Association between self-reported oral health, tooth loss and atherosclerotic burden

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Abstract: Previous studies have suggested that oral diseases may influence the development of atherosclerosis. The aim of this study was to test the hypothesis that poor self-reported oral health (SROH) and tooth loss are positively associated with coronary atherosclerotic burden (CAB). 382 consecutive subjects undergoing coronary angiography were included. Socio-demographic characteristics, cardiovascular risk factors and oral health status were collected using a standardized questionnaire, including data on SROH and use of dental prosthesis. Number of teeth and anthropometric measures were collected through clinical examinations. CAB at coronary angiography was quantified using the Friesinger score (FS). Prevalence ratios (PR) were calculated with Poisson regression analyses. Mean age was 60.3 ± 10.8 years, with 63.2% males. In the bivariate analysis, there was a significant association ($p < 0.05$) between CAB and age (≥ 60y) (PR = 1.01, 95% CI = 1.02–1.16), male gender (PR = 1.11, 95% CI = 1.03–1.19), smoking (PR = 1.08, 95% CI = 1.01–1.16), hypertension (PR = 1.12, 95% CI = 1.03–1.22), diabetes (PR = 1.17, 95% CI = 1.05–1.21), poor SROH (PR = 1.22, 95% CI = 1.02–1.46) and tooth loss (< 20teeth present) (PR = 1.10, 95% CI = 1.02–1.19). The use of dental prosthesis was not associated with CAB. The multivariate models, adjusted for age, gender, smoking, hypertension, diabetes and dyslipidemia showed that poor SROH ($p = 0.03$) and tooth loss ($p = 0.02$) were independently associated with CAB, confirming the study hypothesis.

Descriptors: Atherosclerosis; Risk Factors; Epidemiology; Tooth Loss; Cardiovascular Diseases.

Introduction

Epidemiological studies have suggested that chronic periodontal disease,[1-4] lesions of endodontic origin[5] and tooth loss[6] are associated with cardiovascular disease (CVD) and mortality.

The triggering of an inflammatory response by infectious agents is a potential mechanism correlating infection to the acceleration of atherosclerosis.[7] Coronary atherosclerotic burden (CAB) is a term used to describe the extension of atherosclerosis into coronary vessels.[8] Previous symptomatic atherosclerotic vascular disease (AVD) evaluated by a clinical score of CAB was shown to be an independent predictor of early mortality in patients with first-ever ischemic stroke.[9]

Poor oral health is a major cause of a proinflammatory state and may

accelerate the atherosclerotic process or precipitate a plaque rupture.[10] Poor oral health may also affect eating behavior and contribute to poor nutrition, which has been identified as a risk factor for mortality. Potential pathogenic mechanisms linking oral infections and AVD are based on three main pathways:

- the role of periodontal pathogens and their products in the development of endothelial dysfunction;
- the contribution of oral microorganisms to the formation of fatty streaks and atherosclerotic plaques; and
- the role of oral flora in the modulation and maturation of atheromatous plaques, facilitating their rupture and vascular thrombosis.[10,11]

Studies have found associations between tooth loss and carotid[12] and aortic[13] intima-media thickness, as well as aortic valve sclerosis[14]. In diabetic patients, positive correlations between atherogenic factors and oral hygiene, periodontal disease and tooth loss were found.[15] In another study, tooth loss was associated with inflammatory markers and stroke.[16]

Oral diseases are primarily associated with noncommunicable chronic diseases through shared common risk factors such as age, lifestyle, diet, smoking, and low socioeconomic status. Accordingly, there is some evidence that, after adjusting for these risk factors, the relationship between oral health and CVD may be weakened.[17]

Self-reported health status assessing systemic diseases and health-related conditions are widely used in populational investigations. In the last years, self-reported oral health (SROH) status has been increasingly implemented in dentistry.[18-21]

Few studies have tested the relationship between clinical scores of CAB and oral health in humans. The current study tested this association in a group of southern Brazilian patients using a SROH approach,[19] supplemented by an oral clinical examination measuring the number of teeth. The aim of this study was to test the hypothesis that poor SROH status and tooth loss are positively associated with CAB.

