

## CLINICAL, GROSS AND HISTOPATHOLOGICAL OBSERVATIONS IN SPONTANEOUS CASES OF ASCITES SYNDROME IN BROILER CHICKENS REARED AT LOW ALTITUDE

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### ABSTRACT

A study was carried out on 70100 broiler chickens kept at low altitude (184 meters). Of these, 288 were suffering from spontaneously occurring ascites syndrome. Clinical picture of the syndrome and gross and histological changes were noted. General signs of ascitic broilers were non specific. Gross lesions included accumulation of straw yellow coloured fluid in the abdominal cavity, hydropericardium and right ventricular dilation, induration and fibrosis of the liver, swollen and haemorrhagic kidneys, haemorrhagic intestine along with oedematous and congested lungs. Histopathologically, liver showed congestion and necrosis with proliferation of connective tissue in sinusoidal spaces along with infiltration of heterophils and mononuclear cells. Vacuolar degeneration and lymphocytic aggregation of renal tubules, ectopic cartilaginous masses in the lung lobules, congestion of alveoli and proliferation of connective tissue in interlobular septa with infiltration of inflammatory cells were the main features observed in ascitic broilers.

### INTRODUCTION

Within the past 15 years, alarming increase of ascites syndrome in chickens kept in many low altitude countries has established this ailment as a major cause of deaths with annual losses of 1 billion U.S. \$ globally (Maxwell and Robertson, 1997). High salt intake and hypoxia are the two important factors associated with the development of ascites in commercial broiler chickens. Clinical signs and lesions of sodium chloride toxicity include ascites, difficult breathing, uncoordinated movements and emaciation leading to death (Mohanty and West, 1969). Poor ventilation with accumulation of ammonia inside the shed favours the development of hypoxia which could be another cause of ascites in broiler chickens (Maxwell *et al.*, 1990).

Cardiomegaly/dys-figuration due to right heart hypertrophy/dilatation (Julian *et al.*, 1987), induration, congestion and necrosis of liver (Kapaga, 1990) and intensive passive congestion and ectopic cartilaginous nodules in the lungs of ascitic broilers (Maxwell *et al.*, 1988) have been reported. However, more severe changes occur in ascitic broilers kept at high than low altitudes (Maxwell *et al.*, 1988). This paper describes clinical picture, gross and histopathological changes observed in spontaneous cases of ascites syndrome in broiler chicken kept at low altitude.

### MATERIALS AND METHODS

#### Birds

This study was carried out from November, 1996 to February, 1997 on 70100 broiler chicken belonging to 25 farms located in and around Faisalabad which is located at an altitude of 184 meters. The ascitic birds were searched out from the flocks, clinical observations were recorded and 35 out of 288 birds were randomly selected for further studies.

#### Gross and histopathology

Following slaughter, internal organs were examined for gross lesions. Morbid tissue samples of heart, lungs, liver, intestine and kidneys were preserved in formalin. These samples were dehydrated in ascending grades of ethyl alcohol and cleared in xylene and embedded in paraffin wax. Sections of 4-5  $\mu$ m thickness were cut and stained with Harris haematoxylin and eosin (H & E) stain for histopathological examinations.

### RESULTS

Ascitic birds showed listlessness, dullness and depression, cyanosis of comb, inability to stand,



trembling of head, and sitting in corners. Open mouth breathing, loose faeces, white pasting of vent and distended abdomen full of fluid were the prominent features of the ascitic birds.

#### Postmortem findings

Straw yellow coloured fluid, with or without fibrin clots was observed in the abdominal cavity of all ascitic birds. Hydropericardium of varying degree was a prominent feature of all birds. Heart showed dysfigurement. Generally, it was flabby, cone shaped with dilatation of the right ventricle. Liver was swollen, haemorrhagic, indurated and fibrosed with either irregular or smooth surface. Kidneys were also swollen and haemorrhagic. Either diffused or focal haemorrhagic spots were present in the intestinal mucosa especially in the duodenum. Subcutaneous thigh muscle haemorrhages were seen in a few cases. Lungs were oedematous and congested, with frothy exudate present in and around them.

#### Histopathological findings

The tissue samples taken from the heart of the ascitic birds did not show any significant lesions except mild congestion. However, lesions of varying degree were present in the liver, kidneys, lungs and the intestine. The liver showed congestion and necrosis (Fig. 1) with proliferation of connective tissue in sinusoidal spaces and hepatic triad. Severe infiltration of heterophils and mononuclear cells were present in hepatic triad and occasionally around blood vessels. Lymphocytic aggregation was another significant feature observed in the liver. In kidneys, congestion, vacuolar degeneration (Fig. 2) and lymphocytic aggregation of renal tubules was observed. Necrosis of renal tubules was also observed.

