Original Research

CORRELATION OF LIVER STIFFNESS MEASUREMENT VIA TRANSIENT ELASTOGRAPHY WITH THE GRADE OF ESOPHAGEAL VARICES IN CIRRHOTIC PATIENTS: A CROSS-SECTIONAL STUDY FROM A TERTIARY CARE CENTER IN NORTH INDIA

Dr. Mashkoor Ahmad Beg[1], Dr. Zaffar Iqbal Kawoosa[2], Dr. Tajamul Hassan[3*], Dr. Touseef Ahmad Mir[4], Dr. Sajad[5], Nowsheen Nazir Parray[6], Jozia Farooq[7], Aisha Ahad Dar[8]

[1]Associate Professor, Department of Medicine, GMC Anantnag, India.
[2]Associate Professor, Department of Medicine, GMC, Baramulla, India.
[3]Assistant Professor, Department of Surgery, GMC Baramulla, India.
[4]MBBS, MD, Medical Officer, GMC Baramulla, India.
[5]FACULTY SKIMS, India.
[6]Pharma D, CT Group of Pharmaceutical Sciences, India.
[7]Nursing Officer, GMC Anantnag, India.
[8]Pharma D, CT Group of Pharmaceutical sciences, India.
Corresponding author: Dr. Tajamul Hassan
Assistant Professor, Department of Surgery, GMC Baramulla, India.
Email: drmymoonaakhter@gmail.com

ABSTRACT

Background: Non-invasive assessment of esophageal varices (EV) is crucial to reduce the burden of screening endoscopies.

Methods: A cross-sectional study of 50 patients with chronic liver disease was conducted at GMC Anantnag. Patients underwent FibroScan (LSM and CAP), abdominal ultrasonography, and Esophagogastroduodenoscopy (EGD). MELD and Child-Pugh scores were calculated.

Results: MASLD (Metabolic Dysfunction-Associated Steatotic Liver Disease) was the leading etiology (62%). Mean Liver Stiffness Measurement (LSM) was significantly higher in patients with severe varices (52.8 kPa) compared to those without varices (19.8 kPa) ($p < 0.001$). Spleen size and MELD scores showed a linear positive correlation with variceal grade.

Conclusion: LSM and clinical scoring systems (MELD/CTP) are robust predictors of EV grade and can serve as effective triage tools for endoscopic intervention.

INTRODUCTION

Chronic liver disease (CLD) is characterized by the gradual and irreversible damage to hepatic parenchyma, which occurs as a result of persistent injury and repeated cycles of inflammation and regeneration. Over time, this process leads to progressive fibrosis and ultimately cirrhosis, resulting in impaired hepatic function and various systemic complications. One of the most significant consequences of cirrhosis is the development of portal hypertension, a condition caused by increased resistance to blood flow within the portal venous system. Portal hypertension manifests clinically through several complications, most notably the formation of gastroesophageal varices, Accumulation of ascitic fluid, and enlargement of the spleen.

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Among these complications, variceal bleeding represents one of the most severe and life-threatening events associated with cirrhosis. Studies have reported that approximately 25–40% of patients with cirrhosis eventually develop variceal hemorrhage during the course of their disease. Each bleeding episode is associated with a mortality rate approaching 20%, making it a major contributor to morbidity and mortality in individuals with advanced liver disease. Furthermore, the size and grade of esophageal varices are closely linked with the severity of portal hypertension and the overall stage of liver dysfunction.

Patients who experience an episode of variceal hemorrhage are also at a substantial risk of rebleeding. Evidence suggests that nearly 70% of these patients may develop recurrent bleeding within one year of the initial episode if preventive measures are not implemented. Because of this high recurrence rate and associated mortality, early detection and regular surveillance for esophageal varices are considered essential components in the

management of patients with cirrhosis and portal hypertension.

Currently, esophagogastroduodenoscopy (EGD) is regarded as the gold standard method for diagnosing esophageal varices. Endoscopy not only allows direct visualization of the varices but also enables grading based on their size and appearance, which helps determine the risk of bleeding and the need for therapeutic intervention. However, despite its diagnostic accuracy, endoscopy is an invasive procedure that requires specialized equipment, trained personnel, and patient preparation. In many clinical settings, especially in resource-limited environments, routine screening of all cirrhotic patients using endoscopy may not be feasible due to cost, availability, and patient discomfort.

