Does periodontitis affect mean platelet volume(MPV) and plateletcrit (PCT) levels in healthy adults?



Assistant Prof. Bolu Abant İzzet Baysal University, Dentistry Faculty, Department of Periodontology, Bolu, Turkey.
 Specialist Bolu Abant İzzet Baysal University, Medical Faculty, Department of Cardiology, Bolu, Turkey.

http://dx.doi.org/10.1590/1806-9282.66.2.133

SUMMARY

OBJECTIVE: Periodontitis may stimulate infectious and immune response and cause the development of atherogenesis, coronary heart disease, and myocardial infarction. The aim of this study was to compare the plateletcrit (PCT) and mean platelet volume (MPV) levels derived from complete blood count (CBC) tests in patients suffering from stage 3 periodontitis with those of healthy individuals without periodontal disease.

METHODS: The study included 57 patients (28 females and 29 males) with Stage 3 Periodontitis and 57 volunteering individuals (31 females and 26 males) who were periodontally healthy. The age of study participants ranged from 18 to 50 years. Their periodontal condition was investigated with probing depth (PD), clinical attachment level, bleeding on probing, and plaque index. Leukocyte (WBC) and erythrocyte count (RBC), hemoglobin (Hb) and hematocrit (HCT) levels, mean corpuscular volume (MCV) and red cell distribution width (RDW), thrombocyte count, mean platelet volume (MPV), plateletcrit (PCT), and neutrophil and lymphocyte counts were evaluated based on the CBC test results of the study participants.

RESULTS: PCT, WBC, Neutrophil, and MPV values were found to be significantly higher in the periodontitis group (p<0.05). There were no significant differences in RBC counts, Hb, HCT, MCV, RDW, and platelet and lymphocyte counts between the two study groups (p>0.05).

CONCLUSIONS: PCT and MPV levels may be a more useful marker to determine an increased thrombotic state and inflammatory response in periodontal diseases.

KEYWORDS: Blood cell count. Cardiovascular diseases. Inflammation. Leukocyte count. Periodontitis. Blood platelets. Risk factors.

INTRODUCTION

Periodontitis is an inflammatory disease of chronic nature in the supporting tissues of the teeth. More than one factor is involved in its etiology; however, biofilms of dysbiotic plaques are the main cause of the disease. During the course of periodontitis, the supporting tissues of the teeth are progressively destructed. The harmful effects of periodontitis are not only restricted to the oral cavity but also have an impact on the general health status of the individual. Microorganisms and/or their products and the inflammatory mediators can access the systemic circulation through the ulcerated pocket epithelium and initiate

DATE OF SUBMISSION: 28-Aug-2019 DATE OF ACCEPTANCE: 01-Sep-2019

CORRESPONDING AUTHOR: Mehmet Inanir

Bolu Abant İzzet Baysal University, Medical Faculty, Department of Cardiology 14300, Bolu, Turkey. Tel: 5437941435 Fax: 0374 2546600 E-mail: mdmehmetinanir@yahoo.com

a systemic acute-phase response by activating the immune system. Periodontitis may cause bacteremia, endotoxemia, and low-grade systemic inflammation, and it is potentially associated with the impairment of well-being by causing, among others, cardiovascular diseases (CVDs), adverse pregnancy outcomes, respiratory system diseases, and diabetes mellitus².

Periodontal inflammation can exacerbate systemic conditions through the pathological changes caused by leukocytes. Leukocytes, especially neutrophils, produce a number of specific molecules directly responsible for the inflammatory response, which can be a risk factor for atherosclerosis and cardiovascular complications³. The other major component of the blood is the platelets, which are closely associated with inflammation. When they are activated, pro-inflammatory mediators are released, and pro-inflammatory receptors are exposed. This, in turn, causes platelets to bind to WBC and endothelial cells. Pathogens existing in the periodontal tissues may readily stimulate platelets and WBC, and this activation might be involved in aggravating atherothrombosis⁴.

