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Evaluation of plateletcrit level from platelet indices as a prognostic marker in COVID-19 patients

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Abstract. – OBJECTIVE: COVID-19 disease bears similarities to a wide range of diseases, from simple flu infections to severe acute respiratory distress syndrome and is caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). In this study, we aimed to elucidate the plateletcrit levels in patients with and without mortality who had been admitted to the intensive care unit because of pneumonia associated with SARS-CoV-2.

PATIENTS AND METHODS: In total, 434 patients were evaluated in this retrospective analysis. Their demographic data, comorbid diseases, Acute Physiology and Chronic Health Evaluation (APACHE) II and Sequential Organ Failure Assessment (SOFA) scores, platelet, lymphocyte, white blood cell (WBC) and neutrophil counts; mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), hemoglobin and C-reactive protein (CRP) levels and neutrophil-lymphocyte ratios (NLRs) were obtained from the hospital's electronic database on the days of the patients' intensive care unit admissions. Afterwards, their PLR, PNR, and MPV/PLT ratios were calculated.

RESULTS: APACHE II score, length of hospital stay, WBC count, PCT, PLR, NLR, and CRP levels affected mortality. Increases in hospital stay duration, APACHE II score, platelet-lymphocyte ratio (PLR), and CRP, as well as decreases in PCT percentage, were associated with mortality. ROC curve analysis was performed to determine the success of PCT, PLR, and NLR in predicting mortality in COVID-19 patients and to determine cut-off values for mortality. It was determined that PCT, PLR, and NLR could correctly classify patients at rates of 58.9%, 59.2%, and 66.8% (moderate), respectively. The risk of mortality was higher in patients with PCT values of 0.188 or less, PLR values greater than 293.46, and NLR values greater than 9.49.

CONCLUSIONS: In the COVID-19 patients evaluated in this study, plateletcrit indices could be utilized to predict mortality.

Key Words:
Platelet indices, Plateletcrit, COVID-19.

Abbreviations

%: percentages, APACHE-II: Acute Physiology and Chronic Health Evaluation II, ARDS: Acute Respiratory Distress Syndrome, AUC: area under the curve, CAD: Coronary Artery Disease, CHF: Congestive Heart Failure, CI: confidence interval, COPD: Chronic Obstructive Pulmonary Disease, CRP: C-reactive protein, DM: Diabetes Mellitus, HT: Hypertension, ICU: Intensive Care Unit, MPV: mean platelet volume, MPV/PLT: mean platelet volume-to-platelet count, NLR: neutrophil-to-lymphocyte ratio, OR: odds ratio, PCT: Plateletcrit, PDW: Platelet distribution width, PLR: Platelet-to-lymphocyte ratio, PLT: Platelet, PNR: Platelet-to-neutrophil ratio, ROC: receiver operating characteristics, SD: standard deviation, SOFA: Sequential Organ Failure Assessment, SPSS: Statistics Package for the Social Sciences, WBC: white blood cells.

Introduction

First detected in Wuhan, China, in December 2019, the 2019 coronavirus disease (COVID-19) bears similarities to a wide spectrum of diseases, from simple flu infections to severe acute respiratory distress syndrome (ARDS). It is caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2)[1]. Platelet indices (PI) such as mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) are among the platelet parameters measured in routine complete blood counts. Some studies[2,3] suggest that these parameters may be important in determining the prognoses of some non-hematological diseases.

The probability of developing thrombocytopenia in COVID-19 infection is 13%[4]. In a meta-analysis[5] of nine studies on patients with COVID-19

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infections, decreased platelet count (PLT) was observed in 22.4% of severe patients. The upper PLT limits for thrombocytopenia were 150,000 μL in 7 of the studies and 100,000 μL in 2 of the studies. The PLT decreased by between 27,000-31,000 μL in mild cases and between 29,000-35,000 μL in severe cases. The mechanisms underlying thrombocytopenia are multifactorial; viral infection and mechanical ventilation cause endothelial damage, and platelet activation is associated with platelets' role in the formation of thrombosis in the lungs. Additionally, decreased secretion of platelets from megakaryocytes in the lungs, viral infection of the bone marrow and intravascular coagulation also trigger thrombocytopenia. In the literature[5], there is a close relationship between low PLT and mortality in the COVID-19 patient group.

