

Comparison of neutrophil lymphocyte ratio and platelet lymphocyte ratio with Ranson criteria in acute biliary pancreatitis

Mehmet Ali Karacaer¹  Özgür Kurtkulağı^{1*}  Gökhan Gökten¹  Mehmet Emrah Bayam² 

¹Department of General Surgery, Çanakkale Mehmet Akif Ersoy State Hospital, Çanakkale, Türkiye

²Department of General Surgery, Bursa Yüksek İhtisaa Training and Research Hospital, Bursa, Türkiye

ABSTRACT

Aim: To evaluate the diagnostic value of the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) in predicting the severity of acute biliary pancreatitis, in comparison with the Ranson scoring system.

Methods: A total of 353 patients diagnosed with acute biliary pancreatitis between January 2019 and December 2023 were retrospectively analyzed. Demographic data, laboratory findings, NLR, PLR, and Ranson scores were recorded. Patients were grouped as mild and severe according to clinical severity. The diagnostic performances of NLR, PLR, and Ranson score were assessed using receiver operating characteristic (ROC) curve analysis.

Results: NLR, PLR, and Ranson scores were significantly higher in the severe pancreatitis group compared to the mild group ($p < 0.001$ for all). ROC analysis revealed that Ranson score had the highest area under the curve (AUC = 0.85), followed by NLR (AUC = 0.82) and PLR (AUC = 0.75). Sensitivity and specificity values were acceptable for all three parameters in distinguishing severe disease.

Conclusion: NLR and PLR are simple, cost-effective, and accessible inflammatory markers that may assist in predicting disease severity in acute biliary pancreatitis. Their diagnostic power is comparable to the Ranson scoring system and may support early risk stratification in clinical practice.

Keywords: Inflammation, lymphocytes, acute biliary pancreatitis, neutrophils, platelets.

✉ Özgür Kurtkulağı*

Department of General Surgery,
Çanakkale Mehmet Akif Ersoy State Hospital,
Çanakkale, Türkiye
E-mail: ozgurkurt115@gmail.com

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1. Introduction

Acute pancreatitis is an inflammatory disease in which normally inactive digestive enzymes become prematurely activated within the pancreas, leading to autodigestion and a cascade of systemic inflammatory responses.

This process can result in local, regional, and systemic complications that significantly contribute to the disease burden [1,2]. Despite technological and therapeutic advancements, the morbidity and mortality associated with acute pancreatitis remain high [3]. The majority of patients experience a mild form, often limited to interstitial edema and minimal necrosis at the microscopic level. However, in cases involving severe pancreatic or peripancreatic necrosis, the prognosis worsens considerably [4,5].

The retroperitoneal location of the pancreas complicates both diagnosis and management of

acute pancreatitis. As a cause of acute abdomen, it poses diagnostic challenges due to its deep anatomic position and overlapping symptoms with other abdominal emergencies. Moreover, the progression to systemic complications and organ dysfunction further complicates management, requiring rapid and accurate assessment tools.

Delays in diagnosis, classification discrepancies, comorbidities, and institutional variations in patient care pathways continue to hinder optimal treatment even in well-resourced healthcare settings. Accurate early risk stratification may help reduce hospital stay, avoid unnecessary interventions, and guide clinical decisions such as the need for intensive care admission, surgical or endoscopic intervention (e.g., ERCP), imaging modalities, and antibiotic use [6].

To address these clinical demands, a scoring or biomarker-based system that is accurate, easily applicable, and cost-effective is needed. In recent years, inflammation-based hematologic markers such as the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have been increasingly investigated as prognostic indicators in a wide range of inflammatory conditions, including COVID-19 infection, diabetes mellitus, thyroiditis, inflammatory bowel diseases, and cancer [7-14]. These markers are inexpensive, routinely available, and provide rapid insights into systemic inflammatory status.

This study aimed to investigate the relationship between NLR, PLR, and disease severity in acute biliary pancreatitis, using the Ranson scoring system as a comparative reference. We hypothesize that elevated NLR and PLR values may reflect early inflammatory burden and correlate with higher Ranson scores, contributing to better risk stratification and clinical decision-making.

2. Materials and methods

Patient records from the Department of General Surgery at Bursa Yüksek İhtisas Training and Research Hospital were retrospectively reviewed. A total of 386 patients who were diagnosed with acute pancreatitis between 2011 and 2017 were initially identified. These patients were etiologically classified, and records of 353 patients diagnosed with acute biliary pancreatitis were evaluated through hospital files and available contact information.

