ORIGINAL RESEARCH

Effect of Non-surgical Therapy on Peripheral Blood Count in Smoking and Non-smoking Patients with Generalized Aggressive Periodontitis

Rohit R Thakkar[1], Mayank K Chaturvedi[2], Haresh Kalvani[3], Hardik B Patel[4], Jay H Doshi[5], Ketan Prajapati[6]

ABSTRACT

Introduction: Aggressive periodontitis (AgP) is characterized by rapid progression of attachment and bone loss in healthy individuals with no contributory medical history. A study was planned to evaluate the effect of non-surgical periodontal therapy on white blood cell (WBC) count, differential count, and peripheral platelet count (pplat) count in smokers and non-smokers with generalized AgP.

Materials and Methods: A total of 20 generalized AgP of both sex diagnosed from the outpatients of the Department of Periodontics, Darshan Dental College, Udaipur, Rajasthan, were included for the present study. Estimation of WBC count, differential count, and Pplat count was done using Sysmex KX-21, automated hematology analyzer.

Results: Mean reduction of total WBC was statistically significant ($P < 0.001$) in baseline to 1 month and baseline to 3 months of interval in both groups. Mean reduction of Pplat count was statistically significant ($P < 0.001$) in baseline to 1 month and baseline to 3 months of interval in both groups.

Conclusions: The results of the present study show that there is a definite effect of non-surgical periodontal therapy on total WBC count, differential count, and Pplat count in generalized AgP patients which differs between smokers and non-smokers.

Keywords: Generalized aggressive periodontitis, Non-surgical therapy, Peripheral blood, Systemic inflammation, Total white blood cell count

How to cite this article: Thakkar RR, Chaturvedi MK, Kalvani H, Patel HB, Doshi JH, Prajapati K. Effect of Non-surgical Therapy on Peripheral Blood Count in Smoking and Non-smoking Patients with Generalized Aggressive Periodontitis. Int J Oral Care Res 2018;6(1):S66-70.

Source of support: Nil

Conflicts of interest: None

[1,6]Tutor, [2]Assistant Professor, [3]Professor and Head, [4,5]Reader

[1,3]Department of Periodontology, Siddhpur Dental College and Hospital, Siddhpur, Gujarat, India

[2]Department of Dentistry, GMERS Medical College and Hospital, Patan, Gujarat, India

[4]Department of Conservative Dentistry and Endodontics, Pacific Dental College and Research Centre, Udaipur, Rajasthan, India

[5]Department of Oral and Maxillofacial Surgery, Pacific Dental College and Research Centre, Udaipur, Rajasthan, India

[6]Department of Oral Pathology, Siddhpur Dental College and Hospital, Siddhpur, Gujarat, India

Corresponding Author: Dr. Mayank K Chaturvedi, Assistant Professor, Department of Dentistry, GMERS Medical College and Hospital, Dharpur, Patan, Gujarat – 384 265, India. e-mail: drmayankmk@gmail.com

INTRODUCTION

The oral cavity comprises an ecosystem that is made up of more than 700 bacterial species. When favorable conditions exist, a proliferative change in these pathogens produces clinical disease. It is now generally accepted that much of periodontal destruction observed in periodontitis is host mediated through pro-inflammatory cytokines, by local tissues and by cells of the immune system in response to bacteria and their products such as lipopolysaccharides. Thus, this chronic Gram-negative infection exerts a continuous and sustained insult, which is thought to bring about an alteration in the host systemic status.

Several parameters of systemic inflammation have been identified as markers for cardiovascular diseases. Several direct and indirect pathogenic mechanisms have been discussed to explain the link between infectious disease and atherosclerosis, among them a systemic inflammatory response that may cause elevated levels of established risk factors for atherosclerosis.[1]

Variation in white blood cell (WBC) count has been associated with atherosclerosis in a number of epidemiological studies and is considered to be a risk factor for the disease. It is currently unknown whether periodontal disease affects hematological variables such as WBC. Wakai *et al.* in a cross-sectional study showed a significant rise in platelet count with higher community periodontal index of treatment needs score and showed a slightly upward trend for platelet aggregation. Few studies have analyzed hematological variables in periodontitis patients and the results were contradictory. The only available intervention study failed to show an effect of periodontal therapy on WBC. However, most of these studies dealt with patients with chronic periodontitis of moderate severity.[2,3]

