



## Research Article

## Section: Radiodiagnosis

# Determination of Correlation Between Spleen Stiffness and Endoscopic Esophageal Variceal Grading in Patients with Portal Hypertension, in a Tertiary Care Center in Western India

Dr. Jumana S<sup>\*1</sup> & Dr. Sunita Navani<sup>2</sup>

<sup>1</sup> Junior Resident, Department of Radiodiagnosis, Wockhardt Hospital, Mumbai Central, Mumbai

<sup>2</sup> Head of Department of Radiodiagnosis, Wockhardt Hospital, Mumbai Central, Mumbai, Maharashtra, India

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### \*Corresponding author:

**Dr. Jumana S**

Junior Resident, Department of  
Radiodiagnosis, Wockhardt Hospital,  
Mumbai Central, Mumbai

## ABSTRACT

**Introduction:** Esophageal varices (EV) represent a critical cause of acute upper gastrointestinal bleeding (UGIB), particularly in patients with cirrhosis. The high morbidity and mortality associated with variceal bleeding necessitate accurate and timely diagnosis. Traditional diagnostic methods, such as endoscopy, are invasive. Non-invasive methods like spleen stiffness measurement (SSM) via elastography offer a promising alternative.

**Objective:** The study aims to evaluate the effectiveness of SSM as a non-invasive predictor of esophageal varices' presence and severity in patients with portal hypertension. Additionally, it seeks to correlate SSM with endoscopic variceal grading and compare liver stiffness measurement (LSM), spleen size, liver size, and blood parameters with variceal grading. **Methods:** A prospective cross-sectional observational study was conducted at a tertiary care center in Western India from January 2023 to March 2024. Sixty patients with clinically diagnosed cirrhosis and suspected portal hypertension were included. SSM and LSM were assessed using ultrasound elastography. Endoscopic variceal grading was performed to evaluate the correlation with stiffness measurements and other clinical parameters. **Results:** SSM significantly correlated with esophageal variceal grading (R-squared: 0.651,  $p < 0.001$ ). Higher SSM values were associated with more severe varices. Liver stiffness, spleen size, and platelet count also showed significant correlations with variceal severity, with SSM being the most reliable non-invasive marker. The analysis further demonstrated that elastography-based measurements could reduce the need for invasive procedures like endoscopy in stratifying variceal severity. **Conclusion:** SSM is a reliable non-invasive method for predicting the presence and severity of esophageal varices in patients with portal hypertension. Incorporating SSM into routine clinical practice could reduce the need for invasive diagnostic procedures, enhancing patient care and management. Future research should focus on validating these findings in larger cohorts and exploring the broader applications of SSM in managing portal hypertension complications.

## INTRODUCTION

Esophageal varices (EV) are a critical concern in the context of acute upper gastrointestinal bleeding (UGIB), being one of the most common and severe causes globally. Their prevalence varies worldwide, reflecting regional differences in underlying conditions such as liver diseases and infections like schistosomiasis[1]. EV are a major contributor to mortality from UGIB, with acute variceal bleeding (AVB) being a particularly dangerous complication, arising from clinically significant portal hypertension (CSPH). This

condition poses a substantial challenge not only in terms of patient health but also in its economic impact and burden on healthcare systems[2].

In the United States, EV are identified as the seventh most common cause of gastrointestinal bleeding. Approximately 10% of all UGIB cases in the U.S. are attributed to EV. This statistic underscores their significant role in the spectrum of GI emergencies. In developing countries, the prevalence of schistosomiasis is closely associated with the occurrence of EV, adding another layer of complexity to

the epidemiological landscape. For instance, in regions where schistosomiasis is endemic, up to 30% of patients with this infection may develop EV[3]. In contrast, in Western countries, cirrhosis is the leading cause of EV, with a staggering 85% of cirrhotic patients eventually developing these varices. The incidence of EV is directly related to the severity of liver disease, with an annual development rate of 8% in patients with compensated cirrhosis and even higher rates in those with decompensated cirrhosis[4].

Cirrhosis itself is a chronic liver condition characterized by the histological development of regenerative nodules surrounded by fibrous bands, which occurs in response to long-standing liver injury. This pathological state leads to portal hypertension and can culminate in end-stage liver disease. In India, for instance, liver cirrhosis was responsible for 20 deaths per 100,000 population in 2019, highlighting its significant health impact[5]. However, comprehensive data on portal hypertension and hepatic cirrhosis in India are limited, necessitating reliance on global data to understand these conditions better[6].

