

# RELATIONSHIP BETWEEN LIVER DISEASE SEVERITY (CHILD-PUGH CLASS) AND ENDOSCOPIC FINDINGS IN VARICEAL SCREENING

Original Research (ID: 1694)

Samiyya Rasool Abbasi<sup>\*1</sup>, Dr Shabnam Naveed<sup>2</sup>, Hiba Khairat Rizvi<sup>1</sup>, Momina Mazhar Ali<sup>1</sup>, Dr Zeeshan Ali<sup>3</sup>, Anjli<sup>1</sup>

<sup>1</sup>Post Graduate Trainee (MICU Ward 23), Jinnah Postgraduate Medical Centre

<sup>2</sup>Professor of Medicine (Ward 7), Jinnah Sindh Medical University/ Jinnah Postgraduate Medical Center

<sup>3</sup>Professor of Medicine (MICU Ward 23), Jinnah Sindh Medical University/ Jinnah Postgraduate Medical Center

**Corresponding Author:** Samiyya Rasool Abbasi, [abbasisamiyya@gmail.com](mailto:abbasisamiyya@gmail.com), Post Graduate Trainee (MICU Ward 23), Jinnah Postgraduate Medical Centre

**Acknowledgement:** The authors acknowledge the study participants and endoscopy unit staff for their support during data collection.

Conflict of Interest: None

Grant Support & Financial Support: None

## ABSTRACT

**Background:** Cirrhosis commonly leads to portal hypertension, resulting in gastroesophageal varices and other endoscopic manifestations that may progress to serious bleeding complications. Upper gastrointestinal endoscopy remains the standard method for variceal screening, but access may be limited in busy or resource-constrained settings. The Child-Pugh classification is a simple clinical score for assessing liver disease severity and may help identify patients at greater risk of significant endoscopic abnormalities.

**Objective:** This study aimed to determine the relationship between liver disease severity, assessed by Child-Pugh class, and endoscopic findings among patients undergoing variceal screening.

**Methods:** A cross-sectional observational study was conducted on 100 consecutive patients with chronic liver disease and portal hypertension. Demographic characteristics, clinical history, etiology of liver disease, medication use, laboratory parameters, and Child-Pugh scores were recorded. All patients underwent upper gastrointestinal endoscopy for assessment of esophageal varices, variceal grade, gastric varices, portal hypertensive gastropathy, ascites, and high-risk endoscopic stigmata. Data were analyzed using descriptive statistics, Pearson's chi-square test, linear-by-linear association, Spearman rank correlation, binary logistic regression, Mann-Whitney U test, and receiver operating characteristic curve analysis.

**Results:** The cohort included 66 males and 34 females, with a mean age of  $54.97 \pm 8.22$  years. Esophageal varices were present in 82 patients, including Grade I in 29, Grade II in 21, and Grade III in 32 patients. Gastric varices were observed in 38 patients, portal hypertensive gastropathy in 63, high-risk stigmata in 32, and ascites in 76. Child-Pugh class A, B, and C were present in 25, 45, and 30 patients, respectively. Grade III varices were found in 16.0% of class A, 26.7% of class B, and 53.3% of class C patients. Child-Pugh class was significantly associated with variceal grade ( $\chi^2 = 26.367$ ,  $p < 0.001$ ), and Child-Pugh score showed a positive correlation with variceal severity ( $r_s = 0.413$ ,  $p < 0.001$ ). The area under the curve for Child-Pugh score predicting large varices was 0.671.

**Conclusion:** Higher Child-Pugh class was associated with more severe esophageal varices and clinically important endoscopic findings. Child-Pugh scoring may support risk-based prioritization for endoscopic screening and prophylactic management, especially where endoscopy access is limited.

**Keywords:** Esophageal and Gastric Varices; Gastrointestinal Endoscopy; Liver Cirrhosis; Portal Hypertension; Severity of Illness Index; Splenomegaly; Upper Gastrointestinal Tract

## INTRODUCTION

Cirrhosis represents the advanced and largely irreversible stage of chronic liver disease, characterized by diffuse hepatic fibrosis, distortion of normal liver architecture, and regenerative nodule formation. It commonly develops as a final pathway of long-standing hepatic injury caused by chronic viral hepatitis, alcohol-related liver disease, non-alcoholic fatty liver disease, autoimmune hepatitis, and metabolic disorders. As cirrhosis progresses, deterioration in hepatic function and increasing portal pressure give rise to major complications such as ascites, hepatic encephalopathy, hepatocellular carcinoma, portal hypertension, and variceal haemorrhage. These complications substantially increase morbidity, hospital admissions, healthcare costs, and mortality, while also reducing patients' quality of life and long-term survival (1-3). Portal hypertension is one of the most clinically important consequences of cirrhosis. It develops due to increased resistance to portal venous blood flow through the fibrotic liver, leading to elevated portal pressure and the formation of portosystemic collateral vessels. Among these collaterals, gastroesophageal varices are particularly significant because of their potential to rupture and cause life-threatening upper gastrointestinal bleeding. The thin walls of these varices, combined with persistently raised portal pressure, make them vulnerable to rupture. Variceal bleeding remains one of the most feared complications of cirrhosis, as it is associated with high rates of emergency hospitalization, re-bleeding, intensive care requirement, and death, particularly in patients with advanced hepatic dysfunction (4-5).

