ORIGINAL ARTICLE

Association between central and peripheral blood pressure and periodontal disease in patients with a history of myocardial infarction

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KEY WORDS

cardiovascular risk, central blood pressure, myocardial infarction, periodontal disease, antibodies against *Porphyromonas gingivalis*

ABSTRACT

INTRODUCTION Central and peripheral blood pressure (BP) and periodontal disease (PD) are considered to be related to the risk of cardiovascular disease. However, there is scarce evidence on the association between chronic PD and BP.

OBJECTIVES The aim of the study was to assess the relation between chronic PD, antibodies against *Porphyromonas gingivalis (P.g.)* gingipains and central and peripheral BP in high-risk patients with previous myocardial infarction.

PATIENTS AND METHODS We examined 99 patients (71 men and 28 women) 6 to 18 months after myocardial infarction. The periodontal status was assessed using the Community Periodontal Index (CPI). BP was measured noninvasively using the Mobil-O-Graph device. Antibody titers against *P.g.* gingipains were determined by an enzyme-linked immunosorbent assay. The association between CPI and BP was assessed using logistic regression models.

RESULTS The mean age of participants was 60.5 ±8.7 years. After the adjustment for age, sex, smoking, diabetes, number of antihypertensive drugs, hypercholesterolemia, body mass index, and left ventricular ejection fraction, an association was found between central and peripheral BP and the CPI. Patients from the CPI 3 + 4 group were found to have almost 3 times higher odds of central BP of 130/90 mmHg or higher and more than 3 times higher odds of peripheral BP of 140/90 mmHg or higher compared with patients from the CPI 1 + 2 group.

CONCLUSIONS The severity of PD was associated with increased central and peripheral BP. The association between BP and PD may partially explain the cardiovascular risk related to chronic PD. Proteolytic activity of *P.g.* gingipains was not associated with BP.

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INTRODUCTION Increased blood pressure (BP) is independently associated with the incidence of cardiovascular events such as myocardial infarction (MI), stroke, sudden death, and peripheral arterial disease.[1,2] The prevalence of hypertension in patients with coronary heart disease (CHD) is high and exceeds 70%.[3] BP control is particularly important in patients with CHD as part of secondary prevention; however, it is unsatisfactory in this patient group.[4] Methods of treatment and prevention in patients with CHD have developed rapidly over the last few years. Despite this significant progress, still 25% of patients die within 2 years of acute coronary syndrome.[5]

Central BP is directly responsible for the load of the left ventricle and determines blood supply of the central nervous system. Moreover, it has direct impact on coronary and carotid artery walls, and its increase is one of the main causes of atherosclerosis in these arteries.[6] The high prognostic

value of central BP and central pulse pressure has been confirmed in a number of studies.[7,8] Central pulse pressure is considered to be the best predictor of cardiovascular events in coronary patients.[9]

The prevalence of periodontal disease (PD) in the general European population is estimated to be over 50%.[10] The most pathogenic and etiologic bacteria for PD are *Porphyromonas gingivalis (P.g.)*, *Bacteroides forsythus*, *Treponema denticola*, *Eikenella corrodens*, *Campylobacter rectus*, *Prevotella intermedia*, and *Fusobacterium nucleatum*.[11] *P.g.* is most closely associated with PD and can be detected in 85% of the sites with PD.[12] It has a number of potential virulence factors, such as cysteine proteinases (gingipains, lipopolysaccharide, capsules, and fimbriae).[13] Gingipains contribute to 85% of the total proteolytic activity of *P.g.*[14] As the strongest *P.g.* proteolytic enzyme, gingipains are the key factor responsible for inducing PD and have multidirectional adverse effects on the immune system.[15,16]

The impact of *P.g.* through gingipains may increase the overall inflammatory response, which in turn may lead to increased BP. In most studies on the association between PD and BP, the assessment of the periodontal status was based on the clinical examination or self-reporting. These methods are not capable of measuring a systemic immune response to periodontitis. Such response can be measured by elevated levels of antibodies against periodontopatic bacteria or its virulence factors. The advantage of such measurement is that the clinical manifestation of periodontitis may not reflect immunological susceptibility to a periodontal pathogen, which can vary among individuals.[17]

