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# Precision Medicine in Metabolic Dysfunction-Associated Steatotic Liver Disease: Advances in Diagnosis, Risk Stratification, and Personalized Management

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## ABSTRACT

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) has emerged as one of the most prevalent chronic liver diseases worldwide and represents a growing challenge for healthcare systems due to its close relationship with obesity, insulin resistance, type 2 diabetes mellitus, dyslipidemia, and metabolic syndrome. This review aimed to integrate current scientific evidence regarding the epidemiology, pathophysiology, clinical progression, diagnosis, treatment, and emerging role of precision medicine in MASLD. International scientific literature

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was reviewed through recognized biomedical databases, incorporating original studies, systematic reviews, meta-analyses, clinical practice guidelines, and international consensus statements relevant to the disease. The evidence demonstrates a substantial global burden of MASLD, with particularly high prevalence estimates reported in Latin America and other regions experiencing rapid growth in cardiometabolic disorders. Disease progression is heterogeneous, ranging from hepatic steatosis to metabolic dysfunction-associated steatohepatitis, advanced fibrosis, cirrhosis, and hepatocellular carcinoma, with fibrosis representing one of the most important predictors of adverse clinical outcomes. Advances in non-invasive biomarkers, serum-based fibrosis scores, transient elastography, and magnetic resonance-based techniques have improved risk stratification and reduced dependence on liver biopsy. Lifestyle modification remains fundamental to treatment, while emerging pharmacological therapies provide additional opportunities for patients with specific metabolic and hepatic profiles. The integration of clinical characteristics, metabolic phenotype, fibrosis stage, imaging findings, and biological markers supports the transition toward precision medicine. MASLD should therefore be approached as a multisystem metabolic disease requiring early identification, multidisciplinary management, comprehensive cardiometabolic control, and individualized therapeutic strategies to reduce progression and long-term complications.

***KEYWORDS***

*Metabolic Dysfunction-Associated Steatotic Liver Disease, MASLD, metabolic dysfunction, hepatic steatosis, metabolic syndrome, insulin resistance, liver fibrosis, non-invasive diagnosis, precision medicine, hepatology*

## INTRODUCTION

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) has emerged as the most prevalent chronic liver disease worldwide, representing a major public health challenge due to its rapidly increasing incidence and its close association with obesity, type 2 diabetes mellitus, metabolic syndrome, and cardiovascular disease. The transition from the previous nomenclature of non-alcoholic fatty liver disease (NAFLD) to MASLD reflects a paradigm shift that emphasizes the central role of metabolic dysfunction in disease development and progression while providing a more clinically meaningful framework for diagnosis, research, and patient management [1], [2]. Current epidemiological estimates indicate that nearly one-third of the global adult population is affected by hepatic steatosis associated with metabolic dysfunction, with prevalence continuing to rise in parallel with the worldwide obesity epidemic. This trend is particularly evident in low- and middle-income countries, including many nations in Latin America, where changes in dietary habits, urbanization, and sedentary lifestyles have substantially increased the burden of metabolic disorders and chronic liver disease [5], [7], [10].

Although hepatic steatosis may remain clinically silent for years, MASLD encompasses a broad pathological spectrum ranging from isolated steatosis to metabolic dysfunction-associated steatohepatitis (MASH), progressive fibrosis, cirrhosis, portal hypertension, hepatocellular carcinoma, and liver-related mortality. Importantly, liver complications represent only one aspect of the disease burden. Cardiovascular disease remains the leading cause of death among individuals with MASLD, followed by extrahepatic malignancies and advanced liver disease, highlighting the systemic nature of this condition and reinforcing the need for multidisciplinary management approaches involving hepatologists, internists, endocrinologists, cardiologists, nutritionists, and primary care physicians [3], [6], [12], [18].

During the last decade, advances in molecular biology, genetics, metabolomics, imaging technologies, and artificial intelligence have considerably expanded current understanding of MASLD pathophysiology. Rather than representing a simple accumulation of triglycerides within hepatocytes, MASLD is now recognized as a complex metabolic disorder resulting from the interaction between insulin resistance, adipose tissue dysfunction, chronic low-grade inflammation, oxidative stress, mitochondrial impairment, lipotoxicity, intestinal microbiota alterations, endocrine signaling, and genetic susceptibility. Variants in genes such as *PNPLA3*, *TM6SF2*, *MBOAT7*, and *HSD17B13* have demonstrated significant influence on disease susceptibility, fibrosis progression, and clinical outcomes, supporting the concept that individual genetic profiles contribute substantially to disease heterogeneity [6], [14], [17].

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Recent international consensus statements have also transformed the conceptual framework of fatty liver disease by redefining diagnostic criteria based on positive metabolic characteristics rather than exclusion of alcohol intake. This modification facilitates earlier identification of affected patients, reduces diagnostic ambiguity, and promotes greater inclusion of individuals with overlapping liver conditions frequently encountered in clinical practice. Furthermore, the adoption of standardized nomenclature facilitates international collaboration and improves comparability across epidemiological and clinical studies conducted in different populations [1], [2].

The clinical importance of MASLD extends far beyond hepatology because its prevalence closely parallels the global expansion of obesity, type 2 diabetes mellitus, hypertension, dyslipidemia, chronic kidney disease, and cardiovascular disorders. These metabolic comorbidities not only increase disease prevalence but also accelerate fibrosis progression and worsen long-term prognosis. Several longitudinal cohort studies have demonstrated that fibrosis stage constitutes the strongest independent predictor of liver-related complications, overall mortality, and cardiovascular outcomes, underscoring the necessity of identifying high-risk patients before irreversible hepatic damage occurs [9], [16], [19].

In recent years, substantial progress has been achieved in non-invasive diagnostic strategies. Conventional liver biopsy, although considered the historical reference standard, presents limitations including invasiveness, sampling variability, interobserver differences, procedural risks, and limited feasibility for population-level screening. Consequently, clinical practice has progressively shifted toward multimodal diagnostic algorithms incorporating serum biomarkers, fibrosis scoring systems, vibration-controlled transient elastography, magnetic resonance elastography, proton density fat fraction magnetic resonance imaging, and emerging artificial intelligence-based predictive models. These approaches allow improved risk stratification while reducing unnecessary invasive procedures and facilitating longitudinal patient monitoring [3], [4], [15].

