



## Original Research

# Comparative Diagnostic Performance of Non-Invasive Indices for Predicting Ultrasonographic Hepatic Steatosis in Morbidly Obese Patients: A Single-Center Retrospective Study

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### Abstract

**Objectives:** Morbid obesity is strongly associated with non-alcoholic fatty liver disease (NAFLD), and hepatic steatosis is highly prevalent among candidates for bariatric surgery. Given the invasive nature of liver biopsy, there is a growing need for reliable non-invasive methods to assess hepatic steatosis. This study aimed to compare the diagnostic performance of insulin resistance–based indices (Homeostasis Model Assessment of Insulin Resistance [HOMA-IR] and Quantitative Insulin Sensitivity Check Index [QUICKI]) and biochemical scores (Hepatic Steatosis Index [HSI] and NAFLD Liver Fat Score [NAFLD-LFS]) in predicting ultrasonographically detected hepatic steatosis in patients with morbid obesity.

**Methods:** In this single-center retrospective observational study, 206 patients with morbid obesity who underwent primary laparoscopic sleeve gastrectomy between March 2024 and February 2026 and had available preoperative laboratory and abdominal ultrasonography data were included. Insulin resistance indices (HOMA-IR, QUICKI) and composite scores (HSI and NAFLD-LFS) were calculated. Hepatic steatosis was graded ultrasonographically as grade 1–3 and categorized as mild (grade 1) and moderate-to-severe (grade  $\geq 2$ ) for diagnostic performance analyses. Receiver operating characteristic (ROC) curve analysis was performed to evaluate diagnostic accuracy.

**Results:** Hepatic steatosis was classified as grade 1 in 28.2%, grade 2 in 59.2%, and grade 3 in 12.6% of patients. Steatosis grade showed positive correlations with HOMA-IR ( $r=0.244$ ), HSI ( $r=0.354$ ), NAFLD-LFS ( $r=0.297$ ), HbA1c ( $r=0.274$ ), transaminases, and glucose/insulin levels, and a negative correlation with QUICKI. In ROC analysis, HSI demonstrated the highest diagnostic performance (AUC=0.716), followed by HbA1c (AUC=0.656) and NAFLD-LFS (AUC=0.645). In multivariable analysis, age (OR=1.04), BMI (OR=1.23), and NAFLD-LFS (OR=1.42) were identified as independent predictors, while female sex was associated with lower risk (OR=0.34).

**Conclusion:** In patients with morbid obesity, hepatic steatosis is significantly associated with metabolic parameters and insulin resistance. Among non-invasive indices, HSI demonstrated the highest diagnostic performance. HSI and NAFLD-LFS may serve as practical tools in the preoperative assessment of bariatric surgery candidates.

**Keywords:** Hepatic steatosis, hepatic steatosis index, morbid obesity, NAFLD liver fat score, non-invasive indices

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Morbid obesity is a rapidly increasing global public health problem and is closely associated with metabolic, cardiovascular, and hepatic complications.<sup>[1]</sup> Among these hepatic complications, non-alcoholic fatty liver disease (NAFLD) is one of the most common conditions, recently redefined as metabolic dysfunction-associated steatotic liver disease (MASLD).<sup>[2,3]</sup> NAFLD is characterized by lipid accumulation in hepatocytes and encompasses a broad and progressive clinical spectrum, ranging from simple hepatic steatosis to non-alcoholic steatohepatitis, progressive fibrosis, and cirrhosis.<sup>[4]</sup>

NAFLD is highly prevalent, particularly in individuals with morbid obesity. Studies conducted in patients who are candidates for bariatric surgery have reported that the prevalence of NAFLD ranges between 60% and 90%.<sup>[4,5]</sup> This high prevalence reflects the strong pathophysiological relationship between obesity and hepatic fat accumulation. Insulin resistance is considered one of the main mechanisms underlying impaired hepatic lipid metabolism and increased triglyceride accumulation in hepatocytes.<sup>[6,7]</sup>

