Bangl. J. Vet. Med. (2004). 2 (2): 91–98

IMPORTANT SYSTEMIC AND MISCELLANEOUS DISEASES ASSOCIATED WITH MORBIDITY AND MORTALITY IN COMMERCIAL POULTRY IN BANGLADESH

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ABSTRACT

History and necropsy methods were used to study the systemic and miscellaneous diseases among 1751 moribund and dead Commercial chickens submitted for diagnosis at the BRAC Poultry Disease Diagnostic Centre, Gazipur, during one year period from January to December 2002. Of the 1751 chickens examined, 554 (31.64%) birds were affected with 15 different types of systemic and miscellaneous disorders. These included omphalitis (5.25%), enteritis (9.08%), pneumonia (0.80%), egg peritonitis (2.74%), egg bound (8.28%), salpingitis (0.40%), malabsorption syndrome (0.17%), ascites (0.74%), visceral gout (0.86%), neoplastic disease (1.54%), hydropericardium-hepatitis syndrome (0.23%), big liver and spleen disease (0.06%), fatty liver-haemorrhagic syndrome (0.80%) and cannibalism (0.17%). The possible etiology, single and concurrent occurrences influence of age and seasons on the occurrence of these disorders are described and discussed. It may be concluded from this study that the systemic and miscellaneous disorders are associated in the high rate of morbidity and mortality in commercial poultry with possible complex etiology. Thus further detailed studies are needed to understand the role of etiology in these disease process which would help in designing logical control strategies.

Key words: Morbidity, mortality, poultry, systemic, diseases

INTRODUCTION

The infectious diseases mainly caused by bacteria and viruses have been recognized as outbreak forms associated with high morbidity and mortality in commercial poultry worldwide including Bangladesh (Calnek *et al.*, 1997; Samad, 2000). But the reports on systemic and miscellaneous diseases are very limited in literature because of low prevalence, mild signs and lesions, lack of diagnostic techniques. In addition, there are disease conditions that have multifactorial etiologies including combinations of microbes and microbes plus nutritional or environmental factors. There are no published reports on the occurrence of systemic and miscellaneous diseases in poultry from Bangladesh. This paper reports some important systemic and miscellaneous diseases and disorders in commercial poultry birds.

MATERIALS AND METHODS

This study was conducted on the moribund and / or dead chickens submitted for the diagnosis of diseases at the Bangladesh Rural Advancement Committee (BRAC) Poultry Disease Diagnostic Centre, Nagapara, Gazipur during one year period from January to December 2002. During this one year period, a total of 1751 cases of birds were diagnosed, of which 551 (31.47%) cases were recognized as systemic and miscellaneous diseases on clinical history and post mortem examination as described by Calnek *et al.* (1997). The influence of age, types of birds (brooding, growing, pullet and layers) and seasons on the occurrence of these disorders were analyzed.

RESULTS AND DISCUSSION

The single and mixed, age-wise and season-wise occurrence of systemic and miscellaneous diseases in commercial chickens are presented in Table 1, 2 and 3, respectively.

Omphalitis / yolk sac infection

Omphalitis is characterized by inflammed swollen navel with abnormal yolk material and peritonitis which was recorded in 92 (5.25%) chickens of which 88 (95.65%) as single disease and 4 (4.37%) as dual infection (Table 1). The occurrence of omphalitis in 5.25% chickens supports the report of Al-Sadi *et al.* (2000) who reported 4.0% incidence of omphalitis in chickens, but higher prevalence rate of omphalitis have been reported elsewhere (Ghodasara *et al.*, 1992; Venkanagouda *et al.*, 1996). Significantly ($p < 0.01$) higher prevalence rate was recorded during winter (55.43%) in comparison to summer (22.83%) and rainy (21.74%) seasons (Table 3).

Enteritis

Inflammation of the intestine is called enteritis, which is characterized clinically by diarrhoea. The enteritis cases from which the specific agents or disease could not be recognized are categorized as non-specific enteritis. These non specific enteritis cases were recorded in 159 (9.08%) chickens, of which 89 (55.97%) had single, 60 (37.74%) had dual, 8 (5.08%) had triple and 2 (1.26%) had four types of diseases (Table 1).

