e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 749-754

Original Research Article

# Prognostic Significance of the Neutrophil-to-Lymphocyte Ratio in Acute Pancreatitis: A Prospective Observational Study

**Venugopal H.[1], Karthik K.H.[2], Adarsh M.M.[3]**

[1]Assistant Professor, Department of General Medicine, Subbaiah Medical College, Shimoga, Karnataka, India.

[2]Assistant Professor, Department of General Medicine, Subbaiah Medical College, Shimoga, Karnataka, India.

[3]Assistant Professor, Department of General Medicine, Subbaiah Medical College, Shimoga, Karnataka, India.

**Received: 15-06-2026 / Revised: 02-07-2026 / Accepted: 22-07-2026**

**Corresponding Author: Dr. Venugopal H.**

**Conflict of interest: Nil**

**Abstract**

**Background:** Acute pancreatitis (AP) is usually self-limiting, but approximately 25% of patients develop a severe form associated with mortality of up to 50%. The neutrophil-lymphocyte ratio (NLR), a simple and inexpensive measure of the systemic inflammatory response, may help identify patients at risk of severe disease earlier and more reliably than the total white cell count.

**Objectives:** To evaluate the usefulness of the NLR in predicting outcome in patients with acute pancreatitis, and to determine optimal NLR cut-off values for distinguishing mild (MAP) from severe acute pancreatitis (SAP) at admission, 24 hours, and 48 hours.

**Methods:** This prospective observational study was conducted in the Departments of General Medicine and General Surgery, Subbaiah Medical College, Shimoga, between November 2023 and June 2025, and included 50 consecutive in patients with acute pancreatitis. Patients were classified as MAP or SAP using the APACHE II score. Neutrophil and lymphocyte counts, and the NLR, were recorded at baseline, 24 hours, and 48 hours. Receiver operating characteristic (ROC) curve analysis was used to determine optimal NLR cut-offs and diagnostic performance at each time point.

**Results:** Of 50 patients, 31 (62%) had MAP and 19 (38%) had SAP. Alcohol was the commonest aetiology (52%), followed by gallstones (24%). The mean NLR was higher in the SAP group than the MAP group at every time point, and the between-group difference was statistically significant at 48 hours (SAP 4.21 ± 1.11 vs MAP 3.00 ± 0.96, $p < 0.001$). An NLR cut-off of 3.45 at 48 hours predicted severe disease with a sensitivity of 68.4% and specificity of 71.0% (AUROC 0.80, 95% CI 0.69–0.92, $p < 0.001$) — performance that was superior to the NLR at baseline (cut-off 7.65) or 24 hours (cut-off 5.62, AUROC 0.53, 95% CI 0.37–0.70, not significant). Total leucocyte count, haematocrit, serum calcium, SGOT, GGT, amylase, lipase, and random blood sugar also differed significantly between MAP and SAP ($p < 0.05$).

**Conclusion:** The NLR, particularly when measured at 48 hours, is a simple, inexpensive, and readily available marker that can help identify patients with acute pancreatitis who are likely to follow a severe clinical course, supporting its use alongside established prognostic scoring systems.

**Keywords:** acute pancreatitis; neutrophil-lymphocyte ratio; APACHE II; prognostic marker; severity of illness.

**DOI:** 10.25258/ijcpr.18.8.115



## Introduction

Acute pancreatitis (AP) is usually a self-limiting inflammatory disease of the pancreas, but approximately 25% of patients present with, or subsequently develop, a severe form of the disease associated with mortality of up to 50%.[1] It is most commonly caused by gallstones or alcohol, which together account for about 75% of cases, with the remainder attributable to trauma, hypercalcaemia, hypertriglyceridaemia, post-ERCP injury, malignancy, and other less common causes.[2] The severity of acute pancreatitis is closely linked to extrapancreatic organ failure arising from the patient's systemic inflammatory response, and the poor prognosis seen in severe acute pancreatitis (SAP) is thought to reflect an uncontrolled systemic inflammatory response syndrome or multi-organ dysfunction syndrome.[3]