Liver biopsy is regarded as the gold standard for the assessment of hepatic steatosis; however, its invasive nature and potential risk of complications limit its use in routine clinical practice. Interest in non-invasive methods for evaluating hepatic steatosis has increased in recent years.<sup>[8]</sup> Insulin resistance-based indices and biochemical scores serve as alternative diagnostic tools for the assessment of hepatic steatosis.<sup>[9–11]</sup> However, studies evaluating the relationship between these indices and ultrasonographic hepatic steatosis in morbidly obese individuals, particularly in bariatric surgery candidates, are limited.<sup>[12,13]</sup> Demonstrating this relationship is clinically important for the non-invasive evaluation of hepatic steatosis.

In this study, we evaluated the relationship between insulin resistance-based indices and ultrasonographically detected hepatic steatosis in patients with morbid obesity, and investigated their potential clinical value in predicting ultrasonographically detected hepatic steatosis in candidates for bariatric surgery.

## Methods

### Study Design and Population

This study was designed as a retrospective observational study. The study protocol was approved by the Non-Interventional Clinical Research Ethics Committee of Elazığ Fethi Sekin City Hospital (date: March 26, 2026; decision no: 2026/26-01) and was conducted in accordance with the principles of the Declaration of Helsinki.

The clinical, laboratory, and radiological data of consec-

utive patients who met the inclusion criteria and were scheduled for bariatric surgery for morbid obesity between March 2024 and February 2026 in the General Surgery Department of Elazığ Fethi Sekin City Hospital were analyzed. To ensure population homogeneity, only patients who underwent primary laparoscopic sleeve gastrectomy were included in the study. Patients who underwent gastric bypass or revision bariatric surgery were excluded. All surgical procedures were performed using a standardized laparoscopic sleeve gastrectomy technique.<sup>[14]</sup>

#### *Inclusion Criteria:*

Age  $\geq 18$  years

- Body mass index (BMI)  $\geq 40$  kg/m<sup>2</sup> or  $\geq 35$  kg/m<sup>2</sup> with obesity-related comorbidities
- Undergoing primary laparoscopic sleeve gastrectomy for morbid obesity
- Availability of preoperative laboratory data
- Preoperative assessment of hepatic steatosis by abdominal ultrasonography

#### *Exclusion Criteria:*

- History of alcohol consumption
- Presence of viral hepatitis
- Use of hepatotoxic medications
- Presence of chronic liver disease
- Missing clinical or laboratory data

### Laboratory Measurements and Index Calculations

Preoperative laboratory data were obtained from the hospital information system. Fasting plasma glucose, fasting insulin, AST, ALT, HbA1c, and cortisol levels were recorded.

To evaluate insulin resistance, the homeostasis model assessment of insulin resistance (HOMA-IR) and quantitative insulin sensitivity check index (QUICKI) were calculated.<sup>[15,16]</sup>

HOMA-IR was calculated using the following formula:<sup>[15]</sup>  

$$\text{HOMA-IR} = [\text{fasting glucose (mg/dL)} \times \text{fasting insulin } (\mu\text{U/mL})] / 405$$

QUICKI was calculated using the following formula:<sup>[16]</sup>  

$$\text{QUICKI} = 1 / [\log(\text{fasting insulin } (\mu\text{U/mL})) + \log(\text{fasting glucose (mg/dL)})]$$

To assess hepatic steatosis, the Hepatic Steatosis Index (HSI) and NAFLD liver fat score (NAFLD-LFS) were calculated.<sup>[11,17]</sup>

The HSI was calculated as follows:<sup>[17]</sup>  

$$\text{HSI} = 8 \times (\text{ALT} / \text{AST}) + \text{BMI}$$

Additional +2 points were awarded for female sex and +2 points for the presence of diabetes mellitus.

