Comparison of Salivary Ca, Mg level and pH in patients with generalized chronic periodontitis and healthy individuals in Saudi population - A Clinico - Biochemical study

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Introduction

Human saliva is a fluid with many biological functions essential for the maintenance of oral health.[1] Salivary flow and composition influences calculus formation and periodontal disease. Salivary calcium, magnesium due to its affinity to be readily taken up by plaque, is an important factor not only with regard to the onset of periodontitis but also significantly with regard to dental health. They are one of the most intensely studied potential markers for periodontal disease in saliva.[2,3] Cross-sectional and longitudinal studies have provided strong evidence that smoking is a significant risk factor for periodontal disease.[4] All of the surveys have reported increased quantities of calculus in smokers. It has long been known that smoking causes a marked increase in salivary flow rate as a simple reflex effect and this could explain the tendency of smokers to accumulate increased amounts of calculus. There is some evidence that smoking also increases the mineralizing potential of saliva [4]. An elevated level of salivary calcium in smokers is related to a greater degree of bone loss and lower mineral density of bones than in non-smokers [5,6] Smokers have comparatively higher oral pH than non-smokers. Therefore, there is a great possibility for this pH to extract calcium from the scales deposited on the teeth (or even from their teeth) of these individuals which might result in the elevated levels of salivary calcium, magnesium level.[6]

Aims & Objectives: To estimate, analyze, compare and to correlate the variation in salivary calcium, magnesium levels and pH in periodontally healthy subjects and that of chronic periodontitis of smoker and non-smoker subjects.

Materials & Methods

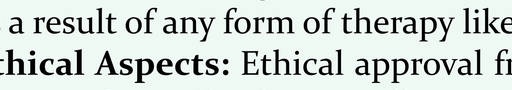
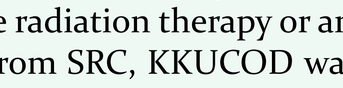
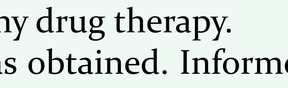
Study Design: The present research is an cross sectional study.
Study Population: Patients attending KKUCOD – OPD.
Total sample size: – 50.
Sampling Method: Judgemental sampling based on:
Inclusion criteria-

1. Patient's age between 25-55 years.
2. Clinical diagnosis of Chronic periodontitis with evident bone loss on radiograph and Probing Pocket Depth (PPD) of ≥ 4 mm and Clinical attachment loss (CAL) of ≥ 1 mm.
3. No history of periodontal therapy in last 6 months.
4. Patients who are current smokers in group III.
5. Subjects with at least 20 permanent teeth present.

Exclusion criteria-

1. Subjects who had undergone any periodontal treatment in the last 6 months.
2. Subjects who had antibiotic therapy in the last 3 months.
3. Subjects with history of smoking, tobacco chewing.
4. Patients suffering from xerostomia due to any systemic or local conditions or as a result of any form of therapy like radiation therapy or any drug therapy.

Ethical Aspects: Ethical approval from SRC, KKUCOD was obtained. Informed consent from all subjects willing to participate was obtained and confidentiality maintained.
Clinical Assessments: Loe & Silness - Gingival Index[GI], Bleeding on probing [BOP], Pus Exudate, Probing Pocket Depth [PPD] & Clinical Attachment Loss [CAL].
Collection of saliva sample and analysis : After periodontal recordings, prior to the collection procedure, the subjects began by rinsing their mouth, thoroughly several times with water and then resting quietly for 3 min. Unstimulated whole saliva (2 ml) was collected by means of the "spitting method" according to the directions given by Navazesh M. (1993).[14]
Samples was then assessed by AVL9180 electrolyte analyzer (Roche, Germany) for calcium ion, magnesium and pH by 'pH litmus test paper.
Statistical Analysis: The data collected was analyzed using SPSS, Version 20. To measure the differences between the means, Mann Whitney U-Test was used. The level of significance was set at 5%.

Results

- The mean PPD score is significantly higher among smokers than non-smokers ($p<0.05$).
- The mean salivary calcium level is significantly higher among smokers than non-smokers ($p<0.05$).
- There were no statistically significant differences in PI, GI, salivary magnesium and pH among smokers than non-smokers ($p>0.05$).

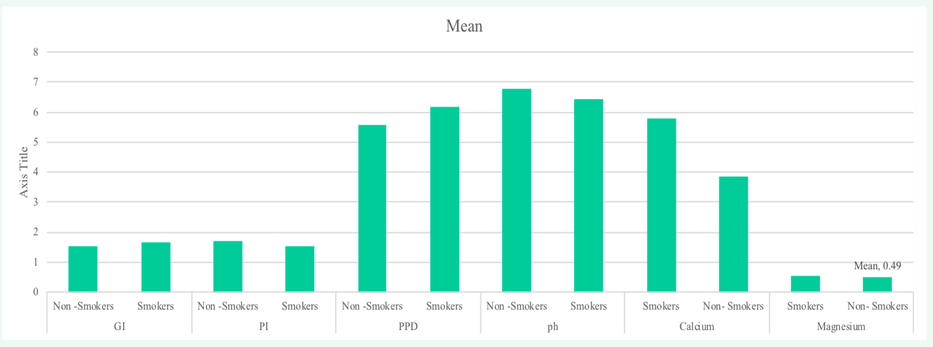


Table 1: Comparison of mean scores between smokers and non-smokers by Mann Whitney U-test

	Groups	N	Mean	SD	Mean Rank	Mann Whitney	Z	P-Value
GI	Non -Smokers	25	1.55	0.39	21.84			
	Smokers	25	1.67	0.29	29.16	221	-1.85	0.064
PI	Non -Smokers	25	1.71	0.49	28.32			
	Smokers	25	1.52	0.35	22.68	242	-1.395	0.163
PPD	Non -Smokers	25	5.57	1.02	20.92			
	Smokers	25	6.17	0.78	30.08	198	-2.432	0.015*
ph	Non -Smokers	25	6.80	0.91	27.64			
	Smokers	25	6.44	0.87	23.36	259	-1.215	0.224
Calcium	Smokers	25	5.79	1.76	33.8			
	Non- Smokers	25	3.87	1.03	17.2	105	-4.03	0.000*
Magnesium	Smokers	25	0.55	0.19	26.92			
	Non- Smokers	25	0.49	0.25	24.08	277	-0.69	0.490

Statistical Significant at 5% level

Discussion

Advances in diagnostic research are moving towards methods whereby the periodontal risk can be identified and quantified by objective measures using biomarkers. Patients with periodontitis may have elevated circulating levels of specific inflammatory markers that can be correlated to the severity of the disease.[4]
In the present study, there was an association with increase in PPD scores to that of increase in scores of calcium levels. These findings suggest that the increase in severity of different levels of periodontal disease which could increase the levels of calcium and pH level. These results were comparable with similar studies. The increase in calcium and PH level will precipitate and forms calculus which in turn causes more bone resorption [5,6]. There was no statistical difference between salivary magnesium levels.

Conclusions

1. Smokers have a higher plaque index and lower gingival index.
2. The level of calcium in saliva is higher in chronic generalized periodontitis individuals compared to those in periodontally healthy individuals.
3. This finding supports the view that higher salivary calcium could act as a risk factor for the development of periodontal diseases, possibly by raising the mineralization potential of dental plaque.

References

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