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The Association Between Dental Status and Systemic Lipid Profile and Inflammatory Mediators in Patients After Myocardial Infarction

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A – research concept and design; **B** – collection and/or assembly of data; **C** – data analysis and interpretation; **D** – writing the article; **E** – critical revision of the article; **F** – final approval of article

Abstract

Background. Many epidemiological studies have proven that local infection may influence the levels of systemic lipid profile and inflammatory mediators.
Objectives. The aim of this research was to evaluate the association between the state of the oral cavity, lipids and inflammatory mediator concentrations in Poles after acute myocardial infarction (MI).
Material and Methods. A total of 134 subjects with a mean age of 54.3 years (± 8.1) were included in the study. Sociodemographic and cardiologic variables were gathered. Subsequently, serum samples were collected for estimation of total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL-C), high-density lipoprotein (HDL-C), high-sensitivity C-reactive protein (hsCRP), fibrinogen and white blood cell counts (WBC). The periodontal parameters measured included bleeding on probing index (BoP), pocket depth (PD), clinical attachment level (CAL), the number of bleeding periodontal pockets (bPP) and the number of lost teeth.
Results. Overall, patients shared high levels of periodontal inflammation and tissue breakdown. Multivariate analysis revealed a significant association between the serum concentration of LDL-C and bPP (standardized coefficient b = 0.3179; p = 0.0009) and PD (b = 0.3186; p = 0.0015); the level of fibrinogen and the number of lost teeth (b = 0.3669; p = 0.0013); WBC and bPP (b = 0.2726; p = 0.0035) independent of age, sex, income, education, atherosclerotic disease in the family, tobacco smoking, arterial hypertension, diabetes mellitus and BMI. No correlations were found regarding hsCRP serum concentration.
Conclusions. To our knowledge, this study demonstrated for the first time that local inflammatory processes in the oral cavity are positively associated with the systemic levels of LDL-C, fibrinogen and WBC in adult Poles. This may underscore relationships between periodontitis and MI as well as potentially impinge on atherosclerotic processes and MI prognosis (**Adv Clin Exp Med 2016, 25, 4, 625–630**).

Key words: inflammation, fibrinogen, cardiovascular disease, lipid profile, C-reactive protein.

Cardiovascular diseases (CVD) are the leading cause of death in Poland, accounting for almost 50% of all deaths. Among the many CVD risk factors, the most important are considered to be lipid disorders, whereas lipid profile evaluation including total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL-C) and high-density lipoprotein (HDL-C) allows an assessment of CVD risk. Elevated concentrations of TG, TC, LDL-C and decreased HDL-C accelerate the development of atherosclerotic plaques [1–3]. The INTER-HEART study by the evaluation of Population Attributable Risk (PAR) showed that dyslipidemia yields a 49% risk of myocardial infarction (MI) [2]. Lipid disorders are common among the population of adult Poles. The extensive LIPIDOGRAM 2004 and LIPIDOGRAM PLUS studies detected a significant deterioration in measures of pro-atherogenic dyslipidemia nationwide [4]. They showed a 7.6% drop in HDL-C and a 2.1% increase in TG.

Research is continuing to better understand the pathogenesis of atherosclerosis and to find etio-

logical factors that may become the target of therapy or may help in diagnostics. According to the latest theories, inflammation plays a key role in the process of atherosclerotic plaque formation. The best known and documented inflammatory markers include C-reactive protein (CRP) and fibrinogen [3, 5].

In patients with stable coronary heart disease (CHD), a higher CRP concentration increases the risk of MI, whereas in patients with unstable CHD and MI history, it yields a worse prognosis and a higher risk of complications [5, 6]. Even after taking into consideration other conventional risk factors, such as arterial hypertension or cholesterol level, CRP remains an independent predictor of coronary risk [7].

In turn, the concentration of fibrinogen in the blood correlates with the severity of atherosclerotic lesions, risk of acute coronary events and mortality of patients [5, 8]. Elevated concentrations of fibrinogen is an unfavorable prognostic factor for patients with CVD through its participation in coagulation processes, altering the hemorheological properties in microcirculation and pro-inflammatory action, increasing the expression of adhesion proteins or through release of inflammatory mediators from endothelial cells [3]. Fibrinogen concentration demonstrates an association with other coronary risk factors such as gender, age, obesity, cholesterol level, blood pressure or diabetes. The post-translational modification of fibrinogen, such as nitration and chlorination of tyrosine residues, oxidation of methionine, histidine and tryptophan residues, formation of dityrosine and carbonyl groups, as well as glycation and homocysteinylation in hyperglycemia and hyperhomocysteinemia, may contribute to an increased risk of arterial and venous thrombosis, which was recently meticulously described [9].