This study's primary aim was to evaluate the plateletcrit level, which is a platelet index, in patients who were admitted to an intensive care unit because of pneumonia associated with SARS-CoV-2.

Patients and Methods

In total, 434 patients were evaluated in this retrospective analysis. The study was initiated after the approval of the ethics committee of the Ankara Atatürk Chest Diseases and Thoracic Surgery training and research hospital of the University of Health Sciences (protocol number 719; dated 25/03/2021). The patients' laboratory results obtained upon their admission to the intensive care unit (ICU) between March 2020 and March 2021 for COVID-19 infection were evaluated within the scope of this study. All procedures followed were in accordance with the ethical standards (institutional and national) of the committee responsible for human experiments and the 1975 Declaration of Helsinki, revised in 2008.

The patients' demographic data, comorbid diseases, Acute Physiology and Chronic Health Evaluation (APACHE) II and Sequential Organ Failure Assessment (SOFA) scores, platelet, lymphocyte, white blood cell and neutrophil counts, and MPV, PDW, PCT, NLR, hemoglobin, CRP and procalcitonin values were obtained from the hospital's electronic database on the days of their intensive care unit admissions. Afterward, their PLR, PNR, and MPV/PLT ratios were calculated. The patients' mortality, hospital stay lengths, and ICU stay lengths were evaluated, and they were divided into two groups (those with mortality and those without mortality).

Inclusion Criteria

Patients aged 18 years and over who were hospitalized in the ICU because of COVID-19 infection were included in this study.

Exclusion Criteria

Patients under 18 years of age, those hospitalized in the ICU for infections other than COVID-19, those with immune and hematologic diseases that could affect platelet indices, and those on medications that could alter platelet indices were excluded from this analysis. Pregnant women and subjects who stayed in the ICU for less than 24 hours were also excluded.

Statistical Analysis

SPSS 26.0 (Statistics Package for the Social Sciences, IBM Corp., Armonk, NY, USA) was utilized for this study. Continuous variables were expressed as means ± standard deviations (SD), and categorical variables were expressed as percentages (%). Pearson's Chi-squared test and Fisher's exact test were used to compare categorical variables. The distribution of data was tested using the Shapiro-Wilk test. A Student's *t*-test was used for pairwise comparisons of normally distributed groups. The Mann-Whitney U test was used for pairwise comparisons of non-normally distributed groups. A p-value less than 0.05 was considered significant.

Multiple logistic regression analysis was used with all predetermined factors with p-values lower than 0.25 in univariate analysis. In multivariate analysis, we considered p-values lower than 0.05 to be statistically significant. Receiver operating characteristic (ROC) analysis was performed to estimate mortality. The area under the curve (AUC), cut-off points, and sensitivity and selectivity values of these cut-off points were calculated. A p-value <0.05 was considered statistically significant in all analyses.

Results

Of the 434 patients included in the study, 162 (37.3%) were female, 272 (62.6%) were male, and their mean age was 67.08 ± 12.82 years. Mortality occurred in 174 (40.1%) of the patients. The patients' demographic and clinical characteristics are detailed in Table I, and their laboratory parameters are detailed in Table II.

APACHE II score, hospital stay duration, WBC, plateletcrit percentage, and PLR, NLR, and

Table I. Patients' demographic and clinical characteristics.