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Table 1. Occurrence of single and concurrent systemic and miscellaneous diseases in commercial chickens

S / N	Diseases	Number of cases (%)				
		Single type	Two type	Three type	Four type	Total
1.	Omphalitis	88 (95.65)	04 (4.37)	–	–	92 (5.25)
2.	Enteritis	89 (55.97)	60 (37.74)	08 (5.08)	02 (1.26)	159 (9.08)
3.	Pneumonia	10 (71.43)	04 (28.57)	–	–	14 (0.80)
4.	Arthritis and tenosynovitis	06 (100.00)	–	–	–	06 (0.34)
5.	Egg peritonitis	23 (47.92)	24 (50.00)	01 (2.08)	–	48 (2.74)
6.	Egg bound	31 (21.38)	100 (68.97)	10 (6.90)	04 (2.76)	145 (8.28)
7.	Salpingitis	05 (71.43)	02 (28.57)	–	–	07 (0.40)
8.	Malabsorption syndrome	03 (100.00)	–	–	–	03 (0.17)
9.	Ascites	07 (53.85)	04 (30.77)	02 (15.38)	–	13 (0.74)
10.	Visceral gout	08 (53.33)	07 (46.67)	–	–	15 (0.86)
11.	Neoplastic diseases	19 (70.37)	07 (25.93)	01 (3.70)	–	27 (1.54)
12.	HHS	02 (50.00)	02 (50.00)	–	–	04 (0.23)
13.	Big liver and spleen diseases	01 (100.00)	–	–	–	01 (0.06)
14.	FLHS	05 (35.71)	03 (21.43)	02 (14.29)	04 (28.57)	14 (0.80)
15.	Cannibalism	03 (100.00)	–	–	–	03 (0.17)
	Systemic diseases (Total)	**300 (54.45)**	**217 (39.38)**	**24 (4.36)**	**10 (1.81)**	**551 (31.47)**

HHS = Hydropericardium-hepatitis syndrome, FLHS = Fatty liver- haemorrhagic syndrome.

Table 2. Age-wise occurrence of systemic and miscellaneous diseases in commercial chickens

S / N	Diseases	Number of cases (%)				
		Brooding upto 2 wks (n = 215)	Growing (>2-8 wks) (n = 421)	Pullet (>8-20 wks) (n = 318)	Layer (>20 wks) (n = 797)	Total (n = 1751)
1.	Omphalitis	90 (97.83)	02 (2.17)	–	–	92 (5.25)
2.	Enteritis	04 (2.52)	44 (27.67)	27 (16.98)	84 (52.83)*	159 (9.08)
3.	Pneumonia	07 (50.00)	05 (35.71)	–	02 (14.29)	14 (0.80)
4.	Arthritis and tenosynovitis	01 (16.67)	–	01 (16.67)	04 (66.67)	06 (0.34)
5.	Egg peritonitis	–	–	05 (10.42)	43 (89.58)	48 (2.74)
6.	Egg bound	–	–	05 (3.45)	140 (96.55)	145 (8.28)
7.	Salpingitis	–	–	02 (28.57)	05 (71.43)	07 (0.40)
8.	Malabsorption syndrome	–	–	03 (100.00)*	–	03 (0.17)
9.	Ascites	–	08 (61.54)	–	05 (38.46)	13 (0.74)
10.	Visceral gout	–	03 (20.00)	06 (40.00)	06 (40.00)	15 (0.86)
11.	Neoplastic diseases	–	–	11 (44.00)	14 (56.00)	27 (1.54)
12.	HHS	–	03 (75.00)	–	01 (25.00)	04 (0.23)
13.	Big liver and spleen diseases	–	–	01 (100.00)	–	01 (0.06)
14.	FLHS	–	01 (7.14)	01 (7.14)	12 (85.71)	14 (0.80)
15.	Cannibalism	–	–	–	03 (100.00)	03 (0.17)
	Systemic diseases (Total)	**102 (18.51)**	**67 (12.16)**	**61 (11.07)**	**321 (58.26)**	**551 (31.47)**

*Differed significantly at ($p < 0.01$), HHS = Hydropericardium-hepatitis syndrome, FLHS = Fatty liver-haemorrhagic syndrome.

