Research Article

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Comprehensive Assessment of Fluctuating Blood Platelet Count in Patients with Chronic Periodontal Diseases: An Original Research Study

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ABSTRACT

Background: As we all are aware that blood platelet count is not very much stable especially in known cases of chronic Periodontitis. Therefore, the sole aim of this study was to evaluate the fluctuating blood platelet count in patients with chronic periodontal diseases.

Materials & Methods: All patients were selected from the department of Periodontology of the institute. Total 120 patients were planned to be studied wherein 60 were healthy (control) while rest 60 patients were suffering from chronic Periodontitis. Periodontal health parameters like plaque index, pocket depth, attachments levels etc. were evaluated and compared to draw final inference.

Results: Statistical analysis was done by statistical software 'Statistical Package for the Social Sciences (SPSS)'. We have ensured the maximum accuracy of results by using appropriate statistical tests that have provided p values, mean, standard deviation, standard error an 95% CI. $P \leq 0.05$ was considered as statistically significant. Out of 120 studied patients, males were 73 and females were 47. P value was significant for the age range 31-35 years.

Conclusion: Within the limitations of the study authors concluded that platelet count was reported to be raised in patients with chronic periodontitis as compared to control group. These findings may be logically correlated with the underlying inflammatory mechanisms and reaction those occurring in the patients with chronic Periodontitis.

Keywords: Periodontitis, Platelet, Inflammation, Gingiva.

INTRODUCTION

Periodontitis is a chronic inflammatory disorder characterized by the devastation of jaw bone, and the subsequent loss of the surrounding connective tissue those support the teeth. Common eliciting factors of inflammation are smoking, diabetes and obesity.[1] Literature has also evidenced that

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periodontitis is a chronic infectious disease of the surrounding tissues of the teeth.[2-5] By the year 2022, non communicable disorders are acquiring an epidemic outline and will be apparently a major cause of life loss in developing countries.[6] Furthermore, studies performed in India have illustrated that every second individual above 36 years of age has periodontal pockets and 31% of total teeth extracted after 36 years of age are due to periodontal dilemmas. Conversely, periodontal pocket is pathologically depended gingival sulcus due to miscellaneous reasons. Periodontitis is one of the most common infections reported in humans, distressing in its most brutal form, roughly 10% of the populace.[7-10] Inflammatory processes of periodontal tissues results in periodontal pocket creation and ulceration of the epithelial layering. Since few decades, blood has been extensively employed as first line of diagnostic tool for estimating the condition of different infections and systemic diseases. Platelets are tiny blood cells that help your body form clots to stop bleeding. If one of your blood vessels gets damaged, it sends out signals that are picked up by platelets. The platelets then rush to the site of damage and form a plug, or clot, to repair the damage. As reported by most of the literature and textbooks, rise in the number of platelets and coupled platelet activation is one of the characteristics of systemic inflammation.[11-13] In addition, it is well known that systemic effects of periodontal diseases are usually initiated by bacteremia arising from pathologic periodontal tissues and the resultant inflammatory reactions. Moreover periodontitis has been frequently linked with higher numbers of platelets. Additionally, few pioneer researchers have done studies and shown that blood platelet counts usually decrease after periodontal therapy. Therefore, it is now becoming clearer that a simultaneous reverse association also exists, i.e., periodontal disease negatively affects systemic health too. This actually potentially led us to think that there could be imperative effect of periodontal diseases on a wide range of organ systems including hematological and lymphatic systems.[14-16] The ultimate aim of this study was to critically evaluate the fluctuating blood platelet count in patients with chronic periodontal diseases.

