

The significance of Systemic Immune-Inflammation Index, pan-immune-inflammation value, and Systemic Inflammation Response Index in the etiopathogenesis of acute pancreatitis

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ABSTRACT

Aims: The Systemic Immune-Inflammation Index (SII), Systemic Inflammation Response Index (SIRI), and pan-immune-inflammation value (PIV) are recently developed markers for systemic inflammatory response. This study aimed to investigate the relationship between SII and SIRI, and serum amylase and lipase levels in patients with acute pancreatitis (AP).

Methods: A total of 101 patients diagnosed with AP and 118 healthy volunteers were included in the study. The diagnosis of AP was based on contrast-enhanced abdominal CT and elevated serum amylase and lipase levels. SII, SIRI, PIV, WBC, CRP, N/L ratios, and other biochemical parameters of all cases were compared with those of the control group.

Results: Upon analysis of the data, serum lipase and amylase levels, as well as SII, SIRI, and PIV indices, were found to be statistically significantly higher in AP patients compared to the control group ($p < 0.001$). Other acute phase reactants, WBC and CRP levels, and the N/L ratio were also statistically significantly higher in AP patients compared to the control group ($p < 0.001$). In ROC analysis, high area under the curve (AUC) values were found for N/L, SII, SIRI, and PIV, which were 0.823, 0.831, 0.890, and 0.895, respectively ($p < 0.001$).

Conclusion: The SII, SIRI, and PIV may be useful in indicating the severity of the disease in patients with AP.

Keywords: Acute pancreatitis, Systemic Immune-Inflammation Index, Systemic Inflammation Response Index, pan-immune-inflammation value, lipase

INTRODUCTION

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas characterized by severe abdominal pain and elevated pancreatic enzymes. In recent years, significant increases in the incidence of AP and related hospitalizations have been observed.^{1,2} Severe AP can lead to the Systemic Inflammatory Response Syndrome (SIRS). Intraacinar activation of proteolytic enzymes is associated with leukocyte chemotaxis, cytokine release, oxidative stress, and microcirculatory damage. The common factor underlying all these mechanisms is an excessive inflammatory response.^{3,4} The establishment of an effective inflammatory response and the maintenance of tissue homeostasis depend on successful coordination among neutrophils, macrophages, and other immune cells, which is controlled by inflammatory mediators (e.g., cytokines and chemokines).^{5,6}

The Systemic Inflammatory Index (SII) was developed by formulating simple parameters (e.g., neutrophil, lymphocyte, and platelet counts) and was first used to assess the prognosis of hepatocellular carcinoma.⁷

The SII, Systemic Inflammation Response Index (SIRI), and pan-immune inflammation value (PIV) are markers developed in recent years for the systemic inflammatory response. The aim of this study was to investigate the relationship between SII, SIRI, and PIV and serum amylase and lipase levels in patients with AP.

METHODS

The study was approved by the Ankara City Hospital, Clinical Researches Ethics Committee (Date: 15.02.2023, Decision No: E223-3380). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Patients' demographic, clinical, laboratory data, and radiological findings were obtained from electronic medical records and hospital computer-generated forms. Cases were divided into two groups (AP and healthy control group). The patients' baseline values and laboratory results were retrieved

from the electronic medical record system, and the groups were compared. The following parameters were analyzed in all groups: WBC, platelets, neutrophils, lymphocytes, monocytes, urea, serum creatinine, ALT, AST, ALP, amylase, lipase, CRP, and the N/L ratio.

To evaluate cases diagnosed with AP, patients' demographic data were assessed along with clinical, laboratory, and radiological findings. Chest and abdominal computed tomography (CT) scan findings were evaluated to determine pancreatic injury.

The diagnosis of AP was made based on the internationally accepted 2012 Atlanta classification, requiring the presence of at least two of the following three criteria: 1) Typical epigastric abdominal pain consistent with AP, 2) Serum amylase and/or lipase levels exceeding three times the upper limit of normal, 3) Findings consistent with AP on cross-sectional imaging methods such as contrast-enhanced CT. Complete blood counts and biochemical parameters at the time of presentation were recorded. The etiology of AP (gallstones, alcohol, hyperlipidemia, idiopathic, etc.) was determined from patient records to the extent possible. However, since the primary objective of this study was to evaluate diagnostic performance and the sample size might be insufficient for subgroup analyses, the potential confounding effect of etiology on inflammatory indices was not examined in detail. Patients were analyzed in two groups: a control group consisting of 118 completely healthy individuals and 101 AP patients.

