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Pathomorphological study of fatty liver hemorrhagic syndrome in layer chickens

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Abstract

Fatty Liver Hemorrhagic Syndrome (FLHS) is an important metabolic disorder of layers characterized by excessive fat retention in the liver resulting in decreased productivity, sudden mortality, and huge economic losses to the poultry industry. The present study was conducted to determine the occurrence and pathological changes associated with FLHS in layer birds presented for postmortem examination at the Department of Veterinary Pathology, College of Veterinary Science, Warangal, Telangana, India, during the period from April to July, 2026. A total of 125 birds were subjected to detailed necropsy examination, of which 20 birds (16%) were diagnosed with FLHS based on gross and histopathological findings. Gross pathological alterations were enlarged, friable, yellowish livers with multifocal to diffuse hemorrhages and hematomas. Excessive fat deposition was evident in the abdominal cavity, gizzard and heart. Histopathology of liver sections revealed hepatocellular vacuolation due to lipid accumulation. The findings confirm that FLHS was one of the cause of mortality among layers during the study period and emphasize the importance of appropriate nutritional and management practices for its prevention.

Keywords: Fatty liver hemorrhagic syndrome, layers, necropsy and histopathology

Introduction

Fatty Liver Hemorrhagic Syndrome (FLHS) is a non-infectious metabolic disorder of layers impacts both egg production and egg quality and leads to sudden mortality and substantial economic losses to the poultry industry. The condition is characterized by excessive hepatic lipid accumulation leading to increased fragility and liver rupture followed by severe internal hemorrhage and death ^[13, 15]. A marked decline in the egg production has been reported by some researchers ^[16, 5]. Although FLHS is predominantly associated with caged layers, it has also been reported in free-range backyard poultry flocks in northern California ^[15].

FLHS is a multifactorial metabolic disorder caused by complex etiological factors, involving genetic, nutritional, environmental and endocrine factors. Housing systems are considered an important risk factor for FLHS. Compared with free-range systems, conventional cage housing predisposes the birds to FLHS due to restricted movement, reducing energy expenditure, and increasing the likelihood of obesity ^[18]. Heat stress is a significant environmental factor associated with FLHS, as reduced thermoregulatory energy expenditure during warmer conditions favors a positive energy balance and consequently increases the risk of FLHS during hot seasons ^[17]. Nutritional imbalance is another important cause of FLHS. Lipotropic factors like choline, methionine, vitamin E and vitamin B12 play role in hepatic lipid transport and mobilization. Deficiency of these nutrients predisposes to fatty liver ^[14]. FLHS was reported in hens fed with high energy diets, low linoleic acid, and low choline ^[19].

The condition is characterized by dysregulation of hepatic lipid homeostasis, wherein hepatic lipid synthesis and accumulation exceed the liver's capacity for lipid oxidation and export ^[14, 16]. When energy intake exceeds the metabolic demands of maintenance and egg production, the surplus energy is converted into triglycerides, which accumulate within hepatocytes, causing hepatic steatosis and hepatocellular enlargement ^[2]. Progressive lipid accumulation compromises normal hepatic structure and integrity, rendering the liver enlarged, fragile and prone to rupture under minor mechanical stress or physiological strain, ultimately causing severe internal hemorrhage and sudden death ^[15, 20].

The present investigation was undertaken to determine the occurrence of FLHS among poultry carcasses presented for postmortem examination and to document the associated gross and microscopic lesions.

Materials and Methods

The present study was carried out in the Department of Veterinary Pathology, College of Veterinary Science, Warangal, Telangana, for a period of four months from April to July, 2026. A total of 125 poultry carcasses were submitted for routine postmortem examination during the study period. A complete necropsy was performed following standard veterinary pathological procedures. Birds showing gross lesions suggestive of FLHS were selected for detailed pathological examination. The available clinical history and observed clinical signs were documented, and all major visceral organs were systematically examined for gross pathological changes. Representative liver tissue samples were collected immediately after necropsy and fixed in 10% neutral buffered formalin for histopathological evaluation. The tissues were processed by paraffin embedding technique, sectioned (5 μ m thick) and stained with Hematoxylin and Eosin, according to standard histological procedures [9].

