

Impact of Non-Surgical Periodontal Treatment on Total WBC Count in Patients with Periodontitis

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Background and objectives: Chronic periodontitis is an inflammatory disorders associated to the accumulation of microbial biofilm and the response of host to this accumulation. In our society the importance of total WBC counts has not yet been investigated with chronic periodontitis. The objective of this study was to investigate the impact of treatment on total WBC counts in subjects with chronic periodontitis.

Materials and methods: A total of 50 subjects were selected for the study. Venous blood samples were taken at base line and 4 weeks after non-surgical periodontal therapy for the study group (group II), and one time for the control group (group I) to estimate total WBC counts. Clinical parameters such as Plaque Index, Gingival Index, probing pocket depth, the clinical attachment level for group II were measured.

Results: The result of the present study shows a highly significant decrease of total WBC counts between group I and II before treatment P value=0.004. Our results indicate that there is a highly significant decrease of Gingival Index and probing pocket depth at base line compared with after treatment P value=0.003 and 0.000 respectively. Also, a significant decrease in Plaque Index and clinical attachment level at base line and 4 weeks following therapy P value=0.035 and 0.026 respectively. In addition, a significant decrease of total WBC counts at base line and 4 weeks following therapy P value=0.016. Statistical tests were done by standard program data processing (MS excel) paired t test.

Conclusion: The present study concludes that total WBC counts were decreased after non-surgical periodontal therapy

Keywords: Non-surgical periodontal therapy, Total WBC counts, Clinical parameters, Chronic periodontitis, Healthy subjects.

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Introduction

Chronic periodontitis is an infectious disease characterized by inflammation within the supporting tissues of the teeth, progressive attachment loss, and bone loss.¹ It is associated with specific bacteria organized in the dental biofilm. However, the onset, progression, and severity of the disease depend on immune response of the individual.^{2,3} With the development of dental biofilm, early signs of an inflammatory reaction occur in the gingival margin (i.e., gingivitis) without real attachment loss. Usually, the complete resolution of this early gingival inflammation follows the optimal plaque control. On the other hand, with neglected oral hygiene, inflammation will progress and eventually results in the loss of the attachment around the teeth.⁴ Even though not all patients with gingivitis develop periodontitis, it is known that all

patients with periodontitis experienced prior gingivitis. The incidence of periodontitis depends on the individual immune response that modifies the beginning and progression of the disease.³⁻⁵

The tissues sustained inflammation results in signaling of fibroblasts and production of proinflammatory cytokines. The peripheral blood antibodies which are specific to oral bacteria and the acute-phase response becomes activated and C-reactive protein [CRP], fibrinogen and complement are produced both by local cells and within the liver. These proteins may further aggravate the local inflammatory response and may affect the initiation or progression of systemic diseases like atherosclerosis. CRP is positively interrelated to interleukin-6 and activates complement, which accounts for the uptake of low density lipoprotein [LDL] by macrophages. Activated WBCs reflect the inflammatory activity of atherosclerosis that perpetuates vascular injury and ischemia.⁶

Leukocytes are the chief component of blood cells for phagocytosis and the first cells of the host defense mechanism against infective agents.⁷ They are an essential part of the innate immune system, during episodes of bacteremia in periodontitis these cells are recruited at higher levels and outflow into the systemic circulation.⁸

Bacteraemia in periodontitis is well established and the extent is directly related to the severity of inflammation of the periodontal tissues. It has been speculated that periodontitis results in a low grade systemic inflammation. Recently several research groups have confirmed that periodontitis is associated with elevated numbers of white blood cells and elevated levels of C-reactive protein.⁹ Which indicate that patients with periodontitis may have a subclinical systemic inflammatory reaction.

Thus, the aim of the present study is to investigate and compare the total WBC count in both the control group (group I) which is free

from any periodontal disease and group II which is the study group, and also to evaluate the impact of non-surgical periodontal treatment on the total WBC count in the study group (group II) before and after treatment.

Materials and methods

Source of Data. All subjects were recruited between April 2017 and November 2017, the study was carried out in the Department of Periodontics, College of Dentistry - Hawler Medical University. Fifty subjects were selected for the study; the subjects were divided into two groups. Group I included 25 healthy periodontium and group II included chronic periodontitis patients. Subjects suffering from any systemic diseases, pregnant women, lactating mothers, undergoing any periodontal therapy, smokers, taking antibiotic or having taken antibiotics three months prior to the research were excluded. All these conditions possibly influence the leukocyte count.¹⁰ The inclusion criteria for group II in this study were as follows: patients chosen have at least 30% or more of the teeth having greater than or equal to 4 mm probing pocket depth, clinical attachment level more than or equal to 3 mm, age 30-55years,¹¹ both males and females.¹² In addition the inclusion criteria for group I in this study were as follows: no gingivitis (GI=0, BOP=0) or periodontitis (CAL=0, PPD=0).

