Endodontic variables and coronary heart disease

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This cross-sectional study was designed to explore a possible association between endodontic disease variables and coronary heart disease (CHD). Dental infections are hypothesized to be linked to atherosclerosis and could be a cause of vascular changes crucial for the development of CHD. Most studies have focused on periodontal disease. To our knowledge, no one has specifically studied endodontic variables as risk factors for the development of CHD. In 1992–93, a representative sample ($n = 1056$) of women in Göteborg, Sweden, aged between 38 and 84 years, took part in a combined dental and medical survey. The dependent variable was CHD, i.e. subjects with angina pectoris and/or a history of myocardial infarction ($n = 106$). The independent variables were number of root-filled teeth (RF), number of teeth with periapical radiolucencies (PA), tooth loss (TL), age, life situation, marital status, smoking, alcohol habits, body mass index, waist-hip ratio, serum cholesterol and triglyceride concentrations, hypertension and diabetes. The multivariate logistic regression analysis did not prove the endodontic variables to be predictive of CHD. Only age and tooth loss were significantly associated with CHD, with OR = 1.07 (CI = 1.03–1.12) and OR = 2.70 (CI = 1.49–4.87), respectively. The bivariate logistic regression analysis showed a positive significant association between subjects with RF = 2 and CHD, but for PA the bivariate analysis did not support an association with CHD. This cross-sectional study did not reveal a significant association between endodontically treated teeth and CHD nor between teeth with periapical disease and CHD. □ *Age; CHD; endodontic; toothloss; women*

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In a number of studies, an association has been suggested between chronic infections and coronary heart disease (CHD) (1–3). Mattila et al. (4) found that dental infection was associated with an increased risk of myocardial infarction, even after adjusting for several known CHD risk factors. Since the Mattila et al. study (4), there have been several cohort and cross-sectional studies suggesting a positive association between toothloss, dental infections/periodontitis and CHD. Most studies show associations with odds ratios (OR) ranging from 1.2 to 1.5 (5). Beck et al. (6) found a significant OR of 1.5 for CHD when comparing subjects with 'high' and 'low' bone loss, and 3.6 for subjects whose teeth all had at least one site with probing depth exceeding 3 mm. They thus found a dose–response relationship between the severity of periodontal disease and risk for CHD. The odds ratio was adjusted for age and other known risk factors. Paunio et al. (7), using number of missing teeth as expression for chronic oral infections, found this variable to be significantly associated with ischemic heart disease. Joshipura et al. (8) found a relative risk (RR) of 1.7 for tooth loss, but only in subjects with pre-existing periodontitis. DeStefano et al. (9) found an increased risk for CHD associated with periodontitis and poor oral hygiene (RR = 1.25) which was even more pronounced for subjects 50 years and younger (RR = 1.72).

This issue is controversial, because there are studies that have not shown any association between poor oral health and CHD. Hujoel et al. (10) found a non-significant increased risk of CHD for gingivitis (hazard ratio (HR) = 1.05) and periodontitis (HR = 1.14). Mattila et al. (11) used information on oral health status, both radiological and clinical data, and found higher dental indices among patients with CHD, but no significant differences between subjects with and without CHD. The authors speculated that the high age of the subjects was one reason for the result.

Although dental infections are prevalent in society, most studies so far have focused on periodontal disease. The periodontal infection shares common features with the infection in necrotic root canals, as the species isolated in infected periodontal pockets are almost the same as those isolated from the infected root canal (12). It could be hypothesized that the same association found between periodontitis and CHD could be revealed between apical periodontitis and CHD, but to our knowledge this possible relationship has not been studied.

The aim of this study was to explore the association between endodontic variables, i.e. root-filled teeth and apical periodontitis, and cardiovascular heart disease.

