ISSN: 2320-9267

Indian Journal of Pharmaceutical and Biological Research (IJPBR)

Journal homepage: www.ijpbr.in

RESEARCH ARTICLE

Relationship between Portal Vein Diameter and Acute Variceal Bleeding in Patients with Chronic Liver Disease: A Cross-Sectional Observational Study

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ABSTRACT

Background: Acute variceal bleeding is a major and potentially life-threatening complication of portal hypertension in chronic liver disease. Although upper gastrointestinal endoscopy remains the standard method for diagnosing and grading varices, its invasive nature and limited accessibility highlight the need for simple non-invasive markers for risk stratification. **Objective:** To evaluate the relationship between portal vein diameter (PVD) and acute variceal bleeding in patients with chronic liver disease, determine an optimal PVD cut-off for predicting acute variceal bleeding, and assess the relationship between PVD and endoscopic grade of esophageal varices. **Methods:** This cross-sectional observational study included 100 adult patients with chronic liver disease, comprising 50 patients with acute variceal bleeding and 50 with non-bleeding varices. Clinical, laboratory, ultrasonographic and endoscopic findings were recorded. PVD was measured by B-mode ultrasonography, and esophageal varices were graded by upper gastrointestinal endoscopy. Group comparisons, one-way ANOVA, receiver operating characteristic (ROC) analysis and binary logistic regression were performed. **Results:** Mean PVD was significantly higher in patients with acute variceal bleeding than in non-bleeders (14.8 ± 1.04 mm vs. 13.5 ± 0.72 mm; $p<0.001$). ROC analysis identified a PVD cut-off of 15.5 mm, with 60% sensitivity, 88% specificity and an area under the curve of 0.817. On binary logistic regression, PVD was a significant predictor of acute variceal bleeding ($\beta=1.187$; OR 3.279, 95% CI 1.975–5.442; $p<0.001$). PVD increased progressively from Grade I to Grade IV varices, but the difference across grades was not statistically significant ($p=0.101$). Higher-grade varices and red wale signs were significantly more frequent among bleeders. **Conclusion:** Portal vein diameter was significantly associated with acute variceal bleeding and demonstrated good discriminatory ability for identifying patients at increased bleeding risk. Ultrasonographic measurement of PVD may serve as a simple, non-invasive adjunct for risk stratification, but it should not replace endoscopic assessment. **Recommendation:** Portal vein diameter should be interpreted together with clinical, laboratory, ultrasonographic and endoscopic parameters. Larger multicentric prospective studies are required to validate the 15.5 mm cut-off and assess its ability to predict future bleeding episodes.

Keywords: Chronic liver disease; portal vein diameter; acute variceal bleeding; hematemesis; portal hypertension; esophageal varices; ultrasonography.

Indian J. Pharm. Biol. Res. (2026): https://doi.org/10.30750/ijpbr.14.3.66

INTRODUCTION

Chronic liver disease is characterized by progressive hepatic injury with inflammation, fibrosis and architectural distortion, ultimately leading to cirrhosis and portal hypertension. Portal hypertension results in the formation of portosystemic collaterals, of which gastroesophageal varices are among the most clinically important. Acute variceal bleeding is a serious complication of portal hypertension and is associated with substantial morbidity and mortality.

Upper gastrointestinal endoscopy is the standard method for detecting and grading esophageal varices and identifying high-risk endoscopic stigmata. Hepatic venous pressure gradient measurement provides a direct hemodynamic assessment of portal hypertension but is invasive and not widely available. These limitations have stimulated interest in simple, repeatable and non-invasive markers that can assist in identifying patients at increased risk of portal hypertensive complications [4, 6, 7].Ultrasonography is widely available and permits assessment of portal venous and other features of portal hypertension. Portal vein diameter is simple to measure and has been evaluated as a potential non-invasive marker of portal hypertension and gastroesophageal varices. Previous studies have reported variable associations between increasing PVD, the presence and severity of esophageal varices, and bleeding risk [11, 15-20].Chouhan *et al.*, reported that a portal vein diameter greater than 1.5 cm predicted bleeding varices with a sensitivity of 55.56% and specificity of 80.70% [20]. Other studies have also demonstrated larger PVD in patients with esophageal varices or higher-grade varices, whereas some recent studies have reported limited discriminatory ability [15, 19-26]. Thus, the clinical utility of PVD remains variable and may depend on the population studied and the outcome assessed.The present study was therefore undertaken to evaluate the relationship between portal vein diameter and acute variceal bleeding in patients with chronic liver disease, to determine an optimal ultrasonographic PVD cut-off for identifying bleeding risk, and to assess the relationship between PVD and the endoscopic grade of esophageal varices.

