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Original Research Article

Neutrophil-To-Lymphocyte Ratio as an Early Predictor of Severity in Acute Pancreatitis: A Prospective Observational Study

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Abstract

Background: Early recognition of severe acute pancreatitis remains difficult because organ failure may evolve after an initially nondiagnostic presentation. The neutrophil-to-lymphocyte ratio (NLR), derived from a routine differential leukocyte count, integrates innate inflammatory activation and stress-associated lymphopenia. This study evaluated admission NLR as an early predictor of acute pancreatitis severity and adverse hospital outcomes.

Methods: A prospective observational study included 180 consecutive adults admitted within 24 hours of symptom onset to a tertiary-care hospital. Acute pancreatitis and disease severity were classified according to the revised Atlanta criteria. Admission NLR was calculated from absolute neutrophil and lymphocyte counts. Bedside Index for Severity in Acute Pancreatitis (BISAP), Acute Physiology and Chronic Health Evaluation II (APACHE II), 48-hour C-reactive protein (CRP), and modified computed tomography severity index were recorded. Receiver-operating-characteristic analysis and multivariable logistic regression were used to evaluate the prediction of severe acute pancreatitis.

Results: Of 180 patients, 108 (60.0%) had mild, 48 (26.7%) had moderately severe, and 24 (13.3%) had severe acute pancreatitis. Median admission NLR increased progressively from 5.2 in mild disease to 6.5 in moderately severe disease and 10.7 in severe pancreatitis ($p<0.001$). NLR predicted severe disease with an area under the receiver-operating-characteristic curve of 0.853 (95% confidence interval [CI]: 0.769–0.920). A cutoff of 6.7 provided 95.8% sensitivity and 66.0% specificity. Severe pancreatitis occurred in 30.1% of patients with NLR ≥6.7 compared with 1.9% of those below this threshold ($p<0.001$). Mortality was 11.0% and 0%, respectively. After adjustment for age, BISAP, and blood urea nitrogen, every one-unit increase in NLR remained independently associated with severe pancreatitis (adjusted odds ratio [OR]: 1.40; 95% CI: 1.15–1.72; $p<0.001$).

Conclusion: Admission NLR provided rapid, inexpensive, and clinically meaningful early risk stratification in acute pancreatitis. Although it did not replace multiparametric assessment or 48-hour CRP, an NLR ≥6.7 identified a high-risk subgroup requiring intensified monitoring and early organ-support planning.

Keywords: Acute Pancreatitis; Neutrophil-To-Lymphocyte Ratio; Severity; Organ Failure; BISAP; Inflammatory Biomarker.

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Introduction

Acute pancreatitis is an inflammatory disorder characterized by a clinical spectrum ranging from self-limited interstitial pancreatic edema to necrotizing disease, persistent organ failure, and death. Its incidence has increased globally, and the disease remains one of the leading causes of gastrointestinal-related hospitalization [1,2]. Although most affected patients recover with supportive treatment, a clinically important minority develop severe acute pancreatitis, in which systemic inflammatory response, microcirculatory dysfunction, and distant-organ injury substantially increase morbidity and mortality. The revised Atlanta classification categorizes acute pancreatitis as mild, moderately severe, or severe according to the presence of local complications and the duration of organ failure [3]. While clinically robust, this classification is partly retrospective because persistent organ failure can only be confirmed after it has continued for more than 48 hours. Early risk stratification is therefore central to effective management. Patients likely to deteriorate may benefit from high-dependency monitoring, individualized fluid resuscitation, early

enteral nutrition, and prompt treatment of respiratory, circulatory, or renal dysfunction. Conventional severity-assessment methods, however, have important limitations. Ranson criteria require measurements collected over 48 hours, APACHE II is relatively complex, and contrast-enhanced computed tomography is not universally indicated at admission. C-reactive protein is commonly used as an inflammatory marker but generally provides its greatest prognostic value after 48 hours. The Bedside Index for Severity in Acute Pancreatitis offers a practical early assessment, but it still requires the evaluation of five separate variables and may not fully capture the intensity of the inflammatory response [4,5]. A simple, inexpensive biomarker obtainable from the first blood sample could consequently have substantial value, particularly in emergency departments and resource-constrained healthcare settings.