MATERIALS & METHODS

The present study was conducted in the department of Periodontology of the institute. Total 120 patients were planned to be studied wherein 60 were healthy (control) while rest 60 patients were suffering from chronic periodontitis. Informed consents were obtained from patients prior to the real execution of the study. Patients suffering from any physical or psychological diseases, pregnant women, smokers and those who received any periodontal treatment within 6 months, were excluded from the study. Patients with any congenital/acquired gross anomaly related to maxillofacial structure were also excluded from the study. The methodology and planned way of study's conduction were explained to the patients. All patients were also informed about the importance and clinical relevance of the study. We had selected total 120 patients inclusive of both sexes, aged between 26 and 50 years. Patient selection process was completed using randomized controlled sampling procedure. Comprehensive periodontal screening was performed for all selected patients. Patients were studied into two groups (of 60 each) based on the periodontal examination results. Group I were healthy patients (control) while Group II patients were known case of chronic periodontitis. Periodontal screening procedure has included estimation of Plaque Index, Gingival Index, Pocket Depth and Clinical Attachment Level. Group I patients showed periodontal pocket depth less than 3 mm, Clinical attachment loss less than 3 mm, with no clinical sign of gingivitis. Group II patients showed more than 3 mm of pocket depth and clinical attachment loss more than 3 mm. Minimum 2 ml of venous blood sample were drawn by venipuncture method in the antecubital fossa and stored in vials containing Ethylene Diamine Tetraacetic Acid [EDTA]. Furthermore, the platelet counts were completed by using an automated cell counter within 24 hours after sample collection, at the hematology laboratory of the institute. Results thus obtained was tabulated and subjected to basic statistical analysis. P value less than 0.05 was considered significant ($p< 0.05$).

RESULTS

All gathered data were sent for statistical analysis using statistical software Statistical Package for the

Social Sciences version 21 (IBM Inc., Armonk, New York, USA). The data was accurately subjected to suitable statistical tests to obtain p values, mean, standard deviation, chi- square test, standard error and 95% CI. Table 1 and Graph 1 shows that out of 120 studied patients, males were 73 and females were 47. Total 15 patients were in the last age group. Total 28 patients were belonging to the age range of 31-35 years. 23 studied patients were falling in the age range of 36-40 years. P value was significant for the age range 31-35 years [0.01]. Table 2 illustrates the basic statistical evaluation of measured platelet count between groups including level of significance evaluation using 'Pearson Chi-Square Test'. The overall level of significance was 'non significant' [0.07]. Table 2 also shows that the mean platelet count in the patients with chronic periodontitis (Group II: 267.39 cells/L) is higher as compared to the control group (Group I: 227.87 cells/L).

Table 1: Age & gender wise distribution of patients.

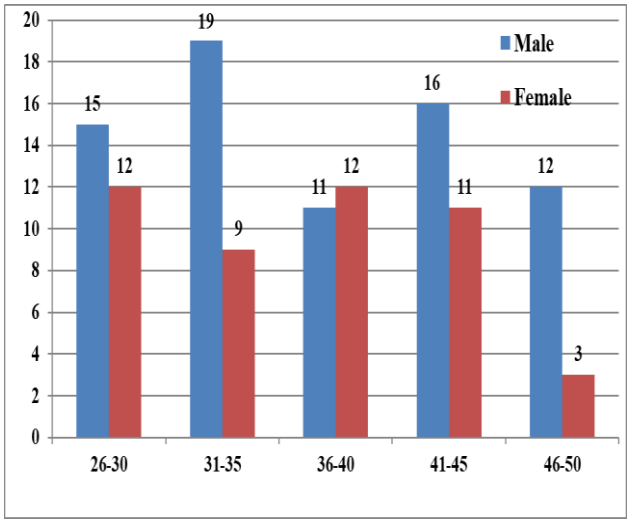
Age Group (Yrs)	Male	Female	Total %	P value
26-30	15	12	27 [22.5 %]	0.09
31-35	19	9	28 [23.3 %]	0.01*
36-40	11	12	23 [19.1 %]	0.70
41-45	16	11	27 [22.5 %]	0.08
46-50	12	3	15 [12.5 %]	0.09
Total	73	47	120 [100%]	***Significant**

***p<0.05 significant**

Table 2: Statistical evaluation of measured platelet count between groups.

Group	N	Platelet count ($X10^9$/L)					P value
		Mean	Standard Deviation	Std. Error	95% CI	Pearson Chi-Square Value	
Group I (Control)	60	227.87	58.64	0.029	1.96	1.0	0.07
Group II (Periodontitis)	60	267.39	54.31	0.084	2.45	1.0	
Total	120	-	-	-	-	-	Non Significant

Graph 1: age & gender wise allocation of patients.



