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ORIGINAL ARTICLE

Breaking new ground: MASLD vs. MAFLD—which holds the key for risk stratification?

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Abstract

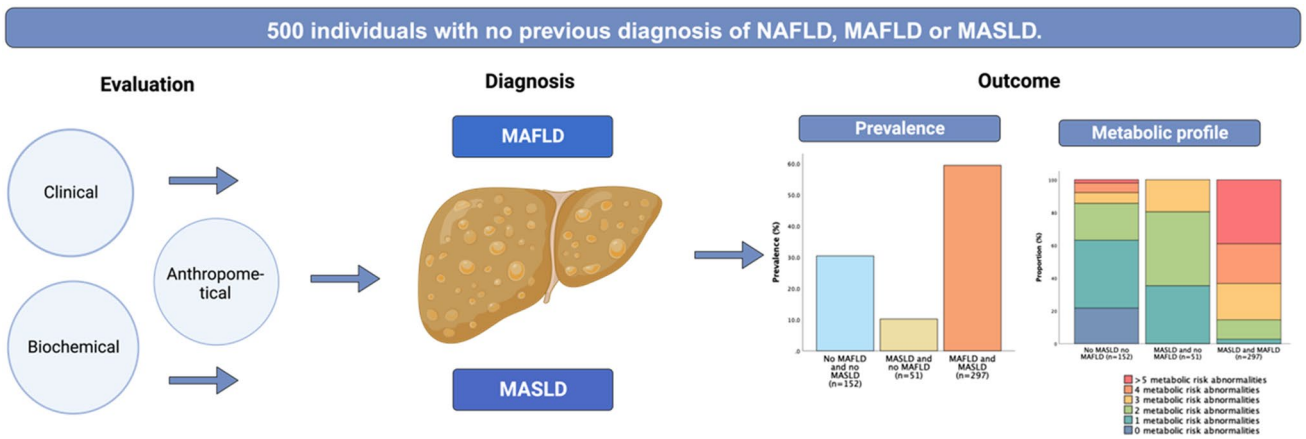
Background The classification and nomenclature of non-alcoholic fatty liver disease (NAFLD) has been the subject of ongoing debate in the medical community. Through the introduction of metabolic dysfunction-associated fatty liver disease (MAFLD) and the later release of metabolic dysfunction-associated steatotic liver disease (MASLD), the limitations associated with NAFLD are intended to be addressed. Both terminologies incorporate the metabolic component of the disease by providing diagnostic criteria that relies on the presence of underlying metabolic risk factors.

Materials and Methods An epidemiologic cross-sectional study of individuals who had undergone abdominal ultrasound and vibration-controlled transient elastography (VCTE) as part of a routine check was performed. We evaluated clinical, anthropometric, and biochemical variables to determine the metabolic profile of each subject.

Results The study included a total of 500 participants, 56.8% (n = 284) males and 43.2% (n = 216) females, with a mean age of 49 ± 10 years. 59.4% (n = 297) were diagnosed with MAFLD and MASLD, 10.2% (n = 51) were diagnosed only with MASLD and 30.4% (n = 152) were not diagnosed with either MAFLD or MASLD. The differences in prevalence were mainly based on the detection of individuals with a BMI < 25 kg/m^2, where MASLD captures the largest number ($p < 0.001$).

Conclusions Although MASLD has a higher capture of lean patients compared to MAFLD, patients with MAFLD and MASLD have a worse metabolic profile than those with only MASLD. Our results provide evidence that MAFLD better identifies patients likely to have a higher risk of liver fibrosis and of disease progression.

Graphical abstract



Keywords Fatty liver · MAFLD · MASLD · Obesity · Cardiovascular risk · Metabolic dysfunction · Diabetes mellitus

Extended author information available on the last page of the article

Background

The prevalence of fatty liver disease (FLD) has increased dramatically in recent decades [1]. FLD is considered one of the most common liver disorders worldwide, with a global prevalence of approximately 38% [2]. Since the introduction of metabolic dysfunction-associated fatty liver disease (MAFLD) [3, 4], the renaming of non-alcoholic fatty liver disease (NAFLD) has been a source of debate. To date, the use of MAFLD has proven superior to the use of NAFLD by demonstrating a greater ability to detect individuals with a higher number of risk factors and a higher risk of developing liver fibrosis [5, 6]. Although the advantages of MAFLD are supported by extensive clinical evidence and its use has been supported by stakeholders around the world [7], no agreement has been reached on which terminology is more appropriate [8–10]. Recently, the terms steatotic liver disease (SLD) and metabolic dysfunction-associated steatotic liver disease (MASLD) have been proposed, with the same aim as MAFLD, to reduce the limitations associated with the previous terminology [11]. Both terminologies incorporate the metabolic component of the disease (with differences) by providing diagnostic criteria that rely on the presence of underlying metabolic risk factors [4, 11] (Fig. 1).