Smoking has been found to increase the risk of periodontitis. It has also been documented that smokers

respond less favorably than non-smokers to non-surgical as well as surgical periodontal treatment. Cigarette smoking is also the strongest predictor of future attachment loss and bone loss. Smoking *per se* induces several changes in the blood such as increased radical generation from peripheral neutrophils and imbalance between proteases/antiproteases. Even passive cigarette smoking is associated with increased leukocyte counts and increased release of reactive oxidants from stimulated neutrophils. As smoking is well-known contributor to elevated WBC counts, other factors like periodontitis may also contribute to elevated WBC counts. Patients with periodontitis exhibit a significantly increased peripheral WBC count, thus suggesting that cigarette smoking and periodontal disease may be an important factor in the elevation of leukocyte counts.[4,5]

Aggressive periodontitis (AgP) is characterized by rapid progression of attachment and bone loss in healthy individuals with no contributory medical history. AgP has a multifactorial etiology and the characteristic tissue destruction is mediated mainly by the aberrant immune response of the host to periodontopathic bacteria. The rapid destruction seen in these cases may be due to a hyperresponsive neutrophil (polymorphonuclear [PMN]) and macrophage phenotype. Several studies have indicated that PMNs from AgP patients may have impaired chemotaxis, defective phagocytosis, and increased superoxide production. The body of evidence points to neutrophil-mediated tissue injury causing the tissue destruction seen in AgP. Christan *et al.* reported a decrease in peripheral leukocyte numbers after non-surgical periodontal therapy in patients with generalized AgP. Elevated leukocyte numbers in the peripheral bloodstream can be presumed in untreated patients with AgP compared to subjects with healthy periodontium.[6] Hence, a study was planned to evaluate the effect of non-surgical periodontal therapy on WBC count, differential count, and peripheral platelet count (pplat) count in smokers and non-smokers with generalized AgP.

MATERIALS AND METHODS

A total of 20 generalized AgP of both sex diagnosed from the outpatients of the Department of Periodontics, Darshan Dental College, Udaipur, Rajasthan, were included for the present study.

Methods of Collection of the Data

Patients were divided into two groups of 10 patients each:

- Group I: (Control group) Individuals diagnosed clinically as suffering from generalized AgP and who were non-smokers
- Group II: (Test group) Individuals diagnosed clinically as suffering from generalized AgP and who were smokers.

All patients underwent non-surgical periodontal therapy at baseline, which consists of thorough scaling and root planing and oral hygiene instructions.

Before dental procedure, blood samples were collected, i.e., baseline, and after therapy at 1 month and 3 months interval. The blood samples were subjected to laboratory analysis for the evaluation of total WBC count, differential count, and platelet count. At baseline, 1 month, and 3 months, the following clinical parameters and blood investigations were evaluated.

Clinical Parameters

1. Modified sulcus bleeding index [MSBI]
2. Pocket probing depth [PPD]
3. Method of collection of blood sample.

Blood Investigation

Venous blood samples were collected and assessed for
1. Estimation of WBC count and differential count
2. Estimation of Pplat count

Laboratory Estimation

Estimation of WBC count, differential count, and Pplat count was done using Sysmex KX-21, automated hematology analyzer.

The mean values and standard deviation, of all the parameters (total leukocyte count [TLC], neutrophils, lymphocytes, monocytes, eosinophils, Pplat count, MSBI, and PPD), were estimated at baseline, 1 month, and 3 months. Student's *t*-test was used to compare between period and groups.

RESULTS

This study was conducted on 20 patients. Ten non-smoker and 10 smoker patients with generalized AgP were enrolled for the study.

Hematological Variables

Total WBC (TLC)

The mean total WBC counts in generalized aggressive periodontitis (GAP) non-smoker were 7620.00 ± 1536.81 at baseline which reduced to 7030.00 ± 1320.82 at 1 month and 5770.00 ± 861.59 at 3 months. While in GAP smoker group, the total WBC counts were 9020.00 ± 1986.51 at baseline and reduced to 8130.00 ± 1523.19 and 7530.00 ± 1594.47 after 1 month and 3 months, respectively [Graph 1]. Mean reduction of TLC was statistically significant ($P < 0.001$) in baseline

to 1 month and baseline to 3 months of interval in both groups.

