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Association of Periodontitis with White Blood Cell and Platelet Count: A Cross-Sectional Study

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Abstract

Background and Aim: Inflammatory diseases can lead to reactive thrombocytosis, an increase in platelet count associated with cardiovascular risks. Periodontal disease has been linked to systemic inflammatory responses. This study aimed to investigate the relationship between periodontitis and platelet and white blood cell (WBC) counts.
Materials and Methods: This cross-sectional study included 630 participants from a health monitoring program. Periodontal status was assessed using the Community Periodontal Index of Treatment Needs (CPITN) and periodontal pocket depth (PPD) with a Williams probe. Blood parameters, including WBC and platelet counts, were measured using a complete blood count (CBC) test. Statistical analysis was performed using SPSS (version 22), with a significance level of 5%.
Results: The participants included 360 women (57.1%) and 270 men (42.9%), with a mean age of 43.41 ± 7.01 years. No significant difference was observed in WBC counts among healthy individuals and those with mild or severe periodontitis ($P = 0.774$). However, there was a significant difference in the gingival index (GI) between the three groups ($P < 0.001$). WBC count was positively correlated with platelet count ($P < 0.001$) but not with CPITN. Additionally, GI and CPITN showed a significant positive correlation ($P < 0.001$).
Conclusion: This study found no significant association between periodontitis and WBC or platelet counts, suggesting that periodontitis may not have a direct impact on these hematologic parameters.

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Introduction

Under certain conditions, such as prolonged poor hygiene, the mouth bacteria weaken the immune system and can cause an inflammatory reaction called periodontitis which is an irreversible disease affecting the gum tissue. [1] Periodontitis is known to be the most common infectious disease in humans with a bacterial origin and can eventually lead to the loss of the connection between periodontal fibers and alveolar bone. [2] According to statistics, 5% to 15% of adults suffer from severe periodontitis. [3]

The penetration of bacteria in the form of a biofilm complex into the gingival sulcus triggers immune cells and results in periodontal inflammation. [4] As a result of tissue chain reactions, various chemical mediators are released, including cytokines and chemokines, which eventually lead to periodontal tissue loss and tissue destruction. [1] The most important symptom of

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periodontitis is the destruction of periodontal tissue, which is the result of the host immune response. [5,6]

Studies have revealed that periodontitis can have a variety of systemic implications in addition to triggering a local inflammatory response. Recent studies have raised the possibility of an association between coronary artery disease and periodontal disease. [7,8] Atherosclerosis and coronary artery disease are linked to periodontal diseases, but the exact process is still unclear. It has been proposed that periodontal infections are more likely to stimulate platelets and leukocytes, [9] and that activated platelets and leukocytes might be a factor in enhanced atherothrombotic activity. [7,10-13] According to studies, [7, 10] people with periodontitis have higher levels of White Blood Count (WBC) and C-reactive protein than people without the condition. Additionally, periodontitis has been linked to higher levels of platelet activation and plasma fibrinogen, which may lead to a pro-coagulant condition and a higher risk of atherosclerosis and cardiovascular disease. [11,12] D'Aiuto et al. showed that controlling periodontitis considerably reduced serum mediators and indicators of acute phase response, indicating that the control of periodontitis could be helpful in the management of atherosclerosis. [13]

The possibility of atheroma plaque formation is increased when platelets and leukocytes that have been stimulated during bacteremia excite additional cells. In order to attach to leukocytes and endothelial cells, activated platelets release pro-inflammatory mediators, which expose pro-inflammatory receptors. [14] It has been suggested that in inflammatory lesions, active platelets control the release of chemokines by monocytes. [15] Platelets are crucial agents in the thrombotic and inflammatory processes due to these capabilities, and platelet activation has been linked to atherosclerosis and coronary artery disease. [14]

As an infection-driven inflammatory disease, periodontitis could lead to a reactive increase in platelet and white blood cell count. This mechanism could partially mediate the well-documented association between periodontitis and atherosclerotic cardiovascular disease. Therefore, this cross-sectional study aimed to test the possible association between periodontitis and platelet-white blood cell count in a representative sample.