Correct identification of individuals at high metabolic risk is crucial, considering the close association between FLD and other complex metabolic diseases [12]. In this regard, cardiovascular disease (CVD) has been identified as the major cause of mortality in patients with FLD [13]. The association between FLD and CVD is attributable to several factors, including obesity, diabetes mellitus and atherogenic dyslipidemia [14]. Obesity, a prevalent condition in contemporary societies, is also a well-known risk factor for both FLD and CVD. Excessive fat accumulation not only affects the liver, but also causes systemic inflammation and metabolic disturbances, contributing significantly to cardiovascular complications [15]. Both disorders also have insulin resistance (IR) as a pathophysiological contributor. IR promotes inflammation and dyslipidemia, which are key drivers of atherosclerosis, the underlying cause of most CVD [16]. As MAFLD progresses, the risk of cardiovascular complications increases, underscoring the critical need for healthcare professionals to recognize and treat both conditions in a comprehensive and integrated manner to optimize patient care and outcomes [17].

In this setting, the motivation for our study lies in the recognition that the MAFLD and MASLD definitions encompass multifaceted aspects of liver disease associated with metabolic abnormalities. Nevertheless, despite their apparent similarities, subtle differences exist that merit meticulous examination. We conducted the present study with the aim

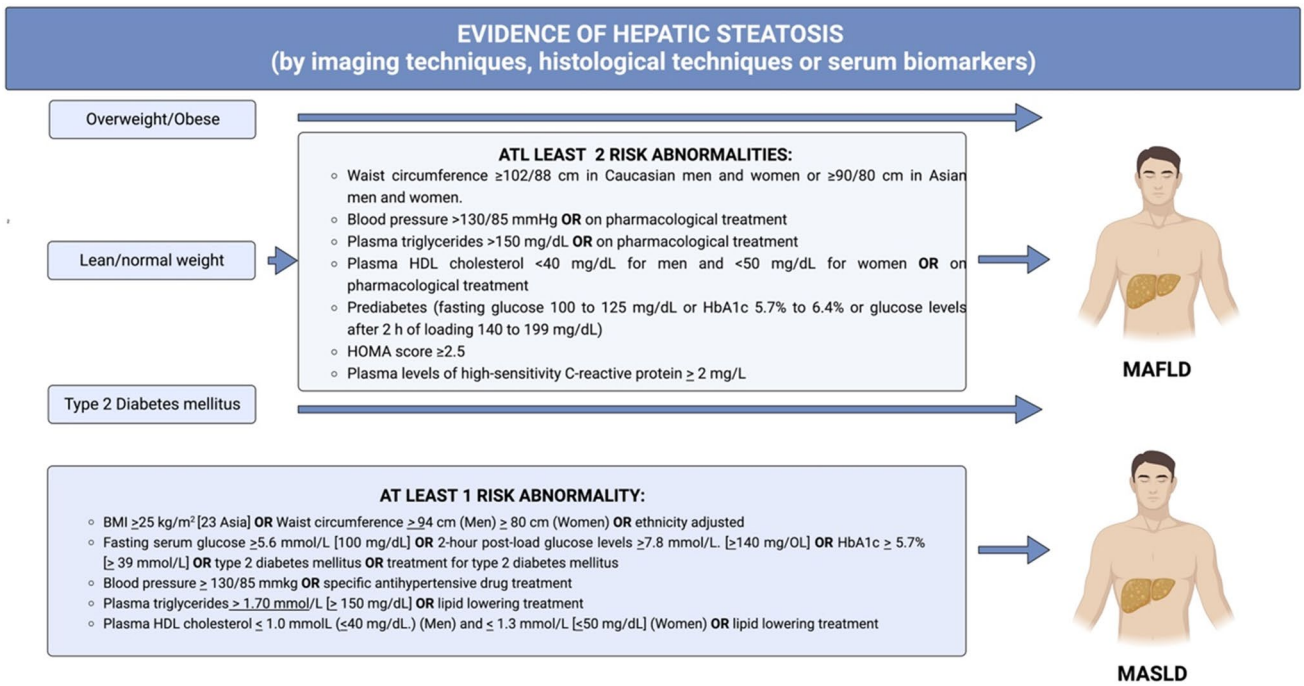


Fig. 1 Overview of MAFLD and MASLD diagnostic criteria. This figure provides a comprehensive visual representation of the diagnostic criteria for Metabolic Associated Fatty Liver Disease (MAFLD) and Metabolic Associated Steatohepatitis (MASLD). It illustrates the key parameters and clinical markers that are considered by each definition

of comparing the use of MAFLD and MASLD and to evaluate the clinical impact of both definitions.

