Evaluation of the Effects of Periodontal Treatment on Levels of Ascorbic Acid in Smokers

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Abstract

Smokers consistently have lower levels of vitamin C, which is important for optimal healing, especially following invasive procedures. Some studies demonstrated that patients undergoing surgery experience significant reductions in systemic vitamin C levels, presumably due to higher metabolic utilization of existing vitamin pools. However, there appear to be no studies evaluating the effect of non-surgical periodontal therapy on plasma levels of vitamin C. The aim of this study was to evaluate if non-surgical periodontal therapy is able to reduce the plasmatic level of ascorbic acid (AA) in smokers. Twenty-six systemically healthy adult (> 40 years) smokers (10 cigarettes/day for > 5 years) who needed scaling and root planing (SRP) for chronic periodontitis were recruited. The sessions of SRP (per quadrant) were scheduled 7 days apart from each other. Blood was collected by venipuncture before the first session of SRP and at the end of the periodontal treatment. The ascorbate concentrations in plasma were assessed according to a published protocol. A paired *t*-test ($p < 0.05$) evaluated the statistical significance of differences between the mean values obtained pre- and post-treatment. In general, there was no significant change in levels of AA; however, in 38% of patients, increased levels of AA in plasma were observed after SRP. In 15% of the patients, no change was noted, while 47% of patients showed a reduction in levels of AA after SRP. It can be concluded that although almost half of individuals presented with reduced levels of ascorbic acid after treatment, SRP did not significantly change the levels of AA in smokers.

Keywords: Smoking, vitamin C, scaling and root planing

Introduction

Tobacco is associated with numerous diseases and is responsible for high mortality rates throughout the world. Several epidemiological studies have demonstrated that smokers have a higher prevalence and severity of periodontitis when compared to non-smokers (Thornton-Evans *et al.*, 2013; Hugoson and Rolandsson, 2011; Natto, 2005). The components of tobacco smoke directly influence periodontal tissues (Bulmanski *et al.*, 2012; Colombo *et al.*, 2012) and nicotine is related to alveolar bone loss, periodontal attachment loss, periodontal pocket formation, tooth loss and poorer periodontal treatment outcomes (Buchwald *et al.*, 2013; Torrungruang *et al.*, 2012; Radvar *et al.*, 2011; Thomson *et al.*, 2007; Silva *et al.*, 2006; Silva *et al.*, 2007; Silva *et al.*, 2010).

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Several studies have indicated that the nutritional status of smokers can be compromised and that smoking is associated with a reduced consumption of antioxidants, especially ascorbic acid (AA) (Vardavas *et al.*, 2008; Dyer *et al.*, 2003). Smokers have reduced concentrations of AA in plasma (Jain *et al.*, 2009; Northrop-Clewes and Thurnham, 2007), presumably representative of reductions in body reserves due to exposure to a large number of oxidants (Church and Pryor, 1985; Morrow *et al.*, 1995). This AA reduction in plasma is so large that it can reach 75% depending on the age, sex and smoking history of the population. Moreover, smokers require a daily intake 40% higher than non-smokers to maintain comparable serum levels (Kallner *et al.*, 1981).

Patients who underwent surgery usually experienced a significant reduction in systemic levels of AA (Vallance, 1988; Ballmer *et al.*, 1994; Dingchao *et al.*, 1994; Schorah *et al.*, 1996; Erhola *et al.*, 1998). Moreover, patients with periodontal disease present with lower levels of AA than healthy subjects (Kuzmanova *et al.*,

2012; Iwasaki *et al.*, 2012). However, it is not clear if less traumatic procedures, such as scaling and root planing (SRP), are able to reduce plasma levels of AA.

Ascorbic acid is essential for humans and is important for wound healing, acting as an antioxidant and nourishing the cells, protecting them from damage caused by free radicals and oxidants (Northrop-Clewes and Thurnham, 2007). Despite the importance of this relationship between smoking, periodontal disease and AA, there appear to be no studies about the systemic change in serum levels of AA after non-surgical periodontal therapy in smokers.