Ethical approval for our study was granted by the Clinical Research Ethics Committee of Bursa Yüksek İhtisaa Training and Research Hospital on 02/11/2016, with decision number 2016/19-05.

Patients were excluded from the study based on the following criteria: those with missing contact details or unreachable by phone (n=16), those diagnosed with acute biliary pancreatitis secondary to ERCP or cholecystectomy (n=15), and patients diagnosed with pancreatic malignancy (n=2).

The diagnosis of acute pancreatitis was established based on the presence of characteristic symptoms along with elevated serum amylase and/or lipase levels. During the imaging phase, patients found to have gallstones or common bile duct stones were classified as having acute biliary pancreatitis.

2.1. Diagnostic criteria for acute pancreatitis included:

- Epigastric or periumbilical pain radiating to the back,
- Nausea and vomiting following the onset of abdominal pain,
- Diffuse abdominal tenderness with voluntary or involuntary guarding,
- Serum or urine amylase levels three times above the upper limit of normal,

- Absence of acute cholecystitis and presence of biliary tract stones or sludge based on ultrasonographic evaluation,
- Confirmation of acute pancreatitis on contrast-enhanced abdominal computed tomography.

The number of days between admission and discharge (or death) was recorded for each patient. Additional data collected included age, sex, imaging findings, and duration of hospitalization. Ranson scores were calculated at the time of admission using established clinical and laboratory parameters. NLR and PLR values were calculated from routine complete blood count measurements.

2.2. Patients were stratified according to disease severity as follows:

- Mild acute pancreatitis: Ranson score < 3
- Severe acute pancreatitis: Ranson score ≥ 3

The following patient groups were excluded from the study:

1. Patients under the age of 18,
2. Patients without biliary sludge or gallstones in the gallbladder or bile ducts,
3. Patients with a history of alcohol use, hypercalcemia, or hyperlipidemia,
4. Patients with concurrent hyperlipidemia,
5. Patients with malignancy in the biliary tract, pancreas, or periampullary region,
6. Patients lacking contact information,
7. Patients who developed acute pancreatitis following ERCP,
8. Patients who had previously undergone biliary or pancreatic diversion surgery,
9. Patients who received treatment by the gastroenterology department,
10. Patients misdiagnosed at admission and diagnosed with acute pancreatitis only after 24 hours.

2.3. Statistical Analysis

All data were recorded using Microsoft Excel 2010 and analyzed using IBM SPSS version 21.0. Descriptive statistics were presented as means \pm standard deviation for continuous variables. ROC (receiver operating characteristic) curve analysis was used to evaluate the diagnostic performance of NLR, PLR, and Ranson scores in predicting severe acute pancreatitis. A p -value < 0.05 was considered statistically significant, and < 0.001 was accepted as highly significant. All results were presented with 95% confidence intervals.

3. Results

A total of 353 patients diagnosed with acute biliary pancreatitis were included in the study. Among them, 231 were female (65%) and 122 were male (34.6%). The mean age was 61.93 ± 16.92 years, ranging from 21 to 92 years. The mean age of female patients was 61.89 ± 17.72 , while that of males was 62.00 ± 15.35 , with no statistically significant difference between genders ($p = 0.953$).

Descriptive statistics of patients including hospitalization duration, glucose, AST, LDH, leukocyte, neutrophil, lymphocyte, platelet counts, NLR and PLR values are summarized in Table 1.

The distribution of Ranson scores among patients is shown in Table 2, with the majority of patients (86.5%) having a score of 0–2, and 13.5% having scores ≥ 3 , indicating severe pancreatitis.

To assess prognostic value, the median NLR value (6.9) was used as a cutoff. A total of 176 patients had NLR < 6.9 , while 177 had NLR ≥ 6.9 . Comparison of these groups revealed statistically significant differences in age, hospital stay, glucose, LDH, leukocyte, neutrophil, lymphocyte counts, and Ranson scores (all $p < 0.001$), as shown in Table 3.

Similarly, for PLR, the median value of 170.5 was used. Patients with $PLR \geq 170.5$ had significantly higher values of age, hospital stay duration, glucose, AST, LDH, leukocyte and neutrophil counts, and Ranson scores, and lower lymphocyte counts compared to those with $PLR < 170.5$ (Table 4).

