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SALIVARY AND GINGIVAL TISSUE MALONDIALDEHYDE LEVELS IN CHRONIC PERIODONTITIS PATIENTS

Dental Science

KEYWORDS:
Periodontitis,Malondialdehyde,Lipid peroxidation, Gingival tissue.

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ABSTRACT

Background and objectives:-Lipid peroxidation plays a role in pathogenesis of periodontal disease and malondialdehyde is one the end products of lipid peroxidation. The objective of the present study was to estimate and compare levels of malondialdehyde in saliva and gingival tissue of subjects with healthy periodontium and with chronic periodontitis.

Materials and methods:-. A total of 40 subjects participated in the study. The subjects were divided among namely
Group A-Total of 20 subjects aged between 30-55 years who have chronic periodontitis
Group B (control) -Total of 20 subjects with healthy periodontium aged between 15-35 years
The values obtained were subjected to students T-test.

Results:-The results showed that the mean salivary and gingival tissue malondialdehyde levels were significantly higher in Group A as compared to Group B subjects.

Conclusion:-?Thus from this study it can be concluded that malondialdehyde can be used as a diagnostic marker for periodontal disease.

INTRODUCTION:

The presence of unpaired valence electrons makes free radicals and reactive oxygen species (ROS) highly reactive.[9] Lipids, proteins, carbohydrates, and nucleotides in the tissues can be chemically modified and damaged by reactive free radicals.[11] Due to the presence of high amount of polyunsaturated fatty acids in cell membrane they are susceptible to the free radical attack and thus affects the hemostatic environment.[14] Studies have shown that oxidative stress is important in pathogenesis of periodontitis.The saliva and gingival crevicular fluid(GCF) of periodontitis patients have higher levels of non specific markers of lipid peroxidation,a hallmark of oxidative stress. Oxidative stress can result due to uncontrolled production of lipid peroxides, causing significant damage to cell integrity. Malondialdehyde(MDA) is a low molecular weight end product formed via the decomposition of certain primary and secondary lipid peroxidation products.[17] With this background the present study was conducted with following objectives:

1.To assess the levels of malondialdehyde in saliva and inflamed gingival tissues of subjects with chronic periodontitis.

2.To assess the levels of malondialdehyde in saliva and healthy gingival tissues of healthy subjects.

3.To compare the levels of malondialdehyde product in saliva and gingival tissues of subjects with chronic periodontitis.

MATERIALS AND METHODOLOGY

Source of data

40 subjects reporting to the outpatient department and Department of Periodontics of A.B.Shetty Memorial Institute of Dental Sciences, were selected –

Method of collection of data.

This study was designed as a case-control study comprising of 40 subjects, inclusive of both sexes and were divided into two groups of 20 patients each

Group A-Total of 20 subjects aged between 30-55 years who have chronic periodontitis.
Group B (control) -Total of 20 subjects with healthy periodontium aged between 15-35 years.

Criteria for selection:

Inclusion Criteria

1.Subjects who have a pocket of probing depth of ≥ 4 mm and loss of attachment of ≥ 3 mm. (For Group A)
2.Subjects should have minimum of 20 teeth.(For Group A)
3.Subject's who have given their informed consent to participate in the study.

Exclusion Criteria

1. Subjects with any systemic disorders .
2. Patients who have received periodontal therapy/antibiotics/anti-inflammatory drugs/ steroids in the past 6 months .
3. Pregnant women, lactating women
4. Subjects who are tobacco users.

A standard proforma consisting of the following data: name, age, sex, medical and past dental history, plaque index (Sillness and Loe), periodontal pocket and clinical attachment for each patient was recorded. Each patient was examined using a mouth mirror and Williams graduated periodontal probe under artificial light.

Method of collection of sample:

Collection of saliva-

After obtaining the subject's consent to participate in the study unstimulated whole saliva samples was collected by expectoration. Subjects were asked not to eat or drink 1 hour prior to the study. Whole saliva samples was used in the study. Saliva samples was obtained in the morning over 5 minutes period with patients seated with instructions to allow saliva to pool in the floor of the mouth. Collected sample was sent immediately for biochemical analysis.[1]

Collection of gingival tissue-

A gingival tissue sample was obtained under local anesthesia, from the subjects undergoing extraction of periodontally diseased tooth /teeth and the healthy tissue samples was collected from patients undergoing extraction for orthodontic reasons. This sample was stored in phosphate buffer and sent immediately for biochemical analysis.

BIOCHEMICAL ANALYSIS:

For estimation of lipid peroxidation levels:-

Thiobarbutaric acid reactive substance estimation.

