

Prevalence, Sonographic Grading and Factors Associated with Metabolic Dysfunction-Associated Steatotic Liver Disease among Adults with Type 2 Diabetes Mellitus: A Multicenter Cross-Sectional Study in Uganda

Abdullahi Ahmed Omar

Kampala International University

Abdisalam Ahmed Sande

abdisalaam77@gmail.com

Kampala International University

Theoneste Hakizimana

Kampala International University

Farah Dubad Abdi

Kampala International University

Abdisamad Guled Hersi

Kampala International University

Mohamed Jayte

Kampala International University

Abdifatah Hersi Karshe

Kampala International University

Isse Muse Osobow

Kampala International University

Ecik Christopher

Kampala International University

Ahmed Hashi Ali

Kampala International University

Sadio Hussein Ibrahim

Kampala International University

Zakaria Ali Ahmed

Kampala International University

Louis Nyende

Kampala International University

Mohamed Elmalik Musa

Kampala International University

Fathi Ali Araye

Kampala International University

Venance Emmanuel Mswelo

Kampala International University

Research Article

Keywords: Metabolic dysfunction-associated steatotic liver disease, Type 2 diabetes mellitus, Hepatic steatosis, Ultrasonography, Poor glycemic control, Obesity, Uganda

Posted Date: October 14th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11173457/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Background

Metabolic dysfunction-associated steatotic liver disease (MASLD) is strongly associated with type 2 diabetes mellitus (T2DM) and is an important cause of chronic liver disease and metabolic morbidity. However, data on MASLD among adults with T2DM in sub-Saharan Africa remain limited. This study aimed to determine the prevalence, sonographic grading, and factors associated with MASLD among adults with T2DM at Kayunga and Hoima Regional Referral Hospitals in Uganda.

Methods

A multicenter hospital-based cross-sectional study was conducted among 422 adults with T2DM attending diabetes clinics at the two study hospitals. Participants underwent clinical assessment, glycated hemoglobin (HbA1c) and triglyceride testing, and abdominal ultrasonography for detection and grading of hepatic steatosis. Patients with known chronic liver disease, viral hepatitis, significant alcohol consumption, or exposure to medications known to cause hepatic steatosis were excluded. Factors independently associated with MASLD were assessed using multivariable logistic regression, with statistical significance defined as $p < 0.05$.

Results

The prevalence of MASLD was 54.5% (230/422). Among participants with MASLD, grade 1 steatosis was most common (175, 76.1%), followed by grade 2 (49, 21.3%) and grade 3 (6, 2.6%). In the multivariable analysis, age (aOR 2.087, 95% CI 1.243–3.504; $p = 0.005$ for 46–59 years; aOR 2.437, 95% CI 1.316–4.511; $p = 0.005$ for ≥ 60 years), hypertension (aOR 1.982, 95% CI 1.198–3.279; $p = 0.008$), obesity (BMI ≥ 30 kg/m²) (aOR 2.237, 95% CI 1.177–4.252; $p = 0.014$), poor glycemic control based on HbA1c (aOR 2.550, 95% CI 1.467–4.434; $p = 0.001$), and triglyceride levels ≥ 2.3 mmol/l (aOR 2.509, 95% CI 1.470–4.284; $p = 0.001$) were independently associated with MASLD.

Conclusion

These findings highlight the importance of incorporating abdominal ultrasonography into the assessment of MASLD, alongside comprehensive management of metabolic risk factors, as part of routine diabetes care to facilitate early identification and management of patients at risk of progressive liver disease.

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is characterized by hepatic steatosis in the presence of cardiometabolic risk factors and is an increasingly recognized cause of chronic liver disease worldwide. It is strongly associated with type 2 diabetes mellitus (T2DM), obesity, dyslipidemia, and hypertension, and may progress to steatohepatitis, liver fibrosis, cirrhosis, and hepatocellular carcinoma. MASLD is also associated with increased cardiovascular morbidity and all-cause mortality [1, 2].

T2DM is a major risk factor for MASLD, with insulin resistance, hyperglycemia, and abnormal lipid metabolism contributing to hepatic fat accumulation [3]. Recent systematic reviews and meta-analyses have reported a high prevalence of MASLD among people with T2DM, with pooled estimates exceeding 50% [4, 5]. Among individuals with T2DM, obesity, poor glycemic control, dyslipidemia, hypertension, and older age have been associated with MASLD [6].

Ultrasonography is widely used to detect and grade hepatic steatosis because it is non-invasive, relatively inexpensive, accessible, and does not involve ionizing radiation. Sonographic hepatic steatosis can be graded according to the degree of increased hepatic echogenicity and attenuation of the ultrasound beam, commonly as mild, moderate, or severe [7]. Although ultrasonography has lower sensitivity for mild steatosis than advanced imaging techniques, its accessibility makes it particularly useful for assessing hepatic steatosis in resource-limited settings [8].

In sub-Saharan Africa, the burden of MASLD among people with T2DM is increasingly recognized, although evidence remains limited and heterogeneous. A systematic review of African studies using the earlier metabolic dysfunction-associated fatty liver disease (MAFLD) terminology reported a pooled prevalence of 48.1% among individuals with T2DM [9]. In Uganda, Enriquez et al. reported MASLD among adults from south-central Uganda, including participants with and without HIV, using the fatty liver index rather than ultrasound-based assessment [10]. However, evidence specifically addressing MASLD among adults with T2DM in Ugandan regional referral hospitals remains limited. Therefore, this study aimed to determine the prevalence, sonographic grading, and factors associated with MASLD among adults with T2DM attending Hoima and Kayunga Regional Referral Hospitals in Uganda.

Methods

Study Design and Setting

This was a multicenter hospital-based cross-sectional study conducted at Kayunga Regional Referral Hospital (KRRH) in central-eastern Uganda and Hoima Regional Referral Hospital (HRRH) in western Uganda. Both hospitals provide specialist medical services to their respective catchment populations and operate diabetes clinics under the Departments of Internal Medicine. Data collection was conducted between April and July 2026.

Study Population and Eligibility Criteria

The study population consisted of adults aged 18 years and above with a confirmed diagnosis of T2DM attending the diabetes clinics at KRRH and HRRH during the study period.

Inclusion Criteria

Adults aged ≥ 18 years with confirmed T2DM who attended the diabetes clinics during the study period, were clinically stable, had decision-making capacity, and provided written informed consent were eligible for participation.

