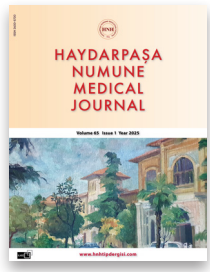
HAYDARPAŞA NUMUNE MEDICAL JOURNAL

DOI: 10.14744/hnhj.2025.87269
Haydarpasa Numune Med J 2026;66(1):58–64



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ORIGINAL ARTICLE

Relationship between Disease Severity and Blood Cell Ratios in Tuberculosis

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Abstract

Introduction: The neutrophil/lymphocyte (NLR), platelet/lymphocyte (PLR), and monocyte/lymphocyte (MLR) ratios are markers indicating the severity of inflammation that can be obtained from the complete blood count (CBC). We examined the relationships between these inflammatory markers (C-reactive protein [CRP], NLR, PLR, and MLR) and disease extent, as evidenced by chest radiography, bacterial burden, and hospital mortality, in patients hospitalized with a bacteriologically confirmed pulmonary tuberculosis (TB) diagnosis.

Methods: The study included 265 patients diagnosed with pulmonary TB without multidrug resistance. CBC and biochemical values were recorded before TB treatment. The disease was defined as mild, moderate, or severe according to the number and location of cavities. The smear grade was used to determine the bacterial burden at the time of diagnosis, and patients were classified into two groups (grade≥3 or <3) depending on the number of bacilli detected in sputum smear microscopy analysis.

Results: A comparison of the NLR (7.32±6.26 vs. 5.14±4.87), PLR (349.61±241.49 vs. 244.52±167.4), and MLR (0.78±0.43 vs. 0.61±0.34) between patients with severe and moderate/mild disease revealed significantly higher values in patients with severe disease ($p<0.001$). These values were not significantly correlated with the smear grade ($p>0.05$). Hospital mortality was observed in nine patients (3%), and the NLR and CRP levels had significant relationships with mortality ($p<0.05$).

Discussion and Conclusion: NLR, PLR, and MLR obtained from the complete blood count, which is a simple and inexpensive method at the time of diagnosis, were found to be associated with the radiological extent of the disease. In addition, these markers may be useful in the early identification of severe disease.

Keywords: Complete blood count, radiology, tuberculosis.

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Submitted Date: 19.03.2024 **Revised Date:** 20.10.2025 **Accepted Date:** 28.10.2025
Haydarpaşa Numune Medical Journal
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Tuberculosis (TB) remains one of the leading causes of death from infectious diseases worldwide [1]. In recent years, rapid advances in diagnostic tools and radiologic imaging techniques have enabled earlier diagnosis of pulmonary TB (PTB) and increased the likelihood of timely treatment initiation.

The delayed immune response mounted by the host against *Mycobacterium tuberculosis* underlies the pathogenesis of TB. After inhalation of tubercle bacilli into the lungs, a complex interaction begins between the pathogen and components of the host immune response, particularly T lymphocytes and macrophages, which play central roles in the development of cavitary lung disease. Various cytokines (e.g., IL-2, IFN-α, IL-6, TNF-α) and platelet-derived growth factor secreted by these cells contribute significantly to the inflammatory process. These cytokines also regulate the recruitment of inflammatory cells into tissues. The severity of inflammation, host immunogenicity, and nutritional status collectively determine disease severity [2,3].

The neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR) are readily obtainable from a complete blood count (CBC) and serve as markers of systemic inflammation. These ratios have been investigated as prognostic indicators in various inflammatory diseases, coronary heart disease, and malignancies [4–6]. The NLR and PLR are elevated in patients with active PTB compared with healthy controls and are associated with disease severity [7–9]. Previous studies have also demonstrated elevated MLR levels during the active phase of TB, which decrease to normal levels following treatment [10]. Moreover, MLR may serve as a predictive marker for the development of active TB [11].

In this study, we aimed to examine the relationships between inflammatory markers (C-reactive protein [CRP], NLR, PLR, and MLR) and radiologic disease extent, smear grade, and mortality in patients hospitalized with a bacteriologically confirmed diagnosis of active PTB.

