

# Total Antioxidant/Oxidant Status in Chronic Periodontitis Patients with Type II Diabetes Following Nonsurgical Periodontal Therapy

Ruby Ramya Vincent, Devapriya Appukuttan, Aruna Balasundaram<sup>1</sup>, Vanaja Krishna Naik<sup>2</sup>, Dhayanand John Victor

Department of Periodontics, SRM Dental College and Hospital, <sup>1</sup>Private Practice, Cure and Care Multispeciality Dental Clinic, Chennai, Tamil Nadu, India, <sup>2</sup>Clinical Teaching Fellow, Academic Faculty, School of Dentistry, University of Leeds, Leeds, England, UK

## Abstract

**Background:** Diabetes and periodontitis are chronic inflammatory diseases characterized by a dysregulated inflammatory response involving oxidative stress (OS). Nonsurgical periodontal therapy (NSPT) eliminates bacterial load followed by reduction in inflammatory burden due to reduced levels of proinflammatory mediators. **Aim:** The study aimed to evaluate the effect of NSPT on the OS biomarkers in the gingival crevicular fluid (GCF) of generalized chronic periodontitis (GCP) patients with Type II diabetes mellitus (DM). **Materials and Methods:** Eighty participants were allotted into Group I, systemically healthy with GCP ( $n = 20$ ); Group II, GCP with Type II DM ( $n = 20$ ); Group III, Type II DM without chronic periodontitis (CP) ( $n = 20$ ); and Group IV, periodontally and systemically healthy controls ( $n = 20$ ). Plaque index, gingival index (GI), probing pocket depth (PPD), and clinical attachment loss (CAL) were recorded. GCF was evaluated for total antioxidant capacity (TAOC), total oxidant status (TOS), and OS index (OSI). Patients in group I and II received NSPT. **Results:** Groups I, II, and III at baseline demonstrated significantly lower GCF TAOC and higher TOS and OSI when compared to Group IV ( $P < 0.001$ ). GI in Group I at baseline negatively correlated with TAOC, whereas PPD and OSI were positively correlated ( $P < 0.05$ ). Following NSPT, significant improvements were observed in the clinical parameters and in the TAOC levels in both group I and II patients ( $P < 0.001$ ). In diabetics with GCP following NSPT, it was observed that OSI positively correlated ( $r = 0.46$ ,  $P < 0.05$ ) with CAL. In group I and II patients, TOS and OSI reduced significantly from baseline ( $P < 0.001$ ). **Conclusions:** Based on the study results, we infer that NSPT can positively modulate the levels of OS biomarkers by restoring the oxidative imbalance. Further, the study underscores the role of periodontal therapy in decreasing oxidative burden in diabetics with periodontal disease.

**Keywords:** Chronic periodontitis, gingival crevicular fluid, oxidative stress index, total antioxidant capacity, total oxidant status, type II diabetes mellitus

## INTRODUCTION

Periodontitis a chronic inflammatory disease is associated with tooth loss and this clinical presumption has been verified by a myriad of studies.<sup>[1,2]</sup> Periodontopathic pathogens are essential for the disease initiation; however, the dysregulated host immunoinflammatory response by means of enhanced secretion of inflammatory mediators and free radicals essentially predisposes to periodontal tissue breakdown.<sup>[3]</sup> Diabetes mellitus (DM) and chronic periodontitis (CP) share a bidirectional relationship, such that diabetic individuals have a two- to three-fold increased risk for developing periodontitis and poor glycemic control is associated with periodontal disease.<sup>[4-7]</sup>

Oxidative stress (OS) regarded as an imbalance between free radicals and antioxidants plays a crucial role in the pathogenesis of chronic diseases.<sup>[8]</sup> This can be determined indirectly by evaluating the total antioxidant capacity (TAOC) and total oxidant status (TOS) in body fluids. The total antioxidants can

**Address for correspondence:** Dr. Devapriya Appukuttan, Department of Periodontics, SRM Dental College and Hospital, Ramapuram, Chennai - 600 089, Tamil Nadu, India. E-mail: devapriyamds@gmail.com

**Submitted:** 26-Jan-2020

**Revised:** 10-Apr-2020

**Accepted:** 30-Apr-2020

**Published:** 20-Jun-2020

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

**For reprints contact:** reprints@medknow.com

**How to cite this article:** Vincent RR, Appukuttan D, Balasundaram A, Naik VK, Victor DJ. Total antioxidant/oxidant status in chronic periodontitis patients with type II diabetes following nonsurgical periodontal therapy. Arch Med Health Sci 2020;8:41-6.