Material and methods

In 1968, the Population Study of Women in Göteborg, Sweden, was initiated. Randomly selected, 1622 women (aged 38, 46, 50, 54, and 60 years) were sampled for a combined medical, psychiatric, and dental examination in

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Table 1. Age distribution between probands with and without cardiovascular heart disease (CHD)

Age	CHD	Non-CHD
38	0	61
50	0	92
62	18	260
70	36	267
74	35	188
78	13	67
84	4	15

which 1462 women took part, corresponding to a participation rate of 90.1% (13). The same women were re-examined in 1980–81 and 1992–93. Of those studied in 1968–69 and 1980–81, 836 took part in the medical study and 702 in the dental study in the follow-up examination in 1992–93. With new probands born 1942 and 1954 added, and extension of the age groups of women born in 1922 and 1930, the medical part of the study, in 1992–93, consisted of 1087 participants. Of those, 1023 took part in the dental study. Our study used information from the 1992–93 examination, providing information for 1056 women regarding cardiovascular disease (14, 15).

The dental substudy included a clinical and radiological examination (panoramic) and a questionnaire. All data were collected during the examination (14). Subjects with CHD were those with angina pectoris and/or a history of myocardial infarction ($n = 106$) and constituted the dependent variable. Angina pectoris was diagnosed using the questionnaire by Rose (16). Myocardial infarction was diagnosed if at least two of the following criteria were present: 1) central chest pain, 2) transient rise of transaminase activities, and 3) typical ECG changes of recent onset (17, 18). The age distribution between those with CHD and those without CHD can be seen in Table 1. We studied the characteristics of women with and without CHD with regard to the independent variables.

The number of teeth (range 0–32) and endodontic variables, such as root-filled teeth (RF = 0, 1, 2, >2) and teeth with periapical destructions (PA = 0, 1, 2, >2), were obtained from the panoramic radiographs. The registrations were done by two calibrated radiologists. A periapical destruction was defined as equal or more than a widened periapical ligament space (19). The other independent variables were taken from the medical part of the study (Table 2) and constituted other well-known risk factors of CHD. Age was used as a continuous variable in the analysis. Diabetes and hypertension were dichotomized (yes/no), where hypertension was answered with 'yes' if the subject had systolic blood pressure $\geq$160 and/or diastolic blood pressure $\geq$95 and/or was treated pharmacologically against hypertension. Cholesterol and triglyceride concentrations were measured as mmol/l and taken from blood samples. Body mass index (weight/height^2) (BMI) and waist-hip ratio (WHR) were calculated.

Table 2. Independent variables. Mean values, standard deviations, and frequencies

	Mean	S
No. of teeth	19.9	7.2
Root-filled teeth (RF)	3.8	3.1
Teeth with periapical destruction (PA)	0.5	0.9
Age	64.7	10.7
Serum cholesterol	6.2	1.1
Serum triglycerides	1.4	0.7
BMI	25.8	4.2
WHR	0.8	0.1
Diabetes	No (1) = 95.1%	
	Yes (2) = 4.9%	
Hypertension	No (0) = 60.2%	
	Yes (1) = 39.8%	
Smoking		
Never	57.5%	(1)
Former	19.8%	(2)
Current	22.7%	(3)
Alcohol habits		
Never	21.5%	(0)
Weekly	70.9%	(1)
Daily	7.6%	(2)
Life situation		
'Acceptable'	85.3%	(0)
'Poor'	14.7%	(1)
Marital status		
Married	57.0%	(1)
Widow	20.8%	(2)
Unmarried/divorced	22.1%	(3)

Smoking was assessed as never, former, or current and alcohol habits as never, weekly, or daily wine consumption. Intake of spirits and beer was excluded because of the very small consumption of spirits, and because of confusing data regarding consumption of beer, where also 'light beer', a non-alcoholic beverage, was considered (20). Life situation was assessed as follows: the participants valued their life situation after being asked 'are you satisfied with your life situation'? on a scale from 1 to 7. The variable was then dichotomized into 'acceptable' (1–4) and 'poor' (5–7) as a crude measure of general well-being. For marital status, the women were categorized as married, widows, or unmarried/divorced.