Table 3. Season-wise clinical occurrence of systemic and miscellaneous diseases in commercial chickens

S/N	Diseases	Number of cases (%)			
		Summer (March-June) (n = 748)	Rainy (July-Oct) (n = 491)	Winter (Nov-Feb) (n = 512)	Total (n = 1751
1.	Omphalitis	21 (22.83)	20 (21.74)	51 (55.43)*	92 (5.25)
2.	Enteritis	69 (43.40)	45 (28.30)	45 (28.30)	159 (9.08)
3.	Pneumonia	02 (14.29)	02 (14.29)	10 (71.43)	14 (0.80)
4.	Arthritis and tenosynovitis	–	05 (83.33)*	01 (16.67)	06 (0.34)
5.	Egg peritonitis	18 (37.5)*	17 (35.42)	13 (27.08)	48 (2.74)
6.	Egg bound	62 (42.760	62 (42.76)	21 (14.48)	145 (8.28)
7.	Salpingitis	05 (71.43)*	–	02 (28.57)	07 (0.40)
8.	Malabsorption syndrome	–	02 (66.67)*	01 (33.33)	03 (0.17)
9.	Ascites	06 (46.15)*	03 (23.08)	04 (30.77)	13 (0.74)
10.	Visceral gout	03 (20.00)	09 (60.00)	03 (20.00)	15 (0.86)
11.	Neoplastic diseases	15 (55.56)*	10 (37.04)	02 (7.41)	27 (1.54)
12.	HHS	–	–	04 (100.00)	04 (0.23)
13.	Big liver and spleen diseases	01 (100.00)	–	–	01 (0.06)
14.	FLHS	05 (35.71)	08 (57.14)*	01 (7.14)	14 (0.80)
15.	Cannibalism	02 (66.67)*	–	01 (33.33)	03 (0.17)
Systemic diseases (Total)		**209 (37.93)**	**183 (33.21)**	**159 (28.86)**	**551 (31.47)**

*Significantly high ($p < 0.01$), HHS = Hydropericardium-hepatitis syndrome, FLHS = Fatty liver-haemorrhagic syndrome.

The 9.08% proportionate prevalence rate of enteritis recorded in this study supports the report of Ghodasara *et al.* (1992) who reported 6.14% enteritis cases in chicken from India. However, Talha *et al.* (2001) reported only 1.05% proportionate incidence rate of non-specific diarrhoea in chickens from Mymensingh. Necropsy examination of 62-day-old broiler bird infected with colibacillosis showed mucus and haemorrhages in intestine and thickened intestinal wall (Fig. 1)

The occurrence of enteritis steadily increased with the increased age of chickens with highest rate recorded in adult layer (52.83%) chickens (Table 2). Talha *et al.* (2001) also reported enteritis in 4 chickens, of which 2 chickens aged between >2 to 8 weeks and 2 chickens aged >20 weeks old. Prevalence rate of enteritis was recorded highest during summer (43.40%) in comparison to rainy (28.30%) and winter (28.30%) seasons (Table 3).

Pneumonia

Inflammation of the lung parenchyma (pneumonia) was recorded in 14 (0.80%) chickens, of which 10 (71.43%) as single and 4 (28.57%) as concurrent disorders (Table 1). Significantly ($p < 0.05$) highest prevalence rate of pneumonia was observed in brooding chicks (50.0%) in comparison to growing (35.71%) and adult layer (14.29%) chickens (Table 2). Significantly ($p < 0.05$) highest infection rate was recorded during winter (71.43%) in comparison to summer (14.29%) and rainy (14.29%) seasons (Table 3). However, Talha *et al.* (2001) reported higher proportionate incidence rate of 5.77% pneumonia (non-specific) in chickens, of which highest incidence in >2 to 8 weeks old chickens. Necropsy examination of broiler chicken infected with pneumonia showed extensive haemorrhage and white to yellowish nodules scattered over the lungs and trachea (Fig. 2).

Arthritis and tenosynovitis

Arthritis and tenosynivitis was recorded in 6 (0.34%) chickens on clinical and necropsy examination. Necropsy examination of each of the six birds showed swollen hock joints and cut surface showed pus in joint cavity (Fig. 3).

