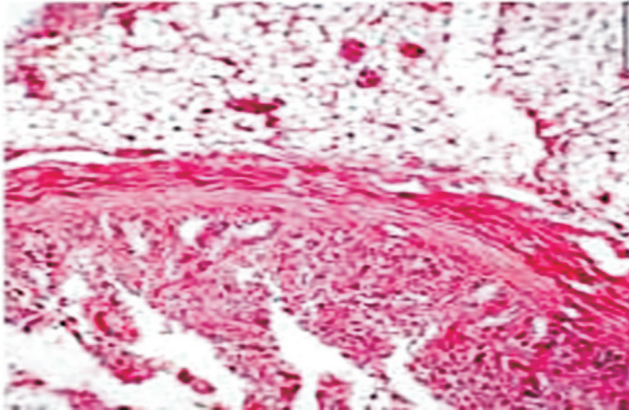


Fig 1. Photomicrograph of intestine showing vascular changes (congestion) extending up to serosal layer in an *E. coli* infected bird. (H. & E. x 33)

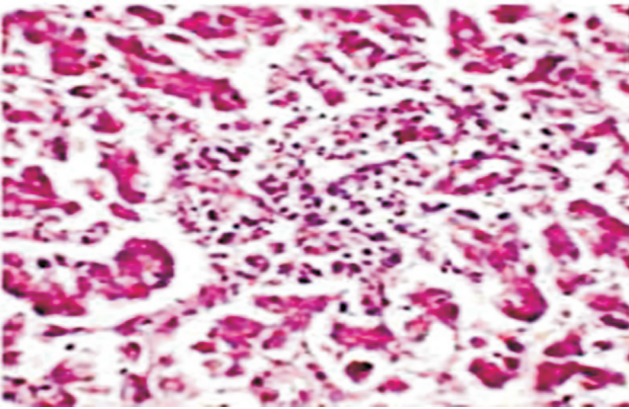


Fig 2. Photomicrograph of liver showing necrotic foci along with aggregation of Kupffer cells, plasma cells and lymphoid cells in a *Salmonella enterica* serovar *Gallinarum* infected bird. (H. & E. x 66)

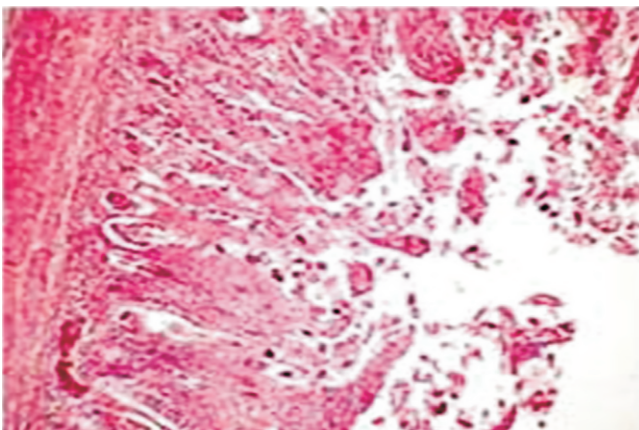


Fig 3. Photomicrograph of intestine showing the presence of oocysts, schizonts and merozoites in epithelial cells of a bird affected with coccidiosis. (H. & E. x 33)

### ***Corynebacterium* Infection**

**Intestines:** Lamina propria was thickened. There was presence of edema and congestion at few places. Mucosal epithelium was desquamated along with mononuclear cell infiltration.

**Liver:** Diffuse fatty changes in the hepatocytes, areas of congestion and haemorrhages, and focal areas of necrosis were evident.

### **PATHOLOGICAL LESIONS ASSOCIATED WITH PARASITIC INFECTIONS**

Coccidiosis was the only parasitic infection observed during faecal examination of birds showing signs of diarrhoea and haemorrhagic enteritis.

**Gross Changes:** There was presence of diarrhoea and the vent was found soiled with faeces. Ballooning of intestines along with white spots was also observed. Haemorrhagic enteritis was a common feature observed during postmortem examination of such birds. Mucosa of the intestine was thickened and the lumen was filled with fluid, blood and tissue debris. In cases of caecal coccidiosis, caeca were enlarged and distended with clotted blood. Liver also appeared enlarged and pale in a few cases.

### **Microscopic Changes**

**Intestines:** Mucosal wall was thickened and edematous. Haemorrhages and inflammation was found extending up to the muscularis layer and sometimes up to serosal layer. Different stages of life cycle of parasite were also observed during microscopic examination of the intestines (Fig. 3). Secretory glands were found to be damaged. Desquamated and necrosed cells were present in the lumen as debris. There was infiltration of macrophages, plasma cells, lymphocytes along with heterophils in the lamina propria.

**Liver:** Degenerative and necrotic changes were observed in the liver along with some areas of congestion and haemorrhages. Necrotic foci were mostly located close to blood vessels with aggregation of leukocytes at certain places.

Similar lesions have also been documented by other workers (Mattiello *et al.*, 2000; Novilla and Carpenter, 2004; Jayanthi *et al.*, 2007).

Based on these findings, it can be concluded that

gastrointestinal disorders were the main cause of poultry mortality which were mainly due to *Salmonella* spp. and *E. coli* infections.

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