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# EVALUATION OF CLINICAL PARAMETERS AND SUPEROXIDE DISMUTASE LEVEL IN POST-MENOPAUSAL WOMEN WITH CHRONIC PERIODONTITIS

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**ABSTRACT**

**Objectives:** To clinically evaluate and compare changes in periodontal parameters and superoxide dismutase activity after non-surgical periodontal therapy with and without micronutrient supplementation in postmenopausal women with periodontitis. **Material And Method:** 60 postmenopausal women with periodontitis were divided into group I and group II. Group I patients was given systemic micronutrient supplementation along with scaling and root planing. Group II patients were treated with scaling and root planing only. Serum and saliva samples were collected and evaluated for SOD level at baseline and 3 months along with clinical parameters. **Result:** Salivary and serum SOD values significantly improved with periodontal treatment. Improvement in systemic enzymatic antioxidant status along with reduction in gingival inflammation and bleeding on probing (%) sites was significantly greater in group I as compared to group II. **Conclusions:** Adjunctive micronutrient supplements reduce periodontal inflammation and improve the status of systemic enzymatic antioxidants in postmenopausal women

## INTRODUCTION:

Periodontitis, one of the most ubiquitous diseases, is characterized by the destruction of connective tissue and bone support following an inflammatory host response secondary to the infection by periodontal bacteria. Almost all forms of periodontal diseases occur as a result of mixed microbial infections within which specific groups of pathogenic bacteria coexist.

In a woman's middle age, through the climacteric course, circulating sex hormone levels change, and this results in several clinical effects that have a potential effect on periodontal status and quality of life.

Estrogen is the predominant sex hormone in women and shows immunoprotective, antioxidants, and anti-inflammatory effects , show better periodontal status compared to post-menopausal women. After menopause with the weakening of estrogen signals women may show more serious periodontal destruction.[1]

Estrogen level is declined in menopause, results in an increase in oxidative stress and a decrease in antioxidants defense mechanism[2] which, may cause oral problems including a paucity of saliva leading to xerostomia, burning mouth syndrome (BMS), increase in the incidence of dental caries, dysesthesia, taste alterations, osteoporotic jaws, atrophic gingivitis, and periodontitis.

Advances in oral and periodontal diagnostic research include the measurement of biochemical markers of tissue destruction, which can be measured in serum, saliva, or gingival crevicular fluid.

The salivary level of various biomarkers and changes in the presence of oral cavity disease. Saliva contains superoxide dismutase, catalase, and glutathione peroxidase as natural enzymatic antioxidants.

Superoxide dismutase constitutes anti inflammatory and antioxidant defense against oxidative stress in the body. The different forms of superoxide dismutase are unequally distributed throughout the body fluid including saliva.

As SOD is highly sensitive to redox changes and can be detected in the saliva and serum, it is taken as a biochemical marker to estimate antioxidant levels in postmenopausal women in the present study. It can be more precisely measured in saliva and serum using SOD kit and ELISA test through Marklund and Marklund method,.[3]

Adjunctive antioxidant micronutrient supplementation in postmenopausal women, is effective against oxidative stress, helps in scavenging free radicals and binds with some of the oxidants, helps in the regulation of the extracellular form of superoxide dismutase, reduces the formation of highly toxic radical formation, improve antioxidant defense and results in the more efficient management of periodontal inflammation and healing.[3]

In the present study, Antoxid capsules are given to the patient as adjunctive micronutrient supplementation for 3 months along with scaling and root planning (SRP) to evaluate its systemic effect on SOD level and effect on periodontal inflammation.

## MATERIAL AND METHOD :

All patients were taken from Periodontology department of the government dental college and hospital Ahmedabad and referred from the gynecological outpatient department of Civil hospital.

The study was carried out in 60 postmenopausal women with periodontitis who had entered natural menopause with their last menstrual period at least 12 months ago having ≥20 teeth and moderate chronic periodontitis having ≥2 interproximal sites with AL ≥4 mm (not on the same tooth), ≥2 interproximal sites with PD ≥5 mm (not on the same tooth) (**According to Eke at al. 2012**)[(4)] of the age group of 40- 65 years.

Patients, Who had undergone oral prophylaxis and Who had taken multivitamins or antioxidant micronutrient supplements in the previous six months, Who are using mouthwashes, Current and former smokers, who had taken

anti-inflammatory or antibiotic drugs within the previous three months and with other medical conditions that could influence the results of periodontal treatment, who had undergone HRT, and who had entered artificial menopause were excluded from the study.

A simple random sampling technique was used for the selection of cases.

Informed consent was obtained from the patients and the ethical committee of the institute approved this clinical trial. The samples were then divided into 30 study groups and 30 control groups.