The neutrophil-to-lymphocyte ratio is calculated by dividing the absolute neutrophil count by the absolute lymphocyte count. It reflects two complementary components of acute physiological stress: neutrophilia caused by cytokine-mediated innate immune activation and relative lymphopenia associated with cortisol release, lymphocyte redistribution, and apoptosis. Zahorec first described NLR as a rapidly available indicator of systemic inflammation and physiological stress in critically ill patients [6]. In acute pancreatitis, pancreatic acinar injury promotes neutrophil recruitment, endothelial adhesion, oxidative burst, protease release, and cytokine amplification.

Simultaneous suppression of adaptive immune cell numbers may reflect more intense systemic stress and greater vulnerability to organ dysfunction.

Several observational studies have linked elevated NLR with pancreatic necrosis, intensive care admission, organ failure, and mortality. Azab et al. reported that higher NLR values were associated with adverse outcomes among patients with acute pancreatitis [7]. Suppiah et al. subsequently demonstrated a progressive relationship between NLR and disease severity and proposed an early prognostic threshold [8]. Nevertheless, reported cutoff values have varied because of differences in blood-sampling time, etiological distributions, disease-severity definitions, and laboratory platforms. The incremental value of NLR alongside BISAP, renal indices, CRP, physiological scoring systems, and imaging also remains incompletely defined.

The present study therefore assessed admission NLR across revised Atlanta severity categories, determined an optimal NLR threshold for severe acute pancreatitis, compared its discriminatory performance with established prognostic indicators, and evaluated its independent association with organ failure and adverse hospital outcomes.

Materials and Methods

This prospective observational study was conducted in the Departments of General Medicine at a tertiary-care hospital over 24 months. Consecutive eligible patients were enrolled at the time of hospital admission. Adults aged 18 years or older had been included when they presented within 24 hours of symptom onset and fulfilled at least two of the following diagnostic criteria: characteristic acute upper abdominal pain; serum amylase or lipase concentrations at least three times the upper reference limit; or imaging findings consistent with acute pancreatitis [3].

Patients had been excluded when they had chronic pancreatitis, pancreatic malignancy, post-endoscopic retrograde cholangiopancreatography pancreatitis, traumatic pancreatitis, active infection unrelated to pancreatitis, hematological disease, autoimmune or inflammatory disease, recent chemotherapy, corticosteroid or immunosuppressive therapy, pregnancy, or incomplete admission differential leukocyte data.

Clinical Assessment and Definitions: Demographic characteristics, comorbidities, symptom duration, etiology, vital signs, urine output, and organ-support requirements had been recorded using a standardized case-report form. The etiology of acute pancreatitis had been assigned using clinical history, liver biochemistry, abdominal ultrasonography, triglyceride concentration, serum calcium, and additional diagnostic tests when clinically indicated.

Disease severity had been classified as mild, moderately severe, or severe according to the revised Atlanta classification. Organ failure had been assessed using the modified Marshall scoring system. Transient organ failure had been defined as organ failure that resolved within 48 hours, whereas persistent organ failure had been defined as organ failure that continued for more than 48 hours. Local complications had included acute peripancreatic fluid collection, acute necrotic collection, pancreatic pseudocyst, and walled-off necrosis.

Laboratory and Imaging Measurements: Venous blood samples had been collected before substantial fluid resuscitation whenever feasible. Complete blood counts had been measured using an automated hematology analyzer. Absolute neutrophil and lymphocyte counts had been extracted directly from the differential leukocyte count. NLR had been calculated by dividing the absolute neutrophil count by the absolute lymphocyte count.

Serum electrolytes, corrected calcium, glucose, creatinine, blood urea nitrogen, liver-function parameters, amylase, and lipase had been analyzed using standard automated laboratory methods. CRP had been measured by immunoturbidimetry at approximately 48 hours after admission.