Exclusion Criteria

Patients with known chronic liver disease, viral hepatitis, or significant alcohol consumption (> 210 g/week for men or > 140 g/week for women) were excluded. Pregnant women and patients receiving medications known to cause hepatic steatosis, including corticosteroids, amiodarone, methotrexate, tamoxifen, and sodium valproate, were also excluded. Patients who were critically ill, required emergency care or surgery, or were unable to provide informed consent were not enrolled.

Sample Size Determination

The sample size was calculated using the Daniel formula for estimating a single population proportion [11]. A pooled prevalence of 48.1% was used, based on an African systematic review and meta-analysis of metabolic dysfunction-associated fatty liver disease (MAFLD) among people with T2DM [9]. Using a 95% confidence level ($Z = 1.96$) and a 5% margin of error ($d = 0.05$), the minimum sample size was 384 participants. To account for potential non-response, a 10% adjustment was added, resulting in a final sample size of 422 participants.

Sampling Procedure

Consecutive recruitment was used, whereby eligible adults presenting to the diabetes clinics during the study period were enrolled until the target sample of 422 participants was reached.

Data Collection Procedures

Questionnaire and Clinical Assessment

A structured and pre-tested questionnaire was used to collect data on socio-demographic characteristics (age, sex, education level, residence, and occupation), diabetes-related characteristics (age at diabetes diagnosis, duration of diabetes, and antidiabetic medication use), and clinical and metabolic factors, including HbA1c, body mass index, hypertension, and triglyceride levels. The questionnaire was available in English, Luganda, and Runyoro according to participants' preferred

language. The translated versions were independently back-translated into English to ensure consistency of meaning.

All participants underwent a clinical examination. Weight was measured using a digital weighing scale and height using a stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2).

Alcohol consumption was assessed separately using a structured questionnaire to determine eligibility for study participation. Weekly alcohol intake was estimated based on the reported type, quantity, frequency, and alcohol strength of beverages consumed.

Information on hypertension was obtained from participants' clinical records. Hypertension was defined as a documented diagnosis of hypertension or current use of antihypertensive medication.

Laboratory Assessment

Approximately 5 mL of venous blood was collected from each participant. Serum triglycerides were measured using an iChem-535 automated clinical chemistry analyzer with an enzymatic colorimetric method. Glycated hemoglobin (HbA1c) was measured using high-performance liquid chromatography (HPLC). Hepatitis B surface antigen (HBsAg) and hepatitis C virus antibody (anti-HCV) were tested using SD Bioline HBsAg and SD Bioline HCV rapid diagnostic tests, respectively. Glycemic control was categorized as good ($\text{HbA1c} < 7\%$) or poor ($\text{HbA1c} \geq 7\%$) according to international diabetes management guidelines.

Abdominal Ultrasonography and MASLD Definition

Abdominal ultrasonography was performed by a qualified radiographer using a GE HealthCare LOGIQ-series ultrasound machine with a low-frequency curvilinear probe (typically 2–5 MHz). Participants underwent standard B-mode examination of the liver in longitudinal, transverse, and oblique planes.

Hepatic echogenicity, visualization of the intrahepatic vessels and portal vein walls, visualization of the diaphragm, and penetration of the ultrasound beam into the liver were assessed. Hepatic steatosis was graded as grade 0 (normal), grade 1 (mild), grade 2 (moderate), or grade 3 (severe). Grade 0 was defined as normal hepatic echogenicity with clear visualization of the portal vein walls and diaphragm. Grade 1 was defined as a minimal diffuse increase in hepatic echogenicity with preserved visualization of the diaphragm and portal vein walls. Grade 2 was defined as a moderate increase in echogenicity with blurring of the diaphragm and reduced visualization of intrahepatic vessels. Grade 3 was defined as marked increase in echogenicity with poor penetration of the ultrasound beam and limited visualization of the diaphragm and posterior right hepatic lobe.

For this study, MASLD was operationally defined as sonographically detected hepatic steatosis in an adult with T2DM in the absence of significant alcohol consumption (> 210 g/week for men or > 140 g/week for women), viral hepatitis, known chronic liver disease, or exposure to medications known to cause hepatic steatosis.

Validity and Reliability of Tools

Content validity of the questionnaire was assessed by two physicians using the Content Validity Index (CVI), with a CVI of ≥ 0.80 considered acceptable. Prior to the main study, the questionnaire was piloted among 10 patients attending the study clinics to assess its clarity, comprehensibility, and reliability.

Data Management and Statistical Analysis

Data were entered and cleaned in Microsoft Excel and subsequently imported into SPSS version 26.0 for analysis. Descriptive statistics were used to summarize participant characteristics. MASLD prevalence was presented with 95% confidence intervals, while sonographic grades were reported as frequencies and percentages.

Associations between categorical variables were assessed using the chi-square test. Variables with $p \leq 0.20$ in the bivariate analysis were included in the multivariable logistic regression model. Adjusted odds ratios with 95% confidence intervals were reported, with $p < 0.05$ considered statistically significant.

Ethical Considerations

Ethical clearance for the study was granted by the Kampala International University Research Ethics Committee. Permission to conduct the study was obtained from the administrations of KRRH and HRRH. Participation was voluntary, and refusal to participate or withdrawal from the study did not affect access to routine medical care. Participant confidentiality was maintained using unique study identification numbers, and personal identifiers were excluded from the analytical dataset. Participants with clinically relevant abnormal findings were referred to the attending clinical team for appropriate management. All data collection procedures were conducted in accordance with standard ethical guidelines.

Results

Characteristics of the Study Participants

The study enrolled 422 adults with T2DM. Females comprised 58.3% of the sample, while 30.1% of participants were younger than 45 years. Most participants (63.5%) were diagnosed with T2DM after age 40 years, and 36.0% had a duration of T2DM exceeding 5 years. Metformin monotherapy was the most commonly reported treatment regimen (59.2%). The complete baseline characteristics are presented in Table 1.