The study was approved by the Ethical Committee of the College of Dentistry - Hawler Medical University. Informed written consent was obtained from all subjects who were selected for the study.

Study Design. After taking the informed consent from the subjects, blood samples were taken under aseptic conditions into EDTA tube (K3EDTA, China) and then were transferred for WBC count estimation. Estimation was done using (ABX Micros ES 60, HORIBA, FRANCE). For all the subjects with chronic periodontitis, complete supragingival and subgingival scaling and root surface debridement were performed using ultrasonic

scalers (NSK varios 350, JAPAN) and Gracey curettes (MEDESY, Italy) in one visit. Good oral hygiene instructions were given for all patients.

Periodontal Examination. For all the subjects with chronic periodontitis, the following indices were recorded before scaling and root planing:

Gingival index (Loe and Silness, 1963)

Score 0: Normal gingiva

Score 1: Mild inflammation, slight change in color, slight edema. No bleeding on probing.

Score 2: Moderate inflammation, redness, edema, and glazing. Bleeding on probing.

Score 3: Severe inflammation, marked redness and edema, ulceration. The tendency toward spontaneous bleeding.

Plaque Index (Silness and Loe, 1964)

Score 0: No plaque

Score 1: A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be seen in situ only after application of disclosing solution or by using the probe on the tooth surface.

Score 2: Moderate accumulation of soft deposits within the gingival pocket, or the tooth and gingival margin which can be seen with the naked eye.

Score 3: Abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin.

Probing pocket depth was assessed by using the calibrated periodontal probe UNC- 15 (MEDESY, Italy).¹² The measurement was taken from the gingival margin to the base of pocket at four surfaces of each tooth (Figure 1). No pressure was used; the probe was allowed to fall by its own weight.¹³

Clinical attachment level was measured by a calibrated periodontal probe (UNC) from cemento-enamel junction (CEJ) to the base of the pocket.¹³

Severity of PD and CAL were estimated (total PD / CAL divided by affected surfaces) (Bortlod *et al*, 2003).¹⁴



Figure 1: Examination of clinical parameter (periodontal pocket depth) by UNC probe

The following indices were recorded at base line and 4 weeks after non-surgical periodontal therapy for group II which was done by two examiners. The sites for measurement were (mid buccal or labial, mesiolabial or buccal, distolabial or buccal, and mid palatal or lingual).

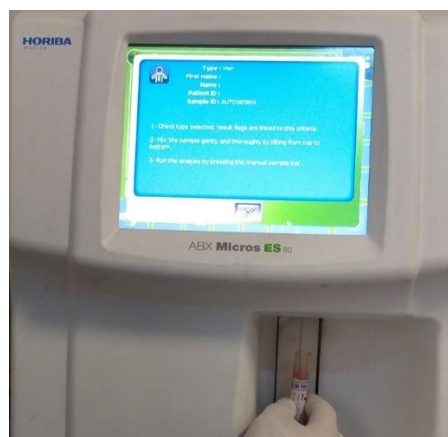


Figure 2: ABX Micros ES 60, HORIBA

Blood Sample Analysis. 2 ml of venous blood sample was taken from the subjects by using disposable syringe (Morningside Pharmaceuticals Ltd, UK) and the blood sample was taken at base line before non-surgical periodontal therapy and 4 weeks after

treatment for chronic periodontitis patients and one time for healthy subjects. The blood samples were analysed using (ABX Micros ES 60, HORIBA, FRANCE) in Laboratory Centre of Hawler Teaching Hospital (Figure 2).

Statistical analysis. Statistical analysis was performed by descriptive and analytical statistical methods using the standard program for data processing (MS excel).

Changes of total WBC counts between group I and II, total WBC counts before and after of group II, Plaque Index, Gingival Index, Probing Pocket Depth, Clinical attachment level following non-surgical periodontal therapy can be seen.

Differences in mean values between groups were compared using Paired *t*-test. Results were considered significant, if *P* value ≤ 0.05.

Results

The mean age of the study subjects was (38 ± 3.90 and 38± 4.22) for control and study groups respectively. In group I, fourteen of them were females and eleven of them were males (Table 1), while in group II, thirteen of them were females and twelve of them were males. The results of the present study show that the mean and the standard deviation of total WBC counts in both groups I and II was (5.95×10³±1.26, 7.04×10³±1.44) /mm³ respectively (Table 2). There was a highly statistically significant decrease in Gingival Index and probing pocket depth (*P*=0.003 and <0.001) after treatment. There was statistically significant decrease in Plaque Index, clinical attachment level and total WBC counts (0.035, 0.026, and 0.016) respectively after treatment (Table 3).

Table 1: The result of demographical data between group I and II.

