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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

# Correlation between clinical parameters of periodontal disease and mean platelet volume in patients with coronary artery disease: a pilot study

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## Abstract

**Background:** Cardiovascular diseases as well as periodontitis can be regarded as current epidemics and have become a social problem. Mean platelet volume (MPV) is a simple, routinely assessed biochemical parameter, which is becoming regarded as a new, independent risk factor of acute coronary syndromes and stroke.

**Aim:** Assessment of a potential relationship between clinical indices of periodontal disease and MPV in relation to the presence of coronary artery disease (CAD) and chronic periodontitis.

**Methods:** The study included 57 individuals aged from 50 to 65 years. Patients were divided into three groups. Group 1 consisted of 19 patients with previously diagnosed CAD and coexisting chronic periodontitis. Group 2 included 18 patients with diagnosed chronic periodontitis with excluded CAD. Group 3 was a control group and consisted of 20 healthy individuals without CAD or periodontitis.

**Results:** Unsatisfactory oral hygiene defined by plaque index (PI) was observed in all patients. Mean PI was significantly higher in Groups 1 and 2 than in Group 3 (76.7% vs. 45.7%, $p < 0.01$). Mean bleeding index (BI) was significantly higher in Group 2 than in Groups 1 and 3 (46.4% vs. 29.8%, $p < 0.05$). Mean periodontal pocket depths (PD) (2.75 mm, 2.93 mm, 1.97 mm, respectively, $p < 0.05$, $p < 0.01$) and clinical attachment loss (CAL) were significantly higher in Groups 1 and 2 than in Group 3 (5.13 mm, 4.79 mm, 1.31 mm, respectively, $p < 0.01$). Mean WBC, fibrinogen and hsCRP were not significantly different among the examined groups (WBC 6.81 G/L vs. 6.71 G/L vs. 6.18 G/L, fibrinogen concentration 4.31 g/L vs. 3.94 g/L vs. 3.67 g/L; hsCRP concentration 4.08 mg/dL vs. 6.61 mg/dL vs. 4.33 mg/dL). In Group 1, MPV was significantly higher than in Group 3 (10.39 fL vs. 9.39 fL, $p < 0.01$). There was a weak, although significant, correlation between periodontal parameters and MPV and correlations between MPV and PD as well as CAL (MPV–PD: $r = 0.45$, $p < 0.05$; MPV–CAL: $r = 0.42$, $p < 0.05$).

**Conclusions:** Chronic periodontitis in patients with CAD results in an increased MPV that may suggest increased platelet activity. This observation could indicate a potential pathophysiological link between chronic periodontitis and an increased risk of acute coronary syndromes.

**Key words:** periodontitis, mean platelet volume, coronary artery disease

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## INTRODUCTION

Chronic periodontitis is a common disease. The prevalence of severe periodontitis, which can result in complete tooth loss, is approximately 20% [1]. It has been reported that the dental biofilm present in chronic periodontitis leads to activation of the inflammatory response, which in long-term observation

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Table 1. Characteristics of studied groups

| | Group 1 | Group 2 | Group 3 | $P_{1, 2}$ | $P_{1, 3}$ | $P_{2, 3}$ |
|---|---|---|---|---|---|---|
| No. of patients | 19 | 18 | 20 | | | |
| Males [%] | 89 | 33 | 28 | < 0.01 | < 0.01 | NS |
| Age [years ± SE] | 60 ± 1.15 | 57 ± 1.25 | 54 ± 0.96 | NS | < 0.01 | NS |
| Treatment with statins [n (%)] | 16 (84.2%) | 1 (5.5%) | 0 | < 0.05 | < 0.01 | NS |
| Treatment with ASA [n (%)] | 19 (100%) | 1 (5.5%) | 0 | < 0.01 | < 0.01 | NS |

ASA — acetylsalicylic acid; Group 1: with previously diagnosed coronary artery disease and coexisting chronic periodontitis; Group 2: with chronic periodontitis and excluded coronary artery disease; Group 3: control group

may contribute to the progression of atherothrombosis. Coronary artery disease (CAD) remains very common and one of the most frequent causes of death [2].

