Waterpipe Smoking Effect on Clinical Periodontal Parameters, Salivary Flow Rate and Salivary pH in Chronic Periodontitis Patient

Saif Mohammed T. Al-Mufti B.D.S., H.D.D.⁽¹⁾ Saif S. Saliem B.D.S, M.Sc.⁽²⁾

ABSTRACT

Background: Waterpipe tobacco smoking has become common especially among young people, Waterpipe smoking misconcepted as a safer mean of smoking, so in this study we will highlight the effect of Waterpipe smoking on periodontal and oral health. **Materials and method.** The selected 100 male subjects of 30-40 years, categorized into 4 groups (each group 25 subject): Waterpipe smoker with healthy periodontium, Waterpipe smoker with chronic periodontitis, Non-smoker with healthy periodontius and Non-smoker with chronic periodontitis. Whole unstimulated saliva was collected. Clinical measurements: plaque index, gingival index, bleeding on probing, salivary flow rate and salivary pH were recorded.

Results. In the healthy groups: plaque index and salivary pH were higher in smokers than non-smokers but with no significant difference (P>0.05). While gingival index and salivary flow rate were higher in smoker than non-smokers and with significant difference (p<0.05). In the chronic periodontitis groups: plaque index, gingival index and salivary flow rate were higher in the non-smokers than smokers and with significant difference (p<0.05). While salivary pH was higher in the non-smokers than smokers than smokers and with significant difference (p<0.05). While salivary pH was higher in the non-smokers than smokers but with no significant difference (p<0.05). Correlation between weekly smoking hours with pH and salivary flow rate, in the smoker healthy groups, showed significant negative correlation, while plaque index showed significant positive correlation at (p<0.05). But in the smokers with chronic periodontitis, only gingival index significantly correlated with weekly smoking hours.

Conclusion. Waterpipe smoking has a detrimental effect on the periodontium and overall oral health. **Keywords:** Waterpipe, smoker, gingivitis, saliva (J Bagh Coll Dentistry 2018; 30(1): 63-68)

INTRODUCTION

Waterpipe tobacco smoking (WPS) is a common practice in Eastern Mediterranean countries. available literature showed that compared to a single cigarette, a 45-min Waterpipe smoking episode involves inhalation of about 100 times the smoking volume⁽¹⁾, Yielded 27–53 times the amount of tar, 3–245 times the amount of polyaromatic hydrocarbons, 6-14 the amount of aldehydes and 4-200 the amount of heavy metals^(2,3) and exposed the smoker to about 5 times more carbon monoxide^(4,5). In addition, blood nicotine levels in Waterpipe users are similar to those of heavy cigarette smokers $^{(6)}$. Periodontal disease is a complex inflammation of the peri-dental supporting structure including gingiva, periodontal ligament, alveolar bone and cementum⁽⁷⁾. Periodontal disease can be widely categorized into gingivitis and periodontitis⁽⁸⁾.

Saliva is a complex secretion, Saliva is a good indicator of the plasma levels of various substances such as hormones and drugs and can therefore be used as a non-invasive method for monitoring plasma concentrations of medicines or other substances⁽⁹⁾. Saliva has some advantages compared to blood and urine, two of the most used diagnostic fluids in laboratory setting. Saliva collection is easy and non-invasive requiring relatively simple instructions for collection and it possesses lower protein content, less complexity and varying composition than serum ^(10, 11), and that is one of the reason that encourage us to do this study.

MATERIALS AND METHODS: The

samples consisted of 100 male subjects of 30-40 years, each study group 25, from the department of Periodontology, at teaching hospital, College of Dentistry, University of Baghdad and from Al Sadir specialized dental center/ directorate of Rissafa /Ministry. All subject enrolled voluntarily and well explained about the aim and purposes of the study and gave informed consent to participate in it, subjects categorized into 4 groups: Waterpipe smoker with healthy periodontium (smoker H), Waterpipe smoker with chronic periodontitis (smoker CP), non-smoker with healthy periodontium (non-smoker H), and non-smoker with chronic periodontitis (non-smoker CP). They were subjected to a questionnaire, including

⁽¹⁾ Master student, Ministry of Health

⁽²⁾ Professor, Department of Periodontics, College of Dentistry, University of Baghdad.

question their name, age, full medical history, dental history, medications and if they smoked or drank alcohol, smoking history and smoking habit for session Followed by saliva collection for the assessment of salivary flow rate and pH followed by full examinations of clinical periodontal parameters of inflammation: Plaque index (PLI), Gingival index (GI) and bleeding on probing (BOP).