### Access this article online

#### Quick Response Code:



**Website:**  
[www.amhsjournal.org](http://www.amhsjournal.org)

**DOI:**  
10.4103/amhs.amhs\_12\_20

be evaluated by TAOC; this analysis is less time-consuming and lowers the cost of estimating individual antioxidants. Oxidants are commonly free radicals that are extremely short-lived *in vivo* and cannot be measured directly; hence, TOS estimates the different oxidant molecules in the sample as most of the oxidant effects are additive. Consequently, both TOS and TAOC serve as valid approaches to the assessment of OS. OS index (OSI) clearly defines the oxidant/antioxidant imbalances in chronic inflammatory diseases. It is calculated as a percentage ratio of TOS to TAOC. It is possibly claimed that this index may be a suitable parameter in the determination of OS and can contribute to a new prospective to the literature of periodontal diseases.<sup>[9,10]</sup>

In this study, we hypothesized that nonsurgical periodontal therapy (NSPT) perhaps positively modulates the OS in diabetic individuals with CP. Therefore, the primary aim was to evaluate the effects of NSPT on oxidative biomarkers in CP with and without type II DM and to further correlate the biochemical markers with the clinical parameters.

## MATERIALS AND METHODS

### Ethical clearance

The present interventional clinical trial was carried out in the Department of Periodontics, SRM Dental College and Hospital, Chennai, from January 2016 to November 2016. Approval was granted by the Institutional Review Board of SRM University (SRMDC/IRB/2014/MDS/No. 505).

### Subject recruitment and allotment

Eighty patients were recruited based on convenience sampling and allotted into four groups: Group I, systemically healthy with generalized CP (GCP) ( $n = 20$ ); Group II, GCP with Type II DM ( $n = 20$ ); Group III, Type II DM without CP ( $n = 20$ ); and Group IV, periodontally and systemically healthy controls ( $n = 20$ ).

The inclusion criteria were patients aged between 25 and 65 years with a minimum of 20 teeth present (at least 5 teeth in each quadrant). The GCP patients (Group I and II) were selected based on the American Academy of Periodontology 1999 classification.<sup>[11]</sup> Patients presenting with a generalized probing pocket depth (PPD) of  $\geq 5$  mm, clinical attachment loss (CAL) of  $\geq 2$  mm in more than 30% of sites, and presence of  $\geq 30\%$  of sites with bleeding on probing were included in the CP (Group I) and CP-DM group (Group II). Patients diagnosed with Type II DM by a diabetologist and under treatment with oral hypoglycemic agents for a minimum duration of 6 months or more were included in Group II and III.

Diabetic individuals with diabetic complications, patients with any other systemic or autoimmune diseases, those under antibiotic therapy for the past 6 months, anti-inflammatory drugs, mouthwashes, vitamins, mineral supplements, or antioxidant therapy within the last 3 months, history of prior periodontal therapy, smokers, alcoholics, and pregnant and lactating women were excluded from the study.

### Periodontal examination

Plaque index (PI),<sup>[12]</sup> gingival index (GI),<sup>[12]</sup> PPD, and CAL were recorded. Four sites around each tooth were scored for the presence/absence of plaque (PI) and for the presence/absence of gingival inflammation. PPD and CAL were recorded for all teeth, 6 sites per tooth using a UNC-15 periodontal probe. All the clinical parameters were assessed at baseline (DA). The patients in Group I and II were reevaluated after 6 weeks following NSPT. A single calibrated examiner (DA) recorded the clinical parameters both at baseline and at 6<sup>th</sup> week following NSPT.

