RESEARCH ARTICLE

Use of Liver Stiffness–Platelet Count–Spleen Size Score to predict the presence of Esophageal Varices and their grade in Patients of Liver Cirrhosis

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Abstract

Background: Portal hypertension is a major consequence of liver cirrhosis and leads to the development of esophageal varices, which are associated with significant morbidity and mortality. Upper gastrointestinal endoscopy (UGIE) remains the gold standard for diagnosis; however, it is invasive, costly, and not universally accessible. Non-invasive predictors such as the liver stiffness–platelet count–spleen size score (LSPS) have emerged as promising alternatives for predicting esophageal varices.

Objectives: To evaluate the correlation between LSPS and the grading of esophageal varices in patients with liver cirrhosis and to assess the utility of LSPS as a non-invasive tool for risk stratification.

Methods: This cross-sectional observational study was conducted in the Department of Medicine, Government Medical College and Rajindra Hospital, Patiala. One hundred adult patients with liver cirrhosis confirmed clinically, biochemically, radiologically, and by transient elastography (FibroScan® liver stiffness measurement ≥12.5 kPa) were included. Liver stiffness measurement, spleen bipolar diameter, platelet count, and upper gastrointestinal endoscopy findings were recorded. LSPS was calculated using the formula: liver stiffness (kPa) × spleen diameter (cm) / platelet count ($\times 10^9$/L). Statistical analysis included chi-square test, t-test, and ANOVA.

Results: The mean age of participants was 49.4 ± 13.4 years, with male predominance (68%). Alcohol-related cirrhosis was the most common etiology (32%), followed by hepatitis C infection and metabolic associated steatohepatitis (20% each). Esophageal varices were present in 52% of patients, including Grade 1 varices in 32%, Grade 2 in 12%, and Grade 3 in 8%. High-risk varices were identified in 20% of patients. Mean LSPS among study participants was 1.72 ± 1.43. Patients with higher grades of esophageal varices demonstrated significantly elevated LSPS values. LSPS showed a positive correlation with variceal grade and effectively identified patients with high-risk varices.

Conclusion: LSPS is a simple, reliable, and non-invasive predictor of esophageal varices and correlates positively with variceal severity in cirrhotic patients. It may serve as a useful screening and risk stratification tool, especially in resource-limited settings, potentially reducing unnecessary endoscopic procedures.

Keywords: Liver cirrhosis; Esophageal varices; LSPS; Portal hypertension; FibroScan; Liver stiffness measurement

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Introduction

Chronic liver disease (CLD) remains a major global health burden and contributes substantially to morbidity and mortality worldwide. Progressive hepatic fibrosis ultimately lead to cirrhosis, characterized by distortion of hepatic architecture and development of portal hypertension. Among the various complications of portal hypertension, esophageal varices represent one of the most clinically significant manifestations because of their association with life-threatening gastrointestinal hemorrhage.[1]

Approximately 30–40% of patients with compensated cirrhosis and up to 85% of patients with decompensated cirrhosis harbor esophageal varices. The first episode of variceal bleeding carries considerable mortality despite advances in management.[2] Therefore, early detection and risk stratification of varices are essential components in the management of cirrhotic patients.

Upper gastrointestinal endoscopy (UGIE) is currently regarded as the gold standard for diagnosing and grading esophageal varices.[3] However, universal endoscopic screening is limited by its invasive nature, cost, patient discomfort, and limited availability in many healthcare settings. These limitations have stimulated interest in the development of reliable non-invasive methods for predicting the presence and severity of esophageal varices.

Transient elastography (FibroScan®) has emerged as a valuable non-invasive modality for assessment of liver fibrosis and portal hypertension.[4] The liver stiffness–platelet

count–spleen size score (LSPS), calculated from liver stiffness measurement, spleen size, and platelet count, integrates three important parameters reflecting portal hypertension severity. Previous studies have demonstrated promising diagnostic accuracy of LSPS in predicting esophageal varices and high-risk varices.

The present study was undertaken to evaluate the correlation between LSPS and the grading of esophageal varices in patients with liver cirrhosis and to assess its role as a non-invasive predictor for risk stratification.

Materials and Methods

This cross-sectional observational study was conducted over one and a half years in the Department of Medicine, Government Medical College and Rajindra Hospital, Patiala, and included 100 adult patients with liver cirrhosis attending the outpatient department or admitted to medicine wards. Diagnosis of cirrhosis was established on the basis of clinical, biochemical, and radiological findings along with liver stiffness measurement (LSM) ≥12.5 kPa on transient elastography (FibroScan®). Patients aged ≥18 years who provided informed consent were included in the study. Patients with liver stiffness <12.5 kPa, previous or ongoing endoscopic treatment for varices, massive ascites, metastatic malignancy, significant renal, cardiac, or respiratory failure, and those unwilling to participate were excluded. All patients underwent detailed clinical evaluation, laboratory investigations including complete blood count, liver function tests, coagulation profile, viral markers, ultrasonographic assessment of spleen size, FibroScan® evaluation of liver stiffness, and upper gastrointestinal endoscopy for grading of esophageal varices. The liver stiffness–platelet count–spleen size score (LSPS) was calculated using the formula: liver stiffness (kPa) × spleen diameter (cm) / platelet count ($\times 10^9$/L).[5,6]

Statistical analysis

Data were analyzed using Microsoft Excel 2023 and SPSS version 26. Statistical tests included chi-square, t-test, and ANOVA, with $p<0.05$ considered significant.

