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THE EFFECT OF SMOKING ON PERIODONTAL DISEASE

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ABSTRACT
Gingivitis is reversible with good oral hygiene. However, in the absence of treatment, or if not controlled it can changed to periodontitis, where the inflammation results in tissue destruction and resorption of alveolar bone and ultimately tooth loss. Cigarette smoking is regarded as one of the most significant risk factors for the development and periodontal disease progression. In this study, the first group GI composed from 15 patients with gingivitis non smokers and the second group GII composed from 15 gingivitis smokers patients while 15 patients with chronic periodontitis non smokers for the third group GIII and 15 patients with chronic periodontitis smokers for the fourth group GIV were enrolled. Plaque Index (PI), Gingival Index (GI) were measured for all groups while Periodontal Probing Depth (PPD) and Clinical Attachment Level (CAL) were measured to assess the pattern of periodontal destruction for patients in the GIII and GIV. Comparisons were made between smoking and non-smoking groups with periodontal diseases. Smokers groups (GII and GIV) demonstrated greater amount of plaque with mean ± SD value (1.346 ± 0.32, 1.6±0.325) respectively than non smokers groups (GI and GIII) (1.16 ± 0.238, 1.22± 0.256) and lesser amount of gingival index in chronic periodontitis smokers group (1.073±0.191) than non smokers groups (1.333± 0.324) with significant difference. There were high significant differences in the intergroup comparison for PPD and CAL between GIII (chronic periodontitis non smokers) and GIV (chronic periodontitis smokers) with T test (-3.624 and -4.729) respectively. Smoking appears to have considerable adverse effects on the inflammatory process, thereby promoting the progression of periodontal disease in smokers.

KEYWORD: gingivitis, chronic periodontitis, cigarette smoking.

INTRODUCTION

Periodontal diseases can affect one or more of the periodontal tissues (e.g. alveolar bone, periodontal ligament, gingiva and cementum). While there are many different periodontal diseases that can affect these supporting tissues, by far the most common ones are plaque-induced inflammatory effects, such as gingivitis and periodontitis. Gingivitis is a non-destructive periodontal disease. The most common form of gingivitis, and the most common form of periodontal disease overall, is in response to the bacteria within the plaque,termed plaque-induced gingivitis. Gingivitis is reversible with good oral hygiene. However, in the absence of treatment, or if not controlled it can changed to periodontitis, where the inflammation results in tissue destruction and resorption of alveolar bone and ultimately tooth loss [1]. The clinical feature that distinguishes periodontitis from gingivitis is the presence of clinically detectable loss of attachment. This often is accompanied by formation of periodontal pocket and changes in the density and height of alveolar bone. In some cases, recession of the marginal gingiva may accompany attachment loss [2]. Chronic periodontitis is a chronic disease characterized by the interaction between Gr-ve bacteria and inflammatory response of the host, which result in tissue destruction and tooth loss [3] .Chronic periodontitis is associated with smoking, inadequate oral hygiene, diabetes, hypertension, rheumatoid arthritis, depression, anxiety, obesity, and other risk factors, including nutrition, alcohol consumption, and socioeconomic status [4], in addition to the effect of the race and the gender. Increases in the rate of disease progression may be caused by the impact of local, systemic, or environmental factors that may influence the normal host-bacterial interaction. Local factors may influence the accumulation of plaque, systemic diseases such as diabetes mellitus and human immune deficiency virus (HIV) infection may influence the host defenses; environmental factors such as stress and cigarette smoking also may influence the response of the host to plaque accumulation [5]. Chronic periodontitis is associated with the accumulation of plaque and calculus and has a slow to moderate rate of disease progression, but periods of more rapid destruction may be observed. This is relating to the length of time that the plaque has been in contact with periodontal structures and the cumulative effect of the disease[6] .The severity of periodontal diseases varies over time, depending on the quantity and quality of the biofilm and the presence of factors modifying the permanence of bacterial plaque[7]. The evidence that the occurrence and severity of periodontal disease are greater in smokers than non-smokers is well documented[8]. Smoking represents a major risk factor for increasing the prevalence and severity of periodontal destruction[9]. The host bacterial interactions normally seen in chronic periodontitis are altered, resulting in more aggressive periodontal breakdown. This imbalance between bacterial challenge and host response may be due to changes in the subgingival plaque composition, with increases in the