Gram's stained smears from lesions identified the Gram positive typical cocci, arranged in clusters (*Staphylococcus* sp.). All the six affected chickens were recorded as single etiology, and in brooding (16.67%), pullet (16.67%) and adult layer (66.67%) birds (Table 2). Of the six cases, 5 (83.83%) recorded in rainy and 1 (16.6%) in winter season (Table 3). Kibenge *et al.* (1982) also reported the morbidity and mortality due to this disease in broiler birds.

Egg peritonitis

The egg peritonitis was recorded in 48 (2.74%) layer chickens on necropsy examination, which showed rupture of egg in the abdominal cavity, deformed and congested ova and thin shell egg in oviduct and sometimes mucus and haemorrhages in the intestine. Cloacal prolapse showing haemorrhagic lesion and protrusion of cloaca around the vent also recorded (Fig. 4).

Of the 48 cases, 23 (47.92%) occurred as single, 24 (50.00%) as two types and only one (2.08%) as three types. Significant ($p < 0.01$) seasonal influence was recorded in summer (37.5%) on the occurrence of egg peritonitis in chickens (Table 3). The 2.74% prevalence of egg peritonitis recorded in this study supports the report of Bhattacharjee *et al.* (1996) who reported 3.52% proportionate incidence of egg peritonitis in layer chickens. This is the disorder of layer chickens which is uniformly distributed through all the seasons of the year.

Egg bound

Egg bound was diagnosed on necropsy examination of chickens which included lodged egg in the cloaca, thin shell egg in abdominal cavity, haemorrhage around the vent and presence of mature egg in the oviduct (Fig. 5).

The term egg bound (impacted egg) is used to describe a condition in which an egg is lodged in the vagina but can not be laid. It may be result from inflammation of the oviduct, partial paralysis of the muscles of the oviduct or production of an egg so large that it is physically impossible for it to be laid. Young pullets laying an unusually large egg are more prone to the problem. When impaction occurs in the uterus or vagina (which is usually the case) egg enclosed by shell membranes may be found in the abdominal cavity. This indicates that eggs continued to form but were refluxed back into the peritoneal cavity. This disorder was recorded in 145 (8.28%) layer chickens, of which 31 (21.38%) as single, 100 (68.97%) as dual, 10 (6.90%) as triple and 4 (2.76%) as four types of mixed disorders (Table 1). The egg bound problem in layer chickens have also been reported from Bangladesh by Bhattacharjee *et al.* (1996) who reported 2.80% incidence of egg bound problem in layer chickens, respectively.

Salpingitis

Inflammation of the fallopian tubes is called salpingitis which was recorded only in 7 (0.40%) chickens, of which 5 (71.43%) as single and 2 (28.57%) as concurrent disorders (Table 1). Necropsy findings included extremely emaciated body, egg packed in oviduct, thin shell egg in oviduct and occasionally ruptured egg found in the abdominal cavity (Fig. 5). This disorder was recorded only in pullet (28.57%) and adult layer (71.43%) chickens, which was found highest rate during summer (71.43%) than winter (28.57%) season. These findings support the reports of Mukhopadhyay *et al.* (1999).

Malabsorption syndrome

Malabsorption was diagnosed on the basis of clinical history and necropsy examinations. Clinical history included diarrhoea, growth retardation (stunting) and poor feathers. Lesions included cachectic body, intact feed materials and mucus in intestine with catarrhal enteritis and sometime proventriculitis.

Viral enteritis in chickens has been reported from many countries by several different names, including malabsorption syndrome, infectious stunting syndrome, 'broiler stunting syndrome', 'pale bird syndrome' and 'helicopter disease' (Calnek *et al.*, 1997). This syndrome was recorded only in 3 (0.17%) chickens and all had single type of disorder, and recorded only in pullet birds (Table 1 & 2). These findings support the earlier report of Bhattacharjee *et al.* (1996) who reported malabsorption syndrome in 0.19% of chickens at pre-production period.