Therefore, this study aimed to evaluate if SRP can reduce the plasma levels of AA in smokers, a population that clearly presents with lower levels of this substance.

Materials and methods

This is a prospective single group study that evaluated the effects of non-surgical periodontal treatment on systemic levels of AA in smokers. Participants in the study signed a consent form authorizing their participation. The study protocol and consent form were approved by the Ethics Committee on Human Research of the State University of Maringá. All clinical procedures were performed in the Dental Clinic of the Department of Dentistry, State University of Maringá, Maringá, Paraná.

Twenty-six systemically healthy adults (> 40 years) smokers (10 cigarettes/day for > 5 years) who needed scaling and root planing (SRP) for the treatment of moderate to severe chronic periodontitis were recruited for the study. Participants were recruited through flyers, announcements and messages posted in the University area and also among those seeking treatment at the Clinic of Dentistry of the State University of Maringá.

Inclusion criteria were a plaque index > 20%, at least 30% of teeth with ≥ 1 site with probing depth ≥ 5 mm and attachment loss ≥ 3 mm at the same site; no mechanical periodontal treatment in the last six months, no oral mucosa lesions, no oral infections, and no acute periodontal disease. Exclusion criteria were pregnancy or lactation, chronic systemic conditions associated with periodontitis, or treatment with antibiotics or vitamin C supplements.

The following parameters were evaluated at baseline for diagnosis and 3 months after treatment: plaque index (PI); bleeding on probing (BoP); probing depth (PD), gingival recession (GR); clinical attachment level (CAL). After clinical examination, diagnosis and treatment planning, smokers with moderate to severe chronic periodontitis were recruited for the study. The sessions of SRP (per quadrant) were scheduled with an interval of 7 days. All patients needed 4 weeks to complete treatment.

Blood was collected by venipuncture before the first session of SRP and at the end of non-surgical periodontal treatment (28 days after baseline). Ascorbic acid concentrations in plasma were evaluated at the Biochemistry Laboratory of the State University of Maringá with high-performance liquid chromatography (HPLC) with electrochemical detection (Leggott *et al.*, 1986; Leggott *et al.*, 1991). Blood was not collected after fasting and no dietary survey was conducted to evaluate vitamin C intake.

The paired *t*-test was used to perform statistical analysis of the results. The outcomes are presented as mean and standard deviation. The significance level was set at 5%.

Results

In the present study the levels of AA were evaluated in 26 smokers (12 women and 14 men) with periodontal disease and an average age of 57.4 ± 6.7 years. Demographic data are shown in *Table 1*.

All measurements of clinical parameters evaluated at baseline and 3 months after periodontal treatment are presented in *Table 2*. All patients showed a positive response to periodontal treatment with an improvement in periodontal parameters.

Figure 1 demonstrates AA levels at baseline and after periodontal treatment in subjects included in this study. The average level of AA in individuals before SRP was 0.07 ± 0.05 μg/mL, and after SRP it was 0.06 ± 0.04 μg/mL. These levels were not statistically significantly different ($p = 0.19$).

In 38% of individuals, a significant increase ($p = 0.02$) of AA levels in plasma was observed after treatment with SRP (from 0.05 ± 0.03 μg/mL to 0.09 ± 0.05 μg/mL). In 15% of individuals, AA was at the same level before and after treatment (0.05 ± 0.04 μg/mL). On the other hand, 47% of patients had a significant reduction ($p = 0.02$) of AA levels (0.09 ± 0.06 μg/mL to 0.05 ± 0.03 μg/mL) after treatment. At the baseline evaluation, the patients in these three groups showed no significant difference in AA levels ($p = 0.19$).

Discussion

In this study, the effect of SRP on plasma levels of AA in smokers was evaluated. The outcomes showed that, in general, SRP was not significantly able to decrease the levels of AA in smokers. At the end of the study, about half of the subjects presented with increased or stable levels of AA. This result could be due to the nutritional aspect of subjects studied (which was not controlled), the extent of trauma and the methodology applied.