BISAP had been calculated during the first 24 hours of hospitalization. APACHE II had been calculated using the worst physiological values obtained during the same period. Contrast-enhanced computed tomography had been performed after 72–96 hours or earlier when clinically indicated. Radiological severity had been graded using the modified computed tomography severity index.

Study Outcomes: The primary outcome had been severe acute pancreatitis, defined by the presence of persistent organ failure. Secondary outcomes had included intensive care unit admission, pancreatic necrosis, local pancreatic or peripancreatic complications, duration of hospitalization, and in-hospital mortality.

The index prognostic test had been admission NLR. Treating clinicians had not used a prespecified NLR cutoff to determine escalation of treatment or admission to intensive care.

Sample Size Estimation: A minimum sample of 168 patients had been estimated to detect an area under the receiver-operating-characteristic curve of at least 0.75 for severe acute pancreatitis, assuming a severe-disease prevalence of approximately 15%, statistical power of 80%, and a two-sided alpha level of 0.05. Enrollment had been increased to 180 patients to account for incomplete clinical or laboratory assessments.

Statistical Analysis: Continuous variables had been examined for distribution using the Shapiro–Wilk test and had been expressed as mean with standard deviation or median with interquartile range, as appropriate. Comparisons between groups had been performed using one-way analysis of variance, the Kruskal–Wallis test, independent-samples t test, or Mann–Whitney U test.

Categorical variables had been presented as frequencies and percentages and had been compared using the chi-square test or Fisher exact test. Spearman rank correlation had been used to evaluate associations between NLR, ordinal disease severity, and other prognostic markers.

Discriminatory performance had been evaluated using receiver-operating-characteristic curves. Optimal prognostic thresholds had been determined using the Youden index. Variables considered clinically relevant or statistically significant in univariable analysis had been entered into a multivariable logistic regression model. Adjusted ORs and corresponding 95% CIs had been calculated. A two-sided p value below 0.05 had been considered statistically significant. Analyses had been performed using R version 4.3 and SPSS version 26.0.

Results

A total of 196 patients were screened during the study period. Sixteen patients were excluded because of delayed presentation, active infection, chronic pancreatitis, immunosuppressive treatment, or incomplete differential leukocyte counts. The final study cohort therefore comprised 180 patients. The mean age of the participants was 45.6±13.0 years, and 132 patients (73.3%) were men. Gallstone disease was the leading etiology and was identified in 77 patients (42.8%), followed by alcohol-related pancreatitis in 63 (35.0%), hypertriglyceridemia in 21 (11.7%), other identifiable causes in 13 (7.2%), and idiopathic pancreatitis in 6 patients (3.3%). According to the revised Atlanta classification, 108 patients (60.0%) had mild, 48 (26.7%) had moderately severe, and 24 (13.3%) had severe acute pancreatitis. Patients with severe disease were older and more frequently male, whereas the distribution of pancreatitis etiologies did not differ significantly between severity categories.

Inflammatory, renal, physiological, and imaging indicators worsened progressively with increasing disease severity. Median admission NLR increased from 5.2 (interquartile range [IQR]: 3.5–6.7) in mild disease to 6.5 (IQR: 4.8–9.6) in moderately severe disease and 10.7 (IQR: 8.1–15.3) in severe pancreatitis ($p<0.001$). Total leukocyte count, 48-hour CRP, blood urea nitrogen, serum creatinine, hematocrit, BISAP, APACHE II, and modified computed tomography severity index also increased significantly across severity categories. In contrast, corrected serum calcium decreased with increasing severity. Admission NLR correlated with revised Atlanta severity (Spearman $\rho=0.46$; $p<0.001$), BISAP ($\rho=0.25$; $p=0.001$), and 48-hour CRP ($\rho=0.34$; $p<0.001$), indicating that NLR captured related but nonidentical components of systemic inflammation and physiological disturbance.

