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HAEMATOLOGICAL AND PLASMA PROTEIN PROFILE IN FAYOUMI HEN SUFFERING FROM ASCITES

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ABSTRACTS

A 9 months old Fayumi layer, having 2.5 kg body weight with distended abdomen was examined. On physical examination, the hen was found anaemic, dyspnoeic and recumbent with pulpy abdomen distended with fluid. Upon aspiration, about 800 ml of straw-coloured fluid was collected which contained proteinitious casts but no fibrin. Haematological examination revealed that RBC counts, haematocrit and WBC counts were 3.5 x 10^{12}/L, 25% and 50 x 10^{9}/L, respectively. Differential leukocytic counts revealed that heterophils, lymphocytes, monocytes and eosinophils were 50, 26, 18 and 6%, respectively, without any basophils. The concentrations of plasma proteins and fibrinogen were 7.9 g/dL and 312 mg/dL, respectively. The hen was given supportive therapy along with antibiotics and flusher.

Key words: Ascites, Fayumi hen, haematology, plasma proteins.

INTRODUCTION

Accumulation of non-inflammatory transudate in one or more of peritoneal cavities or peritoneal spaces is referred as ascites. Fluid frequently accumulates within two ventral hepatic, peritoneal or pericardial spaces and may contain protein clots (Aiello, 1998). Losses up to 30% have been reported in some flocks suffering from ascites (Lopez *et al.*, 1982). Prevalence of ascites syndrome has mostly been reported in fast growing birds i.e. broilers and is not a common finding in layers and other birds with slow growth rate. This paper describes clinical picture and laboratory findings in a Fayoumi bird suffering from ascites. As far our knowledge is concerned, this disease has not been reported in Fayoumi birds until now and this seems to be the first report of ascites in these birds.

CLINICAL/LABORATORY FINDINGS

A 9 months old Fayoumi layer having 2.5 kg body weight with distended abdomen, anorexia, and recumbency, was presented at the Diagnostic Laboratory, Department of Veterinary Pathology, University of Agriculture, Faisalabad during May, 2004. The hen had not been in laying for the last five weeks and was being kept in open yard in a house along with other ten birds. On physical examination, the hen was found anaemic (cyanotic comb), dyspnoeic and recumbent. The abdomen was pulpy and distended with fluid and nearly 800 ml straw coloured fluid was aspirated. Microscopic examination of this fluid revealed presence of proteinitious casts but no fibrin clots.

The blood was collected aseptically from the wing vein and analysed for haematological parameters following the haemocytometer method using Natt and Herrick solution and micro-haematocrit method. Erythrocyte counts, haematocrit and total leukocytic counts were 3.5 x 10^{12}/L, 25% and 50 x 10^{9}/L, respectively. Differential leukocytic counts (DLC) revealed that heterophils, lymphocytes, monocytes and eosinophils were 50, 26, 18, and 6%, respectively without any basophils. Plasma proteins (7.9 g/dl) were measured with the help of clinical refrectometer. Fibrinogen (312 mg/dl) was evaluated following the method described by Benjamin (1978).

TREATMENT

The hen was given supportive therapy along with antibiotics and flusher, i.e. injection Amivicom (Selmore) @ 1.5 ml/kg body weight 1/M for 3 days, Cosumix plus (Hilton Pharma) @ 1 gm/Litre in drinking water for 4-5 days and Nephryl VIRBAC @ 1 ml/Litre only once. However, the bird could not be followed and its fate could not be known.

DISCUSSION

Pathophysiology involved in ascites syndrome is the pulmonary hypertension that can be primary or

secondary. The former type requires exposure to cold environment or high energy feed with increased oxygen for metabolism for its development, whereas the later type usually develops due to hypoxia, hypervolaemia or presence of lung diseases (Julian *et al.,* 1987). Hypoxia, a known factor in the development of secondary hypertension, can be related to high altitude (Odom *et al.,* 1991), other factors contributing for its development can be presence of carbon monoxide (Julian *et al.,* 1987), and hypervolaemia is related to dietary sodium (Mirsalimi *et al.,* 1993).

The possibility of former type of pulmonary hypertension in the present case was ruled out, as the environmental temperature was not cold and these birds were also not on high energy feed. Moreover, Faisalabad is situated at a low altitude, therefore, hypoxia due to high altitude was also ruled out, though cases of ascites in broilers have been reported at low altitude (Habib-ur-Rehman *et al.,* 1999). As these birds were not being kept in confined environment, it nullifies the presence of carbon monoxide for the development of hypoxia. The only possibility left for the development of hypoxia leading to pulmonary hypertension, was the excess of sodium, either in the diet or drinking water. Underground water of Faisalabad city contains high total dissolvable salts. Moreover, fishmeal used in poultry feed is also preserved in sodium, therefore, these factors could lead to development of hypoxia and ascites in this hen.

Pulmonary hypertension leads to hypertrophy and failure of right ventricle in ascites syndrome (Odom *et al.,* 1991). The resultant right ventricular hypertrophy renders right ventricular valves flabby resulting in back flow of blood and fluid to the tissues and ultimately water accumulates in the belly. It may result from increased vascular hydrolic pressure, vascular damage, increased tissue osmotic pressure, decreased tissue oncotic pressure or blockage of lymph drainage. Liver damage caused by aflatoxins or by Carotalaria plant is another cause (Aiello, 1998).

General clinical signs of the disease are non-specific. These include dullness, depression, reluctant to move (Faraz, 1988), listlessness and depression (Coleman and Coleman, 1991) and difficult open mouth breathing (Saif, 2003). In DLC, a particular change was increased heterophils and monocytes at the expense of lymphocytes. This finding is in agreement with the findings of Maxwell *et al.* (1986). It can be concluded from the above discussion that ascites in Fayoumi layers develops through the same mechanism prevailing in broilers and etiology, clinical picture, haematology and pathology remain invariably the same.

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