

## **Prevalence and pathology of colibacillosis in broiler farms at Dinajpur Sadar Upazila**

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### **Abstract**

This study was carried out to investigate the prevalence and pathological lesions of avian colibacillosis in commercial broiler farms at Dinajpur Sadar Upazila (Sub-district) from July to December 2018. A total of 8800 birds from six farms were diagnosed with colibacillosis from clinical signs, post-mortem lesions and histopathological changes. Highest prevalence was in F1 farm (76.2%) and lowest in F6 farm (9.2%). Highest mortality was in F1 farm (9.1%) and lowest in F6 farm (3.3%). Average prevalence was 39.6% and mortality 5.4%. Highest prevalence was at 0-2 weeks of age (69.3%) and lowest at 2-4 weeks of age (9.6%). The most common gross lesions were air sacculitis, omphalitis, pericarditis, perihepatitis, peritonitis and enteritis. The microscopic lesions were haemorrhage and congestion in lung, destruction of intestinal wall and reactive cell infiltration, and thickening of fibrous tissue in pericardium. (*Bangl. vet.* 2021. Vol. 38, No. 1 - 2, 10 – 16)

### **Introduction**

Broiler farming in Bangladesh is a growing industry and plays an important role in the rural development. More than 130 hatcheries produce 3.4 million day-old chicks per week and about 30,000 commercial broiler and layer farms supply 0.3 million tonnes of poultry meat and 5,210 million eggs per year (Rahman, 2003a). One of the major constraints in the development of broiler industry in Bangladesh is disease, which causes about 30% mortality of chickens (Ali, 1994). Bacterial diseases, including colibacillosis, pose a serious threat in Bangladesh.

Colibacillosis in chickens refers to any local or systemic infection caused entirely or partly by *E. coli* strains (Barnes *et al.*, 2003; Ewers *et al.* 2003). It is one of the main causes of economic losses in the poultry industry worldwide (Yogarathnam, 1995; Ewers *et al.*, 2003) with clinical manifestations such as pericarditis, perihepatitis, airsacculitis, peritonitis, salpingitis, panophthalmitis, omphalitis, cellulitis, colisepticaemia, coligranuloma and swollen-head syndrome (Saif *et al.*, 2003).

Faecal contamination of egg may result in the penetration of *E. coli* through the shell membrane and may spread to the chickens during hatching and is often associated with high mortality rates, or it may give rise to yolk sac infection. Day-old chicks may become infected via the yolk sac, but in older chicks the infection is considered to be

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mainly airborne. Broiler chickens up to three weeks of age are highly susceptible to the disease, but chickens of four weeks and older are considered quite resistant to primary colibacillosis (Goren, 1978). Various risk factors may increase the susceptibility of broilers to colibacillosis, e.g., respiratory viruses and environmental factors like dust and high concentrations of ammonia and carbon dioxide. In addition, the *E. coli* concentration in the air of the broiler house is an important factor. Airborne dust particles in broiler houses can contain 10<sup>5</sup>-10<sup>6</sup> *E. coli* bacteria/g and these bacteria may persist for long periods (Harry and Hemsley, 1965).

Broilers suffering from colibacillosis are depressed, and show respiratory distress and growth retardation. Mortality usually remains below 5%, but morbidity often reaches more than 50% (Wray *et al.*, 1996; Vandekerckhove *et al.*, 2004). In severe cases, mortality may reach up to 94% (Haider *et al.*, 2003; Biswas *et al.*, 2006; Roy *et al.*, 2006). There is paucity of reports on colibacillosis in broilers in Dinajpur. The present study was undertaken to study the prevalence of colibacillosis in commercial broiler farms at Dinajpur Sadar Upazila with clinical signs and gross and histopathological alterations, in order to improve management systems.

## **Materials and Methods**

### ***Experimental area and period***

The data were collected from six different farms of Dinajpur Sadar Upazila (Sub-district), and post-mortem examination was done in infected birds. Gross and histopathological studies were conducted from July to December 2018.

