



PROGNOSTIC SIGNIFICANCE OF NEUTROPHIL-TO-LYMPHOCYTE RATIO AND PLATELET-TO-LYMPHOCYTE RATIO IN CHRONIC LIVER DISEASE

General Medicine

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ABSTRACT

Background: Neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are inexpensive indices from a routine complete blood count that may reflect systemic inflammation and portal-hypertension-related haematological change in chronic liver disease. This study evaluated NLR and PLR in cirrhosis, focusing on their association with oesophageal varices (EV) and Child–Pugh severity. **Methods:** This cross-sectional study included 380 adults with cirrhosis undergoing screening endoscopy. NLR and PLR were correlated with EV status, EV grade, and Child–Pugh class. **Results:** EV were present in 261/380 (68.7%) patients. Mean NLR did not differ between patients with and without EV (2.95 ± 0.67 vs 3.04 ± 0.63 ; $p=0.227$). PLR, however, was significantly lower with EV (79.8 ± 51.6 vs 133.1 ± 76.4 ; $p<0.001$), with an AUC of 0.745 and 73.6% sensitivity, 69.7% specificity at a cut-off of ≤ 97 . PLR correlated positively with platelet count ($r=+0.640$) and PC/SD ratio ($r=+0.589$); both NLR and PLR showed weak inverse correlation with Child–Pugh score ($r=-0.101$ and -0.102 , both $p=0.048$). **Conclusion:** PLR, but not NLR, was significantly associated with EV presence and may serve as a simple, inexpensive adjunctive marker of portal-hypertension-related disease burden. Because the study was cross-sectional, these associations reflect disease severity and variceal risk rather than proven long-term prognosis.

KEYWORDS

Chronic Liver Disease; Cirrhosis; Oesophageal Varices; Neutrophil-to-lymphocyte Ratio; Platelet-to-lymphocyte Ratio

INTRODUCTION

Cirrhosis is the end stage of chronic liver injury — fibrosis, regenerative nodules, and a hepatic architecture that no longer works the way it should. What actually determines a patient's course, though, is not just how badly the liver itself has failed. Portal hypertension, systemic inflammation, immune dysregulation, and complications such as ascites, encephalopathy, and gastroesophageal varices all shape outcomes just as much. Variceal rupture in particular can kill quickly, through massive upper gastrointestinal haemorrhage, which is why picking up varices early remains a central part of managing these patients. Globally, the burden of cirrhosis keeps rising, and it falls hardest on low- and middle-income settings, where specialised hepatology services, elastography, and endoscopy are often simply not available — a gap that makes cheap, point-of-care risk-stratification tools genuinely useful rather than a nice-to-have.¹

That gap is part of why the search for cheap, reproducible non-invasive markers of clinically significant portal hypertension has moved well past the usual liver-function tests. Platelet count is a familiar example — it drops in cirrhosis because of splenic sequestration, hypersplenism, and falling thrombopoietin production. Lymphocyte and neutrophil counts shift too, but for a messier set of reasons: systemic inflammation, immune dysfunction, occult infection, physiological stress, and the disease itself as it advances.

The neutrophil-to-lymphocyte ratio (NLR) — neutrophils divided by lymphocytes — is a well-established marker of the balance between innate and adaptive immune activity, used across hepatic, oncological, and cardiovascular disease as a cheap surrogate for systemic inflammatory burden. The platelet-to-lymphocyte ratio (PLR) combines platelet and lymphocyte behaviour, so in principle it should pick up both inflammatory/immune change and the haematological fallout of portal hypertension. Both come from a routine complete blood count, which is exactly why they appeal in resource-constrained settings where dedicated portal-pressure measurement, elastography, or repeat endoscopy are not always an option. Outside hepatology, both ratios already have a substantial track record — elevated NLR and altered PLR have been linked to worse outcomes in oncology, cardiovascular disease, and critical care — so their extension into chronic liver disease is a fairly natural next step. What is less well worked out is how they actually behave in the specific setting of portal hypertension and cirrhosis-related cytopenias, compared with a dedicated hepatic index such as the platelet count/splenic diameter (PC/SD) ratio.

