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PERSPECTIVES ON NON-ALCOHOLIC FATTY LIVER DISEASE IN THE MODERN ERA

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ABSTRACT

This article provides a literature review on non-alcoholic fatty liver disease (NAFLD), a polyetiological condition. Excessive consumption of foods rich in animal fats and easily digestible carbohydrates leads to an influx of free fatty acids (FFAs) from the gastrointestinal tract into the bloodstream, ultimately accumulating in the liver, resulting in steatosis.

Inflammatory mediators actively secreted by adipose tissue directly damage hepatocyte membranes, leading to fibrotic tissue accumulation in the liver. The key feature of NAFLD is its asymptomatic course, with diagnosis often occurring incidentally through laboratory and imaging studies in patients with metabolic syndrome.

As a widespread pathology, NAFLD requires thorough investigation into its pathogenesis and the development of optimal non-invasive diagnostic methods to detect and assess its advanced forms (steatohepatitis, fibrosis, cirrhosis).

KEYWORDS: *Non-Alcoholic Fatty Liver Disease, Steatosis, Steatohepatitis, Liver Fibrosis, Cirrhosis, Non-Invasive Diagnostics.*

MECHANISMS OF METABOLIC DYSFUNCTION IN THE LIVER

There are several mechanisms that contribute to metabolic dysfunction in the liver, classified into:

- Primary (endogenous) factors – including genetic mutations.
- Secondary factors – caused by exogenous and endogenous xenobiotics.

These factors affect hepatocyte function, leading to disruptions in the metabolism of:

- Bilirubin and bile acids
- Proteins and amino acids
- Carbohydrates and glycoproteins
- Lipoproteins and lipids
- Porphyrins, trace elements, and mucopolysaccharides [1,2].

The liver plays a key role in the distribution of dietary components, particularly fatty acids, which enter the bloodstream via the portal vein from the small intestine. In a healthy liver, lipids (primarily triglycerides, cholesterol, and phospholipids) constitute 0.8–1.5% of total liver mass. An increase in lipid levels leads to the development of fatty liver disease.

NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

Non-alcoholic fatty liver disease (NAFLD) is a chronic condition characterized by clinical and morphological changes, including: • Hepatic steatosis (fat accumulation in liver cells) • Steatohepatitis (inflammatory liver disease) • Fibrosis (connective tissue overgrowth) • Cirrhosis (irreversible scarring of liver tissue) NAFLD occurs in individuals who do not consume alcohol excessively, meaning ethanol intake does not exceed 40g/day for men and 20g/day for women [1,2,3,6]. Currently, NAFLD is recognized by the following pathogenetic liver changes: 1. Hepatic steatosis – excessive triglyceride accumulation in hepatocytes (>5% of liver mass). o Small lipid inclusions in hepatocytes (2-3% fat accumulation) can be observed under a light microscope and are considered an early sign of liver steatosis [4,7,9]. 2. Non-alcoholic steatohepatitis (NASH) – a chronic diffuse liver disease characterized by necrotic-inflammatory processes that lead to fibrosis. 3. Liver fibrosis – excessive connective tissue proliferation without structural changes in the organ. 4. Liver cirrhosis (LC) – irreversible replacement of hepatic parenchymal tissue with fibrous connective tissue, forming a nodular liver structure [9,10].

PROGRESSION AND CLINICAL IMPACT

• NAFLD in the steatosis stage is generally benign and progresses slowly.
• NASH often remains asymptomatic for long periods and, without proper treatment, progresses to fibrosis or cirrhosis in 50% of cases.
• In the general population, one-third of NASH patients eventually develop cirrhosis [1,3,5].

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Historical Perspective
• 1884 – Frerichs first described liver changes in patients with diabetes mellitus.
• 1980 – Y. Ludwig and colleagues introduced the term "non-alcoholic steatohepatitis" (NASH) after studying liver pathology in obese and type 2 diabetes (T2DM) patients with no history of hepatotoxic alcohol consumption.
• M. Thaler identified the risk of cirrhosis development due to fatty liver disease.
• 2003 – At the 1st World Congress on Insulin Resistance (Los Angeles), NAFLD was officially recognized as a component of metabolic syndrome, along with obesity, type 2 diabetes, dyslipidemia, and hypertension [3,8,11].

