DOI: 10.14744/cm.2025.40316
Compreh Med 2026;18(1):73-80

RESEARCH ARTICLE

Atherogenic Index of Plasma and Its Relationship with Acute Pancreatitis Severity: A Retrospective Cohort Study

Özgür Yılmaz[1], Sami Çifçi[2]

[1]Department of Internal Medicine, Kanuni Sultan Suleyman Training and Research Hospital, İstanbul, Türkiye
[2]Department of Gastroenterology, Başaksehir Çam and Sakura City Hospital, İstanbul, Türkiye

ABSTRACT

Objective: Acute pancreatitis (AP) varies in severity, making early identification of severe acute pancreatitis (SAP) essential. As lipid imbalance has been increasingly linked to inflammatory activation, the Atherogenic Index of Plasma (AIP) has been proposed as a potential prognostic marker. This study evaluated the association between AIP and AP severity and assessed whether AIP independently predicts SAP.

Materials and Methods: This retrospective cohort included 203 adult patients with acute pancreatitis. Classification of acute pancreatitis followed the Revised Atlanta Criteria. Demographic, laboratory, and lipid data were extracted from electronic records, and AIP was calculated using triglyceride and HDL-cholesterol values. Analyses included group comparisons, correlation testing, comprehensive ROC analyses, multivariable logistic regression, and a covariate-adjusted ROC.

Results: Among 203 patients, 128 had MAP and 75 had SAP. SAP patients showed significantly higher WBC, glucose, creatinine, AST, ALT, and CRP levels (all $p<0.001$) and lower HDL-C ($p=0.024$). Mean AIP was higher in SAP (0.59±0.15 vs. 0.53±0.20; $p=0.016$) but demonstrated limited discrimination (AUC 0.575; cut-off 0.390; sensitivity 96.0%, specificity 22.7%). AIP was significant in unadjusted and WBC-adjusted models ($p=0.023$ and $p=0.043$, respectively), but not in the fully adjusted model including WBC and glucose ($p=0.196$). The covariate-adjusted ROC showed excellent accuracy (AUC 0.892; sensitivity 84.0%, specificity 79.7%).

Conclusion: Although higher in severe cases, AIP did not independently predict severity. While insufficient alone, AIP may contribute supportive value when combined with inflammatory and metabolic markers.

Keywords: Acute pancreatitis, AIP, atherogenic index of plasma, HDL-cholesterol, severe acute pancreatitis, triglycerides

How to cite this article: Yılmaz Ö, Çifçi S. Atherogenic Index of Plasma and Its Relationship with Acute Pancreatitis Severity: A Retrospective Cohort Study. Compreh Med 2026;18(1):73-80

INTRODUCTION

Acute pancreatitis (AP) is an inflammatory disorder of the pancreas with a broad clinical spectrum, ranging from mild, self-limiting disease to severe necrotizing pancreatitis complicated by organ failure and high mortality. Its global incidence has risen steadily over the past two decades, reaching 20–40 cases per 100,000 population annually.[1,2] Despite advances in diagnosis and management, mortality in severe cases may still reach 15–30%.[3,4] Early identification of patients at risk of severe disease is therefore essential to guide intensive monitoring and timely intervention.

According to the 2012 Revised Atlanta Classification (RAC), acute pancreatitis is categorized into mild, moderately severe, and severe forms based on the presence, duration, and consequences of organ failure, as well as local complications.[5] Although this system improves diagnostic consistency, it does not provide real-time prognostic information. Several scoring systems, such as the Bedside Index for Severity in Acute Pancreatitis (BISAP), Ranson's criteria, and the Modified CT Severity Index (MCTSI), are commonly used for early risk assessment; however, their diagnostic performance is variable, and some require imaging or parameters that may not be immediately available in the acute setting.[6–9]

Address for Correspondence: Özgür Yılmaz, Department of Internal Medicine, Kanuni Sultan Suleyman Training and Research Hospital, İstanbul, Türkiye
E-mail: dr_ozguryilmaz@hotmail.com **ORCID ID:** 0009-0000-4976-4771

Received date: 29.11.2025
Revised date: 08.12.2025
Accepted date: 12.12.2025
Online date: 23.01.2026

Comprehensive Medicine published by Kare Media.

Disturbances in lipid metabolism are increasingly recognized in the pathophysiology of AP. Hypertriglyceridemia accounts for 5–10% of cases and is the third most common etiology after gallstones and alcohol.[10] Experimental and clinical studies have shown that triglyceride (TG) hydrolysis by pancreatic lipase generates cytotoxic free fatty acids (FFAs), which induce acinar injury, microvascular dysfunction, and systemic inflammation.[11,12] Conversely, high-density lipoprotein cholesterol (HDL-C) exerts antioxidant and anti-inflammatory effects, and low HDL-C levels have been associated with organ failure and increased disease severity in AP.[13,14] These opposing biological roles suggest that combined lipid indices may provide insights into inflammatory and metabolic stress during early disease.

