The effect of cigarette smoking on salivary IgA and periodontal disease

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ABSTRACT

Background: Chronic periodontitis is an inflammatory disease of tissues supporting the teeth. Salivary compositions have been most intensely studied as a potential marker for periodontal disease. In this study, analysis of saliva provides a simple and non-invasive method of evaluating the role of salivary IgA (s-IgA) levels in periodontal disease by detecting the level of (s-IgA) in patients with chronic periodontitis smokers and non smokers patients and correlate the mean (s-IgA) levels with clinical periodontal parameters Plaque index (PLI) gingival index (GI), probing pocket depth (PPD) and clinical attachment level (CAL).

Materials and Methods: The study samples consists of (15) patients with chronic periodontitis who were non smokers (Group I) and (15) patients with chronic periodontitis who were smokers (Group II) of both gender with an age ranged (35-45) years were the periodontal parameters used in this study (PLI, GI, PPD and CAL), unstimulated salivary sample were collected from all subjects and the levels of salivary IgA (s-IgA) in each sample were analyzed for each group by using enzyme-linked immunosorbent assay (ELISA) technique. A statistical analysis was done by using excel 2013.

Results: There was a significant difference with high mean level in the clinical periodontal parameters in smokers group compared to non smokers with chronic periodontitis (PLI, PPD and CAL) except GI which showed no significant difference between the same groups. The biochemical finding showed significant difference with low mean level for (s-IgA) in smokers group compared to non smokers.

Conclusion: The findings in this study showed that the concentrations of salivary IgA might be used as an indicator for periodontal disease progression in smokers with chronic periodontitis as a resultant to the effect of smoking which lowering the concentration of the salivary IgA and subsequent reducing of the host's defense lead to increase in the progression of periodontal disease.

Keyword: Chronic periodontitis, smokers, (s-IgA), enzyme-linked immunosorbent assay (ELISA), saliva. (J Bagh Coll Dentistry 2015; 27(3):116-119).

INTRODUCTION

Periodontal disease (PD) is a disease with multiple factors that consists of hard and soft dental supporting tissues, microbial colonization, and host immune/inflammatory responses [1]. Smoking is consider a major risk factor for the periodontal disease development and progression [2] . The smoking effects on the periodontal tissue depend on the number of the cigarette smoked daily and the duration of the habit [3] .The higher the occurrence and the severity of periodontitis among cigarette smokers may be explained by the impairment of the host immune system as a result of cigarette smoking. Indeed, it has been shown that polymorph nuclear leukocyte functions such as chemotaxis, phagocytosis, and oxidative burst are decreased by the cigarette smoking substances [4,5].

Saliva is a complex fluid containing large number of host defense factors derived from the different salivary glands and the crevicular fluid [6]. Immunoglobulins (Igs) are protein molecules produced by specialized immune systems in response to the external agents penetrations, such as viruses, bacteria, protozoans, fungi, tumor cells, or tissues that are recognized as foreign because of the cell surface antigens presence [7].

The function of Igs is to bind with specific antigen molecules and, consequently, target bound molecules for inactivation and/or elimination of toxins, micro-organisms and parasites from the organism [8]. The humoral host immune responses play an important role in the oral environment protection because of the capability of antibodies to inhibit the attachment of microorganism to cell surfaces and aggregation/opsonization of these microorganisms [1]. In addition, antibodies are associated with alternative pathways that are also important in the colonization prevention and promotion the lysis of microorganisms, as well as neutralization of the toxic products [1,9] .

Salivary IgA is considered as the principal line of defense in the oral cavity against microorganisms invasions and plays an important role in the interactions of bacterial host [10,11] .

MATERIALS AND METHODS

The study participants included in the study were drawn from patients attending the Department of Periodontics in the Collage of Dentistry, University of Baghdad.

The study population included thirty patients with chronic periodontitis of both gender with an age ranged (35-45) years with no history for any systemic disease, chronic periodontitis in patients was defined as the presence of teeth with probing pocket depth ≥4mm with clinical attachment loss,

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this made according to the international classification system for periodontal disease [12], 15 of them were non smokers (Group I) and 15 were smokers (Group II).The criteria of smoker patients which were regularly smoked at least 10 cigarettes on average per day [13].

