

PERIODONTAL DISEASE AS A RISK FACTOR FOR ATHEROSCLEROSIS

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ABSTRACT

Atherosclerosis has been defined as a progressive process that causes focal thickening of large to medium sized muscular & large elastic arteries. Periodontal disease is destruction of tissue supporting the tooth that occurs following the disease. If the disease progresses without therapeutic intervention final outcome is loss of teeth. The focal progression of the periodontal disease correlates to the inflammatory biomarkers such as pro-inflammatory cytokines & serum antibody titers for pathogenic periodontal bacteria. Various studies have demonstrated a close association between cardiovascular disease & periodontitis & furthermore oral hygiene & periodontal status are closely related to the occurrence of heart attacks.

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INTRODUCTION

Atherosclerosis has been defined as a progressive process that causes focal thickening of large to medium sized muscular & large elastic arteries[1]. The development of atheromatous plaque seems to be relevant to cardiovascular disease as a result of endothelial cell damage and maintenance of the inflammatory reactions in the wall of blood vessels[2].

Studies have demonstrated a close association between cardiovascular disease & periodontitis & furthermore oral hygiene & periodontal status are closely related to the occurrence of heart attacks[3,4].

PERIODONTAL DISEASE

It is destruction of tissue supporting the tooth that occurs following the disease. If the disease progresses without therapeutic intervention final outcome is loss of teeth. The focal progression of the periodontal disease correlates to the inflammatory biomarkers such as pro-inflammatory cytokines & serum antibody titers for pathogenic periodontal bacteria.

FOCAL MEASURES OF PERIODONTAL DISEASE

Different modalities to access exposure variables are:

1. Etiologic bacteria

Periodontopathic bacteria may directly or indirectly contribute to the progression of cardiovascular disease by increased blood platelet co-aggregation, enhanced low density cholesterol in the artery walls, invasion of cardiac & carotid endothelium, high levels of inflamma-



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tory mediators in the circulation & tissues. The amount of the infective load can be estimated through bacterial DNA amplification with nested PCR used fully for investigation.

2. Periodontal disease index

The severity of periodontal disease can be measured by following parameters of periodontal disease such as clinical loss of attachment loss, gingival bleeding index.

3. Bone loss

Radiographs are performed in order to measure the amount of bone loss supporting the diseased teeth.

4. Tooth loss

Measuring the number of remaining teeth in patients with periodontal disease is also important and might be the more objective measure for the definition of periodontal disease

5. Community Periodontal Index for Treatment Needs (CPITN)

CPITN was developed jointly by the International Dental Federation & WHO in 1983. It aims to evaluate periodontal condition and treatment needs [5].

6. Epidemiology for the association between periodontal and coronary artery disease.

In systematic review, examining five perspective studies with approximately 90,000 patients with periodontitis the relative risk for cardiovascular disease was 14% in almost ten years. In same meta-analysis, the relative risk for prevalent periodontal disease was 120% for cases compared to controls and in cross sectional studies that risk was higher in the former group by 60% compared to the latter[6]. The first study evaluated by Matilla showed that myocardial infarction increased compared with their periodontal status with healthy controls from the same population and demonstrated a more deteriorated periodontal status with respect to controls[7].

Many other studies for positive and negative [Table.1 and 2] have been conducted after controlling for different overlapping risk factors and recruiting population free of coronary artery disease. Although in these association the outcome was well defined, periodontal measures varied significantly among the studies. Indeed, tooth loss, bone loss constituted different expressions of periodontitis convicting the validity of the investigated association.

DISCUSSION

Beck JD, Tonetti et al. demonstrated significant correlation between periodontitis and intermediate end points of vascular disease such as intima media thickness, arterial stiffness, endothelial dysfunction and albuminuria[8,9]. The emerging finding by Devariex M, reported positive

First Author (year)	Type of study (duration)	Sample size	Main Finding
Mattila (1989)	Case-control	100 vs. 102	Association between recent myocardial infarction and total dental index
De Stefano (1993)	Retrospective (14 years)	9,760	Severe vs. mild disease: 25% relative risk increase
Beck (1995)	Prospective (4 years)	1,147	50% increase in the risk for CAD in severe periodontitis Patients
Mattila (1995)	Prospective (7 years)	214	In CAD patients dental health predicted incident fatal and nonfatal coronary events
Morrison (1999)	Retrospective (20 years)	21,500	Severe gingivitis predicted for fatal CAD with adjusted OR of 2.15, whereas edentulous status with adjusted OR of 1.90
Jansson (2001)	Prospective (27 years)	1,393	In patients aged <45 years the RR for CVD was 2.70 in those with marginal bone loss of >10% compared to subjects with mean marginal bone loss ≤10 %.