Pplat Count

The mean Pplat counts in GAP non-smoker were 2.40 ± 0.55 at baseline which reduced to 2.22 ± 0.43 at 1 month and 1.81 ± 0.30 at 3 months. While in GAP smoker group, the Pplat counts were 2.74 ± 0.77 at baseline and reduced to 2.46 ± 0.61 and 2.14 ± 0.50 after 1 month and 3 months, respectively [Graph 2]. Mean reduction of Pplat was statistically significant ($P < 0.001$) in baseline to 1 month and baseline to 3 months of interval in both groups.

Clinical Parameters

MSBI

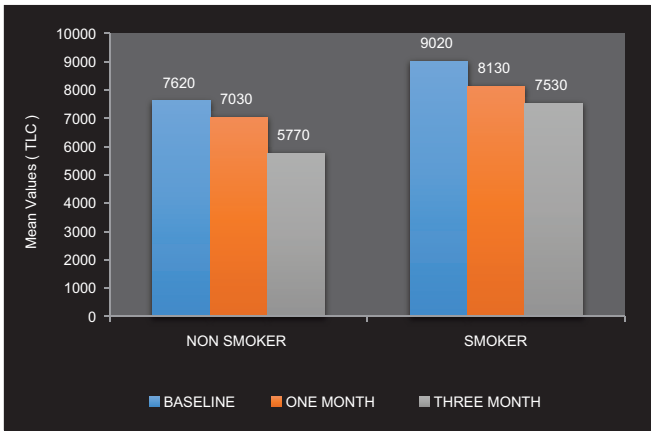
The mean MSBI was1.92 ± 0.28 at baseline, which declined to 1.31 ± 0.31 and 1.22 ± 0.32 at 1 month and 3 months after the periodontal therapy in GAP non-smoker group. In GAP smoker group from baseline which was 1.99 ± 0.25 reduced to 1.36 ± 0.24 and 1.23 ± 0.22 at 1 month and 3 months [Graph 3]. Mean reduction in MSBI at baseline to 1 month and baseline to 3 months after the periodontal therapy was statistically significant ($P < 0.001$) in both groups.

PPD

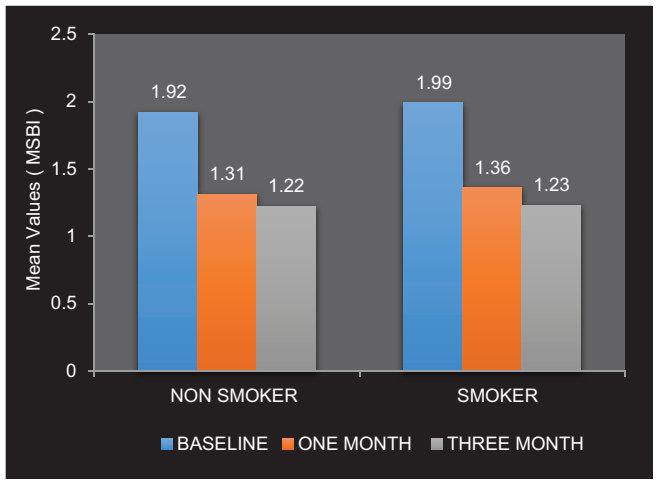
Following the non-surgical periodontal treatment, PPD decreases from 6.70 ± 0.24 at baseline to 5.52 ± 0.25 and 4.82 ± 0.28 after 1 month and 3 months in GAP non-smoker group. While in GAP smoker group, the PPD was 7.16 ± 0.54 at baseline and decreases to 6.23 ± 0.56 and 5.58 ± 0.52 after 1 month and 3 months, respectively [Graph 4]. Reduction in the PPD was statistically significant ($P < 0.001$) in both groups at baseline to 1 month and baseline to 3 months.

