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Hepatic lipidosis in dairy cattle: A comprehensive overview of risk factors, diagnosis and management

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Abstract

In dairy cattle, Hepatic lipidosis (HL), more commonly known as fatty liver syndrome, is a metabolic disease marked by an abnormal buildup of fat in the liver. It frequently occurs as a result of a negative energy balance during the transitional phase, which lasts from late pregnancy until early lactation. Due to hormonal changes and inadequate food absorption, fatty liver develops mostly during the metabolically and physiologically taxing periparturient phase and is associated to an increased risk of infections, inflammations, and diseases. Nutritional risk factors include increased adipose tissue lipolysis or certain foods, hormones, or toxins that change the liver's metabolism. Adipocytes in cow's release substances that resemble hormones, including leptin and probably the pro-inflammatory cytokine TNF α . Risk factors for fatty liver in periparturient cows include limited space or exercise, poor sanitation, high heat, humidity, and poor ventilation. These stressors trigger catecholamine release, increasing NEFA mobilization, reducing feed intake, and raising infection risk during this vulnerable period. Recent diagnostic advancements include the application of biochemical markers (NEFA, BHBA, liver enzymes), ultrasonography, and molecular techniques, enabling earlier, non-invasive, and more accurate detection of HL in dairy cattle.

Keywords: Hepatic lipidosis, risk factors, advances in diagnosis, therapeutic management and prevention

1. Introduction

India's economy relies heavily on livestock, with dairy being the largest agri-commodity. Cattle make up about 36% of total livestock (20th census) and contribute nearly 5% to the national economy. Beyond income and nutrition, dairy cattle play vital social, cultural, and environmental roles in rural livelihoods. The transition period in dairy cows, spanning roughly three weeks before to three weeks after parturition, is marked by profound physiological, nutritional, metabolic, and immunological adaptations as the animal shifts from a non-lactating gestational state to the onset of high milk synthesis. The sharp rise in energy demands for lactation, particularly for glucose required in lactose synthesis, coincides with reduced feed intake near calving, resulting in negative energy balance (NEB) (Sordillo *et al.*, 2013) ^[11]. To compensate, cows mobilize adipose tissue reserves, releasing NEFA to meet metabolic needs. This energy deficit, combined with the physical and metabolic stresses of pregnancy, parturition, and lactation, compromises immune competence and predisposes cows to increased disease susceptibility during this critical phase (Tufarelli *et al.*, 2024) ^[13]. Hepatic lipidosis (HL) also known as fatty liver or fat cow syndrome, is a major metabolic disorder in animals. It develops when the hepatic uptake of lipids exceeds the oxidation and secretion of lipids by the liver. Excess lipids are stored as triacylglycerol (TAG) in the liver and are associated with decreased metabolic functions of the liver (Singh *et al.*, 2024) ^[10]. Liver samples can be evaluated chemically or histologically for liver TAG or total lipid to assess HL. Dairy cattle with HL can be classified as having mild fatty liver (liver TAG 1-5% wet weight; total lipid 5-20% of volume), moderate fatty liver (liver TAG 5-10% wet weight; total lipid 20-40% of volume), or severe fatty liver (liver TAG > 10% wet weight; total lipid > 40% of volume) (Kirovski and Sladojevic, 2017) ^[7].

1.1 Etiology

The etiology of HL in dairy cattle is multifactorial, with negative energy balance (NEB) being the primary predisposing factor. NEB typically arises during the periparturient period when the metabolic energy requirements for lactation exceed dietary energy intake. Nutritional inadequacies, including suboptimal feed quality, low dietary energy density, and imbalanced rations, promote excessive adipose tissue lipolysis, resulting in elevated plasma non-esterified fatty acids (NEFAs) and subsequent hepatic triglyceride accumulation. Concurrently, suboptimal management practices such as inadequate transition cow management, environmental stressors, and poor housing act as contributory risk factors. Moreover, endocrine alterations around parturition, particularly involving insulin resistance, elevated cortisol, and altered growth hormone dynamics, further disrupt lipid mobilization and hepatic lipid oxidation pathways, thereby potentiating hepatocellular fat infiltration and steatosis (Elshafey *et al.*, 2023) [3].

