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RESEARCH ARTICLE

Effects of smoking on local and systemic oxidative stress markers in individuals with periodontitis

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ABSTRACT

Aim: This study aimed to assess the effects of smoking on systemic and local oxidative stress markers in patients with periodontitis.

Methods: A total of 72 patients with periodontitis [38 smokers (S +P+), 34 non-smokers (S-P+)] and 54 periodontally healthy individuals [28 smokers (S+P-), 26 non-smokers (S-P-, control)] were included. After clinical measurements and samplings, the cotinine level, total antioxidant capacity (TAOC), total oxidative status (TOS), and malondialdehyde (MDA) level in the serum and saliva were determined, and the oxidative stress index (OSI) was calculated. Kruskal-Wallis and Mann-Whitney U tests were used for multiple and pairwise comparisons. Correlations were analyzed using Pearson correlation coefficient. $P<0.05$ was considered statistically significant.

Results: Smoking and periodontitis decreased the serum and salivary TAOCs and increased the TOS, MDA level, and OSI. The smokers with periodontitis had the lowest TAOC and the highest TOS, MDA level, and OSI, while the controls had the highest TAOC and the lowest TOS, MDA level, and OSI. The systemic and local effects of smoking seemed more pronounced than those of periodontitis in the oxidative stress study, but no significant difference was identified between the smoking (S+P-) and periodontitis (S-P+) groups. The clinical parameters and oxidative stress markers showed both substantial positive and negative relationships in all groups ($p<0.01$).

Conclusions: It can be concluded that smoking and periodontitis (S+P+) are associated with a decrease in serum and salivary TAOCs and an increase in TOS, MDA levels, and OSI. Smoking has a similar effect as periodontitis on local and systemic oxidative stress, and oxidative stress caused by smoking may be a significant factor in the pathophysiology of periodontitis.

Keywords: Smoking, lipid peroxidation, oxidative stress, periodontitis

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INTRODUCTION

Reactive oxygen species (ROS) are extremely reactive derivatives of oxygen metabolism and can cause tissue destruction through various mechanisms such as DNA damage, lipid peroxidation (LPO), protein damage, oxidation of crucial enzymes, and stimulation of pro-inflammatory cytokine release. The etiology of numerous inflammatory disorders substantially involves ROS, including periodontitis (1,2). In all living organisms, protective antioxidant defense systems have evolved to counteract the damaging effects of ROS (3). In a healthy physiological state, antioxidant defense capability and ROS activity are dynamically balanced. However, oxidative stress may occur, leading to the potential destruction of vital cell components due to diminished antioxidant defense and/or changes in ROS generation or activity (1-4). Although it is not precisely known whether oxidative stress is the cause or result of related diseases, identifying oxidative stress is considered important in clarifying the pathogenic mechanisms of various illnesses (5).

Periodontitis is an inflammatory condition of the supporting tissues of the teeth (6). ROS, LPO products, and antioxidant systems play a crucial role in the pathogenesis of periodontitis (7). According to reports, both oxidative damage and low antioxidant capacity are linked to periodontal disease (8,9). Additionally, local and systemic LPO levels are increased in individuals with periodontitis (9,10).

Smoking is known to be the strongest modifiable risk factor following bacterial plaque accumulation for the incidence and progression of periodontitis (11,12). Smoking can damage periodontal tissues by affecting neutrophil function and cytokine and growth factor production, inhibiting antibody production and fibroblast activity, and decreasing collagen production and vascularity (13).

The high ROS content of cigarettes negatively affects the antioxidant defense mechanism, consequently increasing oxidative stress (11,13). Smoking has also been reported to stimulate the production of ROS from neutrophils (14-16). The presence of the main nicotine metabolite in physiological fluids, cotinine, is a clear sign of current smoking or exposure to cigarette smoke (17). In many studies investigating the relationship between smoking and diseases, cotinine is defined as a chemical determinant of nicotine intake (18).

In the determination of oxidative stress, parameters such as enzymatic and non-enzymatic antioxidant levels, total antioxidant capacity (TAOC), total oxidative status (TOS), LPO product [e.g., malondialdehyde (MDA)] level, and oxidative stress index (OSI) are commonly used (19-21). It has been reported that TAOC decreased while TOS, MDA level, and OSI increased in patients with periodontitis (3,9).

