

Original Research Article

EVALUATION OF CLINICAL, BIOCHEMICAL, AND ULTRASONOGRAPHIC PREDICTORS OF LARGE ESOPHAGEAL VARICES IN PATIENTS WITH CHRONIC LIVER DISEASE: A PROSPECTIVE OBSERVATIONAL STUDY

R. Ramprasath¹, J. Kamaraj², S. Saravanan¹

¹Assistant Professor, Department of General Medicine, Government Theni Medical College and Hospital, Theni, Tamil Nadu, India.

²Associate Professor, Department of General Medicine, Government Dindigul Medical College, Tamil Nadu, India.

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Corresponding Author:

Dr. R. Ramprasath,
Assistant Professor, Department of
General Medicine, Government Theni
Medical College and Hospital, Theni,
Tamil Nadu, India.
Email: dhakshuram0306@gmail.com

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ABSTRACT

Background: Esophageal varices are a major complication of portal hypertension in patients with cirrhosis and are associated with significant morbidity and mortality due to variceal bleeding. Screening endoscopy remains the gold standard for diagnosis; however, it is invasive, costly, and often unnecessary in patients without significant varices. Identification of reliable non-invasive predictors may help reduce unnecessary endoscopic procedures. The objective is to evaluate clinical, biochemical, and ultrasonographic parameters for predicting large esophageal varices in patients with chronic liver disease and portal hypertension.

Materials and Methods: A prospective observational study was conducted among 100 consecutive patients with chronic liver disease and portal hypertension admitted to a tertiary care center. Clinical examination, laboratory investigations, ultrasonography, and upper gastrointestinal endoscopy were performed. Esophageal varices were graded using the Paquet classification. Large esophageal varices were defined as Grade III–IV varices. Univariate and multivariate logistic regression analyses were used to identify independent predictors. Receiver Operating Characteristic (ROC) curves were used to determine optimal cut-off values.

Results: Among 100 patients, 49% had large esophageal varices, 21% had small varices, and 30% had no varices. Univariate analysis demonstrated significant associations between large varices and platelet count ($p=0.02$), serum bilirubin ($p=0.04$), Child-Pugh score ($p=0.003$), spleen size ($p=0.0001$), portal vein diameter ($p=0.001$), and splenic vein diameter ($p=0.001$). Multivariate analysis identified palpable splenomegaly ($p=0.0001$), platelet count ($p=0.001$), spleen size ($p=0.003$), portal vein diameter ($p=0.001$), and splenic vein diameter ($p=0.001$) as independent predictors. A platelet count $<150,000/\text{mm}^3$ demonstrated 72.5% sensitivity and 75% specificity, while spleen size >13 cm showed 88.5% sensitivity and 83% specificity.

Conclusion: Low platelet count, splenomegaly, increased portal vein diameter, and increased splenic vein diameter are significant non-invasive predictors of large esophageal varices. These parameters may aid in selecting high-risk patients for endoscopic screening, thereby reducing unnecessary procedures and healthcare costs.

Keywords: Cirrhosis, Portal Hypertension, Esophageal Varices, Platelet Count, Splenomegaly, Non-invasive Predictors.

INTRODUCTION

Portal hypertension is one of the most important consequences of chronic liver disease and cirrhosis, resulting from increased resistance to portal blood flow and increased portal venous pressure.^[1] The development of portosystemic collateral circulation is a compensatory mechanism that decompresses the portal venous system, leading to the formation of esophageal varices.^[2] Esophageal varices are among the most serious complications of portal hypertension because of their potential to cause life-threatening upper gastrointestinal bleeding.^[3] At the time of diagnosis of liver cirrhosis, esophageal varices are present in approximately 40% of patients with compensated disease and up to 60% of those with decompensated disease.^[4] The prevalence and size of varices tend to increase with disease progression, significantly increasing the risk of variceal hemorrhage.^[5] Variceal bleeding remains a major cause of morbidity and mortality in patients with cirrhosis. The annual incidence of first variceal bleeding is estimated to be around 4% in patients with cirrhosis, with the risk closely related to the size of the varices.^[6] Large esophageal varices are associated with a substantially higher risk of bleeding than small varices due to increased wall tension and portal pressure.^[7] Mortality following an episode of variceal hemorrhage remains high, ranging from 20–25% within the first week despite advances in medical management. Therefore, early identification of patients with large esophageal varices is essential for instituting preventive measures such as non-selective beta-blocker therapy and endoscopic surveillance.^[8] Upper gastrointestinal endoscopy is currently considered the gold standard for detecting and grading esophageal varices.^[9] International guidelines recommend screening endoscopy for all patients with cirrhosis to identify varices at risk of bleeding. However, routine endoscopic screening places a considerable burden on healthcare resources and may expose many patients to unnecessary invasive procedures, especially when large varices are absent.^[10] Since only a proportion of cirrhotic patients develop clinically significant varices, there is increasing interest in identifying reliable non-invasive predictors that can accurately determine the presence of large esophageal varices.^[11,12] Several studies have investigated clinical, biochemical, and ultrasonographic parameters as potential non-invasive markers of esophageal varices. Factors such as thrombocytopenia, splenomegaly, Child-Pugh score, serum bilirubin levels, portal vein diameter, and splenic vein diameter have shown varying degrees of predictive value. These parameters reflect the severity of portal hypertension and hypersplenism and may serve as useful tools for risk stratification. However, the predictive accuracy of these markers varies among different populations due to differences in disease

