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Research Article

PREVALANCE OF PERIODONTITIS IN CARDIOVASCULAR DISEASE PATIENTS: AN OBSERVATIONAL STUDY

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A B S T R A C T

Cardiovascular diseases (CVDs) are a group of disorders of the heart and blood vessels and they include: coronary heart disease, cerebrovascular disease, peripheral arterial disease, rheumatic heart disease, congenital heart disease, deep vein thrombosis and pulmonary embolism. Oral infection models have emerged as useful tool to study the hypothesis that periodontal infection have its association with cardiovascular diseases. Periodontal infections are a leading culprit with studies reporting its association with various systemic conditions. It is also found that periodontal disease is a risk factor for cardiovascular disease but very few studies showed the prevalence rate. The aim of this study is to evaluate the periodontal status of cardiovascular disease patients. The periodontal parameters gingival index, bleeding on probing, probing pocket depth and clinical attachment loss were assessed. Some demographic factors were also considered. A total of 280 cardiac patients were evaluated from the hospitals of Nashik district, Maharashtra. It was found that 130 patients (46.5%) had periodontitis while 91 patients (32.5%) had gingivitis and only 59 patients (21%) had no periodontal disease. Evidence suggests that there is a strong prevalence of periodontitis in cardiovascular patients.

INTRODUCTION

Periodontitis is a chronic infection by oral bacteria that affects the supporting structures of the teeth.1 A mechanism has been proposed whereby the burden of bacterial pathogens, antigens, endotoxins, and inflammatory cytokines of periodontitis contributes to the process of atherogenesis and thromboembolic events. In response to infection and inflammation, susceptible individuals may exhibit greater expression of local and systemic mediators and may thereby be at increased risk for a myocardial infarction or stroke.2It was suggested that periodontal disease, once established provides a biological burden of endotoxin (lip polysaccharide) and inflammatory cytokines, especially thromboxane A2, prostaglandin E2,2 interleukin (IL)1L=1α, and tumor necrosis factor-beta, which serve to initiate and excerbate atherogenesis and thrombembolic events.3 The association of coronary heart disease and periodontal disease may be due to an underlying response trait, which places an individual at high risk for developing both periodontal disease and atherosclerosis.

International Classification of Diseases,9th Revision defined diseases of the circulatory system as follows:

(1) Ischemic heart diseases, (2) cerebrovascular diseases, (3) diseases of arteries, arterioles and capillaries (known as peripheral vascular disease), arterial septal vascular disease (ASVD) affect the heart and blood vessels; which is a major component of the cardiovascular system (CVS).4 It is a chronic process over many years but it can cause acute clinical events including acute coronary syndrome (ACS), myocardial infarction (MI), and strokes.

There are evidences that dental infection, particularly periodontal disease, is possible a risk factor for atherosclerosis coronary artery diseases. Patients who have valvular defects (congenital or acquired as result of post-rheumatic fever) or some other congenital defects such a septal defects or who have prosthetic valves, should receive antibiotic therapy as prophylaxis before dental extraction(high-riskgroup),scaling or periodontal surgery. These are likely to release a significant number of bacteria from the gingiva, particularly periodontal pockets. The role of periodontal infection/ inflammation is a risk factor for atherosclerosis.5 These observations are further corroborated in animal studies that demonstrates that oral infection of atherosclerosis-prone (apolipoprotein E-deficient) mice with P gingivalis resulted in accelerated atherosclerosis and the concomitant presence of Porphyromonas gingivalis DNA in their aortic tissue (Lalla *et al*.,2003) 6. Are centre view of the epidemiologic pattern of periodontitis report arrange in prevalence of severe periodontitis from 1% among 20-29 years to 39% among individuals more than 65 years of age.7 Poor periodontal status was significantly associated with increased C-reactive protein (CRP) and fibrinogenlevels. Another group in vestigated the associate on between