Although smoking has been shown to have detrimental effects on periodontal tissues in numerous studies (12,16), the role played by oxidative stress in this association is still not entirely understood. Accordingly, this study aimed to evaluate the effects of smoking on systemic and local oxidative stress parameters in individuals with periodontitis.

MATERIALS AND METHODS

This study was reviewed and approved by the Ethical Committee of the Karadeniz Technical University Faculty of Medicine (approval number: 2009/44).

Sample size calculation

In the main hypotheses of the study, comparisons between four independent groups were planned. In this study, the sample size was calculated at a 95% confidence level using the G*Power program version 3.1.9.2 (22). The standardized effect size was measured as 0.6386 based on a similar study (CAL: 5.10±0.85 and 4.65±0.52) (23). The minimum sample size for each group was calculated as 20, with a theoretical power of 0.80. Owing to potential mishaps during biochemical analyses, the number of patients was increased by 30% of the calculated sample size, resulting in a minimum sample size of 26 for each group.

Clinical studies

Study groups

The study was conducted in accordance with the Declaration of Helsinki guidelines. A total of 126 individuals were included in this study: 72 patients with periodontitis [38 smokers (S+P+ group), including 17 women and 21 men, mean age: 36.75±8.93 years; 34 non-smokers (S-P+ group), including 16 women and 18 men, mean age: 35.87±8.65 years] and 54 periodontally healthy individuals [28 smokers (S+P- group), including 13 women and 15 men, mean age: 32.17±9.27 years; 26 non-smokers (S-P-/control group), including 12 women and 14 men, mean age: 30.53±8.25 years]. The participants were chosen from those referred to the Karadeniz Technical University Faculty of Dentistry, Department of Periodontology for periodontal issues or routine periodontal checks. The study details were explained to the participants, and written consent was acquired.

In accordance with the standards approved by the 2017 World Workshop, clinical and radiographic evaluations for periodontitis were conducted (24). Patients with stage 3 grade A–C periodontitis were included in the periodontitis groups. Both smoking and non-smoking patients with periodontitis were considered to have poor oral hygiene. There were no prosthetic restorations, and the accumulation of plaque was equivalent to the amount of attachment loss (AL). The control groups (smokers and non-smokers) consisted of periodontally healthy individuals with a PD of ≤3 mm and an AL of ≤1 mm, with adequate dental hygiene, no clinical signs of gingival inflammation, and no prosthetic restorations.

Patients who had no history of any systemic disease, had not received any drug therapy or any antioxidant vitamin therapy in the last 6 months, had not received any periodontal treatment in the last 1 year, were neither pregnant nor lactating, and shared the same dietary preferences and resided in the same geographical area (the Black Sea coastal region of Turkey) were included in the study. Conversely, patients who smoked fewer than 10 cigarettes a day and those who had smoked for less than 2 years were excluded from the study.

Clinical measurements

The PD and CAL (using Williams periodontal probe; 122-006, Hu-Friedy)), gingival index (GI), gingival bleeding index (GBI), and plaque index (PI) were measured in all participants. Radiographs of the entire mouth were taken to assess the extent of periodontal bone loss among the patients. The PD and CAL were assessed at six locations (mesio-buccal, medio-buccal, disto-buccal, mesio-palatal, medio-palatal, and disto-palatal areas) and the GI, GBI, and PI at four locations (mesial, mid-buccal, mid-palatal, and distal areas). The same examiner conducted each clinical and radiographic examination. The participants had a minimum of 20 teeth in total.

Sample collection

Samples were collected in the morning after an overnight fast 2 days after the clinical measurements. The participants were instructed to refrain from eating or drinking in the morning. Before the samples were collected, the participants were evaluated for protocol adherence.

Unstimulated whole saliva samples were collected from the participants. The participants were instructed to rest for 5 min without swallowing their saliva. Pooled saliva at the bottom of the mouth was drained into a collection tube. The saliva was centrifuged at 4,000×g for 10 min at 4°C before analysis. The supernatant fraction was aliquoted into storage vials and then stored in liquid nitrogen until analysis.