### Collection of gingival crevicular fluid

Following selection into the study, clinical parameters were recorded and complete supragingival ultrasonic scaling was completed. To prevent blood contamination of the gingival crevicular fluid (GCF), sample was collected on the morning of the subsequent day, 3–4 h after breakfast. The recruits were made to sit comfortably in an upright position and the area to be sampled was isolated with cotton rolls and slightly air-dried to avoid salivary contamination. Microcapillary pipettes (5  $\mu$ l microcapillary pipettes, Sigma-Aldrich) were gently inserted into the sulcus, and the pipettes were placed in the collection sites for a maximum of 10 min. The deepest sites were chosen for GCF collection and the samples from those sites were pooled so that 15–20  $\mu$ l was obtained. The collected GCF was transferred into Eppendorf tubes, containing 200  $\mu$ l of 20 mM Tris-HCl buffer (pH 6.5) and stored at 80°C, until analysis. The samples were collected at baseline for all patients and 6 weeks after NSPT by a single investigator (AB) for patients in Group I and Group II.

### Nonsurgical periodontal therapy

The patients in Group I and II received NSPT, comprising complete ultrasonic scaling and a full-mouth root surface debridement using ultrasonic scaler (Satellac Scaler unit, Acteon, UK) and area-specific Gracey curettes (Hu-friedy Mfg. Co, Chicago, USA) under local anesthesia (LA). The procedure was carried out by a single clinician ((Ruby Ramya Vincent) RRV). Patients were advised about the possible discomfort and pain that can arise following waning away of the LA effect and were prescribed analgesics. In addition, each patient received personalized oral hygiene instructions and was asked to maintain meticulous oral hygiene including use of mouthwash and warm saline rinse. They were asked to avoid hot, cold, or spicy food for few days due to the interim tooth sensitivity.

### Recall visit

Following NSPT, patients were recalled after 6 weeks for review. The clinical parameters including the PI, GI, PPD, and CAL were reassessed and GCF samples were collected. A similar protocol was followed as before and pooled GCF samples were collected from the same baseline sites and stored until analysis.

### Biochemical assay

The biochemical assay kits were procured from Rel Assay Diagnostics, Turkey. Erel O's automated method of colorimetric analysis was used to measure TAOC<sup>[13]</sup> and TOS.<sup>[14]</sup>

### Total antioxidant assay

The principle behind TAOC assay is that antioxidants in the GCF sample will reduce the dark blue-green colored 2,2'-azino-bis 3-ethylbenzothiazoline-6-sulfonic acid (ABTS) radical to a colorless reduced ABTS form. There is a change of absorbance at 660 nm and was related to the total antioxidant level of the sample. The assay was calibrated with a stable antioxidant standard solution, which is traditionally named as Trolox Equivalent, which is a Vitamin E analog.

### Total oxidant status

TOS assay is based on the principle that oxidants present in the GCF sample oxidize the ferrous ion-chelator complex to ferric ion. The oxidation reaction is prolonged by enhancer molecules, which are abundantly present in the reaction medium. The ferric ion makes a colored complex with the chromogen in an acidic medium. The color intensity, which can be measured spectrophotometrically, is related to the total amount of oxidant molecules present in the sample. The assay is calibrated with hydrogen peroxide and the results are expressed in terms of micromolar hydrogen peroxide equivalent per liter ( $\mu\text{mol H}_2\text{O}_2$  Equiv./L).

### Statistical analysis

IBM SPSS Statistics for Windows, Version 20.0., Armonk, NY, USA: IBM Corp., was used for analysis. Kolmogorov-Smirnov test indicated a normal distribution of data. Hence, parametric tests were applied. One-way analysis of variance (ANOVA) was used to compare means between groups followed by

Tukey's *post hoc* test for pairwise comparisons. Paired *t*-test and Independent *t*-test were applied for intra- and intergroup comparisons, respectively. Pearson's correlation was applied to correlate the clinical and biochemical parameters at baseline and at 6 weeks following NSPT and  $P < 0.05$  was considered to be statistically significant. None of the patients were lost to follow-up following NSPT in either Group I or II.

### RESULTS

Statistical analysis revealed that at baseline, the mean PI and GI scores were higher in Group I, II, and III patients when compared with the Group IV [Table 1]. Intergroup comparison of clinical parameters by one-way ANOVA showed a statistically significant difference between the groups ( $P < 0.001$ ). The mean TAOC values were higher in Group IV than in Group I, II, and III. In addition, the mean TOS and OSI were higher in Group I, II, and III when compared to Group IV. All the biochemical parameters showed a statistically significant difference ( $P < 0.001$ ) between groups at baseline [Table 1]. *Post hoc* analysis following ANOVA at baseline is shown in Table 2.