Materials and Methods

This cross-sectional study was performed on 630 people who were visited within the health monitoring scheme from June 2020 to February 2023 at Mashhad University of Medical Sciences in collaboration with the cohort center of the university. The inclusion criteria were participation in the university cohort plan, being aged 19-65 years, and cooperation in further studies and follow-up. The exclusion criteria were having (fixed and removable) orthodontics, pregnancy, smoking, using anti-inflammatory drugs, and taking anticoagulants. The confounding variables in the study included diabetes and smoking. To prevent confounding, the data of smokers and diabetics were excluded from the data analysis.

In patients with bleeding on probing (BOP), the periodontal pocket depth (PPD), clinical attachment loss (CAL) for Community Periodontal Index of Treatment Needs (CPITN), and the gingival index (GI) were measured and extracted from patients' medical records. In order to minimize interexaminer variation, all clinical periodontal examinations were performed by an experienced periodontist. In the gingival index, normal gums, gums with slight changes, discoloration, and local edema, bleeding gums during probing, and gums with moderate changes, spontaneous bleeding, and severe swelling and discoloration were scored 0, 1, 2, and 3, respectively. [16] CPITN was scored 0 if there was no symptom, 1 if the depth of the PPD was less than 3.5 mm and there was only bleeding, 2 if PPD was less than 3.5 mm and bleeding and subgingival mass were also observed, 3 if PPD was 3.5 to 5.5 mm, and 4 if PPD was greater than 5.5 mm. Using the CPITN scores, the participants were divided into three groups: healthy (individuals with a score of 0 and 1), mild periodontitis (individuals with a score of 2), and severe periodontitis (individuals with a score of 3-4).

PPD was extracted from patients' medical records. The teeth examined for this index were teeth 1 and 6 on the right maxilla, tooth 6 on the left upper mandible, tooth 6 on the right lower mandible, teeth 1 and 6 on the left lower mandible, and tooth 7 in people under 20 years of age. [17] In the next step, blood factors including platelet count examined by CBC test were extracted from patients' medical records. Finally, the chance of platelet count increased and its odds ratio was reported. The odds ratio of greater than 1 (OR>1) indicates a positive relationship between periodontitis and platelet count and a higher chance of increasing the platelet count in people with periodontitis. Moreover, the odds ratio of smaller than 1 (OR<1) indicates the protective effect of periodontitis on platelet count.

Statistical analysis

The data were described using measures of dispersion and central tendency and frequency distribution. Data analysis was also performed using the Spearman correlation coefficient, t-test, and chi-square test. All statistical analyses were performed at the significance level of 0.05 (P= 0.05).

Ethical considerations

We followed all guidelines under the Declaration of Helsinki (DoH) in this cross-sectional study. The researchers declared their commitment to keep the patients' data confidential and use them only for research purposes. This research project was registered on July 8, 2020 in the Organizational Ethics Committee of the School of Dentistry of Mashhad University of Medical Sciences and was approved with the ethics code IR.MUMS.DENTISTRY.REC.1399.046.

Results

In this study, the medical files of 630 persons including 360 women (57.1%) and 270 men (42.9%) with a mean age of 43.41±7.01 years and the age range of 34-62 years were examined for WBCs, platelet count, the gingival index (GI), and CPITN. Since all the research variables had an abnormal distribution, they were analyzed using related statistical tests. Analysis of the participants' data indicated that 516 (81.9%), 46 (7.3%), and 68 patients (10.8%) were in the healthy group, mild periodontitis group, and severe periodontitis group, respectively. The mean age of participants in the healthy group, the mild periodontitis group, and the severe periodontitis group were 43.27±6.93 years, 43.65±7.41 years, and 44.31±7.41 years, respectively. There was no significant difference between the study groups in terms of the mean age (P=0.589) (Table 1).

