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Non-Invasive Parameters of Esophageal Varices in Patients with Liver Cirrhosis in Mmimsr, Mullana, Ambala

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Abstract: ***Background***: Esophageal varices (EV) are a major complication of liver cirrhosis due to portal hypertension. While endoscopy remains the gold standard for detection, it is invasive and costly. Identifying reliable non-invasive parameters is crucial for risk stratification, early intervention, and reducing unnecessary endoscopic procedures in resource-limited settings. ***Aim and Objective***: To identify and evaluate non-invasive clinical, biochemical, and radiological parameters that predict the risk and grade of EV in cirrhotic patients admitted at MMIMSR, Mullana, Ambala. ***Materials and Methods***: This observational cross-sectional study was conducted in the Department of Medicine over 18 months (2023–2025). Fifty cirrhotic patients aged >18 years, diagnosed via clinical assessment, ultrasonography, and lab tests, were enrolled from OPD, wards, and ICU. ***Results***: Majority of patients (41/50) were males. Alcohol was the leading etiology (29 cases), followed by Hepatitis B, C, NASH, and alcohol with Hep B. Mean values included hemoglobin: 9.18 g/dL, platelet count: 113,730/mm^3, PT-INR: 2.04, spleen diameter: 131.3 mm, portal vein diameter: 11.22 mm. Higher variceal grades were associated with alcohol-related cirrhosis, low platelet count, elevated PT-INR, increased liver span, spleen and portal vein diameters, and presence of ascites. The platelet count-to-spleen diameter ratio showed strong inverse correlation with variceal grade. ***Conclusion***: Simple non-invasive parameters such as blood counts and abdominal ultrasonography are effective tools for early EV prediction. These markers are especially valuable in settings with limited endoscopic access, aiding timely initiation of prophylaxis. Larger studies are needed to validate these findings.

Keywords: Esophageal Varices, Liver Cirrhosis, Portal Hypertension, Non-Invasive Predictors, Platelet Count, Spleen Diameter, Platelet-to-Spleen Ratio.

INTRODUCTION

The liver is a vital organ with regenerative ability and diverse physiological functions. It receives nutrient-rich blood via the portal vein and functions as a metabolic gatekeeper, detoxifying xenobiotics and regulating glucose, ammonia, and nutrient metabolism [1]. Given its central role, liver dysfunction manifests as cirrhosis or hepatocellular carcinoma (HCC). Chronic liver disease affects 1.5 billion globally, with cirrhosis causing 1.16 million deaths annually, including 18.3% of Indian cases [1,2]. A shift in etiology from alcohol and infections to metabolic causes like MAFLD is driven by lifestyle changes [3].

Cirrhosis, the end stage of chronic liver injury, is characterized by fibrotic septa, regenerative nodules, and neovascularization. This disrupts hepatic architecture and blood flow, leading to portal hypertension and end-stage liver disease (ESLD) [3]. Hemodynamic changes splanchnic vasodilation, renal vasoconstriction, and fluid retention exacerbate complications such as ascites, encephalopathy, and variceal bleeding. Hyperdynamic circulation with elevated cardiac output and reduced vascular resistance is driven by nitric oxide and other vasodilators [4].

Esophageal varices (EV), a key outcome of portal hypertension, affect up to 90% of cirrhotics during

their lifetime. At diagnosis, 50% have varices, with 5–15% progressing annually; one-third suffer life-threatening bleeding [2-3]. Mortality within 6 weeks of variceal bleed ranges from 11.1% to 40%, with red wale signs, varix size, and hepatic function being key predictors [3]. β-blockers can reduce first bleeding risk by 50% [4,5].

While esophagogastroduodenoscopy (EGD) is the gold standard, Baveno VI recommends omitting it if liver stiffness <20 kPa and platelets >150,000/μL [6]. Non-invasive predictors (platelet count, PC/SD ratio, Child-Pugh class, FibroScan) may reduce unnecessary EGDs [2-4]. This study was conducted to assess the efficacy of such non-invasive parameters for EV detection in cirrhosis at MMIMSR, Mullana, Ambala.

