

From NAFLD to MASLD – a new approach for the definition of fatty liver disease associated with metabolic dysfunction

Od NAFLD do MASLD – nowe podejście do definicji stłuszczeniowej choroby wątroby związanej z zaburzeniami metabolicznymi

Iwona Gorczyca-Głowacka , Łukasz Nawacki, Magdalena Dolecka-Ślusarczyk, Agnieszka Ciba-Stemplewska, Stanisław Głuszek

Collegium Medicum, Jan Kochanowski University, Kielce, Poland

Medical Studies/Studia Medyczne 2025; 41 (1): 45–52

DOI: <https://doi.org/10.5114/ms.2024.145773>

Key words: metabolic syndrome, obesity, diabetes mellitus, steatotic liver disease.

Słowa kluczowe: zespół metaboliczny, otyłość, cukrzyca, stłuszczenie wątroby.

Abstract

Metabolic dysfunction-associated steatotic liver disease (MASLD) is common, affecting a quarter of the population. The pathogenesis of this disease is multifactorial and has been well known for years. After 40 years of using the binding definition of non-alcoholic fatty liver disease (NAFLD), in 2020 new criteria for the diagnosis of metabolic dysfunction-associated fatty liver disease (MAFLD) were proposed, which significantly simplified the diagnosis of the disease, and in 2023 another division of steatotic liver disease (SLD) was proposed based on the aetiology of the disease. The diagnosis of MASLD is clinically important because the presence of this disease significantly influences the prognosis related not only to hepatic but, above all, cardiovascular events.

Streszczenie

Stłuszczeniowa choroba wątroby związana z zaburzeniami metabolicznymi (MASLD) jest częstym schorzeniem i dotyczy co czwartej osoby dorosłej. Patogeneza tej choroby jest wieloczynnikowa i od lat dobrze znana. Po 40 latach obowiązującej definicji niealkoholowego stłuszczenia wątroby (NAFLD) w 2020 roku zaproponowano nowe kryteria rozpoznania stłuszczeniowej choroby wątroby związanej z zaburzeniami metabolicznymi (MAFLD), które istotnie uprościły diagnozę tego schorzenia, a w 2023 roku zaproponowano kolejny podział stłuszczeniowej choroby wątroby (SLD) opierający się na etiologii schorzenia. Rozpoznanie MASLD jest klinicznie istotne, ponieważ obecność tej choroby istotnie wpływa na rokowanie związane ze zdarzeniami nie tylko wątrobowymi, lecz także i przede wszystkim sercowo-naczyniowymi.

Introduction

Metabolic dysfunction-associated fatty liver disease (MAFLD), formerly named non-alcoholic fatty liver disease (NAFLD) and now named metabolic dysfunction-associated steatotic liver disease (MASLD), is a common disease, especially in patients with cardiovascular and metabolic diseases [1]. Clinically, patients with MASLD tend to have components of metabolic syndrome such as obesity, type 2 diabetes mellitus (T2DM), hyperlipidaemia, and hypertension, so it has been referred to as the liver manifestation of metabolic syndrome [2, 3]. The global rise of this disease and its burden on health care outcomes has followed a worrisome and rapid increase in obesity and metabolic disorders [4, 5]. The pathogenesis of MASLD is complex; in addition to metabolic causes usually originating from insulin resistance, secondary causes such

as excessive alcohol consumption, many diseases, and steatogenic drug use may also play an important role in the development of hepatic steatosis [6–8].

Progress in comprehending the complex and multisystem pathophysiology of fatty liver disease has led to a consensus to adopt a more precise and suitable name. The investigation primarily focused on metabolic dysfunction, and as a result, a suggestion was put forth to name it MAFLD [9]. After significant discussions over the past few years, even considering maintaining the existing term due to its long-standing use in numerous studies that – there has been a concern – might become obsolete, the proposal to change the name from NAFLD to MASLD was introduced in a recent multi-society Delphi conference [10].