The exclusion criteria applied were a course of anti inflammatory or antimicrobial therapy within the previous 3 months, a history of regular use of mouth washes, Patients undergoing chemotherapy, radiotherapy, or medications that cause xerostomia.

The clinical parameters, plaque index (PLI) [14], gingival index (GI) [15] probing pocket depth (PPD) [16] and clinical attachment level (CAL) [17] have been clinically recorded. The subject rinses his mouth several times by water and then waits for 1-2 minutes for water clearance and then the unstimulated saliva was collected between 9-12 am. The collected samples centrifuged at 4000^{rpm} for 10 min, freeze at (-20^{o} **C**).

After all the samples were collected, the levels of salivary IgA were estimated by using enzyme-linked immunosorbent assay (ELISA) technique following the guidelines of the commercial kit provided by Demeditec Diagnostic GmbH D-24145 Kiel (Germany). The results were statistically analyzed with t-test and Pearson's coefficient of correlation.

RESULTS

The mean and standard deviation of PLI, GI, PPD, CAL and s-IgA in Group I and Group II were described in the (Table 1) which showed increasing in the mean of these parameters for chronic periodontitis smokers (Group II) as compared to chronic periodontitis non smokers (Group I) except for GI and s-IgA which showed reduction in the mean of these parameters for (Group II) compared to (Group I).

When the mean of clinical and biochemical parameters were compared between groups (Table 2), the PI, PPD, CAL and s-IgA showed significant differences between (Group I) and (Group II) while non significant relationship in the GI were found in the comparison between the same groups. The coefficient of correlation (r) in these groups described in the (Table 3) showed that s-IgA had inversed weak correlation in relation with GI and CAL for Group I.

Table 1: Records the mean and standard deviation of PLI, GI, PPD, CAL and s-IgA in Group I and Group II

Groups	Descriptive Statistic	PLI	GI	PPD	CAL	s-IgA
Group I	**Mean**	1.22	1.506	4.06667	4.12	310.5333
	±SD	0.256905	0.49058	0.144749	0.182052	63.43486
Group II	**Mean**	1.6	1.42	4.65333	4.58667	244.4667
	±SD	0.3251	0.60261	0.60458	0.46578	39.33168

Table 2: Inter group Comparison of means of PL, GI, PPD, CAL and s-IgA between Group I and Group II.

	PLI	GI	PPD	CAL	s-IgA
t-test	1.761	2.145	2.146	1.761	1.7613
P-value	0.001	0.669	0.001	0.001	0.002
Sig	S	NS	S	S	S

S= significant, NS= Non significant

Table 3: Correlation Coefficient (r) s-IgA and PLI, GI, PPD and CAL for Group I and Group II.

Groups		PLI		GI		PPD		CAL	
		r	p	r	p	r	p	r	P
G1	s-IgA	0.1592	0.5707	-0.0012	0.996	0.1428	0.611	-0.2044	0.464
G2		0.1759	0.530	0.0517	0.854	0.1505	0.592	0.3060	0.267

DISCUSSION

There was a significant difference in PLI in chronic periodontitis smokers (Group II) as compared with chronic periodontitis non smokers (Group I) and The findings of higher PLI in smokers are similar to a large body of controlled cross-sectional studies [18-20] and longitudinal studies [21,22]. The increased level of debris which has been observed in smokers group attributed to the personality trait leading to decrease in the oral hygiene and / or increase plaque formation rates [23].

Although there was no significant difference in GI between group I and group II and this might be coincided with the findings of Zuabi et al [24], the

mean of GI in smokers group showed low level than that in non smokers group and this might be due to the effect of smoking which had suppressive effect on the vasculature can be observed through less gingival redness, lower bleeding on probing and fibrous texture of the gingival tissue.

There was a significant difference with high level in the mean of PPD and CAL in smokers group compared with non-smokers group and this could be due to the effect of cigarette smoking on lowering of the Eh (oxidation reduction potential) and this could cause an increase in anaerobic plaque bacteria [25] ,so that lead to loss of balance in the host-bacterial interactions and this might be due to changes in the subgingival plaque composition, with increase in the numbers and / or virulence of pathogenic organisms; the host response changes against bacterial challenge, or a combination of both [26] , These findings were in agreement with Mahuca et al. [27] .

