

EFFECT OF NON-SURGICAL PERIODONTAL THERAPY ON PLATELET INDICES AND LEUCOCYTE COUNT IN CHRONIC PERIODONTITIS SUBJECTS



Dental Science

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ABSTRACT

Blood platelets are a key element linking the processes of hemostasis, inflammation and tissue repair. The role of platelet parameters like mean platelet ratio (MPR), platelet distribution width (PDW) and Platelet crit (PCT) in chronic periodontitis (CP) are not extensively documented. The objective of this study is to evaluate the effect of nonsurgical periodontal therapy (NSPT) on various platelet parameters and leukocytes in patients with periodontitis. 180 subjects were divided into 60 healthy (H), 60 CP and 60 CP subjects recalled after 1 month of NSPT. The present study suggests that all the platelet indices were related with inflammatory status of patients with severe periodontitis.

KEYWORDS

Leukocytes, Platelet Indices, Non Surgical Periodontal Therapy, Chronic Periodontitis

INTRODUCTION

Periodontitis is a chronic inflammatory disease of the supporting structures of tooth¹. Blood platelets are a key element linking the processes of hemostasis, inflammation and tissue repair. Within seconds, platelets respond to an tissue injury or invasion by pathogens through generation of specific signals². Platelet indices have been widely studied as a diagnostic marker to monitor the disease activity. Important platelet parameters - platelet count (PC) which is a part of complete blood cell count³. Platelet crit (PCT), provides data about platelet mass. Platelet distribution width (PDW) reflects variation of platelet size and distribution⁴.

The total number of leukocytes in peripheral blood is used as a diagnostic measure to investigate an infection or inflammatory disease⁵. It has been hypothesized that platelets and leukocytes may be more sensitive to stimulation by periodontal pathogens and might contribute to increased atherothrombotic activity. Mean platelet volume (MPV) and red blood cell distribution width (RDW) are considered as novel indices for inflammation⁶. MPV value was found to be change during inflammation because of the secretion of various cytokines and hormones affecting the proliferation and activation of the platelets⁷. Evidence suggests that the combination of platelet count and MPV that is mean platelet ratio (MPR) which can be obtained by dividing MPV by PLT may be more clinically significant than platelet count or MPV alone⁸.

To the best of our knowledge, this is the first study having an aim to study and investigate the relationships between platelet indices, leukocytes and severe periodontitis and short-term effects of non surgical periodontal treatment on leucocyte count and various platelet indices.

METHOD:

One eighty individuals visiting the Department of periodontics of PMNM Dental College, Bagalkot, Karnataka, India were included. Study was approved by ethical committee of the institution and Informed consent was obtained from all the subjects. Case history was recorded. After screening, the subjects were divided into 3 groups. Group A (Control): 60 systemically and periodontally healthy individuals. Group B: 60 Subjects with CP⁹. Group C: 60 CP subjects who were recalled after 1 month of NSPT.

Clinical periodontal assessments includes: plaque index (PI), gingival index (GI), probing pocket depth (PPD) and clinical attachment level (CAL). Subjects aged between 18 to 60 years, who were systemically healthy. Subjects showing absence of clinical manifestations of periodontal disease with PD ≤ 3 mm or no obvious clinical attachment loss were defined as healthy controls and Subjects diagnosed as CP i.e. at least two interproximal sites with CAL ≥ 6 mm, not on the same tooth; One or more interproximal sites with pocket depth (PD) ≥ 5 mm were included. Subjects with history of SRP or antimicrobial therapy within 6 months, Current or previous smokers, tobacco or alcohol habits, Systemic disease e.g. diabetes mellitus, hypertension or pregnant and those who were under NSAIDs, for the past 3 months and subjects were excluded¹⁰.

LABORATORY ANALYSIS

Three ml of venous blood was collected from all subjects by using routine venipuncture method. WBC and platelet parameters were determined by using an automated cell counter within 24 hours after collection¹¹.

After clinical, radiographic, and blood examinations, all patients

received supragingival scaling and subgingival debridement under local anesthesia. At 1 month after NSPT, periodontal and blood examinations were re-evaluated¹².

STATISTICAL ANALYSIS

Data collected were analyzed using the SPSS software. Results on continuous measurements were presented on Mean SD and results on categorical measurements were presented in Number (%). Level of significance was fixed at $p=0.05$ and any value less than or equal to 0.05 were considered to be statistically significant.

Wilcoxon Signed Rank test & Mann Whitney U test was used to find the significance of study parameters on continuous scale between two groups.

RESULTS

Table 1: Mean Values Of Pi, Gi, Bop, Ppd And Cal.

	Periodontitis, Mean±SD	Healthy, Mean±SD	Chronic periodontitis 1 month after APT Mean±SD
Age(years)	47.03±10.775	28.42±5.881	47.50±8.362
GingivallIndex	2.232±0.3596	0.385±0.168	2.380±0.3042
PlaqueIndex	2.293±1.2213	0.473±0.1867	1.305±0.3352
BOP	0.963±0.3092	0.828±0.4291	1.810±0.3219
PPD(mm)	6.60±1.19	0.0±0.0	4.95±0.872
CAL(mm)	6.07±0.841	0.0±0.0	3.40±1.210

Table 2: Comparison Of Platelet Parameters Like PC, MPV, PDW, PCT, MPR.

