

Evaluation Of The Hepatic Steatosis Index As A Diagnostic Predictor Of Fatty Liver Disease Associated With Metabolic Dysfunction In Type 2 Diabetics

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ABSTRACT

The hepatic steatosis index (HSI) has demonstrated usefulness in detecting metabolic dysfunction-associated fatty liver disease (MAFLD). However, in middle-income countries such as Peru, its predictive capacity has not been validated, nor have optimal cutoff points been established. In this context, the present study evaluates the HSI as a diagnostic predictor of MAFLD in patients with type 2 diabetes mellitus attending a tertiary hospital in northern Peru. **Materials and Methods:** A prospective external validation study of the HSI model was conducted in 175 diabetic patients receiving outpatient care at the Hospital Regional de Chimbote, Peru, between November and December 2024. Based on the collected clinical and biochemical data, each patient's HSI score was calculated using the original model equation. Subsequently, its predictive performance was evaluated through calibration, discrimination, and classification analyses. **Results:** A total of 74.2% of the selected patients were diagnosed with MAFLD. The HSI model showed good discriminative performance, with an AUC of 0.83 (95% CI: 0.77-0.89) and a calibration slope of 1. In our population, an HSI score <36 had a negative likelihood ratio of 0.26, ruling out MAFLD, whereas an HSI >44 yielded a positive likelihood ratio of 24.5, confirming the diagnosis. **Conclusions:** In the present study, the HSI index demonstrated adequate predictive performance for MAFLD. Owing to its ease of application and low cost, this model represents a potentially useful tool in middle- and low-income countries, as it contributes to the identification of type 2 diabetic patients who require diagnostic confirmation of MAFLD through liver ultrasound.

KEYWORDS: Non-alcoholic Fatty Liver Disease; Diabetes Mellitus Type 2; Predictive Models; Index Steatosis Hepatic.

ABBREVIATIONS:

BMI: body mass index; IC : intervalo de confianza; HbA1: glycosylated hemoglobin A1; SD: standard deviation; T2D: type 2 diabetes

INTRODUCTION

Metabolic dysfunction associated fatty liver disease (MAFLD) is currently a highly prevalent chronic illness, given its sustained increase closely linked to the global epidemic of metabolic disorders. In this context, patients with type 2 diabetes (T2D) represent a special high-risk group, as the prevalence of MAFLD in this population ranges from 60% to 80% worldwide [1], 56.8% in South America [2], and 18% in Peru [3].

In response to this issue, several noninvasive models have been proposed for the detection of nonalcoholic hepatic steatosis. Among these, the Hepatic Steatosis Index (HSI) stands out. It was

developed by Jeong et al. [4] in a general Korean population using abdominal ultrasound as the reference standard. Initially, 20 variables were evaluated, of which body mass index (BMI), aspartate aminotransferase (AST), alanine aminotransferase (ALT), the presence of type 2 diabetes mellitus (T2D), and sex collectively demonstrated the best predictive performance, with an area under the curve (AUC) of 0.81 (95% CI: 0.80-0.83). The result is expressed on a scale from 0 to 100, referred to as the Hepatic Steatosis Index (HSI): values <30 rule out fatty liver (negative likelihood ratio = 0.17), whereas values >36 confirm its presence (positive likelihood ratio = 6.51).

The anthropometric and biochemical parameters included in the HSI model are low-cost and easily accessible in outpatient medical care. This model has shown adequate predictive ability for hepatic steatosis in studies conducted in Asian and European populations [5-7]. However, the sociodemographic characteristics, lifestyle, and clinical-metabolic profiles of these populations differ from those observed in patients with T2D, in whom its performance has not yet been evaluated. This gap underscores the need for the present study.

The population in our study meets the diagnostic criteria for both Nonalcoholic Fatty Liver Disease (NAFLD) and MAFLD. Participants were selected after excluding those with significant alcohol consumption or other liver diseases, in accordance with the classical definition of NAFLD. In addition, all participants had T2D, a required criterion for metabolic dysfunction under the MAFLD definition. This dual correspondence allows our results to be compared with findings from previous studies conducted under both definitional frameworks.

Adults with T2D are at increased risk of developing MAFLD due to the frequent coexistence of dyslipidemia, obesity, and insulin resistance [8]. Therefore, the purpose of applying predictive models such as the HSI is to enable the early and noninvasive identification of patients at risk, facilitating timely stratification through the indication of imaging studies or the implementation of metabolic management adjustments. Thus, its main clinical utility lies in serving as a screening tool during the initial evaluation of patients with T2D.

The objective of the present study is to evaluate the predictive performance of the HSI model for the detection of MAFLD in patients with T2D receiving outpatient care at a tertiary hospital in northern Peru.

