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Comparison of inflammatory indices in patients with acute pancreatitis according to age and etiological factors

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Abstract

This study aimed to compare inflammatory indices derived from routine laboratory parameters in patients with acute pancreatitis according to age groups and etiological factors. This retrospective observational study included patients aged ≥ 18 years diagnosed with acute pancreatitis and admitted to the emergency department between January 1, 2020, and December 31, 2025. Age, etiological factors, and laboratory findings obtained at admission and within the first 24 hours were analyzed. Patients were classified into two age groups (< 65 years and ≥ 65 years) and as biliary or non-biliary acute pancreatitis. Inflammatory indices, including neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), pan-immune inflammation value (PIV), leukocyte glucose index (LGI), hemoglobin–albumin–lymphocyte–platelet score (HALP), C-reactive protein-to-albumin ratio (CAR), and CRP–albumin–lymphocyte index (CALLY), were calculated, and comparisons were performed using the Mann–Whitney U test. A total of 564 patients were included. Patients aged ≥ 65 years showed higher inflammatory indices than younger patients in both biliary and non-biliary acute pancreatitis. Inflammatory indices were also higher in patients with biliary etiology across both age groups. In contrast, HALP scores and CALLY values were lower in older patients and in those with biliary acute pancreatitis. Inflammatory indices derived from routine laboratory parameters showed distinct distributions according to age and etiology in acute pancreatitis, indicating age- and etiology-related heterogeneity in laboratory inflammatory profiles. These findings should be interpreted as descriptive and require further validation before any prognostic or clinical implications can be established.

Keywords: Acute pancreatitis, inflammatory indices, age, etiology

Introduction

Acute pancreatitis (AP) is a common gastrointestinal disorder characterized by sudden inflammation of the pancreas, with clinical and biochemical manifestations that may vary across a wide spectrum. Although the disease follows a self-limiting course in the majority of cases, a more complex biological profile may emerge in certain patient groups as a result of an exaggerated inflammatory response [1]. Epidemiological evidence indicates that the incidence of acute pancreatitis increases with advancing age, and that both clinical presentation and laboratory characteristics may differ markedly in elderly patients compared with younger individuals [1,2].

The etiological distribution of acute pancreatitis shows marked differences across age groups. While alcohol consumption and hypertriglyceridemia are more prominent etiological factors in younger patients, biliary pancreatitis is reported as the most common cause in the elderly population [1,2]. This distinction is primarily attributed to the increased prevalence of gallstone disease and age-related changes in metabolic profiles among older individuals.

Inflammation plays a central role in the pathogenesis of acute pancreatitis. The inflammatory process activated following pancreatic tissue injury is characterized by neutrophil infiltration, a reduction in lymphocyte counts, and platelet activation [3]. This cellular response is not confined to local pancreatic inflammation but also leads to systemic biochemical and hematological alterations that can be measured at the systemic level. Therefore, laboratory parameters reflecting inflammation constitute an important component in the assessment of the biological response in acute pancreatitis [3].

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Inflammatory indices derived from routine complete blood count parameters enable a quantitative evaluation of this biological process. Indicators such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII) have attracted increasing attention in recent years as reflections of inflammatory activity in patients with acute pancreatitis [3]. The ease of calculation and availability of these indices from routine laboratory tests have contributed to their widespread use in clinical research [3].

Previous studies have reported that levels of these inflammatory indices may show significant variability in patients with acute pancreatitis and can reveal distinct biochemical patterns of the inflammatory response. However, available data regarding how these indices are distributed across different age groups and the extent to which age-related biological differences are reflected in laboratory findings remain limited [1,2]. In particular, alterations in immune response among elderly patients may render the interpretation of inflammatory indices more complex.

In elderly patients, the laboratory profile of acute pancreatitis is shaped by a different biological background compared with younger individuals. Age-related structural and functional alterations of the immune system (immunosenescence), chronic low-grade inflammation, and the higher burden of comorbid conditions in older populations have been reported to influence the levels and interpretation of inflammatory markers [2,4]. As a result, laboratory values obtained during acute pancreatitis may exhibit distinct patterns across different age groups. Therefore, age emerges as an important variable that should be taken into account when evaluating the laboratory manifestations of the inflammatory response [1].