MASLD is a clinically heterogeneous condition. A smaller percentage of patients have signs of steatohepatitis (NASH), which frequently leads to progres-

sive fibrosis, cirrhosis, and hepatocellular carcinoma. However, hepatic complications among individuals with steatotic liver disease (SLD) are relatively uncommon, compared with cardiovascular disease – specific mortality [11]. Therefore, it is important to identify patients with SLD, especially with MASLD, and implement the procedure to reduce cardiovascular risk. In this study, the terminology of NAFLD and MAFLD is used to refer to the patient populations that were included in clinical trials, and the current name MASLD is used in general terms for the disease in question.

NAFLD/MAFLD epidemiology

The prevalence of MASLD in civilized countries is high and a constant increase in the number of cases is forecast due to the increasing percentage of diseases predisposing to the occurrence of MASLD, mainly obesity and T2DM. The incidence of MASLD varies and depends both on geographical factors and on the occurrence of diseases involved in the pathogenesis of MASLD – mainly components of the metabolic syndrome. In a meta-analysis of 86 studies from 22 countries, the global prevalence of NAFLD was 25.2%; the highest percentage was recorded in the Middle East (31.8%) and South America (30.4%), lower in Europe (23%), and the lowest in Africa (13.5%) [12].

A higher percentage of patients with MASLD than in the general population is observed in patients with overweight or obesity, T2DM, and cardiovascular diseases. Because the main underlying cause of MASLD is insulin resistance resulting from excessive body weight, this disease is observed in 51% of overweight or obese patients [13]. The correlation between MASLD and overweight/obesity is indisputable [14], but it should be remembered that MASLD may also occur in patients with normal body weight, as so-called lean MASLD. Chen *et al.* [12] showed that MAFLD in patients with underweight, normal weight, overweight, and obesity was present in 0.1%, 4%, 27.4%, and 59.8% of the subjects, respectively. MAFLD in patients with body mass index (BMI) < 25 kg/m² but not underweight (BMI < 18.5 kg/m²) was found in 7% of patients in the USA and in 19% of patients in Asia [15, 16]. The pathophysiology of liver steatosis in lean subjects is not clear, and beside metabolic factors, factors such as higher serum levels of secondary bile acids and fibroblast growth factor-19, as well as impairments in gut microbiota profile, have been related to development MASLD [17].

Patients with T2DM are at particular risk of developing MASLD, also due to the higher prevalence of overweight or obesity in this group, which potentiates the effects of factors predisposing to the development of MASLD. In the group of patients with T2DM, approximately 60% of patients are overweight [18]. Epidemiological data concerning NAFLD and T2DM coexistence are not consistent, with estimates ranging

from 30% to 80% of patients (mean: 55.5%). This discrepancy results from the tools used to diagnose fatty liver, difficulties in distinguishing between NAFLD and MAFLD definition, the age of the study population, BMI, and the duration of T2DM [19]. The relationship between T2DM and MASLD is bidirectional. It is very interesting, that there are numerous epidemiological data showing that NAFLD develops before T2DM. A meta-analysis conducted by Mantovani *et al.* [20] of more than 500 studies including more than a year of follow-up found that patients suffering from NAFLD recognized by ultrasonography are more than twice as likely to develop T2DM than those without NAFLD. They also showed that the more severe the steatosis and fibrosis, the higher the T2DM risk. MASLD is more common in elderly patients. In the *National Health and Nutrition Examination Survey* (NHANES), the incidence of MAFLD increased with age, from 23.2% in the 18–39 age group to 43.8% in the over 60 age group [21].

Clinical manifestation of metabolic dysfunction associated steatotic liver disease

MASLD usually has no specific symptoms and is diagnosed accidentally. In the retrospective RESTORE study, in which gastroenterologists and primary care physicians participated, it was shown that most patients with NAFLD presented abdominal symptoms, 64% of the subjects felt fatigue, and as many as 41% of patients reported sleep problems. Physicians declared that 10% of patients with NAFLD did not report any symptoms [22]. On the one hand, the symptomatology of MASLD is non-specific, which may delay diagnosis. On the other hand, the guidelines of scientific societies recommend searching for MASLD among patients with fatty liver with a specific clinical profile, and not among people presenting specific symptoms [23, 24].

