DISEASES ASSOCIATED WITH MORTALITY AND PATHOLOGICAL CHANGES IN COCKEREL BIRDS


Department of Pathology, Faculty of Veterinary Science, Bangladesh Agricultural University, Mymensingh-2202, Bangladesh

ABSTRACT

The diseases associated with mortality and pathological changes were studied in three different cockerel flocks in the rural areas of Thrishal upazilla in Mymensingh district from 5th August to 30th October 2000. Diagnosis of diseases was based on clinical history, characteristic pathological changes and occasionally isolation of the causative agents. The infectious bursal disease (IBD), may sac infection, vitamin E deficiency, coccidiosis and others were diagnosed. The mortality rate in cockerel caused by IBD, may sac infection, vitamin E deficiency, coccidiosis and others were recorded as 7%, 6.67%, 1.72, 0.01% and 0.1% respectively. The characteristic gross lesions of IBD were hemorrhage in thigh and pectoral muscles greasy and macroscopically the bursa showed dead and pyknotic nuclei of lymphocytes. The optical lesions of may sac were thickened with unabsorbed yolk in all cockerel greasy and macroscopically thickening yolk sac due to leukoclastic proliferation and mononuclear infiltration in association with normal fat cells. The vitamin E deficiency was diagnosed grossly by softness, swelling and focal hemorrhage on the cerebellum and histopaclically by proliferation of huge number of giant cells. Grossly, coccidiosis was characterized by swells, thickened, furred and hemorrhagic caeca and macroscopically by hyperplastic, tall columnar epithelial cells contained large number of schizonts and trophozoites in the affected crypts and villi. The findings indicate that IBD is the major disease problem in cokceral farming in Mymensingh.

Key words: Mortality, diseases, pathological changes, cockerel.

INTRODUCTION

The small poultry farmers in Bangladesh usually prefer to cockerel farming due to low cost of 24-day old chicks, required less floor space and feed, higher price of cockerel meat than the broiler and their belief that the cockerels are less susceptible to diseases in comparison to broilers. Although the farmers begin cockerel farming with great enthusiasm but sometimes they become dissatisfied when there is great mortality of cockerels due to disease outbreak.

We have reported the occurrence of diseases associated with morbidity and mortality in cockerel and its relationship with the management (Sai et al., 2002) and this paper describes the mortality and pathological changes of major diseases of cockerels.

MATERIALS AND METHODS

The study was conducted at three different cockerel farms ( = 974 birds) in the rural areas of Thrishal upazilla in Mymensingh district from 5th August to 30th October 2000. The observation was performed from 24 day-old chicks up to marketing (60 days) of the cockerel. The mortality, morbidity, age of infection of various diseases conditions were recorded. The dead birds ( = 87) were collected for necropsy and the diagnoses of different diseases were based on the history, clinical signs and characteristic gross as well as microscopic tissue alterations as described by Chakke et al. (1997). The clinical signs exhibited by the individual bird during illness were recorded in detail in a prescribed form provided by the respective poultry farm's owner. In addition, sometimes two sick birds were kept under careful observation with feed and water ad libitum till death to record the detailed clinical signs along with other abnormalities and all of them were necropsied soon after death. The postmortem examination in all the 87 birds was performed as soon as the dead birds were collected and samples were carried to the laboratory. At necropsy, gross tissue changes were observed and recorded carefully and representative tissue samples containing lesions were fixed in 10% buffered neutral formalin for histopathologic studies. The histopathology of collected tissues was performed following the procedures described by Lima (1968). In addition, some tissue sections were subjected to Gram staining and PAS staining whenever necessary. Necropsy were made from fecal contents and / or caecal scrapings were observed under microscope for detection of coccidial or intestinal oocysts.

Present address: Department of Medicine, Faculty of Veterinary Science, Bangladesh Agricultural University, Mymensingh-2202, Bangladesh, Livestock Research Institute, Mohakhali, Dhaka, Bangladesh, Bangladesh Livestock Research Institute, Savar, Dhaka, Bangladesh.

Copyright © 2003 Bangladesh Society for Veterinary Medicine All rights reserved 1729-7895/2000/0403
Mortality and pathological changes in cockerels

The susceptible sample was collected asexually from yolk sac contents of the yolk sac infected dead cockerels. Special attention was given to the Escherichia coli isolation. Individual single colony from EMB age plate was isolated and identified.

RESULTS AND DISCUSSION

The present investigation identified five maladies responsible for morbidity and mortality in cockerels (Table 1).

