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Pathological evaluation of naturally occurring ascites syndrome in broilers: A case series from Kashmir Valley

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Abstract
This study was undertaken to investigate the pathology of ascites syndrome in poultry rearing in Srinagar and Ganderbal Districts of Kashmir. The surveillance across ten farms in these Districts revealed the occurrence of ascitic conditions in poultry. The mortality rates and flock health indicators were carefully monitored and birds showing clinical symptoms of ascites were isolated for further evaluation. A total number of 100 broilers suspected of ascites were selected for detailed examination. Each suspected carcass underwent systematic necropsy to assess the pathological changes in the affected organs. The Postmortem findings included fluid accumulation in the abdominal cavity, hypertrophy of the right ventricle and enlargement of the liver and kidneys. The histopathological evaluation in of the affected broiler chickens revealed hepatic congestion and edema, myocardial congestion with degeneration of muscle fibers, pulmonary changes such as l edema, congestion, atelectasis & dilation of tertiary bronchi. The microscopic examination of lymphoid organs revealed lymphoid depletion, atrophy of lymphoid follicles and marked infiltration of mononuclear cells in the lymphoid organs. These lesions collectively characterized the pathology of ascites in broilers.

Keywords: Broiler chickens, clinical, gross, histopathological, natural ascites syndrome

Introduction
Pulmonary arterial hypertension is also referred to as Ascites Syndrome (AS), which poses a significant clinical challenge in both humans and broiler chickens (Yang *et al.*, 2016) [27]. In poultry, ascites is a major contributor to flock mortality and its occurrence has been reported to be on the rise in the poultry farms of Kashmir. The syndrome represents a metabolic disorder commonly observed in rapidly growing broilers, which is characterized by abdominal swelling and fluid accumulation within the abdominal cavity (Liu *et al.*, 2016) [15]. The condition develops when the oxygen supply fails to meet the high oxygen demand required for fast growth and feed efficiency (Baghbanzadeh *et al.*, 2008) [1, 4]. The hypoxia initiates the disease by causing peripheral vasodilation, increased cardiac output, elevated pulmonary arterial pressure and ultimately right ventricular hypertrophy (Yang *et al.*, 2002) [26]. The excessive workload placed on the right ventricle due to pulmonary hypertension results in pressure-induced liver cirrhosis and fluid leakage into the abdominal cavity (Khodakaram *et al.*, 2000) [13]. Since oxygen demand is a critical factor, the development of ascites is influenced or exacerbated by elements such as growth rate, high altitudes hypoxic conditions and environmental temperature. Thus, this study focuses on investigating both gross and microscopic tissue alterations caused by ascites in broiler chickens.

Material and Method
A systematic investigation was undertaken to elucidate the occurrence of naturally developing ascites in broiler chickens reared on poultry farms in the Srinagar & Ganderbal District of Kashmir. During the surveillance period, ascitic conditions were identified across ten farms in both districts of Kashmir. The mortality rates and general health parameters of the flocks were meticulously recorded and birds exhibiting clinical manifestations of ascites

were segregated for detailed examination. The ascitic cases in birds were identified by demonstrating abdominal fluid accumulation accompanied by systemic cyanosis of the comb, wattle and skin. A cohort of 100 broilers suspected of ascites was selected for comprehensive analysis.

All suspected carcasses of the birds underwent thorough necropsy to evaluate gross pathomorphological alterations in the affected organs. Each organ was carefully inspected for morphological changes. The tissue specimens from multiple organs were excised and preserved in 10% neutral buffered formalin for subsequent histopathological processing (Luna *et al.*, 1968) [16]. The fixed samples were routinely processed and sections of five microns in thickness were prepared and stained with hematoxylin and eosin (H&E) to facilitate detailed visualization of cellular and tissue architecture. The microscopic examination was conducted to observe pathological changes associated with ascites, allowing for a comprehensive comparison between affected broilers and clinically healthy counterparts. This methodical approach provided a robust framework for the assessment of organ-specific pathologies and the systemic impact of ascitic syndrome in broiler populations (Rehman *et al.*, 2007) [20].

Result

Ascites is also known as water belly, which was detected in broiler chickens reared under intensive production systems. This pathological condition predominantly affected fast-growing strains between 4 and 6 weeks of age, a period that coincides with peak metabolic demand and rapid body weight gain. A noticeable increase in overall mortality was recorded in the affected flocks compared to healthy controls, with sudden deaths occurring in several houses without preceding clinical signs. The Post-mortem examination revealed accumulation of fluid in the abdominal cavity, enlargement of the right ventricle and congestion in the liver and lungs, indicating that cardiovascular dysfunction which was a major contributing factor. The other contributing factors include environmental and management factors, such as high stocking density, inadequate ventilation, cold stress and protein-rich diets, appeared to enhance the prevalence and severity of this pathological condition. The affected birds exhibited varying degrees of weakness, lethargy and reduced activity, reflecting early systemic stress and hypoxia. These initial observations provided a basis for further investigation into the clinical manifestations, gross lesions and histopathological changes associated with ascites in the study population.