In the Western world, the etiology of portal hypertension is most commonly linked to cirrhosis due to alcoholic liver disease, non-alcoholic steatohepatitis (NASH), and hepatitis C infection. Alcohol abuse is a significant public health issue, with approximately 15 million people in the United States affected. Of these, nearly 88,000 deaths annually are attributed to alcohol-related causes, and 10%-15% of individuals with alcohol use disorder develop cirrhosis[7]. The liver, along with the spleen, plays a pivotal role in the splanchnic circulation, and various etiological agents like alcohol, drugs, and viruses can cause irreversible damage to hepatic cells. This damage results in fibrosis and cirrhosis, manifesting as clinically significant portal hypertension and its complications, including esophageal varices[8].

The diagnosis of esophageal varices is typically made through upper gastrointestinal endoscopy. Approximately 50% of patients with cirrhosis will develop esophageal varices, and of these, about 30% will experience variceal bleeding within the first year. Mortality rates for a first episode of variceal bleeding range from 15% to 20%[9]. However, recent advancements have introduced non-invasive methods to predict the presence and severity of portal hypertension and esophageal varices. One such method is transient elastography (TE), which measures liver stiffness by transmitting mechanical waves into the liver tissue and analyzing the resulting wave propagation and tissue deformation. This technique provides valuable information on the level of fibrosis and has become a safe, non-invasive, and easy-to-perform alternative to traditional invasive methods[10].

Portal hypertension, defined as increased resistance to blood flow within the hepatic sinusoids, can lead to significant morbidity and mortality among patients with chronic liver disease. This resistance often translates to the portal vein, ca-

-using increased pressure and backpressure changes throughout the hepatic and portal vein systems[11]. Measuring the hepatic vein pressure gradient remains the gold standard for assessing the degree of portal hypertension, though its invasive nature limits widespread use. Consequently, non-invasive methods such as liver and spleen stiffness measurements have gained traction[12].

Liver and spleen stiffness can be evaluated through advanced ultrasound imaging techniques, providing insights into the severity of underlying fibrosis. This non-invasive approach is especially beneficial in routine clinical practice for assessing portal hypertension among cirrhotic patients[13]. Studies have shown that spleen stiffness measurements (SSM) correlate with the presence and grading of esophageal varices, offering a promising tool for clinicians to predict and manage portal hypertension complications without resorting to invasive procedures. For example, spleen stiffness values greater than 46.5 kPa are significantly associated with the presence of high-risk esophageal varices[14].

A more detailed understanding of these non-invasive techniques is provided by studies that have demonstrated the practicality and efficacy of SSM in clinical settings[15]. For instance, a study involving 150 patients with cirrhosis showed that spleen stiffness measurements could predict the presence of EV with a high degree of accuracy. Patients with spleen stiffness values exceeding 50 kPa were found to have a high likelihood of grade III varices, which are more prone to bleeding. This correlation allows for better stratification of patients based on their risk and aids in timely interventions to prevent severe complications[16].

Moreover, the non-invasive nature of SSM and other ultrasound-based techniques makes them suitable for repeated assessments, which is crucial for monitoring disease progression and the effectiveness of therapeutic interventions[17]. Unlike invasive procedures that carry risks and discomfort, these methods can be safely performed multiple times, providing continuous data to guide clinical decisions[18].

Esophageal varices are a major cause of morbidity and mortality in patients with liver disease, particularly those with cirrhosis. While traditional methods like endoscopy and HVPG measurement remain essential, the advent of non-invasive techniques such as liver and spleen stiffness measurements offer significant advantages[19]. These methods not only reduce the need for invasive procedures but also provide reliable and repeatable data that can improve the management of portal hypertension and its complications. As research continues to validate and refine these techniques, they are likely to become integral to the standard care protocols for patients with chronic liver disease and esophageal varices[20].

The study aims to evaluate the effectiveness of Splenic Stiffness Measurement (SSM) as a non-invasive method for

predicting the presence and severity of esophageal varices in patients with portal hypertension. It seeks to determine the correlation between SSM and endoscopic grading of esophageal varices in these patients. Additionally, the study aims to assess the correlation between liver stiffness measurement (LSM), spleen size, liver size, and blood parameters with the grading of esophageal varices in patients with portal hypertension.

**MATERIALS AND METHODS**

This prospective cross-sectional observational study, conducted at a tertiary care center in Western India from Jan-

-uary 2023 to March 2024, aimed to determine the correlation between spleen stiffness and endoscopic esophageal variceal grading in patients with portal hypertension. Inclusion criteria were suspected hepatic cirrhosis, liver stiffness >1.83 m/s (ARFI), imaging and clinical features of cirrhosis, diagnosed hepatic cirrhosis, and age >18 years. Exclusion criteria included perihepatic/perisplenic fluid, severe obesity, insufficient intercostal space, splenic abnormalities unrelated to cirrhosis, and pregnancy.

