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Lipid Peroxidation And Superoxide Dismutase Levels In Saliva And Gingival Crevicular Fluid In Chronic Periodontitis Patients Pre And Post Periodontal Therapy

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Abstract

Aim & Objectives: The aim of this study is to estimate the GCF and saliva levels of MDA and SOD in chronic Periodontitis patients Pre and Post Periodontal therapy**.**

Materials And Methods: In this study, 30 chronic Periodontitis patients Pre and Post Periodontal therapy of both gender matching in age and sex were included. The analysis of biochemical parameters was done by using diagnostic reagent kit.

Results: In the present study the Mean of GCF and salivary MDA and SOD was lower in post periodontal therapy patients than pre periodontal therapy patients ($P < 0.001$).

Conclusion: The findings suggest that significant relations are present between oxidant status and periodontal status, and that oxidative stress may play an important role, prominently being a consequence from periodontitis.

Keywords: Malondialdehyde (MDA**),** ROS (Reactive Oxygen Species), SOD (superoxide dismutase),Periodontitis, Oxidative stress, GCF (Gingival Crevicular Fluid)

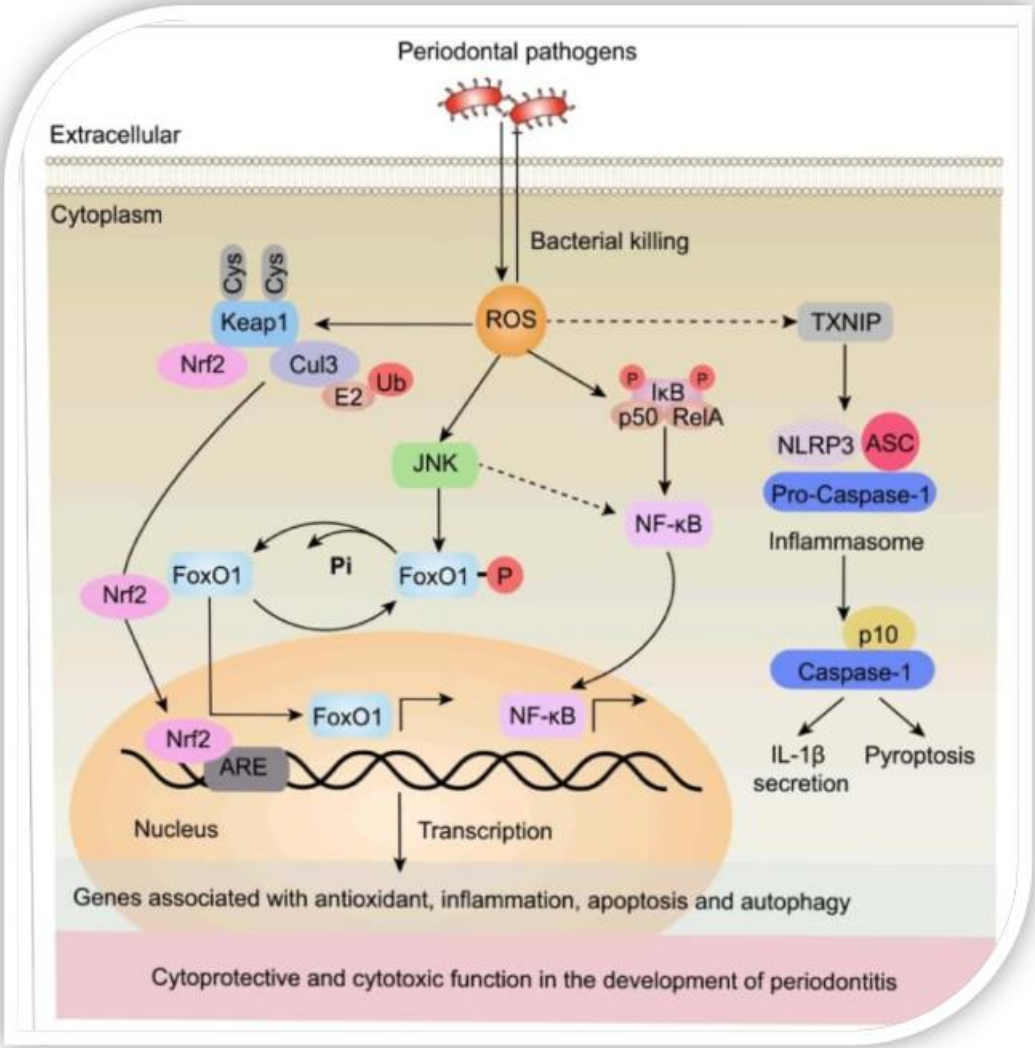
Introduction

Periodontitis is a prevalent inflammatory disease, influencing at least 10% of people worldwide.[1] It can result in the destruction of teeth supporting tissue and ends up with aloss of teeth. In addition, periodontitis has been suggested to have moderate association with several systemic diseases, e.g. cardiovascular disease, diabetes, and adverse pregnancy outcomes.[2] Current concept suggests that this inflammatory disease is initiated by bacterial infection and subsequently progressed by aberrant host response, which mainly contributes to periodontal tissue destruction.[3]