Twenty-four patients (13.3%) developed persistent organ failure and consequently fulfilled the criteria for severe acute pancreatitis. Intensive care unit admission was required in 24 patients (13.3%), pancreatic necrosis was detected in 32 (17.8%), local complications occurred in 42 (23.3%), and 8 patients (4.4%) died during hospitalization. An admission NLR cutoff of 6.7 divided the cohort into 107 patients with lower NLR and 73 patients with higher NLR. Severe pancreatitis occurred in 22 of 73 patients (30.1%) with NLR ≥6.7 compared with 2 of 107 patients (1.9%) with NLR below 6.7

(p<0.001). All eight in-hospital deaths occurred in the higher-NLR group. Median hospital stay was also longer among patients with NLR ≥6.7 than among those with lower values, at 7.7 versus 6.1 days (p<0.001).

For the prediction of severe acute pancreatitis, admission NLR achieved an AUC of 0.853 (95% CI: 0.769–0.920). At the cutoff of 6.7, sensitivity was 95.8% and specificity was 66.0%, supporting the principal role of NLR as an early rule-out and clinical-triage marker. Its discriminatory performance was similar to BISAP, which had an AUC of 0.854, and was greater than that of total leukocyte count, which had an AUC of 0.829. However, its performance was numerically lower than that of 48-hour CRP and APACHE II, which had AUC values of 0.900 and 0.890, respectively.

In multivariable analysis, admission NLR remained independently associated with severe acute pancreatitis after adjustment for age, BISAP ≥3, and blood urea nitrogen ≥25 mg/dL. Every one-unit increase in NLR was associated with a 40% increase in the adjusted odds of severe disease (adjusted OR: 1.40; 95% CI: 1.15–1.72; p<0.001).

Table 1: Baseline Demographic and Etiological Characteristics According To Acute Pancreatitis Severity

Characteristic	Mild, n=108	Moderately severe, n=48	Severe, n=24	p value
Age, years, mean±SD	43.5±12.8	45.2±12.2	55.8±11.3	<0.001
Male sex, n (%)	73 (67.6)	37 (77.1)	22 (91.7)	0.043
Gallstone etiology, n (%)	48 (44.4)	15 (31.2)	14 (58.3)	0.078
Alcohol etiology, n (%)	40 (37.0)	18 (37.5)	5 (20.8)	0.294
Hypertriglyceridemia, n (%)	12 (11.1)	9 (18.8)	0 (0.0)	0.063
Other etiology, n (%)	4 (3.7)	5 (10.4)	4 (16.7)	0.052
Idiopathic etiology, n (%)	4 (3.7)	1 (2.1)	1 (4.2)	0.848

The baseline distribution showed that increasing severity was accompanied by older age and a higher proportion of men, while gallstone- and alcohol-related pancreatitis remained the dominant etiologies throughout the cohort. Etiology itself was not significantly associated with revised Atlanta severity, suggesting that the observed differences in NLR and clinical outcomes were unlikely to be explained solely by the underlying cause. The age gradient was clinically relevant and was therefore retained as a covariate in the multivariable model.

Table 2: Laboratory, Clinical Scoring, and Imaging Parameters across Severity Categories

Parameter	Mild	Moderately severe	Severe	p value
NLR at admission	5.2 (3.5–6.7)	6.5 (4.8–9.6)	10.7 (8.1–15.3)	<0.001
Total leukocyte count, ×10⁹/L	10.9±3.1	13.4±3.1	16.1±3.2	<0.001
CRP at 48 hours, mg/L	65.3 (49.7–93.1)	121.5 (84.8–186.0)	218.2 (165.1–329.4)	<0.001
Blood urea nitrogen, mg/dL	17.2±5.7	24.5±6.5	32.7±5.6	<0.001
Serum creatinine, mg/dL	0.9±0.3	1.2±0.3	1.8±0.3	<0.001
Hematocrit, %	40.6±4.1	43.1±3.3	44.7±3.1	<0.001
Corrected calcium, mg/dL	8.8±0.5	8.3±0.6	7.5±0.4	<0.001
BISAP score	1.0 (0.0–1.0)	2.0 (1.0–3.0)	3.0 (2.0–4.0)	<0.001
APACHE II score	4.9±2.6	7.6±2.6	10.1±2.1	<0.001
Modified CTSI	2.0 (1.0–3.0)	5.0 (4.0–6.0)	8.0 (6.0–9.0)	<0.001

A clear biological gradient was evident across all three severity categories.