**RESULTS**

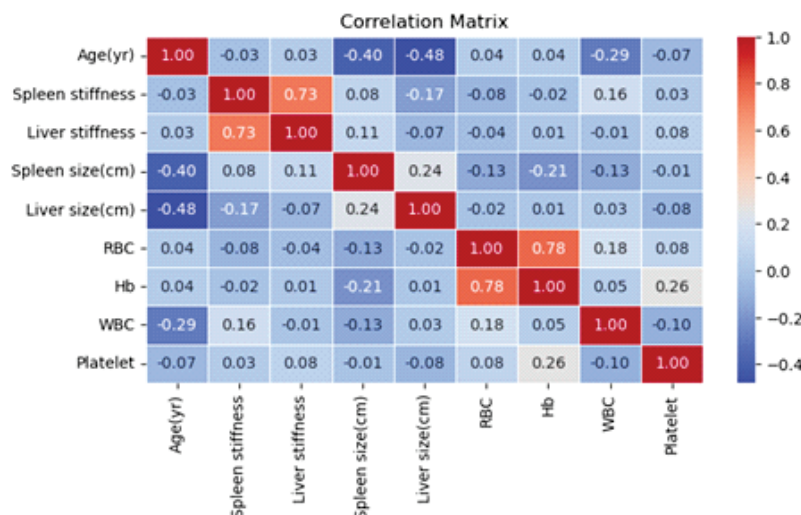
**Table 1: Central tendencies of age, SSM, LSM, spleen size, liver size and blood parameters**

	Age(yr)	Spleen stiffness(m/s)	Liver stiffness(m/s)	Spleen size( cm)	Liver size(cm)
count	60	60	60	60	60
mean	58.68333	3.7115	3.061333	14.60833	14.13167
std	13.55402	0.682073	0.582294	2.543472	2.976831
min	29	1.74	1.51	9.9	9.8
25%	50.5	3.32	2.68375	12.875	11.875
50%	59	3.775	3.0625	14.1	13.3
75%	69.25	4.1875	3.5175	16.425	16.425
max	84	4.93	3.95	22.1	21.4
	RBC( $10^6/\mu\text{l}$ )	Hb(g/dl)	WBC( $10^3/\mu\text{l}$ )	Platelet( $10^5/\mu\text{l}$ )	RBS(mg/dl)
count	60	60	60	60	60
mean	3.153	9.205	7326.333	1.113	115.5717
std	0.817475	2.012202	4074.314	0.76534	39.03573
min	0.93	3.7	1640	0.11	76
25%	2.7075	7.775	4882.5	0.545	89
50%	3.175	9.25	6500	1.02	103
75%	3.6925	10.4	8995	1.335	123.25
max	4.76	14.7	24170	3.58	262

The dataset includes medical measurements from 60 individuals, covering age, spleen and liver stiffness, organ sizes, and blood parameters. The average age is 58.68 years, with mean spleen and liver stiffness of 3.71 and 3.06, respectively. Spleen and liver sizes average 14.61 cm and 14.13 cm. Blood metrics show an average red blood cell count of 3.15, hemoglobin levels of 9.21 g/dL, white blood cell count of 7326.33 per cubic millimeter, and platelet count of 111,300 per cubic millimeter. These measurements provide insights into the individuals' health status and potential medical conditions.

The histogram shows age distribution in the dataset, with most individuals in the 50-60 age range and a slight right skew, indicating more older individuals. Males represent approximately 75% of the dataset, while females constitute 25%. The severity plot shows that males predominantly experience mild severity, followed by moderate and severe levels. In contrast, females have a more evenly spread severity distribution, with a notable presence of severe cases.

**Table 2: Correlation matrix of age, SSM, LSM, spleen size, liver size and blood parameters**



The correlation matrix highlights key associations among variables. Age shows weak to moderate correlations with several parameters, including a negative correlation with spleen and liver sizes. RBC and hemoglobin have a strong positive correlation, indicating a close relationship between these blood parameters. Spleen and liver stiffness display a moderate positive correlation, suggesting a link in liver disease progression. Additionally, platelet count positively correlates with hemoglobin, pointing to potential coagulation dynamics in liver disorders. These correlations emphasize the interplay between age, blood parameters, and organ characteristics in assessing hepatic health.