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How to cite this article: Saini D, Rawat NS, Kumar R, Solanki S, Mukesh, Dheeraj, Rawat, K. Relationship between Portal Vein Diameter and Acute Variceal Bleeding in Patients with Chronic Liver Disease: A Cross-Sectional Observational Study. Indian J. Pharm. Biol. Res. 2026;14(3):411-416

Conflict of Interest: None declared.

Source of Funding: Nil.

Received: 01.06.2026 Revised: 11.06.2026 Accepted: 25.06.2026

MATERIALS AND METHODS

Study Design and Setting

This was a cross-sectional observational study conducted in the Department of General Medicine and the Department of Gastroenterology at Dr. S. N. Medical College and its attached hospitals, Jodhpur, Rajasthan, India. The study was conducted from Institutional Ethics Committee approval until completion of the required sample size.

Study Population

The study population comprised adult inpatients with chronic liver disease diagnosed on clinical, biochemical and imaging criteria, with or without hematemesis due to variceal bleeding. Patients were categorized into a bleeding-varices group and a non-bleeding-varices group.

Sample Size

The sample size was calculated using portal vein diameter values reported by Chouhan et al.[20], using the formula for hypothesis testing for two population means with an alpha error of 0.05 and study power of 80%. In the reference study, mean PVD was 1.55 ± 0.25 cm in the bleeding-varices group and 1.42 ±0.19 cm in the non-bleeding group. The calculated sample size was approximately 46 patients per group. After rounding, 50 patients were included in each group, giving a total sample size of 100 patients.

Inclusion Criteria

- Age ≥18 years.
- Admitted patients with chronic liver disease diagnosed using clinical, biochemical and imaging criteria.
- Patients presenting with or without hematemesis due to variceal bleeding.

Exclusion Criteria

- Hematemesis due to causes other than variceal bleeding, such as peptic ulcer disease, Mallory–Weiss tear or gastric malignancy.
- Pre-hepatic or post-hepatic causes of portal hypertension.
- Hemodynamic instability such as cardiogenic shock.
- Pregnancy.
- History of portal vein thrombosis, splenectomy or portosystemic shunt surgery.
- Current beta-blocker therapy.
- Previous endoscopic or surgical intervention for varices.
- Poor ultrasonographic window, including situations such as morbid obesity or massive ascites interfering with imaging.

Data Collection and Study Procedures

After obtaining written informed consent, data were collected using a structured proforma. Demographic details, duration and etiology of chronic liver disease, history of previous hematemesis and clinical signs were recorded. Laboratory evaluation included complete blood count, renal and liver function tests and coagulation parameters.

Portal vein diameter was measured using B-mode ultrasonography at the point where the portal vein crossed the inferior vena cava, with the patient in the supine position during quiet respiration. Additional ultrasonographic findings including spleen size, ascites and collateral vessels were recorded.

All patients underwent diagnostic upper

gastrointestinal endoscopy to confirm the presence and grade of esophageal varices and to document evidence of recent bleeding. Gastric varices and red wale signs were also recorded.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using IBM SPSS Statistics version 22.0 or later. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Continuous variables were compared using the independent Student's t-test or Mann–Whitney U test according to data distribution. Categorical variables were analyzed using the chi-square test or Fisher's exact test, as appropriate. One-way ANOVA or Kruskal–Wallis testing was used to compare PVD across endoscopic grades of esophageal varices. ROC curve analysis was performed to determine the optimal PVD cut-off for acute variceal bleeding, and binary logistic regression was used to assess PVD as a predictor of bleeding. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 100 patients with chronic liver disease were included: 50 patients with acute variceal bleeding and 50 patients with non-bleeding varices.

Baseline Characteristics

Table 1:Baseline characteristics

Characteristic	Bleeding varices (n=50)	Non-bleeding varices (n=50)	p-value
Mean age, years	47.90 ± 11.09	45.76 ± 12.79	0.374
Male sex	39 (78%)	35 (70%)	0.362
Alcohol-related CLD	22 (44%)	25 (50%)	0.680
Mean duration of CLD, years	4.40 ± 1.20	3.24 ± 1.20	<0.001
Previous hematemesis	29 (58%)	8 (16%)	<0.001

The mean age and sex distribution were comparable between groups. Alcohol-related liver disease was the most common etiology in both groups. The duration of chronic liver disease and a previous history of hematemesis were significantly greater or more frequent, respectively, among patients with bleeding varices.

