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Correlation between Platelet-to-Lymphocyte Ratio, Neutrophil-to-Lymphocyte Ratio and Burden of Thrombus with Disease Severity in Patients with Pulmonary Thromboembolism

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Abstract

Background High neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are markers of subclinical inflammation and have been associated with prognosis and mortality in many diseases. In this study, we evaluated the comparative value of NLR and PLR in identifying high mortality risk in patients hospitalized with acute pulmonary thromboembolism (PTE), and their relationship with the anatomical burden of thrombus.

Methods Patients who were followed up due to PTE were evaluated retrospectively. NLR and PLR were calculated from complete blood counts. The thrombus burden was assessed by the Qanadli score; based on the patients' archival computed tomography angiography images. Mortality prediction was based on an algorithm using the Pulmonary Embolism Severity Index, echocardiographic findings, and troponin levels.

Results Three hundred-two PTE patients were included in the study. Median NLR, PLR, and Qanadli score values were higher in nonsurvivors, with NLR (8.4 [2.2–18.9]) vs. (3.1 [0.4–13.1]), PLR (317 [87.6–525.3]) vs. (124.4 [30–476.3]), and Qanadli scores (21 [3–26]) vs. (9 [1–28]), respectively ($p < 0.001$). We showed that setting a threshold value of >4.45 for NLR and >151.59 for PLR significantly predicts the high mortality-risk group. In the receiver operating characteristic analysis, when the threshold value for the Qanadli score distinguishing between low-risk and high-risk disease was set at 15.5, the sensitivity was calculated as 98.8% and the specificity was 94.9% ($p = 0.001$).

Conclusion This study showed that NLR, PLR, and Qanadli scores can provide essential contributions to the clinician's determination of the anatomical burden of thrombus and disease severity in PTE patients.

Keywords
- pulmonary thromboembolism
- neutrophil-to-lymphocyte ratio
- platelet-to-lymphocyte ratio
- Qanadli score
- mortality risk
- s-PESI

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Introduction

Pulmonary thromboembolism (PTE) is a condition associated with significant mortality and morbidity. It has the potential for recurrence and can pose diagnostic challenges in certain instances.[1] Typically, it develops as a complication of deep vein thrombosis (DVT) and ranks as the third most prevalent cause of cardiovascular mortality. Inflammation plays a crucial role in pathogenesis. Numerous risk factors contribute to intravascular clotting, including trauma, major surgery, advanced age, prolonged travel, and immobilization; additionally, genetic and idiopathic cases also exist. The diagnosis of acute PTE is made by the clinician suspecting the disease and performing appropriate diagnostic tests in line with this suspicion.

Given that it can be incidentally detected clinically or lead to sudden death, the clinician needs to precisely assess the disease severity and promptly start appropriate treatment tailored to the patient.[2] Mortality prediction assessment using the simplified Pulmonary Embolism Severity Index (s-PESI), echocardiography (ECHO) findings, and troponin levels as suggested by the 2019 ESC Guidelines on the Diagnosis and Management of Acute Pulmonary Embolism is a valuable scoring system used to stratify PTE patients in terms of early mortality.[1] These categories also help to guide clinical management and predict outcomes.[1] The person's cardiopulmonary reserve and thrombus burden determine the disease severity. The extent and location of the thrombus can be evaluated by computed tomography (CT) angiography using different scoring methods.[3] Qanadli is among the most frequently used scoring methods.[4]

Numerous studies have explored biomarkers to assess patient prognosis and guide treatment decisions. The neutrophil-to-lymphocyte ratio (NLR) and the platelet-to-lymphocyte ratio (PLR) serve as biomarkers that indicate equilibrium between systemic inflammation and immunity.[5] A high NLR is a subclinical inflammation marker linked to prognosis and mortality across various diseases. Given the pivotal role of inflammation, particularly neutrophils, in the development and clinical course of venous thromboembolism, NLR might serve as a prognostic indicator in this context. NLR has been evaluated as a predictive marker in various diseases, including malignancies and cardiovascular diseases, and is associated with disease course and outcome.[6] Due to its advantage of low cost and easy availability, the diagnostic and prognostic value of PLR has been widely reported in various diseases, including diabetes, cardiovascular diseases, renal diseases, autoimmune diseases, and cancers.[7,8] PLR is also an inflammatory marker and has been shown to have prognostic value in vascular diseases. Inflammation tends to be more pronounced in patients with an increased thrombus burden. Tabakacı et al divided aortic dissection patients into two groups, high PLR and low PLR, and determined that mortality was higher in the high PLR group.[9] Qu et al found that the PLR value was higher, especially in severe and elderly coronavirus disease-2019 (COVID-19) patients, and stated that it was an independent risk factor for patients with severe conditions.[10]