Variable	All n = 434	Mortality		*p*-value
		No = 260	Yes = 174	
Age (year)	68 [20-94]	65.5 [20-94]	72 [40-94]	**< .001**
Gender (male/female)	272/162	163/97	109/65	.992
SOFA	5 [2-18]	4 [2-12]	8 [3-18]	**< .001**
APACHE II	20 [10-38]	17 [10-31]	25 [16-38]	**< .001**
Comorbidity	294 (67.7%)	177 (68.1%)	117 (67.2%)	.855
CAD (n, %)	99 (22.8%)	55 (21.2%)	44 (25.3%)	.315
HT (n, %)	203 (46.8%)	125 (48.1%)	78 (44.8%)	.507
DM (n, %)	113 (26.0%)	70 (26.9%)	43 (24.7%)	.607
COPD (n, %)	69 (15.9%)	34 (13.1%)	35 (20.1%)	.051
Asthma (n, %)	8 (1.82%)	6 (2.3%)	2 (0.5%)	.380
CHF (n, %)	32 (7.4%)	14 (45.4%)	18 (10.3%)	.054
Malignancy (n, %)	26 (6.0%)	16 (6.2%)	10 (5.7%)	.861
Neurological disease (n,%)	26 (6.0%)	12 (4.6%)	14 (8.1%)	.140
Hospital stay (days)	13 [1-59]	15 [4-59]	10 [1-37]	**< .001**
ICU stay (days)	5 [1-36]	5 [3-36]	6 [1-34]	.250

APACHE II: Acute Physiology and Chronic Health Evaluation II, SOFA: Sequential Organ Failure Assessment, COPD: chronic obstructive pulmonary disease, DM: diabetes mellitus, CAD: coronary artery disease, CHF: congestive heart failure, HT: hypertension, ICU: intensive care unit. *Significant difference at $p < 0.05$. *p*-values were calculated using a Student's t-test, Pearson's Chi-squared test, Fisher's exact test, or the Mann-Whiney U test. The data are presented as proportions of cases (%) for qualitative variables and as means (± SD) or medians (min-max) for quantitative variables.

CRP levels affected mortality. Increases in hospital stay duration, APACHE II score, PLR, and CRP were associated with mortality, as were decreases in PCT percentage. The parameters' logistic regression analysis results are shown in Table III.

ROC curve analysis was performed to determine the success of PCT, PLR, and NLR in predicting mortality in the patients and to achieve cut-off values for mortality. We determined that PCT, PLR, and NLR could correctly classify patients at rates of 58.9%, 59.2%, and 66.8% (moderate), respectively (Table IV).

To answer the question of which value should be taken as the cut-off value for this test, the sensitivity and specificity values given as a result of the analysis were examined, and the optimum point was selected. ROC curve analysis of PCT, PLR, and NLR parameters for mortality is shown in Figure 1. The sensitivity, specificity, and cut-off values for PCT were 76.92%, 48.28%, and 0.188, respectively. Therefore, the risk of mortality was higher in cases with PCT values of 0.188 or lower. For PLR, the sensitivity, specificity, and cut-off values were 58.62%, 56.15%, and 293.46, respectively; the mortality risk was higher in cases with PLR values above 293.46. For NLR, the sensitivity, specificity, and cut-off values were 65.9%, 57.0% and 9.49, respectively; the risk of mortality was higher in cases with NLR values above 9.49.

Discussion

PCT, MPV, and PDW are platelet indices that are automatically detected in a hemogram profile. They provide valuable information about platelets' morphology and proliferation kinetics. PCT is the arithmetic product of PLT and platelet volume and is positively correlated with platelet count[6,7]. Studies[8,9] have shown that severe infection, trauma, malignancy, and thrombotic diseases can cause changes in platelet indices.

Thrombocytopenia is common in critically ill patients and indicates disseminated intravascular coagulopathy, severe organ dysfunction, or physiological decompensation in COVID-19 patients[10]. This condition's mechanism is multifactorial. Researchers have suggested that the combination of viral infection and mechanical ventilation in SARS triggers platelet activation, aggregation, and thrombosis in the lungs, causing endothelial damage, which in turn increases platelet consumption. Additionally, because the lungs may be areas of platelet

Table II. Laboratory parameters.