It has been demonstrated that mean platelet volume (MPV), a biochemical parameter which is easy to assess and reproducible, can be regarded as an indicator of an increased platelet reactivity and a predictor of cardiovascular episodes [3]. It has been confirmed that MPV is higher in tobacco smokers and patients with arterial hypertension [4]. Moreover, increased MPV has been found in patients with type 2 diabetes and CAD [5]. Increased MPV has been recognised as an independent risk factor for myocardial infarction [6] and stroke [7]. Furthermore, increased MPV has been associated with poor clinical outcome in survivors of myocardial infarction [8] and with the severity of acute ischaemic cerebrovascular events [9]. We aimed to assess a potential relationship between clinical indices of periodontal disease and MPV in relation to the presence of CAD and chronic periodontitis.

## METHODS

### *Study groups*

The study included 57 individuals (29 females and 28 males) aged from 50 to 65 (mean 56.8 ± 0.93) years. Patients were divided into three groups. Group 1 (n = 19) consisted of patients with previously diagnosed CAD and coexisting chronic periodontitis. Group 2 (n = 18) included patients with chronic periodontitis and excluded CAD. Group 3 (n = 20) was a control group and consisted of healthy individuals, without CAD or periodontitis. Clinical characteristics of the study groups are presented in Table 1.

All patients were subjected to an examination procedure which included the following elements.

Criteria for inclusion in the study were defined as follows:

- age 50–65 years;
- presence of at least seven teeth;
- absence of severe or chronic inflammatory diseases (other than periodontitis);
- absence of autoimmune diseases;
- absence of diabetes mellitus;
- no antibiotic therapy within last three months;
- written consent of the patient to participate in the study.

In Group 1, CAD was confirmed by history of acute coronary syndrome (at least six months prior to the study) and/or by the presence of angiographically visualised significant lesions — at least 50% stenosis in at least one coronary artery.

Chronic periodontitis was diagnosed when the patient had gingival inflammation and progressive destruction of periodontal tissues, confirmed by the values of bleeding index (BI), pocket depth (PD) and clinical attachment loss (CAL).

Each patient was subjected to: (a) periodontal examination; (b) blood tests:

- blood count — including leukocytosis (WBC) and MPV;
- plasma fibrinogen concentration;
- high sensitivity C-reactive protein concentration (hsCRP).

All patients were subjected to clinical examination, including assessment of oral hygiene and condition of periodontal tissues. Oral hygiene was evaluated by the O'Leary simplified PI, progression of periodontal inflammation was evaluated by the Ainamo and Bay BI [10]. Maximum depths of PD, as well as mean and maximum level of CAL, were also measured as an index of the status of periodontal tissues and progression of periodontal inflammation. Inadequate oral hygiene was diagnosed when PI exceeded 20%.

The control group (Group 3) consisted of individuals without signs and symptoms of chronic periodontitis. Average periodontal PD did not exceed the physiological value of 2 mm. Pockets deeper than 2 mm were observed only in third molars, where anatomical conditions of the gingiva justify the presence of physiological pockets deeper than 2 mm. CAL was also observed, indicating age-appropriate periodontal lesions. Neither clinical history nor accessory examinations, electrocardiography nor echocardiography revealed symptoms or signs of cardiovascular disease in these individuals.

The study protocol was approved by the Local Bioethics Committee.

### *Statistical analysis*

Blood tests were performed using commonly available laboratory assays. Leukocytosis was determined by flow cytofluorometry (SYMEX XE-2100D, SYMEX). MPV was also evaluated using the same equipment. CRP levels in serum were determined by the hypersensitive turbidimetric method with the

use of microparticles (Dimension, Siemens). Fibrinogen levels were determined by modified Clauss method (Multifibren U, Siemens). Blood samples were analysed in the first hour after blood donation.

Results were analysed using appropriate modifications of Student's t test, and verified with the Bonferroni-Holm adjustment for multiple comparisons. Results were deemed statistically significantly different when $p < 0.05$. Linear correlation coefficients between studied parameters were determined.