etiology, nutritional status, and severity of liver dysfunction.^[13, 14] In India, chronic liver disease is commonly caused by alcohol-related liver disease and viral hepatitis, and patients often present at an advanced stage of illness. Data regarding the usefulness of non-invasive predictors of large esophageal varices in the Indian population remain limited.^[15] Therefore, this study was undertaken to evaluate the role of clinical, biochemical, and ultrasonographic parameters in predicting the presence of large esophageal varices among patients with chronic liver disease and portal hypertension. Identification of accurate non-invasive predictors may help reduce unnecessary endoscopic procedures, optimize resource utilization, and facilitate timely prophylactic management in high-risk patients.

MATERIALS AND METHODS

Study Design and Setting: This prospective observational study was conducted at the Institute of Internal Medicine, Rajiv Gandhi Government General Hospital, Chennai, a tertiary care referral center. Consecutive newly diagnosed patients with chronic liver disease and portal hypertension were enrolled during the study period after obtaining informed written consent.

Study Population: A total of 100 patients with chronic liver disease and portal hypertension, with or without a history of gastrointestinal bleeding, were included in the study. Patients aged between 18 and 80 years were eligible for enrollment. Patients with hepatocellular carcinoma detected on ultrasonography, primary hematological disorders, current treatment with beta-blockers or nitrates, and those with a history of previous surgical intervention for portal hypertension were excluded.

Clinical Evaluation: All patients underwent detailed clinical assessment at the time of enrollment. Demographic characteristics including age and sex were recorded. Relevant history regarding alcohol consumption, blood transfusion, duration of symptoms, and possible etiological factors was obtained. Clinical examination was performed to identify manifestations of chronic liver disease and portal hypertension, including jaundice, pallor, pedal edema, ascites, hepatic encephalopathy, hepatomegaly, splenomegaly, and abdominal venous collaterals.

Ascites was graded as mild, moderate, or severe based on clinical and ultrasonographic findings. Hepatic encephalopathy was graded according to Conn's classification. Compensated cirrhosis was defined as the absence of ascites and hepatic encephalopathy. Splenomegaly was defined as a spleen diameter greater than 100 mm on ultrasonography.

Laboratory Investigations: All patients underwent routine hematological and biochemical investigations including hemoglobin concentration,

total leukocyte count, platelet count, serum bilirubin, serum albumin, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), serum alkaline phosphatase, and prothrombin time. Serological testing for hepatitis B surface antigen (HBsAg) and antibodies to hepatitis C virus (anti-HCV) was performed to determine the etiology of liver disease. Additional investigations for Wilson's disease, autoimmune liver disease, and hemochromatosis were performed when clinically indicated. In patients with ascites, diagnostic paracentesis was carried out under aseptic precautions. Ascitic fluid albumin and serum-ascites albumin gradient (SAAG) were measured and analyzed.

Ultrasonographic Evaluation: All patients underwent abdominal ultrasonography with Doppler examination after an overnight fast. The parameters evaluated included maximum liver span, liver surface nodularity, spleen size measured along its longest axis, portal vein diameter, splenic vein diameter, presence of portosystemic collaterals, and presence of ascites.

