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Original article

External Validation and Comparisons of Hepatic Steatosis Index (HSI), Dallas Steatosis Index (DSI), and Triglyceride-Glucose (TyG) Index for the Diagnosis of Steatotic Liver Disease (SLD) in Thai Individuals with Cardiometabolic Risk

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Short running title: Validating SLD scores in cardiometabolic risk

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Abstract

Objective: Individuals with cardiometabolic risk are a target population for steatotic liver disease (SLD) screening. Fatty Liver Index is a widely accepted noninvasive score to identify SLD; however, it relies on parameters that are often unavailable in general. We aimed to validate and compare the diagnostic performances of the hepatic steatosis index (HSI), Dallas steatosis index (DSI), and triglyceride-glucose index (TyG) for detecting steatotic liver disease (SLD) in Thai individuals with cardiometabolic risk and to evaluate advanced fibrosis risk according to SLD subclassifications.

Materials and Methods: This cross-sectional study analyzed adults with ≥1 cardiometabolic risk who underwent abdominal ultrasonography (USG) at a university hospital between 2019 and 2023. The diagnostic performances of HSI, DSI, and TyG were evaluated using the area under the receiver operating characteristic curve (AUROC).

Results: Among 5,388 eligible individuals, the prevalence of SLD was 49.4%. DSI demonstrated the highest performance (AUROC: 0.813, 95% confidence interval [CI]: 0.801–0.824), surpassing HSI (AUROC: 0.795, p-value<0.001) and TyG (AUROC: 0.729, p-value<0.001). A DSI cutoff <0.2 yielded 84.4% (95%CI:83.2-85.7%) sensitivity for ruling out SLD, whereas a cutoff ≥0.5 provided 92.5% (95%CI:91.6-93.4%) specificity for ruling in disease. Among 1,355 participants with available alcohol data, the proportion of individuals at high risk of advanced fibrosis (FIB-4 >2.67) was notably higher in the metabolic- and alcohol-associated liver disease (10.7%) and alcohol-associated liver disease (8.1%) groups than in the metabolic dysfunction-associated steatotic liver disease group (1.5%).

Conclusion: DSI outperforms HSI and TyG for detecting SLD and can serve as an effective screening tool in primary care settings without USG.

Keywords: Metabolic Dysfunction-Associated Steatotic Liver Disease; Metabolic dysfunction-associated steatohepatitis; MetALD; Non-Invasive; Dallas Steatosis Index; Hepatic Steatosis Index; Scoring system; Diagnostic test; Metabolic syndrome; Non-alcoholic fatty liver disease

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Introduction

Steatotic liver disease (SLD) represents a significant global health burden, with an estimated worldwide prevalence of 30.0–40.0% among adults, rising to over 50.0% in individuals with cardiometabolic risk factors [1–3]. This condition encompasses a spectrum of liver derangement ranging from simple hepatic steatosis to steatohepatitis with fibrosis and, in its most severe form, progression to cirrhosis and hepatocellular carcinoma (HCC). The clinical presentation of SLD is characteristically asymptomatic in its early stages, making early detection challenging yet crucial for preventing any long-term complications[4]. The disease has emerged as a leading cause of chronic liver disease globally, with metabolic dysfunction-associated steatotic liver disease (MASLD) now representing one of the most common etiologies for liver transplantation and HCC in many countries, surpassing viral hepatitis [5,6].

The identification of individuals at risk of SLD is particularly important in primary care settings [7–9] where providers serve as the frontline for recognizing and screening high-risk populations, including those with cardiometabolic risks [10]. However, significant screening gaps remain; for instance, only 25.7–46.0% of primary care practitioners screen patients with diabetes and obesity, despite recognizing the disease's clinical importance[11]. Ultrasonography (USG) is commonly used to detect hepatic steatosis; however, the limited availability of radiologists, particularly in primary care settings, constrains this approach. In such contexts or in large-scale screenings where USG is not readily accessible, serum biomarkers may serve as practical alternatives for identifying SLD[8].

The fatty liver index (FLI) has emerged as the most widely accepted non-invasive scoring system for identifying individuals with SLD at the population level [8,12–15]. Multiple validation studies have confirmed its utility across different ethnic groups, with particularly robust performance in Asian populations [14,16]. However, the practical implementation of the FLI in routine clinical practice has significant limitations. The inclusion of waist circumference measurements and GGT levels poses logistical challenges, as these parameters are not routinely measured in many primary care settings. Waist circumference assessment requires standardized measurement techniques and additional consultation time, while GGT testing is often not included in standard laboratory panels, potentially increasing costs and delaying diagnosis[17]. These practical

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constraints have prompted the development and validation of alternative scoring systems that incorporate the readily available clinical parameters, including the hepatic steatosis index (HSI), Dallas steatosis index (DSI), and triglyceride-glucose index (TyG), all of which utilize routinely measured laboratory values and the basic anthropometric data commonly available in standard clinical practice [17–19]. In this study, we aimed to validate and compare these scoring systems for the diagnosis of SLD in Thai individuals with cardiometabolic risk factors.