Exclusion criteria:

- 1. Patients who have undergone periodontal treatment in the 3 month period prior to the study, and those with orthodontic appliances also excluded from the study.
- 2. Female patients excluded from this study and that is to avoid potential hormone-induced microcirculatory changes ⁽¹²⁾.
- 3. Patient course of anti-inflammatory or antimicrobial therapy during the last 3 months.
- 4. Gross oral pathology such as oral cancer.
- 5. Cigarette smoker and chronic alcohol drinker.
- 6. Presence of systemic disease, e.g. Diabetes mellitus, cardiovascular disease, Rheumatoid arthritis.....etc.

Collection of Salivary Samples: After the subjects have been selected and before the clinical periodontal parameters examination, the whole unstimulated saliva samples were collected between the hours of 9 -11 a.m., saliva sampling and collection was according to Tenovuo .et al⁽¹³⁾ The volume of unstimulated saliva that collected firstly from each subject at 5 min was recorded, the saliva collected firstly was aspirated from the collection receptacle with a disposable 5 mL sterile syringe avoiding contact with the epithelium. The amount of saliva in ml, divided by the time of duration of the collection was recorded as the salivary flow rate. Only the liquid component of the saliva, not the foam, was measured⁽¹⁴⁾. Where estimation of flow rate (ml/min) was made according to this equation: Flow rate (FR)= Volume (ml) / Time (min) Samples containing blood were discarded. The samples were put in a small cooling box after collection to stop bacterial growth.

Clinical Periodontal Parameters Examination:

- <u>1. Assessment of (PLI)</u>: This was done by using PLI which was introduced by Silness and Löe, 1964⁽¹⁵⁾.
- Assessment of Gingival Inflammation by (GI): By using the criteria of Löe and Silness, 1963⁽¹⁶⁾, that modified by Löe in 1967⁽¹⁷⁾.

3. Assessment of Gingival Bleeding by (BOP): When it bleeds within 30 sec. after walking a periodontal probe inside gingival crevice or pocket of 4 sites (for each tooth), then positive score 1 is given to the site otherwise negative score 0 will be given ⁽¹⁸⁾.

Calibration: kappa test for absolute agreement was used to test measurements obtained by the inter- and intra-examiner calibration. All measurements were recorded by a well calibrated examiner (kappa value > 0.7)

Statistical Analysis:-Data were analyzed using SPSS (statistical package of social science) software version 19. Shapiro-Wilk test was used to check the normality of the data (results are not shown). Since abnormal distribution of the data was assumed in this study, the following statistics were used:

- 1. Descriptive statistics: Including means, medians, standard deviations, values and statistical tables.
- 2. Inferential statistics: Including:
 - a) Mann-Whitney U test.

b) Spearman's correlation coefficient test (r). In the statistical evaluation, the following levels of significance were used: Significant ($p \ge 0.05$)

RESULTS

In table 1 illustrate mean, SD, mean rank and median of PLI, GI, BOP of smokers and non-smokers.

In healthy group, the median of PLI in smokers H (0.750) was higher than of the non-smoker H (0.717), but both the results were found to be of no significant difference (P>0.05). When comparing the median of PLI in periodontitis group only, the findings obtained was that the median of nonsmoker CP(1.132) was more than that of smoker CP(0.982) with highly significant difference (P<0.01). The median of GI in the smokers H (0.866) was higher than those of non-smoker H (0.726), the results was of high significant difference (p<0.01).whereas in the CP group the median of smokers CP (1.019) was less than those of nonsmokers CP (1.148), and results was of significant difference (p<0.05). The median of BOP index in the smoker CP group (46.40) is less than those of non-smoker CP (56.00). The result showed no significant difference between the two groups (p>0.05).

Table 2. illustrates mean, SD, mean rank and median of SFR and salivary pH.