Methodology

The research protocol was approved by the Ethics and Research Committee of the Pontifical Catholic University of Rio Grande do Sul (PUCRS), number 08/04211. All participants provided a signed informed consent form. Consecutive adult patients (≥ 18 years) undergoing coronary angiography to investigate coronary artery disease in the Center for Cardiovascular Diagnosis and Intervention, São Lucas Hospital (Porto Alegre, Brazil), were invited to participate. Emergency cases or patients unable to answer the questionnaire due to physical or mental conditions were excluded. Data were collected prior to the angiography, from October 2008 to December 2009, including a total of 382 individuals. All participants survived after angiography.

Socio-demographic data (age, gender, marital status, education and occupation) and medical cardiovascular risk factors (smoking, hypertension, dyslipidemia, diabetes, family history of coronary heart disease [CHD] and use of statins) were collected using a structured questionnaire. Weight (kg) was measured using an anthropometric calibrated scale (Filizola, São Paulo, Brazil). Height (m) was measured using the stadiometer of the anthropometric scale. The body mass index (BMI) was calculated by dividing the weight by the height squared. Data collection procedures are further described in a previous study.[22]

Information on oral health was collected using a structured questionnaire. Measures of self-perception on oral health included the variables "self-reported oral health (SROH)" (excellent, very good, good, fair or poor)[19,23] and "use of dental prosthesis" (yes or no). Total number of natural teeth was measured by simplified oral clinical examination, performed by a trained non-dentist examiner. Number of teeth (tooth loss) was dichotomized into nonfunctional dentition (< 20 teeth) and functional dentition (≥ 20 teeth).[6]

CAB was evaluated through the Friesinger Score (FS)[22,24] on the diagnostic coronary angiography performed by standard technique. The FS ranges from 0 to 15 and separately scores each of the three main coronary arteries. All coronary lesions were assessed by one interventional cardiolo-

gist, blinded to the oral health data. For analytic purposes, CAB was dichotomized into low (FS ≤ 7) and high (FS > 7), based on the distribution of the FS in the present sample (mean and standard deviation = 7.3 ± 4.0; median = 7).

Data were analyzed using SPSS v.17 (IBM, Chicago, USA). Descriptive statistics (N and %) according to CAB were performed. Prevalence ratios (PR) were calculated with bivariate and multivariate Poisson regression analyses with robust variance.[25] Associations between CAB and SROH and between CAB and number of teeth were determined separately and adjusted for the socio-demographic and medical confounders. Spearman's correlation (r_s) was calculated between SROH and number of teeth. The value for rejection of the null hypothesis was set at $p \leq 0.05$.

Results

Characteristics of the sample in relation to CAB are shown in Table 1. Mean age was 60.3 (± 10.8), ranging from 23 to 89 years, with males (63.3%) predominating. Dental variables revealed that nearly 45% of the participants reported poor or fair oral health status, more than 67% wore dental prosthesis, and only 33% had ≥ 20 teeth.

In the unadjusted analysis (Table 1), there was a significant association between CAB and age (PR = 1.01, 95% CI = 1.02–1.16), gender (PR = 1.11, 95% CI = 1.03–1.19), smoking (PR = 1.08, 95% CI = 1.01–1.16), hypertension (PR = 1.12, 95% CI = 1.03–1.22), diabetes (PR = 1.17, 95% CI = 1.05–1.21), poor SROH (PR = 1.22, 95% CI = 1.02–1.46) and number of teeth < 20 (PR = 1.10, 95% CI = 1.02–1.19).

Multivariate models testing the association of SROH and number of teeth with CAB, after adjusting for age and gender (Model 1) and age, gender, smoking, hypertension, diabetes and dyslipidemia (Model 2) are shown in Tables 2 and 3. Poor SROH (PR = 1.22, 95% CI = 1.02–1.47) or fair SROH (PR = 1.20, 95% CI = 1.02–1.41) and having < 20 teeth (PR = 1.09, 95% CI = 1.02–1.18) were independently associated with CAB after adjustments. A significant correlation between SROH and number of teeth (r_s = 0.23, p < 0.01) was found.