EPIDEMIOLOGY

NAFLD is the most common form of chronic liver disease, accounting for ~70% of all liver disorders [6]. However, the true prevalence remains unclear since most patients do not seek medical attention or present with non-specific symptoms unrelated to gastrointestinal disorders.
Progression and Complications of Non-Alcoholic Fatty Liver Disease (NAFLD)
NAFLD is a slowly progressive disease, and not all patients develop cirrhosis. However, liver fibrosis has been observed in 25% of patients with hepatic steatosis [9,11].
Some authors suggest that:
• 10% of hepatic steatosis cases progress to non-alcoholic steatohepatitis (NASH) within 10 years.
• 5–25% of NASH cases progress to liver cirrhosis.
• 10% of cirrhotic NASH patients develop hepatocellular carcinoma (HCC) within 10 years [2,8,11].
Notably:
• 60–80% of all cryptogenic liver cirrhosis cases are linked to NAFLD [17].
• 10% of liver transplant cases are associated with cirrhotic NASH.
NAFLD is a concern not only for general practitioners and gastroenterologists but also for cardiologists, endocrinologists, and nephrologists, as it increases the risk of:
• Cardiovascular diseases
• Type 2 diabetes mellitus
• Chronic kidney disease [5,8,11]

New Terminology: Metabolic Associated Fatty Liver Disease (MAFLD)
In 2020, an international expert consensus statement introduced a new adaptive concept:
• Metabolic Associated Fatty Liver Disease (MAFLD)
This interpretation highlights the systemic and multifactorial nature of liver parenchymal damage and allows for a more specialized approach to medical care for patients with metabolic syndrome-related liver disease.
Experts who contributed to these clinical guidelines fully support this concept and recommend the use of WHO-approved nosological classifications, including:
• ICD-10 (International Classification of Diseases, 10th edition)
• ICD-11 (International Classification of Diseases, 11th edition) [7,8,11].
Etiopathogenesis of Non-Alcoholic Fatty Liver Disease (NAFLD)
NAFLD has distinct etiopathogenetic features, and based on a literature review, its causes can be classified into primary and secondary factors

PRIMARY FACTORS

These are intrinsic metabolic disorders that predispose an individual to NAFLD, including:
• Sedentary lifestyle
• Metabolic syndrome
• Type 2 diabetes mellitus (T2DM)
• Obesity
• Dyslipidemia
Secondary Factors
These are external or acquired factors that contribute to disease progression, including:
• Medication use:
o Glucocorticoids
o Amiodarone
o Estrogens
o Nonsteroidal anti-inflammatory drugs (NSAIDs)
o Antibiotics
• Nutritional imbalances:
o Starvation or drastic caloric fluctuations

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o Excessive carbohydrate intake
o Parenteral nutrition
o Protein and micronutrient deficiencies
• Digestive and absorptive disorders:
o Chronic gastrointestinal diseases
o Enzyme secretion deficiencies
• Metabolic diseases:
o Wilson-Konovalov disease
o Gout
• Endocrine disorders:
o Hyperthyroidism
• Physiological and environmental factors:
o Pregnancy
o Hypoxia (e.g., anemia, chronic heart failure, respiratory failure)
o Intestinal dysbiosis and gut-derived endotoxemia [5,7,10]
The pathophysiology underlying the progression of NAFLD When too many animal fats and readily digested carbohydrates are consumed, free fatty acids (FFAs) are released from the digestive system into the bloodstream, which then travels to the liver and adipose tissue. As a result, fat cells grow and multiply, causing adipocyte hypertrophy and hyperplasia.