## RESULTS

Basic clinical parameters of the study groups are shown in Table 1 and in Figure 1. All patients in Group 1 were treated with acetylsalicylic acid according to current guidelines (aspirin dose of 75 mg/24 h), whereas in Group 2 only one individual was receiving such treatment. In Group 1, 16 patients were treated with statins. Among patients with chronic periodontitis without coexisting CAD (Group 2), only one was treated with statins.

Comparison of periodontal parameters, leukocytosis (WBC), fibrinogen concentration, hsCRP concentration, and MPV in the studied groups is shown in Table 2.

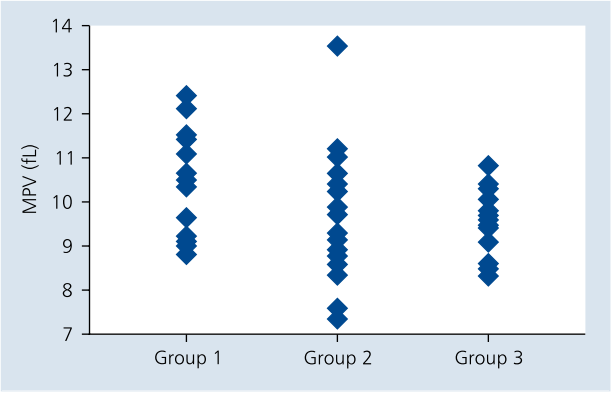


Figure 1. Mean platelet volume in all studied groups (Group 1: with previously diagnosed coronary artery disease [CAD] and coexisting chronic periodontitis; Group 2: with chronic periodontitis and excluded CAD; Group 3: control group)

Unsatisfactory oral hygiene was observed in all patients. Mean PI was significantly higher in Groups 1 and 2 than in the control group. A non-significant trend for higher PI was observed in Group 1 (patients with periodontal disease and CAD), compared to Group 2 (patients with periodontal disease but no CAD).

Mean BI in Group 2 was significantly higher than in Group 1 ($p < 0.05$) and Group 3 ($p < 0.01$). Mean BI in Group 1 (patients with periodontal disease and CAD) was not significantly different to mean BI in the control group, although it should be noted that values in Group 1 tended to be higher. Interestingly, mean PD and CAL (parameters indicating the status of periodontal tissues and progression of periodontal tissue inflammation) were not significantly different between Groups 1 and 2. However, they were significantly lower in Group 3. Mean WBC, fibrinogen and hsCRP concentrations were not significantly different between the examined groups.

Although MPV did not exceed reference values (norm: 7–12 fL) in all examined groups, it was significantly higher in Group 1 than in the control group.

No significant correlation was observed between periodontal indices and evaluated inflammatory parameters (WBC and concentrations of fibrinogen and hsCRP). We observed a weak, but statistically significant, positive correlation between periodontal parameters and MPV: MPV with PD ($r = 0.45$, $p < 0.05$) and CAL ($r = 0.42$, $p < 0.05$) (Table 3, Fig. 2).

## DISCUSSION

Bacteria of dental biofilm present in periodontal pockets, which occur in the majority of the adult population, are a potential source of infection resulting in inflammation of various organs. Each dental procedure, including home oral hygiene, can cause dissemination of $10^{11}$ bacteria per milligram of dental plaque [11]. A close relation between the presence of dental biofilm and the incidence of acute coronary syndromes has been reported. Renvert et al. [12] demonstrated an increased presence of *Streptococci spp.*, *P. gingivalis*, *T. forsythia* and *T. denticola* in subgingival biofilm in patients with CAD, suggesting a close correlation between

