

Review Article

# Role of Platelet-to-Lymphocyte Ratio in Predicting Severity in Acute Pancreatitis: A Systematic Review and Meta-Analysis

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## ABSTRACT

**Background:** Platelet-to-lymphocyte ratio (PLR) is an inexpensive inflammatory index proposed for early severity stratification in acute pancreatitis (AP), but reported associations, thresholds, and diagnostic performance are inconsistent. This review evaluated PLR in relation to AP severity, persistent organ failure, mortality, and other adverse outcomes. **Methods:** PubMed/MEDLINE, Google Scholar, and Semantic Scholar were searched, with a September 2026 update using Elicit. Human primary studies assessing PLR in AP severity or prognosis were eligible. Outcomes were stratified by clinical definition to avoid combining Revised Atlanta severe AP, moderately severe/severe AP, persistent organ failure, mortality, imaging-defined severity, and score-defined severity. Risk of bias was assessed according to study type using QUIPS and QUADAS-2. **Results:** Forty-seven records were screened; 25 primary studies were included. Higher PLR was frequently associated with greater AP severity, but findings were heterogeneous and often attenuated in multivariable or comparative analyses. Reported PLR AUROCs included 0.621-0.720 for severe AP, 0.569-0.731 for persistent organ failure, and 0.693 for 28-day mortality. Sensitivity and specificity varied substantially across thresholds and etiologic subgroups. NLR, CRP-based indices, BISAP, SOFA, modified Marshall, and CT-based scores often showed stronger discrimination. Heterogeneity in severity definitions, timing, etiology, and reporting precluded a single universal PLR threshold or defensible pooled diagnostic estimate. **Conclusion:** PLR is associated with AP severity in several populations but has inconsistent standalone prognostic performance. Its most appropriate role is as an adjunct to established clinical, laboratory, and radiological assessment rather than an independent severity classifier. Standardized prospective studies are needed with predefined outcome-specific thresholds and timing. **Keywords:** Acute Pancreatitis; Platelet-to-Lymphocyte Ratio; PLR; Disease Severity; Persistent Organ Failure; Prognostic Biomarkers; Inflammatory Markers; Risk Stratification

## INTRODUCTION

Acute pancreatitis (AP) is a common gastrointestinal emergency characterized by a highly variable clinical course, ranging from a self-limiting inflammatory illness to severe disease complicated by persistent organ failure, pancreatic or peripancreatic necrosis, and death (1). The Revised Atlanta Classification categorizes AP as mild, moderately severe, or severe; severe acute pancreatitis (SAP) is defined principally by organ failure persisting for more than 48 hours (2). Because deterioration may occur early in the disease course, prompt identification of

patients at risk of persistent organ failure and other adverse outcomes is an important component of initial clinical assessment and triage (1-3).

Several clinical, biochemical, and radiological approaches are used for severity stratification, including the Bedside Index for Severity in Acute Pancreatitis (BISAP), Ranson criteria, Acute Physiology and Chronic Health Evaluation II (APACHE II), Sequential Organ Failure Assessment (SOFA), and computed tomography-based severity indices (3,4). However, no single approach provides optimal early prediction across all clinical settings. Some scoring systems require numerous variables, serial measurements, or observations extending beyond the initial presentation, whereas radiological assessment may not be appropriate or informative at the earliest stage of disease (4). These limitations have maintained interest in rapidly available laboratory biomarkers that can complement clinical assessment and facilitate early risk stratification (4,5).

Systemic inflammation is central to the progression of AP and contributes to endothelial dysfunction, immune dysregulation, microcirculatory disturbance, and organ failure in severe disease (1,3). Consequently, several routinely available hematological and inflammatory indices have been investigated as candidate prognostic biomarkers, including the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), red-cell distribution width, C-reactive protein-based ratios, and other composite inflammatory indices (5). These markers are attractive because they can be derived from routine laboratory investigations, may be available early after admission, and impose little additional cost or testing burden (4,5).

PLR integrates changes in circulating platelet and lymphocyte counts and may therefore reflect components of both inflammatory activation and stress-related immune dysregulation. An accumulating observational literature has linked higher PLR values with greater AP severity, persistent organ failure, and adverse clinical outcomes, although the magnitude and consistency of these associations vary considerably among populations and endpoints (6,7). A recent systematic review and meta-analysis including 20 studies and 5,920 patients found significantly higher PLR values in SAP than in non-SAP, with a pooled standardized mean difference of 1.137 (95% CI 0.752-1.702); importantly, the magnitude of the association differed according to the definition used for severe disease (6). Etiology may also influence PLR performance: in biliary and hypertriglyceridemia-induced AP, PLR values and their relationship with persistent organ failure have differed between etiological subgroups (7).

An association between higher PLR and disease severity, however, does not necessarily imply adequate diagnostic or prognostic discrimination. Individual studies have produced heterogeneous findings. In a prospective cohort, Vo et al. reported an area under the receiver operating characteristic curve (AUROC) of 0.72 for PLR in predicting SAP, compared with 0.82 for NLR (8). Salehi et al. subsequently found that PLR showed the highest Youden index among PLR, NLR, and SII for distinguishing moderate/severe from mild AP, although the PLR threshold yielded a sensitivity of 63.89% and specificity of 78.57% (9). Conversely, a large 2026 cohort evaluating 12 inflammatory indices found that PLR was not retained among the biomarkers significantly associated with moderate-to-severe/severe AP after extensive multivariable adjustment, while the C-reactive protein-to-calcium ratio demonstrated the highest observed discrimination (10). Similarly, Abu-Elfath et al. reported a diagnostic accuracy of 65.9% for PLR, compared with substantially higher performance for BISAP and the computed tomography severity index (11). These findings indicate that PLR may capture inflammatory burden without consistently functioning as a strong standalone severity classifier.

Important methodological heterogeneity further complicates interpretation of the literature. Studies differ in the timing of PLR measurement, pancreatitis etiology, severity definitions, reference standards, outcome selection, and PLR thresholds (6-11). Some evaluate Revised Atlanta-defined SAP, whereas others combine moderately severe and severe disease, use persistent organ failure as the target outcome, classify severity according to imaging or clinical scores, or investigate mortality and other adverse outcomes. Moreover, reported measures range from between-group PLR differences to AUROC, sensitivity, specificity, predictive values, and multivariable effect estimates. Pooling such clinically distinct endpoints without appropriate stratification risks conflating an association with severity with the ability to discriminate or predict a specific clinical outcome (6-11).