In this context, examining differences in laboratory parameters and inflammatory indices between elderly and younger patients diagnosed with acute pancreatitis may provide a more detailed insight into the biological response of the disease [3]. The primary objective was to identify age- and etiology-associated differences in laboratory inflammatory profiles rather than to evaluate the prognostic performance of these indices for disease severity or clinical outcomes. By examining these indices within predefined demographic and etiological strata, the study sought to provide a more detailed description of the biological heterogeneity of the systemic inflammatory response in acute pancreatitis [1]. The specific contribution of this approach is the simultaneous assessment of multiple routinely derived inflammatory and composite indices across both age and etiological strata within the same cohort, allowing a broader characterization of subgroup-related laboratory inflammatory patterns than evaluation of individual indices in isolation. Therefore, this study aimed to characterize and compare routinely derived inflammatory and composite indices in patients with acute pancreatitis according to age groups (< 65 and ≥ 65 years) and etiological categories (biliary and non-biliary).

Material and Methods

This study was designed as a retrospective, observational, and comparative analysis. A total of 564 patients aged 18 years and older who were diagnosed with acute pancreatitis and admitted to the emergency department between January 1, 2020, and December 31, 2025 were included in the study. The diagnosis of acute pancreatitis was established based on the presence of at least two of the following three criteria: typical abdominal pain consistent with acute pancreatitis, serum amylase and/or lipase levels at least three times the upper limit of normal, and characteristic findings of acute pancreatitis on imaging. The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Kırşehir Ahi Evran University Faculty of Medicine Scientific Research Ethics Committee (Decision No: 2026-03/34, Date: 3 February 2026) prior to data collection. Due to the retrospective design of the study and the use of anonymized patient data, informed consent was not required.

Age, etiological category, and routine laboratory findings obtained at admission and within the first 24 hours were retrospectively collected from the hospital information management system. The evaluated laboratory parameters included complete blood count, C-reactive protein (CRP), albumin, and routine biochemical markers.

Using the obtained data, laboratory-derived inflammatory and composite indices were calculated, including the NLR, PLR, eosinophil-to-monocyte ratio (EMR), SII, pan-immune inflammation value (PIV), multi-inflammation indices (MII-1, MII-2, MII-3), hemoglobin–albumin–lymphocyte–platelet score (HALP), C-reactive protein-to-albumin ratio (CAR), C-reactive protein–albumin–lymphocyte index (CALLY), and leukocyte glucose index (LGI).

Patients were stratified into subgroups according to age (< 65 years and ≥ 65 years) and the etiology of acute pancreatitis, and the distributions of inflammatory indices were compared across these groups. For the present analyses, etiology was categorized as biliary or non-biliary. Detailed subclassification of the non-biliary etiologies was not available in the retrospective dataset; therefore, separate analyses according to specific non-biliary causes could not be performed. The analytical framework of the study was designed to characterize differences in laboratory-derived inflammatory profiles across age and etiological groups rather than to assess associations with disease severity or clinical outcomes.

Statistical Analysis

Statistical analyses of the study were performed using the Statistical Package for the Social Sciences (SPSS) software for Windows, version 29.0 (IBM SPSS Statistics for Windows, Version 29.0; Armonk, NY: IBM Corp., USA).

The distribution characteristics of continuous variables were assessed using the Shapiro–Wilk test and histogram inspections. Continuous variables that did not meet the assumption of normality were presented as median and interquartile range

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(IQR). Patients were classified according to age groups (< 65 years and ≥ 65 years) and etiology (biliary and non-biliary). Since the assumption of normality was not satisfied, the Mann–Whitney U test was used for comparisons between two independent groups. Because the analyses were exploratory and descriptive in nature, no formal adjustment for multiple comparisons was applied. Therefore, statistically significant findings should be interpreted as descriptive and hypothesis-generating rather than confirmatory. Because comorbidities, medication use, and other relevant potential confounders were not systematically available in the retrospective dataset, multivariable regression analyses were not performed. Accordingly, the reported subgroup differences should not be interpreted as independent effects of age or etiology.

Accordingly, inflammatory indices (NLR, PLR, EMR, SII, MII-1, MII-2, MII-3, PIV, and LGI), composite nutrition- and inflammation-related indices (HALP, CALLY, and CAR), the urea-to-albumin (UALB), and the CRP-to-lymphocyte ratio (CRP/LYM) were compared between age groups and etiological subgroups.