Endoscopic Evaluation: Upper gastrointestinal endoscopy was performed using a Pentax video gastroscope within 2–3 days of admission. The presence and size of esophageal varices were assessed according to the Paquet grading system. Patients were categorized as having either large esophageal varices (Grade III–IV) or no/small esophageal varices (Grade 0–II). The presence of gastric varices, portal hypertensive gastropathy, portal hypertensive duodenopathy, and rectal varices was also documented. Gastric varices were classified according to Sarin's classification. The diagnosis of cirrhosis was established based on clinical, biochemical, and ultrasonographic findings.

Outcome Measures: The primary outcome of the study was the presence of large esophageal varices detected on upper gastrointestinal endoscopy. Clinical, biochemical, and ultrasonographic parameters were evaluated for their ability to predict the presence of large esophageal varices.

Statistical Analysis: Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) software version 11.0. Continuous variables were analyzed using Student's t-test, while categorical variables were compared using the Chi-square test. Variables found to be statistically significant on univariate analysis were subsequently entered into a multivariate logistic regression model to identify independent predictors of large esophageal varices. Receiver Operating Characteristic (ROC) curve analysis was performed to determine optimal cut-off values and assess the predictive performance of significant variables. Sensitivity, specificity, positive predictive value, and negative predictive value were calculated. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Baseline Characteristics of the Study Population:

A total of 100 patients with chronic liver disease and portal hypertension were included in the study. The median age of the study population was 45 years (range: 18–80 years). There were 70 males and 30 females, with a male-to-female ratio of 2.33:1 [Table 1]. The median duration of symptoms was 90 days (range: 10–240 days). Alcohol-related liver disease was the most common etiology, accounting for 52% of cases, followed by hepatitis B virus infection (21%), autoimmune hepatitis (5%), hepatitis C virus infection (2%), and other causes (20%). According to the Child-Pugh classification, 25 patients belonged to Child-Pugh class A, 35 patients to class B, and 40 patients to class C. Clinically detectable ascites was present in 50 patients, jaundice in 53 patients, pedal edema in 43 patients, splenomegaly in 42 patients, and hepatic encephalopathy in 10 patients. A previous history of gastrointestinal bleeding was documented in 53 patients.

Table 1: Clinical Characteristics of Study Patients

Characteristics	Number of Patients (n=100)	Percentage (%)
Male	70	70
Female	30	30
Alcohol	52	52
Hepatitis B Virus	21	21
Hepatitis C Virus	2	2
Autoimmune Hepatitis	5	5
Others	20	20
Child-Pugh A	25	25
Child-Pugh B	35	35
Child-Pugh C	40	40
Pallor	49	49
Jaundice	53	53
Pedal Edema	43	43
Previous GI Bleed	53	53
Ascites	50	50
Encephalopathy	10	10
Splenomegaly	42	42

Endoscopic Findings: Upper gastrointestinal endoscopy demonstrated esophageal varices in 70 patients. Large esophageal varices (Grade III–IV) were observed in 49 patients, whereas small

esophageal varices (Grade I–II) were present in 21 patients. Thirty patients did not have esophageal varices [Table 2] Thus, the prevalence of large esophageal varices in the study population was 49%.

Table 2: Portal Hypertension Related Endoscopic Findings

Endoscopic Findings	Number of Patients	Percentage (%)
No Varices	30	30.0
Small Varices	21	21.0
Large Varices	49	49.0

Presence of Large Varices According to Child-Pugh Class: The prevalence of large esophageal varices increased with worsening liver function. Large varices were present in 5 of 25 patients (19%) with Child-Pugh class A disease, 12 of 35 patients

(39%) with Child-Pugh class B disease, and 29 of 40 patients (62%) with Child-Pugh class C disease [Table 3] These findings indicate that the frequency of large esophageal varices increased progressively with increasing severity of liver disease.