Accordingly, this systematic review and meta-analysis aimed to provide an updated synthesis of the evidence concerning PLR in AP. The review sought to quantify the association between PLR and clinically defined disease severity where sufficiently comparable data were available, while separately evaluating reported diagnostic and prognostic performance for SAP, moderately severe/severe AP, persistent organ failure, mortality, and other

severity-related outcomes. Particular attention was given to heterogeneity arising from severity definitions, timing of PLR measurement, disease etiology, and reported cut-off values. By distinguishing differences in PLR levels between severity groups from measures of predictive discrimination, this review aimed to clarify whether PLR has sufficient evidence to support use as an independent early prognostic marker or whether its more appropriate role is as an adjunct to established clinical, biochemical, and radiological assessment.

## MATERIALS AND METHODS

### *Study Design and Reporting Framework*

This systematic review and meta-analysis evaluated the association of the platelet-to-lymphocyte ratio (PLR) with severity and adverse outcomes in acute pancreatitis (AP), with a secondary assessment of its reported diagnostic and prognostic performance. The review was structured and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement (12). Because the included literature comprised both prognostic-factor studies and studies reporting diagnostic-performance measures, quantitative analyses were organized according to the clinical question and type of effect estimate rather than combining different outcome definitions or accuracy measures within a single synthesis.

### *Information Sources and Search Strategy*

The source literature search included PubMed/MEDLINE, Google Scholar, and Semantic Scholar. The evidence base was subsequently updated in September 2026 using Elicit as a supplementary literature-discovery platform to identify recent and potentially missed studies. Newly identified candidate articles were cross-checked against PubMed and/or DOI-linked bibliographic records before inclusion.

The search strategy combined terms related to acute pancreatitis and PLR, including: "acute pancreatitis," "platelet-to-lymphocyte ratio," "platelet lymphocyte ratio," "PLR," "severity," "severe acute pancreatitis," "moderately severe acute pancreatitis," "persistent organ failure," "mortality," "prognosis," and "prediction." Boolean combinations were adapted to the requirements of each search platform. Reference lists of relevant systematic reviews and eligible primary studies were also examined for potentially relevant reports. The September 2026 update was specifically designed to capture recently published studies evaluating PLR either as an independent inflammatory marker or as part of comparative or multivariable prognostic models. The search and study-selection process will be reported using a PRISMA 2020 flow diagram (12).

### *Eligibility Criteria*

Studies were eligible if they met the following criteria: they involved human participants with a confirmed diagnosis of acute pancreatitis; reported PLR calculated from platelet and lymphocyte counts; assessed PLR at admission or during the early phase of hospitalization; and evaluated its relationship with clinically relevant severity or prognostic outcomes.

Eligible primary outcomes included severe acute pancreatitis, moderately severe/severe acute pancreatitis, and persistent organ failure. Severe AP was preferentially interpreted according to the Revised Atlanta Classification, in which persistent organ failure lasting more than 48 hours defines severe disease (2). Studies using other explicitly reported severity definitions, including radiological or validated clinical severity criteria, were retained but classified according to the reference standard used by the investigators. Secondary outcomes included mortality, intensive care unit admission, local or systemic complications, need for intervention, length of hospitalization, and correlations with established severity measures such as BISAP, Ranson criteria, APACHE II, SOFA, computed tomography severity index (CTSI), modified CTSI, and the modified Marshall score.

Observational cohort, case-control, cross-sectional diagnostic-accuracy, and prospective prognostic studies were eligible. Reviews, systematic reviews, meta-analyses, editorials, conference material without extractable primary patient data, animal studies, case reports, studies unrelated to AP severity, and studies in which PLR was not evaluated in relation to a severity or prognostic outcome were excluded.

Studies evaluating PLR primarily as a diagnostic marker for the presence of acute pancreatitis, rather than for severity or prognosis among patients already diagnosed with AP, were excluded from the principal synthesis.

### Definition and Harmonization of Outcomes

To minimize clinical heterogeneity, outcome definitions were prespecified for separate synthesis. Studies defining SAP versus non-SAP were considered separately from studies combining moderately severe and severe AP versus mild AP. Persistent organ failure, mortality, intensive care admission, imaging-defined severity, and score-defined high-risk categories were treated as distinct outcomes and were not considered interchangeable with Revised Atlanta-defined SAP.

In particular, a high BISAP, Ranson, APACHE II, SOFA, CTSI, or modified CTSI category was considered a severity-score or imaging-based classification, rather than a direct equivalent of Revised Atlanta-defined severe acute pancreatitis. This distinction was maintained during data extraction, quantitative synthesis, and interpretation. Where studies reported multiple PLR measurements, the value obtained at admission or at the earliest clearly reported time point was prioritized for assessment of early prognostic performance. Later measurements were recorded separately because PLR may change dynamically during the inflammatory course of AP.

### Study Selection

Retrieved records were screened initially by title and abstract. Reports potentially meeting the eligibility criteria were subsequently assessed against the predefined inclusion and exclusion criteria. When multiple publications appeared to originate from the same or overlapping patient cohort, cohort characteristics, recruitment dates, institutions, and outcomes were compared to avoid double counting. Where overlapping reports provided different relevant outcomes, the most complete dataset was used for each respective synthesis. Reasons for exclusion at the full-text stage were categorized for presentation in the PRISMA flow diagram.

### Data Extraction

A standardized data-extraction framework was used to collect the following information from each eligible study: first author, publication year, country, study design, clinical setting, sample size, demographic characteristics, pancreatitis etiology, criteria used to diagnose AP, severity definition, number of patients in each severity category, timing of blood sampling, PLR values, PLR cut-off values, and outcomes assessed. For the quantitative synthesis of PLR levels, group-specific sample sizes, means and standard deviations were extracted for patients with and without the relevant severity outcome when reported. Studies reporting PLR distributions in forms that could not be reliably incorporated into the prespecified quantitative analysis were retained in the qualitative synthesis rather than combined using unsupported assumptions.

For studies assessing diagnostic or prognostic discrimination, the following were extracted when available: area under the receiver operating characteristic curve (AUROC) with 95% confidence interval, PLR threshold, sensitivity, specificity, positive and negative predictive values, and  $2 \times 2$  classification data. Adjusted odds ratios or other multivariable effect estimates were also recorded where available but were analyzed separately from unadjusted diagnostic-performance measures. When a study reported more than one target outcome—for example SAP and mortality—each estimate was assigned to its corresponding outcome category rather than treated as an independent estimate of the same endpoint.

### Primary Quantitative Synthesis

The principal meta-analysis evaluated differences in PLR between clinically comparable severity groups. Because absolute PLR values and their dispersion varied substantially between cohorts, standardized mean differences with 95% confidence intervals were used where appropriate. Hedges'  $g$  was preferred to reduce small-sample bias in standardized effects (16).