According to Ranson scoring, 316 patients

had mild pancreatitis (Ranson 0–2), and 37 had severe pancreatitis (Ranson ≥ 3). Patients with severe pancreatitis had significantly higher age, hospital stay duration, glucose, AST, LDH, leukocyte, neutrophil counts, NLR, and PLR values compared to those with mild disease (Table 5). There was no statistically difference in platelet count between groups ($p = 0.349$).

Table 1. Descriptive statistics of laboratory and clinical parameters.

Variable	N	Minimum	Maximum	Mean \pm SD
Hospital Stay (days)	353	1	24	5.56 \pm 3.25
Glucose	353	59	539	146.09 \pm 65.94
AST	353	11	2796	209.75 \pm 257.09
LDH	353	124	2727	303.01 \pm 193.41
Leukocyte	353	4010	36900	12592.41 \pm 5386.70
Neutrophil	353	2440	33800	10207.96 \pm 5221.61
Lymphocyte	353	210	16200	1568.27 \pm 1178.04
Thrombocyte	353	110000	670000	252096.32 \pm 79029.49
NLR	353	0.50	98.92	9.92 \pm 10.10
PLR	353	8.10	1192.80	223.16 \pm 161.95

Table 2. Distribution of patients according to Ranson score.

Ranson Score	N	Percentage (%)
0	119	33.7
1	122	34.6
2	75	21.2
3	32	9.1
4	5	1.4
Total	353	100.0

Table 3. Comparison of clinical and laboratory parameters according to NLR Cutoff (6.9).

Parameter	NLR < 6.9 (n=176)	NLR \geq 6.9 (n=177)	p-value
Age	58.31 \pm 17.08	65.53 \pm 16.01	<0.001
Hospital Stay (days)	4.44 \pm 2.85	6.67 \pm 3.26	<0.001
Glucose	134.05 \pm 64.21	158.06 \pm 65.63	<0.001
AST	192.18 \pm 210.58	227.22 \pm 295.78	0.201
LDH	265.91 \pm 143.47	339.90 \pm 227.16	<0.001
Leukocyte	9900.63 \pm 3424.83	15268.98 \pm 5649.76	<0.001
Neutrophil	7039.72 \pm 2863.74	13358.31 \pm 5133.91	<0.001
Lymphocyte	2103.13 \pm 1413.74	1036.44 \pm 466.63	<0.001
Thrombocyte	251022.73 \pm 68993.49	253163.84 \pm 88067.97	<0.001
Ranson Score	0.74 \pm 0.85	1.46 \pm 1.03	<0.001

Table 4. Comparison of clinical and laboratory parameters according to PLR cutoff (170.5).

Parameter	PLR < 170.5 (n=174)	PLR ≥ 170.5 (n=179)	p-value
Age	58.75 ± 17.02	65.01 ± 16.28	<0.001
Hospital Stay (days)	4.76 ± 2.88	6.33 ± 3.41	<0.001
Glucose	130.91 ± 54.70	160.84 ± 72.42	<0.001
AST	172.91 ± 180.99	245.56 ± 310.25	0.008
LDH	264.80 ± 125.03	340.15 ± 236.57	<0.001
Leukocyte	11604.60 ± 4762.59	13552.63 ± 5784.01	<0.001
Neutrophil	8581.26 ± 4282.11	11789.22 ± 5567.32	<0.001
Lymphocyte	2142.01 ± 1407.32	1010.56 ± 429.98	<0.001
Thrombocyte	226954.02 ± 63575.25	276536.31 ± 84876.92	<0.001
Ranson Score	0.80 ± 0.91	1.39 ± 1.03	<0.001

Table 5. Comparison of clinical and laboratory parameters between mild (Ranson ≤2) and severe (Ranson ≥3) pancreatitis.

Parameter	Ranson ≤2 (n=316)	Ranson ≥3 (n=37)	p-value
Age	60.19 ± 16.82	76.78 ± 8.45	<0.001
Hospital Stay (days)	5.07 ± 2.82	9.73 ± 3.69	<0.001
Glucose	139.19 ± 61.23	204.97 ± 75.68	<0.001
AST	184.23 ± 199.81	427.65 ± 492.37	<0.001
LDH	280.03 ± 135.92	499.27 ± 399.83	<0.001
Leukocyte	12064.08 ± 4768.84	17104.59 ± 7832.64	<0.001
Neutrophil	9634.68 ± 4618.68	15104.05 ± 7239.71	<0.001
Lymphocyte	1612.88 ± 1211.71	1187.30 ± 745.42	0.037
Thrombocyte	253446.20 ± 79137.81	240567.57 ± 78208.95	0.349
NLR	9.18 ± 9.85	16.18 ± 10.23	<0.001
PLR	217.28 ± 162.33	273.43 ± 151.59	0.046