The result of s-IgA showed significant difference with low mean level in smokers as compared with non smokers group and this might be due to the effect of cigarette smoking which may alter T-cell immunoregulation and B-cell differentiation, generating a decrease in production of s-IgA, which protect the oral mucosa against periodontal pathogenic bacteria.

A low level of salivary s-IgA can be regarded as a risk factor for oral diseases, especially periodontal diseases [28,29] and this was in agreement with Al-Talib [30].As a result to small sample size and the sample taken from the saliva that was less site specificity as compared with the sample taken from gingival crevicular fluid so that there was weak correlation between s-IgA and clinical periodontal parameter in both groups.

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الخلاصه

الخلفيه:- النساغ المزمن هو مرض التهابي يصيب الانسجه الداعمه للاسنان.التراكيب اللعابيه تدرس بصوره مكثفه كعلامه مشتمله لامراض اللثه. في هذه الدراسه تحليل اللعاب يوفر طريقة بسيطة وغير مغزوه لتقييم دور مستويات الاجسام المضاده اللعابيه نوع (أ) في أمراض اللثة عن طريق الكشف عن مستوى هذه المضادات في المرضى الذين يعانون من التهاب اللثة المزمن في المدخنين وغير المدخنين من المرضى وربط مستويات هذه المضادات مع معلمات اللثة السريرية (PLI، GI، PPD وCAL).

المواد وطرق العمل:-عينات الدراسه تتالف من 15 مريض مصابين بالنساغ المزمن وهم من غير المدخنين وهؤلاء هم المجموعه الاولى من العينات والمجموعه الثانيه تتالف من 15 مريض مصاب بالنساغ المزمن وهم من المدخنين وجميع المرضى من كلا الجنسين وتتراوح اعمارهم من (35-45) سنه. مؤشر اللوحة الجرثوميه (PLI) مؤشر اللثة (GI)، سبر عمق الجيب (PPD) ومستوى المرفق السريرية (CAL) هي معلمات اللثة المستخدمة في هذه الدراسة،عينه من اللعاب غير المحفز تجمع من جميع المرضى ومستوى الاجسام المضاده اللعابيه نوع (أ) تحلل في كل عينه لكل مريض في كل مجموعه بواسطة تقنية فحص الانزيم المرتبط مناعيا (ELISA) .يعمل تقييم احصائي لمعرفة مستوى الاجسام المضاده اللعابيه نوع (أ) , معلمات اللثة السريرية وايجاد العلاقه بين معدل مستوى الاجسام المضاده اللعابيه والمعلمات اللثويه السريريه ولكل مجموعه.

النتائج:-كان هناك فرق معنوي مع معدل عالي المستوى في معلمات اللثه السريريه والتي تشمل (PLI, PPD and CAL) في مجموعة المدخنين المصابين بالنساغ المزمن بالمقارنه مع مجموعة الغير المدخنين ماعدا مؤشر اللثه (GI) والتي اظهرت عدم وجود فرق معنوي بين نفس المجاميع .وضحت نتائج الكيمياء الحيويه وجود فرق معنوي مع هبوط بمعدل الاجسام المضاده اللعابيه نوع(أ) في مجموعة المدخنين مقارنة بغير المدخنين .

الاستنتاج:-النتائج في هذه الدراسه بينت ان تراكيز الاجسام المضاده اللعابيه نوع (أ) ربما تستعمل كمؤشر لتقدم مرض اللثه المزمن في المدخنين المصابين بالنساغ المزمن وذلك كمحصله لتاثير التدخين والذي يقلل تركيزالاجسام المضاده اللعابيه نوع (أ) متبوع بانخفاض مقاومة الحائل مما يؤدي الى زيادة التقدم في امراض اللثه.

مفتاح الكلمات:-النساغ المزمن, المدخنين, الاجسام المضاده اللعابيه نوع الفا, فحص الانزيم المرتبط مناعيا, اللعاب