The data reported here come from an institutional dissertation that enrolled 380 patients with cirrhosis at L.N. Medical College &

Research Centre and J.K. Hospital, Bhopal. That original work centred on the PC/SD ratio as a non-invasive predictor of oesophageal varices, but the same dataset also captured NLR and PLR and their clinical correlations. This manuscript takes that same cohort, the same measurements, and the same statistical results, and re-frames the analysis specifically around what NLR and PLR add.

Aim and Objectives

To assess the association of NLR and PLR with the presence of oesophageal varices in adults with cirrhosis; to compare NLR and PLR across grades of oesophageal varices; to examine the relationship of NLR and PLR with Child–Pugh class and other portal-hypertension-related variables; and to assess the diagnostic discrimination of PLR for the presence of oesophageal varices.

Methodology

Study Design and Setting

This was a single-centre, hospital-based, cross-sectional observational study conducted in the Departments of General Medicine and Gastroenterology, L.N. Medical College & Research Centre and J.K. Hospital, Bhopal, a tertiary care teaching hospital. The study period spanned 18 months, comprising protocol and ethics preparation, 12 months of patient recruitment and data collection, and a subsequent period of analysis and report writing. Ethical clearance was obtained from the Institutional Ethics Committee prior to commencement, and the study was conducted in accordance with the principles of the Declaration of Helsinki, with written informed consent obtained from all participants.

Participants

Adults of either sex with established cirrhosis who were undergoing screening endoscopy for oesophageal varices were eligible for inclusion. Inclusion criteria comprised age greater than 18 years and screening endoscopy either at diagnosis or in patients who had never previously undergone screening endoscopy. Patients with active upper gastrointestinal bleeding, a previous history of endoscopic variceal therapy, prior portal-hypertension surgery or transjugular intrahepatic portosystemic shunt, previous beta-blocker prophylaxis, inability to abstain from alcohol, or refusal of consent were excluded.

Clinical and Laboratory Assessment

Detailed clinical history and examination were performed for all participants, including assessment for jaundice, pallor, clubbing, oedema, ascites, hepatomegaly, and splenomegaly. Disease severity was assessed using the Child–Pugh classification. A 5 mL venous blood sample was obtained under aseptic precautions for a complete blood count and routine laboratory evaluation, and platelet, neutrophil,

and lymphocyte counts were recorded from the automated haematology analyser. NLR was calculated as the absolute neutrophil count divided by the absolute lymphocyte count, and PLR as the platelet count divided by the absolute lymphocyte count. Platelet counts were expressed as $\times 10^3/\mu\text{L}$ in the source dataset, and ratios were calculated using the corresponding cell counts.

Ultrasonography and Endoscopy

Abdominal ultrasonography was used to assess splenic diameter and portal venous parameters. Upper gastrointestinal endoscopy was performed by an experienced gastroenterologist after appropriate fasting and served as the reference assessment for the presence and grade of oesophageal varices. Varices were categorised as Grade I (small), Grade II (medium), or Grade III (large), consistent with the classification used in the source dissertation.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using SPSS version 27. Continuous variables were summarised using mean $\pm$ standard deviation; categorical variables were presented as frequencies and percentages. Between-group comparisons used independent-samples testing or the Mann-Whitney U test as appropriate, and categorical variables were assessed with the Chi-square or Fisher's exact test. Receiver operating characteristic (ROC) curve analysis was performed for PLR, with the optimal cut-off selected using Youden's index. Spearman correlation was used to assess relationships between the ratios and clinical variables. A two-sided p-value <0.05 was considered statistically significant throughout.

Analytic Qualification

The source study is cross-sectional and did not include longitudinal mortality, transplant-free survival, or time-to-decompensation follow-up. The term prognostic significance in this manuscript therefore refers to association with disease severity and clinically relevant variceal status, and should not be interpreted as evidence that NLR or PLR independently predicts future mortality or decompensation.

