Oxidant and Antioxidant Status of Peri-Implant Crevicular Fluid in Patients with Sandblasted Acid-Etched and Anodized Dental Implants: a Prospective Clinical Study

Abstract

Background
Exploring oxidant and antioxidant profile around different surface treated dental implants is essential to improve the performance of implants. The purpose of this research was to assess total antioxidant capacity (TAOC), total oxidant status (TOS) and oxidative stress index (OSI) in peri-implant crevicular fluid among patients with sandblasted acid-etched and anodized dental implants.

Materials and Methods
In this prospective clinical study, 78 patients who had undergone implant placement for missing single posterior tooth in mandible using sandblasted acid-etched and anodized dental implants during August 2019 - December 2019 were enrolled and categorized into Group 1: SLA (n=27), Group 2: SLActive (n=26), Group 3: TiUnite (n=25) based on the surface modification of implants. Peri-implant crevicular fluid (PICF) was collected at baseline (3 months after placement, before functional loading) and again at 1 year from implant placement to assess TOS, TAOC, OSI using calorimetric assays.. Statistical analysis was performed using one-way ANOVA for intergroup comparison, followed by Tukey's HSD post hoc for pairwise comparison. For intragroup comparison, paired t test was used.

Results
TOS and OSI in group 3 implants were higher than groups 1 and 2 ($p \leq 0.05$). TAOC in group 3 implants was lower than groups 1 and 2 ($p \leq 0.05$). On pairwise comparison, there was a significant difference between the groups at baseline ($p \leq 0.05$) and 1-year follow up ($p \leq 0.05$). Intragroup comparison showed statistically significant difference in terms of TOS, TAOC and OSI from baseline in all the three groups ($p \leq 0.05$).

Conclusion
Higher total oxidative capacity and oxidative stress index as well as lower antioxidative activity were observed in peri-implant crevicular fluid around TiUnite dental implants.

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Authors

Janagarathinam P. [1]
Rajasekar A. [2*]

1
Post Graduate, Department of Implantology, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, India

2
Associate Professor, Department of Periodontology, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, India

*
Corresponding author

Keywords

Antioxidant, Dental implant, Oxidative stress, Redox, Surface modification.

INTRODUCTION

Dental implants have become an accepted treatment option for missing teeth in recent years. Dr. Branemark introduced the first threaded titanium root-form implant that was documented in 1965 (1). Since then, in order to increase the success rate, implants have undergone significant evolution in size, shape, and surface. However, few individuals experience implant failure due to peri-implant disease. Peri-implant mucositis and peri-implantitis are the two peri-implant diseases. The soft tissues around the implant are affected in peri-implant mucositis, although the surrounding bone remains unaffected. In contrast, peri-implantitis is known to involve the bone (2).

There are numerous methods of modifying the surface of dental implants which include acid treatments, sandblasting or various oxidization mechanisms. In order to create sandblasted-acid etched (SLA) implant surfaces, coarse grit particles are first used to sandblast the implant's macrostructure and then hydrochloric and sulfuric materials are used to etch the surface and create micro-irregularities (3). An advancement over SLA surface is SLActive, where, the implant surface is cleaned using nitrogen protection, hydroxylated, and stored in saline solution. This process makes the surface hydrophilic with greater affinity towards blood clot and angiogenesis, enhancing the earlier stages of osseointegration (4). Another electrochemical method is anodization, which creates a thick titanium dioxide layer thereby making the surface osteoconductive (5).

The surface treatment of dental implants increases the roughness, which in turn help with osseointegration. But these alterations in surface topography also have a minor, tangential effect on microbial adhesion (6). Research evidence suggests that bacterial plaque is the etiology of peri-implant disease similar to periodontal disease (7,8). Although the bacterial plaque causes initiation of peri-implant disease, the bacterial-host interaction contribute to the progression of the disease around implants. Following this, various pro-inflammatory mediators are generated which attracts the neutrophils to the infection site (9).