Clinical and Ultrasonographic Findings

Table 2: Clinical and Ultrasonographic Findings

Variable	Bleeding varices (n=50)	Non-bleeding varices (n=50)	p-value
Pulse rate, beats/min	102.96 ± 8.30	90.36 ± 9.06	<0.001
Systolic BP, mmHg	102.70 ± 9.83	117.02 ± 14.82	<0.001
Diastolic BP, mmHg	66.10 ± 7.20	73.50 ± 7.25	<0.001
Respiratory rate, breaths/min	21.94 ± 3.09	18.98 ± 2.91	<0.001
Pallor	36 (72%)	15 (30%)	<0.001
Pedal edema	28 (56%)	8 (16%)	<0.001
Dilated abdominal veins	29 (58%)	16 (32%)	0.009
Spleen size, cm	16.77 ± 2.80	14.56 ± 2.01	<0.001
Collateral vessels	41 (82%)	24 (48%)	<0.001
Massive ascites	13 (26%)	4 (8%)	0.046

Patients with bleeding varices had higher pulse and respiratory rates and lower systolic and diastolic blood pressures. Pallor, pedal edema and dilated abdominal veins were significantly more frequent. Spleen size, collateral vessels and massive ascites were also more commonly associated with bleeding.

Portal Vein Diameter and Bleeding Status

Table 3: Portal Vein Diameter and Bleeding Status

Group	Mean PVD (mm)	SD	Range (mm)	p-value
Bleeding varices	14.8	1.04	Not reported in manuscript table summary	<0.001
Non-bleeding varices	13.5	0.72	Not reported in manuscript table summary	

Mean portal vein diameter was significantly greater in patients with acute variceal bleeding than in those with non-bleeding varices (14.8 ± 1.04 mm vs. 13.5 ± 0.72 mm; p<0.001).

Endoscopic Findings

Table 4:Endoscopic findings

Endoscopic/clinical variable	Bleeding varices (n=50)	Non-bleeding varices (n=50)	p-value
Grade I esophageal varices	5 (10%)	10 (20%)	<0.001 overall
Grade II esophageal varices	7 (14%)	22 (44%)	
Grade III esophageal varices	26 (52%)	13 (26%)	
Grade IV esophageal varices	12 (24%)	5 (10%)	
Child–Pugh A	1 (2%)	10 (20%)	<0.001 overall
Child–Pugh B	18 (36%)	30 (60%)	
Child–Pugh C	31 (62%)	10 (20%)	
Red wale sign present	36 (72%)	12 (24%)	<0.001
Gastric varices	5 (10%)	4 (8%)	0.727

Grade III and IV esophageal varices were more frequent among bleeders, whereas Grade I and II varices predominated among non-bleeders. Child–Pugh Class C and red wale signs were also significantly more frequent in the bleeding group.

Relationship between Portal Vein Diameter and Variceal Grade

Table 5: Relationship between Portal Vein Diameter and Variceal Grade

Endoscopic grade	n	Mean PVD (mm)	SD
Grade I	15	14.02	0.75
Grade II	29	14.05	0.87
Grade III	39	14.45	1.24
Grade IV	17	14.77	1.11

PVD increased progressively with increasing endoscopic grade, from 14.02 ± 0.75 mm in Grade I to 14.77 ± 1.11 mm in Grade IV. However, the difference across grades was not statistically significant on one-way ANOVA (p=0.101).

Diagnostic Performance of Portal Vein Diameter

Table 6: Diagnostic Performance of Portal Vein Diameter

ROC parameter	Value
Optimal PVD cut-off	15.5 mm
Sensitivity	60%
Specificity	88%
Area under ROC curve (AUC)	0.817

ROC analysis demonstrated good discriminatory ability of PVD for acute variceal bleeding. A cut-off of 15.5 mm provided 60% sensitivity and 88% specificity, with an AUC of 0.817.

Binary Logistic Regression

Table 7: Binary Logistic Regression

Variable	β coefficient	Odds ratio	95% CI	p-value
Portal vein diameter (per 1 mm increase)	1.187	3.279	1.975–5.442	<0.001

Portal vein diameter was a statistically significant predictor of acute variceal bleeding. Each 1 mm increase in PVD was associated with approximately 3.3-fold higher odds of acute variceal bleeding in the fitted binary logistic model.

DISCUSSION

The present study evaluated portal vein diameter as a non-invasive marker associated with acute variceal bleeding in chronic liver disease. The principal finding was a significantly higher mean PVD among patients with bleeding varices compared with those with non-bleeding varices. ROC analysis identified 15.5 mm as the optimal cut-off in this cohort, with good specificity and an AUC of 0.817.