There is evidence on the relation between PD and an increase of peripheral BP,[18,19] although it has not been confirmed in all studies.[20,21] In addition, the relation between PD and BP cannot be explained by known mechanisms. The evidence on the relation between central BP and PD is scarce, with only 2 studies published so far. One of them showed a significant association in patients with hypertension but without cardiovascular disease,[22] and the other—in subjects with diabetes.[23] The latter study also showed impaired vascular health in subjects with chronic or aggressive periodontitis by evaluating pulse wave velocity, augmentation index , and pulse pressure amplification.[23]

So far, the relation between central BP and PD has not been studied in patients with a history of MI. Moreover, no study aimed at assessing the relation between antibody titer against *P.g.* gingipains and central BP in this patient group. Therefore, the aim of this study was to assess the relation between chronic PD and antibody titer against *P.g.* gingipains and central and peripheral BP in patients after MI.

PATIENTS AND METHODS The study group consisted of 99 patients. The inclusion criteria were as follows: hospitalization for MI (with ST-segment elevation on an electrocardiogram) within 6 to 18 months before enrollment to the study, age from 18 to 80 years, and having at least 6 teeth. Patients were recruited from 5 hospitals with cardiac departments, serving the area of Kraków, Poland. In each department, medical records of consecutive patients hospitalized from January 2010 to April 2012 for acute MI (first or recurrent, no prior percutaneous coronary intervention or coronary artery bypass grafting) were reviewed, and patients aged from 18 to 80 years were identified retrospectively, excluding those who died during their in-hospital stay. Medical records of patients fulfilling the inclusion criteria were analyzed using the standardized data collection form, and patients were subject to a dental examination. We recruited 161 patients who met the MI inclusion criterion. The periodontal condition was assessed in participants having at least 6 teeth, according to the World Health Organization (WHO) recommendations (62 patients had less than 6 teeth).[24] The exclusion criteria were as follows: the use of antibiotics during the 2 weeks before the study, immunosuppressive therapy, or chemotherapy.

Data on the history of ischemic heart disease, drug use, demographic characteristics, and the presence of risk factors were collected by an interview using a standard questionnaire. A dental clinical examination was conducted according to the WHO recommendations.[25] Participants were examined by a qualified dentist. A standard intraoral examination was performed, starting from the first to the fourth quadrant. The Community Periodontal Index (CPI) was used to record the periodontal status.[26] The CPI ranged from 0 to 4, with CPI 0 representing the healthy status; CPI 1, bleeding on probing; CPI 2, calculus or conditions for dental plaque retention; CPI 3, 4-mm to 5-mm pocket depth; and CPI 4, ≥6-mm pocket depth. Participants were divided into CPI groups according to the worst finding in any quadrant. Probing was performed on 6 surfaces of each tooth using a periodontal Hu-Friedy probe, type PCPUNC15 (Hu-Friedy Mfg. Co., Chicago, Illinois, United States).

Central and peripheral BP was measured using a Mobil-O-Graph NG oscillometer (IEM GmbH, Stolberg [Rheinland], Germany). The measurements were performed in a quiet room, in a sitting position, and on the right arm after a 10-minute rest. Participants were asked to be at least 30 minutes after consuming the last meal and at least 30 minutes after smoking a cigarette. Hypertension was defined as peripheral BP of 140/90 mmHg or higher and central BP of 130/90 mmHg or higher, as defined in a previous study.[27] A measurement with this device is considered to be reliable. Detailed information on the device and validation of the method was described earlier.[28-30]

Participants were considered smokers if they reported smoking at least 1 cigarette during the month preceding the interview or had at least 10 ppm of carbon monoxide in exhaled air. The concentration of carbon monoxide in exhaled air was

measured using the Smokerlyzer Micro+ (Bedfont Scientific Ltd, Harrietsham, England).