MATERIALS AND METHODS

This observational cross-sectional study will be conducted in the Department of Medicine, MMIMSR, Mullana, Ambala, over 18 months (2023–2025). Fifty adult patients (>18 years) diagnosed with liver cirrhosis based on clinical evaluation, ultrasonography, and relevant laboratory parameters will be enrolled from OPD, medicine wards, and ICU. Ethical clearance will be obtained before study initiation.

Patients presenting with newly diagnosed cirrhosis will be evaluated using a structured proforma documenting demographics, clinical history (including alcohol use), presence of ascites, jaundice, splenomegaly, and hepatic encephalopathy. Biochemical investigations will include liver function tests, platelet count, PT, INR, serum albumin, total protein, and transaminases.

Radiological assessment by abdominal ultrasound will record liver size and echotexture, spleen size, portal vein diameter (PVD), splenic diameter (SD), and presence of ascites. Platelet count to spleen diameter ratio (PLT/SD) and PVD will be calculated. Upper GI endoscopy (UGIE) will grade esophageal varices using the Japanese three-grade classification. Exclusion criteria include history of variceal bleeding, prior banding/sclerotherapy, portal vein thrombosis, hepatoma, or beta-blocker therapy. Data will be analyzed using SPSS v26 with appropriate statistical tests; $p < 0.05$ will be considered significant.

RESULTS

TABLE 1: DEMOGRAPHIC DATA AND OTHER FACTORS DISTRIBUTION OF PATIENTS

		N	%
Age group (years)	≤ 40	5	10%
	41 to 50	21	42%
	51 to 60	14	28%
	61 to 70	7	14%
	>70	3	6%
	Total	50	100%
	Mean	51.52 ± 10.52	
Gender	Males	41	82%
	Females	9	18%
	Total	50	100%
Etiology	Alcohol	29	58%
	NASH	7	14%
	Hepatitis B	4	8%
	Hepatitis C	5	10%
	Alcohol + Hepatitis B	5	10%
	Total	50	100%
Ascites	Mild	20	40%
	Moderate	11	22%
	Massive	9	18%
	Absent	10	20%
	Total	50	100%
Hepatic encephalopathy	Yes	7	14%
	No	43	86%
	Total	50	100%
Modified Child Turcotte Pugh score	Class A	14	28%
	Class B	13	26%
	Class C	23	46%
	Total	50	100%

There were 5 patients who are less than 40 years. 21 patients, 14 patients, 7 patients, 3 patients belonged to 41-50 years, 51 to 60 years, 61 to 70 years &> 70 years of age respectively. There were total 50 patients out of which 41 were males and 9 were females. Alcohol was the leading cause in 29 patients subsequently NASH, Hep B, Hep C, alcohol with Hep B in 7 patients, 4 patients, 5 patients, 5 patients respectively. Mild, moderate & severe ascites was seen in 20 (40%), 11 (22%) and 9 (18%) patients respectively. Ascites was absent 10 (20%) patients. Out of 50 patients, hepatic encephalopathy was present in 7 patients and absent in 43 patients. Out of total 50 patients, 14 patients were in CTP-A, 13 patients and 23 patients were in CTP-B and CTP-C respectively.

TABLE 2: HEMATOBIOCHEMICAL PROFILE

Variable	Mean ± SD
Hb	9.18± 1.94
Platelet	113730± 66313.35
Total bilirubin	4.53 ± 4.88
SGOT	104.69±142.98
SGPT	85.85±126.86
Albumin	2.93± 0.73
Proteins	6.11±1.21
PT-INR	2.04±1.22

The mean haemoglobin, platelet count, total bilirubin, SGOT, SGPT, Albumin, serum protein and PT-INR was 9.18±1.94, 113730±66313.35, 4.53±4.88, 104.69±142.98, 85.85±126.86, 2.93± 0.73, 6.11±1.2, 2.04±1.22 respectively.

