



“Thrombocytopenia and Splenic Enlargement as Predictors of Esophageal Varices in Liver Cirrhosis: A Comprehensive Study”

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Abstract

Background: Esophageal varices are a major complication of portal hypertension in liver cirrhosis and are associated with significant morbidity and mortality due to variceal bleeding. Routine surveillance endoscopy is recommended for detecting varices; however, endoscopic facilities may not be universally available, especially in low-resource settings. Thrombocytopenia and splenomegaly are frequent clinical and ultrasonographic findings in cirrhosis and reflect portal hypertension. Several studies suggest that platelet count, spleen size, and platelet count/spleen diameter ratio can serve as non-invasive predictors of esophageal varices, helping clinicians stratify risk and prioritize endoscopic screening.

Aims: To assess the correlation of platelet count, spleen bipolar diameter, and platelet count/spleen diameter ratio with the presence and grading of esophageal varices in patients with liver cirrhosis without prior evidence of gastrointestinal bleeding, and to identify candidates for surveillance endoscopy.

Methods: This comprehensive study included 50 patients with liver cirrhosis diagnosed based on clinical history, examination findings, impaired liver function tests, deranged coagulation profile, and abdominal ultrasound. Patients with current or previous variceal bleeding, prior endoscopic therapy (sclerotherapy/band ligation), TIPSS/surgery for portal hypertension, recent fever with thrombocytopenia, drug-induced thrombocytopenia, or those receiving beta-blockers/diuretics/antiplatelet drugs were excluded. All participants underwent platelet count assessment and ultrasonographic measurement of spleen bipolar diameter, followed by upper GI endoscopy for detection and grading of esophageal varices. Correlation was analyzed between variceal grade and (1) platelet count, (2) spleen size, and (3) platelet count/spleen diameter ratio.

Results: Among the 50 cirrhotic patients, 88% had esophageal varices while 12% had none. Variceal grading showed Grade I in 36%, Grade II in 36%, and Grade III in 16%. Thrombocytopenia was common: 44% of patients had platelet counts between 50,000–1,00,000/mm³ and 20% had counts <50,000/mm³. Spleen bipolar diameter was 100–200 mm in 60% and <100 mm in 40%. The platelet count/spleen diameter ratio was most frequently 501–1000 (48%), followed by <500 (24%). An inverse correlation was observed between platelet count and the presence/severity of varices, and similarly between platelet count/spleen diameter ratio and variceal grade, indicating that lower values were associated with higher grades of esophageal varices.

Conclusion: Platelet count, spleen bipolar diameter, and especially platelet count/spleen diameter ratio demonstrate clinically useful inverse correlations with the presence and severity of esophageal varices in cirrhosis. These simple, non-invasive parameters may help predict varices and guide early referral for endoscopy in settings where endoscopic screening is limited, enabling earlier identification and prevention of life-threatening variceal bleeding.

Keywords : Liver cirrhosis, Portal hypertension, Esophageal varices, Upper GI endoscopy, Thrombocytopenia, Splenomegaly, Platelet count, Spleen bipolar diameter, Platelet count/spleen diameter ratio, Non-invasive predictors, Variceal grading, Screening / surveillance endoscopy

Introduction

Liver cirrhosis represents the final common pathway of many chronic liver diseases and is characterized by progressive hepatic fibrosis with distortion of normal liver architecture. A major consequence of cirrhosis is portal hypertension, which leads to the development of portosystemic collateral circulation, most importantly esophageal varices. Variceal bleeding is one of the most serious complications of cirrhosis and is associated with high morbidity and mortality. Early identification of esophageal varices is therefore essential to prevent first variceal hemorrhage through timely initiation of prophylactic measures such as non-selective beta blockers or endoscopic variceal ligation.

Upper gastrointestinal endoscopy is considered the gold standard for the diagnosis and grading of esophageal varices. International guidelines recommend routine endoscopic screening of all cirrhotic patients at diagnosis and periodic surveillance thereafter. However, in many regions, routine endoscopic screening is limited due to non-availability of resources, cost constraints, and lack of trained personnel. Hence, there is an increasing need for simple, non-invasive, cost-effective predictors that can identify patients at higher risk of having varices and prioritize them for endoscopy.

