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Comparison of Frequency and Duration of Periodontal Disease with Progression of Coronary Artery Calcium in Patients with Type 1 Diabetes Mellitus versus Non-Diabetics

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Abstract

People with type 1 diabetes mellitus manifest a higher burden of both periodontal disease and coronary artery disease (CAD); however, little is known about their interrelation. Coronary artery calcium (CAC) measures subclinical atherosclerosis and predicts major adverse coronary events. The relation between periodontal disease and CAC progression in individuals with type 1 diabetes has not been previously described. We determined the prevalence and progression of CAC in relation to self-reported periodontal disease. Multivariate logistic and tobit regression models were used to examine the relation between periodontal disease duration and CAC progression and whether this relation differs by diabetes status after controlling for age, sex, total and high-density lipoprotein (HDL) cholesterol, hypertension, smoking, body mass index (BMI), duration of diabetes, and baseline CAC. A total of 473 individuals with type 1 diabetes and 548 non-diabetic individuals were followed for a mean of 6.1 years. At baseline, the prevalence and duration of periodontal disease did not differ between individuals with and without diabetes (14.5% vs. 13.4%, $p=0.60$; 6 vs. 9 years, $p=0.18$). Duration of periodontal disease was not significantly associated with baseline CAC prevalence. In type 1 diabetic individuals, periodontal disease duration was significantly related to CAC progression ($p=0.004$) but not among non-diabetic individuals ($p=0.63$). In conclusion, this study suggests that periodontal disease is an independent predictor of long-term progression of CAC in type 1 diabetic individuals.

Keywords

diabetes mellitus; periodontal disease

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Conflicts of Interest: none

Coronary artery calcium (CAC) is an established marker of atherosclerotic plaque burden that is increased in patients with type 1 diabetes.^{1–2} To date, the influence of periodontal disease on CAC progression in type 1 diabetic individuals has not been established. The Coronary Artery Calcification in Type 1 Diabetes (CACTI) Study was used to examine the relation between periodontal disease and CAC progression over prolonged follow-up. We hypothesized that periodontal disease would be increased in type 1 diabetic individuals compared with non-diabetic individuals, and that the presence and duration of periodontal disease would predict CAC progression.

Methods

Subjects were identified from the CACTI Study. CACTI is a prospective cohort study examining the prevalence of CAC in type 1 diabetic individuals and a comparable group of controls with no history of atherosclerotic cardiovascular disease. Detailed descriptions of the study design have previously been published.² Participants in this study included those who had completed the 6-year (range: 4.1–8.5 years) follow-up visit and had complete data for all covariates (n=1,021). Informed consent was provided by all study participants and the protocol was reviewed and approved by the Colorado Multiple Institutional Review Board.

Physical exam measurements included height, weight, waist and hip circumference, and systolic and diastolic blood pressure. Hypertension was defined as systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg, or treatment with anti-hypertensive medication. A fasting blood sample was collected and stored at -80° C until assayed for measurement of total cholesterol, HDL, and triglyceride levels. Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald equation.³ All subjects were given standardized questionnaires to obtain demographics, medical history, medication use, and current and past smoking status. Subjects were asked if they had ever been told by a dentist or dental hygienist that they have gum disease. If yes, then they were asked the age of diagnosis. Self-reported periodontal disease has been evaluated in other studies and demonstrated to be reasonably accurate.^{4,5}

Two sets of images for CAC scoring were obtained between March 2000 and May 2002 using an ultrafast Imatron C-150XLP electron beam computed tomography scanner (Imatron, San Francisco, CA). The estimated radiation dose for each exam was 2.25 mSv. The CAC score was assessed using the Agatston method and the total volume score using the volumetric method was calculated from the images, as described previously.⁶ Follow-up scans were performed approximately 6 years after the baseline visit between February 2006 and March 2009. Prevalence of CAC was defined as a CAC score >0 . Progression of CAC was defined as the change in the square-root transformed volume score between the follow-up visit and the baseline visit. We used the difference in the square-root transformed volume for all analysis.

