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Platelet Count/Splenic Diameter Ratio as a Non-Invasive Predictor of Esophageal Varices in Cirrhosis: An Observational Study

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Abstract

Background: Esophageal varices are a major complication of portal hypertension in cirrhosis, and endoscopic screening, though the diagnostic standard, is invasive and resource-intensive. The platelet count/splenic diameter (PC/SD) ratio has been proposed as a simple non-invasive predictor of esophageal varices. This study evaluated the diagnostic performance of the PC/SD ratio in patients with cirrhosis attending a tertiary care center. **Materials and Methods:** This cross-sectional observational study included 380 patients with cirrhosis undergoing screening upper gastrointestinal endoscopy. Platelet count and splenic diameter (by ultrasonography) were recorded for each patient, and the PC/SD ratio was calculated and correlated with endoscopic findings. **Results:** Esophageal varices were present in 261 (68.7%) patients. The mean PC/SD ratio was significantly lower in patients with varices than in those without (5.4 ± 2.7 vs. 10.9 ± 3.9; $P < 0.001$) but did not differ meaningfully between variceal grades. At a cut-off of ≤8.3, the PC/SD ratio predicted esophageal varices with 90.4% sensitivity, 89.1% specificity, and an area under the receiver operating characteristic curve of 0.868. The ratio correlated strongly with platelet count ($\rho = +0.916$) and splenic diameter ($\rho = -0.723$) and performed consistently across genders and etiologies of cirrhosis. **Conclusion:** The PC/SD ratio is a simple, inexpensive, and reliable non-invasive marker for predicting the presence of esophageal varices in cirrhosis and may help triage patients for screening endoscopy, particularly in resource-limited settings.

Keywords: Cirrhosis, Esophageal varices, Platelet count, Portal hypertension, Splenic diameter

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Introduction

Cirrhosis is a chronic, progressive disease of the liver characterized by widespread fibrosis and nodular regeneration, resulting in disrupted hepatic architecture, and remains an important cause of morbidity and mortality worldwide.[1] Global Burden of Disease estimates indicate that cirrhosis and other chronic liver diseases account for a substantial and rising share of deaths worldwide, with the burden disproportionately concentrated in low- and middle-income countries, where access to specialized hepatology and endoscopy services is often limited.[2] Esophageal varices, dilated submucosal vessels in the lower esophagus that arise as a consequence of portal hypertension, are among the most clinically significant complications of cirrhosis, since variceal hemorrhage carries a high risk of death, with reported mortality of 15–40% per bleeding episode, particularly in resource-limited settings where late presentation is common.[1]

Screening upper gastrointestinal endoscopy has traditionally been recommended for patients diagnosed with cirrhosis, to detect the presence, size, and high-risk features of esophageal varices and guide prophylactic treatment. The Baveno VII consensus now permits screening endoscopy to be avoided in selected compensated patients on the basis of liver stiffness measurement and platelet count; however, these criteria depend on transient elastography, which is not widely available in many resource-limited settings.[3] Endoscopic screening is invasive, requires specialized equipment and trained personnel, can be uncomfortable for patients, and repeated surveillance is not always feasible in all clinical settings. These constraints have driven considerable interest in identifying reliable non-invasive predictors of esophageal varices that could stratify patients by risk and direct endoscopy toward those most likely to benefit, reducing unnecessary procedures in low-risk patients.

Among the proposed non-invasive predictors, platelet count and splenic diameter, both easily obtained during routine work-up, have received particular attention. Thrombocytopenia in cirrhosis is multifactorial, arising from hypersplenism, reduced thrombopoietin production, and bone marrow suppression, while splenomegaly reflects chronic portal hypertension through increased splenic sequestration of blood and its cellular components. The PC/SD ratio was first proposed by Giannini *et al*. as a simple, inexpensive, and reproducible marker that integrates the combined impact of thrombocytopenia and splenomegaly into a single measure more representative of portal hypertension than either parameter alone,[4] and multiple subsequent studies and meta-analyses have supported a lower PC/SD ratio as a strong indicator of the presence of esophageal varices.[5]

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Nevertheless, considerable inter-study variability in the diagnostic accuracy and optimal cut-off value of the PC/SD ratio has been reported, attributable to differences in population, institution, etiology of cirrhosis, and measurement methodology.[6] Simple laboratory- and ultrasound-based non-invasive criteria have therefore not been universally accepted as a substitute for endoscopy, but in settings where endoscopic facilities are limited, the PC/SD ratio may offer a practical means of triaging patients for screening.[7] This study was undertaken to evaluate the validity of the PC/SD ratio as a non-invasive predictor of esophageal varices in cirrhotic patients attending a tertiary care center in central India, with the aim of identifying validated non-invasive predictors that could reduce reliance on endoscopy, improve patient compliance, and facilitate early identification of high-risk patients who would benefit from prophylactic treatment.

