

DISEASES ASSOCIATED WITH MORTALITY AND PATHOLOGICAL CHANGES IN COCKEREL BIRDS

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ABSTRACT

The diseases associated with mortality and pathologic changes were studied in three different cockerel farms in the rural area of Trishal upazilla in Mymensingh district from day-old chicks up to 60 days of marketing during the period from 5th August to 3rd 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), yolk sac infection, vitamin E deficiency, coccidiosis and others were diagnosed. The mortality rate in cockerels caused by IBD, yolk sac infection, vitamin E deficiency, coccidiosis and others were recorded as 7.29%, 0.62%, 0.72%, 0.21% and 0.10% respectively. The characteristic lesions of IBD were hemorrhage in thigh and pectoral muscles grossly and microscopically the bursa showed dead and pyknotic nuclei of lymphocytes. The typical lesions of yolk sac were thickened with unabsorbed yolk in all chicks grossly and microscopically thickened yolk sac due to fibroblastic proliferation and mononuclear infiltration in association with normal fat cells. The vitamin E deficiency was diagnosed grossly by softened, swollen and focal hemorrhage on the cerebellum and microscopically by proliferation of huge number of glial cells. Grossly coccidiosis was characterized by swollen, thickened, firmer and hemorrhagic caeca and microscopically by hyperplastic, tall columnar epithelial cells contained large number of schizonts and merozoites in the affected crypts and villi. The findings indicate that IBD is the major disease problem in cockerel farming in Mymensingh.

Key words: Mortality, diseases, pathological changes, cockerels

INTRODUCTION

The small poultry farmers in Bangladesh usually prefer to cockerel farming due to low cost of day-old chicks, required less floor space and feed, higher price of cockerel meat than the broiler meat 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 dishearted when there is great mortality of cockerels due to disease outbreak. We have reported the occurrence of diseases associated with morbidity and mortality in cockerels and its relationship with the management (Sil *et al.*, 2002) and this paper describes the mortality and pathological changes of major diseases of cockerels.

MATERIALS AND METHODS

This study was conducted at three different cockerel farms (n = 974 birds) in the rural area of Trishal upazilla in Mymensingh district from 5th August to 3rd October 2000. This investigation was performed from day-old chicks up to marketing (60 days) of the cockerels. The morbidity, mortality, age of affection of various diseases / conditions were recorded. The dead birds (n = 87) were collected for necropsy and the diagnosis of different diseases was based on the history, clinical signs and characteristic gross as well as microscopic tissue alterations as described by Calnek *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 some 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 Luna (1968). In addition, some tissue sections were subjected to Gram's staining and PAS staining whenever necessitated. Smears made from fecal contents and / or caecal scrapings were observed under microscope for detection of coccidial / protozoal oocysts.

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The suspected sample was collected aseptically 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 agar 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

S/N	Diseases	Farm 1 (n = 224)			Farm 2 (n = 250)			Farm 3 (n = 500)			Total (n = 974)	
		Age (days)	No. affected	No. died	Age (days)	No. affected	No. died	Age (days)	No. affected	No. died	Morbidity (%)	Mortality (%)
1	IBD	31-35	224	64	-	-	-	31-35	43	7	267 (27.41)	71 (7.29)
2	Yolk sac infection	-	-	-	7-10	9	6	-	-	-	009 (00.92)	06 (0.62)
3	Vit-E deficiency	-	-	-	19-22	11	5	13-14	6	2	017 (01.75)	07 (0.72)
4	Coccidiosis	-	-	-	56-57	21	2	-	-	-	021 (02.16)	02 (0.21)
5	Others	4	1	1	-	-	-	-	-	-	001 (00.10)	01 (0.10)

IBD = Infectious bursal disease.

Infectious bursal disease

A total of 267 (27.41%) cockerels were found to be affected by infectious bursal disease (IBD) in two different cockerels farms of which 71 (7.29%) cases died.

