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## Ascites syndrome (Water belly) in broilers and its management

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**Abstract**

Ascites syndrome or Pulmonary hypertension is a serious condition of rapidly growing broilers having significant impact on the economy of poultry industry. This report sheds light on the Ascites syndrome in a private broiler farm in Assam comprising of 400 birds. The investigation of the case was enquiry based. The farmer presented 35 birds of 3 weeks old age. The clinical signs were recorded. At necropsy, accumulation of intra-abdominal fluid was observed with clots of fibrin. Gross lesions included fibrinous pericarditis, perihepatitis and hydropericardium. Bacterial examination from the post-mortem specimen revealed the organism to be *Escherichia coli*. Based on the age group, managemental aspect, gross lesions and associated bacteriological findings, the condition was diagnosed as Ascites syndrome. Thus, optimal management, manipulation of diet composition, optimization of brooding temperature, adequate ventilation are helpful practices to decrease the incidence of Ascites syndrome.

**Keywords:** Poultry, broilers, ascites, pulmonary hypertension, *E. coli*
**1. Introduction**

Ascites syndrome is an important metabolic disorder of broiler chickens occurring worldwide. It is characterized by abdominal distention and accumulation of standing fluid in the peritoneal cavity of fast growing broiler chickens [1]. It causes serious economic losses to the poultry industry [2]. It was of then, in the 1960s, when ascitic like syndrome in broilers raised at low altitude were described as "chick oedema factor" and "toxic fat syndrome". During the late 1980s, there was gradual increase in the incidence of ascites and later many reports describing similar symptoms and lesions led to the name 'waterbelly' or Ascites syndrome [3]. The exact mechanism of the condition is complex and is caused by a list of putative and exacerbating factors which includes exposure to environmental conditions such as temperature, lighting and ventilation coupled with nutritional and genetic factors [4]. The process of genetic selection focussing mainly on production traits [5] and a high density feed for fast growing broilers are responsible for the development of ascites. The pathogenesis of this syndrome is unclear but activation of many metabolic mechanism at low temperature and hypoxia stress should be the main influencing factors triggering to the development of ascites. Right ventricular diastolic dysfunction and pulmonary arterial hypertension are the susceptible cause of ascites in broilers respectively [6, 7]. In PHS, the increased demand for oxygen, especially in cold environment is mainly due to the higher metabolic rate of the fast growing broilers. Anatomically, the cardio-pulmonary system of the young broilers are poorly developed and due to which, they are unable to meet the oxygen demand to sustain their faster growth rate. The outcome of this anatomical and physiological limitation is hypoxaemia. Thus, in order to overcome the oxygen inadequacy and to cope up with high venous return, the workload of the cardio-pulmonary system increases which leads to development of many compensatory mechanisms giving rise to pulmonary hypertension and congestive heart failure [8]. Birds that develop ascites do not recover, thus, the consequences are death or condemnation at processing [9] which put forth economic implications for the broiler industry. The objective of this study was to understand the underlying causes of the Ascites syndrome and report its occurrence. Moreover, a strategic managemental and preventive alternative for Ascites syndrome has been discussed.

**2. Materials and Methods****2.1 Case history**

A total of 35 broilers 3 weeks old were brought to the Department of Microbiology, College of Veterinary Science, Assam Agricultural University, Khanapara for investigation.

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The flock were routinely vaccinated against Ranikhet Disease and Infectious Bursal Disease. The broilers were reared in intensive poultry farm. The birds appeared dull and/or depressed with reduced body weight gain and their abdomen were distended. They had abnormally slow heart rate, increased respiratory rate and reduced tolerance to activity. Information on managerial practices and housing conditions revealed that the birds were kept in a colder environment with inadequate ventilation and aeration. The floor space of the poultry shed was inadequate considering the number of birds.

## 2.2 Sample collection and analysis

The birds were subjected for post-mortem examination. Gross evaluation of the organs were done and the lesions observed were recorded systematically. Also, specimens for bacteriological examination were collected aseptically from heart, liver and intestine.

## 2.3 Bacterial Isolation and identification

Specimens were directly inoculated on Blood Agar (BA) media and incubated aerobically at 37 °C for 24 h. The colonies were examined for cultural and morphological characteristics and simultaneously inoculated on differential and selective medium namely, MacConkey Lactose Agar (MLA), and Eosin Methylene Blue Agar (EMB). The cultural characteristics, morphological properties and any changes in the media were recorded. Then, the organism was stained with Gram's Method.

The isolates were identified by biochemical tests (Indole, Methyl Red, Voges Proskauers test)

## 3. Results

### 3.1 Clinical Manifestations

The clinical signs of the affected broiler chickens were depression, distended abdomen with respiratory distress, reduced growth rate and mild diarrhoea.