However, it is important to note that MASLD is viewed as a gradually advancing condition that might not necessarily lead to advanced liver disease in all cases. Simple steatosis has a very good prognosis. It transitions from a state of steatosis without hepatitis to non-alcoholic steatohepatitis (NASH), which can ultimately progress to advanced fibrosis along with associated complications within and beyond the liver. The typical histological advancement from one fibrosis stage to another takes around 14.4 years in the case of MAFLD and approximately 7 years in NASH [25]. Research indicates that around 10–30% of MAFLD patients may advance to NASH, and 10–15% of those cases develop cirrhosis over a time span of 10–20 years [26]. About 10% of patients exhibit hidden cirrhosis through histological analysis. The likelihood of developing hepatocellular carcinoma is relatively low in MAFLD patients, at around 0.44 per 1000 person-years. However, among NASH individuals, the incidence of HCC escalates significantly, reaching

a tenfold increase with an estimated rate of 5.29 per 1000 person-years [27].

The prognosis in patients with MASLD is worse than in patients with proper liver function, which is mainly due to the increased incidence of cardiovascular events. Lauridsen *et al.* [28], in a study with a mean follow-up duration of 26.4 years, stated that patients with NAFLD presented higher mortality than patients from the general population (HR = 1.29; 95% CI: 1.04–1.59), and the authors identified cardiovascular disease as well as liver-related disease to be the main causes of death in patients with NAFLD. A more recent meta-analysis that included 108,711 patients with NAFLD (44% women and 56% men) showed that cardiovascular events and mortality were twice as high in women than in men (OR = 2.12, 95% CI: 1.65–2.73, $p < 0.001$) [29]. The high prevalence of cardiovascular events in patients with MASLD is a result of the complex correlation between hepatic steatosis and the classical and non-classical cardiovascular risk factors commonly observed in this particular population.

There are effective therapies available for the prevention of cardiovascular events in high-risk patients, and MASLD patients fall into this group. Therefore, it is necessary to know the current definitions and criteria for the diagnosis of MASLD to identify patients at risk of cardiovascular events when diagnosing liver disease and to effectively prevent them.

Definitions and criteria for the diagnosis of metabolic dysfunction-associated steatotic liver disease – from NAFLD to MAFLD

This chronological account follows the development from rudimentary explanations of fatty liver during the 1800s to our present-day comprehension of NAFLD in the 21st century. Fatty degeneration of the liver was first described in 1836 by Thomas Addison in patients who consumed excessive alcohol. Only in the late 1970s did people begin to recognize a comparable pathology in individuals with the metabolic syndrome who had minimal or no alcohol intake. While fatty liver had been linked to liver fibrosis and cirrhosis in the late 19th century, its connection to diabetes emerged in the early 1900s. Finally, the term NASH was proposed in 1980, and the term NAFLD in 1986 [30].

For defining NAFLD, 2 conditions must be met: evidence of hepatic steatosis, either by imaging or histology, and lack of secondary causes of hepatic fat accumulation [31]. Secondary causes of hepatic steatosis are as follows: excessive alcohol consumption, hepatitis C (genotype 3), lipodystrophy, acute weight loss (bariatric surgery and starvation), malnutrition, parenteral nutrition, abetalipoproteinaemia, Reye syndrome, acute fatty liver of pregnancy, medications (e.g. corticosteroids, lomitapide, mipomersen, amiodarone, methotrexate, tamoxifen, valproate, and antiretroviral medicines), autoimmune hepatitis,

