

Ascites Syndrome in broiler birds - A case report

Praggaya Priya Lakra*, M.K. Gupta, Vikash Kumar, Shubham Sharma and Amit Kumar Mahto

Department of Veterinary Pathology, College of Veterinary Science and AH, Ranchi, Birsa Agricultural University

Address for Correspondence

Praggaya Priya Lakra, Assistant Professor, Department of Veterinary Pathology, College of Veterinary Science and AH, Ranchi, Birsa Agricultural University, E-mail: praggaya.lakra@gmail.com

Received: 17.3.2025; Accepted: 2.7.2025

ABSTRACT

Five Vencobb broiler birds approximately eight weeks old were submitted for post-mortem examination to the Department of Veterinary Pathology, College of Veterinary Science and Animal Husbandry, Ranchi, Jharkhand, from a nearby private broiler farm. External examination revealed poor body condition and abnormal abdominal distension in all birds. Upon necropsy, approximately 50-60 ml of yellow, watery fluid containing thick, mucus-like yellow clots was found in the abdominal cavity. The liver appeared mottled and friable, the intestines were severely congested, the kidneys showed lesions of nephrosis and the lungs were pneumonic. Histopathological analysis revealed vacuolar degeneration of hepatocytes, congestion of sinusoids and the presence of haemosiderin pigment. The mucosal surface of the proventriculus exhibited degenerative, necrotic and congestive changes. The intestinal villi showed necrosis, epithelial sloughing and karyorrhexis. The heart tissue displayed significant mononuclear cell infiltration - primarily macrophages and epithelioid cells - within the subepicardial region. Based on the clinical history, gross lesions and histopathological findings, the condition was diagnosed as Ascites Syndrome.

Keywords: Ascites Syndrome, broiler, postmortem, Vencobb

Ascites Syndrome (AS), also known as Pulmonary Hypertension Syndrome (PHS), is characterized by the accumulation of fluid within the coelomic cavity. It is a leading cause of morbidity and mortality in the modern broiler industry. In most cases, ascites is diagnosed between 4 and 5 weeks of age¹. Ascites is most commonly seen in fast growing chickens particularly during the winter^{2,3}. In recent years, intensive genetic selection for rapid growth has contributed to the emergence of metabolic disorders, including ascites. Many factors interact to cause ascites, including management practices, environmental conditions and genetic makeup.

Five broiler birds (Vencobb) of about 8 weeks of age were submitted for post-mortem examination in January 2025 from a local poultry farm with a history of mortality that had reached upto 10% in a total strength of 500 birds and was progressively increasing each day. It was also noticed that in the flock about 20% of the birds were smaller in size and weighed lesser than expected at 8 weeks of age. The deaths were sudden and the birds showed signs of poor growth, lethargy, anorexia and distended abdomen.

External examination of the carcasses revealed poor body condition and stunted growth. The abdomen was abnormally distended. Upon opening the carcass, approximately 50-60 ml of yellow colored watery fluid with thick yellow, jelly-like clots were observed within the abdominal cavity (Fig. 1). The liver appeared pale, mottled and friable in consistency. The intestines were severely congested, both kidneys were enlarged and mottled and indicated nephrosis and the lungs were pneumonic. After examining all the organs grossly, samples were collected in 10% Neutral Buffered formalin for histopathology. The tissue were routinely processed, sectioned and stained with Haematoxylin and Eosin (H&E) stain⁴.

Histopathological examination of the intestinal tissue revealed necrosis and sloughing of the villous epithelium, accompanied by nuclear karyorrhexis (Fig. 2). Similar necrotic and karyorrhctic changes were also noted in the crypts, though infiltration of inflammatory cells was minimal. Blood vessels

How to cite this article : Lakra, P.P., Gupta, M.K., Kumar, V., Sharma, S. and Mahto, A.K. 2025. Ascites Syndrome in broiler birds - A case report. Indian J. Vet. Pathol., 49(3) : 278-280.