METHODS

Study Design and Setting

This external validation study was conducted prospectively, collecting clinical and biochemical data from patients with T2D attending the outpatient endocrinology clinic of the Eleazar Guzmán Barrón Regional Hospital (HREGB), a tertiary-level hospital located in the city of Chimbote, northern Peru, between November and December 2024.

Population

Patients with a previous diagnosis of type 2 diabetes (T2D), aged ≥ 25 years, and without a history of significant alcohol consumption (≥ 30 g/day in men and ≥ 20 g/day in women) were included.

Those with a history of viral hepatitis (A, B, or D), chronic liver disease, thyroid disorders, Cushing's syndrome, use of hepatotoxic drugs within the previous six months, renal replacement therapy or hemodialysis, as well as pregnant or breastfeeding women, were excluded.

Sample Size

The sample size was determined according to the recommendation by Peduzzi and Concato [9], who proposed a minimum of 10 events per independent variable included in a logistic regression model. Considering that the HSI model includes five predictors, a minimum of 50 hepatic steatosis events was estimated to be necessary. Assuming an expected prevalence of 60% hepatic steatosis among patients

with T2D [5], a minimum of 84 participants was calculated; however, our final sample consisted of 175 participants.

Preparación de los datos

Los datos fueron registrados en una hoja de cálculo de Microsoft Excel, protegida con contraseña y acceso restringido exclusivamente a los investigadores. Posteriormente, se revisó la base de datos para asegurar la integridad de la información, verificando que cada paciente cuente con los datos completos requeridos en el estudio, eliminando registros duplicados o incompletos.

Data Preparation

The data were recorded in a password protected Microsoft Excel spreadsheet with access restricted exclusively to the investigators. The database was subsequently reviewed to ensure information integrity by verifying that each patient had all the required study data and that duplicate or incomplete records were removed.

Outcome

The predicted outcome, corresponding to the presence of hepatic steatosis, was determined categorically using abdominal ultrasound as the reference standard. The procedure was performed by a single certified radiologist (CMP: 12061297; RNE: 039824) using a VINNO X2 ultrasound device, serial number U0212HY010.

Predictor Variables

The multivariate predictive HSI model includes weight (kg), height (cm), sex (male/female), presence of diabetes, aspartate aminotransferase (AST), and alanine aminotransferase (ALT). Anthropometric variables were obtained directly from the “Diabetes Follow-up Forms” [10], which were prospectively collected by nursing staff from the “Diabetes Epidemiological Surveillance” program, previously trained by the research team.

Procedures

Patients with T2D were identified through the Diabetes Epidemiological Surveillance program at HREGB. Those who met the inclusion criteria and signed informed consent were prospectively evaluated by the research team. At the first contact, anthropometric measurements were performed following the standardized protocol of the Ministerio de Salud del Peru [11]. Subsequently, within 72 hours, a fasting blood sample (≥ 8 hours) was obtained to determine AST (mg/dL) and ALT (IU/L). Samples were processed at the HREGB laboratory following its “Manual de Procedimientos del área de Bioquímica” [12], using automated kinetic methods with internal quality control.

The presence of hepatic steatosis was assessed according to the protocol “Evaluación de esteatosis hepática por atenuación ecográfica” [13]. The radiologist was blinded to the clinical and biochemical data, while the nursing staff and laboratory technicians were unaware of the ultrasound findings, in order to prevent review bias.

Statistical Analysis

To evaluate the predictive performance of the HSI model, a single dataset was used and exported to the R statistical software (version 4.4.1). For external validation, the HSI model was applied to each T2D patient using its original equation [4], defined as:

$$HSI = 8\left(\frac{ALT}{AST}\right) + IMC + 2(\text{if it is T2D}) + 2(\text{if it is Female})$$

To evaluate the model's performance, measures of calibration, discrimination, and classification were employed.

Model calibration was evaluated using the calibration slope, comparing predicted probabilities with observed outcomes within each risk decile. Calibration was considered adequate when the slope ranged from 0.9 to 1.1; acceptable, with slight under or overestimation, when the slope was between 0.8-0.9 or 1.1-1.2; and recalibration was indicated when the slope was below 0.8 or above 1.2 [14].

The model's discriminative ability was assessed using the area under the ROC curve (AUC), interpreted as non-informative when $AUC \leq 0.5$, indicating poor discrimination for values between 0.5 and 0.7, and good discrimination when $AUC \geq 0.7$ [15].

The stratification and classification performance of the HSI model was evaluated using likelihood ratios (LR). Positive LR (LR+) values close to or above 10 were considered strong evidence to confirm the presence of disease, whereas negative LR (LR-) values near or below 0.1 indicated a high probability of its absence [16].