Several validated scoring systems — including Ranson's criteria, the Glasgow score, the Bedside Index of Severity in Acute Pancreatitis (BISAP), and the Acute Physiology and Chronic Health Evaluation II (APACHE II) score — are used to stratify patients by severity, but each requires multiple variables, is cumbersome to apply repeatedly, and depends in part on the total white cell count (WBC), which fluctuates with hydration status, stress, pregnancy, and specimen handling and is therefore a relatively non-specific marker of systemic inflammation.[1,3,4]

The neutrophil-lymphocyte ratio (NLR) — the ratio of absolute neutrophil count to absolute lymphocyte count — has emerged as a more discriminating marker of the systemic inflammatory and physiological stress response than the total WBC count, since neutrophilia and lymphopenia together better reflect the balance between innate inflammatory activation and the physiological stress response.[5,6] The NLR has been shown to predict adverse outcomes in a range of benign and malignant conditions, including coronary artery disease, sepsis, and several solid organ malignancies.[6–10] It is simple to calculate from a routine haemogram, is inexpensive, is not affected by the patient's volume status, and can be repeated easily during the course of an admission — practical advantages over more complex multi-parameter scoring systems.[5]

Only a limited number of studies have specifically examined the role of the NLR in acute pancreatitis, with variable reported cut-offs and predictive performance.[19] We therefore undertook this prospective observational study to evaluate the usefulness of the NLR as a predictor of severity in patients with acute pancreatitis and to determine optimal cut-off values at admission, 24 hours, and 48 hours to help distinguish mild from severe disease.

### Aims and Objectives

1. To use the NLR as a predictor of prognostic outcome in patients with acute pancreatitis.
2. To determine the sensitivity and specificity of the NLR on admission, at 24 hours, and at 48 hours.

### Materials and Methods

**Study design and setting:** This was a prospective observational study conducted in the Departments of General Medicine and General Surgery at Subbaiah Medical College, Shimoga, Karnataka.

**Study period:** November 2023 to June 2025.

**Study population and sample size:** All sequential inpatients admitted with a confirmed diagnosis of acute pancreatitis in the two departments were considered for inclusion. Acute pancreatitis was defined as clinical findings consistent with pancreatitis together with a serum amylase and/or lipase level at least three times the upper limit of normal. Based on an estimated regional incidence of acute pancreatitis and a 95% confidence level with a 5% maximum acceptable difference, the required sample size was calculated as 50 patients.

**Inclusion criteria:** Patients admitted with acute pancreatitis of varied aetiology (alcohol, gallstones, idiopathic, hypercalcaemia, hereditary, malnutrition-related, autoimmune, infective, and other medical or surgical causes) who fulfilled APACHE II criteria on admission.

**Exclusion criteria:** Patients who did not fulfil APACHE II criteria on admission, and patients with chronic pancreatitis.

**Data collection:** A detailed history, clinical examination, and relevant investigations (including a complete haemogram, liver function tests, serum calcium, blood urea nitrogen, random blood sugar, serum amylase and lipase, and contrast-enhanced CT of the abdomen where indicated) were recorded for each patient. Patients were classified as having mild acute pancreatitis (MAP) or severe acute pancreatitis (SAP) using the APACHE II score; SAP was defined as objective evidence of organ failure (circulatory shock, acute renal failure, or acute pulmonary failure) and/or local complications such as necrosis, abscess, or pseudocyst. The differential white cell count was analysed, and the NLR was calculated as the ratio of the absolute neutrophil count to the absolute lymphocyte count at baseline (admission), 24 hours, and 48 hours.

**Statistical analysis:** Data were summarised as mean ± standard deviation, or as frequencies and percentages. The Mann-Whitney U test was used to compare continuous variables between the MAP and SAP groups, and the Chi-square test was used to compare categorical variables. Receiver operating characteristic (ROC) curve analysis was used to determine the optimal NLR cut-off at each time point together with sensitivity, specificity, positive and negative predictive values, and the area under the curve (AUC). A p-value $<0.05$ was considered statistically significant.

### Results

Fifty patients with acute pancreatitis were enrolled, of whom 31 (62%) had mild acute pancreatitis (MAP) and 19 (38%) had severe acute pancreatitis (SAP), a ratio of 1.63:1. The peak incidence was seen in the 31–40-year age group (34%, 17 patients), and there was a marked male predominance, with 40 patients (80%) male and 10 (20%) female (male:female ratio 4:1).