Results

In this study, the distribution of inflammatory indices and selected biochemical parameters was comparatively evaluated in patients with acute pancreatitis according to age groups (< 65 years and ≥ 65 years) and etiology (biliary and non-biliary). The results are presented in a structured manner, beginning with baseline data reflecting the age-group and etiological distribution of the patient population, followed by metabolic and inflammatory ratios, and finally composite indices related to nutrition and inflammation. This approach aims to systematically demonstrate how age- and etiology-related biological differences are reflected in laboratory patterns.

A total of 564 patients with acute pancreatitis were included in the study. Of these patients, 289 (51.2%) were younger than 65 years, while 275 (48.8%) were aged 65 years or older. Regarding etiological distribution, 286 cases (50.7%) were diagnosed with biliary acute pancreatitis, whereas 278 cases (49.3%) had non-biliary acute pancreatitis. The patient population demonstrated a balanced distribution with respect to both age groups and etiological categories (Table 1).

Table 1. Distribution of patients according to age group and etiology

Variable	n (%)
Total number of patients	564
Age group	
< 65 years	289 (51.2)
≥ 65 years	275 (48.8)
Etiology	
Biliary	286 (50.7)
Non-biliary	278 (49.3)

n: number of patients

When evaluated in terms of the glucose-to-potassium ratio (G/K ratio), scores were found to be significantly higher in patients aged ≥ 65 years compared with those aged < 65 years in both biliary and non-biliary acute pancreatitis (biliary: p = .02; non-biliary: p = .008). In addition, within both age groups, patients with biliary etiology exhibited significantly higher G/K values than those with non-biliary acute pancreatitis (< 65 years: p = .003; ≥ 65 years: p = .038).

When the urea-to-albumin ratio was evaluated, values were found to be significantly higher in elderly patients in both biliary and non-biliary groups (p < .001 for both etiologies). In contrast, no statistically significant difference in urea-to-albumin ratio values was observed between biliary and non-biliary etiologies within the same age groups (< 65 years: p = .412; ≥ 65 years: p = .057) (Table 2).

Table 2. Comparison of selected biochemical ratios according to age groups and etiology

Variable	Etiology	< 65 years	≥ 65 years	p†
G/K ratio	**Biliary**	31.85 (14.71)	35.12 (19.33)	.02
	Non-biliary	26.97 (13.57)	30.54 (17.10)	.008
	p‡	.003	.038	
UALB ratio	**Biliary**	0.67 (0.38)	1.02 (0.61)	< .001
	Non-biliary	0.70 (0.41)	1.09 (0.83)	< .001
	p‡	.412	.057	

Data are presented as median (interquartile range, IQR); † Comparison of age groups within the same etiology (Mann–Whitney U test); ‡ Comparison between etiologies within the same age group (Mann–Whitney U test); G/K ratio: glucose-to-potassium ratio, UALB ratio: urea-to-albumin ratio, IQR: interquartile range

When inflammatory indices were evaluated according to age groups and etiology, marked differences were observed among patients with acute pancreatitis. The NLR was found to be significantly higher in patients aged ≥ 65 years compared with those aged < 65 years in both biliary and non-biliary etiologies (p < .001 for both). Moreover, within each age group, NLR values were significantly higher in patients with biliary acute pancreatitis than in those with non-biliary etiology (p < .001).

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When the PLR was examined, a significant increase was observed in elderly patients in both biliary and non-biliary groups (biliary: $p < .001$; non-biliary: $p = .003$). In addition, within both age groups, PLR values were significantly higher in patients with biliary etiology compared with those with non-biliary acute pancreatitis ($p < .001$). Regarding the EMR, no significant age-related difference was detected in the biliary group ($p = .102$), whereas EMR values were significantly lower in patients aged ≥ 65 years in the non-biliary group ($p = .02$). EMR values differed significantly between biliary and non-biliary etiologies in both age groups.

The SII and MII-1, MII-2, and MII-3 were found to be significantly higher in patients aged ≥ 65 years in both biliary and non-biliary etiologies. Moreover, for all of these indices, values were significantly higher in patients with biliary acute pancreatitis compared with those with non-biliary etiology across both age groups ($p < .001$ for all comparisons).

Similarly, the PIV and LGI showed a significant increase in elderly patients across both etiologies. In addition, in both the < 65 and ≥ 65 age groups, PIV and LGI values were significantly higher in patients with biliary acute pancreatitis compared with those with non-biliary etiology (Table 3).

