EFFECT OF DIFFERENT DOSES OF CIGARETTE SMOKING ON PERIODONTAL HEALTH STATUS AND ALVEOLAR BONE LEVEL AMONG DIFFERENT GROUPS OF CURRENT SMOKER PATIENTS (COMPARATIVE STUDY) [+]

تأثير جرع مختلفة من تدخين السجائر على الحالة الصحية للانسجة ما حول السنية ومستوى العظم السنخي بين مجموعات مختلفة من المرضى المستمرين بالتدخين (دراسة مقارنة)

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Abstract:

Background: Cigarette smoking is very common. The aim of this study was to compare the effect of different doses of cigarette smoking on periodontal health status and alveolar bone level among different groups of current smoker patients

Materials and Methods: Seventy five subjects were participated in the study, the subjects with an age range (35-55) years old male, twenty five subjects were light smokers, other twenty five were moderate smokers and the last twenty five one were heavy smokers. All periodontal parameters: plaque index (PLI), gingival index (GI), bleeding on probing (BOP), probing pocket depth (PPD), clinical attachment loss (CAL) and cemento enamel junction to alveolar crest distance (CEJ-AC) were recorded for all teeth except the third molar which was excluded. The system included in the study was charge-coupled device (CCD) digital intra oral radiography by using digital bitewing radiograph, the unit of measurement was from cemento-enamel junction to alveolar crest distance (CEJ-Ac distance) per site in millimeters.

Results: there was high significant difference in mean plaque index between all groups, significant difference in mean gingival index between all groups while there was high significant difference of bleeding sites between all groups. We use scales for ease of estimation for probing pocket depth and clinical attachment loss parameters, it involve the scales (0,1,2,3) for probing pocket depth and scales (1,2,3,4) for clinical attachment level. The results showed that there was high significant difference in all scales between all groups in our study in probing pocket depth and clinical attachment level. High significant difference was found in mean cemento enamel junction –alveolar crest distance between all groups

Conclusions: Heavy smokers group revealed more periodontal tissue destruction and alveolar bone loss than moderate and light smokers groups. Moderate smokers group revealed more periodontal tissue destruction and bone loss than light smokers group by clinical examination and digital radiographic analysis.

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الخلفية: تدخين السجائر شائع جدا، الغاية من هذه الدراسة هي مقارنة تأثير جرع مختلفة من تدخين السجائر على الحالة الصحية للانسجة ما حول السنية ومستوى العظم السنخي بين مجموعات مختلفة من المرضى المستمرين بالتدخين
المواد والطرق: خمسة وسبعون شخص شاركوا في الدراسة، الاشخاص ذكور بمدى عُمر من ٣٥-٥٥ سنة، خمسة وعشرون شخص كَانوا قليلينَ التدخين، خمسة وعشرون آخرون كَانوا معتدلين التدخين ً والخمسة والعشرون الأخيرة كَانوا مدخّنون بإسرافَ.
كل المؤشرات للأنسجة ما حول السنية: مؤشر اللويحة الجرثومية، مؤشر التهاب اللثة، مؤشر النزف عند التسبير، مؤشر عمق الجيوب،مؤشر فقدان الانسجة الرابطة سريريا ومؤشر المسافة من منطقة الاتصال بين الميناء والسمنت الى حافة السنخ احتسبت على الاسنان كلها عدا سن العقل اللذي الغي.
الطريقة المتبعة في هذه الدراسة هي الة الشحن المزدوجة الرقمية الشعاعية لداخل الفم وكذلك باستعمال الفلم الرقمي ذات الاطباق، وحدة القياس كانت من منطقة الاتصال بين الميناء والسمنت الى حافة السنخ مقاسة موقعيا بالمليمترات.
النتائج: هناك فرق معنوي عالي في مؤشر اللويحة الجرثومية بين كل المجاميع، فرق معنوي في مؤشر التهاب اللثة بين كل المجاميع بينما كان هناك فرق معنوي عالي في مؤشر النزف عند التسبير بين كل المجاميع.
وضع مقياس من اربعة درجات لعمق الجيوب مبني على الزيادة في عمق الجيب وكذلك نفس طريقة القياس في مؤشر فقدان النسيج الرابط السريري، النتائج اظهرت هناك فرق معنوي عالي في كل الدرجات لكل مقياس بين كل المجاميع، هناك فرق معنوي عالي في مؤشرالمسافة من منطقة الاتصال بين الميناء والسمنت الى حافة السنخ بين كل المجاميع
الاستنتاجات: اظهرت مجموعة المدخنين بأسراف فقدان في الانسجة ما حول السنية والعظم السنخي اكثر من مجاميع المدخنين القليلين والمعتدلين، مجموعة المدخنين المعتدلين اظهروا فقدان في الانسجة ما حول الاسنان والعظم السنخي اكثر من مجموعة المدخنين القليلين عن طريق الفحص السريري وتحليل الاشعة الرقمية.