Materials and Methods

Study Design and Setting

This was a single-center, hospital-based, cross-sectional observational study conducted in the Department of General Medicine and Department of Gastroenterology, L.N. Medical College and Research Centre and J.K. Hospital, Bhopal, a tertiary care teaching hospital, over a period of 18 months, comprising a 3-month planning phase, a 12-month participant recruitment and data collection phase, and a 3-month data analysis and report-writing phase. Ethical clearance was obtained from the Institutional Ethics Committee before commencement, and the study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants. A detailed history, including demographic details, duration of liver disease, and history of alcohol consumption, was recorded for each participant in a structured case record pro forma.

Participants

Patients aged 18 years or above, of either sex, diagnosed with cirrhosis and undergoing screening upper gastrointestinal endoscopy for esophageal varices, were enrolled using consecutive sampling from among those attending the outpatient and inpatient departments of General Medicine. Patients with active upper gastrointestinal bleeding, a previous history of endoscopic sclerotherapy or band ligation, prior surgery for portal hypertension (including transjugular intrahepatic portosystemic shunt or surgical shunts), previous beta-blocker prophylaxis for varices, inability to abstain from alcohol, or those who declined consent were excluded from the study.

Clinical and Laboratory Assessment

A detailed history and thorough general and systemic examination were performed for all participants, including assessment for jaundice, pallor, clubbing, cyanosis, pedal edema, ascites, hepatomegaly, and splenomegaly, and disease severity was graded using the Child-Pugh classification. A venous blood sample was analyzed for a complete blood count, including platelet count, using an automated hematology analyzer, together with liver function tests and coagulation studies.

Radiological Assessment

All participants underwent abdominal ultrasonography with a curvilinear probe in the supine position, and the maximum bipolar splenic diameter was measured by an experienced radiologist blinded to the clinical and endoscopic findings. The PC/SD ratio was calculated for each patient as platelet count ($\times 10^3/mm^3$) divided by splenic diameter (cm).

Endoscopic Evaluation

All patients underwent upper gastrointestinal endoscopy performed by an experienced gastroenterologist who was blinded to the PC/SD measurements. Esophageal varices, when present, were graded as Grade I (small), Grade II (medium), or Grade III (large) according to standard classification, and the presence of red color signs, gastric varices, and portal hypertensive gastropathy was also recorded.

Sample Size and Data Handling

Sample size was determined based on the prevalence of esophageal varices in cirrhotic patients reported in previous studies, and consecutive sampling was used to enroll all eligible patients meeting the inclusion and exclusion criteria during the study period. Each participant was assigned a unique identification number to maintain confidentiality; de-identified paper forms were stored securely, and electronic data were maintained on a password-protected system accessible only to the research team.

Statistical Analysis

Data were entered in Microsoft Excel 2021 and analyzed using the Statistical Package for the Social Sciences version 27. Continuous variables were summarized as mean ± standard deviation and compared using the independent t-test or Mann–Whitney U-test as appropriate; categorical variables were expressed as frequencies and percentages and compared using the Chi-square or Fisher's exact test. The study population was divided into those with and without esophageal varices on endoscopy, and the PC/SD ratio was compared between groups. Receiver operating characteristic (ROC) curve analysis was performed to determine the area under the curve (AUC), sensitivity, specificity, positive and negative predictive values, and the optimal cut-off (by Youden's index) for the PC/SD ratio in predicting esophageal varices. One-way analysis of variance (ANOVA) was used to compare PC/SD ratio across variceal grades, and Spearman's rank correlation (ρ) was used to assess the relationship between the PC/SD ratio and other clinical and laboratory parameters. A two-tailed $P < 0.05$ was considered statistically significant throughout.

