Association Between Periodontal Disease and Left Ventricle Mass in Essential Hypertension

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Abstract—Chronic periodontitis has been associated with an increased risk for cardiovascular disease. Left ventricular mass is an established independent predictor of cardiovascular disease. In the present cross-sectional study, we tested the association between periodontitis and left ventricular mass in subjects with essential hypertension. One hundred four untreated subjects with essential hypertension underwent clinical examinations, including echocardiographic study, laboratory tests, and assessment of periodontal status according to the community periodontal index of treatment needs (CPITN). With increasing severity of periodontitis, there was a progressive increase in left ventricle mass. Mean values (g/height$^{2.7}$) were 39.0 ($\pm$2.7) in CPITN 0 (periodontal health), 40.2 ($\pm$6.4) in CPITN 1 (gingival bleeding), 42.7 ($\pm$6.8) in CPITN 2 (calculus), 51.4 ($\pm$11.7) in CPITN 3 (pockets 4 to 5 mm), and 76.7 ($\pm$11.3) in CPITN 4 (pockets $\geq$6 mm) (overall F 51.2; $P<0.0001$). Body surface area ($P=0.04$), systolic ($P<0.0001$) and diastolic ($P<0.01$) blood pressure, and left ventricular mass ($P<0.0001$) were determinants of a composite of CPITN 3 and 4. In a multivariate logistic analysis, left ventricular mass was the sole determinant ($P<0.0001$) of CPITN stages 3 and 4. Our findings suggest a direct association between severity of periodontitis and left ventricular mass in subjects with essential hypertension. Periodontal evaluation might contribute to refine cardiovascular risk assessment in hypertensive subjects. **(*Hypertension*. 2003;41:488-492.)**

Key Words: hypertension, essential ■ left ventricular hypertrophy ■ periodontal disease ■ left ventricular mass ■ echocardiography

Periodontium is a complex and highly specialized pressure-sensing system consisting of 4 tissues (cementum, periodontal ligament, alveolar bone, and junctional and sulcular epithelia) supporting the teeth. Of these structures, periodontal ligament is a dynamic tissue with a high rate of remodeling and turnover, which connects the teeth to the alveolar bone.[1]

Prevalence of periodontal disease approaches 14% over a wide age span, including younger and elderly people.[1] Periodontitis begins with a loss of alveolar bone and subsequent formation of a pocked around the tooth, the final stage being tooth mobility and loss.[2] Periodontal pocket can be detected with a periodontal probe and estimated through measurement of distance from gingival margin to the base of the periodontal pocket.[3] In a healthy periodontium, there is no loss of epithelial attachment or pocket formation, and the gingival crevice is $<$2 mm deep.[3] Established risk factors for periodontal disease are dental plaque, calculus, age, genetics, smoking, and diabetes.[4]

At least 9 cohort studies[5–13] examined the association between periodontal disease and coronary heart disease (CHD), with conflicting results. An overview of these studies[14] showed a 15% excess risk of CHD in association with periodontal disease, with 95% confidence intervals ranging from 8% to 122%. To define the underlying mechanisms of such association, several studies, reviewed in depth by Armitage,[15] examined the potential link between periodontal disease and cardiovascular risk factors, including diabetes, smoking, hyperlipidemia, and hypertension. Surprisingly, despite the high prevalence of hypertension in the general population and its leading prognostic importance, few data are available on the relation between elevated blood pressure (BP), hypertensive organ damage, and periodontal disease. Castelli et al[16] found a proliferation of the intima and elastic layers with lumen reduction of vessels feeding the periodontal membrane in hypertensive subjects.[16] In another study, tooth position and movements were affected by the force of BP transmitted through periodontal vessels.[17] Interestingly, periodontal pulsation reflected changes in pulse pressure rather than in mean BP.[17]

Left ventricular (LV) mass is abnormally increased in about one third of people with hypertension,[18] and LV hypertrophy is associated with an excess risk of cardiovascular complications independently of BP and other risk factors.[18] There is also growing evidence that serial changes in LV hypertrophy are independent predictor of outcome.[19,20]

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In view of the high prevalence and the potential interrelationship among elevated BP, cardiac organ damage, and periodontal disease as determinants of cardiovascular risk, we conducted the present study to examine the relation of periodontal disease to BP and LV mass (LVM) in a cohort of subjects with essential hypertension. To eliminate any interference by previous treatment on LVM,[21] only never-treated subjects were included.