Venous blood was collected into plain tubes for serum analysis. Before centrifugation at 1,500×g for 10 min at room temperature, the tubes were maintained at 4°C for an additional 30 min.

Before analysis, the serum samples were aliquoted into cryogenic vials and kept in liquid nitrogen. Each participant served as an analytical unit.

Laboratory studies

TOS assay

The technique created by Erel was used to quantify the serum and salivary TOSs [20]. Hydrogen peroxide (H_2O_2) was used to calibrate the test, and the concentration values were expressed in micromolar H_2O_2 equivalents per liter.

TAOC assay

The serum and salivary TAOCs were measured using commercially available human-specific enzyme-linked immunosorbent assays in accordance with the manufacturer's recommendations (TAOC ELISA Kit, Rel Assay Diagnostics, Gaziantep, Turkey). The Trolox equivalent in millimolar was used to express the findings.

MDA (LPO product) assay

The serum and salivary MDA levels were determined by the high-performance liquid chromatography (HPLC) technique, which was slightly modified from Young and Trimble's method (21). The concentration values were expressed in micromolars.

OSI

The OSI was determined based on the TOS:TAOC ratio. The TAOC expressed in millimolar Trolox equivalents per liter was converted to micromole equivalents per liter. The OSI was calculated using the following formula: OSI=[(TOS in µmol/L)/(TAOC in mmol Trolox equivalent/L)*100].

Cotinine level

The salivary cotinine level was determined using HPLC, as described by Machacek and Jiang (25).

Statistical analyses

The data collected from all participants were imported to the Statistical Package for the Social Sciences (SPSS) for Windows version 16.0 (SPSS Inc., Chicago, IL, USA). Standard descriptive statistics such as means, standard deviations, medians, and frequencies were used to present the characteristics of the sample. The normality of the distribution of the data was examined using the Kolmogorov–Smirnov test. Because the distribution of the data did not meet the requirements for normality and homogeneity of variances, the nonparametric Kruskal–Wallis one-way analysis of variance by ranks and the Mann–Whitney U test were used for the multiple and pairwise comparisons, respectively. The correlations between at least two continuous variables were examined using Pearson's correlation coefficients. $P<0.05$ was considered statistically significant.

RESULTS

Clinical findings

The arithmetic mean values of the clinical parameters are shown in Table 1. All clinical periodontal parameters were significantly higher among the patients with periodontitis than among the periodontally healthy individuals ($p<0.01$). There was no significant difference in the clinical periodontal parameters between the S+P+ and S-P+ groups and between the S+P- and S-P- groups ($p>0.05$).

Laboratory findings (Table 2)

TAOC

The highest and lowest serum and salivary TAOCs were noted in the control and S+P+ groups, respectively (Figure 1).

Table 1. Comparison of clinical parameters in periodontitis groups and periodontally healthy groups

Clinical Parameters	Groups	N	X±SD			Median	χ^2	p
PD (mm)	S+P+	38	3.58	±	0.421*	3.57	103.039	0.001
	S-P+	34	3.52	±	0.607‡	3.42		
	S+P-	28	1.19	±	0.401	1.25		
	S-P-	26	1.2	±	0.395	1.23		
CAL (mm)	S+P+	38	4.17	±	0.739*	4.06	102.91	0.001
	S-P+	34	4.02	±	0.837‡	3.84		
	S+P-	28	1.51	±	0.557	1.55		
	S-P-	26	1.52	±	0.571	1.45		
GI	S+P+	38	1.5	±	0.495*	1.46	107.822	0.001
	S-P+	34	1.79	±	0.325‡	1.75		
	S+P-	28	0.06	±	0.089	0		
	S-P-	26	0.04	±	0.082	0		
GBI (%)	S+P+	38	81.69	±	18.421*	85.87	107.558	0.001
	S-P+	34	87.72	±	11.663‡	88.87		
	S+P-	28	0.03	±	0.06	0		
	S-P-	26	0.02	±	0.048	0		
PI	S+P+	38	2.24	±	0.538*	2.2	108.362	0.001
	S-P+	34	2.06	±	0.436‡	2.02		
	S+P-	28	0.03	±	0.079	0		
	S-P-	26	0.02	±	0.059	0		

Kruskal–Wallis test:

* The S+P+ group is statistically different from the S+P- and ve S-P- groups ($p<0.05$).

