

Prognostic role of systemic inflammatory indices in predicting the severity of acute calculous cholecystitis

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ABSTRACT

BACKGROUND: Acute calculous cholecystitis (ACC) is one of the most common conditions encountered in emergency medicine and surgical practice. Delayed recognition of severe cases can lead to complications such as empyema, gangrene, or perforation, resulting in high morbidity and mortality. While the Tokyo Guidelines provide standardized diagnostic and severity grading criteria, the availability and reliability of imaging may be limited in certain settings. Therefore, there is growing interest in simple and cost-effective biomarkers. This study aimed to evaluate the diagnostic and prognostic value of complete blood count-derived (CBC-derived) systemic inflammatory indices, including neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), neutrophil-to-lymphocyte \times platelet ratio (NLPR), systemic immune-inflammation index (SII), and multiple inflammatory index (MII), in predicting disease severity in patients with ACC.

METHODS: A total of 160 patients diagnosed with ACC in the emergency department between January 2020 and May 2024 were retrospectively analyzed. Patients with acalculous cholecystitis, cholangitis, choledocholithiasis, incomplete data, or age younger than 18 years were excluded. Demographic, clinical, and laboratory findings were reviewed. Receiver operating characteristic (ROC) curve analysis was used to determine optimal cut-off values.

RESULTS: The strongest predictive performance was observed for MII (cut-off=250,01 l; sensitivity 76.9%; specificity 78.9%; area under the curve [AUC]=0.770). NLR (cut-off=8.45) showed 76.9% sensitivity and 68.0% specificity (AUC=0.755). NLPR (cut-off=0.027) had 76.9% sensitivity and 54.4% specificity, while SII (cut-off=2414) achieved 69.2% sensitivity and 72.1% specificity (all $p<0.05$). All indices were significant predictors of severe ACC.

CONCLUSION: CBC-derived systemic inflammatory indices, particularly MII and NLR, are effective, accessible, and inexpensive markers for predicting the severity of ACC. These parameters may complement clinical assessment and assist in decision-making, especially in situations where imaging is unavailable or inconclusive.

Keywords: Severe acute cholecystitis; gallstones; systemic immune-inflammation index; emergency medicine.

INTRODUCTION

Acute calculous cholecystitis (ACC) is among the most common causes of acute abdominal pain and a major indication for urgent surgical intervention worldwide.^[1] Despite advances in imaging, delayed diagnosis or misjudgment of disease severity can result in complications such as empyema, gangrene, or perforation, which substantially increase morbidity and mortality.^[2]

The Tokyo Guidelines (TG13/18) provide standardized diagnostic and severity grading criteria by integrating clinical, laboratory, and imaging findings.^[3,4] They recommend early cholecystectomy for Grade I and II cases, whereas Grade III cases with organ dysfunction require urgent drainage followed by delayed surgery.^[5] However, reliance on imaging reduces applicability in resource-limited settings, and up to 28% of patients—particularly older adults—may present without classical signs such as fever or leukocytosis.^[6]

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Recently, systemic inflammatory indices such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), neutrophil-to-lymphocyte-platelet ratio (NLPR), and systemic immune-inflammation index (SII) have been proposed as cost-effective and widely available biomarkers for disease severity in ACC.^[7-9] Derived from routine complete blood counts, these indices reflect the balance between immune activation and systemic inflammation, and their prognostic value has also been emphasized in emergency medicine more broadly.^[10] Nevertheless, their clinical utility and optimal cut-off values remain uncertain, particularly in patients with ACC.^[9]

This study aimed to evaluate the prognostic performance of systemic inflammatory indices in predicting ACC severity, determine their optimal cut-off values, and explore their potential role in clinical risk stratification.

MATERIALS AND METHODS

A retrospective study was conducted on patients admitted to the Emergency Department of Balikesir University Health Practice and Research Hospital with acute cholecystitis between January 1, 2020 and May 31, 2024. Data were collected from hospital archives and electronic patient records. All patients aged 18 years or older who were admitted with a diagnosis of acute calculous cholecystitis and subsequently evaluated by the General Surgery Department were included in the statistical analysis. Initially, 276 patients were identified; however, 116 patients with acute acalculous cholecystitis, cholangitis, or choledocholithiasis, and 20 patients with incomplete data were excluded from the analysis. Consequently, 160 patients with acute calculous cholecystitis were included in the final analysis.