Material and Methods

Study design and population

This study was a secondary analysis of a previously described retrospective cross-sectional cohort of individuals aged 18 years or older who underwent health check-up programs, including abdominal ultrasonography (USG), at the Srivejchavat Premium Center, Songklanagarind Hospital, a tertiary care university hospital in Southern Thailand, between January 1, 2019, and December 31, 2023 [20].

To address the specific aims of the present study—namely, evaluating non-invasive scoring systems among individuals at cardiometabolic risk—we restricted our analysis to participants presenting with at least one cardiometabolic risk factor. Cardiometabolic risk was defined in accordance with the latest multisociety steatotic liver disease (SLD) nomenclature consensus statement[21], defined as: (1) BMI $\geq$23 kg/m^2 or waist circumference $\geq$90 cm in men and $\geq$80 cm in women, (2) fasting plasma glucose (FPG) $\geq$100 mg/dL, type 2 diabetes or treatment for type 2 diabetes, (3) plasma triglyceride (TG) $\geq$150 mg/dL or use of lipid-lowering therapy, (4) high-density lipoprotein (HDL) $\leq$40 mg/dL in men or $\leq$50 mg/dL in women or current lipid-lowering treatment, (5) blood pressure $\geq$130/85 mmHg or treatment for hypertension.

Individuals positive for HBsAg or anti-HCV, or with missing data required to calculate HSI, DSI, or TyG, were excluded. Consequently, the study population represents a targeted sub-cohort of the original dataset (Fig 1).

To evaluate the burden of SLD across clinical profiles, we evaluated SLD prevalence stratified by each individual cardiometabolic risk component. Furthermore, while SLD screening

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in patients with type 2 diabetes is well established in clinical guidelines, the yield of screening in non-diabetic individuals with obesity is less defined. Because diabetes heavily overlaps with obesity, we specifically evaluated a subgroup of obese individuals without diabetes to isolate the impact of adiposity alone and explore whether screening is warranted prior to the onset of frank glycemic dysregulation.

Data collection and scoring system calculation

Data on eligible patients, including baseline characteristics, laboratory results, and abdominal USG findings, were extracted from the Institute's Digital Innovation and Data Analytics (DIDA) unit. Variables used to calculate the scores included: for HSI – sex, diabetes status, BMI, aspartate aminotransferase (AST), and alanine aminotransferase (ALT); for DSI – age, sex, BMI, diabetes status, hypertension status, ALT, FPG, TG, ethnicity (all participants in this study were categorized as Asian); and for TyG – TG and FPG. Each scoring system was calculated according to the formulas described in the original publications[17,18,22].

Definitions of terms

The presence or absence of hepatic steatosis was determined using USG findings, based on qualitative sonographic features—specifically increased liver echogenicity relative to the renal cortex, attenuation of the ultrasound beam, and vascular blurring—following the methodology described in our previous publication[20]. The risk of advanced fibrosis among individuals with SLD was stratified according to the fibrosis-4 (FIB-4) index as follows: low risk (<1.3, or <2.0 for those over 65 years), indeterminate risk (1.3–2.67, or 2.0–2.67 for those over 65 years), and high risk (≥2.67)(10). The FIB-4 index was calculated using a standard formula[23].

Alcohol consumption data were not systematically collected due to the retrospective nature of the study. In routine clinical practice at our institution, no standardized questionnaire is used to assess alcohol intake; however, attending physicians may inquire about patients' alcohol consumption, including type and quantity. Among individuals with SLD for whom alcohol data were available, participants were further classified as non-drinkers, ex-drinkers, or current drinkers.

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For current drinkers, alcohol intake was quantified in grams per day (g/day) to categorize individuals as having MASLD (≤20 g/day for women, ≤30 g/day for men), MASLD with increased alcohol intake or metabolic- and alcohol-associated liver disease (MetALD; 20–50 g/day for women, 30–60 g/day for men), or alcohol-related liver disease (ALD; >50 g/day for women, >60 g/day for men), according to established criteria[21]. Non-drinkers and ex-drinkers were classified into the MASLD category.

Statistical analysis

All data analyses were carried out using R program version 4.2.2 (R Core Team, Vienna, Austria). Descriptive statistics summarized baseline characteristics and the proportions of individuals in each SLD risk category. Categorical data were presented as counts and percentages, while continuous data were reported as means with standard deviations (S.D.) or medians with interquartile ranges (IQR) when normality assumptions were not met. Differences between individuals with and without SLD were assessed using the Chi-square or Fisher's exact test for categorical variables, and t-tests or rank-sum tests for continuous variables. For three-group comparisons across SLD subtypes (MASLD, MetALD, ALD), the Chi-square test (or Fisher's exact test) was used for categorical variables and the Kruskal-Wallis test for continuous variables. The diagnostic performances of DSI, HSI, and TyG for detecting SLD were evaluated using AUROC, with sensitivity and specificity determined at specified cutoff values. Comparisons of the scoring systems' performances were conducted using DeLong's test.