Differences in characteristics at baseline and follow-up were compared by diabetes status. Parametric continuous data are presented as means and standard deviation. Non-parametric data are presented as the median and interquartile range with the exception of the CAC scores. CAC scores were presented as the prevalence of positive CAC (>0) and as the

median and interquartile range of only the positive values. Categorical data were tabulated as the number of subjects and the percent. Statistical testing to detect differences between groups included the t-test for parametric continuous data, the Wilcoxon rank sum test for non-parametric data, and the chi-square test for categorical data.

Logistic and tobit regression were used to determine the independent effect of periodontal disease presence at baseline and duration on the prevalence and progression of CAC. Previous data suggests that values of change in the square-root transformed volume of CAC >2.5 are robust to interscan variability.⁶ Therefore, we used tobit regression for the progression of CAC models to left censor values of change (<2.5) that may represent interscan variability while estimating linear parameters for values representing significant progression (≥ 2.5). Tobit models assume the presence of a latent outcome variable that equals the observed outcome variable above the censored value, in this case, 2.5 , and is unobserved at or below the censored value. The models use maximum likelihood estimation to linearly regress the latent outcome variable on the covariates, and therefore, the beta coefficients from these models provide an estimate of the linear relationship between the independent variables in the model and the latent outcome variable. Potential confounding variables were considered for inclusion in the models based on a priori criteria: significance in previous studies or confounding the association between the main variable of interest and the outcome by $>10\%$. Age at baseline, sex, total cholesterol, HDL cholesterol, hypertension, smoking, body mass index (BMI), duration of type 1 diabetes, prevalence of periodontal disease, and baseline CAC were included in the final models. Additional variables that were considered for inclusion but not found to meet the a priori criteria were: race, Hispanic ethnicity, years of follow-up, waist circumference, waist-to-hip ratio, triglycerides, on lipid lowering drugs, high sensitivity C-reactive protein (hsCRP), and adiponectin. All analyses were performed using [SAS/STAT] software, Version [9.3] of the SAS System for [Windows] (SAS Institute Inc., Cary, NC, USA).

Results

A total of 473 individuals with established type 1 diabetes and 548 non-diabetic individuals were assessed. Participant characteristics at baseline stratified by diabetes status are presented in Table 1. Those individuals with diabetes were younger and more likely to be receiving medications for hypertension and dyslipidemia. Corresponding diastolic blood pressure, total cholesterol, LDL cholesterol, and triglycerides were lower among those with diabetes compared to those without diabetes. Gender, smoking status, BMI, and hsCRP did not differ between the groups. At baseline, the prevalence and duration of periodontal disease was similar between those with and without diabetes. However, at the 6-year follow-up, the prevalence of periodontal disease was significantly higher in those with diabetes compared to those without diabetes. CAC prevalence, extent, and progression were higher in those with diabetes compared to those without at both the baseline and follow-up exams.

The median CAC score for positive values stratified by diabetes and periodontal disease status is shown in Figure 1. For those with diabetes, subjects with baseline periodontal disease had higher median CAC scores at both the baseline ($p<0.001$) and follow-up

($p=0.002$) examinations. For subjects without diabetes, there were no significant differences between the CAC scores at baseline or follow-up by periodontal disease at baseline.

Table 2 presents the results of age- and gender-adjusted logistic and tobit regression models for CAC prevalence at baseline and progression by the baseline characteristics. Self-reported periodontal disease was significantly associated with progression of CAC, but not prevalence of CAC.

Results of multivariable logistic and tobit regression models of duration of periodontal disease on CAC prevalence and progression are depicted in Table 3. Models are presented for the diabetics and non-diabetics combined and stratified by diabetes status. Duration of periodontal disease was not associated with CAC prevalence in any model. Duration of periodontal disease was also not significantly associated with CAC progression in the combined model. However, duration of periodontal disease was significantly associated with CAC progression in individuals with diabetes, but not those without diabetes (p for interaction = 0.033).