Table 3: Presence of Large Varices According to Child-Pugh Class

Child-Pugh Class	Total Patients	Patients with Varices	Patients with Large Varices	Percentage (%)
A	25	14	5	19
B	35	27	12	39
C	40	33	29	62

Univariate Analysis of Predictors of Large Esophageal Varices: Clinical, laboratory, and ultrasonographic variables were analyzed to identify factors associated with large esophageal varices. Significant associations were observed for platelet count, serum bilirubin, Child-Pugh score, spleen size, portal vein diameter, and splenic vein diameter. Patients with large varices had significantly lower platelet counts than patients with no or small varices (157,725/mm³ vs. 202,781/mm³;

p=0.02). Mean serum bilirubin levels were significantly higher in patients with large varices (3.1 mg/dL vs. 2.2 mg/dL; p=0.04) [Table 4] The mean spleen size was significantly greater among patients with large varices (14.9 cm vs. 11.17 cm; p=0.0001). Likewise, portal vein diameter (13.9 mm vs. 11.3 mm; p=0.001) and splenic vein diameter (9.2 mm vs. 7.8 mm; p=0.001) were significantly higher in patients with large varices.

Table 4: Significant Variables Associated with Large Esophageal Varices on Univariate Analysis

Variable	No/Small Varices	Large Varices	p-value
Platelet Count (/mm ³)	202,781	157,725	0.02
Bilirubin (mg/dL)	2.2	3.1	0.04
Child-Pugh Score	9	9	0.003
Spleen Size (cm)	11.17	14.9	0.0001
Portal Vein Diameter (mm)	11.3	13.9	0.001
Splenic Vein Diameter (mm)	7.8	9.2	0.001

Multivariate Logistic Regression Analysis: Variables found significant on univariate analysis were subjected to multivariate logistic regression analysis to determine independent predictors of large esophageal varices [Table 5] Palpable

splenomegaly, platelet count, spleen diameter, portal vein diameter, and splenic vein diameter remained statistically significant independent predictors. Serum bilirubin lost significance after adjustment for confounding variables.

Table 5: Independent Predictors of Large Esophageal Varices

Predictor	p-value
Palpable Splenomegaly	0.0001
Platelet Count	0.001
Spleen Size	0.003
Portal Vein Diameter	0.001
Splenic Vein Diameter	0.001
Bilirubin	0.08

Diagnostic Accuracy of Non-Invasive Predictors: Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cut-off values for significant predictors. A platelet count below 150,000/mm³ demonstrated a sensitivity of 72.5% and specificity of 75% for

predicting large esophageal varices. A spleen diameter greater than 13 cm showed the highest sensitivity (88.5%) and specificity (83%). A portal vein diameter greater than 11.5 mm had a sensitivity of 76.5% and specificity of 80%, whereas a splenic

vein diameter greater than 8 mm demonstrated a

sensitivity of 70.6% and specificity of 72.6%.

Table 6: Diagnostic Performance of Non-Invasive Predictors

Parameter	Cut-off Value	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Platelet Count	<150,000/mm ³	72.5	75.0	63.8	70.5
Spleen Diameter	>13 cm	88.5	83.0	83.3	75.9
Portal Vein Diameter	>11.5 mm	76.5	80.0	78.0	78.6
Splenic Vein Diameter	>8 mm	70.6	72.6	70.6	72.7

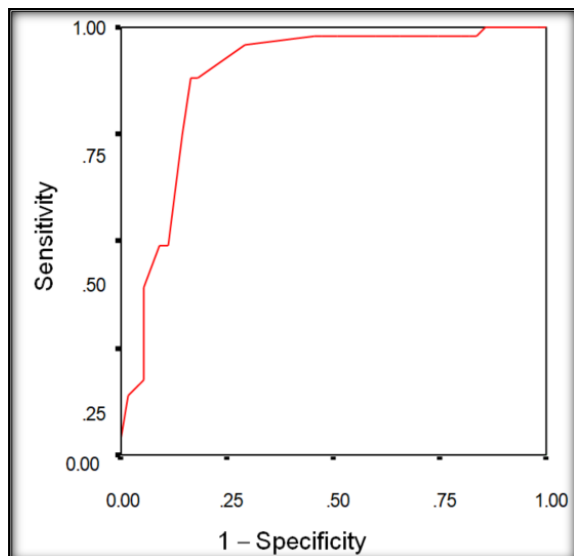


Figure 1: Spleen size: Area under curve: 0.883 [95% CI (0.813-0.912)]

Receiver Operating Characteristic Curve Analysis: ROC curve analysis demonstrated that spleen size was the most accurate predictor of large esophageal varices, with an area under the curve (AUC) of 0.883 (95% CI: 0.813–0.912) [Figure 1] Platelet count showed moderate predictive accuracy with an AUC of 0.701 (95% CI: 0.594–0.808) [Figure 2] Overall, spleen size, platelet count, portal vein diameter, and splenic vein diameter emerged as reliable non-invasive predictors of large esophageal varices in patients with chronic liver disease and portal hypertension.