Clinical signs: The affected broiler chickens exhibited a spectrum of clinical signs characteristic of ascites, reflecting the complex interplay between cardiovascular, respiratory and metabolic disturbances associated with this pathological condition. The birds were consistently depressed, appearing lethargic, slow-moving and often huddled, with reduced responsiveness to environmental stimuli, reluctant to feed or drink, which is indicative of systemic hypoxemia arising from pulmonary hypertension and right-sided heart compromise. A striking clinical feature was abdominal distension, with the abdomen appearing swollen, tense and occasionally shiny due to the accumulation of serous fluid in the coelomic cavity, while in severe cases, the abdominal wall was stretched and the capsule thickened, reflecting portal and systemic venous congestion. This distension is often exacerbated in rapidly growing broilers, particularly in birds aged 4-6 weeks, where the metabolic demand for oxygen exceeds the capacity of the cardiovascular system, promoting fluid accumulation and right ventricular overload. The affected birds also showed respiratory distress, evidenced by rapid, labored breathing, wing extension and open-mouth respiration, as the fluid in the abdomen limited diaphragmatic movement and pulmonary expansion, thereby aggravating systemic hypoxia in advanced stages. The growth performance was markedly reduced, as hypoxia and fatigue led to decreased feed intake, impaired nutrient absorption and poor tissue oxygenation, culminating in stunted growth, lower body weights and disproportionate carcass development relative to unaffected flock mates. Some birds exhibited diarrhea, likely secondary to reduced intestinal perfusion and fluid imbalance resulting from cardiac insufficiency and hepatic congestion, which further contributed to dehydration and electrolyte disturbances. The subtle clinical indicators such as pale combs and wattles, exercise intolerance, reluctance to move and behavioral isolation were frequently observed, reflecting the birds' inability to meet metabolic demands under stress conditions. The environmental factors, including high stocking density, low ambient oxygen levels, cold stress, or poor ventilation, were contributory, as they exacerbate pulmonary hypertension and increase the risk of ascites development. The nutritional influences, such as high-protein diets, rapid growth-promoting feed formulations and imbalanced electrolytes, further predispose birds to right ventricular overload and fluid accumulation. The progression of clinical signs typically begins with mild lethargy, subtle respiratory changes and reduced activity, advancing to pronounced abdominal swelling, severe respiratory compromise, cyanosis and systemic hypoxia. Thus, these observations illustrate that ascites in broilers is a multifactorial, multisystem disorder, where abdominal distension, respiratory distress, reduced growth, gastrointestinal disturbances and behavioral changes are interrelated consequences of pulmonary hypertension, right ventricular failure, impaired oxygen transport, and metabolic stress, which emphasizes the critical importance of early detection, environmental management, dietary modulation and genetic selection in mitigating both animal welfare concerns and economic losses in commercial poultry production.

Pathology

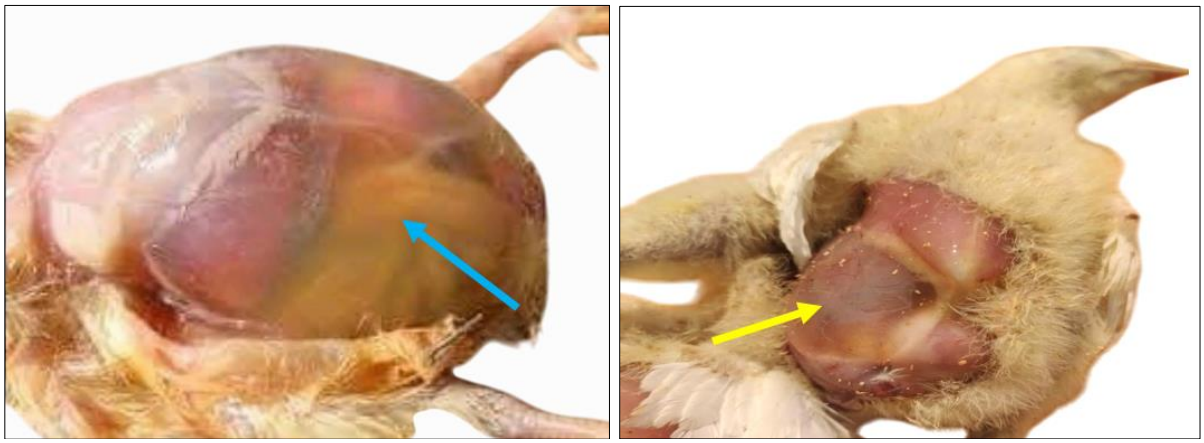
Lungs

Gross Pathology: The lungs displayed a spectrum of pathological changes ranging from mild patchy congestion to severe, diffuse dark-red discoloration, indicative of varying degrees of vascular compromise. The severely affected lungs were markedly enlarged, edematous and heavy, failing to collapse upon opening the thoracic cavity, demonstrating compromised elasticity and airspace expansion. The affected lungs revealed blood-stained frothy fluid often exuded, reflecting alveolar edema and hemorrhage. The areas of pneumonic consolidation were observed in some cases, forming firm, dense lobules due to alveolar exudates. The trachea and bronchi were frequently filled with frothy exudates, sometimes mixed with necrotic material. In few cases, caseous plugs partially obstructed the bronchial lumen, reflecting localized necrotic inflammation. The air sacs generally appeared cloudy, suggesting that the lesions selectively involved the pulmonary parenchyma and

the air sac system. The combination of edema, hemorrhage and consolidation indicated acute inflammatory processes, vascular injury and potential secondary hypoxia.

Histopathology: Microscopically, the lungs exhibited moderate to severe congestion of interstitial vessels and alveolar capillaries, accompanied by interstitial edema, hemorrhage and thickening of the wall of the blood vessel (Fig:1). The pulmonary arterioles demonstrated medial thickening and cystic dilatations, and rare plexiform lesions suggested chronic vascular remodeling, possibly secondary to prolonged hypoxia or pulmonary hypertension. The perivascular leukocytic infiltration was consistently observed, consisting of heterophils and mononuclear cells, indicative of active inflammatory response. The Broncho-interstitial pneumonia manifested as interstitial thickening with infiltration by inflammatory cells and exudates within parabronchi. The hemorrhagic bronchopneumonia showed necrotic debris, hemorrhagic exudates, proliferation of fibrous connective tissue within the interstitium and thickened airspace walls due to hypertrophy of muscular trabeculae. Rarely, osseous metaplasia was noted, representing chronic reparative changes. These histological findings collectively reflected a combination of acute inflammation, vascular compromise, tissue necrosis and reparative remodeling, which could severely impair pulmonary gas exchange and respiratory efficiency.