The Atherogenic Index of Plasma (AIP) combines triglyceride and HDL-C levels into a single indicator of atherogenic dyslipidemia. Because it is derived from routinely measured lipid parameters, AIP reflects the balance between pro-inflammatory triglycerides and the anti-inflammatory, antioxidant functions of HDL-C.[15,16] Recent studies have shown that elevated AIP at admission is associated with a higher likelihood of severe acute pancreatitis. Cho et al.[17] reported that increased AIP independently predicted severe disease in a Korean cohort, while Wu et al.[18] demonstrated that AIP was linked to both severe pancreatitis and the development of acute kidney injury, a major contributor to morbidity. These observations, together with mechanistic evidence that systemic inflammation rapidly decreases HDL-C and increases triglyceride concentrations, support the concept that AIP may serve as an early surrogate marker of metabolic and inflammatory stress in acute pancreatitis.[17]

Although these findings are promising, several uncertainties remain. Reported AIP cut-off values vary across studies, the timing of lipid measurement is not standardized, and data on its additive value over established scoring systems are limited. Further research is therefore needed to clarify whether AIP provides independent and clinically meaningful prognostic information in acute pancreatitis.

The present study aims to evaluate the relationship between the AIP and disease severity in acute pancreatitis and to determine whether AIP serves as an independent predictor of severe acute pancreatitis in a retrospective patient cohort.

MATERIALS and METHODS

Study Design and Settings

This retrospective cohort study included adult patients diagnosed with acute pancreatitis who were hospitalized in the Internal Medicine ward of our tertiary care center between 1 January 2022 and 31 December 2024. Patient data, including demographic characteristics, comorbidities, laboratory values, and imaging findings, were obtained from the institutional electronic medical record system. The diagnosis of acute pancreatitis was established according to the 2012 Revised Atlanta Classification, requiring the presence of at least two of the following: typical abdominal pain, elevation of serum amylase or lipase to at least three times the upper limit of normal, or radiological evidence consistent with acute pancreatitis.[5] Disease severity was defined based on the same criteria; patients without organ failure and without local or systemic complications were categorized as having mild acute pancreatitis (MAP), whereas those with persistent organ failure lasting more than 48 hours, as defined by the Modified Marshall scoring system, were classified as having severe acute pancreatitis (SAP).[19] Patients who met the criteria for "moderately severe" disease, that is, those with transient organ failure or local complications, were included in the MAP group to maintain a binary classification suitable for the analytical design of the study.[20] Exclusion criteria included chronic pancreatitis, pancreatic malignancy, cirrhosis, chronic kidney disease stage ≥ 3, thyroid dysfunction, pregnancy, active systemic infection, chronic inflammatory disorders, use of lipid-lowering therapy within the previous three months, known primary hyperlipidemia, and incomplete laboratory data within 24 hours of admission. After application of these criteria, a total of 203 patients were included in the final analysis.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the principles of the Declaration of Helsinki. The study was approved by the Local Ethics Committee of Kanuni Sultan Suleyman Training and Research Hospital (decision date: 21.04.2025, number: E-80929729-000-274110217). Because of the retrospective nature of the study, the requirement for written informed consent was waived. All data were anonymized before analysis to ensure patient confidentiality.

Calculation of Atherogenic Index of Plasma

Serum TG and HDL-C levels were obtained from admission laboratory records. Measurements were performed using enzymatic colorimetric assays on a Roche Cobas 8000 analyzer (Roche Diagnostics, Mannheim, Germany), according to the hospital's routine laboratory procedures. In this study, both triglycerides (TG) and HDL-cholesterol (HDL-C) were measured in mg/dL. The AIP was calculated using the formula $\log_{10}(TG/HDL\text{-}C)$. Additional laboratory parameters, including amylase, lipase, glucose, calcium, creatinine, and C-reactive protein (CRP), were retrieved from electronic medical records. All lipid measurements (triglycerides and HDL-cholesterol) were obtained within the first 24 hours of hospital admission as part of the routine initial laboratory evaluation. All biochemical analyses were conducted in the hospital's central laboratory following standard operating protocols.

Outcomes

The primary outcome of the study was to determine the association between the AIP and the severity of acute pancreatitis, classified as mild or severe based on the Revised Atlanta Criteria. Secondary outcomes included evaluating the relationship between AIP and key laboratory parameters, assessing its discriminative ability for predicting severe disease through ROC analysis, and determining whether AIP remained an independent predictor of severity in multivariable logistic regression models.

Statistical Analysis

Statistical analyses were performed using Python 13.3 (pandas, numpy, scikit-learn, matplotlib) and IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed with the Kolmogorov–Smirnov and Shapiro–Wilk tests. Normally distributed variables were expressed as mean ± standard deviation (SD), while non-normally distributed variables were summarized as median (Q1–Q3). Group comparisons were conducted using the independent-samples t-test or Welch's t-test when variances were unequal, and the Mann–Whitney U test for non-normally distributed data. Categorical variables were compared with Pearson's chi-square test. Correlations between AIP and clinical or biochemical parameters were examined using Pearson or Spearman correlation coefficients based on variable distribution. To evaluate independent predictors of SAP, multivariable binary logistic regression analysis was performed by incorporating AIP together with white blood cell (WBC) count and serum glucose, which were selected due to their clinical relevance and statistical significance in univariate analyses. Odds ratios (ORs) with 95% confidence intervals (CIs) and Wald statistics were reported. Multicollinearity was assessed through variance inflation factor (VIF) and tolerance values, with VIF <5 considered acceptable. Discriminative performance was evaluated using two receiver operating characteristic (ROC) analyses. First, univariable ROC curves were generated for AIP, triglyceride, and HDL-C to quantify their individual ability to distinguish between mild and severe acute pancreatitis. Second, an adjusted ROC curve was constructed using the predicted probabilities obtained from the final multivariable logistic regression model, in which AIP, white blood cell count, and serum glucose were included as covariates. All ROC analyses were performed in Python, and the area under the curve (AUC), optimal cut-off values, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated using the Youden index. Graphical outputs were generated in high resolution. A two-tailed p-value <0.05 was considered statistically significant.