within the intestinal tissue were congested. Degenerative and necrotic changes with widespread karyorrhexis was observed in mucosal surface of the proventriculus (Fig. 3). Blood vessels were dilated and highly congested. The deeper glandular part showed milder lesions with intact glandular tissue. Lungs revealed severe congestion of both alveolar and larger inter lobular blood vessels with significant dilatation (Fig. 4). No significant inflammatory cell infiltration was observed. Kidney revealed severe congestion of the interstitial blood vessels along with marked degenerative changes in the tubular epithelium suggestive of nephrosis (Fig. 5). The glomeruli exhibited hypoplastic changes, with no notable infiltration of inflammatory cell. Liver exhibited marked vacuolar degeneration

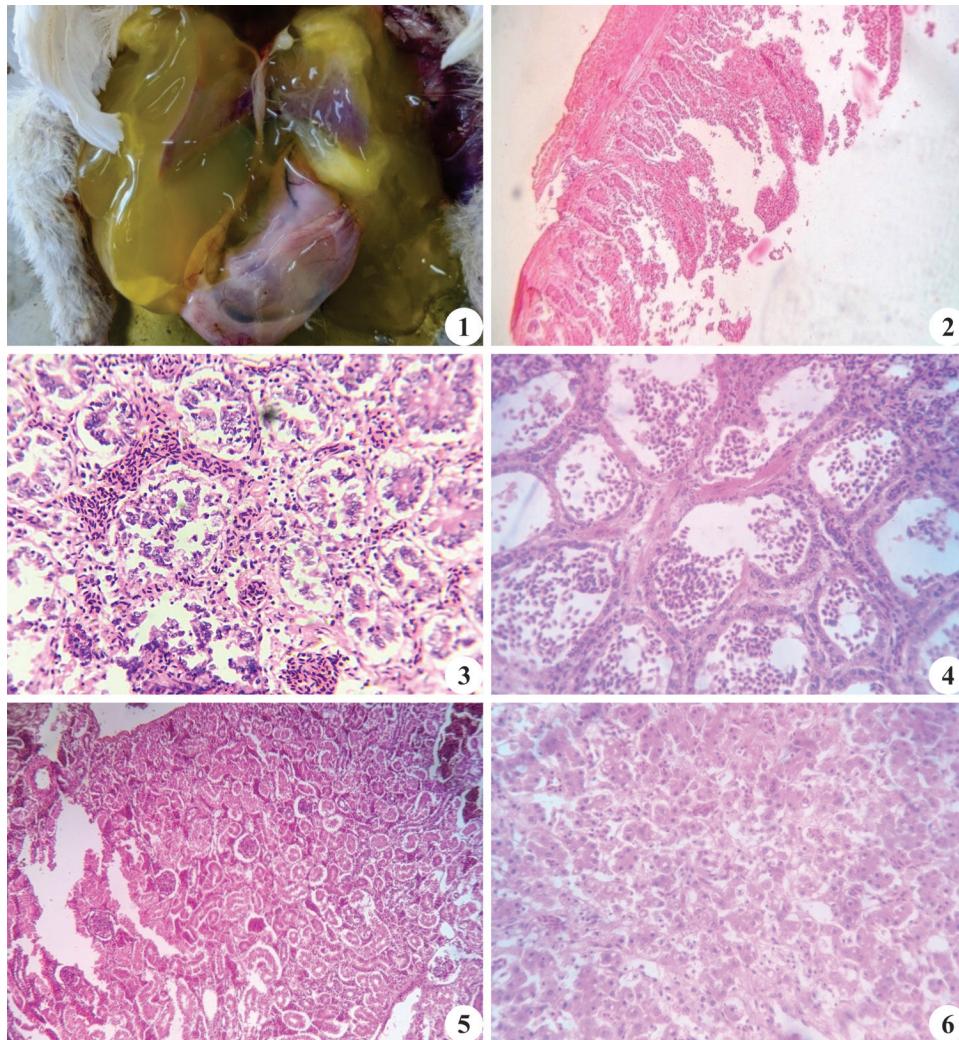


Fig. 1. Yellow colored watery fluid with thick yellow, jelly-like clots in the abdominal cavity; **Fig. 2.** Intestinal epithelium showed necrosis and sloughing of villi with karyorrhectic changes in nuclei (H&E 100X); **Fig. 3.** Section of Proventriculus showing degenerative and necrotic changes in the cells along with dilatation of blood vessels (H&E 400X); **Fig. 4.** Lungs revealed severe congestion of both alveolar and larger inter lobular blood vessels with significant dilatation (H&E 400X); **Fig. 5.** Kidney revealed severe congestion of the interstitial blood vessels along with marked degenerative changes in the tubular epithelium suggestive of nephrosis (H&E 100X); **Fig. 6.** Section of liver showing coagulation necrosis characterized by cytoplasmic acidophilia and pyknosis (H&E 100X).

of hepatocytes along with congested sinusoids and the presence of haemosiderin pigment. In extensive areas hepatocytes revealed coagulation necrosis characterized by cytoplasmic acidophilia and pyknosis (Fig. 6 & 7). Significant fibrosis was observed in the portal tract suggesting initiation of portal cirrhosis were seen in the liver. Heart showed