The development of esophageal and gastric varices is closely related to the severity and duration of portal hypertension. Previous evidence suggests that gastroesophageal varices are present in a substantial proportion of patients at the time cirrhosis is diagnosed, with a higher prevalence among those with decompensated disease. Varices may also increase in size over time as portal pressure worsens, thereby increasing the risk of first variceal bleeding. Large varices and high-risk endoscopic signs, including red wale markings and cherry-red spots, are especially important because they indicate a greater likelihood of rupture. Early identification of patients at risk is therefore essential for timely prophylactic management and prevention of potentially fatal bleeding episodes (6-8). Upper gastrointestinal endoscopy remains the gold standard for detecting and grading gastroesophageal varices. It allows direct visualization of varices, assessment of their size, identification of high-risk bleeding stigmata, and evaluation of associated findings such as portal hypertensive gastropathy and gastric varices. Endoscopy also provides an opportunity for therapeutic intervention, particularly endoscopic variceal ligation, which plays an important role in the primary and secondary prevention of variceal bleeding. However, routine endoscopic screening of all patients with cirrhosis may be difficult in low-resource healthcare settings because of limited endoscopy facilities, high patient burden, procedural costs, and shortage of trained specialists. For this reason, clinically useful predictors are needed to help identify patients who are more likely to have significant varices and should be prioritized for endoscopic evaluation (8-10).

The Child-Pugh classification is one of the most widely used systems for assessing the severity of chronic liver disease. It incorporates five clinical and laboratory parameters: serum bilirubin, serum albumin, prothrombin time or international normalized ratio, ascites, and hepatic encephalopathy. Based on the total score, patients are categorized into Child-Pugh class A, B, or C, representing mild, moderate, and severe hepatic dysfunction, respectively. Beyond its established role in predicting survival and postoperative outcomes, the Child-Pugh score may also reflect the physiological burden of portal hypertension and the likelihood of cirrhosis-related gastrointestinal complications (10-11). Several studies have reported that the frequency and severity of esophageal varices increase with worsening Child-Pugh class. Patients in Child-Pugh class C are more likely to have advanced portal hypertension, impaired synthetic liver function, large varices, gastric varices, portal hypertensive gastropathy, and previous or impending variceal bleeding compared with patients in Child-Pugh class A. This relationship suggests that liver disease severity may serve as a practical clinical marker for predicting endoscopic findings in patients undergoing variceal screening. If such an association is clearly demonstrated, Child-Pugh class could help clinicians stratify patients according to risk and make better use of available endoscopic resources (11-13).

In Pakistan, chronic hepatitis B and hepatitis C remain major contributors to cirrhosis and portal hypertension, creating a considerable burden on tertiary care hospitals. Many patients present late, often after developing decompensated liver disease or complications of portal hypertension. Although variceal screening is an essential component of cirrhosis management, local evidence regarding the relationship between Child-Pugh class and endoscopic findings remains limited. This gap is important because local disease patterns, delayed presentation, healthcare access, and resource limitations may influence both the prevalence and severity of varices. The present study therefore aimed to determine the relationship between liver disease severity, assessed by Child-Pugh class, and endoscopic findings among patients undergoing variceal screening. It was hypothesized that higher Child-Pugh class would be associated with a greater frequency of significant endoscopic abnormalities, including esophageal varices, large varices, gastric varices, and portal hypertensive gastropathy. Establishing this relationship may support more efficient risk stratification and help prioritize high-risk cirrhotic patients for timely endoscopic assessment and preventive management.

## METHODS

This cross-sectional observational study was conducted on 100 consecutive patients with chronic liver disease and portal hypertension who presented for upper gastrointestinal endoscopic evaluation for variceal screening. The study was carried out after approval from the institutional ethical review committee/institutional review board. Written informed consent was obtained from all participants before enrolment, and confidentiality of patient information was maintained throughout the study by using anonymized study records. Patients of either sex with established chronic liver disease and clinical, laboratory, radiological, or endoscopic evidence of portal hypertension were included. Patients were eligible if they were referred for variceal screening and had complete clinical and laboratory data required for Child-Pugh classification. Patients were excluded if upper gastrointestinal endoscopy was contraindicated, if essential laboratory or clinical data were incomplete, if portal hypertension was due to non-cirrhotic causes, or if any coexisting condition prevented reliable assessment of liver disease severity or endoscopic findings.

Baseline demographic and clinical information was recorded for each participant, including age, sex, body mass index, etiology of chronic liver disease, duration of liver disease, history of previous variceal bleeding, and current pharmacological treatment, including diuretics and non-selective beta-blockers. Relevant laboratory investigations, including serum bilirubin, serum albumin, international normalized ratio, and platelet count, were obtained from the medical record or performed as part of routine clinical assessment. The Child-Pugh score was calculated using serum bilirubin, serum albumin, prothrombin time or international normalized ratio, presence and severity of ascites, and hepatic encephalopathy. Patients were then classified into Child-Pugh class A, B, or C, representing mild, moderate, and severe hepatic dysfunction, respectively. All patients underwent upper gastrointestinal endoscopy performed by trained gastroenterologists using a standard endoscopic protocol. Endoscopic findings were documented systematically, including the presence, size, and grade of esophageal varices; presence and type of gastric varices; portal hypertensive gastropathy; and high-risk stigmata such as red wale marks, cherry-red spots, or hematocystic spots. Esophageal varices were graded according to the standard institutional/endoscopic grading system used in the unit, and Grade III varices were considered large varices for analytical purposes. Details of management advised or performed during the index endoscopy, including initiation or continuation of non-selective beta-blocker prophylaxis, endoscopic variceal ligation, or combined therapy, were also recorded.

