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Estimation of salivary uric acid as a biomarker to determine the antioxidant capacity of saliva in smoker and non-smoker periodontitis patient

[1]Dr. Amrita Mishra, PG Student, Department of Periodontology, Sri Aurobindo College of Dentistry, Madhya Pradesh Medical Science University, Indore, M.P.

[2]Dr. Kanteshwari IK, Professor and Head of Department, Department of Periodontology, Sri Aurobindo College of Dentistry, Madhya Pradesh Medical Science University, Indore, M.P.

[3]Dr Gagan Jaiswal, Professor, Department of Periodontology, Sri Aurobindo College of Dentistry, Madhya Pradesh Medical Science University, Indore, M.P.

[4]Dr Rajesh Kumar, Professor, Department of Periodontology, Sri Aurobindo College of Dentistry, Madhya Pradesh Medical Science University, Indore, M.P.

[5]Dr Heena Agrawal, Senior Lecturer, Department of Periodontology, Sri Aurobindo College of Dentistry, Madhya Pradesh Medical Science University, Indore, M.P.

[6]Dr Krati Bakhliwal, PG Student, Department of Periodontology, Sri Aurobindo College of Dentistry, Madhya Pradesh Medical Science University, Indore, M.P

Corresponding Author: Dr. Amrita Mishra, PG Student, Department of Periodontology, Sri Aurobindo College of Dentistry, Madhya Pradesh Medical Science University, Indore, M.P.

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Abstract

Introduction: Progression and severity of periodontitis depend on interaction between several risk factors including smoking. Release of reactive oxygen species is exaggerated in smokers along with reduced production of antioxidants. Assessment of Uric acid in saliva can serve as biomarker to determine the influence of smoking in periodontitis patients.

Aim: Estimation of salivary uric acid as a biomarker to determine the antioxidant capacity of saliva in smokers with Periodontitis

Objectives (i) To assess the influence of smoking in salivary and serum uric acid levels in smokers vs nonsmokers with periodontitis.

(ii) To assess the correlation between serum and salivary uric acid in both groups. Results - Mean salivary uric acid levels 3.53 ± 0.68 mg/dl, and 2.87 ± 0.57 mg/dl were

observed among, non-smokers with Periodontitis, and smokers with Periodontitis groups, respectively. Significantly higher mean salivary uric acid levels in non-smoker group were observed as compared to smoker with Periodontitis.

Conclusion: Smoking has a negative impact in the antioxidant capacity of saliva as we found significant reduction in the level of salivary uric acid level hence worsening the host defense and ultimately periodontitits.

Keywords: Periodontitis, smokers vs nonsmokers, saliva

Introduction

Periodontitis is one of the most widespread chronic inflammatory conditions that affect the tooth supporting structures. It is caused by an intricate interactions between periodontal micro biota and host defense mechanisms(1). Certain periodontopathic bacteria like P. Gingival is provoke the release of certain cytokines like IL-8 and TNF α resulting in an increase in the number and activity of polymorpho nuclear leucocytes (PMN). In response to microbiological insult the host defense cells i.ePMN liberate the singlet oxygen via the respiratory burst (2). This host microbial interaction results in abundant release of reactive oxygen species leading to heightened oxidative damage, connective tissue and bone loss(3). In healthy individuals antioxidants are found in all bodily fluids and tissues, where they protect against free radicals formed endogenously. In conditions like periodontitis this balance is disturbed and total antioxidant capacity of the body fluids is decreased and periodontal disease progression occurs. Along with local factors and specific periodontopathic bacteria certain other risk factors like smoking contribute in the modification of the host response towards periodontal disease (4).Cigarette smoke constitute various free radical ,oxidants ,pro-oxidants, superoxide and reactive nitrogen species that readily react with various biomolecules and cause oxidative stress accounting for decrease host response and delayed wound healing(5). Saliva, being the primary bodily fluid to come in contact with ingested food, drinks, inhaled cigarette smoke (CS), microorganisms, etc., is the first defense against oxidative stress through its antioxidant activity(6).In humans, the most abundant aqueous antioxidant is uric acid, amounting to sixty percent of serum free radical scavenging capacity, and it's action as intracellular free radical scavenger becomes important during metabolic stress, including smoking(7).Thus measurement of uric acid's serum level, indicates the antioxidant capacity. Uric acid present in saliva displays a concentration similar to that of serum. In both smokers and patients with periodontitis the antioxidant capacity is reduced. Uric acid being the most abundant antioxidant, its salivary levels could serve as a prognostic biomarker. Thus the aim of the study is to estimate the potential effect of smoking and periodontitis on salivary and serum uric acid concentrations and to establish the relationship between the two.