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

Microscopically the section of bursa showed dead and pyknotic nuclei of lymphocytes, mainly in medullary area (Fig. 2). Some follicles were edematous and some were atrophied (Fig. 3). At the advanced stage, liquefaction necrosis was observed in the medullary area of the follicles (Fig. 4). Cystic cavities in the medullary area of the follicles and ghosts of some follicle surrounding the heterophils were found (Fig. 4). There was haemorrhage and congestion in the follicles (Fig. 2). Section of thigh muscles showed haemorrhage in between the muscle fibres (Fig. 5). Section of spleen exhibited necrosis of lymphocytes in the white and red pulps. 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, bronchi, alveoli and pleura (Fig. 7). Section of kidneys showed severe haemorrhage and congested blood vessels (Fig. 8). Section of heart exhibited light pink 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 interfollicular blood vessels with thickened walls.

The gross and microscopic lesions noted in IBD are in agreement with those described by Helmboldt and Garner (1964) and Choudhury *et al.* (1996). However, fibrin network in myocardium and severe haemorrhage and congestion in kidneys were noted microscopically in some cases in the present investigation which was not reported earlier by other authors.

Yolk sac infection

During 7 to 10 days of age 6 (0.62%) cockerel chicks died due to omphalitis or yolk sac infection in farm no. 2. Kamal and Hossain (1992) recorded yolk sac infection or omphalitis in 12.54% cases, Mayes (1987) recorded 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 are mainly observed in the yolk sac, which was considerably thickened with unabsorbed yolk in all chicks (Fig. 9). The wall of the yolk sac

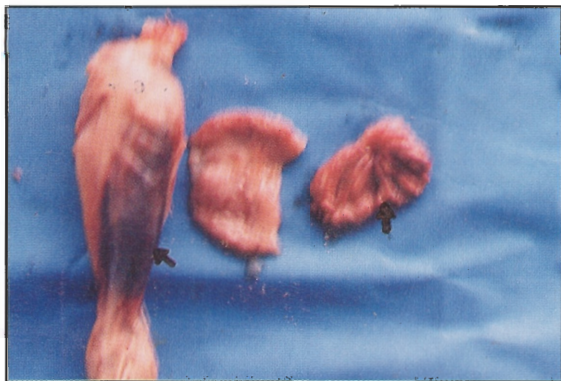


Fig. 1. IBD affected cockerel showing hemorrhage in thigh muscle and bursa.

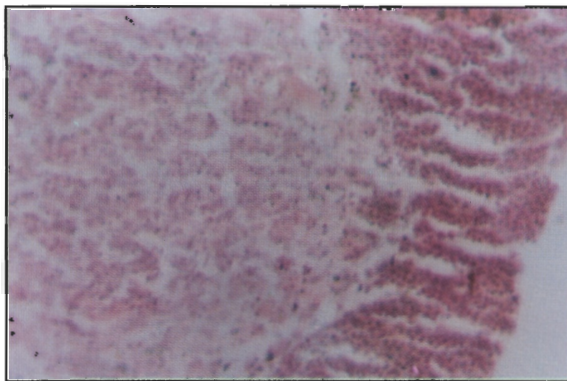


Fig. 2. IBD affected bursa of a cockerel showing dead and pyknotic nuclei of lymphocytes with the presence of hemorrhage in the follicles (H & E, X 84).

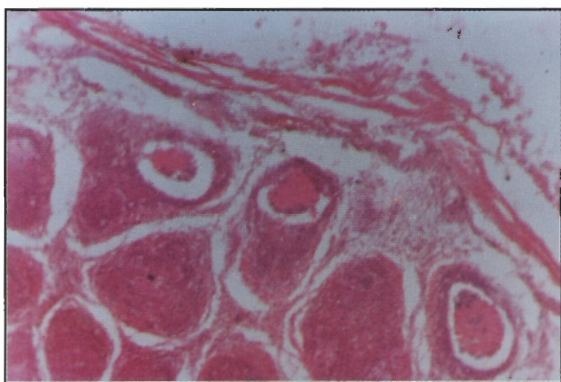


Fig. 3. IBD affected bursa showing interfollicular edema and caseous necrosis in the follicles (H & E, X 333).