Admission NLR, total leukocyte count, renal indices, hemoconcentration, BISAP, APACHE II, CRP, and modified computed tomography severity increased as the disease worsened, whereas corrected calcium decreased. The marked separation of NLR medians between mild and severe pancreatitis supported its use as an early inflammatory signal.

However, overlap between adjacent categories indicated that NLR should complement, rather than replace, physiological scoring and serial clinical reassessment.

Table 3: Clinical Outcomes Stratified By the Admission Nlr Cutoff

Outcome	NLR <6.7, n=107	NLR ≥6.7, n=73	p value
Severe acute pancreatitis	2 (1.9)	22 (30.1)	<0.001
Persistent organ failure	2 (1.9)	22 (30.1)	<0.001
Intensive care unit admission	10 (9.3)	14 (19.2)	0.093
Pancreatic necrosis	14 (13.1)	18 (24.7)	0.073
Local complication	19 (17.8)	23 (31.5)	0.050
In-hospital mortality	0 (0.0)	8 (11.0)	<0.001
Length of stay, days, median (IQR)	6.1 (4.7–7.8)	7.7 (5.5–13.3)	<0.001

Patients above the NLR threshold of 6.7 had an approximately sixteen-fold higher absolute prevalence of severe pancreatitis and accounted for every in-hospital death. The same group also experienced longer hospitalization and numerically more intensive care admissions, pancreatic necrosis, and local complications. The threshold therefore identified a clinically enriched high-risk population. Its moderate specificity meant that some patients would be over-triaged, but this trade-off may be acceptable when the purpose is early surveillance for evolving organ failure.

Table 4: Predictive Performance and Independent Predictors of Severe Acute Pancreatitis

Panel A. Receiver-operating-characteristic analysis

Predictor	AUC	95% CI	Optimal cutoff	Sensitivity, %	Specificity, %
NLR	0.853	0.769–0.920	6.7	95.8	66.0
BISAP score	0.854	0.786–0.911	2.0	95.8	66.0
CRP at 48 hours	0.900	0.823–0.957	132.9 mg/L	91.7	83.3
APACHE II	0.890	0.823–0.947	9.0	79.2	87.2
Total leukocyte count	0.829	0.747–0.907	13.4×10⁹/L	83.3	68.6

Panel B. Multivariable logistic regression

Independent predictor	Adjusted OR	95% CI	p value
Admission NLR, per one-unit increase	1.40	1.15–1.72	<0.001
BISAP ≥3	5.96	1.47–24.20	0.013
Blood urea nitrogen ≥25 mg/dL	27.61	4.26–178.82	<0.001
Age, per one-year increase	1.07	1.01–1.14	0.021

Receiver-operating-characteristic analysis demonstrated good discrimination for admission NLR, with performance comparable to BISAP and better than total leukocyte count alone. CRP at 48 hours and APACHE II were numerically more accurate, but both required either delayed sampling or more extensive data collection. Multivariable regression confirmed that NLR contributed prognostic information beyond age, BISAP, and blood urea nitrogen.

The wide confidence intervals for dichotomous clinical predictors reflected the limited number of severe events and warranted cautious interpretation.