### ***Experimental birds***

A total of 8800 birds were observed. The birds were divided into three groups: 0-2 weeks, 2-4 weeks and >4 weeks of age. Some randomly selected sick and dead birds from each group were collected for post-mortem examination.

### ***Diagnosis of the disease***

Primarily the disease was diagnosed on the basis of history and clinical signs as described by the owner, and the laboratory diagnosis was done by necropsy examination of samples of liver, lung and heart. Gross tissue changes were observed, and samples were preserved in 10% formalin for histopathological studies. Formalin-fixed tissue samples were processed for paraffin embedding, sectioned and stained with haematoxylin and eosin according to standard method (Luna, 1968).

## **Results and Discussion**

### ***Prevalence of colibacillosis***

Six broiler farms of Dinajpur Sadar Upazila were visited on the basis of clinical signs. The number of infected and dead birds were recorded to determine the prevalence and mortality. Overall prevalence of colibacillosis was 39.6% and mortality 5.4%

(Table 1). Highest prevalence was in F1 at 76.2% and lowest in F6 (9.2%). The highest mortality was in F1 (9.1%) and lowest (3.3%) in F6. These results support the reports of Rahman *et al.* (2004) who reported 67.7% and reports of Suha *et al.* (2008) who reported 43.5% morbidity in commercial broiler and layer farms. These results also support the reports of Hossain *et al.* (2008) who reported 60% morbidity in commercial broiler and layer birds. Bhattachajee *et al.* (1996) reported 40.8% and Ahmed *et al.* (2009) reported 52.3% prevalence of *E. coli* in chickens from Bangladesh. Table-2 presents the age-wise prevalence of colibacillosis in infected flocks: highest prevalence was at 0-2 weeks at (69.3%), and lowest at > 4 weeks (9.6%). Similarly, Talha *et al.* (2001) reported higher prevalence of colibacillosis in growing chickens in comparison to adults whereas Bhattachajee *et al.* (1996) reported high prevalence of colibacillosis in both the brooding (12.8%) and layer chickens (5.5 to 8.8%), and this study recorded high prevalence of *E. coli* infection in all age groups of chickens (9.5 to 36.7%).

Table 1: Prevalence of colibacillosis in broiler farms in Dinajpur Sadar Upazila

Name of farms	Total no of birds	No of infected birds	Prevalence of colibacillosis (%)	No of dead birds	Mortality rates (%)
F1	500	381	76.2	35	9.1
F2	2000	207	10.4	07	3.4
F3	1000	449	44.9	24	5.3
F4	800	520	65.0	41	7.9
F5	1500	483	32.2	16	3.3
F6	3000	275	09.2	09	3.3
Total	8800	2315	39.6	118	5.4

Table 2: Prevalence of colibacillosis on the basis of age group in broiler farms in Dinajpur Sadar Upazila

Age groups (Weeks)	Total no. of birds	No. of infected birds	Prevalence (%)
0.2	1300	901	69.3
2-4	2500	932	37.3
>4	5000	482	9.6

### **Clinical Findings**

Respiratory signs were recorded throughout the study period, including coughing, and sneezing with feather ruffling. Birds also showed weakness, reduced appetite, poor growth, omphalitis, depression, loss of weight, dullness, lethargy, soiling of cloaca with semi-solid cheesy material, and faecal material green or white-yellow. Dehydrated birds had dark dry skin, especially noticeable on shank and feet. Younger birds showed omphalitis (navel/yolk sac infection), distended abdomen and

aggregation near to the source of heat, whereas in older birds, production was lower due to tumours. High mortality was observed in younger birds due to yolk-sac infections and high embryonic mortality was seen in breeder flocks. These clinical signs of colibacillosis were mentioned by Ganapathy *et al.* (2000), Omer *et al.* (2010) and Awa wdeh (2017).