The stratification and classification performance of the HSI model was evaluated using likelihood ratios (LRs). Positive likelihood ratio (LR+) values close to or greater than 10 were considered strong evidence for confirming the presence of the disease, whereas negative likelihood ratio (LR-) values near or below 0.1 indicated a high probability of its absence [16].

Since all participants had a diagnosis of T2D, a variable included in the original HSI model equation, a sensitivity analysis was performed excluding this variable. The objective was to determine whether its omission affected the discriminative power of the index (AUC, sensitivity, and specificity) for the identification of MAFLD.

The expected prevalence of hepatic steatosis used for the sample size calculation was 60%, a value considered appropriate to avoid extreme class imbalance. Consequently, no class imbalance correction techniques were applied.

To explore equity in model performance, subgroup-stratified analyses were conducted. The model's discriminative and classification performance were evaluated according to sex, presence of comorbidities, glycemic control ($HbA1c \geq 7\%$, poor control), and duration of T2D (≥ 10 years, long-standing disease). Statistical comparisons of AUCs between subgroups were performed using DeLong's test, with $p < 0.05$ considered statistically significant. These analyses allowed the identification of potential variations in model performance across different subgroups.

Ethical Approval

The study protocol was approved by the Ethics Committee of the School of Human Medicine at Universidad Nacional del Santa and by the Ethics Committee of HREGB. The study was conducted in accordance with the principles of the Declaration of Helsinki [17] and the Council for International Organizations of Medical Sciences guidelines [18]. Informed consent was obtained from all participants.

RESULTS

Participant Flow

During the study period, out of 348 diabetic patients, a total of 250 were randomly selected who were scheduled for outpatient care in the endocrinology department. After applying the selection criteria, 232 met the requirements and were given the informed consent form; among them, 215 agreed to participate. These patients had their blood samples collected and processed the day after the first contact. Subsequently, 200 had available laboratory results and were scheduled to undergo abdominal ultrasound within a maximum of two days. Ultimately, the final sample consisted of 175 patients (Figure 1).

Descriptive Characteristics of the Study Population

When the normality test was applied to the quantitative variables, only BMI showed a parametric distribution, whereas the remaining variables followed a nonparametric distribution.

In the studied population, the mean BMI was 29.62 ± 4.73 kg/m², indicating a predominance of overweight and obesity. The median AST and ALT levels were 22 U/L (IQR: 17-33) and 30 U/L (IQR: 19-32), respectively, values that were generally within normal limits. The median HbA1c was 6.6% (IQR: 6.2-7.0), suggesting that most participants had adequate glycemic control. Among the 175 patients, 130 (74.2%) presented MAFLD, confirmed by ultrasonography, 125 (71.4%) were women, and 112 (64%) had no associated comorbidities.

Patients with hepatic steatosis showed significantly higher values of BMI, waist circumference, ALT, AST, GGT, triglycerides, capillary glucose, and HbA1c ($p < 0.05$). Although liver enzyme levels remained within reference ranges, they were close to the upper limits, and hypertriglyceridemia was also observed. Capillary glucose levels were elevated in both groups, while mean HbA1c values were below 7% in both, yet significantly higher among those with steatosis. No significant differences were found in age, duration of T2D, sex, or presence of comorbidities ($p > 0.05$) (**Table 1**).

The diagnosis of MAFLD was more frequent in females than in males, with 95 cases (76%) versus 35 cases (70%), respectively. Regarding comorbidity status, 48 patients with comorbid conditions (76.1%) had MAFLD, compared to 82 cases (73.2%) among those without comorbidities. Concerning glycemic control, MAFLD was observed in 50 patients (84.7%) with poor control ($\text{HbA1c} \geq 7\%$) and in 80 patients (68.7%) with good control ($\text{HbA1c} < 7\%$). Finally, with respect to disease duration, 87 patients (79%) with long-standing T2D and 43 (66%) with shorter disease duration were found to have MAFLD.

Evaluación del modelo HSI original

En nuestra población, el modelo HSI original mostró un AUC de 0,83 (IC95%: 0,77-0,89) (**Figura 2**), lo que evidencia una buena capacidad discriminativa del modelo HSI para identificar MAFLD. En cuanto a la calibración, la pendiente fue de 1,00 (IC95%: 0,64-1,36; $p < 0,000$) y el intercepto de 0,000 (IC95%: -0,47 a 0,47) (**Figura 3**), indicando una adecuada concordancia entre las probabilidades predichas y las observadas.

Evaluation of the Original HSI Model

In our study population, the original HSI model demonstrated an AUC of 0.83 (95% CI: 0.77-0.89) (**Figure 2**), indicating good discriminative ability for identifying MAFLD. Regarding calibration, the slope was 1.00 (95% CI: 0.64-1.36; $p < 0.001$) and the intercept was 0.000 (95% CI: -0.47 to 0.47) (**Figure 3**), showing an adequate agreement between predicted and observed probabilities.

