

Effect of Scaling and Root Planning in Periodontitis on Peripheral Blood

B Rai, S Kharb

Citation

B Rai, S Kharb. *Effect of Scaling and Root Planning in Periodontitis on Peripheral Blood*. The Internet Journal of Dental Science. 2007 Volume 6 Number 1.

Abstract

Numerous studies linking periodontitis and cardiovascular disease have been observed. We used a treatment intervention model to study the relationship between periodontitis and peripheral blood. We studied 37 subjects (M:F, 16:21, in age group 23-40 years) with severely periodontitis requiring scaling and root planning. Blood samples were obtained: (1) at initial presentation, (2) after 3 weeks of scaling and root planning (3) after 10 weeks of scaling and root planning. After the treatment, there is significant decrease in white blood cells, neutrophil, platelets, while increase hemoglobin and RBC levels. This research paper shows that elimination of periodontitis by scaling and root planning reduces systemic inflammatory markers of cardiovascular risk. It also supports that links between periodontitis and cardiovascular disease exist.

INTRODUCTION

Periodontal disease, a common chronic oral inflammatory disease, is characterized by destruction of soft tissue and bone surrounding the teeth. Epidemiological associations between periodontitis and cardiovascular disease have been reported.^{1,2} Periodontitis and atherosclerosis have complex etiologies, genetic and gender predispositions and may share pathogenic mechanisms as well as common risk factors. Several short term intervention studies have been reported that treatment of periodontitis reduces the serum concentrations of inflammatory markers, such as c-reactive protein, TNF- α , IL-6 which are thought to be initiating factor cardiovascular disease. Hence, the present study was planned, effect of scaling and root planning in periodontitis on peripheral blood.

MATERIAL AND METHODS

Thirty seven (M:F, 16:21, in age group 23-40 years) having at least a minimum of seven sites exhibiting, 6 mm loss of clinical attachment who had been referred to Deptt. of periodontology. The patient had periodontitis characterized by a horizontal loss of supporting tissue by more than 1/3rd of root length with bleeding on probing, furcation involvements of the multi-rooted teeth. In none of the participants was cardiovascular disease or any other ongoing general disease or infections diagnosed. Patients were excluded from the study if they had alcoholic or chronic smoker. In on all these cases, the peripheral blood were

drawn before starting any treatment; three weeks later scaling and root planning; and 10 weeks after scaling and root planning for investigation i.e. total white blood cells count, red blood cell counts, thrombocytes count and hemoglobin (Hb) level and fibrinogen. Plasma fibrinogen was determined according to Clauss method.³ All the statistical analysis were performed using SPSS Software package (version 11.0) and student t-test was applied.

RESULTS

Figure 1

Table 1: (Mean $\hat{A} \pm$ SD) effect of scaling and root planning on blood cell counts, pre-treatment (T₁), past treatment (T₂) (After 3 week of scaling and root planning) and 10 week after scaling and root planning (T₃) counts of total white blood cells, neutrophils, lymphocytes, platelets counts, red blood cells, hemoglobin, fibrinogen.

	Sex	T ₁	T ₂	T ₃
Neutrophils ($\times 10^9/L$)	M	4.8 (3.7-5.8)	4.1 (3.4-5.6)	3.0 (3.1-5.5) ^a
	F	4.7 (3.6-5.6)	4.5 (3.5-5.5)	3.1 (3.1-5.4) ^{ab}
White cell count ($\times 10^9/L$)	M	7.0 (6.1-9.3)	7.2 (6.2-9.6)	6.4 (5.9-8.7) ^a
	F	7.2 (5.9-9.2)	6.4 (5.7-9.3)	6.2 (5.6-9.2) ^b
Red blood cells ($\times 10^{12}/L$)	M	5.1 (4.7-5.7)	5.0 (4.6-5.3)	5.0 (4.6-5.3) ^c
	F	4.8 (4.6-5.1)	4.8 (4.6-5.1)	4.9 (4.7-5.2) ^d
Platelets ($\times 10^{12}/L$)	M	245 (204-300)	230 (207-302)	223 (197-294) ^e
	F	234 (205-301)	226 (206-301)	220 (196-288) ^f
Hemoglobin (g/L)	M	145 (132-164)	146 (133-167)	150 (137-168) ^g
	F	134 (132-162)	136 (133-163)	148 (137-167) ^h
Fibrinogen (g/L)	M	3.7 (2.8-4.8)	3.7 (2.8-4.9)	3.4 (2.7-4.5) ⁱ
	F	3.7 (2.7-4.9)	3.7 (2.9-4.9)	3.6 (2.7-4.4) ^j

*,** = statistically significant difference between T₁ and T₃ and between T₂ and T₃ (p<0.001, p<0.001) a,b = statistically significant difference between T₁ and T₃ and between T₂ and T₃ (p<0.001, p<0.001) c = statistically significant difference between T₁ and T₃ (p<0.01) d = statistically significant difference between T₁ and T₃ (p<0.01) e,f = statistically significant difference between T₁ and T₃ and between T₂ and T₃ (p<0.002, p<0.001) g,h = statistically significant difference between T₁ and T₃ and between T₂ and T₃ (p<0.05, p<0.005) i,j = statistically significant difference between T₂ and T₃ (p<0.001)

The neutrophils, lymphocytes, total WBC and platelets significantly reduced after treatment (Table I). Hemoglobin levels and red blood cells counts showed increase after treatment (Table 1)