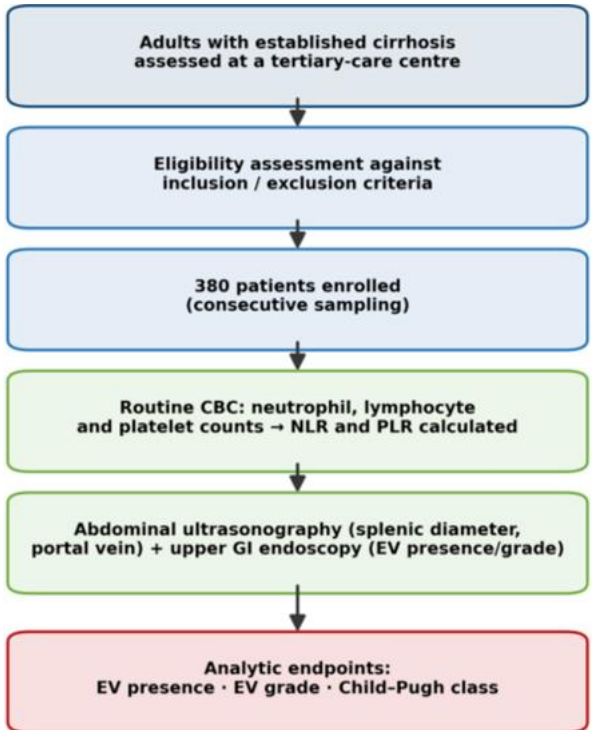


Figure 1. Study Workflow: Patient Selection, Index-Test Measurement, and Reference-Standard Assessment.

RESULTS

Baseline Characteristics

The study included 380 patients with cirrhosis: 350 men (92.1%) and 30 women (7.9%), with a mean age of 40.4 ± 10.9 years. The largest age group was 31 to 40 years (154 patients, 40.5%). Alcohol-related liver disease was the dominant aetiology (302 patients, 79.5%), followed by hepatitis B (24, 6.3%), hepatitis C (8, 2.1%), and other aetiologies including non-alcoholic fatty liver disease/steatohepatitis, primary biliary cholangitis, and cryptogenic cirrhosis (46, 12.1%). The cohort had features of advanced liver disease: mean platelet count was

$103.1 \pm 43.3 \times 10^3/\mu\text{L}$, mean splenic diameter was 15.9 ± 3.4 cm, and mean Child–Pugh score was 10.2 ± 1.6 . Child–Pugh class C accounted for 256 patients (67.4%) and class B for 121 (31.8%); only three patients (0.8%) were class A. Oesophageal varices were detected in 261 patients (68.7%). Mean NLR for the cohort overall was 2.98 ± 0.66 , and mean PLR was 96.5 ± 65.2 (Table 1).

Table 1. Baseline Characteristics of the Study Population (N = 380)

Variable	Finding
Male sex	350 (92.1%)
Mean age	40.4 $\pm$ 10.9 years
Alcohol-related liver disease	302 (79.5%)
Mean platelet count	103.1 $\pm$ 43.3 $\times 10^3/\mu\text{L}$
Mean NLR	2.98 $\pm$ 0.66
Mean PLR	96.5 $\pm$ 65.2
Mean Child–Pugh score	10.2 $\pm$ 1.6
Child–Pugh class C	256 (67.4%)
Oesophageal varices present	261 (68.7%)

NLR and PLR According to Oesophageal-varix Status

Mean NLR was 2.95 ± 0.67 among patients with oesophageal varices and 3.04 ± 0.63 among those without, a difference that was not statistically significant ($p=0.227$), indicating limited discrimination of NLR for the presence of varices in this cohort. PLR showed a substantially different pattern: patients with varices had a mean PLR of 79.8 ± 51.6 , compared with 133.1 ± 76.4 among patients without varices ($p<0.001$) (Table 2, Figure 2). Thus, a lower PLR was strongly associated with the presence of oesophageal varices, while NLR showed no such association.

Table 2. NLR and PLR According to Oesophageal-Varix Status

Marker	With EV (n=261)	Without EV (n=119)	p-value
NLR	2.95 $\pm$ 0.67	3.04 $\pm$ 0.63	0.227
PLR	79.8 $\pm$ 51.6	133.1 $\pm$ 76.4	<0.001

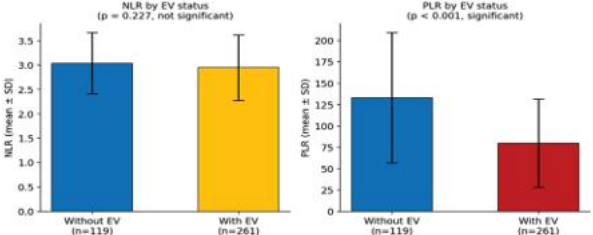


Figure 2. NLR and PLR According to Oesophageal-varix Status (Error Bars Show SD).