Results

Demographic and Clinical Profile

A total of 380 patients with cirrhosis were enrolled, comprising 350 (92.1%) males and 30 (7.9%) females, a male-to-female ratio of approximately 12:1 [Figure 1]. The mean age was 40.4 ± 10.9 years, with the largest proportion in the 31–40-year age group (154, 40.5%). The largest proportions of patients were illiterate (132, 34.7%) and skilled workers (170, 44.7%), followed

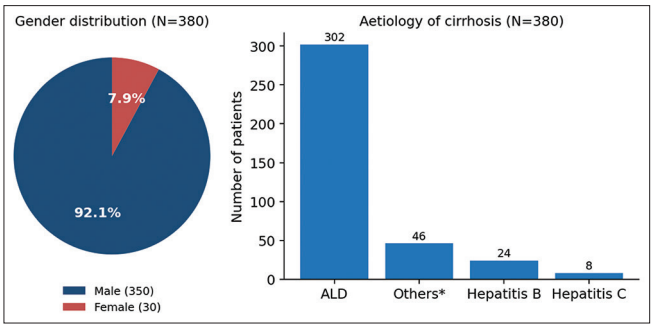


Figure 1: Gender distribution and etiology of cirrhosis among the 380 study patients

by the unemployed (72, 18.9%) and those engaged in business (57, 15.0%); the majority resided in urban areas (254, 66.8%), were Hindu by religion (320, 84.2%), and belonged to socio-economic Class III (137, 36.1%) or Class IV (126, 33.2%) [Table 1].

Alcoholic liver disease (ALD) was the predominant etiology overall (302, 79.5%), followed by other causes, including non-alcoholic fatty liver disease/steatohepatitis, primary biliary cholangitis, and cryptogenic cirrhosis (46, 12.1%), hepatitis B (24, 6.3%), and hepatitis C (8, 2.1%) [Table 2 and Figure 1]. Alcohol consumption was reported in 271 (71.3%) patients overall, with a marked gender disparity (76.9% in males vs. 6.7% in females; $P < 0.001$), consistent with ALD accounting for 82.9% of cases in males compared with 40.0% in females.

Weakness/fatigue (90.0%), abdominal distension (84.5%), and loss of appetite (73.7%) were the most common presenting complaints. On examination, edema (88.7%), pallor (83.7%), and jaundice (77.6%) were the most prevalent clinical signs, and splenomegaly was present in 354 (93.2%) patients and clinical ascites in 299 (78.7%). On general examination, most patients were conscious and oriented (358, 94.2%), while 22 (5.8%) were drowsy, possibly reflecting hepatic encephalopathy; blood pressure was within the normal range in 260 (68.4%) patients, with hypotension noted in 83 (21.8%) and hypertension in 37 (9.7%).

Mean hemoglobin was 9.8 ± 2.0 g/dL, mean platelet count 103.1 ± 43.3 $\times 10^3/\mu L$, mean total bilirubin 4.2 ± 2.9 mg/dL, and mean albumin 2.7 ± 0.8 g/dL, reflecting significant anemia, thrombocytopenia, and hepatic synthetic dysfunction. Coagulation studies revealed a prolonged mean prothrombin time of 20.6 ± 5.4 s and mean activated partial thromboplastin time of 42.4 ± 7.3 s. On ultrasonography, the mean splenic diameter was 15.9 ± 3.4 cm and mean portal vein diameter 14.2 ± 3.0 mm, with a coarse, heterogeneous hepatic echotexture as the predominant finding (282, 74.2%). The majority of patients were classified as Child-Pugh Class C (256, 67.4%), followed by Class B (121, 31.8%) and Class A (3, 0.8%), and Child-Pugh class was significantly associated with the presence of esophageal varices (Chi-square = 6.820, $P = 0.033$).