- **SII is calculated using the following formula:** $SII = \text{platelet count} \times \text{neutrophil} / \text{lymphocyte}$
- **SIRI is calculated using the following formula:** $SIRI = \text{neutrophil} \times \text{monocyte} / \text{lymphocyte count}$
- **PIV is calculated using the following formula:** $PIV = \text{neutrophil} \times \text{monocyte} \times \text{platelet} / \text{lymphocyte count}$

Statistical Analysis

The data were performed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to assess the normality of data distribution. Descriptive statistics were presented as mean $\pm$ standard deviation (SD) for normally distributed variables and as median and interquartile range (IQR) for non-normally distributed variables.

Group comparisons were conducted using student t-test for normally distributed variables and the Kruskal-Wallis test for non-normally distributed variables. Differences in categorical variables were analysed using the Chi-square test or Fisher's exact test, as appropriate.

Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cut-off values for amylase, lipase, SII, SIRI, PIV, and NLR. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 101 patients with AP and 118 healthy controls were included. Baseline demographic and laboratory characteristics are summarised in **Table 1**. There was no significant difference in age between the groups ($p=0.063$).

Patients with AP exhibited a markedly increased systemic inflammatory response, as reflected by significantly elevated

Table 1. Demographic results of control and AP groups

Characteristics	Control group (n=118)	AP group (n=101)	p-value*
Age, median (IQR), range, years	49 (21), 20-79	56 (27), 21-84	0.063 ^a
Gender, male/female	60/58	54/47	0.699 ^b
Length of hospital stay, mean $\pm$ SD	-	6.2 $\pm$ 4.5	-
CT findings			
Normal findings	-	22 (21.8)	-
Pancreatic diffuse edema, thick	-	20 (19.8)	-
Nodular lesion in the pancreas	-	1 (1)	-
Pancreatic duct prominent	-	6 (5.9)	-
Inflammatory contaminations in peripancreatic adipose tissue, fluid	-	48 (47.5)	-
Partial edematous appearance in the pancreas	-	25 (24.8)	-
Fluid around the pancreas	-	12 (11.9)	-
Hypodense appearance in pancreas, necrosis	-	8 (7.9)	-
Mass in the pancreas	-	1 (1)	-
Fatty atrophic changes in the pancreas	-	2 (2)	-
Other findings	-	2 (2)	-
Comorbidities			
Systemic hypertension, n, (%)	-	30 (29.7)	-
Diabetes mellitus, n, (%)	-	17 (16.8)	-
Congestive heart disease, n, (%)	-	11 (10.9)	-
Chronic renal disease, n, (%)	-	4 (4.0)	-
Thyroid dysfunctions, n, (%)	-	5 (5.0)	-
Chronic obstructive pulmonary disease, n, (%)	-	9 (8.9)	-
Other	-	22 (21.8)	-
WBC, $\times 10^9/L$	7.3 (3.1)	9.6 (6.3)	<0.001
NEU, $\times 10^9/L$	4.4 (3.1)	7.9 (5.5)	<0.001
Lymphocytes, $\times 10^9/L$	1.9 (1.1)	1.3 (0.9)	<0.001
Monocytes, $\times 10^9/L$	0.26 (0.15)	0.53 (0.31)	<0.001
Platelet, $\times 10^9/L$	244 (91)	253 (94)	0.111
N/L	2.2 (1.9)	6.3 (8.0)	<0.001
CRP, g/L	0.22 (0.40)	17 (34.20)	<0.001
Amylase, U/L	65 (32)	659 (1029)	<0.001
Lipase, U/L	34 (16)	819 (1859)	<0.001
AST, U/L	22 (17)	76 (167)	<0.001
ALT, U/L	24 (19)	90 (215)	<0.001
Na, mEq/L	140 (3)	139 (4)	0.416
K, mEq/L	4.2 (0.5)	4.1 (0.6)	0.698
Glucose, mg/dl	105 (53)	105 (53)	<0.001
Urea, mg/dl	27 (9)	32 (16)	<0.001
Serum creatinine, mg/dl	0.74 (0.24)	0.79 (0.27)	0.3
SII	554 (500)	1480 (1976)	<0.001
SIRI	0.59 (0.85)	2.8 (5.4)	<0.001
PIV	147 (212)	841 (1160)	<0.001

