How to Cite:
Zagade, H. T., Varma, A. S., Suragimath, G. ., Zope, S., Kale, A., & Mashalkar, V. (2022). Effect of serum lead on salivary visfatin levels in periodontitis patients using smoke or smokeless form of tobacco. *International Journal of Health Sciences*, *6*(S3), 3551–3563. https://doi.org/10.53730/ijhs.v6nS3.6523

Effect of serum lead on salivary visfatin levels in periodontitis patients using smoke or smokeless form of tobacco

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***Abstract*---**To evaluate effect of serum lead on salivary visfatin levels in periodontitis patients using smoke and smokeless form of tobacco. A total of 60 male patients with generalized periodontitis stage II grade B and Grade C were selected for the study. Patients aged 30–65 years were assigned into two groups (30 subjects – Smoking tobacco group, 30 subjects – Smokeless tobacco group). Saliva samples were collected

Manuscript submitted: 27 Dec 2021, Manuscript revised: 09 March 2022, Accepted for publication: 18 April 2022

for analyses of visfatin and blood samples were collected for serum lead level analysis. Quantitative variables were compared using mean values and qualitative variables using proportions. There was statistically significant difference with (p value =0.025) mean increase in Probing pocket depth scores and (p value =0.017) in mean increase in Clinical attachment loss scores among Smokeless form of tobacco users. Mean serum Lead level was significantly high among smoke form (0.0107 + 0.007). Mean salivary visfatin level was significantly high among smoke form group (2.588±1.64). A significant correlation was found between serum Lead and Visfatin among smokeless form of tobacco users. Mean serum lead and salivary visfatin levels were statistically highly significant among the smoke form of tobacco group. A significant correlation was found between serum Lead and Visfatin among smokeless form of tobacco users.

***Keywords*---**Biomarkers, Lead (Pb), Oxidative stress, Periodontitis, Tobacco, Visfatin.

Introduction

Periodontitis is an inflammatory condition primarily considered as infection caused by host interactions/ host immune response towards oral microbiota, under the influence of environmental factors. The rate of disease progression depends on various factors and it varies from person to person. In periodontal disease progression although the microbes are implicated as etiological factor that brings about the inflammatory lesion, it is the chemical mediators of inflammation that play an important role in loss of supporting soft and hard tissue.[1] Chemical mediators are the endogenous molecules that mediate the inflammatory process and play a major role in its amplification, perpetuation and destruction of tissues. These mediators are produced from the various activated cells such as leukocytes, plasma cells, fibroblasts and other connective tissue cells from activated compliment system. Also, they show variation in their concentration with the severity of periodontitis.[2]

The Cigarette smoke contains more than 7000 chemicals. More than half of the periodontal disease cases are attributed to cigarette smoking.[3] Agency for Toxic Substances and Disease Registry (ATSDR) has stated that, the most extensively toxic heavy metals are particularly Lead (Pb) and cadmium (Cd). Lead in tobacco is a particular public health concern because it accumulates into the body.[4] Chronic Lead exposure has shown to affect the bone metabolism and possibly the immune system which suggest, Lead as a potential risk factor for the gingivitis and periodontitis.[5,6.] Recent evidence suggests, extensive elevation in blood Pb levels in mice was associated with increased food intake, body weight, total body fat, and the possibility of excessive production of visfatin.[7]

Visfatin is visceral fat adipokine identified as pre-B-cell colony-enhancing factor. It is mainly secreted by adipose tissues and macrophages. Visfatin is considered as one of the inflammatory adipokine which is available in inflammatory cells and

inflammatory conditions. Periodontal disease shows increase in various proinflammatory cytokine production, that has ability to release biomarker visfatin's expression in periodontal tissues.[8]. Exposure to smoke or smokeless form of tobacco leads to increase in nicotine that impairs the normal prosperity of endothelial cells. Epithelial cells, lining the periodontal pocket wall, are capable of producing visfatin. Therefore, visfatin may have a role in the etiopathogenesis of periodontitis. Thus, the elevation in blood Pb levels might affect the visfatin levels.[9] There is compound yet noteworthy collaboration between cigarette smoking exposure, adipose tissue and inflammatory relation in adipose tissue. With this background, the current study was aimed at evaluation of Effect of serum Pb on Salivary Visfatin levels in Periodontitis patients using smoke or smokeless form of tobacco.

