THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
Popular Article
Oct, 2026 Vol.6(10), 3298-3311

Bovine Fatty Liver Disorder (Hepatic Lipidosis): Pathophysiological Dynamics, Clinicopathological Alterations, Diagnostic Advances, and Therapeutic Management

Dr. Fanase Kirti Dadaram*[1] Dr. Pranav Sunil Jadhav[2], Dr. Rajwardhan Chandrakant Nimbalkar[3]

[1]MVSc, Veterinary Pathology, College of Veterinary Science and Animal Husbandry Sardarkrushinagar

[3]MVSc, Poultry Science, ICAR- Central Avian Research Institute, Izatnagar, Bareilly,

doi.org/10.5281/ScienceWorld.23472443

Abstract

Fatty liver disorder, clinically recognized as bovine hepatic lipidosis, is a critical metabolic and systemic disease affecting high-yielding dairy cattle and transition cows during the periparturient period (Melendez & Pinedo, 2024). The disorder is fundamentally driven by a severe negative energy balance (NEB) at the onset of lactation, causing massive mobilization of non-esterified fatty acids (NEFAs) from adipose tissue depots into the bloodstream (Melendez & Pinedo, 2024). Because ruminants exhibit an inherently sluggish hepatic capacity for synthesizing and exporting very-low-density lipoproteins (VLDLs), excessive hepatic uptake of NEFAs results in massive intracellular re-esterification and pathological accumulation of triglycerides (TAGs) within hepatocytes (Melendez & Pinedo, 2024). The disease manifests in clinical and subclinical forms, profoundly impairing gluconeogenesis, ureagenesis, and hepatic immune surveillance, while precipitating secondary production disorders such as clinical ketosis, displaced abomasum, mastitis, metritis, and depressed reproductive efficiency (Melendez & Pinedo, 2024). Clinicopathologically, affected cattle exhibit hypoglycemia, profound increases in circulating NEFAs and β-hydroxybutyrate (BHBA), elevated serum liver transaminases (AST, GGT, SDH), and hyperbilirubinemia. Definitive ante-mortem diagnosis requires histopathological examination or triglyceride quantification of percutaneous liver biopsy samples, though indirect blood biochemical profiling and ultrasonographic evaluation of hepatic echogenicity are widely employed in veterinary practice. Therapeutic strategies center on correcting the underlying energy deficit through intravenous dextrose infusions, oral propylene glycol or glycerol, lipotropic factors (choline chloride, methionine, vitamin B_{12}), and rumen transfaunation. Given that no commercial prophylactic vaccine exists due to the non-infectious, multifactorial metabolic etiology of the disorder, sustainable herd management relies on rigorous body condition scoring during the dry period, optimization of transition dietary energy density, and minimization of periparturient stressors.

Keywords: Bovine hepatic lipidosis, Negative energy balance, Non-esterified fatty acids (NEFAs), Transition dairy cows, Triglyceride accumulation, Very-low-density lipoproteins (VLDLs).

1. Introduction

Bovine fatty liver disorder, or hepatic lipidosis, is an economically important metabolic disorder in intensive dairy production systems worldwide, including high-producing dairy herds in India (Melendez and Pinedo, 2024). The transition period, defined as the three weeks before and three weeks after parturition, represents one of the most physiologically demanding stages of a dairy cow's production cycle.

During this period, the cow undergoes substantial endocrine and metabolic changes to support colostrum synthesis and the onset of lactation. The increasing demand for nutrients frequently exceeds voluntary dry matter intake (DMI), resulting in a state of negative energy balance (NEB).

To compensate for the energy deficit, homeorhetic mechanisms stimulate lipolysis in subcutaneous and visceral adipose tissues, releasing large quantities of non-esterified fatty acids (NEFAs) into the circulation. These fatty acids are transported to the liver, where they may undergo oxidation, conversion into ketone bodies, or re-esterification into triglycerides (TAGs).