Therapeutic management has similarly undergone profound evolution. Lifestyle modification remains the cornerstone of treatment, emphasizing sustained weight reduction, Mediterranean dietary patterns, structured physical activity, and optimization of metabolic risk factors. However, increasing knowledge regarding disease mechanisms has stimulated the development of targeted pharmacological therapies addressing insulin resistance, inflammation, lipid metabolism, bile acid signaling, fibrogenesis, and incretin pathways. Novel agents including glucagon-like peptide-1 receptor agonists, dual incretin agonists, fibroblast growth factor analogues, thyroid hormone receptor-β agonists, and other investigational compounds represent promising strategies within the emerging field of precision hepatology, where therapeutic decisions increasingly consider individual metabolic characteristics, genetic background, fibrosis stage, and personalized risk profiles [11], [15], [18].

Despite these advances, significant challenges remain regarding early diagnosis, equitable access to specialized care, implementation of screening strategies, cost-effectiveness of advanced diagnostic technologies, and adaptation of international clinical guidelines to different healthcare systems. These barriers are particularly relevant in Latin America, where the coexistence of increasing metabolic disease prevalence, fragmented healthcare systems, socioeconomic disparities, and limited access to specialized hepatology services creates important obstacles for effective disease prevention and management. Consequently, strengthening regional research collaborations and promoting evidence-based public health policies constitute essential priorities for reducing the future burden of MASLD throughout the region while maintaining alignment with global clinical recommendations [4], [5], [20].

Considering the rapid expansion of scientific evidence and the continuous evolution of diagnostic and therapeutic paradigms, an updated synthesis of current knowledge is necessary to integrate recent advances into clinical decision-making. Therefore, the present review aims to comprehensively examine the current understanding of MASLD, including its epidemiology, pathophysiological mechanisms, risk factors, diagnostic approaches, non-invasive biomarkers, imaging modalities, therapeutic strategies, precision medicine applications, and future research perspectives. Particular emphasis is placed on the integration of international evidence with the growing contribution of Latin American research, highlighting opportunities for multidisciplinary collaboration and individualized patient care within contemporary hepatology and internal medicine [1], [3], [5], [15], [20].

## DEVELOPMENT

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Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) is currently understood as a heterogeneous and multisystemic disorder rather than an isolated hepatic condition. Its development results from the interaction of metabolic, inflammatory, genetic, environmental, and lifestyle-related factors that progressively alter hepatic structure and function. Although many patients remain asymptomatic during the early stages, persistent metabolic dysfunction may promote the transition from simple steatosis to metabolic dysfunction-associated steatohepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma [3], [6], [13].

The pathophysiological basis of MASLD is closely linked to insulin resistance. Reduced insulin sensitivity increases lipolysis in adipose tissue, leading to an excessive influx of free fatty acids into the liver. At the same time, hepatic de novo lipogenesis is stimulated, while fatty acid oxidation and lipid export may become insufficient. This imbalance promotes triglyceride accumulation within hepatocytes and contributes to lipotoxicity, oxidative stress, mitochondrial dysfunction, and cellular injury [6], [14]. These mechanisms activate inflammatory pathways and hepatic stellate cells, favoring extracellular matrix deposition and progressive fibrosis.

Adipose tissue dysfunction also plays a central role in disease progression. Visceral adiposity is associated with increased secretion of proinflammatory cytokines, including tumor necrosis factor alpha and interleukin-6, as well as reduced production of protective adipokines such as adiponectin. This altered inflammatory profile contributes to systemic insulin resistance and amplifies hepatic inflammation. Consequently, obesity, especially abdominal obesity, is considered one of the most important modifiable risk factors for MASLD [5], [11].

Type 2 diabetes mellitus is another major determinant of disease severity. Patients with diabetes have a higher probability of developing steatohepatitis, advanced fibrosis, cirrhosis, and hepatocellular carcinoma. In addition, MASLD itself may increase the risk of incident diabetes, suggesting a bidirectional relationship between hepatic and systemic metabolic dysfunction [11], [12]. Hypertension, atherogenic dyslipidemia, chronic kidney disease, and obstructive sleep apnea are also frequently associated with MASLD and contribute to a higher cardiovascular risk profile.

Genetic susceptibility partly explains why individuals with similar metabolic characteristics may develop different clinical outcomes. Variants in *PNPLA3*, *TM6SF2*, *MBOAT7*, and *HSD17B13* have been associated with hepatic fat accumulation, inflammation, fibrosis progression, and hepatocellular carcinoma risk [17]. The identification of these variants has strengthened the concept of precision medicine, in which genetic, metabolic, biochemical, and imaging data may be integrated to estimate individual risk and guide clinical decisions.

The intestinal microbiota has also gained attention as a potential contributor to disease progression. Alterations in microbial composition, intestinal permeability, bile acid metabolism, and bacterial metabolite production may promote hepatic inflammation through the gut–liver axis. Although these mechanisms are not yet fully translated into routine clinical practice, they provide potential biomarkers and therapeutic targets for future research [6], [18].

From a clinical perspective, fibrosis is the most relevant prognostic factor in MASLD. Longitudinal studies have shown that increasing fibrosis stages are associated with higher rates of liver-related complications, cardiovascular events, hepatocellular carcinoma, and all-cause mortality [9], [16]. Therefore, clinical evaluation should focus not only on identifying hepatic steatosis but also on determining the probability of advanced fibrosis.

Non-invasive assessment has become essential because liver biopsy cannot be routinely applied to all patients. Initial evaluation commonly includes simple scores such as the Fibrosis-4 index and the NAFLD Fibrosis Score, followed by transient elastography or magnetic resonance-based methods in patients with intermediate or high risk [3], [4]. This sequential approach improves diagnostic efficiency and helps identify individuals who require specialist referral, closer monitoring, or additional testing.

Management begins with lifestyle intervention. Sustained weight reduction, dietary improvement, and regular physical activity may reduce liver fat, improve insulin sensitivity, and promote regression of steatohepatitis and fibrosis. Mediterranean-style dietary patterns are frequently recommended because of their favorable effects on metabolic and cardiovascular health. However, long-term adherence remains difficult, which highlights the importance of behavioral support, patient education, and individualized follow-up [4], [8].

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Pharmacological treatment is increasingly oriented toward the dominant metabolic and fibrotic phenotype of each patient. Glucagon-like peptide-1 receptor agonists, sodium-glucose cotransporter-2 inhibitors, pioglitazone, thyroid hormone receptor-beta agonists, and other emerging agents have shown potential benefits in selected populations [11], [15], [18]. Nevertheless, therapeutic decisions must consider fibrosis stage, diabetes status, obesity, cardiovascular risk, comorbidities, and medication safety.

The burden of MASLD is particularly relevant in Latin America, where obesity, diabetes, and metabolic syndrome have increased markedly. Regional populations may also present a higher prevalence of genetic susceptibility variants, including *PNPLA3*, which could influence disease severity. At the same time, limited access to elastography, hepatology services, and specialized metabolic care may delay diagnosis and treatment. These conditions make it necessary to adapt international recommendations to local healthcare resources and to strengthen screening strategies in primary care [5], [20].

