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Periodontal Status of Survivors of Acute Myocardial Infarction: A Case-Control Study

Stan tkanek przyzębia u pacjentów po zawale mięśnia sercowego- badanie kliniczno-kontrolne

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

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Summary

Aim: The aim of this multicenter cross-sectional study was to assess a potential relationship between cardiovascular risk profile and chronic periodontitis (CM) during the acute phase of myocardial infarction (MI).

Material/Methods: The study included 417 patients aged under 70 years with diagnosed and invasively managed MI. Blood samples were collected for laboratory parameters, including plasma cholesterol, glucose, CRP, fibrinogen and NTproBNP levels. The clinical evaluation of periodontal status included plaque index (PI), extent of gingival inflammation (BoP), probing pocket depth (PPD), clinical attachment level (CAL) and community periodontal index (CPI).

Results: Moderate periodontitis (CPI-3) was found in 34.6% of subjects and severe periodontitis (CPI-4) in 45.5% of studied patients. The average number of preserved teeth was 16 and it was significantly lower in patients with diabetes (DM). Mutual risk factors, such as age, gender, smoking, arterial hypertension and DM, were very common. Worse periodontal status was associated with peripheral arterial disease (PAD) and with the first MI. Moreover, patients with signs of periodontitis presented significantly higher total cholesterol level, LDL cholesterol and hsCRP levels. Patients with LDL <80mg/dl presented the best periodontal status when compared to the other groups.

Conclusions: The prevalence of CP among patients with acute MI was very high, and it was associated with history of previous MI, PAD, smoking, DM, TC, LDL and hsCRP concentrations. This study pinpointed interplays between CP and cardiovascular events.

Keywords: myocardial infarction • periodontitis • risk factor

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INTRODUCTION

Periodontitis is a chronic inflammatory disease that impairs soft and hard tissues supporting the teeth. Its etiology is based on an interplay between pathogenic microbiota and host immune reaction. When not treated, it can eventually lead to tooth loss. According to epidemiological data, nearly half of individuals aged > 30 years may be affected by various degress of periodontitis [55]. Its prevalence increases with age. periodontitis occurs in 57.2% of adult Poles aged 35-44 years, whereas 41.5% of Poles over 65 years of age have moderate or advanced periodontitis [15, 32].

On the other hand, cardiovascular diseases (CVD) are the main cause of premature death worldwide and are associated with the death of 17.1 million people annually [39]. In Poland in 2010, 456 individuals per 100,000 died of CVD, which amounted to 46.0% of all deaths [59]. Myocardial infarction (MI) was the cause of 17,800 deaths, which accounted for 10.0% of deaths from cardiovascular reasons [59]. This situation is closely associated with lifestyle and the influence of risk factors, among which cigarette smoking, arterial hypertension, overweight, diabetes mellitus, lipid disorders and excessive consumption of sodium result in steadily increasing statistics of cardiovascular events [58]. Recently, the significance of inflammatory and immunological processes has been underscored in the context of their influence on the development of atherosclerotic plaque. Local inflammation aggravated by periodontitis might increase the risk of CVD, including MI events [46]. Consequently, the Fifth Joint Task Force of the European Society of Cardiology (ESC) and Other Societies on Cardiovascular Disease Prevention in Clinical Practice suggested that "periodontitis can be considered a risk indicator for a generally decreased cardiovascular health status" [44]. Patients who have had acute myocardial infarction were characterized by inadequate oral hygiene and advanced periodontal inflammatory changes, which did not significantly change because of constant hygienic negligence despite prophylactic-therapeutic intervention [52, 53]. Moreover, research studies have shown that relative risk estimates for developing CVD in patients with periodontitis range from 1.24 to 1.34 [25]. This risk rises to 1.44 in people under 65 [27]. In the study by Łysek et al. [37] depending on the indicator, the odds of past MI in CP subjects increased 1.2-5.0 times compared to patients without periodontal disease.