Ethics statement

As this study involved secondary analysis of previously collected retrospective data, it was adjudicated exempt from human subject research per the Office of Human Research Ethics Committee (HREC), Prince of Songkla University (REC 68-194-14-1). Informed consent was not required, and a waiver of consent was granted by the HREC. Patient confidentiality was maintained by anonymizing all recorded information, and results were reported in aggregate form.

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Results

Of the 6183 unique individuals who participated in health check-up programs involving USG over the 4-year study period, a total of 795 individuals were excluded due to positive HBV or HCV serology (N=133), missing parameters for DSI, HSI, TyG, or FIB-4 calculation (N=69), and absence of cardiometabolic risk (N=593). Consequently, 5,388 individuals were eligible and included in the analysis (Figure 1).

Prevalence of SLD and participant characteristics

Among the 5,388 individuals with at least one cardiometabolic risk factor, 2,662 (49.4%) had SLD detected by USG. The characteristics of individuals with and without SLD are summarized in Table 1. Although all participants had cardiometabolic risk factors, those with SLD exhibited more pronounced metabolic derangements than those without SLD. Seventy percent of individuals with SLD had three or more cardiometabolic risk factors, compared with 64.8% of those without SLD who had fewer than three risk factors. When examining each component of cardiometabolic risk, the prevalence of SLD varied, as shown in Fig 2. The highest prevalence was observed among individuals with dysglycemia (66.4%, 95% CI: 64.2–68.5%). We were also particularly interested in individuals with obesity (based on BMI criteria) who did not have diabetes; the prevalence of SLD in this group was 51.7% (95%CI: 49.7-53.7%).

Diagnostic performance of biomarkers of interest

The ROC curves of DSI, HSI, and TyG for diagnosing SLD using USG as the reference standard are shown in Fig 3. DSI demonstrated good diagnostic performance with an AUROC of 0.813 (95% CI: 0.801–0.824), which was significantly higher than that of HSI (AUROCs of 0.795, 95% CI: 0.784–0.807, p-value<0.001) and TyG (AUROC 0.729 (95% CI: 0.716–0.743, p-value<0.001). In pairwise comparison, HSI also significantly outperformed TyG ($p < 0.001$).

Given that DSI outperformed the other two scoring systems in detecting SLD, we evaluated its clinical utility using the cutoff values established in the original derivation study rather than re-

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deriving optimal thresholds from our dataset. This approach maintains strict external validation standards, avoiding model overfitting and reflecting real-world clinical application. Applying this dual-cutoff approach, 33.3% (n = 1,794) of participants were categorized as low risk (< 0.2), 38.4% (n = 2,067) fell into the indeterminate zone (0.2–0.5), and 28.3% (n = 1,527) were categorized as high risk (≥0.5). The sensitivity, specificity, positive predictive value, and negative predictive value of DSI at these specified cutoffs are summarized in Table 2.

SLD sub-classifications and liver fibrosis status

Among the 2,662 individuals with SLD, the proportions categorized by FIB-4 strata as low, indeterminate, and high risk of advanced fibrosis were 82.6% (n=2200), 15.8% (n=421), and 1.5% (n=41), respectively.

Alcohol consumption data were available for 1,355 (50.9%) individuals with SLD. Of these, 1290 (95.2%), 28 (2.1%), and 37 (2.7%) were classified as having MASLD, MetALD, and ALD, respectively. The characteristics of the individuals in each subclassification are listed in Table 3. Those with MetALD and ALD were generally younger than those with MASLD. As expected, a male preponderance and significantly higher TG levels were observed in the MetALD and ALD groups compared with the MASLD group. Furthermore, the proportion of individuals at high risk of advanced liver fibrosis differed significantly across the subgroups (1.5% in MASLD [19/1290, exact 95%CI: 0.9%–2.3%], 10.7% in MetALD [3/28, exact 95%CI: 2.3%–28.2%], and 8.1% in ALD [3/37, exact 95%CI: 1.7%–21.9%]; Chi-square test $p < 0.001$).

Discussion

Our study found that the prevalence of SLD in individuals participating in health check-up programs in Thailand who had at least one cardiometabolic risk factor was 49.4%. Using USG as the reference standard, DSI demonstrated good diagnostic performance for detecting SLD in the at-risk population, with an AUROC of 0.813 (95% CI: 0.801–0.824), significantly outperforming HSI and TyG. Among individuals with SLD and available alcohol consumption data, 95.2%, 2.1%, and 2.7% were classified as having MASLD, MetALD, and ALD, respectively. The proportion of

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individuals at high risk of advanced fibrosis was statistically significantly higher in the MetALD (10.7%) and ALD (8.1%) groups than in the MASLD group (1.5%; $p < 0.001$).