‡ The S-P+ group is statistically different from the S+P- and ve S-P- groups ($p<0.05$).

SD: Standart deviation, PD: Pocket Depth, CAL: Clinical Attachment Level, GI: Gingival Index, GBI: Gingival Bleeding Index, PI: Plaque Index

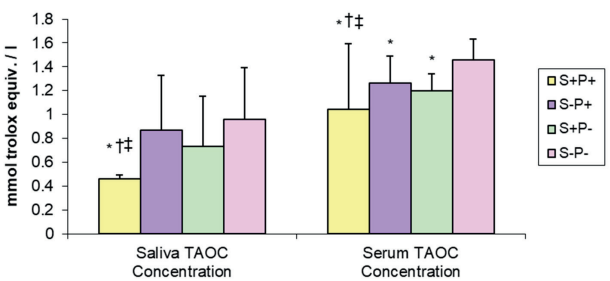

Figure 1. The comparison of the total antioxidant capacity (TAOC) concentrations in serum and saliva between the groups.

* Significant difference as compared with S-P- group ($p<0.05$).
† Significant difference as compared with S-P+ group ($p<0.05$).
‡ Significant difference as compared with S+P- group ($p<0.05$).

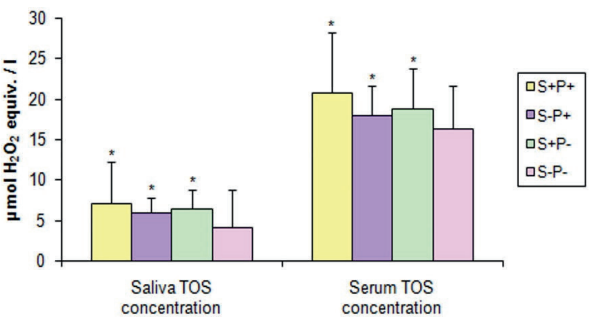

Figure 2. The comparison of the total oxidant status (TOS) concentrations in serum and saliva between the groups.

* Significant difference as compared with S-P- group ($p<0.05$).

Table 2. Comparison of serum and salivary laboratory parameters between periodontitis [smoker (S+P+), non-smoker (S-P+)] and periodontally healthy [smoker (S+P-), non-smoker (S-P-; control)] groups

Laboratory Parameters	Groups	N	X±SD			Median	χ^2	p
Serum TAOC (mmol Trolox Equiv./l)	S+P+	38	1.04	±	0.280*†‡	1.1	47.951	0.000
	S-P+	34	1.26	±	0.232*	1.24		
	S+P-	28	1.20	±	0.143*	1.21		
	S-P-	26	1.46	±	0.174	1.45		
Serum TOS (µmol H_2O_2 Equiv./l)	S+P+	38	20.73	±	7.522*	19.6	8.461	0.037
	S-P+	34	17.96	±	3.71*	18.17		
	S+P-	28	18.87	±	4.92*	19.71		
	S-P-	26	14.38	±	5.219	14.76		
Serum MDA (nmol/ml)	S+P+	38	0.50	±	0.550*	0.27	13.7	0.003
	S-P+	34	0.35	±	0.322	0.21		
	S+P-	28	0.47	±	0.558	0.27		
	S-P-	26	0.20	±	0.13	0.16		
Serum OSI	S+P+	38	2.25	±	1.351*†‡	1.8	32.256	0.000
	S-P+	34	1.47	±	0.492*	1.38		
	S+P-	28	1.59	±	0.421*	1.57		
	S-P-	26	1.04	±	0.385	1.03		
Salivary TAOC (mmol Trolox Equiv./l)	S+P+	38	0.46	±	0.292*†‡	0.39	27.105	0.000
	S-P+	34	0.87	±	0.457	0.86		
	S+P-	28	0.73	±	0.424	0.95		
	S-P-	26	0.96	±	0.433	1.05		
Salivary TOS (µmol H_2O_2 Equiv./l)	S+P+	38	7.14	±	5.137*	6.39	7.951	0.047
	S-P+	34	5.95	±	1.83*	6.14		
	S+P-	28	6.51	±	2.186*	6.39		
	S-P-	26	3.09	±	4.632	0.46		
Salivary MDA (nmol/ml)	S+P+	38	0.10	±	0.035*†‡	0.09	49.582	0.000
	S-P+	34	0.07	±	0.038*	0.06		
	S+P-	28	0.08	±	0.045*	0.07		
	S-P-	26	0.04	±	0.016	0.04		
Salivary OSI	S+P+	38	3.85	±	8.565*	1.72	32.106	0.000
	S-P+	34	1.82	±	3.891*	0.69		
	S+P-	28	2.57	±	4.508*	0.68		
	S-P-	26	0.47	±	0.562	0.23		