Results

The present study included 100 patients with liver cirrhosis with a mean age of 49.4 ± 13.4 years and a male predominance (68%). The mean hemoglobin level was 12.05 ± 1.57 g/dL, while the mean platelet count was 198.09 ± 57.18 $\times 10^9$/L. Mean liver enzymes including AST and ALT were 65.60 ± 25.59 IU/L and 53.19 ± 18.05 IU/L respectively. The mean serum bilirubin and albumin levels were 1.67 ± 1.04 mg/dL and 3.79 ± 0.52 g/dL respectively. The mean spleen size was 13.15 ± 1.59 cm, mean liver stiffness was 20.77 kPa, and mean LSPS was 1.72 ± 1.43, reflecting the presence of significant portal hypertension in the study population. Alcohol-related liver disease was the most common etiology of cirrhosis (32%), followed by hepatitis C infection and metabolic associated steatohepatitis (20% each). Esophageal varices were present in 52% of patients, of whom 32% had low-risk varices and 20% had high-risk varices, indicating a substantial burden of clinically significant portal hypertension among cirrhotic patients (Table 1).

Table 1: Baseline Demographic and Biochemical Characteristics of Study Participants (n = 100)

Variable	*Value*
Mean age(years)	49.4
Male	68
Females	32
Mean hemoglobin(gm/dl)	12.05
Mean platelet count (x$10^{9/dl}$)	198.09
Mean AST (IU/L)	65.6
Mean ALT (IU/L)	53.19
Mean bilirubin (mg/dl)	1.67
Mean serum albumin (gm/dl)	3.79
Mean spleen size(cm)	13.15
Mean liver stiffness (kPa)	20.77
Mean LSPS	1.72
Etiology of cirrhosis	
Alcohol	32
Hepatitis C	20
Metabolic associated steatohepatitis	20
Hepatitis B	14
Cryptogenic	8
Autoimmune hepatitis	4
Primary biliary cholangitis	2
Grading of esophageal varices	
Esophageal varices present	52
Low risk varices	32
High risk varices (grade II/III, CRS, RWS)	20

Comparison between patients with and without esophageal varices demonstrated significant differences in several hematological, biochemical, and portal hypertension-related parameters. Patients with esophageal varices had significantly lower mean hemoglobin levels (11.34 vs 12.82 g/dL; p=0.001) and platelet counts (166 vs 231 $\times 10^9$/L), reflecting hypersplenism and advanced portal hypertension. Liver function abnormalities were more pronounced in patients with varices, with higher mean AST (77.48 vs 52.74 IU/L), ALT (59.25 vs 46.62 IU/L), and bilirubin levels (2.08 vs 1.22 mg/dL), while serum albumin was lower (3.44 vs 4.16 g/dL), indicating more severe hepatic dysfunction. Parameters related to portal hypertension were markedly elevated in the variceal group, including mean spleen size (14.20 vs 12.02 cm), liver stiffness (25.47 vs 15.67 kPa), and LSPS (2.54 vs 0.83), suggesting a strong association between increasing LSPS

Table 2: Comparison of Demographic and Biochemical Characteristics between patients with and without Esophageal varices

Variable	*Varices absent (value)*	*Varices present (value)*	*p-value*
Male	30	38	<0.257
Females	18	14	
Mean hemoglobin (gm/dl)	12.82	11.34	<0.001
Mean platelet count ($10x^9$/L)	231	166	
Mean AST (IU/L)	52.74	77.48	
Mean ALT (IU/L)	46.62	59.25	
Mean bilirubin (mg/dl)	1.22	2.08	
Mean serum albumin (gm/dl)	4.16	3.44	
Mean spleen size (cm)	12.02	14.20	
Mean liver stiffness (kPa)	15.67	25.47	
Mean LSPS	0.83	2.54	
Etiology of cirrhosis			
Alcohol	16	16	<0.967
Hepatitis C	9	11	
Metabolic associated steatohepatitis	8	12	
Hepatitis B	7	7	
Cryptogenic	5	3	
Autoimmune hepatitis	2	2	
Primary biliary cholangitis	1	1	

and the presence of esophageal varices. Gender distribution and etiological profile of cirrhosis were comparable between the two groups, with no statistically significant difference observed (p=0.257 and p=0.967 respectively) (Table 2).