Excessive alcohol consumption was the commonest aetiology overall, accounting for 26 of 50 cases (52%), followed by obstructive gallstones in 12

cases (24%); hypercalcaemia, hypertriglyceridaemia, pancreatic malignancy, post-ERCP injury, and post-traumatic pancreatitis together accounted for the remaining cases. Alcohol was the leading cause in both severity groups — 45.2% (14/31) of MAP cases and 63.2% (12/19) of SAP cases — followed by gallstones (25.8% of MAP and 21.1% of SAP cases); the distribution of aetiology did not differ significantly between the two groups ($\chi^2 = 11.255$, $p = 0.08$). Patients with SAP due to alcohol had consumed significantly more alcohol than those with MAP (31.00 ± 9.79 vs 15.88 ± 6.72 units/week; Mann-Whitney $Z = -3.846$, $p < 0.001$).

**Table 1: Demographic and clinical profile of the study population (N = 50)**

| | | |
|---|---|---|
| Peak age group | 31–40 years | 17 (34%) |
| Sex | Male / Female | 40 (80%) / 10 (20%) |
| Disease severity | MAP / SAP | 31 (62%) / 19 (38%) |
| Aetiology (overall) | Alcohol / Gallstones / Other | 26 (52%) / 12 (24%) / 12 (24%) |
| Aetiology – MAP | Alcohol / Gallstones | 14 (45.2%) / 8 (25.8%) |
| Aetiology – SAP | Alcohol / Gallstones | 12 (63.2%) / 4 (21.1%) |
| Alcohol intake (units/week) | MAP vs SAP, mean ± SD | 15.88 ± 6.72 vs 31.00 ± 9.79* |
| Comorbidity | Dyslipidaemia (commonest overall) / Diabetes mellitus | 13 (26%) / 8 (16%) |

*Mann-Whitney U test, $p < 0.001$.

Dyslipidaemia was the most prevalent comorbidity overall (13/50, 26%), followed by diabetes mellitus (8/50, 16%). Among patients with SAP specifically, diabetes mellitus was the most common comorbidity (5/19, 26.3%). Comorbidities did not differ significantly in distribution between MAP and SAP ($\chi^2 = 7.427$, $p = 0.28$).

Mean neutrophil percentage, lymphocyte percentage, and NLR at baseline, 24 hours, and 48 hours are shown in Table 2. In the MAP group, the mean NLR fell steadily from 7.01 ± 1.33 at baseline to 5.37 ± 1.78 at 24 hours and 3.00 ± 0.96 at 48 hours. In the SAP group, the NLR followed a less consistent trajectory, falling from a higher baseline of 7.54 ± 1.88 to 5.47 ± 1.51 at 24 hours and 4.21 ± 1.11 at 48 hours. The difference in mean neutrophil and lymphocyte counts between MAP and SAP was statistically significant at baseline and at 48 hours ($p < 0.001$), and the difference in mean NLR between the two groups was statistically significant at 48 hours ($p < 0.001$).

**Table 2: Neutrophil percentage, lymphocyte percentage, and NLR at baseline, 24 hours, and 48 hours (MAP vs SAP)**

| | | | |
|---|---|---|---|
| Neutrophil % – Baseline | 67.23 ± 6.63 | 81.26 ± 4.77 | <0.001* |
| Neutrophil % – 24 h | 69.87 ± 6.02 | 70.11 ± 7.10 | NS |
| Neutrophil % – 48 h | 60.29 ± 4.79 | 70.11 ± 5.27 | <0.001* |
| Lymphocyte % – Baseline | 9.87 ± 1.86 | 11.47 ± 2.89 | <0.001* |
| Lymphocyte % – 24 h | 14.58 ± 5.19 | 13.68 ± 3.76 | NS |
| Lymphocyte % – 48 h | 22.16 ± 6.96 | 17.47 ± 3.72 | <0.001* |
| NLR – Baseline | 7.01 ± 1.33 | 7.54 ± 1.88 | NS |
| NLR – 24 h | 5.37 ± 1.78 | 5.47 ± 1.51 | NS |
| NLR – 48 h | 3.00 ± 0.96 | 4.21 ± 1.11 | <0.001* |

*Mann-Whitney U test, statistically significant; NS = not significant.