Kruskal–Wallis test:

* Statistical difference when compared with S-P- groups ($p<0.05$).

† Statistical difference when compared with S-P+ groups ($p<0.05$).

‡ Statistical difference when compared with S+P- groups ($p<0.05$).

SD: Standart deviation, TAOC: Total Antioxidant Capacity, TOS: Total Oxidant Status, MDA: Malondialdehyde, OSI: Oxidative Stress Index

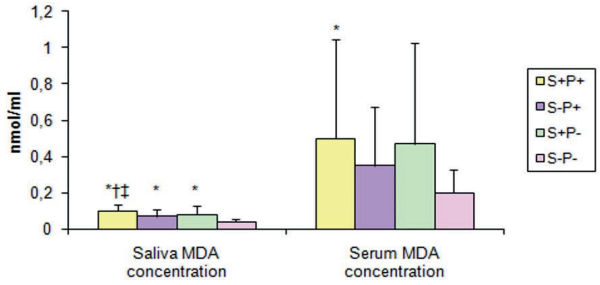

Figure 3. The comparison of the malondialdehyde (MDA) concentrations in serum and saliva between the groups.

* Significant difference as compared with S-P- group (p<0.05).
† Significant difference as compared with S-P+ group (p<0.05).
‡ Significant difference as compared with S+P- group (p<0.05).

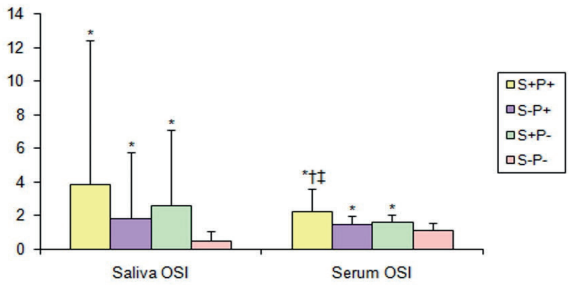

Figure 4. The comparison of the oxidative stress index (OSI) values in serum and saliva between the groups.

* Significant difference as compared with S-P- group (p<0.05).
† Significant difference as compared with S-P+ group (p<0.05).
‡ Significant difference as compared with S+P- group (p<0.05).

TOS

The serum and salivary TOSs were significantly lower in the smoking and/or periodontitis groups than in the control group (p=0.037, p=0.047). The highest and lowest TOSs were observed in the S+P+ and control groups, respectively (Figure 2).

LPO (MDA) level

While the serum MDA level was significantly lower in the S+P+ group than in the control group (p<0.01), no significant difference was found between the other groups (p>0.05). The salivary MDA level was significantly higher in the S+P+ group than in the other groups and the S-P+ and S+P- groups than in the control group (p<0.01). Although the S+P- group had higher serum and salivary MDA levels than the S-P+ group, there was no significant difference between them (p>0.05) (Figure 3). The highest and lowest serum and salivary MDA levels were noted in the S+P+ and control groups, respectively.

OSI

The serum OSI was significantly higher in the S+P+ group than in the other groups and the S-P+ and S+P- groups than in the control group (p<0.01). While the salivary OSI was significantly higher in the smoking and/or periodontitis groups than in the control group (p<0.01), no significant difference was found between the other groups (p>0.05). Further, although the S+P- group had higher serum and salivary OSIs than the S-P+ group, no significant difference was found between them (p>0.05) (Figure 4). The highest and lowest serum and salivary OSIs were observed in the S+P+ and control groups, respectively.

Cotinine level

The salivary cotinine level could only be measured in the S+P+ and S+P- groups. The salivary cotinine level was then significantly higher among the smoking groups than among the non-smoking groups. There was no significant difference in the salivary cotinine level between the S+P+ and S+P- groups (p=0.245) (Table 3).