The stratification and classification performance of the original HSI model in our population, considering a score range from 0 to 100, showed that a Youden-adjusted cutoff value of <36 ($\text{LR-} = 0.26$; sensitivity = 92%) effectively ruled out the presence of MAFLD, whereas an $\text{HSI} \geq 44$ ($\text{LR+} = 24.5$; specificity = 98%) would confirm the diagnosis. Intermediate HSI values (36-44) would require abdominal ultrasonography for diagnostic confirmation (**Table 2**).

Sensitivity analysis

Following the sensitivity analysis, the modified HSI model (excluding the T2D variable) showed an AUC of 0.83 (95% CI: 0.77-0.89) in our population. Regarding calibration, the slope was 1.00 (95% CI: 0.64-1.36; $p < 0.001$) with an intercept of 0.000 (95% CI: -0.47 to 0.47). These results indicate that excluding this variable from the sample did not affect the model's discriminative ability or calibration for detecting MAFLD in patients with T2D.

Subgroup analysis

When the comparative analysis was performed by sex, the AUC was 0.82 (95% CI: 0.70-0.94) in males and 0.84 (95% CI: 0.77-0.91) in females ($p = 0.814$). According to the presence of comorbidities, the AUC was 0.87 (95% CI: 0.78-0.96) in patients with comorbidities and 0.80 (95% CI: 0.72-0.88) in those without comorbidities ($p = 0.272$). Regarding glycemic control, the AUC was 0.80 (95% CI: 0.72-0.88) in patients with good glycemic control and 0.89 (95% CI: 0.81-0.98) in those with poor control ($p = 0.114$). Finally, with respect to T2D duration, the AUC was 0.87 (95% CI: 0.79-0.94) in patients with long-standing T2D and 0.78 (95% CI: 0.67-0.90) in those with shorter disease duration ($p = 0.230$) (Table 3, Figure 4).

Los puntos de corte del modelo HSI original ajustados a nuestra población mantuvieron su robusto desempeño predictivo según sexo, presencia de comorbilidad o el estado de control glucémico. En todos los subgrupos, valores intermedios de HSI con puntaje entre 36 - 44 corresponden a una zona indeterminada (**Tabla 3**).

The cutoff points of the original HSI model, adjusted to our population, maintained their robust predictive performance across sex, presence of comorbidities, and glycemic control status. In all subgroups, intermediate HSI scores between 36 and 44 corresponded to an indeterminate zone (Table 3).

DISCUSSION

In the present external validation study, the HSI model for detecting MAFLD in patients with T2D showed an AUC of 0.83. This value slightly exceeds that reported by Lee et al. [4] in the general Korean population, where the AUC was 0.812 in the derivation cohort and 0.819 in the internal validation cohort. This similarity in performance suggests that the model maintains adequate discriminative ability in a population with different sociodemographic, lifestyle, and clinical-biochemical characteristics, even in a higher-risk group such as patients with T2D.

Regarding calibration, our population showed an adequate agreement between predicted and observed probabilities, indicating that the model preserves its predictive ability in patients with T2D. This finding is relevant, as T2D is a known risk factor for MAFLD and could have potentially influenced the index's performance in this homogeneous cohort. Nevertheless, the sensitivity analysis, excluding the T2D variable from the model, demonstrated that its removal did not alter either the discriminative ability or the calibration (AUC = 0.83; slope = 1), thereby reinforcing the generalizability of the HSI, even in settings where T2D is present.

Similarly, regarding classification ability, the cutoff points adjusted for our population (<36 and ≥ 44) maintained strong predictive performance, with an LR- of 0.26 (sensitivity: 92%) and an LR+ of 24.5 (specificity: 98%), respectively. These values outperform the results obtained with the original thresholds proposed by Lee et al. (<30 and >36) [4]. The observed difference may be attributed to the distinct sociodemographic, lifestyle, and clinical-biochemical characteristics between the two populations: the higher mean BMI and the presence of T2D in our cohort likely shifted the HSI distribution toward higher values.

In the study by Fennoun et al. [19] conducted in Morocco, the HSI demonstrated excellent predictive ability for detecting MAFLD in patients with T2D (AUC = 0.97; sensitivity: 89.5%; specificity: 95.2%; cutoff >36). In our population, the AUC was 0.83, indicating good performance, albeit lower, possibly due to differences in sample characteristics, as Fennoun's hospital-based cohort also exhibited poorer glycemic control. The adjusted cutoff points proposed in our study (<36 and ≥ 44) optimized the model's classification ability without compromising its calibration, supporting the utility and reproducibility of the HSI across different settings and emphasizing the importance of adapting its thresholds to the specific characteristics of each population.