Table 1 - Socio-demographic, medical and dental characteristics of participants (N, %), by coronary atherosclerotic burden (CAB) as measured with the Friesinger Score (FS) **(continued on next page)**.

Variables (N)	Low CAB FS 0–7 N (%)	High CAB FS 8–15 N (%)	p*
Socio-demographic			
Age (382)			
• < 60 years	107 (55.4)	81 (42.9)	
• ≥ 60 years	86 (44.6)	108 (57.1)	0.01
Gender (381)			
• female	84 (43.5)	56 (29.8)	
• male	109 (56.5)	132 (70.2)	< 0.01
Marital status (379)			
• single/divorced/ widowed	58 (30.4)	59 (31.4)	
• married/live together	133 (69.9)	129 (68.6)	0.83
Education (379)			
• up to 8 years	132 (69.1)	128 (68.1)	
• > 8 years	59 (30.9)	60 (31.9)	0.83
Occupation (380)			
• unemployed	5 (2.6)	6 (3.2)	
• formal employee	33 (17.2)	23 (12.2)	0.40
• informal employee	28 (14.6)	31 (16.5)	0.90
• home worker	38 (19.8)	18 (9.6)	0.15
• retired	88 (45.8)	110 (58.5)	0.95
Medical			
BMI (337)			
• normal	38 (22.4)	43 (25.7)	
• overweight	81 (47.6)	71 (42.5)	0.35
• obesity	51 (30.0)	53 (31.7)	0.77
Smoking (379)			
• non-smoker	99 (51.8)	75 (39.9)	
• smoker/ex-smoker	92 (48.2)	113 (60.1)	0.02
Hypertension (380)			
• no	55 (28.6)	32 (17.0)	
• yes	137 (71.4)	156 (83.0)	0.01
Dyslipidemia (379)			
• no	101 (52.6)	81 (43.3)	
• yes	91 (47.4)	106 (56.7)	0.07
Diabetes (379)			
• no	153 (79.7)	122 (65.2)	
• yes	39 (20.3)	65 (34.8)	< 0.01

Table 1 *(continued)*

Family history of CHD (379)			
• no	152 (79.2)	145 (77.5)	
• yes	40 (20.8)	42 (22.5)	0.70
Use of Statins (374)			
• no	110 (57.9)	98 (53.3)	
• yes	80 (42.1)	86 (46.7)	0.37
Dental			
SROH (378)			
• excellent	15 (7.9)	6 (3.2)	
• very good	24 (12.6)	19 (10.2)	0.22
• good	74 (38.7)	72 (38.5)	0.07
• fair	61 (31.9)	68 (36.4)	0.04
• poor	17 (8.9)	22 (11.8)	0.03
Use of dental prosthesis (378)			
• no	123 (64.4)	132 (70.6)	
• yes	68 (35.6)	55 (29.4)	0.20
Number of teeth (374)			
• < 20 teeth	117 (61.3)	135 (73.8)	
• ≥ 20 teeth	74 (38.7)	48 (26.2)	0.01
Total (382)	193 (50.5)	189 (49.5)	-

* *p*-value for bivariate analysis, Poisson regression; BMI = body mass index; CHD = coronary heart disease; SROH = self-reported oral health.

Discussion

The results of this cross-sectional study confirmed the hypothesis that SROH status and number of teeth were significantly associated with CAB in this group of Brazilian patients. Most importantly, this association was independent of other traditional risk factors for CVD, such as age, gender, smoking, hypertension, diabetes and dyslipidemia. To our knowledge, this is one of the first studies to provide evidence of the connection between CAB and oral diseases based on the assessment of SROH status in combination with tooth loss. Nevertheless, inherent limitations of the study design do not allow inferences about causality concerning this association.

The present study evaluated CAB through the FS. Other studies assessed carotid and aortic intima-media thickness or aortic valve sclerosis,[12-14] instead of luminal obstruction of the main coronary arteries quantified using the FS. Unlike other systems for evaluating the extent of CHD, the FS was specifically developed for the assessment of parietal AVD, regardless of the area of perfused myocardium through the stenosis.[22]

Potential mechanisms that link oral infections to

Table 2 - Adjusted models for the association of self-reported oral health (SROH) with coronary atherosclerotic burden (CAB) as measured with the Friesinger Score (FS). N = 378.