A positive standardized mean difference indicated a higher PLR in the more severe or adverse-outcome group. Only studies evaluating sufficiently comparable populations, measurement periods, and target outcomes were pooled within the same analysis. Studies defining strict SAP were not automatically combined with those comparing mild AP against a combined moderately severe/severe AP group. Random-effects models were used because clinical and methodological heterogeneity was anticipated across populations, etiologies, severity definitions, laboratory timing, and study settings. Statistical heterogeneity was assessed using the  $I^2$  statistic, Cochran's  $Q$ , and the between-study variance ( $\tau^2$ ). Heterogeneity measures were interpreted in conjunction with clinical and methodological differences rather than according to rigid numerical thresholds.

### Subgroup and Sensitivity Analyses

Where sufficient studies were available, potential sources of heterogeneity were examined according to severity definition, including Revised Atlanta-defined severity versus alternative criteria; pancreatitis etiology, including unselected AP, acute biliary pancreatitis, and hypertriglyceridemia-induced AP; timing of PLR measurement; and prospective versus retrospective study design. Sensitivity analyses were planned by excluding studies at high risk of bias, studies using non-standard severity definitions, potentially overlapping cohorts, and individual influential studies where appropriate. Leave-one-out analyses were considered when the number of contributing studies was sufficient to permit meaningful assessment. Publication bias was to be explored using funnel-plot asymmetry and formal small-study-effect testing only when an individual meta-analysis contained a sufficient number of studies, generally at least 10, because such assessments are unreliable with very small study sets.

### Diagnostic and Prognostic Performance

Diagnostic and prognostic performance was evaluated separately from the meta-analysis of between-group PLR levels. AUROC, sensitivity, specificity, and threshold values were grouped according to the same target outcome and reference standard. AUROC estimates for SAP were therefore not pooled or directly interpreted together with AUROC values for persistent organ failure, mortality, radiological severity, or score-defined high-risk disease. Likewise, sensitivity and specificity estimates were interpreted in relation to their study-specific PLR thresholds and outcome definitions.

A formal hierarchical or bivariate random-effects diagnostic meta-analysis was considered only when multiple clinically comparable studies reported sufficient threshold-specific information to reconstruct  $2 \times 2$  diagnostic tables. Such models are preferred for jointly synthesizing sensitivity and specificity because these measures are statistically related and threshold-dependent (15). Where the available studies did not provide sufficiently comparable  $2 \times 2$  data, diagnostic performance was summarized descriptively rather than generating a potentially misleading pooled estimate. Reported overall classification accuracy was not combined with mathematically derived balanced accuracy because these statistics are not necessarily equivalent. Balanced accuracy was therefore not used as a substitute for unreported diagnostic accuracy in the revised synthesis.

### Presentation of Diagnostic Estimates

Where confidence intervals were available or could be appropriately derived, study-specific AUROC, sensitivity, and specificity estimates were displayed with 95% confidence intervals. AUROC plots used 0.50 as the reference value for no discriminatory ability. No arbitrary 0.50 reference line was applied to sensitivity or specificity, because 50% does not represent a universal clinically meaningful threshold for either measure.

When confidence intervals or denominators required to calculate them were unavailable, the corresponding result was summarized in the evidence table rather than represented as a conventional forest plot implying precision that had not been reported.

### Risk of Bias Assessment

Because the evidence base contained different study designs and analytical objectives, risk of bias was evaluated according to the type of evidence contributed by each study. Studies contributing prognostic-factor or association data were assessed using domains of the Quality In Prognosis Studies (QUIPS) framework, including study participation, attrition, measurement of the prognostic factor, outcome measurement, confounding, and statistical analysis/reporting (13). Studies contributing diagnostic-accuracy estimates were assessed using QUADAS-2, which evaluates patient selection, conduct and interpretation of the index test, reference standard, and flow and timing, together with concerns regarding applicability (14). A study contributing information to both prognostic and diagnostic syntheses was assessed according to the domains relevant to the respective analysis. Risk-of-bias judgments were reported at study/domain level as low, moderate/unclear where appropriate to the tool, or high risk rather than assigning a single unsupported quality score across all studies.

### Narrative Synthesis

Studies unsuitable for quantitative pooling remained eligible for the systematic review. Narrative synthesis was organized according to the clinical endpoint rather than publication year alone. Findings were grouped into: association of PLR with AP severity; PLR and persistent organ failure; PLR and mortality or other adverse

outcomes; comparative performance against other inflammatory biomarkers; etiology-specific findings; and diagnostic/prognostic discrimination. Particular attention was given to whether PLR remained independently associated with outcomes after multivariable adjustment, whether alternative markers or validated scores demonstrated greater discrimination, and whether results differed according to disease etiology or sampling time.

## RESULTS

### Study Selection

The literature search identified 47 records for screening. Twenty-two records were excluded because they were reviews or meta-analyses, animal studies, case reports, unrelated to acute pancreatitis severity, did not provide a PLR-specific evaluation, or used the abbreviation PLR for an unrelated condition. Twenty-five primary studies fulfilled the eligibility criteria and were included in the qualitative synthesis. Studies were published between 2016 and 2026 and included retrospective and prospective observational designs. Sample sizes ranged from small, highly selected cohorts to large hospital-based populations exceeding 1,900 patients. The included studies differed substantially in study population, pancreatitis etiology, timing of PLR measurement, severity definition, and reported outcome. Several studies evaluated all-cause acute pancreatitis, whereas others focused on acute biliary pancreatitis, hypertriglyceridemia-induced pancreatitis, hyperlipidemic pancreatitis, or acute pancreatitis in pregnancy. Severity was defined using the Revised Atlanta Classification in some studies, whereas others used BISAP, CTSI, modified CTSI, organ failure, mortality, or other clinical outcomes. Consequently, studies were grouped according to their actual clinical endpoint rather than considering all reported severity measures interchangeable.