NAFLD Liver Fat Score was calculated as follows:<sup>[11]</sup>  

$$\text{NAFLD-LFS} = -2.89 + 1.18 \times \text{metabolic syndrome} + 0.45 \times$$

diabetes + 0.15 × insulin + 0.04 × AST – 0.94 × (AST/ALT)

Metabolic syndrome was defined according to the International Diabetes Federation and harmonized criteria as the presence of ≥3 components.<sup>[18]</sup>

### Data Collection and Variables

Demographic and clinical data of the included patients were obtained from electronic medical records. Age, sex, height, weight, and body mass index (BMI) were recorded. Comorbidities, including diabetes mellitus, hypertension, and hyperlipidemia, were also evaluated.

The primary outcome variable was the ultrasonographically determined grade of hepatic steatosis.

### Ultrasonographic Assessment of Hepatic Steatosis

In all patients, the liver parenchyma was evaluated preoperatively using abdominal ultrasonography. Ultrasonographic examinations were performed by experienced radiologists who were blinded to the laboratory results.

Hepatic steatosis was graded based on ultrasonographic findings as follows: grade 1 (mild), grade 2 (moderate), and grade 3 (severe).

For the ROC analysis, hepatic steatosis was categorized into two groups: grade 1 (mild steatosis) and grade ≥2 (moderate-to-severe steatosis).<sup>[19]</sup> However, the number of radiologists was limited, and interobserver variability was not assessed, which should be considered a limitation of the study.

### Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA).

The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test and visual methods (histograms and Q–Q plots). Normally distributed variables were expressed as mean ± standard deviation, while non-normally distributed variables were presented as median (interquartile range). Categorical variables were expressed as numbers and percentages.

For comparisons between groups, the independent samples t-test was used for normally distributed variables, and the Mann–Whitney U test was used for non-normally distributed variables. Categorical variables were compared using the chi-squared test or Fisher's exact test.

The relationships between ultrasonographic hepatic steatosis grade and HOMA-IR, QUICKI, HSI, NAFLD-LFS, HbA1c, and other biochemical parameters were evaluated using Spearman's correlation analysis.

The diagnostic performance of these indices in predicting

ultrasonographically detected hepatic steatosis was evaluated using receiver operating characteristic (ROC) curve analysis. The area under the curve (AUC) with 95% confidence intervals was calculated. Optimal cutoff values were determined using the Youden index.

Multivariate logistic regression analysis was performed to identify independent predictors of hepatic steatosis. Variables that were clinically relevant and had a p-value <0.10 in the univariate analysis were included in the model. Results were presented as odds ratios (ORs) with 95% confidence intervals.

Patients with missing data were excluded from the analysis, and a two-tailed p-value <0.05 was considered statistically significant.

## Results

### Patient Characteristics

A total of 206 patients were included in the study. The median age was 34 years (IQR: 27–45), and 62.6% of the patients were female. The median BMI was 42.97 kg/m<sup>2</sup> (IQR: 40.82–46.06). Ultrasonographic evaluation revealed mild steatosis in 28.2% and moderate-to-severe steatosis in 71.8% of the patients. The baseline characteristics are summarized in Table 1.

### Comparison According to Steatosis Severity

Patients with moderate-to-severe steatosis had significantly higher BMI, glucose metabolism parameters, liver enzyme levels, HSI, and NAFLD-LFS values, whereas QUICKI values were significantly lower. Male sex was more frequent in this group (Table 2).

**Table 1.** Baseline characteristics of the study population

Variable	Total cohort (n=206)
Age, years	34 (27–45)
Female, n (%)	129 (62.6)
Male, n (%)	77 (37.4)
BMI, kg/m <sup>2</sup>	42.97 (40.82–46.06)
Length of hospital stay, days	4 (4–5)
Diabetes mellitus, n (%)	45 (21.8)
Hypertension, n (%)	19 (9.2)
Grade 1 steatosis, n (%)	58 (28.2)
Grade 2 steatosis, n (%)	122 (59.2)
Grade 3 steatosis, n (%)	26 (12.6)

BMI, body mass index; IQR, interquartile range. Continuous variables are presented as mean ± standard deviation or median (interquartile range), as appropriate. Categorical variables are presented as number (percentage).