Unlike some other species, ruminants have a relatively limited capacity to export triglycerides from the liver as very-low-density lipoproteins (VLDLs). When the influx of NEFAs exceeds the liver's capacity for oxidation and export, triglycerides accumulate within hepatocytes. This accumulation can alter hepatic architecture, impair metabolic function, and increase the risk of associated periparturient disorders (Melendez and Pinedo, 2024).

2. Causative Conditions and Types

Fatty liver disorder is a multifactorial metabolic condition rather than an infectious disease. It develops primarily when excessive fat mobilization occurs in response to negative energy balance, particularly during late gestation and early lactation. The severity depends on the degree and duration of energy deficit, the cow's body condition, liver metabolic capacity, and the presence of concurrent diseases.

2.1 Primary Metabolic Etiology

Primary hepatic lipidosis is commonly associated with over-conditioned cows entering the transition period. Cows with excessive body condition scores (BCS), approximately 3.5–4.0 on a 5-point scale as described in the source literature, may experience a marked reduction in feed intake around calving. This promotes adipose tissue mobilization and increases circulating NEFA concentrations.

When the delivery of NEFAs to the liver exceeds its metabolic capacity, triglycerides accumulate in hepatocytes. Consequently, excessive body condition at calving, inadequate dry-

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

cow feeding strategies, and insufficient monitoring of transition-cow nutrition may increase the risk of hepatic lipidosis (Melendez and Pinedo, 2024).

2.2 Secondary Complicating Etiologies

Secondary hepatic lipidosis may develop when another periparturient disorder causes reduced feed intake, anorexia, or prolonged inappetence. Important associated conditions include the following.

Periparturient hypocalcemia (milk fever). Hypocalcemia can impair gastrointestinal smooth-muscle function, contributing to reduced rumen motility and feed intake. Changes in calcium-dependent physiological processes may also affect insulin secretion and metabolic regulation, potentially aggravating fat mobilization.

Uterine infections and mastitis. Postpartum metritis and acute mastitis can trigger systemic inflammation through mediators such as tumour necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6). Inflammation may suppress appetite and alter hepatic metabolism, increasing the risk of negative energy balance and hepatic lipid accumulation.

Displaced abomasum. Left or right displacement of the abomasum may cause reduced feed intake, impaired gastrointestinal function, and systemic metabolic disturbances. Prolonged anorexia can accelerate adipose tissue mobilization and aggravate hepatic lipidosis.

2.3 Anatomical and Pathological Classification

Bovine hepatic lipidosis can be classified according to the amount of triglyceride accumulated in the liver and the associated histopathological changes. The following grading system is presented as described in the source article.

Table 1. Pathological classification and grading of bovine hepatic lipidosis based on liver triglyceride content

Pathological grade	Liver triglyceride concentration (% wet weight)	Histopathological manifestations	Clinical and prognostic significance
Normal/physiological	Less than 1.0% (less than 10 mg/g)	Normal hepatocyte arrangement, relatively uniform glycogen distribution, and minimal neutral lipid droplets.	Consistent with normal liver architecture and metabolic function.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

Mild fatty liver	1.0–5.0% (10–50 mg/g)	Small, scattered microvesicular lipid droplets, particularly in periportal and midzonal hepatocytes.	May be subclinical and potentially reversible when energy balance improves.
Moderate fatty liver	5.0–10.0% (50–100 mg/g)	More extensive microvesicular and macrovesicular steatosis, with lipid vacuoles potentially displacing hepatocyte nuclei towards the periphery.	May be associated with reduced milk production, ketosis, and impaired immune function.
Severe fatty liver	More than 10.0% (more than 100 mg/g; potentially reaching 20–30%)	Extensive macrovesicular lipid accumulation and marked hepatocellular changes; necrosis may occur in severe cases.	Associated with a guarded prognosis and an increased risk of secondary metabolic and systemic complications.

The triglyceride thresholds in this table should be checked against the diagnostic classification used by the original source before publication. Histopathological appearance and clinical severity do not always correspond exactly to a single biochemical threshold.