Bacterial invasion is controlled by neutrophils via oxidative and non-oxidative killing mechanisms. Oxidative killing method involves production of reactive oxygen species by neutrophils and other phagocytes. Reactive oxygen species can damage DNA, lipids, proteins, enzymes and tissues (10). Thus, the body has a variety of defensive antioxidant activities whose primary function is to either eliminate or repair the destruction caused by reactive oxygen species as soon as they develop. Antioxidants are substances that, when present in low concentrations compared to the substrate, will significantly slow down or prevent oxidation of a substrate. Either by scavenging reactive metabolites or transforming the reactive molecules into less reactive ones, antioxidants defend the host (11). Under physiological conditions, reactive oxygen species activity and antioxidant defence capacity are in a dynamic equilibrium. However, when there is a favour towards reactive oxygen species, it results in oxidative stress (12). This imbalance has been suggested as one of the causative factors for peri-implant disease.

In the pathophysiology of chronic diseases, oxidative stress (OS), which is an excess of reactive oxygen species as compared to antioxidants, is a key factor. By assessing the total oxidant status (TOS) and total antioxidant capacity (TAOC), OS can be indirectly established. TAOC assesses the antioxidant capacity of all the antioxidants in a biological sample. This analysis takes less time and is less expensive than estimating individual antioxidants. Since oxidants have a short half-life and cannot be detected directly, TOS calculates the concentrations of the various oxidant molecules in the sample. As a result, both TOS and TAOC are reliable methods for evaluating OS. The oxidant and antioxidant imbalance could be precisely defined by the oxidative stress index (OSI). It is determined using the percentage of TOS to TAOC. Although oxidant and antioxidant levels in peri-implant disease conditions has been studied (13,14), there is a lacuna about the influence of different surface treatments of dental implants on oxidant and antioxidant levels in peri-implant sulcus. In this context, the purpose of this research was to assess the total antioxidant capacity (TAOC), total oxidant status (TOS) and oxidative stress index (OSI) in peri-implant crevicular fluid among patients with sandblasted acid-etched (SLA, SLActive) and anodized (TiUnite) dental implants.

MATERIALS AND METHODS

Study population

In this prospective clinical study, all patients of 25-60 years who had undergone implant placement for missing single posterior tooth in mandible using sandblasted acid-etched and anodized surface dental implants during August 2019 - December 2019 in Department of Periodontics and Implantology, Saveetha Dental College and Hospitals Chennai, India were enrolled according to strict inclusion and exclusion criteria and were categorized based on the surface modification of the dental implants.

The study was carried out in compliance with the 2013 revision of the 1975 Helsinki Declaration. Each participant signed a consent form acknowledging their voluntary participation in the study and the protocol was reviewed and approved by the Institutional Ethical Committee (IHEC/SDC/PERIO/1913/19/TH-01). Sample size was calculated using mean and standard deviation values from a previous study (15)

using G*Power Software, Version 3.0. An α of 0.05 and a power of 80% were selected. The target sample size was 70 implants.

Inclusion criteria

1. Subjects with implant placement for missing single posterior tooth (first or second molar) in mandible
2. Subjects of age between 25 and 60 years
3. Subjects with opposing natural tooth
4. Subjects without systemic diseases
5. Periodontally healthy subjects
6. Subjects with implant placement done in healed extraction sites for a minimum of 6 months
7. Subjects with implant placement done with sufficient bone volume
8. Subjects with implant placement done with the insertion torque of between 35 and 45 Ncm
9. Implants placed subcrestally, verified by digital periapical radiograph
10. Presence of keratinized mucosa width of ⩾2 mm around the implant
11. Subjects with plaque index (PI) score of 0.1-0.9 (Silness and Loe 1964) and gingival index (GI) score of 0.1-1 (Loe and Silness 1963)

Exclusion criteria

1. Immunosuppressed or immunocompromised
2. Subjects with history of radiotherapy or chemotherapy
3. Subjects with underlying systemic illness
4. Pregnant or Lactating women
5. Smokers
6. Poor oral hygiene and motivation
7. Periodontitis patients
8. Subjects with parafunctional habits
9. Subjects with bone metabolic diseases and under treatment with intravenous amino-bisphosphonates
10. Subjects under long term medications
11. Subjects with major bone grafting procedures at implant placement
12. Active inflammation or pathologies adjacent to implant
13. History of extraction due to any cysts, granulomas or tumors.

Seventy-eight subjects with 78 implants fulfilled the study criteria and were divided into three groups, Group 1: SLA (SLA®, Straumann, Basel, Switzerland; n=27), Group 2: SLActive (SLActive®, Straumann, Basel, Switzerland; n=26), Group 3: TiUnite (TiUnite®, Nobel Biocare, Gothenburg, Sweden; n=25) based on the surface modification of the dental implants. All were internal-hex varying platform root analog bone level implants.