Introduction:

The relationship between tobacco smoking and oral diseases including periodontal disease has been studied in several clinical and epidemiological investigations over the years, [1]

Cigarette smoking is very common. Pindporg was one of the first investigators to study the relationship between smoking and periodontal disease [2]. Early studies showed that smokers had higher levels of periodontitis but they also had poorer levels of oral hygiene. [3 a,b], both cross sectional and longitudinal studies provide strong epidemiological evidence that smoking confers considerable risk for periodontal disease [4]. Many studies showed the relationship between the prevalence of periodontal disease with the number of the cigarette smoked per day and the number of years that the patient has smoked [5,6,7].

The effects of smoking on periodontal tissue depend on the number of the cigarette smoked daily and the duration of the habit [8]. In a cross-sectional study [9], it showed that in patients suffering from mild to advanced periodontitis, tobacco consumption increases significantly the value of the periodontal variables. Each extra-cigarette smoked daily elevated the gingival recession value by 2.3 %, PPD by 0.3 %, also they reported that smoking one cigarette per day, up to 10, and up to 20 increased the CAL by 0.5 %, and 10 % respectively.

Factors that are suspected to play a role in periodontal disease are; 1) type of tobacco product; 2) dose of these products; and 3) duration of exposure to smoking [10].

One study [11], reported a positive correlation between serum level of cotinine and probing pocket depth (PPD), clinical attachment loss (CAL) and alveolar bone loss (ABL) among subjects aged 24 to 64 years old suffering from periodontitis.

Other studies [12], showed that the greater the consumption of tobacco, the more alveolar bone loss was measured .Another study concluded that duration had possibly been more important than dosage [13].

However studying the effect of several doses of cigarette smoking on periodontal health status and alveolar bone level has not been studied, on this basis, this work has been conducted.

Materials and methods:

Human sample and study design

Participants included in the study were drawn from patients attending the Department of Periodontics in the Collage of Dentistry, University of Baghdad. Seventy five subjects were enrolled in this study, the subjects with an age range (35-55) years old male. Smoking status was assessed by means of a self-reported questionnaire (appendix) which included general information: name, age, periodontal parameter (plaque index, gingival index, bleeding on probing, probing pocket depth, clinical attachment level and alveolar bone level), general information regarding tooth brushing also information as whether the subject had ever used any tobacco products, number of cigarettes smoker per day and number of years the patient had smoked. The present study was carried out only on the current cigarette smokers, for the last five years [14], those subjects were divided into three groups:-

1- Group I (Light smokers group):- Twenty five subjects regularly smoked <10 cigarettes on average per day [15], with a mean range dose of 6.4 cigarettes / day, and mean duration of 7.2 years.

2- Group II (Moderate smokers group):- Twenty five subjects regularly smoked from 10-19 cigarettes on average per day [15], with a mean range dose of 12 cigarettes / day, and mean duration of 11.04 years.

3- Group III (Heavy smokers group):- Twenty five subjects regularly smoked ≥ 20 cigarettes on average per day [15], with a mean range dose of 24.88 cigarettes / day, and mean duration of 15.76 years.

