

The effect of smoking on Periodontal Health Status, Salivary Flow Rate and Salivary pH : A comparative study



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ABSTRACT

Background: Smoking is considered a major risk factor for development and progression of periodontal disease. Non smokers exposed to secondhand smoke are recognized to be at increased risk of periodontal disease.

Aims of the study: The purpose of this study was to evaluate and compare the effects of smoking and passive smoking on periodontal health status and on the salivary flow rate (SFR) and salivary pH.

Materials and methods: Seventy subjects were enrolled in the study, the subjects with an age range (30-45) year's old males and females without any history of systemic disease. The subjects were divided into three groups :G1(24 non smokers), G2 (21 passive smokers)and G3 (25 smokers). Unstimulated whole saliva was collected for measurement of salivary flow rate and pH. All periodontal parameters (plaque index, gingival index, bleeding on probing) were recorded for each subject.

Results: A significant difference was found in plaque index (PLI) between smokers and non smokers. No significant differences in gingival index (GI) was found for all groups, while there was highly significant difference in the number of bleeding sites. The salivary flow rate and pH were lower in smoker groups in compare to non smoker group.

Conclusion: Smoking was not only responsible for prevalence of periodontitis, but it also caused a decrease in saliva flow rate and pH.

KEYWORDS:

smokers, periodontal health status , saliva flow rate, salivary pH.

I. INTRODUCTION

Periodontal diseases are a group of inflammatory disorders that affect the gingiva, the periodontal ligament (PDL) (the suspensory ligament that anchors the teeth to the bone) and the alveolar bone (that part of the jaw bone that supports the teeth) (Darveau, 2010) [1]

Many potential risk factors of periodontitis have been identified, including smoking, socioeconomic status and stress (Albandar, 2002) [2]. Tobacco smoking is one of the most important risk factors that associated with the destruction of the alveolar bone and loss of attachment in patients with periodontitis (Palmer et al., 2005) [3]. Several studies on the relationship between periodontal diseases and tobacco use have consistently shown that smokers are two to six times more likely to develop periodontitis than non smokers (Razali et al., 2005; Anand et al., 2012) [4,5]. In addition, smoking was found to be a good predictor for further bone loss and attachment loss in smoker patients with periodontitis when compared to non-smoker counterparts (Lima et al., 2008) [6]

Smoking as an environmental factor has been found that it could interact with host cells and affect inflammatory responses to the microbial challenge (Palmer et al. 2005) [3]. The effects of smoking include change in vascular function, monocyte/neutrophil activities, expression of the adhesion molecule, downregulation of anti-inflammatory factors associated with an upregulation of proinflammatory cytokines is involved. (Al-Ghamdi and Anil, 2007; Stampfli and Anderson 2009; Anil et al., 2013) [7,8,9]

Nonsmokers exposed to secondhand smoke are recognized to be at increased risk of periodontitis. Persons exposed to passive smoking had 1.6 times the odds of having periodontal disease compared with those not exposed, after controlling for other covariates (Johnson 2007) [10].

Saliva is the first biological fluid that is exposed to cigarette smoke, which contains numerous toxic compositions responsible for structural and functional changes in saliva (Fox et al 2008) [11]

Saliva is a complex fluid that plays an important role in maintaining good oral conditions. People who have deficiency in saliva secretion, will trouble in nourishing, speaking and swallowing. They are also susceptible to catch oral infections and caries (Miletich 2010) [12]

Saliva can be used as a diagnostic fluid. It is inexpensive, non-invasive and easy to use as adiaagnostic methods (Erdemir et al 2006) [13]. As a clinical tool, saliva has many advantages over serum, including ease of collection, storing and shipping and it can be obtained at low cost in sufficient quantities for analysis. (Zuabi et al 1999) [14]. Saliva also is easier to handle for diagnostic procedures because it does not clot, thus lessening the manipulations required. (Griffiths et al 1992) [15].

Measurement of salivary secretion can be accomplished by different methods:

- a) unstimulated whole saliva secretion
- b) stimulated whole saliva secretion
- c) glandular saliva collection (mainly from parotid glands) with or without stimulation. Unstimulated whole saliva reflects basal salivary flow rate, is present for about 14 hours a day, and is the secretion that provides protection to oral tissues. Stimulated saliva represents physiologic stimulation, and is present for up to 2 hours (Sreebny 2000) [16].