Data were entered and analyzed using IBM SPSS Statistics (version 25). Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean  $\pm$  standard deviation or median with interquartile range, depending on the distribution of data. The association between Child-Pugh class and categorical endoscopic findings, including variceal grade, gastric varices, and portal hypertensive gastropathy, was assessed using cross-tabulation and Pearson's chi-square test. Linear-by-linear association was used to evaluate ordinal trends across increasing Child-Pugh classes. Spearman's rank-order correlation was applied to determine the relationship between numerical Child-Pugh score and severity of esophageal varices. Binary logistic regression analysis was performed to identify independent predictors of large esophageal varices. Variables entered into the model included Child-Pugh score, platelet count, serum bilirubin, and international normalized ratio. The discriminative ability of the Child-Pugh score for detecting large varices was assessed using receiver operating characteristic curve analysis. Median Child-Pugh scores were compared between patients with and without large varices using the Mann-Whitney U test. A p-value of less than 0.05 was considered statistically significant for all analyses. This methodological approach allowed assessment of the relationship between the severity of liver dysfunction and endoscopic findings in patients undergoing variceal screening. It also helped evaluate whether Child-Pugh class and selected laboratory parameters could support risk stratification for clinically significant variceal disease and guide timely surveillance or prophylactic intervention.

## RESULTS

A total of 100 consecutive patients with proven chronic liver disease and portal hypertension were included in the study. The cohort consisted of 66 males (66.0%) and 34 females (34.0%), with a mean age of  $54.97 \pm 8.22$  years and an age range of 35–75 years. The mean body weight was  $66.90 \pm 10.57$  kg, mean height was  $166.92 \pm 9.55$  cm, and mean body mass index was  $24.12 \pm 3.98$  kg/m<sup>2</sup>. The mean duration of chronic liver disease was  $8.22 \pm 3.77$  years, ranging from 1 to 17 years. Hepatitis C virus infection was the most frequent cause of chronic liver disease, observed in 42 patients (42.0%). Hepatitis B virus infection and alcoholic liver disease were each reported in 17 patients (17.0%), while non-alcoholic fatty liver disease/non-alcoholic steatohepatitis was present in 13 patients (13.0%). Autoimmune hepatitis was documented in 7 patients (7.0%), and cryptogenic liver disease was found in 4 patients (4.0%). A history of previous variceal bleeding was present in 38 patients (38.0%), while splenomegaly was documented in 61 patients (61.0%). Non-selective beta-blocker therapy was being used by 51 patients (51.0%), and 54 patients (54.0%) were receiving diuretic treatment.

The mean serum bilirubin level was  $3.21 \pm 1.88$  mg/dL, with a range of 0.4–8.4 mg/dL. The mean serum albumin level was  $2.94 \pm 0.57$  g/dL, ranging from 1.8 to 4.4 g/dL, while the mean international normalized ratio was  $1.66 \pm 0.40$ , with a range of 1.00–2.85. The mean platelet count was  $109.83 \pm 47.15 \times 10^9/L$ , ranging from  $20$  to  $229 \times 10^9/L$ . According to the Child-Pugh classification, 25 patients (25.0%) were categorized as class A, 45 patients (45.0%) as class B, and 30 patients (30.0%) as class C. The mean Child-Pugh score was  $9.29 \pm 2.89$ , with a range of 5–15. Upper gastrointestinal endoscopy showed esophageal varices in 82 patients (82.0%), while 18

patients (18.0%) had no esophageal varices. Based on variceal grading in the total cohort, 29 patients (29.0%) had Grade I varices, 21 patients (21.0%) had Grade II varices, and 32 patients (32.0%) had Grade III varices. High-risk variceal stigmata, including red wale signs, cherry-red spots, or hematocystic spots, were present in 32 patients (32.0%) and absent in 68 patients (68.0%).

Gastric varices were identified in 38 patients (38.0%). Among patients with gastric varices, gastroesophageal varices type 2 were the most common subtype, observed in 18 patients (47.4%), followed by gastroesophageal varices type 1 in 16 patients (42.1%). Isolated gastric varices type 1 were present in 3 patients (7.9%), while isolated gastric varices type 2 were observed in 1 patient (2.6%). Portal hypertensive gastropathy was found in 63 patients (63.0%), including mild portal hypertensive gastropathy in 36 patients (36.0%) and severe portal hypertensive gastropathy in 27 patients (27.0%). Ascites was present in 76 patients (76.0%), including mild ascites in 31 patients (31.0%), moderate ascites in 31 patients (31.0%), and severe ascites in 14 patients (14.0%). At the index endoscopic assessment, 24 patients (24.0%) received no active intervention, 29 patients (29.0%) received beta-blocker prophylaxis alone, 23 patients (23.0%) underwent endoscopic variceal ligation, and 24 patients (24.0%) received combined endoscopic variceal ligation and beta-blocker therapy. The distribution of esophageal variceal grade differed across Child-Pugh classes. Among Child-Pugh class A patients, 11 patients (44.0%) had no varices, 9 patients (36.0%) had Grade I varices, 1 patient (4.0%) had Grade II varices, and 4 patients (16.0%) had Grade III varices. Among Child-Pugh class B patients, 6 patients (13.3%) had no varices, 14 patients (31.1%) had Grade I varices, 13 patients (28.9%) had Grade II varices, and 12 patients (26.7%) had Grade III varices. Among Child-Pugh class C patients, 1 patient (3.3%) had no varices, 6 patients (20.0%) had Grade I varices, 7 patients (23.3%) had Grade II varices, and 16 patients (53.3%) had Grade III varices.

The frequency of any esophageal varices increased from 56.0% in Child-Pugh class A to 86.7% in class B and 96.7% in class C. Grade II or Grade III varices were present in 20.0% of Child-Pugh class A patients, 55.6% of class B patients, and 76.7% of class C patients. Pearson’s chi-square test showed a statistically significant association between Child-Pugh class and esophageal variceal grade ( $\chi^2 = 26.367$ ,  $df = 6$ ,  $p < 0.001$ ). Linear-by-linear association also showed a significant ordinal trend across Child-Pugh classes ( $\chi^2 = 19.891$ ,  $df = 1$ ,  $p < 0.001$ ). One cell (8.3%) had an expected count of less than 5, and the likelihood ratio test remained statistically significant ( $\chi^2 = 26.967$ ,  $df = 6$ ,  $p < 0.001$ ). Spearman’s rank-order correlation analysis showed a statistically significant positive correlation between numerical Child-Pugh score and esophageal variceal grade ( $r_s = 0.413$ ,  $p < 0.001$ ). Patients with higher Child-Pugh scores had higher variceal grades on endoscopic assessment.