Former smokers and never smokers were excluded, all subjects shouldn't have any systemic diseases or under medication affecting on periodontal health. Teeth included in the radiographic examination were from the distal surfaces of first premolars to mesial surfaces of second molars of upper and lower right quadrants, third molars, mesial surfaces of first premolars and the distal surfaces of second molars measurements were omitted [16]. The study is clinical-cross sectional, all participants were carefully informed about the aims of the investigation and they were free to withdraw at any time during the study. Each participant was gave complete medical and dental history to determine their susceptibility to the study. Oral examinations were done in the same visit. All subjects were selected regardless the periodontal health status but adjusted according to age, gender, all of them should be current smokers and had minimum smoking duration (smoking at least 5 years).

Oral examination:

All periodontal variables were recorded on four sites (mesial, buccal, distal and lingual) for all teeth except the third molar which was excluded.

The collected data include:-

(1) Assessment of dental plaque by (plaque index system (PLI)) according to Sillness and Loe [17].
(2) Assessment of gingival inflammation by (gingival index system (GI)) according to Loe and Sillness [18].
(3) Bleeding on probing according to Carrenza and Newman [19].
(4) Probing pocket depth measurement (PPD).

The PPD measurement has been performed using William probe and use a scale for ease of estimation it involve the following criteria:
Scale 0: Those include depth from 0.5-3 mm.
Scale 1: Those include depth from 4-5 mm.
Scale 2: Those include depth from 6-7 mm.
Scale 3: Those include depth more than 8 mm.

(5) Clinical attachment loss (CAL).

It was estimated by using William probe and use a scale for ease of estimation, it involves the following criteria:
Scale 1: those include loss from 1-3 mm.
Scale 2: those include loss from 4-5 mm.
Scale 3: those include loss from 6-7 mm.
Scale 4: those include loss more than 8 mm

Radiographic examination:

The system used in our study was a digital intra oral radiography contained a charged-coupled device, or CCD, sensor with cone angulation at + 10 was operated at 63 KVP and 5 mA, with an exposure time of 1 second. All images were displayed on monitor screen viewed at contrast 10º and brightness 10º, our calibration measurement was done in nearest 0.1 mm.

We use in this radiography, the bite-wing radiograph represented the molar and premolar areas of the right site of dentition. All radiographs were taken by the same operator. To measure the CEJ-AC distance each bitewing radiograph was magnified with magnification factor of 1.3 according to Planmeca OY 2003 manual.

Our standardization on taking the digital bitewing film images by asking the patients to sit down placing his head in straight direction and during the bitewing exposure, the patient closes his teeth during the exposure on the sensor holder. After adjusting the cone angle on +10º to the horizontal plane, we ask the patient to remain as still as possible and we move as far away from the X-ray tube as the length of the cable from the control panel permit. The measurement was done by linear measurement from CEJ to AC by choosing the ruler option of the digital software program. The radiographic landmarks, i.e., the CEJ and AC, were identified.

In cases where the bone level on the bitewing radiograph was indistinct, the bone structure nearest to the crown was scored. If 2 bone levels were detected, the most apical one was scored [20]. If the CEJ on one site could not be detected (because of a filling or caries), the most apical margin of enamel crown or proximal restoration was used.

We use in our study, the absolute technique [21], which stated that the alveolar bone level was assessed as the distance between 2 reference points, CEJ and AC. A distance exceeding 2 mm was considered as bone loss, all measurements were made to the nearest millimeter.

Results:

Inter group comparison for significant difference of mean plaque index scores, mean gingival index scores, percentage of sites with bleeding on probing, percentage of different scales of probing pocket depth, clinical attachment level and mean Cemento-enamel junction to alveolar crest distance were carried out by statistical analysis and the results showed the following:

1-Plaque index (PLI)

The means of plaque index were elevated in heavy smokers group= 1.926±0.433 compared with moderate smokers group=1.779±0.359 and light smokers group=1.421±0.253. The inter group comparison for plaque index by ANOVA test between those groups showed that there was a highly significant difference between them where the p.value <0.001 as shown in table 1, furthermore, by splitting the ANOVA test using LSD analysis for inter group comparison of each two groups separately showed that there was significant difference between group I and group II where p.value <0.05, Highly significant difference between group I and group III where p.value <0.001 and non significant difference between group II and group III where p.value >0.05 as shown in table 2