*(Mattila KJ et al,1989)

Table.1:Positive epidemiological evidence for the association between periodontitis and CAD*[7].

association between periodontal disease measures and blood pressure levels[10].

In the study by Tonetti et al. almost 100 patients with severe periodontitis were randomized in intensive and standard treatment for periodontal disease. Before any treatment, measures of endothelial function, systemic inflammation and molecular mediators mainly derived by endothelial cells were assessed. The two arms of treatment were followed

First Author (year)	Type of study (duration)	Sample size	Main Finding
Joshiyura (1996)	Prospective (6 years)	44,119	No relationship with CAD after adjustment for dietary risk factors.
Howell (2001)	Prospective (12 years)	22,037	Reported PD vs. no PD: no difference in CAD.
Mattila (2000)	Case – control	85 vs. 53	CAD patients vs. controls demonstrated the same magnitude of PD measures after careful adjustment for confounders.
Hujoel (2001)	Prospective NHANES I	4,027	Risk of CAD for those with PD: no difference with edentulous.
Hujoel (2001)	Prospective NHANES I	8,032	PD: no association with coronary events

*(Mattila KJ et al,1989)

Table.2:Negative epidemiological evidence for the association between periodontitis and CAD*[7]

for six months and at the end of the study those who underwent intensive treatment demonstrated ameliorated endothelial mediators dilation and decreased e- selective levels in the context of lower levels of neutrophil counts compared to the standard periodontal therapy group. Over six month follow up period, treatment of periodontitis resulted in amelioration of endothelial function, whereas non treatment was accompanied by the same of endothelial dysfunction[9].

The cross sectional arm of the infection and vascular disease epidemiologic study suggested that patient aged less than 65 yrs demonstrated an association between number of missing teeth and prevalence of carotid plaques, a finding not observed in those edentulous and aged over 65 yrs[10].

Radiographically assessed bone loss was associated with same atherosclerosis oriented outcome with exception of edentulous status[11]. Intimamedia was associated with the periodontal pathogens, over and above the levels of systemic inflammation in another cohort of the INVEST. Finally the levels of systolic and diastolic Blood pressure were higher in those at higher periodontal pathogens as compared with the lower tertile[10].

CONCLUSION

Chronic periodontal infection, like other chronic infection, may play a role in atherogenesis and the pathogenesis of clinical sequence such as myocardial infarction. Periodontal infection can increase the risk of myocardial infarction in several ways. Coronary artery disease is a leading cause of death in most developed countries including the United States. Periodontal disease is also highly prevalent. There is some evidence suggesting that periodontal pathogens could modulate the initiation and perpetuation of atherosclerosis, the prevention and treatment of periodontal infection may be very important in reducing mortality and morbidity, associated with Coronary artery disease.

REFERENCES

1. Pucar, A., Milasin, J., Lekovic, V., et al. Correlation between atherosclerosis and periodontal putative pathogenic infection in coronary and internal mammary arteries. *J Periodontol* 2007; 7: 677-682.
2. Libby. Inflammation and atherosclerosis. *Circulation* 2002;105: 1135-43.
3. Kozarov, E., Sweier, D., et al. Detection of bacterial DNA in atheromatous plaques by Quantitative PCR. *Microbes Infect* 2006; 8: 687-93.
4. Johansson, C., S., Richter A. Periodontal conditions in patients with coronary heart disease: A Case Control Study. *J Clin Periodontol* 2008; 35: 199-205.
5. Costas Thomopoulos, CoatasTsioufis et al. Periodontitis and coronary artery disease: a questioned association between periodontal and vascular plaques. *Am J Cardiovasc Dis* 2011; 1:76-83.
6. Bahekar AA, Singh S et al. The prevalence and incidence of coronary heart disease is significantly increased in periodontitis: A meta analysis. *Am Heart J* 2007; 1(54): 830-837.
- 7*. Mattila KJ et al. Association between dental health and acute myocardial infarction. *BMJ* 1989; 297: 779-791.
8. Beck JD. Relationship of periodontal disease to carotid artery intima media wall thickness: the atherosclerotic risk in communities (ARIC) Study. *ArteriosclerThromb Vasc Biol* 2001; 21:1816-1822.

9. Tonnetti Ms. Treatment of periodontitis and endothelial function. N Engl J Med 2007; 356:911-920.
10. Devariex M. Periodontal microbiota and increased Intima media thickness: oral infections and Vascular Disease: Epidemiologic study (INVEST). Circulation 2005; 111: 576-82.
11. Engebretson SP, Lamster IB et al. Radiographic Measures of Chronic periodontitis and Carotid artery plaque. Stroke 2005; 36:561-66.

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