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Ascaridia galli infection induced gross-pathological changes in broiler chicken

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Abstract

In this study, the effect of *Ascaridia galli* infection on gross-pathological changes in broiler chicken was investigated. The postmortem examinations of broiler experimentally induced *A. galli* revealed gross pathological lesions of infected organs viz., small intestine, liver, lungs and heart. Hemorrhagic enteritis and acute fibrinous enteritis with clotted blood, hemorrhagic and reddish spots on intestinal wall, inflammation and necrotic patches with consolidation of intestinal contents were generally observed. Infected liver showed dark red coloration. Severe congestion, chronic pneumonia, exuded in one lobe of the lung were seen, whereas, necrosis and atrophy in infected heart were notable observations.

Keywords: *Ascaridia galli*, gross-pathology, broiler chicken, Pakistan

The parasitic species pose great economic losses to livestock and wild animals in all parts of the world. In many areas, particularly in tropical and sub- tropical regions, the helminth parasites especially nematodes and cestodes (Norton, 1964; Magwisha *et al.*, 2002) are major cause of death of livestock animals including poultry birds. Nematodes infections cause reduction in food intake, injury to the intestinal wall and hemorrhage resulting in poor weight gain and secondary infection of the birds (Anwar & Rahman, 2002; Skallerup *et al.*, 2005).

Materials and Methods

Collection of samples: 20 broiler chicken of two weeks age were reared and divided into two groups (a) Experimental (b) Control. Both groups were provided similar feeding, housing and related management. High positive *A. galli* eggs infection was induced in experimental chicken of group (a) and left for further two weeks for infection to establish. After two weeks, confirmation of infection was done through coproscopy. The samples collected were brought to the laboratory, Department of Veterinary Parasitology, Faculty of Animal Husbandry & Veterinary Sciences, Sindh Agriculture University, Tandojam, Pakistan.

Post-mortem examination for gross pathology: The postmortem was performed as described by Anjum (1996). Briefly, a transverse cut was made through, the abdominal muscles immediately anterior to the vent on each side with a pair of scissor. Initially, the skin, flesh and rib cage (through joints of ribs) were cut, and then coracoid and clavicles on both sides were cut. The sternum and pectoral muscles were separated from the rest of the body carefully, freeing ventral attachment of heart first. The thoraco-abdominal organs were examined

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for pathological changes. Changes in size, color or shape, exudates, etc. were also noted. The air sacs and lungs were examined along with peritoneum. Abdominal cavity was also opened and examined. Heart and pericardium were removed and examined. Liver was removed by incising the attachments and its color, edges, size, friability, etc. were noted. Gastro-intestinal tract was examined by cutting the intestinal tract at the junction of the proventriculus, oesophagus and at cloaca and was removed from the body cavity along with liver. The loops of intestine were unveiled. Any signs of inflammation, hemorrhages, ulcers, denudations, malfunctions, etc. were examined. Intestines were primarily externally inspected and then cut with scissors to see any lesion on the inner lining (Anjum, 1996).

Gross-pathological examination: Thorough naked eye examination was done to see any gross abnormality of infected and un-infected organs viz., small intestine, heart, liver and lungs. The gross pathology of un-infected and infected birds was done in the laboratory, Department of Veterinary Parasitology, Faculty of Animal Husbandry & Veterinary Sciences, Sindh Agriculture University, Tandojam, Pakistan.

Results

Postmortem of experimentally infected and control birds were revealed gross-pathological changes in target organs; in relation to *A. galli*.

The gross pathological changes during the present study revealed, hemorrhagic enteritis and acute fibrous enteritis with clotted blood, hemorrhagic and reddish spots on intestinal wall, inflammation and necrotic patches with consolidation of intestinal contents observed in most heavily infected birds as compared to the normal (Fig. 1A,B). The gross-pathological changes of liver showed dark red coloration and severe congestion as compared to the normal (Fig. C,D). In case of lungs, chronic pneumonia, exuded in one lobe was noted, and necrotic and atrophied conditions were also noted in heart of infected birds as compared to the normal (Fig. E-H).

Discussion

Gross-pathological study of infected intestine: During present study, the postmortem examinations revealed gross pathological lesions in intestine, hemorrhagic enteritis and acute fibrinous enteritis with clotted blood, hemorrhagic and reddish spots on intestinal wall, inflammation and necrotic patches (Fig. 2).