The prevalence of SLD in our study was higher than that reported in previous studies of general health check-up populations[17,24,25]. This difference is likely attributable to our inclusion criteria, which specifically targeted individuals with at least one cardiometabolic risk factor, a group inherently at higher risk of developing SLD. Notably, among the broader health check-up population screened at our center (N = 6183), the vast majority of individuals exhibited at least one cardiometabolic risk factor, underscoring the high prevalence of metabolic dysfunction in general screening populations. Consistent with this rationale and previous studies [25,26], our analysis confirmed that participants with SLD had significantly worse cardiometabolic profiles, characterized by higher BMI and waist circumference, as well as a greater prevalence of diabetes, hypertension, and dyslipidemia. These findings reinforce the shared pathophysiological mechanisms linking SLD and cardiometabolic disorders.

All three scoring systems (DSI, HSI, and TyG), which are based on routine primary care parameters, showed satisfactory AUROCs for the diagnosis of SLD in this external validation study, consistent with a previous study[19]. However, their performance was slightly lower than that observed in the original development studies[17,18]. This finding is common in external validation studies and may be attributable to differences in ethnicity, study demographics, and the reference standards used to diagnose SLD. Nevertheless, these tools remain valuable for use in primary care and population-based settings.

In our study, DSI achieved the highest AUROC, significantly outperforming earlier-developed HSI and TyG scores. To the best of our knowledge, no prior study has directly compared these three scoring systems. The most relevant study, conducted by Lee et al. [27], reported comparably high diagnostic accuracy for DSI, HSI, and TyG-BMI, with AUROC exceeding 0.89 for all three systems. In contrast to our study, Lee et al. focused on Korean adults younger than 35 years, with 98% of the study population being male. Our cohort was considerably older and included a more balanced sex distribution, which may have contributed to the differences in the findings.

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Furthermore, our findings are consistent with previous research validating DSI in the United States and United Kingdom populations, which demonstrated satisfactory diagnostic performance, with AUROCs exceeding 0.8 for SLD detection [28,29] and a numerically higher AUROC compared with HSI [28]. The superior diagnostic accuracy of DSI may be attributable to the number of variables included: DSI incorporates nine parameters, whereas HSI employs four, and TyG uses only two.

A DSI score below 0.2 demonstrated elevated sensitivity and a strong negative predictive value, indicating that a low DSI threshold can effectively identify individuals at low risk of SLD, potentially obviating the need for further testing. In contrast, a DSI score above 0.5 demonstrated high specificity and a high PPV, suggesting that individuals with a positive score should undergo additional evaluation for SLD. This balance highlights the utility of DSI as both a screening and a confirmatory tool. Importantly, 38.36% (n = 2,067) of our cohort fell into the grey zone (DSI 0.2–0.5). Although these individuals would require secondary diagnostic evaluation (such as ultrasound), this dual-cutoff strategy successfully triaged 61.64% of the screened population into definitive risk categories without immediate imaging. In resource-constrained primary care settings, utilizing DSI as a first-line triage tool can substantially reduce unnecessary ultrasounds by over 60%, focusing advanced diagnostic pathways specifically on the indeterminate subgroup.

In the SLD subgroup with available alcohol consumption data in our cohort, it is unsurprising that the prevalence of MASLD far exceeded that of MetALD and ALD, consistent with the findings of previous studies conducted in other countries [30–32]. Advanced fibrosis, defined as a FIB-4 score >2.67, occurred in approximately 10% of individuals, and it was notably more common in those with MetALD and ALD than in those with MASLD, which had a prevalence of only 1.5%. This finding supports the notion that alcohol exerts additional or potentially synergistic hepatotoxic effects in combination with metabolic stress. The coexistence of alcohol intake and metabolic dysfunction, as observed in MetALD, amplifies hepatic injury through overlapping pathogenic mechanisms, including oxidative stress, impaired lipid metabolism, and gut dysbiosis. These mechanisms collectively intensify hepatic inflammation and fibrogenesis, accelerating the progression from simple fibrosis to steatohepatitis and advanced fibrosis[33]. The synergistic hepatotoxicity of alcohol and metabolic risk factors underscores the pathophysiological continuum between MASLD and ALD, suggesting that even moderate alcohol intake can exacerbate

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metabolic stress and compromise hepatocellular resilience. A similar pattern showing an increased prevalence of advanced fibrosis with alcohol use in patients with SLD has been recently reported[34].

Our study possesses various strengths. We focused on individuals with at least one cardiometabolic risk factor, a population for whom current guidelines recommend SLD screening[10]. We also compared the diagnostic performances of multiple tools that use readily available predictive variables, enhancing the applicability of our findings to primary care settings and population-level research. Additionally, our study included a large sample size with participants exhibiting a range of alcohol consumption patterns, unlike previous studies that often excluded individuals with significant alcohol intake[17,18]. This enabled us to classify patients with SLD into distinct subclassifications: MASLD, MetALD, and ALD.

