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LEAN MASLD: METABOLIC HETEROGENEITY AND IMPLICATIONS FOR DISEASE PROGRESSION

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ABSTRACT

One of the most widespread liver diseases in the world is metabolic dysfunction-associated steatotic liver disease (MASLD), known until recently as non-alcoholic fatty liver disease (NAFLD). Its prevalence is estimated at 25% in the global population. MASLD is defined as a clinical condition characterized by excessive fat accumulation in the liver in the presence of at least one cardiometabolic risk factor. Although obesity plays a significant role in the pathomechanism of MASLD, it is estimated that approximately 10-20% of MASLD cases occur in individuals with a BMI <25 kg/m² (<23 kg/m² in the Asian population). As studies show, individuals with lean MASLD have a higher risk of liver fibrosis and overall mortality.

The aim of this study is a critical review of the current literature regarding the Lean MASLD phenotype, with particular emphasis on its metabolic heterogeneity. The work analyzes unique pathophysiological mechanisms, such as genetic predispositions and adipose tissue distribution disorders, and evaluates their impact on the risk of progression to advanced fibrosis and hepatocellular carcinoma. This work constitutes a literature review, focusing on clinical and cohort studies as well as review papers concerning the pathogenesis, diagnostics, and clinical course of liver steatosis in individuals with a normal BMI.

The literature analysis indicates a significant role of genetic variants, mainly the PNPLA3 polymorphism (I148M), which predisposes lean individuals to disease progression independently of metabolic factors. It has been shown that despite a normal BMI, these patients are often characterized by visceral adipose tissue dysfunction and an altered gut microbiota profile. Metabolic heterogeneity is manifested by the existence of a subgroup of patients with a "metabolically healthy" profile, in whom cardiovascular risk may be lower, but the risk of progression of liver lesions remains significant. The following work emphasizes that Lean MASLD is not a milder form of liver steatosis, and its diagnostics require going beyond the standard BMI measurement. It is necessary to implement personalized monitoring strategies, taking into account the genetic profile and metabolic assessment, to prevent long-term complications in this specific group of patients.

KEYWORDS

Lean MASLD, MASLD, Metabolic Heterogeneity, Liver Fibrosis, PNPLA3, Visceral Adiposity

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1. Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) has become the most common cause of chronic liver disease worldwide. It is estimated to affect approximately 25–30% of the adult population, posing a significant burden on healthcare systems. For decades, this disease was identified almost exclusively with the obesity epidemic and metabolic syndrome. However, increasing clinical evidence points to the existence of a specific phenotype referred to as "Lean MASLD," which occurs in individuals with a normal body mass index (BMI)[1,2].

Lean MASLD is defined in patients whose liver fat content exceeds 5%, despite a BMI below 25 kg/m² (or <23 kg/m² in Asian populations). Although the prevalence of this phenotype varies geographically, meta-analyses suggest that it affects between 10% and even 20% of all cases of liver steatosis. This phenomenon challenges the traditional diagnostic approach based on screening overweight individuals and indicates the existence of more complex pathophysiological mechanisms than mere excessive caloric intake[1].

A key issue in understanding Lean MASLD is the metabolic heterogeneity of these patients. Recent studies emphasize that lean patients are not a homogeneous group. Some of them exhibit a "metabolically obese" phenotype (TOFI – thin on the outside, fat on the inside), characterized by excessive accumulation of visceral adipose tissue, ectopic fat deposition in organs, and occult insulin resistance. Other patients develop steatosis mainly on the basis of strong genetic predispositions, particularly the PNPLA3 gene polymorphism (I148M variant) and TM6SF2, which impair the export of lipids from hepatocytes independently of the total body fat content[3,6].

Contrary to initial assumptions, a lean patient profile does not guarantee a mild disease course. The latest cohort studies indicate that patients with Lean MASLD may show higher overall mortality and faster progression to advanced fibrosis and hepatocellular carcinoma (HCC) compared to patients with obesity. This non-obvious disease dynamics makes understanding the factors determining progression a priority in contemporary gastroenterology. This work aims to summarize current knowledge on metabolic heterogeneity in Lean MASLD and to identify markers allowing for the early identification of high-risk patients[2,3].

2. Materials and Methods

This work is a narrative review, constituting a critical synthesis of contemporary scientific reports regarding Lean MASLD. The aim of the adopted methodology was to identify the key pathophysiological mechanisms and clinical differences characterizing this phenotype.

2.1. Data Collection Strategy

The literature search was conducted in the PubMed and Google Scholar databases, focusing on works published between 2017 and 2026. The following keywords were used: lean NAFLD, lean MASLD, pathogenesis, genetics, PNPLA3, gut-liver axis, and clinical outcomes. Articles published in English were selected for review.