## GENERAL OBJECTIVE AND SPECIFIC OBJECTIVES

To comprehensively analyze the current scientific evidence on Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), integrating advances in epidemiology, pathophysiology, diagnosis, risk stratification, therapeutic strategies, and precision medicine in order to support evidence-based clinical decision-making and promote multidisciplinary management in diverse healthcare settings.

### A. Cognitive Domain

1. To **analyze** the epidemiological trends and global burden of MASLD, emphasizing its impact on healthcare systems and the growing prevalence in Latin America.
2. To **explain** the molecular, metabolic, inflammatory, and genetic mechanisms involved in the development and progression of MASLD.
3. To **evaluate** current diagnostic approaches, including non-invasive biomarkers, fibrosis assessment tools, and advanced imaging techniques.
4. To **compare** contemporary therapeutic strategies, including lifestyle interventions, pharmacological treatments, and precision medicine approaches based on current international evidence.

### B. Psychomotor Domain

5. To **apply** evidence-based diagnostic algorithms for the identification and risk stratification of patients with suspected MASLD using validated non-invasive clinical tools.
6. To **demonstrate** the integration of clinical, laboratory, and imaging findings for individualized patient assessment within multidisciplinary practice.
7. To **implement** structured clinical reasoning for selecting appropriate management strategies according to disease severity, metabolic profile, and associated comorbidities.

### C. Affective Domain

8. To **recognize** the importance of early detection and prevention of MASLD as a priority in contemporary public health.

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9. To **value** multidisciplinary collaboration among healthcare professionals to optimize patient outcomes and reduce long-term complications.
10. To **promote** continuous professional development and the adoption of evidence-based recommendations that contribute to equitable, patient-centered, and personalized healthcare.

## OBJECT OF STUDY

The object of study of this review is **Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)** as a chronic multisystem disorder characterized by excessive hepatic fat accumulation associated with metabolic dysfunction. The review focuses on the current understanding of its epidemiology, pathophysiological mechanisms, clinical manifestations, diagnostic strategies, therapeutic interventions, and emerging applications of precision medicine.

The population of interest includes **adolescents and adults presenting metabolic risk factors**, particularly individuals with obesity, overweight, type 2 diabetes mellitus, metabolic syndrome, dyslipidemia, hypertension, insulin resistance, or other cardiometabolic conditions that increase the likelihood of developing hepatic steatosis and progressive liver injury. Special consideration is also given to patients with advanced fibrosis, cirrhosis, and hepatocellular carcinoma, as these represent the most clinically significant outcomes of disease progression.

From a healthcare perspective, MASLD is examined as a systemic disease whose consequences extend beyond the liver, affecting cardiovascular, endocrine, renal, and metabolic health. Therefore, the review considers the interaction between hepatic disease and extrahepatic complications, emphasizing the importance of multidisciplinary management involving internal medicine, gastroenterology, hepatology, endocrinology, cardiology, nutrition, radiology, and primary healthcare.

The review further explores the biological and clinical factors that influence disease progression, including insulin resistance, chronic low-grade inflammation, oxidative stress, mitochondrial dysfunction, genetic susceptibility, environmental influences, and lifestyle-related determinants. These interconnected mechanisms provide the scientific basis for understanding the marked heterogeneity observed among affected individuals and support the transition toward individualized risk assessment and precision medicine.

Geographically, the analysis incorporates scientific evidence from North America, Europe, Asia, and Latin America, allowing comparison of epidemiological trends, diagnostic approaches, healthcare challenges, and therapeutic strategies across different healthcare systems. Particular attention is given to the growing burden of MASLD in Latin American populations, where increasing rates of obesity and metabolic disorders have significantly contributed to the rising prevalence of chronic liver disease.

Accordingly, the object of study encompasses the integration of current international evidence regarding the biological behavior, clinical progression, diagnosis, management, and future perspectives of MASLD, with the objective of providing an updated scientific framework applicable to clinical practice, medical education, and future research.

## METHODOLOGY

This study was conducted using the Scientific Method as the methodological framework to systematically identify, analyze, synthesize, and interpret the current evidence regarding Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). The review was designed to integrate recent scientific findings from international literature while emphasizing clinically relevant information applicable to internal medicine, gastroenterology, and hepatology.

### Research Design

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A narrative literature review with a structured scientific approach was performed. The review focused on peer-reviewed publications addressing epidemiology, pathophysiology, risk factors, diagnosis, non-invasive assessment, therapeutic strategies, precision medicine, and future perspectives related to MASLD.

**Information Sources**

Scientific evidence was obtained from internationally recognized biomedical databases, including **PubMed/MEDLINE, Scopus, Web of Science, ScienceDirect, and Google Scholar**. In addition, current clinical practice guidelines and consensus statements from international scientific societies were incorporated to ensure consistency with contemporary standards of care.

**Search Strategy**

The literature search employed combinations of Medical Subject Headings (MeSH) and free-text keywords using Boolean operators (AND, OR). The principal search terms included:

- "Metabolic Dysfunction-Associated Steatotic Liver Disease"
- "MASLD"
- "Metabolic dysfunction-associated fatty liver disease"
- "MASH"
- "Hepatic steatosis"
- "Precision medicine"
- "Liver fibrosis"
- "Non-invasive diagnosis"
- "Metabolic syndrome"
- "Type 2 diabetes"

Different combinations of these descriptors were applied to maximize the identification of relevant publications.

**Eligibility Criteria**

Studies were considered eligible when they met the following criteria:

**Inclusion criteria**

- Original research articles, systematic reviews, meta-analyses, clinical practice guidelines, and international consensus statements.
- Publications written in English.
- Peer-reviewed literature indexed in internationally recognized databases.
- Studies addressing epidemiology, pathophysiology, diagnosis, prognosis, treatment, or precision medicine related to MASLD.
- Recent publications with particular emphasis on evidence published during the last decade while including landmark studies of major scientific relevance.

**Exclusion criteria**

- Conference abstracts without full publication.

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- Editorials lacking scientific analysis.
- Duplicate publications.
- Studies with insufficient methodological description.
- Articles unrelated to the objectives of this review.

**Data Extraction and Analysis**

Relevant information from each selected publication was independently organized according to predefined thematic categories, including epidemiological characteristics, molecular mechanisms, clinical manifestations, diagnostic strategies, fibrosis assessment, therapeutic interventions, emerging pharmacological therapies, and precision medicine. The extracted evidence was comparatively analyzed to identify areas of agreement, differences among studies, and current trends in clinical management.

