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ORIGINAL RESEARCH ARTICLE

Association Between Platelet-to-Lymphocyte Ratio and Disease Severity in Patients with Acute Pancreatitis

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Abstract: Background: Platelet-to-lymphocyte ratio (PLR) is an inexpensive inflammatory marker proposed for early severity assessment in acute pancreatitis. This study evaluated the association between admission PLR and disease severity and compared its discriminatory performance with BISAP.**Methods:** A total of 200 adults with acute pancreatitis were classified according to the Revised Atlanta criteria. Admission PLR was compared between mild and moderately severe/severe disease. Multivariable logistic regression adjusted for age, biliary etiology, and blood urea nitrogen (BUN). Receiver operating characteristic analysis compared PLR with BISAP.**Results:** Mild, moderately severe, and severe pancreatitis occurred in 157 (78.5%), 32 (16.0%), and 11 (5.5%) patients, respectively. PLR did not differ significantly between mild and higher-severity disease and was not independently associated with severity (adjusted OR per doubling 0.74, 95% CI 0.40–1.37). PLR showed poor discrimination (AUC 0.443), whereas BISAP performed better (AUC 0.668). BUN was strongly associated with higher severity. PLR was also unrelated to ICU admission, organ failure, necrosis, or length of stay.**Conclusion:** Admission PLR showed limited value as a stand-alone marker of acute pancreatitis severity.

Keywords: acute pancreatitis; platelet-to-lymphocyte ratio; BISAP; disease severity; blood urea nitrogen

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Introduction

Acute pancreatitis is an inflammatory condition that can have a very variable clinical course, from a self-limited disease to pancreatic necrosis, persistent organ failure and death. Early identification of those patients who are likely to develop complicated disease is important as deterioration can occur during the first days of admission and may require intensified monitoring and supportive management. The Revised Atlanta classification defines severity as mild, moderately severe or severe based on local complications and organ failure (present or not and duration) [1].

There are several clinical scoring systems which have been developed for early risk stratification. The Bedside Index for Severity in Acute Pancreatitis (BISAP) is particularly appealing because it is based on five readily available variables obtained within the first 24 hours and has proven to be a useful discriminator for mortality, organ failure and pancreatic necrosis [2]. It has also been proven to be a good prognostic tool in Indian populations, but still requires several clinical parameters for calculation [3]. As a result, simple blood counts have been considered as inexpensive biomarkers, which have been used as simpler adjuncts to assess the severity of the disease at an early stage.

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Acute pancreatitis is associated with systemic inflammation, which involves complex interactions between neutrophils, lymphocytes, platelets, cytokines and endothelial pathways. The platelet-to-lymphocyte ratio (PLR) is a readily available inflammatory index that reflects the changes in platelet count and lymphocyte concentration. Kaplan et al. reported that PLR and the neutrophil-to-lymphocyte ratio (NLR) especially when combined, were correlated with prognosis and mortality in acute pancreatitis [4]. The prognostic significance of the inflammatory indices derived from leukocytes has been corroborated by the associations with persistent organ failure and in-hospital mortality observed in NLR [5].

However, evidence specifically related to PLR is inconsistent. A recent meta-analysis of 20 studies with 5,920 patients showed higher PLR values in severe acute pancreatitis compared to non-severe acute pancreatitis, but also significant differences in the definitions and populations used in the studies [6]. Modern comparative studies have also indicated that PLR, NLR and systemic immune-inflammation index could be useful indicators of severity, but their diagnostic value depends on the threshold and the reference standard used [7]. Comparisons of inflammatory markers and clinical and radiological severity scores also suggest that simple blood markers could be used in addition to validated severity scores [8].

PLR may be clinically useful if it is reliable in identifying patients who are at risk for a more severe course of the disease, and since it is so inexpensive and readily available, it may be. The present study thus examined the association between admission PLR and Revised Atlanta disease severity in acute pancreatitis patients. Secondary outcomes included investigation of relationships with organ failure, pancreatic necrosis, admission to the intensive care unit, hospital length of stay, and mortality, and the comparison of discriminatory ability of PLR with BISAP.