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periodontitis and subclinical atherosclerosis, commonly measured by means of carotid artery intima-media thickness (cIMT) assesments.8 Increased cIMT has been documented to be directly associated with increased risk of MI and stroke (O' Leary *et al*., 1999). Becker *et al*. (2001) provided the first evidence that periodontitis may be linked to subclinical atherosclerosis.9 Several studies have shown that periodontal diseases are associated with heart disease. While a cause and effect relationship has not yet been proven. Patient at risk for infective endocarditis may require antibiotic before adental procedure. Cardiologist and periodontist willed cide if the existing heart condition requires the use of antibiotics before dental procedure.10 Additional studies have pointed out to a relationship between periodontal disease and stroke.In one study that looked out the cause relationship of oral infection as a risk factor for stroke, people diagnosed with acute cerebrovascular ischemia were found more likely to have an oral infection when compared to those in the controlgroup.11

A study done in the late 1980 sby Simonkaetal,12 showed that patiens with myocardial infarction had a higher prevalence of periodontal disease. Since then, there has been a variety of literature studying the association between poor oral health and cardiovascular disease (CVD). Immunostainings of carotid endarterectomy specimens have shown the presence of 2 major odontopathogens, Porphyromonas gingivalis and Streptococcus sanguis, in atherosclerotic plaques.13 Thus, the release of bacteria and proinflammatory mediators such as bacterial endotoxins and cytokines in the blood stream that causes the release of acute phase reactants (such as C-reactive protein) leading to increased inflammatory activity in atherosclerotic lesions may represent the link between periodontal infection andCVD.14,15

Although the evidence of a potentially contributory role of periodontal infections in the natural history of CVD continues to mount, there are well-founded reasons for skepticism. With this in mind, we underwent an observational study with the aim of finding out the prevalence of periodontitis in cardiovascular disease patients.

MATERIALS AND METHODS

Study design and study population

The present study is an observation a investigation of perio dontal clinical parameters of CH Drisk performed on the 280 subjects with cardiovascular diseases. The patients were examined randomly with the history of cardiovascular diseases from the hospitals of district: Nashik, Maharashtra. Males and females with the age range of 20 years to 80 years were selected. Systemic history of which patient was taken. Three clinical parameters are typically recorded in epidemical studies of cardiovascular disease patients to asses prevalence: (1) Bleeding on probing which reflects the presence of an inflammatory infiltrate in gingival tissue, (2) Pocket depth which describes the deepening of gingival sulcus from which dental plaque biofilm can propagate, and (3) Clinical attachment level, which reflects the amount of periodontal tissue loss. Bleeding on probing and increased pocket depth indicates current pathology, whereas attachment levels provide a cumulative measure of loss of support caused by aggregate effects of pathogenetic factors suchas cardiovascular disease and trauma. The periodontal parameters i.e. bleeding on probing, pocket depth and clinical attachment loss. The patients were categorizedas:

Group A: Periodontally Healthy no evidence of clinical attachment loss, clinical inflammation, sulcular bleeding.
Group B: Gingivitis- presence of bleeding on probing at any site.
Group C: Chronic Periodontitis -having moderate to severe alveolar bone loss, clinical attachment loss of equal to or more than 4mm & probing depth of equal to or more than 5mm.

RESULTS

The baseline characteristics of the subjects varied according to severity of periodontal disease. Subjects with more severe disease were more likely to be men and were more from the age group of 51-60 years. (table 1). It was reported that out of 280 patients, 73 were hypertensive while 52 were diabetic and 36 patients had both diabetes and hypertension. (table3) Although the proportion of people who reported having diabetes or high blood pressure increased as severity of periodontal disease increased, some of the increase may be are flection of the older ages of those with more severe disease.254patients had bleeding on probing which accountedto90.72%.Only9.28%had absence of bleeding on probing. (table 4)

