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Original Research Article

Effect of non-surgical periodontal therapy on saliva levels of paraoxonase-1 in healthy and periodontitis subjects: a clinico-biochemical study

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ABSTRACT

Background: Salivary antioxidants play main roles in the defensive mechanism of saliva against free radicals. Prevalence of inflammatory conditions like periodontitis associated with several pathogeneses, knowing the defensive mechanism of salivary antioxidant against free radicals can provide helpful strategies in diagnosis and evaluation of these conditions. The aim of this study was evaluation of the paraoxonase (PON1) levels of saliva in healthy and periodontitis subjects.

Methods: In this clinico-biochemical study 30 patients diagnosed with periodontitis and 30 healthy controls aged between 18 to 60 years, 30 patients were recalled after non-surgical periodontal therapy were selected. Three millilitres (3 ml) of whole unstimulated saliva sample were collected. PON-1 level were determined by a commercially available enzyme-linked immunosorbent assay (ELISA) kit.

Results: The results showed a statistically significant difference in the salivary PON1 levels in pre (66.6600%) and post (89.3200%) treatment samples (p value <0.001). The saliva level of PON1 was improved compared to post treatment. There was an improvement in the clinical parameters, and seen a statistically significant difference with p=0.001.

Conclusions: The reduction in the saliva levels of paraoxonase in periodontitis patients has led to a variation leading to increased ROS and therefore periodontal breakdown. The variation in salivary paraoxonase levels before and after treatment can be taken as an indicator of success of periodontal therapy.

Keywords: High-density lipoproteins, Paraoxonase, Periodontitis, Reactive oxygen species

INTRODUCTION

Periodontitis is a chronic inflammation of the supporting structures of the teeth, together with alveolar bone, cementum and periodontal ligament (PDL) tissue, and shows dreadful destruction of the tooth-supporting tissues, directing to tooth loss, that strikes 10-15% of the developed world population and is the extensive cause of tooth loss in adults.[1,2] The divergent response is characterized by exaggerated inflammation, comprise the release of excess proteolytic enzymes and reactive oxygen species (ROS).[3]

In well balanced state there is a potent equilibrium between ROS activity and antioxidant defence capacity and when that equilibrium shifts in favor of ROS, either by a reduction in antioxidant defences or an increase in ROS production or activity, oxidative stress results.[4] Oxidative stress as a disruption in the pro-oxidant antioxidant balance in favor of the former, directing to potential damage.[5] A growing body of corroboration implicates oxidative stress in the pathobiology of periodontitis.[6] Tissue damage emerge directly from oxidative stress and also indirectly via activation of redox-sensitive gene transcription factors like nuclear factor k-B, which in turn leads to downstream proinflammatory cytokine/chemokine production.[7]

There are various notable antioxidant systems against oxidative stress counting paraoxonase (PON1).[8] Mammalian paraoxonases are unique, highly sustained family of calcium-dependent esterases which includes a family of three enzymes called PON1, PON2 and PON3, located on chromosome 7q21.3-22.1.[9,10]

PON1 and PON3 are synthesized predominantly in the liver from which PON1 and some PON3 are secreted into the blood and incorporated with high-density lipoproteins (HDL). PON2 is not in serum but is expressed in many tissues and cell types.[9]

PON1 is a most explored member of a family of enzymes with high-density lipoprotein-associated esterase/ lactonase, and organophosphatase activity that are widely diffused in mammals, such as rat, rabbit, and mouse, as well as humans.[11,12]

PON1 play many roles such as shielding against oxidative damage and lipid peroxidation, contribution to innate immunity and regulation of cell proliferation/apoptosis. Since they are competent to perform multiple autonomous and often unrelated functions, they are contemplated as "moonlighting proteins".[13]

lactonase activity may constitute an important prevalent feature of the PON enzymes.[14] Which is suggested to be the indigenous ancestral activity of the PON family.[15] In Gram-negative bacterium *Pseudomonas aeruginosa* produces acyl-homoserine lactone (AHL) quorum-sensing (QS) signalling molecules, which regulates the expression of extracellular virulence factors and biofilm formation.[16] PON1 degrades 3OC12-HSL by hydrolysing its lactone ring, whereby blocking porphyromonas aeruginosa quorum-sensing signaling.[17]

