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Assessment of Salivary Composition in Smokers and Non Smokers With Chronic Periodontitis.

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Abstract
Aim: *To assess and compare the salivary composition and periodontal status of smokers and non smokers with chronic periodontitis.*
Materials And Methods: *A total of 100 male subjects were selected from the out patient department of periodontology who fulfilled the criteria of the study and divided into 2 groups- group 1 were non smokers with chronic generalized periodontitis and group 2 consisted of smokers with chronic periodontits.2ml of saliva was collected from the subjects and sent for biochemical analysis(calcium, phosphorous and total proteins). Periodontal parameters, plaque index, gingival index and community periodontal index were recorded.*
Results: *Data was analyzed using student T test and Chi square test. There was high plaque index and CPI index score in smokers compared to non smokers and lower gingival index in smokers compared to non smokers. All these parameters were statistically significant. There was high calcium content and phosphate content in smokers compared to non smokers but was found to be statistically non significant. There was low protein content in smokers compared to non smokers which was statistically significant.*
Conclusion: *The present study exhibited reduced concentrations of total proteins, phosphorus and increased concentration of calcium in whole saliva in smokers with chronic periodontitis. It may thus be concluded that the analysis of salivary composition could be used as an auxiliary means of diagnosis.*

I. Introduction

Tobacco smoking is the practice of burning tobacco and inhaling the resulting smoke (consisting of particle and gaseous phases). The practice may have begun as early as 5000-3000 BC. German scientists identified a link between smoking and lung cancer in the later 1920s, leading to the first anti-smoking campaign in modern history, albeit one truncated by the collapse of the Third Reich at the end of the Second World War[1]. Smoking being one of the major risk factor for the development and progression of periodontitis; also has shown obvious adverse impact on respiratory system, cardiovascular system, bone matrix and hepatocytic function. The primary etiological factor responsible for periodontal disease is 'dental plaque'- a polymicrobial biofilm established on tooth surfaces, and retained if not removed by frequent plaque removal methods [2,3]. As dental plaque is pivotal to periodontal disease, differences with respect to quality of dental plaque are a factor for variability in an individual's risk for periodontal disease.

The noninvasive and simple nature of saliva collection allows for repetition and multiple collection of saliva that can potentially aid in early diagnosis, monitoring disease progression, or treatment responses with minimally trained personnel. This advantage of using saliva attracts investigators, who look for an alternative form of body fluids to simplify a diagnostic procedure.[4] Calcium,phosphate and total proteins are essential minerals within the human body. Positive correlations have been shown between salivary calcium, phosphate and total proteins content and periodontitis and chronic cigarette smoking[5]. Therefore, the purpose of this study was to analyze and compare the effects of smoking on the salivary minerals in chronic periodontitis subjects.

II. Materials and Methods

A total of 200 systemically healthy male patients, aged 25 to 50 years were included in the study. 100 male patients who were non smokers with chronic periodontitis were assigned to the control group and 100 male patients who were smokers with chronic periodontitis were assigned to the test group.

2.1. Inclusion criteria:

1. Subjects who are in age group 25 to 50 years.
2. Systemically healthy subjects.

3. Group I- non smokers with chronic periodontitis having probing depth of equal to or more than 4mm and presence of a minimum of 15 teeth.
4. Group II- smokers with chronic periodontitis having probing depth of equal to or more than 4mm and who smoke more than 5 cigarettes/beedis for a minimum of 3years and a minimum of 15 teeth present.

2.2 Exclusion criteria:

1. Other forms of tobacco usage.
2. Smokers who have quit the habit of cigarette smoking.
3. History of hospitalization or intake of medications in the last 6 months.
4. Previous history of periodontal therapy in last 6 months.

All patients were provided with verbal explanation of the nature of the study, and informed consent was obtained. A detailed systemic and family history were recorded. Gingival and periodontal findings were recorded for each patient following collection of 2ml whole unstimulated saliva.

