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Review Article

Periodontitis and systemic diseases: A systemic review

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ABSTRACT:
Periodontal disease (PD) is among the most common infectious diseases affecting humans. While the burden of periodontal disease on oral health has been extensively investigated, a possible specific relationship between the disease and systemic health is a relatively new area of interest. More recently it has been suggested that PD has an etiological role in the development of atherosclerotic cardiovascular disease, diabetes mellitus, and preterm low-birth weight, among others. It is critical that dentists and physicians are well informed of the potential general health impact of periodontal disease so that they are in a position to knowledgeably counsel patients.
Key words: Periodontitis, Systemic diseases

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INTRODUCTION

A chronic oral infection such as periodontitis is a constant potential source of infection and has been considered as a separate risk factor for cardiovascular diseases, cerebrovascular diseases, peripheral arterial disease, respiratory diseases, and low birth weight. In addition, periodontitis has been described as a potential risk for increased morbidity and mortality for diabetes, insulin resistance, rheumatoid arthritis, obesity, osteoporosis, and complications of pregnancy. In fact, a case of pyogenic liver abscess caused by periodontal bacteria had been reported.[1-3]
Some of these conditions may in turn increase the incidence and severity of periodontal disease by modifying the body's immune response to periodontal bacteria and their by-products. Evidence suggests a bi-directional relationship between periodontitis and systemic diseases. The possible mechanisms or pathways linking oral infections to secondary systemic effect are: metastatic spread of infection from the oral cavity as a result of transient bacteremia, metastatic injury from the effects of circulating oral microbial toxins, and metastatic inflammation caused by immunological injury induced by oral microorganisms.[4, 5] There is tendency for medical and dental specialists to see patient management from regional rather than systemic point of view. In the light of the ever increasing available facts on the role of oral infections like periodontitis on multifarious systemic disorders, it has become necessary to undertake a literature review on the subject.[5]

PERIODONTITIS AND RESPIRATORY DISEASES

As early as 1968, previous authors described the presence of dental diseases in subjects with pulmonary diseases. Oral bacteria can enter the lower respiratory tract by aspiration and cause pneumonia. Severe infections of the lungs can develop after the aspiration of the salivary secretion, especially in patients with periodontitis. 30-40% of aspiration pneumonia and predominantly necrotizing pneumonia or lung abscesses have anaerobes in their aetiology, the most frequent organisms being Proteus gingivalis (PG), Bacteroidesoralis, Eikenellacorrodens, Fuso bacterium nucleatum, Aggregatibacteractinomycetemcomitans (AA), Peptostreptococcus and Clostridium. It is possible that even Streptococcus viridans plays a role in the development and/or progression of pneumonia.[6-8]

Bacteria may have an influence in the exacerbations of Chronic Obstructive Pulmonary Disease (COPD), where the dental plaque may serve as a reservoir of respiratory pathogens. There are a number of possible mechanisms by which bacteria can influence the pathogenesis of respiratory diseases:[6- 8]

- Aspiration of oral pathogens (PG or AA, for example).
- Alteration of the mucous surface by salivary enzymes in periodontitis, leading to an increase in the adhesion and colonization of respiratory pathogens.
- The periodontal disease-associated enzymes may destroy the salivary pellicles on the pathogenic bacteria.[6- 8]

PERIODONTITIS AND DIABETES

A recent prospective study investigated the influence of glycaemic control on the progression of periodontitis during periodontal maintenance therapy (PMT). Type 2 diabetes mellitus (T2DM) control was monitored by HbA1c percentage over a 5-year period. Participants matched for sex and smoking were divided into three groups: 23 with diabetes and poor glycaemic control (PGC), 23 with diabetes and good glycaemic control (GGC), and 46 controls with no diabetes (NDC). Progression of periodontitis was defined as an increase of interproximal clinical attachment loss of ≥3 mm in at least two teeth. Progression of periodontitis was significantly higher in the PGC compared to GGC and NDC participants. Multiple logistic regressions showed that for progression of periodontitis, a HbA1c ≥6.5% had an odds ratio (OR) of 2.9 (95% CI 1.43–9.81). A limitation was the small group sizes which prevented stratification for the effect of smoking. There was adjustment for smoking in the analysis; however, it was not clear if there was an interaction between smoking and glycaemic control. Despite this, and the restricted population that the subjects were recruited from, the study did demonstrate that poor glycaemic control significantly predicted risk for the progression of periodontal destruction in those previously diagnosed with and treated for periodontitis.[9- 14]

A prospective study of 4033 subjects, aged 35–44, with no evidence of periodontitis, defined by a Community Periodontal Index (CPI) score <3, had their fasting plasma glucose (FPG) measured at baseline. The number of newly diagnosed periodontitis cases (CPI score ≥ 3) at re-examination after 5 years of follow-up was 1129 (28.0%) normal FPG, 96 (32.3%) pre-diabetes, and 22 (38.6%) with T2DM at baseline. Multivariable analysis using Cox's proportional hazard regression model showed an increased risk of incident periodontitis for subjects with pre-diabetes (hazard ratio (HR) = 1.25 (95% CI 1.00–1.57)) and T2DM (HR = 1.95 (95% CI 1.22–3.13)) after adjustment for all potential confounders. A novel finding of this study was the significant effect of pre-diabetes on incident periodontitis. A limitation of the study was the use of the CPI score to identify periodontitis. Further weaknesses were lack of information regarding the management of the hyperglycaemia and its possible effects.[9- 14]