Gross Pathology



Photograph depicting a marked collection of straw-colored fluid within the abdominal cavity of Broiler chickens

Liver

Gross Pathology: The liver was prominently swollen, congested and edematous, reflecting systemic vascular compromise. The liver surface was generally smooth, though occasional irregular lobulation was noted, suggesting focal areas of fibrosis or regenerative nodules. The multifocal hemorrhages of varying sizes were observed, ranging from small petechiae to larger ecchymotic patches, indicative of vascular injury or systemic coagulopathy. The severe gross lesions included rupture with large adherent blood clots and in some cases, fibrinous clots of ascitic fluid entirely covered the lobes, demonstrating profound systemic circulatory compromise. The liver tissue was soft, heavy and in some cases, friable on palpation, consistent with edema accumulation. These gross changes suggested combined hepatocellular injury, vascular congestion and inflammatory response.

Histopathology: Microscopically, the liver displayed vascular congestion of central veins and sinusoids (Fig:2), accompanied by hepatocellular degeneration with cytoplasmic eosinophilia and vacuolar changes. The hepatocytes occasionally appeared rounded and individualized, reflecting cellular stress or early necrosis. The focal necrosis was commonly observed, particularly surrounding blood vessels commonly referred to as perivascular necrosis, with infiltration by heterophils and mononuclear cells, indicating an inflammatory response. The glisson's capsule was variably thickened, with cystic dilatation of lymphatic's and leukocytic infiltration. The Kupffer cells proliferated prominently, particularly in subcapsular regions, representing increased phagocytic activity. The hepatocytes sometimes displayed eosinophilic granular cytoplasm, suggesting protein accumulation or degeneration. The diffuse mononuclear infiltration, along with heterophils and lymph leakage from dilated lymphatics, was also evident.. Thus, these histological features reflected a combination of hepatocellular injury, inflammatory infiltration, vascular congestion and early reparative changes, potentially impairing metabolic and synthetic functions of the liver.

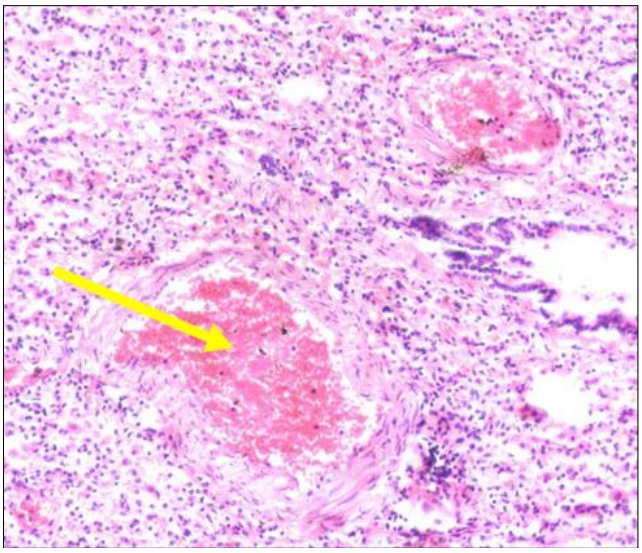


Fig 1: Photomicrograph of the affected lung tissue section showing severe vascular congestion, edema and thickening of wall of blood vessel (H&E, 10×)

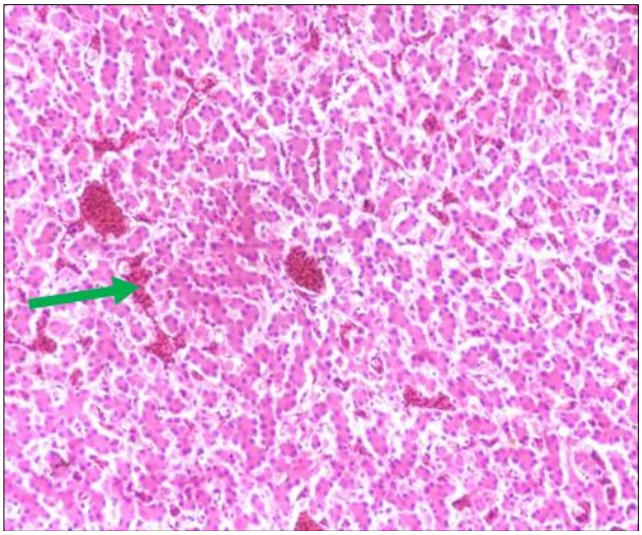


Fig 2: Photomicrograph of the liver tissue section depicting marked sinusoidal congestion (H&E, 10×)

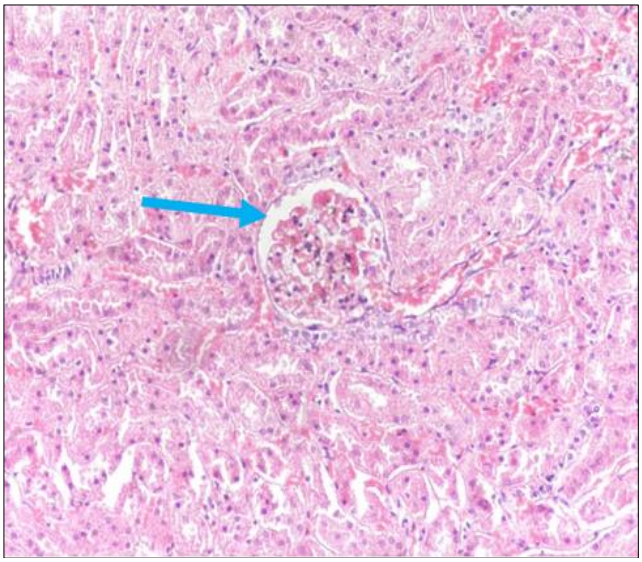


Fig 3: Microscopic analysis of affected kidney tissue section revealing glomerular atrophy and denudation of tubular epithelium (H&E, 10×)