NLR and PLR Across Variceal Grades

Across endoscopic categories, NLR values remained relatively stable: 3.04 ± 0.63 in patients without varices, 2.94 ± 0.67 with Grade I, 3.02 ± 0.67 with Grade II, and 2.85 ± 0.68 with Grade III varices, with no clear monotonic relationship observed. PLR, in contrast, was highest in patients without varices (133.1 ± 76.4) and was lower across all variceal grades: Grade I (80.3 ± 57.9), Grade II (82.2 ± 51.7), and Grade III (75.1 ± 35.5) (Table 3, Figure 3). This pattern indicates that PLR primarily discriminated patients with versus without oesophageal varices, rather than reliably separating individual variceal grades from one another.

Table 3. NLR and PLR Across Endoscopic Variceal Categories

Endoscopic Category	n	NLR (mean $\pm$ SD)	PLR (mean $\pm$ SD)
No varices	119	3.04 $\pm$ 0.63	133.1 $\pm$ 76.4
Grade I	118	2.94 $\pm$ 0.67	80.3 $\pm$ 57.9
Grade II	86	3.02 $\pm$ 0.67	82.2 $\pm$ 51.7
Grade III	57	2.85 $\pm$ 0.68	75.1 $\pm$ 35.5

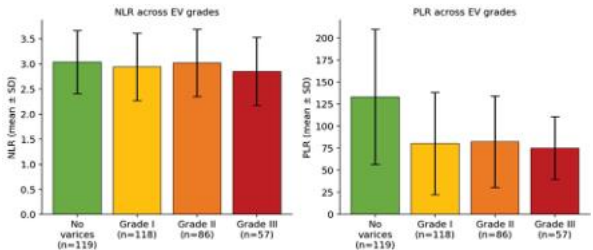


Figure 3. NLR and PLR Across Endoscopic Variceal Grades (Error Bars Show SD).

Relationship with Child–Pugh Class

NLR decreased from 3.39 ± 0.89 in Child–Pugh class A to 3.06 ± 0.68 in class B and 2.93 ± 0.65 in class C; the comparison between classes B and C was borderline but not statistically significant (p=0.067). PLR was 86.0 ± 22.4 in class A, 106.1 ± 59.8 in class B, and 92.1 ± 67.6 in class C, with the class B versus C comparison also borderline (p=0.052) (Table 4, Figure 4).

Table 4. NLR and PLR by Child–Pugh Class

Child–Pugh Class	n	NLR (mean ± SD)	PLR (mean ± SD)
Class A	3	3.39 ± 0.89	86.0 ± 22.4
Class B	121	3.06 ± 0.68	106.1 ± 59.8
Class C	256	2.93 ± 0.65	92.1 ± 67.6

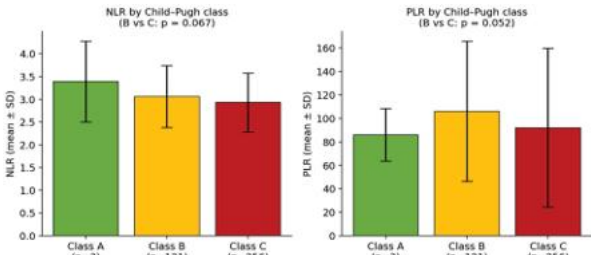


Figure 4. NLR and PLR by Child–Pugh Class (Error Bars Show SD).