In the study by Okada et al. [20], conducted in the general Japanese population, the HSI demonstrated good predictive performance for detecting MAFLD in the subgroup of patients with T2D (AUC = 0.84),

a result similar to that observed in our population (AUC = 0.83). However, unlike the Asian context of Okada's study, where the model was applied without modification, our findings revealed the need to adjust the cutoff points (<36 and ≥ 44) to optimize predictive classification.

Similarly, Chen et al. [21] reported a moderate performance of the HSI for detecting MAFLD in the subgroup of U.S. patients with T2D, with an AUC of 0.719, sensitivity of 88.1%, and specificity of 46.0%, using a cutoff point >38.08 . In contrast, in our population, the HSI demonstrated better predictive performance. These differences may be explained by sociodemographic, lifestyle, and clinical-biochemical variations between populations, particularly the higher prevalence of central obesity and dyslipidemia in our population, which may enhance the model's discriminative ability.

Finally, subgroup analysis revealed that the discriminative ability of the HSI was slightly higher in women, patients with comorbidities, those with poor glycemic control ($\text{HbA1c} \geq 7\%$), and those with long-standing T2D (≥ 10 years). However, these differences were not statistically significant ($p > 0.05$), suggesting an equitable predictive performance of the HSI model across different clinical scenarios in patients with T2D. Similarly, Lee et al. [4] reported comparable AUC values in men and women (0.816 and 0.808, respectively), indicating that sex does not influence the model's discriminative performance.

Clinical Implications

From a clinical standpoint, the HSI model retains its usefulness in our population as a noninvasive, accessible, and low-cost tool, particularly in settings where access to imaging methods is limited. The application of the HSI model could help reduce the need for hepatic ultrasound, prioritizing those with indeterminate results (HSI between 36 and 44).

Limitations

Although this is the first external validation study of the HSI model in a Peruvian cohort, its single-center design may limit the generalization of the findings to other populations with different sociodemographic characteristics or levels of healthcare.

Conclusions

The HSI model demonstrated adequate ability to identify MAFLD in patients with type 2 diabetes, showing good calibration and consistent performance across clinical subgroups. The cutoff points adjusted for our population (<36 and ≥ 44) optimized its classification capacity, defining an intermediate zone (36–44) that requires ultrasound confirmation. These findings support the usefulness of the HSI as a noninvasive, accessible, and reproducible tool for MAFLD screening in clinical settings with limited resources.

Recommendations

It is recommended to conduct prospective, multicenter studies with larger sample sizes to confirm the predictive ability and generalizability of the HSI in other middle and low income countries.

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TABLES Y FIGURES

Table 1. Comparison of clinical, anthropometric, and biochemical variables according to the presence of hepatic steatosis.(n = 175).

VARIABLE	WITH HEPATIC STEATOSIS (n=130)	WITHOUT HEPATIC STEATOSIS (N = 45)	STATISTICAL TEST ^a	p- VALUE ^b
HSI (score)	83	30	w= 65,8	0,000
BMI (kg/m ²)	30,81	26,17	t=-8,21	0,000
Time since diagnosis (years)	12	10	t=-1,50	0,341
Age (years)	62	64	w = 3159	0,425
Waist circumference (cm)	102	94	w=1086	0,000
ALT (U/L)	32	17	w=1220	0,000
AST(U/L)	24,5	18	w=1699	0,000
GGTP (U/L)	36	19	w=1189	0,000
Triglycerides (mg/dl)	210	105	w=943,5	0,000
Preprandial capillary glucose (mg/dl)	168,5	150	w=2065	0,003
HbA1 (%)	6,7	6,4	w=1744	0,000
Female sex	95 (54.3%)	30 (17.1%)	x ² =0,67	0,412
With comorbidity	48 (27.4%)	15 (8.6%)	x ² =0,19	0,665

a. The p-value for parametric quantitative variables was calculated using the Student's t-test (t); for nonparametric quantitative variables, the Wilcoxon test (w) was used; and for qualitative variables, the Chi-square test (χ^2) was applied.

b. Statistically significant difference at $p < 0.05$, indicating an association between the variable and hepatic steatosis.

Table 2. Risk stratification of the HSI model for the diagnosis of hepatic steatosis in patients with type 2 diabetes mellitus.

PREDICTIVE MODEL	ADJUSTED HSI CUTOFF POINT ^a	n(%)	SEN (%)	SP(%)	LR+	LR-
HSI in our population	≥36	150 (86%)	92	31	1,33	0,26
	≥44	63 (36%)	49	98	24,5	0,52

a. The optimal cutoff point was calculated based on the highest Youden index.