marked infiltration of mononuclear cells, primarily macrophages and epithelioid cells in the subepicardium suggestive of pericarditis. The blood vessels were highly congested (Fig. 8). Cardiac muscle fibres also revealed infiltration of mononuclear cells, predominantly lymphocytes. Cardiocytes showed degenerative changes with loss of cross striations (Fig. 9).

The pathogenesis of ascites syndrome may begin with an elevated basal metabolic rate, triggered by

various factors such as cold stress, mild heat exposure, increased physical activity, hyperthyroidism, excessive muscle mass and overeating.

In backyard flocks, cold is regarded to be a major factor in ascites epidemics⁵, due to an increased blood flux out of the bird's lungs to provide the body with internal warmth. Ascites is exacerbated by cold weather by raising pulmonary hypertension and metabolic oxygen demands^{6,7}. Since in the current case the mortality was occurring in the month of January, it can be correlated with the cold factor.

The occurrence of ascites can also be attributed to the diet's nutritional makeup and/or the way that feed is distributed. Ascites in broiler chickens can be caused by significant dietary parameters, such as high feed

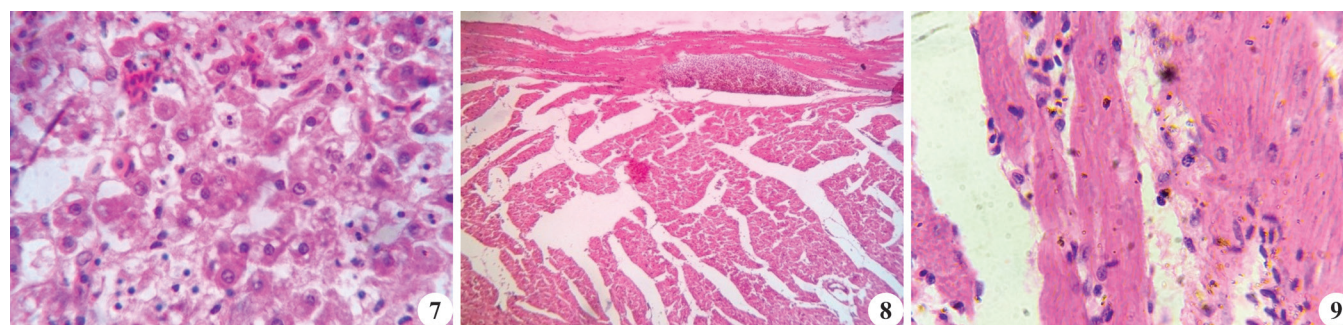


Fig. 7. Liver - Hepatocytes revealed coagulation necrosis characterized by cytoplasmic acidophilia and Pyknosis (H&E 400X); **Fig. 8.** Heart revealed marked infiltration of mononuclear cells, predominantly macrophages and epithelioid cells in the subepicardium suggestive of pericarditis (H&E 100X); **Fig. 9.** Cardiocytes showing loss of cross striations (H&E 1000X).

concentration and an increase in feed consumption, in addition, to feed structure (shape). Diets low in calories have been suggested to lower the prevalence of PHS⁸⁻¹⁰. Apart from temperature and feed, the broilers' genetic makeup can make them susceptible to PHS due to the moderate to high heritability of the genes linked to the disease.

