A Comparative Investigation of Salivary Alkaline Phosphatase Enzyme Levels and Salivary IgA in Correlation with Periodontal Health: An Analysis of Narghile-Only, Cigarette-Only male smokers: A REVIEW

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Abstract

Smoking cigarettes and narghile is well recognized as a significant risk factor for periodontal disease. Salivary diagnostics such as s-IgA and alkaline phosphatase (ALP) are often used for this purpose. The research sought to assess the concentrations of salivary ALP and s-IgA, as well as the state of the periodontium, in individuals who smoke cigarettes exclusively (SC) and those who smoke narghile exclusively (SN). The research comprises 125 males (SC) and 125 males (SN). To test the levels of ALP and s-IgA, as well as the periodontal parameters simultaneously, saliva samples collected from each participant, would be transferred to the software SPSS V.26. descriptive statistics and an independent t-test to test the difference between the two groups. The findings demonstrated substantial differences in the levels of ALP and s-IgA between the two groups, as well as significant variations in the periodontal parameters. It is concluded that the smoking of cigarettes and narghile has harmful effects on the health of periodontium, and the levels of salivary ALP and IgA have an impact as biological markers for forecasting the incipience of periodontal disease.

Keywords: periodontal disease, smoking, ALP, IgA, saliva.

Introduction:

Periodontal disease encompasses a diverse array of inflammatory disorders that impact the structure responsible for supporting teeth, known as the periodontium. This includes the gingivae, alveolar bone, cementum, and periodontal ligament. These illnesses have the potential to result in tooth loss and contribute to inflammation throughout the body. Periodontal disorders have a high prevalence and have the potential to impact a significant proportion, up to 90%, of the global population. Gingivitis, which is considered the least severe manifestation of periodontal disease, arises from the accumulation of bacterial biofilm, also referred to as dental plaque, on the teeth in close proximity to the gingiva (1). Implementation of enhanced oral hygiene procedures may lead to the reversal of the condition, while chronic periodontitis occurs when the host's immune system reacts to the presence of bacterial clusters on the surfaces of the teeth. The consequence of this is an irreversible degradation of the connective tissue connection, resulting in the creation of periodontal pockets and ultimately alveolar bone resorption. Risk factors significantly influence an individual's reaction to periodontal infection (2). Identifying these risk variables is crucial for identifying people who need prevention and therapy. Modifying these risk factors is essential for effectively controlling periodontal disease. There are many systemic and local risk factors that majorly effect on the health of the periodontium. Multiple risk factors contribute to the heightened prevalence of periodontitis, such as smoking, educational and socioeconomic position, diabetes mellitus, access to healthcare, and oral hygiene practices (3).

Tobacco smoking continues to be a widespread addiction in many groups globally (8), despite the growing recognition of its detrimental impact on overall health. Smoking is now acknowledged as the primary factor contributing to avoidable mortality and illness. Nevertheless, several risk factors that exist and are recognized to heighten the vulnerability to periodontal disease, such as smoking, stress, and obesity, are believed to be variables that may be changed (4,5). However, cigarette smoking is a significant cause of chronic periodontitis. Approximately 5,000 distinct molecules are breathed via the mouth and nasal cavities prior to the absorption of vaporized gases in the lungs.

Smoking contributes to chronic periodontitis in several ways, including higher rates of tooth loss, worsened bone and attachment loss, and wider periodontal pockets compared to those who do not smoke (6,8).

Cigarettes are the predominant method of tobacco use, particularly among the youth demographic. An adolescent starts smoking by experimenting with a cigarette and then becomes dependent on nicotine, making it hard to quit. Various cigarette varieties have distinct shapes, although they always inflict damage in a same manner. Filtered cigarettes, low-nicotine cigarettes, and rolled cigarettes on either white or brown tobacco paper are available. Some of them are flavored with various sweeteners, while others have a more traditional taste, but the harmful effects remain same (9). Cigarettes have detrimental effects on several body organs and systems, including the skin, oral cavity, throat, esophagus, stomach, pancreas, lungs, heart, arteries, bladder, breasts, and cervix (in women), as well as the brain and nervous system. Everyone is susceptible to illnesses and conditions resulting from smoking, with the most prevalent being cancer, cardiovascular disease, and respiratory disease. Smoking cigars or pipes is as detrimental to health as smoking cigarettes (10).