Table 3. Comparison of saliva cotinine concentrations of (S+P+) and (S+P-) groups

Laboratory Parameters	Groups	N	X±SD			Median	χ^2	p
Saliva Cotinine (µg/l)	S+P+	38	1867.83		1066.54	2177.5	502	0.245
	S+P-	28	1645.53		1294.73	1232		

SD: Standart deviation.

Table 4. Correlations between clinical parameters, serum and saliva TOS, TAOC, MDA and OSI values, and saliva cotinine in all subjects (r, Pearson's correlation coefficient)

	r	p		r	p
PD-serum TAOC	-0.285	0.001	GI-saliva MDA	0.217	0.003
PD-serum OSI	0.246	0.003	GI-saliva cotinine	0.334	0.001
PD-saliva TAOC	-0.183	0.030	GBI-serum TAOC	-0.311	0.000
PD-saliva TOS	0.185	0.029	GBI-serum OSI	0.260	0.002
PD-saliva MDA	0.262	0.002	GBI-saliva TAOC	-0.167	0.049
PD-saliva cotinine	0.371	0.000	GBI-saliva MDA	0.277	0.001
CAL-serum TAOC	-0.256	0.002	GBI-saliva cotinine	0.374	0.000
CAL-serum OSI	0.225	0.007	PI-serum TAOC	-0.299	0.000
CAL-saliva TAOC	-0.177	0.036	PI-serum TOS	0.198	0.019
CAL-saliva MDA	0.250	0.003	PI-serum OSI	0.275	0.001
CAL-saliva cotinine	0.366	0.000	PI-saliva TAOC	-0.206	0.015
GI-serum TAOC	-0.255	0.002	PI-saliva TOS	0.195	0.021
GI-serum OSI	0.193	0.022	PI-saliva MDA	0.273	0.001
GI-saliva TOS	0.190	0.025	PI-saliva cotinine	0.386	0.000

PD: Pocket Depth, CAL: Clinical Attachment Level, GI: Gingival Index, GBI: Gingival Bleeding Index, PI: Plaque Index, TAOC: Total Antioxidant Capacity, TOS: Total Oxidant Status, MDA: Malondialdehyde, OSI: Oxidative Stress Index

Correlations

The correlations between the clinical parameters and oxidative stress parameters are shown in Table 4. Significant positive and negative correlations were detected between the clinical parameters and oxidative stress parameters in all groups.

DISCUSSION

The present study showed that smoking and periodontitis decreased the serum and salivary TAOCs and increased the TOS, MDA level, and OSI. In the literature, various studies have examined the oxidative and antioxidative status among smokers and non-smokers with periodontitis (10,11,13,26-28). To the best of the authors' knowledge, this study is the first to explore the effects of smoking on the TAOC, TOS, and MDA levels both locally and systemically in individuals with periodontitis. Further, the OSI was used to emphasize the relationship between smoking, periodontitis, and oxidative stress.

Herein, the systemic and local TAOCs were significantly reduced among the smokers with periodontitis compared with those among the periodontally healthy smokers and non-smokers and patients with periodontitis. Guentsch et al. (10) found that smokers with periodontitis had a lower TAOC, while Buduneli et al. (11) reported that gingival inflammation and/or smoking did not change the salivary TAOC in individuals with gingivitis. In addition, the serum, plasma, salivary, and gingival crevicular fluid (GCF) TAOCs were significantly lower among individuals with periodontitis than among healthy controls. Baltacıoğlu et al. (3) reported decreased serum and salivary TAOCs in patients with periodontitis. Other studies have revealed that smoking, independent of periodontitis, reduced plasma and serum antioxidant levels (29-32). These previous findings appear to be consistent with the TAOCs noted in the present study.