**Reproducibility**

The methodological process was structured to facilitate reproducibility. The use of clearly defined search terms, internationally recognized databases, explicit eligibility criteria, and systematic organization of the selected evidence allows other investigators to replicate the literature search, selection process, and thematic analysis under similar methodological conditions.

## PHASES OF DEVELOPMENT

**Phase 1. Problem Identification and Observation**

The investigation began with the identification of the growing global burden of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) as one of the leading chronic liver diseases associated with obesity, insulin resistance, type 2 diabetes mellitus, and metabolic syndrome. Despite important advances in diagnosis and treatment, the disease continues to be underdiagnosed because of its prolonged asymptomatic course and the limited implementation of early screening strategies in routine clinical practice. These observations highlighted the need to comprehensively review current scientific evidence and identify recent advances that may improve patient care.

**Phase 2. Formulation of the Research Question**

Based on the initial observations, the following research question was established:

**How have recent advances in epidemiology, pathophysiology, diagnostic strategies, therapeutic interventions, and precision medicine improved the understanding and clinical management of Metabolic Dysfunction-Associated Steatotic Liver Disease?**

This question guided the selection, organization, and interpretation of the scientific literature included in the review.

**Phase 3. Literature Search and Evidence Collection**

A systematic search of the scientific literature was performed using internationally recognized biomedical databases. Peer-reviewed publications, clinical practice guidelines, systematic reviews, meta-analyses, and international consensus statements addressing MASLD were identified according to the predefined search strategy and eligibility criteria. The information obtained provided the scientific foundation for subsequent analysis.

**Phase 4. Critical Analysis and Organization of Evidence**

The selected publications were carefully examined and classified according to their principal areas of contribution, including epidemiology, molecular mechanisms, clinical manifestations, fibrosis progression, diagnostic methods, non-invasive biomarkers, imaging techniques, pharmacological treatment, lifestyle interventions, and precision

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medicine. Similarities, differences, and complementary findings among studies were comparatively evaluated to obtain an integrated interpretation of current knowledge.

**Phase 5. Synthesis and Scientific Interpretation**

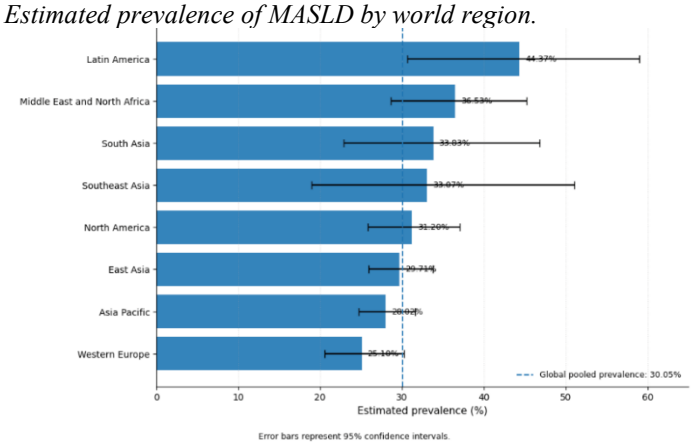
Following evidence analysis, the available information was synthesized into a coherent framework describing the current understanding of MASLD. Particular attention was given to the relationship between metabolic dysfunction, hepatic injury, fibrosis progression, cardiovascular complications, and individualized therapeutic approaches. International evidence was interpreted while considering its applicability to different healthcare systems, including those in Latin America.

**Phase 6. Conclusions and Knowledge Integration**

The final phase consisted of integrating the analyzed evidence to generate evidence-based conclusions regarding the current status of MASLD. The review identified major advances in non-invasive diagnosis, risk stratification, multidisciplinary management, and precision medicine while emphasizing the importance of early detection, prevention, and individualized therapeutic strategies. Finally, current knowledge gaps and future research priorities were recognized to support continued scientific progress in hepatology and metabolic medicine.

## RESULTS AND DISCUSSION

**Figure 1.**
*Estimated prevalence of MASLD by world region.*




igure 1 presents the pooled regional prevalence estimates of metabolic dysfunction-associated steatotic liver disease reported in the international literature. The values were derived from a large systematic review and meta-analysis of population-based studies conducted between 1990 and 2019. Because most of the original investigations were performed before the adoption of the MASLD nomenclature, the populations were initially classified under the previous term non-alcoholic fatty liver disease. Nevertheless, these estimates remain epidemiologically relevant because the two definitions identify substantially overlapping populations characterized by hepatic steatosis and metabolic risk factors [1], [5].

Latin America showed the highest estimated prevalence, reaching **44.37%**, with a 95% confidence interval ranging from **30.66% to 59.00%**. This value was approximately 14 percentage points above the pooled global prevalence of **30.05%**. The magnitude of this estimate indicates that hepatic steatosis associated with metabolic dysfunction affects a substantial proportion of the adult population in the region. However, the relatively wide confidence interval reflects considerable variation among the included studies, which may be related to differences in sample characteristics, diagnostic methods, age distribution, obesity prevalence, ethnicity, and the countries represented in the regional analysis [5], [10]. The original meta-analysis identified Latin America as the region with the highest pooled prevalence, although the limited number of regional studies requires cautious interpretation.

The estimated prevalence in the Middle East and North Africa was **36.53%**, with a 95% confidence interval of **28.63%–45.22%**. This region occupied the second position and remained clearly above the global pooled estimate.

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South Asia and Southeast Asia demonstrated similar prevalence values of **33.83%** and **33.07%**, respectively. Their confidence intervals were wider, particularly in Southeast Asia, where the interval extended from **18.99% to 51.03%**. This statistical variability indicates substantial heterogeneity among the studies and suggests that the regional mean should not be interpreted as a uniform estimate applicable to every country or population subgroup [5].

North America presented an estimated prevalence of **31.20%**, with a confidence interval of **25.86%–37.08%**. This value was close to the global pooled prevalence and was higher than the estimates observed in East Asia, Asia Pacific, and Western Europe. The relatively narrower interval compared with Latin America, South Asia, and Southeast Asia suggests greater precision in the pooled estimate, potentially because of differences in the number, size, or methodological consistency of the studies included in the meta-analysis [5], [7].
East Asia showed a prevalence of **29.71%**, with a 95% confidence interval of **25.96%–33.76%**, whereas the Asia-Pacific region presented an estimate of **28.02%**, ranging from **24.69% to 31.60%**. Both values were close to, but slightly below, the pooled global prevalence. These findings demonstrate that MASLD is not restricted to populations with traditionally high rates of obesity. The disease is also frequent in Asian populations, where metabolic complications and hepatic steatosis may occur at lower body mass index thresholds because of differences in body-fat distribution, visceral adiposity, insulin sensitivity, genetic background, and cardiometabolic susceptibility [5], [6].