DISCUSSION

Saliva, gingival crevicular fluid and tissue lining of oral mucus membrane usually provide the first line of defense. They act particularly for oral cavity and the periodontium. In case of any defect or breach in it, the cellular and molecular elements of the innate immune response are triggered.[17-19] Correct identification of pathologic microorganisms is the sole key to effective innate immunity. Chronic periodontitis is a common infection that manifests as local inflammation affecting the supporting tissues of the teeth. Literature has well evidenced that periodontitis, apart from creating a local inflammatory process, can also put forth a wide range of systemic effects.[20-22] Smoking is considered

as the strongest predictor of attachment loss and bone loss. Several studies showed that the nicotine found in tobacco products initiates the overproduction of cytokines in the body due to lowered oxygen levels.[23-24] Cytokines are chief chemicals mediators those involved in the process of periodontal inflammation. Apart from the cytokine action, several changes in the blood occur including increased leukocyte count and reactive oxidants. Reactive oxidants appear to be induced not only by smoking but also by periodontitis. So in present study we had excluded patients who were smokers. A total hematological analysis is frequently used to assess the presence of systemic infection or inflammation, and the question arises whether periodontal infections can also affect the hematological parameters such as the differential counts of white blood cells, red blood cells, and platelets count.[25] Some of the studies have demonstrated differences in the hematological parameters in subjects with chronic periodontitis as compared to healthy subjects. Additionally, we noticed few studies those were in agreement with our result outcomes. Kalsi DS and associates evaluated the effect of infective-inflammatory periodontal disease and its treatment on commonly assessed blood parameters.[26] They concluded that chronic periodontitis has a considerable effect on the common blood parameters. Gokhale SR and colleagues attempted to find any correlation between signs of anemia and chronic periodontitis.[27] Results of the present study showed that patients suffering from chronic periodontitis have a lower number of erythrocytes and hemoglobin compared to healthy controls. They have also postulated that platelets count could possibly be higher in such circumstances. Rasheed A has also estimated the white blood cell and platelet counts in chronic periodontitis patients.[28] His study also reported higher levels of white blood cells and platelets in periodontitis patients compared to healthy control patients. Therefore these findings were in accordance with our results. Activated platelets regulate chemokine release by the monocytes in inflammatory lesions. Platelets are essential for primary hemostasis and endothelial repair, but also play a key role in atherogenesis and thrombus formation. Platelet count has been associated with vascular and non-vascular death and a recent meta-analysis showed that mean platelet volume is a predictor of cardiovascular risk.

Our study aimed to evaluate the fluctuating blood platelet count in patients with chronic periodontal diseases. We found that the mean platelet count in the patients with chronic periodontitis (Group II: 267.39 cells/L) is higher as compared to the control group (Group I: 227.87 cells/L). Therefore, these findings could be logically correlated with the underlying inflammatory mechanisms and reaction those occurring in the patients with chronic periodontitis.

CONCLUSION

Within the limitations of the study authors concluded that platelet count was reported to be raised in patients with chronic periodontitis as compared to control group. Therefore, it could possibly be inferred that there is a clear-cut effect of periodontal disease and its treatment on blood platelet count. These findings may be logically correlated with the underlying inflammatory mechanisms and reaction those occurring in the patients with chronic periodontitis. However, we expect few other large scale studies to be conducted that could further establish some genuine guidelines in these perspectives.

CONFLICT OF INTEREST

No potential academic conflict of interest relevant to the above article was reported.

REFERENCES

1. Forner L, Larsen T, Kilian M, Holmstrup P. Incidence of bacteremia after chewing, tooth brushing and scaling in individuals with periodontal inflammation. J Clin Periodontol 2006;33:401–7.

2. D'Aiuto F, Parkar M, Nibali L, Suvan J, Lessem J, Tonetti MS. Periodontal infections cause changes in traditional and novel cardiovascular risk factors: results from a randomized controlled clinical trial. Am Heart J 2006;151:977–84.

3. Loos BG. Systemic markers of inflammation in periodontitis. J Periodontol 2005;76:2106–15.

4. Paraskevas S, Huizinga JD, Loos BG. A systematic review and metaanalyses on C-

reactive protein in relation to periodontitis. J Clin Periodontol 2008;35(4):277–90.

5. Klinger MH, Jelkmann W. Role of blood platelets in infection and inflammation. J Interferon Cytokine Res 2002;22:913–22.

6. Wakai K, Kawamura T, Umemura O, et al. Associations of medical status and physical fitness with periodontal disease. J Clin Periodontol 1999;26:664–72.