Limited research evaluated the comparative significance of NLR and PLR with high mortality risk in hospitalized PTE patients and its relationship with the anatomical burden of thrombus.[11–13] In our study, we aim to investigate these relations.

Methods

The Gazi University Faculty of Medicine Clinical Research Ethics Committee approved this retrospective, single-center study on May 30, 2022, under decision number 399. This study was performed in line with the principles of the Declaration of Helsinki. The study included 302 patients we followed up for PTE at our clinic from January 1, 2012 to April 30, 2022. The flowchart of the study is summarized in ►Fig. 1. We recorded demographic data, comorbidities, previous history of DVT and PTE, risk factors, s-PESI score, vital signs (body temperature, pulse rate, blood pressure, oxygen saturation), D-dimer levels, troponin levels, hemogram results, admission blood gas measurements, duration of invasive mechanical ventilation (if applied during hospitalization), and ECHO findings (including main pulmonary artery diameter in millimeters, pulmonary artery pressure, systolic displacement of the tricuspid valve in the annular plane, and thrombus anatomical burden expressed as the occlusion rate of the pulmonary artery vascular bed). The 30-day mortality was determined from patient follow-up records. Patients with acute infection at admission and with active malignancy and pancytopenia were excluded

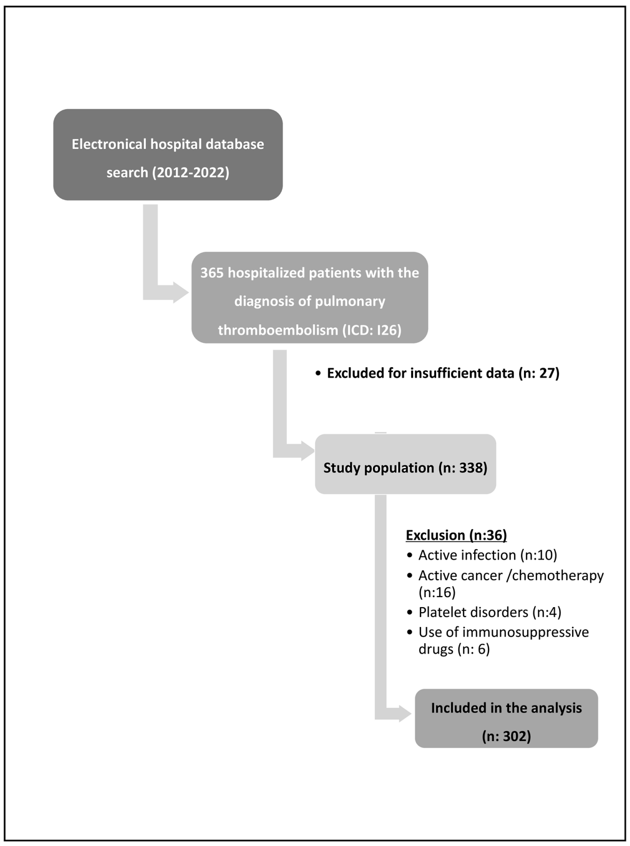

Fig. 1 Flowchart of the study.

from the study. NLR and PLR rates were calculated using the complete blood count values taken at the admission. The NLR was determined by dividing the total neutrophil count by the total lymphocyte count. Similarly, the PLR was calculated by dividing the total platelet count by the lymphocyte count.