Several studies in the literature have reported that high levels of systemic inflammation markers were detected in periodontitis compared to healthy controls. Among these, total white blood cells (WBC), neutrophils, lymphocytes, serum globulin, C-reactive protein, and platelets were listed⁵. Furthermore, it has been suggested that elevated levels of these markers, particularly high leukocyte counts and high levels of C-reactive protein, as well as increased platelet activation, can help establish the relationship between periodontitis and cardiovascular diseases (CVD)⁶.

Complete blood count (CBC) tests are commonly used in clinical practice. Plateletcrit (PCT), mean platelet volume (MPV), and platelet distribution width (PDW) are indices specific to platelet morphology and proliferation kinetics, and these parameters can be derived from CBC7. In the literature, white blood cell (WBC) and platelet counts, PDW, and MPV have been used in several studies to investigate the association between periodontal disease and CVD; however, the results were contradictory. As far as we have observed, the association of PCT with periodontal diseases has not been investigated yet. PCT is an index providing information on the total platelet mass. The following formula is used to calculate PCT: PCT = Platelet count X MPV/100008. The levels of PCT normally vary in range from 0.22% to 0.24%. Therefore, assessing the PCT value can give us more accurate information about inflammation and increased thrombogenic events⁸.

In this study, we aimed to investigate PCT levels in the CBC of patients suffering from stage 3 periodontitis as compared to those of healthy individuals without periodontal disease.

METHODS

Approval by the Clinical Research Ethics Committee of Bolu Abant İzzet Baysal University was obtained prior to the conduct of the study. The study was conducted in compliance with ethical standards according to the current version of the Declaration of Helsinki. After informing the eligible volunteers of the objective and procedures of the study, written consent was obtained from the subjects to enroll them in the study. The sample size was calculated considering Type I errors (0.05), targeted power (0.80), and effect size(0.50) due to the PCT value (p<0.05). The minimum sample size required was calculated as 51.

The study participants comprised 57 patients (28 females and 29 males) with Stage 3 Periodontitis and 57 periodontally healthy subjects (31 females and 26 males). The age range of the participants was between 18 and 50 years. Since individuals older than 50 years might have already developed atherosclerotic processes or might have comorbid diseases, they were excluded from the study as these potential conditions could interfere with the results of the complete blood count tests. Patients were diagnosed with Stage 3 Periodontitis or were determined to be periodontally healthy based on the criteria proposed by the International Workshop for Classification of Periodontal Diseases and Conditions in 2017¹. The study was conducted at the Department of Periodontology between July 2018 and December 2018.

Inclusion and exclusion criteria

Patients were included in the study if they were systemically healthy and found to have a probing depth (PD) of ≥ 6 mm and interdental clinical attachment level (CAL) of ≥ 5 mm, if they had tooth loss of ≤ 4 teeth due to periodontitis, and if they had radiographically detected bone loss reaching the mid-third of the root and beyond. Periodontally healthy subjects were included in the study if they had no radiographically detected bone loss and if they had no sites with clinical attachment loss and no sites with probing depth (PD) of >3 mm in their oral cavity.

Individuals were excluded if they had a history of cardiovascular disease, diabetes mellitus, hypertension, upper respiratory tract infections, smoking, hypo/hyperthyroidism, chronic renal failure, malignancy, any hematological abnormalities, or any medication use such as antiplatelet agents, anticoagulants, antihyperlipidemic, angiotensin-converting enzyme inhibitors, and steroids. Individuals who had been treated for periodontitis in the past 6 months were also excluded.