Among the commonly observed manifestations of portal hypertension are splenomegaly and thrombocytopenia, which occur due to hypersplenism, splenic sequestration of platelets, and altered thrombopoietin production. Several studies have shown that platelet count and spleen size correlate with portal hypertension and may serve as surrogate markers for the presence of esophageal varices. The platelet count to spleen diameter ratio has also been proposed as a more reliable indicator by combining two parameters associated with portal hypertension.

This study aims to evaluate the correlation between platelet count, splenic bipolar diameter, and platelet count/spleen diameter ratio with the presence and grading of esophageal varices in patients with liver cirrhosis. Identifying reliable non-invasive predictors may help reduce unnecessary endoscopies and allow better allocation of resources, especially in low-resource healthcare settings.

Materials and Methods

This study was conducted as a hospital-based observational study on 50 patients diagnosed with liver cirrhosis, admitted to or attending the medicine department during the study period. Cirrhosis was diagnosed based on a combination of clinical history, physical examination, and supportive investigations including liver function tests, coagulation profile, and abdominal ultrasonography. Written informed consent was obtained from all participants prior to inclusion.

Inclusion criteria

All adult patients with confirmed liver cirrhosis, irrespective of etiology, who had no previous history of upper gastrointestinal bleeding and had not undergone prior screening for varices were included.

Exclusion criteria

Patients were excluded if they had:

Current or previous history of hematemesis/melena

Prior endoscopic therapy for varices (sclerotherapy or band ligation)

History of TIPSS or surgery for portal hypertension

Recent fever with thrombocytopenia

Drug-induced thrombocytopenia

Current use of beta-blockers, diuretics, antiplatelet drugs, or any therapy that may influence platelet count or portal pressure

Study procedures

All enrolled patients underwent:

1. Complete hemogram, with special emphasis on platelet count
2. Ultrasonography of abdomen, in which spleen bipolar diameter was measured in millimeters
3. Upper gastrointestinal endoscopy, performed in all subjects to detect the presence of esophageal varices and to grade them based on standard endoscopic criteria into Grade I, Grade II, or Grade III

The following parameters were recorded for each patient:

- Platelet count (per mm³)
- Spleen bipolar diameter (mm)
- Platelet count/spleen diameter ratio, calculated by dividing platelet count by spleen bipolar diameter

Statistical analysis

Data were entered and analyzed to determine correlations between:

- Platelet count and variceal presence/grade
- Spleen size and variceal presence/grade
- Platelet count/spleen diameter ratio and variceal presence/grade

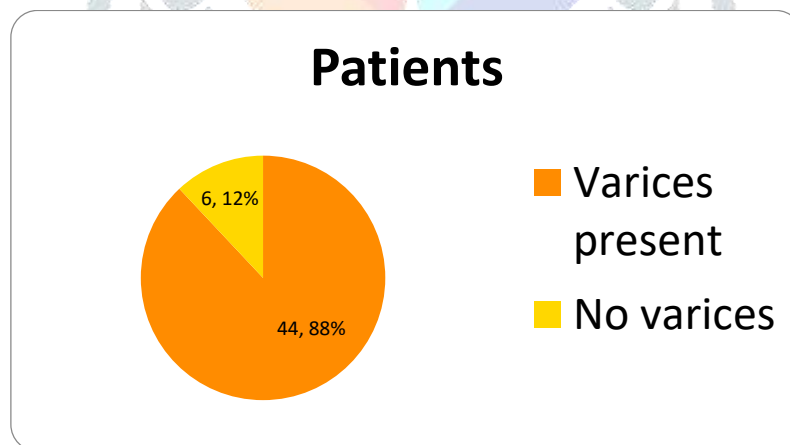
Correlation and comparative analysis were performed, and results were expressed as frequency distribution and association trends. The study aimed to identify whether these non-invasive parameters could act as predictors for esophageal varices in cirrhotic patients.

RESULTS

A total of 50 patients with liver cirrhosis were included in the study. On upper GI endoscopy, 44 patients (88%) were found to have esophageal varices, while 6 patients (12%) had no varices. This highlights the high prevalence of varices among cirrhotic patients even without prior GI bleeding.