**Table 3: OLS Regression results for the correlation between spleen stiffness and EV grading**

OLS Regression Results						
Dep. Variable:	Spleen_stiffness		R-squared:	0.651		
Model:	OLS		Adj. R-squared:	0.588		
Method:	Least Squares		F-statistic:	10.34		
Date:	Thu, 04 Apr 2024		Prob (F-statistic):	8.31e-09		
Time:	01:45:59		Log-Likelihood:	-30.132		
No. Observations:	60		AIC:	80.26		
Df Residuals:	50		BIC:	101.2		
Df Model:	9					
Covariance Type:	nonrobust					
	<b>Coef</b>	<b>Std Err</b>	<b>T</b>	<b>P&gt; t </b>	<b>[0.025</b>	<b>0.975]</b>
<b>Intercept</b>	3.5552	0.068	52.162	0.000	3.418	3.692
<b>F0</b>	-0.5798	0.138	-4.216	0.000	-0.856	-0.304
<b>F10</b>	-0.3517	0.126	-2.793	0.007	-0.605	-0.099
<b>F11</b>	0.3448	0.402	0.858	0.395	-0.463	1.152
<b>F12</b>	0.2498	0.288	0.866	0.390	-0.329	0.829
<b>F20</b>	0.4135	0.156	2.655	0.011	0.101	0.726
<b>F21</b>	0.5673	0.209	2.708	0.009	0.146	0.988
<b>F22</b>	1.0081	0.239	4.224	0.000	0.529	1.487
<b>F30</b>	0.5298	0.209	2.529	0.015	0.109	0.951
<b>F31</b>	0.5031	0.176	2.866	0.006	0.151	0.856
<b>F32</b>	0.8705	0.165	5.291	0.000	0.540	1.201
Omnibus:	26.543		Durbin-Watson:	1.222		
Prob(Omnibus):	0.000		Jarque-Bera (JB):	46.680		
Skew:	-1.507		Prob(JB):	7.30e-11		
Kurtosis:	6.097		Cond. No.	1.33e+16		

The analysis reveals that spleen stiffness measurements (SSM) correlate significantly with esophageal variceal grading. The intercept (3.5552) represents estimated spleen stiffness without varices, red signs, or bleeding. Mild varices (F10) decrease spleen stiffness by 0.3517 units, while moderate (F20-F22) and severe varices (F30-F32) significantly increase it. The model explains 65.1% of spleen stiffness variability (R-squared = 0.651), adjusting to 58.8% for predictors (Adjusted R-squared = 0.588). The F-statistic (10.34, p = 8.31e-09) confirms the model's significance. Low p-values (<0.05) indicate significant coefficients, with spleen stiffness generally increasing as varices severity rises.

**Table 4: p values and key statistics of age, SSM, LSM, spleen size, liver size and Hb across severity levels of EV**

Severity	Mild	Moderate	No Varices	Severe	P-Value
N	17	15	11	17	
Percentage	28.33%	25.0%	18.33%	28.33%	
Age	63.0±14.72	58.27±12.16	56.18±18.91	56.35±8.82	0.434027
Spleen size	14.02±2.38	15.05±2.93	14.24±2.59	15.04±2.37	0.55657
Liver size	13.76±2.78	13.39±2.92	16.07±3.54	13.91±2.52	0.167922
Hemoglobin	9.15±2.06	9.61±1.87	9.41±0.53	8.77±2.66	0.68323
LSM	2.69±0.56	3.31±0.44	2.63±0.47	3.49±0.33	0.0000922
SSM	3.32±0.62	4.13±0.38	2.98±0.32	4.22±0.41	0.0000017

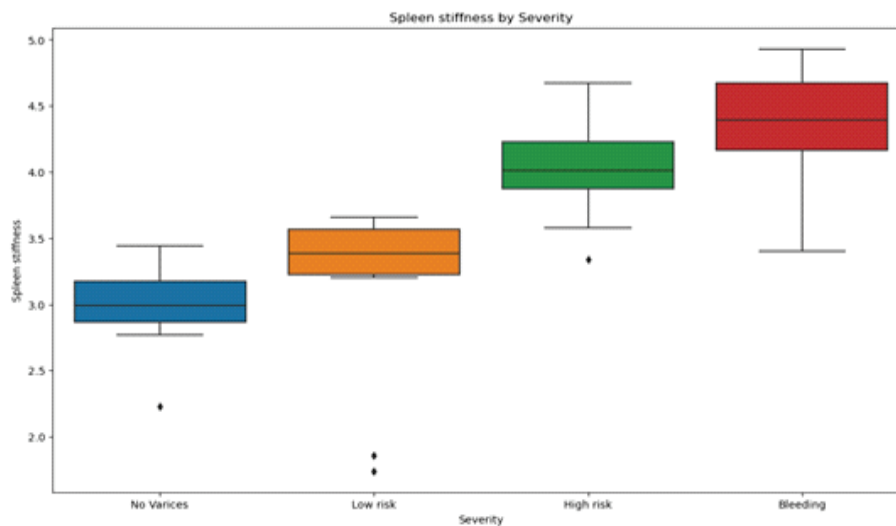
The table presents key statistics and p-values across severity levels—mild, moderate, no varices, and severe. While the distribution of cases is balanced among groups, some parameters hint at differences in disease progression. Age, platelet count, spleen size, liver size, and hemoglobin levels show no significant disparities among severity levels, indicating limited diagnostic value for these factors. However, liver and spleen elastography values vary substantially across severity levels, highlighting their potential as sensitive markers for assessing disease severity. This suggests that elastography is valuable in evaluating the progression of esophageal varices.

