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Effect of Non-Surgical Periodontal Therapy on Plasma and Salivary Superoxide Dismutase Levels in Chronic Generalized Periodontitis Patients

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Abstract

Background: Oxidative stress plays a pivotal role in the pathogenesis of periodontal disease. Superoxide dismutase (SOD), a key antioxidant enzyme, protects periodontal tissues from reactive oxygen species. This study evaluated the effect of non-surgical periodontal therapy (NSPT) on clinical parameters and plasma and salivary SOD level in chronic generalized periodontitis patients.

Aim & Objective: To evaluate and compare the effect of non-surgical periodontal therapy on the clinical parameters and the levels of superoxide dismutase enzyme in patients with chronic generalized periodontitis at baseline and at a four-week interval.

Material & Methods: 60 participants (20–50 years) were divided into two groups: Group I (healthy controls, n=30) and Group II (chronic generalized periodontitis, n=30). Baseline clinical parameters—Plaque Index, Gingival Index, Sulcus Bleeding Index, Probing Pocket

Depth, Recession Depth, and Clinical Attachment Level were recorded. Blood and saliva samples were collected to assess SOD levels. Group II underwent NSPT (scaling and root planing), and parameters were reassessed at 4 weeks.

Results: Group II showed significant post-treatment reduction in Plaque Index, Gingival Index, Sulcus Bleeding Index, and Probing Pocket Depth, along with significant gain in Clinical Attachment Level ($p<0.001$). Plasma and salivary SOD levels significantly increased after NSPT ($p<0.05$). Recession depth changes were found to be statistically non-significant.

Conclusion: NSPT significantly improves both periodontal health and systemic antioxidant defense. Plasma and salivary SOD levels may serve as reliable biomarkers for evaluating periodontal status and therapy outcomes.

Keywords: Chronic periodontitis; oxidative stress; superoxide dismutase; scaling and root planing; periodontal therapy.

Introduction

Periodontitis is a multifactorial, chronic inflammatory disease characterized by the progressive destruction of tooth-supporting structures, ultimately leading to tooth mobility and loss if left untreated. It is initiated by microbial plaque biofilms, but disease progression depends largely on the host's immune-inflammatory response. The dysregulated immune response results in excessive production of inflammatory mediators and reactive oxygen species (ROS), which exacerbate tissue breakdown.

ROS are normally generated by activated neutrophils and macrophages as part of the body's defence mechanism against microbial pathogens. While they serve an essential role in microbial killing, their overproduction creates a state of oxidative stress, where the balance between oxidants and antioxidants is disturbed. This imbalance contributes to lipid peroxidation, protein damage, and DNA fragmentation within periodontal tissues, accelerating periodontal destruction. To counteract oxidative damage, the host relies on antioxidant defence mechanisms, including both enzymatic and non-enzymatic antioxidants. Among the enzymatic antioxidants, superoxide dismutase (SOD) is a key enzyme that catalyses the dismutation of superoxide radicals into hydrogen peroxide and oxygen. Reduced activity of SOD has been demonstrated in the gingival crevicular fluid, saliva, and plasma of periodontitis patients, indicating impaired antioxidant defence at both local and systemic levels.[1,2]

The significance of oxidative stress in periodontitis extends beyond oral health, as mounting evidence links periodontal inflammation with systemic conditions such as diabetes mellitus, cardiovascular disease, and adverse pregnancy outcomes. This association highlights the potential of oxidative stress markers as shared pathways between periodontal and systemic diseases. Non-surgical periodontal therapy (NSPT), primarily comprising scaling and root planing (SRP), remains the cornerstone of periodontal management. It effectively reduces bacterial load, resolves gingival inflammation, and improves clinical attachment. While clinical improvements following NSPT are well-documented, relatively fewer studies have evaluated its effect on systemic and local antioxidant status, particularly SOD activity. Assessing both plasma and salivary SOD levels may provide valuable insights into the role of antioxidant defence in periodontal health and therapy outcomes.[3]

Therefore, the present study was designed to evaluate and compare plasma and salivary SOD levels in healthy individuals and patients with chronic generalized periodontitis, before and after NSPT.

Aim & Objectives

1. To evaluate the clinical parameters and the levels of superoxide dismutase enzyme in participants with clinically healthy gingiva and in patients with chronic generalized periodontitis.
2. To evaluate and compare the effect of non-surgical periodontal therapy on the clinical parameters and the levels of superoxide dismutase enzyme in patients with chronic generalized periodontitis at baseline and at a four-week interval.