DISCUSSION

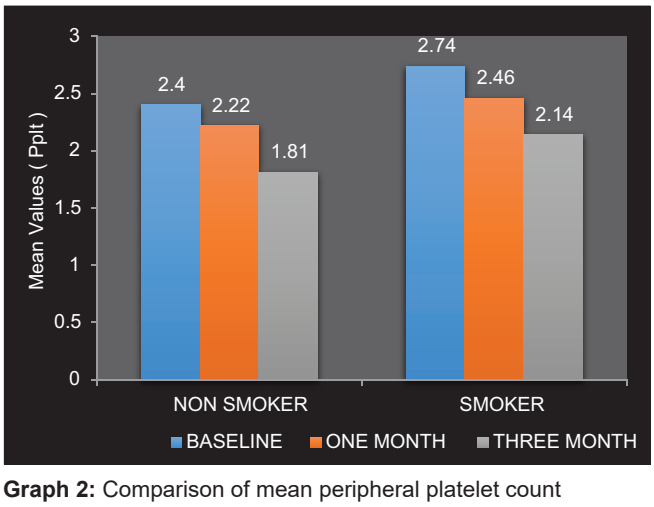
Only few studies have measured leukocyte counts in periodontitis patients. Gustafsson and Asman found no significant differences between 14 patients with periodontitis and 14 healthy patients.[7] In contrast, Loos *et al.* found significantly higher leukocyte counts in 54 patients with chronic generalized periodontitis



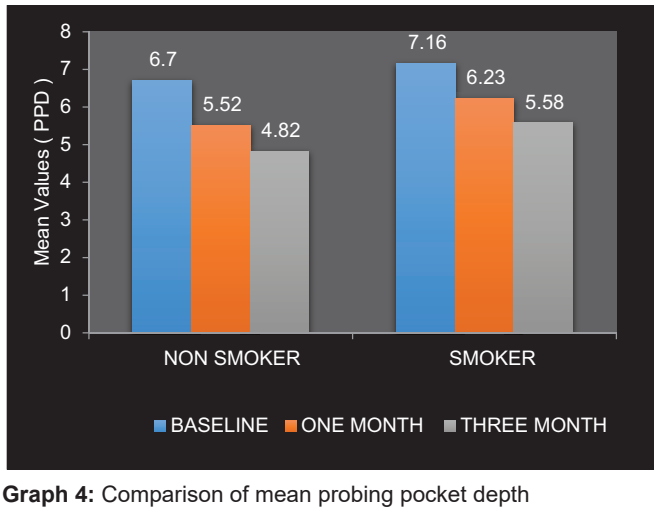
Graph 1: Comparison of mean total white blood cell count



Graph 3: Comparison of mean modified Sulcus Bleeding Index score



Graph 2: Comparison of mean peripheral platelet count



Graph 4: Comparison of mean probing pocket depth

compared to 53 patients with chronic localized periodontitis and 43 healthy controls.[1] Fredriksson *et al.* reported significantly higher leukocyte counts in 17 treated periodontitis patients compared to 17 age- and sex-matched healthy controls. No information was given regarding the smoking habits of the subjects' investigated.[8] In another study by the same group, significantly higher leukocyte counts are reported in non-smoking periodontitis patients compared to non-smoking healthy controls. The difference between smoking periodontitis patients and smoking controls was not statistically significant.[4] The earliest intervention study was published by Christgau *et al.* who studied the effect of periodontal therapy in 20 diabetics and 20 non-diabetic controls with periodontal disease. They also reported leukocyte counts in the course of periodontal therapy. Although the differences between pre- and post-treatment values were not significant, leukocyte counts decreased in both groups after non-surgical periodontal therapy.[9] An another study by Christan *et al.*, who also evaluated the effect of non-surgical periodontal therapy in 27 generalized AgP patients and found that total WBC counts, neutrophil counts, and platelet counts decreased significantly in non-smokers, while in smokers, only platelet counts were reduced significantly after non-surgical periodontal therapy. This was the only study where generalized AgP patients were evaluated.[2] The present study investigated the effect of non-surgical periodontal therapy in young patients with generalized AgP and also evaluated the role of smoking on the outcome of periodontal treatment. This study population was particularly suited to investigate systemic effect of periodontal therapy because these patients have advanced periodontal disease with many remaining teeth. In classic paper on rapidly progressive periodontitis (now known as AgP), Page *et al.* stated that these patients frequently suffer from fever and malaise, indicating that this type of periodontal disease often has systemic sequelae.[10-15] The results of this study are in agreement with studies conducted by Christan *et al.* who observed that periodontal therapy may lead to a significant reduction of WBC counts in non-smoking GAP patients and to a significant reduction of platelet counts in both smokers and non-smokers with GAP.[2] In our study, the effect of periodontal therapy on the leukocyte count was distinct in non-smokers. This is consistent with the results of Fredrikssson *et al.* who found that the combination of periodontitis and smoking alters the blood parameters and there is an increase in levels of leukocyte counts and differential counts in smokers.[4] Loos *et al.* showed that periodontitis results in higher systemic levels of neutrophils and leukocyte counts and it may have systemic sequelae.[1] Thus, the result of the present study indicates that periodontal therapy may lead to a significant reduction of total WBC counts and differential count in non-smoking patients compared to smoking patients and also the significant reduction of platelet counts in both smokers and non-smokers. Hence, it indicates that therapeutic intervention has definite systemic effect on the blood count in GAP patients which differs between smokers and non-smokers.