The salivary flow rate is influenced by the circadian rhythm, it increases during the day and is decreases during sleep. Approximately two thirds of resting whole saliva volume is

produced by the submandibular glands. The parotid glands are responsible for secreting about one half of the stimulated saliva (Dawes, 2008) [17]

Parotid saliva contains a high bicarbonate concentration and thereby the salivary pH is correlated with the salivary flow rate. It has been noticed that patients with low flow rate, had lower bicarbonate concentration and salivary pH (Palomares et al, 2004) [18]

The purpose of this study was to evaluate and compare the effects of smoking and passive smoking on periodontal health status ; salivary flow rate and salivary pH.

II. MATERIAL AND METHOD:

Seventy participants apparently healthy , age 30-45 years were selected randomly regardless the periodontal health status and gender but adjusted according to smoking habit and weather they were exposed to indoor environment cigarette smoke (passive smoking status).

The subjects were divided into three groups:

Group I : (non-smokers group):-Twenty four subjects who had never smoked before and were not exposed to cigarette smoke regularly.

Group II : (passive smokers group):- Twenty one subjects who had been exposed to cigarette smoke at least 2 cigarette/day on ≥ 5 days/wk for at least 5 years (Arinola et al, 2011) [19]

Group III : (smokers group):- Twenty five subjects regularly smoked at least 15 cigarettes on average per day for at least 5 years; current smoker and had not quit smoking (Arinola et al, 2011) [19]

Exclusion criteria included Participant who diagnosed with Sjögren's syndrome, diabetes mellitus, rheumatoid arthritis or HIV. Participant who is on antihypertensive, antihistamines, antidepressants or antipsychotic medications. Participant who had head and neck radiation therapy.

Clinical examination

Clinical periodontal parameters included assessment of plaque index PLI (Silness and Loe in 1964) [20], gingival index GI (Loe 1967) [21], bleeding on probing BOP (Carrenza and Newman, 1996) [22]

Collection of salivary samples

All the individuals were instructed not to eat or drink (except water) at least 1 hour before collection of the samples, the patient is asked to sit in a relaxed position with elbows resting on knee and head hanging down between the arms. The lips are only slightly open and the patient lets the saliva drool passively over the lower lip into the graduated test tube (Tenovuo and Lagerlöf, 1994) [23]. Also the patient shouldn't swallow during the procedure and samples containing blood should be discarded. Saliva was collected between 9-12 am and the collection period was 5 minutes.

Each one of the subjects was asked to droll his saliva into the tube for 5 minutes to determine the salivary flow rate. The tube was labeled with the number of the subject that was previously written on the case sheet. The flow rate of saliva was expressed as milliliter per minute (ml/min).

Salivary pH was then measured using the pH indicating paper. The indicator strip was dipped in the saliva for 30 s and the color on the strip was compared with the standard color chart provided by the manufacturer. Based on the color change of the indicator paper strip, the pH was assessed in comparison with a color chart. Manufacturer's instructions were followed while measuring salivary pH.

Statistical analyses: The study variables were statistically analyzed by using mean, standard deviation, percentage, student t-test, chi-square test and the level of significant was accepted at $P \leq 0.05$ and highly significant when $P \leq 0.001$

III. RESULT

The descriptive statistics for plaque index was shown in Table (1). It was clearly shown that the means of plaque index were elevated in group 3 compared with group 1 and 2

Table (1): Descriptive statistics (mean±SD) of plaque and gingival index in each group.

Statistic	PL.I			G.I		
	G1	G2	G3	G1	G2	G3
Mean	1.052	1.027	1.447	1.029	1.009	1.082
SD±	0.212	0.120	0.391	0.282	0.174	0.085

Inter- group comparison of plaque index using student t-test revealed a non significant difference between group 1 and group 2 and there was significant difference between group 1 and group 3 , while there was highly significant difference between group 2 and group 3 as shown in Table(2)

The mean and SD of gingival index was described in Table (1), the mean of gingival index in group 3 were higher compared with group 1 and 2

Inter-group comparison of gingival index using student t-test revealed anon significant difference among all groups as shown in Table (2).