Material and Methods

This following study was performed in the Department of Periodontology, at School of Dental Sciences, KIMSDU Karad in year 2020-2021. A total of 60 male patients, aged ranging from 30–65 years were assigned into two groups (30 subjects – Smoking tobacco group, 30 subjects – Smokeless tobacco group). Sample size was calculated based on formula $N=2*S^2 (Z1+Z2)^2/(M1-M2)^2$. After due approval from ethical committee and obtaining informed consent, patients were selected based on clustered sampling technique. They were then divided into two groups. Group A (n=30) Stage II Grade B and grade C periodontitis with smoke form of tobacco Habit, Group B (n=30) Stage II Grade B and grade C periodontitis with smokeless form of tobacco Habit. Saliva samples was collected for analyses of visfatin and blood sample were collected for serum lead level analysis. Their basic demographic information, socioeconomic status and lifestyle was documented in a pre-designed case sheet. All the registered participants underwent periodontal examination. Patients of both the groups received oral hygiene instructions and full-mouth ultrasonic scaling. Periodontal parameter measurements such as Gingival index (Loe & Silness 1963), Simplified Oral Hygiene Index (Green and Vermillon, 1964), Russel's Periodontal Index (Russel A. L 1956), Probing pocket depth of all teeth (UNC 15 probe HUFRIEDY) and Clinical attachment loss were recorded.

Results

The data so obtained was compiled systematically. A principal table was organized and the total data was subdivided and distributed meaningfully and presented as individual tables along with graphs. Statistical analysis was done after data compilation. Statistical analysis was done using Statistical Package of Social Science (SPSS Version 22.0; Chicago Inc., USA). Comparison of data was performed by applying specific statistical tests to find out the statistical significance of the comparisons. Quantitative variables comparison was performed using mean values and qualitative variables using proportions. Out of 60, all were male and 30 had habit of Smoke form of tobacco and 30 had habit of smokeless form (chewing) tobacco. Out of 60, 22(36.7%) were having professional degree, 15(25.0%) were graduated, most of the participants 32(53.3%) were skilled worker and 10(16.7%) were doing business or job. Mean age of smoke form group

was 35.87 year and of smokeless form group was 43.40 year. There was statistically higher significance with (p value <0.05) higher level of education and occupation among the smoke form of tobacco users. (Table. 1).

Table 2 reveals oral Hygiene practice & Oral hygiene status of the patients. Out of 60 patients, 35(58.3%) brush their teeth once daily and 25(41.7%) brush twice daily. There was statistically no significant difference found in oral Hygiene practice of the smokers and chewers. (p value =0.067) Out of 60 patient, 39(65.0%) had fair and 18(30.0%) had poor oral hygiene score. There was statistical significance with (p-value =0.027) showing higher oral Hygiene score among smokeless of tobacco. Table 3 reveals comparative evaluation of Mean PPD (Probing Pocket Depth) scores among smoke and Smokeless form of tobacco users. Mean PPD was significantly high among smokeless form group as compare to smoke form. Mean PPD was 4.97±0.718 among smokeless form group and 4.60±0.498 among smoke form respectively. There was statistically significant difference with (p value =0.025) mean increase in PPD scores among Smokeless form of tobacco users. Mean CAL (Clinical attachment loss) was 4.07±0.691 among Smokeless form of Tobacco and 3.63±0.669 among smoke form respectively. There was statistically significant difference with (p value =0.017) in mean increase in CAL scores among Smokeless form of tobacco users.

Mean serum Pb level was significantly high among smoke form (0.0107 + 0.007) as compare to smokeless form (0.0353 + 0.010) of Tobacco. There was statistically high significance among serum Pb level showing increased value in smoke form of tobacco group. (p value =0.001) (Table 4) Mean salivary visfatin level was significantly high among smoke form group (2.588±1.64) as compare to Smokeless form of tobacco group (0.9604±0.022). There was statistically high significance in mean salivary visfatin level scores among smoke form of tobacco group. (p value =0.001). (Table 5). Serum lead had Moderate Positive Significant Correlation i.e., r =0.365* (Pearson's rank correlation coefficient denoted by the Greek letter "r") with PPD and Weak Positive Not- significant Correlation i.e., r= 0.287 with CAL. Salivary Visfatin level had Weak Negative Not- significant Correlation i.e., r=-0.113 with PPD. There was no linear relationship found in between Salivary Visfatin level and CAL. (Table 6) Serum Pb had Moderate Positive Significant Correlation i.e., r=0.390* with CAL and Weak negative, Not-significant Correlation i.e., r= -0.241 with PPD. Salivary Visfatin level had Weak Negative, Not- significant Correlation i.e., r=0.240 with PPD and Moderate Positive Significant Correlation r= 0.389* with CAL. (Table 7). A significant correlation was found between serum Pb and Visfatin among smokeless form of tobacco users. While comparison among the smoke form of tobacco revealed no linear relationship. (Table 8).