Materials and Methods

This retrospective cohort study was conducted at a tertiary training and research hospital with a dedicated TB clinic. The study was approved by the Istanbul Training and Research Hospital Clinical Research Ethics Committee (Decision: 2064; December 6, 2019) and was performed in accordance with the Declaration of Helsinki.

Patients hospitalized in the TB clinic between January 1, 2016, and December 30, 2018, were retrospectively identified through the hospital's electronic database. Patients with positive acid-resistant bacilli (ARB) results on sputum smear microscopy, RT-PCR of sputum samples, or ARB culture of respiratory specimens, as well as those with a new or recurrent diagnosis of PTB, were evaluated. Patients with PTB and pleural involvement were included. Those with extrapulmonary TB without pulmonary involvement or with organ involvement other than pleural disease were excluded. Patients who had initiated anti-TB treatment prior to hospitalization or whose CBC results were not available before treatment were also excluded. Multidrug-resistant TB cases identified through drug-resistance testing were excluded.

Of the 333 patients who met the inclusion criteria, demographic data were recorded. Patients with active malignancy, hematologic disorders, autoimmune diseases, immunosuppressive conditions, HIV infection, acute or chronic renal failure, acute cardiac events, or those receiving hemodialysis were excluded. The final study population consisted of 265 patients. Length of hospital stay and hospitalization outcomes (discharge or death) were recorded. Hospital mortality was defined as death from any cause during hospitalization. Radiologic disease extent was classified according to the criteria described by Dlugovitzky et al. [12]

Disease extent was categorized as follows:

- **Mild disease:** single-lobe involvement without visible cavities or pleural involvement.
- **Moderate disease:** unilateral involvement of one or more lobes with a cavity, or mild disease with pleural involvement or effusion.
- **Severe disease:** bilateral involvement, multiple cavities within a single lobe, or moderate disease with pleural involvement or effusion.

For analysis, patients were grouped as having severe disease or mild-to-moderate disease based on radiologic findings.

Sputum smears were stained using the Ziehl–Neelsen method and examined microscopically for ARB. Smear grading was performed according to standard criteria: 1–9 ARB/100 fields (1+); 1–9 ARB/10 fields (2+); 1–9 ARB/field (3+); and >9 ARB/field (4+) [13]. Smear grade was used to assess bacterial burden and was categorized as ≥3 or <3.

All laboratory samples were collected prior to treatment initiation. CBC analyses were performed using the Mindray BC-6800 Auto Hematology Analyzer. In accordance with hospital laboratory policy, each CBC was processed within 1 hour of sample collection at room temperature.

Absolute neutrophil, lymphocyte, monocyte, and platelet counts were recorded. NLR was calculated as the absolute neutrophil count divided by the absolute lymphocyte count. PLR was calculated as the absolute platelet count divided by the absolute lymphocyte count. MLR was calculated as the absolute monocyte count divided by the absolute lymphocyte count. Serum CRP levels (normal range, 0–5 mg/L) were measured using a Cobas 6000 analyzer (Roche) within 1 hour of sample collection.

Statistical Analysis

Descriptive statistics are presented as median (minimum–maximum) for continuous variables and as percentages for categorical variables. Data normality was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. The Mann–Whitney U test was used to compare nonparametric continuous variables between two independent groups.

Pearson's chi-square test and the continuity correction test were used for categorical data analysis. Receiver operating characteristic (ROC) curve analysis was performed to determine optimal NLR cut-off values. A p value <0.05 was considered statistically significant at a 95% confidence interval. Statistical analyses were conducted using SPSS version 21.0 (IBM Corp., Armonk, NY, USA).

Results

A total of 265 patients were included, comprising 91 women (34.3%) and 174 men (65.7%). The mean age was 43.75±19.58 years. Among patients with available smoking data, 100 (37.7%) had never smoked, 86 (32.5%) were current smokers, and 48 (18.1%) were former smokers. No comorbidities were identified in 181 patients (68.3%), whereas 84 patients (31.7%) had at least one comorbid condition, including chronic obstructive pulmonary disease (COPD), diabetes mellitus (DM), hypertension (HTN), or ischemic heart disease.

Severe cavitary disease was significantly more common in men, current smokers, and patients without comorbidities (p<0.05). No significant association was observed between bacterial burden and radiologic disease extent (p=0.17). Radiologic disease extent did not differ significantly between survivors and non-survivors (p=0.11) (Table 1).