*Comparison of two groups based on Mann-Whitney U, ^bcomparison of two groups based on Chi-square test. The significance level was set at $p < 0.05$. AP: Acute pancreatitis, IQR: Interquartile range, SD: Standard deviation, CT: Computed tomography, WBC: White blood cell, NEU: Neutrophil, CRP: C-reactive protein, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, Na: Sodium, K: Potassium, SII: Systemic Immune-Inflammatory Index, SIRI: Systemic Inflammation Response Index, PIV: Pan immune inflammation value

SII, SIRI, and PIV values compared with controls (all $p < 0.001$) (Table 1).

Correlation analyses showed that inflammatory indices were positively associated with pancreatic enzyme levels in the AP group, whereas no significant correlations were observed in controls (Tables 2 and 3). Notably, no significant correlation was found between lipase and PIV.

Table 2. Correlations between amylase and other parameters

Parameters	Control group (n=118)		Acute pancreatitis group (n=101)	
	rho	p-value	rho	p-value
SII	-0.017	0.853	0.301	0.002
SIRI	-0.011	0.910	0.253	0.011
PIV	-0.069	0.461	0.238	0.018
N/L	0.045	0.630	0.310	0.002

SII: Systemic Immune-Inflammatory Index, SIRI: Systemic Inflammation Response Index, PIV: Pan immune inflammation value, rho: Spearman correlation coefficient, p-values less than .05 were considered significant highlighted in bold

Table 3. Correlations between lipase and other parameters

Parameters	Control group (n=118)		Acute pancreatitis group (n=101)	
	rho	p value	rho	p-value
SII	-0.098	0.294	0.278	0.005
SIRI	-0.114	0.219	0.201	0.046
PIV	-0.122	0.191	0.170	0.092
N/L	-0.109	0.244	0.299	0.003

SII: Systemic Immune-Inflammatory Index, SIRI: Systemic Inflammation Response Index, PIV: Pan immune inflammation value, rho: Spearman correlation coefficient, p-values less than .05 were considered significant highlighted in bold

ROC analysis results are presented in Table 4 and Figure PIV and SIRI demonstrated the highest diagnostic performance, with AUC values of 0.895 and 0.890, respectively, outperforming SII (AUC=0.831) and the N/L ratio (AUC=0.823) (Table 4). Both indices showed high sensitivity (82.2%) with good specificity, supporting their diagnostic value. Overall, these findings highlight PIV and SIRI as the most robust biomarkers for the diagnosis of AP.

DISCUSSION

AP is a common inflammatory disease characterized by sudden onset, rapid progression, and high rates of morbidity and mortality. Therefore, timely and accurate assessment of the clinical condition and prognosis of patients with AP is of critical importance for management strategies. In addition to traditional biochemical and radiological methods used in diagnosis, the role of new-generation indices—which reflect the inflammatory process and are easily accessible and cost-effective—in diagnosis and prognosis has been intensively investigated in recent years.⁸ In this study, the efficacy of markers such as the SII, PIV, and SIRI in the diagnosis of AP

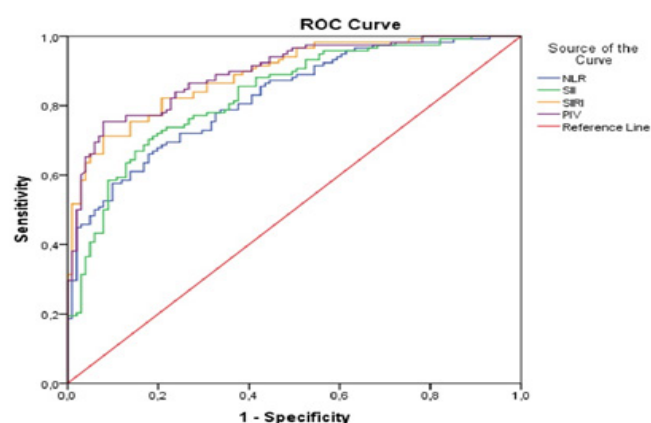


Figure. The ROC curves of NLR, SII, SIRI and PIV in predicting acute pancreatitis on admission

ROC: Receiver operating characteristic, NLR: Neutrophils to lymphocytes ratio, SII: Systemic Immune Inflammatory Index, SIRI: Systemic Inflammation Response Index, PIV: Pan immune inflammation value

was investigated by comparing them with a healthy control group.