Several mechanisms have been suggested that can associate periodontitis with MI, such as metastatic spread of infection after bacteremia, injury caused by microbial toxins and inflammation, endothelial dysfunction and hemorheology [25, 27]. Periodontal microbes have been shown to contribute to the progression of atherothrombosis, as the keystone periodontopathogen *Porphyromonas gingivalis* (the bacteria identified in 85% sites of CP) was able to directly invade arteries and endothelial cells [6]. Periodontitis constitutes a source of origin of continuous proinflammatory mediators and adhesion molecules, such as interleukin (IL)-1, IL-6, tumor necrosis factor (TNF)-α, C-reactive protein (CRP), matrix metalloproteinase (MMP)-1, MMP-8, and MMP-9, which trigger endothelial cells to proatherogenic respones, promote atherosclerotic plaque destabilization [17]. In a study by Punj et al. [46] endothelial function was flawed in patients with CP compared with healthy patients, and the dysfunction was analogous to that noted in the MI group based on flow-mediated dilation (FMD) assessment. Moreover, periodontitis was significantly associated with raised serum levels of CRP and LDL cholesterol, both of which represent biomarkers for endothelial function [28].

In the light of the above mentioned facts, the amount of evidence confirming an association between periodontitis and increased MI risk continues to grow [16, 31, 40, 47, 60]. By the same token, an in vivo study proved that the periodontal microbiota might promote myocarditis and/or myocardial infarction in mice [2]. Moreover, a recent meta-analysis of case-control studies found a significant association between MI and periodontitis [50], the conclusion of which was in line with the review performed by the American Heart Association (AHA) working group and to the report by Joint Workshop of European Federation of Periodontology and the American Academy of Periodontology (EFP/AAP) [36, 56]. Periodontitis can constitute not only a risk factor for MI, but also a condition modifying other primary risk factors associated with occurrence of cardiovascular complications (lipid disorders, arterial hypertension

or diabetes) [1, 2, 3]. However, some epidemiologic studies have found no relationship between periodontitis and MI [36, 44, 56].

The prevalence of CP in our population and its association with MI necessitates a deeper look at the pathogenetic mechanisms linking periodontitis with atherosclerosis and consequently with resulting CVD. Therefore, the objective of this multicenter observational study was the analysis of correlations between the state of periodontal tissues and selected risk factors for ischemic episode in survivors of acute MI.

MATERIAL AND METHODS

Study sample and data collection

The study group included 417 patients (92 women, 325 men) aged 25 to 69 years (mean age 57.0, SD 8.1 years), diagnosed with a non-ST-segment elevation (NSTEMI) or ST-segment elevation myocardial infarction (STEMI), managed invasively in five Polish tertiary referral hospitals (Szczecin, Warsaw, Wrocław, Bialystok and Lublin) on the second or third day of hospitalization. The number of included patients were the following: 179 in Szczecin, 173 in Warsaw, 33 in Wrocław, 21 in Lublin and 11 in Białystok, respectively. The diagnosis of myocardial infarction and management of ACS followed the contemporary guidelines of European Society of Cardiology on NSTEMI and STEMI [20, 53]. The inclusion criteria were MI history and age under 70 years. The exclusion criteria included malignancy, other chronic inflammatory or autoimmune diseases, advanced chronic liver disease, chronic kidney disease stage 4 or 5, rheumatic disease, chronic congestive heart failure as well as edentulism.

All patients participating in the study gave their written consent. The protocol of the study followed ethical standards according to the Helsinki Declaration of 1975, as revised in 2000, and was accepted by local Ethics Committee (KB-145/2011).

The social enquiry and general medical history included the prevalence of smoking (defined as smoking of 10 or more cigarettes a day continuously for at least 5 years), arterial hypertension (defined as diagnosed hypertension treated pharmacologically or systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg, measured in a sitting position after several minutes of rest, using a sphygmomanometer), diabetes mellitus (diagnosed if fasting blood glucose concentration was above 126 mg/dl or if the patient was taking appropriate medications), previous myocardial infarction, peripheral arterial disease.

Blood was collected from 367 fasting participants into a clot tube on the second day after MI and immediately transferred to the laboratory in order to determine the concentrations of serum TC (norm ≤190 mg/dl), TG (norm ≤150 mg/dl), LDL-C (norm ≤115 mg/dl).), HDL-C (male norm >40 mg/dl, female norm >46 mg/dl), hsCRP (reference value: 0-5 mg/l), fibrinogen (reference value: 200-400 mg/dl), accidental plasma glucose (norm 70-99 mg/dl), NT-proBNP (<55 years: male norm <64 pg/ml, female norm <155 pg/ml; 55-65 years: male norm <194 pg/ml, female norm <222 pg/ml).

