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Evaluation of hepatic changes associated with ascites syndrome in broiler chickens and their relationship to weight loss

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Abstract

The fluid accumulation in the abdominal cavity is called ascites (waterbelly). Total mortality due to ascites is higher in broiler poultry, which is capable of faster growth and increased muscle deposition. Therefore, The objective of this study is to investigate the pathological change in the liver of broiler chickens that are affected with ascites syndrome. To conduct this study, a group of samples of broiler chickens infected with ascites were used. The results of body weight at 35 days for bird with ascites showed, the mean values of body weight was very low in the group with ascites as compared with Control groups. And the result of gross lesions showed Ascitic fluid, which is clear and yellow, is present in a significant amount. And also the gross lesions in the liver show, swollen, congestion and with thickness capsule and irregular edges. While The Result of Histopathological of the Liver show Hepatic cells degenerate vacuolar invading, dilatation of hepatic sinusoids and central veins, which is a characteristic of blood stasis with aggregation of inflammatory cells with hypertrophy of hepatocyte. We conclude from the foregoing that liver have a clear gross and histopathological change in broiler chicken with loss Weight.

Keywords: Ascites syndrome, pathological, liver, hypertrophy, broiler chickens

Introduction

Ascites is a significant metabolic disorder in the broiler industry, which can lead to more than 25% of broiler losses and is considered a major non-infectious cause of death among broilers (Maxwell and Robertson, 1997; Balog *et al.*, 2003 and Wideman *et al.*, 2013) [13, 1, 20]. hypoxia is a major factor that may raise the incidence of metabolic disorders, particularly ascites syndrome. Broilers are distinguished by their rapid growth, high feed intake and metabolism rates, and elevated oxygen requirements (Khajali, 2022) [9]. As a result of the oxygen supply and the oxygen needed to maintain rapid growth rates are imbalanced, cold stress raises the demand for oxygen, and broilers that are unable to meet this demand develop ascites syndrome, which is caused by a malfunctioning heart and circulatory system (Decuypere *et al.*, 2000) [4]. Slower growing lines are more susceptible than Since lines chosen for extremely for low feed conversion and high growth capacity to ascites, The altered metabolic needs of rapidly growing broilers have been linked to the genetic background underlying this syndrome. (Chineme *et al.*, 1995; Balog, 2003; Decuypere *et al.*, 2000; De Smit *et al.*, 2005 and Hassanzadeh *et al.*, 2010) [2, 1, 4, 3, 6]. this changes lead to mortality in broiler Chickens because cardiopulmonary failure and lung oedema (Julian, 2000 and Hassanzadeh *et al.*, 2010) [8, 6]. The liver is considered one of the important organs that affected by this syndrome, The study of hepatic alterations at both the macroscopic and histological levels. It is becoming more important to study how the liver changes in ascites syndrome in order to better understand the mechanisms that cause other diseases and come up with better ways to prevent and treat them. Researchers and professionals in the field of veterinary medicine can use an integrated assessment of these changes to create management and nutritional programs that help lower the number of cases of this syndrome and make birds healthier and more productive.

Materials and Methods

Experimental Animals: Taken same of chicken broiler both sex infection with Ascites at

aged 35 day, obtained from clinic vet, were used in this study.

Body Weight

The body weight measurement by using Balance measurement for control and affected birds.

Gross Inspection

The chicken with ascites take from clinic vet record the note, A large volume of clear yellow fluid is present, known as ascites fluid, is revealed when opening the abdominal cavity of an ascites bird. and fixed liver enlargement in 10% buffer formaldehyde solution immediately with record the note lesions on the liver. This study was conducted in clinic veterinary in Babylon.

Histopathological Examination

We took 1x1x1 cm pieces of liver tissue and put them in a

10% buffer formaldehyde solution immediately. Following a 72-hour fixation period, the samples were rinsed with tap water and subjected to a series of alcoholic concentrations ranging from 70% to 100% for two hours at each concentration in order to eliminate any remaining water in the tissues. The specimens were then cleared with xylol and infiltrated in two stages at 58 °C using semi-liquid paraffin wax. Lastly, specimen blocks were created using paraffin wax and cut into 5µm sections with a rotary microtome. We used Haematoxylin and Eosin (H & E) stain to colour all of the tissues, and we looked at the changes in the tissues under a light microscope (Luna, 1968; Mosa and Zenad, 2020) [12, 16].

Results and Discussion

Result of Body Weight: The results of body weight at 35 days for bird with ascites showed, very low in the group with ascites as compared with normal.