**Table 2.** Comparison between patients with mild steatosis and moderate-to-severe steatosis

Variable	Grade 1 (n=58)	Grade ≥2 (n=148)	P
Age, years	29 (25–36)	36.5 (28–46)	0.002
BMI, kg/m <sup>2</sup>	41.45 (40.23–43.28)	43.94 (41.25–47.07)	<0.001
Fasting glucose, mg/dL	99.5 (87.25–110.0)	106 (91–132)	0.033
Fasting insulin, μIU/mL	21.05 (14.22–29.66)	24.39 (15.99–37.36)	0.045
HOMA-IR	5.11 (3.32–8.49)	6.38 (4.51–11.22)	0.01
QUICKI	0.302 (0.283–0.320)	0.293 (0.273–0.307)	0.01
AST, U/L	19 (17–21)	23 (19–30)	<0.001
ALT, U/L	21 (17.25–26)	30.5 (20–43.2)	<0.001
HbA1c, %	5.6 (5.4–5.9)	5.9 (5.6–6.6)	<0.001
HSI	52.85 (51.05–55.26)	55.78 (53.17–60.26)	<0.001
NAFLD-LFS	0.48 (-0.91–1.74)	1.35 (0.20–3.43)	0.001

BMI, body mass index; IQR, interquartile range; HOMA-IR, homeostasis model assessment of insulin resistance; QUICKI, quantitative insulin sensitivity check index; AST, aspartate aminotransferase; ALT, alanine aminotransferase; HbA1c, glycated hemoglobin; HSI, hepatic steatosis index; NAFLD-LFS, nonalcoholic fatty liver disease liver fat score. Comparisons between groups were performed using the Mann-Whitney U test. Continuous variables are presented as median (interquartile range). p<0.05 was considered statistically significant.

**Table 3.** Correlation between steatosis grade and metabolic/biochemical parameters

Variable	Correlation coefficient (r)	p
HOMA-IR	0.244	<0.001
QUICKI	-0.244	<0.001
HSI	0.354	<0.001
NAFLD-LFS	0.297	<0.001
HbA1c	0.274	<0.001
Fasting glucose	0.170	0.015
Fasting insulin	0.195	0.005
AST	0.366	<0.001
ALT	0.354	<0.001
Cortisol	-0.002	0.982

HOMA-IR, homeostasis model assessment of insulin resistance; QUICKI, quantitative insulin sensitivity check index; HSI, hepatic steatosis index; NAFLD-LFS, nonalcoholic fatty liver disease liver fat score; HbA1c, glycated hemoglobin; AST, aspartate aminotransferase; ALT, alanine aminotransferase. Spearman correlation analysis was used. p < 0.05 was considered statistically significant.

### Correlation and ROC Analysis

Steatosis grade was significantly positively correlated with HOMA-IR, HSI, NAFLD-LFS, and liver enzymes, whereas QUICKI was negatively correlated. Cortisol levels were not associated with steatosis grade (Table 3).

ROC analysis demonstrated that HSI had the highest diagnostic performance for predicting moderate-to-severe steatosis (AUC=0.716), followed by HbA1c (AUC=0.656) and NAFLD-LFS (AUC=0.645) (Table 4).

### Multivariable Logistic Regression Analysis

In multivariable analysis, age (OR=1.04), BMI (OR=1.23), and NAFLD-LFS (OR=1.42) were identified as independent predictors of moderate-to-severe steatosis, whereas female sex was associated with lower odds (OR=0.34). Other indices were not independent predictors (Table 5).

