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NON-INVASIVE PREDICTORS OF LARGE ESOPHAGEAL VARICES IN PATIENTS WITH CIRRHOSIS OF LIVER: A CROSS-SECTIONAL STUDY

General Medicine

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ABSTRACT

Background: Esophageal varices are a serious complication of cirrhosis with a high risk of bleeding. Identifying large varices using non-invasive methods can reduce the need for routine endoscopy.**Methods:** This cross-sectional study included 87 patients with cirrhosis and esophageal varices. Clinical, laboratory, and radiological parameters were evaluated. Varices were classified as small or large based on Baveno criteria. Statistical analysis was performed using Chi-square and independent t-test.**Results:** Of the 87 patients, 50 (57%) had small varices and 37 (43%) had large varices. Most laboratory parameters, including hemoglobin, platelet count, liver enzymes, and electrolytes, were not significantly associated with variceal size. Serum creatinine was significantly higher in patients with large varices ($p = 0.0086$). Ascitic fluid parameters, spleen size, portal vein diameter, and platelet count/spleen diameter ratio showed no significant correlation. However, higher Child-Pugh ($p = 0.0167$) and MELD scores ($p = 0.0078$) were significantly associated with large varices.**Conclusion:** Serum creatinine, Child-Pugh score, and MELD score are useful noninvasive predictors of large esophageal varices, whereas other parameters have limited predictive value.

KEYWORDS

Cirrhosis, Esophageal Varices, Meld Score, Child-pugh Score, Noninvasive Predictors

INTRODUCTION

Cirrhosis of the liver represents the end stage of chronic liver disease and is characterized by progressive fibrosis, architectural distortion, and the development of portal hypertension. One of the most clinically significant consequences of portal hypertension is the formation of esophageal varices, which are present in approximately 50% of patients with cirrhosis and increase in prevalence with disease severity [1,2]. Variceal hemorrhage is a life-threatening complication associated with high morbidity and mortality, with reported mortality rates ranging from 15% to 20% despite advances in management [3].

Early identification of patients at risk of developing large esophageal varices is crucial, as these patients benefit from primary prophylaxis with non-selective beta-blockers or endoscopic variceal ligation [4]. Currently, upper gastrointestinal endoscopy is considered the gold standard for the diagnosis and grading of esophageal varices. However, routine endoscopic screening in all cirrhotic patients is resource-intensive, invasive, and may not be feasible in low-resource settings or for repeated surveillance [5].

In view of these limitations, there has been increasing interest in identifying noninvasive predictors of esophageal varices and their severity. Various clinical, biochemical, and radiological parameters—such as platelet count, spleen size, portal vein diameter, and composite scores like Child-Pugh-Turcotte (CPT) and Model for End-Stage Liver Disease (MELD)—have been evaluated as potential surrogate markers of portal hypertension [2,6]. Despite numerous studies, no single parameter has demonstrated consistent reliability across different populations, and the search for accurate, easily accessible, and cost-effective predictors continues.

Therefore, the present study was undertaken to evaluate the role of noninvasive clinical, laboratory, and radiological parameters in predicting the presence of large esophageal varices in patients with cirrhosis of the liver, with the aim of identifying markers that could reduce the need for unnecessary endoscopic screening.

Materials and methods

This cross-sectional study titled *"Noninvasive Predictors of Large Esophageal Varices in Patients with Cirrhosis of Liver"* was conducted at a tertiary care hospital. A total of 87 patients diagnosed with cirrhosis of the liver and esophageal varices were included in the study. All patients aged more than 18 years, irrespective of gender and etiology, who were newly diagnosed with cirrhosis and were willing to participate were enrolled. Patients were excluded if they had a history of hematemesis treated elsewhere, were on prophylactic beta-blocker therapy, had previously undergone endoscopic variceal ligation, sclerotherapy, or transjugular intrahepatic portosystemic shunt (TIPS), or had portal vein thrombosis or hepatocellular carcinoma.