Discussion

The toxicity of the compounds in tobacco smoke depends upon various factors such as dose, route of exposure, age, gender, genetic makeup and nutritional status of person. Arsenic, Cadmium, chromium, lead and mercury are the metals that ranks among one of the priority metals in the list due to their higher predisposition to the toxicity at public health significance.[8]. In our study all the

participants included were male patients with habit of smoke and smokeless form of tobacco use. Out of which 36.7% had professional degree while 53.3% skilled workers, 25.0% were graduates and 16.7% were doing job or business. Mean age of the smokers in smoke form group was 35.8 year while in smokeless form was 43.4 years. Study by Chhabra A. et al in 2021 stated that the use of tobacco is higher among the middle age adults, 24–44 years age group.[10] The prevalence of smokeless tobacco was higher in less educated people compared to smoke form use which was higher among the educated people living in urban areas. This difference among type of tobacco consumption could be due to socioeconomic status of the individual. These findings are consistent with the findings of our study.

In the present study, OHI-S scores were fair (65.0%) and poor (30.0%) among all the participants. The smokeless form of tobacco users showed poor OHI-S score compared to smoke form of tobacco users. In a study by Katuri KK, OHI-S score among smoke form and smokeless form users were fair to poor with mean score 3-4. But the study showed poor OHI-S score among the smokers compared to smokeless form which was contradictory to our study.[11] Calculus formation is increased among smokers due to increased salivary flow and concentration of calcium present in saliva of smokers, immediately after smoking.[12]. In our study, on comparing mean PPD and CAL scores among both the groups, PPD and CAL among the Smokeless form of tobacco group was higher compared to smoke form of tobacco. Which is contradictory to the study by Devi V et al, which showed higher probing pocket depth among smokers.[13] Studies stated that alteration in subgingival microflora [14] causes increase in periodontal disease severity. There is depletion of commensal bacteria's [15] and increase in periodontopathogens.[16] While, Singh GP in his study stated that the smokeless form of tobacco users shows higher impact on all the periodontal health parameters like PPD, CAL, GR, mobility, furcation, lesion. Also, the duration and frequency of use of tobacco has significant effect over the periodontal health.[17] The gingival recession occurs as result of long-term use of these products and causes injury to the adjacent mucosa and gingiva in the oral cavity leading to lesions of mucosa and gingival recession and clinical attachment loss.[18] Tobacco consists of various components which can lead to stimulation of the production of Proinflammatory cytokines, like interleukin (IL)-1, IL-6, IL-8, Tumor necrosis factor-a, transforming growth factor-B which causes the bone resorption in turn causing tissue destruction.[13]

In our study, the mean serum lead level was 4 ug/dl in smoke form of tobacco users and 1 ug/dl among smokeless form of tobacco users. The results of our study are contradictory to NHANES data (1999-2002), where in the mean lead concentration was high among the smokeless form of tobacco users. The geometric mean ratio of blood lead level (BLL) was higher among smoke and smokeless form of tobacco users compared to non-smokers.[19] The study suggests presence of various toxic metals in smokeless form of tobacco which includes Pb, Cd, As, Cu, Hg, and Se.[20] Also, according to WHO report the mean lead levels in smoke and smokeless tobacco form are given as 0.79–5.79 μg/g and 0.28–0.85 μg/g respectively. Lead is present in both forms of tobacco but the concentration is more in smoke form compared to smokeless form, these findings are similar to our study. [21]

In present study on correlation of Serum Lead with clinical parameters, moderate positive significant correlation was found with PPD among smoke form of tobacco group and CAL among smokeless form of tobacco group. Various studies have shown the relationship between prevalence of smoking and the severity of periodontitis. [22] Bone lead concentrations are associated with age and smoking history and that increases in bone lead directly correspond to increases in blood lead levels.[23] Chronic lead exposure may affect bone metabolism, this makes Pb a potential risk factor for periodontitis. Pb also affects bone by initiating an imbalance in the host defense system and the pathogenicity of microorganisms.[24] The periodontal pocket depth in periodontal disease is not only influenced by amount of bone destruction but it also affected by other factors such as inflammatory component of surrounding soft tissue.[25] For lead to cause periodontitis, it requires chronic long term lead exposure causing alteration in the bone metabolism[26], but Pb can lead to imbalance of host defense mechanism and thereby increasing pathogenicity of microorganisms.[26] Thus, this could be two-way mechanism of lead that can influence the periodontal pocket depth and clinical attachment loss in periodontal disease.