A1AT deficiency, Wilson syndrome, and others [32]. NAFLD can be categorized histologically into non-alcoholic fatty liver (NAFL) or NASH. NAFL is defined as the presence of $\geq 5\%$ hepatic steatosis without evidence of hepatocellular injury in the form of hepatocyte ballooning. NASH is defined as the presence of $\geq 5\%$ hepatic steatosis and inflammation with hepatocyte injury (e.g. ballooning), with or without fibrosis [31]. However, there are certain constraints within this disease terminology and definition that have been acknowledged. One such limitation is the realization that fatty liver disease linked with the metabolic syndrome can potentially exist alongside conditions such as alcohol-related or other long-standing liver disorders. Furthermore, as the comprehension of the underlying mechanisms behind liver diseases driven by the metabolic syndrome has grown, there is a growing need to characterize a disease by its inherent qualities rather than defining it by what it lacks. Although it is known that NAFLD often coexists with obesity and T2DM, the clinical picture is not included in the definition, and this is its greatest weakness. It was also emphasized that the term NAFLD does not reflect the metabolic aetiology of the disease, and sheds undue light on an unrelated aetiology – alcohol. To overcome the negative connotation linked to this definition, various scientists collaborated to offer an upgrade in the nomenclature.

In 2020, 32 hepatologists from Europe, South America, North Africa, and the Asia-Pacific region published a consensus statement proposing to rename NAFLD as MAFLD, along with a new definition [9]. This new definition clearly establishes this disease as a metabolic disorder. As depicted in Figure 1, to meet the diagnosis of MAFLD, patients require the presence of hepatic steatosis (histology or imaging or blood markers/scores) accompanied by one of the following 3 features: overweight or obesity (cut-offs according to the ethnicity), T2DM, or signs of metabolic dysfunction defined as having 2 or more metabolic criteria [9]. The definition of MAFLD does not exclude the coexistence of secondary causes, such as alcohol consumption or viral hepatitis underlying steatosis, along with metabolic diseases. The semantic modification of NAFLD as MAFLD highlights the role of metabolic factors in the disease aetiology, which hopefully facilitates understanding of the disease.

Data from population studies suggest that most patients with fatty liver fulfil the definitions of both MAFLD and NAFLD, but up to 10–25% may only meet the criteria of one of the conditions [33, 34]. The percentage of patients with steatosis who have been shown to mismatch the definitions of NAFLD and MAFLD is evidence that the redefinition of “NAFLD” as “MAFLD” is more than a change of single letter. Data analysis from NHANES III showed that MAFLD patients compared with NAFLD were significantly older, had higher BMI, higher proportions of meta-

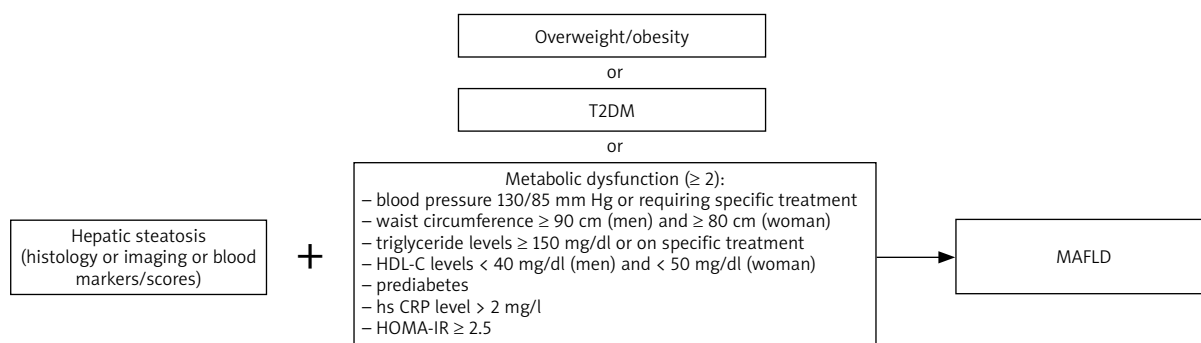


Figure 1. Diagnostic criteria for metabolic-associated fatty liver disease, based on Eslam 2020 [9]

HDL-C – high-density lipoprotein, HOMA-IR – homeostatic model assessment for insulin resistance, hs CRP – high-sensitivity C-reactive protein, MAFLD – metabolic dysfunction-associated fatty liver disease, T2DM – type 2 diabetes mellitus

bolic comorbidities (T2DM, hypertension), and higher HOMA-IR, lipid, and liver enzymes. MAFLD patients with alcohol consumption were younger than those without, and they more likely to be male. They had less metabolic disorder but higher liver enzyme levels. There were more cases with advance fibrosis in MAFLD patients with alcohol consumption. In conclusion, MAFLD definition is more practical than NAFLD for identifying patients with fatty liver disease with a high risk of disease progression [35]. In the Rotterdam Study not the NAFLD-only but also the MAFLD-only group was associated with fibrosis and higher liver stiffness, independent of demographic and lifestyle factors [36].