DENTAL EXAMINATION AND PERIODONTAL MEASUREMENTS

Detailed dental examination and periodontal measurements were conducted in 415 patients before they were discharged. Two patients were not included in the extensive periodontal examination, though they were classified using the Community Periodontal Index. Dental examination was performed according to the standardized protocol by calibrated dentists in artificial light, using a dental mirror and a periodontal probe with colored and engraved markings at 1 mm intervals (Hu-Friedy PCPUNC 15). The number of teeth was determined. The examinations did not include third molars. Dichotomous plaque index (PI) by O'Leary was evaluated on four surfaces of all teeth (mesial, distal, lingual and buccal) [42]. The presence or absence of plaque was determined. The index was calculated as the ratio of surfaces with plaque to all tested surfaces. Bleeding on probing index (BoP) by Ainamo and Bay was evaluated at 4 points around all teeth: mesial-buccal (MB), buccal (B), distal buccal (DB) and lingual (L) [1]. BoP was calculated by dividing the sum of bleeding points by the sum of all test points. Probing pocket depth (PPD) and clinical attachment level (CAL) were evaluated at four sites per tooth: MB, B, DB, L. PPD was defined as the distance from the gingival margin to the bottom of the pocket established by probing (in mm). CAL was defined as the distance between the bottom of the pocket determined by probing and the cemento-enamel junction (in mm). Measurements were rounded down to the nearest mm. Additionally, the number of active (bleeding) pockets above 4 mm deep was recorded. Community periodontal index (CPI) [33] was assessed in 393 patients with the following categories: CPI-0 - no inflammatory symptoms; CPI-1 - presence of bleeding on probing; CPI-2 - presence of supra- and/or subgingival calculus or filling overhangs; CPI-3 - presence of pathological periodontal pockets from 3.5 to 5.5 mm deep; CPI-4 - the presence of pathological periodontal pockets 5.5 mm and deeper. Score CPI-1 was interpreted as gingivitis, while CPI-2 to CPI-4 were considered to indicate signs of periodontitis of different severity.

STATISTICAL ANALYSIS

Statistical analysis was performed using PQ-STAT software. The Mann-Whitney and Kruskal-Wallis tests were used to assess the significance of differences between two and more groups, accordingly. The χ^2 test was used to search for significant differences between the frequencies of analyzed data. Correlations between variables were measured by the Spearman rank correlation test. P-values lower than 0.05 were considered statistically significant.

Missing data were assumed to be independent both of observable variables and of unobservable parameters of interest, and occurred entirely at random. Pairwise deletion analyses were performed, including all cases in which variables of interest were present and thus maximizing all data available by an analysis basis.

RESULTS

All the patients were diagnosed with acute myocardial infarction and subjected to invasive therapy. STEMI was diagnosed in 24.9% of patients while NSTEMI in the remaining 75.1%. History of previous myocardial infarction was positive in 23.3% of studied patients. Most of the patients were current or past smokers of tobacco (almost 80%), arterial hypertension was present in 90.1% of individuals in the study group, whereas diabetes in almost 25% of the patients. During hospitalization patients received dual antiplatelet therapy, statin and ACEI/ARB treatment. Patients with history of previous acute coronary syndrome received long-term antiplatelet therapy with aspirin 75 mg/d and statin therapy.

The results of the periodontal examination showed an average of 12 preserved teeth in women and 18 in men (total median: 16 preserved teeth). Mean values of plaque index (PI) were high in both females and males (76.9% and 78.2% respectively). Also, the BOP index was high, regardless of gender (44.6%). There were significantly more pockets > 4 mm in men. None of the study participants exhibited healthy periodontium when assessed using the Community Periodontal Index. Only 4.1% of the investigated patients presented a satisfactory state of periodontal tissues, with only superficial gums inflammation (CPI-1). Slight periodontitis (CPI-2) was found in 8.4%, moderate periodontitis (CPI-3) in 34.6% and severe periodontitis (CPI-4) in 45.5% of the studied patients. The rest of the patients (11.4%) had only residual dentition, which does not allow us to qualify the patient according to the CPI scale. Clinical characteristics and periodontal status in relation to medical history findings are presented in Table 1. The number of lost teeth, BOP, mean PPD, number of active PPD>4 mm, CAL and CPI were correlated with smoking. The number of teeth in patients with diabetes was significantly lower than among the non-diabetic group. We found no differences in periodontal status between patients with STEMI or NSTEMI.