Methods

We studied 104 consecutive hypertensive subjects (mean age, 57±10) with a clinic systolic BP ≥140 mm Hg and/or diastolic BP ≥90 mm Hg in 3 different visits over 1 month. None of the subjects had been previously treated or screened for hypertension. We used the average of 3 consecutive BP readings taken at distance of ≈1 minute. None of the subjects had secondary hypertension, important concomitant disease, or heart failure on the base of clinic visit, medical histories, physical examinations, 12-lead ECG, laboratory test, and echocardiographic study.

To exclude a possible misleading factor, another inclusion criteria was a history of good oral hygiene. Each subject used to brush the tooth at least twice daily, generally with fluoride toothpaste. Subjects made also regular use of flossing in order to achieve a more effective interdental, and most of them were examined by a dentist at every 6 to 12 months, following accurate preventive instructions for low intake of hypercaloric food and limiting the use of cigarettes.

Echocardiography

The M-mode echocardiographic study of LV was performed according to the American Society of Echocardiography recommendations.[22] The mean value from at least 5 measurements was computed. Relative wall thickness was calculated as twice the end-diastolic posterior wall thickness divided by end-diastolic diameter. LVM was calculated according to Devereux et al[23] and normalized by height elevated to 2.7 ($LVM/h^{2.7}$) according to De Simone et al.[24]

Dental Examination

To assess periodontal condition we used the World Health Organization (WHO) community periodontal index of treatment needs (CPITN) and the specially designed WHO periodontal probe with a sensing force of not >20 g.[25] Briefly, the mouth of each patient was divided into sextants (see Figure 1 for details), and each sextant was examined only if there were ≥2 teeth present and not indicated for extraction; the teeth examined were 17, 16, 11, 26, 27, 47, 46, 31, 36, and 37; for each sextant we recorded the highest index found according to the following score: 0, periodontal health; 1, gingival bleeding; 2, calculus detected during probing; 3, pocket 4- to 5-mm depth; and 4, pocket ≥6-mm depth. Periodontal condition of every patient is reported as the worst sextant CPITN condition.

Data Analysis

We used SPSS, lease 9.0 (SPSS Inc) for statistical analysis. Data are presented as mean (±SD) or frequency in percentage. Differences between groups were performed by 1-way ANOVA or χ^2 analysis when appropriate. Univariate and multiple logistic regression analyses were used to test the relationship of clinical and echocardiographic parameters to moderate-to-severe periodontitis and to determine the probability of a composite of moderate-to-severe periodontitis expressed by a CPITN 3 or 4. Thus, CPITN stage 3 or 4 was coded as 1, and CPITN stage 0, 1, or 2 was coded as 0 in the logistic regression analysis. In 2-tailed tests, $P<0.05$ were considered significant.

Results

The main demographic, clinical, and echocardiographic characteristics of the study population and their distribution among the 5 classes of periodontal condition is reported in