These pathological conditions are induced by the worms as they grab intestinal tissues after absorbing the digested food stuff. Sometimes, worms try to penetrate into the intestinal epithelium, resulting into necrosis and inflammation. Moreover, this may also be due to the fact that embryonated eggs containing second stage larvae may be ingested and hatched in the intestinal wall, and produce gross pathological lesions, including intestinal hemorrhagic enteritis, necrotic patches and reddish spots on the intestinal wall. Soulsby (1982) and Urquhart *et al*., (1996) noted similar conditions. Abrha *et al*., (2014) recorded lesions including enteritis characterized by hemorrhagic, oedematous and thickened wall.

Carlos *et al.*, (1984), William & Linda (2000) and Skallerup *et al*., (2005) also reported injury to the intestinal wall and hemorrhage. Permin & Ranvig (2001) and Magwisha *et al*., (2002) reported petechial bleeding in the intestinal mucosa, nodular hemorrhagic enteritis and inflammation of the proventriculus. Abdelqader *et al*., (2006) and Adang *et al*., (2010) reported that if the number of parasite is quite huge, some may penetrate duodenal or jejunal mucosa and affect their histology. In a more recent study, Sherwin *et al*., (2013) reported pathological lesions including enteritis with hemorrhages, obstruction of the intestinal lumen and altered hormone levels.