### 3.2 Gross lesions

By observing the clinical signs and post-mortem findings, the case was tentatively diagnosed as Ascites syndrome. At necropsy, after the removal of the skin, it was found that the peripheral blood vessels were bluish and congested (Fig 1). There was accumulation of a large volume of clear yellowish fluid in the peritoneal cavity with clots of fibrin (Fig 2). The liver was swollen, congested and had fibrin adherent to the surface with blebs of fluid (Fig 3). There was cloudy thickening of the pericardium, with mild hydropericardium with fibrinous pericarditis and hypertrophy. Moreover, the mesentery were thickened with cloudy appearance, congested and fibrinous (Fig 4).



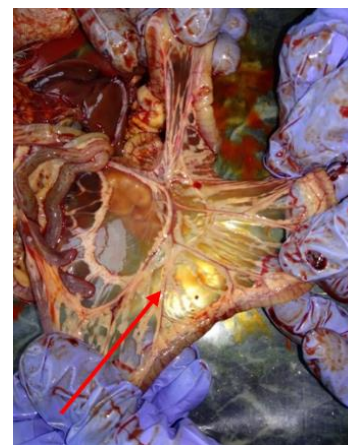
**Fig 1:** Distended abdomen in a 3 weeks Broiler



**Fig 2:** Accumulation of intra-abdominal fluid



**Fig 3:** Enlarged liver with blebs of fluid



**Fig 4:** Congested mesentery with fibrin deposit

### 3.3 Bacterial isolation and identification

Cultures from heart, liver and intestine onto BA plates yielded white to greyish color convex single type of colony. Then, subcultures of the same colonies on MLA and EMB agar revealed lactose fermenting microorganisms. Gram stained smears from these colonies showed Gram negative bacilli. Further, all isolates were positive to Indole and Methyl Red (MR) and negative to Voges Proskaur (VP). Thus, the isolates were identified biochemically as *Escherichia coli*.

## 4. Discussion

Broiler farming today has emerged as one of the fastest sector of the poultry industry due to its productive traits and day by day increasing demand for broiler meat. But improper management results in inefficient production and promotes the development of metabolic disorders. Ascites syndrome in

broilers is one of the serious causes of mortality and economic losses in broiler industry. This investigation was done to report the case of broiler ascites in a private farm in Assam. On analysis of the husbandry practices, it was found that the birds were kept in a closed poultry shed with lower ambient temperature which led to cold stress of the birds. The main predisposing factors that increases the incidence of Ascites syndrome includes lower ambient temperature of the poultry house and significantly lower brooding temperature of the broiler chicks. These factors are reported by many authors and they suggested that evaluation of the different production system such as ambient temperature, brooding temperature, adequate ventilation etc. should be adapted to reduce the incidence of ascites [11, 12, 13, 14].

On necropsy of the birds, a large volume of intra-abdominal clear yellowish fluid was observed which was typical of Ascites syndrome. The gross lesions observed were similar to those described by some authors [10, 15, 16]. However, fibrinous percarditis and perhepatitis with fibrinous messentery may be associated with secondary bacterial infection [10].

Bacteriological examination findings were in accordance with those reported for *E. coli* [17, 18]. To emphasize the fact that the microorganism was *E. coli*, biochemical tests were performed. However, microbial pathogens have not been concerned as a predisposing cause of ascites [17]. Keeping in concern the pathological and physiological evidences from this study, it was assumed that microbial factors (here, *E. coli*) triggers the occurrence of ascites syndrome in broilers coupled with contributing genetic, nutritional and environmental factors. This might be due to exposure of the birds through inhalation to the aerogenic infection.

In this study, the isolates showed resistance against amoxycillin and cephalixin. Ciprofloxacin was moderately sensitive while most sensitive were gentamicin and amikacin.

## 5. Conclusion

This study was done to investigate and report the case of ascites syndrome in a private poultry farm in Assam. Through clinical diagnosis and necropsy, the condition was definite diagnosis of ascites syndrome. As observed in this case report, there are no specific cause or cure to this condition, thus, a better understanding of the underlying causes of ascites is important for its effective prevention. Prompt diagnosis which includes estimation of haematocrit as marker for high rate of erythropoiesis in ascitic birds, which is lower in healthy counterparts [19] is reliable. Moreover, manipulating the management system of poultry rearing will effectively lower the incidence of this condition in the poultry farms. To reduce the incidence of ascites syndrome, a good start from hatchery and brooding management is effective to prevent predisposing the chicks hatched to ascites. Moreover, optimal managemental practices, reducing high stocking density with adequate ventilation, alteration of litter could be applied. Feeding regime can be altered to limit growth rate by nutrient restriction and manipulation of diet composition by feeding mash form of diet rather than crumbled and pelleted form. Also, inclusion of omega-3 fatty acid source, L-carnitine and antioxidants may provide additional protection from reactive oxygen and lipid peroxidation promoting ascites progression in broilers.

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