PON1 has been broadly studied in human medicine. Earlier the attentiveness on this enzyme emerges from the toxicological point of view, by its solicitous role from poisoning by organophosphate derivates. But more latterly research has been fascinated on other clinical aspects such as protective role in vascular disease and use as biomarker of diseases included mainly three situations: (a) oxidative stress, since PON1 act as safeguard against oxidation; (b) inflammation, being considered PON1 as a negative acute phase protein (APP) and (c) liver diseases.[18] PON1 gene polymorphisms have been connected with various human diseases, including coronary heart disease, Parkinson's disease, type 2 diabetes and inflammatory bowel disease.[19]

Saliva has become a standard diagnostic fluid for research and clinics in current years.[20] Saliva being readily available makes the collection process fairly straightforward, even when multiple samples are needed. Its collection is non-invasive making the procedure more acceptable to patients and more conducive to a stress-free appointment.[21]

To the best of our knowledge there were scanty of literature available on salivary PON-1 expression in periodontitis subjects. Therefore, the present study was done to assess the effect of non-surgical periodontal therapy on PON-1 at base line and follow up at 6 weeks of NSPT. Investigation might aid in early detection and proper treatment planning of the periodontal diseases.

METHODS

A total of 60 subjects of age ranging 18-60 years were randomly selected from the outpatient department of periodontics, PMNM Dental College and Hospital, Navanagar, Bagalkot. 30 periodontally heathy subjects, 30 subjects diagnosed as periodontitis and the same were recalled after 6 weeks after non-surgical periodontal therapy. The study was approved by Institutional Ethical Committee of PMNM Dental College and Hospital, Navanagar, Bagalkot. (PMNMDCH/1854/2020-21)

Inclusion criteria and exclusion criteria

Subjects willing to participate in the study and given written consent for the same, patients aged 18-60 years, both males and females who are periodontally healthy, subjects showing absence of clinical manifestations of periodontal disease, patients diagnosed as periodontitis were included in the study.

Patients with systemic diseases, and who had undergone periodontal therapy in the past 6 months, patients on any systemic antibiotics, anti-inflammatory, hormonal therapy, subjects having tobacco in any form, immunosuppressed subjects, history of radiotherapy, wearing complete dentures, having other infections or pathology in oral cavity other than gingivitis and periodontitis were excluded from the study.

A proforma consisting of name, age, sex, medical and dental history, simplified oral hygiene index (OHI-S) (John C. Green and Jack R. Vermillion, 1964), bleeding on probing (Muhlemann and Son, 1971), periodontal disease index (Ramfjord 1959), probing pocket depth (PPD), clinical attachment loss (CAL) was prepared. Clinical examination was done in all the subjects and the above-mentioned parameters were recorded.

Method of collection of samples

Three millilitres (3 ml) of whole unstimulated saliva samples were collected from healthy and periodontitis subjects at base line and after 6 weeks of non-surgical periodontal therapy. On the day of examination samples were collected around 10 a.m. into the 3 ml Eppendorf tube every 60 seconds for 10 minutes or when the subject experiences an urge to swallow the fluid accumulated in the floor of the mouth.[21] The collected saliva samples were centrifuged at 1000xg for 15 minutes at 2-8°C, to remove the cell elements and plaque, and then stored at ≤ -80°C until required. Salivary PON-1 was measured using the human paraoxanase ELSIA kit (Krishgen Biosystems, India) catalogue number KBH2157.

Statistical analysis

Normality of numerical data was checked using Shapiro-Wilk test. Inter group comparison was done using t-test. Descriptive statistics like frequency (n) and percentage (%) of categorical data, mean and standard deviation of numerical data in each group was depicted and compared using chi square test. Intra group comparisons was done using a non-parametric substitute like Wilcoxon signed rank test (for 2 observations). Keeping alpha error at 5% and beta error at 20%, power at 80%, p<0.05 was considered statistically significant. Approximately 29 subjects per group should complete the study at the endpoint follow up, to avoid loss to follow up/attrition.

