How to Cite:
Sultana, F., Mishra, A., Kumar, M. M., Sahithi, A., Reddy, K. K., & Ramya, Y. (2022). Efficiency of periodontal therapy for management of CVS diseases in patient with chronic periodontitis: An original research. *International Journal of Health Sciences*, *6*(S3), 7368–7374. https://doi.org/10.53730/ijhs.v6nS3.7679

Efficiency of periodontal therapy for management of CVS diseases in patient with chronic periodontitis: An original research

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***Abstract*---**To evaluate the effectiveness of periodontal prophylaxis/ treatment in patients with chronic periodontitis already suffering from cardiovascular system (CVS) diseases. 500 Subjects with periodontal diseases (PD) were grouped according to treatment (dental prophylaxis, intensive treatment, and PD without treatment). The Incidence Rates (IRs) of CVS diseases during the 1-year follow-up period were compared among groups. Cox regression analysis adjusted for age, sex, socioeconomic status, residential urbanicity, and comorbidities was used to evaluate the effect of PD treatment on

the incidence of CVS diseases. The IR of CVS diseases among subjects without PD was 0.19%/year. Among those with PD, the IR of CVS diseases was lowest in the dental prophylaxis group (0.11%/year), followed by the intensive treatment (0.28%/year) and PD without treatment (0.31%/year; P<0.001) groups. Cox regression showed that the hazard ratio (HR) for CVS diseases was significantly lower in the dental prophylaxis group. PD is associated with a higher risk of CVS diseases, which can be reduced by dental prophylaxis to maintain periodontal health.

***Keywords*---**periodontal disease, cardiovascular diseases, dental prophylaxis, dental scaling.

Introduction

Periodontitis is one of the most ubiquitous diseases and is characterized by the destruction of connective tissue and dental bone support following an inflammatory host response secondary to infection by periodontal bacteria [1, 2]. Severe periodontitis, which may result in tooth loss, is found in 5–20% of most adult populations worldwide [3–5]. Children and adolescents can have any of the several forms of periodontitis such as aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases [6–8]. It is now generally agreed that almost all forms of periodontal disease occur as a result of mixed microbial infections within which specific groups of pathogenic bacteria coexist [9–11]. Evidence is reviewed on the potential roles of modifiable and nonmodifiable risk factors associated with periodontal disease. An understanding of risk factors is essential for clinical practice. The biological plausibility of the association between periodontal diseases and cardiovascular diseases are well studied and it includes some of the following possible mechanisms: high concentrations of cholesterol and the action of oral bacteria in the process of atherosclerosis or the participation of acute-phase proteins that may increase in chronic periodontitis [10,11]. Several biological mechanisms have been proposed to explain the relationship between periodontal diseases and cardiovascular diseases.

Therefore, periodontitis can probably elicit a systemic inflammatory response and it deserves more attention [12]. Periodontal disease is capable of predisposing to vascular disease due to the rich source of subgingival microbial species and host's response. Furthermore, we must be aware that these diseases share many risk factors and there are evident similarities to the basic pathogenic mechanisms [13]. Periodontitis is associated with the increase in the level of C-reactive protein and fibrinogen, irrespective of coronary diseases. Furthermore, there is evidence that suggests that the increase in the levels of systemic markers of inflammation, such as the C-reactive protein (CRP) and interleukin-6 (IL- 6), is associated with cardiovascular diseases [14]. Bacteremia from periodontitis and dental disease is known to be the primary cause of infective endocarditis [15]. In particular, patients who have undergone heart valve surgery have a significant risk of life-threatening infective endocarditis. Epidemiological and microbiological studies have lent credence to the concept that periodontal disease may be a separate risk

factor for cardiovascular disease, cerebrovascular disease [16], and preterm delivery of low birth weight infants [17]. Wu et al. [18] have shown that periodontal disease is another putative and independent risk factor for cerebrovascular disease, particularly for ischemic stroke. Some studies have found no relationship between periodontitis and ischemic heart disease [19,20] Several studies report positive associations between periodontitis and heart failure. There is evidence from a large Asian study using the Taiwanese National Health Insurance Research Database reporting a significantly higher incidence of atrial fibrillation in individuals with periodontal diseases compared to individuals without periodontal diseases (hazard ratio—HR = 1.31, 95% CI [1.25, 1.36]).[21] Hence, it is imperative to investigate the association between periodontal disease (PD) and cardiovascular diseases, and evaluate the effect of dental prophylaxis on the incidence rate (IR) of CVS diseases.

Aim of the study

To evaluate the effectiveness of periodontal prophylaxis/ treatment in patients with chronic periodontitis already suffering from cardiovascular system (CVS) diseases.