Patients were considered diabetic if they had been previously diagnosed with diabetes or had fasting glucose levels of 7.0 mmol/l or higher. Patients were considered as having hypercholesterolemia if they had been previously diagnosed with hypercholesterolemia or if they had total cholesterol levels of 5.0 mmol/l or higher or low-density lipoprotein cholesterol levels of 3.0 mmol/l or higher.

From each participant, 2 ml of blood were collected, and the serum was separated within 2 hours. Then, using the method described by Zdzalik et al,[31] immunoglobulin G antibody titers against *P.g.* gingipains were determined by an enzyme-linked immunosorbent assay (ELISA). Antibody titers were given in ELISA units. Patients were divided into subgroups according to an antibody titer tertile.

All participants were informed about the study protocol and gave their informed consent to participate in the study. The study was approved by the Bioethics Committee of Jagiellonian University.

Statistical analysis All analyses were done together for men and women. To compare the variables with normal distribution, the t test or analysis of variance was used. The Mann–Whitney or Kruskal–Wallis test was used to test differences between variables without normal distribution. To test differences between categorical variables, the χ^2 test was used. Logistic regression was used to assess the relation between the severity of PD and BP. The results were presented as odds ratios (ORs) with 95% confidence intervals (CIs) for peripheral and central pulse pressure above the median, peripheral BP of 140/90mmHg or higher, and central BP of 130/90 mmHg or higher in the CPI subgroups. The final covariates were age, sex, current smoking, diabetes, number of antihypertensive drugs, hypercholesterolemia, body mass index, and left ventricular ejection fraction. The analyses were performed using the statistical packet, SPSS Software (IBM, Armonk, New York, United States). Statistical significance was accepted at an α level of 0.05.

RESULTS The study sample included 71 men and 28 women. The mean age of participants was 60.5 ±8.71 years. The prevalence of diabetes was 25.3%. There were 16.2% of current smokers. The median number of teeth was 15 ±6.5. All participants took at least 1 hypertensive drug. The mean body mass index was 29.0 kg/m^2. Hypercholesterolemia was diagnosed in 69.4% of the participants. The mean left ventricular ejaculation fraction was 55.0%. Data are presente in **TABLE 1**.

The only participant representing the healthy periodontal status (CPI 0) was classified to the CPI 1 + 2 group. Central and peripheral BP increased with an increase of the CPI (**TABLE 1**). In the CPI 1 + 2 group, 25.5% of the participants had peripheral BP of 140/90 mmHg, and in the CPI 3 + 4 group—58.3%. In addition, in the CPI 1 + 2 group, 31.4% of the participants had central BP of 130/90 mmHg or higher, and in the CPI 3 + 4 group—62.5%. The CPI 1 + 2 and 3 + 4 groups differed significantly in the proportion of patients with high central and peripheral BP. There was no difference in central pulse pressure between the groups. Moreover, there were no differences in other demographic characteristics and cardiovascular risk factors between the groups. (**TABLE 1**).

The relation between BP and the CPI after multivariate adjustment is presented in **TABLE 2**. After adjustment for covariates, patients from the CPI 3 + 4 group were found to have almost 3 times higher odds of central BP of 130/90 mmHg or higher and more than 3 times higher odds of peripheral BP of 140/90 mmHg or higher, compared with patients from the CPI 1 + 2 group. There were no differences in the odds of higher pulse pressure between the CPI 1 + 2 and 3 + 4 groups. BP did not increase with the increasing tertiles of antibody titers (**TABLE 3**).

DISCUSSION Our results are in line with previous reports but also indicate a potentially stronger relation between the severity of PD and central BP. It should be underlined that we studied patients with a history of MI, that is, patients at very high risk. Franek et al[22] showed significant associations between central diastolic BP, left ventricular hypertrophy, and PD in patients with essential hypertension. Our study additionally showed an association between high peripheral and central BP and severity of PD. To our best knowledge, this is the first report showing the relation between PD and BP in high-risk patients with cardiovascular disease. In contrast to the study by Franek et al,[22] we used multivariate adjustments; therefore, we could show independent relations between BP and PD.