DISCUSSION

The chronic infections, such as periodontitis are associated with increased risk for cardiovascular disease. Higher level of leukocyte count in periodontitis have been reported. ⁴ The present study, total leukocytes counts significantly decrease after treatment (Table I, p<0.01). Also, significant decrease in polymorphonuclear leukocytes were observed (Table I, p<0.01) after treatment. Moderately elevated numbers of

leukocytes have been associated with an increased risk for cardiovascular disease. ⁵ Also, since higher numbers of leukocytes increase the blood rheology more cells make blood more viscous, and more cell may adhere to endothelial cells lining the blood vessels, also decreasing blood flow. Reduced blood flow could play a role in relation to cardiovascular, especially in narrow or partly blocked artery due to atherosclerotic plaque formation. ⁶ Low hematocrit value have been reported in the periodontitis patients. Increase hemoglobin levels (Table I, p<0.05) observed in our study which supported early reported. ⁶ In the present study, RBC counts increased significantly in treated patients as compared to without treated patient (Table I, p<0.01). Anemia increases the risk of a cardiovascular event. It has been recently suggested that patients with periodontitis has lower hematocrit and hemoglobin levels after adjustment for confounder. ⁷

The platelets count were significantly decrease in post treated patients as compared to pretreated (Table I, p<0.01). This increase in thrombocyte count could be due to fact that they play an integral role in innate immunity against micro-organism. ^{8,9} Certain proteins from the periodontal pathogen porphyromonas gingivalis can stimulate thrombocytes to aggregate in a similar fashion as the clotting factor thrombin. ^{8,9,10,11,12} Inflammatory and infectious process are known to result in an increase in the number of active thrombocytes; i.e. reactive thrombocytosis. Therefore, increase in circulating thrombocytes could occur in periodontitis patients., p<0.001).

It has been observed that fibrinogen level significantly higher in periodontitis patient as compared to control. ⁴ In this study fibrinogen level decrease significantly after treatment i.e. support the above study. The increased levels of fibrinogen in periodontitis results in its binding to platelets, causing platelet aggregation and promotion of fibrin formation, thus contributing to plasma viscosity. Periodontitis and atherosclerosis may share pathogenic mechanisms and common risk factors. Also, chronic infections and inflammatory conditions such as periodontitis may influence the atherosclerotic process. It is supported by finding of elevated total WBC, thrombocyte and fibrinogen levels while decrease hemoglobin and RBC levels in the present study. Currently, American Heart Association is developing a summary on the inclusion of periodontal screening with previously established measured of risk assessment of cardiovascular disease. Thus, these systemic markers can prove to be useful as a tool for assessment of

cardiovascular risk in periodontal disease.

ACKNOWLEDGMENT

I am very thankful to Dr.Jasdeep Kaur B.D.S ,kapurthala,Prof.B.K.Bahara (Director,Advanced centre of biotechnology),Prof.Simmi Kharb and Dr.Rajnish K Jain (MDS,Endodontic) for kind support during study

CORRESPONDENCE TO

Dr.Balwant Rai e-mail : drbalwantraissct@rediffmail.com
Mobile No. : 091-9812185855

References

1. 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.
2. Rai B, Anand SC, Kharb S. Panoramic radiograph as a detective of cardiovascular risk. World Journal of Medical Science 2006; 1 (2): accepted.
3. Clauss A. Rapid physiological coagulation method in determination of fibrinogen. Acta Haematol 1957; 17: 237-246.
4. Kweider M, Lowe GD, Murray GD, Kinane DF, McGowan DA. Dental disease, fibrinogen and white cell count; links with myocardial infraction? Scott Med J 1993; 38: 73-74.
5. Fredriksson M, Figueredo C, Gustafsson A, Bergstrom K, Asman B. Effect of periodontitis and smoking on blood leukocytes and acute phase protein. J Periodontol 1999; 70: 1355-60.
6. Albert MA, Ridker PM. Inflammatory biomarkers in Africans American : A potential link to accelerated atherosclerosis. Rev Cardiovas Med 2004; 5 (Suppl 3): S22-S27.
7. Merchant A. Whether periodontitis causes anemia cannot be determined. J Evid Base Dent Prac 2002; 2: 329-40.
8. Klinger MH, Jelkmann W. Role of blood platelets in infection and inflammation. J Interferon Cytokine Res 2002; 22: 913-22.
9. Herzberg MC, Weyer MW. Dental plaque, platelets; and cardiovascular diseases. Ann periodontal 1998; 3: 151-60.
10. Lower GD, Vernell JW, Rumby A, Bainton D, Sweetnam PM. C-reactive protein, 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 (4): 403-10.
11. Noack B, Genco RJ, Trevisan M, Grossi S, Zambon JJ. Periodontal infections contribute to elevated systemic c-reactive protein level. J Periodontol 2001; 72 (9): 1296-97.
12. Jadhav PP, Tofler GH. Hemostatic risk factors for cardiovascular disease. In: Triggering of acute coronary syndromes: implications for prevention. Willich SN, Muller JE, editors Dordrecht: Kluwer Academic Publishers, pp. 135-155.

Author Information

Balwant Rai

Editor in Chief Internet Journal Of Dental Science, P.D.M.Dental College and Research Institute

Simmi Kharb, M.D.Biochemistry

P.G.I.M.S