TABLE 3: ULTRASOUND FINDINGS

Variable	Mean ± SD
Liver span	14.673 ± 1.97
Spleen diameter	131.323 ± 23.87
Portal vein diameter	11.223 ± 2.05

Mean liver span, spleen diameter and portal vein diameter was 14.67±1.97, 131.32±23.87, 11.22±2.05 respectively.

TABLE 4: PLATELET COUNT: SPLEEN DIAMETER RATIO

Platelet count: Spleen diameter ratio	Ratio
Mean ± SD	925.17 ± 620.90

The mean platelet count to spleen diameter ratio was found to be 925.17±620.90.

TABLE 5: DISTRIBUTION OF PATIENTS ACCORDING TO UGI ENDOSCOPY FINDINGS

UGI Endoscopy findings	Number of Patients
Grade 0 (No varices)	9 (18%)
Grade I	13 (26%)
Grade II	17 (34%)
Grade III	11 (22%)
Total	50 (100%)

Grade 0(no varices) was seen in 9 (18%) patients. Grade I, Grade II and Grade III varices was seen in 13 (26%), 17 (34%) and 11 (22%) cases respectively

TABLE 6: CORRELATION OF ETIOLOGIC PROFILE WITH GRADE OF EV ON UGIE

	Low risk- Varices Grade 0 - I		High Risk- Varices Grade II - III	
	Number	%age	Number	%age
Alcohol	13	59.09%	16	57.14%
NASH	5	22.73%	2	7.14%
Hepatitis B	1	4.55%	3	10.71%
Hepatitis C	1	4.55%	4	14.29%
Alcohol + Hepatitis B	2	9.09%	3	10.71%

Total	22	100%	28	100%
p-value	0.415			

Out of 50 patients 13 and 16 patients were alcoholic, 5 and 2 patients had NASH, 1 and 3 patients had Hep B, 1 and 4 patients had Hep C, 2 and 3 were alcoholic with hep B in low-risk varices group and greater likelihood varices group respectively.

Table 7: CORRELATION OF HEMATOLOGICAL PROFILE WITH GRADE OF EV ON UGIE

Hematological profile	Low risk- Varices Grade 0 - I		High Risk- Varices Grade II - III		p-value
	Mean	SD	Mean	SD	
Hb	9.53	2.3	8.91	1.6	0.267
Platelet	145068.8	73681.5	89107.1	48261.8	0.002 (Significant)

The mean haemoglobin in low-risk varices group was 9.53±2.3 and high-risk varices group was 8.91±1.6. (p=0.267). The mean Platelet count in low-risk varices group was 145068.8±73681.5 and high-risk varices group was 89107.1±48261.8. (p=0.002).

TABLE 8: CORRELATION OF LIVER FUNCTION TEST WITH GRADE OF EVON UGIE

Hepatic profile	Low risk- Varices Grade 0 - I		High Risk- Varices Grade II – III		p-value
	Mean	SD	Mean	SD	
Total bilirubin (mg/dl)	3.44	3.97	5.38	5.39	0.164
SGOT (U/L)	87.63	81.9	118.1	177.38	0.460
SGPT (U/L)	44.65	21.9	118.2	162.44	0.040 (Significant)

The mean total serum bilirubin in low& high-risk varices group was 3.44±3.97 and 5.38±5.39, respectively. (p=0.164), the mean SGOT in lower-risk varices group & higher-riskvarices group was 87.63±81.9 & 118.1±177.38, respectively. (p=0.460), the mean SGPT in the low-risk varices group and high-riskvarices group was 44.65±21.9 and 118.2±162.44, respectively. (p=0.040)