Our results are in agreement with previous reports on the association between peripheral BP and PD.[19,32,33] Rivas-Tumanyan et al[34] showed that after adjustment for covariates, a severe form of PD was associated with almost 3-fold higher risk for elevated BP (≥140/90 mmHg; OR, 2.93; 95% CI, 1.25–6.84). This association was even stronger when only patients with hypertension were studied (OR, 4.20; 95% CI, 1.28–13.80). This may suggest that the relation between PD and BP is similar in patients with and without atherosclerosis. On the contrary, studies on larger cohorts showed no association between BP and PD. A large study by Rivas-Tumanyan et al,[35] in which 31 543 participants of the Health Professionals' Follow-Up Study (HPFS) were prospectively followed up for 20 years for the outcome of hypertension, found no association between PD measures and incident hypertension in a cohort of middle-aged men. However, the weakness of this study was that the evaluation of hypertension was based on self-reported measurements. Further periodontal pocketing and gingival bleeding

TABLE 1 Associations between the Community Periodontal Index and demographic characteristics, blood pressure, and other cardiovascular risk factors

Parameter	CPI 1+2	CPI 3+4	*P* value	All
	n = 51	n = 48		n = 99
age, y	60.9 ±8.73	60.2 ±8.65	0.7	60.5 ±8.71
men	75.0%	70.1%	0.62	71.3%
years of education, y	11.0 (10.0–14.0)	12.0 (10.0–16.0)	0.65	11.5 (10.0–15.0)
BMI, kg/m^2	28.6 ±4.62	29.5 ±4.59	0.3	29.0 ±4.6
current smokers	23.5%	14.6%	0.26	16.2%
diabetes	25.0%	25.5%	0.95	25.3%
number of antihypertensive drugs	2 (2–2)	2 (1–2)	0.7	2 (2–2)
total cholesterol, mmol/l	4.23 ±0.965	4.22 ±0.885	0.96	4.23 ±0.929
LDL cholesterol, mmol/l	2.26 ±0.831	2.24 ±0.655	0.87	2.25 ±0.745
HDL cholesterol, mmol/l	1.32 ±0.33	1.30 ±0.348	0.7	1.31 ±0.337
triglycerides, mmol/l	1.2 (0.8–1.6)	1.3 (1.0–2.0)	0.13	1.30 (0.90–1.80)
glucose, mmol/l	5.5 (5.2–6.2)	5.7 (5.1–6.4)	0.32	5.6 (5.2–6.3)
hypercholesterolemia	68.0%	70.8%	0.76	69.4%
number of teeth	15 ±7.0	16 ±5.9	0.3	15 ±6.5
antibody titer	8100 (2700–24300)	8100 (8100–24300)	0.23	8100 (2700–24300)
LVEF, %	55.0 (50.0–60.0)	55.0 (50.0–60.0)	0.7	55.0 (50.0–60.0)
pSBP, mmHg	129.0 ±19.14	144.8 ±21.68	0.001	136.6 ±21.80
pDBP, mmHg	78.8 ±10.29	87.4 ±12.48	<0.001	83.0 ±12.14
pPP, mmHg	50.0 ±13.98	57.0 ±16.76	0.03	53.4 ±15.71
cSBP, mmHg	116.3 ±17.19	130.3 ±20.55	<0.001	123.1 ±20.06
cDBP, mmHg	80.6 ±11.01	92.3 ±12.49	<0.001	86.3 ±13.07
cPP, mmHg	34.5 ±13.04	39.5 ±15.59	0.08	36.9 ±14.49
pBP ≥140/90 mmHg	25.5%	58.3%	<0.001	41.4%
cBP ≥130/90 mmHg	31.4%	62.5%	0.002	46.5%

Data are presented as mean ± standard deviation, median (interquartile range), or percentage of patients.