It was reported that 148 (52.85%) patients had a pocket depth of 0-3mm while the remaining 132 (47.15%) patients had pocket depth of more than 4mm. 76 (27.14%) out of 132 patients had shallow pockets while 52 (18.57%) patients had deep pockets (table 5 and 6).It was noted that152 (54.28%) patients had clinic a attachment levelof0-3mm while the remaining 128 (45.72%) had clinical attachment level of more than 4mm. Out of 128 patients, 32, 52 and45 patients had Clinical attachment loss of 4mm, 5mm and 6mm respectively.(table 7 and 8). According to the criteria for group A, group B and group C it was found out that 128 (45.72 %) had periodontitis while 126 (45%) had gingivitis and only 26 (9.28%) had healthy gingiva.t would be said that out of 280 patients, 254 were affected by periodontal disease while 26 were unaffected.

Table 1 Distribution of study population according to age

Age Groups	No.(n)	Percentage (%)
20 - 30 years	31	11.07%
31 – 40 years	45	16.07%
41 -50 years	69	24.64%
51-60 years	83	29.64%
61- 70 years	33	11.78%
71- 80 years	19	6.78%
Total	280	100%

Table 2 Distribution of study population according to gender

Gender	No.(n)	Percentage (%)	Chi-square test	p value, Significance
Male	194	69.28%	17.84	P <0.001, highly significant
Female	86	30.72%		
Total	280	100%		

Table 3 Distribution of study population in relation to prevelance of systemic conditions

Systemic conditions	Number (n)	Percentage (%)
Hypertension	73	26.07%
Diabetes	52	18.57%
Hypertension &diabetes	36	12.85%

Table 4 Distribution of study population based on bleeding on probing

Bleeding on probing	n (%)	Chi – square test	Pvalue, Significance
Present	254 (90.72%)	34.83	p < 0.001, highly significant difference
Absent	26 (9.28%)		
Total	280 (100%)		

Table 5 Distribution of study population based on mean pocket depth

Mean Pocket depth	n (%)	Chi – square test	
0-3 mm	148 (52.85%)	1.47	p = 0.291, no significant difference
≥ 4 mm	132 (47.15%)		
Total	280 (100%)		

Table 6 Distribution of study population based on distribution of mean pocket depth

Mean Pocketdepth	n (%)	Percentage (%)
0-3 mm	148	52.85%
4-5 mm (shallow pockets)	76	27.14%
6-8 mm (deep pocket)	52	18.57%
Total	280	100%

Table 7 Distribution of study population based on mean CAL score

Mean CALscore	n (%)	Chi – square test	
0-3 mm	152 (54.28%)	1.75	p = 0.182, no significant difference
≥ 4 mm	128 (45.72%)		
Total	280 (100%)		

Table 8 Distribution of study population based on distribution of Mean CAL score

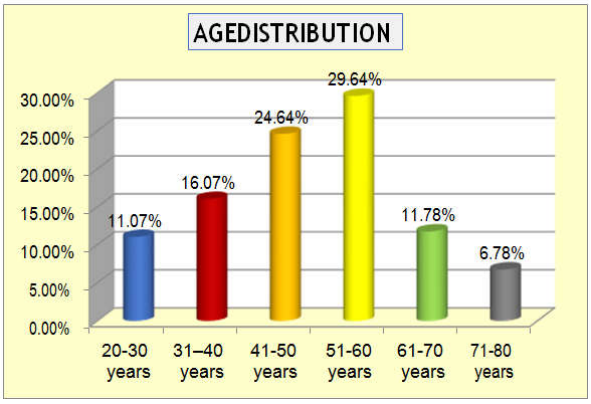
Mean CAL score	n (%)	Percentage (%)
0-3 mm	152	54.28%
4mm	32	11.42%
5 mm	51	18.03%
6 mm	45	16.27%
Total	280	100%