In recent years, reactive oxygen species (ROS) have gained more and more attention because of their central role to the progression of many inflammatory diseases.[4] ROS are described as oxygen free radicals and other non-radical oxygen derivatives involved in oxygen radical production.[5]They are involved in normal cellular metabolism and continuously generated by the cells in most tissues. Another category of substances called antioxidants exist in the cells and can effectively delay or inhibit ROS-induced oxidation.[6] Under physiological conditions, ROS are effectively neutralized by antioxidants, which prevent ROS-mediated tissue damage. When inflammation happens, ROS

production is drastically increased mainly due to cells of innate immune system, e.g. neutrophils and macrophages during the process of phagocytosis via the metabolic pathway of the "respiratory burst" .[4]Subsequently, high levels of ROS cannot be balanced by the antioxidant defense system which leads to the oxidative stress and tissue damage.[6] ROS can directly cause tissue damage involving lipid peroxidation, DNA damage, protein damage, and oxidation of important enzymes; meanwhile they can function as signaling molecules or mediators of inflammation.[7]

Cytoprotective And Cytotoxic Functions In Development Of Periodontitis :



Underlying signaling pathways of ROS regulation in periodontitis. Periodontal pathogen infection can promote ROS generation. In turn, ROS can contribute to the oxidative killing of the pathogens. ROS generated from mitochondria activate the transcription of genes associated with inflammation, apoptosis and autophagy through JNK, NF-κB, and inflammasome-dependent signaling pathways which leads to cytoprotective and cytotoxic effects in the development of periodontitis. ROS activate JNK, which results in the dephosphorylating of FoxO1. ROS have been shown to activate NF-κB in periodontitis. ROS promote excessive inflammation by activating TXNIP, which subsequently activates the NLRP3 inflammasome, elevates the secretion of its substrates such as IL-1β and induces pyroptosis. Meanwhile, ROS interact with cysteine residues in Keap1, disrupting the Keap1-Cul3 ubiquitination system and leading to the release of Nrf2 to the nucleus. In the nucleus, Nrf2 binds to AREs to initiate the transcription of a number of antioxidant genes. Black arrows (↑) and perpendicular lines (⊥)

denote activation and suppression, respectively. Dashed lines denote regulatory relationships that need to be confirmed in periodontitis.[8]

Therefore this study is designed to determine possible associations and relationships between periodontitis and oxidative stress by assessing changes in MDA and SOD levels in GCF and saliva of Chronic periodontitis patient's pre and post periodontal therapy

Material And Methods

This study was carried out in the Department of Periodontics and Department of Biochemistry, Yogita Dental College and hospital khed in collaboration with Department of Biochemistry PIMS&R Islampur, Maharashtra.

This study includes 30 chronic periodontitis patients reported to the the Department of Periodontics after obtaining their informed consent. This study was conducted in age group between 20 to 60 years. The analysis of biochemical parameters was done using standard grade reagent chemicals. MDA levels in saliva and GCF were determined by the method of Young and Trimble [9] and SOD activity was measured by the reduction of nitroblue tetrazolium (NBT) by xanthine–xanthine oxidase system.[10]

The exclusion criteria included subjects of any systemic or metabolic disease, liver disease, vascular diseases, renal artery stenosis, alcoholics, pregnant female and those who were taking any kind of medication in last few years. A record was maintained including current history, diet along with laboratory investigations and previous history of any disease

Distribution Of Study Subjects

Group I	**N = 30 pre periodontal therapy patients.**
Group II	**N = 30 post periodontal therapy patients**

Collection Of Samples

All the samples, prior to and after periodontal therapy, were collected within 48 hours after the clinical measurements in the morning, following an overnight fast. All participants were told not to eat or drink anything or chew gum that morning. The subjects were asked whether they had followed these instructions before samples were collected. Unstimulated whole saliva samples were used in this study. Saliva samples were obtained in the morning, over five-minute periods. Seated patients were instructed to allow saliva to pool in the bottom of the mouth and drain into a collection tube.