2.3 Armamentarium:

- Mouth mirror
- Williams Periodontal probe
- No.5 explorer (Shepherd's hook)
- CPI probe
- Tweezer
- Cotton
- Kidney tray
- Gloves
- Mouth mask
- Ependorff tubes

Clinical parameters recorded were Plaque index (Silness and Loe 1964), Gingival index (Loe and Silness 1963) and Community Periodontal Index (CPI). All the saliva samples were collected prior to clinical measurements. Non-stimulated saliva was collected from the oral cavity where it was allowed to accumulate at the floor of the mouth and was transferred in Eppendorf tubes. Biochemical parameters assessed were total proteins, calcium and phosphorus. Analysis of the calcium, phosphate and total proteins were done by Vitros 5.1FS.The same operator recorded all the clinical data which was entered in Microsoft excel sheet. Student T test was applied to compare both the groups. Chi square test was applied to compare probing depth and loss of attachment in CPI index. Data was subjected to statistical analysis with the Statistical Package for Social Science Software (SPSS,Version 2.0,IBM,US). A value of $p<0.001$ was considered statistically significant.

III. Results

The mean total proteins in smokers is 0.768±0.17 and in non smokers it is 1.03±0.171. There was a statistically significant difference between smokers and non smokers. ($p<0.001$)(TABLE 1).The mean calcium levels in smokers were 2.32±1.16 and in non smokers it was 1.98±1.09.No statistically significant difference was noted. The mean phosphorous levels in smokers were 10.78±2.003 and in non smokers it was 10.13±1.77.No statistically significant difference was noted.(TABLE 1)

The mean plaque score in smokers was 1.1632±0.28 and in non smokers it was 1.42±0.47. There was a statistically significant difference between smokers and non smokers. ($p<0.001$)(TABLE 2)
The mean gingival index score in smokers was 1.16±0.12 and in non smokers it was 1.42±0.49 which was found to be statistically significant. ($p<0.001$).(TABLE 2)

When PD component of CPI was compared, among the nonsmokers about 41% had code1 ,43% had code2 ,16 % had code3 and 0% had code 4 .Among smokers 0% had code 1, 48% had code2, 50.4% had code 3 and 2% had code 4. Chi-square value for probing depth component between the two groups was 60.790 which was statistically significant ($p<0.001$)(TABLE 3). Comparison of LOA component of CPI showed, 52% non smokers had code 1, 48% had code 2. Among smokers 39% had code 0, 54.8% had code 1 and 48% had code 2, 6.5 % had code 3. Chi-square value for LOA component between the two groups was 14.857 which were not found to be statistically significant.(TABLE 4)

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Table 1- Comparison between Total proteins, calcium and phosphorus between smokers and non smokers

	GROUP	N	Mean	Std. Deviation	T	df	P VALUE
TOTAL PROTEINS	NON SMOKERS	100	1.03	0.1714	10.566	197.62	≤0.001
	SMOKERS	100	0.768	0.1792			
CALCIUM	NON SMOKERS	100	1.98	1.091566	-2.176	198	0.031
	SMOKERS	100	2.327	1.162708			
PHOSPHOROUS	NON SMOKERS	100	10.13	1.779	-2.426	195.28	0.016
	SMOKERS	100	10.78	2.003			

TABLE 2-Comparison of PI and GI between smokers and non smokers

	GROUP	N	Mean	Std. Deviation	T	df	P VALUE
PLAQUE INDEX	NON SMOKERS	100	1.34	0.476095	-5.285	160.32	≤0.001
	SMOKERS	100	1.632	0.280451			
GINGIVAL INDEX	NON SMOKERS	100	1.42	0.496045	4.895	112.33	≤0.001
	SMOKERS	100	1.1691	0.129022			

Table 3- Comparison of PD between smokers and non smokers

			GROUP		Total
			NON SMOKERS	SMOKERS	
PROBING DEPTH	1	Count	41	0	41
		% within GROUP	41.0%	0.0%	20.5%
	2	Count	43	48	91
		% within GROUP	43.0%	48.0%	45.5%
	3	Count	16	50	66
		% within GROUP	16.0%	50.0%	33.0%
	4	Count	0	2	2
		% within GROUP	0.0%	2.0%	1.0%
Total		Count	100	100	200
		% within GROUP	100.0%	100.0%	100.0%

Chi-Square Tests			
	Value	Df	P value
Pearson Chi-Square	60.790	3	≤0.001
N of Valid Cases	200		