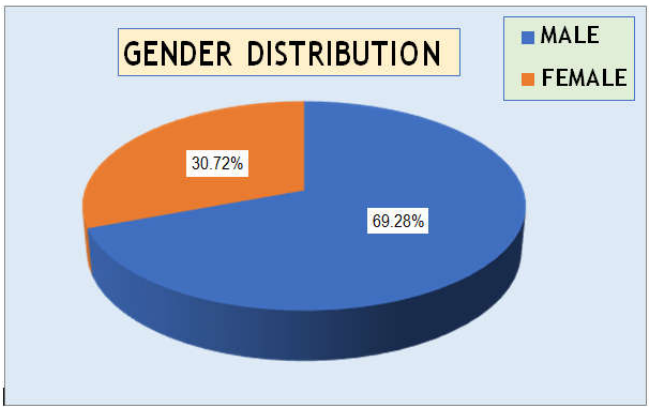
Table 9 Distribution of study population based on frequency of distribution of periodontal disease

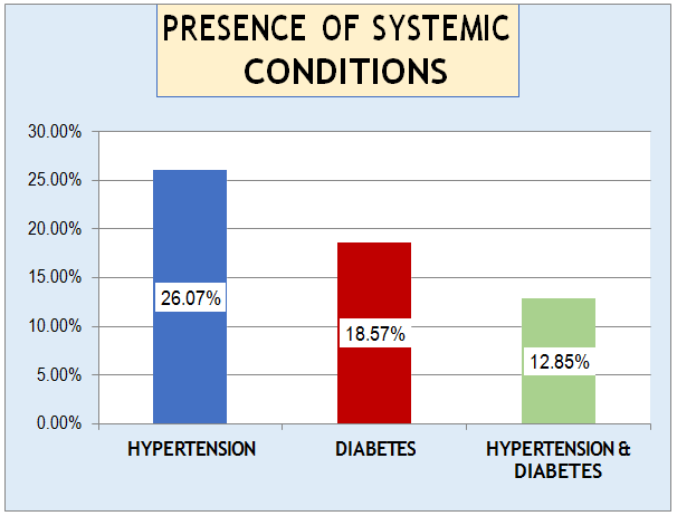
	Number (n)	Percentage (%)
Normal	26	9.28%
Gingivitis	126	45 %
Periodontitis	128	45.72 %
Total	280	100%

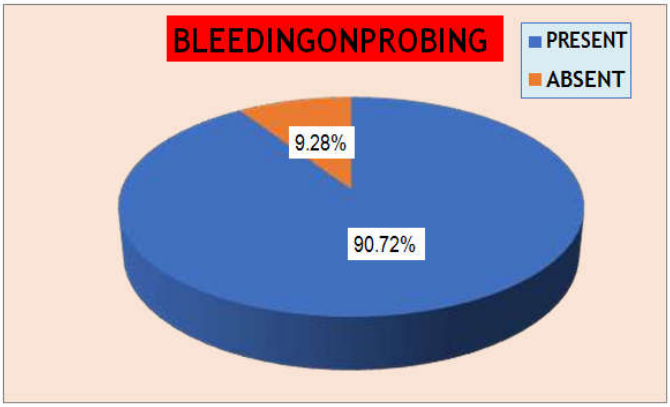
Table 10 Distribution of study population based on frequency of distribution of periodontal disease

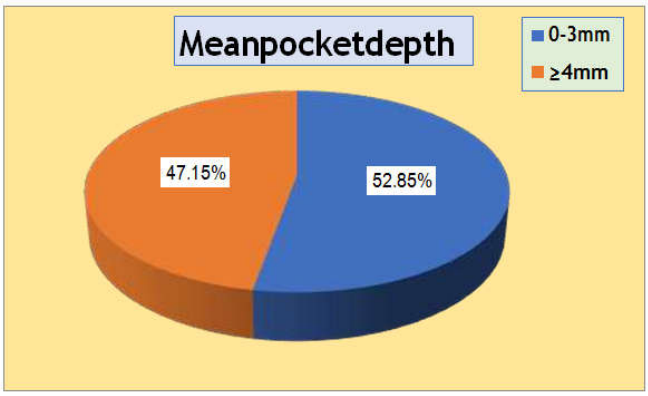
	Number (n)	Percentage (%)	Chi-square test	P value, Significance
Normal	26	9.28%	35.81	p<0.001, highly significant difference
Affected by Periodontal disease	254	90.72%		
Total	280	100%		