TABLE 9: CORRELATION OF BIOCHEMICAL PARAMRTERS WITH GRADE OF EV ON UGIE

Hematological profile	Low risk- Varices Grade 0 - I		High Risk- Varices Grade II - III		p-value
	Mean	SD	Mean	SD	
Albumin (mg/dl)	3.19	0.86	2.71	0.54	0.019 (Significant)
Protein (mg/dl)	6.43	0.99	5.86	1.33	0.100
PT-INR	1.57	0.49	2.39	1.49	0.017 (Significant)

The mean serum albumin in low& high-risk varices group was found to be 3.19±0.86 and 2.71±0.54, respectively. (p=0.019) and mean serum protein in low& high-risk varices group was found to be 6.43±0.99 and 5.86±1.33, respectively. (p=0.100)

The mean PT-INR in low & high-risk varices group was 1.57±0.49 and 2.39±1.49, respectively. (p=0.017)

TABLE 10: CORRELATION OF USG VARIABLES WITH GRADE OF EV ON UGIE

USG variables	Low risk- Varices Grade 0 - I		High Risk- Varices Grade II - III		p-value
	Mean	SD	Mean	SD	
Liver span (cm)	13.97	1.93	15.21	1.86	0.025 (Significant)
Spleen diameter (mm)	119.09	18.19	140.93	23.64	0.008 (Significant)

Portal vein diameter (mm)	10.45	1.50	11.83	2.24	0.016 (Significant)

The mean liver span in low & high-risk varices group was found to be 13.97±1.93 and 15.21±1.86, respectively. (p=0.025). The mean spleen diameter in the low-risk varices and high-riskvarices group was 119.09±18.19 and 140.93±23.64, respectively. (p=0.008) and the mean PVD in low& high-risk varices group was 10.45±1.50 and 11.83±2.24, respectively. (p=0.016)

TABLE 11: CORRELATION OF ASCITES AND HEPATIC ENCEPHALOPATHY WITH GRADE OF EV ON UGIE

Variable	Low risk- Varices Grade 0 - I		High Risk- Varices Grade II - III		p-value
	Number	%age	Number	%age	
Ascites	13	59.09%	27	96.43%	0.0010 (Significant)
Hepatic encephalopathy	2	9.09%	5	17.86%	0.8495

Ascites was present in13 patients and 27 patients, and hepatic encephalopathy was present in 2 and 5 patients in low& high-risk varices group respectively.

TABLE 12: CORRELATION OF MODIFIED CHILD TURCOTTE PUGH SCORE WITH GRADE OF EV ON UGIE

Modified Child Turcotte Pugh score	Low risk- Varices Grade 0 - I		High Risk- Varices Grade II - III		Total	
	Number	%age	Number	%age	Number	%age
Class A	12	54.55%	2	7.14%	14	28%
Class B	5	22.73%	8	28.57%	13	26%
Class C	5	22.73%	15	53.57%	23	46%
Total	22	100	28	100	50	100
p-value	0.0011 (Significant)					

Out of 14 patient in CTP-A , 12 patients belongs to low-risk varices group and 2 patients belong to high-risk varices group. Out of 13 patients in CTP-B , 5 patients belongs to low-riskvarices group and 13 patients belong to high-risk varices group. Out of 23 patients in CTP-C, 5 patients belongs to low-risk varices group and 15 patients belong to high-risk varices group. (p-value 0.0011)

Table 13: Correlation of Platelet count: Spleen diameter ratio with grade of esophageal varices on upper gastro-intestinal endoscopy

Platelet count: Spleen diameter ratio	Low risk- Varices Grade 0 - I	High Risk- Varices Grade II - III	p-value
Mean ±SD	1277.34±690.11	648.46±385.71	0.0001 (Significant)

The PC/SD ratioamong patients of low-risk varices group and high-risk varices group was 1277.34±648.46 and 690.11±385.71, respectively. (p=0.0001)

DISCUSSION

Esophageal variceal bleeding is a life-threatening complication of cirrhosis, with a high mortality rate. Varices occur in 60–80% of cirrhotic patients, with a 25–35% bleeding risk. New varices develop annually in ~5%, and small varices progress at 5–10% per year. As varices enlarge, increased wall tension heightens rupture risk. Despite treatment, first-bleed mortality is ~20%, with 30–40% rebleeding within 72 hours and up to 60% in the first week. Thus, primary and secondary prophylaxis remains essential in managing decompensated chronic liver disease.