Materials and Method

A total of 30 subjects in the age range of 30-65 years with chronic periodontitis were selected from the outpatient department of periodontology at Sri Aurobindo college of dentistry, Indore based on the inclusion and exclusion criteria. After obtaining the approval of the institutional ethical committee all participants were informed about the study procedure. An informed consent form, agreeing to the required treatment plan was obtained. Patients of both sexes who satisfied the study criteria took part in the study. Randomization was followed for allocation of selected 30 subjects in two groups:

Group I: 15 Nonsmokers with chronic periodontitis

Group II: 15 Smokers with chronic periodontitis.

Inclusion criteria

• Current smokers (subjects smoking more than 20 cigarettes /day) within age group of 30-65years having minimum of twenty permanent teeth with probing pocket depth of ≥ 4mm.

• Nonsmokers within age group of 30-65years having minimum of twenty permanent teeth with probing pocket depth of ≥ 4mm.

Exclusion criteria

• Individuals prescribed antibiotic or anti-inflammatory therapy during past 3months. Subjects with high level of uric acid (hyperuricemia)

• Patient who had periodontal surgery in the past 6 months.

• Lactating females

• Pregnant females.

Saliva collection and biochemical analysis

Collection of non-stimulated whole saliva was done in the morning between 8-10 am 2-3 hours after the breakfast to avoid circadian variations.

No attempt was made to stimulate saliva prior to collection.

Group A participants were requested to refrain from smoking for 60 min before salivary collection. In sitting position, subjects were requested to swallow saliva then stay immobile while permitting the saliva to flow passively for 10 min .Five milliliter of un stimulated whole expectorated saliva from each subject was collected into sterile containers after a single mouth rinse with 15.0 ml of water .Estimation of uric acid was done using standard AGAPEE Kit.(9)

Estimation of serum uric acid- 10ml intravenous blood was withdrawn (by a venipuncture of the antecubital vein) and was collected in the sterile centrifuge glass tube .The tubes were centrifuged at 3000rpm for 10mins. At the end of the centrifugation serum was separated. Obtained serum was then used for the assays by standard uricase method. And then concentration of uric acid was calculated.

Statistical analysis: Normality distribution of the data was assessed by applying Kolmogorov-Smirnov tests. It showed significant P value ($P < 0.05$).Suggestive of normal distribution of data. Descriptive statistics of mean and standard deviations and the correlation between serum and salivary uric acid were calculated using un- paired t-test.

Results

A total of 24 males and 06 females participated in the study Mean age of the study participants ranged from 35to 65 years.

Table 1: Gender distribution of the study subjects in different groups

Groups	Gender (n⁄)	
	Male	Female
GroupI	09	06
Group II	15	00
Total	24	06

Table 2: The mean levels of salivary uric acid of, 3.53 ± 0.44 mg/dl, and 2.87 ± 0.43 mg/dl was observed among non-smokers with periodontitis, and smokers with periodontitis, respectively. Comparison of mean salivary uric acid levels in different groups showed statistically significant difference.

Table. No. 2 Comparison of mean salivary uric acid level among different groups (mg/dl)			t-value	p-value
Groups	**Study subjects (n)**	**Mean salivary uric acid levels**		
Group (I)	15	3.53 ± 0.44	2.03	0.025
Group (II)	15	2.87±0.43		

Table 3: The mean levels of serum uric acid of 3.57 ± 0.57 mg/dl, and 3.05 ± 0.68 mg/dl was observed among non-smokers with periodontitis, and smokers with periodontitis, respectively. Comparison of mean serum uric acid levels in different groups showed statistically significant difference $P < 0.05$.