Binary logistic regression was performed to assess factors associated with large esophageal varices, defined as Grade III varices. The overall regression model was statistically significant ( $\chi^2 = 11.153$ ,  $df = 4$ ,  $p = 0.025$ ), with a Nagelkerke  $R^2$  value of 0.148. The Hosmer-Lemeshow goodness-of-fit test showed adequate model fit ( $\chi^2 = 9.582$ ,  $df = 8$ ,  $p = 0.296$ ), and the model correctly classified 72.0% of cases. In the multivariable model, Child-Pugh score had an odds ratio of 1.572 for large varices, with a 95% confidence interval of 0.976–2.532 and a p-value of 0.063. Platelet count had an odds ratio of 0.994, with a 95% confidence interval of 0.979–1.008 and a p-value of 0.378. Serum bilirubin had an odds ratio of 0.738, with a 95% confidence interval of 0.467–1.167 and a p-value of 0.194. The international normalized ratio had an odds ratio of 0.338, with a 95% confidence interval of 0.034–3.315 and a p-value of 0.352. Comparison of Child-Pugh scores between patients with and without large varices showed a statistically significant difference. Patients with Grade III varices had a higher mean rank for Child-Pugh score than patients without Grade III varices, with mean ranks of 62.13 and 45.03, respectively. The Mann-Whitney U test showed this difference to be statistically significant ( $U = 716$ ,  $Z = -2.822$ ,  $p = 0.005$ ). The median Child-Pugh score was 10.0 in patients with Grade III varices and 9.0 in patients without Grade III varices.

Receiver operating characteristic curve analysis was used to assess the performance of the Child-Pugh score for identifying Grade III esophageal varices. The area under the curve was 0.671, showing discrimination above chance level. A Child-Pugh score cut-off of  $\geq 8.5$  yielded a sensitivity of 78.1% and specificity of 41.2%, while a cut-off of  $\geq 10.0$  yielded a sensitivity of 50.0% and specificity of 79.4%.

**Table 1. Baseline Demographic and Clinical Characteristics of the Study Cohort (N = 100)**

Variable	n or Mean $\pm$ SD	% or Range
<b>Age (years)</b>		
Mean $\pm$ SD	54.97 $\pm$ 8.22	35 – 75
<b>Sex</b>		
Male	66	66.0%
Female	34	34.0%
<b>Anthropometric Parameters</b>		

Body weight (kg)	66.90 ± 10.57	47.5 – 106.5
Height (cm)	166.92 ± 9.55	146.4 – 187.2
BMI (kg/m <sup>2</sup> )	24.12 ± 3.98	16.6 – 37.7
Duration of CLD (years)	8.22 ± 3.77	1 – 17
<b>Aetiology of CLD</b>		
Hepatitis C	42	42.0%
Hepatitis B	17	17.0%
Alcoholic liver disease	17	17.0%
NAFLD/NASH	13	13.0%
Autoimmune hepatitis	7	7.0%
Cryptogenic	4	4.0%
<b>Laboratory Parameters</b>		
Serum bilirubin (mg/dL)	3.21 ± 1.88	0.4 – 8.4
Serum albumin (g/dL)	2.94 ± 0.57	1.8 – 4.4
INR	1.66 ± 0.40	1.00 – 2.85
Platelet count (×10 <sup>9</sup> /L)	109.83 ± 47.15	20 – 229
<b>Child-Pugh Classification</b>		
Child-Pugh Score (mean ± SD)	9.29 ± 2.89	5 – 15
Class A	25	25.0%
Class B	45	45.0%
Class C	30	30.0%
<b>Clinical Features</b>		
Past variceal bleeding	38	38.0%
Splenomegaly	61	61.0%
Beta-blocker use	51	51.0%
Diuretic use	54	54.0%

**Table 2. Frequency Distribution of Endoscopic and Clinical Findings (N = 100)**

Endoscopic / Clinical Finding	Frequency (n)	Percent (%)
<b>Esophageal Varices</b>		
Present	82	82.0
Absent	18	18.0
<b>Variceal Grade (among all patients)</b>		
No varices (Grade 0)	18	18.0
Grade I	29	29.0

Grade II	21	21.0
Grade III (large)	32	32.0
<b>High-Risk Variceal Stigmata</b>		
Present	32	32.0
Absent	68	68.0
<b>Gastric Varices</b>		
Present	38	38.0
Absent	62	62.0
<b>Gastric Variceal Type (among those with GV)</b>		
GOV2	18	47.4
GOV1	16	42.1
IGV1	3	7.9
IGV2	1	2.6
<b>Portal Hypertensive Gastropathy</b>		
None	37	37.0
Mild	36	36.0
Severe	27	27.0
<b>Ascites</b>		
None	24	24.0
Mild	31	31.0
Moderate	31	31.0
Severe	14	14.0
<b>Endoscopic / Medical Management</b>		
No intervention	24	24.0
Beta-blocker prophylaxis	29	29.0
Endoscopic variceal ligation (EVL)	23	23.0
EVL + Beta-blocker	24	24.0

**Table 3. Cross-Tabulation of Child-Pugh Classification Against Esophageal Variceal Grade**

Child-Pugh Class	No Varices (Grade 0)	Grade I	Grade II	Grade III	Total
Class A	11 (44.0%)	9 (36.0%)	1 (4.0%)	4 (16.0%)	25
Class B	6 (13.3%)	14 (31.1%)	13 (28.9%)	12 (26.7%)	45
Class C	1 (3.3%)	6 (20.0%)	7 (23.3%)	16 (53.3%)	30
Total	18	29	21	32	100

<b>p-value</b>	$\chi^2 = 26.367, df = 6, p < 0.001$
----------------	--------------------------------------

Data presented as n (row %).  $\chi^2$  = Pearson chi-square statistic.  $p < 0.05$  was considered statistically significant. 1 cell (8.3%) had expected count less than 5; Likelihood Ratio  $\chi^2 = 26.967, df = 6, p < 0.001$ .

