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The effect of non-surgical periodontal therapy on systemic inflammatory markers

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Abstract

Background: Systemic inflammatory markers can be common pathogenic link between various diseases of human body. Periodontitis has also been related with mild elevation of systemic inflammation. It may be responsible for association with cardiovascular, renal, pulmonary diseases, etc. Thus, treatment of periodontitis can reduce risk of systemic diseases.

Aim: To evaluate the effect of non-surgical therapy on Total leucocyte count, Differential count and platelet count in periodontitis patients.

Material and Methods: A 30 patients with chronic periodontitis selected, full mouth scaling and root planing done. Clinical parameters recorded were plaque index, gingival index and probing pocket depth. Haematological parameters recorded were Total Leucocyte count (TLC), Differential count (DC) and Platelet count. Both examinations done on baseline and 2 weeks postoperatively.

Results: Significant reductions in all the clinical and haematological parameters from baseline to 2 weeks postoperatively.

Conclusion: A reduction in the haematological parameters after nonsurgical periodontal therapy may also reduce the risk of atherosclerosis formation in the blood vessel and possibly prevent cardiovascular diseases.

Keywords: Non-surgical periodontal therapy, atherosclerosis, periodontitis

Introduction

Periodontitis is multifactorial chronic inflammatory disease, leads to entry of bacteria or their products into the bloodstream, which in turn activates the host response. Various systemic disease and condition has been shown to be associated with periodontitis in multiple

epidemiologic, experimental, and interventional studies. The most studied associations are diabetes, pregnancy complications, and cardiovascular diseases.[1]

Cardiovascular diseases comprise a variety of heart and vascular conditions including ischemia, atherosclerosis, peripheral artery disease, infective endocarditis, and acute myocardial infarction. Although direct cause effect relation has not been established, periodontitis and cardiovascular disease has been seemed to be associated in various observational studies.[2,7]

Inflammation is recognized to be playing a key role in the pathogenesis of atherosclerosis as well as of periodontitis.[2] Systemic inflammatory mediators like leucocytes, platelets, fibrinogen, C reactive protein (CRP) are shown to increase in both diseases.[1]

Increased white blood cell counts along with elevated level of fibrinogen increases blood viscosity in periodontal inflammation[5]. Which is a predictor of development of atheromatous plaque. Platelets also increased in number in periodontitis.[4] Their role in primary haemostasis due to their capacity to aggregate and form atheromatous plaque, and also through innate immune regulation like production of proinflammatory mediators and expression of multiple surface receptors will increase chances of cardiovascular diseases.[2] Furthermore, Porphyromonas gingivalis a 'keystone pathogen' among microbes causing periodontitis[3], secrete proteolytic enzymes which in turn activates platelets and also modify the expression of cytokines.

After periodontal therapy a systemic inflammatory response triggered for short time followed by gradual reduction.[8] Various interventional studies showed significant reduction in different parameters like platelets and white blood cell count after periodontal treatment.[4,6]

The aim of the present study was to investigate the effect of nonsurgical periodontal therapy on total leukocyte count (TLC), differential leukocyte count (neutrophils, lymphocytes, eosinophils, basophils, and monocytes), and total platelet count in patients with periodontitis.

Material and methods:

A total of 30 patients with periodontitis were selected from the Department of Periodontology, Government Dental college and hospital, Ahmedabad. Systemically, healthy, within the age group of 20–55 years of both the genders having probing pocket depth (PPD) of ≥5 mm at four sites per tooth in different quadrants of the mouth were included.

Patients suffering from any systemic diseases, undergoing any periodontal therapy, taking antibiotic, or having taken antibiotics in the past 3 months, smokers, pregnant women, and lactating mothers were excluded.

The study was approved by the Institutional Ethical Committee. Written consent was obtained from all the patients who were recruited for the study.

Clinical parameters recorded were Plaque Index (PI), Gingival Index (GI) and Probing Pocket Depth (PPD) with help of University of North Carolina (UNC)-15 probe. Non-surgical periodontal therapy consists of full-mouth scaling and root planing along with 0.2%chlorhexidine gluconate mouthwash twice a day for 14 days given.

Venous blood samples were taken on preoperative (baseline) and after two weeks postoperatively. It was obtained at the same time of the day every time, and were immediately transported and processed. The laboratory analysis of total leucocyte count (TLC), differential leucocyte count (DC) and platelet count, were performed by a blinded pathologist. Results obtained were subjected to statistical analysis , ANOVA test was done and p value less than 0.05 was considered as level of significance.

Results

Table 1 shows statistically significant ($p<0.05$) changes in all the clinical parameters from baseline to 2 weeks postoperatively. A significant reduction in Plaque Index decreased from 2.07±0.67 at baseline to 1.67±0.56 after 2 week, Gingival index from 2.01±0.56 at baseline to 0.85±0.87 after 2 weeks and probing pocket depth from 3.74±0.40 at baseline to 2.96±0.34 after 2 week.