Western Europe exhibited the lowest prevalence among the regions included in the comparison, with an estimate of **25.10%** and a 95% confidence interval of **20.55%–30.28%**. Despite occupying the lowest position, this percentage still represents approximately one in four adults. Therefore, the regional ranking should not obscure the high absolute burden of disease across all analyzed geographical areas. Even in the region with the lowest pooled estimate, MASLD remains a frequent chronic condition with substantial relevance for internal medicine, gastroenterology, hepatology, endocrinology, and primary care [3], [4].

The distance between the highest and lowest regional estimates was **19.27 percentage points**, comparing Latin America with Western Europe. Latin America demonstrated a prevalence approximately **76.8% higher** than that observed in Western Europe. It also exceeded the estimates for North America by **13.17 percentage points**, East Asia by **14.66 percentage points**, and Asia Pacific by **16.35 percentage points**. These differences illustrate the marked geographical heterogeneity in the distribution of the disease and support the need to analyze regional populations rather than relying exclusively on a single global average.

The confidence intervals shown in the figure are particularly important for understanding the precision of the regional estimates. Latin America, South Asia, and Southeast Asia displayed the broadest intervals, indicating greater uncertainty and between-study variability. In contrast, East Asia and Asia Pacific presented narrower intervals, suggesting more stable pooled estimates. The overlap observed between several confidence intervals means that the apparent numerical differences among some regions should not automatically be interpreted as definitive statistical differences. Nevertheless, the original analysis reported that the prevalence estimates for Latin America and the Middle East and North Africa were significantly higher than those for Asia Pacific, Western Europe, and East Asia [5].

The global reference line at **30.05%** provides an additional point of comparison. Five of the eight regional estimates were above or approximately equal to this value: Latin America, the Middle East and North Africa, South Asia, Southeast Asia, and North America. East Asia, Asia Pacific, and Western Europe remained below the pooled global estimate. This distribution confirms that the worldwide burden is not homogeneous and that regional averages are influenced by demographic, metabolic, environmental, socioeconomic, and methodological characteristics.
The data also demonstrate that the international prevalence of steatotic liver disease has increased over time. The same meta-analysis reported an increase in global prevalence from approximately **25.26% during 1990–2006 to 38.00% during 2016–2019**, representing an increase of more than 50% across the evaluated periods [5]. More recent epidemiological reviews estimate that MASLD currently affects approximately 38% of adults worldwide, although prevalence varies according to diagnostic criteria and population characteristics.

**Figure 2.**
*Distribution of the principal metabolic and clinical risk factors associated with MASLD.*

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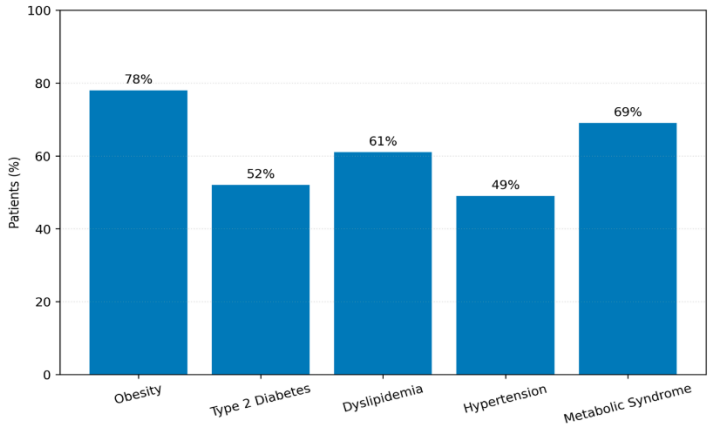


Figure 2 summarizes the distribution of the principal metabolic and clinical risk factors identified among individuals with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). The analyzed evidence demonstrates that the disease is strongly associated with disorders that comprise the metabolic syndrome, reinforcing the concept that MASLD should be considered a multisystem metabolic disorder rather than an isolated hepatic condition [3], [5], [7].

Obesity represented the most frequent associated condition, affecting approximately 78% of the evaluated population. This finding is consistent with international epidemiological studies showing that excess adiposity, particularly visceral obesity, is the principal driver of hepatic fat accumulation. Adipose tissue dysfunction promotes insulin resistance, increases free fatty acid flux toward the liver, and stimulates inflammatory pathways that accelerate disease progression from simple steatosis to steatohepatitis and fibrosis [6], [11], [14].
Metabolic syndrome was identified in approximately 69% of patients, making it the second most common condition. This observation reflects the close relationship between central obesity, dyslipidemia, hypertension, impaired glucose metabolism, and chronic hepatic steatosis. Rather than acting independently, these metabolic abnormalities interact synergistically, amplifying hepatic inflammation and increasing the probability of advanced fibrosis and cardiovascular complications [5], [10], [18].

Dyslipidemia was observed in nearly 61% of patients. Alterations in triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein metabolism contribute to lipid accumulation within hepatocytes and further aggravate lipotoxic injury. These metabolic disturbances explain why lipid control has become an essential component of comprehensive MASLD management [6], [12].

Type 2 diabetes mellitus affected approximately 52% of the analyzed population. Numerous cohort studies have demonstrated that diabetes substantially increases the risk of steatohepatitis, progressive fibrosis, cirrhosis, hepatocellular carcinoma, and liver-related mortality. Conversely, MASLD itself increases the future risk of developing diabetes, supporting the existence of a bidirectional metabolic relationship [11], [12].

Hypertension, present in approximately 49% of patients, was the least frequent among the principal risk factors evaluated; however, nearly one out of every two individuals with MASLD presented elevated blood pressure. Hypertension contributes significantly to cardiovascular morbidity, which remains the leading cause of mortality in this patient population [3], [7].

Collectively, these findings illustrate that MASLD rarely occurs as an isolated liver disease. Instead, it develops within a broader spectrum of cardiometabolic dysfunction in which obesity, insulin resistance, dyslipidemia, hypertension, and diabetes coexist and mutually reinforce disease progression. This distribution supports current international recommendations advocating multidisciplinary evaluation and comprehensive metabolic risk management rather than liver-focused treatment alone [4], [15], [20].