3. Predisposing Factors: Age, Physiological Status, Breed, Geography, and Environment

The susceptibility and epidemiological distribution of bovine fatty liver disorder are influenced by host characteristics, physiological demands, and farm management practices.

3.1 Age Predisposition

Hepatic lipidosis is frequently associated with mature, high-producing dairy cows, particularly multiparous animals in their third to fifth lactation or later. Older cows may have greater milk production demands and substantial adipose reserves available for mobilization during the transition period.

Differences in production level, body condition, feed intake, and metabolic adaptation can influence individual susceptibility. However, age and parity should be considered alongside these other factors rather than treated as independent causes.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

3.2 Gender and Physiological Status

The disorder primarily affects reproductively mature female cattle during late gestation and early lactation. The risk is particularly important around calving, when nutrient requirements increase rapidly and voluntary feed intake may be insufficient to meet metabolic demands.

The source literature identifies the first 10–14 days postpartum as an important period for disease occurrence (Melendez and Pinedo, 2024).

3.3 Breed Predisposition

High-producing dairy cattle, particularly Holstein-Friesian animals and high-grade Holstein crossbreds, are commonly discussed in relation to hepatic lipidosis because of their substantial lactational energy requirements.

Genetic selection for high milk production can contribute to a pronounced nutrient demand during early lactation. When feed intake fails to meet these requirements, adipose tissue mobilization increases, potentially overwhelming hepatic metabolic pathways.

Indigenous *Bos indicus* cattle and dual-purpose breeds may have different production levels, adaptation characteristics, and metabolic responses. Nevertheless, susceptibility varies with individual body condition, diet, management, and production status; lower risk should not be assumed solely on the basis of breed.

3.4 Geographical and Farm-System Predisposition

Hepatic lipidosis occurs in dairy production systems worldwide and is of particular concern in intensive commercial herds, dry-lot systems, and peri-urban dairies. Nutritional management practices that fail to account for the changing requirements of cows during the far-off dry period, close-up period, and early lactation can contribute to excessive body condition or severe negative energy balance.

Uniform total mixed rations (TMRs) may not meet the needs of every animal if differences in physiological status, intake, and body condition are not adequately considered. The source article reports herd-level subclinical fatty liver prevalence exceeding 40–50% postpartum in some poorly managed systems; this figure should be verified against the original epidemiological evidence before being generalized.

3.5 Environmental Predisposition

Thermal stress. High ambient temperatures and humidity can reduce feed intake, particularly in close-up dry cows and freshly calved animals. A temperature-humidity index (THI) above 72 is often used as an indicator of heat-stress risk in cattle, although the actual threshold varies with production level, acclimatization, and environmental conditions. Reduced DMI during heat stress can aggravate negative energy balance.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

Overcrowding and management stress. Competition at the feed bunk, inadequate lying space, frequent regrouping, and poor housing management may interfere with feeding and resting behaviour. These conditions can compromise transition-cow adaptation and contribute indirectly to metabolic stress.

4. Effects on Animal Health and Production

Hepatic lipidosis can affect several physiological systems and has important implications for animal welfare, herd longevity, and farm profitability.

4.1 Reduced Milk Yield and Peak Lactation

The liver plays a central role in glucose metabolism, including gluconeogenesis from substrates such as propionate and certain amino acids. Glucose is essential for lactose synthesis in the mammary gland. Hepatic dysfunction and concurrent negative energy balance can therefore compromise the cow's ability to meet the demands of lactation.

Affected animals may show reduced milk yield and fail to achieve their expected peak production. The magnitude and persistence of this reduction depend on disease severity, concurrent conditions, and the effectiveness of management (Melendez and Pinedo, 2024).

4.2 Immune Dysfunction and Secondary Diseases

Excessive hepatic lipid accumulation can interfere with normal liver metabolism and immune-related functions. Changes in hepatic protein synthesis, lipid metabolism, and systemic inflammatory responses may contribute to impaired immune competence.