Methodology

A retrospective cohort study was conducted amongst 500 adults. Only subjects aged above 20 years were included in the present study. The study population was divided into 3 groups. Patients undergoing - Dental prophylaxis, Intensive treatment (subgingival curettage and root planning), Without periodontal treatment. Patients were studied for a period of 1 year. Patients who developed CVS diseases before PD diagnosis were excluded Statistical analyses were performed using the SAS® statistical package (version 25). The IRs of CVS diseases among patients with PD and control subjects were compared. The x2 test was used for parametric categorical data. A P-value of less than 0.05 was considered significant. A Cox proportional hazards model was used to calculate hazard ratios (HRs) and 95% confidence intervals (CIs) to determine whether PD is a risk factor for the development of CVS diseases.

Results

The IR of CVS diseases, increased with age from 0.03%/year among subjects aged 20–44 years to 0.24% and 0. 61%/year among those aged 45–64 years and >65 years, respectively (P<0.001). The IR of AMI was higher among men than women (0.21% versus 0.13%/year) and was significantly higher among subjects with atrial fibrillation (0.88%/year), diabetes mellitus (0.55%/year), hypertension (0.49%/year), dyslipidemia (0.37%/year), chronic kidney disease (0.87%/year), and peripheral vascular disease (0.52%/year) than people without these comorbidities (all P<0.001). Among those with PD, the IR of CVS diseases, was lowest in the dental prophylaxis group (0.11%/year) and highest in the PD without treatment group (0.31%/year; P<0.001). (Table 1).

Discussion

There is evidence through traces of DNA, RNA or antigens derived from oral bacterial species, mainly periodontal pathogens that have been identified in atherothrombotic tissues. Studies have attempted to correlate the presence of these bacteria in atherothrombotic tissues, with other sample sources (subgingival plaque, serum, etc.), in the same patients, and these suggest that in periodontitis patients there is a higher probability of a positive correlation. At least two studies have demonstrated viable P. gingivalis and A. actinomycetemcomitans in atherothrombotic tissue when culturing the atheroma samples.[22] In vivo and in vitro studies demonstrate the importance of the fimbriae of P. gingivalis to host cell entry and to promote atherothrombotic lesions in experimental models. In vitro experiments have shown that certain bacterial strains expressing P. gingivalis hemagglutinin A (HagA) have an increased capability to adhere and enter human coronary artery endothelial cells (HCAEC). There is evidence that HSPs (Heat shock proteins) from periodontal pathogens (Porphyromonas gingivalis, Tannerella forsythia, Aggregatibacter actinomycetemcomitans and Fusobacterium nucleatum) generate antibodies that can cross-react with human HSPs. These antibodies have been shown to activate cytokine production, as well as monocyte and endothelial cell activation.

The presence of anti-cardiolipin antibodies has been significantly associated with periodontitis patients, which reversed following periodontal therapy. There is some evidence that periodontal pathogens can elicit antibodies that cross-react with cardiolipin.[23] Cross-sectional data of The Scottish Health Surveys from 1995 to 2003 pertaining 11,869 men and women (mean age of 50 years) were linked to a database of hospital admissions and deaths with follow-up until December 2007 (Information Services Division, Edinburgh). Participants who brushed less than once a day exhibited the highest incidence of ACVD (Acute cardiovascular disease) events (HR = 1.7, 95% CI [1.3; 2.3]) compared with those who brushed twice a day, indicating that self-performed oral hygiene routines may reduce the incidence of ACVD.[24] The findings support the concept that inflammatory diseases such as PD may play a role in the pathogenesis of AMI (Acute myocardial infarction). The association of PD with the prevalence of CHD (Congestive heart disease) suggests that inflammation may be a potential mechanism involved in the regulation of the atherosclerotic process.[25,26] Treatment of PD resulting in a beneficial effect on vascular diseases such as AMI and stroke was also reported. A 7-year cohort study by Chen et al showed that frequent tooth scaling reduced the IR and risk of AMI among subjects aged >50 years.[27] In a previous study, it was found that dental prophylaxis reduced the risk of ischemic stroke in subjects with PD, especially those aged 20–44 years.[28] PD has also been demonstrated to contribute to elevated C-reactive protein levels in nondiabetic, non-smoking patients who have experienced AMI. The current study demonstrated that PD is correlated with the risk of AMI, and that dental prophylaxis can significantly reduce the IR of AMI.

Conclusion

Patients who receive regular prophylactic dental treatment are more likely to have healthier periodontal conditions and less likely to have systemic chronic inflammatory reactions, resulting in a lower IR of CVS diseases.

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Tables

Table 1
Univariate analysis of factors affecting the incidence of cardiovascular diseases

Variables	IR (Incidence Rate) (%/year)	x2 test (*P*-value)
Age at baseline		<0.001
20–44 years	0.03	
45–64 years	0.24	
>65 years	0.61	
Gender		<0.001
Female	0.13	
Male	0.21	
Periodontal disease (PD)		<0.001
No PD	0.19	
Dental prophylaxis	0.11	
Intensive treatment	0.28	
PD without treatment	0.31	