Finding	Number	Percentage
Varices present	44	88%
No varices	6	12%

Table 1: Presence of Esophageal Varices

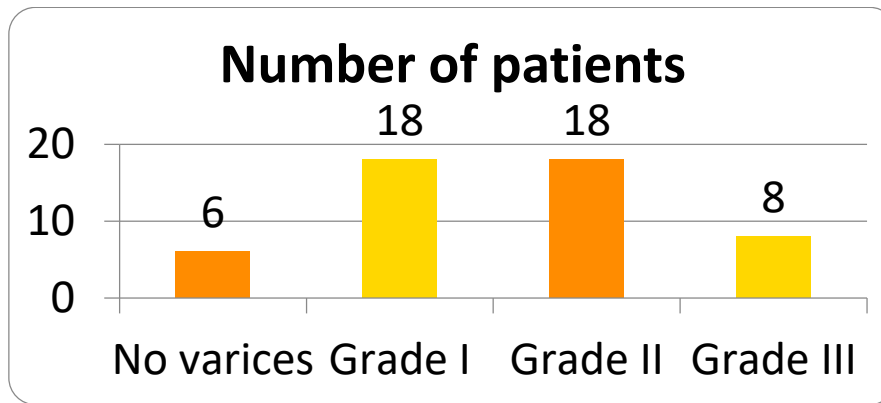


Graph 1: Varices Presence

The distribution of variceal grades showed that Grade I varices were present in 18 patients (36%), Grade II varices in 18 patients (36%), and Grade III varices in 8 patients (16%). Equal frequency of Grade I and Grade II indicates that most patients had early-to-moderate varices, emphasizing the need for systematic screening.

Grade	Number	Percentage
No varices	6	12%
Grade I	18	36%
Grade II	18	36%
Grade III	8	16%

Table 2: Variceal Grade Distribution

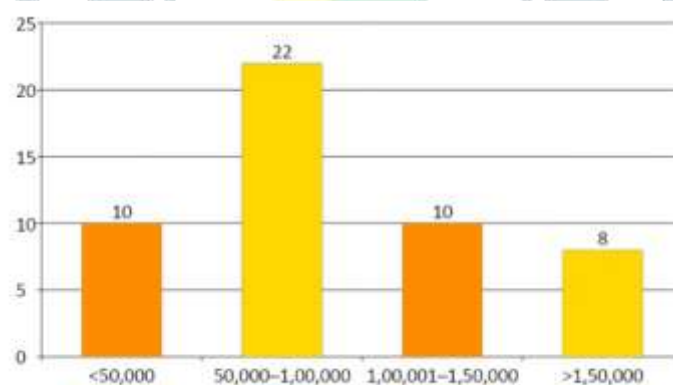


Graph 2: Variceal Grade Distribution

Analysis of platelet count demonstrated that thrombocytopenia was a common finding. 10 patients (20%) had platelet count $<50,000/\text{mm}^3$, 22 patients (44%) had counts between $50,000\text{--}1,00,000/\text{mm}^3$, 10 patients (20%) had $1,00,001\text{--}1,50,000/\text{mm}^3$, and 8 patients (16%) had platelet counts $>1,50,000/\text{mm}^3$. Lower platelet counts were more frequently associated with higher grades of varices, supporting thrombocytopenia as a surrogate marker of portal hypertension.

Platelet Count(mm^3)	Number	Percentage
$<50,000$	10	20%
$50,000\text{--}1,00,000$	22	44%
$1,00,001\text{--}1,50,000$	10	20%
$>1,50,000$	8	16%

Table 3: Platelet Count Distribution

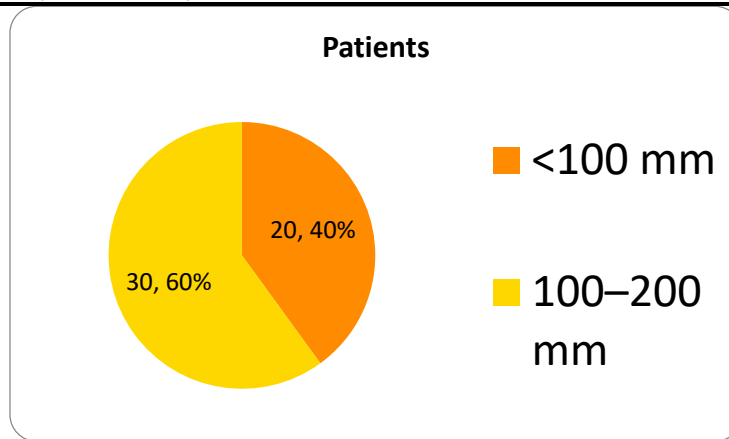


Graph 3: Platelet Count Distribution

On ultrasonography, splenic enlargement was frequently documented. 30 patients (60%) had spleen bipolar diameter between $100\text{--}200\text{ mm}$, while 20 patients (40%) had spleen diameter $<100\text{ mm}$. Patients with larger spleen size showed a greater likelihood of having esophageal varices, especially higher grades, indicating an association between splenomegaly and severity of portal hypertension.