Table 2. Comparison of periodontal and inflammatory parameters, and mean platelet volume in patient groups (mean ± SE)

| | Group 1 (n = 19) | Group 2 (n = 18) | Group 3 (n = 20) | $P_{1,2}$ | $P_{1,3}$ | $P_{2,3}$ |
|---|---|---|---|---|---|---|
| Plaque index [%] | 76.7 ± 5.8 | 65.8 ± 5.0 | 45.7 ± 6.0 | NS | < 0.01 | < 0.05 |
| Bleeding index [%] | 29.8 ± 5.2 | 46.4 ± 5.2 | 18.2 ± 3.70 | < 0.05 | NS | < 0.01 |
| Pocket depth [mm] | 2.75 ± 0.34 | 2.93 ± 0.17 | 1.97 ± 0.10 | NS | < 0.05 | < 0.01 |
| Clinical attachment loss [mm] | 5.13 ± 0.55 | 4.79 ± 0.29 | 1.31 ± 0.35 | NS | < 0.01 | < 0.01 |
| WBC [G/L] | 6.81 ± 0.39 | 6.71 ± 0.39 | 6.18 ± 0.31 | NS | NS | NS |
| hsCRP [mg/dL] | 4.08 ± 0.86 | 6.61 ± 1.55 | 4.33 ± 0.51 | NS | NS | NS |
| Fibrinogen [g/L] | 4.31 ± 0.25 | 3.94 ± 0.25 | 3.67 ± 0.23 | NS | NS | NS |
| MPV [fL] | 10.39 ± 0.30 | 9.72 ± 0.34 | 9.39 ± 0.18 | NS | < 0.01 | NS |

WBC — white blood cell count; hsCRP — high sensitivity C-reactive protein; MPV — mean platelet volume; other abbreviations as in Table 1

Table 3. Correlations between periodontal parameters and mean platelet volume (MPV) in examined groups of patients

| MPV | Plaque index | Bleeding index | Pocket depth | Clinical attachment loss |
|---|---|---|---|---|
| Group 1 | 0.19 | 0.21 | 0.45 | 0.42 |
| Group 2 | 0.16 | 0.20 | –0.15 | –0.06 |
| Group 3 | –0.09 | 0.23 | –0.06 | – |