Results, Discussion and Conclusion

During the study period from April to July, 2026, a total of 125 poultry carcasses were presented for postmortem examination. Among those a 20 birds (16%) were diagnosed with FLHS based on the gross and histopathological findings. Farm-level information revealed that most of the affected birds were found dead without any obvious warning signs. In some cases, a marked decline in egg production, pale combs, lethargy, or unexpected mortality was reported. The rapid progression of FLHS is associated with hepatic rupture and intracoelomic hemorrhage, resulting in acute hemorrhagic shock, pallor of the comb, and sudden death with minimal or no preceding clinical manifestations [6, 11]. Gross pathological examination of the affected birds demonstrated lesions characteristic of FLHS. The most consistent observation was extensive accumulation of adipose tissue throughout the coelomic cavity, resulting in extensive abdominal fat deposition and obesity. Excessive adipose tissue surrounded the visceral organs, reducing the available coelomic space (Fig. 1). The fat deposition was markedly higher in the gizzard compared to other abdominal organs (Fig. 2). The most predominant gross lesions were observed in the liver. The liver was markedly enlarged with rounded margins and appeared soft and friable on palpation. Liver exhibited a pale tan to yellow-brown color with smooth, and glistening surface suggestive of severe hepatic steatosis. The liver lobes revealed prominent congestion and the capsule exhibited multiple subcapsular hematomas accompanied by numerous petechial and ecchymotic hemorrhages distributed over the liver surface (Fig. 3). Severe cases reported rupture of the hepatic parenchyma, accompanied by extensive intracoelomic hemorrhage and formation of large organized blood clots, resulting in a hemoabdomen (Fig. 4). Our results corroborate the findings of previous authors [10, 11]. The hepatic rupture observed in severe cases may be attributed to disruption of the reticulin framework (reticulolysis), which weakens the supporting architecture of the hepatic parenchyma and reduces the mechanical strength of the liver. This structural

compromise, together with extensive lipid accumulation and hepatocellular enlargement, may predispose the liver to hemorrhage and subsequent rupture [4]. In few cases, marked accumulation of coronary fat around the heart was observed, suggesting that excessive energy intake coupled with restricted physical activity may contribute to the development of the syndrome, as previously reported [2, 8]. Hemorrhagic and degenerative ovarian follicles were commonly observed, with rupture of follicular contents and deposition of egg material or presence of malformed egg in the reproductive tract in some cases (Fig. 5), indicating disruption of normal reproductive physiology associated with systemic metabolic imbalance. A fully formed egg was also reported in few cases (Fig. 2). These findings further support the established pathological characteristics of the syndrome described previously [11].

The liver sections revealed severe hepatocellular vacuolation associated with extensive hemorrhage. The hepatocytes were enlarged, characterized by prominent clear vacuoles within the cytoplasm, with eccentrically placed nuclei suggestive of fatty degeneration. Similar histopathological alterations have also been documented by other researchers. [15, 12, 1, 7].

FLHS is commonly has been linked to several management and nutritional factors, such as cage housing system, overcrowding, lack of exercise and high energy diets. In the present study, the occurrence of FLHS may have been associated with cage housing system, high energy diet and restricted exercise. Furthermore, FLHS was observed predominantly in birds during the active laying phase, indicating that the increased metabolic and hormonal demands associated with egg production could contribute to the development of hepatic lipid accumulation [3].

From a management perspective, maintaining an optimal dietary energy level, ensuring adequate ventilation, appropriate supplementation with lipotropic agents to support hepatic lipid metabolism, and minimizing environmental and management-related stressors are important strategies, particularly during the active laying period. Together, these measures may help maintain liver health and reduce the likelihood of FLHS in laying birds.



Fig 1: Marked accumulation of adipose tissue within the coelomic cavity and an enlarged, pale, fatty liver with rounded borders.



Fig 2: Excessive fat deposition in gizzard and a fully formed egg in the reproductive tract.



