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Research Article

The Role of Platelet Mass Index in Predicting Short-Term Mortality in Community-Acquired Pneumonia: An Analytical Study

Trombosit Kütle İndeksinin Toplum Kökenli Pnömonide Kısa Dönem Mortaliteyi Öngörmedeki Rolü: Analitik Bir Çalışma

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Abstract

Purpose: Community-acquired pneumonia (CAP) is a major cause of morbidity and mortality worldwide, particularly among elderly patients and those with comorbidities. Early risk stratification is crucial for optimizing treatment decisions. Platelets play a key role in hemostasis and inflammation, and platelet-related indices have been investigated as potential prognostic markers. The Platelet Mass Index (PMI), calculated as platelet count × mean platelet volume (MPV), has been proposed as a marker of inflammatory severity. This study aims to evaluate the prognostic value of PMI in predicting short-term mortality in hospitalized CAP patients.

Material and Method: This retrospective observational study included adult patients diagnosed with CAP and hospitalized between January 1, 2023, and January 1, 2024. Demographic data, clinical parameters, laboratory findings, and severity scores (PSI, CURB-65) were collected. PMI values were compared between survivors and non-survivors. Statistical analyses included Mann-Whitney U tests, chi-square tests, and logistic regression. The predictive performance of the model was assessed using ROC curve analysis.

Results: A total of 174 CAP patients (67% male, median age 77 years) were analyzed. The most common symptom was dyspnea (82%). No significant difference in PMI was observed between survivors and non-survivors (p=0.33). Higher PSI and CURB-65 scores, lower albumin levels, and elevated respiratory rates were associated with increased mortality. The predictive model for mortality demonstrated an AUC of 0.814.

Conclusion: PMI was not a significant predictor of short-term mortality in hospitalized CAP patients. The inflammatory response in severe cases and potential confounders may have influenced the results. Further prospective studies in broader patient populations are needed to clarify PMI's prognostic role.

Keywords: Community-acquired pneumonia, pneumonia, mortality, platelet, mean platelet volume

Öz

Amaç: Toplum kökenli pnömoni (TKP), özellikle yaşlı hastalar ve komorbiditesi olan bireyler arasında dünya çapında önemli bir morbidite ve mortalite nedenidir. Erken risk sınıflandırması, tedavi kararlarının optimize edilmesi açısından kritik öneme sahiptir. Trombositler, hemostaz ve inflamasyonda kilit rol oynar ve trombositle ilgili indeksler potansiyel prognostik belirteçler olarak araştırılmıştır. Trombosit Kütle İndeksi (TKİ), trombosit sayısı × ortalama trombosit hacmi (OTH) olarak hesaplanır ve inflamatuar şiddetin bir belirteci olarak önerilmiştir. Bu çalışma, hastaneye yatırılan TKP hastalarında TKİ'nin kısa dönem mortaliteyi öngörme değerini değerlendirmeyi amaçlamaktadır.

Gereç ve Yöntem: Bu retrospektif gözlemsel çalışma, 1 Ocak 2023 ile 1 Ocak 2024 tarihleri arasında toplum kökenli pnömoni (TKP) tanısı almış ve hastaneye yatırılmış yetişkin hastaları içermektedir. Demografik veriler, klinik parametreler, laboratuvar bulguları ve hastalık şiddeti skorları (PSI, CURB-65) toplanmıştır. Trombosit Kütle İndeksi (TKİ) değerleri, hayatta kalanlar ve hayatını kaybedenler arasında karşılaştırılmıştır. İstatistiksel analizler arasında Mann-Whitney U testi, ki-kare testi ve lojistik regresyon yer almaktadır. Modelin öngörü performansı ROC eğrisi analizi kullanılarak değerlendirilmiştir.