The AASLD & the Baveno IV Consensus Conference on PH recommend that all patients diagnosed with hepatic cirrhosis undergo screening for EVs at the

time of detection. [6] Routine esophagogastroduodenoscopy (EGD) for all cirrhotic patients is often not economically feasible. A more cost-effective approach involves risk stratification using accessible clinical parameters. Multiple studies have shown that non-invasive predictors such as biochemical markers, clinical findings, and ultrasonographic measurements can reliably identify patients at risk for esophageal varices, reducing reliance on invasive endoscopy for initial evaluation. Hence; the present study was undertaken to identify and study non-invasive investigations (clinical, biochemical, radiological) that could predict the presence and the risk of esophageal varices in the patients with liver cirrhosis admitted in MMIMSR, Mullana, Ambala.

DEMOGRAPHIC DATA AND OTHER FACTORS

There were 5 (10%) patients who aged 40 years or less than 40 years. 21 (42%) patients belonged to the age group of 41-50 years. 14 (28%) subjects belonged to the age group of 51-60 years. There were 7 (14%) subjects that aged between 61-70 years. 3 (6%) subjects aged over 70 years. Mean age of the patients was 51.52 years. In a similar study conducted by Duah et al, mean age of the patients was 45 years. [7] Prihartini et al in a similar study reported mean age of the patients to be 56.6 years. [8] There was total 50 subjects, of which 82 % were males and 18 % were females. Similair to our study, Duah et al reported that 82.86 % of the patients were males [7]. In a similar study conducted by Prihartini et al, 62% of the patients were men while rest of them werewomen. [8] Alcohol was the etiology in 29 (58%) cases. NASH was the cause in 7 (14%) cases. 4 (8%) cases were due to Hepatitis B and 5 (10%) cases were because of hepatitis C. Alcohol and Hepatitis B in combination was the etiology in 5 (10%) cases. Prihartini et al, in a previous study reported that cause was HBV&HCV in 36.2 % & 40.4 % of the patients respectively.[8] Total bilirubin was 4.53 mg/dL. SGOT was 104.69 mg/dL and SGPT was 85.85mg/dL. Duah et al, in a previous study reported that mean SGOT and SGPT levels were found to be 155.06 mg/dL and 65.23 mg/dL respectively.[7] Mild and moderate ascites was seen in 20 (40%) and 11 (22%) cases, respectively. Massive ascites was seen in 9 (18%) cases. While in 10 (20%) cases, ascites was absent. In a previous study conducted by Duah et al, ascites was present in 81.88 % of the patients while it was absent in 18.12 % of the patients [7]. In 7 (14%) cases, hepatic encephalopathy was absent, whereas in 43 (86%) cases, it was absent. Similair to our study, Duah et al reported presence of hepatic encephalopathy in 18.79 % of the patients.[7] 14 (28%) subjects belonged to class A, 13 (26%) subjects belonged to class B and 23 (46%) cases belonged to class C. In a previous study conducted by Duah et al, authors reported that 8.16 %, 43.54 % and 48.3 % of the patients were of class A, class B and class C respectively [7]. Prihartini et al, in another study reported that 28 subjects (59.6%) Child A, 15 subjects (3 1.9%) Child B, and 4 subjects (8.5%) Child C. [8]

HEMATOLOGICAL PROFILE

Mean hemoglobin was discovered to be 9.18 g/dL. The mean platelet count was 113730/mm. In a study conducted by Duah et al, mean hemoglobin concentration and platelet count was 9.19 g/L and 98788.7 /mm respectively.[7]