Surgical procedure

Surgical and restorative procedures in three groups were performed by experienced surgeons of the same institution. The implant osteotomy was carried out using sequential bone drills of increasing diameter following the elevation of full-thickness mucoperiosteal flap via a crestal incision. Using a digital periapical radiograph and a paralleling pin with a diameter of 2 mm, the orientation of the osteotomy was evaluated. All were bone level implants placed 0.5 mm subcrestally, verified by digital periapical radiograph. Titanium-healing abutments were installed. The sizes of every implant in each group are listed in Table 1. The surgical wound was sutured with 4/0 non-absorbable polypropylene monofilament (Orilene®; Orion Sutures Pvt Ltd., Bangalore, India). Antibiotics (Amoxicillin 500 mg three times a day for three days) and analgesics (Zerodol-SP twice a day for two days) were prescribed to all the patients. Following implant surgery, patients were advised to use soft toothbrush and chlorhexidine gel (Hexigel®, ICPA Health Products Ltd., GIDC, Ankleshwar, India) in the operated area after the surgery. Suture removal was done 1 week postoperatively. For three months, all implants were left in a healing state with no functional loading. During stage 2 uncovery procedure (3 months), peri-implant crevicular fluid (PICF) (baseline) was collected to assess the TOS, TAOC and OSI. All the patients were given final cement retained implant-supported porcelain-fused-to-metal prosthetic restoration. Patients were on maintenance visits every 3 months and oral hygiene instructions were reinforced. After the completion of

Implant Dimension	Group 1 (n)	Group 2 (n)	Group 3 (n)	Total (n)
4.1 * 10	18	17		35
4.1 * 12	6	5		11
4.8 * 10	3	4		7
4.3 * 10			12	12
4.3 * 11.5			9	9
5 * 10			4	4
Total (n)	27	26	25	78

Tab. 1 Implants included in the study

the restorative phase (1 year from implant placement), all patients were re-examined and PICF was collected.

Sample collection

Each selected implant site was isolated with sterile cotton, after the supragingival plaque was removed using sterile curettes. Using a 1-5 µL calibrated microcapillary pipette (Sigma-Aldrich®, Missouri, USA), peri-implant crevicular fluid (PICF) was collected. The obtained samples were kept in storage at -20°C for subsequent analysis. Sample collection was done by a single examiner (AR).

Analysis of TOS, TAOC, OSI

TOS and TAOC in PICF were determined using TOS Colorimetric Assay Kit (Elabscience®, USA) and TAOC Colorimetric Assay Kit (Elabscience®, USA) respectively. The colorimetric assays were performed based on manufacturer's instructions. The results of TOS and TAOC were represented as µmol H_2O_2 Equiv./L and mmol Trolox Equiv./L respectively. For OSI calculation, TAOC in mmol Trolox Equiv./L was converted to µmol H_2O_2 Equiv./L and then the percentage of TOS to TAOC was obtained.

Statistical analysis

SPSS Software, Version 23.0 (IBM Corp., Armonk, NY, USA) was utilized to analyse the data. The Kolmogorov-Smirnov test and the Shapiro-Wilk test results followed a parametric distribution. One way ANOVA was used to compare mean age, PI, GI, TOS, TAOC and OSI between the three groups. Gender distribution was assessed using Chi-square test. For pairwise comparison, Tukey's HSD post hoc test was performed. For intragroup comparison, paired t test was used. A statistically significant result was defined as p value less than 0.05.

RESULTS

Table 2 summarizes the demographic characteristics of the study groups. There was a statistical insignificance between the three study groups in relation to age (p = 0.807), gender (p = 0.808), PI (p = 0.864) and GI (p = 0.943).