Statistical analysis

Bivariate logistic regression was used for RF and PA, adjusted for tooth loss (TL), with RF and PA as categorical variables as well as dichotomized (0 vs $\geq$1). TL was dichotomized to loss of $\leq$16 teeth or >16 teeth. Edentulous subjects were not included in the analysis.

Multivariate logistic regression modeling was performed with the measured independent variables and the dependent variable CHD as outcome variable. When comparing groups, the independent t test was used for continuous variables and the χ^2 test for categorical variables.

Table 3. Differences regarding the independent variables between subjects with CHD and without CHD. Means, standard deviations, and frequencies

	With CHD $n = 106$		Without CHD $n = 950$		
	Mean	S	Mean	S	
No. of teeth	12.9	8.9	17.8	9.4	*
Root-filled teeth (RF)	3.0	3.1	3.4	3.2	
Teeth with periapical destruction (PA)	0.4	0.8	0.4	0.8	
Age	71.6	5.6	65.4	10.6	*
Diabetes	8.5%		4.5%		
Hypertension	53.8%		39.3%		*
Serum cholesterol	6.5	1.2	6.2	1.1	*
Serum triglyceride	1.7	0.9	1.4	0.7	*
BMI	27.2	4.7	25.9	4.3	*
WHR	0.84	0.1	0.82	0.1	*
Smoking					*
Never	59.4%		55.9%		
Former	12.3%		20.6%		
Current	28.3%		23.5%		
Alcohol habits					
Never	29.2%		23.5%		
Weekly	64.2%		69.8%		
Daily	6.6%		6.6%		
Lifesituation					*
'Acceptable'	75%		84.9%		
'Poor'	25%		15.1%		
Marital status					
Married	51.9%		54.7%		
Widow	28.8%		23.3%		
Unmarried/Divorced	19.2%		22.1%		

* $P < 0.05$. Independent t test for continuous variables and χ^2 test for categorical variables.

Table 4. Differences regarding the independent variables between subjects with toothloss >16 and ≤16 teeth (TL). Means, standard deviations, and frequencies

	Subjects with TL >16 $n = 245$		Subjects with TL ≤16 $n = 622$		
	Mean	S	Mean	S	
No. of teeth	10.2	4.6	23.7	3.5	*
Root-filled teeth (RF)	3.2	3.0	4.0	3.2	*
Teeth with periapical destruction (PA)	0.5	1.1	0.4	0.8	
Age	69.9	6.7	62.6	11.3	*
CHD	16.6%		5.6%		*
Diabetes	7.4%		3.9%		
Hypertension	52.3%		34.9%		*
Serum cholesterol	6.3	1.1	6.1	1.1	*
Serum triglyceride	1.5	0.65	1.3	0.73	*
BMI	26.7	4.5	25.5	4.0	*
WHR	0.84	0.06	0.82	0.06	*
Smoking					*
Never	50.6%		60.3%		
Former	24.9%		21.8%		
Current	24.5%		17.9%		
Alcohol habits					*
Never	31.1%		17.7%		
Weekly	66.0%		72.8%		
Daily	2.9%		9.5%		
Life situation					
'Acceptable'	82.5%		86.4%		
'Poor'	17.5%		13.6%		
Marital status					*
Married	45.0%		61.7%		
Widow	30.4%		17.1%		
Unmarried/divorced	24.6%		21.2%		

* $P < 0.05$. Independent t test for continuous variables and χ^2 test for categorical variables.

Results

The older probands constituted a significantly larger proportion of the CHD category than the younger probands. There were significant differences between women with CHD and women without CHD, where those with CHD were higher for known CHD risk factors (Table 3).