However, some limitations should be acknowledged. First, due to the retrospective study design, we were unable to compare these scoring systems with a well-established score, specifically the FLI[10], because the GGT parameter was not routinely measured in the health check-up laboratory panel. Second, alcohol consumption data were available for only approximately half of the cohort, as alcohol intake was not systematically recorded using a standardized questionnaire. Because history taking in routine clinical practice was informal, attending physicians may have been more inclined to inquire about and document alcohol use among patients with elevated liver enzymes or overt steatosis on ultrasound. This potential non-random missingness could introduce ascertainment bias into the alcohol-stratified subgroup analyses. Consequently, the small sample sizes within the MetALD and ALD subgroups and potential selection bias necessitate a cautious interpretation of FIB-4-based fibrosis risk across SLD subtypes. Nevertheless, this limitation did not affect the overall diagnosis of SLD. Additionally, this study employed USG as the reference standard for detecting hepatic steatosis. Since USG typically detects fat content above 20.0–30.0%, whereas the standard definition of steatosis is >5% fat, the prevalence of SLD in our study may have been underestimated. Nevertheless, USG was widely used in prior research [18,24] and reflects real-world clinical practice. Although Vibration-Controlled Transient Elastography (VCTE) offers higher sensitivity for detecting steatosis and enables simultaneous assessment of liver fibrosis within a single examination, it was not incorporated into this study because VCTE is not routinely performed as a screening tool in general health check-up programs in Thailand due to cost constraints and limited accessibility. Therefore, abdominal USG remains the standard reference

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for routine health examinations. Finally, as a single-center study conducted at a university hospital in Thailand, involving a health check-up population that voluntarily participated in a program requiring out-of-pocket expenses for USG, the generalizability of our findings to other populations may be constrained.

To the best of our knowledge, this study represents one of the first large-scale external validations comparing the diagnostic accuracy of DSI, HSI, and TyG within an Asian health check-up cohort in Thailand. While DSI demonstrated superior diagnostic performance and its dual-cutoff approach showed potential for streamlining screening, proposing a formal clinical care pathway for nationwide implementation remains premature. Prospective, multicenter investigations with systematic alcohol profiling, FLI comparison, and formal cost-effectiveness analyses are required before integrating DSI into standardized clinical guidelines. Nevertheless, our findings provide valuable foundational evidence highlighting DSI as a promising, accessible tool that warrants further evaluation in primary care settings across Thailand and Southeast Asia.

Conclusion

Approximately half of the individuals with at least one cardiometabolic risk factor had SLD. Among the scoring systems evaluated, DSI demonstrated superior performance compared with HSI and TyG for detecting SLD in this population. In settings where USG or FLI are unavailable, DSI may serve as a practical and reliable screening tool for identifying at-risk patients. Among individuals with SLD, MASLD was significantly more prevalent than MetALD or ALD; however, advanced fibrosis was more common in the MetALD or ALD groups than in those with MASLD.

Declarations

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Institutional review board statement:

This study involved secondary analysis of the previously collected retrospective data and was exempt from human subject research by the Office of Human Research Ethics Committee (HREC), Prince of Songkla University (REC 68-194-14-1). The study was conducted in accordance with the principles of the Declaration of Helsinki and Good Clinical Practice guidelines.

Informed consent statement:

Informed consent was not required, and a waiver of consent was granted by the HREC.

Conflict of interest statement:

All authors declare no competing interests related to this study.

Data availability statement: Due to ethical restrictions involving patient information, the data cannot be shared publicly. The dataset is available upon reasonable request from the corresponding author.

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Clinical trial registration: Not relevant