Table 1: Mean body weight (g) of broiler chickens at 35 days of age

Group	Group one (Ascites group)	Group two (Control group)
Body weight (g)	1300 ± 50 g	1800 ± 50 g

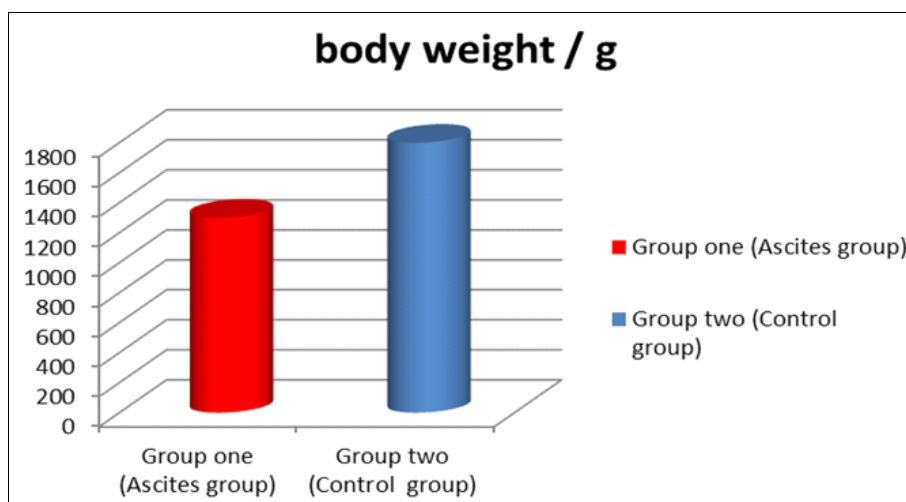


Fig 1: Mean body weight (g) of broiler chickens at 35 days of age

The results of body weight at 35 days for chicken broiler with ascites showed, the mean values of body weight was very low (1300 ± 50 g) as compared with normal chicken broiler, this result may be due to increased oxygen demand and inability of the lung and heart to provide tissues with requisite oxygen to maintain growth rate and nutritional potential. Low-altitude ascites is linked to respiratory disorders and inadequate ventilation (Olkowski and Classen, 1998) [17]. Additionally, The production of ammonia and dust increases the risk of ascites. The spread of disease-causing microorganisms by dust particles can infect or irritate the lung and reduce the flow of oxygen between the environment and birds (Mc Govern *et al.*, 1998) [14]. According to Sun *et al.* (2007) [19], the high incidence rate of AS led to poor growth and low energy and nutrient availability. The accumulation of fluid within the abdominal cavity is a result of this syndrome, which results in right ventricular swelling and heart failure, which results in changes in performance and growth (Miao *et al.*, 2022) [15]. This results in weight changes due to hearing loss and poor absorption of materials (Sun *et al.*, 2007) [19].

Result of Gross Lesions

The result of gross lesions showed present significant amount of Ascitic fluid, which is clear and yellow (Figure 2). And also show the enlargement liver, congestion and with thickness capsule and irregular edges.

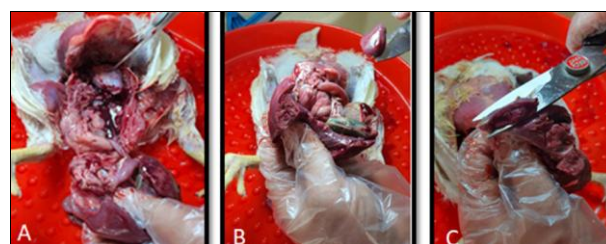


Fig 2: Gross section of broiler chicken with Ascites showing (A) the abdominal cavity ascitic fluid. (B) The liver is swollen, congested with irregular surface and enlargement of gall bladder. (C) The liver is swollen, congested with irregular surface.

Result of Histopathological Lesions

The Result of Histopathological lesion in the Liver show hepatic cells degenerate vacuolar invading and dilatation of

hepatic sinusoids and central veins which is a characteristic of blood stasis with aggregation of inflammatory cells with hypertrophy of hepatocyte and necrosis. (Figures 3, 4, 5 and 6).