Clinical examination

All clinical parameters were evaluated by a single experienced periodontist (G.U), and a calibration exercise was performed to obtain acceptable interexaminer reproducibility. Periodontal examinations were performed with a Williams probe (Hu-Friedy, Chicago, IL, USA). The clinical parameters of PD, plaque index (PI), and clinical attachment level (CAL) were measured for every tooth present in the oral cavity. The measurements were performed at six sites (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, distolingual), and the results were recorded in approximation to the nearest whole millimeter. The distance from the bottom of the pocket to the cementoenamel junction was defined as CAL, which was measured and recorded. The mean PD and the mean CAL values were calculated by dividing the total score of all teeth by the total number of teeth examined during the study. The periodontal probe was carefully and gently introduced into the gingival sulcus to calculate the percentage of BOP, even one site with BOP was recorded as (+) for each individual tooth.

Blood examinations

CBC and platelet volumes were tested simultaneously with optical and impedance measurements (Cell Dyn 3700; Abbott Diagnostics, Lake Forest, Illinois, USA). Platelet count, the levels of hemoglobin (Hb), hematocrit (HCT), red blood cells (RBC), red cell distribution width (RDW), mean corpuscular volume (MCV), neutrophil and lymphocyte counts, total number of WBC, MPV, and PCT were recorded for each patient.

STATISTICAL ANALYSIS

Statistical analyses were conducted with the SPSS software (SPSS 20.0 for Windows, IBM Co, Chicago, IL, USA). The distribution of the variables in study

groups was analyzed by the Kolmogorov-Smirnov test. Normally distributed variables were compared by the t-test, and the results were expressed as mean ± standard deviation. Variables that did not conform to a normal distribution were compared with the Mann-Whitney U test. The chi-square test was used for comparing nonparametric variables between the study groups. A p-value, lower than 0.05, was accepted to indicate a statistical significance.

RESULTS

There were no significant differences in age and gender between the study groups (p>0.05). PD and CAL were found to be statistically different between the groups (p<0.001) (Table 1).

TABLE 1. GENERAL CHARACTERISTICS OF THE STUDY GROUPS

Baseline characteristics	Stage III Periodontitis Mean± SD (n=57)	Control Mean± SD (n=57)	p
Age (years)	37.4±7.0	35.6±7.0	NS
Male/female	29/28	26/31	NS
PD(mm)	6.10±0.72	1.89±0.42	<0.001
CAL(mm)	6.21±0.81	1.85±0.40	<0.001
BOP(%)	63±15	14±4	<0.001
PI	82±17	15±5	<0.001

SD:Standard deviation, PD: Probing Depth, CAL: Clinical attachment level, BOP: Bleeding on probing, Pl: Plaque index

TABLE 2. LABORATORY DATA OF STUDY GROUPS

Periodontitis Mean± SD (n=57)	Control Mean± SD (n=57)	р
8.02±2.68	6.87±1.30	0.004
4.87±0.56	4.96±0.58	0.403
13.92±1.76	14.29±1.69	0.258
41.78±4.94	42.67±4.83	0.337
85.82±6.50	86.17±5.19	0.748
14.80±1.88	15.20±1.51	0.212
258.52±51.11	240.48±54.58	0.073
8.75±1.32	8.22±1.08	0.021
0.223±0.04	0.196±0.04	0.001
4.79±2.36	3.99±1.08	0.021
2.46±1.14	2.14±0.56	0.060
	Mean± SD (n=57) 8.02±2.68 4.87±0.56 13.92±1.76 41.78±4.94 85.82±6.50 14.80±1.88 258.52±51.11 8.75±1.32 0.223±0.04 4.79±2.36	Mean± SD (n=57) Mean± SD (n=57) 8.02±2.68 6.87±1.30 4.87±0.56 4.96±0.58 13.92±1.76 14.29±1.69 41.78±4.94 42.67±4.83 85.82±6.50 86.17±5.19 14.80±1.88 15.20±1.51 258.52±51.11 240.48±54.58 8.75±1.32 8.22±1.08 0.223±0.04 0.196±0.04 4.79±2.36 3.99±1.08

SD:Standard deviation., WBC: White blood cells, RBC: Red blood cells, MCV: Mean Cell Volume, RDW: Red cell distribution width, MPV: Mean platelet volume, PCT: Plateletcrit.