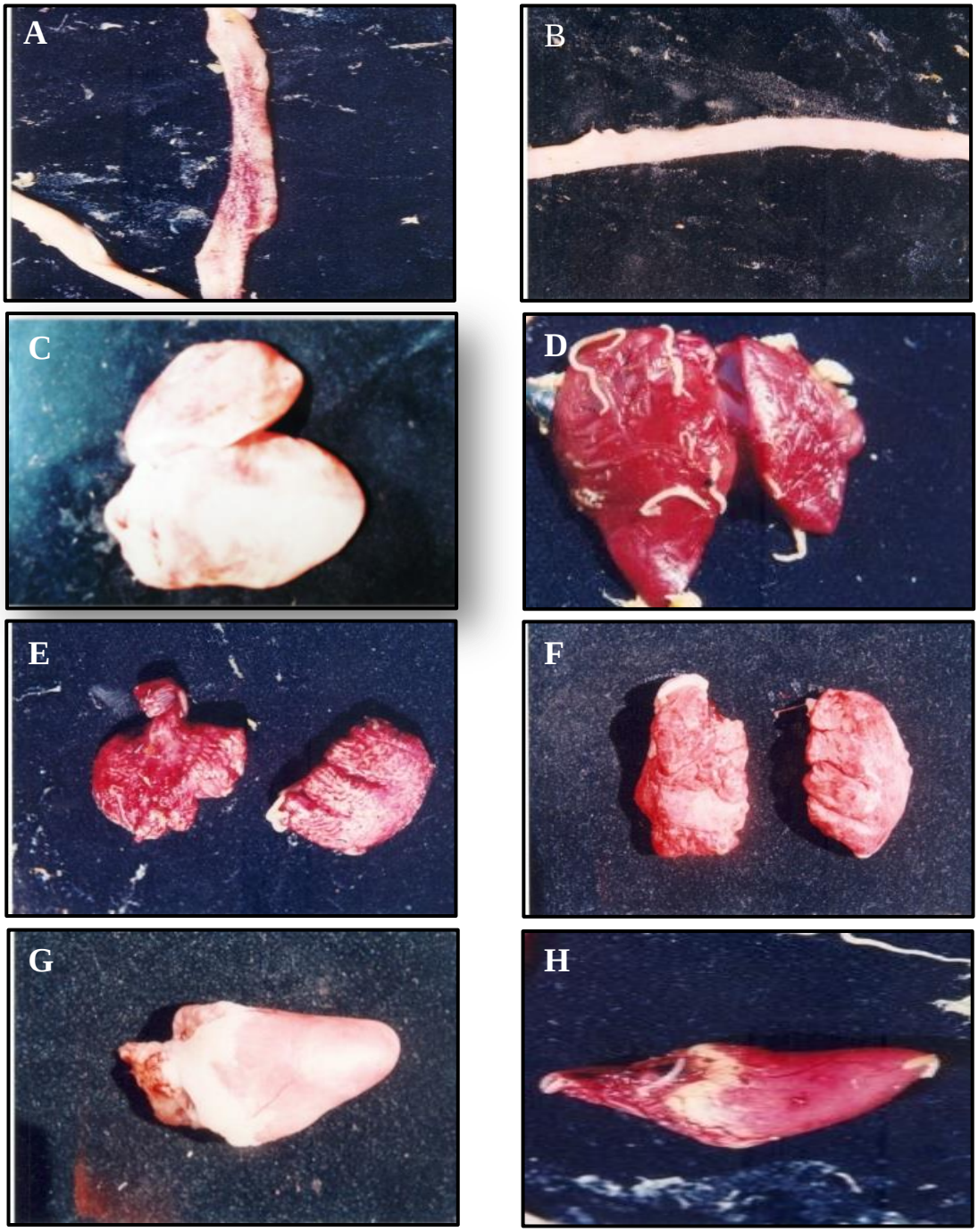

Fig. 1 (A-H). Gross-pathological changes in different organs. A. Normal intestine of broiler chicken; B. Hemorrhagic enteritis and acute fibrinous enteritis in infected intestine; C. Normal liver of broiler; D. Dark red coloration and severe congestion of liver; E. Normal lung of broiler; F. Chronic pneumonia, exuded in one lobe of lung; G. Normal heart of broiler; H. Necrotic and muscle atrophy of heart.

Adang *et al*., (2010) also recorded hemorrhagic lesions in intestine, this may be due to larval migration during tissue phase of life-cycle. The studies indicated that polymorph nuclear and mononuclear cells existed there in necrotized areas.

Gross-pathological study of infected liver: Liver is the metabolic factory, detoxification unit and storage organ in vertebrates. Our findings indicated some gross-pathological changes in liver. Gross-pathological changing of liver showed in (Fig.1D) demonstrates dark red coloration which may be due to blood clotting within hepatic tissues.

In fact, when larvae in large number move towards liver, they damage the tissues resulting into bleeding and finally clotting. Kijlstra & Eijck (2006) also noted dark colour of liver. Gauly *et al*., (2001) and Schou *et al*., (2003) observed that L_2, L_3 larvae can cause milky spots which portray as cloudy whitish spots up to 1.0 cm. in diameter and represented the fibrous repair of inflammatory reactions to passage of larva in the livers.

Gross-pathological study of infected lungs: The present study indicated the gross-pathological changes in the infected lungs (Fig. F). The visual examination demonstrates chronic pneumonia and exudate in one lobe. This condition may be due to larval migration, it was also noted by Abrha *et al*., (2014) who observed exudates and black spots in lungs. Urquhart *et al*., (1996) reported that the L_3 larvae may break through to the alveoli and ascend the bronchi and trachea to be swallowed. He further noted tracheal-migration of third-stage larvae that occurred in the pulmonary alveoli.

The lesions produced by the infective larvae during their migration and development in the tissues of the host lungs sets up an inflammatory reaction that may result in mild to severe respiratory involvement, and which usually heal with few residual lesions. Hemorrhages occurred as the larvae break out in the pulmonary capillaries to enter the alveoli, and in heavy infections loss of bronchiolar epithelium and infiltration of leukocytes alveoli was replaced by cystic spaces, and loss of bronchiolar epithelium and infiltration of leukocytes.

Adang *et al*., (2010) reported the lungs of infected birds with hemorrhagic areas, congested blood vessels and hemosiderosis. There was mononuclear and polymorpho nuclear cellular infiltration at the peribronchiolar and inter-alveolar septae which extended and filled some alveoli. Abrha *et al*., (2014) reported lungs were found to have mild to moderate congestion and hemorrhage, anthracnosis severe congestion, alveoli filled with erythrocytes were also found. Our studies do not agree with the studies of Abrha *et al*., (2014).

Mabon & Reid (1973) observed the larval stages of *Ascaridia galli* in chicken did not migrate into the lungs or other internal organs of the host. The larvae were found associated with the mucosal surface of the jejunum and ileum walls on the 4^{th} to the 12^{th} days of infection. But Taiwo *et al*., (2002) observed *Ascaridia galli* larvae most commonly encountered in the kidney, may backyardize in any tissue, including the liver, lung, myocardium, brain, eye and lymph nodes.

Gross-pathological study of infected heart: Gross pathological study of infected heart (Fig.1 H) showed necrosis and atrophy. This is in partially agreement with Abrha *et al*., (2014), they observed gross lesions of heart, excessive fat deposition around the heart, myocardial pericardial necrosis and fibrinous pericarditis.

Adang *et al*., (2010) reported the heart had focal areas of necrosis of the myocardial cells and few mononuclear and polymorphonuclear cells in the necrotized areas in *A. galli* infection could have some histopathological effects on the heart.

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