Esophageal Varices and the PC/SD Ratio

Esophageal varices were identified on endoscopy in 261 (68.7%) patients; Grade I was most common (118, 45.2%), followed by Grade II (86, 33.0%) and Grade III (57, 21.8%). The mean PC/SD ratio was significantly lower in patients with esophageal varices than in those without (5.4 ± 2.7 vs. 10.9 ± 3.9; $P < 0.001$) [Table 3 and Figure 2]. One-way ANOVA across the four groups (no varices and Grades I–III) was significant ($P < 0.001$); however, this

Table 1: Socio-demographic characteristics of the study population (n=380)

Parameter	*Frequency (n)*	*Percentage*
Male	350	92.1
Female	30	7.9
Age 18–30 years	61	16.1
Age 31–40 years	154	40.5
Age 41–50 years	86	22.6
Age 51–60 years	74	19.5
Age 61–70 years	5	1.3
Illiterate	132	34.7
Urban residence	254	66.8
Socio-economic Class III	137	36.1
Socio-economic Class IV	126	33.2

Table 2: Etiology of cirrhosis and association with esophageal varices (n=380)

Etiology	*n (%)*	*EV present, n (%)*
Alcoholic liver disease	302 (79.5)	202 (66.9)
Hepatitis B	24 (6.3)	19 (79.2)
Hepatitis C	8 (2.1)	4 (50.0)
Others*	46 (12.1)	36 (78.3)
Total	380 (100)	261 (68.7)

*Others include NAFLD/NASH (n=16), primary biliary cholangitis (n=6), and cryptogenic cirrhosis (n=24). NAFLD: Non-alcoholic fatty liver disease, NASH: Non-alcoholic steatohepatitis

Table 3: PC/SD ratio by presence and grade of esophageal varices

Group	*n*	*PC/SD ratio (mean±SD)*	*P-value*
Without EV	119	10.9±3.9	
With EV	261	5.4±2.7	<0.001
Grade I (small)	118	5.4±2.9	
Grade II (medium)	86	5.3±2.6	
Grade III (large)	57	5.4±2.4	<0.001 (ANOVA)

†One-way ANOVA across all four groups (no varices and Grades I–III). ANOVA: Analysis of variance, EV: Esophageal varices, PC/SD: Platelet count/splenic diameter

difference was attributable to the no-varices group, as the PC/SD ratio was virtually identical across Grades I, II, and III (5.4, 5.3, and 5.4, respectively) [Table 3 and Figure 2]. The PC/SD ratio did not differ significantly by gender (7.1 ± 4.0 in males vs. 7.3 ± 4.1 in females; $P = 0.782$) or by etiology of cirrhosis ($P = 0.412$) [Table 4], supporting its broad applicability.

Diagnostic Performance

On ROC curve analysis, the PC/SD ratio predicted the presence of esophageal varices with an AUC of 0.868 (95% confidence interval: 0.808–0.929; $P < 0.001$). At the Youden-optimal cut-off of ≤8.3, sensitivity was 90.4%, specificity 89.1%, positive predictive value 94.8%, negative predictive value 80.9%, and overall diagnostic accuracy 90.0% [Table 5 and Figure 3]. The platelet-to-lymphocyte ratio (PLR) also showed diagnostic value as a complementary marker, with an AUC of 0.745, though its performance was inferior to that of the PC/SD ratio.

Correlation Analysis

The PC/SD ratio correlated strongly and positively with platelet count ($r = +0.916$, $P < 0.001$) and strongly and negatively with splenic diameter ($r = -0.723$, $P < 0.001$), and showed weaker but statistically significant negative correlations with portal

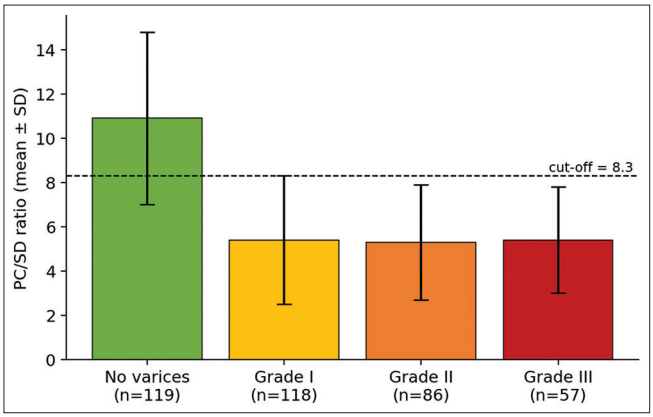


Figure 2: Platelet count/splenic diameter ratio by presence and grade of esophageal varices (error bars show standard deviation); dashed line marks the optimal cut-off (≤8.3)