Variable	All n = 434	Mortality		*p*-value
		No = 260	Yes = 174	
WBC (× 10^9/L)	9.39 [23.0-35.93]	8.17 [2.38-31.83]	10.45 [2.30-35.93]	**< .001**
Neutrophils (× 10^9/L)	5.16 [1.31-34.87]	7.23 [1.31-29.61]	10.37 [1.34-34.87]	**< .001**
Lymphocyte (× 10^9/L)	0.81 [0.05-5.90]	0.88 [0.09-5.90]	0.67 [0.05-5.07]	**< .001**
Hemoglobin (gr/dL)	13.40 ± 1.75	13.47 ± 0.61	13.29 ± 1.94	.306
MPV (f/l)	9.5 [6.25-13.6]	9.5 [6.25-13.6]	9.9 [7.4-13.4]	.213
PDW (%)	16.3 [14.0-18.3]	16.3 [15.3-18.3]	16.3 [14.0-17.5]	.805
PLT (× 10^9/L)	237.0 [23-797]	239 [99-797]	215.5 [23-593]	.082
PCT (%)	0.221 [0.023-0.738]	0.23 [0.101-0.738]	0.231 [0.023-0.533]	**.001**
PLR	271.68 [4.37-1933.33]	266.40 [41.19-1933.33]	347.44 [4.37-1600.00]	**.001**
NLR	10.20 [0.63-118.67]	7.54 [0.63-118.67]	13.98 [1.81-90.77]	**< .001**
PNR	32.26 [1.18-161.82]	35.23 [5.64-161.82]	26.17 [1.18-116.12]	**< .001**
MPV/PLT	0.40 [0.012-0.439]	0.39 [0.012-0.111]	0.42 [0.015-0.439]	.132
CRP (g/L)	117.0 [0.37-562.90]	101.90 [0.37-363.98]	135.11 [3.48-592.90]	**< .001**
Procalcitonin (µg/L)	0.15 [0.01-100]	0.09 [0.01-75]	0.21 [0.01-100]	**< .001**

n: Number, WBC: white blood cell, CRP: C-reactive protein, MPV: mean platelet volume, PDW: platelet distribution width, PCT: plateletcrit, PLR: platelet-lymphocyte ratio, PLT: platelet count, MPV/PLT: mean platelet volume-platelet count ratio, NLR: neutrophil-lymphocyte ratio, PNR: platelet-neutrophil ratio. *Significant difference at $p < 0.05$. *p*-values were calculated using a Student's *t*-test, Pearson's Chi-squared test, Fisher's exact test, or the Mann-Whitney U test. The data are presented as proportions of cases (%) for qualitative variables and as means (± SD) or medians (min-max) for quantitative variables.

release from fully mature megakaryocytes, a decrease or morphological change in the pulmonary capillary bed may lead to disruption of platelets[11].

Coronaviruses can also directly infect bone marrow elements or trigger auto-immune responses against blood cells, resulting in abnormal hematopoiesis. Thrombocytopenia is an important indicator of serious illness in COVID-19 patients, defined in 31.6% of patients with moderate symptoms and in 57.7% of patients with severe infections. However, parameters such as mean platelet volume and reticulated PLT may be useful in risk stratification and clinical decision-making[11,12].

A study[12], involving 245 COVID-19 patients, indicated that NLR, which reflects an elevated inflammatory process, was found to be an independent risk factor for in-hospital mortality, especially in male patients. In another study[13], 61 COVID-19 patients were prospectively evaluated, and in the advanced-age (50 years or older) group with NLRs of 3.13 or greater, serious illness and ICU admission were found to be significantly higher; therefore, NLR was considered an independent disease-related risk factor. In our study, increases in APACHE II score, hospital stay duration, WBC, PLR, NLR, and CRP, as well as decreases in PCT, were associated with mortality.