Ascites

Ascites means dropsical swelling of the abdomen which was recorded only in 13 (0.74%) chickens, of which 7 (53.85%) cases involved single, 4 (30.77%) had dual and 2 (15.38%) had triple types of disorders (Table 1). This disorder was recorded high in rapidly growing (>2 to 8 wks old) chickens (61.54%) in comparison to adult layer (>20 wks old) chickens (38.46%). Although ascites was recorded in all the three seasons of the year but highest prevalence was recorded during summer (46.15%) in comparison to rainy (23.08%) and winter (30.77%). There seems to be no published report on ascites in the inland literature, however, it has been reported elsewhere (Nakamura *et al.*, 1999; Shome *et al.*, 2000).

Although numerous chemical toxicities have been reported to cause ascites in broiler chickens but the most common form of ascites in fast growing broiler chickens is caused by increased hydrostatic vascular pressure. Rapid growth, elevated metabolic rate and therefore, a high oxygen demand impose an increased workload on the heart. This, combined with the insufficient pulmonary capillary capacity of the modern broiler chicken, aggravates the pulmonary hypertension syndrome (PHS) and further precipitates right ventricular hypertrophy. Hypertrophy is soon followed by dilation, right ventricular failure, passive congestion, and then ascites. Any hypertrophy affects the valve and its apposition against the septum, facilitating venous regurgitation, passive congestion and ascites.

Clinically affected broiler chickens were smaller than normal and depressed with ruffled feathers, and severely affected birds showed abdominal distension with reluctance to move, respiratory distress, and cyanosis. Necropsy lesions included hypertrophy and dilatation of the right ventricle with or without accumulation of straw coloured ascetic fluid in the peritoneal cavities, and a generalized passive congestion are characteristic of ascites secondary to PHS. Hydropericardium, protein clots in the ascetic fluid and a fibrotic liver were present depending on the duration of the condition as described by Charlton (2000).

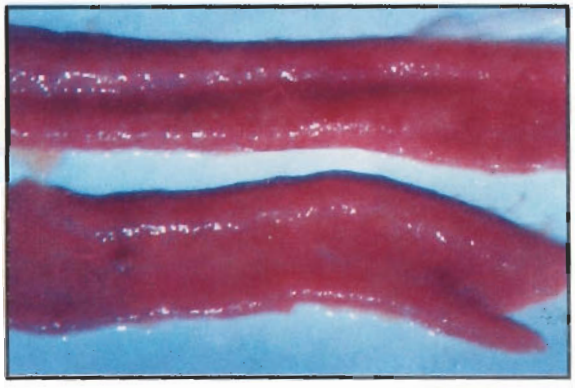

Fig. 1. Cut section of intestine of 62-day old broiler bird experimentally infected with colibacillosis showing mucus and haemorrhage in intestine and thickned intestinal wall.

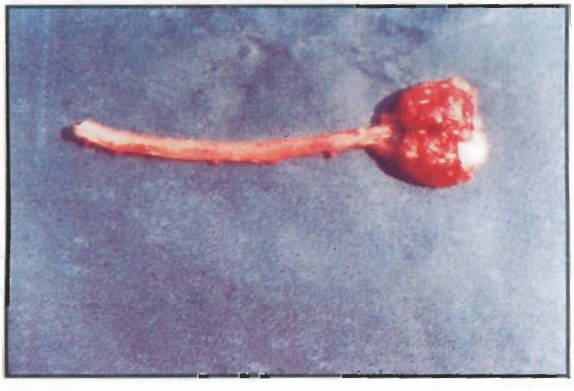

Fig. 2. Trachea and lungs of broiler chicken showing extensive haemorrhage and white to yellowish nodules scattered over the lungs & trachea indicating pneumonia.

Fig. 3. Hock joint of 62-day-old chicken affected with staphylococcosis showing swollen joints and pus (white) in joint cavity on incision.