**1. GROUP I** (study group) – scaling and root planning + systemic administration of micronutrient antioxidants in the form of soft gelatin capsules (ANTOXID)

**2. GROUP II** (control group)– scaling and root planning

Clinical parameters Plaque index (PI), Gingival index (GI), Gingival bleeding index (GBI), Probing pocket depth (PPD), and Clinical attachment level (CAL) were recorded at baseline and 3 months after the completion of nonsurgical periodontal therapy(NSPT).

Saliva and blood samples were collected for biochemical analysis of SOD activity at baseline and 3 months after NSPT.

**Collection of saliva sample**

The participants were asked to refrain from consuming any food or beverages for 2 hours before saliva collection. Following a thorough mouth rinse subjects were instructed to be seated for 5 min and directed not to speak, eat/rinse, and minimize orofacial movements during 10 minutes periods. Then, the subject was instructed to spit the pooled unstimulated saliva from the floor of the mouth into the sterile plastic vial As shown in the figure 1. The plastic vial was then closed and sent to the biochemistry lab for assaying the SOD level.

**Figure 1 Collection Of Saliva**

**Collection of blood samples**

Two milliliters of venous blood was drawn from the antecubital vein of all participants by using a disposable syringe. It was transferred to a sterile plain vacutainer without an additive as shown in figure 2. Then vacutainer was sent to the biochemistry lab for assaying of SOD level. SOD activity in serum and saliva was estimated using Marklund and Marklund technique (1974).

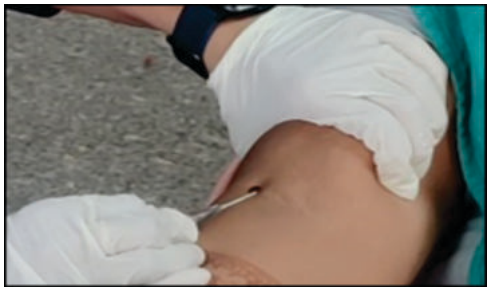

**Figure 2 : Collection Of Blood Sample**

**RESULT:**

The patients in both groups received the intended treatment. Follow-up was taken 3 months after SRP. No complication was observed in patients after treatment.

periodontal and biochemical parameters at baseline and after 3 months of follow-up for both groups are outlined in Table 1.

All the parameters were statistically nonsignificant difference between groups 1 and 2 at baseline. Significant improvements in BOP, GI, PPD, CAL, and PI and in SOD activity (%) in serum and saliva in groups 1 and 2 patients from baseline to 3 months are given in Table 1.

**Table: 1 Comparison Of Periodontal And Biochemical Parameters Of Groups 1 And 2 At Baseline And 3 Months Follow-up Visits.**

| | GROUP I | | | GROUP II | | |
|---|---|---|---|---|---|---|
| | Baseline | Post therapy | P value | Baseline | Post therapy | P value |
| PI | 2.36 ± 0.5 | 1.22 ± 0.3. | <0.001 | 2.58 ± 0.46 | 1.43 ± 0.28 | <0.001 |
| GI | 2.01 ± 0.49 | 0.92 ±0.43 | <0.001 | 1.94 ±0.50 | 1.24 ± 0.33 | <0.001 |
| GBI | 72.71± 18.35 | 23.69 ± 13.02 | <0.001 | 85.60± 19.09 | 40.48 ± 11.62 | <0.001 |
| PPD | 4.39 ± 1.20 | 3.22 ± 0.93 | <0.001 | 5.02 ± 1.11 | 3.91 ± 0.75 | <0.001 |
| CAL | 3.61 ± 1.95 | 1.28 ± 0.98 | <0.001 | 2.91 ± 2.00 | 1.52 ± 1.36 | <0.001 |
| SOD SERUM ACTIVITY | 71.57 ± 2.04 | 143.09 ± 11.27 | <0.001 | 83.89 ± 3.07 | 123.74 ± 5.12 | <0.001 |
| SOD SALIVA ACTIVITY | 48.38 ± 1.02 | 79.90 ± 0.51 | <0.001 | 47.29 ± 1.53 | 62.54 ± 3.5 | <0.001 |

Values are presented as mean ± standard deviation. PPD probing pocket depth, GI -gingival index, PI plaque index, CAL clinical attachment loss, GBI gingival bleeding index SOD super oxide dismutase

**Table: 2 Comparison Of Improvement Of Periodontal And Biochemical Parameters Of The Study Groupsinference: Group I Shows More Improvement In All Parameters As Compared To Group II**

| Parameters | Group I | Group II | P value |
|---|---|---|---|
| ΔGI | 1.09 | 0.7 | <0.001 |
| ΔPI | 1.14 | 1.15 | < 0.001 |
| ΔPPD (mm) | 1.17 | 1.11 | <0.05 |
| ΔBOP (%) | 49.02 | 45.12 | <0.0.05 |
| ΔCAL (mm) | 2.33 | 1.39 | <0.01 |
| ΔSOD serum activity | 71.01 | 39.85 | <0.01 |
| ΔSOD saliva activity | 31.52 | 7.44 | <0.01 |

After treatment, group I showed more reduction in gingival index, gingival bleeding index, probing pocket depth and more clinical attachment gain (2.33) as compared to group II (1.39).