Bulgular: Toplamda 174 toplum kökenli pnömoni (TKP) hastası analiz edilmiştir (%67 erkek, medyan yaş 77 yıl). En yaygın semptom dispne olup %82 oranında gözlenmiştir. Hayatta kalanlar ile hayatını kaybedenler arasında trombosit kütle indeksi (TKİ) açısından anlamlı bir fark bulunmamıştır (p=0,33). Daha yüksek PSI ve CURB-65 skorları, düşük albümin seviyeleri ve artmış solunum hızları, artan mortalite ile ilişkilendirilmiştir. Mortalite tahmin modeli, 0,814 AUC değerine sahip olarak değerlendirilmiştir.

Sonuç: Trombosit Kütle İndeksi (TKİ), hastaneye yatırılan toplum kökenli pnömoni (TKP) hastalarında kısa dönem mortalitenin anlamlı bir prediktörü olarak bulunmamıştır. Şiddetli vakalarda inflamatuar yanıt ve olası kafa karıştırıcı değişkenler sonuçları etkileyebilir. TKİ'nin prognostik rolünü netleştirmek için daha geniş hasta popülasyonlarında ileriye dönük çalışmalar gereklidir.

Anahtar Kelimeler: Bruselloz, Coombs aglütinasyon testi, Seroprevalans

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INTRODUCTION

Pneumonia remains a significant public health concern worldwide, leading to high morbidity and mortality rates. According to the World Health Organization, pneumonia is one of the most common causes of death, particularly among elderly individuals, immunocompromised patients, and those with underlying chronic diseases. Even in developed countries, pneumonia is a major cause of hospital admissions and intensive care unit (ICU) requirements (1-4). In addition to its detrimental effects on individual health, pneumonia also imposes a substantial economic burden due to workforce loss, prolonged hospital stays, and high treatment costs.

A study conducted in the United States reported that the annual cost of community-acquired pneumonia (CAP) exceeds $10 billion in direct healthcare expenditures alone. Furthermore, indirect costs, such as productivity loss and work absenteeism, further amplify this economic burden (5,6). These findings underscore the critical importance of early diagnosis and appropriate treatment of pneumonia at both individual and societal levels.

Platelets play a fundamental role in hemostasis and thrombosis; however, they are also involved in key pathophysiological processes such as inflammation, immune response, and endothelial dysfunction (7,8). Platelets play a crucial role in the pathophysiology of pneumonia beyond their traditional function in hemostasis. They actively contribute to the immune response by recognizing and responding to pathogens through toll-like receptors and inflammatory mediators. During pneumonia, platelets interact with immune cells, endothelial cells, and pathogens, leading to thromboinflammation, which can exacerbate lung injury. Increased platelet activation and aggregation have been associated with disease severity, contributing to microvascular thrombosis and impaired oxygenation. Conversely, platelet-derived factors such as platelet-derived growth factor (PDGF) and transforming growth factor-beta (TGF-β) may support tissue repair. Understanding platelet involvement in pneumonia pathogenesis could lead to novel therapeutic targets for modulating the immune-thrombotic response. Recent studies suggest that platelet count, and platelet-related parameters not only help assess disease severity but may also provide valuable prognostic insights into short-term mortality (9-12). In this context, the Platelet Mass Index (PMI), calculated as the product of platelet count and mean platelet volume (MPV), has emerged as a novel hematological parameter of interest.

PMI reflects both platelet activity and total platelet mass, potentially serving as an indicator of inflammatory severity and microvascular complications (13-17). Although growing evidence supports the prognostic value of PMI in various cardiovascular and inflammatory diseases, its role in predicting short-term mortality in CAP within the emergency department setting remains insufficiently explored.

This study aims to evaluate the role of PMI in predicting short-term mortality among adult patients presenting to the emergency department with CAP. We hypothesize that PMI, as a simple and accessible biomarker, may contribute to the early identification of high-risk patients, ultimately improving clinical decision-making and patient management.

MATERIALS AND METHODS

Study Design and Data Collection

This study was conducted as a retrospective observational analysis using data from patients diagnosed with CAP at an education hospital between January 1, 2023 - January 1, 2024. Patient records were reviewed through the hospital's electronic medical database. The STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) Statement checklist was adhered to in reporting the results and writing the article (18).