Comparison of radiologic disease extent and laboratory findings revealed that monocyte, neutrophil, platelet counts, NLR, PLR, and CRP levels were significantly higher in patients with severe cavitary disease (p<0.001). In contrast, lymphocyte (p=0.003) and albumin levels (p<0.001) were significantly lower in patients with severe disease compared with those with mild-to-moderate disease (Table 2).

ROC curve analysis demonstrated that NLR, PLR, MLR, and CRP predicted radiologic disease extent with cut-off values of 4.7, 232.45, 0.60, and 73.6, respectively. The area under

Table 1. Comparison of demographic and clinical characteristics of patients with radiological extend of disease

Variables	Mild- Moderate (n=127)	Severe (n=138)	p
Age, median (min-max)	39 (14-89)	42 (14-84)	0.587
Sex,n (%)			
Female	59 (46.5)	32 (23.2)	<0.001
Male	68 (53.5)	106 (76.8)	
Smoking history, n (%)			
Non-smokers	55 (50.0)	45 (36.3)	
Current smokers	30 (27.3)	56 (45.2)	0.017
Ex-smokers	25 (22.7)	23 (18.5)	
Comorbidities, n (%)			
Yes	78 (61.4)	103 (74.6)	0.021
No	49 (38.6)	35 (25.4)	
Degree of smear positivity, n (%)			
<3	37 (55.2)	51 (44.7)	0.173
=>3	30 (44.8)	63 (55.3)	
Hospital mortality, n (%)			
No	128 (98.4)	131 (94.9)	0.116
Yes	2 (1.6)	7 (5.1)	

Table 2. Comparison of laboratory results of patients with radiological extend of disease

Variables	Mild- Moderate PTB(n=127)	Severe PTB (n=138)	p value
Monocyte (103/µL)	0.78(0.26-3.13)	0.97(0.15-2.52)	<0.001
Lymphocytes (103/µL)	1.6(0.25-4)	1.47(0.28-4.56)	0.010
Neutrophil (103/µL)	6.39(2.11-15.53)	8.12(1.38-21.65)	<0.001
Platelets (103/µL)	320(79-752)	409(4.24-812)	<0.001
NLR	3.85(0.78-44.84)	5.82(1.34-58.91)	<0.001
PLR	205.42(62.75-1343.48)	280.65(2.34-1507.14)	<0.001
MLR	0.55(0.1-1.8)	0.69(0.2-3.18)	<0.001
CRP (mg/dL)	55.2(0.4-224.9)	105.7(1.4-431)	<0.001
Albumin (g/dL)	3.8(0-36.4)	3.1(1.3-39.1)	<0.001

Values are presented as median (minimun-maximum).
PTB: Pulmonary tuberculosis patients; NLR: Neutrophil/lymphocyte ratio; PLR: Platelet/lymphocyte ratio; MLR: Monocyte/lymphocyte; CRP: C-reactive protein

the curve (AUC) values were 0.681 (0.619–0.743), 0.662 (0.599–0.721), 0.651 (0.587–0.716), and 0.742 (0.685–0.802), respectively. Sensitivity values were 64.2%, 63.3%, 60.8%, and 70.5%, and specificity values were 63.7%, 63.6%, 60.1%, and 69.2%, respectively (Table 3, Fig. 1).

Smear grade was not associated with age, sex, smoking status, or comorbidities (Table 4). Bacterial burden did not differ significantly between survivors and non-survivors (p=0.65). No significant associations were found between bacterial burden and laboratory variables (p>0.05) (Table 5). Likewise, no significant relationships were identified between comorbidities and laboratory parameters (p>0.05).

The hospital mortality rate was 9/255. NLR and CRP levels were significantly higher in non-survivors than in survivors (14.36±13.38 vs 5.99±5.09 and 291.57±356.79 vs 80.93±61.07, respectively; p<0.05). Albumin levels were significantly higher in survivors compared with non-survivors (3.71±3.44 vs 2.61±0.57; p=0.006).

