

Interleukin -6 and Tumor Necrosis Factor-alpha in Gingival Crevicular Fluid of Smoking and Non-Smoking Chronic and Aggressive Periodontitis Patients.

By

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Abstract:

The local host response to periodontitis has been studied by biochemical analysis of gingival crevicular fluid (GCF). GCF cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α), have been shown to have a role in regulating the cellular inflammatory response in the periodontium. Tobacco smoking is strongly associated with destructive periodontal disease, although the mechanisms of its negative influence are not well understood. The aim of this study was to determine the levels of IL-6 and TNF- α in the GCF of chronic (CP) and aggressive (AP) periodontitis patients, and to investigate the relationship between these cytokines and smoking. A total of 36 subjects participated into the study, classified as 20 generalized CP patients, further subdivided into 10 smokers (S) and 10 non-smokers (NS); 16 generalized AP patients, further subclassified into 8 S and 8 NS. The CP patients had severe periodontitis to match the periodontal destruction seen in the AP group. For standardization, smokers were chosen as being heavy smokers (at least 20 cigarettes/day). Initial periodontal therapy was performed to all patients to match amount of plaque, and 1 month post surgery was considered baseline. Clinical parameters including plaque index (PI), retention index (RI), papillary bleeding index (PBI), probing depth (PD) and clinical attachment level (CAL) were recorded at baseline and 3 months post treatment. Full mouth periapical radiographs were taken at baseline and alveolar bone destruction was assessed by the Schei method for diagnosis. GCF was collected by means of filter paper from four sites per patient (with the most severe destruction) at same time intervals. The contents of IL-6 and TNF- α were measured in using ELISA assays. At baseline, all clinical variables did not show significant differences between S and NS in CP patients, except for significant lower PBI in S than NS. At 3 months, S CP patients revealed significantly higher PI and RI scores, whereas NS showed significant reduction in CAL. AP patients demonstrated the same results, but CAL scores did not improve significantly at 3 months in NS. S CP and AP patients demonstrated a non-significant increase in GCF IL-6 and TNF- α concentrations, when compared to NS. In both groups, NS showed a significant decrease in GCF IL-6 concentrations at 3 months compared to baseline. S CP and AP patients showed a significant increase in GCF TNF- α at baseline compared to NS, in addition to a significant decrease in NS at 3 months compared to baseline. GCF IL-6 concentration showed significant negative correlation with PD in NS CP patients, and significant positive correlation with PBI in NS AP patients; whereas GCF TNF- α revealed significant negative correlation with RI in S CP patients. In conclusion, the enhanced production of inflammatory cytokines in the presence of smoking may have clinical consequences, and may elucidate the mechanism of negative influence of tobacco smoking on the periodontium. The reduced improvement following periodontal therapy in S compared to NS may reflect the systemic effect of smoking on both host response and healing process.

Introduction

Periodontal diseases are probably one of the most common chronic diseases in adults, and are initiated by an overgrowth of specific Gram-negative bacterial species which replace the normal microbiota.⁽¹⁾ This may be due to a disequilibrium in the host, which may be caused by several factors, such as modification of the environmental conditions of the infected site, a significant decrease in the proportion of beneficial

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