Spontaneous Occurrence of Ascites in Commercial Broiler Flock and Its Management

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Abstract

The present study was carried out on 100 broiler chicks that were reared in an organized poultry farm. From the above mentioned flock 28 birds died of unknown etiology. The clinical signs and post mortem changes were studied. Most of clinical signs were non-specific except a typical frog sitting posture with a backward leg extension noticed in all birds prior to death. Necropsy revealed accumulation of straw coloured fluid in the abdominal cavity which confirmed ascitic syndrome. After the diagnosis, effective control measures such as immediate withdrawal and replacement of leftover feed and provision of proper ventilation to the unaffected birds stopped further mortality in the flock.

Key words: Ascites, Broiler flock, Management

Introduction

Ascites is a complex problem caused by many interacting factors such as genetics, environment and management (Aftab and Khan, 2005). It can occur in broiler chickens, turkeys, guinea fowls and ducks (Milsavljevic, 2014). Ascites is one of the major causes of mortality and morbidity in modern broiler production (Aftab and Khan, 2005). The ascites syndrome is the primary cause of death for rapidly growing broiler strains, resulting in severe economic loss. It is a metabolic disorder that accounts for over 30% of overall mortality and has become the most noticeable, non-infectious cause of loss in the broiler industry worldwide (Guo et al., 2007). There are many factors that cause ascites such as, high altitude, rapid growth rate, limiting lung volume, provision of high energy rations, cold, poor ventilation, presence of respiratory disease, high sodium and low dietary phosphorus levels, hepatotoxins, mycotoxins and furazolidone in the feed, vitamin E and Se deficiencies and stress (Guo et al., 2007). Among so many causes, identification of the main trigger is still questionable. It was also reported that impaired oxygen supply to sustain a continuous fast growth rate may increase the risk for a higher incidence of ascites.

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syndrome (Singh et al., 2011). The present study depicts the clinical signs, symptoms, post mortem findings observed in ascites syndrome and its clinical management in broiler birds.

**Materials and Methods**

The present report was on coloured synthetic broiler chicks in an organized poultry farm. A total of 100 broiler chicks were procured from a government recognized farm and maintained on standard feeding protocol. They were vaccinated against Marek’s disease at the time of procurement followed by vaccination against Ranikhet disease and infectious bursal disease. A total of 28 chicks died within 15 days from the date of procurement. An acute death pattern was observed among the flock. To ascertain the cause of death and check further mortality a detailed postmortem examination was conducted.

**Results**

*Clinical signs and symptoms*

The birds showed listlessness, dullness, depression, cyanosis of comb, inability to stand, trembling of head and sitting in corners. The predominant sign was marked abdominal swelling leading to reduced mobility and lying down on the floor with a typical frog like posture. Due to abdominal swelling the legs were unable to bear body weight leading to backward extension of legs indicating a false impression of paralysis (Fig.1). Gradually the birds were unable to reach the feed or water source and were deprived of feed or water for a long time.

*Post Mortem Examination*

Post-mortem examination revealed severe abdominal distension with congestion of thigh and breast muscles (Fig.2). The most pathognomonic finding was the accumulation of clear or straw coloured fluid in the abdominal cavity. All had ascitic fluid measuring about 50 to 200 ml per bird that put pressure on all the visceral organs. Besides, there was cardiomegaly, petechial haemorrhages on the epicardium and dilatation of right side of the heart. Right ventricle was slightly enlarged in relation to the normal size. Liver was haemorrhagic, congested and slightly swollen. Kidneys were slightly haemorrhagic, congested and enlarged. In some cases urate deposits were also observed. Lungs, spleens and intestine were moderately congested.

*Diagnosis*

Based on the clinical signs and post mortem findings ascites was diagnosed. Toxicological analysis of the feed sample revealed presence of aflatoxin which might be the cause of feed related toxicity.

*Clinical Management*

After the diagnosis, the remaining birds were treated for ascites. As it is impractical to treat the affected birds, further mortality was checked with the following control measures. The mouldy feed was replaced.
immediately with new feed. As low temperature doubles oxygen demand leading to ascites, it is very much necessary to regulate the temperature in the poultry house. Hence as a second step, stress due to temperature was reduced by providing high power electric bulb in poultry house and by proper insulation during night. Proper ventilation was provided in the shed in order to reduce hypoxia and to reduce the concentration of harmful gases and dust particles in the air.

**Discussion**

The growth rate or body weight gain in broilers has been shown to positively correlate with incidence of ascites (Aftab and Khan, 2005). Ascites is a significant cause of mortality in many flocks of growing broiler chickens and the incidence is increasing day by day (Calnek, 1991; Tafti and Karima, 2000). Managemental practices should aim to provide thermoneutral environment, to limit growth rate and to practice good feeding in order to reduce the chances of ascites. The clinical signs observed in the present study were almost similar to those observed by Rehman et al. (1999). The gross changes included congestion and haemorrhagic lesions in the heart, liver and kidney and these findings were in accordance with Rehman et al. (1999). The right ventricular dialation observed in the present study was also reported earlier (Julian et al., 1987; Wilson et al., 1988; Julian and Goryo, 1990 and Rehman et al., 1999). Rehman et al. (1999) reported right ventricular failure as the prime cause of ascites. The haemorrhage in liver and kidney might be due to the presence of mycotoxin in the feed, which is one of the important causes of ascites as per Guo et al. (2007). Similarly, the presence of urate in the kidney can be correlated with mycotoxicoses.

The present study concluded that ascites might have occurred due to poor managemental practices such as mouldy feed, high energy feed and poor ventilation in the poultry shed. Although the specific etiology of ascites syndrome could not be determined, it appeared that this condition was not likely to be the result of any of the commonly recognized causes of right heart failure and ascites in broilers.

Fig.1. Bird on the floor with backward extension of hind legs.

Fig.2. Distention of abdomen with ascitic fluid.
Conclusion

Ascites is a multifactorial syndrome, caused by interactions among environmental, physiological and genetic factors. Proper nutritional and managerial practices can reduce the chance of occurrence of ascites.

Reference