Early detection of periodontitis among young adult cigarette smokers and non-smokers using cone beam computed tomography

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ABSTRACT

Background: Periodontitis is an inflammatory disease that affects the supporting tissues of the teeth; Smoking is an important risk factor for periodontitis induces alveolar bone loss and cause an imbalance between bone resorption and bone deposition. The purpose of this study is to detect and compare the presence of incipient periodontitis among young smokers and non-smokers by measuring the distance between cement-enamel junction and alveolar crest (CEJ-Ac) using Cone Beam Computed Tomography (CBCT).

Material and methods: The total sample composed of fifty two participants, thirty one smokers and twenty one non-smokers (age range 14-22 years). Periodontal parameters: plaque index (PLI), gingival index (GI) were recorded for all teeth except the third molar while the radiographic analysis using CBCT was recorded on the Ramfjord teeth, the unit of measurement was from cement-enamel junction to alveolar crest distance (CEJ-Ac distance) per site in millimeters.

Results: The results obtained were a non significant difference for PLI, a significant difference of mean of GI between young smokers and non smokers. There was a highly significant difference in the general mean of CEJ-Ac distance between both groups. There was a significant difference between maxillary and mandibular teeth, a non significant difference between right and left sides among young smokers and non smokers.

Conclusion: The CBCT device plays an important role in detection the incipient form of periodontitis among young smokers and non-smokers, so we concluded that there is a highly significant difference in the general mean CEJ-Ac distance between young smokers and non smokers with increase distance in the maxillary teeth than that in the mandibular teeth.

Key words: Incipient periodontitis, cigarette smokers, CBCT. (J Bagh Coll Dentistry 2014; 26(2): 116-121).

INTRODUCTION

Periodontal diseases are bacterial infections of the gingiva, bone and attachment fibers that support the teeth and hold them in the jaw. The main cause of the diseases is bacterial plaque, a sticky, microbial film that constantly forms on teeth**.** (1) Periodontal disease occurs primarily due to bacteria within the gingival crevice or the periodontal pockets, it may be affected indirectly by many other risk factors occurring changes in the vascular system, severity of inflammatory reactions and host immunological responses. (2,3)A clear correlation between the presence of plaque and gingivitis has been established, it doesn't necessary that all individuals with gingivitis will progress to develop periodontitis even in the presence of putative pathogens. (4)

Chronic periodontitis similarly can have its initiation during adolescence and can later in life lead to tooth loss and associated systemic disease**.** The incipient form of chronic periodontitis and aggressive periodontitis can be treated successfully by appropriate intervention (5).

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The incipient periodontitis is often not diagnosed by clinicians for the lack of understanding of the disease and sufficient diagnostic acumens. When the disease is diagnosed, is it chronic or aggressive periodontitis, the tissue damage may be evident. Chronic periodontitis is generally slowly progression of periodontal disease that at any stage may undergo an exacerbation resulting in additional loss of attachment apparatus (6)**.** Smoking was a major factor associated with periodontal destruction in a group of young Jordanian adult's case study (7).

The effects of smoking on periodontal tissue depend on the number of the cigarette smoked daily and the duration of the habit (8)**.** Dental studies have reported that smokers have a greater amount of plaque and calculus deposits than their non-smoking counterparts of comparable age, and the quantity of calculus is correlated with the frequency of smoking (9,10)

The effects of smoking cessation and the use of daily interdental cleaning have not found to be an effective tool to change individual habits. In the future, motivational interviewing may be a more effective method to achieve a behaviour change if an extended education of dental

hygienists within this area will be implemented [11]. Smoking even one cigarette has been suggested to have the potential to cause a decrease in gingival blood flow. [12] Such small but repeated vasoconstrictive attacks and impairment of revascularization due to cigarette smoking may contribute to disruption of immune response and delay in the healing response, leading to an increased risk of periodontal disease. [13] Multiple cross-sectional and longitudinal studies have demonstrated that pocket depth, attachment loss, and alveolar bone loss are more prevalent and severe in patients who smoke compared with non-smokers. [14,15,16]

In vitro studies of the effects of tobacco products on neutrophils have shown detrimental effects on cell movement and the oxidative burst. The immune and inflammatory responses are critical to understanding the pathogenesis of periodontal diseases and they are orchestrated by a number of host-related factors, either intrinsic or induced. [17] The mechanisms by which smoking could influence the host control of bacteria included the effects of carbon monoxide enhancing growth of bacteria, which in turn provide growth factors for anaerobes, and damaging cells involved in the protection of the periodontal environment such as nutrophils, which could be affected by the formation of advanced glycation endproducts (AGEP) by smoking, which is either internal or external in origin. [18]