Table. No. 3 Comparison of mean salivary uric acid level among different groups (mg/dl)			t-value	p-value
Groups (n)	**Study subjects (n)**	**Mean serum uric acid levels**		
Group I	15	3.57±0.57	1.98	0.028
Group II	15	3.05 ±0.68		

Table 4: correlation between the salivary and serum uric acid levels in both the groups

Groups	Group I	Group II	t-value	p-value
Salivary uric acid level	3.53 ± 0.44	2.87±0.43	2.03	0.025
Serum uric acid level	3.57± 0.57	3.05 ± 0.68	1.98	0.028
t- value	-0.20	-0.84		
p- value	0.45	0.20		

Discussion

The role of Reactive Oxygen Species (ROS) in physiological and immune inflammatory reactions is well known. The imbalance between oxidative stress induced by ROS and the concentrations of the antioxidant could lead to oxidative damage(10). Previous researches by Kaufman et al 2000 and Sculley Dv etal in 2002 has suggested that patients with periodontitis are more vulnerable to an imbalance of antioxidant-oxidative stress situations(11) (12). Presence of high concentration of oxidative species in cigarette, makes smoking a momentous source of oxidative stress and an important risk factor for Periodontist (13). Assessment of ROS can serve as a diagnostic tool in early identification of the disease. But as ROS have a short half-life and are thus difficult to quantify in vivo (14). Saliva being a biological fluid is a rich source of antioxidants like uric acid and offer an advantage of sample matrix because it is non-invasive and allows for limitless sampling in a given subject as well as sample collection of large cohorts(15).Based on this information estimation of salivary uric acid levels in smokers and non-smoker periodontitis patients is utilized in this study as a prognostic marker for assessing the effect of smoking in periodontal disease and correlating it with serum uric acid .

Table no. 1 shows the distribution of the gender in the study subjects a total of 24 males and 06 females

participated in the study within the age range of 35 to 65 years. Out of which 09 males were in the nonsmokers group along with 06 females and smoker group constituted of 15 males and no females.

Table no. 2 depicts the mean levels of salivary uric acid 3.53 ± 0.49 mg/dl, and 2.87 ± 0.43 mg/dl was observed among non-smokers with periodontitis, and smokers with periodontitis, respectively. Study results revealed significantly lowered salivary uric acid level among smokers with periodontitis as compared to nonsmokers with periodontitis. Results obtained in our study is consistent with the previous work done by Fatima et al in 2016 , Greabu et al in 2007 Kardesler etal in 2006 and Calsina etal in 2002(9)(14).The possible explanation of this finding is that the smoking certainly alters the periodontal response to microbial challenge. It is well recognized that cigarette smoke contains a large amount of oxidative species and therefore smoking represents a substantial source of oxidative stress (15). The direct pro-oxidant burden of tobacco smoke can contribute to reactive oxygen species-mediated tissue damage through depletion of systemic endogenous antioxidant capacity. Both tobacco smoke and inflammation are sources of reactive oxygen species that could have compromised the antioxidant capacity of saliva. On contrary to our findings the study conducted by Zappacosta et al in 1999 studied the acute influence of smoking a single cigarette on salivary uric acid levels and found no significant difference between smokers and non-smokers(16). And study by Kondakova et al in 1999 showed no statistical differences between uric acid values in the saliva of smokers and non-smokers. The study also found that smoking a cigarette had no impact on the salivary antioxidants level.(5)

Table no. 3 displays the mean serum uric levels .The mean levels of serum uric acid of 3.57 ± 0.57 mg/dl, and 3.05 ± 0.68 mg/dl was observed among non-smokers with periodontitis, and smokers with periodontitis, respectively. Study results revealed significantly lowered serum uric acid level among smokers with periodontitis as compared to nonsmokers with periodontitis.

Results obtained in our study is consistent with the previous work done by Bassam etal in 2006 K. leyan etal in 2005 et al it is attributed to the fact that chronic smoke is a constant source of oxidative stress and reduces endogenous production of antioxidants.