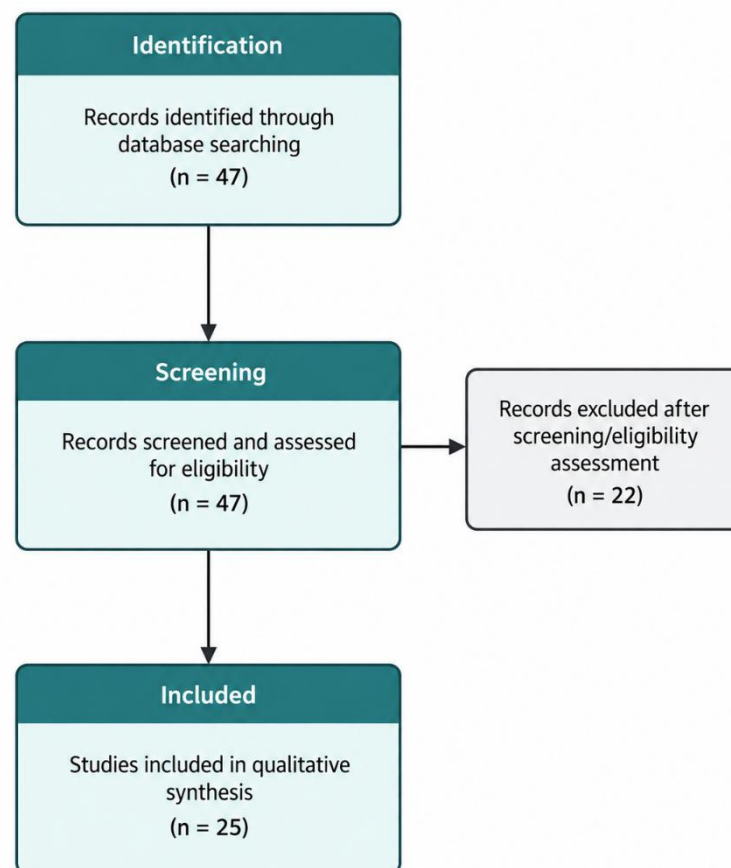


**Figure 1. PRISMA flow diagram of study identification and selection.**

### Characteristics of Included Studies

Most included investigations were retrospective cohort studies, although several prospective observational cohorts were available. PLR was generally calculated from admission or early-hospitalization complete blood

counts, but the timing of measurement was not uniform. The principal study characteristics are summarized in Table 1.

**Table 1. Characteristics of Included Studies**

| Study                        | Design / Sample                     | Population                   | Principal Outcome / Reference Standard           | Main PLR Finding                                                                                 |
|------------------------------|-------------------------------------|------------------------------|--------------------------------------------------|--------------------------------------------------------------------------------------------------|
| Maghsoomi et al., 2025 (17)  | Retrospective; n=150                | AP                           | Multiple severity scores and mortality           | PLR showed minimal prognostic utility; NLR and platelet dynamics were more informative           |
| Afridi et al., 2026 (18)     | Retrospective multicenter; n=272    | Adult AP                     | Revised Atlanta severe vs non-severe AP          | PLR demonstrated weaker predictive performance than NLR and CRP                                  |
| Abu-Elfath et al., 2026 (11) | Observational cohort; n=300         | AP                           | Severe AP                                        | PLR diagnostic accuracy was 65.9%, lower than BISAP and CTSI                                     |
| Ma et al., 2026 (10)         | Retrospective; n=1,981              | Hospitalized AP              | MAP vs MSAP/SAP                                  | PLR was not retained among significant biomarkers in the fully adjusted model                    |
| Salehi et al., 2025 (9)      | Retrospective; n=112                | AP                           | Mild vs moderate/severe AP by modified CTSI      | PLR had the highest Youden index among PLR, NLR, and SII                                         |
| Stutee et al., 2025 (19)     | Prospective; n=80                   | AP                           | BISAP, CTSI and modified CTSI                    | PLR declined during recovery; established scores and LDH showed stronger prognostic associations |
| Li et al., 2025 (20)         | Retrospective; n=230                | HTG-AP                       | Disease progression                              | PLR was evaluated, but D-dimer and CRP-to-lymphocyte ratio were stronger predictors              |
| Pan et al., 2025 (21)        | Retrospective; n=145                | Hyperlipidemic AP            | Mild vs moderate/severe disease                  | PLR contributed to a combined NLR/PLR/PCT/D-dimer model                                          |
| Xu et al., 2024 (7)          | Retrospective; n=771 analyzable     | Biliary and HTG-AP           | Persistent organ failure                         | PLR varied by etiology and showed modest outcome-specific discrimination                         |
| Li et al., 2024 (22)         | Retrospective; n=253                | AP                           | Severe AP                                        | PLR was assessed among inflammatory indices but was not central to the final prediction model    |
| Vo et al., 2023 (8)          | Prospective; n=131                  | AP                           | Severe AP                                        | PLR AUROC 0.72; cut-off 202.7; high negative predictive value                                    |
| Ak et al., 2023 (23)         | Retrospective; n=514                | AP                           | Atlanta severity                                 | Higher PLR was associated with greater disease severity                                          |
| Akdur et al., 2022 (24)      | Retrospective; 171 AP + 59 controls | AP                           | BISAP/mGPS and adverse outcomes                  | NLR and PLR were associated with severity and adverse outcomes                                   |
| Karabuga et al., 2022 (25)   | Retrospective; n=500                | AP                           | BISAP-defined severe disease                     | PLR sensitivity 55.36% and specificity 38.51%; weaker than several comparator markers            |
| Liu et al., 2021 (26)        | Retrospective; n=101                | AP                           | Mild vs severe AP                                | SII outperformed PLR and NLR                                                                     |
| Khan et al., 2021 (27)       | Retrospective; n=154                | Emergency AP                 | CT severity score                                | PLR increased with radiological severity                                                         |
| Junare et al., 2021 (28)     | Prospective; n=160                  | AP                           | Organ failure, ICU care, intervention, mortality | PLR increased with severity, although NLR showed better overall performance                      |
| Pian et al., 2021 (29)       | Retrospective; n=169                | AP                           | Severe vs non-severe AP                          | NLR and prognostic nutritional index were more informative than PLR                              |
| Cifci et al., 2020 (30)      | Retrospective; n=557                | Emergency AP                 | Mortality and length of stay                     | PLR was associated with mortality on ROC analysis                                                |
| Zhou et al., 2019 (31)       | Retrospective; n=406                | AP                           | SAP and 28-day mortality                         | PLR AUROC 0.621 for SAP and 0.693 for mortality                                                  |
| Liu et al., 2019 (32)        | Retrospective                       | AP                           | Persistent organ failure                         | PLR AUROC 0.731; cut-off 173.13                                                                  |
| Kaplan et al., 2018 (33)     | Retrospective; n=142                | AP                           | Prognosis and mortality                          | Combined PLR-NLR assessment was associated with prognosis                                        |
| Cho et al., 2018 (34)        | Prospective; n=243                  | Gallstone/alcohol-related AP | Severe AP                                        | PLR predicted severity in gallstone but not alcoholic pancreatitis                               |
| Wang et al., 2017 (35)       | Retrospective; n=110                | HTG-AP                       | Severe AP                                        | NLR showed stronger discrimination than PLR                                                      |
| Iihan et al., 2016 (36)      | Case-control; 14 AP + 30 controls   | AP in pregnancy              | AP/severity relationship                         | PLR was not significantly discriminatory; NLR was more informative                               |

AP, acute pancreatitis; CTSI, computed tomography severity index; HTG-AP, hypertriglyceridemia-induced acute pancreatitis; MAP, mild acute pancreatitis; MSAP, moderately severe acute pancreatitis; NLR, neutrophil-to-lymphocyte ratio; PCT, procalcitonin; PLR, platelet-to-lymphocyte ratio; SAP, severe acute pancreatitis; SII, systemic immune-inflammation index.