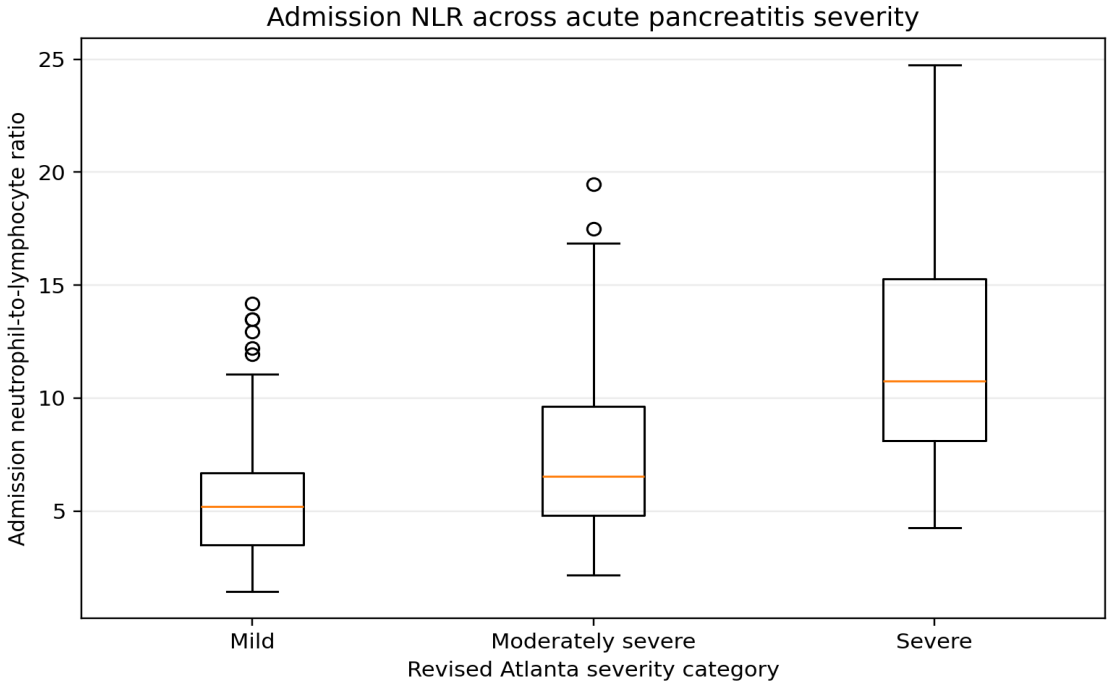

Figure 1: Distribution of admission NLR across revised Atlanta severity categories

The boxplot demonstrated a stepwise upward shift in admission NLR from mild to moderately severe and severe acute pancreatitis. Severe cases had both a higher central value and broader dispersion, consistent with heterogeneous intensity of systemic inflammation. Some overlap remained, particularly between mild and moderately severe disease, explaining why a single threshold could not

provide perfect classification. Nevertheless, the scarcity of severe cases at low NLR values supported the high sensitivity observed at the selected cutoff.