Abbreviations: BMI, body mass index; cDBP, central diastolic blood pressure; CPI, Community Periodontal Index; cPP, central pulse pressure; cSBP, central systolic blood pressure; CVD, cardiovascular disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; n, number of participants; pDBP, peripheral diastolic blood pressure; pPP, peripheral pulse pressure; pSBP, peripheral systolic blood pressure

did not appear to be related to hypertension in nondiabetic, nonsmoking individuals aged from 30 to 49 years old (n = 1296).[36] In another study (n = 1643), additive effects of oral health markers on hypertension showed that, compared with subjects with no component of the oral health markers, the multivariate-adjusted odds ratio of hypertension in those with 3 components was 1.82 (95% CI, 1.23–2.72; *P* = 0.003). These results are partially in line with ours.

The exposure to bacteria etiologic for PD, including *P.g.*, was related to increased peripheral BP in 653 residents of New York City. Subgingival plaque was examined using DNA hybridization. Patients with the highest exposure to etiologic bacteria had more than 3-fold higher probability of hypertension.[18] This is the only published study showing a relation between the titers of antibodies against *P.g.* and the parameters of peripheral pressure. Subjects with antibody titers above the median had significantly higher peripheral BP. In contrary to previous studies, we studied the relation between cysteine proteinase, namely, gingipains produced by *P.g.*, and BP and did not detect a significant association. Moreover, we found no relation between antibody titers against *P.g.* gingipains and the CPI. It is possible that the association between periodontitis and BP is not mediated by the proteolytic activity of *P.g.* gingipains.

In our study, we used the CPI to determine the periodontal condition. The use of the CPI was criticized because of losing an important part of useful information due to: 1) limiting a recording to the worst score per sextant; 2) measurement of several parameters such as gingival bleeding, calculus, and periodontal pockets combined in the same index; and 3) underestimating the pockets greater than 6 mm in older age groups. On the other hand, the CPI is a universal index and allows a wide international comparison of collected data. It is also promoted to be used in epidemiological studies and allows for an effective examination of a population to determine the periodontal condition. In addition, the CPI is easy to use and is readily acceptable by patients.

Our study has several limitations. The cross-sectional nature of the study did not allow to

TABLE 2 Odds ratios for the association between the Community Periodontal Index and increased blood pressure

Parameter		Model A[a]	Model B[b]
		OR (95% CI)	OR (95% CI)
pBP ≥140/90 mmHg	CPI 1 + 2	1	1
	CPI 3 + 4	4.10 (1.78–9.86)	3.38 (1.30–9.42)
cBP ≥130/90 mmHg	CPI 1 + 2	1	1
	CPI 3 + 4	3.77 (1.64–9.02)	2.90 (1.14–7.66)
pPP > median	CPI 1 + 2	1	1
	CPI 3 + 4	2.29 (1.00–5.43)	1.77 (0.65–4.90)
cPP > median	CPI 1 + 2	1	1
	CPI 3 + 4	1.85 (0.82–4.20)	1.37 (0.50–3.78)

a adjusted for age and sex

b adjusted for age, sex, current smoking, diabetes, number of antihypertensive drugs, hypercholesterolemia, body mass index, and left ventricular ejection fraction

Abbreviations: cBP, central blood pressure; CI, confidence interval; OR, odds ratio; pBP, peripheral blood pressure; others, see TABLE 1

TABLE 3 Mean values of central, peripheral, and pulse pressure by tertiles of antibody titers

	pSBP, mmHg	pDBP, mmHg	cSBP, mmHg	cDBP, mmHg	cPP, mmHg	pPP, mmHg
tertile 1	137.2 ±18.1	82.0 ±9.4	122.6 ±17.7	83.8 ±11.0	38.1 ±13.6	55.3 ±13.5
tertile 2	141.5 ±25.0	84.4 ±13.7	128.0 ±22.8	89.7 ±15.0	38.7 ±16.8	56.5 ±17.6
tertile 3	132.8 ±20.9	83.0 ±12.8	119.6 ±19.3	85.7 ±12.8	33.8 ±13.5	49.5 ±15.1
P value	0.256	0.750	0.234	0.208	0.332	0.149

Data are presented as mean ± standard deviation.