2.2. Literature Selection Criteria

The primary inclusion criterion was the substantive relevance of the publications with respect to patients with a BMI < 25 kg/m². Priority was given to the most recent meta-analyses, systematic reviews, and clinical trials with a high strength of evidence. Case reports and texts that did not directly relate to the unique progression mechanisms in the lean population were excluded.

2.3. Rationale and Limitations

The adopted narrative review format allows for a multi-faceted approach to the subject, combining genetic and epidemiological data. Although the selection of sources was based on the author's subjective assessment of their substantive value, efforts were made to ensure the analysis was based on the most current guidelines from international hepatological societies. A limitation of this work is the narrowing of the search to two main databases, which, however, allowed for a synthesis of the most important and influential reports in the discussed area.

3. Epidemiology and Definitions of MASLD

3.1. Evolution of Definitions: From NAFLD to MASLD

For decades, non-alcoholic fatty liver disease (NAFLD) was defined as the presence of steatosis on imaging or histology, while simultaneously excluding excessive alcohol consumption and other secondary causes. However, according to the 2023 international consensus, this term has been replaced by MASLD (Metabolic Dysfunction-Associated Steatotic Liver Disease).

The main assumptions of the new definition include:

Steatosis criterion: confirmation of liver steatosis by imaging (e.g., USG, MRI-PDFF), elastography (CAP), or histology.

Cardiometabolic risk factors (CMRF): at least one of five risk factors (arterial hypertension, glycemic disorders, elevated BMI or waist circumference, dyslipidemia – high TG or low HDL).

Lean MASLD category: A specific subgroup of patients in whom, despite the absence of elevated BMI (<25 kg/m²), the presence of other metabolic disorders or strong genetic predispositions is found.

It is worth emphasizing that the term "non-obese MASLD" (often used interchangeably) is broader and includes patients with a BMI below 30 kg/m² (or <25 kg/m² in Asia), which also accounts for the overweight group. In the metabolic context, however, it is crucial to distinguish that body weight does not always correlate with visceral adipose tissue (VAT) content and ectopic fat[1,4].

3.2. Global Epidemiology

MASLD currently affects approximately 30% of the global population, and it is predicted that this number will increase significantly by 2030 due to the type 2 diabetes epidemic. It is estimated that the lean MASLD phenotype affects on average 10–20% of patients with liver steatosis. Meta-analyses indicate that the highest prevalence of Lean NAFLD is recorded in Asia (approx. 15–20%), while in Western countries it oscillates around 10%. Patients with Lean MASLD are typically younger at the time of diagnosis, more often female, and may exhibit a lower intensity of classic metabolic syndrome features compared to patients with obesity. The increase in incidence in this group is particularly concerning, as these patients are less frequently subjected to routine screening[2,4].

4. Pathophysiological Mechanisms of Lean MASLD

The pathogenesis of fatty liver disease in individuals with a normal body mass represents a unique challenge for modern hepatology, moving beyond the classic model of obesity-induced lipotoxicity. This phenotype is based on a complex interaction between genetic predisposition, adipose tissue dysfunction, and disturbances of the gut-liver axis.

4.1. Dominance of Genetic Factors and Lipid Metabolism Disorders

Unlike patients with a high BMI, for whom the primary factor underlying the pathophysiology of the disease is excessive caloric intake, in lean patients, the so-called "genetic burden" plays a key role. Two genes can be distinguished that play a particular role in the development of lean MASLD. The strongest genetic predictor of Lean MASLD is the I148M variant in the PNPLA3 gene. Under physiological conditions, the PNPLA3 protein exhibits lipolytic activity towards triglycerides in hepatocytes. The point mutation causes a loss of this function and resistance to proteasomal degradation, which leads to the accumulation of the mutated protein on the surface of lipid droplets and blocks access to other lipases. Studies suggest that in lean individuals, this variant is more strongly associated with fibrosis progression than in obese patients, indicating a genetically determined, aggressive form of steatosis. On the other hand, the E167K variant in the TM6SF2 gene leads to the impairment of the formation and secretion of very-low-density lipoproteins (VLDL). This results in lipid retention within hepatocytes while simultaneously lowering serum lipid concentrations, which paradoxically may mask cardiovascular risk in a lean patient[3,5,6].

