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Does Oral Hygiene Trigger Carotid Artery Intima-Media Thickness?

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ABSTRACT

Background and Purpose: The aim of this study was to evaluate whether poor oral hygiene is associated with the intima-media thickness of the carotid arteries, which is one of the predictors of future progression of subclinical atherosclerosis.

Methods: We selected 108 patients during periodontal examinations according to their oral hygiene. The patients had no history of atherosclerotic disease. The results of carotid artery B-mode ultrasonography examinations were analyzed at baseline and after a mean of 7.8 months. Patients were scored on the DMFT index for the number of decayed (D), missing (M), and filled (F) teeth (T). We also used the Silness-Loe plaque index (SLI) to evaluate oral hygiene and dental plaque. The patients were divided into 2 groups according to DMFT and SLI criteria. Group I had a DMFT index of 0 to 3 and an SLI score of 0 or 1; group II had a DMFT index of 4 to 28 and an SLI score of 2 or 3.

Results: Dental status and oral hygiene were significantly associated with carotid artery intima-media thickness. Patients with increasing DMFT and SLI indices were correlated with intima-media thickness of the carotid artery.

Conclusions: Chronic poor oral hygiene and tooth loss are related to subclinical atherosclerotic changes in the carotid arteries and might be indicative of future progression of atherosclerosis.

INTRODUCTION

Cardiovascular diseases (CVDs) have been the most common cause of death and disability in recent decades. It is not true that conventional risk factors for atherosclerosis account for all atherosclerotic entities, and it has been postulated that novel risk factors such as poor oral hygiene and dental or periodontal disease are potentially associated with atherosclerosis [Desvarieux 2004; Schillinger 2006; Southerland 2012]. Several studies have found a close association between CVDs and poor oral hygiene [Ebersole 1997; Desvarieux 2003]. In particular, chronic inflammation triggered by poor oral hygiene has been speculated to play pathophysiological role in the etiology of atherosclerosis [Kiechl 2001; Desvarieux 2005]. In this study, we investigated whether poorer oral hygiene and/or periodontal disease indicate subclinical atherosclerosis.

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METHODS

Outpatients were evaluated for oral hygiene and dental status at Sifa University Hospital's School of Dentistry. DMFT index scores, which describe dental status, were obtained by calculating the number of decayed (D), missing (M), and filled (F) teeth (T). We also used the Silness-Loe plaque index (SLI) to evaluate oral hygiene and dental plaque [Desvarieux, 2003 2004; Schillinger 2006]. The mean index was calculated after evaluation of all teeth and surfaces. A specialized dentist checked patients and decided who could join the study. Ultimately, 108 outpatients with chronic poor oral hygiene were included in the study. We used the DMFT index and the SLI score as criteria for dividing the patients into 2 groups. Group I had DMFT index of 0 to 3, no to mild periodontal disease, an SLI score of 0 or 1, good-to-mild oral hygiene (n = 63; mean SD DMFT index, 0.9 1.16; mean SLI score, 0.63 0.23; 42 men and 21 women with a mean age of 51.75 8.72 years; age range, 39-75 years). Group II had a DMFT index from 4 to 28, severe periodontal disease, an SLI score from 2 to 3, and poor oral hygiene (n = 45; mean DMFT index, 18.92 5.44; mean SLI, 2.56 2.52; 27 men and 18 women with a mean age of 49.15 7.56 years; age range, 32-74 years). The mean follow-up time for all patients was 7.8 months (range, 6.1–8.6 months). Patients were assessed after diagnosis, after treatment, and 8 months later. All patients were treated conservatively for 2 weeks with systemic antibiotics and local treatments.

Carotid arteries were examined with B-mode ultrasonography, and a carotid arterial intima-media thickness of 1 to 2 mm was considered positive; up to 2 mm was strongly positive for subclinical early atherosclerosis. The study also enrolled patients with atherosclerotic carotid artery disease (as defined

by the presence of nonstenotic plaques or some degree of carotid stenosis) who were clinically asymptomatic or symptomatic at the time of screening. For a variety of reasons, 32 patients could not finish the study, and they were excluded from the study. Data were collected from patients between January 2011 and December 2011. Also excluded from the study were patients with diabetes mellitus; high cholesterol or triglyceride levels; hypertension; angina pectoris; a history of myocardial infarction and stroke; a history of rehabilitation, heart surgery, congestive heart failure, peripheral vascular disease, alcohol use, active malignant disease, and/or any immunologic or known chronic inflammatory condition; or current smoking. The study was approved by the institutional review board of the University of Sifa, and all patients gave signed, written informed consent before enrollment.

