



THE EFFECT OF SMOKING ON CLINICAL ATTACHMENT LOSS IN CHRONIC PERIODONTITIS

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ABSTRACT

Chronic periodontitis is an inflammatory disease of the supporting tissues of the teeth which will result in progressive destruction of the periodontal ligament, damage to alveolar bone causing pocket formation, recession, or both. Many risk factors are linked to periodontitis; smoking is one of the strongest risk factors, study the differences of clinical attachment loss between smoker and non smoker. 50 adult subjects aged between (40 -70) years, were divided into two groups: 25 smokers and 25 controlled (non-smoker). The plaque index, gingival index and clinical attachment loss were measured. There was a significant difference in clinical attachment loss and plaque index in smoker group than the control group while gingival index show no significant difference (the gingival index is higher in non-smoker than in smoker). Smoking has a great effect on the clinical attachment loss.

KEYWORD: periodontitis, smoker, clinical attachment loss.

INTRODUCTION

Periodontitis is an inflammatory disease that happens when there is imbalance between pathogenicity of bacteria of dental plaque and the host response to this bacterial attack^[1]. Individual response to periodontal infection is modified by many risk factors; one of the most important lifestyle risk factors is smoking^[2]. Evidence suggested that cigarette smoking is an important risk factor for destructive forms of periodontal diseases. Smoking may be causally associated with periodontitis and may result in less favorable response to treatment^[3]. Different studies had reported that most refractory periodontal condition cases occur in smokers and that there is a positive relation between the quantity, duration of smoking and the amount of bone loss^[4]. Human studies showed that cigarette smoke exposure may cause impairment in the endothelium-dependent vasodilatation in both macro- and micro vascular beds^[5]. The exposure to tobacco will decrease gingival blood flow, this reduction in the blood supply to the periodontium may contribute to higher prevalence and more rate of disease progression in smokers^[6]. Exposure of human gingival fibroblasts to nicotine may reduce the cell viability by time and dose dependent and increase the generation of reactive oxygen species, which peaked at two hours of nicotine exposure^[7,8]. The immune-inflammatory response that occurs in periodontitis will result in an apical migration of the epithelial attachment with loss of periodontal soft and hard tissues^[9]. For this reason, the clinical attachment loss is an important measurement because it represents a

clinical measurement of the loss of connective tissue attachment from the root surface^[10].

MATERIALS & METHODS

The subjects in this study were recruited from the department of periodontology of the teaching hospital in the college of dentistry- University of Baghdad. The study sample included fifty patients equally divided into two groups:

- Smoker
- Non-smoker

All the participants were randomly selected, they were systemically healthy and they didn't use any medication in the last three months. Informed consent was obtained from the participants before starting periodontal examination. The plaque index (PI) ^[11], gingival index (GI) ^[12] and clinical attachment loss (CAL) were measured for each patient. Scaling was done to all the subjects before measuring CAL which was measured from the cemento-enamel junction to the base of pocket for each surface of each tooth by the use of periodontal probe. Statistical analysis was done using Microsoft Excel.

RESULTS

The mean of (PI) was significantly higher in the smoker group than the non-smoker ($P < 0.001$). The mean of the (GI) was higher with no significant difference in the smoker group than the non-smoker group ($P = 0.882$), while the mean of (CAL) was higher in the smoker group than the non-smoker group with high significant difference ($P < 0.001$). (Table 1 and 2)

TABLE 1: Descriptive statistics of periodontal parameters in smoker and non-smoker groups

Groups	Periodontal parameter	Mean
Non-Smoker	PII	1.27±0.34
	GI	1.25±0.33
	CAL	4.35±0.73
Smoker	PII	1.73±0.41
	GI	1.31±0.39
	CAL	6.5±0.81

TABLE 2: t-test of periodontal parameters between smoker and non-smoker

Periodontal parameter	T test	p-value	Sig.
PII	3.94	0.0003	S
GI	0.148	0.882	NS
CAL	8.93	0.0001	HS

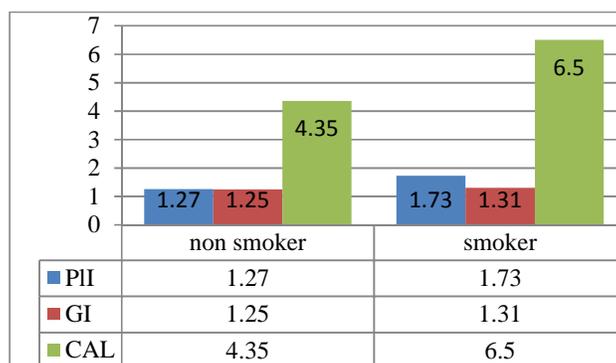


FIGURE 1- illustrating the descriptive statistics of PII, GI and CAL

DISCUSSION

The plaque index was significantly higher in the smoker group, this agrees with early studies that examined the relation between smoking and oral cleanliness, they found that smokers had poorer oral hygiene than non-smokers^[13-16]. Other studies found less plaque in smokers^[17]. There is no evidence that smoking increases the rate of plaque development or effects salivary precipitation. The increase in plaque may be due to heat and accumulated product of combustion causing tobacco stain as well as calculus^[18]. The greater plaque accumulation in smoker may be related to the inadequate oral hygiene measures and also some studies showed that tooth brushing habits in smokers tend to be less favorable than in non-smokers^[19]. Smokers may spend significantly less time brushing and cleaning their teeth which may result in significantly more plaque remaining on their teeth after brushing than non-smokers^[20]. The GI showed no significant difference between smoker and non-smoker and this result not in agree with *studies that* found that GI was higher in non-smoker than in smoker^[21,22]. Regarding CAL, the present study showed that the mean of CAL in the smoker group was higher than the non smoker this could be explained by the change in the sub-gingival plaque composition, the virulence of subgingival bacteria and change in the host response which increase the destruction of periodontium and bone resorption and these results are in agreement with ^[23,24]. The use of nicotine in tobacco can cause damage to the collagen tissues, by increasing the production of collagenase, suppressing the growth of gingival fibroblast, and the production of collagen and fibronectin^[25].

CONCLUSION:

Smoking has an adverse effect on the periodontium by increasing the bone loss associated with periodontitis.

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