0.5 ml sample + 2.5 ml of 20% Trichloric acid + 1ml of Thiobarbuteric acid.This mixture was heated in boiling water bath for 30 mins and then cooled in cold water for 10 min. Chromogen was extracted in 4 ml butanol by centrifugation at 3000 rpm for 10 min. Adsorbance was determined at 530 nm using a spectrophotometer.

RESULTS:

Data was statistically analysed using unpaired t test. SPSS version 17 & MS Excel was used to analyse the data. p<.05 was considered to be statistically significant.

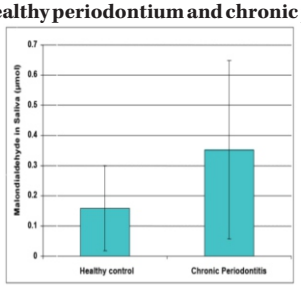
Malondialdehyde saliva (µmol)

group	N	Mean	Std. Deviation	Mean diff	p
Healthy control	20	0.15855	0.140787	0.194300	<.0005 vhs
Chronic Periodontitis	20	0.35285	0.111994		

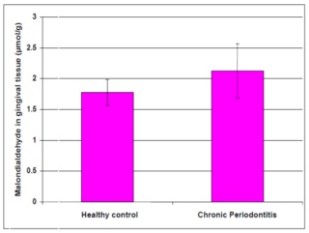
Malondialdehyde gingival tissue (µmol/g)

group	N	Mean	Std. Deviation	Mean diff	p
Healthy control	20	1.78040	0.209347	0.34745	0.004
Chronic Periodontitis	20	2.12785	0.441783		

Comparison of mean salivary malondialdehyde between subjects with healthy periodontium and chronic periodontitis



Comparison of mean gingival tissue malondialdehyde between subjects with healthy periodontium and chronic periodontitis



DISCUSSION

Oxidative stress has a major role in intiating periodontal disease which results from interactions between the host and the pathogenic microbe, which can either be directly result of increased ROS activity /antioxidant deficiency or indirectly as a result of the activation of redox-sensitive transcription factors and the creation of a pro-inflammatory state.[7]

The majority of research work of biomarkers and periodontitis have used GCF as a sample fluid. But collection of GCF is extremely tedious and only shows information about the inflammation at site from which sample has been collected. But on the other hand as the quantity of saliva present is quiet abundant and its collection is much simpler .In addition to this whole saliva shows the state of inflammation from all the sites having periodontitis.[12]

The essential feature of normal cellular metabolism is generation of reactive oxygen species.[10]Nevertheless, they are also highly toxic and damage biological molecules including DNA,proteins and lipids. ROS induce lipid peroxidations (LPO), with related effects on cells. The process of LPO begins when ROS interact with the polyunsaturated fatty acids in membranes or lipoproteins. Excess generation of lipid peroxides can lead to oxidative stress, with marked damage to cell integrity. Since LPO is produced due to oxidative stress, various markers have been used to check this process.

Malondialdehyde (MDA) is the chief and most researched product of polyunsaturated fatty acid peroxidation which can be increased following oxidative stress. Free radical induced LPO, because of high molecule reactivity, has been implicated in the pathogenesis of several pathological disorders including periodontal disease.[18]

Concentration of LPO was found to be increased in gingival tissues of rats with experimental periodontitis has been reported by Sobniec et al.[15]In another study it was demonstrated that the higher levels of TBARS was seen in gingival tissue and plasma of chronic periodontitis patients as compared to healthy subjects .[13]

Cimasoni (1974) has suggested that saliva LPO levels might be used as an indicator of periodontal damage.[4]In a study by Akalin et al., very high levels of MDA was found in the saliva of patients with periodontitis as compared to that of healthy control subjects ($P < 0.05$).[1]In an another study by Tsai et al., had shown a significantly high concentration of LPO product in saliva of patients with periodontal disease as compared to healthy subjects ($P < 0.005$).[16]

In the present study an attempt was made to evaluate and compare levels of lipid peroxidation product-MDA in saliva and gingival tissues of subjects with and without chronic periodontitis using spectrophotometric quantification. The results of this study demonstrated significantly higher levels of malondialdehyde in saliva and gingival tissue of subjects with chronic periodontitis as compared to healthy control subjects and is consistent with the investigations which reported higher levels of MDA in saliva[13,1,16,6] and gingival tissue[15,13] of chronic periodontitis subjects.

Owing to the few number of studies involving assessment of salivary MDA and MDA in gingival tissues,this study suggests that MDA levels could be used as marker to check for the status of periodontal health.

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