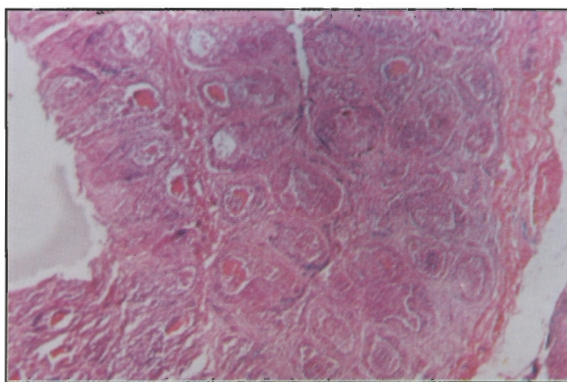


Fig. 4. IBD affected bursa showing cystic cavities in the medullary area and liquefaction necrosis of lymphocytes in the follicles and heterophilic cellular infiltration in and around the necrosed follicles (H & E, X 84)

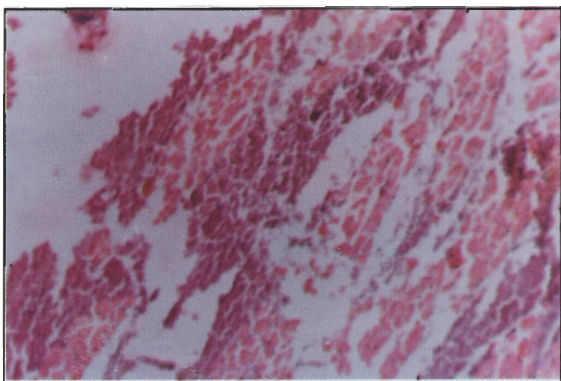


Fig. 5. IBD affected thigh muscle showing hemorrhages in between the muscle fibres (H & E, X 84).

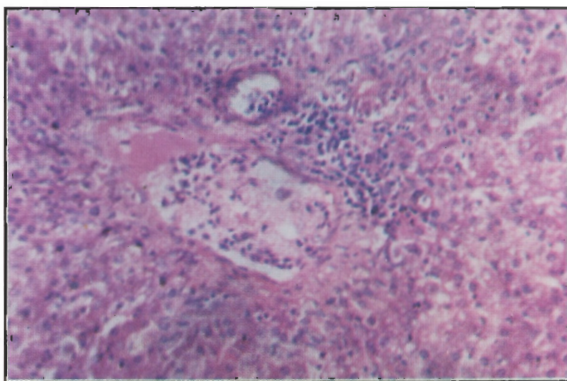


Fig. 6. IBD affected liver of a cockerel bird showing lymphocytic infiltration in portal area (H & E, X 84).

Mortality and pathological changes in cockerels

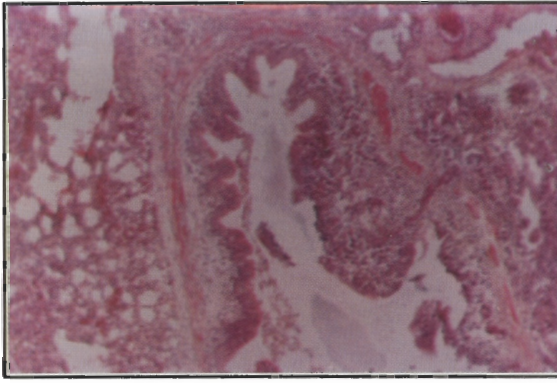


Fig. 7. IBD affected lung showing hemorrhages, congestion and different types of reactive cells (lymphocytes, plasma cells, neutrophils and macrophages) in the wall of bronchiole and alveoli (H & E, X 84)

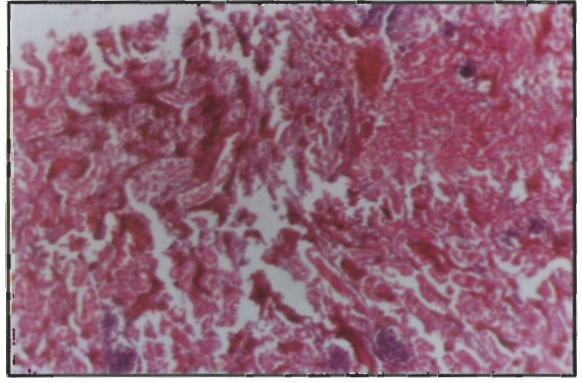


Fig. 8. IBD affected kidney of a cockerel bird showing severe hemorrhage and congested blood vessels (H & E, X 84).