Table no. 4 displays the correlation between the salivary and serum uric acid levels in both the groups. The results obtained in our study depicts no statistically significant difference between saliva and serum uric acid levels positive correlation between serum and salivary uric acid levels as no significant change is observed in the serum and salivary uric acid levels in both the groups. Hence estimation salivary uric acid level can serve as a useful marker in assessment of the antioxidant activity. The results obtained in our study is in accordance with the study assessing the validity, stability, and utility of measuring uric acid in saliva conducted by Jena Riss et al in 2018 in which they have suggested that the serum uric levels have positive correlation with salivary uric acid levels.(17)

Conclusion

Smoking has a negative impact in the antioxidant capacity of saliva as we found significant reduction in the level of salivary uric acid level hence worsening the host defense and ultimately periodontitis. Also the results obtained in our study depict no statistically significant difference between saliva and serum uric acid levels and salivary uric acid levels nearly represent that of serum. Hence salivary sampling can be opted as noninvasive alternative to serum

uric acid levels estimation. For more conclusive and statistically significant result there is need to conduct these studies on larger sample size.

References

1. Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol. 1993;64(1):16–23.
2. Renvert S, Dahlén G, Wikström M. The clinical and microbiological effects of non-surgical periodontal therapy in smokers and non-smokers. J Clin Periodontol. 1998;25(2):153–7.
3. Buduneli N, Kardeşşler L, Isşık H, Willis III CS, Hawkins SI, Kinane DF, et al. Effects of smoking and gingival inflammation on salivary antioxidant capacity. J Clin Periodontol. 2006;33(3):159–64.
4. Abdolsamadi H, Goodarzi MT, Mortazavi H, Robati M, Ahmadi-Motemaye F. Comparison of salivary antioxidants in healthy smoking and non-smoking men. Chang Gung Med J. 2011;34(6):607–11.
5. Kondakova I, Lissi EA, Pizarro M. Total reactive antioxidant potential in human saliva of smokers and non-smokers. IUBMB Life. 1999;47(6):911–20.
6. Miricescu D, Totan A, Calenic B, Mocanu B, Didilescu A, Mohora M, et al. Salivary biomarkers: relationship between oxidative stress and alveolar bone loss in chronic periodontitis. Acta Odontol Scand. 2014;72(1):42–7.
7. Patil PB, Patil BR. Saliva: A diagnostic biomarker of periodontal diseases. J Indian Soc Periodontol. 2011;15(4):310.
8. Pullishery F, Panchmal GS, SiDDique S. Salivary thiocyanate, uric acid and pH as biomarkers of periodontal disease in tobacco users and non-users-an in-vitro study. J Clin Diagn Res JCDR. 2015;9(7):ZC47.
9. Fatima G, Uppin RB, Kasagani S, Tapshetty R, Rao A. Comparison of salivary uric acid level among healthy individuals without periodontitis with that of smokers and non-smokers with periodontitis. J Adv Oral Res. 2016;7(1):24–8.
10. Dahiya R, Blaggana A, Panwar V, Kumar S, Kathuria A, Malik S. Clinical and histological comparison of platelet-rich fibrin versus non-eugenol periodontal dressing in the treatment of gingival hyperpigmentation. J Indian Soc Periodontol. 2019;23(4):345.
11. Kaufman E, Lamster IB. The diagnostic applications of saliva—a review. Crit Rev Oral Biol Med. 2002;13(2):197–212.
12. Sculley DV, Langley-Evans SC. Salivary antioxidants and periodontal disease status. Proc Nutr Soc. 2002;61(1):137–43.
13. Moore S, Calder KA, Miller NJ, Rice-Evans CA. Antioxidant activity of saliva and periodontal disease. Free Radic Res. 1994; 21(6):417–25.
14. Greabu M, Battino M, Totan A, Mohora M, Mitrea N, Totan C, et al. Effect of gas phase and particulate phase of cigarette smoke on salivary antioxidants. What can be the role of vitamin C and pyridoxine? Pharmacol Rep. 2007;59(5):613.
15. Peluso I, Raguzzini A. Salivary and urinary total antioxidant capacity as biomarkers of oxidative stress in humans. Pathol Res Int. 2016;2016.
16. Zappacosta B, Persichilli S, De Sole P, Mordente A, Giardina B. Effect of smoking one cigarette on antioxidant metabolites in the saliva of healthy smokers. Arch Oral Biol. 1999;44(6):485–8.
17. Riis JL, Bryce CI, Matin MJ, Stebbins JL, Kornienko O, Huisstede L van, et al. The validity, stability, and utility of measuring uric acid in saliva. Biomark Med. 2018;12(6):583–96