Table 1. Morbidity and mortality caused by diseases in cockerels

<table>
<thead>
<tr>
<th>S/N</th>
<th>Disease</th>
<th>Farm 1 (n=224)</th>
<th>Farm 2 (n=250)</th>
<th>Farm 3 (n=506)</th>
<th>Total (n=974)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age (days)</td>
<td>No. dead</td>
<td>Age (days)</td>
<td>No. dead</td>
<td>Age (days)</td>
</tr>
<tr>
<td>1</td>
<td>1 BDy</td>
<td>31-35</td>
<td>224</td>
<td>64</td>
<td>31-35</td>
</tr>
<tr>
<td>2</td>
<td>Yolk sac infection</td>
<td>-</td>
<td>-</td>
<td>7-10</td>
<td>9</td>
</tr>
<tr>
<td>3</td>
<td>Vit-E deficiency</td>
<td>-</td>
<td>-</td>
<td>19-22</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>Coccioidiosis</td>
<td>-</td>
<td>-</td>
<td>56-57</td>
<td>21</td>
</tr>
<tr>
<td>5</td>
<td>Others</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
</tbody>
</table>

BDy = Micrococcus bursae disease.

Infectious Bursal Disease

A total of 30 (27.41%) cockerels were found to be affected by infectious bursal disease (IBD) in two different cockerels farms of whom 71 (7.29%) cases died.

Closely the affected birds showed hemorhage in the thigh and pectoral muscles (Fig 1). There was increased mucosa in the intestine. The bursa of Fabricius was filled with yellowish caseous mass. Hemorhage was observed at the junction between proventriculus and gizzard. Lungs were also congested.

Histopathologically the section of bursa showed dead and pyknotic nuclei of lymphocytes, mainly in medulary area (Fig 2). Some follicles were edematous and some were atrophied (Fig 3). As the advanced stage, liquefacitive necrosis was observed in the medulary area of the follicles (Fig 4). Cystic cavities in the medulary area of the follicles and lymphomas of some follicles surrounding the hyperplasic were found (Fig 4). There was hemorrhage and congestion in the follicles (Fig 2). Section of thymus showed hemorrhage in between the muscle fibers (Fig 5). Section of spleen exhibited acrasia of lymphocytes in the white and red pulp. Section of liver showed lymphocytic infiltration in some portal areas (Fig 6). Section of lungs showed broncho-interstitial pneumonia, bronchitis and pleuritis characterized by haemorrhage, congestion and infiltration of lymphocytes, neutrophils, macrophages and plasma cells in the wall of bronchiole, bronchiol, alveoli and pleura (Fig 6). Section of kidney showed severe hemorrhage and congested blood vessels (Fig 6). Section of heart exhibited high peak colored edematous fluid with fibrin network and congested blood vessels in the myocardium. Section of proventriculus exhibited necrosis of the secondary duct of the follicles, congested intercellular tissue blood vessels with thickened walls.

The gross and microscopic lesions noted in IBD are in agreement with those described by Helbigold and Garner (1964) and Chandra et al. (1996). However, fibrin necrosis in myocardium and severe haemorrhage and congestion in lungs were noted macroscopically in some cases in the present investigator which was not reported earlier by other authors.

Yolk sac infection

During 7 to 10 days of age (0.62%) cockerel chicks died due to cannibalism or yolk sac infection in farm no. 2. Karnal and Hinous (1992) recorded yolk sac infection or amphilithias in 12.54% cases. Mayes (1987) reported 0.1 to 7% cases and Byrne and Lowndes (1975) recorded 4.5% cases in chickens against 0.62% cases in cockerels as observed in this study.

At necropsy, all the birds appeared to be highly emaciated and cachectic. Lesions were mainly observed in the yolk sac, which was considerably thickened with unabsorbed yolk in all chicks (Fig 9). The wall of the yolk sac

34
Fig. 1. IBD affected ileum showing hemorrhage in thigh muscle and bursa.

Fig. 2. IBD affected bursa of a ileum showing dead and pyknotic nuclei of lymphocytes with the presence of hemorrhage in the follicles (H & E, x 84).

Fig. 3. IBD affected bursa showing intercellular edema and caseous necrosis in the follicles (H & E, x 333).

Fig. 4. IBD affected bursa showing cystic cavities in the medullary area and liquefaction necrosis of lymphocytes in the follicle and hemopoietic cellular infiltration in and around the necrotic follicles (H & E, X 64).

Fig. 5. IBD affected thigh muscle showing hemmorhage in between the muscle fibres (H & E, X 84).

Fig. 6. IBD affected liver of a ileum bird showing lymphocytic infiltration in portal area (H & E, X 64).

35
Fig. 7: MBD affected liver showing hemorrhages, congestion and different types of reactive cells (lymphocytes, plasma cells, neutrophils and macrophages) in the wall of blood vessels and sinusoids (H & E, X 54).

Fig. 8: MBD affected kidney of a cockerel and showing severe hemorrhage and congested blood vessels (H & E, X 84).

Fig. 9: Thickened with absorbed yolk in Yolk sac infection caused by E. coli in a 8-day-old cockerel chick.

Fig. 10: Thickening of the wall of E. coli infected yolk sac with fibroblastic proliferation and mononuclear cellular infiltration (H & E, X 333).

Fig. 11: Vitamin E deficient brain of a cockerel showing huge number of giant cells and some vacuoles around glial cells (H & E, X 333).