2. Risk Factors and Pathophysiology

2.1 Risk factors

Risk factors for fatty liver can be grouped into 2 categories: nutritional & managemental

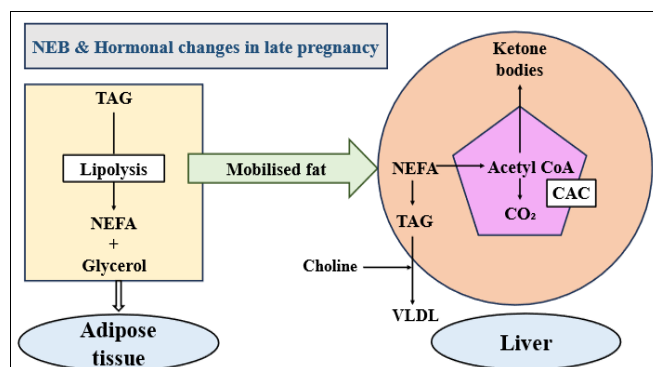
Nutritional risk factors for HL in dairy cattle primarily involve conditions that enhance adipose tissue lipolysis or impair hepatic metabolism. Adipokines such as leptin and potentially TNF α reduce feed intake and insulin sensitivity, while promoting hepatic lipogenesis, catabolism, and inflammation (Herdt, 2000). Sudden dietary changes, high-concentrate feeding, and postpartum high-protein diets predispose cows to ruminal acidosis, endotoxemia, and metabolic imbalance, all of which contribute to triglyceride accumulation in the liver (Murondoti *et al.*, 2002). Importantly, the disorder is more strongly linked to negative energy balance during the periparturient period, rather than amino acid or vitamin deficiencies, with feed changes and spoiled rations further exacerbating the risk (Gross *et al.*, 2023) [6].

Managemental risk factors for HL include poor-quality feedstuffs such as silage with elevated butyrate, which enhance BHBA production and reduce feed intake, as well as abrupt dietary changes or high-concentrate rations that predispose to ruminal acidosis, endotoxemia, and increased ketogenesis (Stober & Scholz, 1991). Environmental and husbandry-related stressors—such as overcrowding, lack of exercise, poor hygiene, high temperature, humidity, and inadequate ventilation—promote catecholamine release, adipose lipolysis, reduced intake, and greater infection risk during the periparturient period. Additional factors like extended gestation, dystocia, and concurrent disease states exacerbate negative energy balance, thereby intensifying hepatic triglyceride accumulation (Kushibiki *et al.*, 2003) [8].

2.2 Pathophysiology

Process HL in dairy cows is a common metabolic disorder associated with negative energy balance (NEB) during the periparturient period. Physiologically, hepatic lipid content rises a few weeks before calving, reaching approximately 20% in the first week postpartum, and gradually decreases to below 5% by week 26 (Bombik *et al.*, 2021) [2]. Failure of metabolic adaptation leads to hypoglycemia, low insulin, and enhanced mobilization of non-esterified fatty acids (NEFAs) from adipose tissue. When lipid influx to the liver exceeds its oxidative and excretory capacity, triacylglycerols (TAGs)

accumulate, resulting in hepatocellular steatosis and impaired liver function (Kirovski & Sladojevic, 2017) [7]. In dairy cattle, fatty acids are primarily synthesized in adipose tissue; excess NEFAs are either oxidized to ketone bodies or esterified into TAGs, predisposing to HL. Elevated NEFA levels are reliable indicators of NEB and excessive fat mobilization, with nearly half of calving cows at risk of metabolic disorders. Biochemical changes, including increased serum β -hydroxybutyrate (BHB), AST, GGT, and GLDH, are strongly associated with hepatic tissue alterations and ketosis, making them valuable markers for assessing metabolic status during early lactation.



3. Clinical Signs

HL in dairy cattle presents with a wide spectrum of clinical manifestations that reflect impaired hepatic function, altered energy metabolism, and secondary systemic effects. General signs of ill-health include anorexia, progressive loss of body condition, lethargy, and a marked reduction in milk yield, often accompanied by concurrent ketosis, characterized by the presence of ketone bodies detectable in breath, urine, or milk. Digestive disturbances are common, with rumen atony leading to reduced motility, predisposing affected cows to abomasal displacement (either left or right) due to decreased feed intake, while diarrhea or constipation may occur as a result of impaired digestive function. Liver-specific signs include jaundice (icterus), caused by impaired bilirubin metabolism, hepatomegaly that may be palpated or visualized by ultrasonography, and pain or sensitivity on palpation of the right flank region due to hepatic enlargement or inflammation. Neurological involvement may manifest as depression, dull mentation, and reduced responsiveness, with severe cases progressing to hepatic encephalopathy, demonstrated by abnormal behaviors such as head pressing, aimless wandering, or ataxia. Reproductive performance is often compromised, with delayed return to estrus, prolonged postpartum intervals, and increased incidence of metritis or retained placenta, attributable to immune suppression and chronic negative energy balance. In both clinical and subclinical instances, metabolic markers are important because high levels of β -hydroxybutyrate (BHB) and non-esterified fatty acids (NEFAs) can be observed before overt clinical indications appear, along with evidence of ketosis including an acetone-like smell in the breath. In extreme cases, cows may show signs of recumbency due to extreme weakness; if this is not addressed promptly, the cow may go into a coma or eventually succumb. These symptoms demonstrate the systemic effects of HL, which go beyond the liver to disrupt energy balance, neurological function, digestive physiology, and reproductive performance, ultimately decreasing dairy herd production and general health.