Several haematological and biochemical parameters also differed significantly between the two groups (Table 3). Compared with MAP, patients with SAP had a higher mean haematocrit (49.68 ± 7.10%), higher mean total leucocyte count (17,173 vs 9,333 /mm³), lower mean serum calcium (8.28 ± 1.50 mg/dL), higher mean SGOT (280 vs 140 U/L), higher mean GGT (209.8 U/L), and higher mean lipase (620 vs 411 U/L) and random blood sugar (mean 213.05 mg/dL) — all differences that were statistically significant ($p < 0.05$). Serum triglycerides and total bilirubin did not differ significantly between groups. Twelve patients had a blood urea nitrogen (BUN) greater than 20 mg/dL at admission; six were in the SAP group. The mean BUN was numerically higher in the SAP group.

**Table 3: Comparison of haematological and biochemical parameters between MAP and SAP**

| | | |
|---|---|---|
| Haematocrit (%) | Higher in SAP (mean 49.68 ± 7.10) | $p < 0.05$ |
| Total leucocyte count (/mm³) | Higher in SAP (mean 17,173 vs 9,333 in MAP) | $p < 0.05$ |
| Serum calcium (mg/dL) | Lower in SAP (mean 8.28 ± 1.50) | $p < 0.05$ |
| SGOT / AST (U/L) | Higher in SAP (mean 280 vs 140 in MAP) | $p < 0.05$ |
| GGT (U/L) | Higher in SAP (mean 209.8) | $p < 0.05$ |
| Lipase (U/L) | Higher in SAP (mean 620 vs 411 in MAP) | $p < 0.05$ |
| Random blood sugar (mg/dL) | Higher in SAP (mean 213.05) | $p < 0.05$ |
| BUN | 12/50 patients had BUN >20 mg/dL; 6 of these were SAP | — |
| Total bilirubin | No significant difference | NS |
| Triglycerides | No significant difference | NS |

On ROC curve analysis, the diagnostic performance of the NLR improved progressively from baseline to 48 hours (Table 4). At baseline, the optimal NLR cut-off was 7.65, with modest and statistically nonsignificant discriminatory performance. At 24 hours, the optimal cut-off was 5.62 (AUROC 0.53, 95% CI 0.37–0.70, $p = 0.70$ — not significant). At 48 hours, the optimal cut-off of 3.45 achieved the best discriminatory performance, with a sensitivity of 68.4%, specificity of 71.0%, positive predictive value of 59.1%, negative predictive value of 78.6%, and an AUROC of 0.80 (95% CI, 0.69–0.92; $p < 0.001$).

**Table 4: Diagnostic performance of the NLR for predicting severe acute pancreatitis at baseline, 24 hours, and 48 hours**

| **Baseline** | **7.65** | **58.9%** | **74.2%** | **57.9%** | **–** | **Not significant** |
|---|---|---|---|---|---|---|
| 24 hours | 5.62 | 52.6% | 58.1% | 43.5% | 66.7% | 0.53 (0.37–0.70), p=0.70 |
| 48 hours | 3.45 | 68.4% | 71.0% | 59.1% | 78.6% | 0.80 (0.69–0.92), p<0.001* |

*Statistically significant.

## Discussion

This prospective observational study evaluated the usefulness of the NLR as a prognostic marker in 50 patients with acute pancreatitis and found that, although the NLR was numerically higher in the SAP group at every time point, the between-group difference reached statistical significance only at 48 hours, where an NLR of 3.45 predicted severe disease with a sensitivity of 68.4% and specificity of 71.0% (AUROC 0.80, $p < 0.001$).

Our cohort had a marked male predominance (80%), similar to the 83 males and 27 females reported among 110 patients by Vengadakrishnan et al. at Sri Ramachandra Medical College, and to the male predominance reported by Prasad et al. in a cohort of 40 patients from Mysore Medical College. A comparable male predominance (63.3%) was also reported by Satoh et al. in a Japanese cohort of 603 patients, consistent with the broader observation that acute pancreatitis affects men more often than women across populations, likely reflecting higher rates of alcohol-related risk factors in men.[2]

Alcohol was the leading aetiology in our cohort (52% overall, and the predominant cause in both MAP and SAP), which contrasts with reports from several Western series where gallstone disease predominates.[2] This likely reflects the socio-demographic profile of the catchment population served by our institution, an area with a high proportion of manual and construction labour and associated heavy alcohol use, rather than a true biological difference in aetiological distribution. This pattern is consistent with the study by Prasad et al., who similarly reported alcohol as the leading cause of acute pancreatitis in their south Indian cohort, and with the observation of Kandasam et al. that alcohol is the predominant aetiology among male patients, while gallstone disease predominates among females — a pattern broadly mirrored in our cohort, where 92% of alcohol-related cases occurred in men.