Comparing between dentate subjects with TL >16 and TL ≤16 showed significant differences; those with TL >16 had fewer root-filled teeth, they were older, they were more often diagnosed with CHD, more often hypertensive, had higher amounts of cholesterol and triglycerides, higher BMI and WHR, were more seldom never-smokers and daily alcohol consumers and were less often married (Table 4).

The bivariate analysis yielded a positive significant association between subjects with RF = 2 and CHD, but not for RF >0 (OR 2.13 (0.94–4.82); $n = 706$), when dichotomized (Table 5). For PA the bivariate analysis did not support a positive association with CHD, PA >0 (OR 1.07 (0.63–1.81); $n = 253$) (Table 6).

In the multivariate analysis no endodontic variable was significantly associated with CHD. Only age and TL >16 were significant predictors for CHD (Table 7).

Table 5. Bivariate logistic regression analysis between number of root-filled teeth (RF) and CHD as dependent variable

No. of RF	OR	CI (95%)	n
RF = 0	1.0	(reference)	129
RF = 1	1.66	(0.57–4.82)	95
RF = 2	3.34	(1.23–9.06)	95
RF >2	2.04	(0.88–4.72)	516

OR = odds ratio.
CI = confidence interval 95%.

Table 6. Bivariate logistic regression analysis between number of teeth with periapical destruction (PA) and CHD as dependent variable

No. of PA	OR	CI (95%)	n
PA = 0	1.0	(reference)	582
PA = 1	0.95	(0.50–1.79)	168
PA = 2	1.53	(0.65–3.62)	59
PA >2	0.88	(0.20–3.94)	26

OR = odds ratio.
CI = confidence interval 95%.

Table 7. Multivariate logistic regression between the independent variables and CHD as dependent variable

	OR	CI (95%)	
TL ≤16	1.0	(reference)	
TL >16	2.70	(1.49–4.87)	*
PA = 0	1.0	(reference)	
PA = 1	0.99	(0.51–1.94)	
PA = 2	1.98	(0.79–5.02)	
PA >2	0.95	(0.21–4.37)	
RF = 0	1.0	(reference)	
RF = 1	1.20	(0.38–3.80)	
RF = 2	2.17	(0.74–6.38)	
RF >2	1.52	(0.62–3.77)	
Age	1.07	(1.03–1.12)	*
Diabetes	1.30	(0.46–3.68)	
Hypertension	1.16	(0.67–2.02)	
Serum cholesterol	1.13	(0.88–1.44)	
Serum triglyceride	1.11	(0.74–1.68)	
Body mass index	1.04	(0.97–1.11)	
Waist-hip ratio	0.95	(0.07–137.5)	
Smoking			
Never	1.0	(reference)	
Former	1.03	(0.50–2.10)	
Current	0.90	(0.42–1.95)	
Alcohol habits			
Never	1.0	(reference)	
Weekly	0.84	(0.46–1.54)	
Daily	1.06	(0.31–3.57)	
Life situation			
'Acceptable'	1.0	(reference)	
'Poor'	1.42	(0.71–2.86)	
Marital status			
Married	1.0	(reference)	
Widow	0.87	(0.46–1.65)	
Unmarried/divorced	0.57	(0.26–1.23)	

* $P < 0.05$.
OR = odds ratio. CI = confidence interval 95%. TL = toothloss. RF = no. of root-filled teeth. PA = no. of teeth with periapical destruction.

Discussion

The objective of this cross-sectional study was to explore the association between endodontic variables and CHD. We found that neither root-filled teeth nor teeth with periapical destruction were significantly associated with CHD. However, age and tooth loss were predictive of CHD.

Infection and its association with atherosclerosis and CHD have received great attention in the past two decades. It is assumed to play a role in the etiopathogenesis of atherosclerosis and to precede the development of cerebral and myocardial infarction (21). Plausible biological mechanisms have been presented to support the observed relationship, but no clear-cut biological explanation is available as yet (6, 21). Chronic infections caused by *Helicobacter pylori* and *Chlamydia pneumoniae* have been studied, as well as dental infections (22). The association between infection and atherosclerosis/CHD, when studied epidemiologically, is generally weak, and reported significant results may be confounded with other well-known risk factors not always controlled for (22).