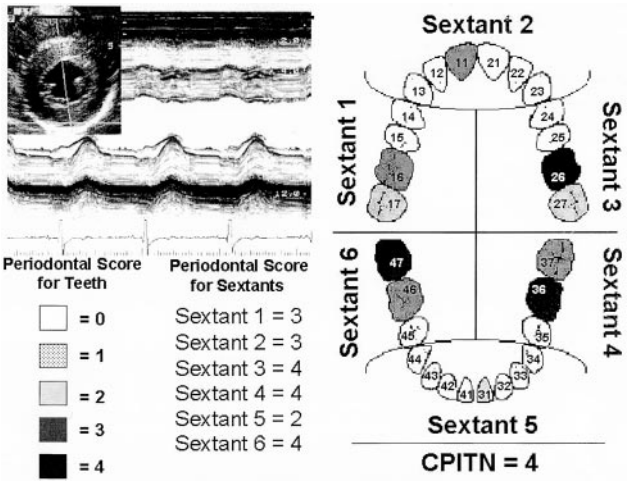

Figure 1. Periodontal status by CPITN in a patient with LV hypertrophy. The mouth is divided into sextants defined by tooth numbers 14, 15, 16, 17, and 18 for sextant 1; tooth numbers 11, 12, 13, 21, 22, and 23 for sextant 2; tooth numbers 24, 25, 26, 27, and 28 for sextant 3; tooth numbers 35, 36, 37, and 38 for sextant 4; tooth numbers 31, 32, 33, 34, 41, 42, 43, and 44 for sextant 5; and tooth numbers 45, 46, 47, and 48 for sextant 6. The first step is attribution of a score to each tooth considered (ie, 17, 16, 11, 26, 27, 47, 46, 31, 36, and 37); the second is attribution to each sextant of the highest score from each single teeth. Finally, periodontal status is reported as the worst sextant CPITN condition.

Table 1. Age, body surface area, heart rate, glucose, and total cholesterol did not differ between the 5 classes. Systolic and diastolic BP gradually increased from CPITN 0 to CPITN 4 ($P<0.0001$ and $P<0.05$, respectively). LVM and prevalence of LVH (defined as $LVM/h^{2.7}$ >49.2 $g/m^{2.7}$ for men and >46.7 $g/m^{2.7}$ for women) progressively increased from CPITN 0 to CPITN 4 ($P<0.0001$). Also interventricular septum thickness ($P<0.0001$), LV internal diameter ($P<0.0001$), and LV posterior wall thickness ($P<0.0001$) increased from CPITN 0 to CPITN 4.

In a logistic regression analysis (Table 2), we tested the univariate association between each single clinical and echocardiographic variable with moderate-to-severe periodontal disease as indicated by a composite of CPITN 3 to 4. Body surface area, systolic and diastolic BP, interventricular septum, LV posterior wall, LV internal diameter, relative wall thickness, and LVM showed an association (all, $P<0.05$) with moderate-to-severe periodontal disease. In a multivariate logistic regression analysis, only LVM (Table 3) maintained an independent association ($P<0.0001$) with periodontal disease CPITN 3 to 4. Body surface area, systolic BP, and diastolic BP did not achieve significance to enter the model. In an additional model that included interventricular septum ($P=0.076$), LV internal diameter ($P=0.168$), posterior wall thickness ($P=0.303$), and LVM ($P=0.028$), only LVM achieved significance.

The progressive increase in LVM with periodontal status and the prevalence of moderate-to-severe periodontitis in the 4 quartiles of LVM (<39.0, 39.0 to 41.9, 42.0 to 53.9, ≥54.0) are depicted in Figure 2. Subjects in the fourth quartile of $LVM/height^{2.7}$ showed the highest probability (85%) of moderate-to-severe periodontitis.

TABLE 1. Main Demographic and Clinical Characteristics and Their Distribution Into the Study Population Stratified by Periodontal Status

Variable	Total Population (N=104)	CPITN 0 Periodontal Health (n=7)	CPITN 1 Gingival Bleeding (n=37)	CPITN 2 Calculus (n=17)	CPITN 3 Pockets, 4–5 mm (n=28)	CPITN 4 Pockets, ≥6 mm (n=15)	F for Trend	*P*
Age, y	57 (10)	56 (9)	57 (11)	57 (9)	55 (11)	62 (6)	0.12	NS
Body surface area, m^2	1.85 (0.22)	1.75 (0.18)	1.78 (0.17)	1.90 (0.19)	1.90 (0.27)	1.89 (0.16)	2.41	NS
Gender, % men	53	30	48	46	50	45	—	NS
Cigarette smoking, %	48	30%	56%	46%	41%	44%	—	NS
Office systolic BP, mm Hg	153 (19)	143 (22)†	147 (15)†	149 (11)*†	162 (19)	172 (16)	8.92	<0.01
Office diastolic BP, mm Hg	96 (11)	94 (5)	95 (9)	91 (8)	99 (13)	101 (11)	2.51	<0.05
Office HR, beats/min	73 (8)	72 (11)	73 (8)	76 (3)	70 (7)	76 (8)	1.60	NS
Glucose, mmol/L	5.63 (1.02)	5.77 (0.53)	5.72 (1.37)	5.31 (0.45)	5.71 (0.78)	5.58 (1.10)	0.55	NS
Total cholesterol, mmol/L	5.65 (1.03)	5.45 (0.80)	5.44 (0.81)	6.12 (1.02)	5.49 (1.26)	5.98 (1.04)	1.95	NS
Triglycerides, mmol/L	1.53 (0.80)	1.09 (0.48)	1.81 (0.92)	1.26 (0.67)	1.37 (0.77)	1.68 (0.52)	2.744	<0.04
Interventricular septum, cm	1.14 (0.25)	0.98 (0.14)†	1.02 (0.24)*†	1.07 (0.23)†	1.20 (0.17)†	1.45 (0.12)	14.59	<0.0001
LV internal diameter, cm	5.01 (0.55)	4.47 (0.35)*†	4.83 (0.44)†	5.02 (0.53)†	5.05 (0.57)†	5.66 (0.24)	10.92	<0.0001
LV posterior wall, cm	0.98 (0.18)	0.91 (0.19)†	0.86 (0.14)*†	0.97 (0.15)†	1.06 (0.18)	1.16 (0.14)	14.03	<0.0001
Relative wall thickness	0.39 (0.08)	0.41 (0.04)	0.36 (0.08)*	0.39 (0.07)	0.43 (0.09)	0.41 (0.05)	3.48	<0.02
LV mass/$h^{2.7}$, g/$m^{2.7}$	48.82 (15.16)	39.05 (2.65)*†	40.20 (6.36)*†	42.69 (6.76)*†	51.41 (11.72)†	76.77 (11.26)*	51.23	<0.0001
LV hypertrophy, %	31	0	8	6	50	93	—	<0.0001