Demographic characteristics, clinical symptoms (e.g., cough, dyspnea, fever), vital signs (e.g., Glasgow Coma Scale, respiratory rate, blood pressure, heart rate, peripheral oxygen saturation), laboratory parameters (e.g., albumin, pH, blood urea nitrogen, creatinine, inflammatory markers), and severity scores (e.g., Pneumonia Severity Index, CURB-65) were recorded.

The Inclusion criteria were hospitalized adult patients (≥18 years old) with a confirmed diagnosis of CAP and available laboratory and clinical data. Patients with missing key data, prior advanced directives limiting treatment, or transferred from another facility without complete records were excluded from the analysis.

Statistical Analysis

The data set was created in the excel program. Statistical analysis was performed using Jamovi (version 2.3). Continuous variables were expressed as median (interquartile range, IQR) due to their non-normal distribution, which was assessed using the Kolmogorov-Smirnov test. Categorical variables were presented as frequencies and percentages.

Comparisons between groups were conducted using the Mann-Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables, as appropriate. The relationship between independent variables and mortality was evaluated using univariate and multivariate logistic regression analyses. To avoid multicollinearity, arterial blood pressure values and renal function parameters were excluded from the multivariate model. The results of the logistic regression analysis were presented as odds ratios with 95% confidence intervals.

The predictive performance of the PMI and the developed model for predicting septic shock was assessed using receiver operating characteristic (ROC) curve analysis. The area under the curve was calculated to determine discriminative ability. The optimal cut-off value was determined using the Youden index. Sensitivity, specificity, positive predictive value, negative predictive value, positive likelihood ratio, and negative likelihood ratio were reported with their respective 95% confidence intervals.

Model fit was evaluated using deviance, Akaike Information Criterion (AIC), and McFadden's R^2. A p-value of <0.05 was considered statistically significant.

Ethical Considerations

This study was approved by the Clinical Research Ethics Committee of the relevant hospital. (ethics committee decision number/date: 554/02.27.2025) and was conducted in accordance with the ethical principles of the Declaration of Helsinki. Since patient data were retrospectively analyzed, the requirement for individual informed consent was waived by the ethics committee. Patient identities were kept confidential, and all data were anonymized and used solely for scientific purposes. Data access was restricted to the researchers, and the highest level of personal privacy and data security was maintained. All procedures were carried out in compliance with the Personal Data Protection Law and Good Clinical Practice guidelines to ensure the protection of patient rights

RESULTS

A total of 174 patients were included in the study, of whom 117 (67%) were male and 57 (33%) were female. The median age of the study population was 77 years (IQR: 67–82). The most common symptom was dyspnea, observed in 143 patients (82%), followed by cough in 69 patients (40%) and fever in 33 patients (19%). Regarding comorbidities, hypertension was the most prevalent (58%), followed by diabetes mellitus (33%), coronary artery disease (29%), chronic obstructive pulmonary disease (26%), and active malignancy (30%). Other baseline characteristics of the study population are presented in Table 1.

Table 1. Descriptive characteristics of patients included in the study and comparison of mortality and survivorship groups.