Table(2): Inter group comparison of mean of plaque and gingival index

Groups	PL.I			G.I		
	t-test	p-value	sig	t-test	p-value	sig
G1 & G2	0.309	0.872	NS	0.167	0.759	NS
G1&G3	-2.514	0.013	S	-0.898	0.461	NS
G2&G3	2.995	0.003	HS	1.482	0.150	NS

The number and percentage of bleeding on probing for all groups were shown in Table (3).The percentage of bleeding sites in group1 was(18.95%), while in group 2 was(11.89%) and in group 3 was(8.58%)

Table (3):The number and percentage of bleeding on probing for all groups.

Score	G1		G2		G3	
	No.	%	No.	%	No.	%
0	2250	81.05	1586	88.11	2322	91.42
1	326	18.95	214	11.89	218	8.58

Statistical analysis using Chi-square test revealed highly significant difference of B.O.P among all groups as shown in Table (4)

Table(4): The Chi-square test of bleeding on probing for all groups.

Groups	Chi-sequar	DF	P-value	Sig
G1 G2 G3	25.042	2	0,000	HS

The descriptive statistics for SFR and pH was shown in Table (5). It was clearly shown that the means were higher in group 1 compared with group 2and 3

Table(5): Descriptive statistics(mean±SD)of salivary flow rate and salivary pH in each group

Statistic	Salivary flow rate (ml/min)			pH		
	G1	G2	G3	G1	G2	G3
Mean	1.04	0.72	0.62	7.1	6.6	6.3
SD±	0.112	0.03	0.031	0.122	0.164	0.071

Inter- group comparison of SFR using student t-test revealed a significant difference among all groups as shown in Table(6)

Inter- group comparison of pH using student t-test revealed a significant difference between group 1 and group 2 and between group 1 and group 3 and there was non significant difference between group 2and group 3 as shown in Table (6)

Table(6):Inter group comparison of mean of salivary flow rate and salivary pH

Groups	SRF			pH		
	t-test	P-value	Sig	t-test	P-value	Sig
G1 & G2	4.809	0.022	S	2.147	0.029	S
G1&G3	3.914	0.013	S	3.198	0.031	S
G2&G3	3.965	0.043	S	3.132	0.75	NS

IV. DISCUSSION

Significant difference was found between smokers group and non-smokers group with more plaque accumulation in smokers than non-smokers, and this was in agreement with the result of the studies done by (Al-Tayeb, 2008; Sreedevi et al, 2012)[\[24,25\]](#) in which they compared plaque accumulation in smokers and non-smokers. On the other hand, the results disagreed with (Giannopoulou et al, 2001)[\[26\]](#) in which they found similar plaque formation rate in smokers and non-smokers. It seems likely that the major factor leading to greater plaque accumulation in smokers is inadequate oral hygiene. Tooth brushing behavior has a marked effect on oral cleanliness; people who brush their teeth frequently have less plaque than those who brush less frequently or only occasionally (Koivusilta et al,2003)[\[27\]](#).The tooth brushing efficiency of smokers was much less than nonsmokers(Bergstrom et al, 2000)[\[28\]](#). There is opinion that male smokers spent significantly less time brushing their teeth, and had significantly more plaque remaining on their teeth after tooth brushing than age-matched, male, non-smokers (Amarasena et al, 2002)[\[29\]](#). Besides, heat and accumulated product of combustion that result in tobacco stain as well as calculus are particular undesirable local irritants that increased with smoking (Al-Bandar et al,2000)[\[30\]](#). On the other hand no statistically significant difference existed in the mean plaque index between non-smokers and passive smokers and highly significant difference between passive smokers and smokers.

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The results showed that there was slight elevated gingival index in smokers group in comparison with non-smokers and the difference between them was non significant. This was in agreement with (Ustün and Alptekin, 2007; Rosa et al, 2008)[31,32] who showed that the gingival inflammation in smokers may be more than non-smokers. The results disagreed with (Al-Tayeb, 2008; Sreedevi et al, 2012) [24,25] who found that smokers had decreased expression of gingival inflammation. According to the results found in the this study ,it was found that smokers had slightly elevated gingival index than non-smokers, one explanation for the result that these alterations of gingival index follow physiologic changes related to the disease process (more plaque accumulation in smokers group lead to more gingival inflammation).