Another marker of the inflammatory host response may be white blood cell count (WBC) in the serum. One study suggested that the concentration of serum neutrophil markers, myeloperoxidase (MPO) and high matrix metalloproteinase (MMP)-8/tissue inhibitor of metalloproteinase (TIMP)-1 ratio increased the risk of recurrent acute coronary syndrome (ACS) [10]. Most recently, neutrophils have been implicated as mediators in periodontitis-myocardial infarction studies [6, 11]. However, the scientific studies in this area are scarce.

Mild chronic inflammation may initiate vascular endothelial dysfunction and impact atherosclerotic plaque formation. Such processes may be located within the oral cavity and occur, for example, in the form of periodontal diseases whose symptoms are bleeding from gingiva or tooth loss. Periodontitis is an infectious disorder that results from the interplay between pathogenic agents and host immune reactions [12]. The majority of published papers, especially retrospective epidemiological studies, supported the association between periodontitis and several systemic diseases and conditions. Consequently, periodontal disease has been applied as a predictor of CVD, diabetes mellitus and metabolic syndrome, chronic obstructive pulmonary disease, pneumonia, chronic kidney disease, cognitive impairment, rheumatoid arthritis, cancer and adverse pregnancy outcomes: preterm birth and low birth weight [13, 14]. In accordance with the European Society of Cardiology (ESC) and Other Societies on Cardiovascular Disease Prevention in Clinical Practice "periodontitis can be considered a risk indicator for a generally decreased cardiovascular health status" [1]. Similarly, the American Heart Association (AHA), as well as the European Federation of Periodontology and the American Academy of Periodontology (EFP/AAP) have concluded that periodontitis imparts increased risk for future CVD, but the evidence for causal link is still missing [13, 14].

In our previous work, we demonstrated a strong positive correlation between the extent of periodontitis and MI independent of classical CVD risk factors [15]. In turn, the aim of this study was to evaluate the association between periodontal tissue status and lipids and selected inflammatory mediator concentrations in the blood of Poles after MI.

Material and Methods

The study was conducted in the First Clinic and Department of Cardiology, Faculty of Medicine, Medical University of Warsaw (MUW) in 2011–2013, observing ethical standards resulting from the Helsinki Declaration of 1975, as revised in 2000. The research was approved by the Bioethics Committee, MUW (KB-145/2011). All patients involved in the study gave written consent to participate in the project. STROBE guidelines were met while conducting and reporting the study.

The research included 134 patients (29 women, 105 men) under 70 years of age who were hospitalized due to a recent MI. MI (STEMI and NSTEMI) was diagnosed in accordance with the criteria of the ESC Guidelines. The mean age was 54.3 years (± 8.1). Exclusion criteria were: 1) stroke history; 2) cancer; 3) rheumatic disease; 4) autoimmune disease; 5) chronic liver disease; 6) chronic renal disease; 7) edentulousness. Recruitment for the study was carried out by a single cardiologist (EN).

Based on data from each patient's medical history and physical examination, the following in-

formation was obtained: Age, income per family member per month (< PLN 800, PLN 800–1500, > PLN 1500), education (primary, secondary and higher), the prevalence of atherosclerosis in the family, smoking, arterial hypertension (systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg in three consecutive measurements carried out at 5-min intervals, or the use of antihypertensive drugs), diabetes (fasting blood glucose level > 126 mg/dL or taking applicable medication), BMI (BMI 25–29.9 kg/m^2 was defined as overweight, and BMI ≥ 30 kg/m^2 as obese).

Fasting venous blood was collected from the patients into a clot tube on the second day after MI and immediately transferred to the Central Laboratory where the following assays were determined: serum level of hsCRP (reference value 0–5 mg/L), fibrinogen (reference value 200–400 mg/dL), white blood cell count (WBC) (standard 4.0–11.0 × 10^3 μL), serum level of TC (standard ≤ 190 mg/dL), TG (standard ≤ 150 mg/dL), LDL-C (standard ≤ 115 mg/dL), HDL-C (standard for men > 40 mg/dL, for women > 46 mg/dL).