Cirrhosis was diagnosed based on a combination of clinical findings, laboratory parameters, and ultrasonographic evidence. After obtaining informed consent, a detailed clinical history was recorded and thorough physical examination was performed according to a predefined proforma.

All patients underwent comprehensive laboratory investigations including complete blood count (hemoglobin and platelet count), liver function tests (serum bilirubin, transaminases—SGOT/AST and SGPT/ALT, total protein, albumin and globulin), coagulation profile (prothrombin time/INR), and renal function tests (blood urea and serum creatinine). Serum electrolytes, including sodium and potassium, were also measured.

Ultrasonographic evaluation of the abdomen was performed in all patients using an Esaote MyLab 50 machine with appropriate probes. Parameters assessed included liver surface nodularity, presence of ascites, and spleen bipolar diameter (in mm). In addition, portal vein Doppler examination was performed, and the portal vein diameter was measured at its widest point just distal to the confluence of the splenic and superior mesenteric veins.

Diagnostic abdominal paracentesis was carried out under aseptic precautions after obtaining consent, and ascitic fluid was analyzed for protein and albumin levels. The Serum-Ascitic Albumin Gradient (SAAG) was calculated by subtracting ascitic fluid albumin from serum albumin.

All patients underwent upper gastrointestinal endoscopy after overnight fasting. The procedure was performed using an Olympus Actera video gastroscope under local anesthesia (5% xylocaine spray) by a single experienced examiner. Patients were evaluated for the presence of esophageal varices, gastric varices, and portal hypertensive gastropathy. Esophageal varices were classified according to the Baveno III and IV consensus criteria into small (<5 mm) and large (>5 mm) varices. Based on this classification, patients were divided into two groups: those with small varices and those with large varices.

The platelet count to spleen diameter ratio was calculated for all patients as platelet count (n/mm³) divided by spleen diameter (mm). Disease severity was further assessed using the Child-Pugh-Turcotte (CPT) score, incorporating serum bilirubin, serum albumin, prothrombin time/INR, ascites, and hepatic encephalopathy. Patients

were categorized into Class A (5–6 points), Class B (7–9 points), and Class C (≥10 points). Additionally, the Model for End-Stage Liver Disease (MELD) score was calculated using the formula: *MELD = 9.6 × ln (serum creatinine) + 3.8 × ln (serum bilirubin) + 11.2 × ln (INR) + 6.4.*

Patients were categorized into two groups based on endoscopic findings (small vs large varices), and clinical, laboratory, and radiological parameters were compared between these groups.

Data were analyzed using SPSS version 18 (SPSS Inc.). Continuous variables were expressed as mean ± standard deviation, median, and range, while categorical variables were expressed as frequencies and percentages. The Chi-square test was used to assess associations between categorical variables, including CPT and MELD scores with variceal size. The independent sample t-test was used to compare mean values of laboratory parameters, ascitic fluid parameters, and ultrasonographic findings between the two groups. A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1: Descriptive statistics for laboratory parameters according to size of varices

Laboratory parameters	Small (Mean ± SD, Median)	Large (Mean ± SD, Median)	P-value*
Hb (gm/dL)	9.20 ± 2.26 (9.3)	8.58 ± 2.11 (9)	0.1386
Platelets (lac/cmm)	1.47 ± 0.67 (1.36)	1.37 ± 0.81 (1.14)	0.4859
Bilirubin (mg/dL)	4.46 ± 4.66 (2.4)	6.27 ± 6.02 (5.1)	0.0613
SGOT (IU/L)	83.09 ± 73.73 (64.3)	93.94 ± 65.93 (90)	0.3791
SGPT (IU/L)	50.58 ± 35.92 (43.1)	63.27 ± 55.97 (46)	0.2475
SGOT/SGPT	1.63 ± 0.93 (1.45)	1.70 ± 0.86 (1.6)	0.7154
Albumin (mg/dL)	2.97 ± 0.92 (2.8)	2.71 ± 0.58 (2.7)	0.1210
Globulin (mg/dL)	3.25 ± 0.79 (3.2)	3.39 ± 0.83 (3.2)	0.4193
INR	1.35 ± 0.41 (1.3)	1.49 ± 0.39 (1.4)	0.0904
Blood urea (mg/dL)	34.34 ± 19.74 (27.65)	39.29 ± 21.66 (34.6)	0.2771
Sr. creatinine (mg/dL)	1.07 ± 0.40 (1)	1.45 ± 0.77 (1.1)	0.0086
Sodium (Na) (mg/dL)	133.66 ± 5.83 (134)	133.41 ± 5.86 (134)	0.8413
Potassium (K) (mg/dL)	4.11 ± 0.71 (4.1)	4.33 ± 0.75 (4.2)	0.1855