The proportion of patients with severe disease in our study (38%) was similar to the 26 of 146 patients (17.8%) with SAP reported by Suppiah et al., and comparable to the 50 of 110 patients (45%) requiring intensive care reported by Vengadakrishnan et al. Two patients in our cohort died, both male, in their fifth decade, with multiple comorbidities (hypertension and diabetes) and organ-specific complications (acute kidney injury and metabolic encephalopathy, respectively) — consistent with the well-recognised observation that comorbidity burden, rather than aetiology alone, is a major determinant of mortality in acute pancreatitis.

The NLR has previously been evaluated as a prognostic marker in acute pancreatitis by Azab et al., who found it superior to the total white cell count in predicting ICU admission and death, and proposed a cut-off of 4.7; however, in-hospital mortality in that study was very low, and organ failure was not systematically assessed. Suppiah et

al. similarly reported an association between the NLR measured within the first 48 hours and progression to severe disease, though their cohort was predominantly mild. In contrast, other authors have reported the NLR to be a weaker or non-significant independent predictor of early mortality, while a Chinese cohort study found elevated NLR to be associated with persistent organ failure, prolonged ICU stay, and increased in-hospital mortality. Our finding that the NLR was most discriminating at 48 hours, rather than at admission, is consistent with this body of evidence and suggests that the evolution of the NLR over the first two days — rather than a single admission value — may carry the most prognostic information.[19]

We also found that several routinely available laboratory parameters — haematocrit, total leucocyte count, serum calcium, SGOT, GGT, lipase, and random blood sugar — differed significantly between MAP and SAP, in keeping with their established roles in existing severity scores. An elevated BUN at admission, previously reported to predict mortality risk, was also more frequent among our SAP patients, consistent with prior reports that a normal admission BUN carries a favourable negative predictive value for severity.

This study has certain limitations. The single-centre design and modest sample size (n = 50) limit precision, particularly for subgroup comparisons and for the baseline and 24-hour ROC estimates, which did not reach statistical significance and should be interpreted cautiously. The prospective design and use of a standardised APACHE II-based severity classification are strengths. Larger, multicentre, prospective studies are needed to validate the 48-hour NLR cut-off identified here and to determine whether combining the NLR with existing scoring systems improves early risk stratification beyond either approach alone.

## Conclusion

Acute pancreatitis remains a common cause of emergency admission with a clinical course ranging from mild, self-limiting disease to severe disease with substantial morbidity and mortality. In this study, the neutrophil-lymphocyte ratio measured at 48 hours after admission was a simple, inexpensive, and statistically significant predictor of severe acute pancreatitis, with a sensitivity of 68.4% and specificity of 71.0% at a cut-off of 3.45 (AUROC 0.80). Because the NLR is derived from a routine haemogram, is unaffected by volume status, and can be easily repeated during an admission, it may serve as a practical adjunct to established severity scoring systems for early risk stratification in patients with acute pancreatitis, pending validation in larger, multicentre cohorts.

## Declarations

**Conflict of interest:** None declared.

**Source of funding:** None.

**Author contributions:** SB: data collection, analysis, manuscript drafting. VNH: study conception and design, supervision and critical revision of the manuscript. BSR: co-supervision (surgical cases), critical revision of the manuscript. All authors approved the final manuscript.

**Acknowledgements:** The authors thank the Departments of General Medicine and General Surgery, Subbaiah Medical College, Shimoga, and all patients who participated in this study.