Following NSPT at 6 weeks, all the clinical parameters improved from baseline with statistical significance in Group I and II ( $P < 0.001$ ) [Table 3]. The mean PI and GI scores reduced in both the groups. There was a reduction in the mean PPD and gain in the mean CAL from baseline in both the groups. There was no statistically significant difference observed in

**Table 1: Descriptive statistics and intergroup comparison of clinical and biochemical parameters evaluated at baseline and at 6 weeks**

Parameters	Group I	Group II	Group III	Group IV	P
PI					
Baseline <sup>#</sup>	1.287±0.45	1.495±0.26	1.518±0.43	0.367±0.16	0.001**
After 6 weeks <sup>‡</sup>	0.495±0.22	0.606±0.25	-	-	0.153
GI					
Baseline <sup>#</sup>	1.372±0.42	1.621±0.24	1.250±0.38	0.000	0.001**
After 6 weeks <sup>‡</sup>	0.498±0.28	0.607±0.25	-	-	0.207
PPD (mm)					
Baseline <sup>#</sup>	5.284±0.22	5.424±0.23	2.305±0.31	1.515±0.22	0.001**
After 6 weeks <sup>‡</sup>	2.571±0.23	2.606±0.30	-	-	0.691
CAL (mm)					
Baseline <sup>#</sup>	2.514±0.38	2.873±0.72	0.000	0.000	0.001**
After 6 weeks <sup>‡</sup>	0.567±0.48	1.024±0.82	-	-	0.039*
TAOC (mmol/L)					
Baseline <sup>#</sup>	0.7551±0.24	0.697±0.19	0.776±0.27	1.202±0.41	0.001**
After 6 weeks <sup>‡</sup>	1.384±0.21	1.165±0.17	-	-	0.001**
TOS ( $\mu\text{mol/L}$ )					
Baseline <sup>#</sup>	9.088±3.70	10.051±3.26	7.848±1.50	5.201±1.30	0.001**
After 6 weeks <sup>‡</sup>	6.300±1.71	7.162±1.53	-	-	0.102
OSI					
Baseline <sup>#</sup>	1.248±0.73	1.395±0.62	1.008±0.52	0.525±0.36	0.001**
After 6 weeks <sup>‡</sup>	0.771±0.44	0.808±0.32	-	-	0.769

\*\* $P < 0.01$ , highly significant, \* $P < 0.05$ , statistically significant, <sup>#</sup>One-way ANOVA, <sup>‡</sup>Independent *t*-test. ANOVA: Analysis of variance, PI: Plaque index, GI: Gingival index, PPD: Probing pocket depth, CAL: Clinical attachment loss, TAOC: Total antioxidant capacity, TOS: Total oxidant status, OSI: Oxidative stress index

**Table 2: Tukey's *post hoc* pairwise comparison of clinical and biochemical parameters at baseline between the four groups**

Variables	Group I versus Group II ( <i>P</i> )	Group I versus Group III ( <i>P</i> )	Group I versus Group IV ( <i>P</i> )	Group II versus Group III ( <i>P</i> )	Group II versus Group IV ( <i>P</i> )	Group III versus Group IV ( <i>P</i> )
PI	0.249	0.169	0.001**	0.997	0.001**	0.001**
GI	0.071	0.619	0.001**	0.002**	0.001**	0.001**
PPD (mm)	0.308	0.001**	0.001**	0.001**	0.001**	0.001**
CAL (mm)	0.034*	0.001**	0.001**	0.001**	0.001**	1.000
TAOC (mmol/L)	0.925	0.996	0.001**	0.831	0.001**	0.001**
TOS (μmol/L)	0.663	0.459	0.001**	0.051	0.001**	0.012*
OSI	0.865	0.541	0.001**	0.157	0.001**	0.052

\**P*<0.05, statistically significant, \*\**P*<0.01, highly significant. PI: Plaque index, GI: Gingival index, PPD: Probing pocket depth, CAL: Clinical attachment loss, TAOC: Total antioxidant capacity, TOS: Total oxidant status, OSI: Oxidative stress index

**Table 3: Intragroup and intergroup comparison of clinical and biochemical parameters from baseline to 6 weeks following nonsurgical periodontal therapy, in Group I and Group II**