Mean Salivary Visfatin level was significantly high among smokeless form of tobacco users as compared to smoke form in the present study. There are very few studies that explored the effect of tobacco smoking on the visfatin levels. Pardo et al. Study in pregnant women stated that there were significant lower levels of adiponectin among smokers.[27] While López-Bermejo et al. found increased cord serum visfatin among the smokers in pregnancy. So, these studies state that, visfatin levels are inversely associated with smoking habit.[28] These lower levels of adiponectin and high levels of visfatin are seemed to be interrelated with the oxidative stress levels and number of free radicles released due to smoking.[29]

Nicotine from the tobacco products is converted to cotinine, cotinine is one of the biomarkers that suggest the changes in redox system in cells. Nicotine and nitrosamine in tobacco leads to increased production of ROS and decrease in uric acid defense system. Thus, there is increase in lipid peroxidation and protein oxidation. This increased level of nitric oxide causes endothelial dysfunction.[30] Visfatin is proinflammatory cytokine, which further leads to release of more proinflammatory cytokines in the endothelial cells such as activation of NF-κB, activation of matrix metalloproteinases and various cytokines and chemokines, IL-6 or monocyte chemotactic protein-1 etc.[31] that contribute to the periodontal inflammation. Studies also suggest *P. gingivalis* can induce visfatin secretion. Thus, visfatin is considered as a biomarker of periodontal disease. [32]

In our study Salivary Visfatin level had weak negative not- significant correlation among both the groups with PPD while there was no linear relationship with CAL among smoke form of tobacco and Moderate Positive Significant among smokeless form of tobacco. A study by Türer ÇC found a positive and significant correlation between visfatin levels in GCF with PPD ≤ 5 mm and PPD ≥ 6 mm.[9] There are not many studies with comparison of visfatin levels among smokers. In our study significant relation of serum lead on salivary visfatin was seen among smokeless form tobacco group. There are very less studies conducted in this relation and

this was the first study to evaluate the effect of serum Lead on salivary visfatin among smoke and smokeless form of tobacco. Numan AT. in his study among Obese and Osteoarthritis patients, stated a positive significant correlation between increased lead exposure, which can lead to increased visfatin.[5]

The current study shows Correlation between Serum Lead and Salivary visfatin level among Non-smokers/Chewers. The various studies state that rises in blood lead levels leads to increase in oxidative stress.[32] Oxidative stress is one of the reasons of cellular injury with production of free radicles.[34] Emission of reactive oxygen species or free radicles causes most of the damage to the periodontal tissues and supporting bone structure.[34] During the inflammation the Polymorphonuclear (PMN) leukocytes are the primary mediators of host immune response and these activated PMN's are responsible for production of ROS and further additional destruction of periodontium. High production of pro-oxidants in turn reduces the ability of the antioxidants to remove them from circulation. Also, the visceral fat deposition and higher amount of free fatty acid through portal and central adiposity both contributes to the oxidative stress. [35]. Neutrophils are believed to the primary source of ROS production in the periodontium. ROS causes direct tissue damage leading to metabolites of lipid peroxidation, DNA Damage and protein damage.[36]

Conclusion

Findings of the study shows increased PPD and CAL among smokeless form of tobacco group. Mean serum lead and salivary visfatin levels were statistically highly significant among the smoke form of tobacco group. On comparing the serum lead with clinical periodontal parameters and, Serum lead had moderate positive significant correlation with PPD in smoke group and with CAL in smokeless from of tobacco group. On comparing salivary Visfatin level with clinical periodontal parameters had Weak Negative, Not- significant correlation with PPD among both groups. While moderate positive significant correlation with CAL among smokeless form of tobacco group. A significant correlation was found between serum Lead and Visfatin among smokeless form of tobacco users.

Acknowledgement: NIL
Funding source: NIL
Conflict of interest: NIL

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Tables

Tables 1
Demographic Characteristic of patients

Parameter		Smoke form tobacco users N (%)	Smokeless form tobacco users N (%)	Total N (%)	Chi Square Value	Significance p value
Education	High School	0(0.0%)	7(23.3%)	7(11.7%)	14.612	0.002(HS)
	Intermediate	6(20.0%)	10(33.3%)	16(26.7%)		
	Graduation	7(23.3%)	8(26.7%)	15(25.0%)		
	Professional Degree	17(56.7%)	5(16.7%)	22(36.7%)		
Occupation	Unemployed (Student)	2(6.7%)	0(0.0%)	2(3.3%)	22.725	0.001(HS)
	Semi Professional (Business/ Job)	3(10.0%)	7(23.3%)	10(16.7%)		
	Professional	0(0.0%)	12(40.0%)	12(20.0%)		
	Skilled Worker (Teacher)	21(70.0%)	11(36.7%)	32(53.3%)		
	Farmer	4(13.3%)	0(0.0%)	4(6.7%)		