During GCF sample collection sites were isolated using cotton rolls and were gently air dried before sampling. It was ensured that the samples were not contaminated by saliva. Collections were performed over 30 seconds with standardized paper strips (Periopaper).

Results

Table no. 1: The mean value of salivary MDA and salivary Superoxide Dismutase (SOD) in chronic Periodontitis patients Pre and Post Periodontal therapy.

Name Of the Parameters	Pre Periodontal therapy Patients (N=30)		Post Periodontal therapy Patients (N=30)		Significance
	Mean ±SD	Std.	Mean ±SD	Std.	

		Error of Mean		Error of Mean	
MDA	0.78 ±0.11 ***	0.021	0.37±0.065	0.011	**P =< 0.001**
Superoxide Dismutase (SOD)	0.19±0.043 ***	0.007	0.092±0.026	0.004	**P = <0.001**

The statistical method uses to compare data was unpaired‘t’ test

*P> 0.05……Not Significant

**P<0.05……...Significant

***P<0.001…...Highly Significant

There is highly statistically significant difference in means of Salivary MDA and SOD (P < 0.001) in pre periodontal therapy patients as compare to post periodontal therapy patients.

In the present study Mean of Salivary MDA and SOD was lower in post periodontal therapy patients than pre periodontal therapy patients (P < 0.001).

Chart I : The mean value of Salivary MDA and salivary Superoxide Dismutase (SOD) in chronic Periodontitis patients Pre and Post Periodontal therapy.

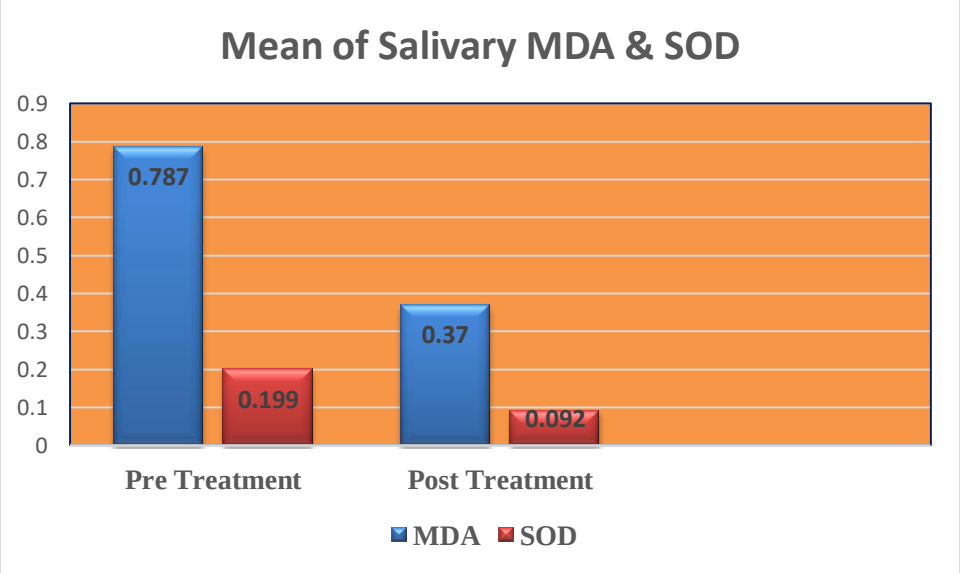


Table no. 2: The mean value of GCF MDA and salivary Superoxide Dismutase (SOD) in chronic Periodontitis patients Pre and Post Periodontal therapy.

Name Of the Parameters	Pre Periodontal therapy Patients (N=30)	Post Periodontal therapy Patients (N=30)	Significance

	Mean ±SD	Std. Error of Mean	Mean ±SD	Std. Error of Mean	
MDA	0.98 ±0.14 ***	0.02	0.66±0.17	0.03	**P =< 0.001**
Superoxide Dismutase (SOD)	0.22±0.02 ***	0.005	0.12±0.02	0.004	**P = <0.001**