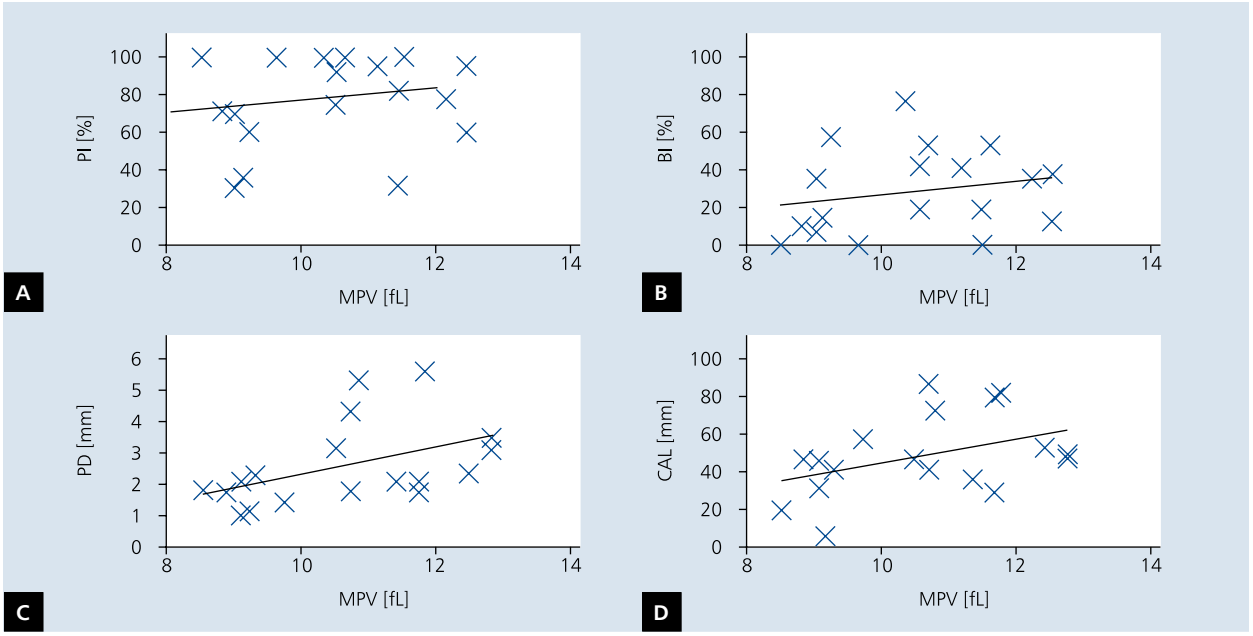


Figure 2. Correlations between mean platelet volume (MPV) and selected periodontal parameters; **A**. Plaque index (PI); **B**. Bleeding index (BI); **C**. Pocket depth (PD); **D**. Clinical attachment loss (CAL)

periodontitis and acute coronary syndromes. Zaremba et al. [13] found *P. gingivalis* and *T. denticola* bacteria in 50% of atheromatous plaque collected intraoperatively during coronary artery bypass grafting in patients with advanced periodontal disease. Furthermore, it has been postulated that periodontitis should be considered an independent risk factor of CAD, particularly in patients without classical CAD risk factors [14].

However, there are no sufficient, long-term studies showing a reduction of prevalence of acute coronary syndromes after periodontal treatment.

In our study, we observed unsatisfactory oral hygiene in both studied groups (Groups 1 and 2), with the lowest index value in the group with CAD. Assessment of inflammation with routine parameters (WBC, fibrinogen and hsCRP plasma levels) did not demonstrate the findings of Kweider et al. [15] who reported that in patients with severe periodontitis, WBC count and serum concentrations of fibrinogen and CRP were more elevated than in controls with healthy periodontal tissues. These authors also observed a positive correlation between fibrinogen levels, WBC and dental indices. One potential explanation for the discrepancy is unsatisfactory oral hygiene in our studied population, which may confound the results. According to recent data, only 1% of the adult Polish population presents a healthy periodontum, while 12% need only plaque removal and oral hygiene instruction [16]. In the patients with CAD, we observed a positive correlation between MPV and periodontal parameters (PD and CAL). Since platelet activation plays an important role in the pathogenesis of atherosclerosis, including acute coronary syndromes, and platelet volume can be regarded as a simple index of platelet activation, this suggests that platelet activation correlates with the activity of periodontal disease.

There are two possible explanations of the significantly higher mean MPV in CAD patients. It may be the result of the synergy of low grade inflammation in periodontal tissues and potentially in coronary arteries; more common periodontal inflammation in Group 2, as expressed by BI, may also be the cause of the observed increase of parameter value.

All patients in Group 1 were subjected to long-term treatment with acetylsalicylic acid; most of them also received statin treatment. Both medications decrease inflammation, which can affect periodontal parameters. Reduced inflammation associated with medication may have affected the

MPV in this group. It may be that the correlation would have been stronger if all the patients had been treated with the abovementioned drugs.

Increased platelet volume is associated with higher platelet reactivity, which results in greater synthesis of thromboxane and greater expression of adhesion molecules [17–19]. A study by Papapanagiotou et al. [20] demonstrated that platelets in patients with periodontitis have increased activity compared to platelets in healthy individuals, indicating a higher risk for coronary atherosclerosis, and consequently for CAD in patients with periodontitis. A study by Nicu et al. [21] suggested that platelets in patients with periodontitis demonstrate increased sensitivity to bacteria of dental biofilm compared to healthy individuals. This observation was confirmed by studies in animal models [22, 23]. Nicu et al. [21] also observed increased platelet activity and more intense formation of platelet-leukocyte complexes as a response to dental biofilm bacteria, which could result in vascular atherosclerosis in patients with periodontitis. Nadar et al. [24] established that patients with systemic hypertension had a higher MPV compared to healthy individuals (7.8 fL vs. 6.36 fL, $p < 0.005$). Subsequent analysis showed that treatment with acetylsalicylic acid correlated with higher MPV and mass. However, administration of statins did not significantly affect MPV values [24].