Table 4- Comparison of LOA between smokers and non smokers

			GROUP		Total
			NON SMOKERS	SMOKERS	
LOSS OF ATTACHMENT	1	Count	52	39	91
		% within GROUP	52.0%	39.0%	45.5%
	2	Count	48	48	96
		% within GROUP	48.0%	48.0%	48.0%
	3	Count	0	13	13
		% within GROUP	0.0%	13.0%	6.5%
Total		Count	100	100	200
		% within GROUP	100.0%	100.0%	100.0%

Chi-Square Tests			
	Value	Df	P value
Pearson Chi-Square	14.857	2	.001
N of Valid Cases	200		

IV. Discussion

Smoking is considered a major risk factor for the development and progression of periodontal diseases. Grossi and coworkers studied the effect of cigarette smoking on the attachment apparatus and alveolar bone height; heavy smokers had greater odds ratio for both attachment loss and alveolar bone loss compared with non-smokers[6,7]. Bergstorm and Floderus Hyrhed in a co-twin control study have shown that smokers exhibited greater disease level than their non-smoker twins. Also, smoking has been shown to affect the response to surgical or nonsurgical periodontal treatment.

Smokers who exhibited greater plaque and calculus formation had also shown elevated calcium concentration and elevated calcium phosphate ratio in plaque. Zuabi *et al.* found that subjects with established periodontitis exhibited elevated concentrations of salivary electrolytes and proteins. In their study, smokers exhibited greater disease level but reduced sodium, calcium and magnesium concentrations.[8]In the present study, the assessment of saliva in smokers and non smokers revealed a higher salivary calcium level in smokers than non smokers but it was not statistically significant. This suggests an existence of an altered calcium metabolism and absorption among smoker subjects with periodontitis. This is in accordance with findings of

Sevon et al[9] and McGregor et al[5] in which they found that the calcium concentration of stimulated saliva of tobacco smokers is higher than non smokers. It maybe speculated that smoking causes a modification in saliva causing an increase in salivary calcium and possibly phosphorus. Sevon et al (2004) showed a decrease in skeletal bone density, a known side effect of smoking maybe reflected in increased levels of salivary calcium.[9]

The assessment of phosphate revealed a higher salivary phosphate level in non smokers compared to smokers but they were not statistically significant. This is in accordance with a study done by Kolte et al[10] and Zuabi et al.[11] In a study done by Erdermir et al[12] the levels of phosphate did not show any significant difference between smokers and nonsmokers.This could be explained by the different techniques used.The assessment of total proteins revealed a higher total protein content in non smokers compared to smokers and it was statistically significant. This is in accordance with Kolte at al[10] and Selvam et al[13]. This could be a result of parasympathetic stimulation of postganglionic neurons in response to nicotine, in the same manner as acetylcholine, because the membrane of these neurons contains the nicotinic type of acetylcholine receptors. Some toxic components of tobacco smoke, unsaturated and saturated aldehydes, couldninteract with thiol rich components, leading to structural and functional modification of these protein molecules.[13]

On comparing the plaque index, smokers showed a higher plaque index score compared to non smokers. This is in accordance with a study done by Kolte et al[14] and Macgregor et al[15] but some studies done by Feldman et al[16] and Machuca et al[17] showed low plaque index score in smokers.

On comparing the gingival index, there was a higher gingival index score for non smokers compared to smokers and was statistically significant. This was in accordance with a study done by Zuabi et al[8] and Preber et al[18]. During smoking it increases the heart rate, cardiac output, and blood pressure by autonomic stimulation, which alsoeffects peripheral vasoconstriction. There is also evidence that nicotine acts directly on blood vessels and capillaries to produce vasoconstriction which reduces bleeding.[19]

The probing depth component and loss of attachment component of community periodontal index score was statistically significant among smokers and non smokers. Haffajee AD et al[20] showed that at all levels of mean attachment loss, smokers exhibited more disease than non smokers.

V. Conclusion

Cigarette smoking is a major risk factor for periodontitis. It is often associated with high susceptibility, comparatively early onset disease, and more severe and wide spread periodontal destruction and treatment failure. Elevated salivary calcium in smokers, emphasizes the role of smoking in the progression of periodontitis. The findings of this study indicate that these that these minerals can be used as biomarkers in periodontal disease. It may further be hypothesized that changes in salivary composition might be useful to establish favorable response to periodontal therapy.

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