Correlation Analysis

NLR demonstrated a weak inverse correlation with Child–Pugh score (r=−0.101, p=0.048), but no significant correlation with platelet count (r=+0.021, p=0.690), splenic diameter (r=−0.033, p=0.519), total bilirubin (r=−0.094, p=0.068), albumin (r=+0.069, p=0.180), or prothrombin time (r=−0.015, p=0.778). PLR, in contrast, correlated positively with platelet count (r=+0.640, p<0.001) and with the PC/SD ratio (r=+0.589, p<0.001), and negatively with splenic diameter (r=−0.308, p<0.001). PLR also showed a weak inverse correlation with Child–Pugh score (r=−0.102, p=0.048), similar in magnitude to that of NLR. NLR and PLR were modestly correlated with one another (r=+0.265, p<0.001) (Table 5, Figure 5).

Table 5. Correlation of NLR and PLR with Clinical and Laboratory Parameters

Variable	NLR: r (p)	PLR: r (p)
Child–Pugh score	−0.101 (0.048)	−0.102 (0.048)
PC/SD ratio	+0.039 (0.443)	+0.589 (<0.001)
Platelet count	+0.021 (0.690)	+0.640 (<0.001)
Splenic diameter	−0.033 (0.519)	−0.308 (<0.001)
Total bilirubin	−0.094 (0.068)	−0.049 (0.340)
Albumin	+0.069 (0.180)	−0.041 (0.428)
Prothrombin time	−0.015 (0.778)	−0.009 (0.857)
NLR vs PLR	+0.265 (<0.001)	—

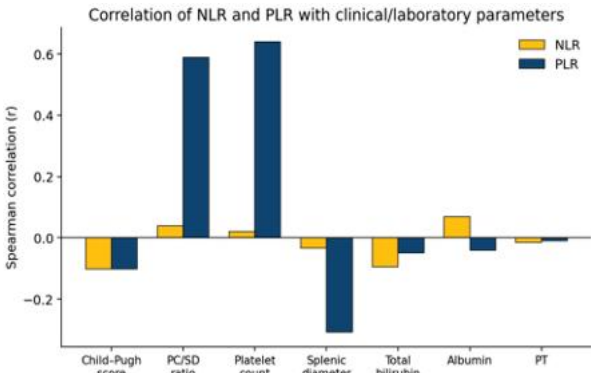


Figure 5. Correlation of NLR and PLR with Clinical and Laboratory Parameters.

Diagnostic Performance of PLR

On ROC curve analysis, PLR predicted the presence of oesophageal varices with an area under the curve of 0.745 (95% CI 0.667–0.823; p<0.001). The optimal cut-off identified by Youden's index was ≤97, providing 73.6% sensitivity and 69.7% specificity. At candidate thresholds of 80, 90, 97, 100, and 120, sensitivity ranged from 59.8%

to 84.7% and specificity ranged from 75.6% to 47.9%, illustrating the expected trade-off between sensitivity and specificity as the cut-off was relaxed (Table 6, Figure 6). Current international guidance, including the Baveno VII consensus and the 2024 AASLD Practice Guidance on portal hypertension, emphasises non-invasive risk stratification using platelet count and liver stiffness measurement to select patients who may safely avoid screening endoscopy;^{2,3} the present findings on PLR are broadly complementary to this direction of travel, since PLR is derived from parameters that are even more universally available than liver stiffness measurement in many primary and secondary care settings.

Table 6. Diagnostic Performance of PLR at Candidate Cut-offs

PLR cut-off	Sensitivity	Specificity	PPV	NPV	Accuracy
≤80	59.8%	75.6%	84.3%	46.2%	64.7%
≤90	68.6%	72.3%	84.4%	51.2%	69.7%
≤97*	73.6%	69.7%	84.2%	54.6%	72.4%
≤100	76.2%	63.9%	82.2%	55.1%	72.4%
≤120	84.7%	47.9%	78.1%	58.8%	73.2%

*Youden-optimal cut-off (AUC 0.745, 95% CI: 0.667–0.823; p<0.001).