RESULTS

A total of 60 subjects consisting of females (n-24), males (n-36) were taken for this study. Among healthy group 46.7% were females and 53.3% males. The mean age of the people in the healthy group was equal to 35.07±10.471 and in periodontitis group was equal to 35.57±12.235.

Table 1: Comparison of the various periodontal parameters in terms of mean (SD) at different time intervals in periodontitis group.

Variable	Time interval	N	Mean	SD	t value	P value
Oral hygiene index simplified (OHIS)	Pre	30	3.517	1.2576	10.964	<0.001**
	Post	30	1.760	0.8916		
Bleeding on probing (BOP)	Pre	30	3.850	0.9299	12.433	<0.001**
	Post	30	2.370	0.8005		
Plaque component (PI)	Pre	30	4.388	0.9218	15.086	<0.001**
	Post	30	2.787	0.8140		
Calculus component (CI)	Pre	30	4.287	0.8476	13.455	<0.001**
	Post	30	2.593	0.8370		
Gingival and periodontal component (PDI)	Pre	30	4.120	0.9590	13.490	<0.001**
	Post	30	2.597	0.8708		
Pocket probing depth (PPD)	Pre	30	4.060	0.7532	15.765	<0.001**
	Post	30	2.533	0.7453		
Clinical attachment loss (CAL)	Pre	30	4.700	0.8773	16.541	<0.001**
	Post	30	2.987	0.7219		

Among the studied clinical periodontal parameters in healthy group OHIS score was equal to 0.770±0.4010 and BOP score was 0.220±0.3699. In periodontitis group after non-surgical periodontal therapy OHI-S, BOP, PI, CI, PDI, PPD, CAL scores were reduced (Table 1). There was a significant difference according to the result of t-test observed between the two groups in terms of p<0.001. Salivary PON1 levels also decreased significantly alongside disease from baseline to after treatment.

The results of the Wilcoxon signed rank test depicted in Table 2 the salivary levels of PON-1 equal to 66.6600 mean (SD) at baseline in periodontitis subjects and 89.3200 after 6 weeks after non-surgical therapy, results showed statistically significant difference in the salivary PON1 levels in pre and post treatment samples (p value <0.001). The saliva levels of PON1 were improved compare to pre and post treatment.

Table 2: Comparison of the saliva levels of paraoxonase-1 in terms of mean (SD) at different time intervals in periodontitis subjects using Wilcoxon signed rank test.

Time interval	N	Mean	SD	Z value	P value
Baseline	30	66.66	58.06078	2.581	0.010*
After 6 weeks	30	89.32	74.39338		

(p<0.05- significant*, p<0.001- highly significant**).

DISCUSSION

The antioxidant enzyme PON1 is mainly secreted by the liver, and in the diffusion bound to HDL transferred to several tissues. These are safe-guard lipids against peroxidation and ward off low-density lipoprotein oxidation, which is a major origin of inflammation which is involved in the commencement of inflammatory diseases such as atherosclerosis and diabetes. Mainly, the PON1 impede the oxidation of LDL, cell membrane lipids hence anti-inflammatory properties.[22]

There was no statical difference of sex distribution between control (16 men, 14 women) and patients with periodontitis (20 men, 10 women) groups in the present study. These results were similar to Baþkol et al, where there was no statistically significant difference of age and sex distribution between patients with osteoporosis and control group and also study conducted by Raadi et al, there was no significant difference in terms of gender.[8] In our study there was unequal distribution of subjects that could have resulted in the non-significant relation between the groups.

Among the studied clinical periodontal parameters, the periodontally healthy group presented the lowest oral hygiene index simplified score (OHI-S). Periodontitis patients presented the highest values. Socransky et al, published that the microflora in plaque is a series of successive waves of colonization by increasing periopathogenic bacteria, culminating in the triumvirate of *Porphyromonas gingivalis* (*P. gingivalis*), *Treponema denticola* and *Tannerella forsythia* (*T. forsythia*).[23] Tahir et al, illustrated in their study NSPT was shown to induce a shift from a pre-dominant Gram negative to a Gram-positive subgingival microbiota in the general population and to significantly reduce total bacteria.[24] Our present study results showed reduction in OHI-S score after 6 weeks, revealing the reduction in the microbial load count to achieve equilibrium between residual microbes and host response to restore gingival health.