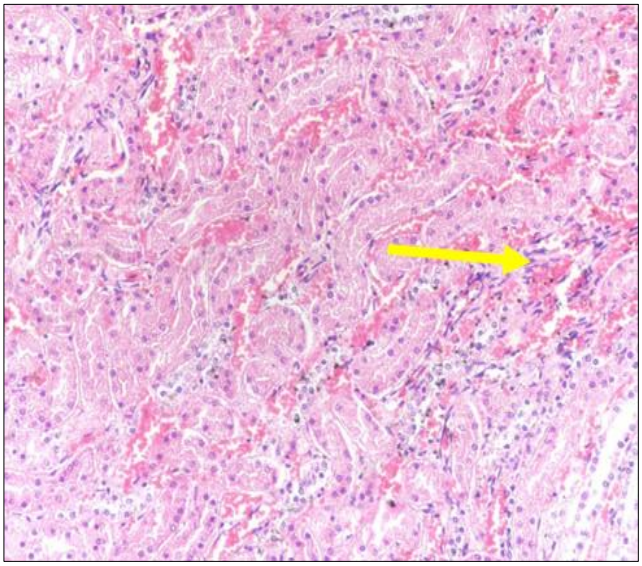


Fig 4: Photomicrograph of the affected kidney tissue section depicting marked interstitial nephritis (H&E, 10×)

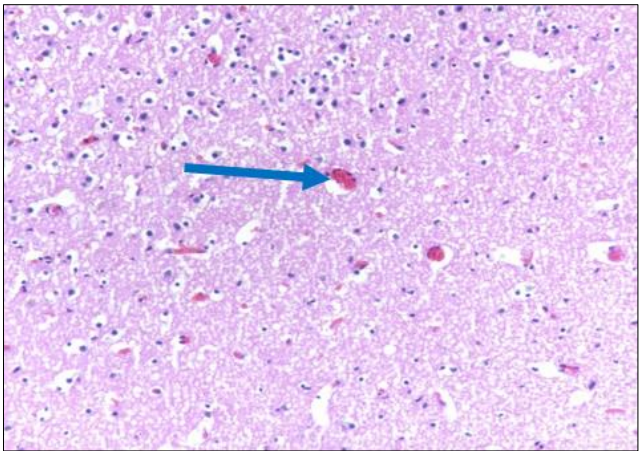


Fig 5: Photomicrograph of Brain tissue section illustrating vascular congestion (H&E, 10×)

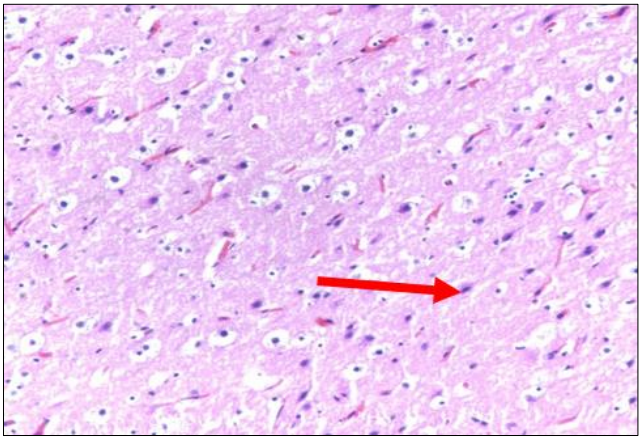


Fig 6: Microscopic analysis of affected Brain tissue section depicting neuronal degeneration (H&E, 10×)

Kidneys

Gross Pathology: The kidneys were swollen, edematous and protruding from the bony sockets, frequently showing dark-red congestion. In some cases, pale to greyish-white mottling was observed, indicating ischemic or degenerative foci. The multifocal hemorrhages were present in several affected birds, reflecting vascular compromise. The cortico-medullary distinction was sometimes obscured and the organ appeared firm to slightly friable, suggesting parenchymal edema and degeneration. These gross changes indicated combined vascular, inflammatory and degenerative processes compromising renal filtration and tubular function.

Histopathology: Microscopically, the kidneys demonstrated mild to severe congestion in cortical and medullary vessels, accompanied by focal to widespread hemorrhages with glomerular atrophy (Fig:3). The swelling of tubular epithelium and denudation of cortical tubular lining were observed in the affected kidneys. The microscopic analysis further reveals interstitial nephritis as evident with presence of inflammatory cells within the intersitium of the affected kidneys (Fig:4). The hyaline and granular casts were present within tubular lumens, indicating impaired filtration and tubular injury. The glomeruli were occasionally swollen, with vacuolated podocytes and focal nephritis. The inflammatory infiltration by mononuclear cells and heterophils was also noted in the interstitium. The sporadic capsular thickening was observed in the affected kidneys.

These histopathological changes suggested both degenerative and inflammatory renal injury, impairing renal excretory and homeostatic functions.