Data are expressed as mean (±SD). BP indicates blood pressure; HR, heart rate; LV, left ventricular.
*P<0.05 vs CPITN 3; †P<0.05 vs CPITN 4.

Discussion

The present study is the first to disclose an independent association between LVM detected by echocardiography and periodontal disease severity, defined by the CPTIN, in untreated subjects with essential hypertension. In a multivariate analysis, LVM was superior to systolic and diastolic BP for prediction of moderate-to-severe periodontitis.

TABLE 2. Results of Univariate Logistic Regression Analyses to Test the Relationship of Clinical and Echocardiographic Parameters to Moderate-to-Severe Periodontal Disease

Variable	β	SE	*P*
Age, y	0.035	0.020	0.088
Body surface area, m^2	2.090	1.008	0.038
Gender, % men	0.18	0.399	0.768
Cigarette smoking, %	0.251	0.400	0.531
Office systolic BP, mm Hg	0.060	0.015	<0.0001
Office diastolic BP, mm Hg	0.054	0.020	<0.01
Office HR, beats/min	−0.037	0.027	0.184
Glucose, mmol/L	0.048	0.194	0.804
Total cholesterol, mmol/L	0.026	0.194	0.804
Triglycerides, mmol/L	0.148	0.255	0.561
Interventricular septum, cm	5.924	1.245	<0.0001
LV internal diameter, cm	1.524	0.414	<0.001
LV posterior wall, cm	7.910	1.706	<0.0001
Relative wall thickness	8.048	2.849	<0.01
LV mass/$h^{2.7}$, g/$m^{2.7}$	0.150	0.031	<0.0001

Periodontitis and Cardiovascular Disease

Over the past few years, several epidemiologic studies examined the potential association between periodontal and cardiovascular disease.[5–13] Infections of periodontal structure could accelerate atherosclerosis by promoting a systemic inflammatory status through release of endotoxins, heat-shock proteins, or acute-phase reactants.[26,27]

Periodontitis and cardiovascular disease might also share some explanatory risk factors such as diabetes mellitus, smoking, and dyslipidemia.[15] Poor glycemic control is an established risk factor for periodontitis, and patients with diabetes mellitus are at increased risk of edentulia.[28] A possible basic mechanism could be the thickening of basement membranes of small vessels in diabetics, leading to reduced transport of oxygen and nutritional substances across the vessels walls.[28] Cigarette smoking is also associated with periodontitis,[29,30] and the underlying mechanisms include an inhibited neutrophil function in saliva and connective tissue, as well as a defective Ig2 antibody response.[29,30] Part of the

TABLE 3. Multiple Logistic Regression Analysis to Test Independent Determinants of Moderate-to-Severe Periodontal Disease Expressed by CPITN 3 or 4