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Figures and Tables

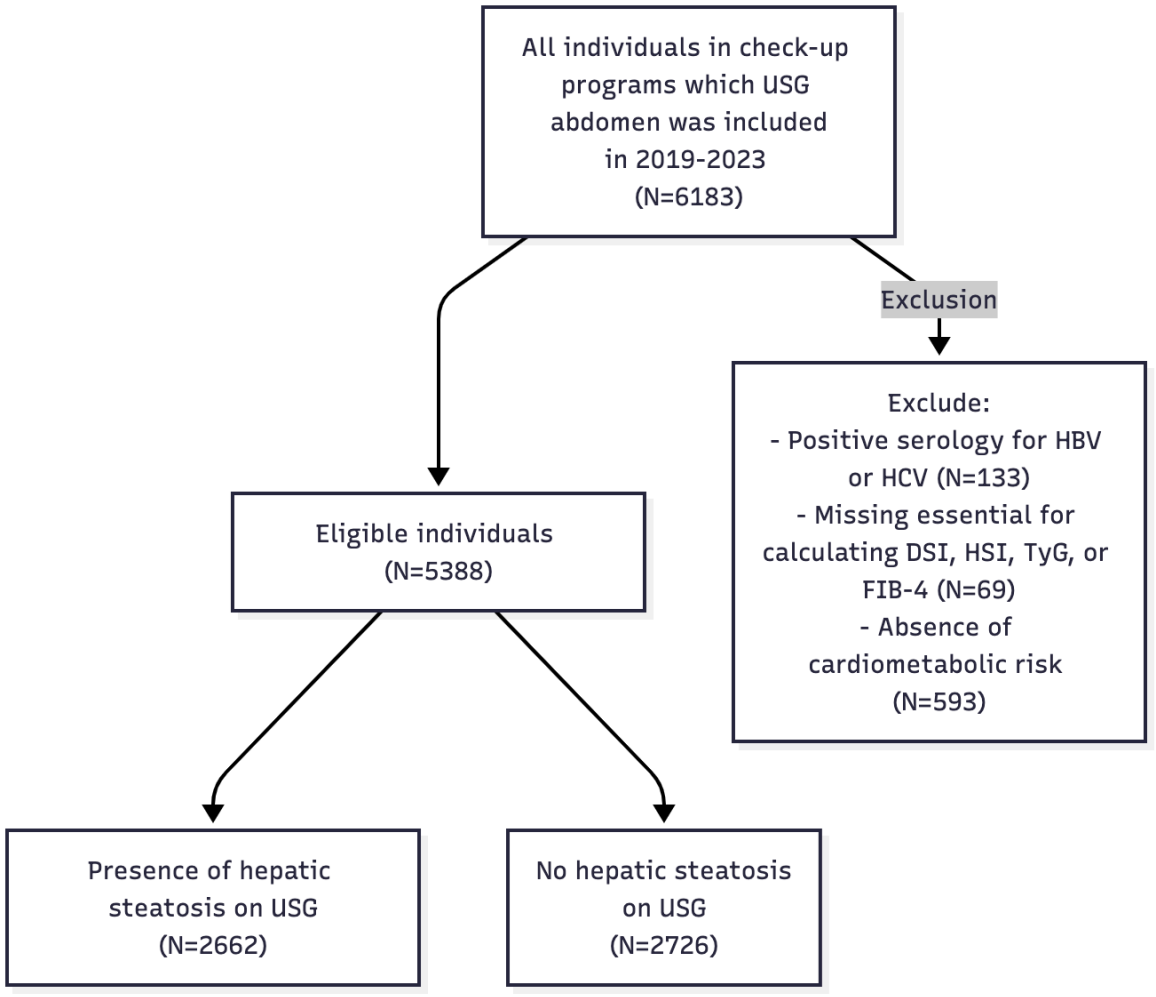

Figure 1. Flow of the study population. FIB-4: Fibrosis-4 index; USG: Ultrasonography; GBV: [define]; FCV: [define]; DSI: Dallas Steatosis Index; HSI: Hepatic Steatosis Index; TyG: Triglyceride–Glucose index; N: number of participants

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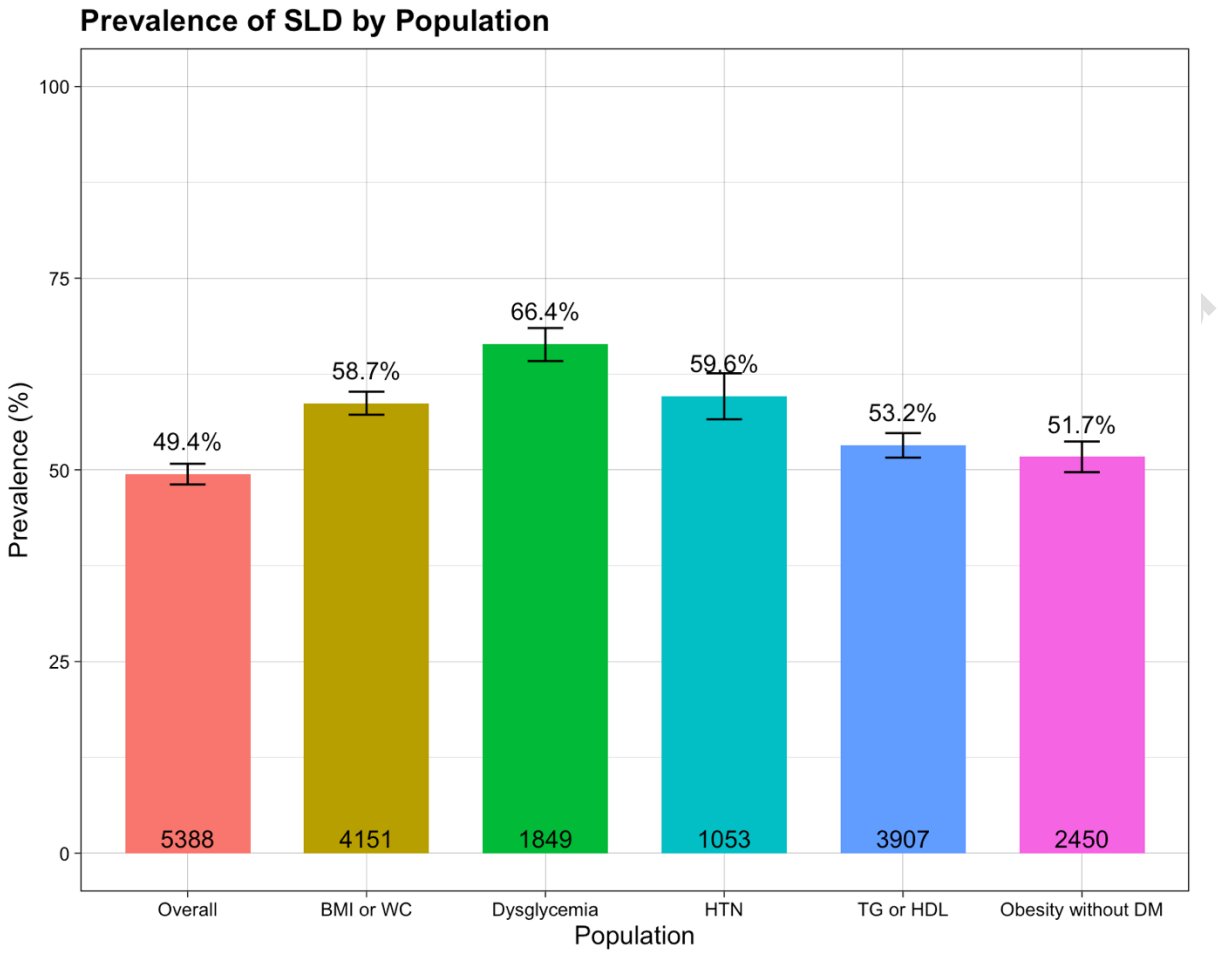