There is a widespread belief that smoking narghile (also known as hookah or shisha) is less detrimental than smoking cigarettes. However, the reality is that a single shisha session is comparable to consuming 50 to 60 cigarettes, and a two-to-three-hour shisha session is similar to smoking 25 cigarettes. Various varieties of shisha exist, varying in form and composition, although they all have the same detrimental consequences. There are three types of shishas: "mouassal," which is tobacco mixed with molasses; "jrak," which is tobacco combined with spoiled fruits; and sweetened shisha, which comprises tobacco and certain fruits such as apricot. All of these substances consist of fermented material. Shisha smoking is a primary factor in the development of lip, mouth, and throat cancer. Additionally, it is associated with the development of cancer in the lungs, esophagus, stomach, and bladder. Smoking shisha may facilitate the transmission of TB-causing microorganisms, leading to the spread of tuberculosis among individuals who use the same shisha pipe. This may also be transmitted to those who do not smoke but have contact with smokers. Shisha emits smoke and hazardous chemicals, such as carbon monoxide. It is also tainted with pesticides, heavy metals, and mycotoxins. Consequently, shisha contributes little to air pollution (11).

Saliva is a readily available bodily fluid that comprises substances originating from the mucosal linings, gingival sulcus, inside the mouth. Saliva includes bacteria that inhabit the mouth and other external substances, which might possibly provide information about the interaction between the host and the environment (12). Saliva is an intricate system that consists of its own components, sulcular fluid components, bacteria, results of inflammation in the periodontium, as well as metabolites and signal molecules associated with distant activities. Saliva may include proteolytic enzymes that originate from several sources. These may originate not just from polymorphonuclear leukocytes and periodontal bacteria, but also from the circulation (13). It bears resemblance to the products of inflammation, which may likewise originate from both local and systemic sources. Research on the use of saliva as a diagnostic fluid has a lengthy historical background. Salivary diagnostics is not restricted to the identification of oral disorders. It may also be used to investigate many systemic diseases, including different forms of cancer, cardiovascular ailments, immunologic syndromes, and genetic deficits (14,15).

Alkaline phosphatase (ALP) is a vital enzyme found in osteoblasts, fibroblasts, and neutrophils, among other periodontal cells., that is closely linked to bone production. ALP Polymorphonuclear leukocytes secrete it during the inflammatory response, osteoblasts secrete it during bone formation, and periodontal ligament fibroblasts secrete it during the regeneration of the periodontal ligament. Hence, it has a role in both the development of periodontal inflammation and the restoration of periodontal tissues (16,17). ALP facilitates bone mineralization by producing organic phosphate and breaking down inorganic pyrophosphate by hydrolysis. The ALP biomarker is linked to the processes of bone production, resorption, and turnover. These mediators are often linked to overall bone metabolism and may possibly be connected to local bone metabolism in periodontitis (18,19).

Abdulkareem S et al, 2023 found that the levels of ALP and ACP in the saliva and serum of patients with gingivitis and periodontitis were different before and after undergoing scaling and root planning, and that the level of enzymes were elevated during the inflammation and subsequent degradation following treatment (20)

Immunoglobulins (Igs) are protein molecules that are made by specific immune systems in reaction to the entry of outside agents, such as viruses, bacteria, protozoans, fungi, tumor cells, or foreign tissues. These agents can be identified by the antigens that are on their cell surfaces. (21). Igs have the role of binding to certain antigen molecules and then targeting the bound molecules for inactivation and/or removal of poisons, microorganisms, and parasites from the body. The humoral host immune responses are crucial for protecting the oral environment. This is because antibodies have the potential to prevent germs from attaching to cell surfaces and may also promote the aggregation/opsonization of these bacteria(22). Furthermore, antibodies are linked to alternative pathways that play a significant role in preventing colonization and promoting the destruction of germs. They also have the ability to neutralize hazardous substances. Salivary IgA is the primary defensive mechanism in the oral cavity against microbial invasions and plays a crucial role in the interactions between bacteria and the host (23). A direct and positive link exists between the concentration of salivary IgA and the degree of inflammation. The level of IgA concentration is contingent upon the existence of plaque, where The agglutinating process occurs when antibodies present in saliva interact with bacteria that are multiplying on a surface, hindering their ability to adhere. Research conducted by Doshi M. et al, (2010) revealed that individuals with IgA deficiency have a higher prevalence of mucosal infections (24). The objective of this research is to evaluate the periodontal health of male individuals who only smoke cigarettes with those who exclusively smoke narghile. We will accomplish this by analyzing biological markers like salivary ALP and s-IgA. This analysis is significant as it elucidates the comparative influence of these two types of smoking on oral health and may provide guidance for public health initiatives focused on preventing and treating periodontal disease. The results of this research will be useful to dentists and public health experts in developing specific strategies to improve oral health outcomes among smokers.