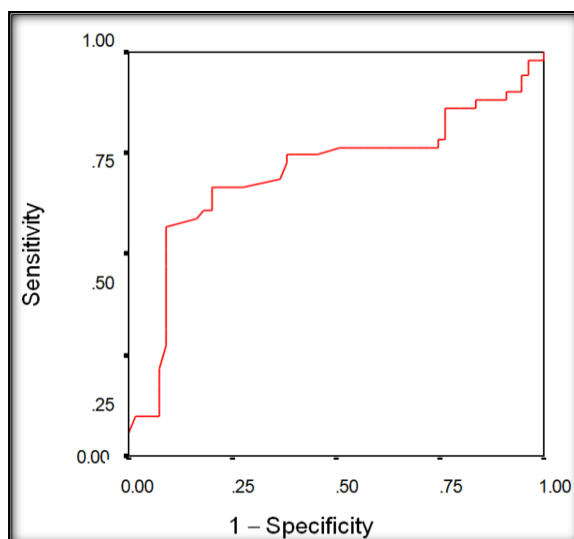


Figure 2: Platelet count: Area under curve: 0.701[95% CI (0.594-0.808)]

DISCUSSION

The present study was conducted to evaluate the usefulness of clinical, biochemical, and ultrasonographic parameters in predicting the presence of large esophageal varices in patients with chronic liver disease and portal hypertension. Early identification of large esophageal varices is clinically important because these patients are at a higher risk of variceal bleeding and are likely to benefit from primary prophylaxis. Since upper gastrointestinal endoscopy is invasive, expensive, and not readily available in all settings, the identification of reliable non-invasive predictors remains an important clinical objective. In the present study, esophageal varices were identified in 70% of patients, while large esophageal varices were present in 49% of patients. The prevalence of large esophageal varices observed in our study was higher than that reported by Thomopoulos et al.^[4] who found large varices in 17.9% of patients, and Chalasani et al.^[16] who reported a prevalence of 20%. The higher prevalence in our study may be attributed to the fact that a substantial proportion of patients belonged to Child-Pugh class C (40%), indicating more advanced liver disease at presentation. Similar observations have been reported in studies from developing countries where patients often seek medical attention at a later stage of disease. The severity of liver dysfunction was associated with an increasing prevalence of large esophageal varices. Large varices were observed in 19% of Child-Pugh class A patients, 39% of Child-Pugh class B patients, and 62% of Child-Pugh class C patients. These findings are in agreement with those reported by Zaman et al.,^[17] who demonstrated that advanced Child-Pugh class was an independent predictor of the presence of esophageal varices and large varices. Progressive worsening of liver function is associated with increasing portal hypertension, which explains the higher prevalence of varices in advanced disease. Among the laboratory parameters evaluated, platelet count emerged as an important predictor of large esophageal varices. Patients with large varices had significantly lower platelet counts than patients with no or small varices (157,725/mm³ versus 202,781/mm³, $p=0.02$). Multivariate analysis confirmed platelet count as an independent predictor ($p=0.001$). A platelet count below 150,000/mm³ demonstrated a sensitivity of 72.5% and specificity of 75% for predicting large varices. These findings are consistent with the studies conducted by

Chalasani et al.,^[16] Zaman et al.,^[17] Zein et al.,^[18] and Thomopoulos et al.,^[4] all of whom identified thrombocytopenia as an important non-invasive marker of esophageal varices. The relationship between thrombocytopenia and varices is likely due to hypersplenism and increased splenic sequestration of platelets resulting from portal hypertension.