Methods

Study design and eligibility criteria

The analysis was conducted in a retrospective cohort design and comprised 200 records, with one qualifying admission per individual. The study was undertaken in S.R. Patil Medical College, Badagandi, Bagalkote, Karnataka, India, during the admission period from December 2025 to February 2026.

Inclusion criteria were age ≥18 years, and at least two of three criteria for acute pancreatitis: characteristic abdominal pain, serum lipase or amylase more than three times the upper reference limit, and characteristic abdominal imaging findings. A complete blood count within 24 hours of admission, calculable platelet-to-lymphocyte ratio (PLR), and sufficient clinical information for Revised Atlanta severity classification were also required. Patients with chronic pancreatitis who did not have a specific acute episode, major haematological disorders and/or myelosuppressive chemotherapy affecting blood counts were excluded, as were duplicate admissions and insufficient information to establish severity.

Exposure, severity and outcomes

Admission PLR was determined as platelet count/absolute lymphocyte count ($\times 10^9$/L). Absolute neutrophil count (ANC) was used as the numerator for the neutrophil-to-lymphocyte ratio (NLR). The Bedside Index for Severity in Acute Pancreatitis (BISAP) was computed in the first 24 hours based on the presence of blood urea nitrogen (BUN) >25 mg/dL, impaired mental status, systemic inflammatory response syndrome, age >60 years, and pleural effusion. If any component was not available, BISAP was considered to be missing.

Severity was based on the Revised Atlanta criteria. Mild disease was defined as disease without organ failure or local or systemic complications. Moderately severe disease included organ failure that lasted less than 48 hours and/or local or systemic complications. Persistent organ failure >48 hours was required for severe disease.

Organ failure was considered as a modified Marshall score ≥2 in any respiratory, renal or cardiovascular system. Moderately severe and severe disease were pooled together to compare with mild disease. Secondary outcomes were: admission to the ICU, organ failure, pancreatic necrosis, invasive mechanical ventilation, vasopressor use, hospital length of stay and in-hospital mortality.

Statistical analysis

Continuous variables were presented as mean (SD) or median (IQR) and categorical variables as n (%). The Mann–Whitney U test with rank-biserial correlation as the effect estimate was used to compare the admission PLR between the two major outcome groups. Haematological markers were compared between the three Revised Atlanta categories using a Kruskal–Wallis test. Spearman correlations were used to determine the relationship between PLR and BISAP, lab markers and length of stay.

Crude and adjusted logistic regression estimated odds ratios per doubling of PLR. Age, biliary aetiology and admission BUN were included in the adjusted model. The area under the curve (AUC) for PLR was estimated using receiver operating

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characteristic analysis in the entire cohort, and PLR was compared to BISAP in those records for which both scores were available. 95% confidence intervals for rank-based effects, AUCs and the paired AUC difference were obtained by bootstrap resampling. Secondary outcome comparisons were made using the Mann–Whitney U test with at least 10 observations in each group. The analysis of pancreatic necrosis was performed on the imaged subset. Available observations were used in analyses without imputation.

Results

Cohort characteristics

The analysis included 200 acute pancreatitis records. Biliary and alcohol-associated disease were the most common aetiologies. Revised Atlanta severity was mild in 157 (78.5%), moderately severe in 32 (16.0%), and severe in 11 (5.5%) records (Tables 1 and 2).

Admission PLR and disease severity

Admission platelet-to-lymphocyte ratio (PLR) distributions overlapped between mild and higher-severity disease (Figure 1). The rank-biserial correlation for the primary comparison was −0.11 (95% CI −0.30 to 0.07), and the three-category comparison gave p = 0.158. PLR correlated positively with the neutrophil-to-lymphocyte ratio (NLR), while its correlation with BISAP was weak (Table 2).

Adjusted association and discrimination

After adjustment for age, biliary aetiology, and blood urea nitrogen, the odds ratio for moderately severe or severe disease per doubling of PLR was 0.74 (95% CI 0.40 to 1.37). In the 178 records with complete BISAP scores, BISAP had a higher AUC than PLR (0.668 vs 0.437, Table 3 and Figure 2).

