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Occurrence and Pathology of Fatty Liver Haemorrhagic Syndrome in Chickens in and Around Hyderabad, India

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Abstract
Fatty liver haemorrhagic syndrome (FLHS) is a non-infectious metabolic disease, mainly of laying hens, in which fat accumulates in the liver and can rupture, causing fatal intra-abdominal haemorrhage. This study describes the occurrence and pathology of FLHS in chickens in and around Hyderabad, Telangana, from July to December 2025. Carcasses were collected from 45 poultry farms and from cases submitted to the Department of Veterinary Pathology, Rajendranagar, for post-mortem examination. FLHS caused 65 deaths across two outbreaks, a case prevalence of 8.56% and the highest disease-specific mortality (3.25%) among the conditions studied. No deaths occurred before 22 days of age. Mortality was 4.00% at 22–28 days and 5.62% in birds older than 29 days. Grossly, the livers were enlarged, soft, friable and discoloured tan to yellow or orange, with intraparenchymal and subcapsular haemorrhages and blood clots in the coelomic cavity. Histopathology showed variable hepatocellular vacuolation, sinusoidal haemorrhages, hepatocyte pyknosis and chromatin margination. The authors conclude that FLHS is an important cause of mortality in older birds in the study area. Keywords: Fatty liver haemorrhagic syndrome, subcapsular haemorrhages, hepatocellular vacuolation, chromatin margination.

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1. Introduction

Over the past five decades, the Indian poultry industry has undergone a profound transformation from traditional, obsolete farming systems to highly intensive commercial operations. Presently, poultry ranks among the most rapidly developing agrobusiness sectors in India, with marked advances in genetics, husbandry, housing, and biosecurity. However, despite these technological gains, the productivity of intensive poultry systems remains constrained by a spectrum of infectious, non- infectious, and metabolic diseases and the impact of which is exacerbated when prevention programmes are inconsistently implemented (Rajendran et al., 2014).

Fatty Liver Haemorrhagic Syndrome (FLHS) is a non-infectious, metabolic disease primarily affecting laying hens, characterized by excessive fat accumulation in the liver, which can lead to hepatic rupture and fatal intra-abdominal haemorrhage. The etiology of FLHS is multifactorial, involving a combination of nutritional, environmental, hormonal, and genetic factors. Key risk factors include the consumption of high-energy diets combined with the restricted physical activity typical of cage housing systems, leading to obese state in birds. The syndrome is most commonly observed in hens that are in active lay, suggesting hormonal involvement, particularly estrogen. Environmental stressors, especially high temperature during summer months, have been shown to increase the incidence of mortality due to FLHS. Diagnosis is typically made during post-mortem examination, as there are few preceding clinical signs other than sudden death. Necropsy reveals characteristic gross lesions, including pale combs and wattles, abundant abdominal fat deposits, and a markedly enlarged, friable, greasy, and yellowish-brown coloured liver. The definitive finding is evidence of hepatic rupture with subcapsular and intraparenchymal hematomas and the presence of large blood clots in coelomic cavity. Interestingly, a retrospective study by Trott et al. (2014) found that 22% of FLHS cases had little to no excess hepatic lipid accumulation, suggesting "haemorrhagic liver syndrome" may be a more accurate term in some instances. Histologically, in liver a significant loss of the reticular fiber framework is a strong predictor of FLHS, though it does not always correlate with the degree of fatty change. Epidemiological studies consistently demonstrate a strong link between FLHS and housing systems. Shini et al. (2019) reported that FLHS was the cause of 74% of deaths in caged hens compared to only 0-5% deaths in barn or free- range systems.

2. Materials and Methods

2.1 Area of Investigation

This study focussed on mortality cases from different broiler and layer farms located in and around Hyderabad, Telangana as well as those brought to Department of Veterinary Pathology, College of Veterinary Science, Rajendranagar for post mortem examination.

2.2 Procedural Methodology

Laboratory investigations were performed on diseases identified throughout the study period (July to December 2025). Carcasses of chicken from a different range of age groups were obtained from 45 poultry farms in and around Hyderabad region and from cases submitted to the Department of Veterinary Pathology for post mortem analysis. Farm owners provided backgrounds including flock size, mortality figures, observed clinical signs, and the age of affected birds.

2.3 Sampling Procedure

Samples were collected from dead birds submitted by different poultry farms to the Department of Veterinary Pathology, C.V. Sc, Rajendranagar for disease diagnosis.

2.4 Gross Pathology

The carcasses were subjected to necropsy examination as per the protocol. Organs were thoroughly examined for presence of gross lesions in gastro intestinal tract and the lesions were recorded. (King et al. 2014)

2.5 Histopathology

The tissue samples of various gastro intestinal organs were collected in 10% neutral buffered formalin for histopathological examination. Tissue samples were trimmed to a thickness not exceeding 4 mm to facilitate adequate penetration of the fixative and were immersed in a fixative. The volume of fixative should be at least ten times to the tissue volume. Following fixation, the specimens were processed by routine histopathological techniques, including dehydration, clearing in xylene and paraffin embedding at 55-65 °C. Paraffin blocks were sectioned into thin ribbons, which were mounted on clean glass slides coated with Mayer's egg albumin, stained with haematoxylin and eosin, and examined under a light microscope (Luna, 1968; Bancroft and Gamble, 2008). All H&E-stained sections were systematically evaluated and the microscopic lesions were recorded.