The median of SFR of smoker H group with nonsmoker H group, smoker H (0.800) appeared higher than non-smoker H (0.450) with significant difference (p<0.05). While in the non-smoker CP group (0.600) had higher SFR than the smoker CP (0.500) and no significant difference was found (p>0.05). The median of salivary pH was equal (7.5) in the non-smoker H and smoker H. But when comparing the mean rank of smoker H (24.10) was higher than non-smoker H (21.63). Also the nonsmokers CP (7.65) was higher than that of smoker CP (7.3), all the comparisons of salivary pH showed no significant difference (p>0.05).

Table 1. Illustrates the statistical difference of plaque index, gingival index and bleeding on probing among
study groups.

Comparison statistics		Healthy		Periodontitis	
		Non-Smokers	Smokers	Non-Smokers	Smokers
Diama	Mean	0.684	0.769	1.180	1.027
	±SD	0.104	0.252	0.252	0.441
	Median	0.717	0.750	1.132	0.982
Index	Mean Rank	21.10	24.52	29.05	18.16
Index	Z	0.868		2.765	
	P-value	0.385		0.006	
	Sig.	NS		HS	
	Mean	0.712	0.856	1.215	1.008
	±SD	0.103	0.202	0.273	0.128
Cincinal	Median	0.726	0.866	1.148	1.019
Gingival	Mean Rank	17.20	27.64	28.33	18.74
muex	Z	2.650		2.433	
	P-value	0.008		0.015	
	Sig.	HS		S.	
	Mean			49.119	44.620
	±SD			20.839	12.430
Bleeding On Probing	Median			56.000	46.400
	Mean Rank			25.40	21.08
	Z			1.100	
	P-value			0.271	
	Sig.			NS	

Table 2. Illustrates statistical differences of salivary flow rate and salivary pH among study groups

Comparison statistics		Healthy		Periodontitis	
		Non-Smokers	Smokers	Non-Smokers	Smokers
Salivary Flow Rate	Mean	0.530	0.720	0.540	0.432
	±SD	0.260	0.210	0.214	0.168
	Median	0.450	0.800	0.600	0.500
	Mean Rank	17.73	27.22	26.83	19.94
	Z	2.430		1.780	
	P-value	0.015		0.075	
	Sig.	S.		NS	
Salivary Ph	Mean	7.400	7.536	7.645	7.384
	±SD	0.581	0.563	0.487	0.426
	Median	7.500	7.500	7.650	7.300
	Mean Rank	21.63	24.10	26.68	20.06
	Z	0.683		1.691	
	P-value	0.523		0.091	
	Sig.	NS		NS	

Correlation statistics of weekly smoking hours (WSH) in table 3. Revealed that in the smoker H group, GI was not correlated (r = -0.170) with WSH and statistically not significant (p>0.05). While pH had a negative correlation (r = -0.453) with the WSH. With statistically significant correlation (p<0.05). SFR also had a negative correlation (r= -0.458) with WSH and with statistical significant (p<0.05). PLI had a positive correlation (r=0.565)with WSH with highly significant correlation (p<0.01). While in the smoker CP group, GI (r= 0.441) had positive correlation with WSH and with statistically significant correlation (p<0.05), while SFR (r=0.010), PLI (r=0.062), BOP (r=0.301) all are not correlated to WSH in smoker CP group with no statistical significance (p>0.05).

Table 3. Illustrates correlational statistics b	etween
variables in study groups	

Correlation statistics		Hours weekly		
		r	P-value	
Smokers Healthy	рН	-0.453	0.023	
	Sal. Flow rate	-0.458	0.021	
	PLI	0.565	0.003	
	GI	-0.170	0.415	
Smokers gingivitis	pН	-0.029	0.890	
	Sal. Flow rate	0.010	0.964	
	BOP	0.301	0.143	
	PLI	0.062	0.768	
	GI	0.441	0.027	

DISCUSSION

The results of this study showed that Waterpipe tobacco smoking, similar to cigarette tobacco smoking, is associated with periodontal disease. Whether Waterpipe smoking directly affects the periodontium by its local effect or indirectly cause periodontal disease through systemic route, so this study was done to investigate the effects of Waterpipe smoking on the periodontium by studying the clinical parameters and the physiology of saliva.