Laboratory parameters	Healthy Mean±SD	CP subjects Mean±SD	CP Subjects 1month after NSPT Mean ±SD
PC	233.37±3.625	252.20±58.567	252.20±57.902
MPV	6.263±0.5593	6.230±0.6392	6.630±0.4774
PDW	14.357±2.1563	14.200±1.5856	15.065±1.2286
PCT	0.1521±0.0279	0.1940±0.0549	0.1680±0.0436
MPR	0.0253±0.0157	0.0315±0.01521	0.0247±0.0071

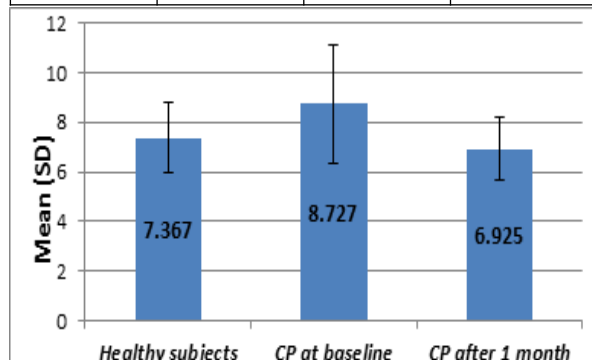


Figure 1: Comparison Of Leukocyte Count Values.

DISCUSSION

Statistically highly significant change was observed in all the clinical parameters like PI, GI, PPD, BOP from baseline to 1 month after NSPT in CP groups (Table 1). This is due to the reinstitution of oral hygiene techniques led to the reduction of gingival inflammation within 4 week of plaque removal which was in accordance with T Srinivas et al⁵.

The results showed higher PC in CP group (252.20±58.567 cells/L) at baseline when compared to the healthy group (233.37 ±32.625 cells/L) as shown in (Tabel 2). This was in agreement with studies by B Rai et al¹³ which could be due to fact that the platelets play an integral role in innate immunity against microorganism. A significant decrease in platelet counts after NSPT (252.20±58.567 vs 239.25±57.902) has been reported, attributable to the decrease in the level of infective-inflammatory periodontal disease following NSPT. Present study showed that there is an increase in number of WBC count in CP group at baseline when compared to healthy controls (8.727±2.3747 Vs 7.367±1.3977) which was in accordance with Devinder Singh et al¹⁴. A reduction in counts of WBCs were

also observed at one month after NSPT (8.727±2.3747 Vs 6.925±1.2639) which was in accordance with studies conducted by Efthymios et al¹². Results are based on fact that when the periodontal inflammatory lesions heal after periodontal therapy, the need for recruitment of inflammatory cells will decrease as well as reduced participation of platelets and leukocytes.

Current study showed decrease in MPV levels in patients with severe periodontitis at baseline when compared with healthy controls (6.263±0.5593 Vs 6.230±0.6392) in accordance with the study conducted by Xian'e Wang et al¹⁰. Increase in levels of MPV was due to the overproduction of proinflammatory cytokines and acute-phase reactants that can suppress the size of platelets and a subsequent release of small-sized platelets from the bone marrow¹¹. In our study significant increase in MPV were observed after NSPT (6.230±0.6392 Vs 6.630±0.4774) and these results may be due to the suppression of inflammation, large-sized platelets were not consumed in inflammatory sites, which resulted in increase of MPV level and these results were in accordance with study by Gasparyan AY et al¹⁶.

This study showed an elevation of PCT volume in CP subjects at baseline when compared to healthy controls (0.1940±0.0549 Vs 0.1680±0.0436). Our results were in concurrent with sahin et al¹⁷, where increase in PCT was significant for inflammatory disease like tuberculosis than the other platelet indices. Subsequent decrease in PCT is observed after one month of NSPT (0.1940±0.0549 Vs 0.1680±0.0436) and this might be due to the less circulating platelets in vasculature due to the cessation of inflammation after NSPT.

This study also showed a decrease in PDW in CP group at baseline when compared to controls (14.357±2.1563 Vs 14.200±1.5856). This may be because of fact that during inflammation, activation of platelet causes morphologic change, including both shapes changing from discoid to spherical and pseudopodia formation resulting in larger PDW value¹⁸. Mean PDW found to be increased after one month of NSPT (14.200±1.5856 Vs 15.065±1.2286). These results might be due to decrease in platelet activation at the site of inflammation making platelet to regain its original discoid shape.

To summarize, this is the first study to evaluate PDW as a marker of incidence of chronic periodontitis. Our finding suggests that PDW could be a potential predictive factor of periodontitis.

Current study is the first to demonstrate the superiority of the MPV/platelet ratio to MPV alone as a predictor for early diagnosis of periodontal disease. This study showed an increase in MPR in chronic periodontitis group at baseline when compared to healthy controls which means that MPR increases in a local inflammatory disease. Therefore, in patients with a higher MPV/platelet ratio indicates a potential need for early diagnosis and treatment. It was interesting to note that MPR were lower in the CP group after NSPT as compared with the baseline in the present evaluation, which may imply that there is a marked decrease due to therapeutic intervention. Consequently the study conducted by Ana Rejec et al¹⁹ did not confirm significant differences in MPV/PLT values between groups of dogs with periodontal disease.

This study showed increase of MPV, Leucocyte count, Platelet count was related to the severe periodontal inflammation, and the value inversed shift after NSPT. The findings suggest that platelet parameters were related with inflammatory status of patients with severe periodontitis. It will be difficult to draw comparisons with our study as there is hardly any available literature addressing MPR as potential systemic biomarkers of CP in humans before and after NSPT.

CONCLUSION

NSPT aims to reduce the number of periodontal pathogens thereby reducing the inflammation. Within the limitations of this investigation, we suggest that MPV (the elevation of which may disrupt the balance between pro- and anti-inflammatory mediators in disease) and MPR (which is a marker of systemic inflammation) and PDW be included as potential parameters to provide clarity to the impact of CP which may have on systemic health.

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