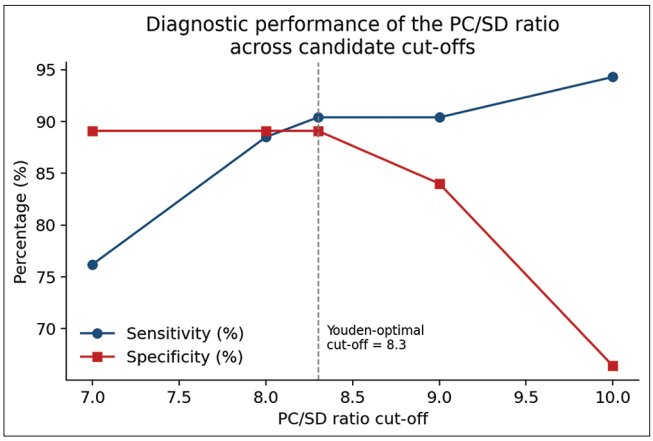


Figure 3: Sensitivity and specificity of the platelet count/splenic diameter ratio across candidate cut-off values

Table 4: PC/SD ratio by gender and etiology of cirrhosis

Subgroup	*Mean PC/SD ratio±SD*	*P-value*
Male	7.1±4.0	0.782
Female	7.3±4.1	
Alcoholic liver disease	6.9±3.8	
Hepatitis B	6.4±3.6	
Hepatitis C	8.2±4.5	
Others	6.4±3.4	0.412 (ANOVA)

ANOVA: Analysis of variance, PC/SD: Platelet count/splenic diameter

Table 5: Diagnostic performance of the PC/SD ratio at candidate cut-offs

Cut-off	*Sensitivity (%)*	*Specificity (%)*	*PPV (%)*	*NPV (%)*	*Accuracy (%)*
≤7.0	76.2	89.1	93.9	63.1	80.3
≤8.0	88.5	89.1	94.7	77.9	88.7
≤8.3*	90.4	89.1	94.8	80.9	90.0
≤9.0	90.4	84.0	92.5	80.0	88.4
≤10.0	94.3	66.4	86.0	84.0	85.5

*Youden-optimal cut-off (AUC 0.868, 95% CI: 0.808–0.929; *P*<0.001).
AUC: Area under the curve, CI: Confidence interval, PPV: Positive predictive value, NPV: Negative predictive value, PC/SD: Platelet count/splenic diameter

Table 6: Correlation of PC/SD ratio with clinical and laboratory parameters

Parameter	*Correlation (r)*	*P-value*
Platelet count	+0.916	<0.001*
Splenic diameter	−0.723	<0.001*
Portal vein diameter	−0.227	<0.001*
PLR	+0.589	<0.001*
Child-Pugh score	−0.109	0.033*
Total bilirubin	−0.031	0.553
Albumin	−0.009	0.859
Hemoglobin	−0.027	0.601
NLR	+0.039	0.443

*Statistically significant (P<0.05). ρ: Spearman's rank correlation coefficient.
PLR: Platelet-to-lymphocyte ratio, NLR: Neutrophil-to-lymphocyte ratio, PC/SD: Platelet count/splenic diameter

vein diameter (r = −0.227, $P < 0.001$) and Child-Pugh score (r = −0.109, $P = 0.033$), and a significant positive correlation with PLR (r = +0.589, $P < 0.001$). Correlations with total bilirubin, albumin, hemoglobin, and neutrophil-to-lymphocyte ratio were not statistically significant, suggesting the PC/SD ratio is driven predominantly by portal hemodynamic changes rather than hepatocellular synthetic function [Table 6].

Discussion

This cross-sectional study of 380 patients with cirrhosis demonstrates that the PC/SD ratio is a robust, inexpensive, non-invasive marker for the presence of esophageal varices, with an AUC of 0.868 and sensitivity and specificity of 90.4% and 89.1% respectively at a cut-off of ≤8.3. Thrombocytopenia in cirrhosis is multifactorial, arising from hypersplenism with splenic sequestration of platelets, reduced hepatic thrombopoietin production, and bone marrow suppression, while splenomegaly reflects chronic portal hypertension; normalizing platelet count to splenic diameter is therefore conceptually attractive since platelet count alone can be misleading and is not solely attributable to portal hypertension.[4]

The marked male predominance observed in this cohort (92.1%; M:F ratio ≈12:1) and mean age of 40.4 ± 10.9 years, concentrated in the 31–40-year age group, are consistent with Kothari *et al*., who studied patients with alcoholic cirrhosis from central India with a mean age of 43.8 ± 10.0 years,[8] and with Mujahid *et al*.[9] and Rangare *et al*.,[10] both of whom reported similar male-predominant, working-age cohorts from Indian tertiary centers. This pattern reflects the higher prevalence of alcohol use disorder among men in India, consistent with ALD being the predominant etiology in this study (79.5%) and alcohol consumption being significantly more common in males than females (76.9% vs. 6.7%; $P < 0.001$).