PPD, CAL and mean CPI were significantly higher in patients suffering from the first myocardial infarction than in patients with a history of previous MI. 10.3% of myocardial infarction patients presented coexisting peripheral arterial disease (PAD) and this group showed significantly more advanced forms of periodontitis, when compared to patients without peripheral artery disease (Table 2). On the other hand, patients with previous myocardial infarction had better periodontal status (as represented by CPI score), when compared to those with no previous cardiovascular events.

Significant differences in lipid profiles according to the periodontal status were found (Table 3). Patients with CPI 2–4 presented a significantly higher total cholesterol level and LDL cholesterol levels when compared to patients with CPI < 2. hsCRP levels were also significantly increased in the former. There were significant differences in periodontal status depending on the LDL levels. Patients with LDL above 120 mg/dl presented worse periodontal status (mean PD, CAL and percentage of pockets ≥ 4 mm with positive BOP) when compared to the other groups (Table 4).

DISCUSSION

The aim of this case-control study of consecutive 417 survivors of acute myocardial infarction was to evaluate potential relationships between chronic periodontitis and cardiovascular risk profile during the acute phase of MI. It should be understood that the etiologies of periodontitis and MI, both of which share some common risk factors, such as age, tobacco smoking and diabetes mellitus, are of a chronic inflammatory nature [36, 44, 56]. All of the above mentioned issues may influence periodontitis as well as MI separately, which may act as confounding factors and affect statistical analyses [7, 31]. Therefore, it is of utmost importance to investigate various dependent and independent risk factors of MI, one of which might be a relationship between MI and chronic periodontitis.

Age and gender are important, non-modifiable risk factors for both periodontal disease and cardiovascular disease. Studies by Persson et al. [45] showed that both of these diseases may occur simultaneously, particularly in individuals over 60 years of age. The occurrence of periodontitis was observed in about 50% of people aged over 60 years, and 55% of subjects were overweight or underwent an episode of stroke or acute coronary syndrome [45]. Our study also showed a higher incidence of periodontal disease in men, as well as higher general percentage of men among patients (recruited consecutively from patients presenting to the ward). Male susceptibility to the occurrence of periodontal disease and cardiovascular disease was also observed by other authors [24, 25, 55].

When analyzing the group of patients after MI it is clear that the number of preserved teeth and the condition of periodontal tissues is much worse compared to epidemiological studies on randomly selected Poles of the same age group [15, 32]. Indeed, the average number of preserved teeth in the group after myocardial infarction was 12 in women and 18 in men (mean 16). While tooth loss can be attributed to many reasons, in CVD patients population this process indicates a preceding accumulation of oral inflammatory processes [34]. Study by Górski et al. [16] showed an average of 24 teeth in the control group of the same age. In the group of patients after myocardial infarction also the percentage of edentulous individuals was high at 16.3% in women and 9.2%

Table 1. Periodontal status of examined patients with myocardial infarction (Mann-Whitney test was used for all the comparisons apart from smoking, where Kruskal-Wallis ANOVA was applied)