Characteristic	N = 174	Survivor group N = 95 (55%)	Mortality group N = 79 (45%)	p-value
Age	77 (67-82)	77 (70-82)	76 (66-81)	0.20
Gender				0.97
Male	117 (67%)	64 (67%)	53 (67%)	
Female	57 (33%)	31 (33%)	26 (33%)	
Cough	69 (40%)	22 (23%)	47 (59%)	<0.001
Dyspnea	143 (82%)	86 (91%)	57 (72%)	0.002
Fever	33 (19%)	10 (11%)	23 (29%)	0.002
Hypertension	101 (58%)	57 (60%)	44 (56%)	0.57
Diabetes Mellitus	57 (33%)	35 (37%)	22 (28%)	0.21
Chronic Obstructive Pulmonary Disease	45 (26%)	24 (25%)	21 (27%)	0.84
Coronary Artery Disease	51 (29%)	29 (31%)	22 (28%)	0.70
History of Stroke	37 (21%)	20 (21%)	17 (22%)	0.94
Chronic Kidney Disease	17 (9.8%)	11 (12%)	6 (7.6%)	0.38
Congestive Heart Failure	28 (16%)	16 (17%)	12 (15%)	0.77
Active Malignancy	52 (30%)	34 (36%)	18 (23%)	0.062
Alzheimer's Disease	27 (16%)	14 (15%)	13 (16%)	0.76
Glasgow Coma Scale	15 (13-15)	15 (13-15)	15 (15-15)	0.011
Systolic Blood Pressure	121 (105-144)	120 (98-142)	124 (112-144)	0.046
Diastolic Blood Pressure	70 (60-80)	70 (52-80)	70 (62-80)	0.40
Heart Rate	98 (85-120)	105 (86-120)	96 (85-111)	0.041
Peripheral Oxygen Saturation	85 (80-89)	80 (79-86)	88 (85-91)	<0.001
Respiratory Rate	24 (20-30)	28 (22-30)	22 (20-24)	<0.001
Serum Albumin Level	32 (28-36)	30 (26-35)	34 (30-38)	<0.001
Aspartate Aminotransferase Level	21 (15-32)	23 (16-32)	20 (15-32)	0.48
Alanine Aminotransferase Level	17 (14-26)	17 (14-24)	18 (12-30)	0.83
C-Reactive Protein Level	118 (58-221)	131 (56-243)	117 (59-203)	0.35
Blood Glucose Level	144 (112-195)	150 (120-222)	140 (102-179)	0.073
Blood Urea Nitrogen Level	48 (36-75)	53 (41-100)	40 (30-64)	<0.001
Serum Creatinine Level	0.90 (0.65-1.35)	1.00 (0.64-1.56)	0.90 (0.67-1.17)	0.27
Serum Sodium Level	136 (133-140)	136 (133-140)	136 (134-139)	0.71
Serum Potassium Level	4.40 (3.99-4.80)	4.40 (3.99-4.90)	4.40 (4.00-4.70)	0.99
White Blood Cell Count	12 (8-18)	12 (8-20)	12 (10-16)	0.73
Neutrophil Count	10 (6-15)	9 (6-16)	10 (8-14)	0.78
Lymphocyte Count	1.02 (0.59-1.59)	0.98 (0.51-1.65)	1.06 (0.72-1.48)	0.49
Hemoglobin Level	11.40 (9.83-12.90)	11.00 (9.70-12.70)	11.60 (10.45-13.75)	0.072
Hematocrit Level	35 (31-40)	35 (30-40)	35 (32-40)	0.45
Platelet Count	264 (192-338)	231 (187-336)	275 (196-342)	0.37
Mean Platelet Volume	9.70 (8.70-10.47)	9.70 (8.70-10.40)	9.70 (8.70-10.55)	0.87
Blood Acidity Level (pH)	7.40 (7.34-7.44)	7.39 (7.29-7.44)	7.42 (7.39-7.45)	<0.001
Partial Pressure of Oxygen	52 (45-56)	48 (42-54)	55 (50-60)	<0.001
Pneumonia Severity Index Score	150 (122-175)	165 (144-188)	123 (102-152)	<0.001
CURB-65 Score				<0.001
0	6 (3.4%)	2 (2.1%)	4 (5.1%)	
1	16 (9.2%)	4 (4.2%)	12 (15%)	
2	81 (47%)	35 (37%)	46 (58%)	
3	44 (25%)	32 (34%)	12 (15%)	
4	22 (13%)	17 (18%)	5 (6.3%)	
5	5 (2.9%)	5 (5.3%)	0 (0%)	
Platelet Mass Index	2,401(1,852-3,330)	2,253 (1,876-3,247)	2,767 (1,788-3,322)	0.33