Table 3. Comparison of inflammatory indices according to age groups and etiology

Variable	Etiology	< 65 years	≥ 65 years	p†
NLR	Biliary	5.12 (5.91)	8.48 (10.02)	< .001
	Non-biliary	2.84 (2.90)	4.66 (5.21)	< .001
	p‡	< .001	< .001	
PLR	Biliary	170.04 (148.55)	227.77 (203.85)	< .001
	Non-biliary	109.18 (73.97)	135.94 (90.34)	.003
	p‡	< .001	< .001	
EMR	Biliary	0.11 (0.18)	0.08 (0.18)	.102
	Non-biliary	0.19 (0.24)	0.14 (0.22)	.02
	p‡	< .001	.001	
SII	Biliary	1409.36 (1536.36)	2152.34 (2509.15)	< .001
	Non-biliary	704.80 (815.72)	1018.50 (1242.47)	< .001
	p‡	< .001	< .001	
MII-1	Biliary	288.22 (497.06)	477.86 (836.03)	< .001
	Non-biliary	76.69 (177.52)	150.84 (252.62)	< .001
	p‡	< .001	< .001	
MII-2	Biliary	9760.45 (14096.52)	11240.55 (22745.71)	.02
	Non-biliary	3150.57 (5558.40)	4476.68 (5887.63)	.02
	p‡	< .001	< .001	
MII-3	Biliary	67224.53 (134704.32)	109901.26 (195090.85)	.002
	Non-biliary	21001.74 (42277.31)	32558.82 (64464.37)	.006
	p‡	< .001	< .001	
PIV	Biliary	826.02 (1098.20)	1185.76 (1540.33)	.007
	Non-biliary	560.62 (686.70)	700.61 (1262.77)	.009
	p‡	< .001	.003	
LGI	Biliary	1.08 (1.03)	1.49 (1.19)	< .001
	Non-biliary	0.84 (0.70)	1.10 (1.10)	.002
	p‡	.003	.002	
CRP/LYM	Biliary	38.99 (50.38)	51.05 (90.49)	.001
	Non-biliary	13.03 (22.54)	18.53 (30.02)	.001
	p‡	< .001	< .001	

Data are presented as median (interquartile range, IQR); NLR: neutrophil-to-lymphocyte ratio, PLR: platelet-to-lymphocyte ratio, EMR: eosinophil-to-monocyte ratio, SII: systemic immune-inflammation index, MII-1, MII-2, and MII-3: multi-inflammation indices, PIV: pan-immune inflammation value, LGI: leukocyte glucose index, CRP/LYM: C-reactive protein-to-lymphocyte ratio; † Comparison of age groups within the same etiology (Mann–Whitney U test); ‡ Comparison between etiologies within the same age group (Mann–Whitney U test)

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When the C-reactive protein–to-lymphocyte ratio (CRP/LYM) was evaluated, a significant increase was observed in patients aged ≥ 65 years in both biliary and non-biliary groups. Additionally, across both age groups, CRP/LYM values were higher in patients with biliary etiology than in those with non-biliary acute pancreatitis ($p < .001$ for all comparisons).

When composite indices reflecting nutritional and inflammatory status were evaluated, significant differences were observed between age groups in both biliary and non-biliary acute pancreatitis patients. The HALP score was found to be significantly lower in patients aged ≥ 65 years compared with those aged < 65 years in both etiologies ($p < .001$ for all comparisons). In addition, across both age groups, HALP scores were significantly lower in patients with biliary etiology than in those with non-biliary acute pancreatitis ($p < .001$).

Similarly, the CALLY index showed a significant decrease in elderly patients in both biliary and non-biliary groups ($p < .001$ for both etiologies). Moreover, in both the < 65 and ≥ 65 age groups, CALLY values were significantly lower in patients with biliary etiology compared with those with non-biliary acute pancreatitis ($p < .001$).

In contrast, the CAR did not differ significantly between age groups. However, in both age categories (< 65 years and ≥ 65 years), CAR values were significantly higher in patients with biliary etiology than in those with non-biliary acute pancreatitis (Table 4).