Currently, it is impractical to detect various oxidant molecules independently, as this approach may not fully reflect the interaction of oxidant molecules with each

other (20). Therefore, assessing the TOS is considered superior to other approaches. In the literature, few studies have evaluated the TOS in individuals with periodontitis (7,31,33). In these studies, the serum, salivary, and GCF TOSs were significantly higher in individuals with periodontitis than in healthy controls, and the TOS increased systemically and locally in individuals with periodontitis. In the present study, the serum and salivary TOSs increased significantly in the smoking and/or periodontitis groups compared with those in the control group, and the highest TOS was noted in the smokers with periodontitis. There was no significant difference between the other groups, except for the control group. These findings indicate that while smoking and/or periodontitis increase oxidative levels, smoking has an additional oxidative effect on periodontitis, although it is not significant.

LPO increases the quantity of end products, especially aldehydes. Therefore, evaluating these products is the current approach for the analysis of oxidative stress. This study also examined the level of MDA, an LPO product, to investigate the effects of smoking and periodontitis on oxidative stress. While the serum MDA level increased among the smokers with periodontitis compared to the controls, there was no significant difference between the other groups, except for the controls. However, the salivary MDA level increased more significantly in the smoking and/or periodontitis groups than did the serum MDA level, and smoking had an additional oxidative effect on periodontitis in terms of the local MDA level. In previous studies examining the serum, salivary, GCF, and gingival MDA levels in individuals with periodontitis, it has been reported that the GCF and salivary MDA levels increase in the presence of periodontitis and smoking, while the systemic MDA level does not change significantly in the presence of periodontitis (9,32-42). These previous findings are similar to the MDA levels observed in the present study.

To the authors' knowledge, only a few studies have focused on the effect of smoking on LPO in patients with periodontitis. Guentsch et al. (10) showed that the MDA levels increased in patients with periodontitis compared to the healthy controls and that the MDA levels were significantly higher in smokers with periodontitis than in periodontally healthy non-smokers. Hendek et al. (43) reported that the GCF level of 4-hydroxynonenal, another LPO product, was significantly high in smokers with periodontitis, but no significant differences were observed in the serum and salivary levels. The present findings are consistent with these previous reports.

The OSI more accurately reflects the degree of oxidative stress. While this index, which is defined as the proportional value between the TOS and TAOC, has been used in various oxidative stress studies in recent years (9,44), it was used in this study to evaluate the relationship between smoking and periodontitis in terms of oxidative injury. In the present study, the serum OSI was significantly higher among the smokers with periodontitis than among the other groups. The salivary OSI was also significantly higher among the smokers with periodontitis, while there were no significant differences between the other groups. These findings show that the systemic and local OSIs are also affected by smoking and periodontitis, in line with the systemic and local TAOCs and TOSs noted herein. While the TAOC is affected by various factors such as nutrition, age, and sex, the TOS is a direct measurement and has certain advantages in predicting oxidative stress. Therefore, the OSI, which is based on the TOS:TAOC ratio, may be more useful in the determination of oxidative stress in individuals with periodontitis.

One of the important limitations of our study is that its cross-sectional design does not allow for an assessment of the causality of the observed relationships. Furthermore, the residual confounding effect of some unmeasured factors (diet, lifestyle, etc.) cannot be completely ruled out.

CONCLUSIONS

In this study, the TAOC, TOS, MDA level, and OSI were investigated in smokers with periodontitis to determine the effects of smoking and periodontitis on oxidative damage. Smoking and/or periodontitis increased the TOS, MDA level, and OSI and decreased the systemic and local TAOCs. These findings indicate that smoking has a similar effect on oxidative stress parameters as periodontitis. However, the much higher amount of oxidative stress seen in both smokers and individuals

with periodontitis may represent an important contribution to understanding the pathophysiology of smoking-related periodontal destruction. The present data could be supported by future studies, emphasizing the importance of efforts that aim to stop smoking in periodontal treatment strategies.

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Ethical approval

This study has been approved by the Ethical Committee of the Karadeniz Technical University Faculty of Medicine (approval date 11/06/2009, number 2009/44). Written informed consent was obtained from the participants.

Author contribution

Surgical and Medical Practices: ÖSA; Concept: ÖSA, EB; Design: EB, AA, ÖSA; Data Collection or Processing: ÖSA, AA, FBY; Analysis or Interpretation: ÖSA, EB; Literature Search: ÖSA, EB, PY, MAK, GA; Writing: ÖSA, EB. All authors reviewed the results and approved the final version of the article.

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Conflict of interest

The authors declare that there is no conflict of interest.

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