2-Gingival index (GI)

The means of gingival index were elevated in heavy smokers group= 1.113±0.125 compared with moderate smokers group=1.064±0.109 and light smokers group=1.01±0.186. The inter group comparison for gingival index by ANOVA test between those groups showed that there was a significant difference between them where the p.value <0.05 as shown in table 1, furthermore, by splitting the ANOVA test using LSD analysis for inter group comparison of each two groups separately showed that there was non significant difference between group I and group II where p.value >0.05, significant difference between group I and group III where p.value <0.05 and non significant difference between group II and group III where p.value >0.05 as shown in table 2

3-Bleeding on probing (BOP)

The sites that bleed were scaled as 1 while that not bleeds were scaled as 0. The number and percentage of non bleeding sites and bleeding sites in group I were [1917 (78.9%) and 511 (21.1%) respectively], in group II, they were [1975 (89.2%) and 241 (10.8%) respectively], while in group III, they were [2069 (94.4%) and 123 (5.6%) respectively] . The inter group comparison by chi-square between those groups showed that there was a high significant difference between them where the p.value <0.001 as shown in table 1.

4- Probing pocket depth (PPD)

The number and percent of sites that were scaled as 0 were 2275 (97.4%) in group I, 1996 (91.8) in group II and 1639 (75.7) in group III. The number and percent of sites that were scaled as 1 were 146 (1.9%) in group I, 196 (7.3%) in group II, and 490 (19.8%) in group III. The number and percent of sites that were scaled as 2 were 7 (0.7%) in group I, 24 (0.9%) in group II, and 60 (3.4%) in group III. The number and percent of sites that were scaled as 3 were 0 (0%) in group I, 0 (0%) also in group II, and 3 (1.1%) in group III. The inter group comparison between those groups was performed by using chi-square and the result showed that there was high significant difference for all scales at (p. <0.001) as shown in table 3

5- Clinical attachment level (CAL)

The number and percent of sites that were scaled as 1 were 2366 (97.5%) in group I, 2035 (91.8%) in group II, and 1659 (75.7%) in group III. The number and percent of sites that were scaled as 2 were 46 (1.9%) in group I, 160 (7.3%) in group II, and 435 (19.8%) in group III. The number and percent of sites that were scaled as 3 were 16 (0.6%) in group I, 21 (0.9%) in group II, and 75 (3.4%) in group III. The number and percent of sites that were scaled as 4 were 0 (0%) in group I, 0 (0%) in group II, and 23 (1.1%) in group III. The inter group comparison between those groups was performed by using chi-square and the result showed that there was high significant difference for all scales at (p. <0.001) as shown in table 3

6-Cementoenamel junction-alveolar crest distance (CEJ-AC) distance

The results showed that the means of CEJ-AC distance were elevated in heavy smokers group= 3.133±0.874 compared with moderate smokers group=2.199±0.69 and light smokers group=1.736±0.419. The inter group comparison for CEJ-AC distance by ANOVA test between those groups showed that there was a high significant difference between them where the p.value <0.001, furthermore, by splitting the ANOVA test using LSD analysis for inter group comparison of each two groups separately showed that there was significant difference between group I and group II where p.value <0.05, high significant difference between group I, group III and between group II, group III where p.value <0.001 as shown in table 4

Discussion:

1-Plaque index

According to the LSD analysis, it showed that, there was significant difference between group I and group II with more plaque accumulation in group II than group I, also high significant difference between group I and group III with more plaque accumulation in group III than group I, and non significant difference between group II and group III with more plaque accumulation in group III than group II. Many studies suggested that heat and accumulated product of combustion that result in tobacco stain as well as calculus are particular undesirable local irritants that increased with smoking [19,22],so with more cigarette smoked per day, there was more stain accumulated and plaque accumulation than less cigarette smoked per day.