Serum and salivary SOD activity significantly increased ($P$<0.01) following periodontal therapy in both groups. Improvement in systemic SOD level was significantly better in group I as compared to group II.

**DISCUSSION:**

Periodontitis is a highly prevalent, multifactorial, chronic inflammatory disease of periodontium leading to the destruction of supportive tissues of teeth and teeth loss. The interaction between microbes present in dental plaque and the host immune response is a major determinant of the

progression and clinical manifestations of periodontal disease. However, there is a multitude of factors like systemic, environmental, behavioral, and genetic factors which directly or indirectly influence this association at multiple levels.[5]

The homeostasis of the periodontium involves complex multifactorial relationships, in which the endocrine system plays an important role. Females seem to be more prone to hormone imbalance than males.[6] They experience hormonal variation under both physiological and non-physiological conditions, which may directly influence the periodontium as it affects the physiology of host-parasite interactions in the oral cavity.[7] Estrogen and Progesterone are responsible for physiological changes in women at specific phases of their life: puberty, menstrual cycle, pregnancy, menopause and post-menopause.

As women approach menopause, the levels of estrogen and progesterone begin to drop, compromising the anti-inflammatory and antioxidant effect of this hormone on the periodontium [6] results in increased oxidative stress and causes a change in lipid profile and enhances lipid peroxidation. [8] Progesterone may compete with glucocorticoids for an osteoblast receptor and inhibit glucocorticoid-induced osteoporosis. Therefore postmenopausal bone density reduction may be the result of a combination of inhibition of osteoclast downregulation by reduced estrogen and the increased cortisol inhibition of osteoblasts via the reduction of competition with progesterone.[6]

Oxidant-antioxidant balance exists in a healthy state. When this balance is disrupted and shifts towards the oxidant side "oxidative stress" results. Which is related to many systemic conditions and diseases such as diabetes [9], rheumatoid arthritis [10], menopause [11], and some oral diseases like oral lichen planus [12], Recurrent aphthous stomatitis [13], OSMF [8], and periodontitis [14] patient.

The antimicrobial activities of PMNs include oxygen-dependent and oxygen-independent mechanisms. The oxygen-dependent pathway involves the production of reactive oxygen species via the metabolic pathway of the "respiratory burst" mechanism [15]. These molecules are capable of initiating periodontal tissue destruction. The production of ROS by PMNs is primarily focused on bacterial killing, but high levels or activities of ROS cannot be balanced by the antioxidant defense system and extracellular release of ROS results in collateral damage to the surrounding tissues.[16]

One of the main components of the antioxidant protection of the body is a group of metal enzymes - SOD.[17] Although, intracellular SOD is the most prominent antioxidant in mammalian tissue,[18] WEI D AT AL have proven the significance of extracellular SOD in plasma and other body fluids.[19]

A decline in the level of estrogen results in a decreased level of Antioxidant enzymes like superoxide dismutase (SOD) in postmenopausal women. [20] The effect of estrogens on SOD expression in humans is apparently due to the activation of the estradiol receptor and was selective for SOD, with no change in the expression of GPX or CAT. Moreover, CAT and the GPX-dependent erythrocyte antioxidant defense were not affected by ovarian hormone disturbances in regularly menstruating women that result in anovulation with markedly lower plasma estradiol concentrations.[21] Estimation of oxidative stress-related tissue destruction products such as malondialdehyde (MDA), total oxidant status, or anti-oxidant levels such as SOD, and glutathione, may act as an indicator for measuring oxidative stress. Hence in the present study, SOD level was evaluated in serum and saliva of postmenopausal women with periodontitis following SRP.