In our study, endodontic variables were in focus as possible risk factors for development of CHD. Teeth with periapical disease are known to harbour bacteria in the necrotic root canal, causing periapical inflammation (23, 24). Even root-filled teeth, with radiologically intact periapical conditions, are known to elicit a chronic inflammatory response periapically in many cases (25).

When accounting for several known confounding factors, no endodontic variable was predictive of CHD in the multivariate analysis. Only age and tooth loss showed a significant association with CHD. The reasons for this might be threefold: DeStefano et al. (9) and Mattila et al. (11) found that the association between dental disease and CHD was most pronounced in subjects 50 years or younger. In our study, no subjects in those age groups were diagnosed with CHD, but since those age groups are small in our sample, the results are inconclusive. Moreover, it is known that males have a higher incidence of CHD than women (5), and our study included only women, which may have influenced the outcome. Marginal periodontitis affects a great surface area (26), probably significantly greater than apical periodontitis. It can therefore be speculated that the inflammatory and microbiological burden may be greater in marginal periodontitis over time. The prevalence of periapical disease was fairly low in our material, while the frequency of root-filled teeth was high compared with other Swedish population studies (27–29, 30). The low prevalence of periapical disease could be explained by the method of detection; most population surveys cited have used full-mouth radiological surveys and it could be argued that our technique, panoramic radiologic survey, might be less sensitive in detecting periapical destruction. On the other hand, Ahlqwist et al. (19) found no differences regarding diagnosis of periapical pathology between panoramic and intraoral radiologic examination essential in an epidemiologic context. The low prevalence of periapical disease could thus be explained by the high prevalence of root-filled teeth. Gothenburg, Sweden, is an urban area with great accessibility to dental care, and in this context it seems just to assume that the low prevalence of periapical disease is a result of endodontic treatment. However, the low prevalence of periapical destruction could also explain the non-significant association between periapical pathology and CHD.

Periodontitis and CHD share common risk factors (31). In our study, tooth loss was the general indicator of dental disease, and it was found that probands with tooth loss shared common features with women diagnosed with CHD. Joshipura et al. (8) found about the same association between tooth loss and known risk factors for CHD, even though they did not present any statistical analysis.

Tooth loss has been significantly associated with CHD in several studies (7, 8, 32). As previously mentioned, subjects with tooth loss shared common etiological features with those with CHD in our study. However, when controlling for other well-known risk factors, tooth loss remained predictive of CHD. Previous dental disease and

dietary factors have been proposed as reasons for this association (33). In our study, we did not control for periodontal disease nor for dietary factors.

The sampling in this study was based on date of birth, and participation rate was high. These factors ensure a high representativity (for detailed information, see reference 15), but our sample was small compared with several other population surveys. However, the included variables could be examined more carefully compared to other studies, where data in some instances have been collected without clinical and/or radiological examination. In our study, medical and dental data were collected by medical and dental staff, respectively, in examinations including laboratory tests, interview, clinical and radiological examination.

Cross-sectional data are not valid for establishing a cause–effect relationship. We cannot therefore conclude that the relationship between age, tooth loss, and CHD is causal. Some authors have emphasized socio-economic factors as crucial confounders when studying possible risk factors for CHD. In our study, we partly adjusted the statistical model with regard to socio-economic and lifestyle factors. We controlled for civil status, a variable that could be accounted for as socio-economic and a life-satisfaction measure, but also for alcohol and smoking habits.

Longitudinal data on this issue, which are imperative for establishing a cause–effect relationship, are scarce. We plan to analyze data from a recent conducted prospective, longitudinal study with a 32-year follow-up.

In conclusion, we found that neither root-filled teeth nor teeth with periapical disease were predictive of CHD in our material.

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