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Research Article

Periodontitis for Dental Health

# Correlative Assessment of Blood Parameters and Periodontal Status in Smokers- A Pilot Project

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**Abstract:** Platelets and leukocytes activated in response to periodontal microorganisms during bacteremia can go on to excite other cells, enhancing the likelihood of atherosclerosis and coronary artery disease. Reports also indicate that decrease in the number of erythrocytes may occur secondary to periodontal disease. Smoking is a known risk factor for periodontal disease by virtue of its adverse effects on the blood cells. It was therefore decided to investigate the relationship between the red blood cel l(RBC), white blood cell (WBC) and platelet counts and periodontal parameters among smokers and non-smokers; to subsequently assess any implication of the systemic effects. Blood counts(RBC, WBC, platelets) for the present study were obtained from peripheral venous blood of 126 patients , both male and female aged between 18-45 years, in each group as follows: GROUP I :Periodontally healthy (n = 42) (non-smokers) GROUP 2: Chronic periodontitis (n = 42)( non-smokers) GROUP 3: Chronic periodontitis (n = 42) ( smokers) The clinical parameters to assess periodontitis included plaque index(PI), bleeding index(BI) and Clinical Attachment Loss( CAL) and history of smoking was elicited based on amount, frequency and duration of smoking. ANOVA tests followed by post HOC –Tukey HSD test revealed that there were significant differences between the 3 groups with respect to PI, BI and RBC counts($p<0.05$). Pearson correlation test revealed a significant correlation between plaque index and platelet counts in group I only($p<0.05$). Additionally, a significant correlation was also observed between WBC and platelet counts in all three groups($p<0.05$).Variations in blood counts in different periodontal situations may pose a risk of systemic inflammation in smokers and non- smokers.

**Keywords:** RBC counts, WBC counts, platelet counts, smoking, periodontal disease

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## 1. INTRODUCTION

Periodontal medicine has gained tremendous attention by researchers worldwide with a lot of focus on the possible association between oral health and systemic diseases with contrasting reports.[1-4] The association between periodontal disease (PD) and systemic health has been emphasized recently, based on inflammatory changes in periodontal tissues caused by bacteria from the oral biofilm. Therefore, a stronger association has been noticed between periodontitis as a risk factor and systemic diseases. The importance of the association between periodontal disease as local infection and systemic diseases requires further investigations and opens new possibilities for an old concept of "focal infection" [5,6] However, the mechanism linking periodontal disease to atherosclerosis and coronary artery disease is not yet clearly understood. Chronic periodontitis is an infection of the tissues supporting the teeth caused by a complex variety of gram negative anaerobic bacteria [7,8]Increase in total leukocyte and neutrophil counts in patients with chronic periodontitis, especially its severe form, can be an indicator of the possible exposure of the body to some systemic disease [9] and might be considered one of the mechanisms that explain how periodontitis is linked to the development of atherosclerosis and cardiovascular disease. [10] It has been hypothesized that platelets and leukocytes may be more sensitive to stimulation by periodontal pathogens [11] and that activated platelets and leukocytes might contribute to the increased atherothrombotic activity.[7, 12-15] Studies have shown that patients suffering from chronic periodontitis have a lower number of erythrocytes and hemoglobin compared to healthy controls. Thus, based on these results it can be concluded that, like any other chronic condition, chronic periodontitis can lead to anemia. [8,16] Tobacco smoking is one of the largest public health threats that face the world killing around 6 million people every year. It has been practiced by over one billion people all over the world, most of them in the developing world. [17] Long term exposure to smoking materials has a direct relation with the periodontal disease; prevalence and severity[18]. It has been reported that smokers are 4-times more likely than nonsmokers to develop periodontitis and smoking is considered the main avoidable risk factor for development and progression of periodontal diseases. [19-21] Effects of smoking on alterations of hemostatic and fibrinolytic system, antioxidant status and hematology parameters have been extensively studied.[22]Smoking can affect the health of periodontal conditions[23] as well as circulating leukocytes and erythrocytes. Concerning the effect of smoking on platelet count, there are only a few reports on this topic. From a cohort study of Green and colleagues, the effect of smoking on the platelet count is still controversial.[24] Keeping this in mind, it was decided to assess and evaluate a correlation, if any, in the periodontal status and the blood parameters of smokers and compare them with the same in nonsmokers and healthy persons.