In this study, PLI and GI in the non-smoker CP was higher than smoker CP, these results were of statistical significant difference, and agree with a previous study done in Sweden, concluded that PLI and oral hygiene were similar in both smokers and non-smokers⁽¹⁹⁾. Also another study showed that the GI was higher in nonsmokers compared to smokers and have a statistical significance⁽²⁰⁾.

In this study although PLI in the smoker H was higher than that of non-smoker H, but no statistical significance found. This result is in agreement with Rad et al.⁽²¹⁾ who found no significant difference in PLI between smoker and non-smoker.

Also GI of smoker H was significantly higher than non-smoker H and this result is agreement with Sreedevi et al.⁽²²⁾ who implied that GI and calculus were significantly higher in smokers than nonsmokers.

In the current study there was no statistical difference in BOP between subgroups of CP (smokers and non-smokers), although non-smokers CP has higher scores than smokers CP, this can be explained by that chronic periodontitis is a multifactorial irreversible and cumulative condition, initiated and propagated by bacteria and host factors⁽²³⁾ and the fundamental mechanisms that lead to the development of chronic periodontitis are closely related to the dynamics of the host immune and inflammatory responses to periodontal pathogens present in the dental biofilm⁽²⁴⁾. Although the SFR in the non-smoker CP was higher than smoker CP, no significant difference found. This result agrees with previous study by Khemiss et al.⁽²⁵⁾ which supports the absence of effect of Waterpipe smoking on SFR while other studies showed similar SFR between smoker and nonsmoker⁽²⁶⁾.

Also SFR in smokers H was higher than nonsmokers H and the comparison was of statistical significant difference. This result agrees with Rad et al.⁽²¹⁾ which concluded that smoking increases the activity of salivary gland when smoking begins thus increases SFR, but long-term smoking significantly reduces SFR.

Salivary pH in smokers H and non-smokers H, also between smokers CP and non-smokers CP there was no significant difference among groups, these results agrees with Ahmadi-Motamayel et al.⁽²⁷⁾ which concluded that no significant differences in pH were noted among individuals with different stages of periodontal disease

When correlation statistics was done between WSH and other variables in the smoker H group revealed that salivary pH has a negative correlation with WSH with statistical significance. This result agrees with a study done on cigarrete smokers by Qamar et al.⁽²⁸⁾ which concluded that salivary pH is significantly reduced with increase in number of packs consumed/day and increased duration of exposure and showed that the pH turns acidic with long term use of tobacco. Also SFR had a negative correlation with WSH and this correlation was of statistical significance. This result is in agreement with previous studies demonstrated that long-term consumption of tobacco in any form, especially smokeless form, is one of the risk factors for reducing SFR^(21,29). Also PLI has a positive correlation with WSH with high statistical significance. This result agrees with Moga et al.⁽³⁰⁾ study that was conducted on healthy smoker and non-smoker participants and found a positive correlation between the PLI, and GI, as part of oral health evaluation, and smoking.

While in the smoker CP, the correlation of WSH with other variable revealed that only GI has a relatively strong positive correlation with WSH with statistical significance. This result disagrees with previous findings implying that the use of tobacco products leads to reduced gingival scores, greater calculus accumulation, and poor oral hygiene and the extent of these periodontal changes increases with the increase in the number of cigarettes smoked daily and the duration of the smoking habit⁽³¹⁾. This can be explained by that Waterpipe smoking can bring more harm to the gingival tissue due to the magnitude of smoke production and higher concentration of toxic substances inherited in the Waterpipe smoke.

CONCLUSION

Smoking Waterpipe have a debilitating effect on oral and periodontal health especially in smokers with chronic periodontitis.