Studies[14,15] on SARS-CoV-2 patients have indicated that high neutrophil counts, lymphopenia, high CRP, and elevated LDH values are the most important determinants of mortality. Fois et al[14] found that leukocytosis, lymphopenia, and increased neutrophil counts were associated with increased disease severity.

Table III. Logistic regression analysis results.

	Univariate analysis					Multivariate analysis				
	Wald	*p*-value	95% CI for EXP (B)	Lower	Upper	Wald	*p*-value	95% CI for EXP (B)	Lower	Upper
Age	0.075	0.784	1.004	0.977	1.031					
APACHE II	32.293	< 0.001	1.385	1.238	1.550	82.044	< .001	1.462	1.347	1.587
SOFA	0.302	0.583	1.049	0.884	1.246					
Hospital stay duration	46.333	< 0.001	0.873	0.840	0.908	47.201	< .001	0.873	0.840	0.908
WBC	7.743	0.05	1.139	1.039	1.249	16.815	< .001	1.171	1.086	1.263
Neutrophile	0.118	0.731	1.024	0.895	1.171					
Lymphocyte	0.177	0.974	0.877	0.476	1.616					
Platetcrit	8.285	0.04	0.001	0.000	0.096	14.136	0.001	0.001	0.000	0.027
PLR	6.306	0.02	1.004	1.001	1.008	9.566	0.002	1.004	1.001	1.007
NLR	3.899	0.031	0.941	0.886	1.000	3.853	0.05	0.956	0.914	1.000
PNR	0.487	0.485	0.990	0.96	1.018					
CRP	7.277	0.007	1.005	1.001	1.008	9.606	< .001	1.005	1.002	1.008
PCT	0.012	0.913	1.004	0.935	1.078					

Wald: wald Chi-squared; OR: odds ratio; CI: confidence interval; Nagelkerke's R^2 (multinominal logistic regression) = 0.672; Hosmer-Lemeshow $p > 0.05$.

Table IV. ROC Curve Analysis for Mortality.

Variable	Cut-off	Sensitivity	Specificity	AUC (95% CI)	*p*-value
Plateletcrit	0.188	76.92	48.28	0.589 (0.531-0.647)	0.002
PLR	293.46	58.62	56.15	0.592 (0.537-0.647)	0.001
NLR	9.49	65.90	57.00	0.668 (0.617-0.719)	< 0.001

SD: sliding distance, APD: anterior-posterior diameter. SCA: cross-sectional area of the spinal canal, DSA: cross-sectional area of the dural sac, LFA: ligamentum flavum cross-sectional area, LFT: ligamentum flavum thickness. VAS: Visual Analogue Scale.

Based on a retrospective analysis of clinical data from 443 COVID-19 patients, Shang et al[15] concluded that NLR, CRP, and PLT indicate disease severity and that although all these parameters should be considered in the clinic, NLR is the best predictor. A meta-analysis[5] of 1,779 COVID-19 patients revealed that thrombocytopenia was associated with a threefold-enhanced risk of severe COVID-19 and that a low PLT level was associated with mortality.

Platelet indices are easily accessible parameters which are being increasingly recognized as markers of inflammation and thrombosis. Reductions in PLT have been proven[9,16] to be an independent risk factor for ICU patients. Simultaneous reductions in PLT and PCT indicate excessive consumption of platelets. MPV is the measure of platelet volume. When platelets are excessively consumed, the bone marrow produces a large number of immature platelets, which have a larger volume than mature ones. Thus, both newly produced platelets with large volumes and mature platelets with small volumes are present in the blood; therefore, both MPV and PDW increase[16]. Thus, instead of measuring only PLT as has been done previously in literature, we measured all the platelet indices to achieve a more comprehensive view of illness severity and an insight into the potential etiology behind changes in platelet indices. Two previously published studies[8,16] have reported that in sepsis and septic shock patients, in-hospital mortality was negatively correlated with PLT and PCT values and positively correlated with MPV and PDW values.