Table 1. Baseline demographic and clinical characteristics of patients with mild and severe acute pancreatitis

Parameters	MAP (n=128)		SAP (n=75)		p
	n	%	n	%	
Age, years	54.5 (34–75)		52 (36–68)		0.725^{U}
Sex					
Female	61	47.7	35	46.7	0.892†
Male	67	52.3	40	53.3	
BMI, kg/m²	27.2±3.6		26.8±4		0.406^{t}
Length of hospital stay (day)	5 (4–6)		9 (7–20)		<0.001^{U}
DM	67	52.3	30	40	0.089†
HT	63	49.2	39	52	0.702†
Smoking (Yes)	66	51.6	39	52	0.952†
Etiology					0.209†
Idiopathic	43	33.6	31	41.3	
Gallstones	29	22.7	8	10.7	
Alcohol-related	25	19.5	20	26.7	
Drug-induced	26	20.3	14	18.7	
Autoimmune	5	3.9	2	2.7	
Pancreatitis type					
Interstitial edematous pancreatitis	116	90.6	30	40	<0.001†
Necrotizing pancreatitis	12	9.4	45	60	

Values are presented as mean±standard deviation (SD), median (interquartile range: Q1–Q3), or number (percentage). ^{U}: Mann–Whitney U test; ^{t}: Independent Samples t-test; †: Pearson chi-square test. MAP: Mild acute pancreatitis; SAP: Severe acute pancreatitis; DM: Diabetes mellitus; HT: Hypertension; BMI: Body mass index

RESULTS

A total of 203 patients were included, comprising 128 with MAP and 75 with SAP. Baseline demographic variables, including age, sex, body mass index, diabetes mellitus, hypertension, smoking status, and etiological factors, did not differ significantly between the two groups (all p>0.05). The median length of hospital stay was markedly longer in the SAP group (9 vs. 5 days; p<0.001). Edematous pancreatitis predominated in MAP, whereas necrotizing pancreatitis was substantially more frequent in SAP (p<0.001). Detailed characteristics are presented in Table 1.

Table 2. Comparison of laboratory and biochemical parameters between mild and severe acute pancreatitis

Parameters	MAP (n=128)	SAP (n=75)	p
WBC, $\times10^9$/L	8.14±1.02	9.07±1.03	<0.001^t
Glucose, mg/dL	118.4±25.5	164.8±35.6	<0.001^t
Creatinine, mg/dL	0.91±0.22	1.38±0.34	<0.001^t
Total Cholesterol, mg/dL	186.6±31.2	182.3±29.4	0.333^t
LDL-C, mg/dL	108.8±27.8	110.5±29	0.678^t
AST, U/L	27 (18–33)	36 (30–45)	<0.001^U
ALT, U/L	24 (19–32)	34 (24–39)	<0.001^U
Amylase, U/L	192 (177–204)	193 (179–210)	0.264^U
Lipase, U/L	616.5 (490–723)	621 (506–739)	0.971^U
Calcium, mg/dL	9.20 (8.75–9.70)	9.30 (9–9.6)	0.584^U
CRP, mg/L	20 (17–22.5)	27 (24–33)	<0.001^U
Triglyceride, mg/dL	162.5 (118.5–216)	165 (134–211)	0.399^U
HDL-C, mg/dL	46 (40–54)	42 (36–49)	0.024^U

Values are presented as mean±standard deviation (SD) or median (interquartile range: Q1–Q3). ^{U}: Mann–Whitney U test; ^{t}: Independent Samples t-test. MAP: Mild acute pancreatitis; SAP: Severe acute pancreatitis; WBC: White blood cell; LDL-C: Low-density lipoprotein cholesterol; HDL-C: High-density lipoprotein cholesterol; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; CRP: C-reactive protein

Biochemical analyses revealed significantly higher white blood cell counts, serum glucose, creatinine, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and CRP levels in SAP patients compared with MAP (all p<0.001). HDL levels were modestly but significantly lower in SAP (p=0.024). No significant differences were observed in total cholesterol, LDL-C, TG, amylase, lipase, or calcium values (Table 2).

AIP values were compared between the MAP and SAP groups. Levene's test indicated that the assumption of equal variances was violated, F(1, 201) =6.84, p=0.010. Accordingly, an independent-samples t-test with unequal variances was performed. The mean AIP value was significantly higher in the SAP group (M=0.59, SD=0.15) compared with the MAP group (M=0.53, SD=0.20), t(188.25)=-2.43, p=0.016, with a mean difference of -0.061 (95% CI: -0.1106 to -0.0114). The distribution of AIP values across severity groups is illustrated in Figure 1.