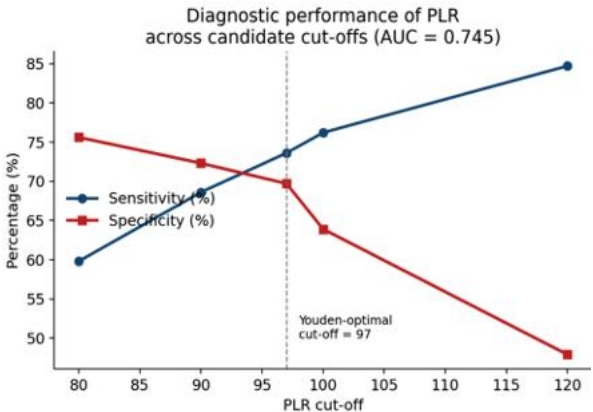


Figure 6. Diagnostic Performance (Sensitivity and Specificity) of PLR Across Candidate Cut-off Values.

DISCUSSION

The headline result here is simple: PLR tracked the presence of oesophageal varices, NLR did not. The gap was not subtle — mean PLR was roughly 40% lower in patients with varices than in those without. That makes physiological sense. Platelet count is heavily shaped by portal hypertension itself, through splenic sequestration and falling hepatic thrombopoietin, whereas lymphocyte counts answer to a different set of pressures — the broader immune disturbance of advanced cirrhosis — that has little to do with portal pressure specifically.

That does not mean inflammation is absent in these patients — it almost certainly is not. NLR is just a ratio, and its numerator and denominator can move in opposite directions at once in advanced liver disease. Inflammation pushes neutrophils up; hypersplenism, marrow suppression, infection, poor nutrition, and immune dysfunction can pull both neutrophils and lymphocytes around at the same time. In a cohort this decompensated, those competing pulls probably cancel out often enough that a single NLR value loses its ability to separate patients with varices from those without, even while the underlying inflammatory state is real.

PLR told a different story, because platelet depletion tracks portal hypertension so closely — through splenic sequestration and reduced thrombopoietin synthesis. The correlations back this up: positive with platelet count (r=+0.640), negative with splenic diameter (r=−0.308). PLR's positive correlation with the PC/SD ratio (r=+0.589) points the same way — it is capturing a slice of the same portal-hypertension biology that the established platelet/spleen metric measures, though PLR also brings lymphocyte count into the picture, so the two markers are not identical. This fits with the wider literature on non-invasive markers in cirrhosis. Chalasani et al. found that a platelet count of 88,000/mm³ or higher, combined with no splenomegaly, marked out patients at low risk of large varices — platelet-based indices keep coming up as central to variceal risk assessment.⁴ Kothari et al., in an alcohol-predominant Indian cohort, and Kumar et al., in a smaller

cross-sectional series, both found platelet count to be one of the few haematological parameters consistently tied to variceal presence or size, while other routine biochemistry fell short — much the same split seen here between platelet-driven PLR and platelet-independent NLR.^{5,6} Amin et al. reported similar platelet-based discrimination in hepatitis-C-related cirrhosis, and Xu et al. extended the same platelet-to-spleen-diameter logic to schistosomiasis-related cirrhosis — together suggesting platelet-driven markers hold up across quite different aetiologies, which lines up with the lack of an aetiology effect on the PC/SD ratio in the source dissertation.^{7,8}

One thing worth flagging about this dataset: it is skewed toward advanced disease — 67.4% Child–Pugh class C, 68.7% with varices already present. Mossie et al., in a comparable Ethiopian cohort, found much the same thing — platelet-based ratios perform better where disease burden and variceal prevalence are already high — and Chen et al.'s meta-analysis of 49 studies on platelet-to-spleen-diameter methodology flagged substantial heterogeneity between studies, largely driven by differences in case mix, aetiology, and disease stage.^{9,10} It is likely that this tertiary-care case mix is part of why PLR's association with variceal status came out as strong as it did, but the same skew limits how far the findings can be pushed toward compensated cirrhosis, where platelet counts and lymphocyte profiles look different. Combined with the heavy alcohol predominance (79.5%) and male skew (92.1%) of the cohort, that argues for external validation in more varied populations before leaning on these numbers too hard. The alcohol predominance itself is not surprising — it mirrors the wider pattern in central India, where chronic alcohol use remains the leading driver of cirrhosis through steatosis, inflammatory injury, and progressive fibrosis.¹¹ But the inflammatory and immune signature of alcohol-driven disease may not generalise cleanly to viral, autoimmune, or metabolic-associated cirrhosis, which is one more reason to treat the NLR findings here cautiously until they have been checked in more aetiologically mixed cohorts.