3. Results

In FLHS, no mortalities were recorded in the 0–7 days, 8–14 days and 15–21 days age groups. In the 22–28

Table 3.1: Age wise mortality pattern of FLHS in chicken

Disease condition	Age group	No.of outbreaks	Total No. of birds in the flock	Mortality no. %	Mortality no. %	PMB	PMD
FLHS	0-7 days	0	0	-	-	-	
FLHS	8-14 days	0	300	-	-	-	
FLHS	15-21 days	0	400	-	-	-	
FLHS	22-28 days	0	500	20	4.0	2.63	30.77
FLHS	>29 days	2	800	45	5.62	5.93	69.23
Total		2	2000	65	9.62	8.56	100

PMD: Proportionate mortality within a disease
PMB: Proportionate mortality between the disease

days age group, 20 mortalities were observed among 500 birds, yielding a mortality percentage of 4.00 and a proportionate mortality distribution of 2.63%. Birds aged above 29 days showed higher involvement, with two outbreaks recorded in 800 birds, resulting in 45 mortalities, corresponding to a mortality percentage of 5.62 and a proportionate mortality distribution of 5.93% (Table 3.1 & Fig 3.1).

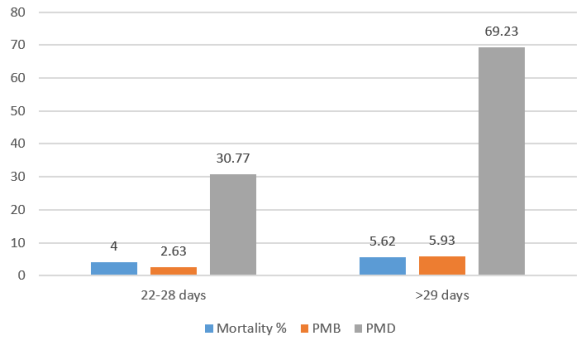


Fig 3.1 Age wise mortality pattern of FLHS in chicken

Fatty Liver Haemorrhagic Syndrome

Grossly, the liver was markedly enlarged, soft, friable and discoloured to tan to yellow or orange. On cut section, intraparenchymal haemorrhages and hematomas were commonly observed. Large blood clots were present within the coelomic cavity, adhered to either the left hepatic lobe (Fig 3.2, Fig 3.3).



Fig. 3.2 Photograph showing hepatic pallor, intracoelomic blood clots, and abundant intracoelomic fat in FLHS of chicken.

Histopathologically, liver sections affected by fatty liver



Fig. 3.3 Photograph showing multifocal to coalescing subcapsular hepatic haemorrhages and extensive blood clots in FLHS of chicken.

haemorrhagic syndrome (FLHS) exhibited considerable variation in severity. The extent of hepatocellular vacuolation ranged widely from occasional mild vacuolar changes to extensive and severe fatty infiltration (Fig 3.4). Additionally, FLHS-affected livers demonstrated subcapsular and/or intraparenchymal hematomas, focal unorganized sinusoidal haemorrhages (Fig 3.6) and in some cases, blood clots adherent to the hepatic capsule. The size and quantity of cytoplasmic vacuoles varied from multiple fine vacuoles to large and solitary vacuoles causing marked distension of hepatocytes (Fig. 3.5). Some hepatocytes revealed pyknosis and margination of chromatin (Fig 3.7).

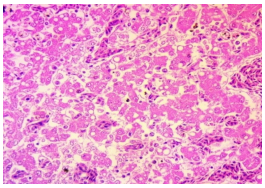


Fig. 3.4 Photomicrograph of liver showing macro vesicular hepatic steatosis in FLHS affected chicken. H&E x400.

4. Discussion

Fatty liver haemorrhagic syndrome accounted for a case prevalence of 8.56% in the present study. Trott et al. (2014) documented FLHS predominantly in older birds. In the present investigation, FLHS was observed

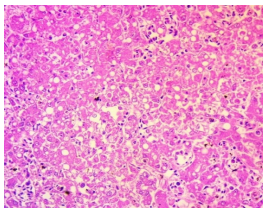


Fig. 3.5 Photomicrograph of liver showing small to medium sized lipid vacuoles inside the cytoplasm of hepatocytes in FLHS affected chicken. HE x400.

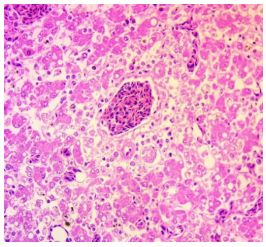


Fig. 3.6 Photomicrograph of liver showing sinusoidal haemorrhages in FLHS affected chicken. H&E x400.

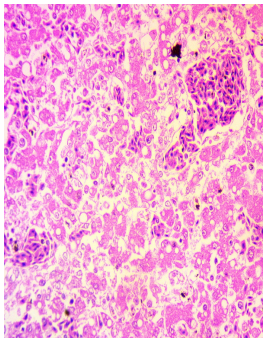


Fig. 3.7 Photomicrograph of liver showing pyknosis and margination of chromatin in hepatocytes in FLHS affected chicken. H&E x400.

mainly in birds above 29 days of age, it is consistent with earlier reports linking the condition to prolonged high-energy feeding and metabolic stress.

Fatty liver haemorrhagic syndrome (FLHS) was recorded in 2 outbreaks, with 65 cases, yielding a case prevalence of 8.56% and the highest disease specific mortality percentage (3.25%). The disease was recorded only in birds above 22 days of age, with higher mortality in birds above 29 days (5.62%) followed by birds at 22-28 days age (4.0%) Grossly, the liver was enlarged, soft, friable, and discoloured with intraparenchymal and subcapsular haemorrhages and blood clots in the coelomic cavity.

Histologically, the liver showed presence of vacuoles in cytoplasm, sinusoidal haemorrhages, hepatocellular degeneration with pyknosis, and margination of chromatin.

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