Some studies have shown that surgical procedures can produce a significant reduction in systemic levels of AA (Vallance, 1988; Ballmer *et al.*, 1994; Dingchao *et al.*, 1994; Schorah *et al.*, 1996; Erhola *et al.*, 1998) as part of oxidative stress, an imbalance of reduced antioxidants (vitamins) and increased pro-oxidants (radicals) (Biesalski, 1997). However, in these patients, perhaps

Table 1. Demographic data of patients included in the study.

	Increased levels of ascorbic acid	Decreased levels of ascorbic acid	Same levels of ascorbic acid before and after scaling and root planing	Total
Age (years)	55.3	59.7	56.8	57.4
Male (n)	5	5	2	12
Female (n)	5	7	2	14

Table 2. Clinical parameters (mean ± SD) at baseline and 3 months after non-surgical periodontal treatment.

	PI	BoP	CAL	GR	PD
Baseline	83.42 ± 9.0	36.36 ± 20.70	4.01 ± 1.13	0.71 ± 0.64	3.30 ± 0.65
3 months	28.33 ± 7.21	13.68 ± 9.89	3.16 ± 1.01	0.93 ± 0.77	2.23 ± 0.49

PI, plaque index; BoP, bleeding on probing; CAL, clinical attachment level; GR, gingival recession; PD, probing depth

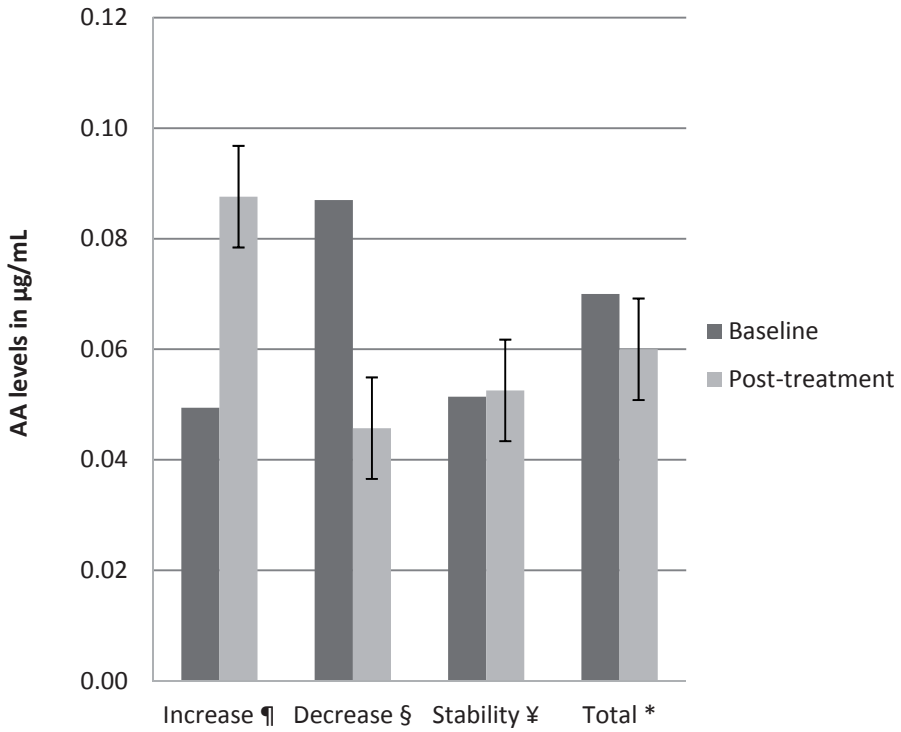

Figure 1. Ascorbic acid (AA) levels in smokers at baseline and after treatment with scaling and root planing (SRP). Paired t-tests were used to determine significant differences in the means; n = 26 individuals. ¶ - p = 0.02; § - p = 0.02; ¥ - Not significant (NS; p > 0.05); * - NS (p = 0.19)

the effect of trauma caused by the SRP (Alves *et al.*, 2005) in the first three weeks was minimal and could not be measured anymore at the end of the fourth week. A recent review (Sculean *et al.*, 2014) showed that between 7-14 days after SRP, long junctional epithelium is already formed and, although complete healing could take longer, the initial phases are over and the metabolic needs are lower. Another recent study in non-smoking patients demonstrated that SRP is able to lower the inflammatory burden and improve systemic oxidant/antioxidant imbalance, increasing vitamin C levels (Aziz *et al.*, 2013a). Moreover, the degree of trauma caused in the last session of SRP was not great enough to cause AA reduction.