Ultrasonography (US) and abdominal computed tomography (CT) were used to detect stones, measure gallbladder wall thickness, identify signs of pericholecystitis, and evaluate both local and systemic inflammatory indicators. For all patients, demographic and clinical data—including age, comorbidities, symptom onset time, and clinical signs—were recorded. Laboratory tests included complete blood count (CBC), hepatic transaminases, bilirubin, urea, creatinine, albumin, international normalized ratio (INR), and C-reactive protein (CRP) at the time of admission.

Patients were classified according to the TG13/18 guidelines into three severity grades: Grade I (mild), Grade II (moderate), and Grade III (severe) acute cholecystitis. Systemic inflammatory biomarkers were calculated from neutrophil (N), platelet (P), and lymphocyte (L) counts obtained from the same blood sample. Based on these values, NLR, PLR, NLPR, SII, and MII (multiple inflammatory index) were calculated as follows: NLR (neutrophil-to-lymphocyte ratio), PLR (platelet-to-lymphocyte ratio), NLPR (neutrophil / (lymphocyte × platelet) ratio), SII ((neutrophils × platelets) / lymphocytes), and MII (SII × CRP).

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the Balikesir University Clinical Research Ethics Committee (Approval Number: 2024/124, Date: 06.08.2024). Written informed consent was waived due to the retrospective nature of the study.

Statistical Analysis

All statistical analyses were performed using SPSS (Statistical Package for the Social Sciences, version 28.0). The normality of numerical data was tested using the Shapiro-Wilk test. Descriptive statistics for numerical data are presented as means ± standard deviations or medians (Q1-Q3). Frequencies (n) and percentages (%) are provided for categorical data. Group comparisons were performed using the Chi-square test for categorical variables and the Kruskal-Wallis H test for numerical variables. Receiver Operating Characteristic (ROC) curve analysis was employed to identify optimal cut-off points by maximizing sensitivity and specificity based on the Youden J index.

RESULTS

A total of 160 patients diagnosed with ACC were included in the study, with a mean age of 56.7±17.5 years (range, 18–92). The majority were classified as moderate cases (Grade II, n=96, 60%), followed by mild (Grade I, n=51, 31.9%) and severe (Grade III, n=13, 8.1%). Female patients accounted for 51.2% of the cohort. Patients with severe ACC (Grade III) were significantly older (68.8±12.9 years, p=0.018) compared to those with Grades I and II, and male patients were more common in Grades II and III. Hypertension (26.9%), diabetes mellitus (12.5%), and coronary artery disease (9.4%) were the most frequent comorbidities, with coronary artery disease significantly more prevalent in Grade III cases (p=0.032). Most patients were admitted more than 72 hours after symptom onset (40.6%). The majority required hospitalization (88.8%), and 61.2% did not undergo surgical intervention (Table 1).

Laboratory analyses revealed significantly higher values in severe cases for leukocyte count (p=0.033), neutrophil count (p=0.015), lymphocyte count (p=0.010), CRP (p=0.001), NLR (p=0.001), PLR (p=0.046), NLPR (p=0.004), SII (p=0.004), and MII (p=0.001). No significant differences were observed for platelet counts, aspartate aminotransferase (AST), or alanine aminotransferase (ALT) levels. Median CRP levels increased progressively with disease severity, ranging from 6.7 mg/L in Grade I to 122.2 mg/L in Grade III (p<0.001). Similarly, NLR increased from 3.5 in mild cases to 11.0 in severe cases (p<0.001). Complete laboratory comparisons are presented in Table 2.

ROC curve analysis demonstrated that NLR, NLPR, CRP, SII, and MII had moderate predictive accuracy for severe ACC (area under the curve [AUC]=0.70–0.88), while PLR and white blood cell count (WBC) showed lower accuracy

Table 1. Comparison of demographic and clinical characteristics of patients categorized according to the TGI3/18 severity grades of acute calculous cholecystitis