#### Association Between PLR and Acute Pancreatitis Severity

Across the included literature, higher PLR was frequently observed in patients with more severe acute pancreatitis; however, the strength and independence of this relationship were inconsistent. Several studies demonstrated higher PLR across increasing severity categories or positive associations with radiological or clinical severity indices (7,23,24,27,28). In contrast, other studies found that PLR lost significance when evaluated together with alternative inflammatory indices or after multivariable adjustment (10,17,18,20,22,26,29). Etiology appeared to modify the relationship between PLR and outcome. Cho et al. found PLR to be predictive of severity in gallstone pancreatitis but not alcoholic pancreatitis (34). Xu et al. likewise demonstrated differences between acute biliary and hypertriglyceridemia-induced pancreatitis. In their cohort, PLR was higher in patients with severe disease in both etiological groups, but its ability to predict persistent organ failure differed markedly between the two populations (7). These observations support outcome- and etiology-specific interpretation rather than application of a single universal PLR threshold.

More recent comparative studies also indicate that PLR is not consistently the strongest inflammatory predictor. Ma et al. evaluated 12 inflammatory indices in 1,981 patients and found that PLR was not retained among the significant biomarkers in the fully adjusted model; the CRP-to-calcium ratio demonstrated the greatest observed discrimination for MSAP/SAP (10). Afridi et al. similarly found NLR and CRP to have stronger relationships with severe AP than PLR (18). Maghsoomi et al. reported minimal prognostic utility for PLR across several severity scores (17).

#### PLR and Persistent Organ Failure

Persistent organ failure was analyzed separately because it represents a clinically distinct endpoint and, under the Revised Atlanta Classification, is central to the definition of severe acute pancreatitis. Liu et al. reported an AUROC of 0.731 for PLR in predicting persistent organ failure, with a cut-off of 173.13 and sensitivity of 95.8% (32). Xu et al. demonstrated substantially different performance according to etiology. For hypertriglyceridemia-induced AP, PLR yielded an AUROC of 0.622 (95% CI 0.546–0.699), sensitivity of 41.3%, and specificity of 82.6%. In acute biliary pancreatitis, the AUROC was lower at 0.569 (95% CI 0.516–0.621), with sensitivity of 72.7% and specificity of 41.8% (7).

In the same Xu cohort, established organ-failure scores generally performed better than PLR. In HTG-AP, SOFA achieved an AUROC of 0.827 and the modified Marshall score 0.772, compared with 0.622 for PLR. In acute biliary pancreatitis, the modified Marshall score and Japanese Severity Score also demonstrated greater discrimination than PLR (7). These data suggest that PLR may be associated with organ-failure risk but has limited standalone discrimination.

#### Diagnostic and Prognostic Performance

The reported performance of PLR varied according to target outcome and reference standard. Therefore, AUROC values for SAP, persistent organ failure, mortality, imaging-defined severity, and score-defined severity were not combined as if they represented a single diagnostic endpoint. For prediction of SAP, Zhou et al. reported a PLR AUROC of 0.621, whereas Vo et al. reported an AUROC of 0.72 (8,31). For persistent organ failure, the reported AUROC values ranged from 0.569 in biliary AP to 0.731 in the Liu cohort (7,32). Zhou et al. reported an AUROC of 0.693 for 28-day mortality (31). Thus, directly reported PLR AUROC values across these different clinical endpoints generally fell within the poor-to-moderate discrimination range, but their clinical meaning differed because the target outcomes were not equivalent.

**Table 2. Reported PLR Diagnostic and Prognostic Performance**

| Study                  | Target Outcome                | PLR Cut-off | AUROC              | Sensitivity | Specificity | Interpretation                                         |
|------------------------|-------------------------------|-------------|--------------------|-------------|-------------|--------------------------------------------------------|
| Zhou et al. (31)       | SAP                           | NR          | 0.621              | NR          | NR          | Limited discrimination                                 |
| Zhou et al. (31)       | 28-day mortality              | NR          | 0.693              | NR          | NR          | Modest discrimination                                  |
| Vo et al. (8)          | SAP                           | 202.7       | 0.720              | NR          | NR          | Moderate discrimination; NPV 91.9%                     |
| Liu et al. (32)        | Persistent organ failure      | 173.13      | 0.731              | 95.8%       | NR          | High sensitivity but incomplete specificity reporting  |
| Xu et al. (7)          | POF, HTG-AP                   | NR          | 0.622              | 41.3%       | 82.6%       | Low sensitivity, relatively high specificity           |
| Xu et al. (7)          | POF, biliary AP               | NR          | 0.569              | 72.7%       | 41.8%       | Limited overall discrimination                         |
| Salehi et al. (9)      | Moderate/severe AP vs mild AP | 21.14       | PLR-specific AUROC | 63.89%      | 78.57%      | Highest Youden index among PLR, NLR and SII            |
| Karabuga et al. (25)   | BISAP-defined severe AP       | NR          | NR                 | 55.36%      | 38.51%      | Weak classification performance                        |
| Abu-Elfath et al. (11) | SAP                           | NR          | NR                 | NR          | NR          | Reported overall PLR diagnostic accuracy 65.9%         |
| Afridi et al. (18)     | Revised Atlanta SAP           | NR          | NR                 | NR          | NR          | PLR predictive accuracy 74.6%; weaker than NLR and CRP |

NR, not reported in the available extract; NPV, negative predictive value; POF, persistent organ failure.