Figure 2. Prevalence of SLD in different subgroups of the population. BMI: Body Mass Index; SLD: Steatotic Liver Disease; HTN: Hypertension; TG: Triglycerides; HDL: High-Density Lipoprotein cholesterol; DM: Diabetes Mellitus; WC: Waist Circumference

Note: Cardiometabolic risk components are defined based on international consensus criteria[21] as follows: BMI or WC (BMI >23 kg/m^2 or WC ≥90 cm in men, ≥80 cm in women); Dysglycemia (fasting plasma glucose >100 mg/dL, type 2 diabetes, or on anti-diabetic treatment); HTN (blood pressure >130/85 mmHg or on antihypertensive treatment; TG or HDL (TG >150 mg/dL, or HDL-C <40 mg/dL in men, <50 mg/dL in women, or on lipid-lowering therapy); Obesity without DM (BMI >23 kg/m^2 without type 2 diabetes).

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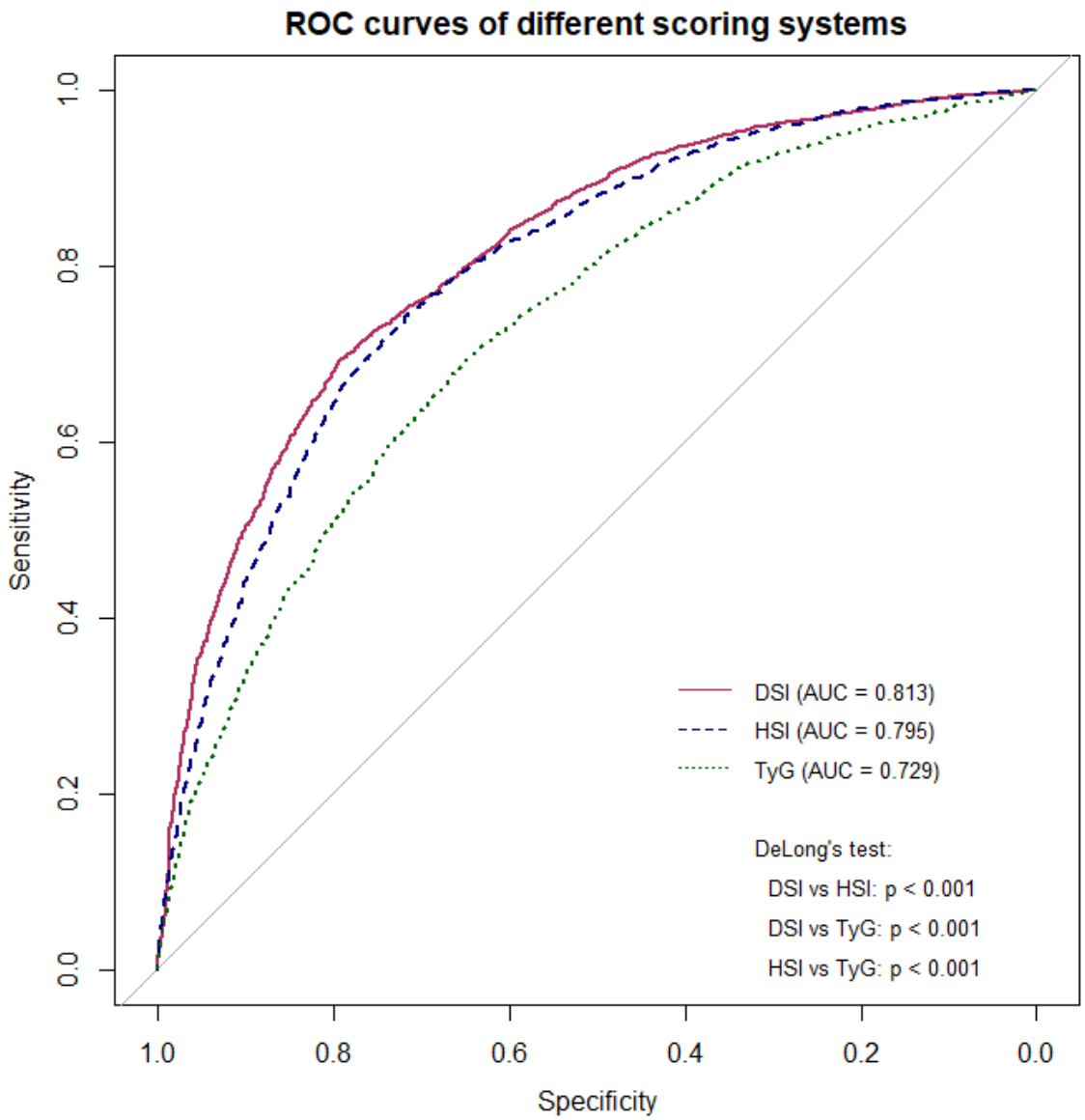

