



Original Research Article

Periodontal disease with risk of cardiovascular complications: A Microbiological study

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A B S T R A C T

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Periodontal disease is characterized by progressive periodontal pathogens. Cardiovascular disease is the leading cause for morbidity and mortality in periodontitis patients. In this, we investigated as the association between periodontal disease and an increased risk of cardiovascular complications; the main aim is that the periodontal infections may increase the systemic inflammatory burden of the host above a threshold that may favour the atherogenic processes. The main objectives as includes, 1. To estimate periodontal pathogen in periodontitis patients, 2. To associate and to search for periodontal conditions related to the increased cardiovascular risk factors, 3. To compare the periodontal condition cases and healthy controls. The case study group included 30 patients diagnosed as periodontal disease patients and 15 healthy controls. The overall periodontal conditions resulted worse in the test group. In particular periodontal conditions such as the presence of deep pockets (probing depth >6 mm) and the loss of more than 12 teeth might represent indicators of a strongly increased risk of cardiovascular disease and microbiological investigations confirmed these findings; Phorphyromonas gingivalis was the most common bacteria. This study supports the existence of an epidemiologic association between periodontal disease and cardiovascular disease and confirms previous data present in the literature. Two periodontal parameters, deep pockets and number of missing teeth, seem to be important risk factors for cardiovascular diseases.

Introduction

Periodontal diseases are pathological processes that affect the periodontal tissues; usually refer as gingivitis and periodontitis. Periodontitis is defined as “an inflammatory disease of the supporting tissues of the tooth caused by specific microorganisms,

resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both”, while gingivitis is inflammation of the gingiva. (Rajendran and Sivapathasundharam., 2012). The essential role of periodontal pathogens in promoting both inflammatory

disease (gingivitis) and destructive disease (periodontitis) is well established. Bacteria are a primary etiologic causative agent in inflammatory periodontal diseases. It affects a large number of individuals, especially adult's oral infection and promotes continuous exposure to bacteria, endotoxin (lipopolysaccharides) and other bacterial products (Page, 1986).

Many bacteria are thought to be involved in the pathogenesis but a restricted group has proved to be the cause of the disease; this list includes *Prevotella intermedia*, *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis* and *Tannerella forsythia*. There was potential linkage between periodontitis and plasma fatty acids profile, an established cardiovascular disease (CVD) risk factor (Ramirez-Tortosa *et al.*, 2010) (Table 1).

The etiology of periodontitis is infectious, a poor level of oral hygiene and the accumulation of dental plaque is the cause of the manifestation of the disease. The pathogenesis of periodontitis is complex and multifactorial, inflammation and host related factors are of crucial importance in the development and severity of the disease (De Nardin, 2001). Periodontitis is the most common mouth disease in the adult population of the industrialized countries (AAP, 1996; NIDR, 1987) and is also the primary cause of tooth loss among the same populations (Flemming, 1999).

Materials and Methods

This cross-sectional studies (Case Control) was conducted from Dec 2013 to Sep 2014 after the protocol was approved by the Institutional Ethics Committee (IEC) of College of Life Sciences, Cancer Hospital & Research Institute (CHRI), Gwalior (M.P), India.

Periodontal Parameters

A full- mouth periodontal examination was performed by a single examiner (N.D) the patients were classified as having mild, moderate or severe periodontitis on the basis of extend and severity index.

Recently, an association between periodontitis and cardiovascular disease (CVD) has received considerable attention. The present study is aimed to assess the possible associations which underlie the pathogenesis of periodontitis and cardiovascular disease (Dhotre *et al.*, 2011).

Results and Discussion

The average age of the two groups was, as expected, not statistically different with a value for the test group of 57.7 and for the control of 55.1 (p value 0.217). The only cardiovascular complication found with a statistically significant difference was the incidence of dyslipidemia, higher in the test group. For all the other risk factors the distribution was similar in both groups (Table 1). Among the periodontal parameters a significant difference was found only for the number of missing teeth (Table 2), and the number of sites probing more than 6mm, as reported in Table 3. This last index was significant however it was investigated: average number per patient, percentage, first and second dichotomization.