**Table 4. Binary Logistic Regression: Predictors of Large (Grade III) Esophageal Varices**

Predictor	B	S.E.	Wald $\chi^2$	p	OR	95% CI
Child-Pugh Score	0.452	0.243	3.458	0.063	1.572	0.976 – 2.532
Platelet Count ( $\times 10^9/L$ )	-0.007	0.007	0.777	0.378	0.994	0.979 – 1.008
Serum Bilirubin (mg/dL)	-0.304	0.234	1.685	0.194	0.738	0.467 – 1.167
INR	-1.085	1.165	0.867	0.352	0.338	0.034 – 3.315
<b>Model Statistics</b>	$\chi^2 = 11.153, df = 4, p = 0.025$ ; Nagelkerke $R^2 = 0.148$ ; H-L $p = 0.296$					

B = logistic regression coefficient; S.E. = standard error; Wald  $\chi^2$  = Wald chi-square; OR = odds ratio; CI = confidence interval; H-L = Hosmer–Lemeshow goodness-of-fit test. Reference category: no large varices (Grade 0–II). Variables entered simultaneously by the Enter method.

**Table 5. ROC Curve Analysis Summary: Child-Pugh Score for Prediction of Large Esophageal Varices**

Test Variable	AUC	Cutoff	Sensitivity	Specificity	p-value
Child-Pugh Score (predicting Grade III varices)	0.671	$\geq 8.5$	78.1%	41.2%	$< 0.001$
Child-Pugh Score (predicting Grade III varices)	0.671	$\geq 10.0$	50.0%	79.4%	$< 0.001$
<b>Mann-Whitney U (Child-Pugh Score by Large Varices)</b>	U = 716; Z = -2.822; p = 0.005				

AUC = area under the receiver operating characteristic curve. Sensitivity and specificity values correspond to the specified cut-off thresholds derived from SPSS curve coordinate analysis. Mann-Whitney U statistics are included for comparison of Child-Pugh score distributions between groups.

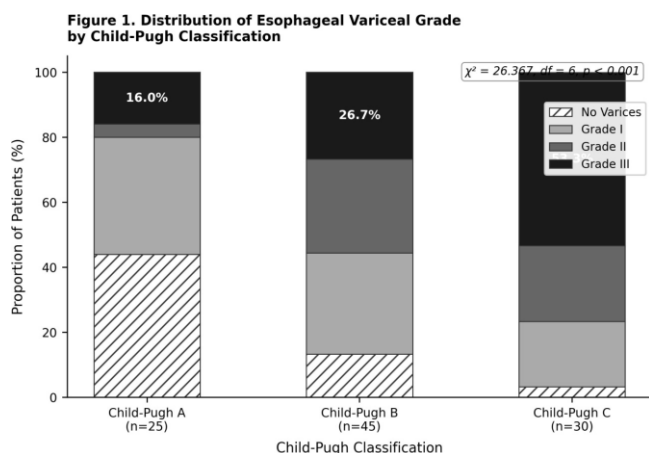


Figure 1. Distribution of esophageal variceal grade stratified by Child-Pugh classification. Each bar represents the proportional distribution of variceal grades (Grade 0 through Grade III) within each Child-Pugh class. Pearson chi-square analysis demonstrated a highly significant association between Child-Pugh class and variceal grade ( $\chi^2 = 26.367, df = 6, p < 0.001$ ). n values represent the number of patients in each class.

**Figure 2. ROC Curve: Child-Pugh Score for Prediction of Large (Grade III) Esophageal Varices**

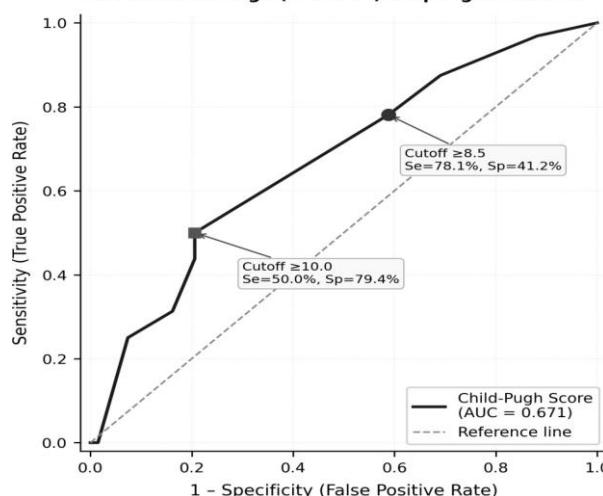


Figure 2. Receiver operating characteristic (ROC) curve for the Child-Pugh score as a predictor of large (Grade III) esophageal varices. The area under the curve (AUC) was 0.671 ( $p < 0.001$ ). The diagonal dashed line represents the reference line of no discrimination. Two clinically relevant cut-off points are annotated: a score of  $\geq 8.5$  (sensitivity 78.1%, specificity 41.2%) and  $\geq 10.0$  (sensitivity 50.0%, specificity 79.4%). Positive test result defined as Grade III varices (n = 32).