Hospital outcomes

ICU admission occurred in 14 (7.0%) records, and in-hospital death in 2 (1.0%). The correlation between admission PLR and length of stay was close to zero (Table4).

Table 1. Baseline characteristics and aetiology

Characteristic	Available n	Summary
Age, years	200	45.80 (14.34)
Female sex	200	56 (28.0%)
BMI, kg/m²	200	25.97 (4.31)
Type 2 diabetes	200	39 (19.5%)
Hypertension	200	44 (22.0%)
Biliary aetiology	200	75 (37.5%)
Alcohol-associated aetiology	200	64 (32.0%)
Other or idiopathic aetiology	200	61 (30.5%)
Admission PLR	200	116.12 (86.71–163.19)
BUN, mg/dL	200	19.20 (15.12–23.42)
BISAP score	178	0.50 (0.00–1.00)

Values are mean (SD), median (IQR), or n (%). BMI, body mass index. BUN, blood urea nitrogen. BISAP, Bedside Index for Severity in Acute Pancreatitis.

Table 2. Admission PLR and related markers by disease severity

Comparison	Mild PLR	Higher-severity PLR	Mann–Whitney U	Rank-biserial r (95% CI)	p
Mild vs higher severity	119.07 (86.80–163.80) n = 157	107.10 (85.71–147.54) n = 43	2,993	−0.11 (−0.30 to 0.07)	0.256

PLR is median (IQR). Higher severity combines moderately severe and severe disease. The effect is oriented as higher severity minus mild.

Marker	Mild (n = 157)	Moderately severe (n = 32)	Severe (n = 11)	H (df = 2)	p
Platelet count (×10⁹/L)	257.00 (213.00–295.00)	226.50 (192.25–265.25)	268.00 (253.50–290.00)	7.87	0.019
Absolute lymphocyte count (×10⁹/L)	2.06 (1.64–2.56)	2.09 (1.78–2.64)	1.93 (1.52–2.52)	0.45	0.800
PLR	119.07 (86.80–163.80)	103.26 (82.61–122.78)	122.17 (97.17–180.22)	3.69	0.158
NLR	3.62 (3.09–4.92)	4.00 (3.12–4.55)	4.74 (3.37–5.68)	1.19	0.552

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Values are median (IQR). H is the Kruskal–Wallis statistic. NLR, neutrophil-to-lymphocyte ratio.

Measure correlated with PLR	n	Spearman ρ (95% CI)	p
BISAP score	178	−0.096 (−0.245 to 0.056)	0.204
CRP (mg/L)	138	0.008 (−0.152 to 0.176)	0.921
BUN (mg/dL)	200	−0.013 (−0.151 to 0.123)	0.853
Serum creatinine (mg/dL)	200	−0.022 (−0.159 to 0.118)	0.758
NLR	200	0.399 (0.264 to 0.519)	<0.001

Correlation intervals are percentile bootstrap estimates. The exploratory p-values are unadjusted for multiple comparisons.

Table 3. Regression and discrimination for higher-severity disease

Predictor	Odds ratio (95% CI)	Wald z	p
PLR per doubling, crude	0.77 (0.43 to 1.39)	−0.87	0.387
PLR per doubling, adjusted	0.74 (0.40 to 1.37)	−0.96	0.336
Age per decade	0.99 (0.77 to 1.29)	−0.04	0.967
Biliary etiology	1.01 (0.47 to 2.19)	0.03	0.972
BUN per 10 mg/dL	6.41 (3.03 to 13.55)	4.86	<0.001

The adjusted model included all four listed predictors and used 200 records with 43 higher-severity events.

ROC analysis	n	Events	AUC or difference (95% CI)
PLR, full cohort	200	43	0.443 (0.349 to 0.545)
PLR, paired cohort	178	39	0.437 (0.339 to 0.541)
BISAP, paired cohort	178	39	0.668 (0.574 to 0.764)
Paired AUC difference, PLR minus BISAP	178	39	−0.230 (−0.365 to −0.093)

AUC, area under the receiver operating characteristic curve. Paired estimates use records with available PLR and BISAP scores.