The present findings confirm those reported by others and indicate a possible relation between periodontal disease and cardiovascular atherosclerosis (De Stefano *et al.*, 1993; Mattila *et al.*, 1989, 1993 and 2000; Josphipura *et al.*, 1996; Genco *et al.*, 1999; Morrison *et al.*, 1999). In particular, all the periodontal parameters are worse in the test than in the control group, even

though for a few of these the statistical significance of these differences is not reached. The most interesting parameters are the presence of sites with PD >6 mm and the average CAL values. With regard to the presence of deep pockets the difference is statistically different whatever the investigation: absolute number, percentage of deep pockets on the total of sites probed, ranked dichotomic non-parametric transformation in both ways it was performed.

This kind of result strongly indicates the possibility that having deep pockets is a risk factor of developing and/or worsening a cardiovascular complication. In fact only deep pockets can harbor significant colonies of periopathogens (Kuramitsu *et al.*, 2001) and can stimulate a significant inflammatory response able to influence systemic health such as the development and/or worsening of cardiovascular atherosclerosis (Wick *et*

al., 1995; Deliargys *et al.*, 2000; Loos *et al.*, 2000; WU *et al.*, 2000; De Nardin, 2001; Noack *et al.*, 2001). The bacterial species isolated in this study are in accordance with the literature (Haffajee *et al.*, 2004), with the exception of *Prevotella* genus, which was more frequent in our geographic area, as well as *S. constellatus*, an opportunistic pathogen collected from cardiopathic and non cardiopathic patients. In conclusion, our findings confirm literature in the direction of a statistical association between periodontitis and atherosclerosis. In particular in this trial the presence of deep pockets appeared to be a strong risk factor for cardiovascular disease. Our study is a case control studies so, despite a strong association, we cannot make any statement of a possible cause-effect relationship between the two conditions. This must be clarified by other studies such as interventional studies or very long cohort prospective studies.

Table.1 Categories of cardiovascular complication cases (as cardiopathic patients) and controls group (healthy patients)

S.NO	Patients	Underlying disease (present)	Cardiopathic (test group)	Non cardiopathic (control group)
1.	Hypertension	11 (40.7%)	3 (18.7%)	0.144
2.	Diabetes	1 (3.7 %)	2 (12.5%)	0.285
3.	Dyslipidemia	22 (81.5%)	4 (25%)	< 0.001
4.	Smoking	13 (48.1%)	7 (43.7%)	0.786

Table.2 Average values of periodontal parameters in test group and healthy control group

S.NO	BoP	%	PD mm	CAL mm	MT n°
1.	Cardiopathic (test)	32.1	2.93	3.45	14.6
2	Non cardiopathic (control)	35	2.81	3.12	7.8
3.	P value	0.572	0.426	0.145	0.045

BoP: Bleeding on Probing; PD: Probing Depth; CAL: Clinical Attachment Level; MT: Missing Teeth; Mm = millimeters

Table.3 Analysis of periodontal parameters in test group and healthy control group

S.NO	CAL	MT sites 4mm<=sites	PD>6mm	PD<6mm
1.	Dichotomic index A: rank 0	Dichotomic index B: rank 0	Average value	Average value
2.	Patients with average CAL <3.5 mm : rank 0	Patients with <12 MT: rank 0	Rank: 0	Rank: 0
3.	Patients with 3 or more sites with PD >6 mm : rank 1	Patients with 1 or more sites with PD >6 mm: rank 0	Rank: 1	Rank: 0
4.	Patients with average CAL >3.5 mm : rank 1	Patients with >12 MT: rank 1	Rank: 1	Rank: 1
5.	Patients with less than 3 sites with PD >6 mm	Patients with no sites with PD >6 mm	Rank: 1	Rank: 0

CAL: Clinical attachment level; MT: Missing teeth; PD: Probing depth

Table.4 Distribution of the investigated periodontal pathogens in test group and control groups

S. NO	Periodontal pathogens	Cardiopathic (test)	Non cardiopathic (control)
1.	<i>Porphyromonas gingivalis</i>	9 (47.4%)	1 (6.6%)
2.	<i>Prevotella intermedia</i>	4 (21%)	0
3.	<i>Prevotella buccae</i>	4 (21%)	1 (6.6%)
4.	<i>Prevotella oralis</i>	4 (21%)	3 (20%)
5.	<i>Tannerella forsythia</i>	2 (10.5%)	0
6.	<i>Fusobacterium nucleatum</i>	4 (21%)	2 (13.3%)
7.	<i>Capnocytophaga sputigena</i>	1 (5.3%)	0
8.	<i>Bifidobacterium</i>	3 (15.8%)	0
9.	<i>Streptococcus constellatus</i>	4 (21%)	7 (46.6%)

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