## 2. MATERIALS AND METHODS

After obtaining the ethical approval from the institution, (H-04-12122018), this cross-sectional study was carried out at dental clinics of IbnSina National College of Medical Sciences, Jeddah, Kingdom of Saudi Arabia throughout the period January-March, 2020. A convenience sample of 126 adults i.e., aged over 18 years (both males and females) was chosen based on the following criteria:

### *2.1. Inclusion Criteria*

- The number of teeth present ≥ 20
- Patients with healthy periodontium /chronic gingivitis(group 1)
- Patients with chronic periodontitis (localised/generalised) (group 2)
- Patients with chronic periodontitis and a history of smoking (group 3)

### *2.2 Exclusion Criteria*

- Presence of any systemic or debilitating diseases
- Recent history or presence of any acute or chronic infections
- Patients with history of any drug intake including antibiotics, analgesics or any other drugs 3 months prior to the study
- Patients who have undergone periodontal therapy in the last 6 months
- Patients who are physically or mentally challenged
- Patients with a history of smoking(group 1&2)

When presented to the dental clinic, demographic data and smoking history were obtained and periodontal examination included clinical attachment loss (CAL) to ascertain periodontal status, gingival bleeding index (BI) and plaque index (PI). Following this, the patients were divided into three separate groups as follows:

**-Group I: included 42 non-smoker adults with clinically healthy periodontium/ chronic gingivitis**
**-Group II: included 42 non- smoker patients with chronic periodontitis**
**-Group III: Included 42 smoker patients with chronic periodontitis**

All the participants were explained the nature of the study and after obtaining consent for the same; were referred to proceed for blood investigations; namely white blood cell count, red blood cell count and platelet count. The patients underwent laboratory tests at various centers under the ministry of health, and from Azizia general medical complex, Jeddah city, KSA.

## 3. STATISTICAL ANALYSIS

The collected data was entered into an excel sheet and then analysed using SPSSV22 software with ANOVA test for comparisons between groups and Post hoc test – Tukey HSD to analyse the significance in the groups. Pearson correlation was done to find any association between periodontal parameters and blood counts in the various groups and also among the blood counts. A 'P' value of < 0.05 was considered statistically significant.

## 4. RESULTS

A total of 126 patients were included in the study which

comprised 59 females (46.8%) and 67 males (53.2%). Of these, group 1 consisted of 27 females and 15 males; group 2 consisted of 23 females and 19 males and group 3 consisted of 9 females and 33 males(TABLE 1). The mean age of the patients of group 1 was 27.26±7.506 ranging from 18-47 years; group 2 was 28.29±5.782 ranging from 19-47 years and group 3 was 36.50±9.290 ranging from 19-55 years(TABLE 2). ANOVA test comparing the clinical periodontal parameters with blood parameters revealed significant changes in plaque index, bleeding index and RBC counts among the 3 groups(P<0.05). However, no significance was observed in WBC counts and platelet counts among the 3 groups(p>0.05) (TABLE 3). Post HOC – Tukey HSD test revealed significant differences between group 1,2 and 3 with regard to plaque index(p<0.05), between group 1 and 2&3(p<0.05) but not between group 2 and 3(p>0.05) with regard to bleeding index; and between group 3 and 1&2 (p<0.05) but not between group 1 and 2(p>0.05) with regard to RBC counts. As already observed in table 3, no significant differences were observed in WBC and platelet counts between the 3 groups(p>0.05)(TABLE 4). Pearson correlation of plaque index with the blood parameters showed significant negative correlation of plaque index with platelet counts in group 1 only(p<0.05). No significant correlation was seen in any other groups with any of the blood counts(p>0.05)(TABLE 5). Similarly, no significant correlation was seen with regard to bleeding index and any of the blood counts among the various groups (p>0.05) (TABLE 6). Correlation assessment of RBC counts with WBC and platelet counts showed no significant correlation between them in all three groups(p>0.05)(TABLE 7). However,a significant positive correlation was observed between WBC counts and platelet counts in all three groups (p<0.05) (TABLE 8).