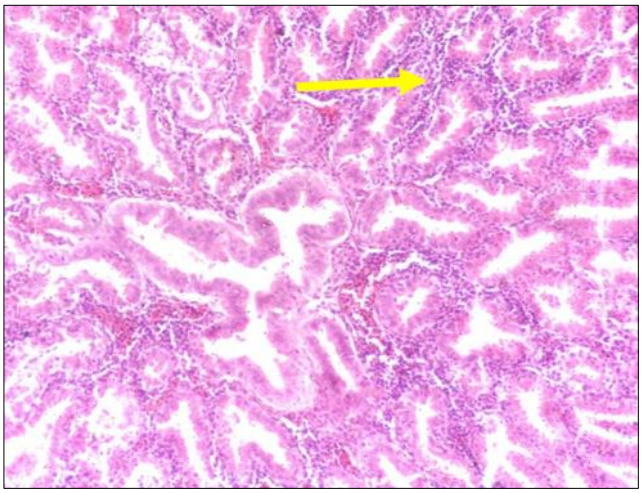


Fig 7: Photomicrograph of proventricular tissue section glandular epithelial degeneration (H&E, 10×)

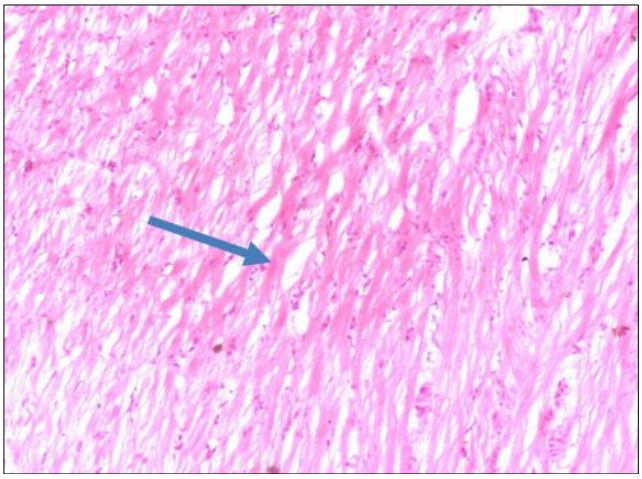


Fig 8: Microscopic analysis of affected heart tissue section revealing separation and disruption of myocardial fibres (H&E, 10×)

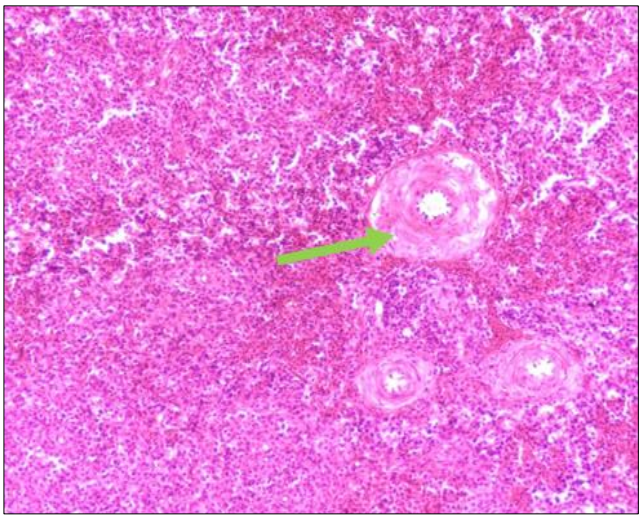


Fig 9: Photomicrograph of the affected spleen tissue section revealing thickening of the wall of the spleenic blood vessel (H&E, 10×)

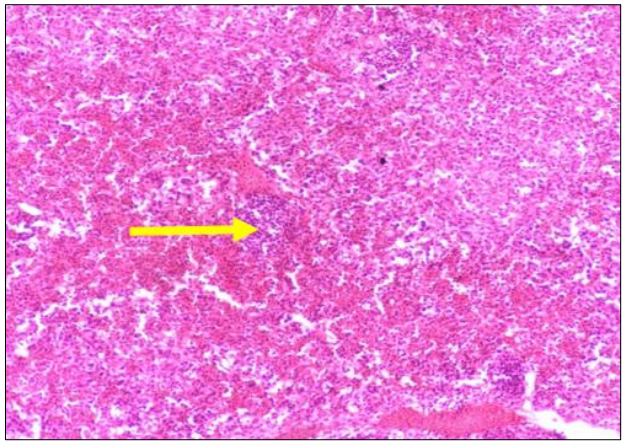


Fig 10: Photomicrograph of the affected spleen tissue section revealing spleenitis as evidenced with infiltration of the inflammatory cells (H&E, 10×)

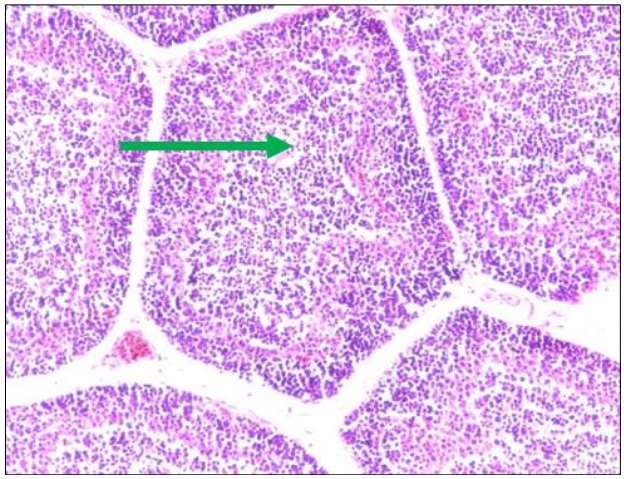


Fig 11: Photomicrograph demonstrating lymphoid depletion within the bursal follicles (H&E, 10×)