Materials and methods

The publication of this article follows the editorial and methodological recommendations of the STROBE Declaration, the Equator Network and the ICMJE.

Patients

We carried out an epidemiologic cross-sectional study of 500 individuals who had undergone abdominal ultrasound and vibration-controlled transient elastography (VCTE) as part of a routine check at Medica Sur Clinic & Foundation in Mexico City. The study enrolled patients through random selection, with no previous diagnosis of NAFLD, MAFLD, or MASLD.

Data collection

All data were collected from review of the medical records. Clinical evaluation, imaging studies and laboratory test were performed the same day. The following information was obtained: age, sex, systolic and diastolic blood pressure (mmHg), body mass index (BMI) (kg/m^2), body fat (%), waist circumference (cm), hip circumference (cm), and waist-hip ratio. The following information was obtained from the laboratory reports: platelets (× 103/µL), creatinine (mg/dL), triglycerides (mg/dL), cholesterol (mg/dL), HDL cholesterol (mg/dL), LDL cholesterol (mg/dL), high sensitivity C-reactive protein (hsCRP) (mg/dL), total bilirubin (mg/dL), glutamic oxaloacetic transaminase (SGOT) (mg/dL), glutamic pyruvic transaminase (SGPT) (mg/dL), alkaline phosphatase (ALP) (mg/dL), gamma glutamyl transferase (GGT) (mg/dL), thyroid stimulating hormone (TSH) (mg/dL), glycosylated hemoglobin (HbA1c) (%).

Diagnosis of fatty liver

The diagnosis of fatty liver by ultrasound was based on the following criteria: heightened contrast between the liver and kidneys, elevated liver parenchyma echogenicity, obscured visualization of intrahepatic vessels, and/or modifications in diaphragm visibility. Liver stiffness (LSM) (kPa) and controlled attenuation parameter (CAP) (dBm) measurements were used for VCTE diagnosis as per manufacturer recommendations using the Fibroscan 502 Touch®.

Diagnosis of MAFLD and MASLD

For the diagnosis of MAFLD, the criteria proposed by the consensus of experts in 2020 was used [18]. The following criteria were considered for diagnosis: evidence of hepatic steatosis, in this case by ultrasound or VCTE, plus one of the following: overweight/obese (BMI ≥ 25 kg/m^2) or lean/normal weight (BMI < 25 kg/m^2) with evidence of metabolic dysregulation. Metabolic dysregulation was defined as the presence of at least two metabolic risk abnormalities: (a) waist circumference ≥ 102/88 cm in men and women, respectively, (b) systolic blood pressure ≥ 130 mm Hg or diastolic blood pressure ≥ 85 mmHg, (c) plasma triglycerides ≥ 150 mg/dL, (d) plasma HDL-cholesterol < 40 mg/dL for men and < 50 mg/dL for women, (e) prediabetes (HbA1c 5.7–6.4%) and (f) hs-CRP > 2 mg/dL.

For the diagnosis of MASLD, the criteria proposed by the multi-society consensus in 2023 was used [11]. These include evidence of hepatic steatosis by ultrasound or VCTE, combined with one of the following: (a) BMI ≥ 25 kg/m^2 or waist circumference > 94/80 cm in men and women, respectively, (b) HbA1c ≥ 5.7%, (c) systolic blood pressure ≥ 130 mm Hg or diastolic blood pressure ≥ 85 mmHg, (d) plasma triglycerides ≥ 150 mg/dL and (e) plasma HDL-cholesterol < 40 mg/dL for men and < 50 mg/dL for women.

Statistical analysis

Continuous variables are expressed as mean, median and range. Categorical variables are expressed as frequencies and percentages. ANOVA and chi-square test were used to analyze differences between groups for quantitative and qualitative variables, respectively with p values < 0.05 considered significant. To assess the congruence and consistency of diagnoses between ultrasound and VCTE methods for MAFLD and MASLD, we employed Pearson's chi-square test and Kappa statistics. A multinomial multivariate model was performed, with the dependent variable being the diagnosis of MAFLD/MASLD.