Univariate analysis revealed significant differences between the two groups. Patients in the mortality group had a higher prevalence of cough (59% vs. 23%, p<0.001), whereas dyspnea (91% vs. 72%, p=0.002) and fever (11% vs. 29%, p=0.002) were more common in the survivor group. Glasgow Coma Scale scores were significantly higher in the positive outcome group [15 (15–15) vs. 15 (13–15), p=0.011]. Among vital signs, systolic blood pressure [124 (112–144) vs. 120 (98–142), p=0.046] and peripheral oxygen saturation [88% (85–91) vs. 80% (79–86), p<0.001] were significantly higher in the positive outcome group, whereas heart rate [96 (85–111) vs. 105 (86–120), p=0.041] and respiratory rate [22 (20–24) vs. 28 (22–30), p<0.001] were lower. There was no significant difference in the PMI between the groups (p = 0.33). The median PMI value was 2,401 (IQR: 1,852–3,330) in the overall study population,

2,253 (IQR: 1,876–3,247) in survivor group, and 2,767 (IQR: 1,788–3,322) in mortality group.

Among laboratory parameters, serum albumin levels were significantly higher in the positive outcome group [34 (30–38) vs. 30 (26–35), p<0.001], whereas blood urea nitrogen levels were lower [40 (30–64) vs. 53 (41–100), p<0.001]. Additionally, blood acidity level (pH) [7.42 (7.39–7.45) vs. 7.39 (7.29–7.44), p<0.001] and partial pressure of oxygen [55 (50–60) vs. 48 (42–54), p<0.001] were significantly higher in the positive outcome group.

The Pneumonia Severity Index (PSI) score was significantly lower in the positive outcome group [123 (102–152) vs. 165 (144–188), p<0.001]. Similarly, the CURB-65 score distribution showed significant differences between the groups (p<0.001), with a higher proportion of patients in the negative outcome group having scores of 3 or higher. The detailed comparison of all parameters between the groups is presented in Table 1.

A multivariate logistic regression analysis was performed to identify independent predictors (Table 2). To avoid multiple collinearity, scores were not included in the model and only respiratory rate was included as a proxy for respiratory parameters.

Table 2. Multivariate Logistic Regression Analysis of Independent Predictors

Predictor	Estimate	SE	Z	p	Odds ratio	Lower 95% Confidence Interval	Upper 95% Confidence Interval
Intercept	171.250	158.338	1.08	0.279	2.74e+7	9.11e-7	8.22e+20
Cough	12.049	0.3873	3.11	0.002	33.364	1.562	7.127
Dyspnea	-0.7373	0.5757	-1.28	0.200	0.4784	0.155	1.479
Fever	0.9820	0.5241	1.87	0.061	26.698	0.956	7.457
Glasgow Coma Scale	0.1439	0.0962	1.50	0.135	11.547	0.956	1.394
Respiratory Rate	0.1124	0.0442	2.54	0.011	11.189	1.026	1.220
Serum Albumin Level	-0.0868	0.0313	-2.78	0.005	0.9168	0.862	0.975
Blood Acidity Level (pH)	-29.932	20.886	-1.43	0.152	0.0501	8.36e-4	3.006
Age	0.0245	0.0175	1.40	0.161	10.248	0.990	1.060

The predictive performance of the developed model for mortality prediction was evaluated. The model demonstrated an accuracy of 69.5%, with a specificity of 64.6% and a sensitivity of 73.7%. The area under the receiver operating characteristic curve was calculated as 0.814, indicating good discriminatory power (Figure 1). The cut-off value was set at 0.5 for classification. These results suggest that the model provides a reliable balance between sensitivity and specificity in predicting mortality.

The model fit analysis indicated that the developed model had a deviance value of 179, an AIC of 197, and a McFadden's R^2 value of 0.254. These results suggest that the model demonstrates a statistically significant fit, with moderate explanatory power.

DISCUSSION

This study evaluated the role of PMI in predicting short-term mortality among adult patients presenting to the emergency department with CAP. The findings revealed no significant difference in PMI between the survivor and mortality groups. To the best of our knowledge, this is the first study to assess the role of PMI in predicting short-term mortality in adult patients with CAP.