In correlation analysis, AIP demonstrated a negative association with age in the MAP group (ρ=-0.232, p=0.008)

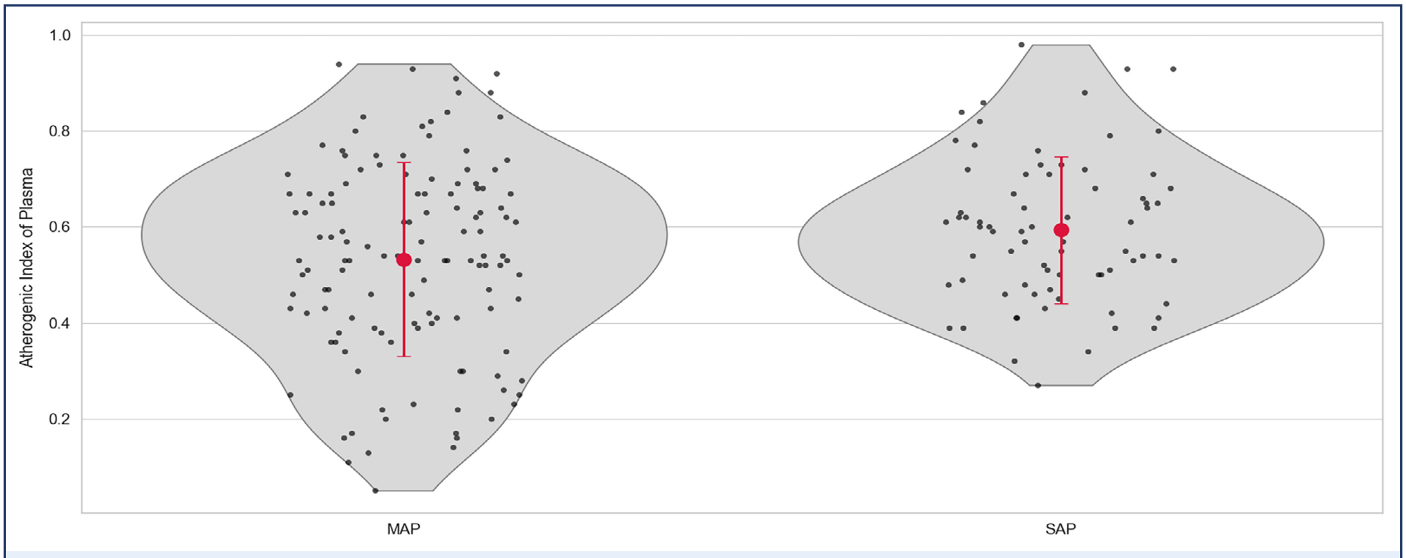

Figure 1. Distribution of AIP values in mild (MAP) and severe (SAP) acute pancreatitis

AIP: Atherogenic index of plasma; MAP: Mild acute pancreatitis; SAP: Severe acute pancreatitis

Table 3. Correlation of AIP with demographic and biochemical variables

	MAP (n=128)		SAP (n=75)	
	r/ρ	p	r/ρ	p
Age[ρ]	**-0.232**	**0.008**	-0.027	0.816
BMI[r]	-0.025	0.783	0.063	0.590
Length of hospital stay[ρ]	-0.020	0.819	-0.134	0.252
WBC[r]	0.069	0.442	-0.009	0.939
Glucose[r]	0.064	0.471	-0.037	0.749
Creatinine[r]	0.113	0.204	0.077	0.513
Total cholesterol[r]	0.041	0.646	-0.036	0.760
LDL-C[r]	-0.020	0.821	-0.199	0.087
AST[ρ]	0.075	0.400	-0.139	0.234
ALT[ρ]	0.057	0.521	**0.258**	**0.025**
Amylase[ρ]	0.115	0.198	-0.166	0.156
Lipase[ρ]	-0.137	0.123	-0.121	0.300
Calcium[ρ]	0.160	0.072	0.075	0.524
CRP[ρ]	0.046	0.604	0.104	0.376

Values are presented as correlation coefficients. [r]: Pearson correlation coefficient; [ρ]: Spearman correlation coefficient. AIP: Atherogenic index of plasma; MAP: Mild acute pancreatitis; SAP: Severe acute pancreatitis; BMI: Body mass index; WBC: White blood cell; LDL-C: Low density lipoprotein cholesterol; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; CRP: C-reactive protein

and a weak positive correlation with ALT in the SAP group (ρ=0.258, p=0.025). No other significant correlations were identified (Table 3).

ROC analysis demonstrated that AIP alone had limited discriminative ability for identifying severe pancreatitis, with an AUC of 0.575. Using a cut-off value of 0.390, AIP yielded 96.0% sensitivity, 22.7% specificity, 42.1% positive predictive value, and 90.6% negative predictive value. TG (AUC 0.536) and HDL-C (AUC 0.405) showed similarly poor performance (Fig. 2).

Binary logistic regression analyses evaluating the contribution of AIP to SAP are presented in Table 4. In Model 1, AIP was significant (OR 6.817, 95% CI 1.307–35.563; p=0.023), although the overall model fit was not significant. In Model 2, AIP remained significant (OR 6.397; p=0.043), and WBC emerged as an independent predictor (OR 2.587; p<0.001). In the fully adjusted Model 3, AIP was no longer significant (p=0.196), whereas both WBC and glucose remained strong independent predictors (both p<0.001). Model performance improved progressively across models (NR^2: 0.06 → 0.27 → 0.61), with no evidence of multicollinearity (VIF<5.0; tolerance >0.20).