The implications of this updated classification are of great importance and will undoubtedly impact the well-being of individuals with metabolic dysfunction. Given the substantial overlap between the NAFLD and MAFLD groups, it is anticipated that individuals classified under MAFLD will possess essentially comparable cardiovascular risks to those categorized under NAFLD. Huang *et al.* [21], using data from the NHANES III database, were among the first to show that MAFLD was associated with a higher risk of cardiovascular mortality compared to NAFLD (HR = 2.01; 95% CI: 1.66–2.44 vs. HR = 1.53; 95% CI: 1.26–1.86, respectively), thus suggesting that the MAFLD definition may better identify patients with a high risk of cardiovascular outcomes. In line with this, Lee *et al.* [37] showed that individuals with MAFLD-only status were at higher risk of incident cardiovascular events (HR = 1.43; 95% CI: 1.41–1.45) compared with those without MAFLD or NAFLD, whereas the association between the NAFLD-only status and risk of cardiovascular outcomes was lower (HR = 1.09; 95% CI: 1.03–1.15). Similarly, a retrospective group of 2985 participants followed for 7 years showed that the MAFLD-only diagnosis was associated with a higher risk of cardiovascular events compared to the control group (HR = 7.2; 95% CI: 2.4–21.5), whilst the NAFLD-only status was not associated with cardiovascular events compared to the non-steatotic group

(HR = 1.90; 95% CI: 0.25–14.8) [38]. In another study the authors showed that patients with either MAFLD or NAFLD had a significantly greater proportion of high coronary artery calcification score, coronary artery disease, higher grade of coronary artery stenosis, and 10-year ASCVD risk than those without MAFLD or NAFLD. Importantly, participants who met the criteria for NAFLD alone were not observed to have increased coronary risk compared with those who met criteria for MAFLD. In conclusion, MAFLD is superior to NAFLD at identifying patients with hepatic steatosis, who are truly at increased risk of cardiovascular outcomes, and this fact was emphasized in an international multidisciplinary consensus statement on MAFLD and the risk of cardiovascular diseases [39].

The latest definition of metabolic dysfunction-associated steatotic liver disease – MASLD diagnosis criteria

Adopting a more contemporary terminology for characterizing fatty liver disease also presents its own constraints. Encompassing various aetiologies of persistent liver conditions within the scope of MAFLD is undoubtedly going to influence a patient's projected course. Assessing the influence of these diverse origins, like viral hepatitis or alcohol misuse disorder, on a patient's prognosis or their susceptibility to cardiovascular ailments could pose challenges. Additionally, as emphasized by numerous studies, MAFLD does not encompass all individuals with hepatic steatosis. Underestimation of the diagnosis of MAFLD may be significant in the group of patients with normal body weight, without T2DM. The definition of MAFLD in the subgroup of patients with metabolic dysregulations includes factors that are not standardized in practice, such as HOMA-IR or CRP level. Ordoñez-Vázquez *et al.* [40] compared the impact of both criteria (NAFLD vs. MAFLD) on the prevalence of the application in overweight and lean patients. This study provided interesting insights into the novel diag-