Results

Population characteristics

In the study sample, 56.8% (n = 284) were male and 43.2% (n = 216) female, with a mean age of 49 years, 95% CI = 48.38 to 50.64, with a median of 50 years. 59.4% (n = 297) were diagnosed with MAFLD and MASLD, 10.2% (n = 51) were diagnosed only with MASLD and 30.4% (n = 152) did not meet MAFLD and MASLD criteria, Table 1 summarizes the baseline characteristics of the population.

Table 1 Sociodemographic, clinical and laboratory characteristics

Variable	Arithmetic mean CI 95%	Median Q25-P75
Age (years)	49.51 48.38 a 50.64	50 43 a 55.50
Systolic blood pressure (mmHg)	114.40 113 a 115.79	113 104 a 123.75
Diastolic blood pressure (mmHg)	73.71 72.85 a 74.56	73 68 a 80
Body mass index (kg/m^2)	25.88 25.53 a 26.24	25.50 23.30 a 28.07
Body fat (%)	28.24 27.49 a 28.98	27.20 22.70 a 33.10
Waist circumference (cm)	90.92 89.90 a 91.94	91 84 a 98
Hip circumference (cm)	100.20 99.36 a 101.05	100 95.25 a 104
Waist Hip Index (numerical value)	0.90 0.89 a 0.90	0.90 0.84 a 0.96
Platelets (× 103/μL)	247.60 242.14 a 253.06	242 206 a 284.75
Creatinine (mg/dL)	0.90 0.88 a 0.91	0.89 0.77 a 1.02
Triglycerides (mg/dL)	130.38 122.40 a 138.37	108.50 76 a 152.75
Cholesterol (mg/dL)	205.17 201.46 a 208.89	205 177.25 a 232
HDL cholesterol (mg/dL)	51.43 49.96 a 52.90	49 41 a 60
LDL cholesterol (mg/dL)	2.73 2.64 a 2.82	2.60 2 a 3.40
High sensitivity C-reactive protein (mg/dL)	2.70 2.24 a 3.17	1.20 0.60 a 2.70
Total bilirubin (mg/dL)	0.97 0.73 a 1.21	0.79 0.64 a 1
Glutamic oxaloacetic transaminase (mg/dL)	25.98 24.91 a 27.06	23.50 20 a 28
Glutamic pyruvic transaminase (mg/dL)	31.62 29.06 a 34.17	25.50 19 a 34
Alkaline phosphatase (mg/dL)	60.03 58.45 a 61.62	57 48 a 69
Gamma glutamyl transferase (mg/dL)	26.87 24.87 a 28.88	20 15 a 30
Thyroid stimulating hormone (mg/dL)	2.47 2.33 a 2.62	2.16 1.47 a 3.09
Glycosylated hemoglobin (%)	5.41 5.36 a 5.46	5.40 5.10 a 5.60
Hepatic stiffness (kPa)	4.20 4.10 a 4.29	4.10 3.50 a 4.80
Controlled attenuation parameter (dBm)	249.21 244.46 a 253.95	247 209 a 280.50

Diagnosis of MAFLD and MASLD

There is no significant difference between the diagnosis of MAFLD and MASLD by ultrasound or VCTE, with a substantial level of concordance (K = 0.644, p = 0.000) for MAFLD and with a moderate concordance level (K = 0.490, p = 0.000) for MASLD, confirming the feasibility of using readily available imaging methods to diagnose the disease.

Comparison of the variation in the proportion of subjects with MAFLD and MASLD

Overall the prevalence of MAFLD was 59.4% (n = 297), while the prevalence of MASLD was 69.9% (n = 348).When we applied the different cut-off points for each of the parameters used in the MAFLD and MASLD diagnostic criteria, we observe that the differences in prevalence are mainly based on the detection of individuals with a BMI < 25 kg/m^2, where MASLD captures the largest number of lean individuals (p < 0.001) (Table 2).

Metabolic profiling of individuals with MAFLD and MASLD

To assess the metabolic profile of individuals, we evaluated the number of metabolic alterations present in the individuals (Fig. 2) as well as their quantification. Individuals with MAFLD and MASLD have a higher metabolic risk profile compared to individuals who only have a diagnosis of MASLD (Table 3). The difference in metabolic risk profiles observed among these groups highlights the importance of discriminating between the two diagnostic categories.