Internal sources include certain systemic conditions such as diabetes, Alzheimers disease, and uremia. [19] External AGEP are produced by the combustion of nicotine in cigarette smoke. Alveolar bone is one of the tissues that is most affected by the progression of periodontal disease. The mechanism of alveolar bone damage produced by smoking is related to the component of tobacco and nicotine metabolites which may act directly as local irritants on the gingival and alveolar bone or indirect because these components are absorbed in the lung which affects the cellular host defense or bone turnover. Another potential mechanism of bone loss in smokers may be the suppression of osteoprotegrin (OPG) production and a change in the receptor activator of nuclear factor–kappa ligand (RANKL) and OPG ratio (RANKL/ OPG ratio). [20, 21]

Computed tomography (CT) is a radiographic technique that using a rotating fan shaped beam to image a thin slice of the patient. The pursuit of 3D information has led to exploring the value of CT for the assessment of the alveolar bone height. [22] The usefulness of CT in analyzing 3D structure of alveolar bony defects in patient with periodontal disease was evaluated. The results showed that differences between CT film and actual reading (surgical) were negligible. It was concluded that the application of CT might be useful for analyzing 3D structure and diagnosis of alveolar bone defect. [23] By the end of the twentieth century and the beginning of the twenty-first, it has become apparent that CBCT imaging may indeed be the next major advancement in dentoalveolar imaging, providing true 3D imaging at a lower cost than conventional CT, with radiation risks similar to current methods of intraoral imaging, including panoramic and full mouth radiographic examination. [24] The CBCT units have been developed specifically for oral and maxillofacial imaging. [25,26]

The technique of CBCT could measure exactly the bone loss in term of distance between CEJ-Ac. CBCT would allow an accurate assessment of bone levels and accurate description of infrabony defects. This study could underline the fact that CBCT allows a very precise assessment of bone craters and furcation involvements. Considering advantages, limitations, risks, and machine-specific variations of CBCT, showed the accuracy and potential applicability of a specific CBCT for radiological periodontal diagnosis. [27] The disadvantages of CBCT imaging are poor soft tissue contrast and artifacts. Poor soft tissue contrast is not usually a problem in dental and maxillofacial imaging, because the main subjects of interest are generally mineralized tissues, i.e. teeth and bones. [28] The use of CBCT in clinical practice has a number of potential advantages over conventional tomography, such as easier image acquisition, greater image accuracy, lower effective radiation dose, faster scan time, and greater cost-effectiveness [26,28,29]. Data from the craniofacial region are often collected at higher resolution in the axial plane than those from conventional CT systems.

MATERIALS AND METHODS

Fifty two male subjects were enrolled in this study, with an age range (14-22) year male. All subjects were drawn from patients attending the special health center for dentistry in AL-Sadar city. Those subjects were divided into two groups:- 1-**Smokers group:** - Thirty one subjects regularly smoked at least 10 cigarettes on average per day [30] for the last two years [16] with an age range (14-22) years. 2-**Non-smokers group:** - Twenty one subjects didn't present any history of smoking with an age range (14-22) years. The exclusive criteria include the following:- 1- Past

smokers. 2- All subjects with any systemic disease. 3- All subjects with medication affecting on periodontal health for at least three months ago (anti inflammatory or antimicrobial therapy). 4- All subjects who smoke other than cigarette. The collected data in clinical examination were PLI and GI and all present teeth included except the third molars, while in radiographical examination the teeth included is Ramfjord teeth. The teeth numbering according to the FDI system are (16,21,24,36,41,44). [31]

The system we use it in our study is Kodak 9500 CBCT. This is the first system that has been installed in Iraq in the special health center of dentistry in AL-Sadar city in the December 2012. CBCT utilizes a cone shaped source of radiation and an area detector and that it acquires a full volume of images in a single rotation with no need for patient movement. By using the CBCT system accompanying software, any number of diagnostic images can be generated. On the coronal view we measured the CEJ-Ac distance in Ramfjord teeth under supervision of special radiologist (the bone loss occurred when the distance was more than 1.5 mm). The measurement was down by linear measurement of the CEJ-Ac distance by choosing the ruler option of the Kodak software program. The slice width used in the anterior teeth was equal to **19.5mm** while in the posterior teeth was equal to **2.1mm.** An extra-oral 3D radiograph was operated at **90Kv** and **10mA** with an exposure time **10.8 seconds**.

RESULTS

It was clearly shown that the mean of plaque index were elevated in Smokers compared with non-smokers, the mean of GI of non smokers group was higher than that of smokers group. (Table 1) It was obviously clear that the general mean of CEJ-Ac distance was elevated in smokers group 2.711 ± 0.463 compared with non-smokers group 0.968 ± 0.345. As a result of a higher levels of CEJ-Ac distance in young smokers than that of non smokers, when we compared CEJ-Ac distance in the maxillary, mandibular, right and left sides between smokers and non smokers we found that the distance in the maxillary and mandibular teeth in smokers is higher than that of non smokers with highly significant differences. The CEJ-Ac distance in the right and left sides is highly significant differences between smokers and non smokers with higher levels in smokers (Table2).

