SALIVARY PAF LEVELS IN EARLY ONSET AND ADULT PERIODONTITIS PATIENTS THROUGHOUT INITIAL PERIODONTAL THERAPY

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INTRODUCTION

Periodontal diseases reflect a constellation of inflammatory mediators which act individually or synergistically to promte disease progression.[1]

Bacteria or their products and components are the driving force behind the observed tissue destruction. Substances from periodontopathic bacteria initiate and drive the inflammatory response and their continued presence is essential for maintenance of the inflammation. Nevertheless, endogenous molecules mediate the inflammatory process and play a major role in its ampification and perpetruation and in the ensuing tissue destruction.[2]

Cellular response to inflammation involves the formation and accumulation of bioactive mediators. Platelet activting factor (PAF) is among the most potent of these mediators, as it leads to cell damage through several mechanisms.[3]

PAF is a family of structurally related, acetylated phospholopids capable of inducing marked pro-inflammatory responses.[4,5] Although originally named for its ability to cause aggregation and histamine release from rabbit platelet,[6] PAF has since been documented to promote a wide range of phlogistic processes which are initiated via specific PAF receptors on various cells and tissues. These processes include the stimulation of diverse targets and effects, such as polymorphonuclear leukocyte (PMN) activation (e.g. chemotaxis, aggregation, lysosomal enzyme release, arachidonic acid metabolism, and superoxide production), monocyte macrophage aggregation and phagocytosis, eosinophil activation, increased vascular permeability, vasoconstriction, and smooth muscle contraction.[4,5,7]

PAF is rapidly synthesized by various inflammatory cells after activation by either immunologically or nonimmunologically triggered signals.[8] Interestingly, PAF is produced by a variety of activated inflammatory cells including many of the same cells which it targets, such as PMN, vascular endothelial cells, monocytes, eosinophils, basopils, platelets and lymphocytes. Thus the pleiotropic effects of these acetylated phospholipids develop as a result of paracrine

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and autocrine stimulation of the inflammatory process.[1]

The presence of PAF in normal human mixed saliva was first reported in 1981 by Cox et al.[9] Pure parotid saliva apparently has no detectable PAF activity, which suggests that PAF in mixed saliva originates from a source other than this salivary gland.[9] Moreover, edentulous, healthy subjects have undetectable or significantly decreased levels of salivary PAF.[10] In combination these results suggest that PAF in mixed saliva may be derived from periodontal tissues.[8]

Subsequent investigations indicate that the gingival crevice appears to be the source of PAF in normal human mixed saliva.[10] Consistent with these observations, the presence of PAF in gingival tissues and crevicular fluid has been associated with clinical signs of periodontal inflammation.[11,12,13]

Salivary PAF levels in periodontitis patients have been correlated with the extent of periodontal disease.[8] Similarly, the levels of PAF in saliva from patients with refractory periodontitis were elevated in comparison to patients who had responded to conventional periodontal therapy and maintenance.[14] Thus a number of separate studies provide the basis for suggestion that PAF a proinflammatory phospholipid autacoid may be involved in periodontal tissue injury and disease.

The crosssectional studies outlined above indicate that the levels of PAF is saliva are correlated with the extent of periodontal disease. However, longitudinal studies to assess the effect of periodontal treatment on salivary PAF periodontitis patients have be insufficient. The purpose of this study thus to evaluate salivary PAF leve throughout initial peiodontal treatme in patients with early onset and adu periodontitis in relation to clinic parameters of the diseases.

MATERIALS AND METHODS

Human subjects:

The subjects of this study were divided into two groups, as follows:

Group I (subdivided into):

Study Group I: Ten early onset periodontitis patients with radiographic evidence of alveolar bone loss, of ages ranging from 19 to 29 years.

Control Group I: Ten healthy control subjects matching their study group in age and sex, enjoying clinically healthy gingiva and no radiographic evidence of bone loss.

Group II (subdivided into):

Study Group II: Ten chronic adult periodontitis patients diagnosed through clinical and radiographic examinations of ages ranging from 35 to 50 years.

Control Group II: Ten healthy control subjects with clinically healthy gingiva and no radiographic evidence of bone loss, matching their study group in age and sex.

The medical and dental history of each subject were reviewed to exclude those suffering from systemic illness. Patients having been subjected to antibiotics or