Multinomial multivariate model

The data in our multinomial mutivariable model (Table 4) suggests that women (compared to men) have 2.3 times the risk of presenting MAFLD and MASLD (OR = 2.30, CI95% = 1.16–4.56); likewise, subjects with an increase in BMI (OR = 2.46, 95%CI = 2.03–2.99) and those with an increase in the percentage of glycosylated hemoglobin (OR = 2.15, 95%CI = 0.80–5.73), have two times the risk of presenting with MAFLD and MASLD. The rest of the

Table 2 Evaluation of the proportions of individuals with MAFLD and MASLD according to the cut-off points of the diagnostic criteria

Cut-off points according to diagnostic criteria	No MASLD and no MAFLD (n = 152) Frequency (%)	MASLD and no MAFLD (n = 51) Frequency (%)	MASLD and MAFLD (n = 297) Frequency (%)	Statistical test Level of significance
Waist circumference				
According to male cut-off points				
< 94 cm	144 (94.7)	45 (88.2)	127 (42.8)	X^2: 124.453 < 0.001
> 94 cm	8 (5.3)	6 (11.8)	170 (57.2)	
According to female cut-off points				
< 80 cm	71 (46.7)	6 (11.8)	12 (4)	X^2: 120.557 < 0.001
> 80 cm	81 (53.3)	45 (88.2)	285 (96)	
HDL cholesterol				
According to male cut-off points				
< 40 mg/dL	14 (9.2)	7 (13.7)	100 (33.7)	X^2: 34.651 < 0.001
> 40 mg/dL	138 (90.8)	44 (86.3)	197 (66.3)	
According to female cut-off points				
< 50 mg/dL	55 (36.2)	18 (35.3)	204 (8.7)	X^2: 46.804 < 0.001
> 50 mg/dL	97 (63.8)	33 (64.7)	93 (31.3)	
Triglycerides				
< 150 mg/dL	147 (96.7)	46 (90.2)	179 (60.3)	X^2: 74.136 < 0.001
> 150 mg/dL	5 (5.3)	5 (9.8)	118 (39.7)	
Blood pressure				
Systolic				
< 130 mmHg	140 (92.1)	47 (92.2)	227 (76.4)	X^2: 18.821 < 0.001
> 130 mmHg	12 (7.9)	4 (7.8)	70 (23.6)	
Diastolic				
< 85 mmHg	145 (95.4)	50 (98)	247 (83.2)	X^2: 16.391 < 0.001
> 85 mmHg	7 (4.6)	1 (2.0)	50 (16.8)	
Glycosylated hemoglobin				
< 5.7%	144 (94.7)	49 (96.1)	213 (71.7)	X^2: 38.163 < 0.001
> 5.7%	8 (5.3)	2 (3.9)	84 (28.3)	
Body mass index				
< 25 kg/m^2	130 (85.5)	51 (100)	42 (14.1)	X^2: 231.341 < 0.001
> 25 kg/m^2	22 (14.5)	0 (0)	255 (85.9)	

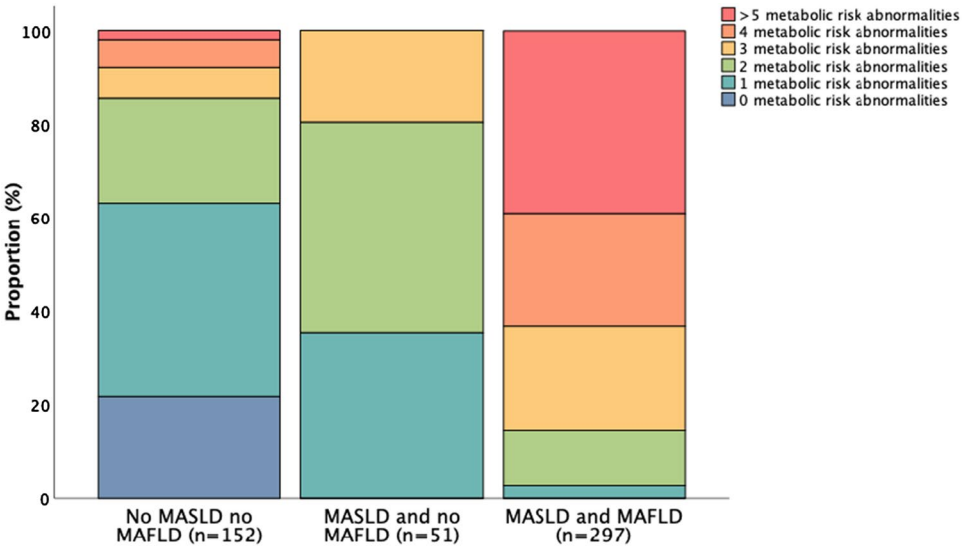


Fig. 2 Distribution of metabolic risk abnormalities among the study groups. The graph offers a comprehensive representation of the proportion of individuals within each group who exhibit varying numbers of metabolic risk abnormalities, ranging from none to more than five

variables show a marked underestimation in the effect of the ORs, tending towards a value of no effect. The model allows us to classify 91% of the subjects presenting as MAFLD and MASLD.