Table 4. Hospital outcomes and admission PLR

Outcome	Events or stay	PLR with event	PLR without event	Statistic	p
ICU admission	14.0 (7.0%) (n = 200)	113.69 (100.53–170.97)	116.18 (85.39–162.69)	U = 1,493	0.362
Transient organ failure	11.0 (5.5%) (n = 200)	108.78 (101.13–113.69)	117.58 (86.41–163.68)	U = 941	0.599
Persistent organ failure	11.0 (5.5%) (n = 200)	122.17 (97.17–180.22)	115.74 (86.41–161.70)	U = 1,198	0.397
Pancreatic necrosis	10.0 (5.8%) (n = 172)	107.24 (68.07–169.78)	115.21 (86.51–163.60)	U = 737	0.635
Invasive mechanical ventilation	2.0 (1.0%) (n = 200)	254.43 (182.66–326.20)	116.12 (86.51–162.69)	—	—
Vasopressor support	2.0 (1.0%) (n = 200)	183.70 (135.89–231.52)	116.12 (86.51–162.69)	—	—
Inhospital mortality	2.0 (1.0%) (n = 200)	98.43 (93.25–103.61)	116.56 (86.51–163.52)	—	—
Length of stay, days	4.50 (3.50–5.95)	—	—	ρ = −0.026	0.716

PLR and length of stay are median (IQR). Mann–Whitney U tests were used when both groups had at least 10 records. Necrosis used the imaged subset. Spearman ρ describes the PLR–length-of-stay correlation.

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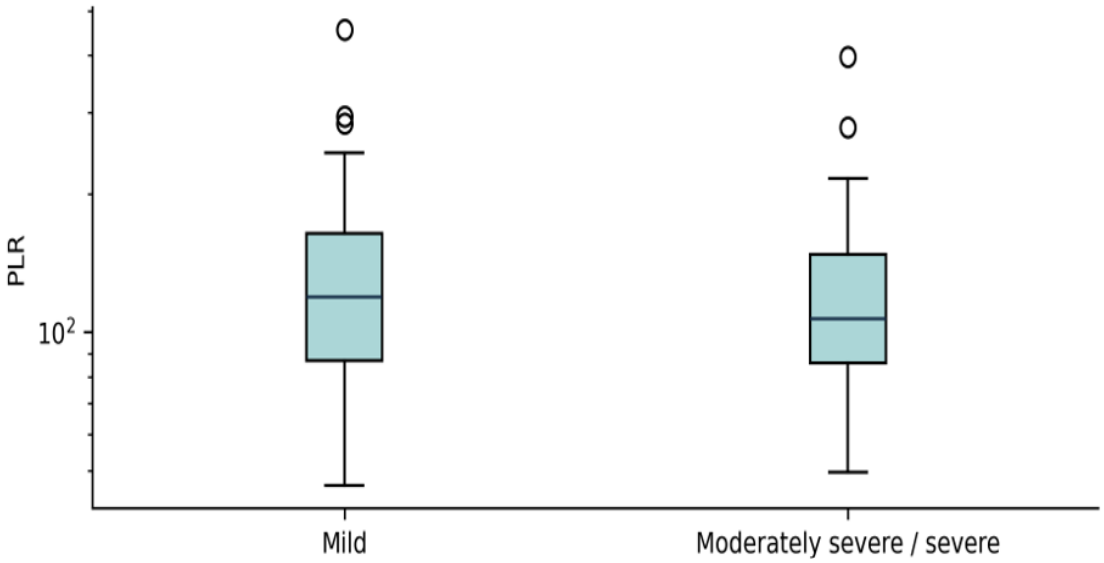

Figure 1. Admission PLR in mild and moderately severe or severe acute pancreatitis. Boxes show the median and interquartile range. The vertical axis uses a logarithmic scale.