The difference in the number of bleeding on probing (BOP)sites among nonsmoker, passive smoker and smoker groups was highly significant difference. The result of this study revealed that the smokers have less number of sites with bleeding on probing than non smoker group and this was in agreement with (Nair et al, 2003; Thomas, 2004; Sreedevi et al, 2012)[33,34,25] while disagreement was recorded with Linden and Mullally (1994)[35] who found that young smokers had in fact more gingival bleeding than non-smokers, the explanation for this finding seemed to be related to the high levels of calculus and plaque reported in this group of young adult. It has been shown that smoking exerts a strong, chronic and dose-dependent suppressive effect on gingival bleeding on probing indicating its effect on gingival blood vessels (Dietrich et al, 2004)[36]. This may be explained by the fact that one of numerous tobacco smokes by-products, nicotine, exerts local vasoconstriction, reducing blood flow, edema and acts to inhibit what are normally early signs of periodontal problems by decreasing gingival inflammation, redness and bleeding (Chen et al, 2001)[37]. Studies have suggested that nicotine increases rate of proliferation of gingival epithelium, thus increasing epithelial thickness among smokers (Gultekin et al, 2008)[38].

Mavropoulos et al in 2003[39] shed some light on the understanding of the vasoactive mechanisms induced by cigarette smoking, and to support the hypothesis that cigarette smoking causes nervously mediated vasoconstriction in the healthy human gingiva. However, the degree of vasoconstriction was far less than in the thumb skin, and was overcome by the evoked rise in arterial perfusion pressure. As a consequence, gingival blood flow increased during smoking. It is speculated that small repeated vasoconstrictive attacks due to cigarette smoking may in the long run contribute to gingival vascular dysfunction and periodontal disease. So smoking has a long-term chronic effect impairing the vasculature of the periodontal tissue rather than a simple vasoconstrictive effect following a smoking episode. The suppressive effect on the vasculature can be observed through less gingival redness and lower bleeding on probing. According to the result of present study, the number of bleeding sites among passive smokers group was reduced when compared with non-smokers.

Secondhand tobacco smoke contains nicotine as well as carcinogens and toxins. Nicotine concentrations in the air in homes of smokers and in workplaces where smoking is permitted typically range on average from 2 to 10 micrograms/m³ (IARC, 2004) [40]. The result showed that passive smoking had an effect on the number and percentage of bleeding sites among passive smoker group which was reduced when compared with non-smokers, although this effect is less than the effect of active smokers. This could be result from the vasoconstrictive actions of nicotine constituent on periodontal tissue. Passive smoking had adverse affects on gums and dental health in a way very similar to direct smoking (Avşar et al 2008, Tomar et al 2008)[41,42].

It can be explained by considering the fact that cigarette smoke is composed of two main phases: a tar phase and a gas phase; both of which are rich in various free radicals and non-radical oxidants. It has been estimated that a single cigarette puff contains approximately, 1014 free radicals in the tar phase, and 1015 radicals in the gas phase (Pryor et al 1983) [43].

In this study there were reduced of salivary flow of smoker in comparison to non smoker and this agree with study done by Edgerton et al 2000 and Aguilar et al 2008 [44,45] who suggested tobacco leads to transient decline in the availability of saliva in the mouth . Also agree with Dyasanoor and Saddu,2014 [46] who found that smoking cause Significant reduction in salivary flow rate .Also agree with Kanwar et al,2013 [47] who found that smoking cause significant decrease in salivary flow rate and pH. but disagree with Palomare et al 2004 [18] who found that smoking do not imply alterations on salivary features (flow and buffer capacity)

Another important finding of this research was evidences of a decreased the salivary flow rate in passive smokers compared to the control group and this result is agree with Rezaei and Sariri 2011[48]

Also it was found that the pH in saliva of smokers and passive smoker was lower than non smokers and this result agree with Parvinen and colleagues 1984 [49] who reported in their study that the amount of pH in the saliva of smokers was lower than both sexes in non-smokers. Avsar and colleagues 2008 [41] also found that salivary pH in smoker were lower than children in the control group. Hosein 2016[50] also found that the pH of saliva in smokers was lower than nonsmokers.

The salivary flow rate and pH are usually correlated, as shown by Palomares et al,[18]. who found that patients with a low flow rate had lower bicarbonate concentration and therefore a lower salivary pH.

V. CONCLUSSION

Cigarette smoking is a complex stimulus which has deleterious effects on oral and periodontal health. It effects on the saliva which is a significant biological fluid by reducing the salivary fiow rate and salivary pH.

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