The dental examination was performed by a calibrated dentist (BG). Calibration was accepted if ≥ 90% of the recordings (pocket depth, clinical attachment level) could be reproduced within a difference of 1.0 mm. The number of teeth present in the oral cavity and the number of extracted teeth were determined. The examinations did not include third molars. Oral hygiene indices (such as plaque index) were not assessed, as probably the period of hospitalization could have negatively impacted the results in this area. Bleeding on probing index (BoP) by Ainamo and Bay was established at four points around all teeth: mesial-buccal (MB), buccal (B), distal-buccal (DB) and lingual (L) by dividing the sum of bleeding points by the sum of all the test points. Pocket depth (PD) and clinical attachment level (CAL) were evaluated at four sites per tooth: MB, B, DB, L. PD was defined as the distance from the gingival margin to the bottom of the pocket established by probing (in mm), and CAL as the distance between the bottom of the pocket and the cementoenamel junction (in mm). Measurements were rounded down to the nearest mm. The number of bleeding pockets with PD ≥ 4 mm (bPP) was also verified. Diagnosis of periodontitis was made in line with the definitions proposed by the Centers for Disease Control and AAP as follows:

– mild periodontitis – 2 or more teeth with CAL ≥ 3 mm on proximal surfaces, or 2 or more teeth with PD ≥ 4 mm on proximal surfaces;

– moderate periodontitis – 2 or more teeth with CAL ≥ 4 mm on proximal surfaces, or 2 or more teeth with PD ≥ 5 mm on proximal surfaces;

– severe periodontitis – 2 or more teeth with CAL ≥ 6 mm on proximal surfaces and 1 or more teeth with PD ≥ 5 mm on proximal surfaces;

– other clinical situations were defined as "no periodontitis" [12].

A statistical analysis of the collected data was performed using PQStat v. 1.4.4 software. The strength of correlations between the biochemical parameters (lipids and selected inflammatory mediators in blood) and dental parameters was evaluated using the Spearman test. In the next step, a multivariate analysis was performed using the linear regression method, where dental parameters were considered to be independent variables while biochemical parameters were reckoned as dependent variables. Apart from the dental parameters, the independent variables included in the first stage were gender and age of the patient (Model I). Subsequently, the remaining assessed CVD risk factors, such as tobacco smoking, arterial hypertension, diabetes mellitus, BMI, atherosclerosis in the family, income and education were incorporated into the analysis (Model II). The threshold for statistical significance was assumed at $p = 0.05$.

Results

Table 1, Table 2 and Table 3 show the demographic, medical and dental characteristics of the study group. Table 1 contains average concentration values of hsCRP, fibrinogen, TC, TG, LDL-C, HDL-C and WBC in the blood.

On the basis of a correlation analysis (Table 4), a correlation between selected biochemical parameters and BoP, PD, bPP, CAL and the number of extracted teeth was determined.

The multivariate analysis resulted in formulation of a few statistically significant models, with biochemical parameters considered as dependent variables, while dental parameters were included among independent variables (Table 5). This analysis has supported the relationships between: 1) fibrinogen concentration and the number of extracted teeth; 2) the number of leukocytes and the number of bPP; 3) LDL-C concentration and PD; 4) LDL-C concentration and the number of bPP, also when other factors such as age, gender, tobacco smoking, arterial hypertension, diabetes mellitus, body mass disorders, family occurrence of atherosclerosis, income and education were included.

Discussion

Our study showed a positive correlation between oral health and the blood levels of LDL-C,

Table 1. Demographic and medical characteristics of the study group (N = 134)

Demographic and medical variables	
Age, mean ± SD	54.3 ± 8.1
Gender, n (%) women men	 29 (21.6) 105 (78.4)
Income, n (%) > PLN 1500 PLN 800–1500 < PLN 800	 45 (33.6) 50 (37.3) 39 (29.1)
Education, n (%) higher secondary primary	 27 (20.2) 61 (45.5) 46 (34.3)
Atherosclerosis in family, n (%)	21 (15.7%)
Smoking habit, n (%) never past current	 27 (20.2) 23 (17.2) 84 (62.7)
Smoking, pack-years, mean ± SD	22.3 ± 19.4
Arterial hypertension, n (%)	101 (75.9)
Systolic blood pressure (mm Hg), mean ± SD	136.7 ± 20.9
Diastolic blood pressure (mm Hg), mean ± SD	80.1 ± 11.6
Diabetes mellitus, n (%)	28 (20.9)
BMI (kg/m^2), mean ± SD	28.7 ± 5.0
Body weight, n (%) normal overweight obesity	 28 (21.4) 57 (43.5) 46 (35.1)

SD – standard deviation; n – number; BMI – body mass index.