**Figure 3. Endoscopic and Clinical Severity Parameters**

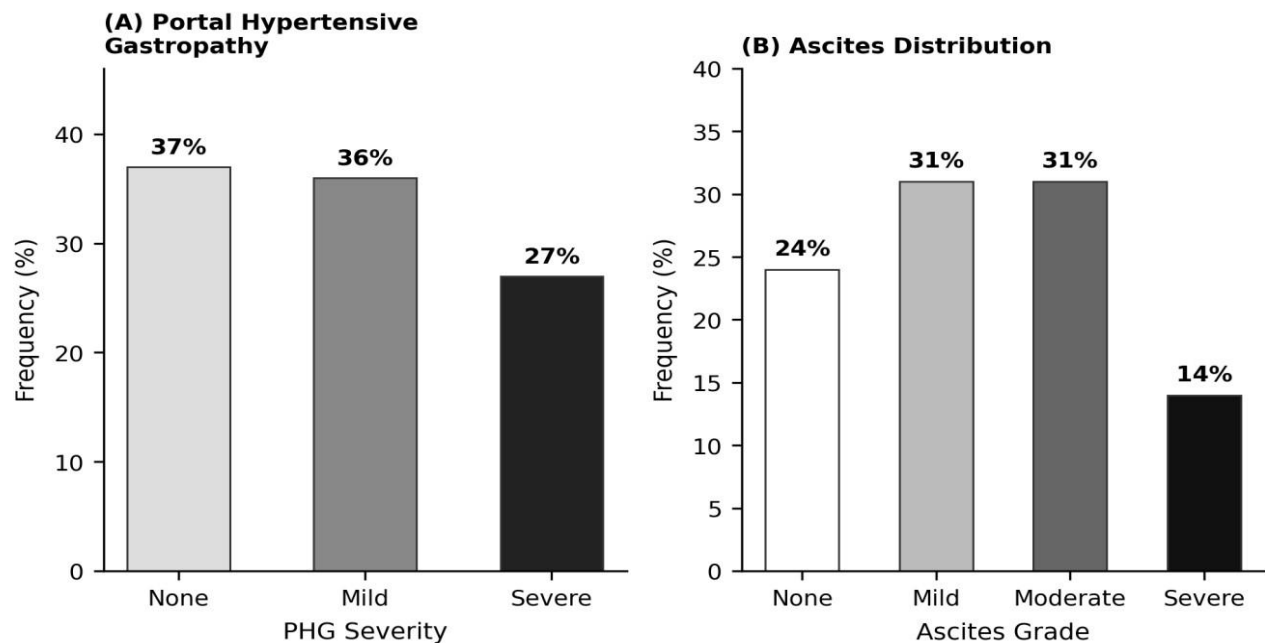


Figure 3. Distribution of portal hypertensive gastropathy (PHG) severity (Panel A) and ascites grade (Panel B) in the study cohort (N = 100). PHG was present in 63% of patients (mild 36.0%, severe 27.0%). Ascites was absent in 24.0% of patients; among those with ascites, mild and moderate degrees were equally prevalent (31.0% each). Values represent percentages of the total cohort.

## DISCUSSION

The present study demonstrated a high burden of esophageal varices among patients with chronic liver disease and portal hypertension undergoing variceal screening. Esophageal varices were identified in 82.0% of patients, while Grade III varices were present in 32.0%. A clear stepwise increase in variceal severity was observed with worsening Child-Pugh class. Patients in Child-Pugh class A more commonly had absent or low-grade varices, whereas patients in classes B and C showed progressively higher frequencies of Grade II and Grade III varices. The association between Child-Pugh class and variceal grade was statistically significant, and the positive correlation between numerical Child-Pugh score and variceal grade further supported the relationship between worsening hepatic dysfunction and more advanced variceal disease. These findings were consistent with previous literature showing that progression of cirrhosis and hepatic decompensation are closely related to the development and enlargement of gastroesophageal varices. Earlier studies and systematic reviews reported that patients with advanced Child-Pugh class had higher portal pressure, greater prevalence of varices, larger variceal size, and increased risk of variceal bleeding and mortality (14-16). The present findings therefore supported the clinical relevance of Child-Pugh classification not only as a prognostic tool for survival, but also as a practical marker of portal hypertensive complications. This association was particularly important in clinical environments where routine endoscopic access may be limited and where simple bedside risk stratification can assist in prioritizing patients for timely endoscopic assessment.

The observed pathophysiological relationship between Child-Pugh class and variceal severity was biologically plausible. As cirrhosis advances, progressive fibrosis, nodular regeneration, and architectural distortion increase intrahepatic vascular resistance. This process is accompanied by splanchnic vasodilation and increased portal venous inflow, leading to sustained portal hypertension. Over time, collateral vessels develop and enlarge, resulting in gastroesophageal varices. Patients with advanced Child-Pugh class also tend to have impaired hepatic synthetic function, hypoalbuminemia, coagulopathy, ascites, and encephalopathy, reflecting a more decompensated hemodynamic state. Previous research has shown that endoscopic severity of varices parallels increasing hepatic venous pressure gradient, which provides a physiological basis for the association observed in this study (16-19). The prevalence of gastric varices in the present cohort was 38.0%, which was broadly comparable to observational studies reporting gastric varices in a substantial proportion of patients with portal hypertension. Some previous studies reported lower frequencies, usually around 20–30%, which may be related to differences in disease severity, study setting, etiology of cirrhosis, and referral patterns. In this study, gastroesophageal varices type 2 were the most frequent gastric variceal subtype, followed by gastroesophageal varices type 1. This pattern suggested a clinically relevant burden of advanced portal hypertensive disease in the cohort. Portal hypertensive gastropathy was also common, being present in 63.0%

of patients, including severe portal hypertensive gastropathy in 27.0%. Similar observations have been reported in previous work, where portal hypertensive gastropathy was more closely related to portal pressure and decompensated cirrhosis than to isolated laboratory abnormalities (14).