Mortality prediction assessment was made based on an algorithm using combined parameters and scores (s-PESI, ECHO findings, troponin levels) as suggested by the 2019 ESC Guidelines on the Diagnosis and Management of Acute Pulmonary Embolism.[1] The s-PESI values for the patients were determined as described in the literature.[14]

Radiologists assessed the thrombus burden by calculating the Qanadli score using archival CT angiography images of the patients using the methodology described in the existing literature. In the context of the Qanadli score, each lung undergoes evaluation across 10 segments (3 segments for the upper lobes, 2 for the middle lobe or lingula, and 5 for the lower lobes). A weighting factor is assigned based on the degree of vascular obstruction: 0 points for no thrombus, 1 for partially occlusive thrombus, and 2 for total occlusion. The maximum score per patient is 40.[4]

Statistical Analysis

The statistical analysis used IBM SPSS (Statistical Package for the Social Sciences) version 26.0 (SPSS Inc., Chicago, Illinois, United States). In descriptive statistical analyses, continuous variables were reported as mean ± standard deviation (if the data follow a normal distribution) and median (minimum–maximum, if the data do not follow a normal distribution). Categorical variables were presented as numbers (count of occurrences) and percentages (% - relative frequency). The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test, and based on the test results, either parametric or nonparametric statistical tests were employed. The Kruskal–Wallis test was used for the comparison of more than two groups with nonnormal distribution. In cases where significant differences were detected, post-hoc analysis using the Dunn procedure with Bonferroni correction was performed to determine which specific pairs of groups exhibited significant differences. The Mann–Whitney U-test was used for the comparison of two nonparametric groups. ROC (receiver operating characteristic) analysis was employed to assess whether the NLR and PLR could serve as diagnostic indicators for mortality risk in patients diagnosed with PTE and whether they could be utilized for this purpose. Spearman correlation analysis was employed to assess the relationships between the Qanadli score and s-PESI, NLR, and PLR. The statistical significance level was accepted as $p < 0.05$.

Results

A total of 302 patients, 168 (55.6%) females and 134 (44.4%) males, were included in the study, and the median age was 62 ± 16 (min: 21, max: 95). Demographic data of the patients are shown in ►Table 1. Laboratory values of the patients are shown in ►Table 2.

Table 1 Demographic data of the patients

	n (%), mean ± SD
Age	62 ± 16
Sex (female)	168 (55.6%)
Mortality risk	
High risk	86 (28.5%)
Intermediate risk	119 (39.4%)
Intermediate-low risk	66 (21.9%)
Intermediate-high risk	53 (17.5%)
Low risk	97 (32.1%)
Comorbidities	
History of PTE	33 (10.9%)
Hypertension	126 (41.7%)
Diabetes mellitus	60 (19.9%)
Cerebrovascular event	10 (3.3%)
Heart failure	40 (13.2%)
Atrial fibrillation	48 (15.9%)
COPD	33 (10.9%)
Asthma	46 (15.2%)
Chronic renal failure	13 (4.3%)

Abbreviations: COPD, chronic obstructive pulmonary disease; PTE, pulmonary thromboembolism; SD, standard deviation.
Note: Values are expressed as mean ± SD and individuals (percentage).

Table 2 Mean laboratory values of the patients included in the study

D-Dimer (μg/mL)	3.07 ± 3.15
Troponin I (μg/L)	54.85 ± 130.89
Hemoglobin (g/dL) (12–14.6)	12.76 ± 1.91
WBC ($\times 10^9$/L)	14.36 ± 6.5
Neutrophil ($\times 10^9$/L)	7.19 ± 3.25
Lymphocyte ($\times 10^9$/L)	2.14 ± 0.84
PLT ($\times 10^9$/L)	267.37 ± 89.23

Abbreviations: PLT, platelet count; WBC, white blood cell count.
Note: Values are expressed as mean ± standard deviation.

Upon analyzing the mortality risk groups as described before, 86 patients (28.5%) were in a high mortality risk group, 119 patients (39.4%) were in an intermediate mortality risk group (53 patients were in intermediate-high and 66 patients were in intermediate-low mortality risk group), and 97 patients (32.1%) were in a low mortality risk group. s-PESI values are 1 (1–5), 1 (1–3), 1 (0–2), and 0 (0–0), in high, intermediate-high, intermediate-low, and low mortality risk groups, respectively.