Variables	Unadjusted PR and 95% CI	p	Model 1* Adjusted PR and 95% CI	p	Model 2* Adjusted PR and 95% CI	p
SROH						
excellent	1.00		1.00		1.00	
very good	1.12 (0.93–1.35)	0.22	1.11 (0.92–1.33)	0.26	1.13 (0.94–1.35)	0.20
good	1.16 (0.99–1.36)	0.07	1.15 (0.98–1.35)	0.08	1.16 (0.99–1.37)	0.07
fair	1.19 (1.01–1.39)	0.04	1.19 (1.02–1.39)	0.03	1.20 (1.02–1.41)	0.03
poor	1.22 (1.02–1.46)	0.03	1.26 (1.05–1.50)	0.01	1.22 (1.02–1.47)	0.03

p = *p*-value, Poisson regression. (*) adjusted through Poisson regression for: Model 1 - age, gender; Model 2 - age, gender, smoking, hypertension, diabetes, dyslipidemia.

Table 3 - Adjusted models for the association of number of teeth with coronary atherosclerotic burden (CAB) as measured with the Friesinger Score (FS). N = 374.

Variables	Unadjusted PR and 95% CI	p	Model 1** Adjusted PR and 95% CI	p	Model 2** Adjusted PR and 95% CI	p
Number of teeth						
≥ 20 teeth	1.00		1.00		1.00	
< 20 teeth	1.10 (1.02–1.19)	0.01	1.10 (1.02–1.19)	0.01	1.09 (1.02–1.18)	0.02

p = *p*-value, Poisson regression. (*) adjusted through Poisson regression for: Model 1 - age, gender; Model 2 - age, gender, smoking, hypertension, diabetes, dyslipidemia.

atherogenesis are based on the role of periodontal pathogens and their products in the development of endothelial dysfunction, formation of fatty streaks and maturation of atherosclerotic plaques, with their rupture and vascular thrombosis.[11] Our results confirm previous findings in which periodontal disease,[15] lesions of endodontic origin[5] or tooth loss[12-14,16] were significantly associated with AVD,[12-14] atherogenic risk factors[15] or cardiovascular events such as stroke[16] and CHD.[5] Nevertheless, other studies failed to find this association.[17,26]

The methodologies of previous studies testing the association between oral health and CVD differed from that of the present study. A cohort study[3] measured the mean bone loss and the probing pocket depth scores *per* tooth. In other studies, subjects received a periodontal examination[4] or a periodontal microbiological evaluation,[2] and a carotid scan using high-resolution ultrasound was the method used to evaluate subclinical AVD. Importantly, there is extensive variability in the literature regarding definitions of the oral exposure, including salivary flow, reported periodontal disease, number of teeth, oral organisms, antibodies to oral organisms, and different periodontal disease parameters.[1]

This study focused on the assessment of SROH and number of teeth, instead of investigating microbiological, clinical and radiographic parameters. The use of SROH measures provides relevant cost and time savings in large epidemiological surveys.[19,21] SROH is a known Likert-type scale and previous studies have provided consistent evidence of the construct validity of this model.[19,23]

Limitations of the SROH approach must be clarified. Self-perceived oral health was shown to be better in individuals with more teeth and recent dental treatment and worse in those with tooth mobility, coronal decay and medical problems.[19] SROH measures showed valid estimates for variables such as number of teeth, fillings, root canal therapy and prosthesis, but was less accurate for the assessment of dental caries and periodontal disease.[20] In fact, it is known that self-perceived oral health is influenced not only by dental clinical oral status, but also by social and psychological issues.[27]

This study includes self-perceived measures of disease, not only related to dental variables, but also to medical cardiovascular risk factors. Diagnosis of chronic diseases by a physician would result in more reliable information regarding the presence of comorbidities. Although SROH is a known valid measure, the analysis would benefit from the inclusion of a detailed oral clinical evaluation. Unfortunately, such data were not collected due to hospital service characteristics. However, the significant correlation between SROH and number of teeth found here indicates that the accuracy of self-perceived oral measures was not divergent from the actual clinical findings in this sample.