Table 2 shows statistically significant decrease in haematological parameters. Total Leucocyte Count (TLC) was 6566/mm3 at baseline, and at and at 2 weeks follow-up it was 6036/mm3, ($P < 0.05$). There was a statistically significant decrease in platelet count from 2.01 lac/mm3 preoperatively to 1.91 lac/mm3 at 2 weeks postoperatively ($P < 0.05$). There was no statistically significant difference in the differential leukocyte counts after nonsurgical periodontal therapy.

Discussion

Periodontitis and cardiovascular disease association has been explained via various mechanisms like bacterial dissemination, periodontal pathogen in atherogenic plaque and systemic inflammatory markers like C-reactive protein, interleukins, platelets, fibrinogen, white blood cells, dyslipidaemia, genetic risk factors, antibodies against periodontal microorganisms etc.[2]

Present study was aimed to evaluate effect of nonsurgical periodontal therapy on systemic inflammatory markers. A total number of 30 patients with chronic periodontitis had been selected. Full mouth scaling and root planing done with adjunctive use of 0.2% chlorhexidine gluconate mouthwash 10ml twice daily for 14 days. Clinical parameters assessed were Plaque Index(PI), Gingial Index(GI) and Probing Pocket Depth(PPD) at baseline and after 2 week postoperatively. Venous blood sample taken from antecubital vein and immediately processed in laboratory and evaluated by blinded pathologist for various haematological parameters like Total Leucocyte count(TLC), Differential count(Neutrophils, Basophils, Eosinophils, Monocytes and lymphocytes) and Platelet counts. Blood sample was taken just

Leucocytes are integral part of immunity. A direct dose–response relationship has been observed between increasing levels of leucocyte count and graded increase in cardiovascular disease risk [10]. Higher leucocyte counts have also been associated with increased periodontal inflammation[9]. Which is mainly due to an increase in the numbers of polymorphonuclear leukocytes (PMNs) as they represent the first line of defense and recruited at higher levels during episodes of bacteremia in periodontitis.[11] Leucocytes increases viscosity of blood and create hypercoagulable state which predispose to atherogenic plaque formation. [5]

In present study, a statistically significant decrease in TLC was observed two weeks after scaling and root planing (from 6566 / mm3 at baseline to 6036 / mm3 at 2 weeks postoperatively.) Similar findings were also reported by Christan et al.[6] who showed reduction in total leucocyte count from $7.4 \cdot 10^9$/l to $5.8 \cdot 10^9$/l after treatment. A significant reduction in neutrophil counts also observed; from 61% at baseline to 56% after 2 weeks. These results are in accordance with Kalsi DS et al [12] who showed significant reduction in neutrophil as they are cells of acute inflammation, and plaque-induced periodontitis is an infective-inflammatory disease.

Other white blood cells e.g., lymphocytes, eosinophils and monocytes also showed reduction but it was statistically nonsignificant. Similar findings were observed in other studies which showed minimal effect of periodontal therapy on agranulocytes.[11,13]

Thaulow et al.[14] studied that platelet counts were positively related to the risk of cardiovascular death. Periodontal inflammation also has been related to increased platelet count[4] which can be considered as a

"reactive thrombocytosis"[11]. Platelets are responsible for primary haemostatic plug formation and coaggregation has important role in atherogenesis. These cells are able to express multiple surface receptors, such as P-selectins or toll-like receptors that can interact with bacteria like P. gingivalis. As a result of activation by periodontal pathogen and their products, platelets release a wide variety of proinflammatory mediators and form aggregates with other cells, such as neutrophils or monocytes etc. Hence, an increase in platelets might be another underlying mechanism for the possible link between periodontal inflammation and CVD.

In present study reduction in platelet count was observed from 2.01 lac/mm^3 at baseline to 1.9 lac/mm^3 at 2 weeks postoperatively. Similar findings were shown by Christan et al.[6] who showed a decrease in platelet counts after periodontal therapy from 2.54×10^3 to 2.25×10^3.

Conclusion

Periodontitis and cardiovascular disease association has been proved via link of systemic inflammation markers. Positive effect of non-surgical periodontal therapy on inflammatory markers may explain its importance in reduction of risk of cardiovascular disease.

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Legend Tables

Table 1: Clinical parameters at different time interval

Clinical parameters	Baseline	At 2 weeks	p Value
Plaque Index (PI)	2.07±0.67	1.67±0.56	0.01
Gingival Index (GI)	2.01±0.56	0.85±0.87	0.03
Probing Pocket Depth (PPD)	3.74±0.40	2.96±0.34	0.02

Table 2: Haematological Parameters at different time interval

Haematological parameters	Base line	At 2 weeks	p Value
Total leucocyte count(/mm3)	6566	6036	0.007
Neutrophils(%)	61	56	0.04
Lymphocyte(%)	39	37	0.1
Eosinophils(%)	15	14	0.2
Monocytes(%)	3.53	3.10	0.07
Platelet count(lac/mm3)	2.01	1.91	0.02