2. Modified adipose tissue endocrine function: Pro-inflammatory mediators such as tumor necrosis factor-alpha (TNF-α), eicosanoids (EOCs), and interleukin-6 (IL-6) are secreted in greater amounts.
Chronic low-grade inflammation brought on by these inflammatory mediators develops gradually [3].
Additionally, an excessive influx of FFAs into the portal circulation and liver results in:
• Lipid imbalance – Excess fat storage surpasses hepatic lipid metabolism capacity.
• Hepatocyte steatosis – Accumulation of triglyceride-filled lipid vacuoles in hepatocytes.
• Hepatocyte damage – Pro-inflammatory mediators directly impair hepatocyte membranes, leading to:
o Cytochrome P450 activation
o Lipid peroxidation (LPO)
o Oxidative stress, causing hepatocyte apoptosis and necrosis
o Fibrotic tissue deposition
Steatosis to Steatohepatitis Progression Mechanisms
Elevated FFAs, which cause direct damage to hepatocyte membranes, and increased TNF-α secretion by adipose tissue are linked to the change from simple steatosis to non-alcoholic steatohepatitis (NASH).
Cytochrome P450 activation; oxidative stress and lipid peroxidation (LPO); excessive reactive oxygen species (ROS) accumulation; toxic xenobiotic production; and endotoxemia brought on by gut dysbiosis [1, 2, 5, 6].
Gut Microbiota's Function in NAFLD
The importance of gut microbiota in the development of obesity and non-alcoholic fatty liver disease has been highlighted by recent research. Dysbiosis of the intestinal tract causes:
Increased intestinal permeability leads to a higher absorption of endotoxins;
bacterial toxins, such as lipopolysaccharides, intensify hepatic inflammation; fatty livers are more vulnerable to TNF-α, IL-1, IL-6, and IL-8; and hepatocyte necrosis and fibrosis occur more quickly [4].
Therefore, an imbalance in the gut microbiota exacerbates the course of NAFLD by causing morphological and fibrotic liver alterations.
The Gut Microbiota's Contribution to the Development of NAFLD
In non-alcoholic fatty liver disease (NAFLD), dysbiosis of the gut microbiota aggravates fibrotic and morphological alterations in the liver, resulting in:
• Hepatocyte dystrophy and elevated disease histological activity.
• Sinusoidal mononuclear cell activation, which fuels inflammation.
Impaired bile synthesis and excretion.
• Increased sensitivity of fatty liver to pro-inflammatory cytokines (TNF-α, IL-1, IL-6, IL-8), which promotes hepatocyte necrosis and fibrosis [1,6,8].

CLINICAL PRESENTATION AND DIAGNOSIS OF NAFLD
Scientific studies indicate that NAFLD is often asymptomatic, and the disease is frequently diagnosed incidentally through laboratory or imaging tests performed on patients with metabolic syndrome.
Non-Alcoholic Steatohepatitis (NASH) Symptoms
NASH presents with non-specific symptoms that may indicate liver damage but do not reliably determine disease severity. The most common symptoms include:
• Asthenovegetative syndrome (fatigue, weakness, irritability).

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• Dyskinetic syndrome – a feeling of heaviness or discomfort in the right upper quadrant, which may be transient or persistent.
• Itching, anorexia, dyspeptic symptoms, and jaundice, when combined with portal hypertension, Physical Examination and Additional Diagnostic Criteria
• Hepatomegaly (liver enlargement) is detected in 50–75% of NAFLD patients [5].
• Additional diagnostic tests are indicated in cases of:
o Asymptomatic elevation of aminotransferase levels.
o Unexplained persistent hepatomegaly.
o Hepatomegaly detected on radiological imaging.
o Exclusion of other potential causes of liver enlargement.
In rare cases, patients with NASH may exhibit chronic liver disease signs, such as:
• Telangiectasia (spider veins).
• Palmar erythema (reddening of the palms).
NAFLD-related symptoms are detected in 10–15% of individuals without clinical manifestations Important Factors in the Diagnosis of NAFLD

In order to evaluate risk factors, a thorough patient history is essential. First, the following circumstances need to be ruled out:
• Liver illness from alcohol.
• Viral hepatitis B and C, which are chronic.
• Hemochromatosis that is inherited.
• Wilson's illness.
• Liver autoimmune disorders.

Risk Factors for NAFLD Progression to NASH and Fibrosis Numerous clinical observations have identified high-risk factors for the progression of non-alcoholic fatty liver disease (NAFLD) to non-alcoholic steatohepatitis (NASH) and liver fibrosis, including:
• Age over 45 years
• Female sex
• Body mass index (BMI) > 28 kg/m²
• ALT elevation ≥ 2 times the upper normal limit
• Triglyceride levels > 1.7 mmol/L
• Arterial hypertension
• Type 2 diabetes mellitus (T2DM)
• Insulin resistance index (HOMA-IR) > 5

The presence of two or more of these criteria indicates a high risk of liver fibrosis.
It is important to note that gradual weight loss can potentially reverse NAFLD and NASH. However, rapid weight loss has been associated with disease progression from one stage to another.
Laboratory and Instrumental Diagnostics for NAFLD
Liver Enzyme Abnormalities
• ALT and AST activity increases 4–5 times the normal range but AST/ALT ratio remains < 1 (ALT is usually higher).
• Alkaline phosphatase (ALP) and gamma-glutamyl transpeptidase (GGT) levels increase in 40–60% of cases (typically not exceeding twice the upper limit).

