ISSN: 2581-5989
PubMed - National Library of Medicine - ID: 101738774

International Journal of Dental Science and Innovative Research (IJDSIR)

IJDSIR : Dental Publication Service

Available Online at: www.ijdsir.com

Volume – 7, Issue – 3, June – 2024, Page No. : 140 - 145

Volumetric analysis of gingival crevicular fluid in smokers versus nonsmokers: A cross sectional study

[1]Dr Virender S, [2]Dr T Prasanth, [3]Dr Ruchi Harish

[1-3]Department of Periodontology, Army Dental Centre(R&R), Dhaula Kuan, New Delhi.

Corresponding Author: Dr T Prasanth, Department of Periodontology, Army Dental Centre(R&R), Dhaula Kuan, New Delhi.

Citation of this Article: Dr Virender S, Dr T Prasanth, Dr Ruchi Harish, "Volumetric analysis of gingival crevicular fluid in smokers versus nonsmokers: A cross sectional study", IJDSIR- June – 2024, Volume –7, Issue - 3, P. No. 140 – 145.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Introduction: Smoking is highly prevalent and an accepted risk factor for increasing the prevalence and severity of periodontal destruction. At comparable plaque levels smokers do not express gingivitis symptoms as clearly as non-smokers due to peripheral vasoconstriction. The present study was undertaken to understand the cumulative effects of smoking in Gingival Crevicular Fluid(GCF) which have not been examined in the past.

Aim: The aim of this study is to evaluate the effects of smoking on GCF volume in clinically healthy individuals.

Materials and Methods: After obtaining the informed consent and approval from the Institutional ethical committee, 30 Periodontally healthy males were enrolled in the study from general OPD of a tertiary care dental centre located in Northern part of country. Subjects were matched for age and gender and were divided equally between smokers and non-smokers. Clinical attachment levels (CAL) and pocket depths (PD) were obtained at six points (mesial, distal and midpoints) of all teeth using UNC-15 periodontal probe. Following periodontal examinations, samples of GCF were obtained using Periopaper (Periostrips) from four of the individuals' teeth 16,21,36 and 41 for evaluating the volume(in µl). The statistical significance of differences between groups were tested according to paired 't' test.

Results: Smokers had significantly higher mean plaque index (PI) and gingival crevicular fluid (GCF) volumes (P =.019 and P =.027, respectively) as compared to non smokers.

Conclusion: The present study showed significantly increased GCF volume in smokers as compared to non-smokers population. The elevated volume of GCF indicates the presence of sub-clinical inflammatory component, despite that the Gingival Index is equivalent for both the groups.

Keywords: Tobacco smoking, Gingival crevicular fluid, Gingival index, Plaque index, Periodontal health.

Introduction

An accepted risk factor for periodontal disease is Smoking.[1]Studies examining the correlation between smoking and periodontal disease have repeatedly shown that smokers have worse periodontal effects and are more likely to have established periodontal disease than non-smokers.[2]Smokers exhibit a decreased propensity to produce inflammation in response to plaque biofilm buildup in human subjects when compared to nonsmokers.[3-4]Smokers tend to have greater numbers of deeper periodontal pockets and mean probing pocket depth.[5]According to longitudinal research, young people who smoke more than 15 cigarettes a day have the highest risk of losing their teeth.[6]Smoking also detrimentally affects the neutrophils and macrophages, which are important as gingival immunocompetent cells, especially, smoking impairs neutrophils chemotaxis and or phagocytosis.[7]

Smokers will not exhibit gingivitis symptoms as clearly as non-smokers at identical plaque levels. This was first observed by Bergstrom and Floderus-Myrhed[8] using a population of Swedish twins, and controlling for plaque index. The effect of smoking on the clinical gingivitis response is a function of the amount of challenge as well as smoking exposure. Under normal conditions of gingival health or with low levels of gingivitis there will be no detectable differences between smokers and nonsmokers. Smokers' gingivitis reactivity will be comparatively repressed at high or rising levels of plaque challenge; this suppression will increase with the challenge and smoking exposure. Smokers therefore, seldom will show highly increased gingivitis levels, not even in the presence of severe disease.[9]

There are a number of theories as to how smoking tobacco negatively impacts the periodontium, but the exact process is still unknown. There have been reports of both systemically and locally produced effects on the periodontium. Smoking tends to conceal gingival irritation by narrowing the coronary arteries and the gingiva's blood vessels.[10]

The possible roll of Gingival crevicular fluid (GCF) in oral defense was first elucidated by pioneering work of Waerhaug[11] and Brill and Krausse[12] during 1950s. Its presence in clinically normal sulci can be explained by the fact that gingiva that appears clinically normal invariably exhibits inflammation when examined microscopically.[13]When there is inflammation, there is more gingival fluid present, which can occasionally be correlated with the degree of inflammation. The mastication of coarse meals, brushing your teeth, massaging your gingiva, ovulation, hormonal contraceptives, and smoking all cause an increase in the generation of gingival fluid.[14] GCF volume is a well known marker of gingival health and have been used in many studies.[15]It has been shown that smoking causes a temporary, but noticeable, increase in gingival fluid flow; however, the long-term effects of smoking on GCF have not been studied. The current study's objective was to assess how smoking affects GCF volume in people who are clinically healthy.