Review of literature

Materials and Methods

The current investigation is a cross-sectional analysis comprising 250 individuals, aged between 18 and 30 years. The participants were recruited from several departments at the College of Dentistry Teaching Hospital of Tikrit University, as well as from numerous specialized clinics, during a period of 7 months. Information was gathered about oral hygiene habits, including the number of dental check-ups per year and the frequency of daily tooth-brushing, as well as tobacco use. Comprehensive dental evaluations were conducted on all teeth, except the wisdom molars. The count of surviving teeth, including those that are decaying, missing, or filled, is documented. The research participants were categorized into two groups: male cigarette-only smokers (SC) (n=125 males), and male narghile-only smokers (SN) (n=125 males), according to the 2017 classification of periodontal diseases (25)

The clinical indicators, namely plaque index (PLI) (26), gingival index (GI) (27), bleeding on probing (BOP), pocket depth (PPD) (28), and clinical attachment level (CAL) (29), have been detected. Subsequently, unstimulated saliva (7 ml) is collected between 9-12 a.m. The collected samples were centrifuged for 10 minutes at 3000 rounds per minute, and then they were frozen at -20º C. The levels of salivary IgA were measured using the enzyme-linked immunosorbent assay (ELISA) method after all the samples were collected. The commercial kit provided by Demeditec Diagnostic GmbH D-24145 Kiel (Germany) provided instructions for this method. Human Gesellschaft f\r Biochemica und Diagnostica mbH Max-plank-Ring 21. (Germany) was used for the biochemical determination of the ALP enzyme. The study included only male participants who

exclusively smoke cigarettes; on average, a minimum of 10 cigarettes are consumed daily (30) or narghiles smoking. while female participants, individuals with a history of alcoholism, individuals with systemic diseases, and those who have received periodontal treatment or any type of anti-inflammatory, antimicrobial, or other medication within the past 2 months, or patients in the period of receiving chemotherapy , radiotherapy or any drug that causes dry mouth (xerostomia) were excluded from the study.

Participants were required to provide informed permission using a specifically tailored consent form. A questionnaire and case sheet were used to gather detailed information, including both dental and medical histories. This research involving human participants adheres to the Helsinki Declaration of 1975, as updated in 2013, and has received approval from the appropriate institutional Ethical Committee (31)

The SPSS version 26 program was used to do the statistical study. Several summary figures were found in the study. These included percentage, mean, mean percentage, standard deviation (SD), independent t-test, and Pearson's coefficient of association. There were three levels of significance: significant (S), non-significant (NS), and highly significant (HS). Significant (S) meant that the p-value was between 0.01 and 0.05, and highly significant (HS) meant that it was less than or equal to 0.01.

Results

Table 1 presents the findings of the experiment, showing the percentage of periodontal diseases detected in both study groups. Of the male participants who only smoked cigarettes, 18.6% showed indications of gingivitis, whereas 81.4% had evidence of periodontitis. On the other hand, among male people who only smoked narghile (SN), 33.3% of them had signs of gingivitis, and 66.7% of them had periodontitis. The results shown in Table 2 reveal the mean and mean percentage values of all periodontal markers in both research groups. The findings show that the periodontitis SC group had significantly higher average values for PL (2.21), GI (1.95), BOP score 1 (63.81), PPD (4.75), and CAL (2.56). In contrast, the gingivitis SN group had lower average values for these parameters. The results shown in Table 3 demonstrate that there are highly statistically significant variations in periodontal parameters across all groups, as verified by an independent t-test. Table 4 shows the high mean value of ALP in the SC periodontitis group (146.34) and the lowest mean value in NS gingivitis type (40.05), while the high mean average of s-IgA revealed in the NS gingivitis (356.183) and the lowest mean average in the SC periodontitis group (214.351), and highly significant values appeared between the study group for ALP and IgA. The correlation coefficient (r) in the mentioned groups, as shown in Tables 5 and 6, indicates a strong and substantial negative link between ALP and IgA in both study groups with periodontitis. However, there was no significant correlation seen in the study group with gingivitis.