Lipid and Glucose Metabolism Abnormalities
• Hypertriglyceridemia and hypercholesterolemia
• Hyperglycemia (fasting glucose > 6.1 mmol/L)
• Impaired glucose tolerance (IGT) and elevated C-peptide levels

Other Biochemical Markers
• Hypoalbuminemia
• Mild hyperbilirubinemia (30–35 mmol/L)
• Thrombocytopenia and prolonged prothrombin time

Key Clinical Differences Between Simple Hepatic Steatosis and NASH
The main distinguishing factor between simple fatty liver (hepatic steatosis) and NASH is the degree of biochemical hepatocellular injury (cytolysis syndrome).
• Cytolysis (hepatocellular damage) is observed in 50–90% of NASH patients, particularly in specialized clinical settings.
Although AST is more common in cirrhotic patients, ALT is frequently greater than AST.
• Insulin resistance (IR) is indicated by ALT levels and metabolic indicators, and ALT levels may be employed as an extra biomarker for IR patients.

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• Advanced fibrosis and NASH may be present when low ALT levels are accompanied by a high BMI.

NAFLD IMAGING METHODS AND DIAGNOSTIC MARKERS

The presence of cytokeratin-18 (CK18-Asp396) fragments in serum, which are released during hepatocyte apoptosis mediated by activated caspases, is a hallmark of non-alcoholic fatty liver disease (NAFLD). Elevated levels of CK18 fragments are highly specific for NASH, setting it apart from simple steatosis.This test has a sensitivity of 85.7% and a specificity of 99.9% [2,3].

Non-Invasive Methods for Assessing Liver Steatosis, Inflammation, and Fibrosis

Non-invasive tests such as FibroScan and FibroMax have been introduced into clinical practice to evaluate steatosis, inflammation, and fibrosis [4,6].

•FibroTest sensitivity and specificity range between 70–90%.

• However, due to the lack of direct histopathological validation with liver biopsy findings, FibroTest is not considered a primary diagnostic tool for NAFLD.

Since standard liver function tests (LFTs) are non-specific and do not always correlate with histological changes (such as inflammation, trauma, or fibrosis), liver biopsy remains the "gold standard" for diagnosing NAFLD, staging disease severity, and assessing therapeutic response.

Scoring Systems for NASH Risk Assessment

Several indices have been developed to estimate the risk of NASH:

• HAIR Index (Hypertension, ALT > 40 U/L, Insulin Resistance)

o Sensitivity: 80%

o Specificity: 89% [31]

• BAAT Index

o BMI > 28 kg/m²

o Age > 50 years

o ALT > 2 times the upper normal limit

o Elevated triglycerides

o A BAAT score <1 nearly excludes NASH (100% negative predictive value) [2,3,10].

- NAFLD Score (J.-H. Lee, JNYOX formula)

o Formula: 8 × ALT/AST + BMI

o NAFLD unlikely if score < 31

Probability of NAFLD >90% if score > 36

Specificity: 91.2% [1,3]

Imaging Techniques for NAFLD Diagnosis

Instrumental diagnostics for NAFLD include:

• Computed tomography (CT)

• Magnetic resonance imaging (MRI)

• Abdominal ultrasound (USG) – assessing hepatic echogenicity and hepatomegaly [8]

Sensitivity and specificity of imaging modalities:

• Ultrasound: 45% sensitivity, 90% specificity

• MRI: 90.9% sensitivity, 94% specificity [3,5]

Ultrasound and Imaging Modalities for NAFLD Diagnosis

If a patient has no clinical symptoms but liver function tests (LFTs) show abnormalities, and a liver biopsy is not feasible, ultrasound (USG) is considered an objective method for diagnosing hepatic steatosis.