The pathophysiology of AP is characterized by a complex inflammatory cascade that develops following damage to pancreatic acinar cells. Neutrophils and macrophages play a central role in this process.⁶ In particular, neutrophil extracellular traps (NETs) and neutrophil infiltration have been identified as key mechanisms amplifying local and systemic inflammation in pancreatic tissue. Gukovsky and colleagues⁵ have highlighted common mechanisms linking inflammation, autophagy, and obesity in the pathogenesis of pancreatitis and pancreatic cancer. The systemic response observed in AP is a result of a cytokine storm, macrophage-mediated inflammation, and uncontrolled neutrophil infiltration.⁸ The reflection of these complex processes in numerical changes in peripheral blood cells makes hematological indices valuable and dynamic tools for evaluating the pathophysiology of AP.

In clinical practice, serum amylase and lipase levels have long been accepted as standard markers for the diagnosis of AP. In our study, we found that levels of these enzymes were statistically significantly higher in the AP group compared to the control group ($p < 0.001$). Additionally, levels of C-reactive protein (CRP), another common marker of inflammation, were also significantly higher in the AP group ($p < 0.001$). However, these traditional markers have certain limitations. Amylase and lipase levels may not always correlate with disease severity, may exhibit low specificity, or may remain within normal limits in some cases.⁸ CRP, on the other hand, has limited sensitivity in the early diagnostic phase because it peaks 24–48 hours after the onset of inflammation.

Traditionally used parameters such as CRP, WBC, or the N/L ratio reflect only certain aspects of the inflammatory process. In contrast, multicomponent indices such as PIV and SIRI provide a more comprehensive picture of the immune system

Table 4. The parameters in diagnosis of patients with acute pancreatitis on admission

Variables	Cut-off value	AUC (95% CI)	Sensitivity (%)	Specificity (%)	p-value
Systemic Immune-Inflammatory Index	≥ 1131.77	0.831 (0.778-0.884)	80.5	64.4	< 0.05
Systemic Inflammation Response Index	≥ 1.49	0.890 (0.849-0.931)	82.2	79.2	< 0.05
Pan immune inflammation value	≥ 385.41	0.895 (0.855-0.936)	82.2	77.2	< 0.05
N/L	≥ 3.96	0.823 (0.770-0.877)	80.5	62.4	< 0.05

AUC: Area under the curve, CI: Confidence interval

and the inflammatory response by incorporating neutrophils, monocytes, lymphocytes, and, in the case of PIV, platelets into their formulas. In our study, the fact that PIV and SIRI demonstrated superior AUC values compared to the N/L ratio and SII supports the hypothesis that this comprehensive approach enhances diagnostic accuracy. In particular, the fact that PIV includes platelets—which play a critical role in coagulation and hemostasis—in addition to inflammatory cells, gives it a theoretical advantage in a complex disease such as AP, where microcirculatory damage is a primary concern.

Our study found that SII demonstrated high efficacy in the diagnosis of AP (AUC: 0.831, 80.5% sensitivity, 64.4% specificity). SII reflects immune-inflammatory interactions by combining neutrophils, lymphocytes, and platelets. Hu and colleagues⁷ noted that SII was initially developed for prognostic prediction in hepatocellular carcinoma but is also valuable in hematological malignancies. Subsequently, the use of SII has expanded to a wide range of conditions, including invasive vulvar cancer⁹ and asthma and drug-induced respiratory diseases.¹⁰ Most studies in the literature have evaluated the role of SII in AP in terms of disease severity and prognosis. For example, Liu et al.¹¹ reported a high AUC value of 0.920 in distinguishing severe AP (SAP), while Zhang et al.¹² and Bıyık et al.¹³ also highlighted the success of SII in prognosis prediction. The fact that the AUC value obtained in our study differs somewhat from these studies can be explained by our focus on “diagnosis” rather than “prognosis.” However, our achievement of a higher performance than the AUC value of 0.739 reported by Salehi et al. for AP severity demonstrates that SII is a strong candidate in the diagnostic process as well.

