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Original Study

Evaluation of oxidative stress levels in patients undergoing endodontic treatment- an in vivo study

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ABSTRACT

Introduction :

Any entity with the aptitude to function independently has one or more unpaired electrons. Free radicals called reactive oxygen species are produced by regular physiological cell processes. Oxidative damage to cellular macromolecules occurs when the formation of reactive oxidative species overwhelms the antioxidant defense of the cells, which can result in a variety of clinical diseases. The destruction of vulnerable lipids and amino acids in proteins at locations has been thought to be the cause of metabolic dysfunction during pathogenesis.

Aim:

To assess the levels of oxidative stress in endodontic therapy patients and to correlate the relationship between malondialdehyde and total antioxidant status in patients before and after endodontic therapy.

Materials and Methods:

Thirty patients who had been undergoing endodontic treatment and met the inclusion criteria had 2 ml of unstimulated saliva collected prior to surgery. After a month following obturation, post-operative samples were drawn and tested for the plasma's ability to reduce ferric iron and the antioxidant strength of thiobarbituric acid reactive substances.

Results:

The total antioxidant status and pre- and post-operative antioxidant levels differed markedly (P value <0.05).

Conclusion:

This study underscores the communication between apical periodontitis and oxidative stress. It is essential to analyse the principles of this parameter to reduce the damage that the tissues may endure due to increased reactive oxygen species. Moreover, through appropriate endodontic treatment, a good oxidative balance can be reestablished, thereby avoiding the danger of having chronic focus, that causes damages with potential generalized consequences.

***Keywords:** Oxidative stress, Reactive oxygen species, Salivary biomarkers, Total antioxidant status.*

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INTRODUCTION

The cell's extremely potent antioxidant systems effectively control reactive oxygen species without causing any negative side effects. A disparity between the production of reactive oxygen species (ROS) and antioxidant defence leads to oxidative stress, which through a series of events deregulates cellular functions and causes a variety of pathological conditions, such as metabolic dysfunction in nearly all vital organs and cardiovascular dysfunction, as well as neurodegenerative diseases and gastroduodenal pathogenesis.[1] Cell death follows the oxidation of DNA, glycoxidation, and membrane lipoproteins brought on by the free radical-mediated oxidative stress.[2,3] The neighbouring cells are attacked by ROS, necrotic agents, and proteases from injured cells, causing tissue injury. It has been noted that significant oxidative stress can result from tissue damage.[4,5] ROS is primarily protected from catalytic elimination.

Living cells include antioxidants, which remove reactive oxygen from inside cells. In addition to defending against potentially detrimental consequences of high oxidative stress, biological antioxidants in human nutrition, intracellular antioxidants, and enzyme systems also prevent several pathological disorders.[6,7] Free radicals are stabilized and made inactive by antioxidants before they can injure cells. They are required to keep cellular and systemic health at its best.

The earliest interaction between dietary components and biological tissues takes place in the oral cavity where saliva, a complex fluid, is present.[8] There are limited investigations on the connection between salivary antioxidants and dental, gingival, and oral disorders, despite the critical relevance of salivary antioxidant defence mechanisms.[9,10,11] Saliva contains countless levels of all enzymatic and molecular antioxidants. The main antioxidant in saliva is uric acid, which is water soluble. Low amounts of the lipid-soluble antioxidants that proteins transport are present10. Oral peroxidase, which is made up of the peroxidase enzymes salivary peroxidase (SPO) and myeloperoxidase (MPO), is the main enzyme in saliva.[12,13] Thiobarbituric acid reactive substance (TBARS), malondialdehyde, and isoprostanes13 are the indicators for lipid peroxidation.

Acrolein is a biomarker for protein denaturation that is measured using the Carbonyl assay.[14,15] Superoxide dismutase and ferric reducing antioxidant capacity are among the indicators for total antioxidant status (FRAP). The purpose of this study was to assess the levels of oxidative stress in endodontic therapy patients and to determine the association between malondialdehyde and total antioxidant status in patients before and after endodontic therapy.

There was no difference between pre-operative and post-operative levels of malondialdehyde and overall antioxidant status, according to the null hypothesis. Another possibility was that the levels of malondialdehyde and overall antioxidant status were different between pre- and post-operative periods.

MATERIALS AND METHODS:

The study was conducted in the Endodontics and Conservative Dentistry Department. Before the study got started, it acquired ethical approval. The study included thirty patients who met the inclusion requirements. Patients must be between the ages of 18 and 45, have a diagnosis of symptomatic apical periodontitis, and have a pain score of at least 5. Among the grounds for exclusion are the existence of any periradicular pathology, before using analgesics, Asymptomatic teeth, smokers, drinkers, systemic diseases, and the presence of several active carious lesions are other risk factors.