#### Sensitivity and Specificity

Threshold-based sensitivity and specificity demonstrated substantial between-study variability. Salehi et al. reported sensitivity of 63.89% and specificity of 78.57% at a PLR threshold of 21.14 for differentiating moderate/severe from mild AP (9). Karabuga et al., using BISAP-defined severity, reported sensitivity of 55.36% and specificity of only 38.51% (25). For persistent organ failure, Xu et al. reported opposite sensitivity-specificity profiles between etiological subgroups: PLR was relatively specific but insensitive in HTG-AP, whereas it was more sensitive but poorly specific in biliary AP (7). This variation demonstrates substantial threshold and spectrum effects and does not support identification of a single PLR cut-off applicable across all forms of acute pancreatitis.

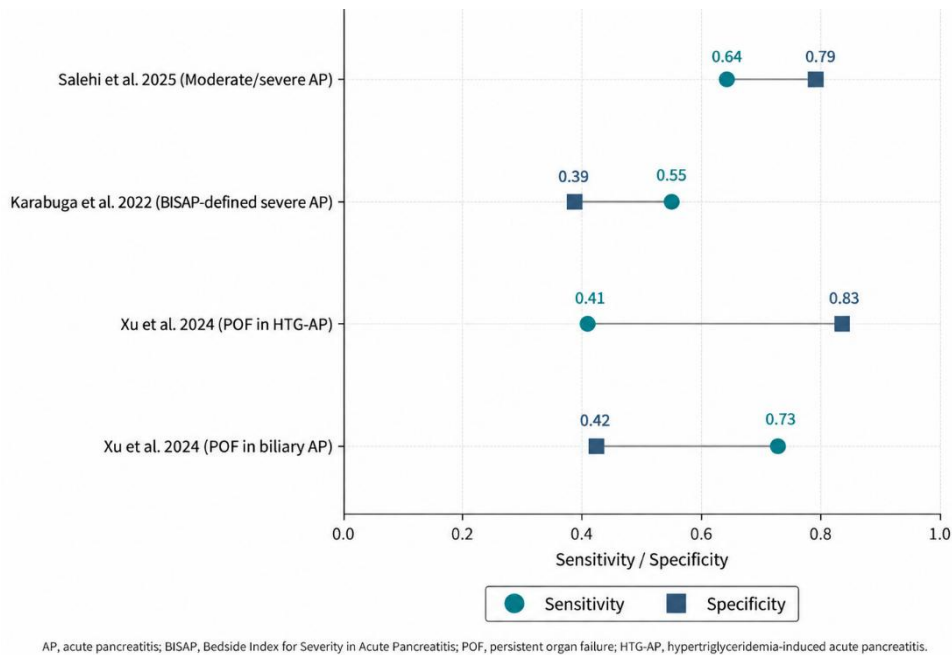


**Figure 2. Outcome-specific PLR sensitivity and specificity.**

#### Descriptive AUROC Synthesis

Because the reported AUROC estimates referred to clinically different target conditions, no overall mean AUROC was calculated. Instead, values were grouped by endpoint.

#### Performance Relative to Other Biomarkers and Severity Scores

PLR was rarely the strongest predictor when compared directly with other inflammatory markers or established scoring systems. Zhou et al. found BISAP, SOFA, Ranson, APACHE II, and RDW to provide greater discrimination for important outcomes than PLR (31). Vo et al. reported an AUROC of 0.82 for NLR compared with 0.72 for PLR in predicting SAP (8). Karabuga et al. found substantially better diagnostic performance for CRP, CRP/albumin, and NLR than for PLR (25).

More recent studies reinforced this pattern. Ma et al. identified CRP-based composite indices, particularly the CRP-to-calcium ratio, as stronger predictors than PLR (10). Abu-Elfath et al. reported diagnostic accuracy of 96.4% for BISAP and 100% for CTSI compared with 65.9% for PLR (11). Afridi et al. likewise demonstrated stronger associations and predictive performance for NLR and CRP than PLR (18). Conversely, Salehi et al. found PLR to have the highest Youden index among PLR, NLR, and SII for their specific moderate/severe AP classification, illustrating that relative performance was not uniform across cohorts (9).

#### Risk of Bias and Methodological Heterogeneity

The evidence base was dominated by retrospective, single-center observational studies, with fewer prospective cohorts (8,19,28,34). The principal methodological concerns were non-consecutive or incompletely described patient selection, differences in the timing of PLR measurement, inconsistent adjustment for potential confounders, heterogeneous severity reference standards, and incomplete reporting of PLR-specific diagnostic accuracy.

A further concern was outcome heterogeneity. Revised Atlanta-defined SAP, combined MSAP/SAP, persistent organ failure, BISAP-defined severity, CT-defined severity, ICU admission, and mortality were used across different studies. These outcomes were therefore not considered interchangeable. Diagnostic-accuracy studies were particularly limited by variable PLR thresholds and incomplete reporting of  $2 \times 2$  classification data.