Materials and Methods

30 periodontally healthy patients who reported to the general OPD of a tertiary care dental centre located in Northern part of country, were selected and divided equally between smoker and non smokers groups after obtaining the written informed consent. The study population included age matched 15 malesas the amount of GCF secreted can be influenced by ovulation during menstrual cycles or use of hormonal contraceptives in female. The patients did not have any teeth with probing depth (PD) >3mm. Subjects with potential confounding factors such as diseases of oral hard or soft tissues,

history of diabetes, hepatitis, or HIV infection, in need for premedication for dental treatment, chronic usage of anti-inflammatory drugs, immunosuppressive chemotherapy, history of any disease known to severely compromised immune function, current acute necrotizing ulcerative gingivitis, or use of orthodontic appliances were excluded during screening. The study included both smokers and nonsmokers where smoking status was classified based on cigarette consumption calculated in pack years.[16] Subjects were selected on the basis of reported smoking habits, namely, smokers who claimed to have smoked at least half a pack (10 cigarettes)/day for the past 5 years at the minimum. Non-smokers were defined as people who had not smoked > 100 cigarettes in their life till now and not currently smoking. The study was approved by Institutional Ethical Committee at the institute, and written informed consent was obtained from all participants before inclusion in the study in accordance with the Helsinki declaration.

Clinical recordings

Prior to gingival crevicular fluid collection, supra gingival plaque was scored using plaque index (PI).[17] Gingival inflammation was scored using gingival index (GI).[18] Full mouth PD were obtained at six points (mesial, distal and midpoints) of all teeth using a conventional periodontal probe. All clinical data were recorded by one examiner (KU).

Crevicular fluid sampling

Gingival Index, Plaque Index and probing pocket depths of the subjects were recorded for teeth no 16, 12, 24, 36, 32 & 44 using explorer & UNC – 15 probe, and GCF was collected from teeth no 16, 21, 36 & 41 using periostrips after the individual tooth site was air dried and isolated with cotton rolls. Readings were recorded using dry periostrips and Periotron 8000 for calibrating the instrument. The periostrips were placed inside the gingival sulci passively till resistance is felt and kept in situ for 30 seconds and immediately placed between the jaws of the periotron so that all but 1 mm of the white part of the strip ends up between the sensors when they are brought together. The strips contaminated with blood or saliva were discarded. If the residual crevicular fluid or debris is left on the sensors, the reading of the periotronmay not be zero for a fresh dry strip. Consequently, the sensors were gently wiped with a paper tissue wetted with a small amount of 70% ethyl alcohol. The sensors were dried with dry paper tissue before each recording. The readings were converted to actual volume (µl) using calibration curve for periotron.

Statistical analysis

Collected Data were expressed as means and standard deviations. The statistical significance of differences between groups were tested according to paired ‘t’ test. The null hypothesis was rejected at $P < .05$. For statistical analysis SPSS 13 program was used (SPSS Inc., Chicago, IL)