Splenomegaly was another important predictor identified in the present study. Clinically palpable splenomegaly and ultrasonographic spleen diameter were both significantly associated with large esophageal varices. Spleen size greater than 13 cm demonstrated a sensitivity of 88.5% and specificity of 83%, making it the most accurate predictor in our study. The area under the ROC curve for spleen size was 0.883, indicating excellent discriminatory ability. Similar findings have been reported by Thomopoulos et al.,^[4] Madhotra et al.,^[19] Chalasani et al.,^[16] and Fagundes et al.,^[20] who identified splenomegaly as a significant predictor of esophageal varices. Enlargement of the spleen reflects the severity of portal hypertension and therefore correlates closely with the presence of large varices. Portal vein diameter was also found to be significantly associated with large esophageal varices. Patients with large varices had a mean portal vein diameter of 13.9 mm compared to 11.3 mm in patients without large varices ($p=0.001$). A portal vein diameter greater than 11.5 mm yielded a sensitivity of 76.5% and specificity of 80%. These findings are comparable to those reported by Cottone et al.,^[21] and Prihatini et al.,^[22] who demonstrated that increased portal vein diameter was associated with the presence of esophageal varices. Increased portal vein diameter reflects elevated portal venous pressure and may therefore serve as an indirect marker of portal hypertension severity. Similarly, splenic vein diameter emerged as an independent predictor of large esophageal varices. Patients with large varices had significantly larger splenic vein diameters than those without large varices (9.2 mm versus 7.8 mm, $p=0.001$). A splenic vein diameter greater than 8 mm demonstrated reasonable sensitivity and specificity. Although fewer studies have evaluated splenic vein diameter as a predictor, our findings support its usefulness as a non-invasive marker of clinically significant portal hypertension. Serum bilirubin levels were significantly associated with large varices on univariate analysis ($p=0.04$) but failed to retain significance on multivariate analysis ($p=0.08$). Similar observations have been reported in several previous studies where bilirubin was associated with disease severity but did not independently predict the presence of large varices after adjustment for other variables. This suggests that bilirubin reflects overall hepatic dysfunction rather than portal hypertension alone.

Interestingly, several clinical and laboratory parameters including age, sex, hemoglobin concentration, white blood cell count, serum

albumin, prothrombin time, liver size, and etiology of liver disease were not significantly associated with large esophageal varices. These findings are similar to those reported by Sharma et al.^[23] and Amarapurkar et al.,^[24] who found limited predictive value for these variables. The lack of association may be due to the multifactorial nature of portal hypertension and the influence of confounding factors on these parameters. Among all evaluated variables, spleen size demonstrated the highest diagnostic accuracy, followed by portal vein diameter and platelet count. These findings suggest that a combination of clinical examination and simple ultrasonographic parameters may be useful for identifying patients at high risk for large esophageal varices. Such an approach may reduce the number of unnecessary screening endoscopies, decrease healthcare costs, and improve patient comfort without compromising clinical care. The findings of the present study are consistent with previous literature and demonstrate that thrombocytopenia, splenomegaly, increased portal vein diameter, and increased splenic vein diameter are reliable non-invasive predictors of large esophageal varices. These parameters may help clinicians identify patients who would benefit most from endoscopic screening and prophylactic therapy.

Limitations of the Study: The present study has certain limitations that should be considered while interpreting the findings. It was conducted at a single tertiary care center and included a relatively small sample size of 100 patients, which may limit the generalizability of the results to other populations and healthcare settings. A large proportion of the study participants belonged to Child-Pugh class B and C, indicating advanced liver disease; therefore, the findings may not be directly applicable to patients with compensated cirrhosis or early-stage chronic liver disease. The grading of esophageal varices was based on endoscopic assessment, which is subject to inter-observer variability despite the use of standard grading criteria. In addition, only selected clinical, biochemical, and ultrasonographic parameters were evaluated, while other promising non-invasive markers such as transient elastography, liver stiffness measurement, and platelet count-to-spleen diameter ratio were not assessed. Further large-scale multicenter prospective studies are needed to validate these findings and improve predictive accuracy.

CONCLUSION

The present study demonstrated that large esophageal varices are a common complication in patients with chronic liver disease and portal hypertension, with a prevalence of 49% in the study population. Among the various clinical, biochemical, and ultrasonographic parameters

evaluated, low platelet count, palpable splenomegaly, increased spleen size, increased portal vein diameter, and increased splenic vein diameter were identified as independent predictors of large esophageal varices. Spleen size greater than 13 cm showed the highest diagnostic accuracy, while a platelet count below 150,000/mm³ was also significantly associated with the presence of large varices. These findings suggest that simple, readily available, and non-invasive parameters can be used to identify patients at high risk for large esophageal varices. The application of these predictors may help reduce the number of unnecessary screening endoscopies, decrease healthcare costs, and improve patient comfort. Larger multicenter studies are warranted to validate these findings and establish reliable non-invasive screening strategies.

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