The effect of smoking and passive smoking on periodontal health status and salivary enzymes level

(Comparative and Biochemical study)

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in

Periodontics

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Abstract

Background: Smoking is considered a major risk factor for development and progression of periodontal disease. Environmental tobacco smoke (ETS) exposure is probably one of the most important public health hazards in the community. Non smokers exposed to secondhand smoke are recognized to be at increased risk of periodontal disease. There is no risk-free level of exposure to secondhand smoke.

Aims of the study: The purpose of this study was to evaluate the effects of smoking and passive smoking on periodontal health status and on the salivary levels of alkaline phosphatase (ALP), lactate dehydrogenase (LDH) and creatine kinase (CK), and to correlate the level of these enzymes with clinical parameters of periodontal health status in each group.

Materials and methods: Seventy two subjects were enrolled in the study, the subjects with an age range (30-45) year's old males and females without any history of systemic disease. The subjects were divided into two study groups (22 passive smokers and 25 smokers) and control group (25 non smokers).

Unstimulated saliva sample was collected and all periodontal parameters (plaque index, gingival index, bleeding on probing, probing pocket depth and clinical attachment level) were recorded and for each subject. The collected saliva was centrifuged and clear supernatant saliva was collected and kept frozen until biochemical analysis of salivary enzymes.

Results: The result showed that the mean ALP in saliva was significantly higher in smoker than the non smoker group. Passive smoker group had higher level of salivary ALP than the non smoker group but the difference between them was not significant.

There was a highly significant difference in the salivary LDH and CK levels between smokers and non-smokers groups. The level of salivary LDH and CK in passive smoker group was significantly higher than the non smoker group.

A significant difference was found in plaque index (PLI) between smokers and non smokers. No significant difference existed in the mean PLI between passive smokers and non smokers.

No significant differences in gingival index (GI) was found for all groups, while there was highly significant difference in the number of bleeding sites.

A highly significant difference in probing pocket depth (PPD) was found among all groups. There was increase in the total number of all scores of PPD (scores 1and 2) in smokers compared with non-smokers except for score 0 which was decreased. The passive smoker group also showed increased PPD with its different scores in (scores 1, 2 and 3) compared with non-smokers group except for score 0 which was decreased.

There was a highly significant difference in the in the clinical attachment level (CAL) among all groups. There was increased CAL with its different scores in smokers group when compared with non-smokers group. The passive smokers showed increase percentage of CAL only in score 1 compared with non-smokers.

Conclusions: smokers group revealed more periodontal tissue destruction and alveolar bone loss than non-smokers group represented by deeper pockets and more clinical attachment level, also it was concluded that passive smoking have negative impact on periodontal health and can consider as risk factor for periodontal disease. Salivary enzymes (ALP, LDH and CK) are considered as good biochemical markers of periodontal tissue destruction and can be used to evaluate the effect of smoking and passive smoking on periodontal health status.