		Number of lost teeth [median (Q1–Q3)]	PI (%) mean ± SD	BOP(%) mean ± SD	PD, mm mean ± SD	PD≥4 mm + BOP mean ± SD	PD≥4 mm + BOP (%) mean ± SD	CAL, mm mean ± SD	CPI = 4 (%)	CPI (median (Q1–Q3))
Gender	Females (22.1%)	16 (10-24)	76.9 ± 25.4	45.0 ± 30.8%	2.8 ± 1.3	6 (2-15)	24.8 ± 25.1	3.9 ± 2.0	33.7%	3 (3-4)
	Males (77.9%)	10 (5-19)	77.6 ± 22,2	44.2 ± 28.6%	2.8 ± 1.0	11 (5-22)	27.3 ± 24.6	3.9 ± 2.2	45.5%	4 (3-4)
	P	< 0.01	0.73	0.95	0.62	< 0.01	0.26	0.83	0.19	< 0.05
Type of MI	STEMI (75,1%)	12 (6-20)	77.1 ± 23.6	43.6 ± 28.2%	2.9 ± 1.1	11 (4-21)	27.6 ± 25.6	4.0 ± 2.2	46.7%	4 (3-4)
	NSTEMI (24,9%)	13 (7-21)	78.8 ± 20.3	42.4 ± 28.3%	2.7 ± 0.9	8 (3-16)	26.0 ± 23.2	3.9 ± 2.2	40.7%	3 (3-4)
	P	0.63	0.79	0.78	0.33	0.17	0.81	0.43	0.61	0.55
History of MI	Yes (12.7%)	10 (5-18)	79.7 ± 21.5	41.1 ± 27.8%	2.1 ± 0.8	6 (0-15)	16.7 ± 19.0	2.4 ± 2.3	40.4%	3 (2-4)
	No (87.3%)	12 (6-20)	77.1 ± 23.1	44.8 ± 29.2%	3.0 ± 1.0	10 (5-21)	28.4 ± 25.4	4.1 ± 2.0	46.3%	4 (3-4)
	P	0.23	0.55	0.45	< 0.01	< 0.01	< 0.01	< 0.01	0.71	0.01
Hyper-tension	Yes (90.1%)	12 (7-20)	78.5 ± 22.4	46.1 ± 28.8%	3.0 ± 1.1	10 (5-20)	29.3 ± 25.4	4.2 ± 2.0	48.5%	4 (3-4)
	No (9.9%)	12 (6-17)	86.0 ± 17.9	50.5 ± 30.5%	2.8 ± 0.9	8 (3-21)	23.4 ± 21.9	4.1 ± 1.8	47.1%	3 (3-4)
	p	0.15	0.06	0.39	0.35	0.48	0.20	0.54	0.95	0.48
Peripheral arterial disease	Yes (10.3%)	15 (10-20)	90.7 ± 15.3	68.2 ± 30.4%	3.1 ± 0.9	9 (2-16)	31.6 ± 26.6	5.1 ± 2.0	45.5%	3 (3-4)
	No (89.7%)	10 (5-17)	82.0 ± 20.3	46.2 ± 29.6%	2.7 ± 1.1	9 (2-19)	23.4 ± 22.4	3.4 ± 2.1	44.2%	3 (3-4)
	P	0.02	0.03	< 0.01	0.07	0.86	0.10	< 0.01	0.89	0.62
Smoking	At present (49.3%)	14 (7-21)	80.4 ± 20.5	48.0 ± 28.7%	3.1 ± 1.2	12 (6-22)	32.8 ± 27.0	4.5 ± 2.0	50.7%	4 (3-4)
	In the past (27.8%)	11 (5-20)	74.1 ± 23.4	41.1 ± 29.1%	2.6 ± 0.9	7 (2–17)	22.0 ± 22.5	3.2 ± 2.2	37.1%	3 (3-4)
	Never (22.9%)	10 (5-17)	75.1 ± 26.0	40.6 ± 28.6	2.6 ± 0.7	9 (3-18)	20.1 ± 19.1	3.3 ± 1.9	43.4%	3 (3-4)
	P	0.01	0.06	0.04	< 0.01	< 0.01	< 0.01	< 0.01	0.36	0.01
Diabetes mellitus	Yes (24.2%)	16 (7-22)	79.7 ± 20.5	46.3 ± 31.6%	2.8 ± 0.9	7 (2-14)	25.2 ± 23.7	4.3 ± 2.0	41.9%	3 (3-4)
	No (75.8%)	11 (6-20)	76.5 ± 23.6	43.3 ± 27.9%	2.9 ± 1.1	11 (4-22)	27.4 ± 25.1	3.8 ± 2.2	47.0%	4 (3-4)
	P	0.01	0.36	0.59	0.87	0.01	0.46	0.09	0.68	0.49

in men (mean 10.8%), in the control group examined by the above-mentioned author the number of edentulous patients was 2.5%. The majority of studies have reported on an association between the number of missing teeth and MI status [3, 31]. Research by Desvarieux et al. [9] on the relation between the number of teeth and progression of atherosclerosis and risk of myocardial infarction showed plaque in carotid arteries in 46% of individuals who had lost from 0 to 9 teeth and in 60% of individuals who had lost more than 10 teeth. Also, studies by Schillinger et al. [49] indicated that toothless patients had more advanced atherosclerotic lesions in carotid arte-

Table 2. Relationship between periodontal health and previous myocardial infarction or peripheral arterial disease (PAD) in patients with acute MI (χ^2 test)

Severity of periodontitis	PAD + (10.3%)	P	PAD – (89.7%)	Previous MI + (12.7%)	P	Previous MI – (87.3%)
Median CPI < 2	4.5%		17.9%	28.6%		9.0%
Median CPI 2 or 3	50.0%	p = 0.0187	57.6%	55.1%	p = 0.0005	59.2%
Median CPI >3	45.5%		24.5%	16.3%		31.8%

ries. Holmlund et al. [22] reported that individuals with more than 10 teeth demonstrated a 7-fold increase in the risk of mortality from a heart attack compared to persons with more than 25 teeth, as well as that more advanced atherosclerotic lesions in carotid arteries are present in individuals with fewer teeth. In own study the number of teeth in patients with diabetes was significantly lower than among non-diabetic group. Surprisingly, these patients presented lower number of active PPD > 4 mm, which may be related to higher tooth loss, suggesting more oral cavity sanitation procedures being performed in those suffering from diabetes.