From baseline to 6 weeks following NSPT	<i>t</i>	<i>P</i>
PI		
Intragroup Group I <sup>†</sup>	12.873	0.001**
Intragroup Group II <sup>†</sup>	13.619	0.001**
Intergroup Group I versus II <sup>‡</sup>	-1.457	0.153
GI		
Intragroup Group I <sup>†</sup>	13.238	0.001**
Intragroup Group II <sup>†</sup>	23.942	0.001**
Intergroup Group I versus II <sup>‡</sup>	-1.283	0.207
PPD (mm)		
Intragroup Group I <sup>†</sup>	39.046	0.001**
Intragroup Group II <sup>†</sup>	45.821	0.001**
Intergroup Group I versus II <sup>‡</sup>	-0.400	0.691
CAL (mm)		
Intragroup Group I <sup>†</sup>	29.515	0.001**
Intragroup Group II <sup>†</sup>	25.523	0.001**
Intergroup Group I versus II <sup>‡</sup>	-2.137	0.039*
TAOC (mmol/L)		
Intragroup Group I <sup>†</sup>	-10.643	0.001**
Intragroup Group II <sup>†</sup>	-9.188	0.001**
Intergroup Group I versus II <sup>‡</sup>	3.597	0.001**
TOS (μmol/L)		
Intragroup Group I <sup>†</sup>	4.255	0.001**
Intragroup Group II <sup>†</sup>	4.430	0.001**
Intergroup Group I versus II <sup>‡</sup>	1.673	0.102
OSI		
Intragroup Group I <sup>†</sup>	5.822	0.001**
Intragroup Group II <sup>†</sup>	4.556	0.001**
Intergroup Group I versus II <sup>‡</sup>	0.295	0.769

\**P*<0.05, statistically significant, \*\**P*<0.01, highly significant, <sup>†</sup>Paired *t*-test, <sup>‡</sup>Independent *t*-test. PI: Plaque index, GI: Gingival index, PPD: Probing pocket depth, CAL: Clinical attachment loss, TAOC: Total antioxidant capacity, TOS: Total oxidant status, OSI: Oxidative stress index

the PI, GI, and PPD between group I and II. The gain in the mean CAL following NSPT showed statistically significant difference between the groups (*P* < 0.05) [Table 3].

The mean TAOC improved following NSPT in both the Groups (I and II) and was statistically significant (*P* < 0.001) [Table 3].

TOS and OSI levels reduced significantly from following NSPT in both Groups I and II (*P* < 0.001) [Table 3]. The decrease in the oxidant levels in both the groups indicates the effectiveness of NSPT in modulating the OS markers in periodontal disease. It was also observed that periodontitis patients had greater improvements in TAOC levels in comparison to patients with coexisting type II DM, with significant difference statistically (*P* < 0.001) [Table 3].

Correlation showed that the GI and TAOC were negatively correlated and PPD and the OSI were positively correlated at baseline in Group I (*P* < 0.05) [Table 4]. The CAL positively correlated with OSI post-NSPT in Group II (*P* < 0.05) [Table 4].

## DISCUSSION

Total antioxidants levels in the present study were lower in diabetic and CP patients when compared to periodontally and systemically healthy controls; this observation was similar to the findings of Atabay *et al.*<sup>[15]</sup> Likewise, reduced serum and GCF TAOC have been demonstrated among pregnant,<sup>[16]</sup> preeclamptic,<sup>[17]</sup> and postmenopausal women<sup>[18]</sup> with CP indicating a decline in both systemic and local antioxidant defense system. On the contrary, Esen *et al.*<sup>[9]</sup> demonstrated no significant difference in the GCF TAOC levels in periodontitis patients when compared to healthy controls. Wei *et al.*<sup>[19]</sup> and Su *et al.*<sup>[20]</sup> reported higher antioxidant activity in CP patients, which was in disagreement with the present study and these authors ascribed their findings to a possibly upregulated antioxidant enzyme system in cells and tissues as local response to increase in OS associated with periodontal inflammation.

