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The effects of non-surgical periodontal therapy on oxidant and anti-oxidant status in smokers with chronic periodontitis

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ABSTRACT

Aim: The aim of this study was to determine the effect of non-surgical periodontal treatment on gingival crevicular fluid (GCF) and serum oxidant–antioxidant levels in smoking and non-smoking patients with chronic periodontitis.

Methods: Twenty-nine patients with chronic periodontitis (15 smokers (CP-S) and 14 non-smokers (CP-NS)) and 20 periodontally healthy subjects (10 smokers (H-S) and 10 non-smokers (H-NS)) totalling 49 subjects were included in this study. GCF was collected from at least two pre-selected sites (one moderate and one deep pocket) in patients with CP. In the healthy group, GCF samples were collected from one site. Probing pocket depth, clinical attachment level (CAL), gingival and plaque indices, and bleeding on probing were measured. To determine serum total oxidant status (TOS) and total antioxidant status (TAS), venous blood was drawn from each subject. The GCF, serum sampling, and clinical measurements were recorded at baseline and 6 weeks after periodontal treatment.

Results: The study showed statistically significant improvement of clinical parameters after periodontal treatment in both smokers and non-smokers. In the CP-S group, there were no significant differences in GCF TAS levels at both moderate and deep pocket sites between baseline and 6 weeks ($p > 0.05$). GCF TAS levels in the CP-NS groups were significantly increased ($p < 0.05$) at moderate and deep pocket sites between baseline and 6 weeks. GCF TOS levels in the CP-S groups were significantly decreased ($p < 0.05$) at deep pocket sites between baseline and 6 weeks. There was no significant difference in serum TAS levels of the all periodontitis patient groups between at baseline and 6 weeks ($p > 0.05$). Serum TOS levels in the CP-S and CP-NS groups were significantly decreased ($p < 0.05$) after periodontal treatments.

Conclusions: The periodontal treatment improves the clinical parameters in both smokers and non-smokers. These results confirm that non-surgical periodontal therapy can reduce oxidative stress.

1. Introduction

Periodontal diseases result from the complex interaction between pathogenic bacteria and the host's immuno-inflammatory responses. It is believed that while the primary etiological agent is specific, predominantly gram-negative anaerobic or facultative bacteria within the subgingival biofilm,[1] the majority of periodontal tissue destruction is caused by an inappropriate host response to those microorganisms and their products.[1,2]

It is widely accepted that the host response to subgingival bacteria plays a critical role in periodontal pathogenesis[3] and that pathogenic processes are modified by environmental and

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acquired risk factors such as smoking.[4] For example, smokers demonstrate 2.6–6 times increased prevalence of periodontal diseases compared to non-smokers[5] and a reduced response to periodontal treatment.[6,7]

Chronic periodontitis (CP) is initiated by the sub-gingival biofilm,[8] but the progression of destructive disease appears to depend upon an abnormal host response to those organisms.[3,9,10] Over the past few years, strong evidence has emerged to implicate oxidative stress in the pathogenesis of periodontitis.[10,11] Free radicals and reactive oxygen species (ROS) are essential to many normal biologic processes. Low levels of certain free radicals and ROS can stimulate the growth of fibroblasts and epithelial cells in culture, whereas higher levels may result in tissue injury.[12] The deleterious effects of increased oxidative stress are termed oxidative damage; generally, they appear after exposure to a relatively high concentration of ROS and/or a decrease in antioxidant defense system against ROS.