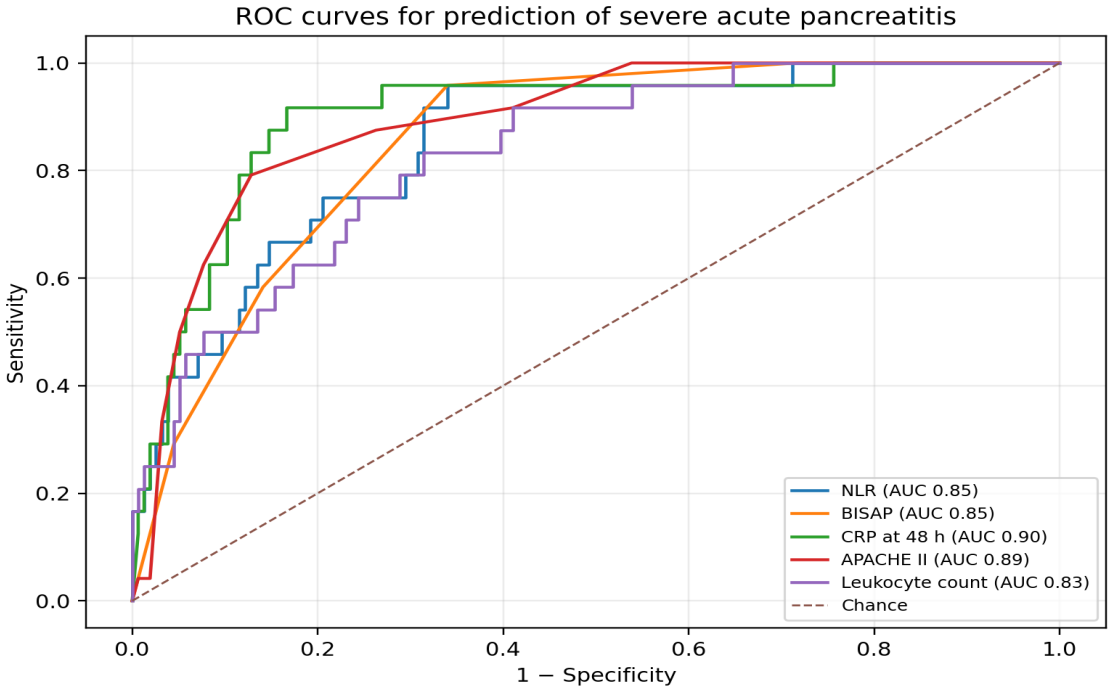

Figure 2: Receiver-operating-characteristic curves for prediction of severe acute pancreatitis

The receiver-operating-characteristic curves illustrated that admission NLR achieved clinically useful early discrimination without outperforming all established comparators. NLR and BISAP had nearly identical AUCs, while APACHE II and 48-hour CRP showed higher curves. NLR nevertheless offered an operational advantage because it was available from the initial complete blood count and required no additional testing beyond two absolute cell counts. Its most appropriate role is therefore rapid triage followed by confirmation through serial physiology, renal trends, and CRP.

Discussion

The present study demonstrated a strong and graded association between admission NLR and the severity of acute pancreatitis. Median NLR more than doubled between mild and severe disease, and a threshold of 6.7 identified severe pancreatitis with 95.8% sensitivity. Importantly, NLR remained independently associated with persistent organ failure after adjustment for age, BISAP, and blood urea nitrogen.

These findings support NLR as an immediately available indicator of early systemic inflammatory burden rather than merely a surrogate for total leukocytosis. The findings were consistent with the early study by Azab et al. [7], which associated elevated NLR with adverse outcomes in acute pancreatitis. Suppiah et al. [8] similarly observed progressive NLR elevation across severity categories and identified an optimal prognostic threshold. Jeon and Park [9] found that NLR measured early during hospitalization predicted unfavorable outcomes and suggested that serial NLR values could further strengthen prognostic assessment. Across these investigations, optimal cutoff values differed. Such variability was expected because NLR is influenced by the timing of blood sampling, hydration status, infection, steroid exposure, age, and the relative proportions of necrotizing, gallstone-related, or alcohol-related disease. The cutoff derived in the present study should consequently be interpreted as a locally generated triage threshold rather than a universal biological boundary.

The discriminatory performance of NLR was comparable to that of BISAP in the present cohort. This result was consistent with the original population-based development of BISAP by Wu et al. [5] and its prospective validation by Singh et al. [11], both of which established the value of simple early variables in predicting mortality and organ failure. Papachristou et al. [10] found that BISAP, APACHE II, Ranson criteria, and computed tomography severity had broadly similar prognostic utility. Mounzer et al. [12] further showed that several conventional scoring systems predicted persistent organ failure with only moderate accuracy. The present findings reinforce the principle that no isolated test provides complete prognostic certainty. NLR may simplify the initial

assessment but should be interpreted alongside organ function, physiological disturbance, and the subsequent clinical trajectory.

The biological plausibility of NLR as a severity marker is substantial. Pancreatic acinar injury activates damage-associated molecular pathways, complement, cytokines, and chemokines that recruit and prime circulating neutrophils. Activated neutrophils contribute to endothelial injury, increased capillary permeability, microvascular thrombosis, oxidative stress, and distant pulmonary and renal injury.