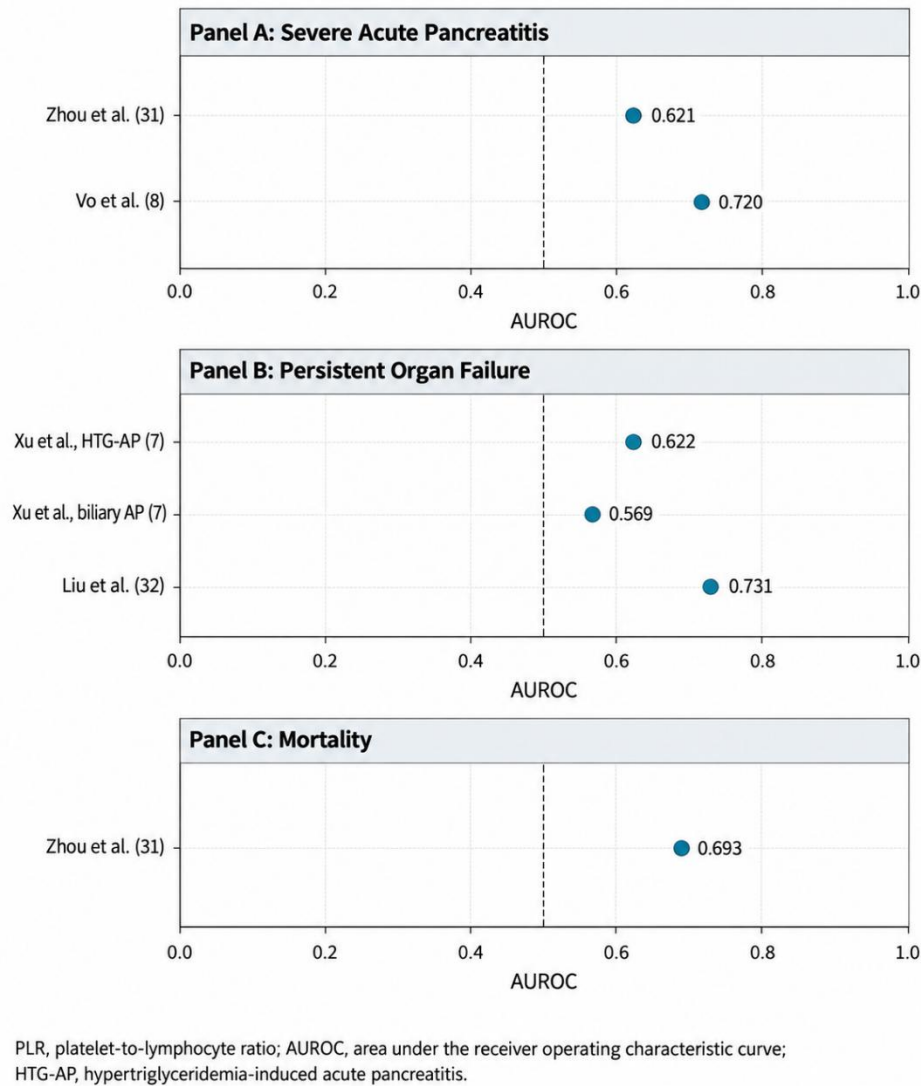


**Figure 3.** Descriptive plot of platelet-to-lymphocyte ratio (PLR) area under the receiver operating characteristic curve (AUROC) values stratified by clinical outcome. For severe acute pancreatitis, AUROC values were 0.621 for Zhou et al. (31) and 0.720 for Vo et al. (8). For persistent organ failure, AUROC values were 0.622 for Xu et al. in hypertriglyceridemia-induced acute pancreatitis, 0.569 for Xu et al. in biliary acute pancreatitis (7), and 0.731 for Liu et al. (32). For mortality, Zhou et al. reported an AUROC of 0.693 (31). The vertical reference line at AUROC = 0.50 represents discrimination no better than chance. Values are presented descriptively at the study level and are not pooled because of clinical heterogeneity across outcomes and incomplete reporting of confidence intervals.

#### Overall Evidence Pattern

Taken together, the included studies support an association between elevated PLR and greater inflammatory or clinical severity in several acute pancreatitis populations, but the relationship was not universal and was frequently attenuated when PLR was compared with stronger inflammatory markers or established clinical scores. PLR performance also varied according to pancreatitis etiology, outcome definition, timing of measurement, and threshold selection. The most consistent interpretation of the evidence is therefore that PLR provides adjunctive prognostic information, rather than functioning as a sufficiently accurate standalone classifier of SAP, persistent organ failure, or mortality. No universal PLR threshold could be identified from the available studies.

**Table 3.** Principal Sources of Methodological Heterogeneity

| Domain                     | Common Finding                                                      | Potential Effect on Interpretation             |
|----------------------------|---------------------------------------------------------------------|------------------------------------------------|
| <b>Study design</b>        | Predominantly retrospective cohorts                                 | Selection bias and residual confounding        |
| <b>Setting</b>             | Mostly single-center studies                                        | Reduced external validity                      |
| <b>PLR timing</b>          | Admission, ≤24 h, ≤48 h, and serial later measurements              | PLR may vary with disease evolution            |
| <b>Severity definition</b> | Revised Atlanta, BISAP, CTSI/MCTSI, organ failure, mortality        | Limits direct between-study comparability      |
| <b>Etiology</b>            | Mixed AP, biliary AP, HTG-AP, alcoholic AP, pregnancy-associated AP | Possible spectrum and etiological effects      |
| <b>PLR threshold</b>       | Substantial variation or no threshold reported                      | Prevents universal cut-off determination       |
| <b>Accuracy reporting</b>  | AUROC, sensitivity, specificity and 2×2 data incompletely reported  | Limits formal diagnostic meta-analysis         |
| <b>Comparator markers</b>  | NLR, CRP, SII, RDW and clinical scores varied across studies        | Relative performance depends on comparator set |

## DISCUSSION

This systematic review synthesized 25 primary studies examining the platelet-to-lymphocyte ratio (PLR) in relation to acute pancreatitis (AP) severity and prognosis. The overall evidence indicates that PLR frequently increases with worsening disease severity, but its performance as a standalone prognostic or discriminatory biomarker is inconsistent. This distinction is important: a biomarker may differ significantly between severity groups without providing sufficient discrimination to classify individual patients reliably. Across studies reporting receiver-operating-characteristic analyses, PLR AUROC values were generally modest, ranging from 0.621 to 0.720 for severe acute pancreatitis (SAP), from 0.569 to 0.731 for persistent organ failure (POF), and reaching 0.693 for 28-day mortality (7,8,31,32). Differences in outcome definitions, PLR thresholds, disease etiology, measurement timing, and reference standards prevented interpretation of these estimates as a single diagnostic construct.

The present findings should be interpreted in the context of the recent systematic review and meta-analysis by Ibrahim et al., which included 20 studies and 5,920 patients and demonstrated significantly higher PLR values in SAP than in non-SAP, with a pooled standardized mean difference of 1.137 (95% CI 0.752–1.702) (6). The association was stronger among studies applying Revised Atlanta criteria, but significant heterogeneity according to the definition of SAP was identified (6). The present review extends that evidence by emphasizing the distinction between between-group association and patient-level prognostic discrimination and by incorporating more recent studies published through 2026. This distinction helps reconcile apparently conflicting findings: PLR may be statistically elevated in patients with more severe disease while still showing only limited-to-moderate AUROC, sensitivity, or specificity.

Recent primary studies reinforce this interpretation. Vo et al. reported an AUROC of 0.720 for PLR in predicting SAP, whereas NLR achieved an AUROC of 0.820 (8). Salehi et al. found PLR to have the highest Youden index among PLR, NLR, and systemic immune-inflammation index in their cohort, but at the reported threshold its sensitivity was 63.89% and specificity 78.57% (9). In contrast, Ma et al., in a large cohort of 1,981 patients, did not retain PLR among the inflammatory indices independently associated with moderate-to-severe/severe AP after extensive multivariable adjustment; CRP-based indices performed more strongly (10). Similarly, Abu-Elfath et al. reported an overall diagnostic accuracy of 65.9% for PLR, compared with substantially greater performance for BISAP and CTSI (11), while Afridi et al. observed stronger predictive relationships for NLR and CRP than for PLR (18). Collectively, these findings argue against interpreting PLR as an independently sufficient marker of AP severity.