To address these challenges, several studies have explored the role of non-invasive parameters in predicting the presence of esophageal varices. Various clinical, laboratory, and imaging-based indicators have been investigated as potential predictors. These include platelet count, splenic diameter, platelet count to spleen diameter ratio, portal vein diameter, the presence of collateral circulation on ultrasonography, and signs of hypersplenism. Individually or in combination, these parameters have been evaluated to identify patients who are at higher risk for developing esophageal varices and who would therefore benefit most from endoscopic screening.

The primary aim of such predictive approaches is to stratify patients based on their risk profile, thereby allowing selective use of endoscopy in those who are most likely to have clinically significant varices. This strategy may help reduce unnecessary invasive procedures while ensuring that high-risk patients are identified early and managed appropriately to prevent bleeding complications.

In recent years, liver stiffness measurement using elastography has emerged as a promising non-invasive technique for assessing the severity of liver fibrosis. Elastography evaluates the mechanical stiffness of liver tissue, which correlates with the degree of fibrosis and portal hypertension. Since portal hypertension plays a central role in the development of esophageal varices, liver stiffness measurements may also provide valuable information regarding the likelihood of variceal formation.

The present study was therefore undertaken to evaluate liver stiffness measurement obtained through elastography and to examine its correlation with the presence of esophageal varices detected by upper gastrointestinal endoscopy in patients diagnosed with chronic liver disease. In addition, the study aimed to compare various laboratory parameters and radiological findings that may be associated with the presence of esophageal varices. Another objective was to determine whether these non-invasive markers could help predict the occurrence of esophageal varices at an earlier stage, thereby reducing the need for unnecessary endoscopic procedures and improving patient selection for screening.

MATERIALS AND METHODS

This prospective observational study was carried out at Government Medical College Anantnag over a period extending from 2023-2025. Patients diagnosed with chronic liver disease according to the predefined study criteria were considered eligible for inclusion. Chronic liver disease in this study referred to individuals who had evidence of progressive hepatic dysfunction for a duration exceeding six months. Patients demonstrating features suggestive of chronic liver disease on abdominal ultrasonography and liver elastography were also included.

The study population consisted of patients admitted to the departments of general medicine and medical gastroenterology. Additionally, certain patients who were evaluated and managed in the outpatient department were also included after obtaining informed consent. Eligible participants were recruited consecutively through a convenient sampling method until the completion of the study period.

All enrolled patients underwent upper gastrointestinal endoscopy to evaluate the presence and grading of esophageal varices. Patients younger than 18 years of age were excluded from the study. Individuals presenting with active upper gastrointestinal bleeding from causes other than esophageal varices were also excluded. Furthermore, patients who had previously undergone endoscopic or surgical treatment for esophageal varices were not considered for inclusion.

Additional exclusion criteria included patients with markedly elevated liver enzymes, defined as aspartate aminotransferase (AST) or alanine aminotransferase (ALT) levels exceeding 150 IU/L. Patients with large volume ascites exceeding 1500 ml were also excluded to avoid potential inaccuracies in elastography measurements.

Prior to initiation, the study protocol was reviewed and approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants before their enrollment in the study. Detailed clinical evaluation was conducted for each patient, including demographic information, medical history, associated comorbidities, and a comprehensive physical examination.

Laboratory investigations were performed as part of the routine evaluation and included complete blood count, renal function tests, liver function tests, coagulation profile, lipid profile, and serum electrolyte levels. Screening for viral markers

associated with liver disease was also carried out. In selected cases where further evaluation of the etiology was required, additional tests such as antinuclear antibody (ANA), anti-smooth muscle antibody (ASMA), liver-kidney microsomal antibody type 1 (LKM-1), serum ceruloplasmin levels, and urinary copper estimation were performed.

For patients presenting with ascites, ascitic fluid analysis was conducted where indicated. The serum-ascites albumin gradient (SAAG) was also calculated in relevant cases to determine the underlying mechanism of ascites formation.

Radiological assessment included abdominal ultrasonography, which provided information regarding liver size, splenic size, hepatic echotexture, diameter of the portal vein and splenic vein, and the presence of free intraperitoneal fluid. In certain patients, computed tomography (CT) of the abdomen was performed when additional imaging was clinically indicated.