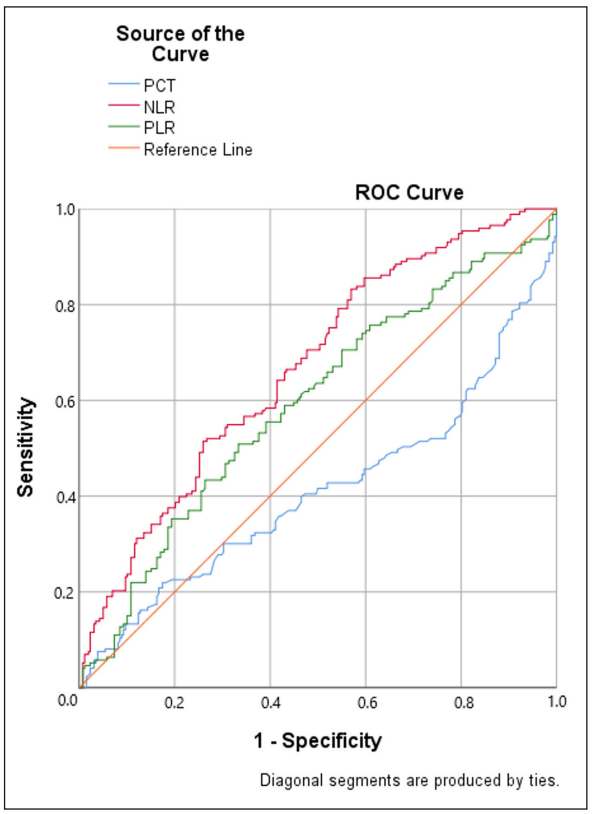

Figure 1. ROC Curve Analysis.

Bleibel et al[17] have suggested that PLT is an indicator of the degree of hemorrhage and inflammatory response. The possible explanation for the link between platelet indices and mortality is related to inflammation. Activation of the coagulation system, severe infection, trauma, systemic inflammatory response syndrome, and thrombotic diseases could all produce changes in PLT indices. The inflammatory response is widely accepted to be significantly associated with adverse clinical outcomes in ICU patients, and PCT is associated with increased mortality risk[9,16]. Our results are consistent with other studies[18-20] on trauma, cardiovascular medicine, and patients waiting on a liver transplant list.

As aforementioned, PCT is a measurement derived from the PLT and the mean platelet volume. PCT, MPV, MPV/platelet ratio, and several other parameters have been used as predictive indicators for many inflammatory diseases. By considering COVID-19 pneumonia as an inflammatory process based on its similarity to ARDS, we suggest using platelet indices as predictors of clinical course. In one study[21], patients who died in intensive care, probably due to sepsis, presented with low PCT levels. Like in those sepsis patients, PCT levels were lower in severe COVID-19 pneumonia patients. In

our study, PCT results showed a decrease in patients with mortality. ROC curve analysis was performed to determine the relevance of PCT, PLR, and NLR in predicting mortality in COVID-19 patients and to determine cut-off values for mortality. We determined that PCT, PLR, and NLR could correctly classify patients at rates of 58.9%, 59.2%, and 66.8%, respectively. For PCT, the sensitivity, specificity, and cut-off values were 76.92%, 48.28%, and 0.188, respectively. Thus, the mortality risk was elevated in cases with PCT values of 0.188 or lower[22]. Measuring all platelet indices, rather than only PLT, can provide a more comprehensive idea of disease severity. A previously published study[23] on septic shock patients reported that mortality was negatively correlated with PLT and PCT values and positively correlated with MPV and PDW values. Zhang et al[24], in a study of adult ICU patients, found that patients with low PLT values, low PCT values, high MPV values, or high PDW values had shorter survival times compared to patients with normal values. In another study[16], in which 1,556 patients, platelet indices were measured upon ICU admission and evaluated, high MPV and PDW values and low PLT and PCT values were associated with elevated mortality risk.