Figure 3. ROC curves of DSI, HSI, and TyG in the diagnosis of SLD in individuals with cardiometabolic risk. DSI: Dallas Steatosis Index; HSI: Hepatic Steatosis Index; TyG: Triglyceride–Glucose index; SLD: Steatotic Liver Disease; ROC: Receiver Operating Characteristic curve

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Table 1. Characteristics of individuals with and without SLD

Characteristic	No SLD (N=2726)	SLD (N=2662)	Total (N=5388)	p-value
Age, years, median (IQR)	56 (45,62.8)	56 (47,62)	56 (46,62)	0.271
Sex				< 0.001
Female	1530 (56.1)	1244 (46.7)	2774 (51.5)	
Male	1196 (43.9)	1418 (53.3)	2614 (48.5)	
Waist circumference, cm, median (IQR)	82 (76,88)	91 (85,97)	86 (79,93)	< 0.001
BMI, kg/m^2, median (IQR)	23.42 (21.5,25.4)	26.53 (24.4,29.1)	24.87 (22.8,27.5)	< 0.001
FPG, mg/dL, median (IQR)	92 (88,99)	98 (92,108)	95 (90,103)	< 0.001
TG, mg/dL, median (IQR)	92 (70,125)	131 (96,178)	109 (81,153)	< 0.001
Total cholesterol, mg/dL, median (IQR)	211 (184,241)	210 (181,239)	210 (182,240)	0.049
HDL, mg/dL, median (IQR)	57.5 (48.6,68.7)	49.2 (42.3,57.3)	53.1 (44.77,63.4)	< 0.001
LDL, mg/dL, mean (S.D.)	144.81 (41.3)	146.7 (41.8)	145.74 (41.5)	0.095
AST, IU/mL, median (IQR)	21 (18,26)	23 (19,29)	22 (19,27)	< 0.001
ALT, IU/mL, median (IQR)	18 (14,25)	25 (19,37)	21 (15,30)	< 0.001

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Platelets, x10^9/L, median (IQR)	256 (221,296.8)	264 (229,306)	261 (224,301)	< 0.001
Creatinine, mg/dL, median (IQR)	0.79 (0.66,0.97)	0.82 (0.67,0.99)	0.81 (0.67,0.98)	0.002
Diabetes	100 (3.7)	300 (11.3)	400 (7.4)	< 0.001
Hypertension	425 (15.6)	628 (23.6)	1053 (19.5)	< 0.001
Dyslipidemia	1485 (54.5)	1562 (58.7)	3047 (56.6)	0.002
CAD	62 (2.3)	74 (2.8)	136 (2.5)	0.273
Stroke	39 (1.4)	39 (1.5)	78 (1.5)	1.000
CKD	30 (1.1)	37 (1.4)	67 (1.2)	0.403
Number of cardiometabolic risks				< 0.001
1	919 (33.9)	245 (9.2)	1164 (21.7)	
2	840 (30.9)	550 (20.7)	1390 (25.9)	
3	655 (24.1)	905 (34.0)	1560 (29.0)	
4	260 (9.6)	716 (26.9)	976 (18.2)	
5	41 (1.5)	243 (9.1)	284 (5.3)	

BMI: body mass index; USG: ultrasonography; SLD: steatotic liver disease; FPG: fasting plasma glucose; TG: triglycerides; HDL: high-density lipoprotein; LDL: low-density lipoprotein; AST: aspartate aminotransferase; ALT: alanine aminotransferase; CAD: coronary artery disease; CKD: chronic kidney disease; IQR: interquartile range; S.D.: standard deviation.

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Table 2. Diagnostic performance indices of DSI in the diagnosis of SLD in individuals with cardiometabolic risk

Cutoff level	Sensitivity (95%CI)	Specificity (95%CI)	NPV (95%CI)	PPV (95%CI)
DSI <0.2	86.5% (85.3-87.8)	60.1% (58.5-61.8)	84.4% (83.2-85.7)	64.1% (63.1-65.1)
DSI ≥0.5	47.4% (45.6-49.3)	92.5% (91.6-93.5)	68.1% (67.3-69.0)	84.0% (82.1-85.7)

DSI: Diabetes Screening Index; SLD: Steatotic Liver Disease; NPV: Negative Predictive Value; PPV: Positive Predictive Value.

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