BIOCHEMICAL VARIABLES

Mean albumin levels were 2.93. The amounts of proteins were found to be 6.11. The PT-INR was 2.04. In a previous study conducted by Duah et al, mean albumin levels were found to be 2.64 while total proteins were found to be 6.98. PT-INR in their study was found to be 1.87.[7]

USG VARIABLES

Mean liver span was 14.67. Mean spleen diameter was 131.32 mm. The mean portal vein diameter was 11.22. Duah et al, in a similar study reported mean spleen diameter to be 138.28 mm.[7]

PC/SD RATIO

The mean platelet count to spleen diameter ratio was found to be 925.17±620.90. The minimum value was 170.58 and the maximum value was 2909.09. In a previous study conducted by Duah et al, mean PLT count to spleen diameter ratio was found to be 855.36.[7]

UGI ENDOSCOPY FINDINGS

Grade I pathology was seen in 9 (18%) cases. Grade II pathology was seen in 13 (26%) cases. Grade III and Grade IV pathologies were seen in 17 (34%) and 11 (22%) cases.

COMPARISON OF AGE

Mean age of the patients with low& high-risk varices was 45.3 years and 43.1 years; on comparing, non-significant results were obtained. Sarangapani A et al in a similar study reported that mean age of the patients with low-risk varices and high-risk varices was 43.3 years and 42.5 years (p-value >0.05). [9] In another study conducted by Prihartini et al,mean age of the patients with low& high-risk varices was 57 years and 56 years (p-value > 0.05).Dakshinamoorthy et al in another study reported that mean age of the patients with and without varices was 57.84 years and 55.75 years respectively. [8,10]

COMPARISON OF GENDER

77.27 % and 85.71 % of the patients with low-risk varices and high-risk varices were males. Both high-risk and low-risk varices were comparable in terms of gender-wise distribution. Sarangapani A et al in a similar study also reported similar gender-wise distribution among patients with low-risk varices and high-risk varices (p-value >0.05).[9] In another study conducted by Duah et al, 78.52 % and 66.67 % of the patients with varices and without varices were males (p-value >0.05).[7] Dakshinamoorthy et al in another study reported that 90 % of the patients with varices and 75 % of the patients without varices were males. [10]

COMPARISON OF ETIOLOGIC PROFILE

Both the low-risk varices group and high-risk varices group were comparable in terms of etiologic profile. Similar to our study, Sarangapani A et al also reported similar in a similar study also reported similar etiologic profile among patients with low-risk varices and high-risk varices (p-value >0.05). [9] In a study conducted by Prihartini et al, Esophageal varices were high risk in 36 subjects (76.6%) out of47 study subjects, comprising 19 Child A subjects from 28 subjects (67.9%), 13 Child B subjects out of 15 subjects (86.7%), & 4 Child C subjects (100%). 11 subjects (23.4%) had low-risk varices, comprising 9 Child A subjects and 2 Child B subjects.[8]

COMPARISON OF HEMATOLOGICAL PROFILE

Mean Hb among patients with low-risk varices and high-risk varices was 9.53 g/dL and 8.91 g/dL respectively. Mean platelet count among patients with low-risk varices and high-risk varices was 145068.8 and 89107.1 respectively. Mean platelet count among patients with high-risk varices was significantly lower in comparison to patients with low-risk varices (p-value < 0.05). Our results were in concordance with the previous authors who also reported similar findings. Sarangapani A et al also reported similar Hb levels among patients withlow-risk varices and high-risk varices. In their study also, mean platelet count was significantly lower among patients with high-risk varices (157725) in comparison to patients with low-risk varices (202781). [9] Chandail VS et al, in a similar study reported that mean platelet count among patients with and without varices was 131.32 and 172.65 (p-value < 0.05); similar to our results. [11] Zaman et al reported that groups without varices had a higher mean platelet count (1,28,500) than the group with small varices (mean platelet count, 1,07,800) and platelet count of <90,000 raised the likelihood of having EV by nearly 2.5-fold. [12]