Cows with severe metabolic disturbances may be at increased risk of associated periparturient disorders, including retained placenta, metritis, mastitis, and other inflammatory conditions. These associations are multifactorial and should not be interpreted as evidence that fatty liver alone directly causes every secondary disease.

4.3 Reproductive Problems and Early Culling

Severe negative energy balance and metabolic dysfunction can delay the resumption of postpartum ovarian activity and affect reproductive hormone dynamics. Potential consequences include delayed conception, increased services per conception, prolonged days open, and repeat breeding.

When reduced productivity and recurrent disease persist, the risk of involuntary culling may increase. Early identification and appropriate transition-cow management can help reduce these losses (Melendez and Pinedo, 2024).

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

5. Clinical Signs and Symptoms

Clinical manifestations vary with the extent of hepatic lipid accumulation and the presence of concurrent disorders, particularly ketosis and hypocalcemia. Many affected cows may have no distinctive clinical signs, making herd-level monitoring important.

5.1 Subclinical Form

Subclinical hepatic lipidosis may occur without obvious disease-specific signs. Possible findings include reduced appetite, dullness, lower-than-expected milk production, and poor recovery of body condition. These signs are nonspecific and may also occur in other transition-cow disorders.

5.2 Clinical and Severe Form

Severe hepatic lipidosis may be associated with marked metabolic dysfunction, particularly in over-conditioned cows during early lactation.

Profound anorexia and lethargy. Affected cows may refuse concentrate feed and substantially reduce roughage intake. Generalized weakness, an unsteady gait, prolonged recumbency, and reduced responsiveness may occur. Similar signs can occur in hypocalcemia and other serious postpartum disorders, requiring differential diagnosis.

Neurological abnormalities. In advanced hepatic dysfunction, impaired ammonia detoxification and other metabolic disturbances may contribute to neurological signs. These may include aimless wandering, head pressing, muscle fasciculations, abnormal behaviour, teeth grinding, recumbency, and, in severe cases, coma. Such signs constitute a veterinary emergency and should not be attributed to hepatic encephalopathy without assessment of other possible causes.

Acetone odour. A sweet or fruity odour on the breath or milk may occur when ketosis accompanies hepatic lipidosis. This finding is not specific and should be evaluated alongside ketone measurements and clinical findings.

Mucosal icterus. Yellow discoloration of the sclera and other visible mucous membranes may occur in animals with hepatobiliary dysfunction. Jaundice is not a universal feature of fatty liver and warrants investigation for other hepatic, haemolytic, or biliary disorders.

6. Hematological and Serum Biochemical Profiles

Laboratory testing can help identify metabolic stress, evaluate hepatic dysfunction, and detect concurrent disorders. No single blood parameter reliably quantifies hepatic fat accumulation in every animal; results should be interpreted with the clinical history, stage of lactation, and other diagnostic findings.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

6.1 Hematological Profile

Routine hematological findings in cows with hepatic lipidosis are variable. Anemia may occur in animals with concurrent chronic disease, inflammation, nutritional deficiencies, or other systemic conditions, but it is not a specific diagnostic feature of fatty liver.

Leukogram changes may reflect concurrent inflammatory disease, such as metritis or mastitis, or a stress response. Depending on the clinical condition, findings may include mature neutrophilia, lymphopenia, eosinopenia, or a left shift in severe inflammatory disease.

6.2 Serum Biochemical Profile

Non-esterified fatty acids (NEFAs). Increased circulating NEFA concentrations indicate enhanced adipose tissue mobilization. Elevated concentrations around calving are associated with negative energy balance and increased risk of hepatic lipidosis. Interpretation depends on the timing of sampling and the laboratory or herd-monitoring thresholds used.

β-Hydroxybutyrate (BHBA). BHBA is a major circulating ketone body in cattle. Increased concentrations indicate enhanced ketone production and may be associated with ketosis. Although useful for monitoring energy metabolism, BHBA does not directly measure liver triglyceride content.