REFERENCES

- 1. World Health Organization. Waterpipe tobacco smoking: health effects, research needs and recommended actions by regulators. Geneva: World Health Organization. 2005.
- Shihadeh A. Investigation of mainstream smoke aerosol of the argileh water pipe. Food Chemic Toxicol 2003; 41(1): 143-52.
- Shihadeh A, Saleh R. Polycyclic aromatic hydrocarbons, carbon monoxide, "tar", and nicotine in the mainstream smoke aerosol of the narghile water pipe. Food Chemic Toxicol 2005; 43(5):655-61.
- El-Nachef WN, Hammond SK. Exhaled carbon monoxide with waterpipe use in US students. J Am Med Assoc 2008; 299(1):36-8.
- Maziak W, Rastam S, Shihadeh AL, Bazzi A, Ibrahim I, Zaatari GS, Ward KD, Eissenberg T. Nicotine exposure in daily waterpipe smokers and its relation to puff topography. Addictive Behav 2011; 36(4): 397-9.
- 6. Neergaard J, Singh P, Job J, Montgomery S. Waterpipe smoking and nicotine exposure: a review of the current evidence. Nicotine Tobacco Res 2007; 9(10): 987-94.
- 7. Darveau RP. Periodontitis: a polymicrobial disruption of host homeostasis. Nat Rev Microbiol 2010; 8(7): 481-90.

- Lindhe J, Niklaus P Lang, Thorkild Karring. Clinical Periodontology and Implant Dentistry. 5th Ed, 2008. Wiley-Blackwell.
- 9. Hofman LF. Human saliva as a diagnostic specimen. J Nutrition 2001; 131(5):1621S-5S.
- 10. Nunes LA, Brenzikofer R, Macedo DV. Reference intervals for saliva analytes collected by a standardized method in a physically active population. Clinic Biochem 2011; 44(17): 1440-4.
- 11. Liu J, Duan Y. Saliva: A potential media for disease diagnostics and monitoring. Oral Oncol 2012; 48(7):569-77.
- 12. Lindhe J, Brånemark PI. Changes in vascular permeability after local application of sex hormones. J Periodont Res 1967; 2(4):259-65.
- Tenovuo J, Lagerlöf F. Saliva. Thylstrup A, Fejerskov O. Textbook of clinical cariology. 2nd ed. Copenhagen: Munksgaard. 1994: 713-17.
- 14. Alves C, Brandão M, Andion J, Menezes R. Use of graduated syringes for measuring salivary flow rate: a pilot study. Braz Dent J 2010; 21(5):401-4.
- 15. Silness J, Löe H. Periodontal disease in pregnancy II. Correlation between oral hygiene and periodontal condition. Acta odontologica scandinavica. 1964; 22(1):121-35.
- Löe H, Silness J. Periodontal disease in pregnancy I. Prevalence and severity. Acta Odontol Scand 1963; 21(6): 533-51.
- 17. Löe H. The gingival index, the plaque index and the retention index systems. J Periodontol 1967; 38(6 Part II):610-6.
- 18. Newman MG, Takei HH, Carranza FA. Clinical Periodontology, 2002.
- Axelsson P, Paulartder J, Lindhe J. Relationship between smoking and dental status in 35-, 50-, 65-, and 75-year-old individuals. J Clin Periodontol 1998; 25(4):297-305.
- Naderi NJ, Semyari H, Elahinia Z. The impact of smoking on gingiva: a histopathological study. Iran J Pathol 2015; 10(3):214.
- Rad M, Kakoie S, Brojeni FN, Pourdamghan N. Effect of long-term smoking on whole-mouth salivary flow rate and oral health. J Dent Res Dent Clin Dent Prospects 2010; 4(4):110.
- 22. Sreedevi M, Ramesh A, Dwarakanath C. Periodontal status in smokers and nonsmokers: a clinical, microbiological, and histopathological study. Int J Dentistry 2012; 14: 1-10.
- 23. Kinane DF. Causation and pathogenesis of periodontal disease. Periodontology 2000. 2001; 25(1):8-20.
- Gemmell E, Seymour GJ. Immunoregulatory control of Th1/Th2 cytokine profiles in periodontal disease. Periodontology 2000. 2004; 35(1):21-41.
- 25. Khemiss M, Ben Khelifa M, Ben Saad H. Preliminary findings on the correlation of saliva pH, buffering capacity, flow rate and consistency in relation to waterpipe tobacco smoking. Liby J Med 2017; 12(1):1-7.
- 26. Islas-Granillo H, Borges-Yañez SA, Medina-Solís CE, Galan-Vidal CA, Navarrete-Hernández JJ, Escoffié-Ramirez M, Maupomé G. Salivary parameters (salivary flow, pH and buffering capacity) in stimulated saliva of Mexican elders 60 years old and older. West Indian Med J 2014; 63(7):758.
- Ahmadi-Motamayel F, Falsafi P, Goodarzi MT, Poorolajal J. Comparison of Salivary pH, Buffering Capacity and Alkaline Phosphatase in Smokers and Healthy Non-Smokers: Retrospective cohort study. Sultan Qaboos Univ Med J 2016;16(3):e317.