4. Discussion

The most common cause of acute pancreatitis is the presence of sludge or stones in the gallbladder or common bile duct. It is estimated that 3–8% of patients with symptomatic gallstones develop acute pancreatitis [7]. Gallbladder stones are the most preventable cause of acute pancreatitis, as shown in multiple studies [8,9]. Elevated liver enzymes, including bilirubin, ALP, GGT, ALT, and AST, often indicate a biliary origin of acute pancreatitis [10,11]. In our cohort, all patients demonstrated such elevations, and all were diagnosed with acute biliary pancreatitis.

Determining the severity of acute pancreatitis is crucial in guiding appropriate treatment strategies. Various laboratory and imaging-based scoring systems, such as Ranson's criteria, the APACHE II score, the Balthazar score, and the CT severity index, have been developed for this purpose [12–15]. In this study, we used the Ranson scoring system based on parameters available at hospital admission. A Ranson score ≥3 was considered as an indicator of severe pancreatitis, consistent with previous literature [16].

Approximately 80% of acute pancreatitis cases are classified as mild and self-limiting,

while the remaining 20% may involve complications and carry a significant risk of mortality. Therefore, easily accessible and reliable indicators for predicting prognosis and length of hospitalization are of critical importance.

We evaluated NLR and PLR values in comparison with Ranson scores and other clinical parameters. Neutrophils, lymphocytes, and platelets play key roles in the inflammatory process, and their ratios—NLR and PLR—can be calculated from peripheral blood samples. These markers have been associated with poor prognosis in peripheral vascular diseases, coronary artery disease, gynecologic cancers, hepatobiliary malignancies, and systemic inflammation [17-19].

Azab et al. were among the first to study the utility of NLR in acute pancreatitis, reporting that an NLR >4.7 was predictive of ICU admission and prolonged hospitalization (20). Most studies have adopted a cutoff value of ≥ 5 for prognostic purposes. Suppiah et al. found that high NLR was associated with readmission and severity, while Binnetoğlu et al. questioned its prognostic accuracy [21,22]. Zhang et al. showed that elevated NLR levels at admission correlated with multi-organ dysfunction, longer ICU stays, and in-hospital mortality in Chinese patients [23]. In a study by Suppiah et al., high NLR values differentiated between mild and severe pancreatitis and decreased in recovering patients, while remaining elevated in severe cases [24].

Our study findings align with the literature. We observed that both NLR and PLR levels were significantly higher in patients with severe pancreatitis (Ranson ≥ 3). Additionally, we found that NLR and PLR values correlated positively with glucose levels and age, both of which have been previously identified as negative prognostic factors [25-28]. The mean

age in our study was 61.93 years, and increasing age was associated with more severe disease and higher NLR/PLR values.

Consistent with prior research, gender did not show a significant correlation with disease severity or NLR/PLR values in our study [29]. However, patients with severe pancreatitis had longer hospital stays and higher inflammatory marker levels. Interestingly, PLR showed a significant correlation with serum AST levels, while NLR did not. Both markers, however, were positively correlated with LDH, an enzyme released due to pancreatic cell damage and known to rise during inflammation [30].

We conclude that NLR and PLR are simple, effective, and low-cost tools for assessing pancreatitis severity. Still, our study has limitations. As a retrospective and single-center study, it may be subject to selection bias and lacks prospective validation. The absence of complete 24-hour Ranson data and the inability to document complications due to limited records also pose constraints. Future multicenter, prospective studies are necessary to validate these findings and refine cutoff thresholds.

4.1. Conclusion

Our findings indicate that NLR and PLR levels are significantly elevated in cases of acute biliary pancreatitis, particularly in patients with severe disease. These markers were also associated with higher age, leukocyte count, and glucose, LDH, and AST levels. A clear relationship between increased NLR/PLR levels and longer hospital stay was also observed.

Given that traditional scoring systems such as Ranson require multiple parameters over time and can be impractical in urgent clinical settings, NLR and PLR offer rapid, cost-effective alternatives for initial risk stratification. These markers can assist

clinicians in identifying high-risk patients upon admission and guide timely interventions.

To establish NLR and PLR as reliable components of future scoring systems, additional prospective studies with larger, diverse patient populations are warranted.

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