PCT is part of a complete blood count examination. Few studies[25-27] report the use of this parameter to predict clinical outcomes, including mortality. In 2021, Yardimci et al[25] examined platelet indices as a prognostic indicator in their study on a COVID-19 patient group and found that PLT and ratios were significantly associated with mortality in COVID-19 patients. Elevated first-day levels of CRP, lactate dehydrogenase (LDH), ferritin, MPV/PCT, PDW/PLT, PDW/PCT, and third-day levels of CRP, LDH, ferritin, procalcitonin, PDW, MPV/PCT, PDW/PLT, and PDW/PCT were also associated with poor prognoses. In the ROC curve analysis, the AUC values for MPV/PCT, PDW/PLT, and PDW/PCT levels, as prognostic indicators, were 0.710, 0.708, and 0.712, respectively. In their 2020 study, Sudharsono et al[26] showed that PCT and red blood cell distribution width (RDW-CV) were significantly associated ($p < .001$) with hospital mortality in 1,053 patients with acute coronary syndrome. The cut-off values for PCT and RDW-CV levels in ROC analysis were 0.25 (AUC = 0.65, $p < .001$) and 13.05 (AUC = 0.69, $p < .001$), respectively. In their 2011 study on 1,673 trauma patients, Lam et al[27] found significant differences between survivors and non-survivors in WBC count, absolute neutrophil count, segmented neutrophil count, red blood cell counts, and platelet indices. In the ROC curve analysis, the AUC values of PLT and PCT levels as indicators of 7-day in-hospital mortality were 0.684 and 0.696, respectively. In our study, the cut-off values for PCT, PLR, and NLR as hospital mortality indicators in COVID-19 patients were 0.188 (AUC = 0.589, $p < .002$), 293.46 (AUC = 0.592, $p < .001$) and 9.49 (AUC = 0.668, $p < .001$), respectively. We believe that these values can be used as indicators of mortality and prognosis in patient admission, follow-up, and treatment management.

Limitations and Strengths

Our study was limited in several ways. First, it was a single-center retrospective study and included a relatively small number of patients. Second, blood parameters were assessed using a single measurement taken upon ICU admission, and no serial measurements were taken. Therefore, the risk of type II error should be considered when evaluating our study's results. Our results should be supported by studies with larger sample sizes.

One strength of our study is that very few studies in the literature have investigated the relationship between platelet indices, particularly PCT, and mortality in COVID-19 patients. Additionally, platelet indices are low-cost parameters that can be easily evaluated. Increasing the use of these parameters, especially in intensive care units, for disease evaluation and follow-up may provide cost-effective treatment management. Considering the increasing number of patients and current ICU bed occupancy, PCT level, one of the platelet indexes, can be considered as an indicator of disease severity upon ICU admission.

Conclusions

Regarding the results of this research, platelet indices can be used to predict mortality in patients admitted to the ICU because of SARS-CoV-2-associated pneumonia. Additionally, PCT may be a predictive factor for mortality.

Funding

This research has not received any special funding.

Conflict of Interests

The authors declare that they have no competing interests.

Ethics Approval

Approval was obtained from the ethics committee at the University of Health Sciences, Ankara Atatürk Chest Diseases and Thoracic Surgery Training and Research Hospital (protocol number 719, date 25/03/2021). All procedures followed were in accordance with the ethical standards (institutional and national) of the committee responsible for human experiments and the 1975 Declaration of Helsinki.

Informed Consent

Not required, due to the retrospective nature of the study.

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Authors' Contributions

Semih Aydemir: conception, data collection, literature review, writer.
Fatih Segmen: data collection, critical review.
Onur Kucuk: writer, literature review, analysis, and interpretation.

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