The statistical method uses to compare data was unpaired‘t’ test

*P> 0.05……Not Significant

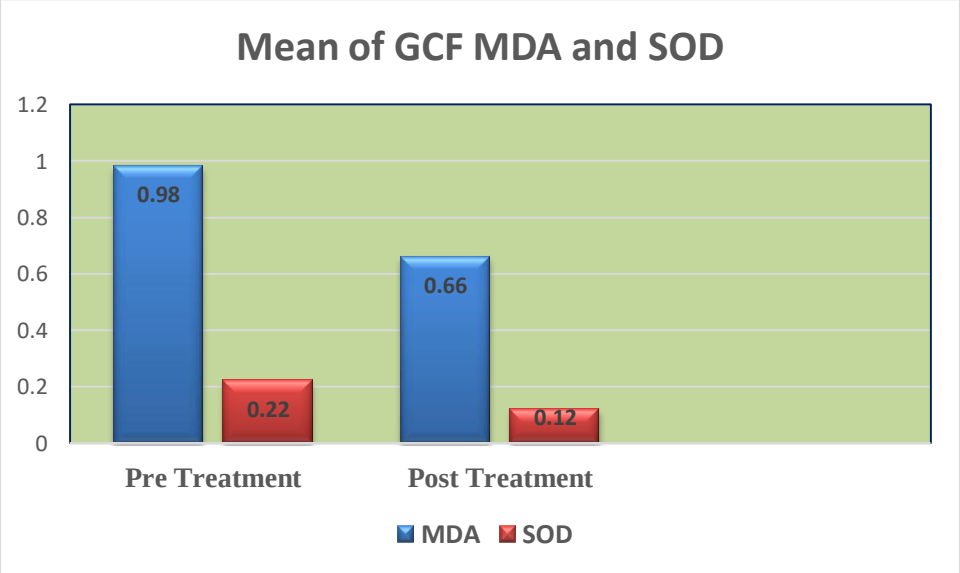
**P<0.05……...Significant

***P<0.001…...Highly Significant

There is highly statistically significant difference in means of GCF MDA and SOD (P < 0.001) in pre periodontal therapy patients as compare to post periodontal therapy patients.

In the present study Mean of GCF MDA and SOD was lower in post periodontal therapy patients than pre periodontal therapy patients (P < 0.001).

Chart II : The mean value of GCF MDA and salivary Superoxide Dismutase (SOD) in chronic Periodontitis patients Pre and Post Periodontal therapy.



Discussion

Patients with periodontal disease are more susceptible to an imbalance of antioxidant oxidative stress situation.[11,12] ROS have a very short life and are therefore not easy to detect. However, ROS-related tissue destruction can be measured by the final product of LPO, such as MDA,[13,14] which is the principal and most studied product of polyunsaturated fatty acid peroxidation. It is worth noting that some studies [15, 16] have indicated that the measurement of 4-hydroxynonenal, acrolein and isoprotane might be more logical than MDA because MDA is only one of many aldehydes formed during lipid peroxidation and can also arise from free radical

attack on sialic acid and deoxyribose. In the present study, we found that the Mean of GCF and salivary MDA and SOD levels was lower in post periodontal therapy patients than pre periodontal therapy patients ($P < 0.001$). Our results are partially in accordance with other studies demonstrating an increased LPO (MDA) and SOD level in GCF of periodontitis patients.[17,18] Nevertheless, Sobaniec and Sobaniec-Lotowska[19] found that rats with periodontitis had higher blood lipid and peroxidation concentrations than periodontaly healthy ones. The increased MDA level in GCF detected in our study suggests an increase in the level of MDA in periodontium and oral environment in periodontitis.

The present data also suggests that MDA concentration in GCF was significantly higher than that in saliva. This is in agreement with Tsai et al. [17] who described a possible association of higher MDA concentrations with an increased percentage of GCF in Comparison to saliva of periodontitis patients.

The present findings indicated lower SOD activity in GCF and saliva in post periodontal therapy patients than pre periodontal therapy patients ($P < 0.001$) This finding confirms several observations in the literature about oxidant–antioxidant imbalance in the pathological process of periodontitis.

However, consistent with our finding, other studies [20, 21] have shown that SOD activity increases with the progression of inflammation in Chronic Periodontitis patients. The human periodontal ligament has been shown to possess the enzyme SOD, which might afford biological protection against ROS, particularly O_2 during the inflammatory response.[22]Bacterial lipopolysaccharide was also shown to stimulate O_2 release from gingival fibroblast, suggesting that the induction of SOD may represent an important defense mechanism of the fibroblast during inflammation.[23] In the present study, increased gingival and salivary SOD activity level in Chronic Periodontitis seems to support the above findings.

Conclusion

The results of the present study suggest that a significant oxidative stress may occur in periodontitis.The findings also suggest that significant relations are present between oxidant status and periodontal status, and that oxidative stress may play an important role, prominently being a consequence of periodontitis. However, further studies are needed to confirm whether oxidant status is a cause of periodontitis which might be targeted for the therapy of periodontitis.

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