**Table 5. p values and key statistics of various parameters of dataset across severity levels of EV**

	Mild	Moderate	No Varices	Severe	P-Value
N	17	15	11	17	
Percentage	28.33%	25.0%	18.33%	28.33%	
Age	63.0±14.72	58.27±12.16	56.18±18.91	56.35±8.82	0.434027
Platelet Count	1.07±0.63	0.97±0.65	1.76±1.11	0.84±0.37	0.204170
Spleen Diameter	14.02±2.38	15.05±2.93	14.24±2.59	15.04±2.37	0.556574
Portal Vein Diameter	13.76±2.78	13.39±2.92	16.07±3.54	13.91±2.52	0.167923
Hemoglobin	9.15±2.06	9.61±1.87	9.41±0.53	8.77±2.66	0.683231
Liver Elastography	2.69±0.56	3.31±0.44	2.63±0.47	3.49±0.33	0.000009
Spleen Elastography	3.32±0.62	4.13±0.38	2.98±0.32	4.22±0.41	0.000000
RBS	122.76±42.32	106.75±19.54	119.36±43.41	113.71±46.64	0.728599
T.Bilirubin	5.63±6.9	6.4±9.62	6.99±8.22	3.89±4.88	0.689034
D.Bilirubin	3.8±5.64	4.76±8.61	5.55±7.42	2.13±3.21	0.526503
I.Bilirubin	1.83±1.85	1.63±1.22	1.44±0.91	1.76±1.93	0.938444
AST	79.38±76.57	81.15±50.02	97.91±53.18	76.35±50.8	0.549017
ALT	40.69±50.69	45.85±24.19	58.73±47.11	40.59±21.62	0.163169
ALP	142.22±82.89	137.49±97.2	149.09±129.2	107.35±43.27	0.580753
GGT	83.59±78.89	75.2±62.19	238.0±234.0	66.82±59.35	0.013301

INR	2.94±4.35	1.97±0.9	1.39±0.36	1.61±0.56	0.184687
S. Creatinine	1.84±1.29	1.32±0.65	1.23±0.79	1.15±0.74	0.400459
SBUN	25.35±20.27	21.38±11.96	14.0±6.13	18.35±6.38	0.365517
Na	136.84±5.63	133.33±5.05	138.0±3.61	135.12±7.62	0.140691
K	3.97±0.66	4.19±0.64	3.78±0.87	4.16±0.8	0.451252
Cl	103.53±5.15	102.73±6.27	103.91±4.32	106.59±9.55	0.383198
T.protein	7.06±0.81	6.27±1.02	7.02±1.2	6.62±0.85	0.187869
Albumin	2.91±0.59	2.73±0.39	3.16±0.6	2.86±0.44	0.187116
Globulin	4.13±0.66	3.54±0.9	3.87±0.91	3.76±0.74	0.239794
A_G_ratio	0.72±0.2	0.81±0.23	0.84±0.19	0.79±0.22	0.439319

There is no significant association between age, platelet count, spleen size, liver size, hemoglobin levels, and disease severity. However, liver and spleen stiffness, as well as GGT levels, show significant associations with severity, indicating their potential as important indicators of disease progression. Other variables, such as bilirubin levels, A/G ratio, and various electrolyte and protein levels, do not exhibit significant associations with severity. This highlights the relevance of elastography and GGT in assessing the severity of the condition.



Graph 1: Box and whisker plot: SSM across severity levels of EV

The "No Varices" group shows the lowest spleen stiffness values, indicating a less severe condition. The "Low Risk" group has slightly higher, but still relatively low, spleen stiffness values, suggesting mild spleen involvement. The "High Risk" group exhibits moderate to high spleen stiffness values, reflecting more significant spleen involvement compared to "Low Risk" and "No Varices." The "Bleeding" category shows the highest spleen stiffness values, indicating severe spleen impact and advanced disease stage. The visualization effectively illustrates the progression of spleen stiffness from "No Varices" to "Bleeding," aligning with clinical expectations of increasing severity

Table 6: Key statistics of SSM across severity levels of EV

Severity	count	mean	std	min	25%	50%	75%	max
No Varices	11.0	2.975455	0.3154484	2.23	2.8650	2.990	3.1750	3.4
Low risk	14.0	3.203571	0.6145126	1.74	3.2225	3.385	3.5675	3.6
High risk	23.0	4.036087	0.3169857	3.34	3.8750	4.010	4.2250	4.6
Bleeding	12.0	4.356667	0.4616243	3.40	4.1625	4.395	4.6725	4.9

This dataset provides descriptive statistics for four groups classified by the severity of a condition related to varices: No Varices, Low Risk, High Risk, and Bleeding. The table includes the count of observations, mean values, standard deviation (std), minimum (min), 25th percentile (25%), median (50%), 75th percentile (75%), and maximum (max) values for each group. The data indicates increasing mean values and higher standard deviations as the severity progresses from No Varices to Bleeding, with "Bleeding" showing the highest mean and variability, reflecting more severe cases with greater outcome variance.