numbers and/or virulence of pathogenic organisms; changes in the host response to the bacterial challenge; or a combination of both[10]. In both cross sectional and longitudinal studies which provide strong epidemiological evidence that smoking confers considerable risk for periodontal disease[11]. Tonetti reported that smokers are between 2 and 14 times more likely to develop periodontal disease than non-smokers[12]. Abu-Ta'a *et al* observed that the older adults smokers are approximately 3 times more likely to have severe periodontal disease[13]. The central role of cigarette smoking in the etiology of periodontal attachment loss, a role in large part to the substantial relationship between smoking and severe periodontal disease[14]. The nicotine administration causes a rise in the blood pressure, an increase in heart rate, an increase in the respiratory rate and decreased skin temperature due to peripheral vasoconstriction[15].

MATERIALS & METHODS

Subjects 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 sixty male only. Those subjects divided into four groups and each group composed from 15 patientss. The first group GI (non smokers with gingivitis), the second group GII (smokers with gingivitis), the third group GIII (non smokers with chronic periodontitis) and the fourth group GIV (smokers with chronic periodontitis). The criteria for the selection of patients with chronic periodontitis include the presence of teeth with probing pocket depth ≥4mm with clinical attachment loss and this made according to the international classification system for periodontal disease[16]. The smokers groups include patients regularly smoked at least 10 cigarettes on average per day, for the last five years[17]. All patients with an age range from (30-50) year's old and with no history for any systemic disease. 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, any previous periodontal treatment, habits like chewing gum and previous chemotherapy, radiation therapy, or medications that cause xerostomia. The participants with chronic periodontitis were examined intra orally to determine the plaque index (PLI) (Silness and Loe 1964)[18], Gingival index (GLI) Loe and Sillness (1967)[19] , probing pocket depth (PPD)[20] and clinical attachment level (CAL)[21] while the examination of patients with gingivitis include PLI and GI only. The data were processed and analyzed using the statistics package for social sciences (excel 2013). Both descriptive (Statistical tables. b. Mean c. Standard deviation (SD) and inferential statistics Student (t-test) were used.

RESULTS

In this study the mean ±SD of PLI and GLI in (Group I) and (Group II) were (1.16 ±0.238, 0.75±0.367) and (1.36±0.320, 1.153±0.442) respectivly while the mean ±SD of PLI and GLI in (Group III and (Group IV) were (1.22 ±0.256, 0.75±0.367) and (1.333±0.324, 1.073±0.191) respectivly as showed in table1 and 2.The intergroup comparison between GI and GII for PLI and GLI were no significant difference with T test (1.74 and 1.67) respectively while the intergroup comparison between GIII and GIV for PLI (High significant with T test 3.55) and for GLI (no significant difference with T test 2.67) as showed in table3. The mean and SD for PLI in the GIII and GIV (1.22±0.256,1.6±0.325) respectively while for GIL (1.333±0.324,1.073±0.191) respectively as showed in table 1 and there were high signifcant differences in the comparison between GIII and GIV for PLI with T test 3.55 and significant difference for GLI with T test 2.67 as showed in table 2 and 3.The descriptive statistcs for the mean and SD of PPD for GIII and GIV (4.03±0.08,4.58±0.57) respectively while the CAL for the same groups were (4.12±0.18,5.52±1.12) respectively as showed in table 1. The intergroup comparison between GIII and GIV for PPD and CAL were high significant difference with T test (-3.624) and (- 4.729**)** respectively as showed in table 4.In the comparison between GI (gingivitis non smokers) and GIII (chronic periodontitis non smokers) for PLI and GLI there were no significant differences between them while the intergroup comparison for PLI and GLI between GII (gingivitis smokers) and GIV (chronic periodontitis smokers),there were significant differences between them as showed in table 2 and 3.

