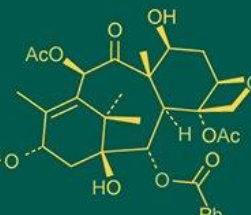
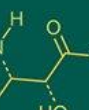
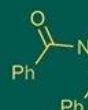


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2026): 5.29
IJABR 2026; 10(9): 46-49
www.biochemjournal.com
Received: 19-06-2026
Accepted: 22-07-2026

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Pathological characterization of Fatty Liver Haemorrhagic Syndrome (FLHS) in cross bred native chicken

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DOI: <https://www.doi.org/10.33545/26174693.2026.v10.i9a.9825>

Abstract

Fatty Liver Haemorrhagic Syndrome (FLHS) is an important metabolic disorder affecting laying hens, characterized by excessive lipid accumulation in the liver, hepatic haemorrhages, and sudden mortality. The present study reports a pathological investigation of FLHS in crossbred native layer chickens from a desi poultry farm in Theni district. The farm comprises of totally 2000 birds which experienced sudden mortality and a marked decline in egg production. Vaccination and deworming were regularly followed and biosecurity measures were done appropriately. Postmortem examination revealed excessive accumulation of abdominal fat, with fatty infiltration around the liver, heart, intestine, and gizzard. The liver appeared enlarged, pale, greasy, and friable. Histopathological examination of the liver showed severe hepatic steatosis, characterized by distended hepatocytes containing large clear vacuoles that displaced the nuclei to the periphery. Similar vacuolar changes were observed in the cardiac muscle. These findings confirmed the diagnosis of FLHS. The study highlights the significance of gross and histopathological examination as reliable diagnostic tools and emphasizes the role of nutritional, metabolic, and environmental factors in the pathogenesis of FLHS in laying chickens.

Keywords: Fatty liver haemorrhagic syndrome, hepatic steatosis, layer chicken, histopathology

Introduction

Fatty Liver Haemorrhagic Syndrome (FLHS) is a metabolic disorder of poultry characterized by excessive fat accumulation in the liver accompanied by hepatic haemorrhages and sudden death (Butler, 1976; Julian, 2005) ^[1, 5]. The condition is most commonly observed in high-producing laying hens, particularly those reared under cage systems and exposed to high environmental temperatures (Whitehead, 1982; Crespo and Shivaprasad, 2003) ^[14, 2].

FLHS is frequently associated with a decline in egg production and sporadic mortality, resulting in substantial economic losses to poultry producers (Butler, 1976; Julian, 2005) ^[1, 5]. Death often occurs suddenly due to massive liver rupture and internal haemorrhage, sometimes without any preceding clinical signs (Crespo and Shivaprasad, 2003) ^[2].

There are fundamental differences in carbohydrate and fat metabolism between birds and mammals. Birds maintain blood glucose levels that are approximately three to four times higher than those of mammals. Unlike mammals, where adipose tissue is the major site of lipogenesis, the liver is the primary site of fatty acid synthesis in birds. During embryonic life, lipid metabolism depends largely on yolk-derived fat. After hatching, hepatic lipogenesis increases rapidly to support growth and development (Whitehead, 1982) ^[14].

As chickens reach sexual maturity and commence egg laying, hepatic lipogenesis increases dramatically to meet the demand for yolk precursor synthesis, including very low-density lipoproteins (VLDL) and vitellogenin (Hermier, 1997) ^[4]. Consequently, laying hens exhibit elevated plasma lipid levels and increased fat storage in the liver. When lipid synthesis exceeds hepatic export, excessive fat accumulates in hepatocytes, predisposing birds to FLHS (Butler, 1976; Julian, 2005) ^[1, 5].

FLHS is considered a non-infectious, multifactorial condition. Nutritional factors such as excess dietary energy, high carbohydrate intake, low protein diets, deficiencies of lipotropic factors (choline, methionine, biotin), fungal toxins and anti-nutritional substances have been implicated in its development (Whitehead, 1982; Leeson and Summers, 2001; Julian, 2005) ^[14, 9, 5].