Most laboratory parameters did not show a statistically significant association with variceal size ($p > 0.05$). Although bilirubin and INR were higher in patients with large varices, these differences were not statistically significant. Serum creatinine was significantly higher in patients with large varices ($p = 0.0086$), suggesting a possible association between renal dysfunction and severity of portal hypertension.

Table 2: Descriptive statistics for ascitic fluid parameters according to size of varices

Ascitic fluid parameters	Small (Mean ± SD, Median)	Large (Mean ± SD, Median)	P-value*
TLC (per cmm)	299.83 ± 515.12 (200)	217.13 ± 279.36 (100)	0.3399
Protein (mg/dL)	1.36 ± 0.87 (1.2)	1.43 ± 0.73 (1.19)	0.6850
SAAG	1.73 ± 0.70 (1.4)	1.60 ± 0.52 (1.4)	0.3213

Ascitic fluid parameters did not show any statistically significant difference between groups, indicating limited utility in predicting variceal size.

Table 3: Descriptive statistics for USG spleen size according to size of varices

Parameter	Small (Mean ± SD, Median)	Large (Mean ± SD, Median)	P-value*
USG spleen size (mm)	128.96 ± 23.96 (130)	130.16 ± 22.85 (130)	0.8128

No statistically significant association was observed between spleen size and variceal size.

Table 4: Descriptive statistics for portal vein Doppler parameters according to size of varices

Portal vein Doppler	Small (Mean ± SD, Median)	Large (Mean ± SD, Median)	P-value*
Splenic vein (mm)	9.32 ± 0.71 (9)	9.59 ± 0.64 (10)	0.0638
Portal vein (mm)	11.57 ± 1.25 (11)	12.03 ± 1.27 (12)	0.0976

Although portal and splenic vein diameters were higher in patients with large varices, the differences were not statistically significant.

Table 5: Descriptive statistics for platelet count/spleen diameter ratio according to size of varices

Parameter	Small (Mean ± SD, Median)	Large (Mean ± SD, Median)	P-value*
Platelet count/spleen diameter ratio	1165.18 ± 546.88 (1077.86)	1077.30 ± 741.28 (918.18)	0.5448

No statistically significant association was found between platelet count/spleen diameter ratio and variceal size.

Table 6: Distribution of subjects as per CPT score and size of varices

CPT score	Small n (%)	Large n (%)	Total n (%)	P-value*
A	3 (6)	1 (3)	4 (5)	0.0167
B	36 (72)	17 (46)	53 (61)	
C	11 (22)	19 (51)	30 (34)	
Total	50	37	87	-

A statistically significant association was observed between CPT score and variceal size, with higher CPT grades (especially Class C) more common in patients with large varices.

Table 7: Distribution of subjects as per MELD score and size of varices

MELD score	Small n (%)	Large n (%)	Total n (%)	P-value*
<10	10 (20)	3 (8)	10 (11)	0.0078
11–20	32 (64)	16 (43)	46 (53)	
21–30	7 (14)	13 (35)	24 (28)	
≥31	1 (2)	5 (14)	7 (8)	
Total	50 (57)	37 (43)	87	-

MELD score showed a statistically significant association with variceal size, with higher scores observed more frequently in patients with large varices.