Discussion

Previously, the ability of MAFLD to detect individuals with a high-risk metabolic profile and an increased risk of disease progression has been demonstrated [19, 20]. As we demonstrate, while both MAFLD and MASLD criteria require the presence of metabolic dysfunction, an intriguing distinction arises when we consider the number of patients included in each definition. The MASLD definition appears to encompass a larger number of individuals, which translates into a higher prevalence of the disease. This divergence in prevalence raises an important question: does a higher prevalence automatically make one definition superior to the other, especially in the case of a condition as widespread worldwide as FLD? [21, 22]. This forces us to scrutinize the true essence of these definitions and their impact on patient care.

The key to answer this question resides in the ability to precisely identify high-risk individuals. The change from NAFLD to MAFLD was initiated with the overall goal of redefining the disease to better reflect its underlying metabolic nature and to help in risk-stratification [23]. The change in nomenclature was a response to our evolving understanding of the disease, recognizing that metabolic dysfunction is not only associated with the disease, but is in fact intrinsic to it [1, 24–26]. Therefore, the primary concern here is not simply the prevalence of the disease, but rather its accuracy and more importantly clinical relevance for identifying those at increased risk of adverse outcomes [5, 20, 27, 28]. We observed that the difference between MAFLD and MASLD is principally localized to the detection of lean individuals; the basis for this is the number of metabolic risk abnormalities required for a diagnosis. For MASLD, lean individuals must have at least one metabolic risk abnormality, while for MAFLD they must have two. This may lead to overdiagnosis or misclassification of individuals without high metabolic risk under the MASLD definition. This comes as no surprise since several studies suggest that NAFLD and MASLD identify identical patient groups (98%) and hence would not be expected to risk stratify patients any better than NAFLD. In turn, this deficiency limits the ability of the MASLD definition firstly to identify homogenous patient groups (3 in the case of MAFLD—overweight/obese, normal weight with two metabolic risk factors and those with type 2 diabetes and at least 5 for MASLD) with different clinical features in cross sectional studies and different disease trajectories, as well as different risks of liver fibrosis [29]. These aspects are critical at the bedside for clinical management.

The main limitation of our study is the sample size as some of the clinical differences between MAFLD and MASLD could not be substantiated by statistical analysis. In addition, the categorization of the dependent variable implies a mismatch between the subgroups of subjects when contrasting the estimators of interest, whether proportions or means. Furthermore, the lack of adequate representation of individuals with MAFLD or MASLD with significant liver fibrosis is a limitation. Consequently, we were unable to explore this critical aspect of the disease in depth, limiting the scope of our conclusions and our insights into the advanced stages of MAFLD and MASLD. These limitations underscore the need for larger and more diverse data sets to reach more robust conclusions about clinical differences and disease progression rates in MAFLD and MASLD.

Table 3 Clinical, anthropometrical, and cardiometabolic characteristics of participants