Discussion

Type 1 diabetic individuals are at increased risk for the development of both periodontal disease and CAD, but the interrelation between these conditions is not fully understood. Given the longitudinal nature of the study, we further investigated the relation between diabetes and periodontal disease on the progression of CAC over a 6-year period. Although periodontal disease did not differ at baseline between the diabetic and non-diabetic groups, the duration of periodontal disease was significantly associated with progression of CAC, an established marker for atherosclerosis, among individuals with diabetes. Progression of CAC is an important clinical marker as it is associated with an increased risk of CAD events and all-cause mortality even after adjustment for standard cardiovascular disease risk factors and baseline CAC burden.^{7–8}

While epidemiologic studies support an association between periodontal disease and atherosclerotic disease independent of traditional risk factors, a causative relationship has not yet been established.⁹ However, our results demonstrate an association between periodontal disease and CAC progression only in the diabetic group. These results are consistent with a prior large, cross-sectional study that reported patients with diabetes and severe periodontal disease were more likely to have increased carotid artery intima-media thickness, acoustic shadowing which is evidence of advanced atheroma lesions, and CAD.¹⁰ The current study extends those findings with regard to CAC progression over a long-term follow-up and suggests that periodontal disease in type 1 diabetic individuals should be identified given a potentially magnified risk for development of CAD.

Periodontal disease is a known complication of diabetes.¹¹ Immune response to the underlying microbial infection can result in a chronic inflammatory state as periodontal disease is associated with increased markers of chronic inflammation.^{12–13} We speculate that chronic inflammation due to periodontal disease in combination with the hyperglycemic and inflammatory states associated with diabetes, especially in type 1 diabetes, could result in

accelerated progression of atherosclerosis. While we adjusted for hemoglobin A1C and evaluated hsCRP for inclusion in our models, these measures were taken at a single point in time and may not accurately reflect actual burden of hyperglycemia and inflammation over long-term follow-up. In addition, hsCRP is only one of many cytokines that could be pathologically increased in periodontal disease.

The clinical relevance of an association between periodontal disease and cardiovascular disease is an emerging area of study. Whether treatment of periodontal disease could decrease systemic inflammation and thereby reduce the risk of cardiovascular events is of principal interest. Local periodontal therapy has demonstrated a reduction in systemic inflammation as determined by hsCRP.^{14–15} One prospective study demonstrated a reduction in carotid artery intima-media thickness with local therapy.¹⁵ Therapies targeting systemic inflammation may improve periodontal disease status. A recent prospective, randomized, double-blind study demonstrated that patients receiving high dose statin for 12 weeks showed a significant reduction in periodontal inflammation as determined by ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography.¹⁶ However, there is no definitive data to suggest that treatment of periodontal disease decreases cardiovascular event rates.

Limitations for the current study include the use of a questionnaire to determine presence and duration of periodontal disease rather than clinical examination, although reasonable accuracy of self-reported periodontal disease has been demonstrated in a recent analysis from the Women's Health Initiative and in a representative US adult population from the 2009–2010 National Health and Nutrition Examination Survey.^{4,5} In addition, a similar question to the one used in this study: “Has any dentist/hygienist told you that you have deep pockets?” was previously shown to have a sensitivity of 55% and a specificity of 90%.¹⁷ This suggests that underreporting is likely, which would bias results towards the null. The subject's ability to recall the age at diagnosis could also be a factor that could bias estimates. Clinical examination would undoubtedly result in better measures of periodontal health and determine the severity of disease, which could result in the identification of the highest risk individuals. Clinical examination, however, would be unable to provide the duration of periodontal disease. Nonetheless, we were able to longitudinally study a large group of type 1 diabetic individuals who are highly susceptible to cardiovascular disease and demonstrate that periodontal disease is independently associated with long-term progression of CAC. Younger, type 1 diabetic individuals could represent a novel population worthy of therapy targeted both to oral health and risk factor control to decrease the burden of cardiovascular disease.

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Figure 1. Median (IQR) CAC score by Diabetes Status at Baseline and Followup
CAC measured in Agatston units and interquartile range (IQR). Light bars represent self-reported periodontal disease at baseline. Dark bars represent no periodontal disease at baseline.