According to published research research, a limited number of genes have a major role in the heritability of ascites⁵. In an investigation mutant genes were shown to increase pulmonary artery reconstruction and be related to ascites incidence^{11,12}.

In conclusion, the diagnosis in this case was supported by a comprehensive gross and histopathological examination, along with the clinical history. The clinical signs were in accordance with the previously reported earlier worker¹³. Feed restriction and nutrient concentration reduction in the diet can limit growth and prevent ascites-related death¹⁴. Ascites is a complex condition resulting from the interaction of physiological, environmental and management factors. However, its incidence can be minimized through proper management, medical intervention and nutritional strategies.

Financial support & sponsorship: None

Conflicts of Interest: None

Use of Artificial Intelligence (AI)-Assisted Technology for manuscript preparation: The authors confirm that there was no use of AI-assisted technology for assisting in the writing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Das S and Deka P. 2019. Ascites syndrome (Water belly) in broiler and its management. *J Entomol Zool Stud* **7**: 388-390.
2. Wideman RF, Rhoads DD, Erf GF and Anthony NB. 2013. Pulmonary arterial hypertension (ascites syndrome) in broilers: a review. *Poult Sci* **92**: 64-83.
3. Maxwell MH and Robertson GW. 1997. World broiler ascites survey. *Poult Internat* **36**: 16-30.
4. Luna LG. 1968. Manual of histologic staining methods of the Armed Forces Institute of Pathology. 3rd Edition, McGraw-Hill, New York.
5. Kalmar ID, Daisy V and Greet PJ. 2013. Broiler ascites syndrome: Collateral damage from efficient feed to meat conversion. *The Vet J* **197**: 155-164.
6. Julian RJ. 2000. Physiological management and environmental triggers of the ascites syndrome: a review. *Avian Pathol* **29**: 519-527.
7. Stoliz JL, Rosenbaum LM, Jeong D and Odom TW. 1992. Ascites syndrome, mortality and cardiological responses of broiler chickens subjected to cold exposure. *Poult Sci* **71**: 4.
8. Coello CL, Arce MJ and Avila GE. 2000. Management techniques to reduce incidence of ascites and SDS. Proceeding of the XXI World's Poultry Science Association Congress, Canada.
9. Balog JM. 2003. Ascites syndrome (pulmonary hypertension syndrome) in broiler chickens: Are we seeing the light at the end of the tunnel. *Avian Poult Biol Rev* **14**: 99-126.
10. Ozkin S, Piavnik I, and Yahav S. 2006. Effects of early feed restriction on performance and ascites development in broiler chickens subsequently raised at low ambient temperature. *J Appl Poult Res* **15**: 9-19.
11. Sufang Cheng, Xin LIU, Pei LIU, Guyue LI, Xiaoquan GUO and Lin LI. 2021. Dysregulated expression of mRNA and SNP in pulmonary artery remodeling in ascites syndrome in broilers. *Poult Sci* **100**: 1-8.
12. Dey S, Parveen A, Tarrant KJ, Licknack T, Kong BC, Anthony NB and Rhoads DD. 2018. Whole genome resequencing identifies the CPQ gene as a determinant of ascites syndrome in broilers. *PloS One* **13**: e0189544.
13. Olkowski AA, Wajnarowicz C, Rathgeber BM, Abbott JA and Classen HL. 2003. Lesions of pericardium and their significance in the aetiology of heart failure in broiler chickens. *Res Vet Sci* **74**: 203-211.
14. Camacho-fernandez D, Lopez C, Avila E and Arce J. 2002. Evaluation of different dietary treatments to reduce the ascites syndrome and their effect on corporal characteristics in broiler chickens. *J Appl Poult Res* **11**: 164-174.