To evaluate AIP's discriminative capacity after adjustment for confounders, a model-based ROC curve was generated using predicted probabilities from the fully adjusted logistic regression model (Model 3). This adjusted ROC demonstrated excellent discriminative performance (AUC 0.892), with 84.0% sensitivity, 79.7% specificity, 70.8% PPV, and 89.5% NPV (Fig. 3).

DISCUSSION

This study evaluated the predictive performance of the AIP in acute pancreatitis and demonstrated that AIP values were significantly higher in patients with SAP compared with those with mild disease. The group difference was also

Table 4. Evaluation of AIP's contribution to severe acute pancreatitis using different binary logistic regression models

	B	SE	Wald	df	p	OR	95% CI	
							LL	UL
Model 1								
AIP	1.919	0.843	5.187	1	0.023	6.817	1.307	35.563
Model 2								
AIP	1.856	0.915	4.112	1	0.043	6.397	1.064	38.458
WBC	0.951	0.184	26.814	1	<001	2.587	1.805	3.708
Model 3								
AIP	1.519	1.175	1.670	1	0.196	4.566	0.456	45.699
WBC	0.917	0.230	15.861	1	<001	2.501	1.593	3.926
Glucose	0.054	0.009	38.011	1	<001	1.055	1.037	1.073

All models were adjusted for age, BMI, sex (female), smoking status (non-smoker), diabetes mellitus (yes), and hypertension (yes). Model 1: NR^2=0.06, χ^2=9.12, p=0.238; Model 2: NR^2=0.27, χ^2=44.99, p<0.001; Model 3: NR^2=0.61, χ^2=119.28, p<0.001. B: Regression coefficient; SE: Standard error; OR: Odds ratio; 95% CI: 95% confidence interval; LL: Lower limit; UL: Upper limit; Wald: Wald test value; df: Degrees of freedom; p: significance level; NR^2: Nagelkerke R-squared; χ^2: Chi-square test; VIF: Variance inflation factor; AIP: Atherogenic index of plasma; WBC: White blood cell

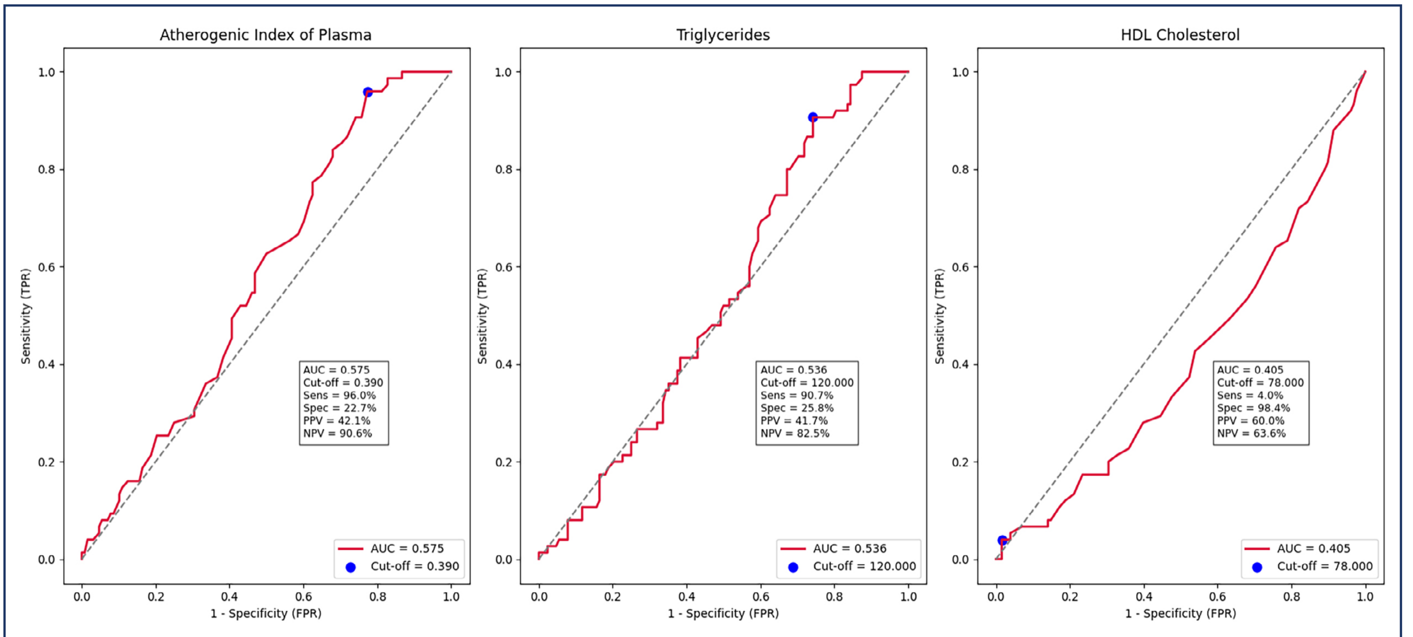

Figure 2. ROC curves showing the discriminative performance of AIP, triglyceride, and HDL-C for identifying severe acute pancreatitis

ROC: Receiver operating characteristic; AIP: Atherogenic index of plasma; HDL-C: High-density lipoprotein cholesterol