Table 1
Characteristics of study participants

Characteristic	Frequency	Percentage
Age (years)		
< 45	127	30.1
46–59	181	42.9
60+	114	27.0
Sex		
Male	176	41.7
Female	246	58.3
Education level		
Primary	234	55.4
Secondary	173	41.0
Tertiary	15	3.6
Residence		
Rural	302	71.6
Urban	120	28.4
Income (monthly/Ugx)		
< 250K	331	78.4
250-500K	60	14.2
> 500K	31	7.3
Occupation		
Formal employment	76	18.0
Student	47	11.1
Business	48	11.4
Un-employed	53	12.6
Farmer	198	46.9
Age at diagnosis		
< 40 years	154	36.5

Characteristic	Frequency	Percentage
Age (years)		
≥ 40 years	268	63.5
DM duration		
≤ 5 years	270	64.0
> 5 years	152	36.0
Antidiabetic medication		
BIG alone	250	59.2
BIG + SFU	86	20.4
BIG + Insulin	9	2.1
Insulin alone	77	18.2
Hypertension		
No	275	65.2
Yes	147	34.8
BMI		
18.5–24.9	165	39.1
< 18.5	34	8.1
25-29.9	138	32.7
≥ 30	85	20.1
HbA1c		
< 7	329	78.0
≥ 7	93	22.0
Triglyceride levels		
< 1.8	165	39.1
1.8–2.2	120	28.4
2.3+	137	32.5

BIG=Biguanide antidiabetics, SFU= sulfonylurea antidiabetics, HbA1c=Glycated hemoglobin, BMI = Body mass index.

Prevalence of MASLD among adults with T2DM

Among the 422 participants, 230 had MASLD, giving a prevalence of 54.5% (95% CI 49.5–59.5). The prevalence of MASLD among the study participants is illustrated in Fig. 1.

Sonographic grading of hepatic steatosis among adults with T2DM

Among the 230 participants with MASLD, 175 (76.1%; 95% CI 70.9–81.7) had grade 1 steatosis, 49 (21.3%; 95% CI 15.7–27.0) had grade 2 steatosis, and 6 (2.6%; 95% CI 0.9–4.8) had grade 3 steatosis. The distribution of sonographic grades is presented in Fig. 2.

Factors associated with MASLD among adults with T2DM

Variables with $p \leq 0.20$ in the bivariate analysis were considered for inclusion in the multivariable model. Age category, residence, duration of T2DM, hypertension, BMI, HbA1c category, and triglyceride level met this criterion and were included in the adjusted analysis. Detailed results are shown in Table 2.

<

Table 2
Bivariable analysis of factors associated with MASLD among adults with T2DM.

Characteristic	No Hepatic steatosis, N = 192	Hepatic steatosis, N = 230	Bivariable analysis		
			cOR	95% CI	P value
Age (years)					
< 45	82(42.7)	45(19.6)	Ref		
46–59	74(38.5)	107(46.5)	2.635	1.648–4.212	< 0.001
60+	36(18.8)	78(33.9)	3.948	2.308–6.753	< 0.001
Sex					
Male	85(44.3)	91(39.6)	Ref		
Female	107(55.7)	139(60.4)	1.213	0.823–1.790	0.329
Education level					
Primary	127(66.1)	107(46.5)	0.562	0.194–1.629	0.288
Secondary	59(30.7)	114(49.6)	1.288	0.438–3.792	0.646
Tertiary	6(3.1)	9(3.9)	Ref		
Residence					
Rural	146(76.0)	156(67.8)	Ref		
Urban	46(24.0)	74(32.2)	1.506	0.978–2.318	0.063
Income (monthly)					
< 250K	159(82.8)	172(74.8)	0.891	0.425–1.866	0.759
250-500K	19(9.9)	41(17.8)	1.777	0.728–4.337	0.207
> 500K	14(7.3)	17(7.4)	Ref		
Occupation					
Formal employment	38(19.8)	38(16.5)	Ref		
Student	29(15.1)	18(7.8)	0.621	0.296–1.301	0.207

Characteristic	No Hepatic steatosis, N = 192	Hepatic steatosis, N = 230	Bivariable analysis		
			cOR	95% CI	P value
Age (years)					
Business	19(9.9)	29(12.6)	1.526	0.734–3.176	0.258
Un-employed	21(10.9)	32(13.9)	1.524	0.749–3.102	0.245
Farmer	85(44.3)	113(49.1)	1.329	0.782–2.260	0.293
Age at diagnosis					
< 40 years	74(38.5)	80(34.8)	Ref		
≥ 40 years	118(61.5)	150(65.2)	1.176	0.790–1.750	0.425
DM duration					
≤ 5 years	138(71.9)	132(57.4)	Ref		
> 5 years	54(28.1)	98(42.6)	1.897	1.260–2.856	0.002
Antidiabetic currently					
BIG alone	112(58.3)	138(60.0)	Ref		
BIG + SFU	36(18.8)	50(21.7)	1.127	0.687–1.850	0.636
BIG + Insulin	4(2.1)	5(2.2)	1.014	0.266–3.867	0.983
Insulin alone	40(20.8)	37(16.1)	0.751	0.450–1.252	0.272
Hypertension					
No	145(75.5)	130(56.5)	Ref		
Yes	47(24.5)	100(43.5)	2.373	1.560–3.611	< 0.001
BMI					
18.5–24.9	85(44.3)	80(34.8)	Ref		
< 18.5	20(10.4)	14(6.1)	0.744	0.352–1.572	0.438

Characteristic	No Hepatic steatosis, N = 192	Hepatic steatosis, N = 230	Bivariable analysis		
			cOR	95% CI	P value
Age (years)					
25-29.9	63(32.8)	75(32.6)	1.265	0.804–1.990	0.310
≥ 30	24(12.5)	61(26.5)	2.701	1.539–4.739	0.001
HbA1c					
< 7	166(86.5)	163(70.9)	Ref		
≥ 7	26(13.5)	67(29.1)	2.624	1.589–4.335	< 0.001
Triglyceride levels					
< 1.8	77(40.1)	88(38.3)	Ref		
1.8–2.2	78(40.6)	42(18.3)	0.471	0.290–0.764	0.002
2.3+	37(19.3)	100(43.5)	2.365	1.455–3.843	0.001

cOR= Crude odds ratio, CI= Confidence interval, BIG=Biguanide antidiabetics, SFU= sulfonylurea antidiabetics, HbA1c=Glycated hemoglobin, BMI=Body mass index.