Based on the available evidence to date, it seems likely that the level of metabolic control influences future periodontal disease risk. However, further longitudinal studies are warranted to assess what effect improved glycaemic control has on the progression of periodontitis. The evidence examining the influence of pre-diabetes as a condition that can potentially adversely affect periodontal condition is inconclusive and also requires further investigation as evidence can both be found for and against.[9- 14]

PERIODONTAL DISEASE AND CARDIOVASCULAR DISEASE (CVD)[15- 19]

Cardiovascular disease (CVD) is a common cause of death, accounting for 29% of deaths worldwide. Estimates from the year 2002 show that more than 70 million Americans were diagnosed with one of the forms of CVD, which include high blood pressure, coronary heart disease (myocardial infarction and angina pectoris), peripheral arterial disease, and stroke, with atherosclerosis as the principal cause of all CVDs. Atherosclerosis is thus responsible for 50% of all mortality in the United States, Europe, and Japan. After adjustment of other risk factors, studies indicate that severe periodontal disease is associated with a 25% to 90% increase in risk for CVD.22 One study showed that 91% of patients with CVD demonstrated moderate to severe periodontitis, while 66% of cardiologically healthy patients had periodontitis. The same study showed a statistically significant correlation between coronary artery disease and periodontitis.[15- 19]

Periodontal disease may be associated with CVD due to mutual risk factors for atherogenesis and periodontal disease. In order to consider periodontal disease as a risk factor for atherosclerosis and other CVDs, the presence of pathogens associated with periodontal infection should be localized in serum or atheromatous plaques. Investigating this by sampling carotid atheromatous plaques, Cairo et al.24 detected T. forsynthensis DNA in 79%, F. nucleatum in 63%, P. intermedia in 53%, P. gingivalis in 37%, and A. actinomycetemcomitans in 5% of the samples from carotid atheroma patients. In addition to carotid, coronary, and aortic atherosclerotic plaques, these various oral bacteria were also detected in occluded arteries from patients with Buerger Disease. One would expect that these pathogens would induce the release of proinflammatory cytokines. Etiologically, gentle mastication releases bacterial endotoxins from the oral cavity into the bloodstream, inducing cytokine production (TNF, IL-1, and PGE2). Further, animal studies should be able to demonstrate atherosclerosis induced by periodontal pathogens. Animal models provide a more thorough

understanding of the pathogenesis of CVD; specifically, with the use of gene-targeted animals such as the apolipoprotein E-knockout (apoE −/−) mouse.[15- 19]

Etiologically, the chronic presence of periodontal microbes can lead to atherogenesis via two pathways: (1) direct invasion of the arterial wall23 and (2) the release, in response to infection, of systemic inflammatory mediators with atherogenic effects.30 These pathogens, especially P. gingivalis, have demonstrated the ability to interact with the endothelial surface and to induce smooth-cell proliferation, causing damage and impairing the vasomotor functionality of the endothelial cells.2,26,31,32 Serum C-reactive protein (CRP) plays a role in endothelial dysfunction, and elevated levels of CRP provide insight into the linking of periodontal disease and CVD.2,33–36 In patients with periodontal disease who have elevated plasma levels of both fibrinogen37 and TNF-alpha, there is an association with increased carotid intima-media thickness (IMT).38 IMT and left ventricular mass (LVM) are alternative, yet valuable tools in measuring carotid atherosclerosis.5,19,22,39 However, our understanding of the mechanism linking these inflammatory markers with atherosclerosis progression is unclear.[20- 25]

Recent studies have shown that CRP may directly interfere with endothelial nitric oxide (NO) availability, by both decreasing the expression of NO synthase and simultaneously increasing the production of reactive oxygen, which inactivates NO. Elevated CRP serum levels are the signal feature of the transition from stable coronary artery disease to the formation of a platelet-rich thrombus following plaque rupture or erosion.4 These findings shed light on the fact that endothelial activity, associated with elevated CRP serum levels, is characterized by the impaired systemic bioavailability of NO in coronary artery disease patients. Further investigation of this hypothesis (i.e., the rote of CRP on NO) has led to the discovery that CRP serum levels are important in predicting the availability of NO in the systemic circulation in coronary artery disease patients.[23- 25]

CONCLUSION

Although periodontal disease is the most prevalent infectious oral condition but is treatable and preventable. The reduction in the incidence and prevalence of periodontal disease can result in lowering its associated systemic diseases and complications. Decreased periodontal disease burden can minimize treatment needs and can reduce financial impact on health-care systems. High prevalence of periodontal disease also necessitates the establishment of surveillance system for oral diseases in the community. Preventive programs for periodontal disease should utilize common risk approaches to reduce the magnitude of other chronic diseases. Cost-effective strategies would also enhance interdisciplinary collaborations among health-care providers. Health-care providers should be familiar with perio-systemic link and should be able to diagnose and refer the patients to specialized dental or periodontal care to improve the quality of life of their patients.