Pneumonia remains a leading cause of morbidity and mortality worldwide, particularly among elderly patients and those with comorbid conditions. It is characterized by an acute infection of the lung parenchyma, leading to inflammation, alveolar consolidation, and impaired gas exchange (19,20). The etiology of pneumonia is diverse, including bacterial, viral, fungal, and atypical pathogens. Streptococcus pneumoniae remains the most common bacterial cause, while viral pneumonias, such as those due to influenza and SARS-CoV-2, have gained increasing recognition, especially in the context of recent pandemics (21-25). The differential diagnosis of pneumonia is broad and includes conditions such as acute bronchitis, congestive heart failure with pulmonary edema, pulmonary embolism, and interstitial lung diseases. Clinical presentation, radiological findings, and laboratory parameters help distinguish pneumonia from these mimicking conditions (26,27). However, overlapping symptoms such as dyspnea, cough, and fever can complicate diagnosis, necessitating the use of biomarkers, imaging modalities, and microbiological studies for confirmation. Given the significant disease burden, accurate risk stratification remains essential for optimizing treatment decisions, including hospitalization criteria and antimicrobial therapy selection (28,29). Various scoring systems, such as the CURB-65 and Pneumonia Severity Index (PSI), assist in this process, yet additional prognostic markers are still being investigated to improve patient outcomes.

Platelets are increasingly recognized as key mediators of inflammation, bridging hemostasis with immune system activation in various disease states, including infectious and non-infectious inflammatory conditions (30-32). Beyond their role in coagulation, platelets actively participate in the immune response by releasing pro-inflammatory cytokines, interacting with leukocytes, and modulating endothelial function. In infections such as pneumonia, platelet activation enhances neutrophil extracellular trap formation, contributing to bacterial clearance. However, excessive platelet activation can lead to endothelial injury, microvascular thrombosis, and organ dysfunction,

exacerbating disease severity. Similar mechanisms are observed in sterile inflammatory conditions such as acute pancreatitis, where platelet-derived mediators amplify the systemic inflammatory response, promoting microcirculatory disturbances and worsening disease outcomes (33,34). Platelet indices, particularly MPV and platelet-to-lymphocyte ratio, have been associated with disease severity in both infectious and inflammatory conditions. In pneumonia, elevated MPV has been linked to increased mortality risk, likely reflecting heightened platelet activation and consumption in severe inflammation. These findings suggest that platelet parameters could serve as valuable biomarkers in predicting outcomes in inflammatory diseases, yet their precise role requires further investigation (30-34). Understanding platelet involvement in inflammation may offer new insights into disease pathophysiology and potential therapeutic targets.

Platelets play a crucial role not only in hemostasis and thrombosis but also in immune response and inflammation, making them significant contributors to the pathophysiology of CAP. Several studies have highlighted the prognostic value of platelet-related parameters in predicting mortality and disease severity in CAP patients. Cho et al. investigated the relationship between the MPV to platelet ratio (MPR) and short-term mortality in CAP patients. Their findings demonstrated that higher MPR levels were significantly associated with increased mortality, suggesting that platelet indices could serve as prognostic markers in CAP (35). Similarly, Golcuk et al. examined the combination of MPV, and the CURB-65 score to predict 28-day mortality. Their study revealed that incorporating MPV into the CURB-65 score improved its predictive accuracy, underscoring the potential of platelet indices in risk stratification (36). Moulis et al. explored the impact of platelet counts within the normal range on CAP prognosis. Their analysis of 12,905 patients found that platelet counts at both the lower and upper ends of the normal spectrum were associated with altered mortality risks. These findings suggest that even within normal limits, platelet levels may reflect underlying disease severity and systemic inflammatory responses in CAP patients (37). ElMaraghy et al. further emphasized the role of platelets in CAP severity and outcomes. Their study demonstrated a significant relationship between platelet abnormalities—thrombocytopenia and thrombocytosis—and the CURB-65 score, respiratory complications, and overall mortality. This suggests that platelet count deviations may serve as valuable markers of disease progression (38). Finally, Huang et al. assessed the diagnostic value of various blood parameters, including platelet-to-lymphocyte ratio and neutrophil-to-lymphocyte ratio, in CAP patients. Their findings indicated that elevated platelet-to-lymphocyte ratio and neutrophil-to-lymphocyte ratio levels correlated with higher disease severity and prolonged hospitalization (39). This supports the growing evidence that platelet indices and related hematological markers can aid in the early identification of high-risk CAP patients. While platelet count abnormalities have been shown to be associated with CAP severity and mortality, they do not fully capture the functional and dynamic aspects of platelet involvement in systemic inflammation.