One of the most striking findings of our study is SIRI's superior performance in the diagnosis of AP (AUC: 0.890, 82.2% sensitivity, 79.2% specificity). With its composition including neutrophils, monocytes, and lymphocytes, SIRI more comprehensively reflects the complex cellular interactions in the pathophysiology of AP. The prognostic value of SIRI has previously been demonstrated in stroke,¹⁴ ANCA-associated vasculitis,¹⁵ and various malignancies.¹⁶ Specifically regarding AP, Bıyık et al.¹³ reported that SIRI is a potential biomarker for predicting disease severity (AUC: 0.782). The AUC value of 0.890 obtained in our study suggests that SIRI has a much stronger potential not only in prognosis but also in the early diagnosis phase. Indeed, recent studies by Wu et al.¹⁷ and Araiza-Rodríguez et al.¹⁸ have demonstrated a dose-dependent relationship between SIRI and AP severity, thereby reinforcing the clinical significance of this index.

Similarly, PIV, which demonstrated the highest diagnostic performance in our study (AUC: 0.895, 82.2% sensitivity, 77.2% specificity), emerges as a promising marker for the diagnosis of AP. By integrating neutrophils, monocytes, lymphocytes, and platelets, PIV overcomes the limitations of parameters that focus on only two cell types, such as NLR or PLR1.^{19,20} The prognostic value of PIV has been investigated in many malignancies; a meta-analysis by Guven et al.²¹ demonstrated that it is a strong biomarker in various types of cancer.

A very recent study by Zhang and Hu²² demonstrated that PIV is an independent and strong predictor of in-hospital

mortality in critically ill patients with AP (OR: 1.21, $p=0.015$). Our study makes a critical contribution to this literature by demonstrating that PIV is not only a reliable tool for prognosis but also an extremely reliable tool for diagnosing AP at the time of emergency department presentation (AUC: 0.895). These findings indicate that PIV is one of the most comprehensive markers reflecting “pan-immune” activation in the pathophysiology of AP.

In conclusion, our ROC analysis data demonstrated that PIV and SIRI exhibit superior accuracy in the diagnosis of AP compared to N/L (AUC: 0.823) and SII (AUC: 0.831). The high AUC values of these three new indices demonstrate that they offer low-cost, rapid, and reliable tools for supporting the diagnosis and quickly assessing the severity of inflammation in patients with suspected AP in emergency department settings. The importance of hematological ratios, emphasized by researchers such as Ventriglia et al.¹⁹ and Cao et al.²⁰ in various pathologies, has been taken to a further level in the diagnosis of AP in our study (PIV and SIRI). Validation of our findings through broader and prospective studies will facilitate the incorporation of these indices into routine clinical practice.

Limitations

Our study has several limitations. Its retrospective design and single-center nature may limit the generalizability of the findings. Additionally, no subgroup analyses were conducted to assess the specific effects of AP etiology (gallstones, alcohol, etc.) on these indices. Most importantly, external validation through prospective studies in different populations is required to fully integrate these high diagnostic performance values into clinical practice.

CONCLUSION

This study has demonstrated that easily accessible and cost-effective markers such as SII, SIRI, and PIV exhibit high diagnostic performance in the diagnosis of AP. In particular, the fact that PIV and SIRI demonstrate superior AUC values compared to N/L and SII in predicting AP diagnosis underscores the clinical potential of these new-generation indices. These markers can be used as rapid and reliable tools to support standard diagnostic methods in patients presenting with suspected AP. Our findings require validation through broader, prospective studies aimed at better understanding the role of these indices in the pathophysiology of AP and determining their place in clinical practice.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was approved by the Ankara City Hospital, Clinical Researches Ethics Committee (Date: 15.02.2023, Decision No: E223-3380).

Informed Consent

This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: AK; Design: AK, GA; Data Collection and/or Processing: AK, GA, EÇ; Analysis and/or Interpretation: AK, GA; Literature Review: AK, EÇ; Writing the Article: AK, EÇ; Critical Review: AK, GA.

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