TABLE 1: Statistical description for PLI, GI, PPD and CAL findings for GI, GII, GIII and GIV

Clinical	GI		GII		GIII		GIV	
parameters	MEAN	SD	MEAN	SD	MEAN	SD	MEAN	SD
PLI	1.16	0.238	1.346	0.320	1.22	0.256	1.6	0.325
GI	0.75	0.367	1.153	0.442	1.333	0.324	1.073	0.191
PPD					4.03	0.08	4.58	0.57
CAL					4.12	0.18	5.52	1.12

TABLE 2: Inter groups Comparison of the plaque index mean for GI, GII, GIII and GIV

	T test	P value	Sig
GI and GII	1.74	0.09	NS
GIII and GIV	3.55	0.001	HS
GI and GIII	0.65	0.51	NS
GIV and GII	2.11	0.04	S

TABLE 3: Inter groups Comparison of means of gingival index for GI, GII, GIII and GIV

	T test	P value	Sig
GI and GII	1.67	0.222	NS
GIII and GIV	2.67	0.012	S
GI and GIII	0.64	0.525	NS
GIV and GII	2.87	0.008	HS

TABLE 4: Inter groups Comparison of the PPD and CAL mean for GIII and GIV

	Pocket Depth			Clinical Attachment Loss		
Group	t-test	p-value	Sig	t-test	p-value	Sig
Group III – Group IV	-3.624	0.002761	HS	-4.729	0.000323	HS

DISCUSSION

The mean level of PLI for GII slightly higher than non-smoker GI, in addition there was high significant difference in the mean level for GIV thannon smoker III and this finding is in agreement with the other previous studies (Haffajee & Socransky, 2001[22]; Calsina *et al*., 2002[23]; Maddipati *et al*., 2012[24]; Sreedhar & Shobha, 2012[25]).This increased plaque level which have been observed in smokers have been tentatively attributed to personality traits as the level of education, leading to decreased oral hygiene habits in smokers. Also the higher prevalence of dental plaque in smokers than non-smokers could be due to 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-Bander *et al*., 2000[26]). There was no significant difference between between GI and GII and this was in agreement with one study show no differences in the inflammatory status between smokers and non-smokers (Haffajee & Socransky, 2001[22]) while there was significant difference for GLI in the comparison between GIII and GIV with lesser level of inflammation in smokers GIV as compared to GIII and these results in agreement with studies like (Muller H-P *et al*. [27]; Basima & Omar, 2012[28] and Maddipati *et al*., 2012[24]). The explanation for the previous results was that cigarette smoking causes peripheral vessels vasoconstriction. Therefore this constrictive effect on gingival vessels would result in the suppression of vascular properties of inflammation such as bleeding, redness and exudation. So in smokers, suppression of the normally developing gingival inflammatory reaction associated with plaque may be due to tobacco smoke products that interfere with the vascular inflammatory response. The severity of inflammation increased in chronic periodontitis smokers GIV with high level of significance as compared to gingivitis smokers GII and this could be due to the greater amount of plaque in GIV in which represented the major etiological factor in periodontal disease and it is expected to be accumulating more in chronic periodontitis, in addition smoking is one of the most prevalent risk factors for chronic periodontitis. Regarding the PPD and CAL, the present study clarified that the values of these parameters were high significant difference in smokers GIV than non smokers GIII and these results in agreement with (Haffajee & Socransky, 2001[22]; Calsina *et al*., 2002 [23]and Basima & Omar, 2012[28]). The explanation for the increasing in the destruction of the periodontium and the bone resorbtion might be due to changes in the sub gingival plaque composition, with increase in the numbers and / or virulence of pathogenic organisms; changes in the host response to the bacterial challenge, or a combination of both.

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