Abbreviations: HSI = Hepatic Steatosis Index; % = number of patients with HSI ≥ cutoff point; SEN = sensitivity; SP = specificity; LR+ = positive likelihood ratio; LR- = negative likelihood ratio.

Table 3. Subgroup risk stratification of the HSI model for the diagnosis of hepatic steatosis in patients with type 2 diabetes mellitus.

ADJUSTED HSI CUTOFF POINT ^a	n(%)	SEN (%)	SP(%)	LR+	LR-	AUC (IC95%)	p- VALUE ^a
MALE SUBGROUP (n=50)							
≥36	43(86%)	91	27	1,25	0,33	0,82 (IC95%: 0,70–0,94)	0,814
≥44	20(40%)	54	93	7,71	0,49		
FEMALE SUBGROUP (n = 125)							
≥36	107(86%)	92	33	1,37	0,24	0,84 (IC95%: 0,77–0,91)	0,272
≥44	43(34%)	45	99	45	0,56		
SUBGROUP WITH COMORBIDITY PRESENT (n=63)							
≥36	53(84%)	92	40	1,53	0,19	0,87 (IC95%: 0,78–0,96)	0,272
≥44	25(37%)	52	99	52	0,48		
SUBGROUP WITHOUT COMORBIDITY (n=112)							

≥36	91(81%)	91	27	1,25	0,33	0,80 (IC95%: 0,72–0,88)
≥44	85(76%)	45	97	15	0,57	

SUBGROUP WITH GOOD GLYCEMIC CONTROL (HbA1 <7%) (n=116)

≥36	96(83%)	90	33	1,34	0,30	0,80 (IC95%: 0,72–0,88)
≥44	36(31%)	44	97	14,6 7	0,58	0,114

SUBGROUP WITH POOR GLYCEMIC CONTROL (HbA1 ≥7%) (n= 59)

≥36	54(92%)	94	22	1,21	0,27	0,89 (IC95%:0,81–0,98)
≥44	27(46%)	54	99	54	0,46	

SUBGROUP WITH LONG-STADING T2D (Disease duration ≥10 years)(N=110)

≥36	96(87%)	93	35	1.43	0.20	0,87 (IC95%: 0,79-0,94)
≥44	44(40%)	51	99	51	0.49	0,230

SUBGROUP WITH SHORTER T2D DURATION (Disease duration <10 years) (n=65)

≥36	54(83%)	89	27	1.22	0.41	0,78 (IC95%: 0,67-0,90)
≥44	19(29%)	42	95	8.40	0.61	

a. The optimal cutoff point was calculated based on the highest Youden index. Abbreviations: n = participants; HSI = Hepatic Steatosis Index; % = number of patients with HSI ≥ cutoff point; SEN = sensitivity; SP = specificity; LR+ = positive likelihood ratio; LR– = negative likelihood ratio.