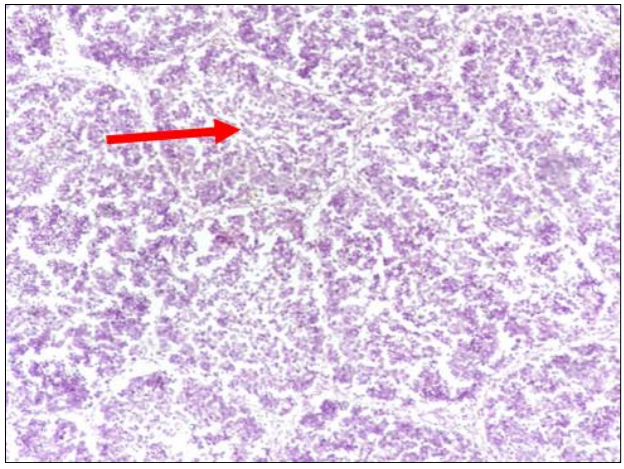


Fig 12: Microscopic analysis of affected thymus revealing atrophic thymic follicles caused due to severe cortical lymphoid depletion (H&E, 10×)

Brain: Gross Pathology: The brain appeared largely normal on gross examination, although mild to severe congestion of meningeal vessels was occasionally observed. The surface vessels appeared distended and subtle cortical or cerebellar discoloration was noted in severe cases, indicating vascular compromise.

Histopathology: Microscopically, vascular congestion was observed in the brain especially choroid plexus, cerebrum and cerebellum (Fig:5). The microscopic analysis of the affected brain tissue section revealing neuronal necrosis and degeneration (Fig:6). The necrotic neurons were shrunken, with cytoplasmic basophilia, pyknosis, karyorrhexis and karyolysis, often accompanied by satellitosis, neuronophagia and neuropil vacuolation. The occasional pronounced gliosis was noted. The medullary tracts showed mild demyelination and Purkinje cell degeneration in the cerebellum associated with vascular congestion. These histological changes reflected hypoxic-ischemic injury, inflammatory response and neuronal loss, potentially impairing central nervous system function.

Gastrointestinal Tract

Gross Pathology: The proventriculus occasionally exhibited mild to moderate edema, with thickened and friable mucosa corresponding to underlying vascular congestion.**.** The intestines consistently showed marked congestion of both mucosal and serosal surfaces. The luminal contents varied from semisolid to fluid, suggesting disturbed motility and absorption. The diffuse hemorrhages were prominent in the duodenum and jejunum, ranging from petechiae to larger ecchymotic patches. The mucosa frequently appeared velvety, and in severe cases, mucosal folds were flattened or eroded, with dark-red streaks indicative of submucosal hemorrhage. The mesenteric vessels were congested and mild distension suggested systemic circulatory stress. These findings indicated active enteritis, vascular compromise and functional impairment, including diarrhea, malabsorption and susceptibility to secondary infections.

Histopathology: Microscopically, the proventriculus exhibited mucosal and submucosal congestion with interstitial edema separating glandular and stromal components. The microscopic analysis of the affected proventricular tissue section further revealing glandular epithelial degeneration (Fig:7) The mild cellular infiltration by heterophils and mononuclear cells was evident. In the intestines, congestion of mucosal and submucosal vessels was more pronounced, with interstitial edema and lymphatic dilatation. Goblet cells displayed mucous degeneration, and the lamina propria contained heterophils and mononuclear cells, indicative of inflammatory changes. The villous blunting, epithelial desquamation and focal necrosis were observed in severe areas. The submucosal edema sometimes extended into muscularis layers and capillary networks were engorged, consistent with gross hyperemia. Thus, these histological findings suggested inflammatory enteritis with vascular compromise, explaining functional disturbances such as impaired nutrient absorption and fluid loss.

Thyroid Gland

Gross Pathology: The thyroid gland frequently appeared enlarged, swollen and congested, with brown discoloration, suggesting vascular compromise and interstitial edema accumulation. The glandular enlargement was mostly symmetrical, though localized focal enlargements were occasionally noted, potentially representing areas of hyperemia or early inflammation. The capsule appeared tense and the surface sometimes showed fine granularity, reflecting subcapsular congestion. On palpation, the gland was sometimes soft and friable, consistent with cellular degeneration or inflammatory infiltration. Thus, these gross changes indicate that the thyroid was under systemic stress, with possible vascular congestion and altered function, potentially impacting thyroid hormone production.

Histopathology: Microscopic examination revealed pronounced congestion in interstitial and capsular blood vessels, occasionally with perivascular edema. The affected thyroid follicles are varied in size, with many lined by squamous epithelium and containing homogeneous colloid, while active follicles showed cuboidal to columnar epithelium with peripheral colloid vacuolation, reflecting heightened secretory activity. The microscopic analysis further revealed mild perivascular mononuclear infiltration, indicating a subtle inflammatory response. The follicular cells sometimes displayed cytoplasmic vacuolation and degeneration, suggesting systemic stress or metabolic imbalance. These histological changes collectively reflect vascular congestion, altered follicular activity and early degenerative processes, which may affect thyroid hormone synthesis and endocrine homeostasis.

Parathyroid Gland

Gross Pathology: The parathyroid glands were enlarged and firm in consistency, with dark-red discoloration, indicative of vascular congestion. The gross pathological changes also include surface irregularities, hemorrhage or necrosis, suggesting altered structural integrity.

Histopathology: Microscopically, parathyroid architecture was altered, with densely packed cords of basophilic chief cells and scattered oxyphil cells. The vascular congestion, necrosis, inflammation, or parenchymal disruption was noted in the affected tissue section. These findings indicate functional changes, altered calcium homeostasis and parathyroid hormone secretion.