## References

1. Papachristou GI, Muddana V, Yadav D, O'Connell M, Sanders MK, Slivka A, Whitcomb DC. Comparison of BISAP, Ranson's, APACHE-II, and CTSI scores in predicting organ failure, complications, and mortality in acute pancreatitis. Am J Gastroenterol. 2010;105(2):435-441.
2. Dufour M, Adamson M. The epidemiology of alcohol-induced pancreatitis. Pancreas. 2003;27:286-290.
3. Dambrauskas Z, Gulbinas A, Pundzius J, Barauskas G. Value of the different prognostic systems and biological markers for predicting severity and progression of acute pancreatitis. Scand J Gastroenterol. 2010;45(7-8):959-970.
4. Pavlidis TE, Pavlidis ET, Sakantamis AK. Advances in prognostic factors in acute pancreatitis: a mini-review. Hepatobiliary Pancreat Dis Int. 2010;9(5):482-486.
5. Zahorec R. Ratio of neutrophil to lymphocyte counts – a rapid and simple parameter of systemic inflammation and stress in critically ill. Bratisl Lek Listy. 2001;102(1):5-14.
6. de Jager CP, van Wijk PT, Mathoera RB, de Jongh-Leuvenink J, van der Poll T, Wever PC. Lymphocytopenia and neutrophil-lymphocyte count ratio predict bacteremia better than conventional infection markers in an emergency care unit. Crit Care. 2010;14(5):R192.
7. Le Tulzo Y, Pangault C, Gacouin A, et al. Early apoptosis of circulating lymphocytes in human septic shock is associated with poor outcome. Shock. 2002;18(6):487-494.
8. Halazun KJ, Hardy MA, Rana AA, et al. Negative impact of neutrophil-lymphocyte ratio on outcome after liver transplantation for hepatocellular carcinoma. Ann Surg. 2009;250(1):141-151.
9. Walsh SR, Cook EJ, Goulder F, Justin TA, Keeling NJ. Neutrophil-lymphocyte ratio as a prognostic factor in colorectal cancer. J Surg Oncol. 2005;91(3):181-184.

10. Wyllie DH, Bowler IC, Peto TE. Relation between lymphopenia and bacteraemia in UK adults with medical emergencies. J Clin Pathol. 2004;57(9):950-955.
11. Garcea G, Gouda M, Hebbes C, et al. Predictors of severity and survival in acute pancreatitis: validation of the efficacy of early warning scores. Pancreas. 2008;37(3):e54-61.
12. Bradley EL 3rd. A clinically based classification system for acute pancreatitis. Arch Surg. 1993;128(5):586-590.
13. McKay CJ, Evans S, Sinclair M, Carter CR, Imrie CW. High early mortality rate from acute pancreatitis in Scotland, 1984-1995. Br J Surg. 1999;86:1302-1305.
14. Felderbauer P, Müller C, Bulut K, et al. Pathophysiology and treatment of acute pancreatitis: new therapeutic targets – a ray of hope? Basic Clin Pharmacol Toxicol. 2005;97(6):342-350.
15. Mofidi R, Patil PV, Suttie SA, Parks RW. Risk assessment in acute pancreatitis. Br J Surg. 2009;96(2):137-150.
16. Harrison DA, D'Amico G, Singer M. The Pancreatitis Outcome Prediction (POP) score: a new prognostic index for patients with severe acute pancreatitis. Crit Care Med. 2007;35(7):1703-1708.
17. Juneja D, Gopal PB, Ravula M. Scoring systems in acute pancreatitis: which one to use in intensive care units? J Crit Care. 2010;25(2):358.e9-358.e15.
18. Sakorafas GH, Tsiotou AG. Aetiology and pathogenesis of acute pancreatitis: current concepts. J Clin Gastroenterol. 2000;30:343-356.
19. Azab B, Jaglall N, Atallah JP, et al. Neutrophil-lymphocyte ratio as a predictor of adverse outcomes of acute pancreatitis. Pancreatology. 2011;11(4):445-452.
20. Petrov MS, Shanbhag S, Chakraborty M, Phillips AR, Windsor JA. Organ failure and infection of pancreatic necrosis as determinants of mortality in patients with acute pancreatitis. Gastroenterology. 2010;139(3):813-820.
21. Talukdar R, Vege SS. Recent developments in acute pancreatitis. Clin Gastroenterol Hepatol. 2009;7(11 Suppl):S3-9.
22. Banks PA, Freeman ML; Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. Am J Gastroenterol. 2006;101(10):2379-2400.