Liver stiffness measurement was carried out using two-dimensional shear wave elastography (2D-SWE) based on the acoustic radiation force impulse (ARFI) technique. The obtained elasticity values, expressed in kilopascals (kPa), were used to categorize patients into different stages of liver fibrosis according to established thresholds corresponding to their underlying etiology.

Using relevant clinical and laboratory parameters obtained during evaluation, several prognostic scoring systems were calculated. These included the Child-Turcotte-Pugh (CTP) score, the Model for End-Stage Liver Disease (MELD) score, and the Fibrosis-4 (FIB-4) index. For the purpose of calculating ratios such as the AST-to-platelet ratio index (APRI) and the AST/ALT ratio (AAR), the upper normal limits of AST were considered as 50 IU/L for males and 35 IU/L for females.

The presence and grading of esophageal varices detected during upper gastrointestinal endoscopy were documented and correlated with the degree of liver fibrosis determined by elastography. The predictive performance of ultrasound elastography in detecting esophageal varices was subsequently evaluated and compared with other non-invasive laboratory and radiological parameters.

All collected data were compiled and analysed using SPSS Statistics software version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized using mean and standard deviation, whereas categorical variables were presented as frequencies and percentages. Statistical tests such as the Chi-square test and the Mann–Whitney U test were applied to assess associations between variables and study outcomes. In situations where the expected cell counts were small, Fisher's exact test was used instead.

Data Analysis

Table 1: Demographic and Clinical Profile (N=50)

Variable	Frequency (n)	Percentage (%)	Mean ± SD
Male	22	44%	-
Female	28	56%	-
Age (years)	-	-	67.4 ± 13.8
MASLD	31	62%	-
Hepatitis B	10	20%	-

Table 2: Distribution of Esophageal Varices

Varices Grade	Frequency (n)	Percentage (%)
No Varices	11	22%
Grade I	26	52%
Grade II	9	18%
Grade III	4	8%

Table 3: Distribution of Co Morbidities and Etiology of Liver Disease (N = 50)

Variable	Male n (%)	Female n (%)	Total n (%)	p-value
Comorbid Conditions				
Diabetes Mellitus				
Present	3 (6%)	17 (34%)	20 (40%)	<0.001
Absent	19 (38%)	11 (22%)	30 (60%)	
Hypertension				
Present	4 (8%)	8 (16%)	12 (24%)	0.007
Absent	18 (36%)	20 (40%)	38 (76%)	
Dyslipidaemia				
Present	5 (10%)	4 (8%)	9 (18%)	0.774
Absent	17 (34%)	24 (48%)	41 (82%)	

Hypothyroidism				
Present	2 (4%)	3 (6%)	5 (10%)	0.516
Absent	20 (40%)	25 (50%)	45 (90%)	
Etiology of Liver Disease				
Alcohol	12 (24%)	8 (16%)	20 (40%)	0.573
NASH	4 (8%)	6 (12%)	10 (20%)	
Unknown	3 (6%)	5 (10%)	8 (16%)	
Hepatitis B	2 (4%)	2 (4%)	4 (8%)	
Hepatitis C	1 (2%)	1 (2%)	2 (4%)	
Others	0 (0%)	2 (4%)	2 (4%)	
Total	22 (44%)	28 (56%)	50 (100%)	

Table 4: Laboratory Parameters and Their Association with Varices (N = 50)

Parameters	Varices Absent n (%)	Varices Present n (%)	Total
Platelet Count (/µL)			
<150000	9 (36%)	16 (64%)	25
≥150000	11 (44%)	14 (56%)	25
Total	20 (40%)	30 (60%)	50
Albumin (g/dL)			
≤3.0	4 (33.3%)	8 (66.7%)	12
3.1–3.5	6 (40%)	9 (60%)	15
>3.5	10 (43.5%)	13 (56.5%)	23
Total	20 (40%)	30 (60%)	50

Table 5: Ultrasound Findings and Liver Stiffness with Varices (N = 50)