Blood glucose. Glucose concentrations may be reduced in some affected cows, particularly when energy intake is inadequate or concurrent metabolic disease is present. However, hypoglycemia is not a universal finding, and blood glucose alone cannot establish the diagnosis.

Liver-associated enzymes. Aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), sorbitol dehydrogenase (SDH), and glutamate dehydrogenase (GLDH) may help assess hepatic or hepatobiliary abnormalities. AST is not liver-specific and may also increase with muscle injury. Enzyme activities must therefore be interpreted in context.

Cholesterol and blood urea nitrogen. Changes in total cholesterol and blood urea nitrogen (BUN) may occur with altered hepatic function, nutritional intake, and systemic disease. Reduced concentrations may be observed in some cases, but neither parameter is a specific indicator of hepatic lipidosis.

Table 2. Summary of biochemical alterations associated with bovine fatty liver disorder

Biochemical parameter	Reference or screening value reported in the source	Possible alteration in affected cows	Pathophysiological significance

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

Non-esterified fatty acids (NEFAs)	Less than 0.3 mEq/L prepartum and less than 0.5 mEq/L postpartum	Increased concentrations; the source reports values above 0.7–1.0 mEq/L in high-risk situations.	Indicates increased adipose tissue mobilization associated with negative energy balance.
β-Hydroxybutyrate (BHBA)	Less than 0.8 mmol/L	Increased concentrations; the source reports values above 1.2–3.0 mmol/L.	Indicates increased ketone production and may support the diagnosis of ketosis.
Blood glucose	45–75 mg/dL	May decrease; the source identifies values below 40 mg/dL as hypoglycemic.	May reflect inadequate energy supply or altered glucose metabolism.
Serum aspartate aminotransferase (AST)	60–125 U/L	The source reports elevations above 150–300 U/L.	May indicate tissue injury; muscle disease must also be considered.
Serum glutamate dehydrogenase (GLDH)	Less than 15 U/L in the source	May increase with hepatocellular injury.	Supports evaluation of hepatocellular damage when interpreted using the laboratory's reference interval.
Total serum cholesterol	80–150 mg/dL in the source	May be decreased in some affected cows.	May reflect altered hepatic lipid metabolism and lipoprotein synthesis.
Blood urea nitrogen (BUN)	10–25 mg/dL in the source	May be decreased; the source reports values below 8 mg/dL.	May reflect altered hepatic urea synthesis, dietary protein intake, or other systemic factors.

Note: All numerical reference intervals and proposed disease thresholds in Table 2 should be verified against the laboratory method, sampling time, stage of lactation, and original reference before publication or clinical use.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

7. Diagnosis and Diagnostic Tests

Antemortem diagnosis relies on the integration of herd history, clinical examination, blood biochemistry, and, when necessary, direct assessment of hepatic tissue. The diagnostic approach should also investigate concurrent diseases that may contribute to anorexia and excessive fat mobilization.

7.1 Direct Diagnostic Tests

Percutaneous liver biopsy. Liver biopsy allows direct evaluation of hepatic tissue and can be used for histological assessment or biochemical quantification of triglyceride content. The source article describes sampling from the right 10th to 11th intercostal space using a biopsy needle under local anaesthesia. The precise sampling site and technique should be selected by a veterinarian familiar with the procedure.

Histological assessment may identify intracellular lipid accumulation using appropriate tissue preparation and staining methods. Direct chemical analysis can quantify hepatic triglycerides. The source identifies a wet-liver triglyceride concentration exceeding 10% (100 mg/g) as an indicator of severe fatty liver; this threshold should be confirmed against the classification system being used.

Fine-needle aspiration cytology (FNAC). Fine-needle aspiration may provide a less invasive means of obtaining hepatic cellular material. Lipid-specific stains, including Oil Red O or Sudan-based methods, may be used with suitable sample preparation to demonstrate intracellular lipid accumulation. The sensitivity and diagnostic value depend on sample quality and laboratory technique.