Normally, there is a balance between ROS and antioxidants that may be disturbed by a variety of factors, including smoking. Smoking increases ROS production and is a significant source of oxidative stress.[13,14] It depletes systemic endogenous antioxidant capacity, resulting in increased pro-oxidant burden.[15]

Periodontal disease is associated with reduced total antioxidant status (TAS) and increased oxidative damage within the oral cavity.[14,16,17] Recently, it was demonstrated that smoking increases the levels of free radicals in periodontal tissues.[18] Palmer et al.[19] reported that cigarette smoke contains a large amount of oxidative species, and therefore smoking represents a significant source of oxidative stress. In addition, decreased antioxidant levels in blood, gingival tissue, saliva, and GCF have been shown in periodontitis and gingivitis patients who smoke.[15,18,20]

The reduction of oxidative stress and increase in antioxidant capacity after the non-surgical periodontal therapy were reported in literature.[9,10,21] Chapple[22] indicated that periodontitis could be associated with reduced local antioxidant defense and suggested that systemic and local TAS levels in CP might reflect increased oxygen radical activity during periodontal inflammation and can be restored to control subject levels by successful non-surgical therapy. Furthermore, Kim et al.[23] found that the TAS in saliva decreased directly after SRP. With time, it increased slightly and was relatively unchanged compared to the baseline. It was assumed that nonsurgical therapy did not improve the TAS in severe chronic periodontitis patients with non-smoking. D'Aiuto et al.[24] demonstrated that acute increases in reactive oxygen metabolites in serum and systemic inflammation occurred following periodontal therapy in smokers. In contrast, Guentsch et al. suggested that non-surgical periodontal treatment leads to a reduction of malondialdehyde and glutathione peroxidase to levels comparable to healthy controls.

As yet, the relationship between smoking and GCF serum oxidant–antioxidant status in periodontitis has not been clarified. Possible alterations in GCF and serum antioxidant composition may influence clinical periodontal status as well as the response to non-surgical periodontal treatment in smokers. Thus, the aim of this study was to determine the effect of non-surgical periodontal treatment on GCF and serum antioxidant levels in smoking and non-smoking patients with chronic periodontitis.

2. Material and methods

2.1. Study population

Twenty-nine otherwise healthy chronic periodontitis patients (15 smokers (CP-S), 14 non-smokers (CP-NS)), and 20 systemically and periodontally healthy volunteer subjects (10 smokers (H-S), 10 non-smokers (H-NS)), were selected at Cumhuriyet University Faculty of Dentistry, Department of Periodontology for periodontal problems. Written informed consent was obtained from all subjects and the study protocol was approved by the Medical Ethics Committee of Cumhuriyet University.

All subjects were systemically healthy. Subjects were excluded from the study if they had a taken course of non-steroidal, anti-inflammatory drugs or antimicrobial drugs within a 3 month period before the study began; were pregnant or lactating; had used mouthwashes or vitamin supplements within the previous 3 months; had a history of current drug use; or had special dietary requirements.

The selection of patients was made according to the criteria approved by the 1999 International Workshop for the Classification of Periodontal Diseases and Conditions.[25] Twenty-nine subjects with generalized chronic periodontitis characterized by at least 30% teeth with pockets >5 mm were recruited from new patients of the department. X-rays were taken from all subjects. Cigarette consumption was determined by verbal questioning. Smokers were enrolled if they regularly smoked ≥ 20 cigarettes/day, and non-smokers were characterized as not having smoked cigarettes in their lifetime. Healthy control groups had no attachment loss with the teeth having periodontal pockets ≤ 3 mm and no bleeding on probing and they had no radiographic evidence of alveolar bone loss. These individuals were systemically and periodontally healthy volunteers.

2.2. Clinical measurements and non-surgical periodontal therapy

Prior to crevicular fluid collection, plaque index (PI),[26] gingival index (GI),[26] probing pocket depth (PD), clinical attachment level (CAL), and presence of bleeding on probing (BOP) were measured. Based on the initial probing PD measurements, the study sites were further classified and at least one moderate (4–6 mm) and at least one deep (>6 mm) pockets of teeth were selected per subject.[27,28] PD and CAL measures were obtained using a Williams' periodontal probe. All clinical periodontal measurements were performed by the same examiner (A.A.). In all subjects, individual acrylic stents were fabricated with grooves as reference points for the clinical measurements. After recording the baseline measurements, non-surgical therapy, which consists of oral hygiene instructions, scaling, and root planning (SRP), was performed on subjects with periodontitis.[29] The SRP procedure was performed quadrant per quadrant under local anaesthesia in four visits using