Table 2. Biochemical characteristics of the study group (N = 134)

Biochemical parameters	
hsCRP (mg/L), mean ± SD	16.6 ± 28.9
Fibrinogen (mg/dL), mean ± SD	354.1 ± 101.2
WBC (10^3 µL), mean ± SD	10.8 ± 6.7
TC (mg/dL), mean ± SD	190.5 ± 51.6
TG (mg/dL), mean ± SD	150.0 ± 78.6
LDL-C (mg/dL), mean ± SD	116.9 ± 39.3
HDL-C (mg/dL), mean ± SD	43.1 ± 10.2

SD – standard deviation; hsCRP – high-sensitivity; C-reactive protein; WBC – white blood cells; TC – total cholesterol; TG – triglycerides; LDL-C – low-density lipoprotein; HDL-C – high density lipoprotein.

Table 3. Dental characteristics of the study group (N = 134)

Bleeding on probing index (BoP) (%), mean ± SD	49.9 ± 29.9
Pocket depth (PD) (mm), mean ± SD	2.9 ± 1.1
Bleeding periodontal pockets (bPP) (n), mean ± SD	14.3 ± 14.1
Clinical attachment level (CAL) (mm), mean ± SD	4.2 ± 2.0
Periodontal status, n (%) no periodontitis mild periodontitis moderate periodontitis severe periodontitis	 7 (5.2) 7 (5.2) 45 (33.6) 75 (56.0)
Lost teeth, median (Q1–Q3) (n)	10 (6–17)

SD – standard deviation; n – number.

fibrinogen and WBC count in patients after MI. A multivariate analysis confirmed that these correlations were independent of other CVD risk factors.

The most important relationships seem to be between PD/bPP and LDL-C concentration, because a strong positive correlation between LDL-C serum level and cardiovascular risk in both sexes has been proven, regardless of the presence of clinically overt CVD [1, 2]. These outcomes show that both periodontal tissue breakdown (PD) and level of inflammation (bPP) impinge on the lipid serum level. The results of scientific research on LDL-C concentration in the blood of patients with CVD depending on the state of periodontal tissues are ambiguous [16, 17]. However, the majority of reports on patients without cardiovascular burden indicate an impact of periodontitis on lipid disorders expressed by elevated levels of LDL-C, TC and TG, and a reduced level of HDL-C [3, 18–20]. Most recently, Sangwan et al. [18] observed positive and significant correlations between PD and TG, TC and LDL-C serum concentrations, as well as between CAL and TC, and CAL and LDL-C, after adjusting for confounders. Regression analysis revealed a positive association between PD and TC ($p < 0.001$), along with correlation between CAL and LDL-C ($p = 0.013$). Moreover, the intake of statins was associated with increasing serum osteoprotegerin level, which seemed to have a protective effect against bone breakdown and periodontal attachment loss. Osteoprotegerin as a soluble decoy receptor of RANKL competes for binding to RANK and, as a result, inhibits osteoclast differentiation and activation. Another study showed significant association between BoP and TG, TC and LDL-C concentrations [20]. In our research,

Table 4. Correlations between biochemical parameters and dental status (Spearman test)

	Bleeding on probing index (BoP) (%)	**Pocket depth (PD) (mm)**	**Number of bleeding periodontal pockets (bPP)**	**Clinical attachment level (CAL) (mm)**	**Number of missing teeth**
hsCRP (mg/L)	r = 0.04 p = 0.6775	r = 0.06 p = 0.4834	r = –0.03 p = 0.7120	r = 0.01 p = 0.9269	r = 0.09 p = 0.2603
Fibrinogen (mg/dL)	r = 0.19 p = 0.0349 *	r = 0.10 p = 0.2549	r = –0.15 p = 0.0904	r = 0.14 p = 0.1052	r = 0.31 p = 0.0003*
WBC (10^3 μL)	r = –0.03 p = 0.7685	r = 0.09 p = 0.3006	r = 0.17 p = 0.0498*	r = 0.04 p = 0.6500	r = –.017 p = 0.0494*
TC (mg/dL)	r = 0.01 p = 0.8740	r = 0.14 p = 0.1129	r = 0.21 p = 0.0193*	r = 0.01 p = 0.8905	r = –0.11 p = 0.1762
TG (mg/dL)	r = –0.12 p = 0.1683	r = –0.01 p = 0.9513	r = 0.02 p = 0.7988	r = –0.06 p = 0.4687	r = –0.07 p = 0.3741
LDL-C (mg/dL)	r = 0.03 p = 0.7655	r = 0.24 p = 0.0100*	r = 0.28 p = 0.0020*	r = 0.08 p = 0.3976	r = –0.09 p = 0.3224
HDL-C (mg/dL)	r = 0.11 p = 0.2210	r = 0.08 p = 0.3976	r = 0.21 p = 0.0150*	r = 0.13 p = 0.1528	r = 0 p = 0.9456