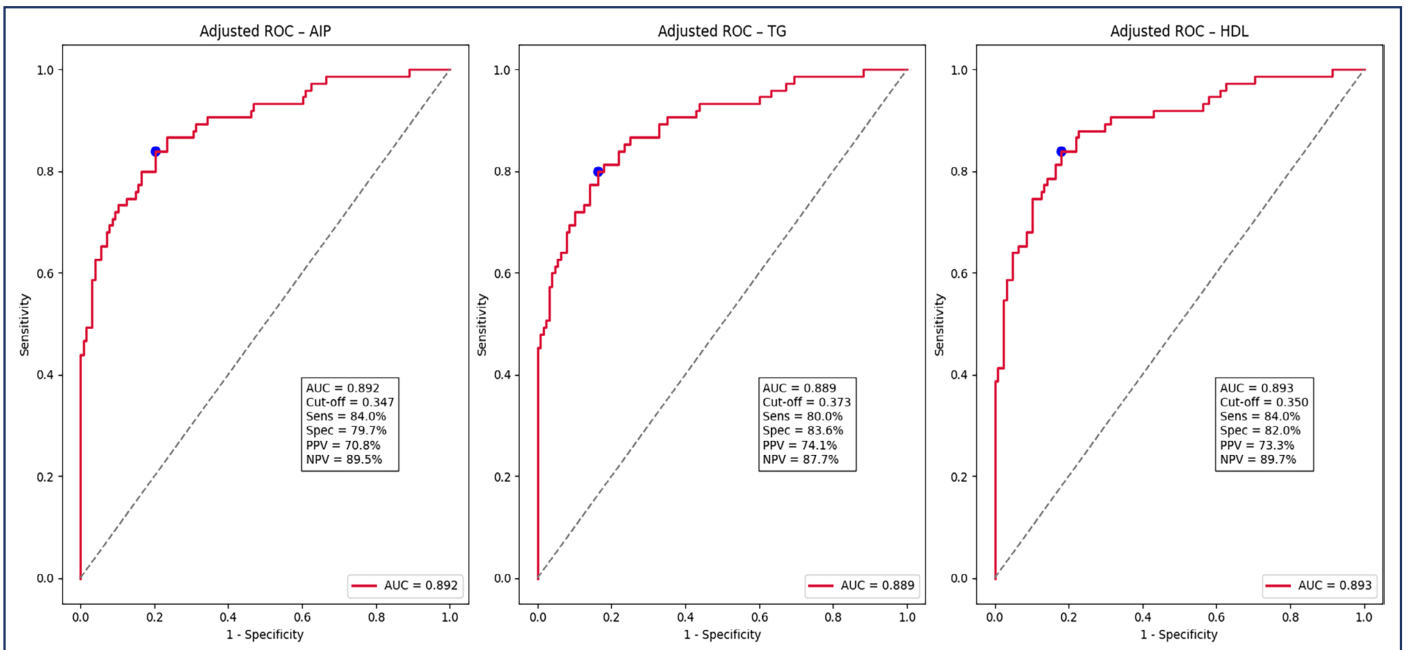

Figure 3. ROC curve of AIP adjusted for WBC and glucose using the final multivariable logistic regression model (Model 3)

ROC: Receiver operating characteristic; AIP: Atherogenic index of plasma; WBC: White blood cell

clearly depicted in the AIP distribution (Fig. 1), highlighting the shift toward higher metabolic–inflammatory burden in SAP. Furthermore, AIP showed modest correlations with age in MAP and with ALT in SAP, suggesting partial overlap between lipid dysregulation, hepatocellular stress, and systemic inflammation. However, despite these associations, AIP exhibited only limited discriminatory capacity and, critically, did not remain an independent predictor once white blood cell (WBC) count and serum glucose were included in the multivariable logistic regression model. These findings indicate that AIP alone cannot serve as a reliable standalone biomarker for early severity assessment.

Our results partially align with earlier studies reporting elevated AIP levels in SAP. In a prospective cohort, Cho et al.[17] demonstrated that AIP was significantly higher in SAP compared with mild AP and remained an independent predictor after multivariable adjustment. Similarly, Wu et al.[18] found AIP to be significantly associated with AKI and SAP, although the reported AUC values, approximately 0.65, indicated only moderate discriminative performance. In both studies, AIP demonstrated predictive value for severe acute pancreatitis; however, neither achieved high diagnostic accuracy. Consistent with these observations, the AIP ROC performance in our cohort was similarly modest (AUC 0.575), reinforcing the conclusion that AIP alone has limited discriminative strength. These findings, supported by prior clinical evidence showing that triglyceride elevation and HDL reduction parallel inflammatory severity in acute pancreatitis—particularly in hypertriglyceridemia-associated cases—suggest that AIP largely reflects the metabolic response to systemic inflammation rather than functioning as a robust independent prognostic marker.[17,21,22]

Our multivariable findings diverge from earlier reports largely because prior studies did not adjust for essential inflammatory and metabolic predictors, particularly WBC and glucose. They often relied on smaller or more homogeneous cohorts and seldom evaluated AIP alongside a broader panel of laboratory parameters. By incorporating these critical covariates within a structured multivariable binary logistic regression framework, our study is among the first to demonstrate that AIP loses its apparent predictive significance once key upstream physiological determinants are accounted for. This methodological distinction likely explains the discrepancy between our results and previously published associations.