Adrenal Gland

Gross Pathology: The adrenal glands showed enlargement, with dark-red discoloration of the cortex and medulla, reflecting vascular congestion and hemorrhage. The capsule was occasionally tense, suggesting edema and vascular engorgement. The minor hemorrhagic streaks were sporadically observed, indicating localized vascular compromise. These findings emphasize the adrenal gland's sensitivity to systemic stress.

Histopathology: Microscopically, vascular congestion was evident in both cortex and medulla, with occasional focal hemorrhages. The cortical necrosis was observed, especially in the zona fasciculata and zona reticularis. The medullary chromaffin cells remained largely intact, although there was mild perivascular edema and sporadic leukocytic infiltration. These histological changes suggest impaired adrenal perfusion, which may influence corticosteroid secretion, affecting stress response, electrolyte balance and systemic homeostasis.

Spleen

Gross Pathology: The spleen was consistently enlarged, congested and dark-red, indicating systemic vascular compromise and circulatory stress. The hemorrhagic foci were observed, varying from pinpoint petechiae to larger

ecchymotic patches and in more severe cases, there was pronounced edema, producing a mottled parenchymal surface and a thickened, tense capsule. The splenic parenchyma was soft to friable, reflecting compromised tissue integrity, likely due to vascular congestion and interstitial fluid accumulation. These gross features suggest potential functional impairment, as these structural alterations could hinder normal hematopoietic and immune functions of the spleen.

Histopathology: The microscopic examination of the affected spleen revealed severe congestion of the red pulp, frequently accompanied by multifocal hemorrhages and thickening of the wall of the spleenic blood vessel (Fig:9). The white pulp and periarteriolar lymphoid sheaths showed lymphoid depletion, indicating a reduction in immune cell populations. The multifocal necrosis was noted, associated with thickening of vascular walls, cystic dilation and leakage of lymph into surrounding interstitial tissue. The necrotic areas were infiltrated by inflammatory cells particularly heterophils (Fig:10). These findings collectively indicate immune exhaustion, impaired hematopoietic function and reduced immunologic competence, emphasizing the spleen's dual role in vascular regulation and systemic immune defense.

Bursa of Fabricius

Gross Pathology: The bursa was enlarged, edematous, congested and frequently hemorrhagic, with soft, friable mucosa and serous exudates within the lumen, indicating severe vascular compromise and active inflammatory processes. These gross changes are consistent with impaired lymphoid organ function and likely compromised B-cell development, which would have direct implications for humoral immunity.

Histopathology: Microscopically, the bursa exhibited vascular congestion and interstitial edema, with focal hemorrhages observed in both medullary and cortical regions. The lymphoid depletion ranged from mild to severe and infiltration by heterophils intermixed with mononuclear cells was noted in the mucosa and submucosa (Fig:11). There was also degeneration of the mucosal epithelium and formation of cystic spaces, indicating structural compromise. Thus, these histopathological findings collectively reflect severe immunosuppression, impaired lymphoid tissue architecture, and defective humoral immunity due to compromised B-cell development, underscoring the bursa's critical role in maintaining adaptive immune responses.

Thymus

Gross Pathology: The thymus exhibited swelling, edema and congestion, with prominent lobular contours resulting from interstitial fluid accumulation. The surface was occasionally discolored and the tissue was soft and friable, consistent with early degenerative changes secondary to systemic stress or circulatory compromise. These gross pathological changes indicate structural disruption and potential functional impairment of T-cell maturation, suggesting that the organ's capacity for generating mature T lymphocytes may be compromised under systemic stress conditions.

Histopathology: Microscopically, the thymus showed pronounced vascular congestion and interstitial edema, with marked cortical lymphoid depletion, reflecting a loss of functional T lymphocytes from thymic lymphoid follicles (Fig:12). Additionally, heterophil infiltration was observed within Hassall's corpuscles and the surrounding stromal tissue, indicative of a systemic inflammatory or stress response. Thus, these histological changes suggest immunosuppression, reduced thymic output of mature T cells and compromised adaptive immunity, which may predispose the birds to increased susceptibility to infections.

Discussion

Broiler production has become one of the fastest-growing sectors of the poultry industry due to rapid growth rates, efficient feed conversion and high demand for meat. Despite these advantages, intensive production exposes birds to metabolic disorders, especially under improper management practices such as low ambient temperatures, poor ventilation and suboptimal brooding (Baghbanzadeh *et al.,* 2008) [1, 4]. Among these disorders, ascites syndrome is a significant cause of mortality and economic loss in broiler flocks. This syndrome arises when birds' physiological capacity to cope with rapid growth and environmental stressors is exceeded, resulting in cardiopulmonary imbalance and fluid accumulation in the abdominal cavity (Wideman *et al.*, 2013) [24]. The optimizing environmental conditions, including precise control of ambient temperature, ventilation and brooding practices, is therefore essential to prevent ascites and maintain flock health (Malan *et al.*, 2003; Tekeli *et al.*, 2014) [17, 23].

Clinically, affected broilers exhibit lethargy, stunted growth, ruffled feathers and increased respiratory effort, including open-mouth breathing and exaggerated abdominal movements as compensatory responses to hypoxemia (Baghbanzadeh and Decuypere, 2008) [1, 4]. The abdominal distension due to fluid accumulation imparts a swollen appearance, while cyanosis of combs, wattles and extremities reflects underlying hypoxia (Malan *et al.*, 2003; Maxwell *et al.*, 1986) [17, 18]. The reduced feed intake, sudden mortality and signs of right-sided heart failure including accumulation of ascitic fluid in the coelomic cavity are common, particularly under cold stress or poor ventilation (Shafi *et al.*, 2025) [21].