### Discussion

This study demonstrated that insulin resistance-related indices and biochemical parameters were significantly associated with ultrasonographic hepatic steatosis in patients with morbid obesity undergoing bariatric surgery. Patients with moderate-to-severe steatosis exhibited a markedly adverse metabolic profile, and HSI showed the highest diagnostic performance, whereas age, BMI, and NAFLD-LFS emerged as independent predictors. These findings suggest that hepatic steatosis in patients with morbid obesity represents a multifactorial metabolic condition that cannot be explained by insulin resistance alone.<sup>[20,21]</sup>

**Table 4.** Diagnostic performance of non-invasive indices for predicting moderate-to-severe steatosis: ROC analysis

Parameter	AUC	Cut-off	Sensitivity (%)	Specificity (%)
HOMA-IR	0.616 (0.536–0.696)	5.65	60.8	58.6
QUICKI	0.616 (0.536–0.696)	0.298	60.8	58.6
HSI	0.716 (0.642–0.790)	54.51	68.0	70.7
NAFLD-LFS	0.645 (0.565–0.724)	-0.051	79.7	43.1
HbA1c	0.656 (0.577–0.735)	5.8	63.3	62.1

AUC, area under the curve; CI, confidence interval; HOMA-IR, homeostasis model assessment of insulin resistance; QUICKI, quantitative insulin sensitivity check index; HSI, hepatic steatosis index; NAFLD-LFS, nonalcoholic fatty liver disease liver fat score; HbA1c, glycated hemoglobin. Receiver operating characteristic (ROC) analysis was performed to evaluate the diagnostic performance of each parameter. p<0.05 was considered statistically significant.

**Table 5.** Multivariable logistic regression analysis for moderate-to-severe steatosis

Variable	OR	95% CI	p
Age	1.04	1.01–1.08	0.022
Female sex	0.34	0.15–0.78	0.011
BMI	1.23	1.03–1.46	0.020
HOMA-IR	0.92	0.80–1.06	0.243
HSI	1.02	0.88–1.17	0.807
NAFLD-LFS	1.42	1.01–2.00	0.042
HbA1c	1.31	0.86–2.00	0.214

OR, odds ratio; CI, confidence interval; BMI, body mass index; HOMA-IR, homeostasis model assessment of insulin resistance; HSI, hepatic steatosis index; NAFLD-LFS, nonalcoholic fatty liver disease liver fat score; HbA1c, glycated hemoglobin. Multivariable logistic regression analysis was performed to identify independent predictors of moderate-to-severe steatosis.  $p < 0.05$  was considered statistically significant.

Although ultrasonography is widely used in clinical practice, it is an operator-dependent modality with limited sensitivity, particularly in detecting mild hepatic steatosis (<20% liver fat content). Therefore, the findings of this study should be interpreted within the context of ultrasonography-based assessment and reflect ultrasonographically detected hepatic steatosis rather than histopathologically confirmed hepatic steatosis.<sup>[8,19]</sup>

Insulin resistance is widely recognized as a central mechanism in the pathogenesis of hepatic steatosis, primarily through increased lipolysis, elevated free fatty acid flux, and enhanced hepatic de novo lipogenesis. In line with previous studies, HOMA-IR and QUICKI were significantly associated with steatosis severity in our cohort.<sup>[20,22]</sup> However, the loss of significance of HOMA-IR in multivariable analysis indicates that insulin resistance alone is insufficient to explain hepatic fat accumulation in morbid obesity. Additional mechanisms, including adipocyte dysfunction, chronic low-grade inflammation, and metabolic reprogramming of hepatocytes, likely play critical roles.<sup>[23,24]</sup>

A key finding of this study is the superior diagnostic performance of HSI compared to other parameters. Composite indices appear to outperform single biochemical markers, reflecting the multidimensional nature of hepatic steatosis. The superior performance of HSI in this cohort may be attributed to the high metabolic burden associated with morbid obesity, where indices incorporating anthropometric parameters provide additional discriminative value beyond insulin resistance alone.<sup>[20,25]</sup> The inclusion of BMI and transaminase ratios in HSI likely enhances its ability to capture both metabolic burden and hepatic injury.<sup>[26,27]</sup> Similarly, NAFLD-LFS remained an independent predictor,

possibly due to its incorporation of insulin resistance and metabolic syndrome components.<sup>[20]</sup> The identified cut-off value of HSI may be useful in clinical decision-making, particularly for stratifying patients according to the risk of moderate-to-severe steatosis.