Post-menopausal conditions alter the responses of periodontal tissue to microorganisms.[22]. Nonsurgical periodontal treatment results in a significant reduction in periodontal parameters.[23]

Micronutrient supplementation may be useful to maintain a balanced ROS environment in postmenopausal women with chronic periodontitis.[24]

Antioxidant micronutrients including beta-carotene, zinc, manganese and selenium may be effective in suppressing the activation of proinflammatory pathways through the quenching of free radicals. These trace elements act as cofactors for antioxidant enzymes. Copper and zinc act as a cofactor for cytosolic SOD, Manganese for mitochondrial SOD and selenium for glutathione peroxidase.[25] This may help to increase the antioxidant level in serum and saliva after adjunctive supplementation. So, Antoxid was used as an adjunctive micronutrient supplementation in the present study, which contains carotene, selenium, zinc, copper, and manganese.

Reduction in plaque index after 3 months may be due to a decrease in plaque accumulation as the plaque retaining area was reduced allowing the patient to maintain good oral hygiene and repeated reinforcement of oral hygiene habits in patients.

Reduction in the gingival index and gingival bleeding index and probing depth were found to be statistically significant at 3 months. This indicates that both the treatment procedures are equally effective in improving gingival health at 3 months.[24] A shift of balance in favor of antioxidants resulted in an improved reduction of gingival inflammation, greater reduction in BOP (%) sites, and more reduction in probing depth in group 1 as compared to group 2 suggesting more effective prevention of periodontal loss through adjunctive micronutrient supplementation during the maintenance phase in this population group.[26,27]

The effect of menopause is more evident in serum antioxidant analysis. The effect of periodontitis is more evident in saliva and gingival crevicular fluid.[28].

Evaluation of the data at baseline revealed that SOD levels in serum and saliva were significantly lower in both groups. It indicates a compromised oxidant-antioxidant balance in postmenopausal chronic periodontitis patients. The decreased level of SOD activity may be due to its high utilization to protect the cells from the injurious effects of superoxide radicals.[13] due to lower antioxidant activity in saliva and[29,30]Depressed levels of serum antioxidant levels.[31,32] of chronic periodontitis patients.[33]

According to **Godin & Wohaieb 1988[(34)],** SOD activity increases directly after the occurrence of oxidative stress. The human periodontal ligament possess the enzyme superoxide dismutase, which might afford biological protection against free radicals, particularly $O2^-$, during the inflammatory response[35]. Bacterial Lipopolysaccharides stimulate $O2^-$ release from the gingival fibroblast, suggesting that the induction of superoxide dismutase may represent an important defense mechanism of the fibroblast during inflammation.[36] Increased superoxide dismutase activity level in inflamed gingiva from chronic periodontitis patients may indicate the increased $O2^-$ generation by neutrophils invaded at the disease site, and increase oxidative stress, which in turn caused an increased need for superoxide dismutase production to establish the reactive oxygen species-antioxidant balance to protect the tissue. The levels of total oxidative stress and SOD values were significantly higher in the chronic periodontitis group and total oxidative stress and SOD levels significantly decreased after periodontal therapy.[3]In the present study, Significant improvement in SOD level in

group I as compared to group II patients may be attributed to adjunctive supplementation of antioxidants. Plasma levels of micronutrients, along with some components of the enzymatic antioxidant system, are found to be increased with multi-nutrient supplementation [38] The increased level of SOD activity after SRP in group II may reduce oxidative stress both locally and systemically. This can be due to decreased level of periodontal pathogens post non-surgical periodontal therapy thereby decreasing the inflammation, which in turn reduces the ROS production and enables the body defense to restore the physiologic pro antioxidant balance. Thus, Successful NSPT is highly effective in restoring SOD level. However, the SOD levels at follow-up in chronic periodontitis patients remained inferior to those of the healthy individuals.[39,40]

**Parth Purwar et al. (2014)**. and **Neha Singh et al. (2013)** suggested that non-surgical therapy improves clinical parameters and increase the antioxidant defense in saliva and plasma in chronic periodontitis patients.[41,42]

The study was conducted in a single center with the availability of a very limited number of samples. To overcome this limitation, we need to perform multi-centered longitudinal studies with large sample sizes to substantiate these results and adequate provisions need to be made to overcome this limitation.

Another limitation of this study is that it was done for a short duration, long-term follow-ups are required to confirm the impact of micronutrients on the outcome of periodontal therapy in postmenopausal women before it can be generally recommended in a clinical setting.

## CONCLUSION:

Postmenopausal women have a low level of SOD activity as compared to other individuals. A decreased activity of antioxidant enzymes increases the levels of reactive oxygen species. Hence, it can be postulated that Menopause is a potential risk factor for the initiation, progression and severity of periodontal disease. Nonsurgical periodontal therapy reduces the levels of oxidative stress associated with periodontal inflammation. Along with that Adjunctive supplementation of antioxidant micronutrients results in the more efficient management of periodontal inflammation by increasing the activity of SOD enzyme in biological body fluid serum and saliva.

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