Prior to the surgery, the patient's vocal pre-operative pain score was recorded using the Visual Analog scale, and written consent was obtained from them. Prior to the application of local anaesthesia, 2ml of unstimulated saliva was collected. Until it was transported to the lab, the saliva was kept in a sterile container. Saliva was collected prior to surgery, and then local anaesthesia was provided, the access was opened, the working length was completed, followed by cleaning and shape, and a closed dressing was applied. After one week, root canal obturation was completed. After one month, post-operative salivary samples were taken.

The collected samples were tested for thiobarbituric acid reactive substances (TBARS) and ferric reducing ability of plasma (FRAP) (TBARS). The FRAP assay involves mixing 1 ml of saliva with 2.5% phosphate buffer and 1% potassium ferricyanide, and then incubating the mixture at 50C for 30 minutes. After centrifuging it at 6500 rpm for 10 minutes, 2.5 ml of 10% trichloroacetic acid, 2.5 ml of distilled water, and 0.5 ml of 0.1% ferric chloride were added. A UV spectrophotometer was used to measure absorbance at 700 nm. TBARS assay: 1 milliliter of saliva was combined with 200 microliters of 20% trichloroacetic acid, 0.67% thiobarbituric acid, and 2.51% linoleic acid. These were heated for 10 minutes in a water bath, cooled, and then centrifuged at 3000 rpm. After that, it was put through a UV spectrophotometric study at 532 nm.

Statistical Analysis:

Means and standard deviations were individually calculated for each of the parameters used in our investigation. Using the One-Way ANOVA test, a difference in the parameters between the assessment periods was examined. Statistical significance was defined as a P value of 0.05.

RESULTS:

The present study's findings demonstrated that post-endodontic therapy levels of malondialdehyde, a FRAP assay biomarker, were reduced (Table 1). Malondialdehyde levels were lower, which suggests that antioxidant levels have increased during endodontic therapy.

Table1: Measurements of FRAP assay

	Mean	Standard Deviation	P-Value
Pre Op	27.1300	7.38577	0.01
Post Op	29.6850	7.51572	0.01

On subjecting to TBARS assay, the total antioxidant status increased, thereby the oxidative stress levels decreased post endodontic therapy (Table 2).

Table2: Measurements of TBARS assay

	Mean	Standard Deviation	P-Value
Pre Op	36.9150	6.65923	0.012
Post Op	33.8035	7.02003	0.012

DISCUSSION

Chronic apical periodontitis is an inflammatory condition that affects the alveolar bone that supports the teeth's connective tissue, hard tissue, and gums. According to Kornman, the development of periodontal disease is significantly influenced by the biochemical changes brought on by innate immunity, even if the basic cause of the condition is bacterial activation of immune inflammatory processes. The key mediators of the host defence mechanism against spreading periodontal pathogenic bacteria are polymorphonuclear (PMN) leukocytes. Periodontal tissues are destroyed when PMNs get activated because they generate a lot of reactive oxygen species (ROS).[16,17] This study's goal was to determine whether endodontic therapy and levels of oxidative stress are related.

Even though reactive oxygen species have been linked to a varied range of illnesses, endodontic diseases and oxidative stress have not been the subject of any studies. Therefore, the purpose of the current research was to investigate the connection between systemic oxidative stress and apical periodontitis. The null hypothesis was rejected since there was a difference in the levels of malondialdehyde and overall antioxidant status between pre- and post-operative periods. It was established that the levels of malondialdehyde and the overall antioxidant status varied between the pre-operative and post-operative stages.

The findings of this investigation showed that people with chronic apical periodontitis are more vulnerable to oxidative stress in general than the general population. It has been established that this poses a threat to everyone's health. However, a chronic endodontic disease's impact on stages of systemic oxidative stress can be a significant contributory factor in the pathogenesis of some serious systemic illnesses.[18] Up to this point, studies have only evaluated the variations found in the crevicular fluid of patients under investigation. There are not much research that have investigated this topic in literature.

Therefore, since endodontic therapy returns antioxidant status to normal levels, the incidence of oxidative imbalance in patients with chronic apical periodontitis is extremely obvious. Thus, the current study has shown a link between oxidative stress and chronic apical periodontitis. It has been shown by biological evidence that individuals with chronic apical periodontitis have a considerable increase in systemic oxidative stress. The fact that levels in all samples changed during the pre-operative and post-operative stages of treatment was a significant finding of this investigation. This is because as the inflammation is controlled, the body's innate antioxidant level rises, as determined by the FRAP assay, and the level of oxidative stress declines, as shown by the TBARS assay.[19] Additionally, there is a link between the disease's presence and elevated levels of oxidative stress in patients of untreated or undiagnosed apical periodontitis.[20]

CONCLUSION

The correlation between Oxidative Stress and Chronic Apical Periodontitis is highlighted in the current study, making it crucial to examine the standards of this parameter to prevent damage to the periodontal tissues even in the nonexistence of symptoms. These results also suggest that a favourable oxidative balance can be restored with appropriate endodontic therapy, minimizing the possibility of having a chronic focus leading to damages with significant repercussions.

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Conflicts of interest - There are no conflicts of interest.

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