Table 4. Comparison of composite nutrition- and inflammation-related indices according to age groups and etiology

Variable	Etiology	< 65 years	≥ 65 years	p†
HALP	Biliary	3.39 (2.59)	2.31 (2.17)	< .001
	Non-biliary	5.49 (3.95)	4.10 (3.27)	< .001
	p‡	< .001	< .001	
CALLY	Biliary	11.53 (17.48)	7.87 (12.18)	< .001
	Non-biliary	34.09 (63.34)	21.23 (34.43)	< .001
	p‡	< .001	< .001	
CAR	Biliary	12.40 (19.62)	13.25 (18.85)	.24
	Non-biliary	6.53 (10.33)	7.63 (11.19)	.10
	p‡	< .001	< .001	

Data are presented as median (interquartile range, IQR); HALP: hemoglobin–albumin–lymphocyte–platelet score, CALLY: C-reactive protein–albumin–lymphocyte index, CAR: C-reactive protein-to-albumin ratio; † Comparison of age groups within the same etiology (Mann–Whitney U test); ‡ Comparison between etiologies within the same age group (Mann–Whitney U test)

Discussion

In this study, the distribution of inflammatory indices derived from routine laboratory parameters was evaluated in patients with acute pancreatitis across age (< 65 and ≥ 65 years) and etiological (biliary and non-biliary) axes. Our findings indicate that indices reflecting the systemic inflammatory response tend to be higher in elderly patients and in those with biliary etiology, whereas certain composite indices integrating inflammatory and nutritional status are observed at lower levels in these patient groups.

The principal contribution of the present study lies in the simultaneous evaluation of a broad panel of routinely derived inflammatory and composite indices across both age and etiological strata within the same cohort. This two-dimensional comparative approach provides a more detailed characterization of laboratory inflammatory heterogeneity than studies focusing on a single index, a single age category, or a single etiological subgroup.

A consistent finding across the present analyses was the higher distribution of several inflammatory indices, including NLR, PLR, SII, PIV, and related composite measures, in patients aged ≥ 65 years. Rather than demonstrating prognostic significance, this pattern indicates that older and younger patients exhibit distinct laboratory inflammatory profiles in acute pancreatitis. This observation suggests that patient age may represent an important contextual factor when interpreting inflammation-related indices, although the clinical implications of these differences require further validation. Previous studies have described age-related differences in systemic inflammation and biochemical characteristics in acute pancreatitis, supporting the plausibility of this finding [2,5-8].

The observed increase, particularly in neutrophil- and lymphocyte-based indices, is consistent with the concepts of immunosenescence and "inflammaging" described in advanced age. Previous studies have reported that cellular-based inflammatory indices such as NLR, PLR, and SII are detected at higher levels in elderly patients with acute pancreatitis and that this elevation may reflect the systemic inflammatory response [2,5,9-11]. These findings further support the presence of age-related differences in laboratory inflammatory profiles.

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A second notable finding was the consistently higher distribution of several inflammatory indices in patients with biliary acute pancreatitis compared with those classified as non-biliary. This pattern suggests that etiological background is another factor associated with variation in laboratory inflammatory profiles and supports the interpretation of these indices within an etiological context rather than in isolation.

Previous studies have also reported differences in inflammatory and biochemical characteristics across etiological forms of acute pancreatitis, providing biological plausibility for this observation [2,3,12-15]. However, because the non-biliary category could not be further subclassified in the present dataset, these findings should be interpreted as comparisons between broad etiological categories and not as effects attributable to specific non-biliary causes.

An additional contribution of the present study is the demonstration that the observed subgroup differences were not limited to indices that increase with systemic inflammation. Composite indices integrating inflammatory and nutritional components, particularly HALP and CALLY, showed the opposite pattern, with lower values in older patients and in those with biliary etiology. Evaluating these indices alongside conventional inflammation-based measures provides a broader description of the laboratory phenotype by capturing complementary dimensions of the biological response. Previous studies have suggested that albumin-based and composite indices may reflect interactions among inflammation, nutritional status, and physiological reserve [2,4,6,16–18]. However, because nutritional status, chronic disease burden, and related determinants were not directly assessed in the present study, the mechanisms underlying these differences cannot be established.