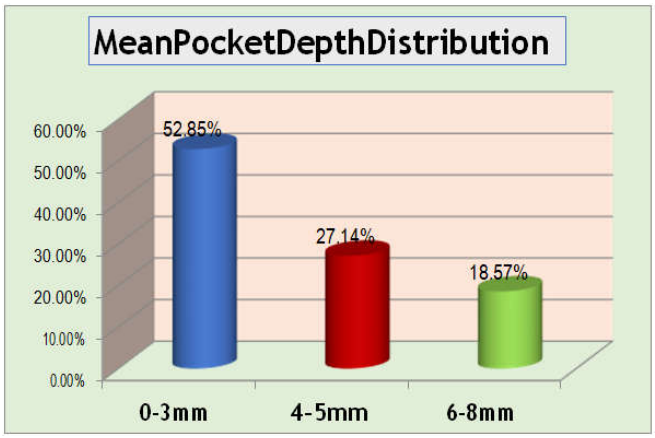


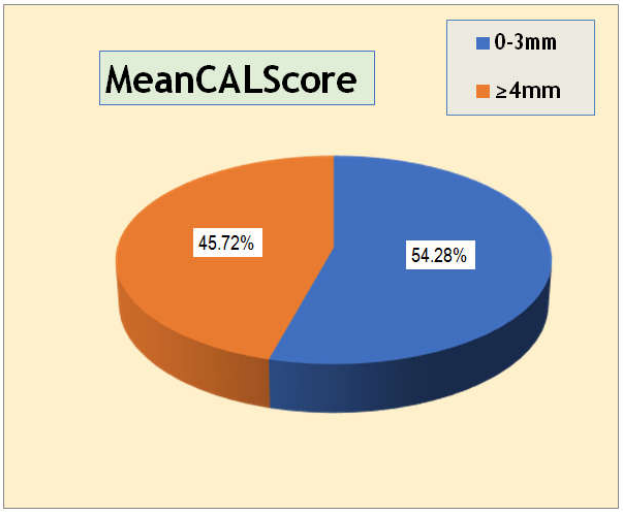












DISCUSSION

Patients with periodontal disease share many of the same risk factors aspatients with cardiovascular disease including age, gender (predominantly male),lower socio economic status,stress,andsmoking.16 Additionally, alarge proportion of patients with periodontal disease also exhibit cardiovascular disease.17 These observation ssuggest that periodontal disease and athero sclerosis share similar or common etiological pathways. In 2003, Scannapieco *et al*. conducted a systematic review of the evidence supporting or refusing any relationship.18

The purpose of this study was to examine the possible associations between individual clinical parameters of periodontitis (BOP, PD, and CAL)and system ic disease CHD patients with periodontitis. The results indicate that BOP, the clinical parameter that provides a measure of periodontal tissue inflammation. (Greenstein *et al*. 1981, Engelberger *et al*. 1983). It is strongly associated with CRP, a systemic inflammatory biomarker and CHD risk factor (Wang *et al*. 2002). A strong association between periodontitis and CHD has been established from epidemiological studies (Humphrey *et al*.2008, Dietrich *et al*.2013, Tonetti & Van Dyke 2013).This association is indirectly supported by intervention trials that Examined the effects of periodontal therapy on CHD risk markers (D'Aiuto *et al*.2004, 2006, Seinost *et al*. 2005,Hussain Bokhari *et al*.2009,Of fenbacher *et al*.2009,Bokhari et al.2012.