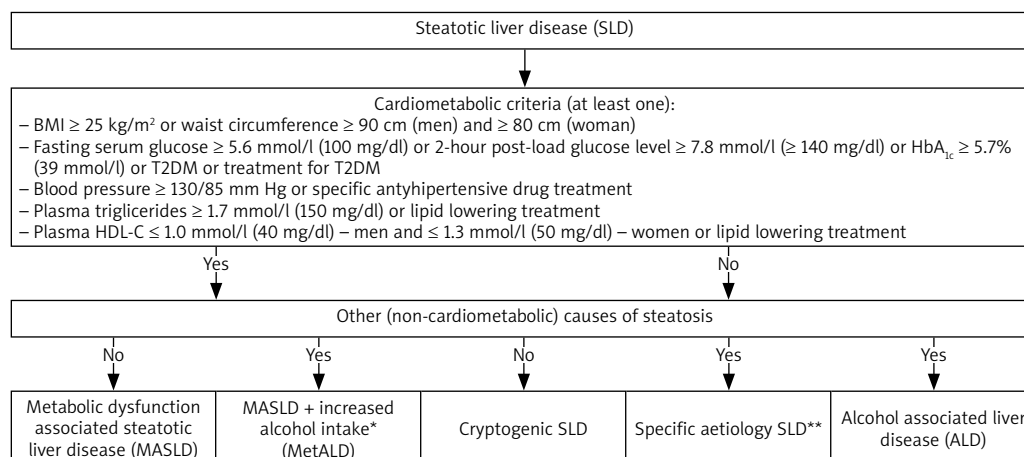


Figure 2. Diagnostic criteria of steatotic liver disease – based on Rinella 2023 [10]

*Weekly alcohol consumption greater than 140 g for women, 210 g for men, **secondary to applied medication, Wilson's disease, hypobetalipoproteinaemia, celiac disease, etc. ALD – alcohol-related liver disease, BMI – body mass index, HbA_{1c} – haemoglobin A_{1c}, HDL-C – high-density lipoprotein, HOMA-IR – homeostatic model assessment for insulin resistance, MASLD – metabolic dysfunction associated steatotic liver disease, MetALD – metabolic alcohol-related liver disease, NAFLD – nonalcoholic fatty liver disease, SLD – steatotic liver disease, T2DM – type 2 diabetes mellitus.

nostic criteria of MAFLD. A total of 3847 patients who were evaluated for liver steatosis by transient elastography were included and divided according to BMI (≤ 25 kg/m² and < 25 kg/m²) and classified as NAFLD or MAFLD according to metabolic abnormalities. The concordance of the diagnoses of MAFLD and NAFLD was high in the group of overweight patients. In this group (61%) the prevalence NAFLD was 63.6% and 65.3% for MAFLD ($p = 0.22$). In contrast, the prevalence of MAFLD was lower (7.9% vs. 18.3%, $p < 0.001$) in lean patients. In conclusion, with MAFLD criteria, the prevalence is lower in lean patients, but patients with high risk of progression of liver disease for steatosis were identified according to their metabolic dysfunction.

The robust connection in terms of both epidemiology and pathogenesis among NAFLD, metabolic dysfunction, and insulin resistance prompted an external expert committee to propose a diagnosis based on affirmative criteria rather than exclusionary ones, like “nonalcoholic”. It was almost universally agreed upon that the criteria should be encompassing enough to detect individuals with cardiometabolic risk factors and obesity, taking into account regional and ethnic distinctions. Additionally, the consensus was that the criteria should involve easily accessible and simple-to-measure parameters for broad applicability across various clinical scenarios. Lastly, the chosen diagnostic criteria were aligned with cardiometabolic risk factors that are commonly linked to insulin resistance, and are thoroughly established and confirmed within the realm of cardiovascular disease.

In June 2023, experts from the hepatology societies European Association for the Study of the Liver

(EASL), La Asociación Latinoamericana para el Estudio del Hígado (ALEH), and the American Association for the Study of Liver Diseases (AASLD) published a consensus recommending the division of SLD depending on aetiology (Figure 2):

- MASLD - fatty liver in patients with at least one of 5 cardiometabolic risk factors,
- MetALD - MASLD and increased alcohol consumption, defined as a weekly consumption of at least 140 g (women) or 210 g (men),
- ALD (alcohol related liver disease) – fatty liver associated solely with alcohol consumption,
- SLD secondary to other diseases,
- SLD cryptogenic [10].

According to the presented current definition, SLD may be diagnosed by imaging or biopsy. The main message for practitioners resulting from this document is the replacement of NAFLD notion with MASLD and NASH with MASH (metabolic dysfunction-associated steatohepatitis).