We found a high prevalence of advanced periodontitis among patients with acute coronary syndrome. Only 12% of investigated subjects showed satisfactory periodontal status. The state of periodontal tissues evaluated by the number and percentage of patients with pockets above 4 mm and average CAL was higher compared to the control group in the study by Górski et al. [16], amounting to 14.3 vs 8.5 and 4.2 vs 2.5, respectively. No differences in periodontal status between patients with STEMI or NTEMI were detected. The recent metanalysis by Shi et al. found that MI patients had worse periodontal and oral hygiene status, as well as fewer teeth than did control subjects [50]. The differences in the means of PPD and CAL between MI patients and control subjects were 1.209 and 1.000 mm, respectively. Similarly, MI patients presented a higher level of plaque and BoP, which indicated poor oral hygiene. Likewise, they lost more than 4 teeth more than the control subjects (difference in means: 4.122, 95% CI: 2.012-6.232, p=0.000). Quite unexpectedly, however, periodontitis was more advanced in patients hospitalized due to the first myocardial infarction than in patients with a history of previous MI. The prevalence of severe periodontitis almost doubled in the former 31.8% vs 16.3% (p<0.01). A possible explanation of this finding could be that patients with a history of myocardial infarction received statins which can show a beneficial effect on periodontitis [19, 48]. By the same token, resolvins show an ability to regulate neutrophilic infiltration, downregulates cytokines and prostaglandins, stimulates macrophage-mediated phagocytosis of microbes and cellular debris [21, 41]. Moreover, there is also some data suggesting the potential preventive effect of low dose aspirin on periodontitis [11]. Most probably, patients with previous MI were more aware of CHD risk factors and were undertaking preventive measures, including control of oral hygiene. 10.3% of studied myocardial patients presented coexisting peripheral arterial disease and this group showed significantly more advanced signs of periodontitis when compared to patients with MI but without peripheral artery disease. They presented a higher number of lost teeth and increased BOP.

Disorders in lipidogram (increased TC, TG, LDL-C and decreased HDL-C) as well as in hsCRP concentration show strong correlations with the occurrence of CVD [10, 30]. Interestingly, we observed a positive rela-

Table 3. Laboratory tests based on periodontal status according to CPI values (Spearman rank test)

	Median CPI < 2 (periodontal health or signs of gingivitis) (11.8%)	Median CPI ≥ 2 (signs of periodontitis) (88.2%)	P
Total cholesterol (TC) mg/dl	180.4 (42.8)	203.7 (50.0)	0.0072
Low-density cholesterol (LDL) mg/dl	107.6 (36.5)	127.9 (43.3)	0.0069
Triglycerides (TG) mg/dl	162.0 (115.5)	161.3 (106.5)	0.3
hsC-reactive protein (CRP) mg/dl	20.6 (43.3)	25.0 (47.5)	0.0294
Accidental Plasma Glucose mg/dl	120.5 (48.3)	121.5 (53.3)	0.8
Fibrinogen mg/dl	366.9 (132.3)	361.4 (103.6)	0.7
NT-proBNP pg/ml	1477 (1489)	1189 (1424)	0.7

Table 4. Clinical periodontal status and selected laboratory test according to low density cholesterol (LDL) values intervals