In the present study we obtained higher BOP scores in periodontitis group than healthy group, scores were reduced after 6 weeks of NSPT. Umeda et al, at their, clinical reports suggested that NSPT reduces the total number of gingival sites that bleed during probing, facilitating a transition of oral microbiota from gram-negative to gram-positive bacteria.[25] Additionally, NSPT reduces the black-pigmented species and spirochetes, with a concomitant increase in coccoid cells. In our study periodontitis subjects had higher values of BOP compared to subjects recalled after 6 weeks of NSPT. Reduction in microbial load after NSPT suggests improvement in the inflammatory component of gingival epithelium and connective tissue.

Pocket Probing Depth score was more in periodontitis group compared to periodontitis subjects after 6 weeks of NSPT. Studies conducted by Chen et al, have showed the nonsurgical phase scaling and root planing considerably reduce Gram-negative microbes such as *Porphyromonas gingivalis*.[26] In our study after successful NSPT there might be shift towards a more dominant population of Gram-positive microbes and which correlates with a reduction in the pocket depth associated with gingival health.

Clinical attachment loss score in periodontitis group and periodontitis after 6 weeks of NSPT, there was a significant difference seen between the two groups ($p<0.01$). Our study was in accordance with the study done by Hamdi et al, where they got statistically highly significant ($p<0.001$) between the study group and control group.[27] All the clinical parameters (PI, CI and PDI) showed statistically significant intergroup analysis with periodontitis and periodontitis after 6 weeks of NSPT. Healthy subjects showed statistically significant with least PI, CI and PDI score. Some studies that inspected either PON1 levels or PON1 status (plasma PON1 activity levels) have showed that low PON1 levels are a risk factor for vascular diseases.[28]

The study done by Cagirci et al, perceived lower serum PON1 activity in patients with calcific aortic valve stenosis by using spectrophotometry and found that this phenomenon positively corresponded with disease severity.[29] Also, Mackenzie study in which both serum PON-1 concentration and activity in infected individuals of type 2 diabetes was reduced compared to the control group.[30]

Study conducted by Abbott and Ikda described PON-1 decreases in patients with diabetes, although the decrease in PON-1 in this study is alike to our study, but in this study, the concentration of PON-1 was measured.[31,32] In our study, the amount of enzyme activity has been studied.

Compared with the controls, in our study, periodontitis patients showed a significant decrease of PON1 activity. The low PON1 activities in periodontitis patients may result from the damage of PON1 proteins by the action of high amounts of reactive oxygen species produced in periodontitis patients.

Preliminary studies have showed that IL-6 and TNF-α (which is produced in periodontitis disease) have impaired PON1 gene expression, which put forward that inflammatory marker might have an effect on PON1 activity.[33]

Most of the previously mentioned studies, reported that the association among PON1, inflammation and oxidative stress, which can occur during the development of inflammatory conditions.[34] However, PON1 can be affected by other inflammatory disorders like in ulcerative colitis patients, characterized by inflammation and oxidative stress, serum PON1 levels were found to be decreased, and their activity was found to be reduced in ulcerative colitis and Crohn's diseases.[35]

The increased PON levels may be due to reduction in oxidative stress after scaling and root planing, which is in line with a study conducted by Kinane et al, that averted those inflammatory condition like chronic periodontitis is predominantly correlated with increased oxidative stress. An increase in ROS particularly with neutrophils being involved in the pathogenesis because of the creation of oxidative burst during phagocytosis and killing.[36]

PON1 activity was corresponded well with disease severity and inflammatory response, it would be fascinating to evaluate its potential as a predictor of disease eruption/remission in follow-up studies.[37]

The smaller sample size, evaluation of PON1 in other differences such as race and population variations, which does not allow us to generalize these findings on to the entire population. Therefore, further studies with larger sample size, evaluation of PON1 in saliva and a multi-center level study are required to consolidate the findings of the present study .

CONCLUSION

There is certainly a necessity of further studies in order to illuminate the role of PON1 in numerous clinical conditions allied with oxidative stress and inflammation, in addition its use of biomarker for diagnosis and treatment of these diseases. Overall, this research study could uplift researchers to explore the wide areas of PON1 in periodontal medicine.

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