In a meta-analysis of 24 studies, comprising 6,000 patients, Chu et al. [25] demonstrated that MPV is increased in patients with stable CAD, and that it can be a risk factor for death in patients after myocardial infarction. It has been suggested that MPV could be regarded as a prognostic marker in cardiovascular diseases. Assessment of MPV is easily available, reproducible and inexpensive. Our data provides a potential link between periodontitis and increased MPV.

### *Limitations of the study*

Important limitations of our study should be mentioned. First of all, the studied group comprised a small number of patients. Therefore our results can be considered hypothetical — adequately powered prospective cohort trials are warranted to verify our findings. The distribution of the sexes in the studied groups was uneven, which can significantly influence the results. Moreover, despite lengthy efforts, we were not able to collect patients with CAD without contaminant periodontitis.

## CONCLUSIONS

Our observations could indicate a potential pathophysiological link between chronic periodontitis and an increased risk of CAD.

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***Conflict of interest:*** *none declared*

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# Ocena korelacji między parametrami klinicznymi choroby przyzębia i średnią objętością płytki krwi u pacjentów z chorobą wieńcową: doniesienie wstępne

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## Streszczenie

**Wstęp:** Choroby sercowo-naczyniowe oraz choroby przyzębia można nazwać współczesnymi epidemiami i problemem społecznym. Oznaczenie średniej objętości płytki krwi (MPV) jest prostym, rutynowo wykonywanym badaniem biochemicznym. Parametr ten uważa się obecnie za nowy, niezależny czynnik ryzyka wystąpienia zawału serca i udaru mózgu.

**Cel:** Celem pracy była ocena potencjalnego związku między klinicznymi wskaźnikami zapalenia przyzębia a MPV w zależności od występowania choroby wieńcowej i przewlekłego zapalenia przyzębia.

**Metody:** W badaniu wzięło udział 57 osób podzielonych na 3 grupy: Grupa 1 liczyła 19 pacjentów z chorobą wieńcową i współistniejącym przewlekłym zapaleniem przyzębia, Grupa 2 — 18 pacjentów z przewlekłym zapaleniem przyzębia, bez choroby wieńcowej, Grupa 3 — 20 zdrowych osób, u których nie stwierdzono choroby wieńcowej ani aktywnego zapalenia przyzębia.

**Wyniki:** U wszystkich badanych osób stwierdzono niezadowalającą higienę jamy ustnej. Średnie wartości wskaźnika płytki (PI) były istotnie statystycznie wyższe w Grupach 1 i 2 niż w Grupie 3 (76,7% *vs*. 65,8% *vs*. 45,7%). Średnie wartości wskaźnika krwawienia (BI) były istotnie wyższe w Grupie 2 niż w Grupach 1 i 3 (29,8% *vs*. 46,4% *vs*. 18,2%). Średnie wartości głębokości kieszonki przyzębnej (PD) (2,75 mm *vs*. 2,93 mm *vs*. 1,97 mm) i utraty przyczepu łącznotkankowego (CAL) były istotnie statystycznie wyższe w Grupach 1 i 2 niż w Grupie 3 (5,13 mm *vs*. 4,79 mm *vs*. 1,31 mm). Średnie wartości leukocytozy, stężenia fibrynogenu i białka C-reaktywnego (CRP) nie różniły się między grupami. W Grupie 1 stwierdzono istotną statystycznie różnicę w wartościach MPV w porównaniu z Grupą 3 (10,39 fl *vs*. 9,39 fl). Analiza korelacji między parametrami klinicznymi oceniającymi stan tkanek przyzębia wykazała słabą, ale istotną statystycznie korelację między MPV a PD i CAL (MPV–PD: $r = 0,45$; $p < 0,05$; MPV–CAL: $r = 0,42$; $p < 0,05$).

**Wnioski:** W trakcie przewlekłego zapalenia przyzębia u pacjentów z chorobą wieńcową stwierdza się większą MPV, co może wskazywać na większą aktywację płytek i sugerować potencjalny mechanizm zwiększonego ryzyka wystąpienia ostrych epizodów wieńcowych.

**Słowa kluczowe:** przewlekłe zapalenie przyzębia, średnia objętość płytki krwi, choroba wieńcowa

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