Dental Examination

In this study, we used the World Health Organization–approved dental indices to quantify dental disease: the DMFT is a measure of dental status, and the SLI is a measure of oral hygiene and dental plaque. All dental examinations were performed by specially trained dentists blinded to the patients' clinical and ultrasound data. Dental examinations took place 1 week before the initial ultrasound examination. The oral health parameters of all study participants were recorded at the beginning of the study. We evaluated all 28 teeth according to the DMFT index; we excluded the third molar teeth from the study. The DMFT index uses dental status and the amount of dental caries in an individual to express caries prevalence numerically. The SLI score assesses the state of oral hygiene and dental plaque accumulation by measurement of both soft and mineralized deposits at 4 sites per tooth (mesiobuccal, midbuccal, distobuccal, and midlingual). All periodontal findings were taken by means of the half-mouth method at all 4 gingival areas of the tooth and marked with a score from 0 to 3. Dental plaque was scored as follows: 0, no plaque; 1, a film of plaque adhering to the free gingival margin and adjacent area of the tooth that can be seen in samples from the tooth surfaces; 2, moderate accumulation of soft deposits within the gingival pocket or on the tooth and gingival margin that can be seen with the naked eye; 3, an abundance of soft matter within the gingival pocket and/or the tooth and gingival margin.

For patients who were toothless, an SLI score was obtained from the prosthesis. An SLI score of 0 or 1 was defined as absent or mild, and a score of 2 or 3 was defined as serious.

Carotid B-Mode Ultrasonography

The carotid arterial intima-media thickness and/or stenosis were measured by experienced radiologists using B-mode ultrasonography. The extracranial carotid arteries were examined bilaterally with a 7.5-MHz linear array transducer (Antares; Siemens, Munich, Germany). The operators were blinded to the patients' clinical data and dental status. The patients were in a supine position with their head turned slightly away from the operator. Measurements were taken in longitudinal and transverse planes on the far wall of the common carotid artery 1 cm from the bulb, the bifurcation, the internal carotid artery, and the external carotid artery. The intima-media thickness was defined as the distance between the leading edges of the lumen-intima echo and the media-adventitia echo. Subclinical atherosclerosis was defined as a mean carotid artery intima-media thickness of >1 mm, as assessed by B-mode ultrasound. The 1-mm cutpoint was chosen because of its clinical and prognostic significance and its association with the subsequent development of coronary artery disease. Carotid plaque was defined as a localized intima-media thickening of >1 mm with at least a 100% increase in thickness compared with adjacent wall segments. The presence of plaque diagnosed during the examination was defined as early atherosclerosis. Plaques were present if they protruded into the lumen or showed localized roughness with an increased echogenicity or an area of increased thickness of the intima-media layer. Plaque presence was defined as 1 plaque in any of the carotid arteries. To compensate for the stretching effect of arterial distension secondary to increased arterial pressure on wall thickness, we chose patients whose systolic arterial pressures were <140 mm Hg.

Continuous data are displayed as the mean SD. Categorical data are expressed as proportions. Categorical variables were analyzed with the chi-square test or the Fisher exact test, as appropriate. In all studies, *P* values <.05 were considered statistically significant.

RESULTS

The 2 groups of patients had similar baseline demographic and clinical characteristics. The mean ages of the 2 groups were not significantly different (Table 1). Baseline measurements revealed that group II had mean values for carotid artery intima-media thickness that were significantly higher than those of group I (*P* = .001). Dental status was not correlated with oral hygiene (Table 2). The mean DMFT index scores were 0.9 1.16 in group I and 18.92 5.44 in group II. The groups' respective mean SLI scores were 0.63 0.23 and 2.56 2.52. Figures 1 and 2 depict carotid artery intima-media thickness as measured by B-mode ultrasonography. The B-mode ultrasonographic examination revealed 6 patients (9.52%) in group I and 39 patients (86.66%) in group II with an intima-media thickness >1 mm (Table 2). This proportion was 89% in patients who were toothless.

Increasing DMFT and SLI scores were correlated with the intima-media thickness of the carotid artery. Intima-media thickness was >1 mm in 83% of the patients with a DMFT index >20 and in 71% of patients with an SLI score >2; however, an intima-media thickness >1 mm was observed in only 11% of patients with a DMFT index <3 and in 7.1% of patients with an SLI score of 0 or 1. Significantly more patients with an intima-media thickness >2 mm had a DMFT index score >20. The intima-media thickness was >1 mm in 11% of patients with just teeth fillings, which was significantly lower than the rate for patients with decayed and missing teeth (*P* = .0001). The number of missing teeth was significantly associated with the baseline thickness of the carotid artery intima-media. In addition, toothless patients had a significantly higher degree of thickness at baseline (*P* = .006).