The patients' median NLR ($n = 302$) and PLR ($n = 302$) values were 3.5 (min–max: 0.42–18.9) and 132.9 (min–max:

Table 3 Median NLR and PLR values in different clinical mortality risk groups

	Low risk	Intermediate-low risk	Intermediate-high risk	High risk	*p*-Value
NLR	1.8 (0.4–4.7)[a]	2.8 (1.0–11.8)[a]	4.7 (2.4–17.7)[a]	7.5 (2.9–18.9)[a]	<**0.001***
PLR	89.7 (28.9–177.6)[b]	115.8 (54.0–356.5)[b]	159.0 (71.8–412.8)[b]	235.6 (103.6–540.0)[b]	<**0.001***
Qanadli score	2.0 (1.0–6.0)[c]	8.0 (5.0–25.0)[c]	14.0(8.0–21.0)[c]	19.0 (15.0–28.0)[c]	<**0.001***

Abbreviations: NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio.
Note: Values are expressed as median (confidence interval). *The analysis was conducted using the Kruskal–Wallis test.
[a]Results of post-hoc analysis using the Dunn procedure with Bonferroni correction for all pairwise comparisons showed $p < 0.001$.
[b]Results of post-hoc analysis using the Dunn procedure with Bonferroni correction for all pairwise comparisons showed $p < 0.001$.
[c]Results of post-hoc analysis using the Dunn procedure with Bonferroni correction for all pairwise comparisons showed $p < 0.001$.

29–540), respectively. NLR and PLR values significantly increased as mortality risk increased ($p < 0.001$ for all pairwise group comparisons). NLR and PLR values in different clinical mortality risk groups are shown in ►Table 3. The Qanadli score was 19.0 (15.0–28.0), 14.0 (8.0–21.0), 8.0 (5.0–25.0), and 2.0 (1.0–6.0) in high, intermediate-high, intermediate-low, and low mortality risk groups, respectively. The Kruskal–Wallis test was employed to evaluate group differences. Following this, post-hoc analysis using the Dunn procedure with Bonferroni correction was performed to account for multiple comparisons. The results indicated significant differences between all groups, particularly in low-risk patients, where the Qanadli score was significantly lower compared with other groups ($p < 0.001$; ►Table 3). A positive correlation was observed between the total Qanadli score and median s-PESI of the total patient group ($p < 0.001$, $r = 0.830$), NLR ($p < 0.001$, $r = 0.865$), and PLR ($p < 0.001$, $r = 0.773$). Twenty-five (8.3%) of the patients included in the study experienced mortality during the 30-day follow-up period. In patients who died within 30 days, NLR (8.4 [2.2–18.9]), PLR (317 [87.6–525.3]), and Qanadli scores (21 [3–26]) were found to be significantly higher compared with other patients (NLR (3.1 [0.4–13.1]), PLR (124.4 [30–476.3]), Qanadli scores (9 [1–28]) ($p < 0.001$; $p < 0.001$; $p < 0.001$). A significant positive correlation has been identified between 30-day mortality and NLR ($p < 0.001$, $r = 0.312$), PLR ($p < 0.001$, $r = 0.312$), and the Qanadli score ($p < 0.001$, $r = 0.269$), calculated using Spearman's correlation coefficient.

We conducted a ROC analysis to identify the optimal cutoff value for the Qanadli score in predicting the mortality risk. During the ROC analysis, when the Qanadli score threshold for differentiating between low-risk and high-risk diseases was set at 15.5, the sensitivity was found to be 98.8%, and the specificity was 94.9% ($p = 0.001$; ►Fig. 2 and ►Table 4).

We showed that setting a threshold value >4.45 for NLR (AUC [area under the curve] = 0.96, sensitivity: 98.4%, specificity: 85.4%, $p < 0.001$) and >151.59 for PLR (AUC = 0.93, sensitivity: 87.3%, specificity: 79.8%, $p < 0.001$) is significant in predicting the high-mortality risk group (►Fig. 3, ►Table 4).

Discussion

Exploring novel prognostic factors in patients with PTE offers valuable insights for the clinical management of these patients. This prognostic assessment helps provide a more accurate prediction of short-term adverse events. Numerous studies have investigated NLR and PLR for this specific purpose. Our study evaluated the association between these ratios, thrombus anatomical burden, and mortality risk prediction. Additionally, these parameters were found to be correlated with mortality rate.