Abbreviations: see TABLE 1

address the problem of the causal association between the variables. Our results could be confounded by the use of BP-lowering drugs by study participants. However, for ethical reasons, it was not possible to ask participants to stop taking antihypertensive drugs. Another limitation is that BP was measured only once. Although it is an accepted method in epidemiological studies, our measurements could be affected by strong within-person variation. However, despite this, we found a significant relation between periodontitis and BP, which supports the hypothesis that chronic inflammation may be related to a more stable increase in BP or even higher risk of hypertension. In future studies, it would be worth using 24-hour BP monitoring to evaluate the effect of PD on BP more thoroughly. Furthermore, we targeted patients with CHD, who were otherwise healthy, and patients who did not take antibiotics or drugs that seriously affect inflammatory response, but cases of silent or hidden inflammation might have affected our results. Finally, we studied a relatively small number of participants, so the statistical power of our sample is limited.

In conclusion, there is a similar association between PD and central and peripheral BP. The association between PD and BP may partially explain the cardiovascular risk related to chronic PD. High antibody titer against *P.g.* gingipains is not related to central or peripheral BP.

Contribution statement RŁ, RW-W, and AŁ designed the study and collected the data. RŁ, PJ, MP, KS, AM, and AP were responsible for data interpretation. MP, KS, and AM performed the statistical analysis of the data. RŁ, PJ, TW, DC, and AP made the final revision of the manuscript. KS and AP coordinated funding for the project. All authors edited and approved the final version of the manuscript.

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Związek między centralnym oraz obwodowym ciśnieniem tętniczym a chorobą przyzębia u pacjentów po zawale mięśnia sercowego

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SŁOWA KLUCZOWE

centralne ciśnienie tętnicze, choroba przyzębia, przeciwciała przeciw *Porphyromonas gingivalis*, ryzyko sercowo-naczyniowe, zawał mięśnia sercowego

STRESZCZENIE

WPROWADZENIE Uznaje się, że centralne i obwodowe ciśnienie tętnicze (*blood pressure* – BP) oraz choroba przyzębia związane są z ryzykiem chorób sercowo-naczyniowych, jednak dowody na związek pomiędzy przewlekłą chorobą przyzębia a BP są ubogie.

CELE Celem badania była ocena związku między przewlekłą chorobą przyzębia, przeciwciałami przeciw gingipainom *Porphyromonas gingivalis* (*P.g.*) a centralnym i obwodowym BP u pacjentów wysokiego ryzyka po wcześniejszym zawale mięśnia sercowego.

PACJENCI I METODY Przebadano 99 pacjentów (71 mężczyzn i 28 kobiet) 6–18 miesięcy po zawale mięśnia sercowego. Stan przyzębia oceniano przy pomocy wskaźnika Community Periodontal Index (CPI). BP mierzono w sposób nieinwazyjny przy użyciu urządzenia Mobil-O-Graph. Miana przeciwciał przeciw gingipainom *P.g.* oznaczano za pomocą testu immunoenzymatycznego. Związek pomiędzy CPI a BP oceniano za pomocą modeli regresji logistycznej.

WYNIKI Średnia wieku uczestników wynosiła 60,5 ±8,7 roku. Po uwzględnieniu wieku, płci, palenia tytoniu, cukrzycy, liczby leków przeciwnadciśnieniowych, hipercholesterolemii, wskaźnika masy ciała oraz frakcji wyrzutowej lewej komory stwierdzono zależność pomiędzy centralnym i obwodowym BP a CPI. Pacjenci z grupy CPI 3+4 mieli prawie 3 razy większe szanse na centralne BP ≥130/90 mm Hg i >3 razy większe szanse na obwodowe BP ≥140/90 mm Hg w porównaniu z pacjentami z grupy CPI 1+2.

WNIOSKI Zaawansowanie choroby przyzębia związane było z podwyższonym centralnym oraz obwodowym BP. Związek między BP a chorobą przyzębia może częściowo tłumaczyć wzrost ryzyka sercowo-naczyniowego związanego z przewlekłą chorobą przyzębia. Aktywność proteolityczna gingipain *P.g.* nie była związana z BP.

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