Number of teeth is a surrogate variable commonly used to access history of periodontal disease. Most studies, however, do not consider that tooth loss may occur not only due to periodontitis, but also due to dental caries, endodontic infections or trauma. Periodontal disease is the main reason for tooth loss at the tooth level, but caries/endodontic disease is the most common cause of tooth loss at the individual level.[28] Tooth loss is one of the strongest populational oral health indicators, working as a "registry" of the history of both periodontal and endodontic diseases. Tooth loss is not only a biological process; it can also involve factors such as attitudes of patients and providers, access to care issues and dental care delivery systems.

The weak but statistically significant association between oral status and AVD estimated in this study may raise questions about the "statistical significance" *versus* its "clinical relevance". In this study, the PR of classic risk factors for CVD were similar to those found for dental variables. The low but statistically significant PR do not affect the clinical relevance of these classic risk factors for CVD. The aim of our study was to test the hypothesis of the association rather than exploring its clinical relevance, which may only be considered after future evidence from longitudinal studies.

Interestingly, the correlation between oral diseases with AVD and CVD is apparent in different studies, irrespective of the variability in oral exposures and vascular outcomes. The present findings using SROH measures encourage future investigations of this association in large epidemiological longitudi-

nal studies, which would contribute to a better understanding of this multifactorial relationship.

Conclusion

SROH status and number of teeth were independently associated with CAB, measured by the FS, in a group of Brazilian patients, thus confirming the study hypothesis.

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References

1. Beck JD, Offenbacher S. Systemic effects of periodontitis: epidemiology of periodontal disease and cardiovascular disease. J Periodontol. 2005 Nov;76(11 Suppl):2089-100.
2. Desvarieux M, Demmer RT, Rundek T, Boden-Albala B, Jacobs DR Jr, Sacco RL, et al. Periodontal microbiota and carotid intima-media thickness: the Oral Infections and Vascular Disease Epidemiology Study (INVEST). Circulation. 2005 Feb 8;111(5):576-82.
3. Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. J Periodontol. 1996 Oct;67(10 Suppl):1123-37.
4. Desvarieux M, Demmer RT, Rundek T, Boden-Albala B, Jacobs DR Jr, Papapanou PN, et al. Relationship between periodontal disease, tooth loss, and carotid artery plaque: the Oral Infections and Vascular Disease Epidemiology Study (INVEST). Stroke. 2003 Sep;34(9):2120-5.
5. Caplan DJ, Chasen JB, Krall EA, Cai J, Kang S, Garcia RI, et al. Lesions of endodontic origin and risk of coronary heart disease. J Dent Res. 2006 Nov;85(11):996-1000.
6. Padilha DM, Hilgert JB, Hugo FN, Bos AJ, Ferrucci L. Number of teeth and mortality risk in the Baltimore Longitudinal Study of Aging. J Gerontol A Biol Sci Med Sci. 2008 Jul;63(7):739-44.
7. Hayashi C, Viereck J, Hua N, Phinikaridou A, Madrigal AG, Gibson FC 3rd, et al. Porphyromonas gingivalis accelerates inflammatory atherosclerosis in the innominate artery of ApoE deficient mice. Atherosclerosis. 2011 Mar;215(1):52-9.
8. Guerrero M, Harjai K, Stone GW, Brodie B, Cox D, Boura J, et al. Usefulness of the presence of peripheral vascular disease in predicting mortality in acute myocardial infarction patients treated with primary angioplasty (from the Primary Angioplasty in Myocardial Infarction Database). Am J Cardiol. 2005 Sep 1;96(5):649-54.
9. Roquer J, Ois A, Rodriguez-Campello A, Gomis M, Munteis E, Jimenez-Conde J, et al. Atherosclerotic burden and early mortality in acute ischemic stroke. Arch Neurol. 2007 May;64(5):699-704.
10. Cotti E, Dessi C, Piras A, Mercuro G. Can a chronic dental infection be considered a cause of cardiovascular disease? A review of the literature. Int J Cardiol. 2011 Apr 1;148(1):4-10.
11. Kebschull M, Demmer RT, Papapanou PN. "Gum bug, leave my heart alone!"--epidemiologic and mechanistic evidence linking periodontal infections and atherosclerosis. J Dent Res. 2010 Sep;89(9):879-902.
12. Chin UJ, Ji S, Lee SY, Ryu JJ, Lee JB, Shin C, et al. Relationship between tooth loss and carotid intima-media thickness in Korean adults. J Adv Prosthodont. 2010 Dec;2(4):122-7.
13. Castillo R, Fields A, Qureshi G, Salciccioli L, Kassotis J, Lazar JM. Relationship between aortic atherosclerosis and dental loss in an inner-city population. Angiology. 2009 Jun-Jul;60(3):346-50.
14. Völzke H, Schwahn C, Hummel A, Wolff B, Kleine V, Robinson DM, et al. Tooth loss is independently associated with the risk of acquired aortic valve sclerosis. Am Heart J. 2005;150(6):1198-203.
15. Furukawa T, Wakai K, Yamanouchi K, Oshida Y, Miyao M, Watanabe T, et al. Associations of periodontal damage and tooth loss with atherogenic factors among patients with type 2 diabetes mellitus. Intern Med. 2007;46(17):1359-64.
16. You Z, Cushman M, Jenny NS, Howard G. Tooth loss, systemic inflammation, and prevalent stroke among participants in the reasons for geographic and racial difference in stroke (REGARDS) study. Atherosclerosis. 2009 Apr;203(2):615-9.
17. Tuominen R, Reunanen A, Paunio M, Paunio I, Aromaa A. Oral health indicators poorly predict coronary heart disease deaths. J Dent Res. 2003 Sep;82(9):713-8.
18. Douglass CW, Berlin J, Tennstedt S. The validity of self-reported oral health status in the elderly. J Public Health Dent. 1991 Fall;51(4):220-2.
19. Jones JA, Kressin NR, Spiro A 3rd, Randall CW, Miller DR, Hayes C, et al. Self-reported and clinical oral health in users of VA health care. J Gerontol A Biol Sci Med Sci. 2001 Jan;56(1):M55-62.
20. Pitiphat W, Garcia RI, Douglass CW, Joshipura KJ. Validation of self-reported oral health measures. J Public Health Dent. 2002 Spring;62(2):122-8.
21. Gomes MS, Hugo FN, Hilgert JB, Padilha DM, Simonsick EM, Ferrucci L, et al. Validity of self-reported history of endodontic treatment in the Baltimore Longitudinal Study of Aging. J Endod. 2012 May;38(5):589-93.