4. Advances in Diagnosis

Parameter	Correlation	Coefficient P value	Significance
TP (g/dl)	$r = -0.3931$	$P = 0.0076$	**
Albumin (g/dl)	$r = -0.2904$	$P = 0.0530$	Ns
TB (mg/dl)	$r = 0.04178$	$P = 0.7852$	Ns
AST (U/L)	$r = 0.8556$	$P < 0.0001$	****
GGT (U/L)	$r = 0.7986$	$P < 0.0001$	****
TG (mg/dL)	$r = -0.5973$	$P < 0.0001$	****
Glucose (mg/dL)	$r = -0.6074$	$P < 0.0001$	****
Cholesterol (mg/dL)	$r = -0.5284$	$P = 0.0002$	***
ALP (U/L)	$r = 0.5087$	$P = 0.0004$	***

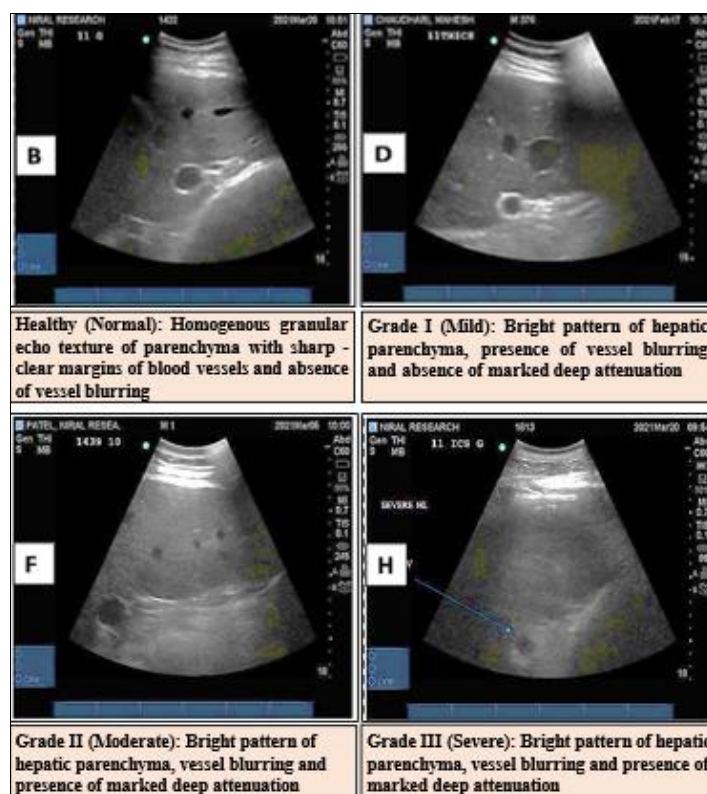
4.1 Analysis of serum metabolites

In HL, biochemical profiling revealed significantly elevated levels of β -hydroxybutyrate (BHBA) and non-esterified fatty acids (NEFA) in moderate and severe cases compared to healthy animals, with both markers showing strong positive correlations with ultrasonographic severity grading. Total protein (TP), albumin, and total bilirubin (TB) did not differ significantly between HL and healthy groups; however, TP demonstrated a negative correlation with disease grading, while albumin and TB correlations were not significant. Hepatic enzymes AST and GGT were markedly increased in moderate and severe HL, with significant intergroup differences, and both showed strong positive correlations with ultrasonographic scores. Alkaline phosphatase (ALP) was

significantly higher in severe HL and positively correlated with grading. Conversely, triglycerides (TG) and glucose levels were significantly reduced in moderate and severe HL compared to healthy controls, indicating metabolic impairment associated with disease progression (Patel *et al.*, 2022) [9].

4.2 Ultrasonographic examination

The Ultrasonographic evaluation of the liver was performed in standing animals with a 2–5 MHz convex transducer, applied over the right 9th–12th intercostal spaces after proper clipping and preparation. Scanning was conducted from caudal to cranial and dorsal to ventral directions, with image interpretation based on established echogenic features to classify HL (HL) into normal, mild, moderate, and severe grades. Normal livers exhibited a homogeneous granular echotexture with sharp vascular margins and no vessel blurring. Mild HL (Grade I) was characterized by increased parenchymal echogenicity with vessel blurring but without deep attenuation, while moderate HL (Grade II) showed similar echogenicity with vessel blurring and distinct deep attenuation. Severe HL (Grade III) demonstrated marked hyperechogenicity, prominent deep attenuation, poor vascular visibility, and enhanced near-field brightness, with additional assessment of portal vein diameter and depth aiding disease grading (Fiore *et al.*, 2018) [4] (Patel *et al.*, 2022) [9].