PCT was found to be higher in the periodontitis group, and this difference was statistically significantly higher (p=0.001) (Table 2). The periodontitis group was found to have statistically significant higher levels of WBC count and MPV compared to the control group (p=0.004, p=0.021; respectively). PCT, MPV, and WBC distribution of the control and periodontitis group are shown in Figure-1. There were no statistically significant differences between the two groups regarding the other parameters investigated in the study, including the RBC count, HB and HCT levels, MCV, and RDW (p>0.05)(Table 2). The correlations between the parameters were also tested in the two groups (N=114). In Table 3, the statistically significant correlations between the parameters investigated are shown.

DISCUSSION

Recent studies demonstrated that periodontitis and systemic diseases such as cardiovascular diseases and diabetes were strongly related. Aggravated systemic inflammatory responses and reactions to maintain homeostasis may be the key factors to provide insight into the relationship between periodontal disease and systemic conditions⁹.

It is known that infections increase WBC and neutrophil counts, and it has been proposed that these increases might be linking infections with systemic diseases, including CVDs¹⁰. In our study, an increase in the WBC and neutrophil counts were detected in the periodontitis group compared to the healthy controls. The presence of a high number of leukocytes in the systemic circulation makes the blood more viscous, facilitating the adherence of the circulating cells to the endothelial lining of the blood vessels. This latter change also increases the viscosity of the blood. Decreased blood flow might be involved in the development of CVD. Similar to our results, Kumar et al. 11 reported that patients suffering from periodontitis had high WBC counts compared to controls, and the differences were statistically significant. Conversely, the study by Rao et al. 12 showed no statistically significant differences in WBC count between both groups.

Platelets play a crucial role in managing vascular integrity and regulating hemostasis, and they are involved in the fundamental biological process of chronic inflammation associated with disease pathology, thrombosis, and atherogenesis¹³. Platelet activity can be evaluated with platelet indices such as MPV, platelet counts, PDW, and PCT. Previous studies have

P = 0.001P=0.004 P=0.021 0.4 10 10 0.35 0,3 0,25 0,2 0,15 0,1 0,05 WBC ■ Control Periodontitis Period ontitis

FIGURE 1. PCT, WBC AND MPW DISTRIBUTIONS OF CONTROL AND PERIDONTITIS GROUPS

MPV: Mean platelet volume, PCT: Plateletcrit, WBC: White blood cells

TABLE 3. THE STATISTICALLY SIGNIFICANT CORRELATIONS BETWEEN BLOOD COUNT PARAMETERS AND CLINICAL PERIODONTAL PARAMETERS

	WBC	WBC		MPV		PCT		Neutrophil	
	r	P value	r	P value	r	P value	r	P value	
Mean PD	0.221*	0.019	0.253**	0.007	0.344**	p<0.001	0.202*	0.032	
Mean CAL	0.237*	0.012	0.251**	0.007	0.344**	p<0.001	0.221*	0.019	
ВОР	0.210*	0.026	0.271**	0.004	0.359**	p<0.001	0.195*	0.040	

Correlation analysis was performed using Pearson's correlation analyses. ** Correlation is significant at the 0.01 level. *Correlation is significant at the 0.05 level. MPV: Mean platelet volume, PCT: Plateletcrit, WBC: White blood cells. PD: Probing Depth, CAL: Clinical attachment level, BOP: Bleeding on probing

shown that platelet counts increase in cardiovascular diseases and vascular complications^{14,15}. In our study, we detected a higher platelet count in the periodontitis group, but the difference was not statistically significant. This higher platelet count may be explained due to dental plaque bacteria, including the periodontal pathogen *Porphyromonas gingivalis*, which induces platelet activation and aggregation⁶. Similarly, several studies in the literature observed higher platelet counts in periodontitis patients^{4,16}. Differently, Kumar et al.¹¹ reported statistically lower platelet counts in the periodontitis group.