High-risk endoscopic stigmata were present in 32.0% of patients. These included red wale signs, cherry-red spots, and hematocystic spots, which are clinically important because they have been associated with increased risk of variceal bleeding. The coexistence of large varices and high-risk stigmata has been emphasized in previous consensus-based and observational literature as a major indication for primary prophylaxis, including non-selective beta-blockers, endoscopic variceal ligation, or combined management where appropriate (15). The presence of these findings in nearly one-third of the study population highlighted the importance of timely endoscopic screening in patients with portal hypertension, particularly those with advanced liver dysfunction. In multivariable analysis, the Child-Pugh score showed the strongest association with Grade III esophageal varices, although it reached only marginal statistical significance after adjustment for platelet count, serum bilirubin, and international normalized ratio. Platelet count, serum bilirubin, and international normalized ratio did not independently predict large varices in the adjusted model. This finding suggested that liver disease severity as a composite clinical score may be more informative than isolated laboratory parameters. However, the result should be interpreted cautiously because serum bilirubin and international normalized ratio are already components of the Child-Pugh score, and their simultaneous inclusion in the same regression model may have reduced the independent effect of each variable. Previous studies have similarly suggested that composite scoring systems may perform better than individual biomarkers because they integrate clinical decompensation and laboratory dysfunction into a single severity measure (16-19).

Receiver operating characteristic analysis showed that the Child-Pugh score had moderate discriminatory ability for identifying Grade III esophageal varices, with an area under the curve of 0.671. A lower threshold of  $\geq 8.5$  provided higher sensitivity, while a threshold of  $\geq 10.0$  provided higher specificity. This finding suggested that Child-Pugh score may help support clinical prioritization but should not replace endoscopy. In settings where missing large varices carries substantial risk, a more sensitive threshold may be useful for early referral. Conversely, in settings with constrained endoscopic resources, a more specific threshold may assist in identifying patients most likely to have large varices. Nevertheless, the moderate area under the curve indicated that Child-Pugh score alone was insufficient as a standalone screening tool and should be used alongside clinical judgment, platelet count, splenomegaly, liver stiffness where available, and other non-invasive markers. The findings had practical relevance for healthcare systems with a high burden of chronic viral hepatitis and limited access to endoscopy. In such settings, many patients present with established cirrhosis or decompensated liver disease, making risk-based referral strategies clinically valuable. The progressive increase in variceal severity from Child-Pugh class A to class C indicated that patients with advanced Child-Pugh class should be considered a higher-priority group for endoscopic screening and prophylactic planning. The results also supported the continued use of Child-Pugh classification as a simple and accessible clinical tool in routine hepatology and gastroenterology practice.

A key strength of this study was the use of endoscopic confirmation in all enrolled patients, which strengthened the reliability of outcome assessment. The inclusion of consecutive patients reduced selection within the eligible clinic population, and the study evaluated a broad range of clinically relevant endoscopic findings, including esophageal varices, gastric varices, portal hypertensive gastropathy, and high-risk stigmata. The analysis also included several complementary statistical methods, including chi-square testing, ordinal trend assessment, correlation analysis, logistic regression, and receiver operating characteristic analysis, allowing the relationship between Child-Pugh severity and variceal disease to be assessed from different analytical perspectives. This study also had limitations. Its cross-sectional design did not allow assessment of causality, temporal progression, or future bleeding risk. The study was conducted at a single centre with a sample size of 100 patients, which may limit generalizability to wider populations with different etiological patterns, healthcare access, or disease severity. A considerable proportion of patients had previous variceal bleeding or were already receiving beta-blockers, which could have influenced the grade and appearance of varices at the time of endoscopy. Prior endoscopic therapy, if present, may also alter variceal morphology and should be accounted for in future analyses. In addition, hepatic venous pressure gradient, transient elastography, spleen stiffness, and other validated non-invasive markers were not assessed, limiting comparison with contemporary risk-stratification approaches. Possible multicollinearity in regression analysis also needed consideration because some laboratory variables overlapped with the components of the Child-Pugh score.

Future longitudinal studies with larger multicentre samples would help clarify whether worsening Child-Pugh class predicts variceal progression, first bleeding episode, re-bleeding, or mortality over time. Further research should also compare Child-Pugh classification with other non-invasive markers such as platelet count-to-spleen diameter ratio, liver stiffness, spleen stiffness, and Model for End-Stage Liver Disease score. Stratified analysis excluding patients with previous variceal bleeding or prior variceal treatment would provide a clearer estimate of screening-stage disease. Such studies may help develop locally applicable risk-stratification models for identifying cirrhotic patients who require urgent endoscopy and prophylactic intervention. Overall, the findings showed that increasing Child-Pugh class was significantly associated with higher esophageal variceal grade and a greater frequency of large varices among patients with chronic liver disease and portal hypertension. Although Child-Pugh score alone had only moderate discriminatory performance, it remained a clinically useful and easily available marker for identifying patients at higher risk of significant variceal

disease. These results supported the role of Child-Pugh classification as part of a broader risk-based approach to variceal screening and preventive management.

## CONCLUSION

This study concluded that liver disease severity, as assessed by Child-Pugh classification, was closely related to endoscopic findings in patients undergoing variceal screening. Patients with more advanced hepatic dysfunction were more likely to have higher-grade esophageal varices and clinically important portal hypertensive changes. Although Child-Pugh score should not replace upper gastrointestinal endoscopy, it may serve as a simple and practical clinical tool for identifying patients who require earlier endoscopic assessment and timely prophylactic management. These findings support risk-based screening strategies, particularly in healthcare settings where endoscopic resources are limited.