The result of this study is the CEJ-Ac distance in the max teeth is higher than that of the mandibular teeth with a highly significant difference among smokers and a significant difference among non-smokers. A non significant difference in the level of the alveolar bone loss was recorded between right and left sides in smokers and non-smokers (Table 3).

In smokers there was a non significant negative correlation between the mean of PLI and CEJ-Ac distance while, in non-smokers there was a significant positive correlation. It appears that there was a non significant positive correlation between the mild gingivitis and CEJ-Ac distance in both groups while there was a non significant negative correlation between the moderate gingivitis and CEJ-Ac distance (Table 4).

Table 1: Descriptive statistics and Inter group Comparison of means of plaque index and gingival index between smokers and non- smokers.

	Group	Mean	± SD	t-test	P-value	sig
PLI	**Smokers**	1.325	0.471	1.446	0.154	NS
	Non-Smokers	1.138	0.429			
GI	**Smokers**	0.974	0.288	2.896	0.006	S
	Non-Smokers	1.228	0.341			

Table 2: Descriptive statistics and Inter group Comparison of means of CEJ-Ac distance of smokers and non- smokers.

Group	Mean	± SD	t-test	P-value	sig
Smokers	2.711	0.463	14.687	0.000	HS
Non-Smokers	0.968	0.345			
Max. teeth Smokers	3.0097	0.544	14.087	0.000	HS
Max. teeth Non-Smokers	1.1094	0.353			
Mand. Teeth Smokers	2.413	0.481	12.436	0.000	HS
Mand. Teeth Non-Smokers	0.834	0.398			
Right Smokers	2.6555	0.463	12.511	0.000	HS
Right Non-Smokers	1.0595	0.433			
Left Smokers	2.768	0.506	14.981	0.000	HS
Left Non-Smokers	0.885	0.332			

Table 3: Descriptive statistics and intra group Comparison of means of CEJ-Ac distance in maxillary and mandibular teeth, right and left side.

		Mean	± SD	t-test	P-value	sig
Smokers	**Max.**	3.0097	0.544	4.576	0.000	HS
	Mand.	2.4129	0.481			
	RIGHT	2.655	0.463	0.918	0.363	NS
	LEFT	2.7677	0.5061			
Non-Smokers	**Max**	1.109	0.353	2.375	0.022	S
	Mand	0.834	0.398			
	RIGHT	1.0595	0.433	1.466	0.15	NS
	LEFT	0.8849	0.332			

Table 4: The coefficient of person correlation (r) of the level of CEJ-Ac distance with plaque index, mild and moderate gingivitis among smokers and non-smokers and their level of significant differences

	Group	r	P-value	Sig
Plaque Index	**Smokers**	-0.035	0.850	NS
	Non-smokers	0.530	0.013	S
Mild Gingivitis	**Smokers**	0.22	0.431	NS
	Non-smokers	0.999	0.031	NS
Moderate Gingivitis	**Smokers**	- 0.04	0.880	NS
	Non- Smokers	- 0.309	0.288	NS

DISCUSSION

The mechanism of alveolar bone damage produced by smoking is related to the components of tobacco and nicotine metabolites which may act directly as local irritants on the gingival and alveolar bone or systemically because these components are absorbed in the lung, which affects the cellular host defense or bone turnover. Nicotine can suppress the proliferation of cultured osteoblasts while stimulating osteoblast alkaline phosphatase activity. [32]

Recently, some in vitro studies provided other possible intimate mechanisms by which smoking may affect bone metabolism. Rosa et al. [32] reported that nicotine increased the secretion of IL-6 and TNF-α in osteoblasts and also nicotin increased the production of tissue-type plasminogen activator, PGE2 and MMP, thereby tipping the balance between bone matrix formation and resorption toward the latter process. Al Qutub [33] observed a higher mean alveolar bone loss in the max. teeth than the mand. in smokers and non smokers.

Bergstrom [34] found that the alveolar bone loss was more prominent in the maxillary teeth than the mandibular teeth in the form of percentage. The possible explanation could be that the cortical bone in maxilla is thinner, less dense and more rigid than that in mandible; therefore, the maxillary alveolar bone undergoes resorption more readily than that of the mandible. In non smokers, the positive correlation between PLI and bone loss suggest that, the bacterial plaque play a major and an important role in alveolar bone loss. Since this study conducted on young age group of

patient, longer time using tobacco could have a stronger effect on the bone destruction.

Sch tzle et al. [35] demonstrated that, in smokers and non smokers younger than 30 years of age, there was little or no difference in the standard of oral hygiene, so the bone loss independent of plaque levels and severity of gingivitis. In conclusion the CBCT reveal that the prevalence of incipient bone loss among smokers was 100% while it was 4.76% among non-smokers. There was a highly significant difference in the mean CEJ-Ac distance between smokers and non smokers with higher mean CEJ-Ac distance in smokers than non-smokers group. A significant difference in the amount of bone loss between maxillary and mandibular teeth for both groups was found.

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