**Table 1: Frequency distribution**

| | | | Group 1 | Group 2 | Group 3 | Total |
|---|---|---|---|---|---|---|
| Gender | Female | Count | 27 | 23 | 9 | 59 |
| | | % | 64.3% | 54.8% | 21.4% | 46.8% |
| | Male | Count | 15 | 19 | 33 | 67 |
| | | % | 35.7% | 45.2% | 78.6% | 53.2% |

**Table 2: Descriptive statistics (AGE)**

| | N | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|---|
| Group 1 | 42 | 18 | 47 | 27.26 | 7.506 |
| Group 2 | 42 | 19 | 47 | 28.29 | 5.782 |
| Group 3 | 42 | 19 | 55 | 36.50 | 9.290 |

**Table 3: Comparison of clinical periodontal parameters and blood parameters in the 3 groups using ANOVA**

| Variable | | Group 1 | Group 1 | Group 1 | AVOVA(F) | P-value |
|---|---|---|---|---|---|---|
| Plaque index | Mean | 0.25543 | 0.46902 | 0.59893 | 28.404 | 0.00 |
| | Std. Deviation | 0.220483 | 0.206427 | 0.205472 | | |
| Bleeding index | Mean | 0.09698 | 0.2526 | 0.22543 | 23.777 | 0.00 |
| | Std. Deviation | 0.097087 | 0.153986 | 0.058954 | | |
| RBC counts | Mean | 5.131 | 5.2729 | 5.6874 | 7.179 | 0.001 |
| | Std. Deviation | 0.4177 | 0.75905 | 0.84652 | | |
| WBC counts | Mean | 8.1479 | 8.39 | 8.2936 | 0.09 | 0.914 |
| | Std. Deviation | 2.34078 | 2.25957 | 3.21248 | | |
| Platelet counts | Mean | 261.14 | 290.88 | 270.14 | 1.82 | 0.166 |
| | Std. Deviation | 69.579 | 69.507 | 80.157 | | |

**Table 4: Multiple comparison (Post HOC test – Tukey HSD)**

| Dependent Variable | (I) group | (J) group | Mean Difference (I-J) | Sig. |
|---|---|---|---|---|
| Plaque index | 1.00 | 2.00 | -.213595* | .000 |
| | | 3.00 | -.343500* | .000 |
| | 2.00 | 1.00 | .213595* | .000 |
| | | 3.00 | -.129905* | .015 |
| | 3.00 | 1.00 | .343500* | .000 |
| | | 2.00 | .129905* | .015 |
| Bleeding index | 1.00 | 2.00 | -.155619* | .000 |
| | | 3.00 | -.128452* | .000 |
| | 2.00 | 1.00 | .155619* | .000 |
| | | 3.00 | .027167 | .499 |
| | 3.00 | 1.00 | .128452* | .000 |
| | | 2.00 | -.027167 | .499 |
| RBC counts | 1.00 | 2.00 | -.14190 | .623 |

| | | | | |
|---|---|---|---|---|
| | | 3.00 | -.55643* | .001 |
| | 2.00 | 1.00 | .14190 | .623 |
| | | 3.00 | -.41452* | .021 |
| | 3.00 | 1.00 | .55643* | .001 |
| | | 2.00 | .41452* | .021 |
| WBC counts | 1.00 | 2.00 | -.24214 | .907 |
| | | 3.00 | -.14571 | .965 |
| | 2.00 | 1.00 | .24214 | .907 |
| | | 3.00 | .09643 | .985 |
| | 3.00 | 1.00 | .14571 | .965 |
| | | 2.00 | -.09643 | .985 |
| Platelet counts | 1.00 | 2.00 | -29.738 | .155 |
| | | 3.00 | -9.000 | .840 |
| | 2.00 | 1.00 | 29.738 | .155 |
| | | 3.00 | 20.738 | .399 |
| | 3.00 | 1.00 | 9.000 | .840 |
| | | 2.00 | -20.738 | .399 |

**Table 5: Pearson Correlation of Plaque Index with different Blood parameters**

| Group | Statistics | Blood parameters | | |
|---|---|---|---|---|
| | | RBC counts | WBC counts | Platelet counts |
| **Group 1** | Pearson Correlation | -.014 | -.186 | -.339* |
| | Sig. (2-tailed) | .932 | .239 | .028 |
| **Group 2** | Pearson Correlation | .224 | -.167 | .098 |
| | Sig. (2-tailed) | .154 | .292 | .535 |
| **Group 3** | Pearson Correlation | .189 | .059 | -.209 |
| | Sig. (2-tailed) | .229 | .709 | .183 |