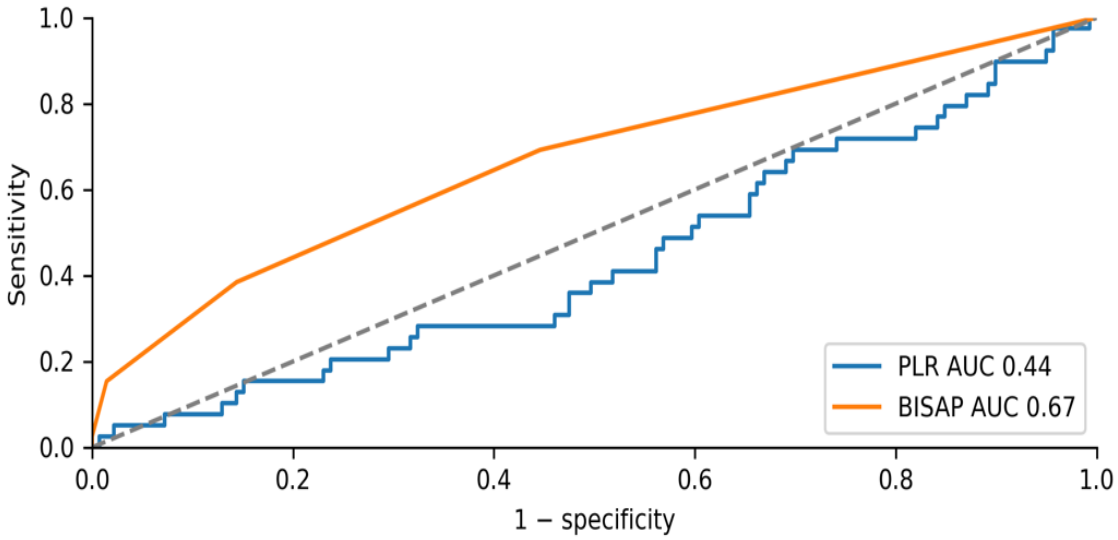

Figure 2. Receiver operating characteristic curves for admission PLR and BISAP in 178 records with both measures, including 39 higher-severity episodes.

Discussion

In this group of 200 patients with acute pancreatitis, the platelet-to-lymphocyte ratio (PLR) measured at admission did not show a link to how severe the disease was. The middle PLR values overlapped a lot between patients with disease and those with more severe disease. No clear pattern appeared when comparing moderately severe and severe categories of the Revised Atlanta classification. After accounting for age biliary cause and blood urea nitrogen (BUN) each doubling of PLR still did not connect with more severe disease. The ability to separate patients by severity was poor (AUC 0.443). The BISAP score performed better though still only moderate (AUC 0.668). PLR also did not relate to ICU admission organ failure, pancreatic tissue death or length of hospital stay. These results cast doubt on using admission PLR as a severity marker for patients with acute pancreatitis who are not preselected.

The findings differ from Cho et al. [9] Who followed 243 patients with gallstone or alcohol related pancreatitis. The PLR was much higher in gallstone pancreatitis than in pancreatitis (344.1 versus 177.8) showing that the cause of pancreatitis strongly affects the PLR. In gallstone pancreatitis the PLR could predict organ failure, with an AUC of 0.638. A PLR threshold of 229.1 gave 70.4% sensitivity and 52.3% specificity. The PLR did not predict organ failure in alcoholic pancreatitis. The difference between causes may partly explain the

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performance of PLR in the mixed cohort, where only 37.5% of patients had biliary disease.

Akdur et al. [10] Reported results in 171 patients with acute pancreatitis. The average PLR was much higher in patients than in controls (221.8 versus 83.3). Both PLR and the neutrophil-to-lymphocyte ratio (NLR) were higher in groups with severe disease. The authors also found that these ratios helped spot complications of pancreatitis. These findings show that PLR is a marker of inflammation. However telling the difference between a person and someone with pancreatitis is not the same as telling how severe the disease is in someone who already has pancreatitis. The results suggest that the latter task is much harder.