Table 1. Demographic and Clinical Data for the Study Patients*

Parameter	Group I (n = 63): DMFT, 0-3; SLI, 0-1	Group II (n = 45): DMFT, 4-28; SLI, 2-3	*P*
Age, y	51.62 ± 4.98	49.15 ± 5.26	.260
Male patients, ± n (%)	42 (64.6)	27 (60.1)	.076
BMI, kg/m^2	26 ± 3	27 ± 2	.120
Systolic blood pressure, mm Hg	136 ± 21	139 ± 24	.317
Diastolic blood pressure, mm Hg	80 ± 14	81 ± 12	.125
Family history, n (%)	12 (19.04)	9 (20)	.915
Fasting plasma glucose, mg/dL	92 ± 12	94 ± 15	.356
Total cholesterol, mg/dL	214 ± 28	216 ± 32	.134
Triglycerides, mg/dL	195 ± 18	191 ± 22	.075
Uric acid, mg/dL	5.1 ± 2.2	5.2 ± 2.6	.952
DMFT index	0.9 ± 1.16	18.92 ± 5.44	.0001
SLI score	0.63 ± 0.23	2.56 ± 2.52	.001
CA-IMT, mm	0.61 ± 0.1	1.76 ± 0.14	.001
hs-CRP, mg/L	2.9 ± 1.4	4.9 ± 2.8	.001
Smoking, n (%)	16 (25.39)	12 (26.66)	.256

*Data are presented as the mean ± SD or n (%), as indicated. DMFT indicates decayed, missing, filled teeth; SLI, Silness-Loe plaque index; BMI, body mass index; CA-IMT, carotid artery intima-media thickness; hs-CRP, high-sensitivity C-reactive protein.

Table 2. Carotid Arterial Ultrasound Results and High-Sensitivity C-Reactive Protein (hs-CRP) Levels in the 2 Groups of Patients*

Parameter	Group I (n = 63): DMFT, 0-3; SLI, 0-1	Group II (n = 45): DMFT, 4-28; SLI, 2-3	*P*
Baseline, n (%)			
Carotid intima-media thickness			
1-2 mm	5 (7.93)	28 (62.22)	.0001
>2 mm	1 (1.58)	11 (24.44)	.0001
hs-CRP (up to 3 mg/L)	7 (11)	40 (89)	.0001
After 8 months, n (%)			
Carotid intima-media thickness			
1-2 mm	6 (9.52)	31 (68.8)	.001
>2 mm	2 (3.17)	12 (26.6)	.0001
hs-CRP (up to 3 mg/L)	5 (8)	34 (75.5)	.0001

*DMFT indicates decayed, missing, filled teeth; SLI, Silness-Loe plaque index.

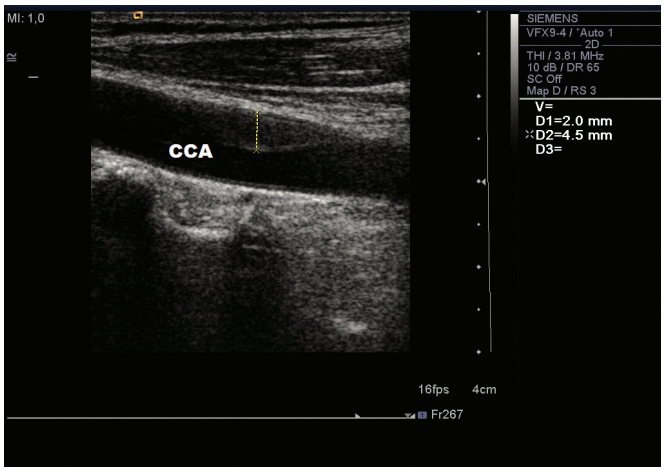

Figure 1. Carotid artery intima-media thickness measured by B-mode ultrasonography. CCA indicates common carotid artery.

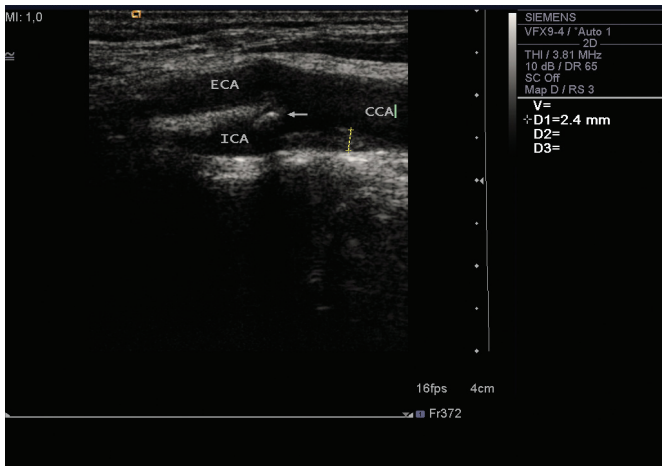

Figure 2. The intima-media thickness of a carotid artery as measured by B-mode ultrasonography. Dotted line indicates media-intima thickness. ECA indicates external carotid artery; ICA, internal carotid artery; CCA, common carotid artery.