Pathological examination demonstrates systemic effects of pulmonary hypertension, hypoxia and circulatory compromise, resulting in multi-organ dysfunction. The lungs show congestion, edema, dark-red discoloration and consolidation, often with frothy or caseous exudates in the airways, reflecting acute inflammation and vascular injury (Biswas *et al.*, 2007) [3]. Microscopically, interstitial and alveolar congestion, hemorrhage, hypertrophy of pulmonary arterioles, perivascular leukocytic infiltration and rare plexiform lesions indicate both acute injury and chronic vascular remodeling due to prolonged hypoxia (Wideman *et al.*, 2011; Bautista-Ortega *et al.*,, 2012) [25, 2]. Some cases of affected lung tissue section also reveals atelectasis, heterophilic infiltration and smooth muscle hypertrophy in the capillaries (Suleiman *et al.*, 2018) [22].

The liver is swollen, congested, edematous and hemorrhagic with fibrinous clots and irregular lobulation, reflecting systemic vascular compromise (Dimple *et al.*, 2005) [8]. Histopathology reveals central vein and sinusoidal congestion, hepatocellular degeneration, perivascular

necrosis, heterophil and mononuclear infiltration and Kupffer cell proliferation, indicating impaired metabolic and synthetic function (Davis *et al.,* 2012) [6]. The affected Kidneys show congestion, edema, hemorrhage, glomerular atrophy, denudation of renal tubular epithelium, tubular epithelial swelling, and glomerular vacuolation, reflecting degenerative and inflammatory injury (Lorenzoni *et al.*, 2008) [14]. Some severe cases of affected Kidney also reveal tubular degeneration, epithelial sloughing and lymphocytic aggregation in ascitic birds (Shafi *et al.,* 2023). These pathological changes collectively indicate that pulmonary lesions and hypoxemia trigger systemic vascular and inflammatory responses, leading to hepatic & renal dysfunction (Wideman *et al.,* 2013) [24]. The histopathological changes in the heart include hydropericardium, pericardial edema, separation of myocardial fibers, vascular congestion and myofibril degeneration (Janwari *et al.,* 2018; Umara *et al.,* 2021) [11,]. The brain appears grossly congested but microscopically demonstrates necrosis, satellitosis, perivascular space dilation, neuronal degeneration, neuronophagia, vascular congestion, mild gliosis and demyelination, indicating hypoxic-ischemic injury (Moayyedian *et al.,* 2011, Shafi *et al.*, 2025) [19, 21].

The gastrointestinal tract displays mucosal and serosal congestion, edema, hemorrhage, villous blunting, epithelial desquamation and lymphatic dilation, indicative of enteritis and functional impairment (Habib-ur-Rehman *et al.,* 2007) [20, 7]. The endocrine organs exhibit variable changes as the thyroid shows congestion with follicular vacuolation, while adrenal glands have cortical and medullary congestion, hemorrhage and perivascular edema, reflecting compromised hormone secretion (Bautista-Ortega and Ruiz-Feria, 2010) [2]. The affected lymphoid organs including the spleen, thymus and bursa of Fabricius exhibit vascular congestion, hemorrhage, lymphoid depletion and interstitial edema, suggesting immunosuppression (Kluess *et al.,* 2012; Ruiz-Feria *et al.*, 2009) [12].

The region-specific studies in Kashmir further emphasize the epidemiology and pathophysiology of pulmonary arterial hypertension (PHS) under temperate climatic conditions. The mortality patterns indicate higher susceptibility in younger birds and during rapid growth periods (Janwari *et al.*, 2019; Shafi *et al.,* 2025) [21]. Clinically, affected birds exhibit right ventricular hypertrophy, abdominal fluid accumulation, elevated hematocrit, leukocyte imbalances, and serum biochemical disturbances, reflecting multi-organ stress (Janwari *et al.,* 2018a) [8]. The epidemiological surveys report revealed that high PHS prevalence are influenced by environmental management, seasonal fluctuations and genetic predisposition (Janwari *et al.*, 2018b) [9]. Gross and histopathological examinations reveal pulmonary congestion, right ventricular dilation, hepatic congestion, ascites, vascular remodeling of pulmonary arterioles, cardiomyocyte degeneration, interstitial hemorrhages and hepatic sinusoidal congestion (Shafi *et al.*, 2025 Janwari *et al.,* 2018c) [21, 10]. The degenerative and inflammatory changes in the lymphoid organs like spleen, bursa, thymus and caecal tonsils further indicate compromised immunity in stressed broilers (Shafi *et al.*, 2025) [21].

Thus, the ascites in broilers arise from a multifactorial combination of environmental stress, rapid growth, nutritional imbalances and genetic predisposition. The effective management requires a holistic approach, including optimized housing and ventilation, precise control of temperature and lighting, balanced diets and selective breeding for cardiovascular resilience. The early monitoring and timely interventions are critical to prevent disease progression, reduce mortality and maintain both bird welfare and productivity. Thus, it is important to address this issue through integrated, proactive strategies ensures sustainable and healthy broiler production (Wideman *et al.,* 2013) [24].

Conclusion

The pathological alterations observed in ascitic broilers include lymphoid depletion, infiltration of mononuclear cells and necrotic changes in vital organs, which collectively suggest marked immune suppression and systemic stress. The pulmonary lesions such as congestion, edema and bronchial dilatation further emphasize the substantial respiratory involvement associated with ascites. These extensive tissue damages point out the profound impact of the condition on multiple vital organs of the affected birds.

Conflict of Interest: The authors declare that they have no conflict of interest.

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