Shifting from the NAFLD definition to the MASLD one, where the focus moves from excluding other liver diseases to emphasizing specific, primarily cardiometabolic risk factors, introduces potential limitations. Firstly, the fundamental metabolic dysfunction that underlies MASLD is insulin resistance, and the chosen metabolic risk factors do not uniformly predict insulin resistance. For instance, HDL-C and diastolic blood pressure only exhibit a weak association with insulin resistance. Secondly, both steatosis and insulin resistance could manifest even without any cardiometabolic risk factors, particularly among younger adults. In situations of uncertainty, if a clinician holds a strong suspicion of underlying metabolic dysfunction despite

Table 1. Comparison of definition and diagnosis criteria of fatty liver disease of metabolic aetiology

Parameter	Non-alcoholic fatty liver disease (NAFLD)	Metabolic dysfunction-associated fatty liver disease (MAFLD)	Metabolic dysfunction-associated steatotic liver disease (MASLD)
Author	Schaffner <i>et al.</i>	Eslam <i>et al.</i>	Rinella <i>et al.</i>
Year of publication	1986	2020	2023
Diagnosis criteria			
Liver steatosis	Required	Required	Required
Metabolic criteria	Not required	One of the following criteria: 1. Overweight/obesity 2. T2DM 3. Metabolic dysfunction (at least two): – hypertension – abdominal obesity – hypertriglyceridaemia – low HDL-C level – prediabetes – hs CRP level > 2 mg/l – HOMA-IR ≥ 2.5	Cardiometabolic criteria (at least one): – Overweight/obesity/abdominal obesity – T2DM/prediabetes – Hypertension – Hypertriglyceridaemia – Low HDL-C level
Non-metabolic reasons of liver steatosis	Not acceptable	Acceptable	Not acceptable

HDL-C – high-density lipoprotein, HOMA-IR – homeostatic model assessment for insulin resistance, hsCRP – high sensitivity C-reactive protein, MAFLD – metabolic dysfunction-associated fatty liver disease, MASLD – metabolic dysfunction associated steatotic liver disease, NAFLD – nonalcoholic fatty liver disease, T2DM – type 2 diabetes mellitus.

the lack of cardiometabolic risk factors, the classification “possible MASLD” could be considered, awaiting further tests such as HOMA-IR or OGTT.

Song *et al.* [41] examined data of 1016 randomly selected community patients who had undergone proton-magnetic resonance spectroscopy from 2008 to 2010. The population prevalence of NAFLD, MASLD, and MAFLD was 25.7% (95% CI: 23.1–28.5%), 26.7% (95% CI: 24.0–29.5%), and 25.9% (95% CI: 23.2–28.7%), respectively. The authors concluded that the MASLD definition reclassified fewer patients with NAFLD than the MAFLD definition, and discrepancy between MASLD and NAFLD was minimal.

Table 1 presents the criteria for the diagnosis of NAFLD, MAFLD, and MASLD. It seems that the last definition of the disease best reflects its aetiology, but studies are needed to determine whether it is better than the previous one in predicting the progression of liver disease and cardiovascular events.

Summary

Fatty liver disease with metabolic aetiology is a civilization disease that results in an increased risk of cardiovascular events. Therefore, it is necessary to know the current criteria for its diagnosis. In recent years, the definition has changed from NAFLD to MAFLD and finally to MASLD. The criteria for diagnosis and the nomenclature differ significantly; all definitions have strengths but also limitations. To put

it simply, they share one common criterion: the presence of fatty liver, but they differ in the clinical profile assigned to a given definition.

Funding

Project financed under the program the Minister of Education and Science called “Regional Initiative of Excellence” in the years 2019–2023, project no. 024/RID/2018/19, amount of financing 11,999,000 PLN.

Ethical approval

Not applicable.

Conflict of interest

The authors declare no conflict of interest.

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Address for correspondence:

Iwona Gorczyca-Głowacka
Collegium Medicum
 Jan Kochanowski University
 Kielce, Poland
 Phone: +48 604 407 956
 E-mail: iwona.gorczyca@interia.pl

Received: 16.08.2023

Accepted: 5.08.2024

Online publication: 31.12.2024