Table 7: Correlation matrix of all parameters in dataset

	Age(yr)	Spleen stiffness	Liver stiffness	Spleen size(cm)	Liver size(cm)	RBC	Hb	WBC	Platelet	RBS	T. Bilirubin	D. Bilirubin	I. Bilirubin	AST	ALT	ALP	GGT	INR	S. Creatinine	S.BUN	Na+	K+	Cl-	T. protein	Albumin	Globulin	AVG ratio	
Age(yr)	1.00	0.03	0.03	-0.40	-0.48	0.04	0.04	-0.29	0.07	0.12	-0.29	-0.27	-0.28	0.08	0.03	0.03	0.05	0.13	0.21	0.32	0.22	0.21	0.07	0.27	0.15	0.22	0.07	
Spleen stiffness	0.03	1.00	0.73	0.08	-0.17	-0.08	0.02	0.16	0.03	0.37	-0.03	-0.06	0.13	-0.04	-0.03	-0.04	0.27	0.04	-0.02	0.16	-0.17	0.10	0.11	0.38	-0.39	-0.21	-0.02	
Liver stiffness	0.03	0.73	1.00	0.11	-0.07	-0.04	0.01	0.06	-0.25	-0.01	-0.03	0.09	-0.11	0.08	-0.14	0.30	0.04	-0.04	-0.09	0.16	-0.17	0.14	0.04	0.07	-0.30	-0.26	-0.03	
Spleen size(cm)	-0.40	0.08	0.11	1.00	0.24	-0.13	0.21	-0.13	0.01	0.05	0.14	-0.18	-0.15	0.11	-0.03	0.06	-0.11	0.18	0.15	0.03	0.06	-0.01	-0.07	0.04	-0.01	0.08	0.17	
Liver size(cm)	-0.48	-0.17	-0.07	0.24	1.00	0.02	0.01	0.03	0.06	0.00	0.23	0.14	0.19	0.35	0.09	-0.06	-0.23	0.10	-0.13	0.04	-0.13	0.04	0.16	0.09	0.13	-0.12	-0.02	
RBC	0.04	-0.08	-0.04	-0.13	-0.02	1.00	0.78	1.00	0.05	0.26	0.18	0.08	0.15	-0.20	-0.15	-0.38	0.02	0.16	0.11	0.10	-0.33	-0.29	-0.11	-0.17	-0.03	0.00	0.16	-0.09
Hb	0.04	-0.02	0.01	-0.21	0.01	0.78	1.00	0.05	0.26	0.06	0.03	0.06	-0.12	0.17	0.14	0.16	0.07	-0.25	-0.21	-0.12	-0.24	-0.00	-0.12	0.07	0.04	0.07	-0.03	
WBC	-0.29	0.16	-0.01	-0.13	0.03	0.18	0.05	1.00	-0.10	-0.15	0.19	0.18	0.19	0.14	0.08	-0.14	0.13	0.05	0.09	0.12	-0.05	0.33	0.13	0.14	0.01	0.19	-0.12	
Platelet	-0.07	0.03	0.08	-0.01	-0.08	0.08	0.26	-0.10	1.00	0.05	-0.09	-0.08	-0.09	-0.02	0.11	0.12	0.03	-0.02	-0.09	-0.02	-0.19	-0.03	-0.20	0.16	0.05	-0.16	0.09	
RBS	0.12	0.37	0.25	0.05	0.00	0.15	0.06	-0.15	0.05	1.00	-0.20	-0.17	-0.25	-0.22	-0.14	-0.06	-0.14	-0.08	-0.13	-0.11	0.12	0.14	0.11	0.21	0.30	0.07	0.13	
T. Bilirubin	-0.29	0.03	-0.01	-0.17	0.24	-0.29	0.03	0.19	-0.09	-0.20	1.00	0.99	0.72	0.59	0.94	0.18	0.06	0.26	0.11	-0.01	-0.18	-0.18	-0.15	0.35	-0.14	0.51	-0.44	
D. Bilirubin	-0.27	-0.06	-0.03	-0.17	0.28	-0.15	0.06	0.18	-0.08	-0.17	0.99	1.00	0.59	0.61	0.97	0.24	0.11	0.17	0.06	-0.07	-0.13	-0.21	-0.10	0.35	-0.14	0.51	-0.44	