On comparing TOS at baseline, group 3 implants demonstrated significantly higher values than groups 1 and 2 (4.73±0.88 µmol H2O2 Equiv./L vs. 3.34±0.26 µmol H2O2 Equiv./L and 2.44±0.12 µmol H_2O_2 Equiv./L, respectively; p = 0.000). Also at 1 year, it was group 3 implants those with significantly higher TOS when compared with groups 1 and 2 (6.45±0.42 µmol H_2O_2 Equiv./L vs. 5.28±0.48 µmol H_2O_2 Equiv./L and 4.43±0.21 µmol H2O2 Equiv./L, respectively; p = 0.000). Additionally, on pairwise comparison, there was a statistically significant difference between group 1 and group 2 (p=0.000), group 1 and group 3 (p=0.000), group 2 and group 3 (p=0.000) at baseline and 1-year follow up (Table 3).

At baseline, the TAOC in group 3 implants was

	Group 1 (n=27)	Group 2 (n=26)	Group 3 (n=25)	p value
Age (years)	42.16±10.67	42.12±9.51	40.56±9.30	0.807
Gender (M/F)	14/13	12/14	12/13	0.808
PI	0.59±0.04	0.60±0.03	0.61±0.04	0.864
GI	0.58±0.03	0.56±0.04	0.58±0.02	0.943

Tab. 2 Demographic data of the study population

Type of implants	Baseline (3 months)	1 year
Group 1	Mean±SD: 3.34±0.26	Mean±SD: 5.28±0.48
Group 2	Mean±SD: 2.44±0.12	Mean±SD: 4.43±0.21
Group 3	Mean±SD: 4.73±0.88	Mean±SD: 6.45±0.42
ANOVA Test	p = 0.000*	p = 0.000*
Tukey's HSD post hoc test	Group 1 vs Group 2 Mean Difference: 0.901 p = 0.000*	Group 1 vs Group 2 Mean Difference: 0.849 p = 0.000*
	Group 1 vs Group 3 Mean Difference: -1.394 p = 0.000*	Group 1 vs Group 3 Mean Difference: -1.165 p = 0.000*
	Group 2 vs Group 3 Mean Difference: -2.295 p = 0.000*	Group 2 vs Group 3 Mean Difference: -2.014 p = 0.000*
*Statistically significant		

Tab. 3 Comparison of TOS between three types of implants at different time periods

Type of implants	Baseline (3 months)	1 year
Group 1	Mean±SD: 1.29±0.13	Mean±SD: 1.18±0.11
Group 2	Mean±SD: 1.84±0.12	Mean±SD: 1.56±0.06
Group 3	Mean±SD: 0.81±0.17	Mean±SD: 0.42±0.02
ANOVA Test	p = 0.000*	p = 0.000*
Tukey's HSD post hoc test	Group 1 vs Group 2 Mean Difference: -0.541 p = 0.000*	Group 1 vs Group 2 Mean Difference: -0.384 p = 0.000*
	Group 1 vs Group 3 Mean Difference: 0.472 p = 0.000*	Group 1 vs Group 3 Mean Difference: 0.762 p = 0.000*
	Group 2 vs Group 3 Mean Difference: 1.013 p = 0.000*	Group 2 vs Group 3 Mean Difference: 1.146 p = 0.000*
*Statistically significant		

Tab. 4 Comparison of TAOC between three types of implants at different time periods

Type of implants	Baseline (3 months)	1 year
Group 1	Mean±SD: 0.29±0.03	Mean±SD: 0.41±0.05
Group 2	Mean±SD: 0.15±0.01	Mean±SD: 0.23±0.04
Group 3	Mean±SD: 0.82±0.18	Mean±SD: 1.14±0.06
ANOVA Test	p = 0.000*	p = 0.000*
Tukey's HSD post hoc test	Group 1 vs Group 2 Mean Difference: 0.181 p = 0.000*	Group 1 vs Group 2 Mean Difference: 0.129 p = 0.000*
	Group 1 vs Group 3 Mean Difference: -0.412 p = 0.000*	Group 1 vs Group 3 Mean Difference: -0.855 p = 0.000*
	Group 2 vs Group 3 Mean Difference: -0.593 p = 0.000*	Group 2 vs Group 3 Mean Difference: -0.984 p = 0.000*
*Statistically significant		

Tab. 5 Comparison of OSI between three types of implants at different time periods

significantly lower than groups 1 and 2 (0.81±0.17 mmol Trolox Equiv./L vs. 1.29±0.13 mmol Trolox Equiv./L and 1.84±0.12 mmol Trolox Equiv./L, respectively; p = 0.000). Also at 1 year, it was group 3 implants those with significantly lower TAOC when compared with groups 1 and 2 (0.42±0.02 mmol Trolox Equiv./L vs. 1.18±0.11 mmol Trolox Equiv./L and 1.56±0.06 mmol Trolox Equiv./L, respectively; p = 0.000). Additionally, on pairwise comparison, there was a statistically significant difference between group 1 and group 2 (p=0.000), group 1 and group 3 (p=0.000), group 2 and group 3 (p=0.000) at baseline and 1-year follow up (Table 4).