Mean Age (Year)		35.87 Year	43.40 Year			

HS=Highly Significant

Table 2
Comparison of OHI-S score of Smoke from and Smokeless form

Parameter		Smoke form tobacco users N (%)	Smokeless form tobacco users N (%)	Total N (%)	Chi Square Value	Significance 'P' Value
Oral Hygiene Practice	Once Daily	14(46.7%)	21(70.0%)	35(58.3%)	3.360	0.067(NS)
	Twice Daily	16(53.3%)	9(30.0%)	25(41.7%)		
Oral Hygiene Index Simplified	Good	3(10.0%)	0(0.0%)	3(5.0%)	7.197	0.027(S)
	Fair	22(73.3%)	17(56.7%)	39(65.0%)		
	Poor	5(16.7%)	13(43.3%)	18(30.0%)		

NS= Not Significant, S= Significant

Table 3
Comparative evaluation of Mean PPD and CAL scores among smoke and Smokeless form of Tobacco Users

Groups	Number	Mean PPD score		Mean CAL	
		Mean	SD	Mean	SD
Smoke form tobacco users	30	4.60	0.498	3.63	0.669
Smokeless form tobacco users	30	4.97	0.718	4.07	0.691
Unpaired student 't' test value	2.297			2.467	
Significance 'p' value	0.025(s)			0.017(S)	

PPD= probing pocket depth, CAL= Clinical attachment loss, SD= Standard Deviation, S significant

Table 4
Comparative evaluation of Mean Serum Lead Level among smoke and Smokeless form of tobacco users

Groups	Number	Mean Serum Lead Level	
		Mean	SD
Smoke form tobacco users	30	0.0353	0.015
Smokeless form tobacco users	30	0.0107	0.007
Unpaired Student 't' test Value	7.613		
Significance 'P' Value	0.001(HS)		

SD= Standard Deviation, HS= Highly Significant

Table 5
Comparative evaluation of Mean Salivary Visfatin level among smoke and Smokeless form of tobacco users

Groups	Number	Mean Salivary Visfatin level	
		Mean	SD
Smoke form tobacco users	30	2.588	1.64
Smokeless form tobacco users	30	0.9604	0.022
Unpaired Student 't' test Value	5.426		
Significance 'P' Value	0.001(HS)		

SD= standard Deviation, HS= Highly Significant

Table 6
Pearson's Correlation of Serum Lead and Salivary Visfatin level with Clinical periodontitis parameters among smoke form group

	Serum Lead	Salivary Visfatin level
PPD		
Pearson's Correlation Coefficient	0.365*	-0.113
Significance	0.047(S)	0.553(NS)
Inference	Moderate Positive Significant Correlation	Weak Negative Not- significant Correlation
CAL		
Pearson's Correlation Coefficient	0.287	-0.032
Significance	0.124(NS)	0.868(NS)
Inference	Weak Positive Not- significant Correlation	No Linear relationship

*. Correlation is significant at the 0.05 level (2-tailed). PPD= probing pocket depth, CAL= Clinical attachment loss, S=Significant, NS= Not significant

Table 7
Pearson's Correlation of Serum Lead and Salivary Visfatin level with Clinical periodontitis parameters among smokeless form of tobacco group

	Serum Lead	Salivary Visfatin level
PPD		
Pearson's Correlation Coefficient	-0.241	-0.240
Significance	0.200(NS)	0.210(NS)
Inference	Weak Negative Not- significant Correlation	Weak Negative Not- significant Correlation
CAL		
Pearson's Correlation Coefficient	0.390*	0.389*

Significance	0.033(S)	0.034(S)
Inference	Moderate Positive Signiifcant Correlation	Moderate Positive Signiifcant Correlation

*. Correlation is significant at the 0.05 level (2-tailed). PPD= probing pocket depth, CAL= Clinical attachment loss, S=Significant, NS= Not significant

Table 8
Pearson's Correlation between Serum Lead and Salivary Visfatin level among smoke and smokeless form of tobacco

Serum lead	Salivary visfatin	
	Smoke form	Smokeless form
Pearson's Correlation Coefficient	0.002	1.000**
Significance	0.992	0.001(HS)
Inference	No linear Relationship	Perfact Significant Correlation

**. Correlation is significant at the 0.01 level (2-tailed). HS= Highly Significantss