In recent years, it has been reported that MPV can also be used as a marker of inflammation in different inflammatory diseases¹⁷. Different studies in the literature have reported that MPV has a positive or negative correlation with inflammatory activity^{17,18}. Thus, Ekici et al.¹⁹ reported a strong association between MPV and angiographic severity of coronary artery disease. In our study, MPV value was found to be higher in the group of patients with periodontitis. Czerniuk et al. 18 reported similar findings. On the other hand, some studies in the literature have demonstrated decreased MPV in periodontitis^{20,21}, and others concluded that MPV values were not statistically different between the periodontitis groups and periodontally healthy groups^{22,23}. In the studies we have mentioned, there are different and contradictory results regarding MPV and platelet counts. These might be due to the different severity of periodontitis in the studies and differences in the technological methods and modes of measurements used. Also, genetic and environmental factors may affect platelet indices of different populations. Also, in our study, there were no statistically significant differences between the two groups in RBC count, HB, and HCT levels. Conversely, Rao et al.¹² found that the mean Hb level in the periodontitis group was statistically lower compared to the control group.

In the literature, some studies concluded that PCT is a reliable indicator for the diagnosis and treatment of several diseases^{24,25}. According to our findings, PCT was significantly higher in the periodontitis group and positively correlated to periodontal clinical parameters that described the severity of periodontal disease. This data might provide more accurate insight into the platelet mass and function in periodontal diseases. There was increasing evidence that platelet indices, including MPV and PCT, were found to be significantly

associated with vascular risk factors²⁶. In the study by Aslan et al.²⁷, 230 patients with carotid artery disease were included, and high PCT levels were demonstrated to be statistically higher in patients with major adverse cardiac and cerebrovascular events. Furthermore, a study showed that there was a strong relationship between PCT and saphenous vein disease and slow coronary flow²⁸. Therefore, high PCT levels in systemically healthy individuals with periodontitis may pose a risk for systemic diseases such as atherosclerotic events.

Study limitations

The limitation of our study is the small patient population (57 patients) and the cross-sectional study design. Since the participants did not undergo coronary angiography, we were unable to demonstrate the relationship between PCT and coronary artery disease directly. Further prospective and randomized studies with larger stratified populations are needed to reveal possible effects of periodontitis on CVDs.

CONCLUSION

As far as we know, this is the first clinical study to investigate PCT values derived from CBC in patients with periodontitis. We conclude that periodontitis may elevate WBC, MPV, and PCT levels compared to healthy control patients. Furthermore, the prevention and treatment of periodontitis may decrease serum mediators and markers of acute-phase response and may be beneficial in the control of atherosclerosis and other systemic inflammatory diseases.

Disclosure statement

No potential conflict of interest was reported by the authors.

Authors' contributions

Concept, study design, and project management were done by Dr. Erdal and Dr. Ustaoglu; statistics and writing were done by Dr. Inanir and Dr. Ustaoglu.

Conflict of interest

The authors declare that there is no conflict of interest and funding support. The Clinical Research Ethics Committee of the Bolu Abant İzzet Baysal University approved this study.

RESUMO

OBJETIVO: A periodontite pode estimular a resposta infecciosa e imunitária e causar o desenvolvimento da aterogênese, doença coronária e infarto do miocárdio. O objetivo deste estudo foi comparar os níveis de plaquetócrito (PCT) e de volume médio de plaquetas (VMP) derivados dos testes de hemograma completo (CBC) em doentes que sofrem de periodontite de fase 3 com os de indivíduos saudáveis, sem doença periodontal.

MÉTODOS: O estudo incluiu 57 doentes (28 mulheres e 29 homens) com periodontite de fase 3 e 57 voluntários (31 mulheres e 26 homens) que eram periodontalmente saudáveis. A idade dos participantes do estudo variou de 18 a 50 anos. A condição periodontal dos participantes do estudo foi investigada com profundidade de sonda (PD), nível de ligação clínica, hemorragia na sonda e índice de placas. Contagem de leucócitos (WBC) e eritrócitos (RBC), níveis de hemoglobina (Hb) e hematócrito (HCT), volume corpuscular médio (VCM) e largura de distribuição das células vermelhas (RDW), contagem de trombócitos, volume plaquetário médio (MPV), plaquetócrito (PCT) e contagem de neutrófilos e linfócitos foram avaliados com base nos resultados do teste CBC dos participantes do estudo.

