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Research Article

EFFECT OF STANDARD PERIODONTAL THERAPY ON WHITE BLOOD CELL COUNT IN GENERALIZED AGGRESSIVE PERIODONTITIS

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A B S T R A C T

Aim: The aim of the present study was to investigate the effect of standard periodontal therapy (scaling and root planing) on white blood cell count and the differential blood count in patients with generalized aggressive periodontitis (GAP). **Materials and Methods:** 24 adult periodontitis patients with previously untreated GAP were subjected to 2 sessions of oral hygiene procedure. Afterwards, the patients were treated by scaling and root planing under local anaesthesia. Periodontal examinations were performed after supragingival pretreatment and three months after subgingival therapy. Pocket probing depth (PPD) and bleeding on probing were measured with UNC 15 probe. Accompanying clinical evaluation venous blood samples were taken to analyse the WBC counts and differential blood counts. For statistical analysis non-parametric tests were utilized. **Results:** PPD and bleeding on probing (BoP) improved significantly after therapy. Following periodontal treatment WBC counts, neutrophil counts decreased significantly. **Conclusion:** The results indicate that a therapeutical intervention may have a systemic effect on the blood count in GAP patients.

INTRODUCTION

Due local inflammatory and infectious nature of periodontitis (Socransky 1977, Theilade 1986),[1, 2] many studies have focused on the interaction between the pathogenic bacteria and the host response (Socransky & Haffajee 1992).[3] Bacteria with varying pathogenicity have been identified (Slots & Genco 1984, Socransky *et al*. 1998)[3,4] and correlated with various forms of periodontitis (Haffajee *et al*. 1998, Moore *et al*. 1991, Slots & Listgarten 1988). [5,6,7] It has been shown that periodontal bacteria or their products can directly invade the periodontal tissue (Allenspach Petrzilka & Guggenheim 1983, Meyer *et al*. 1991)[8,9] and gain access to the systemic circulation. Transient bacteraemia can be provoked by a variety of oral manipulations. The extent of bacteraemia from oral origin appears to be directly related to the severity of gingival inflammation (Allenspach Petrzilka & Guggenheim 1983, Silver *et al*. 1979).[8,10]

Periodontal disease has been associated with atherosclerosis (Beck *et al*. 1996, DeStefano *et al*. 1993)[11,12] and several direct and indirect pathogenetic mechanisms explain the link between infectious disease and atherosclerosis (Danesh *et al*. 1997),[13] among them a systemic inflammatory response that may cause elevated levels of established risk factors for atherosclerosis. The white blood cell count (WBC) has been associated with atherosclerosis in a number of epidemiological studies and is considered to be a risk factor for the disease (Ensrud & Grimm 1992).[14] Few studies have analysed hematological variables in periodontitis patients and the results were contradictory (Frederiksson 1999, Fredriksson *et al*. 1998, Loos *et al*. 2000). [15,16,17] Intervention study by christgau *et al*. 1998 failed to show an effect of periodontal therapy on WBC.[18] However, most of these studies dealt with patients with chronic periodontitis of moderate severity. Interventional study conducted by christan *et al* (2002)[19] in generalized aggressive periodontitis patients reported that following periodontal treatment WBC counts, neutrophil and platelet counts decreased significantly in non-smokers, while in smokers only platelet counts were significantly reduced.

The aim of the present study was to investigate the effect of non-surgical periodontal therapy on white blood cell count and the differential blood count in patients with generalized aggressive periodontitis (GAP).

MATERIAL AND METHODS

Patient Group

A convenient sample of 24 adult patients with previously untreated GAP were recruited for the study. Screening examination included a medical history, in particular smoking habits, radiographs and full-mouth pocket depth measurements to confirm the diagnosis of GAP. Inclusion criteria: age

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between 18–40 years, minimum of 20 teeth, pocket depth >6 mm on at least 3 teeth/quadrant, radiographical evidence of horizontal and vertical bone loss. Exclusion criteria: systemic antibiotic use during the study or preceding 6 months, systemic disorders, smoking, pregnancy and lactation. All patients partaking in this study signed the consent form.

Treatment

All patients were treated and evaluated by one experienced periodontics. The clinical study protocol consisted of 2 visits of full-mouth supragingival professional tooth cleaning (scaling and polishing) and oral hygiene instructions within a 2 week period. Oral hygiene was asssessed using the plaque index (PI) by sillness and loe and to assess gingival inflammation, pocket probing depth (PPD), bleeding on probing were recorded with the UNC 15 probe. After the oral hygiene phase baseline measurements including the described oral hygiene index, PPD and bleeding on probing were recorded.