	GRADE I	GRADE II	GRADE III	TOTAL	p
Age, mean±SD	53.43±16.15	56.82±18.14	68.77±12.89	56.71±17.51	0.018 ^{1*}
Female, N, %	32	45	5	82 (51.2%)	0.117 ²
Male, N, %	19	51	8	78 (48.8%)	
US, N, %	42	70	9	121 (75.6%)	0.369 ²
CT, N, %	31	61	8	100 (62.5%)	0.945 ²
Comorbidities					
Hypertension	11	27	5	43 (26.9%)	0.435 ²
Diabetes mellitus	5	13	2	20 (12.5%)	0.760 ²
Coronary artery disease	2	9	4	15 (9.4%)	0.032 ²
Chronic respiratory disease	2	3	0	5 (3.1%)	0.630 ²
Complaint initiation period					
<24 h, N	18	23	1	42 (26.3%)	0.121 ²
24-72 h, N	18	31	4	53 (33.1%)	
>72 h, N	15	42	8	65 (40.6%)	
Hospitalization					
Discharged, N	8	1	0	9 (5.6%)	0.0222 ³
Hospitalized, N	40	90	12	142 (88.8%)	
Transfer, N	2	4	1	7 (4.4%)	
Treatment rejection	1	1	0	2 (1.3%)	
Length of hospitalization, days, mean±SD	4.38±1.78	5.17±2.36	6.58±3.18	5.06±2.35	0.119 ¹
Surgery					
Non-surgical management	28	63	7	98 (61.20%)	0.601 ²
<30 days surgery	15	18	3	36 (22.50%)	
>30 days surgery	8	15	3	26 (16.25%)	
TOTAL	51 (31.9%)	96 (60%)	13 (8.1%)	160 (100%)	

¹ANOVA; ²Pearson chi-square or Fisher's exact test. Data are presented as mean±standard deviation or number (percentage). *P<0.05. SD: Standard deviation; US: Ultrasonography; CT: Computed tomography; ACC: Acute calculous cholecystitis.

(AUC=0.54–0.69). The optimal cut-off values were: NLR 8.45 (sensitivity 76.9%, specificity 68.0%), PLR 219.2 (sensitivity 69.2%, specificity 68.7%), NLPR 0.027 (sensitivity 76.9%, specificity 54.4%), CRP 78.45 mg/L (sensitivity 76.9%, specificity 66.0%), SII 2414 (sensitivity 69.2%, specificity 72.1%), and MII 250,011 (sensitivity 76.9%, specificity 78.9%). Among these, MII (AUC=0.770) and NLR (AUC=0.755) were the strongest predictors of severe ACC, whereas WBC had poor discriminative performance (AUC=0.541, p=0.628). Detailed results are summarized in Table 3 and illustrated in Figure 1.

DISCUSSION

Cholelithiasis and cholecystitis remain among the most common causes of abdominal pain requiring emergency department (ED) evaluation, often leading to hospital admission and urgent surgical intervention. Accurate early recognition

of severe cases is crucial because delayed treatment can result in complications such as empyema, gangrene, or perforation, which substantially increase morbidity and mortality. Although the Tokyo Guidelines (TG18) offer a standardized framework for diagnosis and severity grading, they depend primarily on imaging modalities, which may not be consistently available in resource-limited environments and are subject to interobserver variability.^[11] Therefore, there is growing interest in cost-effective biomarkers that are widely available, reproducible, and capable of supporting clinical decision-making in the ED.

Systemic inflammatory indices have been extensively studied in recent years as prognostic markers in a range of acute and chronic diseases.^[12] These indices, including the neutrophil-to-lymphocyte ratio, neutrophil-to-lymphocyte-platelet ratio, platelet-to-lymphocyte ratio, systemic immune-inflammation

Table 2. Laboratory findings of patients with acute calculous cholecystitis, stratified by severity

	Mean±SD	Q1	Q3	P
Hemoglobin, g/dL	12.9±1.82	11.72	14.1	0.958
White blood cells, /mm ³	12.41±5.14	9.03	14.83	0.033*
Neutrophils, ×10 ³ /mm ³	9.89±5.04	6.7	12.2	0.015*
Lymphocytes, ×10 ³ /mm ³	1.59±0.95	1	1.9	0.01*
Platelets, ×10 ³ /mm ³	263.52±80.66	209.5	304	0.508
BUN, mg/dL	15.78±7.99	10.4	19.04	0.016*
Urea, mg/dL	33.77±17.08	22.25	40.75	0.019*
Creatinine, mg/dL	0.94±0.34	0.71	1.07	0.007*
AST, IU/L	67.21±108.92	18	61.75	0.447
ALT, IU/L	69.22±116.86	14	56	0.419
GGT, IU/L	109.15±176.45	22	88.5	0.921
ALP, IU/L	104.58±94.43	61.25	109.5	0.327
Total bilirubin, mg/dL	1.23±1.17	0.56	1.42	0.005*
Direct bilirubin, mg/dL	0.46±0.76	0.11	0.46	0.006*
Indirect bilirubin, mg/dL	0.77±0.55	0.41	0.96	0.037*
INR (sec)	1.08±0.13	1	1.14	0.001*
CRP, mg/dL	69.43±72.32	6.73	122.25	0.001*
Albumin, g/dL	36.28±5.12	33	40	0.001*
NLR	9.1±8.97	3.46	11.05	0.001*
PLR	217.25±157.28	132.09	256.6	0.046*
NLPR	0.04±0.05	0.01	0.05	0.004*
SII	2329.27±2511	905.87	2661.67	0.004*
MII	215,183.86±371,341	8711.5	251,233.5	0.001*