A distinguishing strength of our work is the comprehensive multivariable modeling approach that incorporated major inflammatory and metabolic predictors. In contrast with earlier studies, our analysis showed that WBC and glucose were strong and independent determinants of SAP (both $p<0.001$), and their inclusion in the binary logistic regression models eliminated the statistical significance of AIP. This finding is biologically plausible: AIP is derived from triglyceride and HDL concentrations, which undergo rapid cytokine-driven alterations during acute inflammatory states. Feingold and Grunfeld demonstrated that TNF-α and IL-6 markedly suppress HDL synthesis and accelerate HDL clearance during the acute-phase response, while Pirillo and Catapano reported proportional HDL reductions with increasing inflammatory severity.[23,24] Additionally, prior clinical and experimental data have shown that stress hyperglycemia and hypertriglyceridemia contribute to metabolic instability and correlate with AP severity.[25] Taken together, these observations suggest that AIP reflects the broader metabolic-inflammatory milieu rather than functioning as an independent prognostic marker, supporting its use as a potential adjunctive but not standalone indicator.

The strong performance of our fully adjusted model with an AUC of 0.892 further underscores the superiority of composite approaches integrating inflammatory and metabolic indicators. This finding is consistent with the broader AP literature. Venkatesh et al.[26] showed that composite models incorporating inflammatory and biochemical parameters outperform single biomarkers. Mofidi et al.[27] reported that inflammatory markers, particularly WBC, were strongly associated with organ failure and mortality. Our findings extend this evidence by demonstrating a substantial improvement in predictive performance when WBC and glucose are included alongside AIP in a multivariable framework.

Nevertheless, some limitations merit consideration. The retrospective design may introduce residual confounding despite adjustment. The sample size, while larger than many prior AIP–AP studies, may still not be sufficient to detect smaller effect sizes in subgroup analyses. In addition, lipid measurements were taken at a single time point; future work should evaluate dynamic lipid changes across different disease stages. Moreover, because this was a single-center study, the results may reflect local laboratory standards, patient demographics, and institutional management practices, which may limit broader applicability. Variations in early resuscitation intensity, analgesic strategies, nutritional timing, and imaging schedules could also have influenced lipid profiles and clinical parameters. Additionally, although standardized severity criteria were applied, subtle differences in clinical documentation or timing of assessment may have introduced classification variability.

CONCLUSION

AIP levels were higher in severe acute pancreatitis, but its independent significance diminished after adjusting for inflammatory and metabolic markers. While AIP alone is insufficient for early severity assessment, it may still provide supportive value when incorporated into multivariable models alongside WBC and glucose. Future prospective studies should clarify its additive role within integrated prediction frameworks.

Disclosures

Ethics Committee Approval: The study was approved by the Kanuni Sultan Suleyman Training and Research Hospital Ethics Committee (No: E-80929729-000-274110217, Date: 21/04/2025).

Informed Consent: Because of the retrospective nature of the study, the requirement for written informed consent was waived. All data were anonymized before analysis to ensure patient confidentiality.

Conflict of Interest Statement: No conflict of interest was declared by the authors.

Funding: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: No AI technologies utilized.

Author Contributions: Concept: Ö.Y., S.Ç.; Design: Ö.Y.; Supervision: Ö.Y., S.Ç.; Data Collection: Ö.Y., S.Ç.; Analysis or Interpretation: S.Ç.; Literature Search: Ö.Y., S.Ç.; Writing: Ö.Y.; Critical Review: Ö.Y., S.Ç.

Peer-review: Externally peer-reviewed.