Table 1**Participant Characteristics at Baseline**

Variable	Overall (n=1021)	Diabetes (n=473)	No Diabetes (n=548)	p-value
Age (years) *	39 (±9)	37 (±9)	41 (±9)	<0.001
Sex (male) †	496 (48.6%)	217 (45.9%)	279 (50.9%)	0.11
Race (white) †	911 (89.6%)	448 (95.1%)	463 (84.8%)	<0.001
Hispanic †	67 (6.6%)	16 (3.4%)	51 (9.4%)	<0.001
Past smoking †, ‡	214 (21.3%)	94 (20.3%)	120 (22.2%)	0.46
Current smoking †, ‡	94 (9.4%)	50 (10.8%)	44 (8.2%)	0.15
Hypertension †	273 (26.8%)	190 (40.3%)	83 (15.2%)	<0.001
Antihypertensive drugs †	203 (19.9%)	163 (34.5%)	40 (7.3%)	<0.001
Lipid lowering drugs †	112 (11.0%)	77 (16.3%)	35 (6.4%)	<0.001
Hemoglobin A _{1c} (%) *	6.6 (±1.5)	7.9 (±1.2)	5.5 (±0.4)	<0.001
Systolic blood pressure (mmHg) *	115.4 (±12.8)	116.7 (±13.3)	114.3 (±12.3)	0.004
Diastolic blood pressure (mmHg) *	78.1 (±8.5)	77.1 (±8.6)	79.0 (±8.4)	<0.001
Total cholesterol (mg/dL) *	184 (±37)	173 (±33)	193 (±37)	<0.001
High-density lipoprotein cholesterol (mg/dL) *	53 (±16)	56 (±17)	51 (±15)	<0.001
Low-density lipoprotein cholesterol (mg/dL) *	108 (±32)	99 (±28)	116 (±33)	<0.001
Triglycerides (mg/dL) §	90 (67–129)	76 (60–105)	104 (77–155)	<0.001
Body mass index (kg/m ²) *	26.2 (±4.5)	26.1 (±4.2)	26.2 (±4.8)	0.74
C-reactive protein (mg/l) §	1.2 (0.89–2.0)	1.2 (0.89–2.1)	1.2 (0.88–1.9)	0.49
Self-reported periodontal disease at baseline †	141 (13.9%)	68 (14.5%)	73 (13.4%)	0.60
Self-reported periodontal disease at follow-up †	185 (18.2%)	103 (21.9%)	82 (15.0%)	0.005
Duration of periodontal disease (years) §	9 (3–13)	6 (2–11)	9 (4–15)	0.18
Duration of diabetes (years) *	N/A	23 (±9)	N/A	N/A
Coronary artery calcification prevalence (>0) at baseline †	317 (31.1%)	169 (35.7%)	148 (27.0%)	0.003
Coronary artery calcification prevalence (>0) at follow-up †	534 (52.9%)	278 (59.5%)	256 (47.2%)	<0.001
Coronary artery calcification score of positives at baseline §	8.2 (1.4–72.4)	14.6 (1.4–98.9)	4.7 (1.1–31.8)	0.011
Coronary artery calcification score of positives at follow-up §	13.7 (2.2–100.4)	30.9 (3.3–201.7)	8.0 (1.5–51.2)	<0.001
Coronary artery calcification progression prevalence //	359 (35.2%)	201 (42.5%)	158 (28.8%)	<0.001
Coronary artery calcification progression 2.5 §, ¶	5.7 (3.9–9.5)	6.5 (4.2–10.9)	5.0 (3.6–7.6)	<0.001

* Data presented as mean ±SD, p-value from t-test

† Data presented as number (%), p-value from chi-square or Fisher's exact

‡ Referent category never smoking

[§]Data presented as median (interquartile range), p-value from Wilcoxon rank sum test

// Data presented as number (%) of those with progression of CAC (≥ 2.5 square-root transformed units difference in volume scores between baseline and follow-up)

[¶]Difference in square-root transformed volume scores between baseline and follow-up for those with positive progression (≥ 2.5) only

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Table 2

Factors Influencing Coronary Artery Calcium Prevalence at Baseline and Progression