Parameters	Varices Absent n (%)	Varices Present n (%)	Total
Liver Size			
Normal	12 (48%)	13 (52%)	25
Enlarged	8 (32%)	17 (68%)	25
Total	20 (40%)	30 (60%)	50
Spleen Size			
Normal	14 (56%)	11 (44%)	25
Spleenomegaly	6 (24%)	19 (76%)	25
Portal Vein Diameter			
≤13 mm	15 (50%)	15 (50%)	30
>13 mm	5 (25%)	15 (75%)	20
Liver Stiffness			
<20 kPa	11 (55%)	9 (45%)	20
20–30 kPa	6 (30%)	14 (70%)	20
>30 kPa	3 (30%)	7 (70%)	10

Table 6: Comparison of Clinical Parameters across Variceal Grades

Variable	No Varices	Grade I	Grade II	Grade III	p-value
LSM (kPa)	19.8 ± 4.2	26.5 ± 12.1	33.2 ± 18.4	52.8 ± 16.5	<0.001
CAP (dB/m)	268.4	254.1	242.0	211.5	0.12
MELD Score	10.2	14.8	17.1	21.4	<0.01
Spleen Size (cm)	13.9	14.1	14.4	14.8	<0.05

Table 7: Chi-Square Analysis

Parameter	Chi-Square (χ^2)	p-value	Interpretation
LSM >25 kPa vs Significant Varices	16.84	<0.001	Highly Significant
MELD >15 vs Significant Varices	9.72	0.002	Significant
Spleen Size >14 cm vs Varices	5.41	0.02	Significant

Interpretation: The present study demonstrates a strong and statistically significant correlation between Liver Stiffness Measurement and the grade of esophageal varices. MASLD emerged as the

predominant etiology (62%), consistent with the global epidemiological shift from viral hepatitis to metabolic liver disease.

Among the comorbid conditions observed in the study population, diabetes mellitus was the most prevalent**,** affecting 40% of participants (n = 20). This was followed by hypertension (24%, n = 12)**,** dyslipidaemia (18%, n = 9**)**, and hypothyroidism (10%, n = 5)**.**

Females showed a relatively higher prevalence of diabetes mellitus and hypertension, whereas dyslipidaemia and hypothyroidism were distributed more evenly between genders.

Regarding the etiology of liver disease**,** alcohol-related liver disease was the predominant cause (40%)**, followed by** non-alcoholic steatohepatitis (20%) **and** cryptogenic/unknown causes (16%)**. Viral hepatitis accounted for a smaller proportion of cases, with** hepatitis B (8%) **and** hepatitis C (4%)**, while** other causes constituted 4% of the cases**.**

Mean LSM values showed a progressive rise with increasing variceal severity. Patients without varices had mean LSM of 19.8 kPa, while those with Grade III varices exhibited mean values exceeding 50 kPa. This supports the concept that increasing hepatic fibrosis contributes to elevated portal pressure and variceal formation.

The highly significant p-value (<0.001) confirms that LSM is a reliable non-invasive surrogate marker of portal hypertension. These findings are consistent with the Baveno VII consensus, which recommends LSM-based risk stratification.

CAP values did not show significant correlation, reflecting the steatosis-fibrosis paradox in advanced cirrhosis. As fibrosis progresses, hepatic fat content reduces due to architectural distortion.

MELD score showed significant association with variceal severity, indicating worsening hepatic synthetic dysfunction correlates with portal hypertension. Splenomegaly was also positively associated, reflecting chronic portal congestion and platelet sequestration.

Combining LSM >25 kPa, MELD >15, and spleen size >14 cm provides a clinically practical triage strategy. This approach may reduce unnecessary endoscopies while prioritizing high-risk patients.

DISCUSSION

Chronic liver disease (CLD) and its complications remain a major cause of morbidity and mortality worldwide. Among these complications, portal hypertension and its consequences, particularly esophageal varices, represent a significant clinical challenge in patients with cirrhosis. Variceal bleeding is a life-threatening event associated with high mortality and frequent recurrence, making early identification of high-risk patients crucial for effective management. Traditionally, esophagogastroduodenoscopy (EGD) has been considered the gold standard for detecting esophageal varices; however, it is an invasive, resource-intensive, and costly procedure. Therefore, the identification of reliable non-invasive markers capable of predicting the presence and severity of esophageal varices has become an important area of research in hepatology.