Collectively, these studies highlight the clinical significance of platelet-related parameters in CAP prognosis. The findings suggest that platelet indices, including MPV, MPR, and platelet-to-lymphocyte ratio, could serve as valuable biomarkers for mortality prediction and disease severity assessment. Future research should focus on integrating these parameters into clinical decision-making models to enhance risk stratification and optimize patient management strategies.

In our study, no significant relationship was found between PMI and CAP prognosis. There may be several possible reasons for this result. First of all, our study included only hospitalized CAP patients, and since this group of patients usually has a more severe clinical course, their inflammatory responses may be more homogeneous. This may have made possible variations that may clarify the prognostic value of PMI. In addition, factors such as the development of sepsis, coagulopathy, and bone marrow suppression in severe CAP cases may cause changes in the relationship between platelet count and MPV. It is thought that PMI may cease to be a significant prognostic indicator, especially in critically ill patients, due to platelet consumption or dysfunction. In addition, accompanying comorbidities (e.g. chronic kidney disease, liver cirrhosis, hematological diseases) may have created additional variability in PMI values by affecting platelet production and function. Our results suggest that the use of PMI as a prognostic marker in CAP may vary depending on the patient population and the severity of the disease. Further studies in larger patient groups and different CAP subtypes may contribute to a better understanding of this relationship.

Our study has some limitations. First, there is a risk of information bias due to its retrospective design. Since the data were obtained from hospital records, incomplete or incorrect records may have made it difficult to correctly evaluate some variables. In particular, variability between laboratory results and clinical evaluations may affect data integrity. Secondly, selection bias is an important limitation of our study. Including only hospitalized CAP patients may have excluded milder cases and limited the generalizability of our results to the general CAP population. This may lead us to ignore the possibility that the prognostic value of PMI may be different, especially in mild and outpatient patients. Thirdly, confounding bias should be considered. Although multivariate analysis was performed for some variables such

as age, comorbidities, and severity of infection in our study, it was not possible to eliminate the effect of other potential confounding factors (e.g., antibiotic use, concomitant inflammatory diseases, or immune status). With prospective and larger-scale studies, the impact of these factors can be better evaluated, and more definitive conclusions can be reached regarding the true prognostic value of PMI.

In conclusion, no statistically significant relationship was found between PMI and short-term mortality in hospitalized CAP patients in our study. This situation can be explained by the fact that our study included only hospitalized severely ill patients, the inflammatory response varied in different stages of the disease, and the inability to fully control possible confounding factors. However, there are studies in the literature showing that platelet parameters may have prognostic value in CAP patients. Prospective and large-scale studies covering different patient groups are necessary to better understand the prognostic value of PMI.

Declarations

Ethics Committee Approval: Ethics committee approval was obtained from Clinical Research Ethics Committee of the relevant hospital. (ethics committee decision number/date: 554/02.27.2025). This study was conducted according to the principles of the Declaration of Helsinki.

Authorship Contributions: Concept: SO, IA, AO. Design: SO, IA, AO. Data Collection or Processing: SO, IA, AO. Analysis or Interpretation: SO, IA, AO. Literature Search: SO, IA, AO. Writing: SO, IA, AO. All authors approved the final version of the manuscript.

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