Variables	No MAFLD No MASLD (N = 152)		MASLD and not MAFLD (N = 51)		MAFLD and MASLD (N = 297)		Mean difference IC95 MASLD And Not MAFLD group vs MAFLD And MASLD group	Statistical test Level of significance
	Arithmetic mean CI 95%	Median Q25-P75	Arithmetic mean CI95%	Median Q25-P75	Arithmetic mean CI 95%	Median Q25-P75		
Systolic blood pressure (mmHg)	109.70 107.55–11.85	110 100–118	104.61 99.53–109.69	105 99–112	118.48 116.74–120.23	118 109–128	− 13.877 − 19.35 to − 8.40	ANOVA = 18,021 p = 0.000
Diastolic Blood Pressure (mmHg)	70.67 69.14–72.20	70 64–78.75	68.57 66.29–70.84	70 64–73	76.14 75.08–77.21	75 69.50–81	− 7.576 3.25 to 7.70	ANOVA = 12,280 p = 0.000
Body Mass Index (kg/m2)	22.65 22.28–23.02	22.80 21.20–24.27	23.10 22.70–23.51	23.30 22.30 – 24.30	28.01 27.60–28.43	27.50 25.70–29.55	− 4.90919 − 6.0371 to − 3.7813	ANOVA = 23,362 p = 0.000
Body fat (%)	23.83 22.63–25.03	23.20 18.60–28.60	26.29 24.70–27.87	26.10 21.50–30.90	30.83 29.87–31.78	29.20 25–36.10	− 4.54017 − 7.3994 to − 1.6810	ANOVA = 0.797 p = 0.372
Waist circumference (cm)	81.08 79.66–82.51	82 75–88	86.43 84.53–88.32	86 82–92	96.73 95.63–97.83	96 90.50–102	− 10.3057 − 13,637 to − 6,974	ANOVA = 3,325 p = 0.069
Hip circumference (cm)	95.21 94.34–96.08	96 92–99	97.52 96.49–98.56	97 95–100	103.22 102–104.44	102 99–107	− 5.6969 − 8,918 to − 2,476	ANOVA = 1,653 p = 0.199
Waist Hip Index (numerical value)	0.85 0.83–0.86	0.85 0.80–0.91	0.87 0.85–0.89	0.87 0.85–0.92	0.93 0.92–0.94	0.94 0.88–0.99	− 0.05620 − 0.0873 to − 0.0251	ANOVA = 1,555 p = 0.213
Platelets (×103/μL)	251.78 241.89–261.68	249.50 205.50–285.75	255.82 237.31–274.33	239 206–304	244.04 237–251.09	239 206–282	11.77383 − 10.8502 to 34.3979	ANOVA = 0.732 p = 0.393
Creatinine (mg/dL)	0.89 0.86–0.92	0.89 0.77–0.99	0.85 0.79–0.91	0.82 0.72–1.02	0.91 0.88–0.93	0.90 0.78–1.03	− 0.05843 − 0.1276 to 0.0108	ANOVA = 3,048 p = 0.081
Triglycerides (mg/dL)	86.68 81.17–92.18	79.50 63 – 107.75	96.96 85.85–108.07	88 70—124	158.49 146.46–170.52	134 94.50 – 186.50	− 61.531 − 92.26 to − 30.80	ANOVA = 4,171 p = 0.042
Cholesterol (mg/dL)	198.26 191.40–205.11	196 172.25 – 225.75	206.82 195.79 to 217.85	214 175—233	208.43 203.60 to 213.26	208 182.50—234	− 1.607 − 16.96 to 13.74	ANOVA = 0.308 p = 0.579
HDL cholesterol (mg/dL)	57.28 55.11–59.44	56.50 48–66	58.09 51.77–64.42	54 27–63	47.30 45.49–49.11	45 38–54	10.7953 4,971 to 16,619	ANOVA = 5,961 p = 0.015
LDL cholesterol (mg/dL)	2.28 2.13–2.43	2.20 1.50–2.70	2.52 2.30–2.74	2.40 2.00–3.20	3.00 2.88–3.12	2.90 2.20–3.60	− 0.47720 − 0.8397 to − 0.1147	ANOVA = 0.642 p = 0.423
High sensitivity C-reactive protein (mg/dL)	2.60 1.35–3.84	0.80 0.40–1.90	1.45 0.77–2.13	1.00 0.40–1.40	2.97 2.53–3.41	1.60 0.80–3.40	− 1.51858 − 3.4372 to 0.400	ANOVA = 2,889 p = 0.090
Total bilirubin (mg/dL)	1.22 0.44–2.00	0.76 0.62	0.89 0.81–0.98	0.87 0.65–1.05	0.85 0.81–0.89	0.81 0.64–1.01	0.04163 − 0.9456 to 1.0288	ANOVA = 0.124 p = 0.725

Table 3 (continued)