Environmental factors including high ambient temperature, lack of exercise and stress further exacerbate lipid accumulation in the liver (Crespo and Shivaprasad, 2003) [2]. Genetic predisposition and hormonal influences, particularly estrogen-induced lipogenesis, also play a significant role (Hermier, 1997) [4].

Although FLHS has been extensively reported in commercial layer flocks, documentation of the condition in crossbred native chickens is limited. Hence, the present study was undertaken to describe the gross and histopathological features of FLHS in crossbred native layer chickens.

Materials and Methods:

A desi chicken farm in Theni district reported sudden mortality among adult laying birds along with a noticeable reduction in egg production. Representative carcasses were submitted for postmortem examination. A detailed necropsy was performed following standard pathological procedures (Luna, 1968) [10].

Tissue samples from the liver and heart were collected under aseptic conditions and fixed in 10% neutral buffered formalin. After fixation, tissues were washed overnight under running tap water, dehydrated in ascending grades of

alcohol, cleared in xylene and embedded in paraffin wax. Sections of 5 µm thickness were cut using a microtome and stained with haematoxylin and eosin (H&E) as per standard protocol (Luna, 1968) [10].

Results and Discussion

Gross Pathological Findings

Gross pathological examination revealed excessive accumulation of fat in the abdominal cavity. The abdominal fat was observed to be creamy white in one bird and yellowish in another, indicating variation in lipid deposition and chronicity of the condition. The heart was surrounded by abundant fat, and the gizzard appeared embedded within adipose tissue. The intestines were also encased in excessive fat deposits.

The liver was enlarged, pale, greasy and friable. These gross changes are attributed to excessive triglyceride accumulation within hepatocytes, leading to compromised structural integrity of hepatic tissue (Julian, 2005) [5].