*P<0.05. BUN: Blood urea nitrogen; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; GGT: Gamma-glutamyl transferase; ALP: Alkaline phosphatase; INR: International normalized ratio; CRP: C-reactive protein; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; NLPR: Neutrophil-to-lymphocyte-platelet ratio; SII: Systemic immune-inflammation index; MII: Multiple inflammatory index.

Table 3. Receiver operating characteristic (ROC) curve analysis of systemic inflammatory indices for predicting severe acute calculous cholecystitis

	AUC	Cut-off	Youden J Index	P value	Sensitivity (%)	Specificity (%)
NLR	0.755 (0.609-0.90)	8.45	0.450	0.002*	76.92	68.03
PLR	0.696 (0.514-0.878)	219.2	0.379	0.019*	69.23	68.71
NLPR	0.735 (0.544-0.866)	0.027	0.313	0.015*	76.92	54.42
WBC	0.541 (0.365-0.716)	11.35	0.119	0.628	61.54	50.34
CRP	0.728 (0.585-0.872)	78.45	0.429	0.006*	76.90	66.00
SII	0.717 (0.558-0.877)	2414	0.413	0.009*	69.23	72.11
MII	0.770 (0.625-0.916)	250,011	0.558	0.001*	76.90	78.90

AUC: Area under the curve; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; NLPR: Neutrophil-to-lymphocyte-platelet ratio; WBC: White blood cell count; CRP: C-reactive protein; SII: Systemic immune-inflammation index; MII: Multiple inflammatory index.

index, and multiple inflammatory index, are derived from routine complete blood count parameters. They reflect the interplay between immune activation and systemic inflamma-

tion. In acute calculous cholecystitis, gallbladder obstruction and bacterial invasion activate innate immunity, triggering neutrophil proliferation, lymphocyte apoptosis, and platelet-

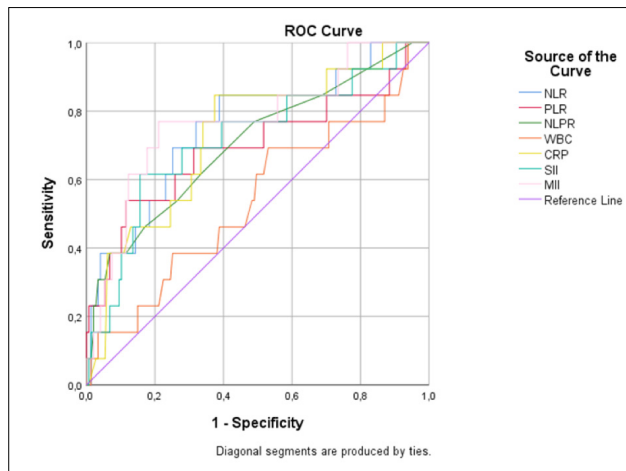


Figure 1. ROC curve analysis of systemic inflammatory indices (NLR, PLR, NLPR, WBC, CRP, SII, and MII) for predicting severe acute calculous cholecystitis. ROC: Receiver operating characteristic; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; NLPR: Neutrophil-to-lymphocyte-platelet ratio; WBC: White blood cell count; CRP: C-reactive protein; SII: Systemic immune-inflammation index; MII: Multiple inflammatory index.

mediated microvascular injury. Pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α) play a central role in this cascade. IL-6 amplifies inflammatory signaling through NF- κ B (nuclear factor kappa-light-chain-enhancer of activated B cells) pathways and induces hepatic synthesis of C-reactive protein, while TNF- α promotes neutrophil recruitment and vascular injury.^[13,14] Elevated levels of IL-6 and TNF- α have been reported in patients with ACC, where they correlate with disease severity and complications.^[15] Moreover, clinical and meta-analytic studies confirm the prognostic value of these cytokines in acute inflammatory diseases.^[16,17] This mechanistic background explains why indices such as NLR, NLPR, and SII increase with greater disease severity.