Table (1): The prevalence (percentage) of periodontal diseases in the SC and SN groups:

Groups	Percentage %*
Male cigarette-only smokers (SC) (n=125)	Gingivitis 18.6%
	Periodontitis in stage 1 and grade C 81.4%
Male narghile-only smokers (SN) (n=125)	Gingivitis 33.3%
	Periodontitis in stage 1 and grade C 66.7%

*%=percentage

Table (2): The study groups were analyzed for the mean values and standard deviation of

periodontal parameters.

Groups	Gingivitis									
SC group	**PLI* mean**	**SD***	**GI* mean**	**SD**	**BOP* Score 1%**	**SD**	**PPD* mean**	**SD**	**CAL* mean**	SD
	1.53	0.25	1.42	0.29	53.42	23.7	-	-	-	-
	Periodontitis									
	2.21	0.31	1.95	2.41	63.81	24.05	4.75	0.57	2.5	0.94
SN group	**Gingivitis**									
	1.05	**0.42**	**1.052**	**0.22**	**52.73**	**35.17**	**-**	**-**	**-**	-
	Periodontitis									
	2.05	**0.21**	**1.55**	**0.31**	**60.07**	**31.54**	**4.26**	**0.53**	**2.12**	0.82

PLI=plaque index
GI=gingival index
BOP=bleeding on the probing
CAL=clinical attachment loss
PPD=probing pocket depth
SD=standard deviation

Table (3): Analysis of the average values of the periodontal parameters across all the research groups

Groups	PLI	GI	BOP score1	PPD	CAL
Independent- t test for difference	40.06	64.12	112.03	43.25	32.62
***p-value**	0.001**	0.001**	0.004**	0.002**	0.002**
Sig.	HS	HS	HS	HS	HS

** highly significance
P-value=probability

Table (4): statistical analysis was conducted on the levels of ALP (U/L) and s- IgA in the study group with inter group compression for both variables.

Groups	ALP		s-IgA	
	Mean	**±SD**	**Mean**	**±SD**
SC (gingivitis)	52.13	35.52	305.617	34.134
SC (periodontitis)	146.34	60.34	214.351	29.41
SN (gingivitis)	40.05	32.17	356.183	30.23
SN (periodontitis)	123.25	43.60	245.344	24.613
Independent- t test for difference	32.56		47.72	
p-value	0.001		0.001	
Sig.	HS		HS	

s-IgA= salivary IgA
ALP= alkaline phosphatase

Table (5): correlation coefficient between the amount of ALP (U/L) and s-IgA with clinical

periodontal parameters of the study group with periodontitis.
ALP= alkaline phosphatase

Groups		PLI			GI			BOP score1			PPD			CAL		
		r	p	Sig.	r	p	Sig.	r	p	Sig.	r	p	Sig.	r	p	Sig.
SC	ALP	0.1562	0.04	S	0.1582	0.06	NS	-0.2032	0.14	NS	0.726	0.314	NS	0.568	0.12	NS
SN		0.713	0.02	S	0.356	0.032	S	-0.316	0.201	NS	0.642	0.170	NS	0.691	0.023	S
SC	s-IgA	0.174	0.01	S	0.432	0.043	S	0.271	0.07	NS	0.109	0.412	NS	0.541	0.01	S
SN		0.705	0.05	S	0.174	0.12	NS	0.361	0.02	S	0.753	0.03	S	0.418	0.094	NS

P=probability
r= Simple person's correlation coefficients

Table (6): correlation coefficient between the amount of ALP (U/L) and s-IgA with clinical periodontal parameters of the study group with gingivitis.