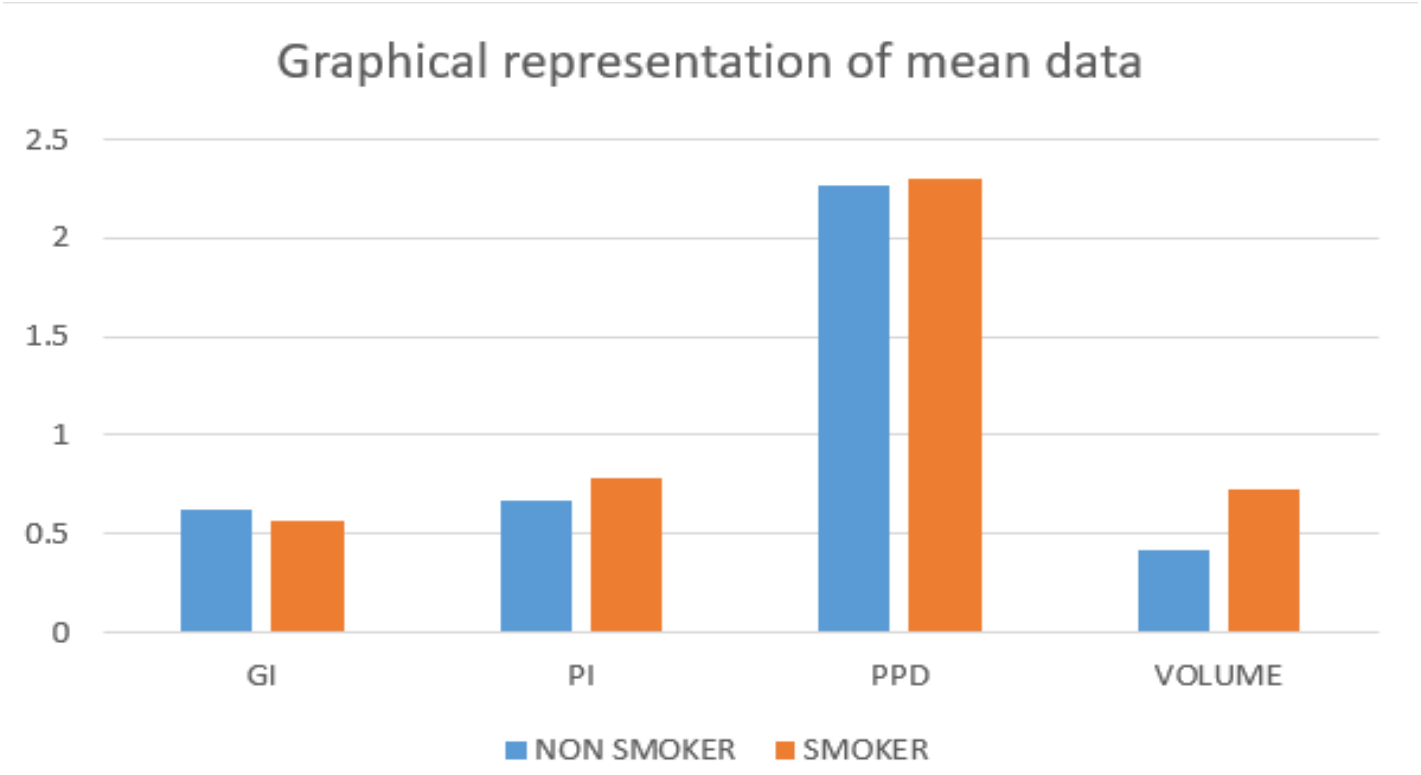
Results

Age and gender matched 15 smokers, and 15 non-smokers were enrolled for this study. All the participants were males. Table 1 shows the group statics of each group, including age, GI, PI,PPD,Periotron recordings and GCF volume (µl).

The graphical representation below shows that PI scores were significant different between smokers and non-smokers ($P = .019$). The PI scores were higher in smokers. The GI scores were not significantly different between groups ($P = .109$). PPD scores were not significantly different between groups ($P = .207$). The GCF volumes were significant different between smokers and non-smokers ($P = .027$). The GCF volumes were higher in smokers.

Table 1: Group Statistics

	1=NONSMOKER 2=SMOKERS	N	Mean	Std. Deviation	Std. Error Mean
Age	1	15	35.5333	5.18055	1.33761
	2	15	35.4667	4.42181	1.14171
GI	1	15	.6200	.21778	.05623
	2	15	.5700	.21044	.05434
PI	1	15	.6667	.14960	.03863
	2	15	.7800	.18593	.04801
PPD	1	15	2.2667	.45774	.11819
	2	15	2.6000	.50709	.13093
PERIOTRON	1	15	55.4000	3.21103	.82908
	2	15	80.6000	2.84228	.73387
VOLUME	1	15	.4200	.04140	.01069
	2	15	.7267	.05936	.01533



Discussion

In this study GCF volumes and PI scores were found higher in smokers. It is documented that GCF amountcan be affected by ovulation or hormonal contraceptives,therefore female subjects were excluded from this study.

Additionally, McLaughlin et al have shown that smoking cigarettes increases the GCF volume.[19] In a recent study blood flow of gingiva was measured with a laser Doppler and an increase in the gingival blood flow rate was observed. Authors mentioned that normally smoking causes a vasoconstriction, However, the degree of vasoconstriction was far less than in the thumb skin, and overcome by the evoked rise in arterial perfusion pressure.

Since smoking causes gingival blood flow to rise, it is hypothesized that smoking-related mild, recurrent vasoconstrictive episodes may eventually lead to gingival vascular dysfunction and periodontal disease.[20]Although the GI was similar to that of non-smokers, the increased volume of GCF shows the presence of masked inflammation in smokers.

The study by McLaughlin et al indicates that smoking produces a marked transient increase in GCF flow-rate, which might reflect changes in blood flow known to be produced by nicotine.[19]

This study with limited sample size showed that smoking group's elevated PI levelsare expected to effect GCF volume. Although at higher PI, the GI of the two groups were not statistically different. This result confirms that smoking decreases signs of GI and increases the GCF volume.

Conclusion

Smokers had significantly higher mean plaque index and GCF volume by 84% and 57% respectively compared to non smokers. GCF serves as an invaluable source of research material in microbial, host and wound healing longitudinal studies on human subjects group.

The study's findings demonstrated that smokers had a greater GCF volume than non-smokers. It is well known that the degree of inflammation increases with GCF volume. Smokers' elevated volume of GCF indicates the presence of a hidden inflammation, despite their GI being equivalent to that of non-smokers.

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