Variable	β	SE	*P*
Constant	−11.60	3.586	0.001
Body surface area, m^2	0.403	1.385	0.771
Office systolic BP, mm Hg	0.015	0.020	0.468
Office diastolic BP, mm Hg	0.030	0.029	0.299
LV mass/$h^{2.7}$, g/$m^{2.7}$	0.143	0.036	<0.0001

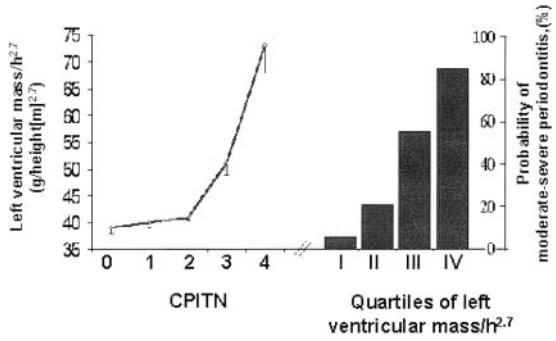

Figure 2. Distribution of mean LVM (±SE) into the study population stratified by periodontal status (CPITN) and probability of moderate-severe periodontitis according to quartiles of LVM.

association between periodontitis and CHD has been attributed to cigarette smoking as a confounding factor.[31] The association between elevated LDL cholesterol and periodontal disease seems to hold mostly in the male sex.[31]

Periodontitis and Hypertension

Despite the high prevalence of hypertension in the general population and its leading prognostic importance, only a few studies addressed the relation between BP and periodontal disease.[16,17] In the present study, systolic BP progressively increased with severity of periodontal disease, whereas diastolic BP did not show any significant changes. LVM showed also a progressive increase with the severity of periodontal disease. Because of the likelihood of residual confounding, we tested whether the association between LVM and periodontal disease was independent of BP. Indeed, the multivariate logistic regression analysis showed that LVM was an independent predictor of moderate-to-severe periodontitis.

The potential mechanisms underlying the association between LVM and periodontitis remain speculative. In hypertensive subjects, hypertrophic heart and periodontium may share microcirculatory dysfunction and arteriolar and capillary rarefaction.[32,33] Pressure overload could be involved at a time in inducing development of LV hypertrophy and generalized narrowing of the luminal diameter of microvessels. The resulting vascular rarefaction may lead to ischemia at cardiac and periodontal level. Because office BP determined by conventional sphygmomanometry is subject to important intrasubject and intersubject variability, increased LVM and periodontal disease may represent more stable biological assays, reflecting chronically elevated BP levels. Insulin resistance might be another potential mechanism involved in the pathogenesis of LV hypertrophy in subjects with periodontitis, but further research is needed to verify such hypothesis.[34] In a previous study from our laboratory, insulin and insulin growth factor I were independent determinants of LVM and geometry in subjects with essential hypertension, and such relation was independent of office and ambulatory BP.[35] Unfortunately, insulin resistance was not assessed in the present study.

Some limitations of this study deserve mention. Ambulatory BP, which is more accurate than office BP to predict the degree of LV hypertrophy,[36] was not measured. Use of ambulatory BP might provide a more restrictive assessment of the association between LVM and periodontal disease. Furthermore, because all subjects were white, caution is needed in extrapolating results to different ethnic groups.

Clinical Implications

Our findings have clinical implications. It is widely established that LVM is associated with an increased risk of a variety of adverse cardiovascular events independently of BP levels.[18,37,38] Our data suggest that periodontal disease can provide an easily accessible biological assay for a more accurate definition of cardiovascular risk profile in individual subjects. Thus, a simple periodontal evaluation might be useful to refine cardiovascular risk assessment in hypertensive subjects.

Perspectives

We found an association between periodontal disease, expressed by the CPITN and LVM in untreated subjects with essential hypertension. Such association was independent of BP and other determinants of LVM. Although the present study was not designed to investigate the potential mechanisms underlying the association between periodontal disease and LVM, it is tempting to speculate that pressure overload might accelerate microcirculatory changes leading to capillary rarefaction and subsequent ischemia at both periodontal and cardiac level. Periodontal disease could provide an easily accessible biological assay for a more accurate definition of cardiovascular risk profile in individual subjects.

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