***. Correlation is significant at the 0.05 level (2-tailed).***

**Table 6: Pearson Correlation of Bleeding Index with different Blood parameters**

| Group | Statistics | Blood parameters | | |
|---|---|---|---|---|
| | | RBC counts | WBC counts | Platelet counts |
| **Group 1** | Pearson Correlation | -.201 | -.162 | -.236 |
| | Sig. (2-tailed) | .203 | .305 | .133 |
| **Group 2** | Pearson Correlation | .191 | .132 | .041 |
| | Sig. (2-tailed) | .226 | .406 | .798 |
| **Group 3** | Pearson Correlation | .271 | .080 | -.097 |
| | Sig. (2-tailed) | .083 | .613 | .539 |

**Table 7: Pearson Correlation of RBC count with different Blood parameters**

| Group | Statistics | Blood parameters | |
|---|---|---|---|
| | | WBC counts | Platelet counts |
| **Healthy** | Pearson Correlation | .255 | .288 |
| | Sig. (2-tailed) | .104 | .146 |
| **Non Smokers** | Pearson Correlation | -.005 | .188 |
| | Sig. (2-tailed) | .974 | .232 |
| **Smokers** | Pearson Correlation | .290 | .253 |
| | Sig. (2-tailed) | .062 | .107 |

**Table 8: Pearson Correlation of WBC count with Platelet counts**

| Group | Statistics | Blood parameters |
|---|---|---|
| | | Platelet counts |
| **Healthy** | Pearson Correlation | .470** |
| | Sig. (2-tailed) | .002 |
| **Non Smokers** | Pearson Correlation | .585** |
| | Sig. (2-tailed) | .000 |
| **Smokers** | Pearson Correlation | .490** |
| | Sig. (2-tailed) | .001 |