Table 1: Descriptive statistics of the participants' demographic characteristics in the three study groups

Groups	Gender		Age (Mean±SD)	Age range
	Females	Males		
Healthy (n=516)	288 (55.8%)	228 (44.2%)	43.27±6.93	35-69
Mild periodontitis (n = 46)	32 (69.8%)	14 (30.4%)	43.65±7.41	35-62
Severe periodontitis (n = 68)	40 (58.8%)	28 (41.2%)	44.31±7.41	34-60
Total (n=630)	360 (57.1%)	270 (42.9%)	43.41±7.01	34-62
Test results	X^2 = 0.187	P = 0.187	X^2 =1.06	X^2 =1.06
			P = 0.589	P=0.589

The number (percentage) of male patients in the healthy, mild periodontitis, and severe periodontitis groups were 228 (44.2%), 14 (30.4%), and 28 (41.2%), respectively. Overall, the number of men and women was not significantly different in the three study groups (P=0.18). Moreover, the participants were homogenous in terms of age and gender in the three study groups (Table 1).

As indicated in Table 2, the lowest and highest WBC values were observed in the healthy group. The lowest and highest WBC ranges (the difference between the maximum and minimum values) were reported in the mild periodontitis and healthy groups, respectively. Moreover, the mean±SD WBC count (per 103 cells) in the healthy, mild periodontitis, and severe periodontitis groups were 6±1.40, 6.01±1.37, and 6.20±1.51, respectively. The lowest and highest PLT values were observed in the healthy and mild periodontitis groups, respectively. In addition, the lowest and highest PLT ranges were observed in the mild periodontitis and healthy groups, respectively. The mean±SD PLT count (µL) in the healthy, mild periodontitis, and severe periodontitis groups were 225.18±51.19, 231.91±52.92, and 226.74±64.20, respectively (Table 2):

Table 2: Comparison of WBC and PLT in the three study groups

Variable	Group	Number	Mean	SD	Minimum	Maximum	Median	Kruskal-Wallis
WBC[1]	Healthy	516	6.00	1.40	2.2	12.5	5.85	X^2 =0.51
	Mild periodontitis	46	6.01	1.37	3.8	10.5	5.70	P= 0.774
	Severe periodontitis	68	6.20	1,51	4.0	11.9	5.90	
PLT[2]	Healthy	516	225.18	51.90	98.0	522.0	222.00	X^2 =0.70
	Mild periodontitis	46	231.91	52.92	138.0	389.0	225.50	P = 0.704
	Severe periodontitis	68	226.74	64.20	137.0	534.0	221.00	

The data presented in Table 3 suggest that the mild GI was the most frequent in all three groups followed by zero GI in the healthy and mild periodontitis groups. However, moderate GI was observed in the severe periodontitis group. In general, in the severe periodontitis group, the GI value was higher compared to the other two study groups. The GI distribution was statistically significant among the three study groups (P<0.001, Table 3).

[1] White Blood Cell

[2] Platelets

Table 3: Comparison of GI distribution in the three study groups

Group		GI (score)				Total	Kendall's tau-b (τb) correlation
		None (0)	Mild (1)	Moderate (2)	Severe (3)		
Healthy	Number	231	239	39	7	**516**	Tb= 0.21
	%	44.8	46.3	7.6	1.4	100	P<0.001
Mild periodontitis	Number	19	22	5	0	46	
	%	41.3	47.8	10.9	0	100	
Severe periodontitis	Number	9	33	20	6	68	
	%	13.2	48.5	29.4	8.8	100	
Total	Number	259	294	64	13	630	
	%	41.1	46.7	10.2	2.1	100	

In the healthy group, age was not significantly correlated with any of the variables, and WBC had a positive significant relationship with PLT, but was not significantly correlated with GI. In addition, PLT was not significantly related to GI. In the mild periodontitis group, age was negatively and significantly associated with PLT (P= 0.026), but it was not significantly correlated with either WBC or GI. WBC was not significantly associated with PLT and GI. Similarly, PLT was not significantly correlated with GI. In the severe periodontitis group, age was not significantly correlated with any of the variables. However, WBC was directly and significantly correlated with PLT (P= 0.010). In contrast, WBC and PLT were not significantly related to GI as indicated in Table 4.