Unlike most of the other evaluated indices, CAR did not differ significantly between age groups, although higher values were observed in patients with biliary etiology. This divergence from the age-related pattern observed for several other indices suggests that not all routinely derived inflammatory measures respond uniformly across demographic and etiological strata. Previous studies have evaluated CAR as a marker of inflammatory status in acute pancreatitis, but its distribution across age groups has not been consistently demonstrated [11,16,19]. In the present study, the differing pattern of CAR further illustrates the heterogeneity among laboratory-derived inflammatory indices and supports the value of evaluating multiple complementary measures rather than relying on a single index.

A major strength of this study is the simultaneous evaluation of a broad spectrum of routinely derived inflammatory, nutritional, and composite indices within the same patient cohort. Rather than examining a single biomarker or a single patient characteristic, the study compared these indices across both age and etiological dimensions, allowing identification of convergent as well as divergent laboratory patterns among patient subgroups. This integrated comparative approach provides a broader characterization of inflammatory heterogeneity in acute pancreatitis and may help contextualize the interpretation of individual indices according to patient age and etiological background. Nevertheless, these findings should be considered descriptive and hypothesis-generating because their prognostic and clinical utility was not directly evaluated.

Taken together, these findings suggest that age and etiology represent important contextual factors in the interpretation of laboratory-derived inflammatory indices in acute pancreatitis. The simultaneous evaluation of multiple indices demonstrated that inflammatory profiles are not uniform across patient subgroups and that different indices may reflect distinct biological dimensions of the disease. This broader comparative perspective represents the main contribution of the present study and provides a basis for further investigation of how routinely available laboratory indices vary across demographic and etiological subgroups. However, because disease severity and clinical outcomes were not directly evaluated, the present findings should be regarded as descriptive and hypothesis-generating rather than as evidence of prognostic or clinical utility [1,2,5,6,10,12].

Limitations

This study should be interpreted in light of several limitations. First, the retrospective and single-center design precludes causal inferences and may limit the generalizability of the findings. Data were obtained retrospectively from the hospital information system, which may have introduced potential biases related to missing records and heterogeneity in measurement timing. Second, the analysis was restricted to laboratory parameters obtained at admission and within the early phase of hospitalization; therefore, temporal changes in inflammatory indices during the disease course could not be evaluated.

In addition, standardized clinical severity measures, including the Revised Atlanta Classification and organ failure status, as well as clinical outcomes such as complication development, intensive care unit requirement, and mortality, were not included in the present analysis. Therefore, the observed differences in inflammatory indices should be interpreted as differences in laboratory inflammatory profiles rather than as markers of disease severity or prognosis. The prognostic relevance of these age- and etiology-associated differences requires further evaluation in studies incorporating standardized severity classifications and clinically relevant outcomes.

Moreover, etiological classification was limited to broad biliary and non-biliary categories. Because detailed subclassification of non-biliary etiologies was not available, the potential heterogeneity within this group could not be addressed through separate subgroup analyses and may have influenced the observed etiological comparisons.

Furthermore, because multiple statistical comparisons were performed and no formal adjustment for multiplicity was applied,

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the findings should be interpreted cautiously and regarded as exploratory and hypothesis-generating until confirmed by future prospective studies.

Finally, detailed baseline characteristics, including sex, comorbidities, medication use, nutritional status, and other relevant clinical variables, were not systematically available in the retrospective dataset. Therefore, these variables could not be compared between groups or incorporated into multivariable analyses. Because several of these factors may independently influence inflammatory and nutritional indices, residual confounding may have contributed to the observed differences, particularly those associated with age. Accordingly, the observed subgroup differences should not be interpreted as independent effects of age or etiology.

Conclusion

Inflammatory indices derived from routinely available laboratory parameters showed different distributions according to age and broad etiological categories in patients with acute pancreatitis. Older patients and those with biliary etiology demonstrated distinct inflammatory and composite laboratory profiles; however, these findings are descriptive and should not be interpreted as evidence of disease severity, prognosis, or clinical utility. Given the retrospective, single-center design and the absence of standardized severity and clinical outcome data, prospective multicenter studies with appropriate adjustment for potential confounders are needed to determine the clinical relevance of these observations.

Ethical Approval

This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Kırşehir Ahi Evran University Faculty of Medicine Scientific Research Ethics Committee (Decision No: 2026-03/34, Date: 3 February 2026) prior to the initiation of the study.

Patient Consent

Due to the retrospective design of the study and the use of anonymized patient data, informed consent was not required.

Conflict of Interest

The authors confirm the absence of any conflicts associated with this work.

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