COMPARISON OF HEPATIC PROFILE

In our study, hepatic profile parameters were higher in the high-risk varices group, with total bilirubin (5.38 ± 5.39 vs 3.44 ± 3.97; p=0.164) and SGOT (118.1 ± 177.38 vs 87.63 ± 81.9; p=0.460) showing non-significant differences, while SGPT was significantly elevated (118.2 ± 162.44 vs 44.65 ± 21.9; p=0.040). Similarly, Prihartini et al. reported higher mean bilirubin in high-risk patients (1.78 vs 1.07; p > 0.05), supporting our findings of modest hepatic profile elevation in high-risk varices.[8]

COMPARISON OF BIOCHEMICAL PROFILE

High-risk varices were associated with significantly lower albumin levels (2.71 vs 3.19; p=0.019) and higher PT-INR (2.39 vs 1.57), indicating worsened liver function. Prihartini et al. similarly reported lower albumin in high-risk groups, supporting the link between hypoalbuminemia, coagulopathy, and variceal severity in cirrhotic patients [8]. Chandail VS et al in a similar study, reported that mean PT-INR among patients with and without varices was 1.75 and 1.26, respectively [11]. Manohar TP et al. found the INR value indicated to be used as a marker of predicting oesophageal varices with a p-value of 0.015 and on ROC analysis of 75.5%, a significant association between INR values and the size of varices.[13]

COMPARISON OF USG VARIABLES

In our study, liver span, spleen diameter, and portal vein diameter were significantly higher in patients with high-risk varices. These findings suggest a strong association between these ultrasonographic parameters and variceal severity. Prihartini et al. similarly reported increased portal vein diameter in high-risk groups, supporting our results.[8] Chandail VS et al. demonstrated in their study that patients with esophageal varices had a significantly greater mean spleen diameter (133.0 mm) compared to those without varices (122.2 mm). Additionally, they reported a significantly increased mean portal vein diameter in patients with varices (14.48 mm) versus those without (13.3 mm). [11] The utility of splenomegaly as a non-invasive predictive marker for esophageal varices was also highlighted by Chalasani and colleagues.[14] Similarly, Dakshinamoorthy et al. found that the spleen diameter was markedly larger in patients with varices (156.9 mm) compared to those without (128.6 mm). [10] Supporting these findings, Cherian et al. observed a significantly increased spleen diameter in patients with large varices (180 mm) relative to those with small varices (155 mm), consistent with the findings of our study. [15] Furthermore, Amarapurkar et al. reported that splenomegaly alone served as a significant predictor for the development of large esophageal varices.[16] In a prospective study, Sharma et al. identified splenomegaly and thrombocytopenia as independent

predictors for the presence of large esophageal varices. [17]

COMPARISON OF ASCITES AND HEPATIC ENCEPHALOPATHY

In our study, ascites was significantly more common in patients with high-risk varices (96.43%) compared to those with low-risk varices (59.09%) (p=0.0010), indicating a strong association between ascites and variceal severity. Hepatic encephalopathy was observed in 17.86% of the high-risk group and 9.09% of the low-risk group (p=0.8495), showing no significant correlation. Similarly, Duah et al. reported hepatic encephalopathy in 19.26% of patients with esophageal varices and 13.33% without varices, with the difference being statistically insignificant ($p > 0.05$), thereby aligning with our findings regarding hepatic encephalopathy and variceal risk stratification. [7] Similarly, Cherian et al. reported hepatic encephalopathy in 29.63% of patients with large varices and in 12.84% of those with small varices, which also was not statistically significant ($p > 0.05$). [15] Consistent with the findings of our study, Thomopoulos et al. identified the presence of ascites as an independent predictor for large esophageal varices. [18] Supporting this, Kassim et al. found ascites to be significantly more prevalent among patients with varices (75%) compared to those without varices (25%), aligning with the observations made in our analysis. [19]