RESULTADO: Verificou-se que os valores de PCT, WBC, neutrófilos e MPV eram significativamente mais elevados no grupo da periodontite (p<0,05). Não houve diferenças significativas nas contagens de glóbulos vermelhos, Hb, HCT, MCV, RDW; nem nas contagens de plaquetas e linfócitos entre os dois grupos estudados (p>0,05).

CONCLUSÃO: Os níveis de PCT e MPV podem ser um marcador mais útil para determinar um estado trombótico aumentado e a resposta inflamatória em doenças periodontais.

PALAVRAS-CHAVE: Contagem de células sanguíneas. Doenças cardiovasculares. Inflamação. Contagem de leucócitos. Periodontite. Plaquetas. Fatores de risco.

REFERENCES

- Papapanou PN, Sanz M, Buduneli N, Dietrich T, Feres M, Fine DH, et al. Periodontitis: consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. | Periodontol. 2018;89(Suppl 1):S173-82.
- Papapanou PN. Systemic effects of periodontitis: lessons learned from research on atherosclerotic vascular disease and adverse pregnancy outcomes. Int Dent J. 2015;65(6):283-91.
- Martinez-Herrera M, López-Domènech S, Silvestre FJ, Silvestre-Rangil J, Bañuls C, Victor VM, et al. Chronic periodontitis impairs polymorphonuclear leucocyte-endothelium cell interactions and oxidative stress in humans. J Clin Periodontol. 2018;45(12):1429-39.
- Nicu EA, Van der Velden U, Nieuwland R, Everts V, Loos BG. Elevated platelet and leukocyte response to oral bacteria in periodontitis. J Thromb Haemost. 2009;7(1):162-70.
- Nicu EA, Rijkschroeff P, Wartewig E, Nazmi K, Loos BG. Characterization of oral polymorphonuclear neutrophils in periodontitis patients: a case-control study. BMC Oral Health. 2018;18(1):149.
- Monteiro AM, Jardini MA, Alves S, Giampaoli V, Aubin EC, Figueiredo Neto AM, et al. Cardiovascular disease parameters in periodontitis. J Periodontol. 2009;80(3):378-88.
- Wiwanitkit V. Plateletcrit, mean platelet volume, platelet distribution width: its expected values and correlation with parallel red blood cell parameters. Clin Appl Thromb Hemost. 2004;10(2):175-8.
- 8. Chandrashekar V. Plateletcrit as a screening tool for detection of platelet quantitative disorders. J Hematol. 2013;2(1):22-6.
- Slade GD, Ghezzi EM, Heiss G, Beck JD, Riche E, Offenbacher S. Relationship between periodontal disease and C-reactive protein among adults in the Atherosclerosis Risk in Communities study. Arch Intern Med. 2003;163(10):1172-9.
- Wheeler JG, Mussolino ME, Gillum RF, Danesh J. Associations between differential leucocyte count and incident coronary heart disease: 1764 incident cases from seven prospective studies of 30,374 individuals. Eur Heart I. 2004;25(15):1287-92.
- Kumar BP, Khaitan T, Ramaswamy P, Sreenivasulu P, Uday G, Velugubantla RG. Association of chronic periodontitis with white blood cell and platelet count: a case control study. J Clin Exp Dent. 2014;6(3):e214-7.
- Rao PK, Reddy RV, Mapare SA, Nag VR, Gowtham K, Arora D. An investigation of blood hemogram and estimation of serum iron and protein levels in aggressive periodontitis patients: a clinic biochemical study. J Contemp Dent Pract. 2013;14(5):852-7.
- **13.** Thomas MR, Storey RF. The role of platelets in inflammation. Thromb Haemost. 2015;114(3):449-58.
- Li N, Hu H, Lindqvist M, Wikström-Jonsson E, Goodall AH, Hjemdahl P. Platelet-leukocyte cross talk in whole blood. Arterioscler Thromb Vasc Biol. 2000;20(12):2702-8.