Chronic periodontal disease induces a low-grade inflammation, thereby creating an oxidative environment and reducing the TAOC capacity. This depletion of antioxidants could be ascribed toward the necessity to constantly counterbalance the exaggerated ROS activity generated during periodontal inflammation. It was observed from the current study that among all the four groups, GCF TAOC at baseline was lowest in patients with GCP and DM; this perhaps is attributed to hyperglycemia that amplifies inflammation in periodontal tissues. Advanced glycation end products (AGE) bind to receptor



**Table 4: Pearson's correlation P value is shown between the clinical and the biochemical parameters evaluated at baseline and following nonsurgical periodontal therapy in Group I and II**

	TAOC	TOS	OSI
Correlations at baseline in Group I			
Group I			
PI	0.415	0.872	0.561
GI	0.032*	0.648	0.378
PPD	0.247	0.240	0.042*
CAL	0.089	0.869	0.335
Group II			
PI	0.557	0.430	0.462
GI	0.694	0.701	0.653
PPD	0.794	0.130	0.319
CAL	0.451	0.292	0.898
Correlations at 6 weeks following NSPT			
Group I			
PI	0.649	0.862	0.502
GI	0.701	0.626	0.383
PPD	0.795	0.058	0.800
CAL	0.259	0.908	0.562
Group II			
PI	0.860	0.281	0.202
GI	0.880	0.187	0.821
PPD	0.347	0.777	0.713
CAL	0.564	0.635	0.041*

\* $P < 0.05$ , statistically significant. PI: Plaque index, GI: Gingival index, PPD: Probing pocket depth, CAL: Clinical attachment loss, TAOC: Total antioxidant capacity, TOS: Total oxidant status, OSI: Oxidative stress index

for AGE resulting in upregulated production of proinflammatory cytokines which further increases the intensity of periodontal inflammation in diabetics. Furthermore, these glycosylated products enhance neutrophil priming and increase nicotinamide adenine dinucleotide phosphate oxidase activity leading to excessive free radical production, thus contributing to impaired antioxidant capacity. These pathologic mechanisms in DM together with the preexisting periodontal disease could be responsible for the exaggerated periodontal destruction seen in diabetics and also may explain the greater risk for periodontitis in diabetics.

The GCF TAOC at baseline negatively correlated with gingival index in group I; this could be attributed to the reduced antioxidant levels associated with amplified inflammatory burden in the gingival tissues in periodontitis. Atabay *et al.*<sup>[15]</sup> showed a similar negative correlation between GCF TAOC and all the clinical parameters including PI, GI, BOP, PPD, and CAL ( $r = -0.809, -0.844, -0.891, -0.756, \text{ and } -0.721$ , respectively, and  $P < 0.01$ ). Akpinar *et al.*<sup>[21]</sup> demonstrated a negative correlation between GCF TAOC with PI ( $r = -0.578, P < 0.05$ ) in nonsmokers with CP and a positive correlation between GCF TAOC and PPD ( $r = 0.636, P < 0.05$ ) in smokers with CP.

Examination of baseline GCF TOS in the current study showed significantly higher levels in patients with CP, CP

with Type II DM, and DM (Group I, II, and III, respectively) when compared to the periodontally and systemically healthy controls (Group IV). Further, the highest total oxidant levels were seen in Group II patients. When comparing the baseline GCF TOS and TAOC levels in the present study, the imbalance in oxidant and antioxidant status is clearly evident with the balance tipping in favor of elevated oxidants probably related to increased OS implicated in both the disease processes.

OSI values were significantly higher in Group I, II, and III than Group IV at baseline ( $P < 0.01$ ). This is possibly due to increased oxidant status in both periodontal disease and DM. Our results were supported by Wei *et al.*<sup>[19]</sup> and Akalin *et al.*<sup>[22]</sup> Both the authors reported elevated levels of serum, saliva, and crevicular fluid total oxidant levels in CP patients and ascribed their findings to increased OS both locally and systemically by periodontal disease. However, Zhang *et al.*<sup>[23]</sup> showed no such difference in the salivary TOS levels between CP and healthy controls, which was in disagreement with the present study.