In the adjusted model, increasing age, hypertension, obesity (BMI ≥ 30 kg/m²), poor glycemic control, and triglyceride levels ≥ 2.3 mmol/L were independently associated with MASLD. Participants aged 46–59 years had higher odds of MASLD (aOR 2.087, 95% CI 1.243–3.504; p = 0.005), as did those aged ≥ 60 years (aOR 2.437, 95% CI 1.316–4.511; p = 0.005). Hypertension (aOR 1.982, 95% CI 1.198–3.279; p = 0.008), obesity (aOR 2.237, 95% CI 1.177–4.252; p = 0.014), poor glycemic control (aOR 2.550, 95% CI 1.467–4.434; p = 0.001), and triglyceride levels ≥ 2.3 mmol/L (aOR 2.509, 95% CI 1.470–4.284; p = 0.001) were also associated with MASLD. Detailed results are presented in Table 3.

Table 3
Multivariable analysis of factors associated with MASLD among adults with T2DM.

Characteristic	Bivariable analysis			Multivariable analysis		
	cOR	95% CI	P value	aOR	95% CI	P value
Age (years)						
< 45	Ref					
46–59	2.635	1.648–4.212	< 0.001	2.087	1.243–3.504	0.005
60+	3.948	2.308–6.753	< 0.001	2.437	1.316–4.511	0.005
Residence						
Rural	Ref					
Urban	1.506	0.978–2.318	0.063	0.981	0.572–1.682	0.945
DM duration						
≤ 5 years	Ref					
> 5 years	1.897	1.260–2.856	0.002	1.372	0.841–2.236	0.202
Hypertension						
No	Ref					
Yes	2.373	1.560–3.611	< 0.001	1.982	1.198–3.279	0.008
BMI						
18.5–24.9	Ref					
< 18.5	0.744	0.352–1.572	0.438	0.791	0.332–1.880	0.595
25–29.9	1.265	0.804–1.990	0.310	1.345	0.807–2.241	0.256
≥ 30	2.701	1.539–4.739	0.001	2.237	1.177–4.252	0.014
HbA1c						
< 7	Ref					
≥ 7	2.624	1.589–4.335	< 0.001	2.550	1.467–4.434	0.001
Triglyceride levels						
< 1.8	Ref					
1.8–2.2	0.471	0.290–0.764	0.002	0.602	0.351–1.034	0.066
2.3+	2.365	1.455–3.843	0.001	2.509	1.470–4.284	0.001

cOR= Crude odds ratio, aOR= adjusted odds ratio, CI= Confidence interval, HbA1c=Glycated hemoglobin, BMI=Body mass index.

Discussion

Prevalence of MASLD

In this study, the prevalence of MASLD among adults with T2DM at KRRH and HRRH was 54.5%. This is comparable to a study from Saudi Arabia, where MASLD was identified in 54.5% of adults with T2DM using abdominal ultrasonography [12]. Similarly, a recent systematic review and meta-analysis from India reported a pooled MASLD prevalence of 56.9% among adults with T2DM, with a prevalence of 55.6% among studies using ultrasonography [4]. In Tanzania, MASLD was identified in 51.6% of adults with T2DM using transient elastography [13]. Despite the use of a different imaging modality, this prevalence was also comparable to our findings.

Conversely, the prevalence observed in our study was lower than that reported in another study from Saudi Arabia, where 68.4% of adults with T2DM had MASLD diagnosed by ultrasonography [14]. It was also lower than the global pooled prevalence of 65.33% reported in a systematic review and meta-analysis involving more than 2.2 million patients with T2DM [5]. These differences may reflect variations in the age and metabolic characteristics of study populations, study settings, and diagnostic approaches.

The prevalence in our study was higher than the 48.1% pooled prevalence reported in an African systematic review and meta-analysis of MAFLD among people with T2DM. The pooled prevalence was 44.7% in sub-Saharan Africa and 47.0% among studies using ultrasonography [9]. The prevalence was also higher than the 34% reported in an Indian study of adults with T2DM and BMI ≥ 25 kg/m², in which MASLD was diagnosed using abdominal ultrasonography [15]. Differences in participant characteristics, metabolic risk profiles, sampling methods, and diagnostic criteria may explain the observed variation in prevalence estimates.

Taken together, these findings indicate that MASLD is common among adults with T2DM in Uganda, with a prevalence broadly comparable to recent estimates from other diabetic populations. The consistency of our findings with ultrasound-based estimates from Saudi Arabia and India, as well as the Tanzanian estimate obtained using transient elastography, supports the substantial burden of MASLD among adults with T2DM. In resource-limited settings, incorporating abdominal ultrasonography into the assessment of MASLD as part of routine diabetes care may facilitate earlier identification and management of patients at risk of progressive liver disease.

Sonographic grading of hepatic steatosis

In this study, grade 1 steatosis was the predominant sonographic grade, accounting for 76.1% of participants with MASLD, followed by grade 2 (21.3%) and grade 3 (2.6%). This pattern is comparable to

a recent study from Saudi Arabia, where grade 1 steatosis accounted for 69.6% of participants with MASLD, followed by grade 2 (27.8%) and grade 3 (2.6%) [14]. The predominance of mild steatosis in both studies suggests a broadly similar distribution of sonographic severity among adults with T2DM.

Conversely, our findings differ from those reported in Egypt, where among adults with T2DM and ultrasound-diagnosed MAFLD, 30.9% had grade 1, 49.6% had grade 2, and 19.4% had grade 3 steatosis [16]. Similarly, a study from Nigeria reported grade 1, grade 2, and grade 3 steatosis in 16.3%, 32.5%, and 20.0% respectively, among patients with T2DM and ultrasound-diagnosed non-alcoholic fatty liver disease (NAFLD) [17]. The higher proportions of moderate and severe steatosis in these studies may reflect differences in age distribution, obesity, duration and control of diabetes, metabolic risk profiles, referral patterns, and ultrasound grading criteria.

The predominance of mild sonographic steatosis in our study indicates that most participants with MASLD had detectable hepatic fat accumulation without advanced sonographic steatosis. However, ultrasound grading reflects the extent of hepatic steatosis and does not directly assess liver fibrosis or steatohepatitis. Therefore, the low proportion of grade 3 steatosis should not be interpreted as absence of clinically significant liver disease. These findings highlight the potential value of abdominal ultrasonography for identifying hepatic steatosis during routine diabetes care, followed by assessment with additional non-invasive tools for liver fibrosis when clinically indicated.

Factors associated with MASLD

In this study, increasing age, hypertension, obesity, poor glycemic control, and elevated triglyceride levels were independently associated with MASLD.