	LDL < 80 mg/dl (17.3%)	LDL = 80-120 mg/dl (31.2%)	LDL > 120 mg.dl (51.5%)	P value (Kruskal-Wallis test)
Number of lost teeth	12 (8-21)	12 (5-21)	12 (6-20)	0.0535
PI	73.6 ± 26.3%	77.1 ± 24.0%	78.5 ± 21.1%	0.67
BOP	39.9 ± 29.0%	42.5 ± 27,.%	46.5 ± 28.3%	0.2
mean PD	2.8 ± 1.0	2.7 ± 1.0 [a]	3.1 ± 1.1 [a]	0.0124 [a]
PD≥4 mm + BOP	23.7 ± 25.5%	24.9 ± 25.5%	30.7 ± 24.6%	0.0211
mean CAL	3.7 ± 2.4	3.6 ± 2.4 [b]	4.3 ± 2.0 [b]	0.013 [b]
median CPI	3 (2-3)	3 (2-4)	3 (3-4)	0.0535
Plasma Glucose	122.1 ± 54.9	115.3 ± 53.8	120.5 ± 41.1	0.33
CRP	27.6 ± 63.1	21.5 ± 45.5	21.8 ± 36.8	0.15
Fibrinogen	273.0 ± 174.5	309.8 ± 158.1	346.5 ± 156.1	0.49

tionship between plasma total cholesterol, LDL cholesterol and hsCRP levels and periodontitis severity. Patients with severe periodontitis presented the highest concentrations of the above mentioned. This confirms findings of another study in which positive and significant correlations between PPD, CAL and lipid disorders and hsCRP concentration were observed, even after adjusting for confounders [12, 38, 43, 48]. CRP levels in patients with CHD and CP equaled 7.3 mg/L, in patients with periodontitis only 2.4 mg/L, and in individuals with healthy periodontium, 1.4 mg/L [35]. 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) [5].

In our study we observed a correlation between smoking and average pocket depth, the number and percentage pockets above 4 mm and an average loss of clinical attachment loss. The study also shows that most people who experienced a heart attack were past or present cigarette smokers. Results obtained by other authors confirm our data. Hyman et al. [26] demonstrated that smokers with concomitant advanced periodontitis were at an 8-fold higher risk of myocardial infarction. Tobacco smoking is undoubtedly the most important risk factor for both CVD and CP [14, 55]. Studies have shown that in the case of the death of a smoker before 50 years of age due to CVD, there is an 80% probability that such a death was caused by the addiction [18].

Another modifiable risk factor for both diseases is diabetes. Own study shows that 25% of the patients after myocardial infarction also suffered from coexisting diabetes. Wożakowska-Kapłon et al. [60] observed diabetes in 34% of MI patients younger than 60 years old. The risk of periodontitis progression in patients with poorly controlled diabetes is 11 times higher than in healthy controls [54]. Studies by Kannel and McGee [29] showed that diabetes increases the risk of fatal cardiovascular complications 1.7 times in men and 2.1 times in women. The INTERHEART study showed that diabetes increases the risk of MI 3.08 times and of mortality in patients with acute coronary syndrome [61]. On the other hand, studies by Gerstein et al. [13] indicated that the risk of MI rises with values of HbA1C > 5.4%, and any growth in hemoglobin concentration by one percentage point independently increases the risk of MI by additional 19%.

In our study group, more than 90% of patients with MI had arterial hypertension, which is a very important risk factor for cardiovascular disease, and recent studies increasingly indicate a relationship between the state of marginal periodontal tissues and blood pressure values [4]. This correlation appears to result from the outflow of bacteria associated with periodontitis, which may be responsible for the increase in both systolic and diastolic blood pressure through an immune reaction by activating T lymphocytes. The immune reaction may cause increased sensitivity of the body to the action of angiotensin II [8].

Our study has some limitations that need to be taken into account. First, it was an observational study and as such it did not allow us to draw definite conclusions on causality. Although all patients were treated according to current recommendations for acute coronary syndromes, their management was not adjudicated. Second, limited data on previous therapy was available. Third, while staying in the hospital patients might not have maintained their daily oral hygiene, which could contribute to higher plaque indices and BoP. Moreover, the correlations with BoP may be partially explained by the use of antiplatelet medications. More well-designed

and high-quality studies on the relationship between MI and periodontitis, especially those evaluating the effect of periodontal treatment on the reduction of MI events, should be carried out.

CONCLUSIONS

In summary, MI patients had poor periodontal health and more than 88% of them presented with CP. Moreover, severe periodontitis almost doubled in patients suffering from the first MI as compared to those with the second episode of acute MI. At the same time, total cholesterol, LDL and CRP levels were above the desirable range in this group of patients. Other mutual risk factors, such as age, smoking, arterial hypertension and diabetes mellitus, were also very prevalent. All of the abovementioned data may suggest that patients diagnosed with chronic periodontitis may possess a greater risk of developing cardiovascular diseases such as myocardial infarction. It remains to be seen whether the list of known risk factors for MI will be supplemented by periodontitis.

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