I. Bilirubin	-0.28	0.13	0.09	-0.10	-0.00	-0.36	-0.12	0.19	-0.09	-0.25	0.72	0.59	1.00	0.31	0.11	-0.12	-0.14	0.53	0.27	0.23	-0.32	0.01	-0.32	0.21	-0.13	0.34	-0.31	
AST	-0.08	0.04	-0.11	-0.17	0.23	0.02	0.17	0.14	-0.02	0.22	0.59	0.61	0.31	1.00	0.79	0.30	0.26	0.04	0.06	-0.06	-0.14	-0.20	0.08	0.16	-0.26	0.37	-0.38	
ALT	0.03	-0.03	-0.08	-0.18	0.14	0.16	0.14	0.08	0.11	0.14	0.79	0.79	0.79	1.00	0.42	0.39	0.26	0.01	-0.02	-0.09	-0.19	-0.17	-0.12	-0.02	0.17	0.09	-0.11	
ALP	-0.03	0.01	-0.14	-0.15	0.19	0.11	0.16	-0.14	0.12	-0.06	0.18	0.24	-0.12	0.30	0.42	1.00	0.60	-0.09	-0.12	-0.14	-0.18	-0.05	-0.08	0.10	0.20	0.13	-0.15	
GGT	-0.05	0.27	0.30	-0.11	0.35	0.10	0.07	-0.13	0.03	-0.14	0.06	0.11	-0.14	0.26	0.99	0.60	1.00	0.14	-0.17	-0.22	0.03	-0.18	-0.02	0.10	0.20	0.03	0.10	
INR	0.13	0.04	-0.04	-0.03	0.09	-0.33	-0.25	0.05	-0.02	0.08	0.26	0.17	0.53	0.04	-0.01	-0.09	-0.14	1.00	0.54	0.41	-0.24	0.18	0.28	0.06	-0.03	0.06	-0.09	
S. Creatinine	0.21	-0.02	-0.09	0.06	-0.06	-0.29	-0.21	0.09	-0.09	-0.13	0.11	0.06	0.27	0.06	-0.02	-0.12	-0.17	0.54	1.00	0.57	-0.06	-0.02	-0.14	0.14	0.31	0.02	-0.25	
S.BUN	0.32	0.16	0.16	0.01	-0.23	-0.11	-0.12	0.12	-0.02	-0.11	0.01	-0.07	0.23	-0.06	-0.09	-0.14	0.22	0.41	0.57	1.00	-0.06	0.18	-0.15	0.25	0.23	-0.15	-0.05	
Na+	0.22	-0.19	-0.17	-0.07	0.10	-0.17	-0.24	-0.05	-0.19	0.12	-0.18	-0.13	-0.32	-0.14	-0.19	-0.18	0.03	-0.24	-0.06	-0.06	1.00	-0.29	0.69	0.06	0.10	-0.13	0.19	
K+	-0.21	0.11	0.04	-0.04	-0.13	0.17	-0.00	0.33	-0.03	0.14	-0.18	-0.21	0.01	-0.20	-0.17	-0.05	-0.18	0.18	-0.02	0.18	-0.29	1.00	-0.02	0.05	0.17	-0.05	0.11	
Cl-	0.07	0.11	0.07	-0.01	0.04	-0.03	-0.12	0.13	-0.20	0.11	-0.15	-0.10	-0.32	0.08	-0.12	-0.09	-0.02	-0.28	-0.14	-0.15	0.09	-0.02	1.00	0.06	-0.01	-0.06	0.09	
T. protein	-0.27	0.30	-0.30	-0.08	0.16	0.00	0.07	0.14	-0.16	0.21	0.15	0.10	-0.32	0.08	-0.12	-0.09	-0.02	0.28	-0.14	-0.15	0.09	-0.02	1.00	0.56	0.05	-0.33		
Albumin	-0.15	0.30	-0.26	-0.17	0.09	0.16	0.04	-0.01	-0.05	0.30	-0.14	-0.14	-0.13	-0.28	-0.17	-0.02	0.20	0.16	-0.02	-0.14	-0.25	-0.06	0.05	1.00	0.04	0.56		
Globulin	-0.22	-0.21	-0.20	-0.01	0.13	-0.09	0.07	0.19	-0.16	0.07	0.51	0.51	0.34	0.37	0.09	0.13	-0.00	0.08	0.02	-0.15	-0.13	-0.05	0.04	1.00	0.04	0.56		
AVG ratio	0.07	-0.02	0.03	-0.12	-0.02	0.16	-0.03	-0.12	0.09	0.13	-0.44	-0.44	-0.31	-0.30	-0.11	-0.15	0.10	-0.09	-0.25	-0.05	0.19	0.09	0.33	0.75	1.00	0.75		