HsCRP – high–sensitivity; C–reactive protein; WBC – white blood cells; TC – total cholesterol; TG – triglycerides; LDL-C – low-density lipoprotein; HDL-C – high density lipoprotein; * statistical significance.

after multivariate analysis based on the classic CVD risk factors, no independent correlations between concentrations of TC, TG, HDL-C, and dental status were observed, although the univariate analysis suggested the existence of such relationships.

Fibrinogen is a protein synthesized by hepatocytes and fibroblasts in response to inflammation [8]. It has been thought to increase the risk of coronary heart disease not only through its participation in coagulation processes, but also through pro-inflammatory action. In our study, a significant correlation between fibrinogen and the number of lost teeth was observed. The number of lost teeth may be a reflection of inflammatory processes in the oral cavity, ongoing in conditions of increased genetic susceptibility which may predispose a patient to both periodontitis and CVD. It should be noted that, in this study, tooth loss has been registered in a cross-sectional design and thus may not be entirely due to periodontal disease. However, patients shared high levels of periodontal inflammation and tissue breakdown which indicated long-term periodontal diseases. Previously published reports have highlighted an impact of the status of periodontal tissues on fibrinogen concentration in the blood of patients with CVD [3, 21, 22]. Seringec et al. [21] showed that patients with chronic periodontitis had significantly higher fibrinogen and globulin levels, as well as higher plasma viscosity and erythrocyte aggregation tendency when compared with healthy periodontium. Most recently, an association between BoP and fibrinogen concentration was proven [22]. Quite similarly in our study, a strong correlation between BoP and fibrinogen was observed (p = 0.0587), which may reflect the potential significance of local inflammation for systemic inflammatory burden.

Decidedly, the greatest attention in the literature was devoted to CRP. Many authors have demonstrated that CRP concentration is higher in patients with periodontitis and CVD compared to patients with only one of these two disorders [3, 16, 20, 23, 24]. This was also confirmed in a meta-analysis by Paraskevas et al. [25]. CRP levels in patients with CHD and chronic periodontitis equaled 7.3 mg/L, in patients with periodontitis only 2.4 mg/L, and in individuals with healthy periodontium, 1.4 mg/L [23]. Patients with diabetes mellitus, acute MI and periodontitis had considerably higher hsCRP serum levels (5.31 mg/L) than non-diabetic patients (2.38 mg/L) [26]. On the basis of the literature, it can be concluded that inflammation in the oral cavity affects CRP concentration in the blood, which reflects the dynamics of inflammatory reactions in the body. In our study, however, no such correlation was observed, which may be related to the fact that, in patients after MI, a sudden increase in CRP levels is observed, and that, in turn, may mask its possible association with periodontal status.

Currently, researchers postulate the existence of several mechanisms that might explain the po-

Table 5. Multivariate analysis showing correlations between selected biochemical parameters and dental status

Dependent variables in distinct models	Model I			Model II		
	independent variables	standardized coefficient b (for dental parameter only)	p (for dental parameter only)	independent variables	standardized coefficient b (for dental parameter only)	p (for dental parameter only)
Fibrinogen (mg/dL)	age gender BoP (%)	0.1741	0.0479*	as in Model I + tobacco smoking, arterial hypertension, diabetes mellitus, BMI, atherosclerosis in the family, income and education	0.1798	0.0587
Fibrinogen (mg/dL)	age gender number of lost teeth	0.2579	0.0139*		0.3669	0.0013*
WBC (10^3 µL)	age gender number of bPP	0.2620	0.0030*		0.2726	0.0035*
WBC (10^3 µL)	age gender number of lost teeth	–0.1067	0.2731		–0.1038	0.3860
TC (mg/dL)	age gender number of bPP	0.1220	0.1669		0.1486	0.1043
LDL-C (mg/dL)	age gender PD (mm)	0.2250	0.0162*		0.3186	0.0015*
LDL-C (mg/dL)	age gender number of bPP	0.2528	0.0064*		0.3179	0.0009*
HDL-C (mg/dL)	age gender number of bPP	0.1660	0.0591		0.1721	0.0587