At the same time, catecholamine and cortisol responses promote lymphocyte redistribution and apoptosis. The NLR therefore combines the prognostic information contained in both innate immune activation and adaptive immune suppression. The lower AUC observed for total leukocyte count in this study supported this integrated biological interpretation. Zahorec's description of NLR as an index of physiological stress [6] and the normal-population data reported by Forget et al. [22] further contextualize why substantially elevated NLR values may indicate disproportionate systemic stress.

Established severity-assessment tools remained important comparators. Ranson et al. [13] introduced objective prognostic criteria for acute pancreatitis, but their complete application required 48 hours of observation. Larvin and McMahon [14] demonstrated the usefulness of APACHE II for repeated physiological assessment, whereas Knaus et al. [20] provided the broader critical-care framework on which APACHE II had been developed. Balthazar et al. [15] established the prognostic importance of computed tomography, and Brown et al. [17] identified hemoconcentration as an early marker of pancreatic necrosis and organ failure. Harshit Kumar and Singh Griwan [16] subsequently confirmed that BISAP, APACHE II, Ranson criteria, and modified computed tomography severity index each provided useful but imperfect prognostic information under revised Atlanta definitions.

In the present study, 48-hour CRP and APACHE II modestly outperformed NLR. This represented a partially contradictory but clinically coherent finding. NLR was available more rapidly and required minimal calculation, whereas delayed biomarkers and multiparametric physiological scores provided greater specificity. The purpose of NLR should therefore not be to displace established severity tools but to provide an immediate, inexpensive risk signal before more comprehensive information becomes available.

The clinical implication of an elevated NLR is not that every patient requires intensive care admission. Rather, an NLR ≥6.7 should prompt more frequent assessment of vital signs, careful monitoring of fluid balance and urine output, repeat hematological and renal testing, and early evaluation for respiratory, circulatory, or renal dysfunction. Contemporary clinical guidelines emphasize early risk assessment, enteral nutrition, and treatment based on evolving organ failure rather than prophylactic invasive intervention [4,18,19]. NLR is particularly suitable for this staged approach because it is inexpensive, reproducible, and readily accessible even when advanced imaging or complex scoring systems are unavailable.

The present study had several limitations. First, it was conducted at a single tertiary-care center, and the number of patients with severe disease was relatively small. Second, the derived NLR threshold was not evaluated in an external validation cohort. Third, NLR may have been affected by unmeasured factors such as occult infection, smoking, obesity, medication exposure, and prehospital fluid resuscitation. Fourth, only admission NLR was evaluated as the primary index, whereas serial changes in NLR may have provided additional prognostic information. Fifth, computed tomography was performed according to clinical indication rather than uniformly in every patient, potentially introducing verification bias.

Finally, the observational design established an association between NLR and severity but could not establish a causal relationship.

Future multicentre studies should validate population-specific thresholds, assess changes in NLR during the first 24–72 hours, and investigate whether combining NLR with BISAP or electronic early-warning systems improves model calibration and discrimination. Interventional studies should also determine whether NLR-guided monitoring strategies improve clinical decision-making, resource allocation, or patient-centered outcomes.

Conclusion

Admission neutrophil-to-lymphocyte ratio was strongly and independently associated with severe acute pancreatitis, persistent organ failure, prolonged hospitalization, and in-hospital mortality. An NLR cutoff of 6.7 provided high sensitivity and identified a subgroup with markedly increased risk while remaining immediately obtainable from the initial complete blood count. NLR did not replace 48-hour CRP, APACHE II, imaging, or repeated clinical assessment, but it provided a practical first-line triage signal with performance comparable to BISAP. Incorporating NLR into early bedside evaluation may improve prioritization of monitoring and organ-support readiness, particularly in resource-limited settings.

External validation and evaluation of serial NLR trajectories are required before universal threshold-based implementation.

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