ALD accounted for the majority of cases in this study, consistent with Kothari *et al*.'s alcoholic-cirrhosis-predominant cohort from central India,[8] and with the broader epidemiological pattern in which chronic alcohol consumption drives progression to cirrhosis through steatosis, inflammation, and fibrosis.[11] Weakness/fatigue, abdominal distension, and loss of appetite were the leading presenting complaints, and splenomegaly and ascites were the most prevalent examination findings, in keeping with prior reports linking splenomegaly and thrombocytopenia to variceal risk, including Chalasani *et al*., who showed that patients with a platelet count of 88,000/mm^3 or more and no splenomegaly had a low risk of large varices, whereas those with splenomegaly or a lower platelet count had a substantially higher risk.[12]

The prevalence of esophageal varices in this cohort (68.7%) is comparable to that reported by Kothari *et al*.,[8] Rangare *et al*. (75%),[10] Mujahid *et al*. (70%),[9] and the original description by Giannini *et al*. (61%),[4] and lies within the 60–80% range described in other Indian series. The mean PC/SD ratio was markedly lower in patients with esophageal varices than in those without (5.4 ± 2.7 vs. 10.9 ± 3.9; $P < 0.001$), and the diagnostic performance observed here (AUC 0.868, sensitivity 90.4%, specificity 89.1% at a cut-off of ≤8.3) is concordant with the original description by Giannini *et al*., who proposed a cut-off of 909 (platelet count per mm^3, splenic diameter in mm) with a sensitivity of 100% and specificity of 93% in 145 cirrhotic patients, and identified the PC/SD ratio as the only parameter independently associated with esophageal varices on multivariate analysis.[4] This was subsequently validated in a multicenter study of 218 patients by the same group, in which the 909 cut-off yielded a sensitivity of 91.5% and specificity of 67.0%.[13] The apparent numerical difference between our cut-off (≤8.3) and the original 909 largely reflects the difference in units used for PC/SD (platelet count $\times 10^3/mm^3$ per cm of splenic diameter here, versus platelet count per mm3 per mm in the original description); when rescaled, our cut-off corresponds to approximately 830 on the Giannini scale, modestly lower than the original threshold.. A systematic review and meta-analysis of 49 studies by Chen *et al*. reported a pooled area under the summary ROC curve of 0.872 for the diagnosis of varices of any grade, closely concordant with our finding,[6] and comparable diagnostic performance has been reported by Mossie *et al*. in an Ethiopian cohort (AUC 0.835)[14] and by Qi *et al*. in patients with hepatitis-B-related cirrhosis (AUC 0.865).[15]

The prevalence of esophageal varices also varied by etiology in this cohort, being highest with hepatitis B (79.2%), followed by other causes (78.3%) and ALD (66.9%), while hepatitis C showed a lower rate (50.0%), although in a small subgroup ($n = 8$); this pattern suggests that variceal development is driven predominantly by the severity of portal hypertension rather than the specific underlying etiology, consistent with the broadly similar 60–80% prevalence range reported across the Indian studies cited above irrespective of the dominant etiology in each cohort. Natarajan *et al*., in an evaluation of 50 cirrhotic patients, similarly found that patients with varices had a lower mean PC/SD ratio than those without, using an optimized cut-off of approximately 870,[16] while Qi *et al*., studying 139 patients with hepatitis-B-related cirrhosis, reported an AUC of 0.865 with an optimal threshold of 1013.2 achieving 88.5% sensitivity and 69.2% specificity, and identified the PC/SD ratio as an independent predictor of varices on multivariate analysis (odds ratio 3.569, $P < 0.001$).[15] These findings, together with ours, support the PC/SD ratio as a reproducible predictor across a range of populations, cut-off values, and cirrhosis etiologies, even though the precise optimal threshold appears to vary modestly with local population characteristics and measurement units.