4.2. The TOFI Phenotype and Visceral Adipose Tissue Dysfunction

Despite a normal BMI, patients with Lean MASLD exhibit a specific body composition architecture. A key concept here is metabolically obese normal weight (MONW), which often corresponds to the characteristics of the TOFI phenotype – Thin on the Outside, Fat on the Inside. In these patients, there is a preferential accumulation of adipose tissue within the mesentery and omentum (visceral fat) and in skeletal muscles. Visceral adipose tissue (VAT) is characterized by a high rate of lipolysis and resistance to the inhibitory effect of insulin, resulting in a constant influx of free fatty acids (FFA) to the liver via the portal circulation. A significant reduction in adiponectin concentration is also observed, which under physiological conditions enhances fatty acid oxidation in mitochondria through the activation of AMPK kinase. Adiponectin deficiency, along with a simultaneous increase in pro-inflammatory cytokine levels (TNF-alpha, IL-6), induces a state of chronic low-grade inflammation, promoting the transition from simple steatosis to inflammation, that is MASH - Metabolic Dysfunction-Associated Steatohepatitis, formerly known as NASH [5,7].

4.3. Insulin Resistance and De Novo Lipogenesis (DNL)

In lean patients, the phenomenon of selective hepatic insulin resistance occurs. Despite the absence of systemic obesity, insulin ceases to effectively inhibit gluconeogenesis but retains the capacity to stimulate lipogenesis through the SREBP-1c protein pathway. The increased influx of substrates (glucose and fructose) and hyperinsulinemia stimulate *de novo* fatty acid synthesis, which is particularly evident in lean patients with diets rich in simple sugars. Furthermore, fatty acid overload leads to impaired beta-oxidation and increased production of reactive oxygen species (ROS), resulting in DNA damage in hepatocytes and the activation of hepatic stellate cells (HSC), which are responsible for fibrosis[6].

4.4. Gut Microbiota Interactions (Gut-Liver Axis)

Recent studies have shown that Lean MASLD is associated with a unique microbiological signature, distinct from that observed in obese individuals. In lean individuals, a lower species diversity and an increased abundance of endogenous ethanol-producing bacteria are found. Changes in the microbiome lead to damage of the intestinal barrier. Lipopolysaccharides (LPS) penetrate into the portal circulation, where, by binding to TLR4 receptors on hepatic macrophages, they initiate an inflammatory cascade, which is a key element of disease progression to MASH in patients without classic metabolic syndrome[8].

5. Metabolic Heterogeneity

Metabolic heterogeneity is an immanent feature of Lean MASLD, determining the difficulties in unifying diagnostic and therapeutic strategies. Recent scientific evidence allows for the identification of at least three main subphenotypes within this population, differing in pathogenesis and the risk of extrahepatic complications.

5.1. Metabolically Unhealthy Phenotype (MONW – Metabolically Obese Normal Weight)

The most widely described subphenotype is the group of patients referred to as metabolically obese with normal weight (MONW). These patients, despite a normal BMI, exhibit a biochemical and metabolic profile identical to patients with clinical obesity. The key element is the TOFI (Thin-on-the-Outside, Fat-on-the-Inside) phenomenon, characterized by a disproportionately high volume of visceral adipose tissue (VAT) and ectopic fat, with a simultaneous deficit of subcutaneous adipose tissue (SAT). This phenotype is characterized by pronounced peripheral and hepatic insulin resistance, atherogenic dyslipidemia (elevated triglycerides, low HDL cholesterol), and adipocyte dysfunction manifested by hypoadiponectinemia. It is also associated with the highest risk of major adverse cardiovascular events (MACE) and complications related to metabolic syndrome (Type 2 diabetes)[6,7].

5.2. Genetic-Driven Lean MASLD

In contrast to the MONW group, patients with the genetic subphenotype may not exhibit classic features of insulin resistance or abdominal obesity. The main causative factor is the presence of the previously mentioned PNPLA3 and TM6SF2 variants. In these patients, lipid accumulation in hepatocytes results from a primary, genetically determined enzymatic defect and occurs independently of systemic inflammation or excess adipose tissue. In this case, steatosis is not the result of metabolic 'overloading' of the body, but rather a local disturbance of lipid homeostasis, which can lead to a false sense of clinical security despite the silent progression of fibrotic changes[3,6].

5.3. The Role of Sarcopenia and Myosteatosis in Metabolic Heterogeneity

In the assessment of the metabolic heterogeneity of MASLD, the skeletal muscle-liver axis is increasingly recognized as a key determinant of clinical outcomes. Even in patients with a normal BMI, the disease phenotype often involves a combination of muscle mass deficiency (sarcopenia) and impaired muscle quality due to fatty infiltration (myosteatosis). Evidence indicates a bidirectional and reciprocal interaction: skeletal muscle dysfunction drives the progression of liver pathology, while intrinsic liver disease conversely exacerbates muscle degradation.