Figure 1. Patient selection flowchart

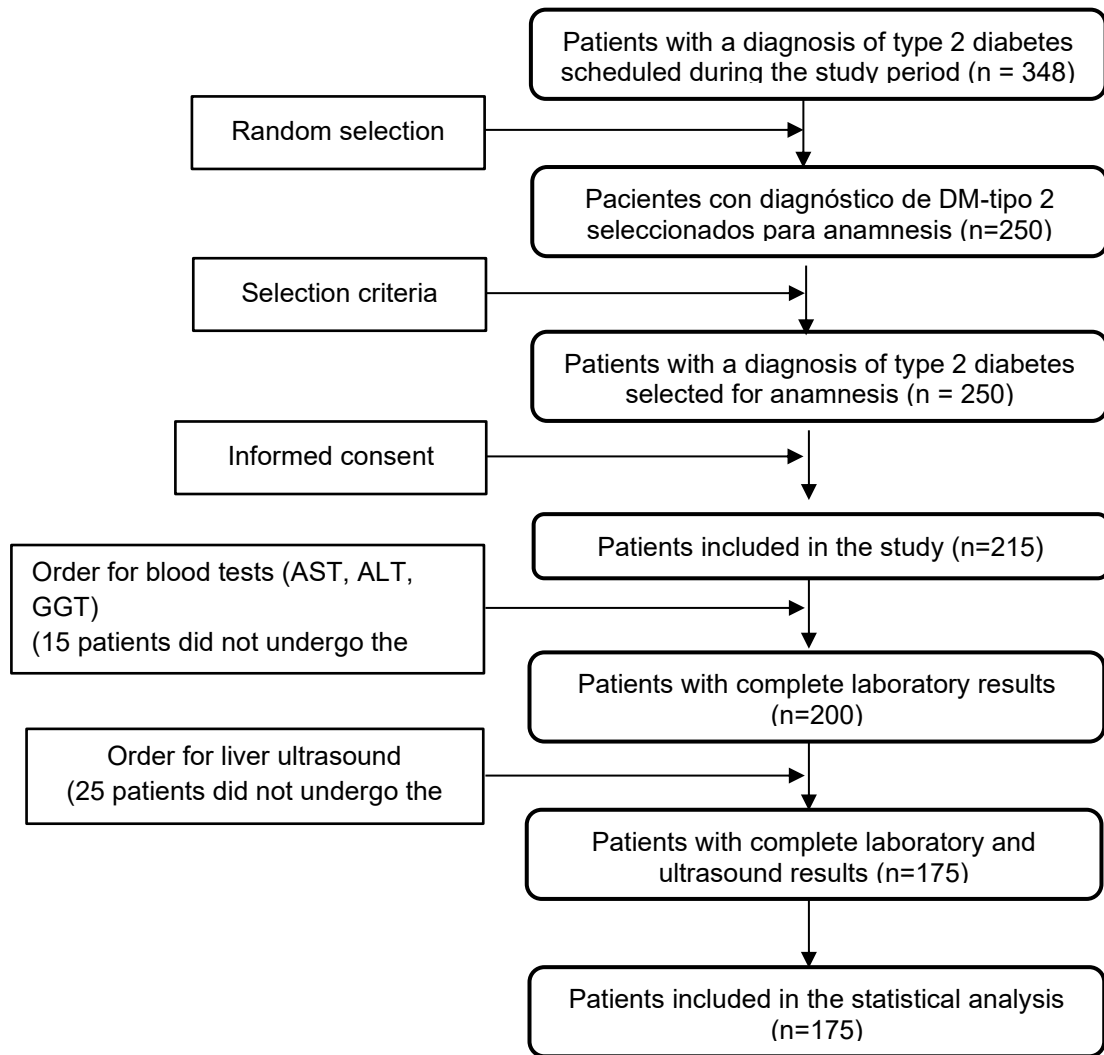


Figure 2. ROC curve of the HSI predictive model for the diagnosis of hepatic steatosis in patients with type 2 diabetes mellitus.

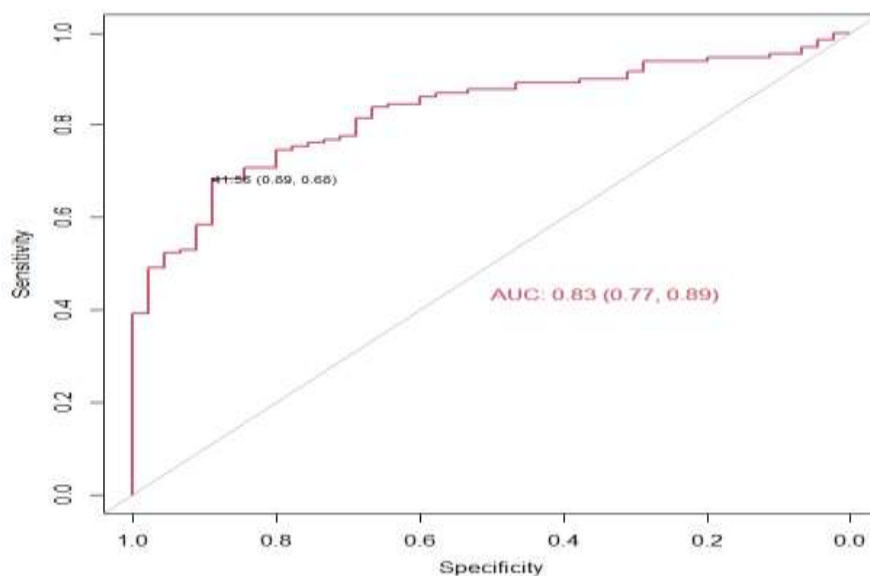


Figure 3. Calibration curve of the HSI model for the diagnosis of hepatic steatosis in patients with type 2 diabetes mellitus.

