Guest Editorial

Periodontal and Cardiovascular Diseases: An Intriguing Association

Dental and periodontal diseases are common in our population due to lack of proper emphasis on dental hygiene and high incidence of smoking (cigarettes, hukka, bidi) and tobacco chewing. Doctors should be concerned about dental hygiene not only as a cause of caries, tooth loss and periodontal diseases but also because of its association with metabolic syndrome. Increased prevalence of the metabolic syndrome has been reported after adjusting for traditional risk factors, as high as 2.3 times higher among patients with severe periodontal disease [1]. Higher prevalence of metabolic syndrome has been linked to increasing severity of periodontal disease, with a 12% increase in the risk for metabolic syndrome per 10% increase in the measurement of gingival bleeding. Meta-analysis of trials investigating the link between periodontal disease and cardiovascular disease (CVD) found increased relative risk of CVD 1.19-1.24 times in individuals with periodontal disease [2,3]. This link was more intensive for individuals <65 yrs of age (RR=1.44). Periodontal disease has been independently linked to increased incidence of fatal cardiac events and more strongly to the occurrence of stroke. Edentulism also increased the risk for coronary heart disease (CHD) [3]. Even poor oral hygiene without periodontal disease, defined by the degree of dental debris and calculus, also has been found to increase the risk for CHD suggesting a continuum of risk [4].

Atherosclerotic vascular disease has long been understood to be a result of deranged lipid homeostasis with vessel wall deposition, but lately role of chronic persistent inflammation has gained prominence in its progression and also in precipitating cardiovascular events by producing rupture of thin fibrous plaque cap. Hence, there is search for factors which could possibly aggravate this intravascular inflammatory condition.

Chronic localized inflammation of the dental sockets in periodontitis could possibly spill over into the systemic circulation and add to the existing cardiovascular risk, as has been postulated in the paper of Tandon S, et al published in this issue [5]. Periodontal disease has been shown to produce an increase in the degree of intravascular inflammation. It has been associated with increases in the levels of inflammatory markers, including an increase in serum C-reactive protein (CRP) levels of over 40% and an increase in plasma fibrinogen levels [6]. However, literature is wanting on a strong association between periodontal disease and serum lipid values.

Literature review shows that definition of periodontitis is heterogeneous in most of the studies. Results of studies investigating a casual role have been equivocal, however results of meta-analysis do show a increased risk of CVD with periodontitis. Both periodontitis and cardiovascular disease have high prevalence in the population and that produces a confounding effect. Moreover both share common risk factors like smoking, increasing age, genetics and stress which could be the reason for their coexistence [7]. However, studies conclude that a positive relationship exists even after accounting for confounding risk factors [3].

Postulations suggest that periodontitis and CVD may share a common pathophysiological mechanism or putative physiological defect. Aryl hydrocarbons present in cigarette smoke could inhibit bone formation especially in the presence of bacterial cofactors as in periodontal disease, promoting periodontal bone loss [8]. The same aryl hydrocarbons have been implicated in promoting vascular disease as measured by vascular calcification [9]. Similarly, activation of matrix metalloproteases has been implicated in periodontal tissue breakdown and also destabilization of atheromatous plaque and even development of heart failure [10,11].

Henceforth, coexistence of periodontitis and increased atherosclerotic risk might signify affliction of two different organ systems from a common pathological process. As of now evidence is insufficient to signify a causal role of periodontitis in progression of atherosclerosis especially in the backdrop of failure of antibiotic therapy to reduce cardiovascular events. Hence, antibiotic therapy as a mode of secondary prevention for CVD is not recommended but physicians should certainly lay emphasis to dental health hygiene and prevention of periodontitis as a primary preventive measure. This has been adequately advocated in the present article of Tandon S, et al [5].

References

1. D'Aiuto F, Sabbah W, Netuveli G, et al. Association of the metabolic syndrome with severe periodontitis in a large U.S. population-based survey. J Clin Endoncrinol Metab. 2008;93:3989-3994.
2. Janket SJ, Baird AE, Chuang SK, Jones JA. Meta-analysis of periodontal disease and risk of coronary heart disease. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003;95 (5):559-569. Abstract
3. Humphrey LL, Fu R, Buckley DI, Freeman M, Helfand M. Periodontal disease and coronary heart disease incidence: a systematic review and meta-analysis. J Gen Intern Med. 2008;23(12):2079-2086
4. DeStefano F, Anda RF, Kahn HS, Williamson DF, Russell CM. Dental disease and risk of coronary heart disease and mortality. BMJ. 1993;306:688-691. Abstract
5. Tandon S, Dhingra MS, Lamba AK, Verma M, Munjal A, Faraz F. Effect of periodontal therapy on serum lipid levels. Indian Journal of Medical Specialities 2010; 1:8-12.
6. Wu T, Trevisan M, Genco RJ, Falkner KL, Dorn JP, Sempos CT. Examination of the relation between periodontal health status and cardiovascular risk factors: serum total and high density lipoprotein cholesterol, C-reactive protein, and plasma fibrinogen. Am J Epidemiol. 2000;151:273-282. Abstract
7. Hujoel PP, Drangsholt M, Spiekerman C, DeRouen TA. Periodontitis-systemic disease associations in the presence of smoking – causal or coincidental? Periodontol 2002: 30: 51-60.
8. Singh SU, Casper RF, Fritz PC et al, Inhibition of dioxin effects on bone formation in vitro by a newly described aryl hydrocarbon antagonist, resveratrol. J Endocrinol 2000: 167: 183-195
9. Usman, OA. The effects of aryl hydrocarbon on vascular calcification in the warfarin-vitamin K rat model. 2004 Masters Thesis, University of Toronto. World Health Organization. The world health report 1995: Bridging the gaps. Geneva: WHO, 1995: 1.
10. Lee HM, Ciancio SG, Tuter G, Ryan ME, Komaroff E, Golub LM. Subantimicrobial dose doxycycline efficacy as a matrix metalloproteinase inhibitor in chronic periodontitis patients is enhanced when combined with a non-steroidal anti-inflammatory drug. J Periodontol 2004: 75: 453-463.
11. Matsumura S, Iwanaga S, Mochizuki S, Okamoto H, Ogawa S, Okada Y. Targeted deletion or pharmacological inhibition of MMP-2 prevents cardiac rupture after myocardial infarction in mice. J Clin Invest 2005:115: 599-609.

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