### Gross lesions

During post-mortem, different forms of colibacillosis were observed. Air sacculitis was characterized by caseous exudation in the airsac with thickened and cloudy air sac membranes (Fig. 1). Omphalitis was characterized by unabsorbed yolk sac with viscid, yellow-green to watery, yellow-brown or caseous material (Fig. 2). Fibrinous pericarditis was observed with light yellow and fibrinous exudation in the pericardial sac (Fig. 3). Enteritis was seen in every dead bird, with exudation in intestinal lumen, blackish fluid in intestine and sometimes haemorrhagic and catarrhal exudation in intestinal lumen (Fig. 4). There was often yellowish-white covering on liver as perihepatitis, and peritonitis with fibrin and free yolk sac infection in the peritoneal cavity were common in laying hens. Acute septicaemia was observed with greenish liver and congested pectoral muscle and sometimes small white necrotic foci were found in liver. These forms of colibacillosis were reported by Shah *et al.* (2003); Rahman *et al.* (2004); Landman and Comelissen (2006); Nakamura *et al.* (2007) and Chowdhury *et al.* (2009).



Fig. 1: Air sacculitis characterized by Airsac contain a caseous exudates and the airsac membranes become thicker and cloudy in appearance



Fig. 2: Omphalitis or Yolk sac infection characterized by Unabsorbed yolk sac with viscid, yellowgreen to watery, yellow-brown or caseous material (Black arrow)

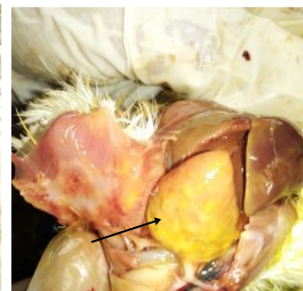


Fig. 3: Fibrinous pericarditis characterized by Light yellow and fibrinous exudates in the pericardial sac (Black arrow)



Fig. 4: Enteritis characterized by (A) Deposition of exudation of fluid in intestine (Black arrow), (B) Blackish deposition of fluid in intestine (Black arrow) (C) Haemorrhage with Catarrhal exudation in intestinal lumen (Black arrow)



### Microscopic lesions

Haemorrhage and congestion in lungs, thickening of fibrous tissue in pericardium and destruction of intestinal wall with reactive cell infiltration (Fig. 5) were recorded. Similar lesions were observed by Talha *et al.* (2001); Islam *et al.* (2003); Nakamura *et al.* (2007) and Khaton *et al.* (2008).

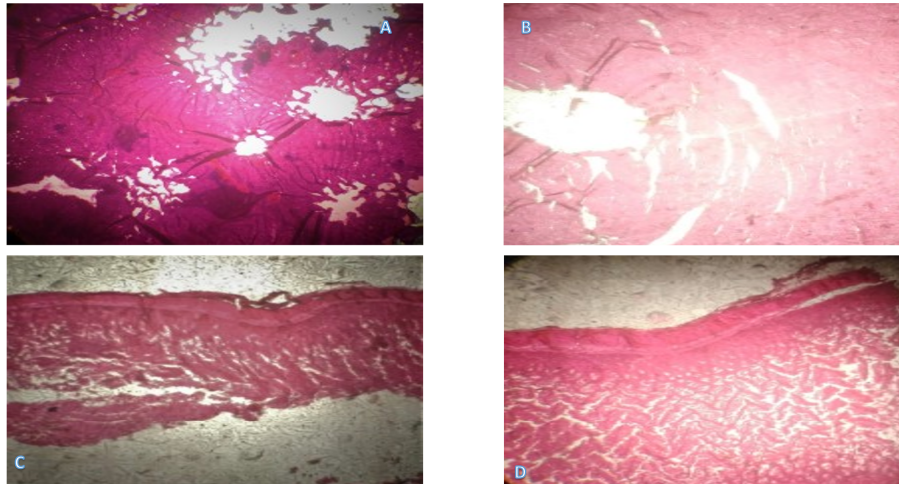


Figure.5 Microscopic Lesions found in different organs associated with colibacillosis (A) hemorrhage and congestion in lung, (B) thickening of fibrous tissue in pericardium due to pericarditis, (C) and (D) Destruction of Intestinal wall with reactive cell infiltration

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