Table 4: The correlations between the study variables

Groups	Variables	Indicators	WBC	PLT	GI[3]
Healthy	Age	Spearman correlation	-0.050	-0.049	-0.008
		P-value	0.255	0.268	0.851
	WBC	Spearman correlation	1.000	0.263**	-0.084
		P-value		0.000	0.057
	PLT	Spearman correlation		1.000	-0.031
		P-value			0.483
Mild periodontitis	Age	Spearman correlation	-0.155	-0.328*	0.198
		P-value	0.304	0.026	0.187
	WBC	Spearman correlation		0.285	0.137
		P-value		0.054	0.363
	PLT	Spearman correlation		46	-0.016
		P-value			0.918
Severe periodontitis	Age	Spearman correlation	0.055	-0.130	0.057
		P-value	0.655	0.292	0.645
	WBC	Spearman correlation		0.310*	0.108
		P-value		0.010	0.380
	PLT	Spearman correlation			0.105
		P-value			0.394

**: P<0.0; * P<0.05

As indicated in Table 5, age was not significantly correlated with any of the variables. WBC was directly and significantly correlated with PLT (P<0.001), but was not significantly correlated with CPITN and GI. Moreover, PLT was not significantly associated with CPITN and GI. CPITN had a direct and significant relationship with GI (Table 5):

Discussion

Blood platelets and WBC in patients with periodontitis and healthy individuals were measured in the present study. The obtained results showed no significant relationship between periodontitis and platelet count. Similarly, Kumar et al. found no significant correlation between periodontitis and platelet count. [18] Furthermore, Iqbal et al. found no significant difference in platelet count between normal individuals and patients with invasive periodontitis, and platelet count was slightly higher in healthy individuals. [19] The result of another study showed no statistically significant difference in terms of platelet count (226.93 ±50.90) in healthy and periodontitis (214.60±51.72)

[3] Gingival Index

groups. [20] However, Wang et al. reported that platelet count was significantly lower in patients with periodontitis compared to healthy individuals. [21]

Contrary to the results of the present study, Al-Rasheed showed a significant relationship between periodontitis and platelet count. [22] Sharma et al. found that people with chronic periodontitis had higher platelet counts than healthy people and people with acute periodontitis, but there was no significant difference between healthy individuals and patients with acute periodontitis. [23] Romandini et al. reported that periodontitis resulted in a significant increase in platelet count in affected patients compared to healthy individuals. [24]

Table 5: The relationships between the variables

Variables	Indicators	WBC	PLT	CPITN	GI
Age	Spearman correlation	-0.075	-0.048	0.039	0.024
	P-value	0.061	0.231	0.332	0.543
WBC	Spearman correlation	1.000	0.271**	0.002	-0.030
	P-value		<0.001	0.965	0.457
PLT	Spearman correlation		1.000	0.021	-0.043
	P-value			0.598	0.276
CPITN[4]	Spearman correlation			1.000	0.226**
	P-value				<0.001

**: P<0.0; * P<0.05

The findings of the present study showed no significant relationship between different study groups in terms of WBC. Similarly, Iqbal found no significant difference between patients with invasive periodontitis and healthy individuals in terms of the mean number of WBCs. [19] Moreover, in another study, researchers observed no significant difference between patients with periodontitis and healthy individuals in terms of WBC. [25] Consistently, Romandini et al. found no significant difference between different groups of patients with mild and severe periodontitis and healthy individuals in terms of WBC. [24]