In periodontitis patients, sites with BOP are histologically characterized by a three fold increase in the volumetric density of inflammatory cells (lymphocytes, macrophages and monocytes), compared to sites without BOP (Cooperetal.1983). The significant association between BOP, a clinical marker of localized tissue inflammation, and a marker of systemic inflammation (CRP)is consistent with the recently reported strong association between periodontal tissue metabolic activities, a possible surrogate for periodontal inflammation, and histologically assessed atheroscle roticplaque inflammation in patients who underwent carotid endarterectomy (Fifer et al.2011). The results of this study have potential limplications for future studies (inclusion criteria, periodontal therapy endpoints) investigating the association between periodontal and cardiovascular disease.

In the present study, there is a strong prevalence of periodontitis and cardiovascular disease. In most cross-sectional and longitudinal studies indicate that periodontitis and cardiovascular diseases are positively associated. Clinical trials have shown that periodontal treatment can positively modulate surrogate markers of cardiovascular disease. In 2018, a systematic review documented that periodontal treatment results in significant reductions in serum C-reactive protein and leukocyte counts (Roca-Millan *et al*. 2018). However, no definitive randomized controlled trials have been conducted examining the effects of periodontal interventions on incident cardiovascular disease. Such studies would need to be long term, may involve ethical issues regarding the appropriate treatment allocated to the control arm, and would be invariably quite costly.

Three main path way slinking oral infection to secondary disease were suggested (Thoden Van Velzen*et al*., 1984): (1) metastatic infection, secondary to the oral infection, due to transient bacteremia (presumably resulting primarily in endocarditis) ; (2) systemic inflammation from immunologic injury caused by oral bacteria; and (3) system icvascular injury due to oral microbial endotoxins. Bacterial endotoxin may affect endothelial integrity, plasma lipoprotein metabolism, blood coagulation, platelet function, and prostaglandin synthesis (Syrjanen, 1990). Bacteria lendotoxin increases cytokine secretion (Offenbacher *et al*.,1993; Shapira *et al*.,1994), which could elevate inflammatory markers (Feyand Fuller, 1987; Fahmy and Young,1993; Rogers *et al*.,1994). Participants with periodontal disease showed elevated fibrinogen and white blood cell levels (Kweider *et al*.,1993), which may be causally related to CHD (Friedman *et al*.,1974; Kannel *et al*., 1987; Yarnell *et al*.,1991; Ensrud and Grimm,1992; Ernst, 1993;

To flerand Jadhav, 1996). There is also some evidence linking dental infection with Von Willebr and factor (Mattila *et al*., 1989b), which in turn has been associated with CHD (Thompson et al.,1995).

It is now clear from the epidemiologic studies that a potential link exists between Periodontal disease and CVD oral healthcare. Professional scan identify patients who are unaware of the ir risk of developing serious complications as a result of CVD and who are in need of CVD and those who need medical intervention. Prospective interventional studies are required to determine the exact link between PD and CVD as well as to evaluate whether periodontal treatment may reduce the risk of developing CVD. However, the challenge remains whether periodontal disease can be considered one

among the traditional risk factor for CVD as the link established from different studies is not limited to a recent CVD. PD Seems to be associated with no more than a modest increase (−20%) in cardiovascular risk in general population. As the ongoing studies report and confirm the strength of the association between periodontal disease and CVD overtime, the oral health-care professionals and medical professionals have to prepare for better planning of prevention programs. It seems from the scientific evidence gathered so far tha interventional care remains invaluable not only for oral health but also general health aswell.

In the present study, no histological analysis was made nor any association were determined between the risk factors of CVD and of periodontitis. Future studies are needed to determine these ascepts.

CONCLUSION

There is a strong prevalence, periodontal infections are epidemiologically associated with CVD; that is, periodontal infections seem to be found more frequently in patients with CVD.

However, thecriticalquestionofwhetherperiodontalinfectionsareariskfactorfororcontributecausallyto CVD and cerebro vascular disease remains unanswered.

Future well-designed prospective cohort studies with uniform definitions of periodontal disease and CHD in vestigating the definite role of periodontal pathogen burden on the occurrence of CHD and management of patients with PD to reduce the future risk of development of CHD are necessary.

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