In the present cohort, cut-off values were determined for NLR (8.45), PLR (219.2), NLPR (0.027), CRP (78.45 mg/L), SII (2414), and MII (250,011). Patients exceeding these thresholds were more likely to develop complications such as gangrenous cholecystitis or perforation. Elevated NLR reflects disproportionate neutrophil activation relative to lymphocyte suppression, while high MII captures the combined influence of SII and CRP, integrating both cellular and humoral aspects of the inflammatory process. This study is consistent with Efgan et al.^[7] (2025), who demonstrated that inflammatory indices, particularly NLR and SII, are reliable predictors of complications in ACC. However, this study is the first to define and propose a specific MII cut-off, thereby extending the clinical applicability of these indices in severity assessment. This finding is also consistent with Afzal et al.^[18] (2025), who reported elevated NLPR values in patients requiring conversion to open cholecystectomy due to severe inflammation. Similarly, Özkan et al.^[19] (2024) demonstrated the diagnostic utility of NLPR in acute appendicitis, with excellent discrimi-

natory performance (AUC=0.94). These findings suggest that such indices may serve as early warning markers of clinical deterioration, potentially preceding radiological evidence of severe disease.

Our results align with previous studies. Efgan et al.^[7] (2025) demonstrated that elevated NLR and SII were predictive of complications and extended hospitalization. Turhan et al.^[20] (2022) similarly reported significantly higher NLR and PLR levels in complicated ACC, supporting the role of systemic inflammation in disease progression. The systemic inflammation response index (SIRI) has also been proposed as a marker of chronic inflammatory processes contributing to gallstone pathogenesis rather than acute exacerbations.^[20] Meng and Liu (2023) observed a significant correlation between higher SII levels and gallstone formation in younger adults, suggesting that inflammatory indices may also play a role in early biliary pathology.^[21,22] Collectively, these findings highlight the broader utility of systemic indices across the spectrum of gallbladder disease.

Molecular and biochemical markers provide additional insight. Shan et al.^[17] (2024) reported that combining IL-6 with procalcitonin (PCT) and neutrophil count achieved excellent discriminatory power (AUC \approx 0.90) for severe ACC, underscoring IL-6 as a central mediator of the inflammatory cascade. Chen et al.^[4] (2024) identified lower cut-off values for NLR, PLR, SII, and NLPR than those observed in the present cohort, yet confirmed the same trend: higher values were consistently associated with disease severity. Variations may reflect differences in patient populations and the timing of laboratory testing. In elderly patients, Xia et al.^[5] (2023) reported NLR thresholds of 5–7 for distinguishing purulent or gangrenous cholecystitis, while Prabhu et al.^[23] (2024) prospectively demonstrated that NLR >10 effectively distinguished severe forms, with high sensitivity and specificity.

Additional evidence also supports the clinical utility of these indices. Prakash and Hasan (2022) demonstrated that NLR was strongly associated with CT findings such as gallbladder distension, increased pericholecystic fat, and pericholecystic fluid collection, highlighting its ability to reflect radiological severity.^[24] Li et al.^[22] (2024) evaluated biomarkers including NLR, PLR, SII, and the Prognostic Nutrition Index (PNI) in acute cholangitis, reporting significantly higher NLR and SII levels in severe cases, which they attributed to systemic inflammation and immune dysregulation. Similarly, Kler et al.^[25] (2022) found that NLR was significantly higher in patients with acute cholecystitis compared with non-acute cholecystitis (non-AC) controls, and notably higher in severe compared to uncomplicated cases, supporting its role as an independent predictor.

Other emerging biomarkers may further enhance prognostic accuracy. Yodying et al.^[26] (2025) demonstrated that the neutrophil percentage-to-albumin ratio (NPAR) >21.5 had excellent predictive performance (AUC=0.906) for conser-

vative treatment failure, outperforming NLR and PLR. Likewise, Güneş et al.^[27] (2024) reported that both the C-reactive protein-to-albumin ratio (CAR >1.86) and systemic immune-inflammation index (SII >1327.69) were independent predictors of severe ACC and perioperative complications. These findings underscore the value of combining inflammatory and nutritional indices to refine risk stratification in ACC.