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

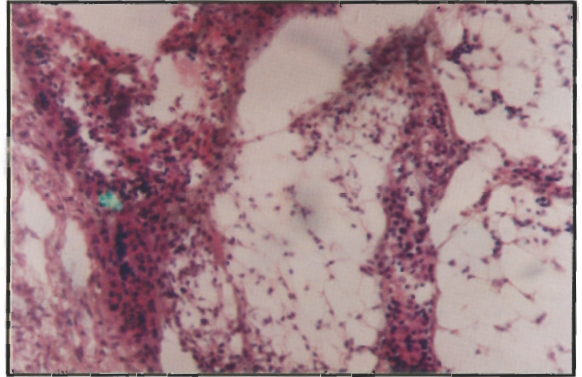


Fig. 10. Thickening of the wall of *E. coli* infected yolk sac with fibroblastic proliferation and mononuclear cellular infiltration (H & E, X 333).

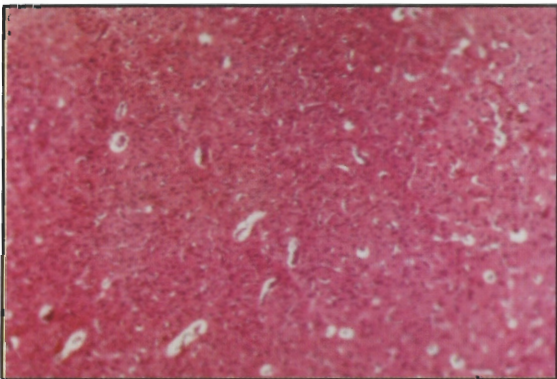


Fig. 11. Vitamin E deficient brain of a cockerel showing huge number of glial cells and some vacuoles around glial cells (H & E, X 333).

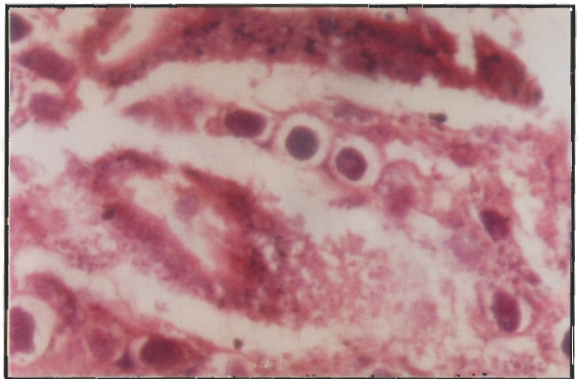


Fig. 12. Coccidiosis affected cecum of a cockerel showing numerous scizonts and merozoites in the epithelia of the villi, hemorrhage and congestion, infiltration of leukocytes and desquamation of epithelia (H & E, X 333).

was inflamed, thickened and edematous. There were congested blood vessels around the yolk. The yolk sac contents appeared to be cheesy, 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 infected yolk sac was thickened due to fibroblastic proliferation and mononuclear infiltration in association with normal fat cells (Fig. 10). Section of liver showed necrosis of hepatocytes with distortion of hepatic cords. Section of lungs revealed reactive cells (heterophils and lymphocytes) in the alveoli.

The gross and microscopic lesions noted in yolk sac infection were similar to those described by Calnek *et al.* (1997) and Kamal and Hossain (1992). However, necrosis in liver and fibroblastic 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 a total 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 recorded 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 Hossain (1992), Bhattacharjee *et al.* (1996) and Talha *et al.* (2001) recorded 17.36%, 9.40% and 5.51% 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 coccidiostats in their farms.

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

Microscopically the section of the carcass showed haemorrhage, congestion, necrosis and varying degrees of inflammatory reaction. The lining of the villi exhibited necrosis, distortion and resulted in the denuding of the caecal 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 caeca was infiltrated with lymphocytes, heterophils, eosinophils and plasma cells. There was disruption of caecal glands. There were also presence of oocysts, sporozoites and merozoites within and outside the epithelia. Lumens of the caeca 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 Sil *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 marked infiltration of mononuclear cells and lymphocytes in the alveoli, bronchi and in the interstitium and some parabronchioles filled with exudate and reactive cells. Section of liver showed vacuolar degeneration, congested sinusoid and reactive cells in hepatic parenchyma 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.

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