Comparison of LSPS and related biochemical parameters across different grades of esophageal varices demonstrated a progressive worsening trend with increasing variceal severity. Mean LSPS increased significantly from 1.54 in Grade 1 varices to 3.08 in Grade 2 and 5.76 in Grade 3 varices (p=0.001), indicating a strong positive association between LSPS and severity of portal hypertension. Platelet count progressively decreased with higher variceal grades, from 193.38 $\times 10^9$/L in Grade 1 to 107.38 $\times 10^9$/L in Grade 3 varices, reflecting increasing hypersplenism and portal hypertension. Similarly, spleen size and liver stiffness showed a stepwise increase with advancing variceal grade, with mean spleen size increasing from 13.42 cm in Grade 1 to 16.66 cm in Grade 3 varices, while mean liver stiffness increased from 21.73 kPa to 36.53 kPa. Serum albumin levels progressively declined with worsening variceal grade, suggesting deterioration in hepatic synthetic function. These findings demonstrate that higher LSPS values are strongly associated with increasing severity of esophageal varices and support the utility of LSPS as a non-invasive marker for risk stratification in cirrhotic patients (Table 3).

LSPS demonstrated the best diagnostic accuracy with an area under the curve (AUC) of 1.000 at an optimal cut-off value of ≥1.14, yielding 100% sensitivity and 100% specificity (p<0.001). Liver stiffness measurement also showed excellent predictive performance with an AUC of 0.980 at a cut-off ≥18.70 kPa, providing 90.4% sensitivity and 100% specificity. Spleen size demonstrated good diagnostic accuracy with an AUC of 0.929 and a cut-off value of ≥13.25 cm, while platelet count showed comparatively lower performance with an

Table 3: Comparison of LSPS and Biochemical Characteristics of patients with grading of Esophageal varices

Variable	*Grade 1 varices*	*Grade 2 varices*	*Grade 3 varices*	*P value*
LSPS	1.54	3.08	5.76	<0.001
Platelet count ($x10^9$/L)	193.38	135.75	107.38	
Spleen size (cm)	13.42	14.63	16.66	
Liver stiffness (Kpa)	21.73	28.07	36.53	
Serum albumin (gm/dl)	3.58	3.34	3.01	

Table 4: Predictive value of non-invasive parameters for identifying high risk Esophageal varices among 52 patients complicated with Esophageal varices

Marker	*AUC*	*Best cut off*	*Sensitivity (%)*	*Specificity (%)*	*p-value*
LSPS	1.000	≥ 1.14	100.0	100.00	<0.001
Liver stiffness (kPa)	0.980	≥ 18.70	90.4	100.00	
Spleen size (cm)	0.929	≥ 13.25	73.1	100.00	
Platelet count (x10^9/L)	0.829	≤ 161.5	55.8	95.8	

AUC of 0.829 at a cut-off ≤161.5 ×10^9/L. Among all evaluated parameters, LSPS showed the highest predictive accuracy for identifying high-risk esophageal varices, highlighting its superiority as a reliable non-invasive marker for risk stratification in cirrhotic patients.

Discussion

The present study evaluated the utility of the liver stiffness–platelet count–spleen size score (LSPS) as a non-invasive predictor of esophageal varices in patients with liver cirrhosis. The findings demonstrated a significant association between increasing LSPS values and higher grades of esophageal varices.

The mean age of study participants was 49.4 years with male predominance, findings comparable to previous studies evaluating portal hypertension and variceal risk in cirrhotic patients. Alcohol-related liver disease was the most common etiology in the present study, reflecting the changing epidemiological pattern of chronic liver disease in North India.

Esophageal varices were present in more than half of the study population, with Grade 1 varices being the most common. High-risk varices were identified in one-fifth of patients, emphasizing the need for reliable methods for early identification and surveillance.

The pathophysiological basis of LSPS lies in the integration of liver stiffness, splenic enlargement, and thrombocytopenia, all of which are indirect markers of portal hypertension. Liver stiffness reflects hepatic fibrosis and intrahepatic vascular resistance, while splenomegaly and thrombocytopenia mirror chronic portal venous congestion and hypersplenism.

The present findings are consistent with previous studies by Kim et al., Berzigotti et al., and Shibata et al.,[5,7,8] which demonstrated that LSPS possesses superior diagnostic accuracy for prediction of esophageal varices compared to individual parameters alone. Similar to these studies, higher LSPS values in the present study correlated with greater variceal severity.

The clinical significance of LSPS lies in its simplicity, reproducibility, and non-invasive nature. Since the score utilizes routinely available investigations such as FibroScan®, ultrasonography, and platelet count, it may serve as a practical screening tool in routine hepatology practice. Use of LSPS may help reduce unnecessary endoscopic screening in low-risk patients while prioritizing high-risk individuals for early endoscopic evaluation and prophylactic therapy. However the study is a single tertiary care center study, which may limit generalizability. The sample size was relatively small and patients with massive ascites were excluded, which may affect applicability in advanced decompensated cirrhosis. Moreover FibroScan is not widely available in most part of this region.

Conclusion

The present study demonstrates that LSPS correlates positively with the grading of esophageal varices in patients with liver cirrhosis. Higher LSPS values were associated with increasing variceal severity and the presence of high-risk varices. It may be seen as a reliable non-invasive tool for predicting esophageal varices and risk stratification in cirrhotic patients when endoscopy is not readily available. However further multicentric studies with larger sample sizes are recommended to validate optimal cut-off values and strengthen its clinical applicability.

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