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Assessment of Hematological Parameters in patients with Chronic Periodontitis after Non-Surgical Periodontal Therapy

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Abstract

Background: Periodontitis is a persistent potential source of infection and has been considered as a separate risk factor for some cardiovascular, respiratory, endocrine, musculoskeletal, and reproductive system related abnormalities. Oral health impacts on the general well-being, and if comprehensive health care is ever to be achieved, oral health should not be seen as a separate, distant and less important area of health, which is totally unrelated to lifespan and its quality. The objective of this study is to evaluate the effect of nonsurgical periodontal therapy on various hematological parameters in patients with chronic periodontitis.

Material and Methods: A total of 30 patients with untreated chronic periodontitis were recruited. Blood was drawn from antecubital fossa as per WHO guidelines in the morning hours and the blood samples were then immediately transported to the laboratory for analysis of all the haematological parameters. All the clinical parameters (Plaque Index, Gingival Index and Probing pocket depth) and hematological parameters (Total leucocyte count (TLC), ESR and Platelet count) were assessed preoperatively at baseline and 2 weeks after non-surgical periodontal therapy.

Results: The study results showed that there was a statistically significant decrease in TLC, ESR, and platelet

count from baseline to 2 weeks postoperatively following nonsurgical periodontal therapy.

Conclusion: From this study, it can be concluded that there is improvement in the hematological parameters after nonsurgical periodontal therapy, which might also reduce the risk of atherosclerosis formation in the blood vessel and perhaps prevent cardiovascular diseases.

Keywords: Non-surgical periodontal therapy, Oral health, Periodontitis, Platelet count, Total leucocyte count

Introduction

Periodontitis is defined as "an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with increased probing depth formation, recession, or both.[1] Several studies [2,3] have displayed the invasion of periodonto-pathogens into the periodontal tissue and thereby into the systemic circulation predisposing the patient to an increased incidence of chronic low level bacteremia. Inflammation is recognized as playing a crucial role in the pathogenesis of atherosclerosis and cardiovascular disease which are the major health problems in developing nations. Inflammatory cells and cytokines are important not only in the initiation of plaque formation in the blood vessel wall but also in the maintenance and rupture of the plaque and subsequent thrombotic complications.[4] Usually, the total number of white blood cells and erythrocyte sedimentation rates in peripheral blood was used as a diagnostic measure to investigate whether a given individual suffers from an inflammatory disease or infection.[5] Leukocytes are an integral part of the innate immune system; these cells are recruited at higher levels during episodes of bacteraemia in periodontitis or leakage into the systemic circulation.[5] Periodontitis has also been shown to be associated with an increase in plasma fibrinogen and also increase in platelet activation → procoagulant state and thus increased risk for atherosclerosis and CVD.[6] Changes in cellular and molecular components of peripheral blood in periodontitis may explain at least in part, that periodontitis is associated with CVD's, thromboembolic events, and atherosclerotic changes.[7] The aim of the present study was to investigate the effect of non-surgical periodontal therapy on total leukocyte count (TLC), total platelet count and erythrocyte sedimentation rate (ESR) in patients with chronic periodontitis.

Materials and Method

A total of 30 patients with untreated chronic periodontitis were recruited from the Department of Periodontics, Kamineni Institute of Dental Sciences, Narketpally, Nalgonda, Telangana.

Inclusion criteria

- Age groups of 20-55 years of both genders were included.
- Systemically, healthy patients.
- Patients with probing pocket depth of ≥ 5mm along with radiographic evidence of bone loss.

Exclusion criteria

- Use of tobacco in any form.
- Pregnant and lactating women.
- Systemically compromised patients.
- Patients with a history of drug intake known to affect the periodontium.

Clinical measurements

Following clinical parameters were recorded at baseline and after 2 weeks:

- Plaque Index (PI) (Loe and Silness)[8]
- Gingival Index (GI) (Silness and Loe)[8]
- Probing pocket depth (PPD)

UNC -15 probe was used to record PPD

Blood Sample Collection

Under aseptic conditions peripheral venous blood was drawn from antecubital fossa as per WHO guidelines in the morning hours, and the blood samples were then immediately transported to the laboratory for analysis of all the haematological parameters. All the clinical and hematological parameters were assessed preoperatively at baseline and within 2 weeks after non-surgical periodontal therapy.

Periodontal Therapy

On the first appointment, all patients received routine oral hygiene instructions and one-stage full-mouth conventional SRP employing both hand instruments and a piezoelectric ultrasonic hand piece under local anaesthesia of 2% lidocaine with 1:80,000 adrenaline.