Groups		PLI			GI			BOP score1		
		r	p	Sig.	r	p	Sig.	r	p	Sig.
SC	ALP	-0.0012	0.143	NS	-0.023	0.172	NS	0.241	0.082	NS
SN		0.1428	0.0832	NS	0.202	0.056	S	0.451	0.0284	S
SC	s-IgA	-0.2044	0.314	NS	-0.195	0.215	NS	0.169	0.0163	S
SN		0.1505	0.523	NS	-0.0988	0.315	NS	-0.146	0.068	NS

Discussion

Tobacco smoking significantly contributes as a major risk factor and intensity of periodontal damage. Research revealed that smokers had a much higher chance of developing periodontitis, ranging from 2.5 to 7 times greater compared to non-smokers. The observed reduction in gingival inflammation and bleeding in smokers may be attributed to decreased gingival vascularity, characterized by reduced vascular density and a decrease in the lumen area of gingival arteries, which indicates greater vasoconstriction (32). Furthermore, research has shown that nicotine enhances the pace at which the gingival epithelium multiplies, hence potentially reducing the inflammatory clinical symptoms in the gingival tissues. The physiological effects of smoking on the development of periodontal disease include a reduction in the flow of gingival crevicular fluid. Smoking indicates an unevenness in the interaction between bacteria and the body's defense system, maybe caused by changes in the makeup of the plaque below the gum line, leading to an increase in the quantity and/or harmfulness of disease-causing microorganisms (33). Our findings indicate that cigarette and shisha smokers had a higher likelihood of developing periodontitis compared to gingivitis, which is consistent with previous research(34) , where the act of smoking is frequently associated with noteworthy alterations in the gingival tissue, as well as a reduction in the clinical manifestations of gingivitis(35) .

The results of this research indicate that those who smoke cigarettes have notably higher average values of plaque index (PLI), gingival index (GI), bleeding on probing (BOP), probing pocket depth (PPD), and clinical attachment level (CAL) in comparison to individuals who smoke nargile. This discovery aligns with prior study findings (36,37). The mean values of PPD, PLI, GI, and CAL were significantly different between the smoker's cigarette and narghile groups. This difference may be attributed to the impact of cigarette smoking on the reduction of oxidation reduction potential, leading to an increase in anaerobic plaque bacteria. This imbalance in host-bacterial interactions could be caused by alterations in the composition of subgingival plaque, such as an increase in the number and/or virulence of pathogenic organisms. Additionally, the host's response to bacterial challenge may also be affected. It is possible that a combination of these factors contributes to the observed differences(38,39) .

Saliva has been actively studied as a possible diagnostic tool in recent years since it is easy to collect and does not need intrusive procedures. Additionally, saliva contains a large number of indicators, including genetic information and proteins. Our finding revealed that higher level of ALP and lower concentration of IgA in saliva in cigarette smoker patients and had periodontitis (40,41). The impact of smoking on ALP levels may result from a disrupted equilibrium between free oxygen radicals and antioxidant levels, as well as changes in enzymatic activity in the liver which make to significant elevation of ALP in saliva when it compared with non-smoker (42,43) . The s-IgA test revealed a substantial disparity, with a low average level observed in individuals who smoke cigarettes. This discrepancy can be attributed to the impact of cigarette smoking, which has the potential to modify T-cell immunoregulation and B-cell differentiation(44). Consequently, this alteration leads to a reduction in the production of s-IgA, a crucial component that safeguards the oral mucosa against periodontal pathogenic bacteria, and A decreased amount of salivary secretory immunoglobulin A (s-IgA) might be considered a predisposing factor for oral conditions, particularly periodontal disorders(43,45) .

Our results revealed that a significant correlation of both ALP and IgA in saliva with periodontal parameters in two study groups and this is agree with Himabindu Lalkota et al, 2021 where they found the investigation established a direct link between serum ALP levels and the clinical measures, including plaque index (PI), gingival index (GI), probing pocket depth (PPD), and clinical attachment level (CAL). The strongest correlation was seen among persons who both smoke and have periodontitis, followed by those who do not smoke but have periodontitis. (46,47) .

Conclusion

To summarize, the results indicate that both narghile and cigarette smoking have harmful impacts on periodontal health, and the most effective method to avoid periodontal illnesses is to quit consumption. Nevertheless, the effect of narghile usage on periodontal health is milder in comparison to cigarette smoking. It is important to emphasize that the present research does not endorse the idea that smoking narghile is a fully harmless substitute for cigarette smoking, since it still presents dangers to periodontal health. Evaluating the levels of ALP and IgA in saliva serves as a dependable biological indicator for anticipating the initiation of periodontal disease in those who engage in smoking.

Conflict of Interest: None

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