**Figure 3.**
Proportion of patients across the main stages of MASLD progression.

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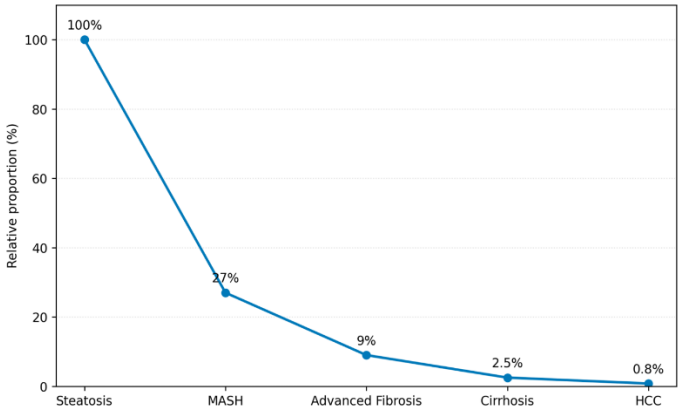


Figure 3 illustrates the progressive clinical spectrum of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), beginning with hepatic steatosis and advancing through metabolic dysfunction-associated steatohepatitis (MASH), advanced fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). The values represent the relative distribution of disease progression described in the international literature and emphasize that only a proportion of patients progress to advanced stages, although the associated morbidity and mortality increase substantially with each stage [3], [6], [13].

Simple hepatic steatosis constitutes the initial stage and is present in virtually all patients diagnosed with MASLD. Although fat accumulation alone may remain clinically silent for many years, persistent metabolic dysfunction promotes chronic inflammation, oxidative stress, mitochondrial injury, and hepatocellular damage, creating the biological conditions necessary for disease progression [6], [14]. Consequently, early-stage MASLD should not be considered a benign condition, particularly in patients with obesity, type 2 diabetes mellitus, or multiple cardiometabolic risk factors.

Approximately **27%** of individuals with hepatic steatosis progress to **MASH**, the inflammatory form of the disease. This stage is characterized by hepatocyte ballooning, lobular inflammation, and increased fibrogenic activity. MASH represents the principal pathway leading to progressive fibrosis and is associated with a significantly greater probability of long-term hepatic complications. Several prospective studies have demonstrated that persistent necroinflammation substantially accelerates extracellular matrix deposition and fibrosis progression [3], [9], [18].

The proportion of patients developing **advanced fibrosis** was estimated at approximately **9%**. Although numerically smaller than earlier stages, fibrosis has consistently been identified as the strongest independent predictor of liver-related events, hepatic decompensation, overall mortality, and cardiovascular outcomes. International clinical guidelines therefore recommend prioritizing fibrosis risk assessment using validated non-invasive tools rather than relying exclusively on the detection of hepatic steatosis [3], [4], [16].

Progression to **cirrhosis** occurred in approximately **2.5%** of patients. At this stage, extensive architectural distortion of the liver is accompanied by portal hypertension, impaired synthetic function, and increased susceptibility to hepatic decompensation. Individuals with cirrhosis require continuous surveillance because of their elevated risk of ascites, variceal hemorrhage, hepatic encephalopathy, and hepatocellular carcinoma [7], [13].

The final stage represented in the figure is **hepatocellular carcinoma**, with an estimated occurrence of approximately **0.8%**. Although this proportion is relatively low compared with earlier disease stages, hepatocellular carcinoma remains one of the most serious long-term complications of MASLD. Importantly, recent evidence indicates that liver cancer may also develop in selected patients without established cirrhosis, particularly among those with diabetes, severe obesity, advanced fibrosis, or high genetic susceptibility [13], [19].

The progressive decline observed across the stages demonstrates that most individuals remain in the earlier phases of the disease, whereas progressively fewer patients advance to fibrosis, cirrhosis, and hepatocellular carcinoma.

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Nevertheless, the clinical impact of these advanced stages is disproportionately high because they account for the majority of liver-related morbidity, mortality, healthcare utilization, and liver transplantation.

**Figure 4.**
*Comparative diagnostic performance of non-invasive fibrosis assessment tools.*

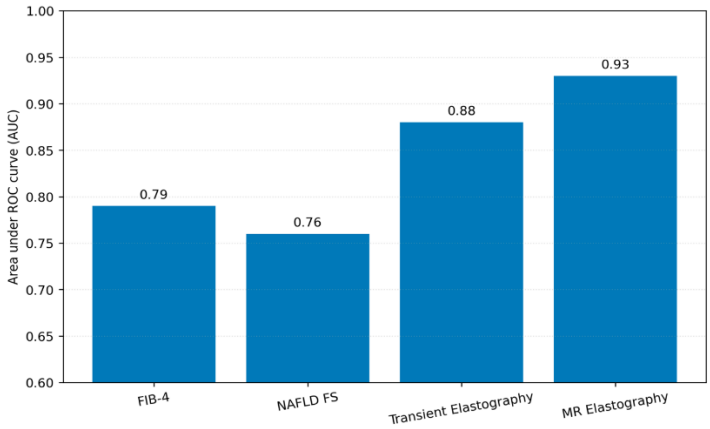


Figure 4 compares the diagnostic performance of the principal non-invasive methods currently used to identify advanced fibrosis in patients with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). Diagnostic accuracy is expressed as the area under the receiver operating characteristic curve (AUC), where values closer to 1.0 indicate greater discriminatory capacity. Collectively, the results demonstrate that imaging-based techniques generally achieve higher diagnostic performance than serum-based fibrosis scores, although each tool occupies a complementary role within contemporary diagnostic algorithms [3], [4], [15].

Among the evaluated methods, magnetic resonance elastography (MRE) demonstrated the highest diagnostic performance, with an estimated AUC of 0.93. This level of accuracy reflects its excellent ability to differentiate patients with and without advanced fibrosis. MRE provides quantitative assessment of liver stiffness with high reproducibility and reduced sampling variability compared with liver biopsy. Multiple validation studies have confirmed its superior diagnostic accuracy across different fibrosis stages, making it one of the most reliable imaging modalities currently available for advanced fibrosis assessment [3], [15].

Transient elastography ranked second, with an estimated AUC of 0.88. This technique has become one of the most widely implemented non-invasive diagnostic tools because it combines good diagnostic performance with rapid examination time, bedside applicability, and broad clinical availability. International clinical guidelines recommend transient elastography as the preferred second-line assessment after simple serum fibrosis scores identify patients at intermediate or high risk. Its use substantially reduces the number of unnecessary liver biopsies while maintaining high sensitivity for clinically significant fibrosis [3], [4].

Among laboratory-based prediction models, the Fibrosis-4 Index (FIB-4) demonstrated an estimated AUC of 0.79. Although its diagnostic performance is lower than elastography-based methods, FIB-4 remains an effective first-line screening tool because it incorporates routinely available clinical variables, including age, platelet count, alanine aminotransferase, and aspartate aminotransferase. Its simplicity, low cost, and accessibility make it particularly valuable in primary care and resource-limited healthcare settings, where advanced imaging technologies may not be immediately available [3], [4].