The correlation matrix reveals relationships between variables, with coefficients ranging from -1 to 1. Age shows weak negative correlations with spleen size (-0.40) and liver size (-0.48), indicating slight decreases in organ size with age. Spleen and liver stiffness are strongly correlated (0.73), suggesting they increase together. RBC and Hb exhibit a strong positive correlation (0.78), reflecting their close connection. T. Bilirubin and D. Bilirubin are nearly collinear (0.99). AST and ALT also correlate strongly (0.75), showing similar variation patterns. T. Protein and Albumin have a positive correlation (0.56), linking overall protein and albumin levels.

**DISCUSSION**

Portal hypertension, a complication of chronic liver disease, often results in esophageal varices, posing a risk of life-threatening bleeding. Accurate variceal assessment is crucial. Spleen stiffness, measured via elastography, is emerging as a non-invasive marker of portal hypertension severity. This study in Western India aims to correlate spleen stiffness with variceal grading, potentially reducing invasive endoscopy and enhancing patient management. Numerous studies support this research's significance[21]. Khanna R. et al. (2018) studied Idiopathic Portal Hypertension (IPH) and Extrahepatic Portal Vein Obstruction (EHPVO), both marked

by significant splenomegaly and normal hepatic venous pressure gradients. IPH is associated with infections, autoimmunity, and prothrombotic states, while EHPVO is linked to prothrombotic disorders and local factors. Our study highlights spleen stiffness as a non-invasive indicator of variceal severity in portal hypertension patients, potentially reducing the need for invasive procedures like endoscopy and improving patient care and risk stratification[22]. Kausar S. et al. (2021) found a strong negative correlation between platelet count and variceal size in cirrhosis patients, with lower counts predicting larger varices. Our study, inste-

ad, shows that spleen stiffness correlates with variceal severity, offering a reliable, non-invasive alternative to endoscopy for assessing variceal severity, enhancing patient care[23]. Our study examines demographic characteristics within a dataset, focusing on age and gender distributions. The age distribution peaks in middle age, with a slight skew towards older participants, indicating a predominantly adult population. Gender distribution shows a male predominance, with 75% of participants being male. These insights are crucial for interpreting the study's findings within this demographic context[24].

El-Toukhy N. et al. (2020) explored non-invasive prediction of esophageal varices in cirrhotic patients using transient elastography (Fibroscan®) to measure spleen stiffness. The study found significant correlations between spleen stiffness, liver stiffness, and variceal presence. Patients with varices had larger spleen sizes, greater portal vein diameters, more ascites, and higher liver stiffness. Our study supports these findings by analyzing demographic characteristics and correlations between age, blood parameters, and organ sizes, emphasizing the potential of non-invasive tools like elastography for assessing portal hypertension and predicting variceal presence in clinical practice[25].

Monga A et al. examined clinical and biochemical parameters linked to esophageal varices in cirrhotic patients, finding a male predominance and correlations between variceal presence, Child-Pugh class, platelet count, and spleen size. Our study focuses on the relationship between spleen stiffness and variceal severity, demonstrating its potential as a non-invasive predictor, supporting the use of transient elastography in clinical practice[26]. Roy A et al. (2024) investigated magnetic resonance elastography (MRE) for assessing varices in obese patients with NAFLD-CC. They found that MRE liver stiffness measurements (LSM) were significantly higher in patients with varices, unlike ARFI LSM. Platelet count and MRE-LSM predicted high-risk varices. Similarly, our study confirmed liver and spleen stiffness as valuable markers for assessing disease severity and guiding clinical management[27].

Uong P et al. (2023) investigated esophageal varices (EVs) prevalence in 303 cirrhotic patients undergoing variceal screening. They found that 66% had EVs, with grade 2 varices without red signs and grade 1 varices most common. Males had a higher EV prevalence, though not statistically significant. Additionally, 24.1% had large EVs, underscoring the importance of screening for variceal bleeding risk[28].

Our study found significant correlations in cirrhotic patients, particularly between spleen and liver stiffness, indicating a connection between portal hypertension and liver fibrosis. Red blood cell count also correlated positively with other variables. These insights highlight the importance of variceal screening and risk stratification in improving cirrhosis management and outcomes[29]. Zoughlami A et al. (2023) assessed non-invasive variceal screening in virus-related ch-

-ronic advanced liver disease (cACLD). They found that liver stiffness measurements (LSM) and biomarkers like FIB-4 and APRI reduce unnecessary esophagogastroduodenoscopies (EGD) while maintaining high predictive value. The study highlights the role of non-invasive tools in improving patient care and resource allocation, especially in low-resource settings[30].

## CONCLUSION

Esophageal varices are a major cause of morbidity and mortality in cirrhotic patients, traditionally diagnosed through upper gastrointestinal endoscopy. Recently, spleen stiffness measurement by sonoelastography has emerged as a non-invasive method for predicting portal hypertension and its complications. This study, conducted at a tertiary care center in Western India, found a significant correlation between spleen stiffness and the severity of esophageal varices. Higher spleen stiffness values were associated with more severe varices, suggesting that spleen stiffness could serve as a reliable non-invasive marker. Incorporating spleen stiffness measurements into routine practice could reduce the need for invasive procedures, improve patient comfort, and enhance clinical management of portal hypertension. Further research is needed to validate these findings and explore the broader application of spleen stiffness in predicting complications of portal hypertension.

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