From a clinical perspective, incorporating these indices into ED workflows may enhance triage and treatment planning. For example, patients presenting with NLR >8.5 or CRP >78 mg/L could be prioritized for early surgical consultation and closer monitoring, even when imaging findings are inconclusive. Integrating systemic indices with TG18 severity grading may provide a more objective framework for risk stratification, improving decision-making in both high-volume tertiary centers and resource-limited hospitals. Such strategies could reduce delays, lower complication rates, and optimize resource utilization.

This research has several limitations. It was retrospective and single-centered, with a relatively small cohort size, which may restrict generalizability. Laboratory parameters were obtained only at admission; serial evaluations could yield additional prognostic insight. Nonetheless, the findings provide preliminary cut-off values for systemic inflammatory indices in ACC and demonstrate their potential association with disease severity.

These results may serve as a reference point for future investigations. In particular, prospective multicenter studies and analyses integrating inflammatory indices with established clinical scores or molecular biomarkers could clarify their role in risk stratification and guide the development of more comprehensive prognostic models.

CONCLUSION

Acute calculous cholecystitis can be assessed rapidly and cost-effectively using systemic inflammatory markers such as NLR, PLR, NLRP, SII, and MII, which are derived from routine complete blood counts. These indices are inexpensive, widely available, and can be integrated into standard diagnostic protocols without additional resource burden, offering a practical alternative in settings where advanced imaging is limited. Early identification of severe cases through these markers may facilitate timely surgical intervention, reduce complication rates, and optimize hospital outcomes. Future multicenter studies are warranted to confirm optimal cut-off values and validate their utility across diverse populations.

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ORİJİNAL ÇALIŞMA - ÖZ

Akut taşlı kolesistitin ciddiyetini tahmin etmede sistemik inflamatuvar indekslerin prognostik rolü

AMAÇ: Akut kolesistit, acil tıp ve cerrahi kliniklerinde en sık karşılaşılan ve tedavi edilen acil durumlardan biridir. Akut taşlı kolesistit (ATK) olgularında inflamasyonun şiddetini ve kapsamını öngörmeye kullanılabilecek sistemik inflamatuvar parametreleri değerlendiren çalışmalar sınırlıdır. Bu çalışmanın amacı, nötrofil/lenfosit oranı (NLR), trombosit/lenfosit oranı (PLR), nötrofil/lenfosit × trombosit oranı (NLPR), sistemik immün-inflamasyon indeksi (SII) ve çoklu inflamasyon indeksi (MII) gibi tam kan sayımı (TKS) kaynaklı parametrelerin ATK'nın ayırıcı tanısı ve şiddetinin belirlenmesindeki tanılabilirliğini araştırmaktır.

GEREÇ VE YÖNTEM: Çalışmaya 1 Ocak 2020-31 Mayıs 2024 tarihleri arasında acil servise başvuran ve genel cerrahi tarafından ATK tanısı doğrulanmış 160 hasta dahil edildi. Akut kalkülöz kolesistit, kolanjit, koledokolitiazis tanısı alan, eksik verisi bulunan ve 18 yaş altı hastalar çalışma dışı bırakıldı. Hastaların demografik, klinik, laboratuvar ve görüntüleme bulguları incelendi; NLR, PLR, NLPR, SII ve MII hesaplandı. Parametrelerin şiddet öngörüsündeki etkinliği ROC analizi ile değerlendirildi.

BULGULAR: En yüksek prediktif değer MII'de saptandı (kesme değeri=250.011; duyarlılık %76.9; özgüllük %78.9; AUC=0.770). NLR için 8.45 kesme değeri %76.9 duyarlılık ve %68,0 özgüllük gösterdi. NLPR (cut-off=0.027) %76.9 duyarlılık ve %54.4 özgüllük; SII (cut-off=2414) ise %69.2 duyarlılık ve %72.1 özgüllük sağladı (tümü p<0.05).

SONUÇ: Sistemik inflamasyon indeksleri, özellikle MII ve NLR, ATK şiddetinin değerlendirilmesinde etkili, ucuz ve erişilebilir biyobelirteçlerdir. Bu parametreler, özellikle görüntüleme yöntemlerinin sınırlı olduğu durumlarda klinik karar sürecine önemli katkı sağlayabilir.

Anahtar sözcükler: Acil tıp; safra taşı; sistemik immün-inflamasyon indeksi; şiddetli akut kolesistit.

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