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Effects of Light Smoking on Salivary Levels of Alkaline Phosphatase and Osteocalcin in Chronic Periodontitis Patients

A Thesis
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Abstract

Background Chronic periodontitis is an inflammatory disease that affects the supporting tissues of the teeth and it is common among adults. Smoking is an important risk factor for periodontitis that induces alveolar bone loss. Alkaline phosphatase enzyme is involved in the destruction of the human periodontium. It is produced by many cells such as polymorphonuclear leukocytes, osteoblasts, macrophages and fibroblasts within the area of the periodontium and gingival crevice. Osteocalcin is one of the most abundant matrix proteins found in bones and the only matrix protein synthesized exclusively there. Small osteocalcin fragments are found in areas of bone remodeling and are actually degradation products of the bone matrix.

Aims of the study 1. Investigate whether light smoker chronic periodontitis patients exhibit different salivary concentrations of Alkaline Phosphatase and Osteocalcin compared to the non-smoker counterpart and compare to light smoker and non-smoker control groups and 2.Correlate the clinical periodontal parameters (plaque index, gingival index, bleeding on probing, probing pocket depth and clinical attachment level) to the biochemical findings in light smokers and non-smokers chronic periodontitis and control groups. 3. Estimation of the salivary PH and flow rate and correlate them with clinical periodontal and biochemical parameters.

Materials and Methods Five ml of unstimulated whole saliva samples and full-mouth clinical periodontal recordings (plaque index, gingival index, bleeding on probing, probing pocket depth and clinical attachment level) were obtained from study groups (25 light smokers and 33 non-smokers subjects, both with chronic periodontitis) and control groups (8 light smokers and 13 non-smokers subjects, both with healthy periodontium). All subjects were systemically healthy males, with age range (30-50) years. Salivary Alkaline phosphatase and Osteocalcin levels were determined by Colorimetric and Enzyme-linked Immunosorbent Assay, respectively. Salivary PH and flow rate were also measured.

Results Smoker chronic periodontitis patients revealed non-significant differences in clinical periodontal parameters with non-smoker counterparts **(P > 0.05)** in terms of plaque index, probing pocket depth, clinical attachment loss, salivary PH and flow rate, with slight increase in plaque index value in smoker chronic periodontitis group**(1.42±0.46)** than non-smoker chronic periodontitis group **(1.38±0.50)**, while there was slight decrease in mean value of probing pocket depth **(3.90±1.78)**, clinical attachment level **(3.22±0.93)** and PH **(7.58±0.50)** in smoker chronic periodontitis group than non-smoker chronic periodontitis group **(4.27±1.98), (3.74±0.85), (7.73±0.33)** respectively. Flow rate value was higher in smoker chronic periodontitis **(4.80±2.69)** than non-smoker chronic periodontitis group **(4.67±2.09).** While there were highly significant differences in terms of gingival index and bleeding on probing **(P ≤ 0.01**). Osteocalcin levels were lower in smoker chronic periodontitis group (**0.13±0.20**) than non-smoker chronic periodontitis group (**1.09±2.26**) with significant difference **(0.05 ≥ P > 0.01)**. Highly significant, strong, positive correlations were found between Osteocalcin concentration with plaque index and PH in non-smoker control group. Correlation analysis between Osteocalcin concentration and flow rate in smoker chronic periodontitis group revealed a significant, moderate, positive correlation.

Mean of Alkaline phosphatase level was lower in smoker chronic periodontitis **(11.14±4.53)** than non-smoker chronic periodontitis **(11.45±4.17)** with a non-significant difference, while there was a significant difference in Alkaline phosphatase concentrations between smoker and non-smoker control subgroups. Correlation analysis between ALP concentration and clinical periodontal parameters (plaque index, gingival index, bleeding on probing, probing pocket depth and clinical attachment level) in chronic periodontitis smoker group appear non-significant, weak, negative and related non-significantly, weakly, positively with PH and flow rate. Clinical attachment level and PH correlated non-significantly, weakly, negatively with Alkaline phosphatase concentration, while plaque index, gingival index, bleeding on probing, probing pocket depth and flow rate related non-significantly,

weakly, positively with Alkaline phosphatase concentration in chronic periodontitis non-smoker group.

There was a significant, moderate, negative correlation between salivary PH and bleeding on probing and significant, weak, negative correlation regarding probing pocket depth in smoker chronic periodontitis group.

A significant, weak, positive correlation between salivary flow rate and clinical attachment level was found in non-smoker chronic periodontitis group.

There were non-significant differences between smoker chronic periodontitis and smoker control groups in terms of salivary PH, flow rate, Osteocalcin and Alkaline phosphatase concentrations. There were non-significant differences between non-smoker chronic periodontitis and non-smoker control groups in terms of PH, flow rate, Osteocalcin and Alkaline phosphatase concentrations.

There were non-significant, weak increase in Alkaline phosphatase concentration compared to the increase in Osteocalcin concentration in smoker and non-smoker chronic periodontitis and smoker control groups, while there was a non-significant, weak decrease in Alkaline phosphatase concentration with increase in Osteocalcin concentration in non-smoker control group.

Conclusion Within the limits of this study, it may be suggested that suppression of salivary Osteocalcin levels by smoking and weak increase in Alkaline phosphatase in smokers groups, may explain the deleterious effects of smoking on periodontal health status.