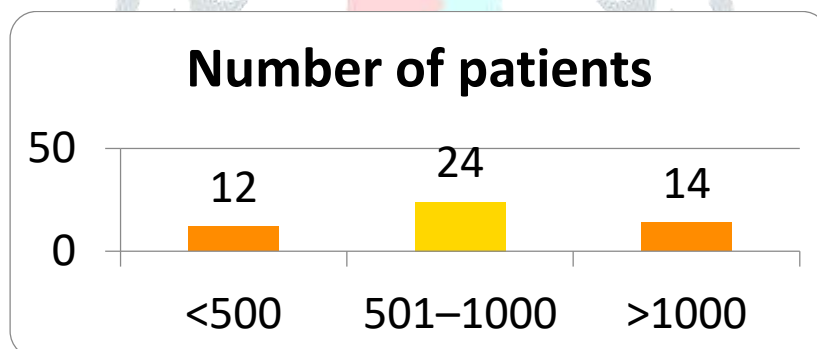
Spleen size (mm)	Number	Percentage
<100	20	40%
$100\text{--}200$	30	60%

Table 4: Spleen Bipolar Diameter

**Graph 4: Spleen Diameter Distribution**

The platelet count/spleen diameter ratio, a combined marker, showed that 24 patients (48%) had a ratio between 501–1000, 12 patients (24%) had a ratio <500, and 14 patients (28%) had a ratio >1000. Lower ratios were more strongly linked with higher grades of varices, suggesting this ratio may be a useful non-invasive predictor for variceal severity.

Ratio	Number	Percentage
<500	12	24%
501–1000	24	48%
>1000	14	28%

Table 5: Platelet Count / Spleen Diameter Ratio**Graph 5: Platelet/Spleen Ratio Distribution**

Overall, the study findings suggest that low platelet count, increased spleen diameter, and particularly a reduced platelet count/spleen diameter ratio correlate with the presence and grading of esophageal varices, supporting their use as cost-effective tools for triaging cirrhotic patients for endoscopic screening.

Discussion

Esophageal varices are among the most important clinical consequences of portal hypertension in liver cirrhosis and represent a major cause of morbidity and mortality due to the risk of life-threatening upper gastrointestinal bleeding. Although upper GI endoscopy is the gold standard for diagnosing and grading varices, routine endoscopic screening may not be feasible for all cirrhotic patients due to limited resources, cost, and accessibility issues, particularly in low-resource settings. Hence, identifying reliable non-invasive predictors of esophageal varices is clinically valuable for triaging patients who require early endoscopic evaluation.

In the present study, a very high prevalence of esophageal varices was observed (88%) among cirrhotic patients without previous history of gastrointestinal bleeding. This finding reflects the silent nature of portal hypertension and emphasizes that varices can develop early and remain asymptomatic until bleeding occurs. Most patients had Grade I and Grade II varices (each 36%), while Grade III varices were present in 16%. The predominance of lower-grade varices suggests that many patients could benefit from early detection and prophylactic measures before progression to large varices and bleeding risk.

Thrombocytopenia was a common hematological finding in this cohort. The majority of patients had platelet counts between 50,000–1,00,000/mm³, and 20% had severe thrombocytopenia (<50,000/mm³). Platelet count demonstrated an inverse relationship with variceal presence and severity—patients with lower platelet counts were more likely to have varices, particularly higher grades. This association can be explained by portal hypertension-induced hypersplenism, which leads to increased splenic sequestration and destruction of platelets, and also by reduced hepatic thrombopoietin production in advanced liver disease. Therefore, platelet count serves as an inexpensive and widely available marker reflecting portal hypertension severity.

Ultrasound measurement of spleen bipolar diameter also showed significant relevance. Splenomegaly was common, with 60% having spleen size between 100–200 mm. Larger spleen size correlated with higher grade varices, supporting the concept that splenic congestion is a surrogate marker of increased portal venous pressure. Ultrasonography being non-invasive and routinely performed in cirrhotic patients further supports its utility in predictive models.