Materials and Methods

Study Design and Population

This was a randomized, controlled, interventional study conducted in the Department of Periodontology and Oral Implantology, Desh Bhagat Dental College and Hospital, Mandi Gobindgarh, Punjab, India. The study was approved by the Institutional Ethical Committee and carried out in accordance with the Declaration of Helsinki (2013 revision). Written informed consent was obtained from all participants before inclusion.

A total of 60 participants, aged between 20–50 years, were recruited from the outpatient department. Participants were systemically healthy and were allocated into two groups:

- **Group I (Control group, n=30):** Periodontally healthy individuals, showing probing depth ≤3 mm, no clinical attachment loss, and no radiographic signs of bone loss.
- **Group II (Test group, n=30):** Patients diagnosed with chronic generalized periodontitis, with probing depth ≥5 mm and clinical attachment loss ≥4 mm affecting more than 30% of sites, and radiographic evidence of alveolar bone loss.

Inclusion criteria

- Age between 20 and 50 years.
- Presence of at least 20 natural teeth.
- For Group II: diagnosis of chronic generalized periodontitis according to the American Academy of Periodontology (1999) criteria.
- For Group I: clinically healthy periodontium with no history of periodontal therapy.

Exclusion criteria

- History of smoking or tobacco use in any form.
- Pregnancy or lactation.
- Presence of systemic diseases such as diabetes mellitus, cardiovascular disease, or immunological disorders.
- Antibiotic or anti-inflammatory drug intake within 6 months prior to study.
- Previous periodontal therapy in the last 12 months.
- Patients unwilling to comply with study protocol.

Examiner Calibration

All periodontal measurements were recorded by a single calibrated examiner using a UNC-15 periodontal probe. Examiner calibration was performed on 10 non-study patients with chronic periodontitis, and measurements were repeated after 48 hours. Intra-examiner reproducibility was calculated, and >90% agreement within ±1 mm was achieved before commencement of the study.

Clinical Parameters

At baseline and 4 weeks after treatment, the following periodontal parameters were recorded:

- **Plaque Index (PI):** Silness and Loe, 1964 – to assess oral hygiene.
- **Gingival Index (GI):** Loe and Silness, 1963 – to assess gingival inflammation.
- **Sulcus Bleeding Index (SBI):** Muhlemann and Son, 1971 – to record bleeding on probing.
- **Probing Pocket Depth (PPD):** Distance from gingival margin to base of pocket.

- **Clinical Attachment Level (CAL):** Calculated as PPD + RD.
- **Recession Depth (RD):** Distance from cementoenamel junction (CEJ) to gingival margin.

All indices were recorded at six sites per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, distolingual).

Intervention

Patients in Group II underwent non-surgical periodontal therapy (NSPT), consisting of full-mouth scaling and root planing (SRP).

- Procedures were performed under local anaesthesia using ultrasonic scalers and hand instruments (Gracey curettes).
- Each patient received two treatment sessions within 7 days.
- After instrumentation, patients were instructed in oral hygiene maintenance, including modified Bass brushing technique and use of interdental aids.
- No systemic antibiotics, antiseptic mouth rinses, or host-modulation agents were prescribed during the study to eliminate confounding effects.
- Supportive motivation and reinforcement of oral hygiene instructions were provided at follow-up visits.

Sample Collection

At baseline and at 4 weeks after NSPT, saliva and blood samples were collected from both groups.

- **Saliva collection:** Unstimulated whole saliva was collected in sterile containers between 9:00–11:00 am to minimize circadian variation. Participants were instructed to avoid food, drinks (except water), and oral hygiene practices for at least 2 hours prior to collection. Approximately 2–3 mL of saliva was collected by the passive drool method and immediately stored on ice.
- **Blood collection:** 5 mL of venous blood was drawn from the antecubital vein under aseptic conditions using EDTA-coated vacutainers. Samples were centrifuged at 3000 rpm for 10 minutes to separate plasma.

  Both saliva and plasma samples were stored at –20°C until biochemical analysis.

Biochemical Analysis of Super Oxide Dismutase (SOD)

Superoxide dismutase (SOD) activity was estimated spectrophotometrically using the McCord and Fridovich method (1971). This method is based on the ability of SOD to inhibit the auto-oxidation of pyrogallol.

- The rate of pyrogallol oxidation was monitored at 420 nm using a UV-visible spectrophotometer.
- One unit of SOD activity was defined as the amount of enzyme required to cause 50% inhibition of pyrogallol auto-oxidation per minute under assay conditions.
- Results were expressed as units per millilitre (U/mL) for plasma and units per milligram protein (U/mg protein) for saliva.