Variables	No MAFLD No MASLD (N = 152)		MASLD and not MAFLD (N = 51)		MAFLD and MASLD (N = 297)		Mean difference IC95 MASLD And Not MAFLD group vs MAFLD And MASLD group	Statistical test Level of significance
	Arithmetic mean CI 95%	Median Q25-P75	Arithmetic mean CI95%	Median Q25-P75	Arithmetic mean CI 95%	Median Q25-P75		
Glutamic Oxaloacetic Transaminase (mg/dL)	23.32 22.21–24.42	22 19–26	23.82 21.51–26.14	22 18–28	27.72 26.06–29.37	24 21–29	− 3.894 − 8.29 to 0.51	ANOVA = 0.888 p = 0.346
Glutamic Pyruvic Transaminase (mg/dL)	24.20 21.71–26.70	21 17–26.75	25.67 21.19–30.14	20 17–32	36.43 32.47–40.39	29 22–40	− 10.764 − 21.16 to − 0.37	ANOVA = 1.201 p = 0.274
Alkaline phosphatase (mg/dL)	54.26 51.98–56.54	53 44–62	59.06 54.97–63.15	60 49–68	63.15 60.93–65.38	61 50–75	− 4.096 − 10.50 to 2.31	ANOVA = 0.018 p = 0.894
Gamma Glutamyl Transferase (mg/dL)	21.01 18.20–23.83	16 13–22.75	23.33 17.15–29.52	16 13–23	30.48 27.68–33.28	22 18–36	− 7.148 − 15.30 to 1.00	ANOVA = 0.526 p = 0.469
Thyroid Stimulating Hormone (mg/dL)	2.28 2.05 to 2.50	1.85 1.37 – 2.85	2.27 1.89 to 2.66	2.17 1.23 – 3.14	2.61 2.41 to 2.81	2.22 1.51 – 3.30	− 0.33373 − 0.9241 to 0.2567	ANOVA = 0.494 p = 0.482
Glycosylated hemoglobin (%)	5.24 5.20–5.29	5.20 5.10 – 5.40	5.24 5.16 to 5.31	5.30 5.00 – 5.40	5.53 5.45–5.60	5.50 5.20 – 5.70	− 0.2905 0.102 to 0.479	ANOVA = 3,655 p = 0.056
Hepatic stiffness (kPa)	3.90 3.77 to 4.04	3.90 3.20 – 4.50	4.10 3.59 to 4.62	4 3.30 – 4.50	4.36 4.25 to 4.48	4.30 3.70 – 4.90	− 0.2628 − 0.647 to 0.121	ANOVA = 0.043 p = 0.836
Controlled attenuation parameter (dBm)	203.99 198.37–209.60	202.50 182.25—223	229.76 220.31 to 239.22	232 203—255	275.69 270.24 to 281.14	267 244.50—307	− 45.926 − 61.56 to − 30.29	ANOVA = 2,490 p = 0.115

Conclusion

Both MAFLD and MASLD identify individuals with hepatic steatosis and metabolic dysfunction. Although MASLD encompasses a larger cohort, this should not be a factor that determines selecting one definition over the other. Rather, clinical emphasis should remain on accurately identifying high-risk individuals, consistent with the overall goal of redefining and reclassifying fatty liver disease in the context of its metabolic underpinnings. The use of MAFLD and MASLD definitions have significant implications for clinical practice. Recognizing the differences in diagnostic accuracy between both, health care professionals should be aware of the limitations of each category. This is especially important to avoid over-diagnosis or misclassification of patients at low metabolic risk. Regarding future research, long-term studies are needed to understand how the different subtypes of MAFLD and MASLD progress over time and how they respond to different medical and lifestyle interventions. It is also important to conduct studies to validate and ensure the consistency of the MAFLD and MASLD classification criteria to avoid confusion and ensure the global applicability of this new nomenclature. Future research and clinical practice will undoubtedly provide insight into the utility and implications of these evolving definitions for the management of this globally important health problem.

Table 4 Multivariate model of polytomic logistic regression

Variables	Wald's test Level of significance	Odss ratio CI 95%
Sex (gender reference: male)	8.08 p = 0.004	4.28 1.57–11.68
Ultrasound hepatic steatosis	55.77 p = 0.000	90.20 27.67–294
FibroScan hepatic steatosis	44.309 p = 0.000	37.73 12.95–109.91
Systolic blood pressure (mmHg)	0.633 p = 0.426	1.01 0.980–1.04
Diastolic blood pressure (mmHg)	0.000 p = 0.995	1.0 0.939–1.06
Body Mass Index (kg/m^2)	50.919 p = 0.000	2.85 2.14–3.81
Triglycerides (mg/dL)	15.812 p = 0.000	1.02 1.01–1.03
HDL cholesterol (mg/dL)	1.667 p = 0.197	0.98 0.96–1.0
Glycosylated hemoglobin (%)	3.595 p = 0.058	4.23 0.95–18.82
Constant	p = 0	

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Data availability All data supporting the findings of this study are available within the paper.

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the Ethics Committee of Medica Sur Clinic & Foundation (protocol code 2021-EXT-552 and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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