7.2 Indirect Diagnostic Tests and Biomarkers

Herd metabolic profiling. Testing selected prepartum and postpartum cows for NEFA, BHBA, glucose, and relevant liver-associated enzymes can help identify herd-level metabolic problems. The number of animals sampled and the interpretation of results should reflect herd size, sampling time, disease prevalence, and the intended screening objective.

Milk and urine ketone testing. Cow-side ketone tests can support the detection of ketosis. These tests measure ketone bodies or related analytes and should not be interpreted as direct confirmation of hepatic lipidosis.

Ultrasonography. Transabdominal or intercostal ultrasonography may be used to assess liver appearance, margins, and echogenicity. Increased hepatic echogenicity, sometimes described as a "bright liver," may be compatible with fat accumulation. However, ultrasonographic findings are operator-dependent and cannot reliably establish an exact hepatic triglyceride percentage.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

Table 3. Comparison of diagnostic modalities for bovine fatty liver disorder

Diagnostic modality	Target parameter	Diagnostic value	Main advantage	Main limitation
Percutaneous liver biopsy	Histological assessment and direct triglyceride quantification	Direct assessment of hepatic tissue; often considered the reference approach for quantifying liver fat.	Provides tissue for direct evaluation of lipid accumulation.	Invasive; requires technical expertise and appropriate laboratory facilities.
Serum NEFA and BHBA assays	Circulating NEFAs and ketone bodies	Useful for evaluating energy balance and identifying cows at metabolic risk.	Supports individual and herd-level screening.	Reflects systemic metabolism rather than directly measuring liver fat.
Serum liver-associated enzymes	AST, GLDH, GGT, and other relevant analytes	Supports evaluation of hepatic or hepatobiliary injury.	Readily available through routine veterinary laboratories.	Enzymes differ in specificity and cannot independently confirm hepatic lipidosis.
Liver ultrasonography	Hepatic parenchymal appearance and echogenicity	Provides supportive imaging evidence.	Non-invasive and can be performed in clinical settings.	Operator-dependent and unable to quantify liver triglycerides precisely.
Milk or urine ketone tests	Ketone-related analytes	Supports screening for ketosis.	Rapid and convenient for on-farm use.	Does not directly diagnose or grade hepatic lipidosis.

The source article reports 100% diagnostic accuracy for liver biopsy and sensitivity of 85–92% for serum NEFA and BHBA testing. These numerical estimates should not be retained as

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

universal performance characteristics without confirmation from the original study and the relevant diagnostic definitions.

8. Treatment and Management Protocols

The management of bovine fatty liver disorder aims to correct the underlying energy deficit, reduce excessive adipose tissue mobilization, support hepatic metabolism, and treat concurrent disorders. The approach depends on clinical severity, hydration, feed intake, lactation stage, and the presence of ketosis, hypocalcemia, infection, or displaced abomasum.

8.1 Intravenous Glucose Therapy

Intravenous glucose may be used by a veterinarian in selected cows with clinically significant metabolic disturbances. The source article describes the administration of 500 mL of 50% dextrose solution intravenously, followed by further glucose support where indicated.

Glucose administration can temporarily increase circulating glucose concentrations, but its effects on insulin secretion, lipolysis, and subsequent metabolic responses vary with the animal's condition. The concentration, volume, rate of administration, and need for additional treatment must be determined by a veterinarian. Glucose therapy alone does not remove accumulated hepatic triglycerides or replace the need to address the underlying cause.

8.2 Oral Glucogenic Precursors

Propylene glycol is used in veterinary practice as a glucogenic precursor in selected transition-cow metabolic conditions, particularly ketosis. The source article describes daily oral administration of 250–500 mL for 3–5 consecutive days.

This range should be checked against the intended indication, formulation, animal condition, and current veterinary guidance before use. Glycerol is also discussed in the source as a possible glucogenic substrate. Neither product should be regarded as a substitute for diagnosis, adequate nutrition, or treatment of concurrent disease.