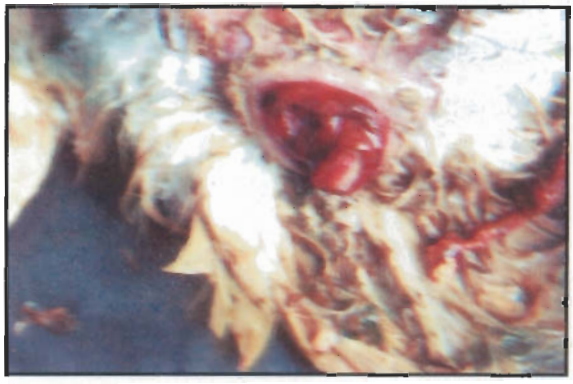

Fig. 4. Cloacal prolapse occur in egg peritonitis showing haemorrhagic lesion and protrusion of cloaca arround the vent.

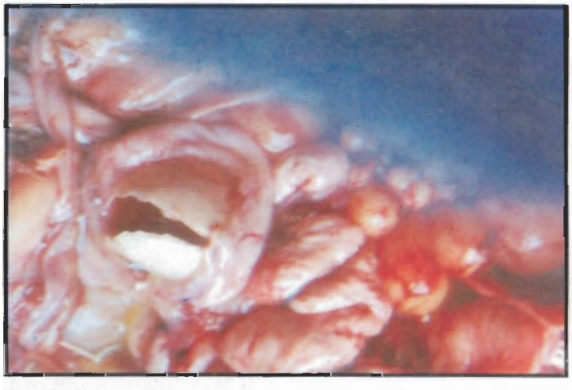

Fig. 5. Internal organs including ova and oviducts of a layer bird affected with salpingitis and egg bound syndrome showing congested ova and oviduct, fully formed cracked shell egg in oviduct, distended oviducts with unlaid caseated egg materials.

Fig. 6. Internal organs of a layers affected with visceral gout showing chalky white material on heart, liver, spleen, lungs in whole visceral organs and in on the ova.

Visceral gout

Visceral gout was diagnosed on necropsy examination of 15 (0.86%) chickens and the lesions showed chalky white crystals of urates especially in the kidney and also in the pericardium, liver, lung, spleen and oviduct (Fig. 6). In addition, kidneys were enlarged and paler than normal. Gout is a common finding during necropsy of poultry which is the result of abnormal accumulation of urates and occurs as two distinct syndromes articular gout and visceral gout. Visceral gout, which has also been called visceral urate deposition, which is characterized by precipitation of urates in the kidneys and on serous surfaces of the visceral organs. As it has been reproduced by feeding high protein diets, it is tempting to infer that it results from excess production of uric acid. Visceral urate deposition is generally due to a failure of urinary excretion, which may be due to obstruction of ureters, renal damage, or dehydration. However, dehydration due to water deprivation is a common cause of visceral gout in domestic poultry. The deposition on serosal surfaces appear grossly as a white chalky coating, while these within viscera may only be recognized microscopically. Of the 15 visceral gout cases, 8 (53.33%) had single and 7 (46.67%) with concurrent disorders (Table 1). Prevalence rate of gout was higher in pullets (40.0%) and adult layers (40.0%) in comparison to growing (20.0%) but nil up to 2 weeks old chickens (Table 2). Highest prevalence rate was recorded during rainy (60.0%) in comparison to summer (20.0%) and winter (20.0%) seasons (Table 3). The proportionate prevalence rate of 0.86% with higher incidence in adult chickens are in conformity with the report of Talha *et al.* (2001) who reported 0.79% incidence rate in >20 weeks old chickens.

Neoplastic diseases

Neoplastic diseases other than avian leucosis were recorded in 27 (1.54%) chickens of which 19 (70.37%) as single, 7 (25.93%) as dual and 1 (3.70%) as triple types of mixed disorders. Neoplastic disease was observed only in chickens >8 to 20 weeks (44.0%) and >20 weeks (56.0%) old chickens, with all the three seasons of the years. The neoplastic diseases have been described elsewhere (Calnek *et al.*, 1997; Charlton, 2000).