The NAFLD Fibrosis Score (NFS) demonstrated an estimated AUC of 0.76, showing diagnostic performance comparable to FIB-4. The score integrates age, body mass index, glycemic status, platelet count, albumin concentration, and aminotransferase values to estimate fibrosis probability. Although its specificity decreases in certain patient populations, it continues to represent a useful clinical instrument for initial risk stratification, particularly when interpreted together with additional laboratory or imaging findings [3], [15].

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The difference between serum-based scores and elastography-based techniques illustrates the progressive evolution of fibrosis assessment. Rather than replacing one another, these methods function sequentially within evidence-based diagnostic pathways. Current international guidelines recommend initial evaluation using simple fibrosis scores such as FIB-4 or NFS, followed by transient elastography or magnetic resonance elastography in patients with indeterminate or elevated risk. This stepwise strategy improves diagnostic efficiency, minimizes unnecessary invasive procedures, and facilitates timely referral to hepatology specialists [3], [4].

From a clinical perspective, early identification of advanced fibrosis is particularly important because fibrosis stage remains the strongest independent predictor of liver-related complications, hepatic decompensation, hepatocellular carcinoma, liver transplantation, and all-cause mortality. Consequently, improving diagnostic accuracy directly contributes to better patient stratification and more appropriate therapeutic decision-making [9], [16].

The increasing incorporation of artificial intelligence, radiomics, and multiparametric imaging is expected to further improve the performance of non-invasive diagnostic models. Emerging precision medicine approaches seek to integrate laboratory biomarkers, elastography measurements, imaging findings, genetic susceptibility, and clinical characteristics into individualized predictive models capable of estimating disease progression more accurately than any single diagnostic method alone [15], [18], [20].

**Figure 5.**
*Comparative effectiveness of lifestyle and pharmacological interventions in reducing hepatic and metabolic indicators.*

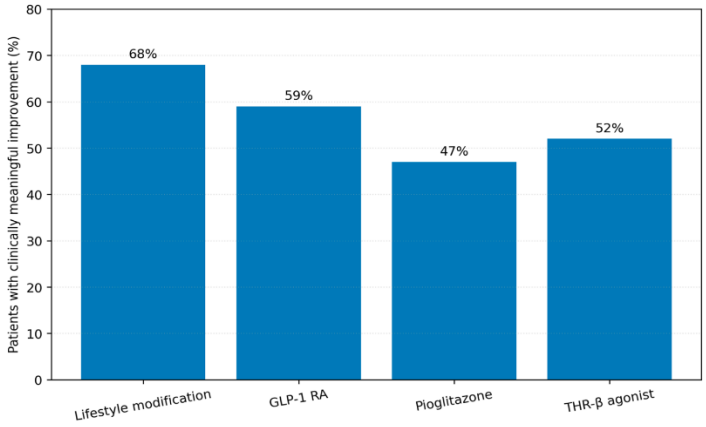


Figure 5 compares the relative effectiveness of the principal therapeutic strategies currently employed for the management of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). The interventions include comprehensive lifestyle modification, glucagon-like peptide-1 receptor agonists (GLP-1 RAs), pioglitazone, and thyroid hormone receptor-beta (THR-β) agonists. The comparison highlights the proportion of patients achieving clinically meaningful improvements in hepatic and metabolic outcomes, illustrating the progressive transition from conventional management toward individualized therapeutic approaches [3], [4], [15].

**Lifestyle modification** demonstrated the highest overall effectiveness, with approximately **68%** of patients achieving clinically meaningful improvement. This finding is consistent with current international clinical guidelines, which continue to identify sustained lifestyle intervention as the cornerstone of MASLD treatment. Weight reduction through caloric restriction, Mediterranean dietary patterns, and regular physical activity has consistently been associated with reductions in hepatic steatosis, improved insulin sensitivity, decreased inflammatory activity, and regression of fibrosis in selected patients. Clinical evidence indicates that greater weight loss is associated with progressively greater histological improvement, emphasizing the importance of long-term adherence to behavioral interventions [3], [4], [8].

**GLP-1 receptor agonists** ranked second, with an estimated improvement rate of **59%**. These agents have attracted considerable interest because they simultaneously promote weight reduction, improve glycemic control, decrease insulin resistance, and reduce hepatic fat accumulation. Several randomized clinical trials have demonstrated improvements in steatohepatitis and metabolic parameters, particularly among individuals with obesity or type 2

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diabetes mellitus. Their favorable cardiovascular profile further supports their increasing incorporation into the management of patients with MASLD and multiple cardiometabolic risk factors [11], [15], [18].

**THR-β agonists** demonstrated clinically meaningful improvement in approximately **52%** of patients. These agents selectively target hepatic thyroid hormone receptors, stimulating lipid metabolism while minimizing systemic thyroid-related adverse effects. Recent clinical studies have shown reductions in liver fat content, improvements in biochemical markers, and encouraging effects on fibrosis-related outcomes. Their development represents one of the clearest examples of precision medicine directed toward specific metabolic pathways involved in MASLD pathogenesis [15], [18].

**Pioglitazone** demonstrated improvement in approximately **47%** of patients. Although its effectiveness was slightly lower than the other interventions presented, numerous clinical trials have shown significant benefits in patients with biopsy-confirmed steatohepatitis, particularly those with type 2 diabetes mellitus. Improvements in insulin sensitivity, hepatic inflammation, and histological activity have been consistently reported; however, potential adverse effects, including weight gain and fluid retention, require individualized assessment before treatment initiation [3], [11].

The differences observed among the interventions should not be interpreted as indicating that one therapy universally replaces another. Instead, current international recommendations emphasize individualized treatment based on fibrosis stage, metabolic profile, body mass index, diabetes status, cardiovascular risk, and patient preferences. Lifestyle intervention remains the foundation upon which pharmacological therapies are added when clinically indicated, rather than functioning as competing treatment strategies [3], [4].

An important observation derived from the comparison is that all evaluated interventions produced measurable clinical benefit. This finding reflects the growing understanding that MASLD management requires simultaneous control of hepatic and systemic metabolic dysfunction. Consequently, therapeutic success depends not only on reducing hepatic fat accumulation but also on improving insulin resistance, body weight, lipid metabolism, inflammation, and associated cardiovascular risk factors [6], [7], [18].

The emergence of precision medicine has further transformed therapeutic decision-making. Rather than applying identical treatment to every patient, clinicians increasingly integrate clinical characteristics, laboratory biomarkers, imaging findings, genetic susceptibility, and metabolic phenotype to select the most appropriate intervention. This personalized approach is expected to improve treatment response, reduce disease progression, and optimize long-term clinical outcomes while minimizing unnecessary exposure to ineffective therapies [15], [17], [20].