The NLR, which is straightforward, cost-effective, and readily available, is a clinical parameter reflecting the vigor of the host's inflammatory response. During infection or inflammation, neutrophil levels in the blood rise while lymphocyte levels decline. Numerous studies have shown that elevated NLR may be an independent predictor of prognosis in a variety of clinical conditions, including malignancies, chronic obstructive pulmonary disease (COPD), sepsis, ischemic stroke, retinal artery occlusion, cardiovascular disease, acute respiratory distress syndrome, and fibrotic liver diseases.[15–17]

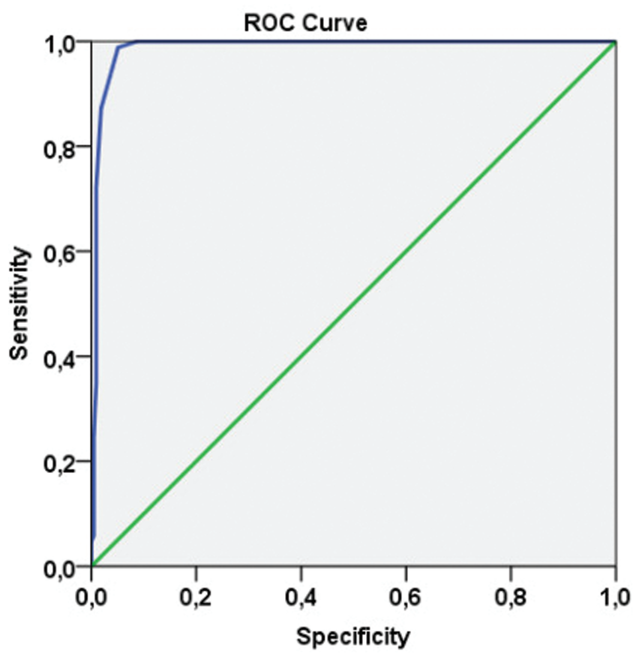

Fig. 2 The receiver operating characteristic curve illustrates the sensitivity and specificity of the Qanadli score in differentiating between low-risk and high-risk diseases in patients with pulmonary thromboembolism.

Table 4 ROC analysis results of NLR, PLR, and Qanadli score values in predicting mortality risk

	Cut-off value	Sensitivity (%)	Specificity (%)	AUC	*p*-Value
NLR	4.45	98.4	85.4	0.96	0.001
PLR	151.59	87.3	79.8	0.93	0.001
Qanadli score	15.5	98.8	94.9	0.98	0.001

Abbreviations: AUC, area under the curve; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio.

PLR, a clinically established inflammatory marker, is easily calculated and widely appreciated. Recent studies show elevated PLR reflects inflammation, atherosclerosis, and platelet activation. Recent research has acknowledged its utility in diagnosing and predicting outcomes for various infectious diseases, including COVID-19 infection; furthermore, it has been the subject of many studies alongside NLR.[18] In a retrospective study conducted at a single center, researchers investigated the prognostic significance of NLR and PLR in differentiating COVID-19 patients with and without pneumonia, and they were higher in patients with pneumonia.[19] In another retrospective study involving COPD patients, NLR and PLR values were 2.24 ± 0.56 and 157.1 ± 28.36, respectively. Notably, both ratios were significantly elevated compared with the control group ($p < 0.001$). These rates were higher during the exacerbation than during the stable period.[20]

In another study investigating the NLR and the PLR in patients diagnosed with DVT, it was found that these ratios were statistically significantly higher in the patient group compared with the control group. Notably, the study emphasized that PLR could serve as an effective biomarker for accelerating the diagnostic process in patients with suspected DVT.[21] In recent years, it has been suggested that NLR also has a prognostic role in acute PTE. A retrospective study that assessed prognostic markers affecting 30-day mortality, and NLR, PLR, N-terminal pro-brain Natriuretic Peptide, and d-dimer values were significantly increased in patients who died within 30 days.[22] In a different retrospective study, a significant positive relationship was found between high NLR values and mortality within 30 days and 1 year. This study showed that NLR with a median value above 5.12 was associated with a higher risk of 30-day and 1-year mortality.[23] Another study retrospectively screened 82 patients with PTE; the cut-off value for PLR in patients with high s-PESI values was 156, and the cut-off value for NLR was 3.56.[24] Previous studies have shown a relationship between PLR and a high risk of in-hospital mortality in patients with acute PTE and found it to be correlated with established risk scores.[25]