The demographic profile was comparable between the two groups. Most patients were middle-aged, and males predominated in both groups. Alcohol-related liver disease was the most common etiology, but

the etiological distribution did not differ significantly between bleeders and non-bleeders. In contrast, a longer duration of chronic liver disease and a previous history of hematemesis were significantly associated with the bleeding group, consistent with the progressive nature of portal hypertension and the known tendency for recurrent variceal hemorrhage.Patients with acute variceal bleeding showed tachycardia, lower blood pressure and higher respiratory rate, consistent with the hemodynamic consequences of acute blood loss. Pallor was more frequent in the bleeding group, while pedal edema, dilated abdominal veins, larger spleen size, collateral vessels and massive ascites reflected more advanced portal hypertension and liver disease.The mean PVD of 14.8 ± 1.04 mm in bleeders was significantly higher than 13.5 ± 0.72 mm in non-bleeders. This observation is consistent with Chouhan *et al.,* who reported an association between larger PVD and bleeding varices and identified a threshold above 15 mm with sensitivity of 55.56% and specificity of 80.70% [20]. Other studies have also reported an association between increased PVD and esophageal varices or advanced variceal severity [11, 15, 17-19, 28].The ROC-derived cut-off of 15.5 mm is clinically relevant because of its high specificity of 88% in the present cohort. However, the sensitivity of 60% indicates that a lower PVD cannot exclude bleeding risk. Therefore, PVD should be considered an adjunctive risk marker rather than a replacement for endoscopy or comprehensive assessment of portal hypertension.Higher grades of esophageal varices were strongly associated with bleeding. Grade III and Grade IV varices predominated among bleeders, whereas Grade II varices were most common among non-bleeders. Red wale signs were also markedly more frequent in the bleeding group. These findings support established endoscopic concepts that larger varices and high-risk surface stigmata are associated with an increased risk of hemorrhage [33, 35, 49].Although mean PVD increased progressively from Grade I to Grade IV varices, the association was not statistically significant. This differs from studies reporting a significant correlation between PVD and variceal grade, including studies by Khan *et al.,* and Patel *et al.,* [17, 18] but is consistent with reports suggesting limited ability of PVD alone to discriminate variceal severity [23, 26]. The discrepancy may reflect the multifactorial nature of portal hypertension, collateral formation, physiological and respiratory variation, and ultrasonographic measurement variability. Binary logistic regression showed that increasing PVD was a significant predictor of acute variceal bleeding in the fitted model, with an odds ratio of 3.279 for each 1 mm increase. This strengthens the observed association between PVD and bleeding status. Nevertheless, because the present study was cross-sectional, the findings demonstrate association and discrimination within the study population rather than prospective prediction of future bleeding episodes.

LIMITATIONS

- The cross-sectional observational design allows assessment of association but cannot establish causality.
- Hepatic venous pressure gradient measurement was not performed, markers such as transient elastography, platelet count-to-spleen diameter ratio, portal vein velocity, congestion index and liver stiffness measurements were not evaluated.
- Patients were categorized according to bleeding status at presentation, and longitudinal follow-up was not undertaken; therefore, prediction of future bleeding episodes could not be assessed.

CONCLUSION

Portal vein diameter was significantly associated with acute variceal bleeding in patients with chronic liver disease. Patients with bleeding varices had a significantly greater mean PVD than non-bleeders, and a PVD cut-off of 15.5 mm demonstrated good discriminatory ability with high specificity. PVD increased numerically with higher grades of esophageal varices but did not show a statistically significant association with variceal grade. Ultrasonographic measurement of PVD may therefore serve as a simple, non-invasive and widely available adjunct for identifying patients at increased risk of acute variceal bleeding. It should, however, be interpreted in conjunction with clinical, laboratory, ultrasonographic and endoscopic findings and should not replace endoscopic assessment.

RECOMMENDATIONS

- PVD may be incorporated into routine ultrasonographic assessment of patients with chronic liver disease as an adjunctive marker of bleeding risk.
- Patients with a PVD around or above 15.5 mm may merit prompt endoscopic assessment, particularly where immediate endoscopy for all patients is not feasible.
- PVD should be interpreted together with factors such as liver disease severity, spleen size, ascites, collateral vessels, previous hematemesis, variceal grade and red wale signs.
- Future studies should evaluate composite models combining PVD with other non-invasive markers of portal hypertension.

Ethics Approval: Institutional Ethics Committee approval was obtained before enrollment. Written informed consent was obtained from eligible participants.

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