The subsequent non-surgical treatment consisted of subgingival debridement using ultrasonic instruments and Gracey curettes under local anaesthesia in 4 sessions within 2 weeks. 3 months after the completion of the subgingival scaling the post-treatment measurements were performed.

Blood Sampling

Prior to any dental manipulation at baseline and at 3 months after completion of subgingival scaling, samples of 3 ml venous blood were collected in test tubes containing EDTA. The laboratory analysis of WBC count and differential blood count was done by Sysmex XT-2000i hematological autoanalyser, which uses Fluorescence flow cytometry to measure WBCS.[20]

Statistical Analysis

Non-parametric methods were utilized for statistical analysis. P-values are given for the comparison between baseline and after therapy values (Wilcoxon test)

RESULTS

All 24 patients, 11 females and 13 males, with a median age of 35 years completed the study.

Clinical Treatment Outcome

The differences of the clinical periodontal measurements between pre and post-treatment values are shown in Table 1. There was a marked reduction in sites that bled on probing. The probing depth reduction was also significant.

Table 1 Intragroup comparision of clinical parameters at baseline and at 3 months

Clinical parameters		Mean ± SD	P - value
Plaque index	Baseline	2.13 ± .23	<.001
	3 months	1.42 ± .18	
Bleeding on probing	Baseline	1.02 ± .20	<.001
	3 months	0.46 ± .22	
Pocket depth	Baseline	5.13± .49	<.001
	3 months	3.34 ± .52	

Blood Samples

The results of blood sample analysis are presented in Table 2. WBC count and differential blood count were within the normal range for all patients. WBC and neutrophil count decreased from baseline to 3 months after therapy, for all other haematological variables no significant differences between baseline and post-treatment values were found.

Table 2 Total number of white blood cell count and neutrophil count in $10^3/\mu$l at baseline and 3 months

		Mean	P value
WBCS	Baseline	7.1 (6.2–8.4)	< .01
	3 months	5.4 (4.5–6.9)	
Neutrophils	Baseline	4.10 (3.42–5.21)	<.01
	3 months	3.11 (2.47–4.70)	

DISCUSSION

As infections lead to increased leukocyte count, therefore increased leukocyte counts due to an inflammatory response have been discussed as one factor linking infections to cardiovascular disease (Danesh *et al*. 1997).[13] Several epidemiological studies have demonstrated leukocyte count to be independent predictor of future coronary heart disease (Grimm *et al*. 1985, Zalokar *et al*.1981).

Only few studies have measured leukocyte counts in periodontitis patients. Gustafsson & Asman (1996) found no significant differences between 14 periodontitis patients and 14 healthy controls. In contrast Loos *et al*. (2000) found significantly higher leukocyte counts in 54 patients with generalized periodontitis compared to 53 patients with localized periodontitis and 43 healthy controls.[17] Fredriksson *et al*. (1998) reported significantly higher leukocyte counts in 17 treated periodontitis patients compared to 17 age and sex matched healthy controls.[16] In another study by the same group, significantly higher leukocyte counts are reported in non-smoking periodontitis patients compared to non-smoking healthy controls (Frederiksson 1999).[15] The difference between smoking periodontitis patients and smoking controls was not statistically significant. An intervention study was published by Christgau *et al*. (1998) who studied the effect of periodontal therapy in 20 diabetics and 20 non-diabetic controls with periodontal disease. They also reported leukocyte counts in the course of periodontal therapy. Although the differences between pre- and post treatment values were not significant, leukocyte counts decreased in both groups after non-surgical periodontal therapy.[18] Another interventional study conducted by christan *et al* (2002) reported that following periodontal treatment WBC counts, neutrophil and platelet counts decreased significantly in non-smokers (p®0.004), while in smokers only platelet counts were significantly reduced (p½0.006). Non-smokers showed a significantly higher reduction of WBC counts (p½0.005) and neutrophils (p½0.001) compared to smokers.[19]

The present study investigated the effect of non-surgical periodontal therapy in young adult patients with aggressive periodontitis. Periodontal therapy decreased the leukocyte count in the study population. The results are consistent with the results of Frederiksson (1999) and christan *et al* (2002).

CONCLUSION

The results of the present study indicate that periodontal therapy may lead to a significant reduction of leukocyte counts, thus periodontal therapy has systemic effects and has the potential to ameliorate an established cardiovascular risk factor.

Conflicts of Interest: None
Source of Support: Nil

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