

## After Scaling and Root Planning Lower Systemic Inflammatory and Thrombotic Marker of Cardiovascular Risk

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**Abstract:** Numerous studies of link between periodontitis and cardiovascular disease has been observed. We used a treatment intervention model to study the relationship between periodontitis and systemic inflammatory and thrombotic cardiovascular indicators of risk. We studied 47 subjects (M:F, 25:22, in age group 30-40 years) with severely periodontitis requiring scaling and root planning. Blood samples were obtained: (1) at initial presentation, (2) after 2 weeks of scaling and root planning (3) after 10 weeks of scaling and root planning. After the treatment, there is significant decrease in c-reactive protein, fibrinogen, white blood cells, neurophil, platelets, while increase hemoglobin and RBC levels. This research paper shows that elimination of periodontitis by scaling and root planning reduces systemic inflammatory and thrombotic markers of cardiovascular risk. Also, supports that links between periodontitis and cardiovascular disease exists.

**Key words:** Systemic markers • cardiovascular marker • periodontitis

### 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 aetiologies, 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- $\alpha$ , IL-6 which are thought to be initiating factor cardiovascular disease. Hence, the present study was planned, effect of systemic inflammatory and thrombotic marker after periodontal treatment.

### MATERIALS AND METHODS

Forty seven (M:F, 25:22, in age group 30-40 years) having atleast 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 1/3<sup>rd</sup> 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; two 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 fibrinogen and c-reactive protein. Plasma was obtained after centrifugation at 1500 g for 10 min and stored at 4°C until analysis of c-reactive protein. CRP was analysed by a immuno assay (using two monoclonal antibodies with sensitivity 0.3 mg l<sup>-1</sup> of medix diacor). Plasma fibrinogen was determined according to Clauss method.<sup>3</sup> All the statistical analysis were performed using SPSS Software package (version 7.0) and student t-test was applied.

### RESULTS

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

Table 1: (Mean±SD) effect of scaling and root planning on blood cell counts, pre-treatment (T<sub>1</sub>), past treatment (T<sub>2</sub>) (After 2 week of scaling and root planning) and 10 week after scaling and root planning (T<sub>3</sub>) counts of total white blood cells, neutrophils, lymphocytes, platelets counts, red blood cells, hemoglobin, c-reactive proteins, fibrinogen

	Sex	T <sub>1</sub>	T <sub>2</sub>	T <sub>3</sub>
Neutrophils (X10 <sup>9</sup> l)	M	4.9 (3.7-5.8)	4.3 (3.4-5.6)	3.9 (3.1-5.5)*
	F	4.8 (3.6-5.6)	4.6 (3.5-5.5)	3.8 (3.1-5.4)**
White cell count (X10 <sup>9</sup> l)	M	7.3 (6.1-9.3)	7.1 (6.2-9.6)	6.5 (5.9-8.7) <sup>a</sup>
	F	7.1 (5.9-9.2)	6.7 (5.7-9.3)	6.3 (5.6-9.2) <sup>b</sup>
Red blood cells (X10 <sup>12</sup> l)	M	5.0 (4.7-5.7)	5.1 (4.6-5.3)	5.1 (4.6-5.3) <sup>c</sup>
	F	4.9 (4.6-5.1)	4.9 (4.6-5.1)	4.9 (4.7-5.2) <sup>d</sup>
Platelets (X10 <sup>12</sup> l)	M	240 (204-300)	235 (207-302)	231 (197-294) <sup>e</sup>
	F	240 (205-301)	233 (206-301)	225 (196-288) <sup>f</sup>
Hemoglobin (g l <sup>-1</sup> )	M	146 (132-164)	147 (133-167)	149 (137-168) <sup>g</sup>
	F	145 (132-162)	146 (133-163)	146 (137-167) <sup>h</sup>
CRP (mg l <sup>-1</sup> )	M	2.6 (0.8-6.7)	2.1 (0.7-5.9)	1.7 (0.5-5.3) <sup>i</sup>
	F	2.6 (0.9-6.8)	2.0 (0.8-5.8)	1.8 (0.6-5.2) <sup>j</sup>
Fibrinogen (g l <sup>-1</sup> )	M	3.7 (2.8-4.8)	3.7 (2.8-4.9)	3.5 (2.7-4.5) <sup>k</sup>
	F	3.7 (2.7-4.9)	3.7 (2.9-4.9)	3.5 (2.7-4.4) <sup>l</sup>