Some findings should be interpreted in the context of the underlying mathematical relationships. The identical diagnostic performance of HOMA-IR and QUICKI is expected, as they are inverse transformations of the same variables. Moreover, the lack of independent predictive value of HSI despite its strong ROC performance likely reflects collinearity with BMI and liver enzymes. Likewise, HOMA-IR and HbA1c lost significance after adjustment, whereas NAFLD-LFS retained its predictive value because of its composite structure. These observations highlight the complex and interrelated nature of the metabolic determinants of hepatic steatosis.

Previous studies in the general population have frequently identified the Fatty Liver Index (FLI) as the most accurate non-invasive marker; however, in high-risk populations such as morbidly obese individuals, HSI and NAFLD-LFS have also demonstrated strong diagnostic performance.<sup>[26,28]</sup> The prominence of HSI in our cohort suggests that indices that incorporate anthropometric and hepatic parameters may better capture the spectrum of steatosis in this specific population.<sup>[21,29]</sup>

Although non-invasive indices have certain advantages, biopsy-based studies indicate that these tools may have limited accuracy in quantifying hepatic fat content at the individual level.<sup>[21,28]</sup> Therefore, such indices should be considered complementary tools for screening and risk stratification rather than replacements for histopathological or advanced imaging methods.

From a clinical perspective, our findings support the use of simple, cost-effective, and non-invasive indices in the pre-operative evaluation of bariatric surgery candidates. These tools may facilitate the early identification of high-risk patients and guide the selection of individuals who require further diagnostic evaluation. These findings suggest that non-invasive indices may help reduce the need for additional imaging or invasive diagnostic procedures, such as liver biopsy, in selected bariatric surgery candidates. In clinical practice, the use of HSI may reduce the need for additional imaging or invasive evaluation in selected bariatric surgery candidates with a high probability of steatosis.<sup>[30–32]</sup>

This study has several strengths, including a relatively large sample size, a homogeneous surgical population, and a comparative evaluation of multiple indices within the same cohort. However, certain limitations should be acknowledged. The retrospective design introduces poten-

tial selection bias. The use of ultrasonography instead of liver biopsy may limit diagnostic accuracy. Additionally, the restriction of the study population to morbidly obese patients undergoing bariatric surgery limits generalizability. The absence of detailed lipid profile parameters is another limitation.

Future studies should validate these findings using prospective, multicenter designs that incorporate histopathological confirmation. The development of combined predictive models and the integration of artificial intelligence-based approaches may improve the non-invasive assessment of hepatic steatosis. Population-specific scoring systems tailored to morbid obesity also represent a promising area for further research.<sup>[24,33,34]</sup> Taken together, these results support the clinical utility of non-invasive indices as accessible tools for risk stratification in bariatric populations.

## Conclusion

HSI and NAFLD-LFS demonstrated superior diagnostic performance compared with single insulin resistance markers in the assessment of ultrasonographically detected hepatic steatosis. Age, BMI, and NAFLD-LFS were identified as independent predictors of moderate-to-severe steatosis, indicating that the disease is driven by multifactorial metabolic mechanisms beyond insulin resistance alone.

These indices may serve as practical and cost-effective tools in the preoperative evaluation of bariatric surgery candidates. However, prospective, multicenter studies with histopathological confirmation are required to validate these findings. The development of combined indices and artificial intelligence-based models may further improve diagnostic accuracy.

## Disclosures

**Ethics Committee Approval:** This study was approved by The Elazığ Fethi Sekin City Hospital Non-Interventional Clinical Research Ethics Committee (Decision No: 2026/26-01; 26.03.2026).

**Informed Consent:** Informed consent was waived by the Institutional Review Board due to the retrospective design.

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