ROC analysis for hospital mortality showed an NLR cut-off value of 7.2, with an AUC of 0.729 (0.530–0.928). Sensitivity and specificity were 66.7% and 73.1%, respectively. For CRP, the cut-off value was 73.6, with an AUC of 0.799 (0.634–0.963). Sensitivity and specificity were 71.4% and 70.1%, respectively (Fig. 2).

Discussion

In this study, NLR, PLR, and MLR values obtained from the complete blood count were compared between patients with radiologically severe disease and those with moderate or mild disease. These values were significantly higher in patients with severe disease. No significant correlation was found between these markers and smear grade (p>0.05). Hospital mortality occurred in nine patients (0.03%), and both NLR and CRP levels were significantly associated with mortality (p<0.05).

A well-established association exists between disease severity and the intensity of inflammation in patients with

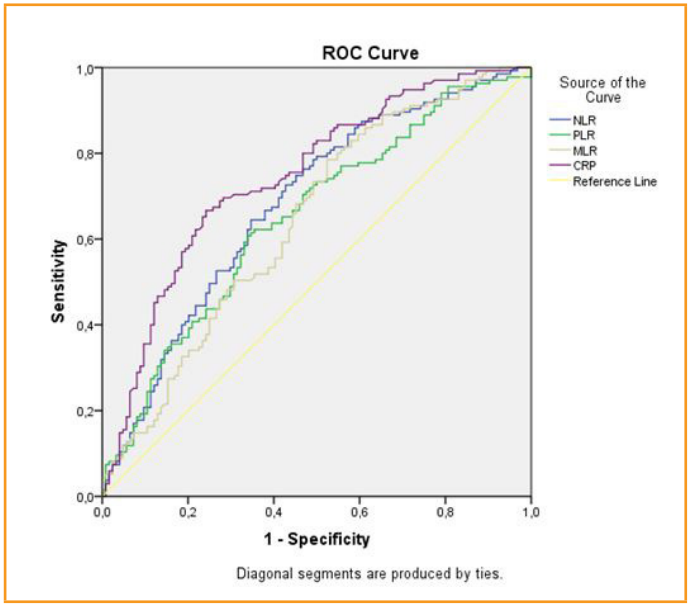

Figure 1. Receiver operating characteristic (ROC) curves of blood neutrophil/lymphocyte ratio (NLR), platelet/lymphocyte ratio (PLR), monocyte/lymphocyte (MLR) and C-reactive protein (CRP) in detecting radiological extent of disease.

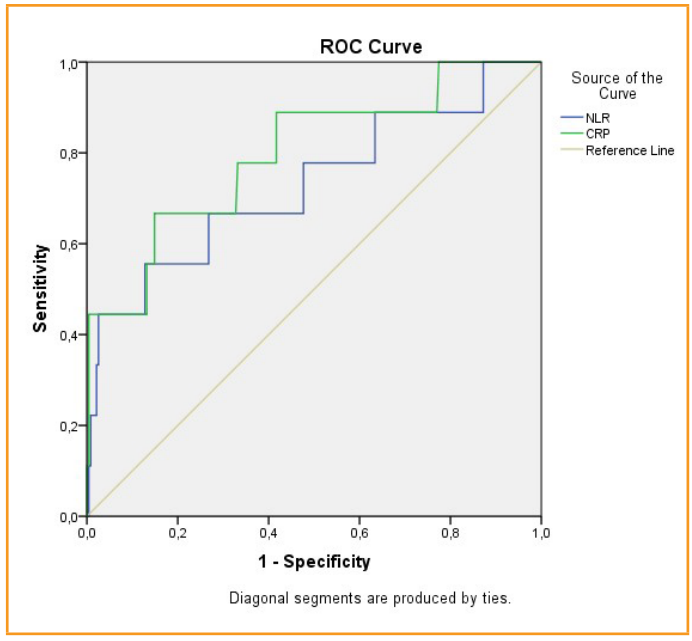

Figure 2. Receiver operating characteristic curves for NLR, CRP, in predicting hospital mortality.