\*,\*\* = Statistically significant difference between T<sub>1</sub> and T<sub>3</sub> and between T<sub>2</sub> and T<sub>3</sub> (p<0.001, p<0.001), a, b = Statistically significant difference between T<sub>1</sub> and T<sub>3</sub> and between T<sub>2</sub> and T<sub>3</sub> (p<0.001, p<0.001), c = Statistically significant difference between T<sub>1</sub> and T<sub>3</sub> (p<0.01), d = Statistically significant difference between T<sub>1</sub> and T<sub>3</sub> (p<0.01), e, f = Statistically significant difference between T<sub>1</sub> and T<sub>3</sub> and between T<sub>2</sub> and T<sub>3</sub> (p<0.002, p<0.001), g, h = Statistically significant difference between T<sub>1</sub> and T<sub>3</sub> and between T<sub>2</sub> and T<sub>3</sub> (p<0.05, p<0.005), i, j = Statistically significant difference between T<sub>2</sub> and T<sub>3</sub> (p<0.001)

increase after treatment (Table 1). The CRP and fibrinogen level decrease 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 1, p<0.01). Also, significant decrease in polymorphonuclear leukocytes were observed (Table 1, 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 1, 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 1, 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 1, 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]. 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. The CRP might be indicator of chronic infective processes possibly correlated with risk of coronary heart disease, such as infection by *Chlamydia pneumonia* or chronic gastric infection with *Helicobacter pylori* [10]. The lower plasma levels of CRP were observed in the present study in post-treated patients as compared to pre-treated patients (Table 1, p<0.001). Recent studies also have

reported elevated CRP levels among these with periodontitis [12]. It has been suggested that CRP is valuable marker in assessment of cardiovascular risk and strong associated of CRP with periodontal disease has been reported in literature [12].

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. Epidemiological studies have shown that increased level of CRP fibrinogen are strong predictors of cardiovascular disease [12].

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, fibrinogen and CRP 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.

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#### REFERENCES

1. Slade, G.D., E.M. Ghezzi and G.R. Heiss, 2003. Relationship between periodontal disease and c-reactive protein among adults in atherosclerosis risk in community study. *Arch. Int. Med.*, 63: 1972-79.
2. Rai, B., S.C. Anand and S. Kharb, 2006. Panoramic radiograph as a detective of cardiovascular risk. *World J. Med. Sci.*, 1: 000-000. (When final write pp:)
3. Clauss, A., 1957 Rapid physiological coagulation method in determination of fibrinogen. *Acta Haematol.*, 17: 237-246.
4. Kweider, M., G.D. Lowe, G.D. Murray, D.F. Kinane and D.A. McGowan, 1993. Dental disease, fibrinogen and white cell count; links with myocardial infraction? *Scott. Med. J.*, 38: 73-74.
5. Fredriksson, M., C. Figueredo, A. Gustafsson, K. Bergstrom and B. Asman, 1999. Effect of periodontitis and smoking on blood leukocytes and acute phase protein. *J. Periodontol.*, 70: 1355-60.
6. Albert, M.A. and P.M. Ridker, 2004. Inflammatory biomarkers in Africans American : A potential link to accelerated atherosclerosis. *Rev. Cardiovas Med.*, 5: S22-S27.
7. Merchant, A., 2002. Whether periodontitis causes anemia cannot be determined. *J. Evid. Base Dent. Prac.*, 2: 329-340.
8. Klinger, M.H. and W. Jelkmann, 2002. Role of blood platelets in infection and inflammation. *J. Interferon Cytokine Res.*, 22: 913-922.
9. Herzberg, M.C. and M.W. Weyer, 1998. Dental plaque, platelets; and cardiovascular diseases. *Ann. Periodontol.*, 3: 151-160.
10. Lower, G.D., J.W. Vernell, A. Rumby, D. Bainton and P.M. Sweetnam, 2001. 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.*, 21: 403-410.
11. Noack, B., R.J. Genco, M. Trevisan, S. Grossi and J.J. Zambon, 2001. Periodontal infections contribute to elevated systemic c-reactive protein level. *J. Periodontol.*, 72: 1296-1297.
12. Jadhav, P.P. and G.H. Tofler, 1996. Hemostatic risk factors for cardiovascular disease. In: *Triggering of acute coronary syndromes: implications for prevention.* Willich, S.N. and J.E. Muller, editors Dordrecht: Kluwer Academic Publishers, pp: 135-155.