The lungs showed pneumonic lesions with congestion of alveoli (Fig. 3), thickening of alveolar septa, and infiltration of heterophils in alveolar septa. In severe cases, proliferation of connective tissue with infiltration of inflammatory cells was present in interlobular septa. Inflammatory cells, especially heterophils, were also present in the walls of bronchioles. Ectopic cartilaginous masses were observed in the lobules of lungs (Fig. 4) and in some cases atelectasis was also evident. Intestinal tissue of ascitic birds showed sloughing of villi, presence of necrotic material in the lumen and congestion of mucosa.

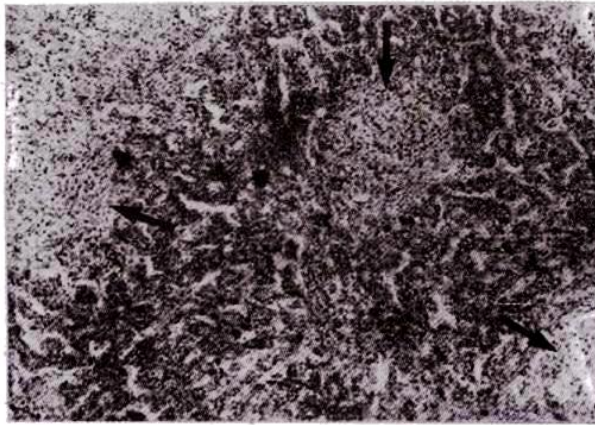


Fig. 1: Photomicrograph of liver from ascitic chicken showing necrosis. (H & E, 100 x)

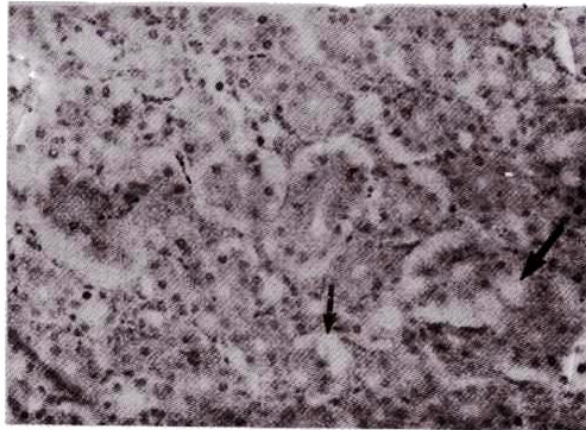


Fig. 2: Vacuolar degeneration in kidney of chicken suffered from ascites syndrome. (H & E, 200 x)

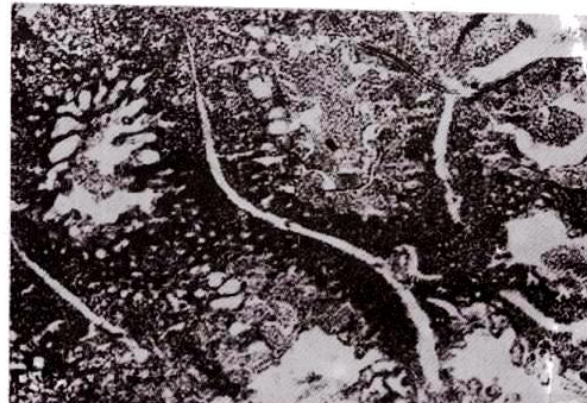


Fig. 3: Congested alveoli in ascitic chicken (H & E, 40 x)



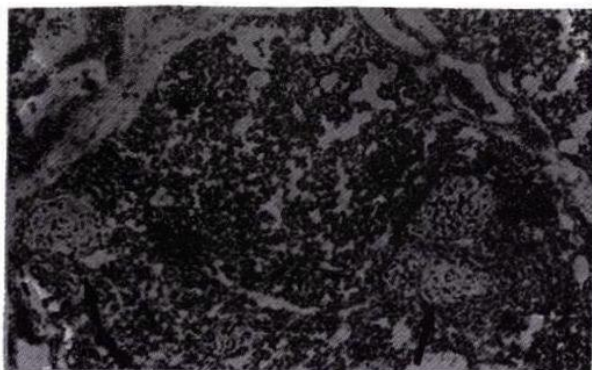


Fig. 4: Ectopic cartilaginous masses in lung lobules of ascitic chicken (H & E, 100 x)

## DISCUSSION

General clinical signs of ascitic broilers observed in this study were non specific. Dullness and depression, sleepiness and reluctant to move have been reported by Faraz (1988) and Lu *et al.* (1992). Difficult or open mouth breathing (Calnek, 1991), diarrhoea (Faraz, 1988), ruffled feathers, abnormal position of head (Madej *et al.*, 1992) and listlessness and depression (Coleman and Coleman, 1991) were also observed in the present study.