Fig 5: Hemorrhagic ovarian follicles with a malformed egg in the reproductive tract.



Fig 3: The liver lobes show prominent congestion and hepatomegaly accompanied by multifocal dark reddish-purple areas/hemorrhages on the surface and abdominal fat pad occupying the lower visceral cavity.

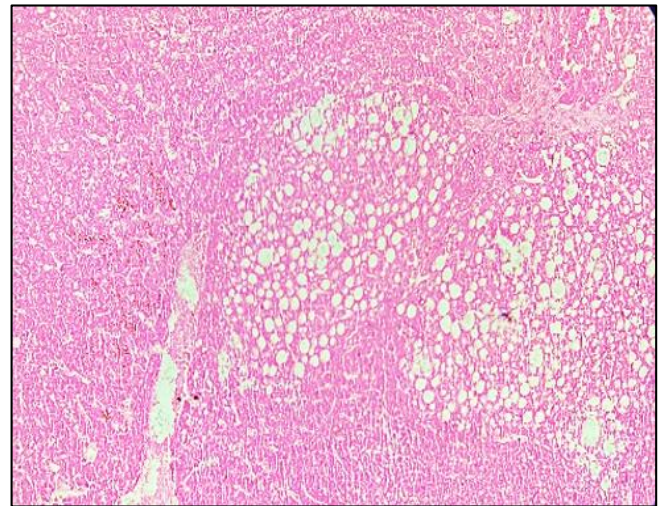


Fig 6: Numerous distinct, clear vacuoles within the hepatocytes. Areas of normal hepatic cords are interspersed with patches of severe fatty infiltration, disrupting the standard lobular architecture.

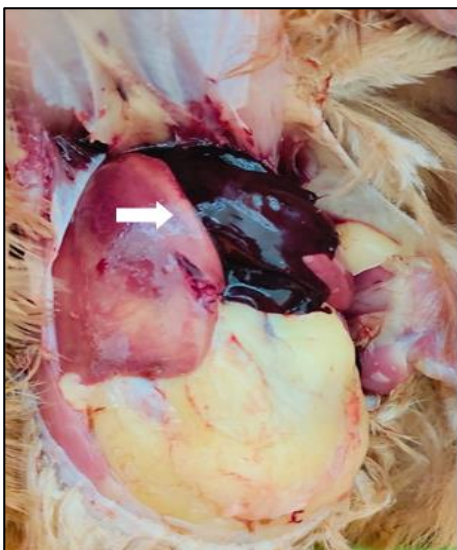


Fig 4: Ruptured liver lobe accompanied by extensive hemorrhage and formation of large organized blood clot.

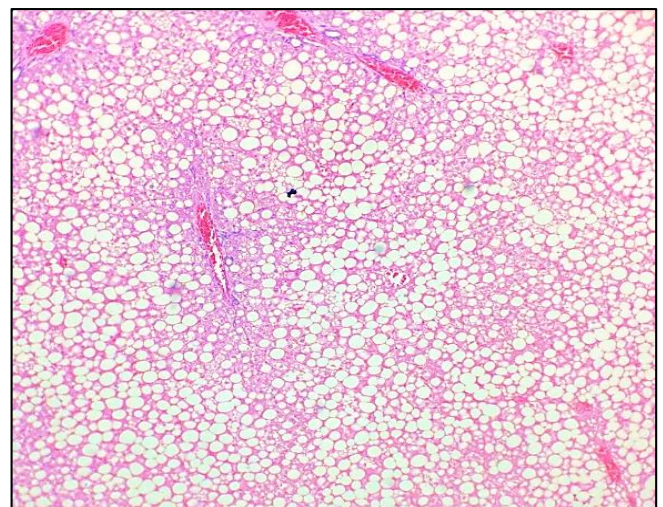


Fig 7: Hepatocytes are distended by numerous well-defined, clear lipid vacuoles, displacing the nuclei to the periphery. Portal triads and central veins are visible amidst the diffusely vacuolated parenchyma, accompanied by moderate congestion.

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Conflict of Interest

All authors declare that they have no conflict of interest

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