2-gingival index

According to the LSD analysis, it showed that, there was non significant difference between group I and group II with slightly elevated gingival index in group II than group I, significant difference between group I and group III with elevated gingival index in group III than group I, and non significant difference between group II and group III with slightly elevated gingival index in group III than group II. So, according to our results there was increase in gingival inflammation whenever there was increase in the quantity of cigarette smoked per day, it can be explained by the alterations of gingival index follow the physiologic changes related to the disease process (more plaque accumulation in group III lead to more gingival inflammation than in group II and group I which showed less plaque accumulation). Also, the measurement were done by the gingival index scores considered as subjective measurement in which there may be over estimation or under estimation during calculation of mean gingival index for all groups with greater variability in different scores of gingival index (0, 1, 2, 3).

3-Bleeding on probing

The difference in the amount of gingival bleeding between different groups in our study was highly significant. According to our result, the number of bleeding sites among group III was greatly reduced when compared with group I and group II, the reduced bleeding has proposed to be caused by nicotine induced vasoconstriction [23], which was proportional to the amount of cigarettes smoked per day. Tobacco use has been associated with reduced permeability of peripheral blood vessels [24]. The measurement done by bleeding on probing was more objective as it indicate the presence of bleeding or not (score 0, 1), so, definitely it indicate the effect of smoking on blood circulation and the actual physiological process that happened in different groups of our study as it was clearly shown in bleeding on probing which showed highly significant difference between all groups.

4-Probing pocket depth

The significant increase in scale 1 in group III (490) when compared with group II (196) and group I (146) was clearly observed in this scale because the progress and severity of periodontal disease started at pocket depth 4-5 mm (pathological pocket formation), and all groups were selected in our study regardless the periodontal health status (all groups had chronic periodontitis) but the severity of PPD was more concentrated in scale 1 and this is may be due to cigarette smoking dose effect. The slightly increase in scales 2,3 in group III (60,3) than in groups II (24,0),group I (7,0) and in group II than in group I, it can be explained that among these two scales, the increase PPD usually associated with advanced disease in patients older than the age range in this study (35-55 years age) as it was clearly shown that periodontal disease is a chronic disease with more disease progression according to age and require more time to show its destructive effects. We can explained the general increase in PPD in group III compared with group II and group I from the fact that there is imbalance in the host-bacterial interactions and this imbalance between bacterial challenge and host response may be due to changes in the composition of the sub gingival plaque, 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. The imbalance became more severe and destructive when there was increase in cigarettes smoked per day which was more observed in group III then in group II and finally in group I

5-Clinical attachment level (CAL)

The general explanation for increase CAL in group III when compared with group II and group I can be derived from the same explanations of increased PPD in group III than in group II and group I which were listed previously as both of them represent the most important features of chronic periodontitis and reflect the progress and severity of periodontal tissue destruction.

6-Cementoenamel junction-alveolar crest distance (CEJ-AC) distance

According to the LSD analysis, it showed that, there was significant difference between group I and group II with slightly elevated CEJ-AC distance in group II than group I, high significant difference between group I and group III with elevated CEJ-AC distance in group III than group I, and high significant difference between group II and group III with slightly elevated CEJ-AC distance in group III than group II. There is experimental evidence to suggest that nicotine as well as cigarette smoke had detrimental effects on bone cells and osteoprogenitor cells [25], also, it's further realized that several other agents in Cigarette smoke may exert a toxic action on bone cell metabolism causing retardation or obstruction of regenerative functions, such an action would lead to an imbalance between build-up and breakdown functions [26]. The factors involved in the bone destruction in periodontal disease

are bacterial and host mediated. Bacterial plaque products induce the differentiation of bone progenitor cells into osteoclasts and stimulate gingival cells to release mediators that have the same effect [27]. Also plaque products and inflammatory mediators can also act directly on osteoblasts or their progenitors, inhibiting their action and reducing their numbers [28]. All these changes could be more pronounced in group III than in group II, group I and in group II more than in group I.