Grossly, liver from ascitic birds showed variable degree of induration, swelling and either smooth or dimpled surface. Histologically, congestion (Julian *et al.* 1989a; Biswas *et al.*, 1995) and variable degree of necrosis along with fibrosis in sinusoidal spaces (Biswas *et al.*, 1995) were the main features observed in ascitic broilers in the present study. However, hyperplasia and capsular fibrosis of liver of ascitic broilers reported by Wilson *et al.* (1988) were not observed in the present study.

Pneumonic changes observed in ascitic broilers, could be consequences of pulmonary hypertension and right ventricular failure. Lungs showed congestion, oedema, consolidation and ectopic osseous nodules in the present study had also been reported by Maxwell *et al.* (1988) and Biswas *et al.* (1995). However, hypertrophy of parabronchial smooth muscles in ascitic broilers reported by Wilson *et al.* (1988) was not recorded in the present study.

Accumulation of non inflammatory transudate in one or more of the peritoneal cavities is the main feature of ascities syndrome in broiler chickens. Rigid and molded lungs in thoracic cavity with their inability to expand too much, narrow pulmonary capillaries with little capacity to dilate, unproportionate growth of

lungs than rest of the body (Jordan, 1990), and nucleated erythrocytes with reduced ability to deform (Mirsalimi and Julian, 1991) maneuver in the development of increased resistance in pulmonary blood flow resulting in pulmonary hypertension in the broiler chicken. High dietary sodium is also responsible for reduced erythrocytes leading to pulmonary hypertension (Mirsalimi *et al.*, 1992).

Pulmonary hypertension can be primary or secondary. The former can result from the exposure to cold environment and/or high energy feed requiring increased oxygen for metabolism in association with genetic potential of rapid growth (Julian *et al.*, 1988a). The later usually develops due to hypoxia, hypervolaemia or presence of lung diseases (Julian *et al.*, 1987). Hypoxia, a known factor in the development of secondary pulmonary hypertension, might be due to high altitude (Odom *et al.*, 1991), severe rickets, presence of carbon monoxide and lung disease (Julian, 1987, Julian *et al.*, 1988b), whereas increased dietary sodium leads to hypervolaemia (Mirsalimi *et al.*, 1993).

Pulmonary hypertension leads to hypertrophy and failure of right ventricle (Julian and Goryo, 1990; Odom *et al.*, 1991). Right atrio-ventricular valve does not effectively seal the atrio-ventricular orifice and blood pressure rises in the vena cava, resulting in portal hypertension, leakage of plasma from the liver, obstruction of lymph return to the vena cava which ultimately ends in ascites (Julian and Goryo, 1990).

Dilation of the right ventricle in ascitic broilers observed in the present study has also been reported by Julian *et al.* (1987), Wilson *et al.* (1989) and Julian and Goryo (1990). Flabby and cone shaped appearance of the heart in ascitic broilers could be due to dilation of right ventricle. Congestion observed in histological sections of the heart tissue from ascitic birds is in agreement with the findings of Biswas *et al.* (1995). However, focal degeneration of myocardium and myofibril disorganization reported by Cerruti-Sola *et al.* (1988) and Maxwell and Mbugua (1990) could not be confirmed in this study.

As stated earlier, portal hypertension secondary to right ventricular failure intensifies the liver damage which is the most common cause of ascites in chicken (Julian *et al.*, 1986). Damaged liver due to amyloidosis, hepatic fibrosis or cholangiohepatitis can also increase hydraulic pressure in the hepatic portal system resulting in ascites. However, right ventricular failure causes both ascites and liver damage. Therefore, liver lesions could be primary or secondary. Since right ventricular failure was observed in all ascitic birds in the present study, it appears that right ventricular failure was the cause of ascities while



the liver lesions observed in the present study were secondary.

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