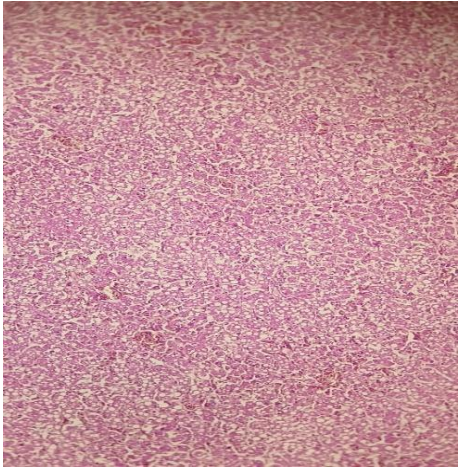
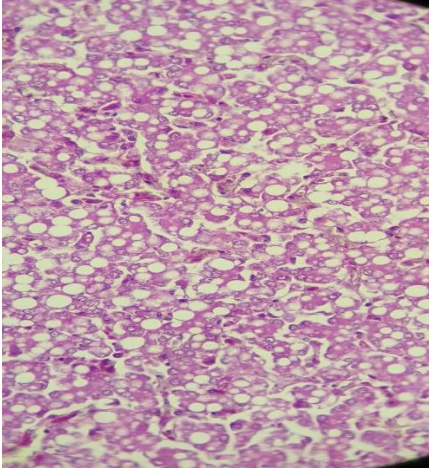
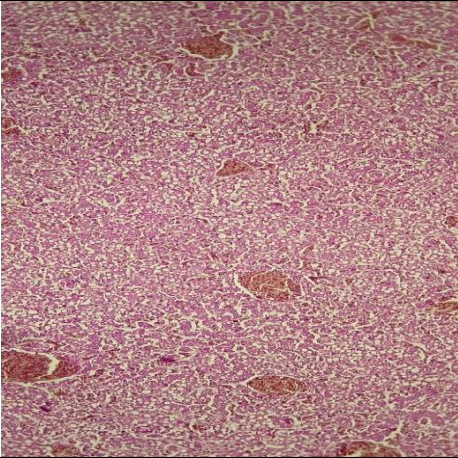
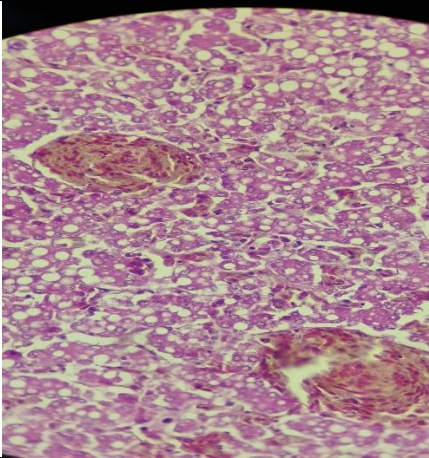
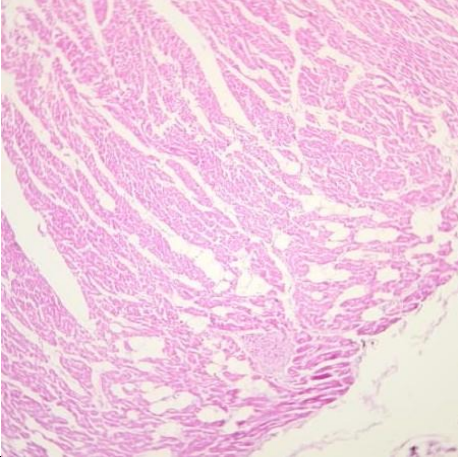
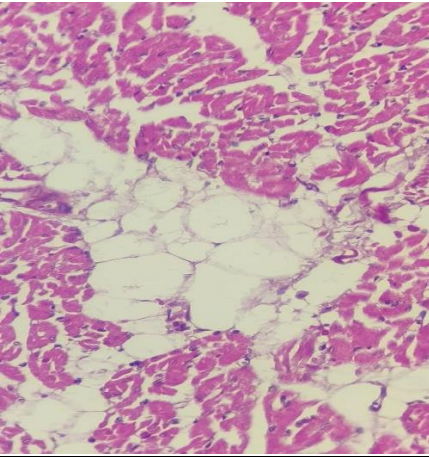
Similar gross lesions have been reported by earlier workers (Whitehead, 1981; Trott *et al.*, 2014, Butler, 1976; Crespo and Shivaprasad, 2003) [14, 13, 1, 2] who described enlarged, yellowish, soft livers with extensive fat infiltration and a tendency for spontaneous haemorrhage

Abdominal fat deposition (creamy whitish colour)	Abdominal fat deposition (Yellowish colour)
	
Fat accumulation in the liver	Gizzard surrounded by fat
	
Fat accumulation around the intestine of layer chicken	Heart surrounded by fat
	

Histopathological Findings

Histopathological examination of the liver revealed severe hepatic steatosis. Hepatocytes were markedly distended with large clear cytoplasmic vacuoles that displaced the nuclei to the periphery, producing a typical signet-ring appearance. These vacuoles represent lipid droplets dissolved during routine histological processing (Luna, 1968)^[10]. In several areas, hepatocytes showed degenerative changes, cytoplasmic vacuolation and focal necrosis. Severe hepatic steatosis compromises cellular metabolism and increases susceptibility to hypoxia, mechanical rupture and

haemorrhage (Trott *et al.*, 2014)^[13]. These findings are consistent with previous reports describing hepatocellular degeneration, vacuolation and necrosis in FLHS-affected birds (Butler, 1976; Crespo and Shivaprasad, 2003)^[1, 2]. Histological sections of the heart revealed clear vacuoles within the myocardial fibers, indicating fatty infiltration. Although liver lesions are the hallmark of FLHS, lipid deposition in other organs such as the heart has been reported in advanced cases and may contribute to sudden death (Julian, 2005)^[5].