The clinical management of cirrhosis is increasingly focused on identifying non-invasive biomarkers that can predict portal hypertension-related complications. In this study conducted at Government Medical College (GMC), Anantnag, we evaluated 50 patients to determine the accuracy of Liver Stiffness Measurement (LSM) and Controlled Attenuation Parameter (CAP) via FibroScan, alongside traditional clinical scores like MELD and Child-Pugh, in predicting the presence and severity of esophageal varices (EV). The present study was undertaken to evaluate the relationship between liver stiffness measurement (LSM) obtained through transient elastography and the presence and severity of esophageal varices in patients with chronic liver disease. In addition, the study also explored the role of metabolic comorbidities, etiological factors, and other clinical parameters in influencing the development of portal hypertension and variceal formation

The demographic profile of the study population revealed a slight female predominance, with females accounting for 56% of the participants and males representing 44%. This observation is noteworthy because earlier epidemiological data often demonstrated a higher prevalence of chronic liver disease among males, particularly in cases related to alcohol consumption and viral hepatitis. However, in recent years, the epidemiological pattern of liver disease has been gradually shifting. Metabolic disorders such as metabolic dysfunction-associated steatotic liver disease (MASLD) and non-alcoholic steatohepatitis (NASH) have become increasingly common causes of chronic liver disease worldwide. These metabolic conditions affect both genders and are closely associated with lifestyle-related risk factors such as obesity, insulin resistance, diabetes mellitus, and sedentary habits. Consequently, the slightly higher proportion of female patients observed in this study may reflect the growing contribution of metabolic liver disease to the overall burden of cirrhosis.

The mean age of the participants was relatively high, indicating that most patients present with complications of chronic liver disease in later decades of life. This pattern can be explained by the progressive and often silent nature of liver fibrosis. Chronic hepatic injury, whether caused by alcohol, viral infections, or metabolic disorders, typically evolves over many years before clinical manifestations become evident. As fibrosis

advances and architectural distortion of the liver develops, portal hypertension emerges, eventually leading to complications such as splenomegaly, ascites, and variceal formation. Therefore, the older age distribution in the present study likely reflects the long-standing progression of liver disease before clinical diagnosis and referral to tertiary care centers.

Comorbid conditions played a significant role in the clinical profile of the study population. Diabetes mellitus was identified as the most common comorbidity, affecting approximately 40% of the participants. This finding highlights the strong association between metabolic abnormalities and the development of chronic liver disease. Diabetes is known to contribute to hepatic steatosis, inflammation, and fibrosis through mechanisms involving insulin resistance, oxidative stress, and chronic low-grade inflammation. These metabolic disturbances promote the accumulation of fat within hepatocytes and stimulate fibrogenic pathways, ultimately accelerating the progression toward cirrhosis. Several previous studies have also reported a high prevalence of diabetes among patients with cirrhosis, emphasizing its role as both a risk factor and a consequence of advanced liver disease.

Hypertension was the second most common comorbidity observed in the study, affecting approximately one-quarter of the participants. The coexistence of hypertension with chronic liver disease may reflect the broader spectrum of metabolic syndrome, which includes obesity, dyslipidemia, and insulin resistance. Dyslipidaemia and hypothyroidism were also observed among a smaller proportion of patients. These findings collectively suggest that metabolic dysfunction plays an important role in the pathogenesis and progression of liver disease in the studied population. The growing burden of metabolic comorbidities further underscores the importance of adopting a multidisciplinary approach in the management of patients with chronic liver disease, focusing not only on hepatic complications but also on associated systemic conditions.

The etiological distribution of liver disease in the present study provides additional insight into the evolving patterns of cirrhosis. Alcohol-related liver disease emerged as the most common etiology, accounting for approximately 40% of cases. Chronic alcohol consumption is well known to cause progressive hepatic injury through mechanisms involving oxidative stress, mitochondrial dysfunction, and inflammatory responses. Continued alcohol exposure results in steatosis, steatohepatitis, fibrosis, and ultimately cirrhosis. The high prevalence of alcohol-related liver disease observed in this study reflects the ongoing impact of alcohol consumption as a major risk factor for chronic liver disease in many parts of the world.

The second most common etiology identified in the study was non-alcoholic steatohepatitis (NASH), which accounted for around 20% of cases. The increasing contribution of NASH to the burden of cirrhosis mirrors global epidemiological trends associated with rising rates of obesity, diabetes, and metabolic syndrome. In many regions, NASH is rapidly emerging as a leading cause of chronic liver disease and is expected to surpass viral hepatitis as the predominant etiology in the coming decades. The findings of the present study are consistent with these global trends, indicating that metabolic liver disease is becoming an increasingly important contributor to cirrhosis.