Although the PC/SD ratio clearly distinguished patients with and without varices, it did not differ meaningfully between variceal grades in this cohort, indicating that the ratio is better suited to detecting the presence of varices than to estimating their size. Amin et al. likewise reported significantly lower PC/SD ratios in patients with varices due to hepatitis-C-related cirrhosis,[17] and Baig et al. found the PC/SD ratio had the highest accuracy among non-invasive parameters in an Indian cohort of 150 cirrhotic patients.[18] In contrast, El-Sherbiny et al. reported a significant negative correlation between the PC/SD ratio and variceal grade in patients with hepatitis-C-related cirrhosis,[19] a gradient not observed in the present cohort, possibly reflecting its predominantly decompensated, alcohol-related population. The strong positive correlation with platelet count ($\rho = +0.916$) and negative correlation with splenic diameter ($\rho = -0.723$) driving the ratio downward. This is in agreement with El-Sherbiny *et al*., who reported a significant negative correlation between the PC/SD ratio and variceal grade in patients with hepatitis-C-related cirrhosis.[19]

Importantly, the PC/SD ratio performed consistently across genders ($P = 0.782$) and etiologies ($P = 0.412$) in this study, mirroring the multicenter validation by Giannini *et al*., in which sensitivity exceeded 80% across etiological subgroups,[13] and supporting its use as a broadly applicable screening tool irrespective of the underlying cause of cirrhosis or patient sex, notwithstanding the relatively small female subgroup in this cohort ($n = 30$). Similarly, Xu *et al*. validated the PC/SD ratio specifically in patients with schistosomiasis-related cirrhosis, further supporting its applicability across diverse etiologies.[20]

Given that platelet count and abdominal ultrasonography are already obtained as part of the routine work-up of cirrhotic patients, the PC/SD ratio can be calculated at essentially no additional cost, making it particularly attractive as a triage tool in resource-limited settings where endoscopic capacity is constrained. Identifying patients with a low pre-test probability of varices, defined here as a PC/SD ratio above 8.3, could allow endoscopy to be prioritized for higher-risk patients, potentially reducing the burden on endoscopy services. However, with a negative predictive value of 80.9%, approximately one in five patients above this threshold still had varices, so the ratio should be used to prioritize rather than replace endoscopy. The combined use of the PC/SD ratio alongside PLR and Child-Pugh scoring may offer a more comprehensive, still entirely non-invasive, approach to risk-stratifying cirrhotic patients before endoscopy is considered.

Limitations

This study has certain limitations. It was conducted at a single tertiary care center, and the findings may not be generalizable to the broader population or to peripheral healthcare settings; the referral nature of the center likely accounts for the predominance of decompensated (Child-Pugh Class C) patients in this cohort. The cross-sectional design does not allow assessment of temporal relationships or follow-up of variceal progression or bleeding risk over time. The female subgroup was small ($n = 30$), limiting the power of gender-specific analyses, and the predominantly alcoholic etiology of this cohort may limit applicability to populations with a different etiological mix. The optimal cut-off was derived and evaluated within the same cohort without external validation in an independent population, and splenic diameter measurement, though performed by an experienced radiologist blinded to clinical and endoscopic findings, remains subject to inter-observer variability that could affect reproducibility. The ratio did not discriminate between variceal grades, so its ability to identify large (high-risk) varices specifically was not established. Finally, this study did not compare the PC/SD ratio against newer non-invasive modalities such as transient elastography or spleen stiffness measurement.

Conclusion

In this observational study of 380 patients with cirrhosis, the PC/SD ratio was significantly lower in patients with esophageal varices

than in those without, although it did not differ between variceal grades. At a cut-off of ≤8.3, the ratio predicted the presence of esophageal varices with 90.4% sensitivity and 89.1% specificity, and performed consistently across genders and etiologies of cirrhosis. Given that platelet count and splenic ultrasonography are already obtained as part of the routine evaluation of cirrhotic patients, the PC/SD ratio represents a simple, inexpensive, and reliable non-invasive tool that could help triage patients for screening endoscopy, reduce the burden on endoscopy services, and improve the cost-effectiveness of variceal screening, particularly in resource-limited settings. External validation in independent, multicenter cohorts, ideally incorporating comparison with newer modalities such as transient elastography and spleen stiffness measurement, is warranted before the ratio can be recommended for routine clinical use in place of endoscopic screening.

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