An AUC of 0.745 is moderate, not excellent — enough to be useful, not enough to stand in for endoscopy on its own. The same source dissertation found the PC/SD ratio performed noticeably better for the same outcome (AUC 0.868, 95% CI 0.808–0.929), which says that adding splenic size to platelet-based information buys real discriminatory power that PLR alone does not have. Even so, PLR keeps a practical edge: it needs nothing beyond a routine complete blood count, so it works in places where ultrasonography or endoscopy simply are not available — a reasonable first pass before anything more resource-intensive.

One word needs care here: prognostic. Because the design is cross-sectional, what the data actually show is an association with varices and Child–Pugh status that already exist at the time of testing — not a prediction of what happens next. Proving genuine prognostic value would need a prospective study, serial measurements, and real clinical endpoints: bleeding, decompensation, transplant-free survival, death. Until that exists, these findings are best read as a snapshot of disease severity, not a forecast. The distinction is not just semantic. Flagging patients who already have varices is useful for triage; predicting who will bleed or die over the next year or two is a different and much bigger claim, and the two should not get blurred together when these results are discussed with clinicians or patients.

In practice, PLR costs nothing extra — it comes straight off a complete blood count that most cirrhotic patients already have. A PLR of 97 or below picked up varices with 73.6% sensitivity and 69.7% specificity in this cohort, which is good enough to help flag patients who deserve closer attention or earlier endoscopy, particularly where endoscopy capacity is tight. NLR, on this data, does not clear that bar — it should not be used on its own to screen for varices, even though it may still earn its keep in other settings such as infection or malignancy. A sensible next step would be testing whether PLR adds anything once combined with platelet count, splenic size or the PC/SD ratio, and Child–Pugh class, rather than relying on any single marker alone. Between the two ratios examined here, PLR is clearly the more useful one for portal hypertension and variceal risk; NLR, despite its track record as a general inflammatory marker elsewhere, did not pull its weight for oesophageal varices in this cohort.

Limitations

A few limitations need to be stated plainly. The cross-sectional design rules out any real assessment of change over time or genuine long-term prognosis, and outcome data — bleeding, decompensation, mortality

— simply were not collected. Being single-centre and tertiary-care brings the usual referral and spectrum bias, and the cohort skews heavily toward decompensated disease (67.4% Child–Pugh class C), which limits how far these results travel to compensated cirrhosis. Women made up only 7.9% of the sample, so sex-specific conclusions carry little weight, and with alcohol-related disease at 79.5% of cases, performance may look different in cohorts dominated by viral, metabolic, or autoimmune cirrhosis. The PLR cut-off reported here was derived and tested in the same cohort, with no external validation. And finally, this manuscript works from the aggregate results in the source dissertation rather than re-created patient-level data; those aggregate numbers were not independently re-verified against the original case records as part of this specific re-analysis.

CONCLUSION

Across this cohort of 380 patients with cirrhosis, PLR came out clearly more useful than NLR for identifying oesophageal varices — significantly lower in patients with varices, an AUC of 0.745, and a correlation pattern with platelet count, splenic diameter, and the PC/SD ratio that all points the same direction. NLR did not discriminate variceal status and showed no real pattern across variceal grade, though both ratios shared a similarly weak inverse link with Child–Pugh score. What this supports is PLR as a simple, cheap, adjunctive marker of portal-hypertension-related disease burden, readily available from a routine blood count. What it does not yet support is calling either ratio a validated long-term prognostic biomarker — that needs longitudinal data on bleeding, decompensation, and mortality, and the PLR cut-off proposed here needs testing in cohorts beyond this one before it belongs in routine clinical decision-making.

Conflict of Interest

The authors declare no potential conflicts of interest with respect to research, authorship and/or publication of this article.

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Data Availability and Source Integrity

This manuscript is based on the aggregate statistical results contained in the institutional dissertation from which it is derived; individual-level patient data were not reproduced or fabricated for this manuscript. Figures are visualisations of the aggregate values reported in the dissertation. Because the study is cross-sectional, claims of independent long-term prognosis would require additional follow-up data that are not present in the source dataset.

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