REFERENCES

1. Tezal M, Grossi SG, Genco RJ. Is periodontitis associated with oral neoplasms? J Periodontol. 2006;77:1465–82.
2. Rosenquist K. Risk factors in oral and oropharyngeal squamous cell carcinoma: A population-based case-control study in southern Sweden. Swed Dent J. 2005;179:1–66.
3. Michaud DS, Liu Y, Meyer M, Giovannucci E, Joshipura K. Periodontal disease, tooth loss, and cancer risk in male health professionals: A prospective cohort study. Lancet Oncol. 2008;9:550–08.
4. Meyer MS, Joshipura K, Giovannuci E, Michandi DS. Periodontal disease and cancer. Cancer causes control. 2008;19:895–07.
5. Fitzpatrick SG, Katz J. The association between periodontal disease and cancer: A review of the literature. J Dent. 2010;38:83–5.
6. Tezal M, Sullivian MA, Hyland A, Marshall JR, Stoler D, Reid MA, et al. Chronic periodontitis and the incidence of head and neck squamous cell carcinoma. Cancer Epidemiol Biomarkers Prev. 2009;18:2409–12.
7. Yu-Kang T, Gilthorpe MS. Commentary: Is tooth loss good or bad for general health? Int J Epidemiol. 2005;34:475–6.
8. Jürgensen N, Petersen PE. Oral health and the impact of socio-behavioural factors in a cross sectional survey of 12-year old school children in Laos. BMC Oral Health. 2009;9:29.
9. Morita I, Nakagaki H, Kato K, Murakami T, Tsuboi S, Hayashizaki J, et al. Relationship between number of natural teeth in older Japanese people and health related functioning. J Oral Rehabil. 2007;34:428–32.
10. Abnet CC, Qiao YL, Dawsey SM, Dong ZW, Taylor PR, Mark SD. Tooth loss is associated with increased risk of total death and death from upper gastrointestinal cancer, heart disease, and stroke in a Chinese population based cohort. Int J Epidemiol. 2005;34:467–74.
11. Nakanishi N, Fukuda H, Takatorige T, Tatara K. Relationship between self-assessed masticatory disability and 9-year mortality in a cohort of community-residing elderly people. J Am Geriatr Soc. 2005;53:54–8.
12. Touger-Decker R, van Loveren C. Sugars and dental caries. Am J ClinNutr. 2003;78:881–92.
13. Bimstein E, Katz J. Obesity in children: A challenge that pediatric dentistry should not ignore-review of literature. J ClinPediatr Dent. 2009;34:103–6.
14. Scannapieco FA, Bush RB, Paju S. Periodontal disease as a risk factor for adverse pregnancy outcomes: A systematic review. Ann Periodontol. 2003;8(1):70–78.
15. Scannapieco FA, Bush RB, Paju S. Associations between periodontal disease and risk for atherosclerosis: A systematic review. Ann Periodontol. 2003;8(1):38.

16. Khader YS, Albashaireh ZS, Alomari MA. Periodontal diseases and the risk of coronary heart and cerebrovascular diseases: a meta-analysis. J Periodontol. 2004;75(8):1046–53.
17. Seymour RA, Preshaw PM, et al. Cardiovascular disease and periodontology. J ClinPeriodontol. 2003;30(4):279–92.
18. Hujoel PP. Does chronic periodontitis cause coronary heart disease? A review of the literature. JADA. 2002;133(supplement):31S.
19. Council on Cardiovascular Disease in the Young, Oral health and cardiovascular disease. American Heart Association. Available at:“ www.americanheart.org/presenter.jhtml?identifier=30 16026.” Accessed Nov. 4, 2004.
20. Bosnjak A, et al. Advances in the Relationship between Periodontitis and Systemic Diseases. ActaStomatol Croat. 2001;35:br.
21. Gilbert GH, Miller MK, Duncan RP, Ringelberg ML, Dolan TA, Foerster U. Tooth-specific and person-level predictors of 24-month tooth loss among older adults. Community Dent Oral Epidemiol. 1999;27:372–85.
22. Chi H, Messas E, Levine RA, Graves DT, Amar S. Interleukin-1 receptor signaling mediates atherosclerosis associated with bacterial exposure and/or a high-fat diet in a murine apolipoprotein E heterozygote model: pharmacotherapeutic implications. Circulation. 2004;110:1678–85.
23. Li L, Messas E, Batista EL, Jr, et al. Porphyromonasgingivalis infection accelerates the progression of atherosclerosis in a heterozygous apolipoprotein E-deficient murine model. Circulation. 2002;105:861–7.
24. Skyler J, Oddo C. Diabetes trends in the USA. DiabetesMetab Res Rev. 2002;18:S21–6.
25. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995–2025: prevalence, numerical estimates, and projections. Diabetes Care. 1998;21:1414–31.