Notably, skeletal muscle insulin resistance in MASLD may be even more strongly associated with the disease than hepatic insulin resistance. Reduced peripheral glucose uptake is an early harbinger of metabolic failure, frequently preceding pancreatic beta-cell dysfunction by decades. This is particularly relevant in lean MASLD, where clinical studies using the hyperinsulinemic-euglycemic clamp have demonstrated that peripheral glucose disposal rates are approximately 45% lower than in healthy, weight-matched controls. Sarcopenia, which has a prevalence ranging from 10% to 70% in MASLD populations, serves as an independent risk factor for advanced fibrosis and increased mortality, underscoring its central role in the metabolic diversity of this condition. Thus, muscle-targeted assessments are vital for characterizing the heterogeneous nature of lean MASLD [5,7].

5.4. Gut-Liver Axis and Microbiome Variability

The gut-liver axis in patients with Lean MASLD presents a different microbiota architecture than in individuals with a high BMI. Metagenomic studies indicate a specific increase in the Bacteroidetes to Firmicutes ratio and an overrepresentation of the Lachnospiraceae and Ruminococcus families. In contrast to classic MASLD, in lean individuals, the key differentiating factor is the increased production of endogenous ethanol. This leads to a specific upregulation of cytochrome P450 2E1, resulting in the generation of high levels of oxidative stress and damage to mitochondrial DNA (mtDNA) even with low caloric intake.

The above metagenomic distinctions emphasize that in individuals with a normal body mass, liver pathology can evolve through pathways similar to toxic organ damage, which significantly differentiates their risk profile from patients with classic obesity[8].

6. Disease Progression and Clinical Implications

The clinical course of Lean MASLD stands in contradiction to the traditional view of liver damage associated solely with obesity. Understanding the dynamics of progression in this group is crucial for early intervention and improving the prognosis of normal-weight patients.

6.1. Fibrosis Dynamics and Imaging Diagnostics

Despite the lower severity of systemic metabolic syndrome features, lean patients paradoxically exhibit a higher risk of advanced fibrosis at the time of diagnosis. Histopathological data suggest that in lean individuals, the process of fibrogenesis may proceed in a more insidious and aggressive manner. Faster progression to F3-F4 stages in lean patients suggests that intracellular mechanisms (oxidative stress, mitochondrial dysfunction) are stronger stimuli for stellate cells than excess adipose tissue. A normal BMI cannot be an exclusionary factor in hepatological diagnostics. The widespread use of non-invasive fibrosis assessment methods, such as liver elastography (FibroScan) or magnetic resonance imaging (MRI-PDFF), is recommended for every patient with an unclear etiology of elevated liver enzymes, even in the absence of abdominal obesity features[9,10].

6.2. Oncological Risk and HCC Surveillance

Another undeniable threat in the lean population is the specific development of hepatocellular carcinoma (HCC). Lean patients more frequently develop HCC in a liver not affected by cirrhosis. This mechanism is likely driven by the direct mutagenic effect of oxidative stress and endogenous ethanol (see Section 4.4). Standard oncological surveillance programs, which focus exclusively on patients with cirrhosis, may be insufficient for the Lean MASLD population. The implication is to consider earlier screenings (Ultrasound, AFP) in lean patients with confirmed MASH and a high genetic risk score (e.g., PNPLA3 homozygotes)[2,3,7].

6.3. Cardiovascular and Metabolic Risk

Extrahepatic risk in lean patients is highly phenotype-dependent, requiring an individual approach to cardiological care. Patients with the MONW/TOFI phenotype exhibit high cardiovascular mortality (MACE) resulting from metabolic dysfunction hidden under a normal BMI. In contrast, in genetic patients, this risk is lower but still significant due to the atherogenic VLDL profile induced by the TM6SF2 variant. Lean NAFLD patients require control of cardiovascular risk factors. The fact that a patient is lean should not delay the implementation of pharmacotherapy if the lipid profile and HOMA-IR index indicate metabolic dysfunction[3,10].

6.4. Prognosis and Overall Mortality – The "Lean Paradox"

Paradoxically, overall mortality in Lean MASLD patients is often higher than in overweight patients (the so-called "lean paradox"). Recent studies have shown that lean patients have an 11% higher mortality from liver-related and overall causes compared to obese patients. This poor prognosis may result from delayed diagnosis, a lack of appropriate therapeutic standards dedicated to lean individuals, and lower metabolic reserves. This suggests a need for separate diagnostic pathways that do not rely on traditional anthropometric indicators[7,10,11].

7. Conclusions

Analysis of scientific reports regarding Lean MASLD allows for the formulation of the conclusion that this entity constitutes a unique clinical challenge. A normal BMI does not exclude the risk of advanced metabolic disorders, and the significant heterogeneity of this population indicates the necessity for personalization of medical care. Due to the misleading phenotype and often normal results of routine biochemical tests, it is essential to modernize screening protocols. The 'Lean MASLD paradox' phenomenon, manifested by higher mortality and the risk of HCC in the absence of cirrhosis, represents a critical challenge for contemporary hepatology, requiring the implementation of more restrictive surveillance for this patient population.

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