When OSI was compared between the three groups, the OSI in group 3 implants was significantly higher than groups 1 and 2 (0.82±0.18 µmol H_2O_2 Equiv./L vs. 0.29±0.03 µmol H_2O_2 Equiv./L and 0.15±0.01 µmol H_2O_2 Equiv./L, respectively; p = 0.000). Also at 1 year, it was group 3 implants those with significantly higher OSI when compared with groups 1 and 2 (1.14±0.06 µmol H2O2 Equiv./L vs. 0.41±0.05 µmol H_2O_2 Equiv./L and 0.23±0.04 µmol H_2O_2 Equiv./L, respectively; p = 0.000). Additionally, on pairwise comparison, there was a statistically significant difference between group 1 and group 2 (p=0.000), group 1 and group 3 (p=0.000), group 2 and group 3 (p=0.000) at baseline and 1-year follow up (Table 5).

Table 6 depicts the intragroup comparison of TOS, TAOC and OSI. There was a statistically significant difference in all the parameters from baseline in all the three groups (p<0.05).

DISCUSSION

It is believed that bacterial adherence to the implant surface plays a significant role in the pathophysiology of peri-implantitis. Similar mechanisms that drive periodontitis also drive the onset and progression of peri-implantitis. Peri-implantitis is thought to be caused by microorganisms similar to those that cause periodontitis. Red complex organisms, including *Porphyromonas gingivalis*, *Tanneralla forsythia*, and

Type of implants	Baseline TOS – 1 year TOS		Baseline TAOC – 1 year TAOC		Baseline OSI – 1 year OSI	
	t	p value	t	p value	t	p value
Group 1	-19.214	0.000*	2.721	0.012*	-8.947	0.000*
Group 2	-47.566	0.000*	11.798	0.000*	-8.677	0.000*
Group 3	-19.710	0.000*	11.765	0.000*	-7.811	0.000*
*Statistically significant						

Tab. 6 Intragroup comparison of TOS, TAOC and OSI (Paired t test)

Treponema denticola, are known to be prevalent in periodontal diseases (16). The abiotic surface, which seems to alter the microbial community as pathogenic is another risk factor for peri-implantitis (17). Surface materials must therefore impede initial microbial attachment in order for implants to survive. They must be particularly equipped to combat microorganisms linked to the peri-implant disease, as indicated above (18). To reduce the incidence of peri-implantitis and promote effective osseointegration, significant advancements have been made in the surface treatment of dental implants. Research in implantology is largely directed toward improving success rates by enhancing osseointegration between the implant and the surrounding bone (19-23). Such efforts aim to optimize bone–implant integration, which forms the cornerstone of long-term stability and functional success (24,25). However, the ideal implant surface must strike a delicate balance between providing adequate osteoconductive properties and exhibiting antibacterial activity (26). Achieving this balance remains challenging, as increasing surface roughness can enhance bone formation but may also promote biofilm development.

The dental implants considered in the present study were SLA, SLActive and TiUnite, all the three implants differ in terms of surface modification. The microbial profile of implants is greatly influenced by the surface topography and chemical makeup of the implant, so it is possible that topographical differences between various implant surfaces will have an impact on the microbial profile (27), which can further alter the redox potential and affects the balance between oxidants and antioxidants. Research supports the idea that oxidative stress is the primary cause of a number of clinical diseases (28). Oxidative stress can inhibit the osteoblast cell proliferation, which is essential for successful osseointegration (29). Therefore, exploring the oxidant status, antioxidant status and oxidative stress around different surface treated dental implants is essential to improve the performance of implants.

Currently, the three dental implants that are most commonly utilised in clinical settings are SLA, SLActive, and TiUnite. Despite the fact that the three dental implants have different surface characteristics, it is unclear which implant surface least likely alters the redox potential. This is the first study of its kind to assess the total antioxidant capacity (TAOC), total oxidant status (TOS) and oxidative stress index (OSI) in peri-implant crevicular fluid among patients with SLA, SLActive and TiUnite dental implants.