Table 3. ROC analysis results of NLR, PLR, MLR and CRP for radiological extent of disease

	Cut-Off value	AUC	Sensitivity%	Specificity%	p
NLR	4.6574	0.681 (0.619-0.743)	64.1	63.2	<0.001
PLR	232.45	0.662 (0.599-0.721)	63.4	63.2	<0.001
MLR	0.6010	0.651(0.587-0.716)	60.7	60.0	<0.001
CRP	73.561	0.742(0.685-0.802)	70.4	69.4	<0.001

ROC: Receiver operating characteristic, AUC: The area under the curve; NLR: Neutrophil/lymphocyte ratio; PLR: Platelet/lymphocyte ratio; MLR: Monocyte/lymphocyte ratio; CRP: C-reactive protein

Table 4. Comparison of demographic and clinical characteristics with microscopic degree of smear positivity

Variables	ARB<3 (n=88)	ARB≥3 (n=93)	p
Age, median (min-max)	40 (15-85)	44 (15-89)	0.533
Sex			
Female, n (%)	27 (30.7)	22 (23.7)	0.286
Male, n (%)	61 (69.3)	71 (76.3)	
Smoking history, n (%)			
Nonsmokers	27 (37.8)	30 (36.1)	0.630
Current smokers	35 (43.8)	39 (47.0)	
Ex-smokers	18 (22.5)	14 (16.9)	
Comorbidities, n (%)			
Yes	60 (64.5)	59 (60.8)	0.599
No	33 (35.5)	38 (39.2)	
Hospital mortality, n (%)			
No	89 (95.7)	94 (96.9)	0.659
Yes	4 (4.3)	3 (3.1)	

ARB: Acid-resistant bacilli

Table 5. Comparison of laboratory results and microscopicdegree of smear positivity

Variables	ARB<3 (n=88)	ARB≥3 (n=93)	p
Monocyte (10^3/µL)	0.94 (0.33-3.13)	0.96 (0.15-2.52)	0.469
Lymphocytes (10^3/µL)	1.45 (0.28-4.56)	1.54 (0.31-3.17)	0.883
Neutrophil (10^3/µL)	7.01 (2.75-17.08)	8.02 (3.44-21.65)	0.091
PLT	376 (138-752)	382 (4.24-812)	0.619
NLR	4.97 (0.87-25.88)	5.82 (1.75-58.91)	0.158
PLR	262.1 (70.23-1507.14)	275.35 (2.34-1487.88)	0.894
MLR	0.65 (0.1-1.86)	0.69 (0.11-3.18)	0.478
CRP (mg/dL)	87.5 (0.8-238.5)	111.15 (7.7-289)	0.075
Albumin (g/dL)	3.2 (0-36.4)	3.1 (1.3-39.1)	0.80

Values are presented as median (minimum-maximum). ARB: Acid-resistant bacilli; NLR: Neutrophil/lymphocyte ratio; PLR: Platelet/lymphocyte ratio; MLR: Monocyte/lymphocyte ratio; CRP: C-reactive protein

PTB. Proinflammatory cytokines such as IFN-γ, TNF-α, and IL-17A have been reported to be significantly elevated in PTB patients with bilateral and cavitary involvement compared with individuals with latent infection and healthy controls [14].

The release of cytokines into the bloodstream leads to increased levels of acute-phase proteins and inflammatory markers. During the inflammatory response, TNF is released early and promotes lymphocyte apoptosis [14]. Macrophages and CD4+ T lymphocytes play central roles in TB pathogenesis through the development of cell-mediated immunity. Neutrophils, the most abundant subtype of white blood cells, are key components of the innate immune system [15]. In conditions such as trauma, surgery, bacteremia, and sepsis, peripheral neutrophil counts increase while lymphocyte counts decrease. Previous studies have demonstrated that NLR and PLR are associated with disease severity and prognosis in nosocomial pneumonia and sepsis [16,17]. Elevated NLR and PLR have also been identified as early predictive markers of mycobacterial infection in patients with chronic obstructive pulmonary disease (COPD) complicated by PTB [18]. Furthermore, NLR has been shown to be higher in TB patients than in those with sarcoidosis and may serve as a useful marker for differentiating between these two conditions [19]. Consistent with our findings, NLR, PLR, CRP, and platelet count have previously been reported as potential biomarkers of disease severity [20,21].