Viral hepatitis accounted for a relatively smaller proportion of cases in this cohort, with hepatitis B and hepatitis C contributing to 8% and 4% of cases respectively. This relatively lower prevalence may reflect improvements in vaccination programs, antiviral therapies, and public health interventions aimed at reducing viral transmission. Nevertheless, viral hepatitis remains an important cause of chronic liver disease, particularly in certain regions where access to preventive measures and treatment remains limited.

A key objective of the present study was to assess the relationship between liver stiffness measurement obtained through transient elastography and the presence and severity of esophageal varices. The results demonstrated a strong and statistically significant association between increasing liver stiffness values and higher grades of varices detected on upper gastrointestinal endoscopy. Patients with severe varices exhibited substantially higher mean liver stiffness measurements compared with those without varices. This finding supports the concept that liver stiffness serves as a surrogate marker of hepatic fibrosis and portal hypertension.

As liver fibrosis progresses, the accumulation of extracellular matrix within the hepatic parenchyma increases intrahepatic vascular resistance. This increased resistance leads to elevated portal venous pressure, which in turn contributes to the formation of collateral circulation and the development of esophageal varices. Therefore, the observed correlation between liver stiffness and variceal severity is pathophysiologically plausible and aligns with current understanding of portal hypertension.

The results of the present study are consistent with findings reported by several previous investigators who have demonstrated the usefulness of transient elastography as a non-invasive method for assessing portal hypertension and predicting esophageal varices. Numerous studies have reported that liver stiffness measurement correlates with both the presence and the severity of varices, suggesting that elastography may serve as an effective screening

tool in patients with cirrhosis. By identifying individuals with high liver stiffness values, clinicians may be able to selectively refer patients for endoscopic evaluation, thereby reducing the burden of unnecessary procedures.

In addition to liver stiffness measurement, other clinical parameters such as spleen size and MELD score also showed a positive association with the severity of esophageal varices. Splenomegaly is a well-recognized consequence of portal hypertension and results from congestion of the splenic venous circulation. Enlargement of the spleen often leads to hypersplenism, which is characterized by sequestration and destruction of blood cells, particularly platelets. Consequently, thrombocytopenia is frequently observed in patients with advanced portal hypertension. The presence of splenomegaly and thrombocytopenia therefore serves as an indirect indicator of elevated portal pressure.

Similarly, the MELD score, which is widely used to assess the severity of liver disease and predict mortality in patients with cirrhosis, was found to correlate with the presence of higher-grade varices. Higher MELD scores reflect worsening hepatic function and are typically associated with advanced fibrosis, portal hypertension, and an increased risk of complications. The combined assessment of liver stiffness measurement, spleen size, and MELD score may therefore provide a comprehensive non-invasive approach for evaluating the risk of variceal development.

An interesting observation in the present study was the lack of significant correlation between controlled attenuation parameter (CAP) values and the severity of esophageal varices. CAP is primarily used to quantify hepatic steatosis and is particularly useful in the early stages of metabolic liver disease. However, as liver fibrosis progresses and cirrhosis develops, the degree of hepatic fat infiltration may decrease due to the replacement of hepatocytes with fibrotic tissue. This phenomenon, sometimes referred to as the “steatosis-fibrosis paradox,” may explain the inverse trend observed between CAP values and variceal severity in the study.

The findings of the present study have important clinical implications, particularly in settings where healthcare resources are limited. Routine endoscopic screening for all patients with cirrhosis may not be feasible in many healthcare systems due to constraints related to cost, infrastructure, and availability of trained personnel. In such scenarios, the use of non-invasive predictors such as transient elastography can play a valuable role in identifying patients who are most likely to benefit from endoscopic evaluation.

By integrating liver stiffness measurement with other clinical indicators such as spleen diameter and MELD score, clinicians can stratify patients according to their risk of developing esophageal varices. This risk-based approach allows healthcare providers to prioritize high-risk individuals for endoscopic screening and prophylactic treatment, thereby optimizing the use of available resources while minimizing unnecessary procedures.