- **15.** Schneider DJ. Factors contributing to increased platelet reactivity in people with diabetes. Diabetes Care. 2009;32(4):525-7.
- **16.** Al-Rasheed A. Elevation of white blood cells and platelet counts in patients having chronic periodontitis. Saudi Dent J. 2012;24(1):17-21.
- 17. Wada H, Dohi T, Miyauchi K, Shitara J, Endo H, Doi S, et al. Mean platelet volume and long-term cardiovascular outcomes in patients with stable coronary artery disease. Atherosclerosis. 2018;277:108-12.
- 18. Czerniuk MR, Bartoszewicz Z, Dudzik-Niewiadomska I, Pilecki T, Górska R, Filipiak KJ. Simple platelet markers: mean platelet volume and congestive heart failure coexistent with periodontal disease. Pilot studies. Cardiol J. 2019;26(3):253-9.
- Ekici B, Erkan AF, Alhan A, Sayin I, Ayli M, Töre HF. Is mean platelet volume associated with the angiographic severity of coronary artery disease? Kardiol Pol. 2013;71(8):832-8.
- **20.** Wang X, Meng H, Xu L, Chen Z, Shi D, Lv D. Mean platelet volume as an inflammatory marker in patients with severe periodontitis. Platelets. 2015;26(1):67-71.
- 21. Zhan Y, Lu R, Meng H, Wang X, Sun X, Hou J. The role of platelets in inflammatory immune responses in generalized aggressive periodontitis. J Clin Periodontol. 2017;44(2):150-7.
- 22. Temelli B, Yetkin Ay Z, Aksoy F, Büyükbayram HI, Kumbul Doguc D, Uskun E, et al. Platelet indices (mean platelet volume and platelet distribution width) have correlations with periodontal inflamed surface area in coronary artery disease patients: a pilot study. J Periodontol. 2018;89(10):1203-12.
- **23.** Androsz-Kowalska O, Jankowski K, Rymarczyk Z, Kowalski J, Pruszczyk P, Górska R. Correlation between clinical parameters of periodontal disease and mean platelet volume in patients with coronary artery disease: a pilot study. Kardiol Pol. 2013;71(6):600-5.
- 24. Cetin MS, Ozcan Cetin EH, Akdi A, Aras D, Topaloglu S, Temizhan A, et al. Platelet distribution width and plateletcrit: novel biomarkers of ST elevation myocardial infarction in young patients. Kardiol Pol. 2017;75(10):1005-12.
- 25. Zhang F, Chen Z, Wang P, Hu X, Gao Y, He J. Combination of platelet count and mean platelet volume (COP-MPV) predicts postoperative prognosis in both resectable early and advanced stage esophageal squamous cell cancer patients. Tumour Biol. 2016;37(7):9323-31.
- **26.** Boos CJ, Lip GY. Platelet activation and cardiovascular outcomes in acute coronary syndromes. J Thromb Haemost. 2006;4(12):2542-3.
- 27. Aslan S, Demir AR, Demir Y, Tasbulak Ö, Altunova M, Karakayali M, et al. Usefulness of plateletcrit in the prediction of major adverse cardiac and cerebrovascular events in patients with carotid artery stenosis. Vascular. 2019;27(5):479-86.
- 28. Akpinar I, Sayin MR, Gursoy YC, Aktop Z, Karabag T, Kucuk E, et al. Plate-letcrit and red cell distribution width are independent predictors of the slow coronary flow phenomenon. J Cardiol. 2014;63(2):112-8.

REV ASSOC MED BRAS 2020; 66(2):133-138