USG is advised for patients who have one or more NASH risk factors and for tracking the course of the illness over time.

Important Ultrasound Characteristics of Hepatic Steatosis

1. The echogenic signal is distal attenuated (because to the high fat content).
2. Hepatic echogenicity that is diffusely elevated ("bright liver" sign).
3. The liver has a higher echogenicity than the kidneys.
4. Vascular structures that are blurred as a result of significant fat infiltration.

Ultrasound's limitations when evaluating cirrhosis and fibrosis

Ultrasound is helpful, although it is not always able to identify severe cirrhosis or fibrosis.

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Ultrasound is helpful, although it is not always able to identify severe cirrhosis or fibrosis.

In these situations, hepatic fat infiltration may be detected with the use of magnetic resonance imaging (MRI) and computed tomography (CT).

Important CT and MRI Characteristics of NAFLD • CT results: Fat accumulation is indicated by a lower hepatic attenuation index.

The benefits of MRI

High tissue contrast and the capacity to image whole organs in several planes

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Table 1.
Stepwise Algorithm for Diagnosing NAFLD

Diagnostic Parameter	Information Scope
Medical History	•**Assess alcohol consumption (non-hepatotoxic dose:** <20 g/day ethanol for women, <40 g/day for men**).** • **Identify other** metabolic syndrome components**:** obesity, type 2 diabetes mellitus (T2DM), dyslipidemia, arterial hypertension**.** • **Exclude** hepatotoxic drug use **within the past** 3 months**.** • **Rule out** risk factors for viral hepatitis**: blood contact, transfusion, dental visits, tattoos, piercings.** • **Exclude** hereditary liver diseases**: Wilson's disease, hemochromatosis**
Physical Examination	• **Assess** anthropometric parameters **(**BMI >30, waist circumference (WC), hip circumference (HC), WC/HC ratio**).** • **Rule out** alcoholic liver disease stigmas**.** • **Rule out** severe cholestatic syndrome**.** • **Check for** melanoderma (hyperpigmentation)**.** • **Screen for** portal hypertension **and** hepatic encephalopathy
Laboratory Tests	• Biochemical blood panel **(cytolysis, cholestasis, immune inflammation, hepatic insufficiency),** coagulation profile, lipid panel**.** • Carbohydrate metabolism assessment **(**glucose, insulin, HOMA-IR index**).** • Viral hepatitis screening **(**HBsAg, anti-HBc, anti-HCV**).** • Autoimmune hepatitis panel **(**IgG, IgM, anti-actin antibodies, ANA, ASMA, AMA**).** • Hemochromatosis screening **(**serum transferrin saturation, ferritin, genetic testing for C282Y, H63D mutations**).** • Wilson's disease screening **(**serum ceruloplasmin, 24-hour urine copper excretion**).** • Hepatocellular carcinoma marker **(**alpha-fetoprotein (AFP)**).**
Ultrasound, CT, MRI	• Ultrasound signs of hepatic steatosis**.** • **Exclude** focal liver lesions**.**
Liver Biopsy	•Macrovacuolar hepatic steatosis**.** •Hepatitis with predominant lobular inflammation**.** • Fibrosis signs**.**

CLINICAL ASPECTS AND DIAGNOSIS OF NAFLD

After ruling out all other possible causes of liver damage, mainly drug-, alcohol-, and viral-induced liver disease, non-alcoholic fatty liver disease (NAFLD) is diagnosed.

Furthermore, people of all ages, particularly older patients, should have their risk of acquiring diffuse liver illnesses taken into account. Comorbid diseases might result from the frequent coexistence of NAFLD with other metabolic disorders, especially metabolic syndrome.

As a result, a wide range of laboratory and imaging tests should be included in the diagnostic evaluation of suspected NAFLD.

CONCLUSION

The significant prevalence of non-alcoholic fatty liver disease (NAFLD) calls for continued investigation into its etiology as well as the creation of non-invasive diagnostic techniques to identify more complex and advanced stages of the illness, including cirrhosis, fibrosis, and steatohepatitis.

Knowing how NAFLD is multifactorial and how it contributes to the emergence of multimorbidity enables:

Choosing efficient pharmaceutical and non-pharmacological treatments; determining priority treatment plans; and accurately assessing prognoses

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