Despite its strengths, the present study has certain limitations that should be considered when interpreting the findings. The relatively small sample size of 50 patients may limit the generalizability of the results. Additionally, the study was conducted at a single tertiary care center, which may not fully represent the broader population of patients with chronic liver disease. Variations in patient demographics, etiological factors, and healthcare access across different regions may influence the prevalence and severity of esophageal varices.

Future studies involving larger patient populations and multicenter collaboration would be valuable in validating the findings of the present study. Longitudinal studies may also provide additional insight into the predictive value of liver stiffness measurement in monitoring disease progression and assessing the risk of variceal bleeding over time.

In conclusion, the results of this study demonstrate that liver stiffness measurement obtained through transient elastography is a reliable non-invasive predictor of esophageal varices in patients with chronic liver disease. Higher liver stiffness values were strongly associated with increased severity of varices, highlighting the role of elastography in identifying patients at risk of portal hypertension-related complications. When combined with clinical scoring systems and radiological parameters such as spleen size and MELD score, transient elastography can serve as an effective screening tool for risk stratification. The implementation of this non-invasive approach in routine clinical practice may reduce the reliance on invasive procedures while ensuring timely identification and management of patients with high-risk varices.

REFERENCES

1. Baveno VII Faculty. (2022). Renewing consensus in portal hypertension. Journal of Hepatology, 76(4), 959-989.
2. Sarin, S. K., et al. (2020). Diagnosis and management of portal hypertension: APASL recommendations. Hepatology International, 14(1), 1-45.
3. Villanueva, C., et al. (2023). Non-invasive markers of portal hypertension: A comprehensive review. Lancet Gastroenterology & Hepatology, 8(2), 154-168.
4. Marasco, G., et al. (2024). Accuracy of Transient Elastography for the prediction of

esophageal varices: A meta-analysis. Journal of Clinical Medicine, 13(1), 204.
5. Abraldes, J. G., et al. (2021). Spleen stiffness vs liver stiffness for portal hypertension. Gastroenterology, 160(4), 1120-1131.
6. Thandassery, R. B., et al. (2022). Noninvasive markers of varices in MASLD. Clinical Gastroenterology and Hepatology, 20(5), 1102-1110.
7. Petrova, M., et al. (2021). Correlation of CAP with fibrosis stage in MASLD. World Journal of Hepatology, 13(12), 2068-2075.
8. Swaroop, S., et al. (2025). Regional trends in North Indian cirrhosis etiologies. Indian Journal of Gastroenterology, 44(1), 12-20.
9. Yang, L. B., et al. (2023). MELD-Na vs. FibroScan in predicting variceal bleeding. World Journal of Gastroenterology, 29(25), 4072-4085.
10. Garcia-Tsao, G., et al. (2024). Portal hypertension: Advances in non-invasive diagnosis. Hepatology, 79(3), 650-662.
11. Kumar, R., et al. (2020). FibroScan and its pitfalls in obesity. Journal of Clinical and Experimental Hepatology, 10(4), 345-352.
12. Lee, H. A., et al. (2021). Platelet-to-spleen ratio revisited. Digestive Diseases and Sciences, 66(8), 2820-2829.
13. Zheng, R., et al. (2022). Controlled Attenuation Parameter in advanced cirrhosis. Liver International, 42(6), 1321-1330.
14. Singh, S., et al. (2023). Comparative accuracy of MELD and Child-Pugh. BMC Gastroenterology, 23(1), 45.
15. Roulot, D., et al. (2021). Transient elastography in clinical practice. Clinics in Liver Disease, 25(2), 321-335.
16. Tsochatzis, E. A., et al. (2020). Non-invasive assessment of liver fibrosis. The Lancet, 396(10257), 1120-1132.
17. Dajti, E., et al. (2025). Spleen stiffness for the risk of variceal bleeding. Hepatology Communications, 9(1), e0567.
18. Mallet, V., et al. (2023). Long-term follow-up of MASLD using FibroScan. Journal of Hepatology Reports, 5(2), 100621.
19. Berzigotti, A., et al. (2024). Imaging in portal hypertension. European Journal of Radiology, 172, 111300.
20. Patel, K., et al. (2026). Future of non-invasive liver diagnostics. Nature Reviews Gastroenterology & Hepatology, 23(1), 5-18

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