JOURNAL OF CLINICAL AND DIAGNOSTIC RESEARCH

How to cite this article:
RAI B, KHARB S, ANAND SC. EFFECT OF TREATMENT OF PERIODONTITIS ON C-REACTIVE PROTEIN, TISSUE PLASMINOGEN ACTIVATOR AND HIGH-SERUM/LOW DENSITY LIPOPROTEIN IN CHOLESTEROL: A PILOT STUDY. Journal of Clinical and Diagnostic Research [serial online] 2008 April [cited: 2008 Apr 7]; 2:786-788.
Available from
http://www.jcdr.net/back_issues.asp?issn=0973-709x&year=2007&month=April&volume=2&issue=2&page=786-788&id=41

SHORT ARTICLE

Effect of Treatment of Periodontitis on C-reactive Protein, Tissue Plasminogen Activator and High-serum/Low Density Lipoprotein in Cholesterol: A Pilot Study

RAI B, KHARB S, ANAND SC

ABSTRACT

Background: Periodontitis is associated with elevated levels of C-reactive protein (CRP), tissue plasminogen activator (tPA) and low density lipoprotein cholesterol (LDL-C), recently postulated as coronary heart disease or cardiovascular disease risk factor.

Material and Methods: (CRP), (tPA) and (LDL-C) levels were measured in 12 patients (7M:5F) with periodontitis, before and after treatment (scaling and root planning).

Results: Periodontal treatment significantly decreased CRP, t-PA and LDL-C levels in these individuals, and it may thus decrease their CHD risk.

Key words: Periodontitis, Biomarker, Scaling, Root Planning.

Dr. Balwant Rai , Village – Bhangu, Distt. Sirsa, Post Office – Sahuwala I. India. E-mail: drbalwantraissct@rediffmail.com
Mobile No : 091-9812185855

Introduction

Periodontitis is defined as a group of conditions that cause inflammation and destruction of the attachment apparatus of teeth. Epidemiological associations between periodontitis and cardiovascular disease have been reported.[1,2] This could directly be due to the direct action of periodontal pathogens or their products on endothelial cells via transient bacteraemia, or indirectly due to products of the inflammatory response.[3-5] Periodontitis and atherosclerosis have complex aetiologies and genetic and gender predispositions, and may share pathogenic mechanisms as well as common risk factors. Also, increased levels of serum LDL-C, and chronic inflammatory markers CRP and t-PA (a parameter of endothelial function) have been related to increased cardiovascular risk.[6-8] Several short term inventory studies have reported that treatment of periodontitis reduces the serum concentrations of inflammatory markers.[9]

Materials and Methods

Twelve M:F 7:5 [Table/Fig 1](in age group of 31-40 years), having at least a minimum of seven sites exhibiting 6 mm loss as clinical attachment and the number of teeth in the dentition of at least 20, were studied. Patients were excluded from the study if they were alcoholic or chronic smokers, since they are a predisposing factor for periodontitis. Informed consent was obtained from all the included subjects. None of the patients selected, had any history of chronic inflammatory disease, diabetes, hypertension, or use of steroids or drugs. In all these cases, the peripheral blood was drawn before starting any treatment, one week later after scaling and root planning; and 3 weeks after scaling and root planning for investigation i.e. CRP, LDL-C and t-PA antigen. Plasma was obtained after centrifugation at 1500 g for 10 min, and was stored at -4^0C until analysis. CRP was analysed by immuno assay (using Mentura, CA, USA). t-PA concentrations were determined by ELISA (Imulgse tPA, Biopool International), and LDL-C was estimated enzymatically. The data was analyzed by SPSS 7.0.

Results

The purpose

CRP, t-PA and LDL-C levels were elevated in periodontitis. i.e. after treatment, patients fell significantly from T1 to T3 and from T2 to T3 [Table/Fig 2, $p<0.01$]

Table/Fig I: Characteristics of the participants in the study

Age	35 ± 6.2 years
Number of completed case	12
Gender	7 Male, 5 Female
Average number of teeth	21 ± 3.2
Average number of periodontal involved side	9.0 ± 1.3
Average probing pocket depth (William probe)	7.5 ± 1.2

Table/Fig 2: Effect of scaling and root planning on CRP, t-PA and CRP (pretreatment (T_1), one week later scaling and root planning (T_2), two week later scaling and root planning (T_3) means ± SD

Parameters	T_1(pre-treatment)	T_2 (one week after scaling and root planning)	T_3 (two weak after scaling and root planning)
CRP (mg/l)	2.8 ± 0.3 (1.3-5.2)	2.3 ± 0.2 (1.1-4.7)	1.9 ±0.3 (0.6-4.2)
t-PA (mg/dl)	13.9± 1.3 (7.2-22.6)	12.5± 1.2 (6.9-21.3)	11.9± 1.3 (5.3-17.5)
LDL-C (mg/dl)	187± 3.4 (158-281)	183± 3.7 (154-263)	172± 3.3 (142-259)