22. Chagas P, Caramori P, Barcellos C, Galdino TP, Gomes I, Schwanke CH. Association of different anthropometric measures and indices with coronary atherosclerotic burden. Arq Bras Cardiol. 2011 Sep;97(5):397-401.
23. Gilbert GH, Duncan RP, Heft MW, Dolan TA, Vogel WB. Multidimensionality of oral health in dentate adults. Med Care. 1998 Jul;36(7):988-1001.
24. Javed F, Aziz EF, Nadkarni GN, Khan SA, Sabharwal MS, Malhan R, et al. The association of the Friesinger score and pulse pressure in an urban South Asian patient population: pulse pressure, an independent predictor of coronary artery disease. J Am Soc Hypertens. 2010 May-Jun;4(3):142-7.
25. Barros AJ, Hirakata VN. Alternatives for logistic regression in cross-sectional studies: an empirical comparison of models that directly estimate the prevalence ratio. BMC Med Res Methodol. 2003 Oct 20;3:21.
26. Joshipura KJ, Rimm EB, Douglass CW, Trichopoulos D, Ascherio A, Willett WC. Poor oral health and coronary heart disease. J Dent Res. 1996 Sep;75(9):1631-6.
27. Martins AB, Santos CM, Hilgert JB, Marchi RJ, Hugo FN, Padilha DMP. Resilience and self-perceived oral health: a hierarchical approach. J Am Geriatr Soc. 2011 Apr;59(4):725-31.
28. Phipps KR, Stevens VJ. Relative contribution of caries and periodontal disease in adult tooth loss for an HMO dental population. J Public Health Dent. 1995 Fall;55(4):250-2.