Importantly, the platelet count/spleen diameter ratio appeared to be a stronger composite predictor than either parameter alone. Nearly one-fourth of patients had a ratio <500, and lower ratios were more frequently associated with higher grades of varices. This ratio integrates two independent effects of portal hypertension—splenic enlargement and platelet sequestration—thereby improving predictive performance. Such a combined marker may assist clinicians in selecting high-risk cirrhotic patients for urgent endoscopic screening and reducing unnecessary endoscopies in low-risk individuals.

Overall, the study supports the clinical usefulness of platelet count, spleen size, and platelet count/spleen diameter ratio as non-invasive indicators of esophageal varices. Incorporation of these parameters into screening strategies could lead to early diagnosis, timely prophylaxis, and reduction in variceal bleeding-related morbidity and mortality, especially where endoscopic facilities are limited.

Conclusion

Esophageal varices are a common and serious complication of portal hypertension in liver cirrhosis, and early identification is essential to prevent first variceal hemorrhage and related mortality. Although upper gastrointestinal endoscopy is the gold standard for detection and grading of varices, routine screening for all cirrhotic patients may not always be practical because of limited accessibility, cost, and resource constraints. Therefore, non-invasive predictors that can reliably identify patients at higher risk of varices are of great clinical importance.

In this study, a high prevalence of esophageal varices (88%) was observed among cirrhotic patients without previous gastrointestinal bleeding, emphasizing that varices can occur silently and may remain undetected until bleeding develops. Most patients had Grade I and Grade II varices, indicating a significant opportunity for early detection and initiation of prophylactic interventions before progression to large varices.

The findings demonstrate that platelet count and spleen bipolar diameter correlate with the presence and severity of varices. Thrombocytopenia and splenomegaly reflect underlying portal hypertension and hypersplenism, making them useful indicators in cirrhosis patients. Most importantly, the platelet count/spleen diameter ratio showed a stronger association with variceal grading, suggesting it may serve as a more dependable composite non-invasive marker.

Thus, low platelet count, increased spleen diameter, and a reduced platelet count/spleen diameter ratio can be used as practical, cost-effective tools to predict esophageal varices and stratify cirrhotic patients for endoscopic screening. Applying these parameters in routine clinical practice may help optimize resource utilization, reduce unnecessary endoscopies, and ensure timely endoscopic evaluation for high-risk patients. Ultimately, such an approach can improve early intervention strategies, reduce the incidence of variceal bleeding, and contribute to better outcomes in patients with liver cirrhosis.

References

1. Garcia-Tsao G, Sanyal AJ, Grace ND, Carey W. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Hepatology*. 2007;46(3):922–938.
2. de Franchis R; Baveno VI Faculty. Expanding consensus in portal hypertension: Report of the Baveno VI consensus workshop. *J Hepatol*. 2015;63(3):743–752.
3. North Italian Endoscopic Club for the Study and Treatment of Esophageal Varices. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices. *N Engl J Med*. 1988;319(15):983–989.

4. Giannini E, Botta F, Borro P, et al. Platelet count/spleen diameter ratio: Proposal and validation of a non-invasive parameter to predict the presence of esophageal varices in patients with liver cirrhosis. *Gut*. 2003;52(8):1200–1205.
5. Giannini EG, Zaman A, Kreil A, et al. Platelet count/spleen diameter ratio for the noninvasive diagnosis of esophageal varices: Results of a multicenter prospective study. *Am J Gastroenterol*. 2006;101(11):2511–2519.
6. Chalasani N, Imperiale TF, Ismail A, et al. Predictors of large esophageal varices in patients with cirrhosis. *Am J Gastroenterol*. 1999;94(11):3285–3291.
7. Zaman A, Hapke R, Flora K, Rosen HR, Benner K. Factors predicting the presence of esophageal or gastric varices in patients with advanced liver disease. *Am J Gastroenterol*. 1999;94(11):3292–3296.
8. Sarin SK, Kumar A. Gastric varices: Profile, classification and management. *Am J Gastroenterol*. 1989;84(10):1244–1249.
9. Christensen E, Fauerholdt L, Schlichting P, et al. Aspects of the natural history of gastrointestinal bleeding in cirrhosis and the effect of portal hypertension. *Hepatology*. 1981;1(4):350–356.
10. Bosch J, Abraldes JG, Berzigotti A, García-Pagán JC. Portal hypertension and gastrointestinal bleeding. *Semin Liver Dis*. 2008;28(1):3–25.
11. Tripathi D, Stanley AJ, Hayes PC, et al. UK guidelines on the management of variceal haemorrhage in cirrhotic patients. *Gut*. 2015;64(11):1680–1704.