***Correlation is significant at the 0.01 level (2-tailed).***

## 5. DISCUSSION

Evidence of an association between decrease in red blood cell parameters with increase in the severity of periodontal destruction has been demonstrated by many researchers.[8,25,26] In addition, a number of studies have demonstrated elevation of total leukocyte count in periodontitis.[7,27] Such an increased leukocyte count in periodontitis may carry a high risk of coronary heart disease because there are several plausible mechanisms by which the WBCs can promote atherosclerosis, thrombosis, and myocardial ischemia [11,28,29] Previous studies have also suggested an increase in platelet levels and activation in periodontitis patients.[14] It has been demonstrated that dental plaque bacteria, including the periodontal pathogen Porphyromonasgingivalis, induces platelet activation and aggregation.[30] Continuous cigarette smoking has been shown to produce severe adverse effects on haematological parameters (e.g., hemoglobin, white blood cells count, mean corpuscular volume, mean corpuscular hemoglobin concentration, red blood cells count and hematocrit)[22]. Smoking may also contribute to the incidence of atherosclerosis, as well as acute complications, especially thrombosis . It is believed that disturbances of platelet function, especially aggregation, is the essential mechanism responsible for this pathology. However, the effect of smoking on the quantity of platelets might be another contributing factor.[31,32] With such diverse evidence, this pilot project was conducted to understand the correlations between the clinical periodontal parameters and blood cell counts in different periodontal conditions. Additionally, it was also decided to assess whether a history of smoking may have an impact on these correlations since smoking is an established risk factor for both periodontal disease and hematologic parameters. To the knowledge of the authors, this is one of the few studies correlating clinical periodontal parameters and the three blood counts in patients with healthy periodontium, periodontitis and smokers with periodontitis patients. The study group(n=146) consisted of nearly equal distribution of male(53.2%) and female(46.8%) patients with the smokers with periodontitis group comprising a larger number of males(n=33). The average age of the participants in all three groups was around 27-28 years, with the smokers with periodontitis group having a slightly higher mean age(36.5 years). ANOVA test comparison followed by post HOC Tukey test of the three groups with respect to clinical periodontal parameters and blood parameters showed that there were significant differences between all 3 groups with plaque index, bleeding index and RBC counts. Highly significant differences were observed with plaque index and bleeding index in the periodontally healthy group compared to the periodontitis group and smokers with periodontitis group and also between the periodontitis group and smokers with periodontitis group. This is an obvious finding since patients with little or no plaque accumulation have a lower chance of inflammation which translates to little or no bleeding and hence havehealthy periodontal tissues. On the contrary, higher plaque scores directly relate to a higher risk of inflammation, bleeding and periodontitis as was observed in the periodontitis group. Higher plaque scores have been reported in smokers especially due to the tar deposits on the teeth surfaces favoring increased plaque deposition. However, low bleeding scores have been reported in smokers because of the vasoconstriction of blood vessels which has been observed in the mean bleeding scores in smokers with periodontitis group. There were significant differences between the RBC counts of non-smokers(healthy periodontium group and periodontitis group) and smokers(with periodontitis). This is in accordance with the findings of Lakshmi et al [33] who concluded that among the smokers the RBC count was significantly increased as the intensity of smoking increases. Whitehead et al. in their study observed that hemoglobin concentration and hematocrit was significantly increased in those smoking more than 10 cigarettes per day[34]. Increased number of erythrocytes and values of hematocrit in smokers can be explained by the fact that tissue hypoxia caused by increased creation of carboxyhemoglobin leads to increased secretion of erythropoietin, thus increasing erythropoiesis. Carbon monoxide from tobacco smoke also leads to an increase in permeability of the capillaries which decreases the volume of plasma, which finally mimics the condition of polycythemia, characterized by an increased share of the erythrocytes in the blood volume, which is reflected also through increased values of hematocrit.[35,36]Malenica et alalso concluded that continuous cigarette smoking increases erythrocyte count, hemoglobin concentration, hematocrit, leukocyte count, mean corpuscular volume and mean corpuscular hemoglobin concentration and these alterations might be associated with a greater risk for developing atherosclerosis, polycythemia vera, chronic obstructive pulmonary disease and/or cardiovascular diseases. [22] There were no significant differences between the three groups with regard to WBC and platelet counts. This is in contrast to the findings of Alrasheed et al [10] who concluded that chronic periodontitis may elevate WBC and platelet counts compared to healthy control patients and might be considered one of the mechanisms that explain how periodontitis is linked to the development of atherosclerosis and cardiovascular disease and also Melancia et al[22] who concluded that continuous smoking increases leukocyte counts. However, this is in accordance with the findings of Suwansaksri et al 2004 [37] who found no difference between the platelet counts of smokers and non-smokers. On the other hand, Pearson correlation analysis revealed significant negative correlation of plaque index with the platelet counts in the periodontally healthy group only, thereby suggesting low plaque scores and little or no periodontal inflammation seen in this group are associated with higher platelet counts, which again is in contrast to the findings of Al Rasheed et al [10] who concluded otherwise. However, no correlation was observed between the plaque scores and RBC and WBC counts and also between bleeding scores and the blood cell counts in all the groups, thus contradicting the findings of several researchers as mentioned above. Additionally correlation analysis of RBC counts with WBC and platelet counts was inconclusive as no statistical significance was observed among the three groups. However, a significant correlation was observed between WBC counts and platelet counts in all three groups thereby suggesting that irrespective of the periodontal and smoking status, these two blood parameters increase or decrease in accordance with each other and without any association with the RBC counts. The exact reason for this association is not very clear but increase in WBC and platelet counts have been reported in chronic periodontal disease [10] and also in smokers.[38] Our study found higher mean WBC and platelet counts in periodontitis group followed by smokers and healthy periodontium group. Periodontitis with all its clinical symptoms and consequences can pose a potential risk of systemic exposure to inflammatory stress with variations in blood cell counts and thus may adversely affect the systemic

status of the patient. As suggested in all previous studies, blood parameters may be altered either by periodontal infection or smoking or even a combination of both of them; and this in turn, may pose a risk of systemic disease such as atherosclerosis and coronary heart disease among others.

## 6. CONCLUSION

Thus the results of our study though not in keeping with some researchers, do suggest that periodontal inflammation alone and/or in combination with smoking may increase the risk of systemic disease by virtue of changes in the blood parameters. The exact nature of the systemic inflammatory response evoked is still unclear as also whether reduction in periodontal inflammatoryresponse would consequently diminish this risk. The obvious next step in this investigation would be to monitor the effects of periodontal treatment and smoking cessation on the blood cell counts which has been planned as the next leg of this project.

## 7. AUTHORS CONTRIBUTION STATEMENT

Dr Shreya Shetty conceptualized and designed the study and collected relevant literature about the same. She was also the main person responsible for final drafting and completion of the manuscript. Dr.Mawadah, Dr.Halah, DrMazen, Dr.Jumanah and Dr.Shorooq were mainly responsible for collection of data, compilation and initial drafting of the manuscript. Ms.Khammarunissa performed the statistical analysis and necessary inputs were given towards the designing of the manuscript with regard to results and discussion. Overall, all authors participated in the methodology and final results and contributed to the final manuscript.

## 8. CONFLICT OF INTEREST

Conflict of interest declared none

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