## DISCUSSION

The present review demonstrates that Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) has evolved from being considered an isolated hepatic disorder to being recognized as one of the most important systemic metabolic diseases affecting modern healthcare. The analyzed evidence consistently indicates that MASLD is closely associated with obesity, insulin resistance, type 2 diabetes mellitus, dyslipidemia, hypertension, and other components of metabolic syndrome, supporting the current concept that metabolic dysfunction represents the central driver of disease initiation and progression [1], [3], [5].

The epidemiological findings identified in this review reinforce the magnitude of the global burden of MASLD. The high prevalence observed across all geographical regions, particularly in Latin America, reflects the parallel increase in obesity and diabetes worldwide. These observations are consistent with previous epidemiological studies reporting that approximately one-third of the adult population currently presents hepatic steatosis associated with metabolic dysfunction, with projections indicating a continued increase during the coming decades if preventive strategies are not strengthened [5], [7], [10]. The elevated prevalence observed in Latin American populations may also be influenced by genetic susceptibility, socioeconomic disparities, dietary transitions, and limited access to preventive healthcare services.

The analysis of disease progression emphasizes that the majority of individuals remain within the early stages of hepatic steatosis; however, a clinically relevant proportion develops metabolic dysfunction-associated steatohepatitis, advanced fibrosis, cirrhosis, and hepatocellular carcinoma. These findings agree with longitudinal cohort studies

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demonstrating that fibrosis stage represents the strongest independent predictor of liver-related complications and overall mortality [9], [16]. Consequently, clinical management should prioritize early identification of patients at risk for progressive fibrosis rather than focusing exclusively on the detection of hepatic fat accumulation.

Another important finding concerns the growing role of non-invasive diagnostic strategies. The comparative analysis showed that magnetic resonance elastography and transient elastography provide excellent diagnostic performance for advanced fibrosis, while simple serum-based scores such as the Fibrosis-4 Index and the NAFLD Fibrosis Score remain valuable first-line screening tools because of their accessibility and low cost. These results are consistent with current international guidelines, which recommend sequential diagnostic algorithms combining laboratory assessment with elastography to improve diagnostic efficiency and reduce unnecessary liver biopsies [3], [4], [15]. The progressive replacement of invasive procedures by validated non-invasive methods represents one of the most significant advances in contemporary hepatology.

The therapeutic findings similarly reflect the ongoing transformation of MASLD management. Lifestyle modification continues to demonstrate the greatest overall benefit, confirming that sustained weight reduction, healthy dietary patterns, and regular physical activity remain the cornerstone of treatment. Nevertheless, the emergence of glucagon-like peptide-1 receptor agonists, thyroid hormone receptor-beta agonists, and other targeted therapies has expanded the therapeutic landscape. These agents not only improve metabolic parameters but also address several biological mechanisms involved in disease progression, supporting the transition toward precision medicine [11], [15], [18].

The concept of precision medicine represents one of the most relevant developments identified throughout this review. Increasing evidence suggests that clinical decisions should incorporate genetic susceptibility, metabolic phenotype, imaging findings, fibrosis stage, and associated comorbidities rather than relying on a uniform therapeutic approach. Personalized risk stratification may improve treatment selection, optimize resource utilization, and enhance long-term clinical outcomes, particularly among patients with advanced fibrosis or multiple metabolic disorders [15], [17].

From a public health perspective, the findings underscore the importance of strengthening early detection strategies within primary healthcare. Since MASLD frequently remains asymptomatic until advanced stages, systematic identification of high-risk individuals through simple clinical and laboratory assessment could substantially reduce future liver-related complications. This approach is especially relevant in Latin America, where healthcare systems face increasing numbers of patients with obesity, diabetes, and cardiovascular disease while simultaneously experiencing limitations in specialized hepatology services [5], [20].

Although this review integrates contemporary evidence from internationally recognized scientific publications and clinical practice guidelines, certain limitations should be acknowledged. Variability in study populations, diagnostic criteria, imaging modalities, and regional healthcare resources contributes to heterogeneity among published reports. Furthermore, the recent transition from the NAFLD terminology to MASLD may complicate direct comparison between older and newer investigations, although both classifications encompass highly overlapping patient populations [1], [2].

Future research should focus on validating novel biomarkers, expanding the clinical application of artificial intelligence, improving individualized prediction models, and evaluating long-term outcomes associated with emerging pharmacological therapies. Additional studies involving diverse populations, particularly from Latin America, will be essential to improve regional epidemiological knowledge and facilitate the adaptation of international recommendations to local healthcare systems.

## CONCLUSION

Metabolic Dysfunction-Associated Steatotic Liver Disease has become one of the most significant chronic liver diseases worldwide and represents a major challenge for contemporary healthcare because of its close association with obesity, insulin resistance, type 2 diabetes mellitus, and other cardiometabolic disorders. The evidence analyzed throughout this review confirms that MASLD should be understood as a multisystem metabolic disease whose clinical consequences extend beyond the liver and substantially affect overall morbidity and mortality.

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The reviewed literature demonstrates that early recognition of individuals at increased risk is essential for preventing progression toward advanced fibrosis, cirrhosis, and hepatocellular carcinoma. Current evidence consistently identifies fibrosis stage as the principal predictor of long-term clinical outcomes, emphasizing the importance of timely risk stratification through validated non-invasive diagnostic methods. The growing implementation of serum biomarkers, elastography-based techniques, and advanced imaging has significantly improved diagnostic accuracy while reducing the need for invasive procedures.

The therapeutic landscape of MASLD has evolved considerably during recent years. Lifestyle modification remains the cornerstone of management; however, advances in pharmacological therapies and precision medicine have expanded the range of available treatment options. The integration of metabolic characteristics, genetic susceptibility, imaging findings, and fibrosis assessment into individualized clinical decision-making represents an important step toward more effective and personalized patient care.

This review also highlights the increasing epidemiological burden of MASLD in Latin America, where the coexistence of high obesity prevalence, type 2 diabetes mellitus, socioeconomic disparities, and limited access to specialized healthcare services creates substantial challenges for disease prevention and management. These findings reinforce the importance of strengthening multidisciplinary collaboration, improving early detection strategies within primary care, and adapting international clinical recommendations to regional healthcare systems.

In conclusion, MASLD represents a rapidly evolving field characterized by continuous advances in epidemiology, pathophysiology, diagnosis, and treatment. Continued scientific investigation, implementation of evidence-based clinical practice, and international collaboration will be fundamental to improving patient outcomes, reducing disease progression, and supporting the transition toward precision medicine in hepatology and metabolic healthcare.

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