Increasing age was independently associated with MASLD in our study. This finding is consistent with a study from Pakistan, where increasing age was associated with greater NAFLD severity among adults with and without T2DM [18]. More recent MASLD-specific evidence from India also found that increasing age was independently associated with advanced fibrosis among patients with biopsy-proven MASLD, including those with T2DM [19]. Age-related changes in insulin sensitivity, lipid metabolism, and mitochondrial function, together with cumulative exposure to metabolic risk factors, may contribute to hepatic fat accumulation and disease progression [20].

Hypertension was also independently associated with MASLD, consistent with findings from a study in Tanzania, where elevated diastolic blood pressure was independently associated with MASLD among adults with T2DM [13]. This finding is also supported by a systematic review and meta-analysis of observational cohort studies, which found that hypertension was independently associated with an increased risk of incident NAFLD [21]. The relationship between hypertension and hepatic steatosis may be explained by shared mechanisms including insulin resistance, endothelial dysfunction, chronic inflammation, and activation of the renin–angiotensin–aldosterone system, which promote hepatic lipid accumulation and elevated blood pressure. Hypertension may also impair hepatic microcirculation, potentially contributing to progression of fatty liver disease [22, 23].

Obesity (BMI ≥ 30 kg/m²) was similarly independently associated with MASLD, consistent with findings from studies conducted in Saudi Arabia and Egypt, where higher BMI or obesity was associated with MASLD or MAFLD among adults with T2DM [12, 16]. Studies from Scotland, Bahrain, and Ethiopia also reported BMI or obesity as factors independently associated with NAFLD among adults with T2DM [24–26]. Obesity increases adipose tissue lipolysis, resulting in excessive delivery of free fatty acids to the liver, while visceral adiposity promotes inflammatory cytokine production and reduces adiponectin, thereby worsening insulin resistance and hepatic fat accumulation [27].

Poor glycemic control (HbA1c $\geq 7\%$) was likewise independently associated with MASLD, consistent with findings from studies conducted in Egypt, Nigeria and Scotland, where higher HbA1c or poor glycemic control was associated with NAFLD or MAFLD among adults with T2DM [16, 17, 24]. A recent prospective study of patients with biopsy-proven MASLD also found that higher mean HbA1c was independently associated with progression of liver stiffness, particularly among individuals with T2DM [28]. Persistent hyperglycemia may promote hepatic steatosis through increased hepatic de novo lipogenesis, oxidative stress, and impaired mitochondrial fatty acid oxidation, while worsening insulin resistance and triglyceride accumulation within hepatocytes [29].

Elevated triglyceride levels (≥ 2.3 mmol/l) were additionally independently associated with MASLD, consistent with findings from studies conducted in Egypt and Romania, where higher triglyceride levels were independently associated with MAFLD among adults with T2DM [16, 30]. Similarly, studies from Scotland, China, and Ethiopia reported elevated triglycerides as independent factors associated with NAFLD or MASLD among adults with T2DM [24, 31, 32]. Elevated circulating triglycerides reflect underlying insulin resistance and increased hepatic lipid flux, while increased hepatic triglyceride synthesis, reduced very-low-density lipoprotein clearance, and impaired fatty acid oxidation contribute to hepatic lipid accumulation [33].

Strengths and Limitations

To our knowledge, this is the first multicenter study in Uganda to evaluate the prevalence, sonographic grading, and factors associated with MASLD among adults with T2DM. The inclusion of participants from two regional referral hospitals in different regions of Uganda provides broader geographic representation and may enhance the generalizability of the findings. In addition, objective assessment of hepatic steatosis by ultrasonography and measurement of key metabolic variables, including BMI, HbA1c, and triglycerides, strengthened the validity of the findings. The use of sonographic grading also provided information on the distribution of steatosis severity beyond overall MASLD prevalence.

Several limitations should be acknowledged. Because of the cross-sectional design, temporal or causal relationships between the identified factors and MASLD cannot be established. The hospital-based nature of the study introduces potential selection bias, as patients attending regional referral hospitals may not be representative of the broader population of adults with T2DM, limiting generalizability. Ultrasonography was used to assess hepatic steatosis rather than liver biopsy, magnetic resonance

imaging, or elastography. Although ultrasonography is non-invasive, accessible, and suitable for screening in resource-limited settings, it has limited sensitivity for mild steatosis and cannot assess steatohepatitis or liver fibrosis. Furthermore, liver function tests and a complete lipid profile were not performed because of financial constraints, limiting assessment of hepatic biochemical abnormalities and other lipid parameters. Overall, the study provides useful multicenter data on the burden and metabolic factors associated with MASLD among adults with T2DM in Uganda.

Conclusion

The prevalence of MASLD among adults with T2DM at KRRH and HRRH was 54.5%. Most participants with MASLD had grade 1 (mild) sonographic steatosis. Increasing age, hypertension, obesity, poor glycemic control ($\text{HbA1c} \geq 7\%$), and elevated triglyceride levels ($\geq 2.3 \text{ mmol/l}$) were independently associated with MASLD. These findings highlight the importance of incorporating abdominal ultrasonography into the assessment of MASLD, alongside comprehensive management of metabolic risk factors, as part of routine diabetes care to facilitate early identification and management of patients at risk of progressive liver disease.

Recommendations

1. Routine assessment for MASLD with abdominal ultrasonography should be considered as part of diabetes care, particularly for patients with increasing age, obesity, hypertension, poor glycemic control, or triglyceride levels $\geq 2.3 \text{ mmol/l}$.
2. Adults with T2DM should receive counseling and regular follow-up focused on weight management, healthy dietary practices, physical activity, and optimal glycemic and lipid control to reduce metabolic risk and hepatic fat accumulation.
3. Regional referral hospitals should establish appropriate pathways for further assessment and management of patients identified with MASLD, including evaluation for liver fibrosis when clinically indicated.
4. Further multicenter, prospective studies incorporating non-invasive assessment of liver fibrosis are needed to establish temporal relationships, assess disease progression, and guide national MASLD screening and management strategies.

Abbreviations

MASLD

Metabolic dysfunction-associated steatotic liver disease

T2DM

Type 2 diabetes mellitus

HRRH

Hoima Regional Referral Hospital

KRRH

Kayunga Regional Referral Hospital

HbA1c

Glycated hemoglobin

MAFLD

Metabolic dysfunction-associated fatty liver disease

NAFLD

Non-alcoholic fatty liver disease

BMI

Body mass index

Declarations

Ethical Approval and Consent to Participate

Ethical clearance for this study was obtained from the Research Ethics Committee of Kampala International University (Ref No: KIU-2025-1933). Administrative permission to conduct the study was also obtained from the management of KRRH and HRRH. Written informed consent was obtained from all participants prior to enrollment in the study. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Consent for Publication

Written informed consent for publication of the study findings and accompanying data was obtained from all participants.