Hydropericardium-hepatitis syndrome:

The hydropericardium-hepatitis syndrome (HHS), caused by an adenovirus serotype 4 is an acute infectious disease of chickens, characterized by excess accumulation of fluid in the pericardial sac and multifocal hepatic necrosis which have given rise to the common name of the disease HHS. However, HHS was first recognized in flocks in Angara Goth near Karachi, Pakistan in late 1987 and accordingly the HHS was initially referred to as 'Angara disease' (Anjum *et al.*, 1989; Anjum, 1990; Cheema *et al.*, 1989) and then it has been reported from many countries of the world (Calnek *et al.*, 1997) including Bangladesh (Rahman *et al.*, 2001). The HHS was recorded in 4 (0.23%) broiler birds of which 2 (50%) as single etiology and 2 (50%) as in concurrent infections. The existence of this disease has been reported as outbreak form Bangladesh. The causative adenovirus serotype 4 is also known to cause immunosuppression and concurrent infection might be the cause of increased severity of the disease. The HHS was recorded in growing (75.0%) as well as adult (25.0%) chickens, and only in winter seasons (100%). Although the disease was initially reported in 15 to 35 days old broiler but 4 to 15 weeks old breeder flocks were also affected with this HHS. The vertical transmission of avian adenovirus 4 (AAV-4) has been reported from persistently infected breeder stocks to their progeny, and only pathological investigation of HHS in broiler chickens have been made from Bangladesh (Rahman *et al.*, 2001). Therefore, identification of the causative virus of HHS would be required to detect the exact status of the disease for its prevention under local conditions.

Big liver and spleen disease

The big liver and spleen disease (BLS) was recognized in only one (0.06%) chicken on post mortem lesions which included good body condition with empty crops, and lesions on the enlarged liver and spleen. This BLS case was recorded in pullet of > 8 to 20 weeks old and during summer season (Table 2 & 3). There are no published inland reports to compare this observation but it has been reported in broiler birds elsewhere (C larke *et al.*, 1990; Crerar and Cross, 1994a, 1994b).

Fatty liver-haemorrhagic syndrome

The fatty liver-haemorrhagic syndrome (FLHS) was diagnosed on necropsy examination which showed clotted blood on the liver, congested liver and bleeding from the liver, paleness of the whole body and visceral organ. Excess abdominal fat, enlarged pale and friable liver. In some cases deformed ova in oviduct and ruptured egg were also found. The FLHS was recorded in 14 (0.80%) chickens, of which 5 (35.71%) had single, 3 (21.43%) as dual, 2 (14.29%) as triple and 4 (28.57%) as four types of disorders associated with this syndrome. This syndrome was found highest in adult layers (85.71%) in comparison to pullet (7.14%) and grower (7.14%) chickens. These observations support the earlier report of Bhattacharjee *et al.* (1996) who reported 1.44% incidence of FLHS in adult chickens from Bangladesh. However, this syndrome has been recognized in laying hens in many countries of the

world. It has been reported that excessive consumption of high-energy diets in birds whose exercise is restricted in cages is considered to result in a positive energy balance and excessive fat deposition. The occurrence of this syndrome was recorded significantly ($p < 0.05$) at higher rates during hot weather of rainy (57.14%) and summer (35.71%) in comparison to winter (7.14%) which indicate that excessive fat deposition might be compounded by hot weather. These findings support the observation of Reddy *et al.* (1997). Low calcium levels of dietary calcium may result in increased feed consumption, liver fat and liver haemorrhage (Calnek *et al.*, 1997).

Cannibalism

Cannibalism is also called vices, a definition comprehensive enough to include 'bad habit'. Many forms of cannibalism occur in domestic fowl recorded in captivity, the most common ones being feather pulling, and vent, head and toe picking. Feather pulling occurs in any age of birds and in severe cases bird, may die due to haemorrhage. Cannibalism was recorded in only in 3 (0.17%) birds and all the cases were identified as a single disorder. All the three cases were recorded in layers (>20 wks old) chickens, of which significantly ($p < 0.05$) higher prevalence rate during summer (66.67%) in comparison to winter (33.33%) months. Comparatively higher prevalence rate of cannibalism has been reported by Bhattacharjee *et al.* (1996) who reported 2.01% prevalence rate, of which highest prevalence rate (78.13%) at peak production (>20 wks old) of hens. Many cases of cannibalism have been suggested but conditions reported as predisposing to cannibalism are light intensity, dense stocking, nutritional, vitamin and mineral deficiency, being without feed for too long, insufficient feeder or drinker space, irritation from external parasites etc have all been mentioned (Calnek *et al.*, 1997; Charlton, 2000).

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