Variable	Coronary Artery Calcium Prevalence	Coronary Artery Calcium Progression *	
	OR (95% CI) [†]	B ±SE	p-value
Age (years) [‡]	1.09 (1.07, 1.11)	0.34 ±0.03	0.001
Type 1 diabetes	2.57 (1.88, 3.50)	5.3 ±0.53	<0.001
Male [§]	3.02 (2.25, 4.04)	3.7 ±0.54	0.001
Race (Non-white vs white)	0.79 (0.49, 1.29)	−1.4 ±0.90	0.12
Hispanic	1.05 (0.60, 1.86)	−0.33 ±0.37	0.38
Past smoking	1.24 (0.87, 1.76)	0.001 ±0.66	0.99
Current smoking	1.83 (1.13, 2.96)	2.5 ±0.87	0.004
Hypertension	2.69 (1.97, 3.68)	5.2 ±0.54	<0.001
Antihypertensive drugs	2.94 (2.08, 4.16)	5.3 ±0.58	<0.001
Lipid lowering drugs	2.97 (1.92, 4.59)	4.9 ±0.73	<0.001
Hemoglobin A _{1c} (%)	1.40 (1.26, 1.55)	1.65 ±0.17	0.001
Systolic blood pressure (mmHg)	1.03 (1.02, 1.04)	0.16 ±0.02	<0.001
Diastolic blood pressure (mmHg)	1.02 (1.00, 1.04)	0.09 ±0.03	0.004
Total cholesterol (mg/dL)	1.00 (1.00, 1.01)	−0.02 ±0.007	0.03
High-density lipoprotein cholesterol (mg/dL)	0.99 (0.98, 1.00)	−0.03 ±0.02	0.10
Low-density lipoprotein cholesterol (mg/dL)	1.00 (1.00, 1.01)	−0.02 ±0.009	0.048
Log triglycerides (mg/dL)	1.54 (1.18, 2.03)	0.44 ±0.50	0.381
Body mass index (kg/m ²)	1.15 (1.11, 1.19)	0.30 ±0.06	<0.001
Log C-reactive protein (mg/l)	1.53 (1.22, 1.92)	1.3 ±0.42	0.002
Self-reported periodontal disease	1.16 (0.78, 1.74)	1.2 ±0.73	0.090
Duration of diabetes (years)	1.05 (1.02, 1.07)	0.24 ±0.05	<0.001

* Tobit regression with censored values <2.5 square-root transformed units

[†] Models adjusted for age and gender[‡] Models adjusted for gender[§] Models adjusted for age

Table 3

Duration of Periodontal Disease and Coronary Artery Calcium Prevalence and Progression

Variable	CAC Prevalence		CAC Progression*			
	Minimally Adjusted [†]	Adjusted [‡]	Minimally Adjusted [†]	Adjusted [§]		
	OR (95% CI)	OR (95% CI)	$\beta \pm SE$	p-value	$\beta \pm SE$	p-value
Periodontal Disease duration (yrs; N=1021))	1.01 (0.97, 1.06)	1.00 (0.95, 1.06)	0.24 $\pm$ 0.09	0.01	0.14 $\pm$ 0.07	0.06
Periodontal Disease duration (yrs) among Type 1 Diabetes (N=473)	1.07 (0.99, 1.15)	1.03 (0.94, 1.13)	0.57 $\pm$ 0.15	<0.001	0.36 $\pm$ 0.12	0.004
Periodontal Disease duration (yrs) among non-diabetics (N=548)	0.99 (0.92, 1.07)	0.98 (0.90, 1.07)	-0.03 $\pm$ 0.10	0.79	-0.04 $\pm$ 0.07	0.63

p-value for interaction term of duration of periodontal disease*diabetes = 0.033

* Tobit regression with censored values <2.5 square-root transformed units

[†] Adjusted for prevalence of periodontal disease[‡] Adjusted for age, sex, diabetes, total cholesterol, high-density lipoprotein cholesterol, hypertension, smoking, body mass index, and duration of diabetes[§] Adjusted for age, sex, diabetes, total cholesterol, high-density lipoprotein cholesterol, hypertension, smoking, body mass index, baseline coronary artery calcification prevalence, and duration of diabetes