BoP – bleeding on probing index; WBC – white blood cells; bPP – the number of bleeding periodontal pockets; TC – total cholesterol; LDL-C – low-density lipoprotein; PD – pocket depth; HDL-C – high density lipoprotein; * – statistical significance.

tential impact of periodontitis on the increased risk of CVD. It is most likely that elevated levels of lipopolysaccharides (LPS) in the blood stimulate the liver to increase synthesis of CRP, fibrinogen and cholesterol, which forms complexes with endotoxins and plasma lipids, intensifying their atherogenicity. Periopathogens and released proinflammatory mediators also contribute to oxidation of LDL phospholipids and their modification to ox-LDL and to modification of HDL-C by reducing the content of cholesterol esters and sphingomyelin as well as molecules of apolipoprotein (apo) A-I, whereas inflammatory HDL-C loses the ability to inhibit LDL oxidation processes [27]. It has been found, that *Aggregatibacter actinomycetemcomitans* increased the oxidation of LDL-C through oxidative stress involving NADPH oxidase- and myeloperoxidase-derived reactive oxygen species in apolipoprotein E-deficient spontaneously hyperlipidemic (Apoe(shl)) mice [28]. Macrophages in the intima, through phagocytosis of oxidized LDL, are transformed into foam cells. Foam cells activated by T lymphocytes secrete free oxygen radicals responsible for generalized oxidative stress, and numerous cytokines and growth factors that stimulate connective tissue cells and smooth muscle cells to proliferation. These mechanisms may act in concert to promote systemic inflammation in periodontal patients and to promote atherosclerosis.

Periodontal pockets deeper than 4 mm create anaerobic conditions, thus providing a reservoir of anaerobic bacteria, which may lead to an increased number of WBC in the blood, representing the body's response to bacterial load, which was also observed in our study. A recent study by Huda et al. [29] showed a statistically significant increase (two-fold, $p < 0.05$) in oral neutrophil counts in

patients with periodontitis compared to those without periodontal disease. Another work indicated that high serum neutrophil markers (MPO, MMP-9/TIMP-1 ratio) reflected an increased risk of recurrent ACS [10]. Neutrophils and leukocytes might underlie relationships of periodontitis with MI. Marfil-Álvarez et al. [11] demonstrated that the extent of periodontitis was positively associated with acute MI size as measured by blood troponin I and myoglobin levels. In that study, the extent of periodontitis, as measured by the Arbes Index, accounted for the levels of neutrophils ($p < 0.01$), total leukocytes ($p < 0.02$) and myoglobin ($p < 0.05$). Interesting observations were made by Taylor et al. [30] indicating that the removal of all teeth in patients with periodontopathy contributes to a decrease in the levels of CRP and fibrinogen, and to WBC reduction.

When interpreting the data in this study, some of its limitations must be considered. The biochemical markers were determined during hospitalization of the patients on the second day after MI. It would be recommended to perform further assessments at different time intervals after MI, to see how their concentrations in the blood are changing. None of the subjects had healthy periodontium, and the occurrence of periodontitis was very common. Therefore, it would be valuable to compare the concentrations of lipids and inflammatory mediators in patients after MI with healthy periodontium, in patients without CVD but with periodontitis, and in patients without CVD and with a healthy periodontium. It would be particularly difficult to distinguish a group of patients after MI and with healthy periodontium. Another issue is the predominance of men in the study group, which results from the more frequent occurrence of MI in men under 70 years than in women. This was taken into account in the statistical analysis, where multivariate analysis was adjusted for gender.

The results of this study have considerable clinical significance for the Polish population, as they indicate the potential impact of periodontal tissue status and the number of extracted teeth in patients after recent MI on the concentrations of lipids and inflammatory mediators in the blood, which may translate into a reduced effectiveness of CVD treatment strategies and increased coronary risk. These mechanisms might underline a previously observed association of periodontitis with MI [15]. Consequently, dental treatment leading to the elimination of inflammation in the oral cavity could contribute to better control of lipid disorders and the reduction of inflammatory mediator concentrations in the blood, and thus improve the efficiency of MI treatment.

Because of the high mortality due to CVD, it seems appropriate to develop guidelines for a basic algorithm for dental prophylaxis and therapy that could be used quickly, economically and beneficially for the patient's health, to reduce the infectious load of the body associated with periodontal foci of infection.

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