Competing Interests

The authors declare that they have no competing interests.

Funding

This research received no specific funding from any public, commercial, or not-for-profit organizations.

Author Contribution

AAO conceived and designed the study, collected and analyzed data, and drafted the manuscript. AAS, TH, FDA, AGH, MJ, AHK, IMO, EC, AHA, SHI, ZAA and LN contributed to data collection and validation. MEM, FAA, and VEM provided supervision, critical revisions, and methodological guidance. AAS assisted with visualization and manuscript editing. VEM and AAS performed statistical analysis and data visualization. All authors reviewed and approved the final manuscript.

Acknowledgement

The authors sincerely thank all study participants and the staff of Kayunga and Hoima regional Referral Hospitals for their valuable contributions to this research.

Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

References

1. Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. *Hepatology*. 2023;78(6):1966–86. 10.1097/HEP.0000000000000520.
2. European Association for the Study of the Liver (EASL), European Association for the Study of Diabetes (EASD). European Association for the Study of Obesity (EASO). EASL-EASD-EASO Clinical Practice Guidelines on the Management of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). *Obes Facts*. 2024;17(4):374–444. 10.1159/000539371.
3. Cusi K, Abdelmalek MF, Apovian CM, Balapattabi K, Bannuru RR, Barb D, et al. Metabolic dysfunction-associated steatotic liver disease (MASLD) in people with diabetes: the need for screening and early intervention. A consensus report of the American Diabetes Association. *Diabetes Care*. 2025;48(7):1057–82. 10.2337/dci24-0094.
4. Singhai A, Meena R, Joshi R, Khadanga S, Jhaj R, Goyal A, et al. Prevalence of metabolic dysfunction associated steatotic liver disease in type 2 diabetes mellitus in India: a systematic review and meta-analysis. *Indian J Med Res*. 2026;164(1):72–80. 10.25259/IJMR_2480_2025.
5. Younossi ZM, Golabi P, Price JK, Owringi S, Gundu-Rao N, Satchi R, et al. The global epidemiology of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among patients with type 2 diabetes. *Clin Gastroenterol Hepatol*. 2024;22(10):1999–e20108. 10.1016/j.cgh.2024.03.006.
6. Potra M, Popescu S, Timar B, Lazar S, Gaita L, Braha A, et al. Determinants of metabolic dysfunction-associated steatotic liver diseases in patients with type 2 diabetes mellitus. *J Clin Med*. 2026;15(16):6421. 10.3390/jcm15166421.
7. Kang CC, Wang TE, Liu CY, Chen MJ, Wang HY, Chang CW, et al. Update on imaging-based noninvasive methods for assessing hepatic steatosis in nonalcoholic fatty liver disease. *J Med Ultrasound*. 2024;32(2):116–20. 10.4103/jmu.jmu_88_23.
8. Spencer-Sandino M, Balakrishnan M, Wynne D, et al. Inter- and intra-observer agreement in ultrasound diagnosis of steatotic liver disease: implications for screening in resource-limited settings. *Sci Rep*. 2025;15:29819. 10.1038/s41598-025-07862-1.

9. Ekpor E, Akyirem S, Adade Duodu P. Prevalence of metabolic dysfunction-associated fatty liver disease and its association with glycemic control in persons with type 2 diabetes in Africa: a systematic review and meta-analysis. *PLOS Glob Public Health*. 2024;4(5):e0002835. 10.1371/journal.pgph.0002835.
10. Enriquez R, Homsy M, Ssekubugu R, Nabukalu D, Zeebari Z, Marrone G, et al. Prevalence and risk factors of metabolic dysfunction-associated steatotic liver disease in south Central Uganda: a cross-sectional survey. *Aliment Pharmacol Ther*. 2024;59(9):1111–21. 10.1111/apt.17931.
11. Daniel WW, Cross CL. Determination of sample size for estimating proportions. *Biostatistics: a foundation for analysis in the health sciences*. 8th ed. New York: Wiley; 1999. pp. 189–90.
12. Elnasieh AM, Almesned M, Al Hazmi AN, Alturki A, Alhawaidi FI, Alhadlq RK, et al. Prevalence and risk factors of metabolic dysfunction-associated steatotic liver disease in patients with type 2 diabetes mellitus at a tertiary center in Saudi Arabia: cross-sectional questionnaire study. *JMIR Diabetes*. 2025;10:e77772. 10.2196/77772.
13. Malindisa E, Kafumu I, Rweyendera A, Mkemangwa E, Majinge D, Nkandala I, et al. Metabolic dysfunction-associated steatotic liver disease in patients with type 2 diabetes mellitus in Tanzania: prevalence and predictors. *BMC Endocr Disord*. 2026;26(1):67. 10.1186/s12902-026-02179-0.
14. Hadadi I, Adam M, Musa J, Gareeballah M, Alqahtani A, Kanbayti M. Exploring the prevalence and coexistence of metabolic dysfunction-associated steatotic liver disease in type 2 diabetes mellitus patients using ultrasound: a cross-sectional study. *Curr Med Imaging*. 2025;21:e15734056354807. 10.2174/0115734056354807241217043210.
15. Budhwani P, Goswani B, Debnath S, Shukla A. Prevalence of metabolic dysfunction-associated steatotic liver disease among overweight and obese patients with type 2 diabetes mellitus. *Cureus*. 2025;17(12):e99670. 10.7759/cureus.99670.
16. Semeya AA, Hafez RSAA, Ewais SHM, Mostafa SM, Eldeeb A, Elgamal R, et al. Screening for metabolic-associated fatty liver disease in type 2 diabetes patients using non-invasive scores and ultrasound: a cross-sectional study in Egypt. *BMC Gastroenterol*. 2025;25(1):639. 10.1186/s12876-025-03844-9.
17. Afolabi BI, Ibitoye BO, Ikem RT, Omisore AD, Idowu BM, Soyoye DO. The relationship between glycaemic control and non-alcoholic fatty liver disease in Nigerian type 2 diabetic patients. *J Natl Med Assoc*. 2018;110(3):256–64. 10.1016/j.jnma.2017.06.001.
18. Iqbal MZ, Irfan A, Ejaz M, Gardazi A, Syed HBS. Evaluating non-alcoholic fatty liver disease in individuals with and without type 2 diabetes: comparative cross-sectional study using ultrasound imaging. *Life Sci*. 2025;6(3):344–9. 10.37185/LnS.1.1.772.
19. Choudhary NS, Duseja A, De A, Mehta M, Singh SP, Anirvan P, et al. Type 2 diabetes mellitus and risk of advanced fibrosis in patients with metabolic dysfunction-associated steatotic liver disease (MASLD): the results of multi-centre Indian Consortium on MASLD (ICOM-D) study. *J Clin Exp Hepatol*. 2026 Mar-Apr;16(2):103468. 10.1016/j.jceh.2025.103468.