Oral and maxillofacial Surgery and Periodontics 67

- 28. Qamar A, Baig S, Ali A, Zehra N, Memon MA. Resting salivary flow rate and pH decreases in chewable tobacco users. ; Brit J Med Medic Res. 2016; 11(3):1-9
- 29. Kanwar A, Sah K, Grover N, Chandra S, Singh RR. Longterm effect of tobacco on resting whole mouth salivary flow rate and pH: An institutional based comparative study. Eur J Gener Dentistry 2013; 2(3):296
- 30. Moga M, Bosca AB, Bondor CI, Ilea A, Lucaciu OP, Ionel A, Man MA, Rajnoveanu RM, Câmpian RS. Assessment of the correlations between nicotine dependence, exhaled carbon monoxide levels and oral hygiene status: an observational study. Clujul Med 2017; 90(1): 99.
- Malhotra R, Kapoor A, Grover V, Kaushal S. Nicotine and periodontal tissues. J Indian Soc Periodontol 2010; 14(1):72.

الخلاصة

نبذة عن البحث: تدخين الاركيلة امست ظاهرة منتشرة وخصوصا بين الشباب بسبب الاعتقاد الخاطئ حول كونها وسيلة امينة لتدخين التبغ. في هذه الدر اسة سنسلط الضوء على تاثير تدخين الاركيلة على صحة اللثة والفم.

المواد والطرائق المستعملة في البحث: تتالف العينة من ١٠٠ متطوع في الفئة العمرية بين ٣٠ ال ٤٠ سنة. تم تقسيمهم الى ٤ مجاميع (كل مجموعة ٢٥ متطوع) كالتالي : مدخن اركيلة ذو لثه سليمة, مدخن اركيلة ولديه التهاب نسيج ماحول الاسنان المزمن , غير مدخن ذو لثة سليمة , غير مدخن ولديه التهاب نسيج ماحول الاسنان المزمن. تم جمع اللعاب غير محفز التكوين من كل متطوع واخذ القياسات السريرية فيما يخص اللثة وهي مؤشر الصفيحة الجرثومية و موشر التهاب اللثة والنزف. هذا اضافة الى تسجيل معدل تدفق اللعاب والمؤشر الهيدروجيني.

النتائج: المجموعتين ذوي اللثة السليمة ,وجد ان مؤشر الطبقة الجرثومية والموشر الهيدروجيني للعاب كان اكثر عند المدخنين منهم لكن مع عدم وجود فرق احصائي معنوي ,لكن مؤشر التهاب الثة ومعدل تدفق اللعاب كان اكثر عند المدخنين ايضا مع وجود فرق احصائي معنوي. المجموعتين الذين لديهم التهاب نسيج ماحول الاسنان المزمن , وجد ان مؤشر الطبقة لاجرثومية ومؤشر التهاب اللثة ومعدل تدفق اللعاب كان اكثر عند وجود فرق احصائي معنوي . التحليل الاحصائي للدراسة اوجد علاقة سابية بين عدد ساعات التدخين العاب كان اكثر عند غير المدخنين منهم مع معدل تدفق اللعاب من جهة أخرى عند المدخنين الذين لديهم وجود فرق العاب كان اكثر عند عبر المدخنين منهم مع الاسبو عية اللعاب من جهة أخرى عند المدخنين الذين لديهم لثة سليمة , بينما وجد ان مؤشر الطبقة الجرثومية له علاقة العاب كان اكثر عند المدخنين منهم مع الاسبو عية للعاب من جهة الحرى عند المدخنين الذين لديهم لثة سليمة , بينما وجد ان مؤشر الطبقة الجرثومية له علاقة التخين منهم مع

الاستنتاج: تدخين الاركيلة له تاثير ضار على انسجة ماحول الاسنان و صحة الفم .