Other inflammation markers support this idea. Li et al. [11] Examined 359 patients, including 31 who did not survive and found that NLR was much higher in the non-survivors. The NLR had an AUC of 0.804 for predicting death using a cutoff of 16.64 with 82.4% sensitivity and 75.6% specificity. However severe pancreatitis was linked to C-reactive protein (CRP) red-cell distribution. The prognostic nutritional index, not just to NLR. Therefore even ratios that predict death may not work the way for other outcomes in pancreatitis.

Park [12] also studied NLR in 490 patients. The NLR measured at admission had limited ability to distinguish severe pancreatitis (AUC 0.59) or organ failure (AUC 0.62). When NLR was measured after admission the ability to predict ICU admission improved with AUCs around 0.73–0.74. The best NLR cut-off at admission for disease was 4.76. These results suggest that the way inflammation changes over time gives useful information than a single measurement at admission. The same idea may apply to PLR. The study looked at the admission PLR and did not use later changes in platelet or lymphocyte counts.

Han et al. [13] Showed the value of monitoring inflammation over time in 1,639 patients. Both NLR and the amount of fluid retained were higher in pancreatitis and were linked to the Ranson score and length of hospital stay. An admission NLR of 9.64 or higher gave about 77–82% sensitivity for disease. When NLR was combined with retention the predictive ability matched the Ranson score. These findings support using markers together where blood-cell ratios add to information about body function and fluid status rather than standing alone.

The direct comparison with BISAP is especially important. Cho et al. [14] Examined 161 patients, 21 of whom had pancreatitis. A BISAP score of 2 or more predicted disease with 61.9% sensitivity and 72.1% specificity and the score had an AUC of 0.74. The APACHE-II score performed better with an AUC of 0.78 but the differences between the scores were not statistically significant. In the study BISAP again did better, than PLR with an AUC difference of 0.23. This makes sense because BISAP uses BUN, the inflammatory response, age, mental state and pleural fluid so it captures many aspects of disease severity that a single blood-cell ratio cannot.

Recent data further support combining inflammatory markers with established scores. Lu et al. [15] Studied 113 patients. Observed progressive increases in BISAP, CRP and NLR from mild through moderately severe to severe pancreatitis. The respective AUCs for disease were 0.885 for BISAP, 0.814 for CRP and 0.714 for NLR while the combined model reached an AUC of 0.953 with 86.7% sensitivity and 100% specificity. These findings, considered alongside our PLR discrimination suggest that simple cell-count ratios may have greater value as components of multimodal assessment than as replacements for validated clinical scores.

An additional finding in our study was the association of BUN with higher severity (adjusted OR 6.41 per 10 mg/dL) whereas PLR remained non-significant. This supports the importance of consequences of pancreatitis including intravascular depletion and organ dysfunction, over nonspecific inflammatory ratios. The moderate correlation between PLR and NLR ($\rho = 0.399$) coupled with correlations between PLR and BISAP, CRP, BUN or creatinine further suggests that PLR captures only a limited component of the severity phenotype.

Several limitations should be considered. The number of cases was small restricting precision for uncommon outcomes such as mechanical ventilation and mortality. PLR was measured at admission preventing evaluation of temporal changes. Etiologic heterogeneity may have diluted associations that're specific to biliary or other pancreatitis subtypes. BISAP was unavailable in a proportion of records although the paired ROC comparison used the patients for both markers. Nevertheless the consistent absence of association across severity categories, multivariable analysis and secondary outcomes argues against stand-alone prognostic utility for admission PLR in this cohort.

Admission PLR was not associated with Revised Atlanta severity. Showed poor discrimination, for moderately severe or severe acute pancreatitis. BISAP performed better while BUN demonstrated a strong independent association with severity.

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PLR may reflect inflammatory activity but these findings do not support its use as an isolated early severity marker. Future studies should evaluate PLR measurements and their incremental value when combined with established clinical scores.

Conclusion

Admission PLR was not associated with Revised Atlanta severity and showed poor discrimination for moderately severe or severe acute pancreatitis. BISAP performed substantially better, while BUN demonstrated a strong independent association with severity. PLR may reflect systemic inflammatory activity, but these findings do not support its use as an isolated early severity marker. Future studies should evaluate serial PLR measurements and their incremental value when combined with established clinical scores.

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