20. He QJ, Li YF, Zhao LT, Lin CT, Yu CY, Wang D. Recent advances in age-related metabolic dysfunction-associated steatotic liver disease. *World J Gastroenterol*. 2024;30(7):652–62. 10.3748/wjg.v30.i7.652.
21. Li G, Peng Y, Chen Z, Li H, Liu D, Ye X. Bidirectional association between hypertension and NAFLD: a systematic review and meta-analysis of observational studies. *Int J Endocrinol*. 2022;2022:8463640. 10.1155/2022/8463640.
22. Nakagami H. Mechanisms underlying the bidirectional association between nonalcoholic fatty liver disease and hypertension. *Hypertens Res*. 2023;46:539–41. 10.1038/s41440-022-01117-6.
23. Nasiri-Ansari N, Androutsakos T, Flessa CM, Kyrou I, Siasos G, Randeva HS, et al. Endothelial cell dysfunction and nonalcoholic fatty liver disease (NAFLD): a concise review. *Cells*. 2022;11(16):2511. 10.3390/cells11162511.
24. Williamson RM, Price JF, Glancy S, Perry E, Nee LD, Hayes PC, et al. Prevalence of and risk factors for hepatic steatosis and nonalcoholic fatty liver disease in people with type 2 diabetes: the Edinburgh Type 2 Diabetes Study. *Diabetes Care*. 2011;34(5):1139–44. 10.2337/dc10-2229.
25. Mohamed AM, Isa HM, Ali MS, Dadi A, Kadhim Z. Prevalence of non-alcoholic fatty liver disease among patients with diabetes mellitus attending primary health care centers in Bahrain. *Oman Med J*. 2022;37(2):e350. 10.5001/omj.2022.53.
26. Berhe EA, Sema RA, Tesfaye YM, Berhane AA, Dawit M, Gebrehiwot EM, et al. Assessing nonalcoholic fatty liver disease in type 2 diabetes patients at a tertiary hospital, Addis Ababa, Ethiopia. *J Diabetes Res*. 2025;2025:5555842. 10.1155/jdr/5555842.
27. Saponaro C, Sabatini S, Gaggini M, Carli F, Rosso C, Positano V, et al. Adipose tissue dysfunction and visceral fat are associated with hepatic insulin resistance and severity of NASH even in lean individuals. *Liver Int*. 2022;42(11):2418–27. 10.1111/liv.15377.
28. Huang DQ, Wilson LA, Behling C, Amangurbanova M, Kleiner DE, Kowdley KV, et al. Liver stiffness progression in biopsy-proven metabolic dysfunction-associated steatotic disease among people with diabetes versus people without diabetes: a prospective multicenter study. *Hepatology*. 2025;81(5):1553–63. 10.1097/HEP.0000000000001015.
29. Demir M, Bornstein SR, Mantzoros CS, Perakakis N. Liver fat as risk factor of hepatic and cardiometabolic diseases. *Obes Rev*. 2023;24(10):e13612. 10.1111/obr.13612.
30. Efrem IC, Moța M, Vladu IM, Mitrea A, Clenciu D, Timofticiuc DCP, et al. A study of biomarkers associated with metabolic dysfunction-associated fatty liver disease in patients with type 2 diabetes. *Diagnostics (Basel)*. 2022;12(10):2426. 10.3390/diagnostics12102426.
31. Chen L, Jiang L. Clinico-pathological features and related risk factors of type 2 diabetes mellitus complicated with nonalcoholic fatty liver. *Pak J Med Sci*. 2022;38(7):1771–5. 10.12669/pjms.38.7.6289.
32. Aydagnum GB, Tefera AS, Getahun GT, Melaku EE. Predictive factors of metabolic dysfunction-associated steatotic liver disease (MASLD) among individuals with cardiometabolic risk factors:

multicenter cross-sectional study, North East Ethiopia. BMC Gastroenterol. 2026;26(1):161. 10.1186/s12876-026-04656-1.

33. Heeren J, Scheja L. Metabolic-associated fatty liver disease and lipoprotein metabolism. Mol Metab. 2021;50:101238. 10.1016/j.molmet.2021.101238.