Table 1: Inter group comparison of mean plaque index, mean gingival index (by ANOVA test) and percents of scores 0, 1 of bleeding on probing (by Chi-square test) between Group I, group II and group III

	Plaque index			Gingival index			Bleeding on probing			
	mean	F-test	p.value	mean	F-test	p.value	Score 0 %	Score 1 %	Chi-square	p.value
Group I	1.421	15.87	0.000	1.01	3.24	0.045	78.9	21.1	256.7	0.00
Group II	1.779			1.064			89.2	10.8		
Group III	1.926			1.113			94.4	5.6		

Table 2: LSD analysis (splitting the ANOVA test) of plaque index and gingival index

LSD analysis	Plaque index		Gingival index	
	P.value	Sig.	P.value	Sig.
Group I and Group II	0.011	S	0.29	NS
Group I and Group III	0.00	HS	0.027	S
Group II and Group III	0.12	NS	0.086	NS

Table 3: Inter group comparison of percents of scales 0,1,2,3 of probing pocket depth and scales 1, 2, 3, 4 of clinical attachment level between Group I, group II and group III

	Probing pocket depth				Clinical attachment level			
	Scale 0%	Scale 1%	Scale 2 %	Scale 3 %	Scale 1%	Scale 2 %	Scale 3 %	Scale 4 %
Group I	97.4	1.9	0.7	0	97.5	1.9	0.6	0
GroupII	91.8	7.3	0.9	0	91.8	7.3	0.9	0
Group III	75.7	19.8	3.4	1.1	75.7	19.8	3.4	1.1
Chi-square	266.68				589.3			
p.value	0.00				0.00			

Table 4: Inter group comparison (by ANOVA test) and LSD analysis of mean Cementoenamel junction-alveolar crest distance (CEJ-AC) distance between Group I, group II and group III

Groups	CEJ-AC distance			LSD analysis	CEJ-AC distance	
	mean	f-test	p.value		p.value	Sig.
Group I	1.736	26.80	0.00	Group I and Group II	0.0067	S
Group II	2.199			Group I and Group III	0.00	HS
Group III	3.133			Group II and Group III	0.001	HS

Refernces:

1- Annemarie A, Dorthe H. “An "S-shaped" relationship between smoking duration and alveolar bone loss: Generating a hypothesis”. *J Periodontol*, Vol.72, pp. 1164-1171, 2001.

2- Pindporg J. “Tobacco and gingivitis. I. Statistical examination of the significance of tobacco in the development of ulceromembranous gingivitis and in the formation of calculus”. *J Dent Res*, Vol. 26, pp. 261-264, 1947.

3(a) - Brandtzaeg P, Jamison H. “A Study of periodontal health and oral Hygiene in Norwegian army recruits”. *J Periodontol*, Vol. 35, pp. 302, 1964.

(b)- Bradtzaeg P, Jamison H. “The effect of controlled cleansing of the teeth on periodontal health and oral hygiene in Norwegian army recruits”. *J Periodontol* , Vol. 35,pp. 308, 1964

4-Molloy J, Wolff LF, Lopez-guzman A Hodges JS. “The association of periodontal disease parameters with systemic medical conditions and tobacco use”. *J Clin Periodontol*, Vol. 31, pp. 625-632, 2004.

5- Grossi SG, Zamobon JJ, Ho AW., Koch G, Dunford R, Machtei EE, Norderyd OM, Genco RJ. “Assessment of risk for periodontal disease. I. Risk indicators for attachment loss”. *J Periodontol*, Vol. 65, pp. 260-267, 1994.

6- Grossi SG, Genco RJ, Machtei EE, Ho AW, Koch G, Dunford R, Zambon JJ, Hausmann E. “Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss”. *J Periodontol*, Vol. 66, pp. 23-29, 1995.

7- Haber J, Kent R. “Cigarette smoking in a periodontal practice”. *J Periodontol*, Vol. 63, pp. 100-106, 1992.

8- Calcina G, Ramon J, Echeverria J. “Effects of smoking on periodontal tissue”. *J Clin Periodontol* ,Vol. 29, pp. 771-776, 2002.