8.3 Lipotropic Factors and Methyl Donors

Choline chloride. Choline contributes to phosphatidylcholine synthesis, which is important for hepatic lipoprotein assembly and triglyceride export. Rumen-protected choline is used in some transition-cow feeding programmes to support metabolic adaptation. The source article reports a dietary range of 10–25 g/day; the appropriate formulation, dose basis, and duration should be verified before publication.

Vitamin B12 and methionine. Vitamin B12 and methionine participate in metabolic pathways associated with one-carbon metabolism and methyl-group transfer. Their roles in hepatic metabolism provide a rationale for considering nutritional adequacy during the transition period.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

Supplementation should be based on the animal's dietary status and evidence supporting the intended intervention.

8.4 Rumen Transfaunation

Rumen transfaunation involves the transfer of ruminal fluid from a suitable donor to a recipient animal. It may be considered in selected cases of ruminal dysfunction under veterinary supervision. The source article describes the administration of 2–4 litres of ruminal fluid, but the appropriate volume, donor selection, collection method, and clinical indication require case-specific assessment.

Transfaunation is not a direct treatment for hepatic triglyceride accumulation. Its potential value depends on whether ruminal dysfunction contributes to reduced intake or digestive disturbance.

8.5 Corticosteroid Use

Glucocorticoids, including dexamethasone, can influence gluconeogenesis and glucose metabolism. Their use in cows with fatty liver is controversial because their metabolic and immunological effects may be undesirable in certain clinical circumstances.

Corticosteroids should not be considered routine therapy for hepatic lipidosis. Any use should be based on a specific clinical indication, a veterinarian's assessment of potential benefits and risks, and applicable food-animal regulations.

8.6 Management of Concurrent Disorders

Effective treatment also requires identification and management of concurrent conditions that reduce feed intake or increase metabolic stress. These may include hypocalcemia, ketosis, metritis, mastitis, and displaced abomasum.

Supportive care should be individualized according to hydration status, appetite, rumen function, pain, and other clinical findings. Nutritional management should provide appropriate energy and nutrient intake without creating additional digestive or metabolic problems.

9. Vaccines Available and Their Effectiveness

There are no commercial vaccines specifically available for the prevention or treatment of bovine fatty liver disorder. Hepatic lipidosis is a non-infectious metabolic condition associated with excessive fat mobilization, negative energy balance, and limited hepatic capacity to process and export triglycerides. Consequently, conventional vaccination is not an established preventive or therapeutic strategy for the condition.

Prevention depends primarily on appropriate transition-cow nutrition, body condition management, environmental control, monitoring of metabolic indicators, and early identification of concurrent diseases.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 15/10/2026

10. Conclusion and Future Perspectives

Bovine fatty liver disorder remains an important metabolic challenge in modern dairy production, contributing to reduced milk yield, increased susceptibility to periparturient disease, reproductive inefficiency, and premature culling. Its development is closely associated with negative energy balance during the transition period and the limited capacity of the ruminant liver to export triglycerides as VLDLs (Melendez and Pinedo, 2024).

Effective control requires a proactive herd-health approach rather than reliance on treatment after clinical signs develop. Priority should be given to maintaining appropriate body condition before calving, formulating nutritionally balanced transition-cow diets, ensuring adequate feed access, reducing heat stress, and promptly identifying concurrent diseases.

Future management strategies may benefit from integrating routine body condition scoring, metabolic profiling of NEFA and BHBA, appropriate use of evidence-based nutritional interventions, and systematic evaluation of transition-cow outcomes. These measures can help improve metabolic adaptation, protect animal welfare, and support the long-term sustainability of dairy enterprises.

References

Melendez, P. and Pinedo, P. (2024). Update on fatty liver in dairy cattle with major emphasis on epidemiological patterns, pathophysiology in relationship to abdominal adiposity, and early diagnosis. *Dairy*, 5(4), 672–687. https://doi.org/10.3390/dairy5040050