Liver showing fat vacuoles	
Liver shows various size vacuoles with haemorrhages (10x)	Liver shows macro and micro vacuoles (40x)
	
Liver showing haemorrhage	
Liver with vacuoles and congestion (10x)	Liver with various size vacuoles with congestion and haemorrhage (40x)
	
Heart with fat vacuoles	
Heart shows deposition of fat (10x)	Heart shows numerous fat cell in myocardium (40x)
	

Pathogenesis and Predisposing Factors

The pathogenesis of FLHS is closely associated with excessive hepatic lipogenesis and impaired lipid export. Estrogen stimulates hepatic lipid synthesis during egg production, while excess dietary energy further aggravates fat accumulation (Hermier, 1997) ^[4]. High ambient temperature reduces physical activity and alters metabolic pathways, increasing susceptibility to FLHS (Whitehead, 1981; Crespo and Shivaprasad, 2003) ^[14, 2].

Nutritional deficiencies of lipotropic factors impair the mobilization and transport of fat from the liver, promoting steatosis (Leeson and Summers, 2001) ^[9]. Mycotoxins and anti-nutritional factors can further damage hepatic function and exacerbate lipid accumulation (Julian, 2005) ^[5].

Nutritional and Toxic Factors

Deficiencies of lipotropic factors such as choline, methionine and biotin impair the mobilization and transport of fat from the liver, increasing the risk of hepatic steatosis (Leeson and Summers, 2001) ^[9]. Choline deficiency, in particular, disrupts phospholipid synthesis required for lipoprotein formation, leading to fat retention in hepatocytes (Whitehead, 1981) ^[14].

Mycotoxins and anti-nutritional factors present in feed ingredients may further compromise hepatic function. Certain mycotoxins are known to interfere with lipid metabolism and induce oxidative damage to hepatocytes, thereby aggravating FLHS (Julian, 2005) ^[5].

Economic and Welfare Implications

FLHS causes significant economic losses due to sudden mortality, decreased egg production and reduced feed efficiency (Butler, 1976; Julian, 2005) ^[1, 5]. Subclinical cases may remain undiagnosed, yet they contribute to poor flock performance and increased production costs.

From an animal welfare perspective, FLHS represents a serious metabolic stress condition. Sudden death due to internal haemorrhage underscores the importance of preventive management strategies aimed at maintaining metabolic balance and liver health.

Diagnosis and Preventive Strategies

Gross and histopathological examinations are the most reliable diagnostic tools for FLHS (Trott *et al.*, 2014) ^[13]. While biochemical indicators such as elevated liver enzymes and plasma lipid levels may support diagnosis, definitive confirmation requires pathological evaluation.

Preventive measures include:

- Balanced diets with controlled energy levels
- Adequate protein and supplementation of lipotropic factors
- Avoidance of mycotoxin-contaminated feed
- Environmental management to reduce heat stress
- Housing systems that allow sufficient physical activity

Genetic selection for birds with improved metabolic efficiency may also reduce susceptibility to FLHS in the long term (Hermier, 1997) ^[4].

Conclusion

The present investigation provides a comprehensive pathological characterization of Fatty Liver Haemorrhagic Syndrome in crossbred native chickens. The combination of extensive abdominal fat deposition, enlarged pale greasy liver and severe hepatic steatosis confirms the diagnosis of FLHS. Histopathological findings of hepatocellular

vacuolation, degeneration and focal necrosis underscore the severity of hepatic damage associated with this condition. The study highlights the multifactorial nature of FLHS and emphasizes the critical role of nutrition, environment and management in its prevention. Early diagnosis through pathological examination and implementation of appropriate preventive strategies are essential to minimize economic losses and improve poultry health and welfare.

Disclaimer

The authors declare that no generative artificial intelligence tools, including large language models or text-to-image generators, were used in the preparation, writing, or editing of this manuscript.

Competing Interests

Authors have declared that no competing interests exist.

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