4.3 Liver Pathology

HL in dairy cattle results from excessive accumulation of triacylglycerols (TAG) in the liver, causing enlargement, swelling, pale to yellow discoloration, and infiltration of other organs, with severe cases showing necrosis, myocarditis, and involution of endocrine and lymphatic tissues. Histopathological confirmation through liver biopsy, the gold standard, involves guided needle aspiration to obtain cytology samples for diagnosis. Clinically, biochemical markers

provide critical insights: elevated AST and GGT indicate hepatocellular and biliary damage, increased bilirubin reflects impaired liver function, and high NEFA and BHBA levels denote excessive fat mobilization and ketosis. Additionally, hypoglycemia, altered triglycerides, and reduced albumin indicate impaired gluconeogenesis, disrupted lipid metabolism, and compromised protein synthesis, respectively, making these parameters valuable for assessing severity and metabolic impact of HL in dairy cattle.

4.4 Body Condition Score

In dairy cattle, subcutaneous fat deposition in the rump or hump region provides a subjective estimate of body fat, but back fat thickness (BFT) measurement by B-mode ultrasonography offers greater accuracy. In this study, BFT was assessed using a portable ultrasound machine with a 5–8 MHz linear transducer, after shaving and applying coupling gel to the sacral site (9–11 cm cranial to the tuber ischia). The probe was placed vertically along the line between the tuber ischia and tuber coxae, and images were recorded at a depth of 4.2–4.8 cm using the inbuilt caliper function (Batista *et al.*, 2022)^[1].

5. Therapeutic Management & Prevention

Treatment of HL depends on the extent of lipid infiltration and underlying causes, with general management focused on improving feed intake using fresh, high-quality forage (Stöber and Scholz, 1991)^[12]. To correct negative energy balance, glucose with calcium and magnesium salts may be administered. Glucose precursors such as propylene glycol @ 250–300 g/cow twice daily P.O. or glycerol up to 500 g/cow twice daily P.O. increase blood glucose and reduce fat mobilization, while intravenous infusion of 500 ml of 50% dextrose provides an immediate glucose source. Lipotropic agents act as hepatoprotective compounds and support hepatocyte regeneration. Rumen-protected niacin improves lipid metabolism, energy balance, and oxidative stress management. Hormonal therapies with insulin (reducing NEFA mobilization) or glucagon (supporting reversal of hepatic lipidosis) may be used under close monitoring to prevent adverse effects. Antioxidant supplementation helps limit oxidative stress, protect hepatocyte membranes, and enhance immune function. Rumen transfaunation (10–15 L ruminal fluid) from a healthy donor cow restores microflora, improves feed intake and digestion, reduces toxins, and supports hepatic recovery (Fiore *et al.*, 2016)^[5].

Prevention of hepatic lipidosis in dairy cattle requires integrated nutritional, management, and monitoring strategies to maintain energy balance and liver health, particularly during the transition period. Nutritional management focuses on providing energy-dense feeds such as propylene glycol and glycerol, avoiding over-conditioning (ideal BCS: 3.0–3.5), promoting high dry matter intake (DMI), and ensuring balanced protein with rumen-protected sources. Transition care includes introducing pre-calving diets 2–3 weeks before parturition, minimizing stress, ensuring proper housing, ventilation, and space, and supporting DMI with fresh, palatable feed. Supplementation with rumen-protected choline, methionine, niacin, vitamin E, and selenium supports lipid metabolism and antioxidant defense. Regular health monitoring through BCS scoring, metabolic profiling, feed intake tracking, ketone testing, and veterinary checkups enables early detection of at-risk cows and timely intervention. Sound management practices—including use of TMR to reduce feed sorting, heat abatement strategies, stress reduction, prompt calving assistance, and veterinarian-guided feeding protocols—further reduce fatty liver risk and enhance herd productivity and longevity.

6. Conclusion

HL in dairy cattle is difficult to diagnose which leads huge economic losses in modern cattle husbandry. Poor nutrition and management are potential risk factors for fatty liver in lactating dairy cows. Clinical symptoms usually occur with severe liver damage when treatment is no longer effective.

Regular examination of animals is needed to reveal the scale of the disease. Ultrasonography examinations along with analysis of serum metabolites is very much helpful for identification of disease. Histopathology is essential diagnostic modality for diagnosis of grades of hepatic lipidosis. Prevention involving an appropriate nutrition during every stage of dairy production is essential.

Conflict of Interest

Not available

Financial Support

Not available

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