REFERENCES

1. Iannuzzi JP, King JA, Leong JH, Quan J, Windsor JW, Tanyingoh D, et al. Global ıncidence of acute pancreatitis ıs ıncreasing over time: A Systematic review and meta-analysis. Gastroenterology 2022;162:122 –34. [CrossRef]
2. Li T, Qin C, Zhao B, Li Z, Zhao Y, Lin C, et al. Global and regional burden of pancreatitis: epidemiological trends, risk factors, and projections to 2050 from the global burden of disease study 2021. BMC Gastroenterol 2024;24:398. [CrossRef]
3. Trikudanathan G, Yazici C, Evans Phillips A, Forsmark CE. Diagnosis and Management of Acute Pancreatitis. Gastroenterology 2024;167:673–88. [CrossRef]
4. Petrov MS, Yadav D. Global epidemiology and holistic prevention of pancreatitis. Nat Rev Gastroenterol Hepatol 2019;16:175–84. [CrossRef]
5. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al.; Acute Pancreatitis Classification Working Group. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62:102–11. [CrossRef]
6. Wu BU, Johannes RS, Sun X, Tabak Y, Conwell DL, Banks PA. The early prediction of mortality in acute pancreatitis: a large population-based study. Gut 2008;57:1698–703. [CrossRef]
7. Gao W, Yang HX, Ma CE. The Value of BISAP score for predicting mortality and severity in acute pancreatitis: A systematic review and meta-analysis. PLoS One 2015;10:e0130412. Erratum in: PLoS One 2015;10:e0142025. [CrossRef]
8. Mortele KJ, Wiesner W, Intriere L, Shankar S, Zou KH, Kalantari BN, et al. A modified CT severity index for evaluating acute pancreatitis: improved correlation with patient outcome. AJR Am J Roentgenol 2004;183:1261–5. [CrossRef]
9. Mounzer R, Langmead CJ, Wu BU, Evans AC, Bishehsari F, Muddana V, et al. Comparison of existing clinical scoring systems to predict persistent organ failure in patients with acute pancreatitis. Gastroenterology 2012;142:1476–82. [CrossRef]
10. Song K, Wu Z, Meng J, Tian W, Zheng S, Mu D, et al. Hypertriglyceridemia as a risk factor for complications of acute pancreatitis and the development of a severity prediction model. HPB (Oxford) 2023;25:1065–73. [CrossRef]
11. Zhang X, Jin T, Shi N, Yao L, Yang X, Han C, et al. Mechanisms of pancreatic ınjury ınduced by basic amino acids differ between L-arginine, L-ornithine, and L-histidine. Front Physiol 2019;9:1922. [CrossRef]
12. Sun YM, Gao F, Chen X, Zhang J. The relationship between triglyceride level and the severity of acute hypertriglyceridemic pancreatitis in Chinese patients. Turk J Gastroenterol 2020;31:633–9. [CrossRef]
13. Wu Q, Zhong X, Fu M, Yang H, Bo H, Liao X, et al. High-density lipoprotein cholesterol to low-density lipoprotein cholesterol ratio in early assessment of disease severity and outcome in patients with acute pancreatitis admitted to the ICU. BMC Gastroenterol 2020;20:164. [CrossRef]
14. Venegas-Tamayo AR, Peña-Veites OM, Hernández-González MA, Barrientos-Alvarado C. Decreased HDL-C levels as a predictor of organ failure in acute pancreatitis in the emergency department. Life (Basel) 2023;13:1602. [CrossRef]
15. Chen M, Fang CY, Guo JC, Pang LM, Zhou Y, Hong Y, et al. Predictive value of atherogenic index of plasma and atherogenic index of plasma combined with low-density lipoprotein cholesterol for the risk of acute myocardial infarction. Front Cardiovasc Med 2023;10:1117362. [CrossRef]
16. Dobiásová M. AIP--aterogenní index plazmy jako významný prediktor kardiovaskulárního rizika: od výzkumu do praxe [AIP--atherogenic index of plasma as a significant predictor of cardiovascular risk: from research to practice]. Vnitr Lek 2006;52:64–71. [Czech]
17. Cho SK, Kim JW, Huh JH, Lee KJ. Atherogenic ındex of plasma ıs a potential biomarker for severe acute pancreatitis: A prospective observational study. J Clin Med 2020;9:2982. [CrossRef]
18. Wu W, Li Y, Zhang Y, Chen X, Zhang C, Qu X, et al. Atherogenic index of plasma as a novel predictor for acute kidney injury and disease severity in acute pancreatitis: a retrospective cohort study. Lipids Health Dis 2025;24:111. [CrossRef]
19. Marshall JC, Cook DJ, Christou NV, Bernard GR, Sprung CL, Sibbald WJ. Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. Crit Care Med 1995;23:1638–52. [CrossRef]
20. Vege SS, DiMagno MJ, Forsmark CE, Martel M, Barkun AN. Initial Medical Treatment of Acute Pancreatitis: American Gastroenterological Association Institute Technical Review. Gastroenterology 2018;154:1103–39. [CrossRef]
21. Hołyński J, Modzelewski B. Rola lipidów i lipoprotein w rozwoju ostrego zapalenia trzustki [The role of lipids and lipoproteins in acute pancreatitis]. Pol Merkur Lekarski 2001;11:517–21. [Polish]
22. Hidalgo NJ, Pando E, Alberti P, Vidal L, Mata R, Fernandez N, et al. Elevated serum triglyceride levels in acute pancreatitis: A parameter to be measured and considered early. World J Surg 2022;46:1758–67. [CrossRef]
23. Feingold KR, Grunfeld C. Effect of Inflammation and Infection on Lipids and Lipoproteins. 2025. In: Feingold KR, Ahmed SF, Anawalt B, Blackman MR, Boyce A, Chrousos G, et al., editors. Endotext. South Dartmouth (MA): MDText.com, Inc.; 2000.
24. Pirillo A, Catapano AL, Norata GD. HDL in infectious diseases and sepsis. Handb Exp Pharmacol 2015;224:483–508. [CrossRef]
25. Guan Y, Liu G, Tang F, Wu X, Shi J, Huang Q. Stress hyperglycemia in acute pancreatitis: From mechanisms to prognostic implications. Life Sci 2025;365:123469. [CrossRef]
26. Venkatesh NR, Vijayakumar C, Balasubramaniyan G, Chinnakkulam Kandhasamy S, Sundaramurthi S, G S S, Srinivasan K. Comparison of different scoring systems in predicting the severity of acute pancreatitis: A prospective observational study. Cureus 2020;12:e6943. [CrossRef]
27. Mofidi R, Duff MD, Wigmore SJ, Madhavan KK, Garden OJ, Parks RW. Association between early systemic inflammatory response, severity of multiorgan dysfunction and death in acute pancreatitis. Br J Surg 2006;93:738–44. [CrossRef]