In group II, toothless patients (9%) who had a dental prosthesis were included as a separate category in the calculations of the SLI score. In analyzing these patients, we found that toothless patients had a significantly higher risk for disease progression than patients in group I (P = .001).

Values for high-sensitivity C-reactive protein (hs-CRP) were significantly higher in group II than in group I, both at baseline and after 8 months (P = .001). hs-CRP levels were >3 mg/L in 11% of patients in group I and in 89% of patients in group II. Both the DMFT index and the SLI score were significantly associated with the hs-CRP level at baseline and at the 8-month follow-up. Among the patients who received treatment, intima-media thickness values were similar after 8 months, but hs-CRP levels were significantly lower (P = .001).

No deaths were recorded during follow-up (mean, 7.8 months; range, 5.1-8.6 months). During the follow-up period, only 2 patients (3.17%) in group I showed progression in carotid artery intima-media thickness. On the other hand, 4 patients (8.8%) in group II showed progression of carotid artery intima-media thickness. Patients with progressive atherosclerosis had significantly higher DMFT and SLI scores than did patients with stable disease. Further analysis of the subcategories of the DMFT index in the 8-month follow-up measurements revealed that the number of missing teeth was strongly associated with disease progression (P = .001). The number of decayed teeth was also significantly associated with disease progression (P = .02), but the number of filled teeth was not (P = .09).

DISCUSSION

CVD is a major cause of morbidity and mortality worldwide. In the last 10 years, an increasing number of epidemiologic investigations have studied the possible association between inflammatory diseases or chronic infections (i.e. periodontal infections) and CVDs [Sanz 2010; Cotti 2011; Pessoa 2011]. According to these studies, atherosclerosis is considered a process closely related to inflammation. We know that infectious or inflammatory diseases, elevated levels of inflammatory markers, and autoimmune processes can contribute to the development of atherosclerosis [Pessoa 2011]. In addition, the results of several studies published during the last 2 decades have indicated that oral diseases, periodontal inflammation, and especially poor oral hygiene may also act as risk factors for the development of atherosclerosis via chronic inflammation [Beck 1999; Lorenz 2007; Bartels 2012].

In particular, chronic microbial infection, including several periodontal pathogens, may play an important role in the development of atherosclerotic disease [Desvarieux 2005; Niessner 2006; Hoke 2011]. How does periodontal disease cause thickening of the arterial intima-media wall (which is a predictor of subclinical atherosclerosis)? The answer to this question is still unclear [Sabeti 2005; Schillinger 2005]. It remains uncertain whether an immune response to pathogens or the pathogen itself triggers the progression of atherosclerotic disease [Desvarieux 2003; Beck 2005]. We confirm that missing and decayed teeth, rather than filled teeth, are significantly associated with atherosclerosis progression. hs-CRP levels are low in patients with treated caries, and treated caries do not seem to play a major role in promoting atherosclerosis. Poor oral hygiene with infection, a trigger for systemic inflammation, has previously been suggested to correlate with carotid intima-media thickness, a surrogate marker of atherosclerosis [Schillinger 2006].

Elter et al [2006] proposed a potential mechanism for vascular dysfunction in the presence of periodontal disease. A study by Tonetti et al [2007] concluded that intensive periodontal infection led to acute, short-term systemic inflammation and endothelial dysfunction. In addition, periodontal disease has been shown to be a strong predictor of mortality from ischemic heart disease and diabetic nephropathy among Pima Indians with type 2 diabetes [Saremi 2005]. In addition, investigators in another study postulated that

poor oral hygiene might be an insidious cause of endothelial dysfunction and future cardiovascular events [Blum 2007].

The present study has clearly demonstrated a significant relationship between dental diseases (especially tooth loss) and subclinical atherosclerosis. We conclude that a high DMFT index is associated with the thickness of the carotid intima-media wall, a marker of early initiation of atherosclerotic lesions. The clinical implications derived from our study are that once a dentist diagnoses advanced dental disease or signs of poor oral hygiene, the patient should be referred to an internist for further screening. If necessary, cardiovascular risk factors can then be assessed. Demonstrating an association between inflammation, dental indices, poor oral hygiene, and disease progression, however, requires more long-term studies. The mean follow-up period of 8 months is a limitation of this study. Therefore, we will continue our follow-up investigation to monitor disease progression.

We should mention the other limitations of our study. Data on pathogen levels or immune responses to pathogens were not available for our population. We could not determine an individual's propensity for developing an inflammatory reaction. Microbial aspects, which have been shown to be more specific than clinical signs of poor oral hygiene, were not evaluated in our study. In addition, long-term periodontal status was not very well known. We investigated only clinical measures of dental and periodontal disease.

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