In literature, TOS and TAOC were assessed in periodontitis and peri-implantitis. Patients with periodontitis had higher levels of TOS and lower levels of TAOC in their serum and saliva, according to research by Baltacioglu E et al. (30). Additionally, it was claimed that there was a substantial association between TOS and TAOC and periodontal markers, indicating an aggravation of the inflammatory process (31). Also, total salivary antioxidant levels and clinical periodontal parameters were found to be correlated by Novakovic N et al., who assessed the oral cavity's total antioxidant activity. They illustrated the relevance of measuring TAOC in saliva for predicting the prognosis of periodontal therapy (32).

Furthermore, Drafta S et al., found a statistically significant negative correlation between TAOC and bone loss, indicating TAOC may eventually become a risk factor for peri-implant bone loss (33). Similar to this, it was observed that peri-implantitis patients had greater levels of salivary oxidative stress indicators than healthy individuals (34). According to Liskmann S et al., increased production of reactive oxygen species in peri-implant disease creates an environment of excessive oxidative stress, which may play a significant role in the deterioration of peri-implant tissues (35).

The present study revealed that TOS, TAOC and OSI activities were within the normal range, however, TOS and OSI levels were high and TAOC activity was less comparatively around TiUnite dental implants followed by SLA and SLActive implants. The surface characteristics of the implants might be accountable for this variation. According to Albouy JP et al., implants with a TiUnite surface demonstrated a faster rate of disease progression than implants with a SLA surface. Moreover, peri-implantitis progressed more significantly and had a worse treatment outcome around implants with a TiUnite surface than around implants with a SLA surface (36). Furthermore, histological assessment revealed that osseointegration was more around SLA implants than around TiUnite implants (37). Although it has been demonstrated

that increased roughness promotes bone to implant contact, it also affects bacterial adhesion and biofilm formation (38). Both in vitro and in vivo studies have demonstrated a relationship between implant surface roughness and the propensity for bacterial adhesion (39,40).The surface of TiUnite have grooves and pits that shield bacteria from shear forces and promote persistent adherence (41). As a result, the TiUnite surface may provide a suitable atmosphere for bacterial adherence, which might create an inflammatory milieu, resulting in oxidative stress.

Also, it was observed in the present study that TOS and OSI were significantly low around SLActive surfaces. Surface study has shown that the SLActive surface being hydrophilic, prevents the hydrophobic organisms from attaching to its surface. Additionally, it was discovered that the hydrophilic substrates considerably have reduced amount of bacterial adhesion (42). According to an in vitro study, *P. gingivalis* demonstrate hydrophobic activity and are less attracted to hydrophilic surfaces (43). On hydrophilic surfaces in the culture media, *A. actinomycetemcomitans* and *F. nucleatum* also exhibit lower levels (44). This surface characteristic aids in reducing the adhesion of pathogenic bacteria to the SLActive surface, hence affecting the environment in the least possible way.

Collectively, the present study supports that dental implants' surface treatments have an impact on the tissue around them. Surface features that may impact bacterial adherence and generate a homeostatic imbalance are one possible explanation for the variation in TOS, TAOC and OSI activities among different implant systems. Eventhough the difference in oxidant and antioxidant activities are being negligible, it might affect the osseointegration thereby hamper the long-term success of the dental implants. Further studies are warranted to assess the influence of structural characteristics of dental implants on the microbiological and immunological pathways to substantiate these findings.

In summary, implants with different surface treatments might affect the redox balance, leading to reduction in total antioxidant status and increase in total oxidant capacity and oxidative stress. Quantification of these levels periodically might help in predicting peri-implant risk, which in turn helps to initiate early therapeutic intervention.

CONCLUSION

Higher total oxidative capacity and oxidative stress index as well as lower antioxidative activity were observed in peri-implant crevicular fluid around TiUnite dental implants as compared to SLA and SLActive implants.

Contribution statement

Both authors have made substantial contributions to conception and design of the study. PJ has been involved in data collection, data analysis. PJ and AR have been involved in data interpretation, drafting the manuscript and revising it critically and have given final approval of the version to be published.

Conflict of interest statement

The authors have no conflicts of interest to declare.

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