7. Christan C, Dietrich T, Hagewald S, Kage A, Bernimoulin JP. White blood cell count in generalized aggressive periodontitis after nonsurgical therapy. J Clin Periodontol 2002;29:201–6.

8. Herzberg MC, Weyer MW. Dental plaque, platelets, and cardiovascular diseases. Ann Periodontol 1998;3:151–60.

9. Lourbakos A, Yuan Y, Jenkins AL, et al. Activation of proteaseactivated receptors by gingipains from Porphyromonas gingivalis leads to platelet aggregation: a new trait in microbial pathogenicity. Blood 2001;97:3790–7.

10. Weyrich AS, Lindemann S, Tolley ND, et al. Change in protein phenotype without a nucleus: translational control in platelets. Semin Thromb Hemost 2004;30:491–8.

11. Kamath S, Blann AD, Lip GY. Platelet activation: assessment and quantification. Eur Heart J 2001;22:1561– 71.

12. Janket SJ, Baird AE, Chuang SK, Jones JA. Meta-analysis of periodontal disease and risk of coronary heart disease and stroke. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95:559–69.

13. Tonetti MS, D'Aiuto F, Nibali L, et al. Treatment of periodontitis and endothelial function. N Engl J Med 2007;356:911–20.

14. Pussinen PJ, Tuomisto K, Jousilahti P, Havulinna AS, Sundvall J, Salomaa V. Endotoxemia, immune response to periodontal pathogens, and systemic inflammation associate with incident cardiovascular disease events. Arterioscler Thromb Vasc Biol 2007;27:1433–9.

15. Pussinen PJ, Alfthan G, Jousilahti P, Paju S, Tuomilehto J. Systemic exposure to Porphyromonas gingivalis predicts incident stroke. Atherosclerosis 2007;193:222–8.

16. Holmlund A, Holm G, Lind L. Severity of periodontal disease and number of remaining teeth are related to the prevalence of myocardial infarction and hypertension in a study based on 4,254 subjects. J Periodontol 2006;77:1173–8.

17. Mustapha IZ, Debrey S, Oladubu M, Ugarte R. Markers of systemic bacterial exposure in periodontal disease and cardiovascular disease risk: a systematic review and meta-analysis. J Periodontol 2007;78:2289–302.

18. Bizzarro S, van der Velden U, ten Heggeler JM, et al. Periodontitis is characterized by elevated PAI-1 activity. J Clin Periodontol 2007;34:574–80.

19. Schonbeck U, Mach F, Libby P. CD154 (CD40 ligand). Int J Biochem Cell Biol 2000;32:687–93.

20. Gawaz M. Role of platelets in coronary thrombosis and reperfusion of ischemic myocardium. Cardiovasc Res 2004;61:498–511.

21. Freedman JE. CD40-CD40L and platelet function: beyond hemostasis. Circ Res 2003;92:944–6.

22. Yamamoto T, Tsuneishi M, Furuta M, Ekuni D, Morita M, Hirata Y. Relationship between decrease of erythrocyte count and progression of periodontal disease in a rural Japanese population. J Periodontol 2011;82:106-13.

23. Banthia R, Jain P, Banthia P, Belludi S, Parwani S, Jain A. Effect of phase I periodontal therapy on pro-coagulant state in chronic periodontitis patients – A clinical and haematological study. J Ir Dent Assoc 2013;59:183–8.

24. Rastogi P, Singhal R, Sethi A, Agarwal A, Singh VK, Sethi R. Assessment of the effect of periodontal treatment in patients with

coronary artery disease: A pilot survey. J Cardiovasc Dis Res 2012;3:124–7.

25. Holmes MB, Sobel BE, Howard DB, Schneider DJ. Differences between activation thresholds for platelet P-selectin and glycoprotein IIb–IIIa expression and their clinical implications. Thromb Res 1999;95:75–82.

26. Kalsi DS, Sood A, Mundi S, Sharma V. Effect of scaling and root planing on blood counts in patients with chronic generalized periodontitis. Indian J Dent Sci 2017;9:109-13.

27. Gokhale SR, Sumanth S, Padhye AM. Evaluation of blood parameters in patients with chronic periodontitis for signs of anemia. J Periodontol 2010;81:1202-6.

28. Al-Rasheed A. Elevation of white blood cells and platelet counts in patients having chronic periodontitis. Saudi Dent J 2012;24:17–2.