Demographical data	
Gender (group I)	14 female 11 male
Gender (group II)	13 female and 12 male
Age (group I and II)	30-55years
Mean age (group I)	38 ± 3.90
Mean age (group II)	38± 4.22

Table 2: Comparing total WBC count between group I and II before treatment was done by independent t test; group I and group II after treatment was done by independent t test, group II (before and after) was done by paired t test.

Group I	5.95×10 ³ ±1.26	0.004 **
Group II (Before)	7.04×10 ³ ±1.44	
Group I	5.95×10 ³ ±1.26	0.211
Group II (After)	6.20×10 ³ ±1.12	
Group II (Before)	7.04×10 ³ ±1.44	0.016
Group II (After)	6.20×10 ³ ±1.12	

* Significant, ** highly significant

Table 3: Comparing clinical parameters in group II (between before and after treatment) were done by paired t test.

Clinical parameters		Mean \pm Std. Deviation	P value
PI	Before	1.68\pm0.52	0.035 *
	After	1.42 \pm0.46	
GI	Before	1.60\pm0.49	0.003 **
	After	1.16\pm0.57	
PPD	Before	4.48\pm0.44	<0.001 **
	After	3.72\pm0.44	
CAL	Before	3.45\pm1.04	0.026 *
	After	2.89\pm0.90	

Discussion

The present study investigates and compares total WBC count in both groups and the effect of treatment (scaling and root planing) on total W.B.C. count in chronic periodontitis patients. The current study shows the beneficial effects of periodontal treatment (scaling and root planing) on total WBC count and clinical periodontal parameters (PI, GI, PPD, CAL).

The results of our study shows a highly significant difference in total leukocytes counts in patients with chronic periodontitis compared with healthy subjects. This difference in value is in accordance with the finding of Vaishali et al¹⁵, who reported that the mean value of leukocyte count in patients with chronic periodontitis was higher than control group. So it is evident that the leukocyte count in patients with chronic periodontitis is higher when compared with control group. This finding of the study confirmed the concept according to which stronger inflammation of the periodontium of subjects with chronic periodontitis could be the result of increased leukocyte count in peripheral blood, which is in agreement with the basic function of leukocytes in infection and inflammation.

Kweider¹⁶ has shown that periodontitis patients have significantly higher levels of fibrinogen and leukocytes when compared to control subjects. Inflammation of oral diseases such as periodontitis most likely influences the

circulatory total leukocyte counts considerably. Total leukocyte counts as indicators of inflammation at the same time show the association between oral disease, especially periodontitis, and systemic diseases in which the infection is an etiologic factor (cardiovascular diseases, especially myocardial infarction).¹⁷

Results showed that there is obvious decrease in WBC count after non- surgical therapy. Patients with higher levels of normal range of WBC count came to lower level of normal range of WBC count. This correlates with the finding of Gokhale et al,¹⁸ that The WBC count decreased after non-surgical treatment of generalised chronic periodontitis. As increase in WBC count can act as risk factor for many systemic diseases, so to avoid the risk of systemic disease, non-surgical therapy should be advocated.

Many authors reported that there was a statistically significant decrease in total WBC counts following nonsurgical periodontal therapy.¹⁹⁻²²

With the inflamed periodontal tissue, the cellular and molecular mechanisms are unified, so that such connections and consequences are not restricted to periodontal tissue only, they also cause systemic effects.²¹

Gingival index and Plaque indices were reduced post therapy, these are in agreement with the finding of Gokhale et al,¹⁸ which

showed reduction in plaque scores and gingival scores as the patient started maintaining oral hygiene after oral hygiene instructions and inflammation got subsided after non-surgical therapy.

Also, PPD and CAL were reduced after therapy; these correlate with the same results of Joseph and Janam²³ who evaluated the efficiency of periodontal treatment was confirmed by the significant reduction in probing pockets depth, clinical attachment loss, simplified oral hygiene index and papillary bleeding index.

The results of our study indicate that the group with healthy subjects has lower level of total leukocyte counts (within normal range) than the group of chronic periodontitis (within normal range). Also, our results revealed that the periodontal infection could be the cause of increased WBC count (within normal range) in systemically healthy patients. After treatment, a decrease in WBC count is seen. So, the patients chosen were systemically healthy with no other foci of infection other than chronic periodontal disease.

Conclusion

We concluded that the present study shows a highly significant difference between healthy subjects and chronic periodontitis patients in total WBC counts. The total W.B.C. counts decreased after non-surgical periodontal treatment as compared to before treatment.

Conflicts of interest

The authors reported no conflicts of interest.

Acknowledgement

We would like to thank the Department of Periodontology for their helping in completing this study. Also, we would like to thank the assistances in analyzing the data (Laboratory Centre in Hawler Teaching Hospital). In addition, we would like to thank a statistician who performed a statistical analysis of our results.

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