Statistical Analysis

Data were compiled and analyzed using SPSS version 17.0 (SPSS Inc., Chicago, IL, USA). Descriptive statistics (mean ± standard deviation) were calculated for all clinical and biochemical parameters. Normality of data distribution was checked using the Shapiro-Wilk test. Intergroup comparisons (Group I vs. Group II) were made using the independent t-test. Intragroup comparisons (baseline vs. 4 weeks) were analyzed using the paired t-test. The level of statistical significance was set at $p < 0.05$.

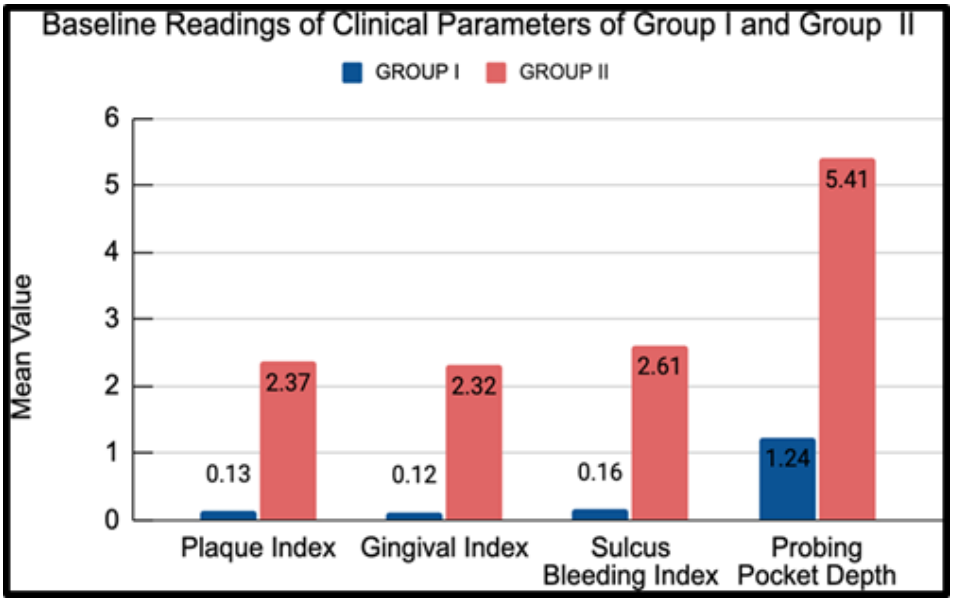
Results

Demographic Characteristics

60 participants completed the study, with 30 individuals in each group. Group I (controls) included 15 males and 15 females (mean age: 34.2 ± 5.8 years), while Group II (periodontitis) comprised 16 males and 14 females (mean age: 35.7 ± 6.2 years). There were no statistically significant differences in age or gender distribution between the groups (p>0.05).

Clinical Parameters

At baseline, Group II exhibited significantly higher mean values of Plaque Index (PI), Gingival Index (GI), and Sulcus Bleeding Index (SBI) as compared with Group I (p<0.001). Mean Probing Pocket Depth (PPD) and Clinical Attachment Level (CAL) were also significantly greater in Group II (p<0.001), indicating active periodontal destruction. Recession Depth (RD) did not differ significantly between the groups (p>0.005).



Graph 1: Baseline readings of all the clinical parameters of Group I and Group II

Following non-surgical periodontal therapy (NSPT), Group II demonstrated marked improvement. At 4 weeks, there was a significant reduction in PI, GI, SBI, and PPD compared with baseline (p<0.001). CAL showed significant gain (p<0.001), whereas RD increased slightly but was not found to be statistically significant (p>0.005). Group I (controls) showed no significant changes in any clinical parameter during the study period.