It was observed that a statistically significant increase in GCF TAOC and a decrease in TOS levels in both Group I and II patients ( $P < 0.01$ ) were seen following NSPT; this was similar to the findings of Bansal *et al.*<sup>[24]</sup> These authors showed an increase in plasma antioxidants level 3 weeks following NSPT in CP patients (baseline TAC  $792.33 \pm 124.33 \mu\text{mol/L}$ ; following NSPT  $989.75 \pm 96.80 \mu\text{mol/L}$ ). Chapple *et al.*<sup>[25]</sup> hypothesized that GCF TAOC levels increased to the levels of the healthy controls following periodontal therapy and the restoration of TAOC to healthy levels following NSPT indicated that the imbalance possibly resulted from periodontal inflammation. Wei *et al.*<sup>[19]</sup> showed reduced serum, saliva, and GCF TOS levels following initial periodontal therapy in CP group compared with the baseline levels. On the contrary, Toker *et al.*<sup>[26]</sup> failed to demonstrate any significant change in the levels of GCF TAOC and TOS at baseline and after 6 weeks following NSPT in nonsmokers with CP when compared to the healthy controls.

The OSI values reduced following periodontal therapy from baseline in both the groups; this is due to the increase in the TAOC levels and reduction in TOS achieved following periodontal therapy. Thus, OSI can be utilized as a biomarker for OS and a prognostic indicator following periodontal therapy. In this study, OSI positively correlated with PPD at baseline in CP patients and with CAL in group II patients following NSPT, suggesting that periodontal disease severity is associated with OS and reduction in OS -induced inflammation following periodontal therapy improved clinical parameters. The principal study by Esen *et al.*<sup>[9]</sup> evaluated the effects of CP and rheumatoid arthritis on serum and GCF TAOC, TOS, and OSI. Similar to the present study, baseline OSI values were higher and statistically significant in CP patients than periodontally healthy controls. Another study by Baltacıoğlu *et al.*<sup>[10]</sup> investigated serum and salivary levels of TOS, TAOC, and OSI from patients with CP and generalized aggressive periodontitis (GAgP). An elevated TOS and OSI

levels were seen in CP and GAgP than periodontally healthy controls. Their study demonstrated a positive correlation between clinical parameters and OSI, thus supporting our findings. Therefore, both the above studies supported the results from the current study. Hence, this study demonstrated that NSPT successfully reduces the OS in the periodontal tissues in CP patients with and without diabetes.

In the current study, there could have been a possible influence of oral hypoglycemic drugs on the antioxidant levels in diabetic patients, since studies have shown an improvement in antioxidants levels following their administration. However, this factor cannot be controlled as the patients cannot be refrained from their drugs. Hemoglobin a1c levels were not assessed in the present study. Hence, the glycemic control of the diabetic patients was not validated. This is a confounding factor as the glycemic levels can influence the levels of OS biomarkers in these patients and therefore a possible study limitation.

## CONCLUSION

This study further underscores the prevailing presumption and adds on to the scientific understanding that OS persists not only in patients with chronic periodontal disease *per se* but also in those with coexisting type II DM. Furthermore, patients diagnosed with both CP and diabetes have heightened OS levels and nonsurgical periodontal intervention reduces the systemic inflammatory load in diabetic patients and harmonizes the imbalance prevailing between free radicals and antioxidants, thereby possibly preventing the progression of periodontal destruction in these individuals. The current study further corroborates the use of OSI as a marker for periodontal disease activity in type II diabetics with periodontal disease.

## Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

## REFERENCES

- Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease: Current concepts. *J Periodontol* 1992;63:322-31.
- Moore WE, Moore LV. The bacteria of periodontal diseases. *Periodontol* 2000 1994;5:66-77.
- Kinane DF, Lappin DF. Immune processes in periodontal disease: A review. *Ann Periodontol* 2002;7:62-71.
- Mealey BL, Oates TW; American Academy of Periodontology. Diabetes mellitus and periodontal diseases. *J Periodontol* 2006;77:1289-303.
- Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: An epidemiologic perspective. *Ann Periodontol* 2001;6:99-112.
- Löe H. Periodontal disease. The sixth complication of diabetes mellitus. *Diabetes Care* 1993;16:329-34.
- Ohlrich EJ, Cullinan MP, Leichter JW. Diabetes, periodontitis, and the subgingival microbiota. *J Oral Microbiol* 2010;2:5818.
- Sies H. Oxidative stress: Oxidants and antioxidants. *Exp Physiol* 1997;82:291-5.
- Esen C, Alkan BA, Kırnap M, Akgül O, Işıkoğlu S, Erel O. The effects of chronic periodontitis and rheumatoid arthritis on serum and gingival crevicular fluid total antioxidant/oxidant status and oxidative stress index. *J Periodontol* 2012;83:773-9.
- Baltacıoğlu E, Yuva P, Aydın G, Alver A, Kahraman C, Karabulut E, *et al.* Lipid peroxidation levels and total oxidant/antioxidant status in serum and saliva from patients with chronic and aggressive periodontitis. Oxidative stress index: A new biomarker for periodontal disease? *J Periodontol* 2014;85:1432-41.
- Armitage GC. Periodontal diagnoses and classification of periodontal diseases. *Periodontol* 2000 2004;34:9-21.
- Löe H. The gingival index, the plaque index and the retention index systems. *J Periodontol* 1967;38:610-6.
- Erel O. A novel automated method to measure total antioxidant response against potent free radical reactions. *Clin Biochem* 2004;37:112-9.
- Erel O. A new automated colorimetric method for measuring total oxidant status. *Clin Biochem* 2005;38:1103-11.
- Atabay VE, Lutfioğlu M, Avci B, Sakallioğlu EE, Aydoğdu A. Obesity and oxidative stress in patients with different periodontal status: A case-control study. *J Periodontol Res* 2017;52:51-60.
- Akalin FA, Baltacıoğlu E, Alver A, Karabulut E. Total antioxidant capacity and superoxide dismutase activity levels in serum and gingival crevicular fluid in pregnant women with chronic periodontitis. *J Periodontol* 2009;80:457-67.
- Canakci V, Yildirim A, Canakci CF, Eltas A, Cicek Y, Canakci H. Total antioxidant capacity and antioxidant enzymes in serum, saliva, and gingival crevicular fluid of preeclamptic women with and without periodontal disease. *J Periodontol* 2007;78:1602-11.
- Baltacıoğlu E, Akalin FA, Alver A, Balaban F, Unsal M, Karabulut E. Total antioxidant capacity and superoxide dismutase activity levels in serum and gingival crevicular fluid in post-menopausal women with chronic periodontitis. *J Clin Periodontol* 2006;33:385-92.
- Wei D, Zhang XL, Wang YZ, Yang CX, Chen G. Lipid peroxidation levels, total oxidant status and superoxide dismutase in serum, saliva and gingival crevicular fluid in chronic periodontitis patients before and after periodontal therapy. *Aust Dent J* 2010;55:70-8.
- Su H, Gornitsky M, Velly AM, Yu H, Benarroch M, Schipper HM. Salivary DNA, lipid, and protein oxidation in nonsmokers with periodontal disease. *Free Radic Biol Med* 2009;46:914-21.
- Akpınar A, Toker H, Ozdemir H, Bostancı V, Aydın H. The effects of non-surgical periodontal therapy on oxidant and anti-oxidant status in smokers with chronic periodontitis. *Arch Oral Biol* 2013;58:717-23.
- Akalin FA, Baltacıoğlu E, Alver A, Karabulut E. Lipid peroxidation levels and total oxidant status in serum, saliva and gingival crevicular fluid in patients with chronic periodontitis. *J Clin Periodontol* 2007;34:558-65.
- Zhang T, Andrukho O, Haririan H, Müller-Kern M, Liu S, Liu Z, *et al.* Total antioxidant capacity and total oxidant status in saliva of periodontitis patients in relation to bacterial load. *Front Cell Infect Microbiol* 2015;5:97.
- Bansal N, Gupta ND, Bey A, Sharma VK, Gupta N, Trivedi H. Impact of nonsurgical periodontal therapy on total antioxidant capacity in chronic periodontitis patients. *J Indian Soc Periodontol* 2017;21:291-5.
- Chapple IL, Brock GR, Milward MR, Ling N, Matthews JB. Compromised GCF total antioxidant capacity in periodontitis: Cause or effect? *J Clin Periodontol* 2007;34:103-10.
- Toker H, Akpınar A, Aydın H, Poyraz O. Influence of smoking on interleukin-1beta level, oxidant status and antioxidant status in gingival crevicular fluid from chronic periodontitis patients before and after periodontal treatment. *J Periodontol Res* 2012;47:572-7.