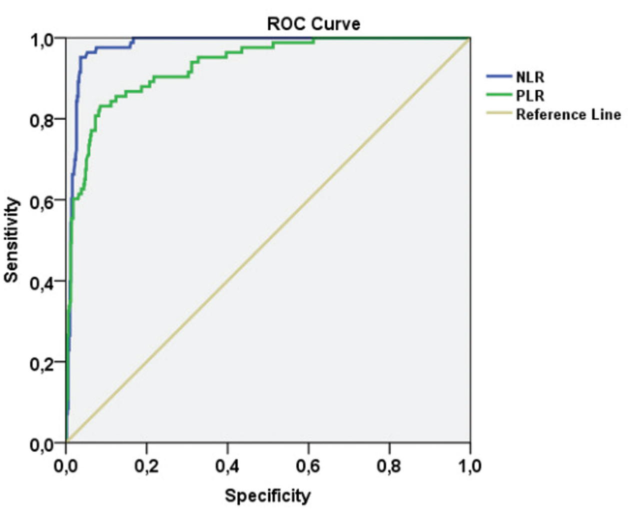

Fig. 3 Receiver operating characteristic curve illustrating the sensitivity and specificity of NLR and PLR to predict a high-risk group in patients with pulmonary thromboembolism. NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio.

Similar results were obtained in our study, with higher values of these ratios observed in patients diagnosed with PTE. We also observed higher NLR and PLR values in patients with increasing mortality risk and patients with a fatal course within 30 days.

Various clot burden scores in the lungs have been described. Among them, the Qanadli PTE index is simple and easily calculated, which was derived from the amount and location of the thrombus on CT images based on a study.[26] The Qanadli scoring method has been the subject of research in many studies. It is stated that it can distinguish between high and low mortality risk in acute PTE.[4,27] In a retrospective study, patients were categorized into two groups based on a Qanadli score cut-off of 18 points, as determined by ROC curve analysis. It was demonstrated that patients with a cut-off score greater than 18 had elevated levels of laboratory tests (troponin and D-dimer), and the findings from ECHO and CT angiography were more pronounced.[28] This score has been shown to have a linear correlation with various variables associated with increased morbidity and mortality.[29] In the study by El-Menyar et al[30], a significant correlation was observed between the Qanadli score and the right ventricular to left ventricular index. According to the ROC curve, a Qanadli score above 17.5 was effective in detecting right ventricular dysfunction and predicting mortality. In a different study, a significant correlation between the Qanadli score and NLR was observed, which is consistent with the findings of our study.[13] As per our research, no existing literature has directly compared the Qanadli score with these indices.

However, our study has some limitations. The most important of these is that our study is single-center and retrospective. Because of the retrospective design, control group cannot be included.

Conclusion

In conclusion, this study demonstrated that NLR, PLR values, and Qanadli score can make significant contributions to the clinician in predicting disease severity and mortality risk especially in situations where immediate access to ECHO and troponin values is not possible. These simple parameters can help clinicians to identify patients who need close follow-up.

There are limited studies evaluating the thrombus burden, NLR, and PLR in PTE patients. In the future, new prospective studies will more clearly define their role in determining mortality risk in PTE patients and guiding the treatment.

Author Contributions

A.M. and İ.K.O. designed and coordinated the study, participated in data acquisition and interpretation, and drafted the article. A.M., K.T., G.J., A.O., M.T., S.D., S.A., and O.G. participated in the data collection. Radiologic images were evaluated and reviewed by A.C.K.K., S.K.Ö., and G.E. Statistical analyses were performed by Z.Y. and reviewed in detail by İ.K.O. All authors participated in the review and revision of the manuscript. All authors have approved and take responsibility for the final version of the manuscript.

Ethical Declarations

The study was approved by the Gazi University Clinical Research Ethics Committee (Date: 30.05.2022 and Decision No: 399).

Submission Declaration

This study has yet to be previously published or is under consideration for publication elsewhere.

Competing Interests

The authors have no relevant financial or nonfinancial interests to disclose.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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