Table 1: Baseline Readings of Clinical Parameters of Group I and Group II

Group	Group I	Group II	GRP I VS GRP II (p- value)
Plaque Index (PI)	0.13 ± 0.017	2.37 ± 0.15	<0.001**
Gingival Index (GI)	0.12 ± 0.029	2.32 ± 0.21	<0.001**
Sulcus Bleeding Index (SBI)	0.16 ± 0.04	2.61 ± 0.16	<0.001**
Probing Pocket Depth (PD)	1.24 ± 0.18	5.41 ± 0.41	<0.001**

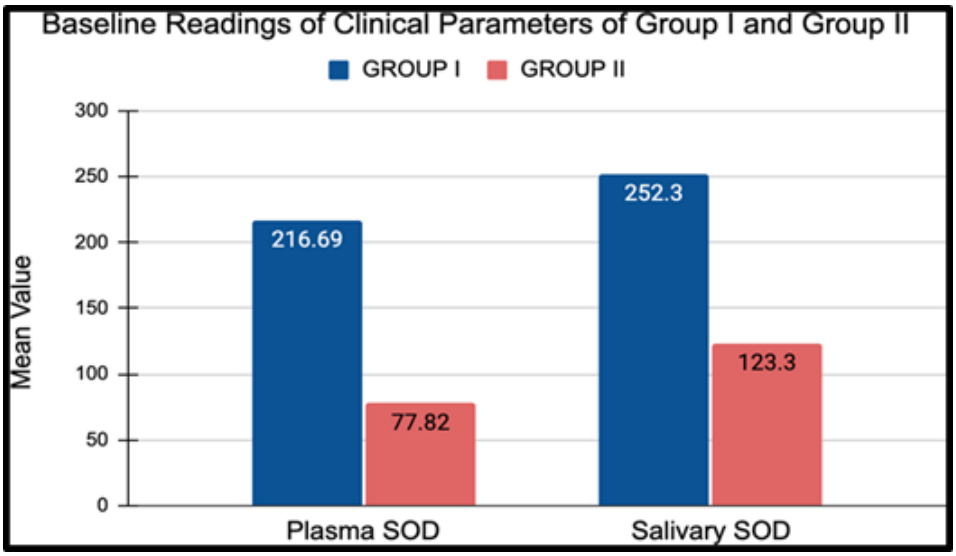
Biochemical Parameters

At baseline, plasma and salivary superoxide dismutase (SOD) activity were significantly lower in Group II compared to Group I (p<0.005). After NSPT, Group II showed significant increases in both plasma and salivary SOD levels (p<0.005), reflecting restoration of antioxidant defence. No significant changes were observed in Group I across the two time points.

Table 2: Baseline Readings of Biochemical Parameters of Group I and Group II

Group	Group I	Group II	GRP I VS GRP II (p- value)
Plasma Superoxide Dismutase (SOD)	216.69 ± 82.40	77.82 ± 36.28	<0.001**
Salivary Superoxide Dismutase (SOD)	252.30 ± 79.14	123.30 ± 45.69	<0.001**

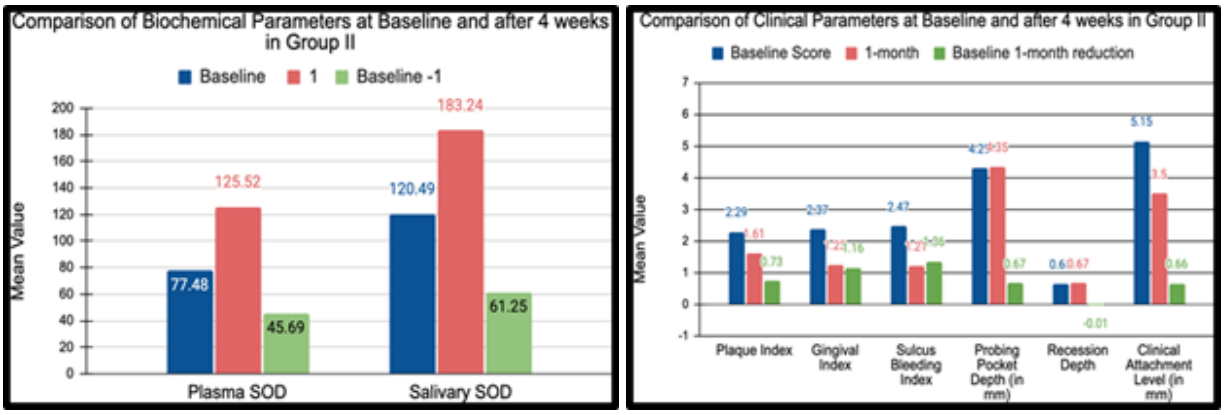
** P<0.001: Highly Significant, * P<0.05: Significant, P> 0.05: Not significant



Graph 2: Baseline readings of Biochemical parameter of Group I and Group II

Intergroup Comparison

At 4 weeks, Group II demonstrated significant improvement in clinical and biochemical parameters compared to baseline values and difference between Group II and Group I were found to be decreased. However, PPD, CAL and SOD levels in Group II while improved remained marginally different from controls (p<0.005).