Contrary to the results obtained in the present study, al-Rashhed reported that people with chronic periodontitis had significantly higher WBCs than healthy people. [22] Furthermore, Kumar showed that the mean WBC was significantly higher in two groups of patients with periodontitis aged 36-40 and 46-50 years compared to healthy individuals, but WBC was not significantly different between the patients aged 30-35 years and the patients aged 41-45 years. [18] In another study, Sharma showed that WBC was higher in patients with chronic acute periodontitis compared to those with acute periodontitis healthy individuals, but was significantly lower in patients with acute periodontitis compared to healthy individuals. [23] Therefore, further studies are needed to comment on the relationship between periodontitis and increased WBC. The number of WBCs is affected by several factors and is also more likely to increase in people with chronic periodontitis since an increase in WBC is characteristic of chronic infections. Similar studies have addressed this issue with much smaller sample sizes and in different populations. [21, 23]

It seems that WBC and blood platelets count increase in the presence of systemic infections. In the studies conducted by Al-Rsheed and A. Sharma, the platelet count was higher in people with chronic periodontitis. [22,23] However, studies with a larger sample size in different populations are needed to prove this. The results of the study conducted by Romandini et al. on 5197 people showed that periodontitis can independently lead to an increase in platelets, but the researchers stated that this increase can be affected by several factors, including the person's weight, gender, and smoking status. [24]

Cardiovascular disease and periodontitis have many common risk factors, which is why these conditions have been considered more frequently by researchers in recent years. There are several reasons for the connection between these two diseases, but the exact mechanism remains unresolved. Some studies have shown that periodontal bacteria or their products can enter the circulatory system and increase the risk of systemic diseases. [26] An increase or decrease in the number of white blood cells or inflammatory proteins in the peripheral blood can be indicative of a systemic infection in the body. This increase in WBC may also be observed in people with periodontitis. [27] It seems that WBC and platelets are likely to increase systemic infections. Al-Rashhed and Sharma showed that platelet counts were higher in people with chronic periodontitis. [22, 23] A study performed by Romandini on 5,197 people showed that periodontitis alone could lead to an increase in platelets. However, the researchers

[4] Community Periodontal Index of Treatment Needs

suggested that this increase could be affected by several factors, including the person's weight, gender, and smoking status. [24] Thrombosis secondary to infections and infectious diseases is a physiological mechanism. In periodontitis, bacterial products and cytokines released from the immune system can enter the bloodstream and eventually lead to a low-grade infectious condition in the body. [28] The increase in secondary systemic infection relative to periodontal infections is characterized by an increase in biomarkers such as IL-6, IL-1, CRP, and TNF-a. [29] Increased IL-6 can eventually lead to increased platelet production by the liver, which in turn leads to increased platelet count. However, it is not clear at what stage increased platelet counts can be observed in periodontitis.

It can take a long time from the onset of periodontal disease to the release of cytokines and bacterial products into the bloodstream. However, the time varies from person to person and population to population and can be influenced by many factors such as weight, smoking habits, alcohol consumption, and genetics. [29] The results of this study showed that an increase in the number of WBCs had a direct and significant relationship with increased platelet count. It should be noted that since WBC and platelets are both markers of systemic infections, increased secretion of cytokines and inflammatory factors can affect both. [30]

Conclusion

The findings of the present study indicated that periodontitis was not associated with a significant change in the number of WBCs. Moreover, there was no association between periodontitis and platelet count compared to healthy individuals. In all groups, age had no significant correlation with any of the variables. Furthermore, the increase in the number of WBC had a direct and significant relationship with the increase in platelets. Eventually, since increasing of WBC and platelets are both markers of systemic infections, an increase in the secretion of cytokines and inflammatory factors can have an effect on the increase of both.

Limitations of the study

One of the limitations of the present study was that we did not measure various indicators such as platelet size, CRP, blood albumin, and blood globin in addition to WBC and platelets due to the numerous risk factors of this disease which could be effective to be identified.

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Conflict of interest

The authors declared no conflict of interest.

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