Figures

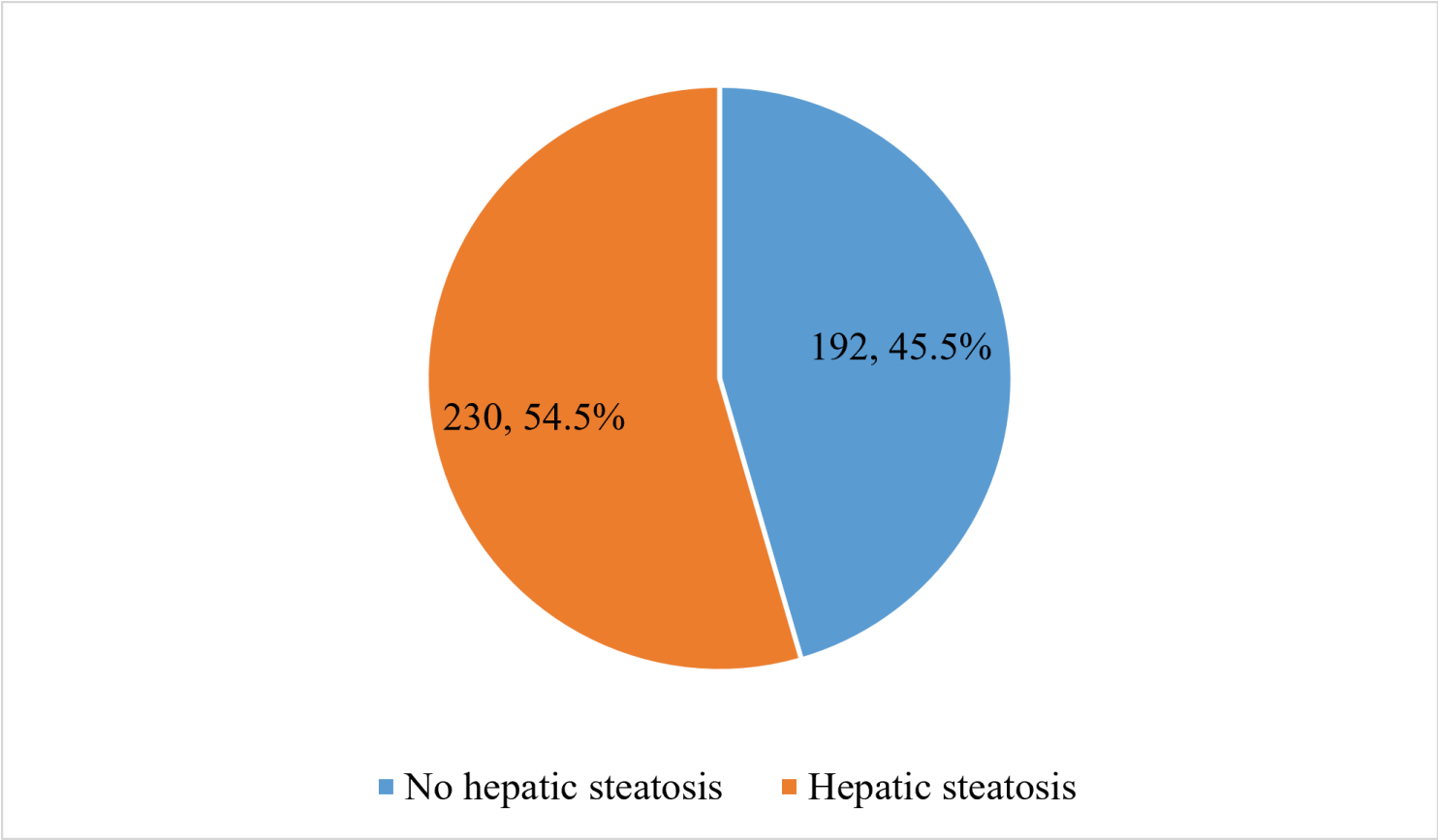


Figure 1

Prevalence of MASLD among adults with T2DM.

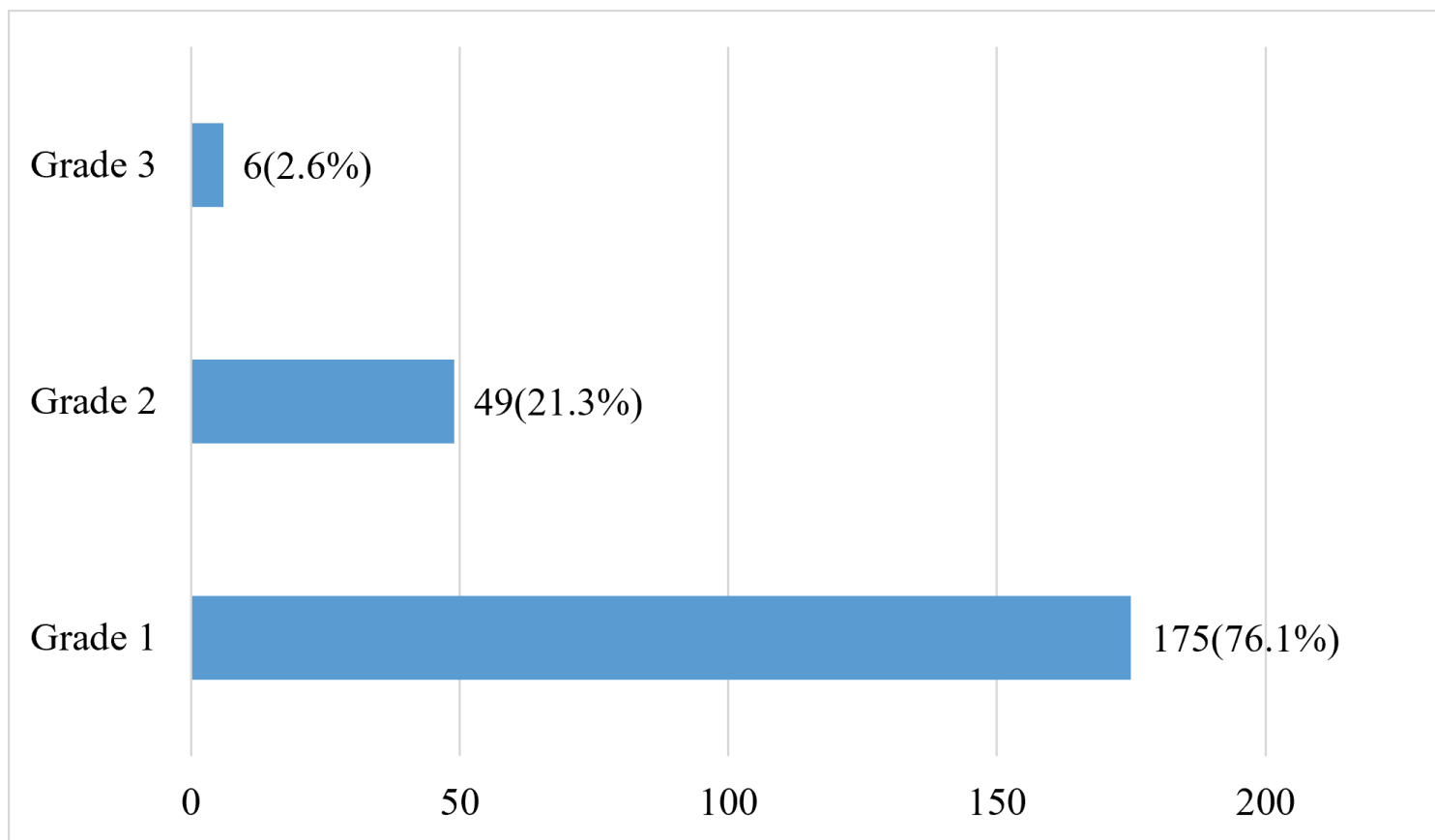


Figure 2

Sonographic grading of hepatic steatosis among adults with T2DM.