In the bone marrow, proinflammatory cytokines stimulate the differentiation of mononuclear phagocytic cells (monocytes) and their release into the circulation. Monocytes have a relatively long lifespan in the bloodstream and subsequently differentiate into tissue macrophages, playing a crucial role in the host response to *M. tuberculosis*. Absolute monocyte count and MLR have been identified as predictive factors for active TB in HIV-infected children [22,23]. La Manna et al. [24] reported that MLR, markedly elevated during active infection, decreased with treatment, and that both absolute monocyte count and MLR were significantly higher in patients with active TB compared with those with latent infection and healthy controls.

The reduction in lymphocyte count observed in PTB and the association between elevated MLR and disease severity are consistent with the relationship between systemic inflammation and disease progression. Although CBC-derived parameters were associated with disease severity in our study, these markers are nonspecific and may also be elevated in other chronic lung diseases, including sarcoidosis, COPD, and lung cancer [25–27]. Therefore, further research is required before these parameters can be reliably used to predict disease severity and mortality in PTB rather than serving solely as supportive diagnostic indicators.

High-resolution computed tomography findings, such as diffuse infiltration and cavity density, have been associated with bacterial burden in PTB patients. Individuals with multiple cavitary lesions and a high bacillary load are at increased risk of drug resistance and may require prolonged treatment, potentially resulting in higher morbidity and mortality. However, a meta-analysis evaluating factors associated with hospital mortality in TB patients concluded

that ARB positivity on microscopic examination was not associated with mortality [28].

Unsal et al. [21] demonstrated that PTB patients had elevated levels of IL-6, a hematopoietic cytokine associated with more diffuse lung involvement and ARB positivity. In contrast, no significant association was observed between bacterial burden and radiologic disease extent in our study. Additionally, we found no significant relationships between inflammatory blood markers and bacterial burden, cavitary disease, or mortality. The retrospective design of the study may have introduced limitations, including incomplete data and variability in sputum quality and microscopic evaluation, which may have influenced smear grading accuracy.

The reported hospital mortality rate among PTB patients ranges from 2% to 12% [29]. Mortality is influenced by multiple factors, and our findings are consistent with previous studies identifying low albumin levels as a significant predictor of mortality. In our study, CRP and NLR were significantly higher in non-survivors than in survivors. The mortality rate in our cohort was lower than that reported in the literature. Using an NLR cut-off value of 7.15, the sensitivity and specificity for predicting mortality were 63.7% and 77.1%, respectively. With a CRP cut-off value of 73.5, sensitivity and specificity were 70.4% and 69.4%, respectively. Neither NLR nor CRP demonstrated clear superiority in predicting mortality. Because only a small number of patients died (n=9), multivariate analysis was not feasible. Although these findings appear meaningful, they should be validated in larger cohorts with a greater number of mortality events.

Although patients with immunosuppressive conditions (e.g., active cancer, hematologic disease, steroid use, and HIV infection) were excluded, individuals with diabetes mellitus, asthma-COPD, and hypertension were included. Multivariate analysis suggested that comorbidities did not significantly affect laboratory parameters. However, concomitant infections could not be excluded due to the retrospective nature of the study, representing a key limitation. Another limitation is that sputum samples were collected at different time points, which may have influenced smear grading results.

Conclusion

In conclusion, in patients with untreated pulmonary tuberculosis, NLR, PLR, and MLR—readily obtained from CBC analysis—may serve as markers reflecting radiologic disease extent. However, these inflammatory markers were not significantly associated with bacterial burden. Moreover, their effectiveness in predicting mortality in PTB patients has not been definitively established. Larger prospective studies including more mortality cases are needed to clarify their prognostic value.

Ethics Committee Approval: This study was approved by the Istanbul Training and Research Hospital Clinical Research Ethics Committee (Decision: 2064; Date: 6.12.2019).

Informed Consent: We obtained written informed consent from all participants.

Conflict of Interest: The authors declare that there is no conflict of interest.

Financial Disclosure: The authors declared that this study has received no financial support.

Use of AI for Writing Assistance: None declared.

Authorship Contributions: Concept: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.; Design: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.; Supervision: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.; Materials: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.; Data collection and/or processing: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.; Analysis and/or interpretation: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.; Literature review: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.; Writing: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.; Critical review: G.G., N.Ş.V., B.A.B., E.N.A.S., P.P., E.B., E.C.S., M. G.O., S.A.

Peer-review: Externally peer-reviewed.

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