Statistical Analysis

Clinical parameters like Plaque index and gingival index scores were compared by Wilcoxon matched pairs test and PPD scores by dependent t test. Hematological parameters like Total leukocyte count (cells/cumm) scores, PCV (Vol %), MCV (fl), MCH (pg), ESR (mm/first hour) and Platelet count (lakhs/cumm) by dependent t test by dependent t test. MCHC (%) scores by Wilcoxon matched pairs test.

Results

Statistically significant changes in all the clinical parameters (PI, GI and PPD) from baseline to 2 weeks postoperatively (Table 1). Statistically significant decrease in TLC following 2 weeks after scaling and root planing (at baseline TLC was 7703/mm^3 and at 2 weeks follow-up TLC was 6350/mm^3 ($P < 0.0001$). There was a statistically significant decrease in platelet count from 2.64 lac/mm^3 preoperatively to 2.13 lac/mm^3 at 2 weeks postoperatively ($P < 0.0001$). Statistically significant decrease in ESR following 2 weeks after scaling and root planing (at baseline ESR was 24.50/mm3/first hour and at 2 weeks follow-up TLC was 15.03/mm3/first hour, $P < 0.0001$) (Table 2).

Discussion

The results of the present study show the beneficial effects of periodontal treatment on possible risk factors for atherosclerotic events due to the improvement of hematological parameters in patients with chronic periodontitis. There was decrease in all the clinical and hematological parameters at the end of 2 weeks when compared to baseline. There was statistically significant decrease in plaque index, gingival index and MCHC at the end of 2 weeks ($p<0.05$). Leukocytes are the major component of blood cells of the phagocytosis and first cells of the host defense mechanism against infective agents. Periodontitis is a bacterial infection; neutrophils are the initially predominant cells of host defense and have a significant role in inflammation and pathogenesis. Many authors reported that there was a statistically significant decrease in TLC from baseline to 2 weeks following non-surgical periodontal therapy.[9] These findings were similar to our study where TLC was reduced. Platelets have their main function in hemostasis, but they also play a role in inflammatory and immune processes. Their number increases in chronic inflammation.[10] An epidemiological study in 732 patients by Griesshammer et al.[11] concluded that elevated platelet counts (thrombocytosis) was the underlying cause of periodontal infection in 21% of the subjects studied. Wakai et al[12] in their study reported increased platelet counts in patients with periodontitis. Thaulow et al.[13] assessed the possible association between platelet count and the coronary heart diseases over 150 patients where he found that platelet counts were positively related to the risk of cardiovascular death. In our study also, there was significant decrease in platelet counts after SRP. Similar results were reported by Christan et al.[2] who showed

decrease in platelet counts after periodontal. In our study, there was a decrease in ESR after SRP due to a decrease in chronic infection which is in accordance with the study by Hutter et al.[14] The determination of ESR is helpful in assessing the progress of patients treated for certain chronic inflammatory disorders.

Limitations of the study and future prospective

The date from the present study has provided with the evidence that periodontal diseases may act as risk factors for cardiovascular diseases. However, the exact mechanism how the periodontal infections contribute to cardiovascular diseases is still unclear. Because of extensive research is going on in this field, more clarity in the mechanisms involved in the association of periodontal diseases to cardiovascular diseases is expected in future.

Conclusion

Non- surgical periodontal therapy objects to reduce the number of periodontal pathogens thereby reduces the inflammation, which may indirectly decrease the risk of CVD's. The positive effect of non-surgical periodontal therapy in reducing leukocyte count, platelet count and ESR could be helpful in preventing CVD's.

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Legend Tables

Table 1: Clinical parameters at baseline and 2 weeks

Clinical Parameters	Mean ± Standard Deviation		
	Baseline	2 weeks	P-value
Plaque Index (PI)	1.68±0.15	0.81±0.07	0.0360*
Gingival Index (GI)	1.69±0.15	0.86±0.05	0.0170*
Probing Pocket Depth (PPD)	4.10±0.33	2.80±0.36	0.5620

*p<0.05

Table 2: Hematological values at baseline and 2 weeks

Hematological Parameters	Mean ± Standard Deviation		
	Baseline	2 weeks	P-value
Total leukocyte count (cells/cumm)	7703.33±1658.93	6350.00±1295.28	0.9420
Packed Cell Volume (Vol %)	37.31±5.88	35.72±5.27	0.4120
Mean Corpuscular Volume (fl)	84.17±4.21	81.77±3.25	0.5550
Mean Corpuscular Hemoglobin (pg)	28.27±1.58	26.87±1.07	0.9920
Mean Corpuscular Hemoglobin Concentration (%)	33.63±1.46	29.84±6.17	0.0010*
Platelet count (lakhs/cumm)	2.64±0.69	2.13±0.56	0.5550
Erythrocyte Sedimentation Rate (mm/first hour)	24.50±11.47	15.03±8.15	0.4880

*p<0.05