COMPARISON OF MODIFIED CHILD TURCOTTE PUGH SCORE

In our study, high-risk varices were significantly associated with more severe liver dysfunction as per the Modified Child-Turcotte-Pugh (CTP) classification. Among patients with low-risk varices, 54.55% were Class A, 22.73% Class B, and 22.73% Class C. In contrast, only 7.14% of high-risk variceal patients were Class A, while 28.57% were Class B and a notable 53.57% were Class C (p=0.0011). This indicates that patients with high-risk varices tend to have more advanced hepatic decompensation. These findings align with the study by Cherian et al., who reported that large varices were significantly more prevalent in CTP Class C patients (48.15%) compared to small varices (12.84%), further supporting the correlation between variceal size and severity of liver disease. [15]

COMPARISON OF PLATELET COUNT: SPLEEN DIAMETER RATIO

The mean platelet count: spleen diameter ratio among the subjects of low-risk group and high-risk group was 1277.34±648.46 and 690.11±385.71, respectively. (p=0.0001). Hence; patients with high-risk varices were associated with significantly reduced platelet count: spleen diameter ratio in comparison to patients with low-risk varices. Dakshinamoorthy et al. [10] demonstrated that the platelet count-to-spleen diameter (PC/SD) ratio was significantly reduced in patients presenting with esophageal varices. This finding aligns with the research by Barrera et al. [20] and González-Ojeda et al. [21] , both of whom identified the PC/SD ratio as a reliable non-invasive predictor of esophageal varices. In a separate investigation, Cherian et al.[15] reported that the mean PC/SD ratio among patients with large varices was notably lower (462.5) compared to those with small varices (699.33), with the difference being statistically significant ($p < 0.05$).

The PC/SD ratio was originally introduced by Giannini et al. [22] as a dependable non-invasive tool for predicting the presence of esophageal varices, a finding that was later corroborated by a multicenter study predominantly involving individuals with hepatitis C virus (HCV)-associated cirrhosis. Supporting this observation, Agha et al. [23] reported similar outcomes in a comparable HCV-positive cohort. Additionally, Sen et al.[24] affirmed the sensitivity of the PC/SD ratio as a predictive marker in patients with HCV-induced cirrhosis.

Uppalapati S et al. highlighted portal vein diameter as a reliable non-invasive marker for predicting esophageal varices. A diameter >13 mm was significantly associated with variceal presence and severity, showing high sensitivity and specificity. This supports abdominal ultrasonography as a valuable screening tool for guiding early EGD and prophylaxis. [25]

Anbukumar T et al. evaluated non-invasive predictors of portal hypertension in patients with esophageal varices, noting 84% male predominance and alcohol-related cirrhosis in 76%. Most had grade II/III varices. Significant correlations were found with thrombocytopenia, Fibroscan-based liver stiffness, portal vein diameter, and PC/SD ratio, supporting early non-invasive risk stratification.[26]
PT-INR and ultrasonographic parameters, despite limited CBC and LFT utility, show promise as non-invasive predictors of varices, aiding early prophylaxis in resource-limited settings and high endoscopy workload scenarios.

CONCLUSION

The study found that alcohol-related cirrhosis was significantly associated with higher-grade esophageal varices. Low platelet count, elevated PT-INR, increased liver span, spleen diameter, portal vein diameter, and presence of ascites showed strong correlation with variceal severity. The platelet count to spleen diameter ratio was a significant inverse predictor of variceal grade. Routine labs and abdominal ultrasonography were effective, accessible, and affordable tools for early variceal detection. In

settings where UGIE is not feasible, these non-invasive parameters offer practical risk stratification and help guide prophylaxis decisions. Larger multicenter studies are needed to validate their predictive value across diverse populations.

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