Possibly, if the periodontal treatment were done all in one week or as a full-mouth debridement and the AA levels evaluated immediately afterward, the degree of trauma inflicted by the SRP (Alves *et al.*, 2005) could have significantly reduced AA levels. A study by Vandekerckhove *et al.* (1996) demonstrated higher rises in body temperature and more cases of herpes labialis in patients treated with full-mouth debridement than in those treated with traditional SRP because of greater bacteremia and a stronger immunological response. These facts demonstrate that full-mouth debridement is more traumatic to periodontal tissues than traditional SRP.

Smokers (Jain *et al.*, 2009; Northrop-Clewes and Thurnham, 2007) and patients with periodontal disease (Kuzmanova *et al.*, 2012; Iwasaki *et al.*, 2012) present with reduced levels of plasma AA compared to non-smokers. Smokers present with concentrations of AA in plasma that are reduced up to 75% compared to non-smokers, depending on age, sex and smoking history of the population (Jain *et al.*, 2009; Northrop-Clewes and Thurnham, 2007). As the patients of this study are smokers with periodontal disease, it is fair to infer that they probably have even lower levels of AA (Aziz *et al.*, 2013b). Therefore, it can be speculated that the decrease in plasma AA would not be significant in these patients due to the already reduced levels.

On the other hand, it was observed that almost half of individuals showed a reduced level of AA in plasma after treatment. In these individuals, the trauma caused by SRP promoted a reduction of 0.04 μg/mL, almost 50% of the initial levels.

It can be conjectured that SRP promotes changes and trauma in the periodontal ligament and other supporting tissues that causes a reduction of AA. Possibly, this reduction is related to the use of AA by fibroblasts to repair the periodontal ligament. Ascorbic acid plays an important role in human physiology, including a role in the formation of collagen fibers (Yan *et al.*, 2013; Li and Schellhorn, 2007; Clark *et al.*, 2002), because it is a cofactor for two enzymes, the lysyl and prolyl hydroxylase, which act on the stage of hydroxylation of collagen. Thus, the function of AA as a cofactor is to prevent oxidation of the iron present in the chemical structure of these enzymes and consequently protect them against self-inactivation (Parsons *et al.*, 2006; Senturk *et al.*, 2004; Tsuchiya and Bates, 1998). Ascorbic acid also stimulates collagen synthesis; it acts by increasing the gene expression of fibroblasts (Yan *et al.*, 2013; Senturk *et al.*, 2004).

According to Palmer *et al.* (2005), nicotine negatively affects proliferation of gingival fibroblasts, collagenase and fibronectin production, and destruction of type I collagen. Malhotra *et al.* (2010) showed that nicotine has an adverse effect on microcirculation and blood flow, inducing chronic vasoconstriction in periodontal tissues, and interfering with the periodontal healing process by inhibition of the growth of gingival fibroblasts and production of fibronectin and collagen, and promotion of collagen destruction.

Another variable that could interfere with the final outcomes is the difference in nutrition status among patients. Unlike most animals, humans are unable to synthesize AA and must rely on dietary intake to meet their physiological needs. Because it is water soluble, AA is not maintained well in the body and requires a continuous intake to maintain adequate body stores (Northrop-Clewes and Thurnham, 2007). As a nutrition survey was not done, some patients could have had a higher intake of vitamin C-containing food during treatment, which could interfere with the final outcome.

Thus, it can be concluded that non-surgical periodontal treatment did not cause significant changes in plasma levels of AA in smoking patients. However, it was observed that almost half of smokers showed a significant decrease in the levels of AA. So, further studies are needed to confirm the effects of periodontal treatment on the levels of AA in patients with periodontal disease.

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