CONCLUSIONS

The results of the present study show that there is a definite effect of non-surgical periodontal therapy on total WBC count, differential count, and Pplat count in generalized AgP patients which differs between smokers and non-smokers.

REFERENCES

1. Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PM, van der Velden U. Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients. J Periodontol 2000;71:1528-34.
2. Christan C, Dietrich T, Hägewald S, Kage A, Bernimoulin JP. White blood cell count in generalized aggressive periodontitis after non-surgical therapy. J Clin Periodontol 2002;29:201-6.
3. Wakai K, Kawamura T, Umemura O, Hara Y, Machida J, Anno T, *et al.* Associations of medical status and physical fitness with periodontal disease. J Clin Periodontol 1999;26:664-72.
4. Fredriksson MI, Figueredo CM, Gustafsson A, Bergström KG, Asman BE. Effect of periodontitis and smoking on blood leukocytes and acute-phase proteins. J Periodontol 1999;70:1355-60.
5. Renvert S, Dahlén G, Wikström M. The clinical and microbiological effects of non-surgical periodontal therapy in smokers and non-smokers. J Clin Periodontol 1998;25:153-7.
6. Shi D, Meng H, Xu L, Zhang L, Chen Z, Feng X, *et al.* Systemic inflammation markers in patients with aggressive periodontitis: A pilot study. J Periodontol 2008;79:2340-6.
7. Gustafsson A, Asman B. Increased release of free oxygen radicals from peripheral neutrophils in adult periodontitis after fc delta-receptor stimulation. J Clin Periodontol 1996;23:38-44.
8. Fredriksson M, Gustafsson A, Asman B, Bergström K. Hyper-reactive peripheral neutrophils in adult periodontitis: Generation of chemiluminescence and intracellular hydrogen peroxide after *in vitro* priming and fcgammaR-stimulation. J Clin Periodontol 1998;25:394-8.
9. Christgau M, Palitzsch KD, Schmalz G, Kreiner U, Frenzel S. Healing response to non-surgical periodontal therapy in patients with diabetes mellitus: Clinical, microbiological, and immunologic results. J Clin Periodontol 1998;25:112-24.
10. Page RC, Altman LC, Ebersole JL, Vandesteen GE, Dahlberg WH, Williams BL, *et al.* Rapidly progressive periodontitis. A distinct clinical condition. J Periodontol 1983;54:197-209.
11. Roshna T, Thomas R, Nandakumar K. A case-control study

on the association of human leukocyte antigen-a 9 and b 15 alleles with generalized aggressive periodontitis in an Indian population. J Periodontol 2006;77:1954-63.
12. Havemose-Poulsen A, Westergaard J, Stoltze K, Skjødt H, Danneskiold-Samsøe B, Locht H, *et al.* Periodontal and hematological characteristics associated with aggressive periodontitis, juvenile idiopathic arthritis, and rheumatoid arthritis. J Periodontol 2006;77:280-8.
13. Kachlany SC. Aggregatibacter actinomycetemcomitans leukotoxin: From threat to therapy. J Dent Res 2010;89:561-70.
14. Schenkein HA, Koertge TE, Brooks CN, Sabatini R, Purkall DE, Tew JG, *et al.* IL-17 in sera from patients with aggressive periodontitis. J Dent Res 2010;89:943-7.
15. Liu RK, Cao CF, Meng HX, Gao Y. Polymorphonuclear neutrophils and their mediators in gingival tissues from generalized aggressive periodontitis. J Periodontol 2001;72:1545-53.