Fig. 12: Coccioidoites alveolaris oocysts of a cockerel showing numerous saccoids and meningoites in the epithelium of the villi, hemorrhage and congestion, infiltration of leukocytes and desquamated epithelium (H & E, X 333).
was inflamed, thickened and edematous. There were congested blood vessels around the yolk. The yolk sac contents appeared to be chesty, yellow-brown in color. The livers of the few affected chicks were pale. In the nutrient agar media, the organism of yolk sac contents produced convex, smooth, and colorless colonies. In the EMB agar media, the yolk sac isolate produced smooth colonies with metallic sheen. So it was suspected that cockerels were died due to yolk sac infection caused by E. coli in farm no. 2.

Microscopically, the wall of the inflamed yolk sac was thickened due to fibroelastic proliferation and mononuclear infiltration in association with normal fat cells (Fig. 10). Section of liver showed necrosis of hepatocytes with distortion of hepatic cords. Sections of lungs revealed reactive cells (heterophils and lymphocytes) in the alveoli.

The gross and microscopic lesions noted in yolk sac infection were similar as those described by Calnek et al. (1997) and Kamal and Hossian (1992). However, necrosis in liver and fibroelastic proliferation and infiltration of mononuclear cells in lumen of alveoli were noted microscopically in some cases in the present study, which was not reported earlier by the other authors.

Vitamin-E deficiency

This study revealed that 7 of 7 (0.72%) birds died due to Vitamin-E deficiency in farm no. 2 and 3 at 13 to 22 days of age. Talha et al. (2001) reported 2.89% cases of birds died due to Vitamin-E deficiency disorder. Bhattacharjee et al. (1996) reported 8.22% cases related to malnutrition.

On postmortem examination, Vitamin-E deficient birds exhibited softened, swollen and focal haemorrhage on the cerebellum, edematous meninges and also congested cerebral blood vessels indicating characteristic encephalomalacia. Microscopically there was proliferation of huge number of glial cells and some vacuoles were found around the glial cells in the brain (Fig. 11). The blood vessels were markedly congested.

The clinical signs exhibited by the Vitamin-E deficient cockerels and associated pathologic lesions in the present study in this malady are almost similar to those reported by Sarker (1976) and Talha et al. (2001).

Coccidiosis

An outbreak of coccidiosis was recorded in farm no. 2. The total number of cockerels in this farm was 250 and disease appeared at 56 to 57 days of age. A total of 2 (0.21%) cockerels died due to coccidiosis out of 21 affected cockerels. Kamal and Hossian (1992), Bhattacharjee et al. (1996) and Talha et al. (2001) recorded 17.36%, 9.40% and 5.15% mortality of chickens due to coccidiosis. In the present study, this occurrence was recorded as 2.16% in cockerels. The incidence of coccidiosis reported in this study and previous reports indicates that coccidiosis is decreasing in Bangladesh. This might be due to the awareness of the farmers and routine use of coccidiostat in their farms.

Gross changes observed at necropsy in all the affected cockerels were confined to the cecaum. The affected ceca were swollen and haemorrhagic, the walls of ceca appeared thickened and firmer in consistency; the lumen of ceca was filled with blood tinged exudate. The semi-solid contents were mixed up with blood and blood stained necrotic tissue debris. The examination of the scraping taken from the affected cecal wall revealed the presence of large number of oocysts under light microscope in all the cases. Moreover, smear made with cecal contents showed the presence of large number of oocysts.

Microscopically the section of the cecal showed haemorrhage, congestion, necrosis and varying degrees of inflammatory reaction. The lining of the villi exhibited necrosis, distorsion and result in the denudation of the cecal villi. The affected crypts and villi lined with hyperplastic, tall columnar epithelial cells contained large number of schizonts and merozoites (Fig. 12). The mucosa of the affected ceca was infiltrated with lymphocytes, heterophils, eosinophils and plasma cells. There was disruption of glandular. There were also presence of oocytes, spermatozoids and merozoites within and outside the epithelia. Lumen of the ceca filled with desquamated tissue debris, clotted blood and numerous schizonts, merozoites and sporozoites of Eimeria.

The gross and microscopic changes observed in this study due to coccidiosis are in conformity with the earlier reports of Sill et al. (2002) and Talha et al. (2001).

Miscellaneous disease condition

In the present investigation, the cause of death of one case (0.01%) from farm no. 1 could not be ascertained, so, grouped into miscellaneous condition. At necropsy, there was no gross lesion. Microscopically, section of the lungs exhibited focal infiltration of mononuclear cells and lymphocytes in the alveoli, bronchi and in the interstitium and some perivascularly filled with exudate and reactive cells. Section of liver showed vacuolar degeneration, congested sinusoid and reactive cells in hepatic parenchyme and in the lumen of blood vessels. There was also infiltration of inflammatory cells in myocardium and in the wall and lumen of the blood vessels.

37
REFERENCES