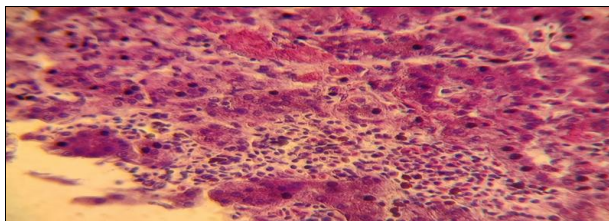


Fig 3: In the liver with ascites the Histopathological changes shows necrosis with severe MNCs Aggregation with of hepatocyte with congestion and hypertrophy of hepatocyte. (H&E stain 10X)

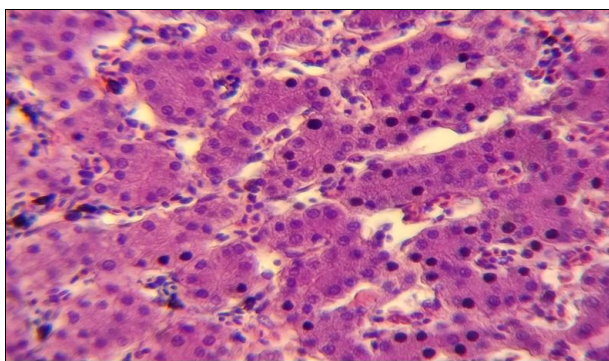


Fig 4: The section in the liver with Ascites show dilatation of hepatic sinusoids with hypertrophy of hepatocyte. (H&E stain 20 X).

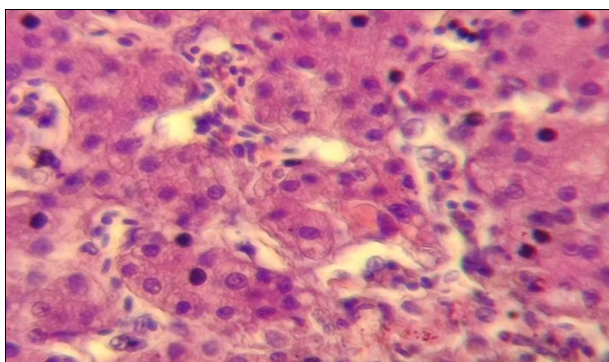


Fig 5: The section In the liver with ascites the Histopathological changes shows dilatation of hepatic sinusoids with hypertrophy of hepatocyte With inflammatory cells. (H&E stain 40X).

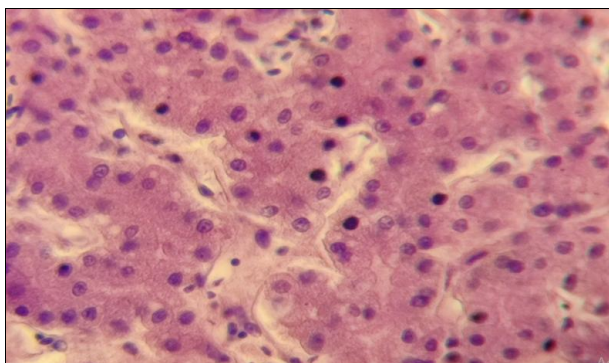


Fig 6: The section in the liver with Ascites shows d hypertrophy of hepatocyte with edema. (H&E stain 40X).

As it is a significant cause of mortality in growing broiler

chickens and the incidence appears to be increasing and Accumulation of non-inflammatory fluid in the cavity is reported after pulmonary hypertension in broilers. A high-energy diet and the genetic predisposition for rapid growth can cause primary pulmonary hypertension. (Wideman *et al.*, 2013) [20]. Additionally, The most common cause of this outcome is venous hypertension brought on by right heart failure in response to elevated pulmonary resistance. Other possible causes include, vascular damage, increased vascular hydraulic pressure, decreased vascular oncotic (usually colloidal) pressure or increased tissue oncotic pressure (Guyton and Hall, 2005) [5]. The Result of Histopathological lesion in the Liver show Hepatic cells degenerate vacuolar invading and dilatation of hepatic sinusoids and central veins, which is a characteristic of blood stasis with aggregation of inflammatory cells with hypertrophy of hepatocyte. This result may be due to The liver produces albumin, and if the liver and hart are harmed by mycotoxins and other chemicals, the amount of albumin in the blood decreases, which lowers the koloidosmotskog pressure value. Edema is then caused by the presence of blood in the free fluid that enters the intercellular spaces through the capillary walls (Knezevic and Milanka, 1996; Jasim *et al.*, 2019) [11, 7]. The accident ascites due to hypoxia it is consider major factor in this synderom special in broiler chicken, and this lead to hart failure and high pulmonary pressure this lead to infiltration fluid in cavity of bird. This condition caused by inability the respiratory system's in meeting due to increased demand for oxygen in rapid growth (Wideman and Hamal, 2023) [21].

Conclusion

We conclude from the foregoing that liver have a clear gross and Histopathological change included swollen, congestion and with thickness capsule and irregular edges. While Histopathological of the Liver show Hepatic cells degenerate vacuolar invading and dilation is occurring in the hepatic sinusoids and central veins, which is a characteristic of blood stasis with aggregation of inflammatory cells with hypertrophy of hepatocyte with Wight loss in broiler chicken.

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