Persistent organ failure provides a particularly relevant endpoint because it is central to the Revised Atlanta definition of SAP (2). Liu et al. reported an AUROC of 0.731 for PLR in predicting POF and a sensitivity of 95.8% at a threshold of 173.13 (32). However, Xu et al. demonstrated considerable etiological variation: the AUROC was 0.622 in hypertriglyceridemia-induced AP and 0.569 in acute biliary pancreatitis, with markedly different sensitivity-specificity profiles between these subgroups (7). In the same study, SOFA and modified Marshall scores provided substantially greater discrimination for POF than PLR (7). These findings demonstrate why severity definitions and etiological subgroups should not be pooled indiscriminately and suggest that PLR performance may depend partly on the pathophysiological context in which AP develops.

Etiology-related variation was also evident in earlier studies. Cho et al. found PLR to be associated with severity in gallstone pancreatitis but not alcoholic pancreatitis (34), whereas Wang et al. found NLR to perform more consistently than PLR in hypertriglyceridemia-induced AP (35). PLR is biologically plausible as an inflammatory index because it incorporates changes in both platelet and lymphocyte compartments, yet these cellular components may be influenced by the cause of pancreatitis, systemic inflammatory burden, immune response, comorbidity, infection, and disease stage (5). Consequently, a single PLR value is unlikely to capture the multidimensional pathophysiology responsible for persistent organ failure and other complications.

Timing represents another important source of heterogeneity. Some studies assessed PLR immediately on admission, whereas others used measurements during the first 24–48 hours or evaluated serial values during hospitalization. AP is a dynamic inflammatory condition, and prognostic information contained in hematological indices may evolve over time (1,3,5). Studies evaluating admission PLR therefore address a different clinical question from studies assessing changes after resuscitation or during recovery. Future synthesis should preserve these time windows rather than treating PLR measurements obtained at different disease stages as equivalent.

The variability in reported thresholds further limits clinical translation. Thresholds differed widely across studies and were commonly derived within individual datasets using ROC optimization rather than being prospectively prespecified and externally validated. Karabuga et al. reported relatively poor sensitivity and specificity for PLR when severity was defined using BISAP (25), whereas Salehi et al. reported a more favorable specificity using a different threshold and modified CTSI-based outcome (9). Such variation may reflect differences in disease spectrum, prevalence of severe disease, laboratory timing, etiological composition, and choice of reference standard rather than a true universal biological threshold. On the available evidence, no single PLR cut-off can be recommended for general use across AP populations (7,8,10).

The comparative evidence is also clinically informative. PLR was commonly evaluated alongside NLR, RDW, CRP, CRP-derived ratios, SII, D-dimer, and established clinical scores (8-11,17-29). In many cohorts, NLR, CRP-related indices, BISAP, SOFA, modified Marshall score, or CT-based severity assessment performed at least as well as, and frequently better than, PLR (11,17-19,25,26,31,32). This does not render PLR clinically irrelevant. Because PLR is inexpensive, rapidly calculated, and derived from routine complete blood counts, it may contribute useful supplementary information, especially where more complex testing is unavailable. Its value is therefore more plausibly adjunctive than substitutive: PLR may contribute to an early multidimensional assessment but should not replace established clinical evaluation, organ-failure assessment, or validated prognostic scoring systems.

An additional methodological issue concerns the distinction between SAP, combined MSAP/SAP, POF, mortality, radiological severity, and score-defined high-risk disease. Earlier presentations of these data risked treating such outcomes as interchangeable. The Revised Atlanta Classification defines SAP according to persistent organ failure, whereas BISAP, CTSI, modified CTSI, and other scores are predictors or severity descriptors rather than equivalent reference diagnoses (2). Separating these endpoints in the present synthesis substantially improves interpretability and prevents artificial aggregation of fundamentally different clinical questions.

#### Strengths and Limitations

A principal strength of this review is its outcome-specific approach. Rather than treating all severity-related outcomes as equivalent, the synthesis distinguishes SAP, MSAP/SAP, POF, mortality, radiological severity, and score-defined disease. It also separates evidence that PLR is associated with severity from evidence that PLR can discriminate individual patients with clinically important outcomes. The literature was updated through September 2026, allowing recent studies to be considered alongside earlier evidence (7-11,17-36). Risk-of-bias assessment was aligned with study purpose through the use of QUIPS for prognostic-factor evidence and QUADAS-2 for diagnostic-accuracy evidence (13,14).

Several limitations remain. Most primary studies were retrospective and single-center, with considerable variation in patient selection, etiology, PLR measurement timing, severity definition, and statistical adjustment. Diagnostic-accuracy reporting was frequently incomplete, particularly with respect to confidence intervals, predetermined thresholds, and reconstructable 2×2 data. Some studies used severity scores or imaging classifications as reference standards rather than Revised Atlanta clinical outcomes. These limitations prevented a defensible pooled diagnostic-accuracy estimate and limited identification of a clinically generalizable PLR threshold. Although group-level PLR differences have previously been meta-analyzed (6), sufficient homogeneous and consistently reported summary data were not available within the present extraction to justify presenting a new pooled effect estimate without additional assumptions.

#### Implications for Research

Future studies should be prospective, multicenter, and adequately powered, with PLR measured at prespecified time points, preferably including the initial admission sample. Revised Atlanta-defined SAP and POF should be analyzed as standardized primary outcomes, while mortality and other complications should remain separate secondary endpoints. Investigators should report PLR distributions, prespecified thresholds, AUROC with confidence intervals, sensitivity, specificity, and complete 2×2 classification data. External validation is essential before any threshold is proposed for routine clinical use. Equally important, future studies should determine whether PLR adds clinically meaningful incremental information to readily available predictors such as NLR, CRP, BISAP, and early organ-failure assessment rather than evaluating PLR only in isolation.

## CONCLUSION

PLR is frequently elevated in patients with more severe acute pancreatitis and therefore reflects aspects of the systemic inflammatory response. However, its diagnostic and prognostic performance varies substantially according to the clinical endpoint, disease etiology, timing of measurement, severity definition, and selected threshold. Available AUROC and threshold-based data indicate predominantly limited-to-moderate standalone discrimination, and several established clinical scores and alternative inflammatory biomarkers perform more consistently.

PLR should therefore be considered an adjunctive, readily available inflammatory marker rather than an independent classifier of severe acute pancreatitis, persistent organ failure, or mortality. Current evidence does not support a universal PLR threshold. Prospective multicenter validation using standardized Revised Atlanta outcomes, prespecified sampling times, complete diagnostic-accuracy reporting, and assessment of incremental value over established prognostic tools is required before routine PLR-based clinical decision-making can be recommended.

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