$P<0.01$. As compared to T_1

Discussion

Cumulative evidences support a casual association between periodontal infection and artherolerotic cardiovascular, or its sequelae[10]. Earlier evidences from interventional studies suggest that controls of periodontal infections may result in the improved levels of markers of systemic inflammations and measures of endothelial dysfunctions.[11] Plasma levels of CRP as an inflammatory marker have been accepted as systemic markers for risk of cardiovascular disease.[9] D'Aiuto et al reported reductions of CRP levels, six months after periodontal therapy.[12] The present study is an exploratory study which showed that high blood levels of CRP, t-PA and LDL-C are established indicators of risk for CVD, which reduced in patients with periodontitis after scaling and root planning. Our findings are consistent with those reported in literature.[13-15] Also, plasma t-PA levels was increased in periodontitis patients. Increased plasma t-PA level is a marker of endothelial dysfunction, and has been associated with increased risk of CHD.[8-9] Endothelial dysfunctions has been reported to be associated with increased risk for atherosclerosis in healthy patients, and have been considered to be valuable in the early stages, before manifestations or formation of atherosclerotic plaque. Following treatment, the tPA levels fell significantly, and plasma LDL cholesterol levels were higher in these patients and reduced following treatment. To the best of our knowledge, this is the first study reporting the effect of scaling and root planning in periodontitis, to decrease LDL-C, tPA and CRP levels.

Conclusions

Since the number of patients in this study was relatively small, these results need to be interpreted with caution. Nevertheless, these results are compatible with the view that only "susceptible" individuals react to periodontitis with increase t-PA, CRP and LDC-C, and that these individuals were not necessarily the ones with the most severe disease. Treating periodontitis by scaling and root planning lowered t-PA, CRP and LDL-C levels in these individuals, which could also possibly reduce their CHD risk. It may be concluded that biomarkers of inflammation ie. dyslipidaemia and endothelial dysfunction, rather than haemostatic factors, may be potential medicators of the periodontal cardiovascular disease relationship. The extent to which the control of periodontal infections results in lower incidence of CHD, needs to be addressed in future studies.

Acknowledgement:

We are thankful to Jain Diagnostic Centre, New Delhi for Sponsorship and caring out the lab investigations and Dr.Jasdeep Kaur, Resident, BJS dental College, LDH, 162 Kapurthala (member of our group) for helping during study.

References

[1] Rai B, Anand SC, Kharb S. Panoramic radiograph as a detective of cardiovascular risk. World J Med Sci 2006; (1) :92-93

[2] Slade GD, Ghezzi EM, Heiss GR. Relationship between periodontal disease and c-reactive protein among adults in atherosclerosis risk in community study. Arch Int. Med. 2003; 63: 1972-79.

[3] Choi JI, Chung SW, Kang HS, Rhim BY, Kim SJ, Kim SJ. Establishment of porphyromonas gingivalis heat-shock protein specific t-cell lines from atherosclerosis patients. J Dent Res 2002; 81: 344-48.

[4] Geerts So, Nys M, De MP, Charpentier J, Albert A, Legrand V. Systemic release of endotoxins induced by gentle mastication: association with periodontitis severity. J Periodontol 2002; 73: 73-78.

[5] Ridker PM, Brown NJ, Vaughan DE, Harrison DG, Mehta JL. Established and emerging plasma biomarkers in the prediction of first atherothrombotic events. Circulation 2004; 109 (25 Suppl I): IV 6-IV19.
[6] Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. Circulation 1998; 97: 1837-47.
[7] Thompson SG, Kienast J, Dyke SD, Haverkate F, Vande Loo JC. Hemostatic factors and the risk of myocardial infraction or sudden death in patients with angina pectoris. Enropean concerted action on thrombosis and disabilities angina pectoris study group. N Engl J Med. 1995; 332: 635-641.
[8] Ridker PM, Buring JE, Shih J, Matias M, Hennekens CH. Prospective study of c-reactive protein and risk of future cardiovascular events among apparently healthy women. Circulation 1998; 98: 731-733.
[9] Rai B, Anand SC. After scaling and root planning lower systemic inflammatory and thrombotic marker of cardiovascular risk. World J Med Sci 2006; 1(2).
[10] Beck JD, Offenbacker S. Systemic effect of periodontitis: epidemiology of periodontal disease and cardiovascular disease. J Periodontol 2005 (Sppl); 76: 2089-2100.
[11] Behle JH, Papapanou PN. Periodontal infections and atherosclerotic vascular disease: an update. International Dental Journal 2006; 56: 256-262.
[12] D'Aiuto F, Ready D, Tonettims S. Periodontal disease and c-reactive protein-associated cardiovascular risk. J Periodontal Res 2004; 39: 236-241.
[13] Taylor BA, Tafler GH, CARey HMR, Morel-Kopp Mc, Philcox S, Carter TR, Ellit MJ, Kall AD, Ward C, Skhenck K et al. Full mouth extraction lower systemic inflammatory and thrombotic markers of cardiovascular risk. J Dent Res 2006; 85 (1): 74-78.
[14] Joshipura KJ, Wand HC, Merchant AT, Rimm EB. Periodontal disease and biomarkers related to cardiovascular disease. J Dent Res 2004; 83 (2): 151-155.
[15] Lower GD, Vernell JW, Rumby A, Bainton D, Sweetram PM. C-reactive, fibrino-D-dimer and incident ischemic heart disease in the speedwell study are inflammation and fibrin turnover linked in pathogenesis? Artheroscles Thromb Vasc Biol 2001; 21: 403-410.