9- Martinez-Canut P, Lorca A, Magan R. “Smoking and periodontal disease severity”. *J Clin Periodontol*, Vol. 22, pp. 743-749, 1995. (Abstract).

10- WHO/IARC. “IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans”. Lyon: WHO/IARC, pp. 210-232, 1986.

11- Gonzalez YM, DeNardin A, Grossi SG, Machtei EE, Genco RJ, DeNardin E. “Serum cotonine levels, smoking and periodontal attachment loss”. *J Dent Res*., Vol. 75, pp. 796-802, 1996 .

12- Bolin A, Eklund G, Frithiof L, Lavstedt S. “The effect of changed smoking habits on marginal alveolar bone loss”. *Swed Dent J*, Vol. 17, pp. 211-216,1993.

13- Jette A, Feldman H, Tennstedt S. “Tobacco use: A modifiable risk factor for dental disease among the elderly”. *American J of Public Health*, Vol. 83, pp. 1271-1276,1993.

14- Machuca G, Rosales I, Lacalle JR, Machuca C, Bullon P. “Effect of cigarette smoking on periodontal status of healthy young adults”. *J Periodontol*, Vol. 71, pp. 73-78, 2000.

15- Okuyemi KS, Ahluwalia JS, Richter KP, Mayo MS, Resnicow K. “Differences among African American light, moderate, and heavy smokers”. *Nicotine Tob. Res*, Vol. 3, No.1, pp. 45-50, 2001.

16- Bergstrom J, Eliasson S, Preber H. “Cigarette smoking and periodontal bone loss”. *J Clin Periodontol*, Vol. 62, pp. 242-246, 1991.

17- Sillness J, Loe H. “Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition”. *Acta Odont Scand*, Vol. 22, pp. 121-135, 1964.

18- Loe H, Sillness J. “Periodontal disease in pregnancy. I. prevalence and severity”. *Acta Odont Scand*, Vol. 21, pp. 533, 1963.

19- Carranza FA, Newman MG. *Clinical periodontolgy*. 8th ed. W.B. Saunders Co., pp. 169, 356-357, 1996.
20- Wang H, Ma L, Li y, cho CH. "Exposure to cigarette smoke increases apoptosis in the rat gastric mucosa through a reactive oxygen species-mediated and p53-independent pathway". *Free Radical Biol Med* , Vol. 28, pp. 1125-1131, 1991.
21- Albander J, Rise J, Gjermo P, Johansen J. "Radigraphic quantification of alveolar bone level changes: A 2-year longitudinal study in man". *J Clin Periodontol*, Vol. 13, pp. 195-200, 1986.
22- Al-Bander JM, Stekfus GF, Adesanya MR, Winn DM. "Cigar, pipe and cigarette smoking as risk factors for periodontal disease and tooth loss". *J Periodontol* , Vol. 71, pp. 1874-1881, 2000
23- Preber H, Bergstrom J. "Occurrence of gingival bleeding in smoker and non-smoker patients". *Acta Odontol Scand*, Vol. 43, pp. 315-320, 1985.
24- Powell JT. "Vascular damage from smoking: Disease mechanisms at the arterial wall". *Vascular Medicine*, Vol. 3, pp. 21-28, 1998.
25- Walker L, Preston M, Magnay J, Thomas P, E1 Haj A. "Nicotine regulation of c-foc and osteopontin expression in human-derived osteoblast-like cells and human trabecular bone organ culture". *Bone*, Vol. 28, pp. 603-608, 2001.
26- Baljoon M, Natto S, Bergstrom J. "Long-term effect of smoking on vertical periodontal bone loss". *J Clin periodontal*, Vol. 32, pp. 789-797, 2005.
27- Schwartz Z, Goultschin J, Dean DD. "Mechanisms of alveolar bone destruction in periodontitis". *Periodontology*, Vol. 14, pp. 158, 2000.
28- Fermin A. Carranza. "Bone loss and patterns of bone destruction". *Carranza's Clinical Periodontology* ,9th Edition, pp. 354-370, 2002.