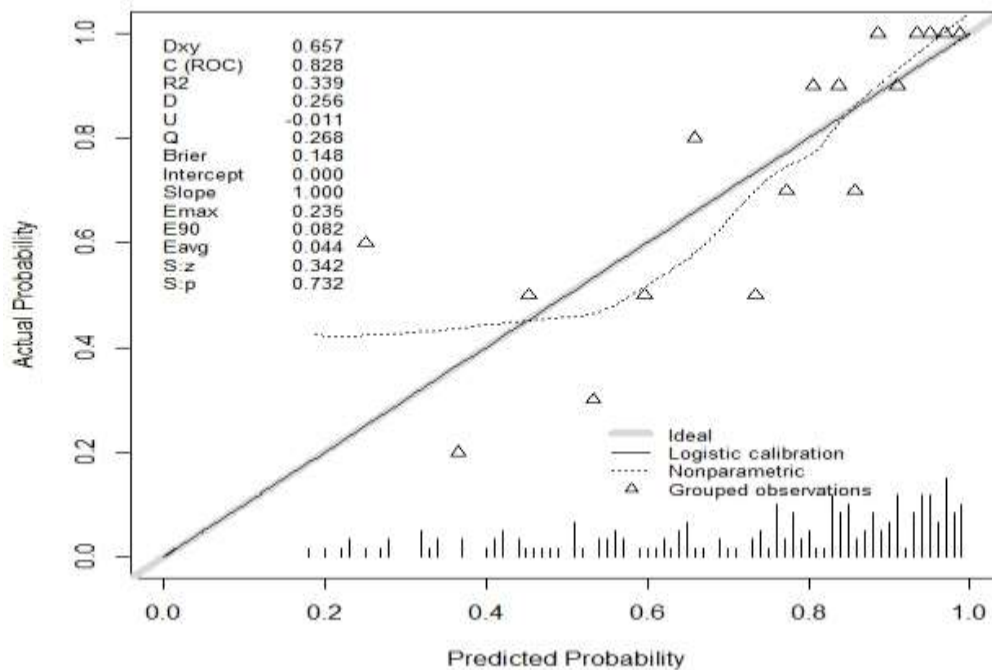
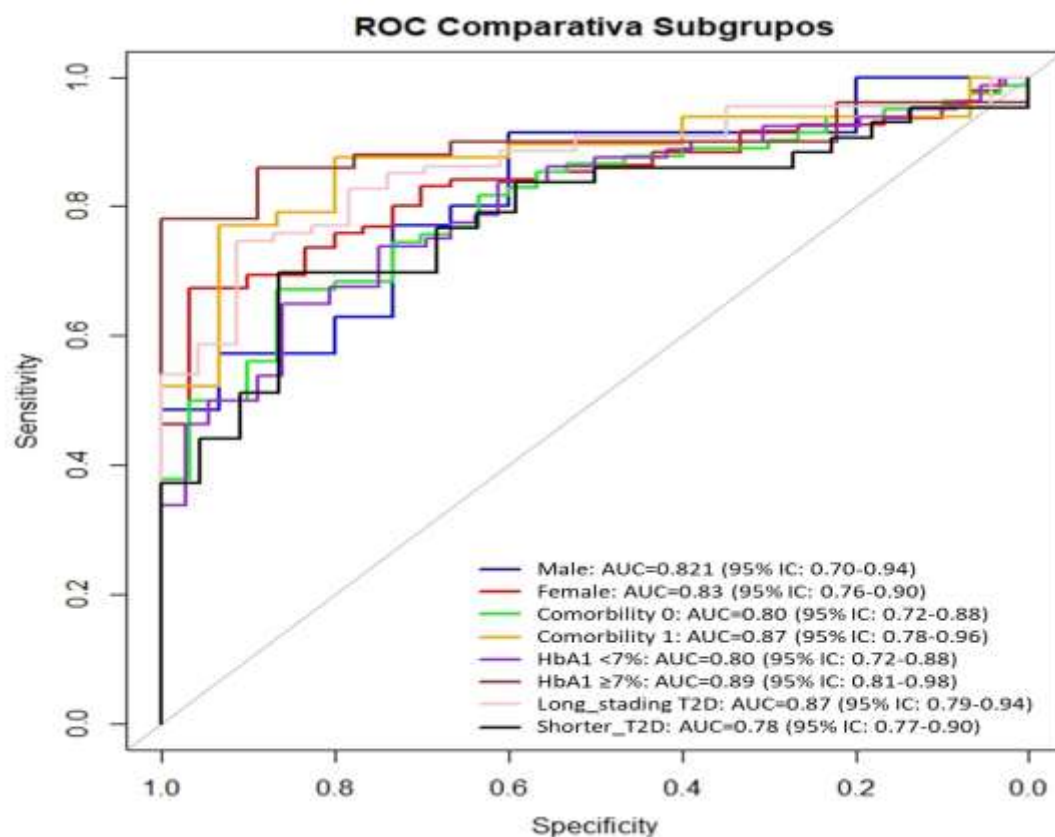


Figure 4. ROC curves of the HSI model for the diagnosis of hepatic steatosis in patients with type 2 diabetes mellitus, according to subgroup analysis.



Note: The analysis included subgroups by sex, presence (1) or absence (0) of comorbidities, glycemic control (HbA1c <7% vs. ≥7%), and duration of T2D (long and short evolution). The AUC values (95% CI) for each subgroup are shown.

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