## AUTHOR CONTRIBUTION

Author	Contribution
Samiyya Rasool Abbasi	Conceptualization, Methodology, Formal Analysis, Writing - Original Draft, Validation, Supervision
Dr Shabnam Naveed	Methodology, Investigation, Data Curation, Writing - Review & Editing
Hiba Khairat Rizvi	Investigation, Data Curation, Formal Analysis, Software
Momina Mazhar Ali	Software, Validation, Writing - Original Draft
Dr Zeeshan Ali	Formal Analysis, Writing - Review & Editing
Anjli	Writing - Review & Editing, Assistance with Data Curation

## REFERENCES

1. Li S, Huang P, Jeyarajan AJ, et al. Assessment of non-invasive markers for the prediction of esophageal variceal hemorrhage. *Front Med (Lausanne)*. 2021;8:770836. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8672133/>
2. Tsois A, Marlar CA. Use Of The Child Pugh Score In Liver Disease. Treasure Island, FL: StatPearls [Internet], StatPearls Publishing; 2024. [PubMed]
3. MELD Na (UNOS/OPTN). MDCalc; 2024. Available from: <https://www.mdcalc.com/calc/78/meld-score-model-end-stage-liver-disease-12older>
4. Horvatits T, Mahmud N, Serper M, et al. MELD-lactate predicts poor outcome in variceal bleeding in cirrhosis. *Dig Dis Sci*. 2023;68:1042–1050. doi: 10.1007/s10620-022-07744-w.
5. Kamada Y, Munekage K, Nakahara T, et al. The FIB-4 index predicts the development of liver-related events, extrahepatic cancers, and coronary vascular disease in patients with NAFLD. *Nutrients*. 2022;15:66. Available from: <https://pubmed.ncbi.nlm.nih.gov/36615725/>
6. Fibrosis-4 (FIB-4) index for liver fibrosis. MDCalc; 2024. Available from: <https://www.mdcalc.com/calc/2200/fibrosis-4-fib-4-index-liver-fibrosis>
7. de Franchis R, Bosch J, Garcia-Tsao G, Reiberger T, Ripoll C, Baveno VII Faculty. Baveno VII: Renewing consensus in portal hypertension. *J Hepatol*. 2022;76(4):959-974. doi:10.1016/j.jhep.2021.12.022
8. Sarin SK, Lahoti D, Saxena SP, Murthy NS, Makwana UK. Prevalence, classification and natural history of gastric varices: a long-term follow-up study in 568 portal hypertension patients. *Hepatology (Baltimore)*. 1992;16(6):1343–1349. doi:10.1002/hep.1840160607
9. Sharma P, Kumar A, Sharma BC, Sarin SK. Comparison of scoring systems (Child Pugh vs MELD) for prediction of high risk esophageal varices in cirrhosis. *Clin Exp Gastroenterol*. 2021;14:19–27
10. Garcia-Tsao G, Abraldes JG, Rich NE, Wong VW. AGA Clinical Practice Update on the Use of Vasoactive Drugs and Intravenous Albumin in Cirrhosis: Expert Review. *Gastroenterology*. 2024;166(1):202-10.

11. Liu C, Zhang L, Zhang S, Li X, Wong YJ, Liang X, et al. Carvedilol to prevent decompensation of cirrhosis in patients with clinically significant portal hypertension stratified by liver stiffness: study protocol for a randomised, double-blind, placebo-controlled, multicentre trial in China. *BMJ Open*. 2024;14(7):e081623.
12. Yang L. [Current Interventional Management of Acute Upper Gastrointestinal Bleeding]. *Sichuan Da Xue Xue Bao Yi Xue Ban*. 2022;53(3):361-6.
13. Baumgartner K, Cooper J, Smith A, St Louis J. Liver Disease: Cirrhosis. *FP Essent*. 2021;511:36-43.
14. Ackermann O, Bernard O, Franchi-Abella S, Almes M, Tanase C, Jacquemin E, et al. The Natural History of Gastroesophageal Varices in Children With Portal Hypertension. *Gastroenterology*. 2026;170(1):188-98.
15. Nicoară-Farcău O, Han G, Rudler M, Angrisani D, Monescillo A, Torres F, et al. Pre-emptive TIPS in high-risk acute variceal bleeding. An updated and revised individual patient data meta-analysis. *Hepatology*. 2024;79(3):624-35.
16. Premkumar M, Dhiman RK, Duseja A, Mehtani R, Taneja S, Gupta E, et al. Recompensation of Chronic Hepatitis C-Related Decompensated Cirrhosis Following Direct-Acting Antiviral Therapy: Prospective Cohort Study From a Hepatitis C Virus Elimination Program. *Gastroenterology*. 2024;167(7):1429-45.
17. Zhang W, Huang Y, Xiang H, Zhang L, Yuan L, Wang X, et al. Timing of endoscopy for acute variceal bleeding in patients with cirrhosis (CHESS1905): A nationwide cohort study. *Hepatol Commun*. 2023;7(5).
18. Lv Y, Chen H, Luo B, Bai W, Li K, Wang Z, et al. Transjugular intrahepatic portosystemic shunt with or without gastro-oesophageal variceal embolisation for the prevention of variceal rebleeding: a randomised controlled trial. *Lancet Gastroenterol Hepatol*. 2022;7(8):736-46.
19. Wang Q, Zhao H, Deng Y, Zheng H, Xiang H, Nan Y, et al. Validation of Baveno VII criteria for recompensation in entecavir-treated patients with hepatitis B-related decompensated cirrhosis. *J Hepatol*. 2022;77(6):1564-72.