Graph 3: Intergroup comparison of clinical and biochemical parameters of Group I and Group II

Table 3: Comparison of Clinical Parameters at Baseline and After 4 Weeks in Group Ii

Clinical Parameters	Baseline Score	4th week Score	Baseline - 4th week reduction		
			Mean ± SD	t value	p-Value
Plaque Index (PI)	2.29 ± 0.161	1.61 ± 0.11	0.73 ± 0.15	25.43	<0.001**

Gingival Index (GI)	2.37 ± 0.34	1.25 ± 0.15	1.16 ± 0.15	35.80	<0.001**
Sulcus Bleeding Index (SBI)	2.47 ± 0.12	1.21 ± 0.18	1.36 ± 0.14	42.94	<0.001**
Probing Pocket Depth (PPD)	4.29 ± 0.19	4.35 ± 0.20	0.67 ± 0.12	20.18	<0.001**
Recession Depth (RD)	0.65 ± 0.15	0.67 ± 0.15	-0.01 ± 0.04	-1.53	<0.001**
Clinical Attachment Level (CAL)	5.15 ± 0.26	3.50 ± 0.21	0.66 ± 0.13	18.60	<0.001**

Discussion

The present study demonstrated reduced plasma and salivary SOD levels in patients with chronic generalized periodontitis, supporting previous findings that oxidative stress is a central mechanism in periodontal destruction.[4,5,6] Following NSPT, both clinical and biochemical improvements were observed, confirming that therapy restores antioxidant balance by reducing ROS burden.[7,8,9] These results suggest that SOD levels in saliva and plasma may serve as non-invasive biomarkers for periodontal disease activity and treatment response. This aligns with prior studies that emphasized the role of oxidative stress markers in systemic diseases such as diabetes, cardiovascular conditions, and adverse pregnancy outcomes. Thus, the findings of this study not only reinforce the role of oxidative stress in periodontal pathology but also highlight the systemic implications of periodontal disease. Another important point is the clinical significance of NSPT as the gold standard of periodontal therapy. Although surgical interventions and adjunctive therapies such as local drug delivery, laser therapy, and host-modulation agents are often discussed, NSPT continues to remain the cornerstone of treatment due to its predictable efficacy in reducing inflammation and microbial load.

The present study further strengthens this evidence by showing a concurrent improvement in host antioxidant defense mechanisms post-therapy.

It is noteworthy that SOD activity was measured in both saliva and plasma, offering insights into local as well as systemic responses to periodontal therapy. Saliva, being an easily accessible diagnostic fluid, may serve as a chairside biomarker for periodontal disease monitoring. On the other hand, plasma reflects the systemic burden of oxidative stress, thereby linking periodontal health with overall systemic well-being.

Despite these encouraging findings, certain limitations must be acknowledged. The relatively short follow-up period (4 weeks) limits assessment of long-term stability of SOD improvements post-therapy.[10] In addition, the sample size, though adequate for preliminary analysis, may not fully represent broader population heterogeneity. Future studies with larger cohorts, longer follow-ups, and evaluation of additional oxidative stress biomarkers (such as catalase, glutathione peroxidase, and malondialdehyde) are warranted to provide a more comprehensive understanding.

Furthermore, the role of lifestyle factors such as smoking, diet, and systemic conditions like diabetes could not be fully evaluated in the present study. As oxidative stress is influenced by multiple systemic variables, controlling for these confounders in future research would provide more accurate correlations.[11] Additionally, interventional studies testing antioxidant supplementation alongside NSPT could offer valuable insights into potential adjunctive strategies for managing oxidative stress in periodontitis.[12]

To summarize, the present findings underscore the dual role of NSPT in controlling local inflammation and restoring host antioxidant capacity. They provide strong evidence supporting the utility of SOD as a biomarker for

disease activity and treatment outcomes, with potential applications in both research and clinical practice.

Conclusion

Oxidative stress developed due to periodontal tissue damage could have influenced the level of antioxidant enzyme in plasma and saliva. Thus, this indicates that periodontitis can be one of the factors for decreased levels of antioxidants in our body. Superoxide dismutase (SOD) is an important antioxidant enzyme that protects tissues against oxidative injury from free oxygen radicals generated by various metabolic processes. Non-surgical periodontal therapy significantly improves periodontal health and enhances systemic and local antioxidant defense by increasing plasma and salivary SOD levels. SOD can be considered a reliable biomarker for evaluating periodontal disease progression and therapeutic outcomes.

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