DISCUSSION

In the present study, multiple clinical, biochemical, and radiological parameters were evaluated for their association with the size of esophageal varices in patients with portal hypertension. The findings suggest that while several parameters showed trends toward association, only a few demonstrated statistically significant correlations.

Among laboratory parameters, serum creatinine was the only variable that showed a statistically significant association with variceal size, with higher levels observed in patients with large varices ($p = 0.0086$). This finding highlights the potential link between renal dysfunction and advanced portal hypertension, possibly reflecting systemic hemodynamic alterations and hepatorenal physiology in decompensated liver disease. Similar observations have been reported in previous studies, where worsening renal function has been associated with increased severity of cirrhosis and its complications, including varices [7,8].

Although bilirubin and INR were higher in patients with large varices, these differences did not reach statistical significance. These parameters are markers of hepatic synthetic and excretory function, and their elevation in advanced disease has been consistently documented [9,10]. The lack of statistical significance in this study may be attributed to sample size or variability within groups.

Other laboratory parameters, including hemoglobin, platelet count, liver enzymes, albumin, and electrolytes, did not show significant associations with variceal size. While thrombocytopenia has traditionally been considered a surrogate marker of portal hypertension due to hypersplenism, its predictive value for variceal size remains inconsistent across studies [11,12]. Our findings support the notion that platelet count alone may not be a reliable predictor.

Ascitic fluid parameters, including TLC, protein, and SAAG, also did not demonstrate any significant association with variceal size. This suggests that while ascites reflects portal hypertension, its biochemical characteristics may not directly correlate with the severity of varices. Similar conclusions have been drawn in earlier studies [13].

Radiological parameters such as spleen size and portal vein Doppler measurements (portal vein and splenic vein diameters) were higher in patients with large varices but did not reach statistical significance. Previous literature has shown mixed results, with some studies demonstrating significant correlations while others, like ours, reporting limited predictive value [14,15]. These discrepancies may be due to differences in measurement techniques, patient populations, and disease severity.

The platelet count to spleen diameter ratio, proposed as a non-invasive predictor of esophageal varices, did not show a statistically significant association in this study. While some studies have validated its utility as a screening tool [16], others have questioned its reliability, particularly in heterogeneous populations [17]. Our findings align with the latter, suggesting limited applicability in predicting variceal size.

Importantly, CPT (Child-Pugh-Turcotte) score demonstrated a statistically significant association with variceal size ($p = 0.0167$), with higher classes (especially Class C) being more prevalent among patients with large varices. This underscores the role of overall liver dysfunction in the progression of portal hypertension and variceal formation. Similar associations have been well documented in the literature [9,18].

Likewise, the MELD score showed a significant correlation with variceal size ($p = 0.0078$), with higher scores more frequently observed in patients with large varices. MELD score, being an objective and widely used prognostic tool, reflects disease severity and has been linked to complications of cirrhosis, including variceal bleeding risk [8,18]. Our findings reinforce its utility as a predictor of disease severity.

Overall, this study suggests that while individual laboratory and imaging parameters have limited predictive value, composite scoring systems such as CPT and MELD scores are more reliable indicators of variceal severity. The significant association of serum creatinine further emphasizes the importance of renal function in advanced liver disease.

Limitations

The study is limited by its sample size and single-center design, which may affect generalizability. Additionally, dynamic parameters such as hepatic venous pressure gradient (HVPG), which is considered the gold standard for assessing portal hypertension, were not included.

Conclusion (Discussion ending tone)

In conclusion, CPT score, MELD score, and serum creatinine were significantly associated with variceal size, whereas other laboratory, ascitic, and radiological parameters showed limited predictive utility. These findings support the use of composite clinical scoring systems over isolated parameters in assessing the severity of portal hypertension.

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