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Salivary pH and oxidative stress levels among peri-implantitis patients

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Abstract:
Dental implants are a dependable treatment for tooth replacement, but peri-implant infections can compromise long-term treatment success. This comparative study evaluated 60 patients, equally divided into peri-implant health, peri-implant mucositis, and peri-implantitis groups, by assessing clinical peri-implant parameters, salivary pH, malondialdehyde, and total antioxidant capacity. Patients with peri-implantitis showed higher plaque index, bleeding on probing, probing depth, marginal bone loss, and malondialdehyde levels, with lower salivary pH and total antioxidant capacity. These findings indicate an association between peri-implant disease and altered salivary pH and oxidative stress status. Thus, data shows that salivary pH and oxidative stress markers as potential adjunctive tools for early assessment of peri-implant disease.

Keywords: Dental implants, peri-implantitis, peri-implant mucositis, salivary pH, oxidative stress, malondialdehyde, total antioxidant capacity

Background:
Dental implants are widely utilised to replace missing teeth and have showed substantial long-term efficacy. Nonetheless, biological problems associated with implants are increasingly emerging as a prevalent clinical issue. Peri-implant health denotes an implant location devoid of clinical inflammation, whereas peri-implant mucositis is characterised mostly by soft tissue inflammation. Peri-implantitis is a more severe disorder characterised by inflammation and progressive loss of supporting bone surrounding the implant **[1, 2].** These diseases are affected by plaque buildup, inadequate oral hygiene, tobacco use, systemic disorders, a history of periodontal disease, and inconsistent maintenance care **[3, 4]**. The early identification of peri-implant disease is crucial, as clinical manifestations may not appear until tissue inflammation has advanced. Traditional clinical and radiological assessments are crucial; nevertheless, there is growing interest in simple chairside indicators to facilitate early diagnosis. Saliva is a valuable diagnostic fluid due to its ease of collection, reproducibility, and noninvasive nature for the patient. It signifies changes in the oral environment and includes numerous molecular markers associated with inflammation, tissue degradation, and microbial activity **[5, 6]**. Salivary pH is an additional metric to consider. A reduction in salivary pH may facilitate bacterial growth, plaque formation, and an inflammatory oral environment. Previous studies suggest that salivary pH may vary with the severity of periodontal disease, indicating its possible use as an additional diagnostic tool **[7]**. Oxidative stress is a crucial physiological process in peri-implant disease. During inflammation, host cells release reactive oxygen species. When oxidants surpass the capability of antioxidant defenses, they can induce deterioration of connective tissue and loss of bone density **[8]**. Recent assessments have highlighted the potential importance of salivary oxidative stress markers in differentiating between peri-implant health and disease **[9, 10].** Thus, assessing salivary pH in conjunction with oxidative stress levels may provide a simple, non-invasive approach to comprehending the biological changes linked to peri-implant health and disease. Therefore, it is of interest to evaluate salivary parameters in individuals with peri-implant health and peri-implant disease.

Methodology:
This research was executed as an observational, analytical, cross-sectional study within the Department of Periodontology. Patients attending for implant follow-up, maintenance, or routine periodontal assessment were screened for the study. The research was conducted over six months following approval from the Institutional Ethics Committee.

Sample size:
The sample size was determined utilising G*Power software version 3.1. One-way ANOVA was used for sample size estimation because the study compared three groups. The alpha error was established at 0.05, the power at 80%, and the confidence level at 95%. The minimum requisite sample size was 60 individuals based on these characteristics. Hence, 20 patients were incorporated into each group. Sixty patients were enrolled and categorised into three groups: Group I (n=20) comprised patients with excellent or good peri-implant health, Group II (n=20) included those with peri-implant mucositis, and Group III (n=20) consisted of patients with peri-implantitis.

Eligibility criteria:
Individuals aged 25 to 65 years were incorporated into the study. Patients with at least one dental implant functioning for 12 months or longer were selected. Participants who consented to provide unstimulated saliva samples were included. Patients who had not undergone periodontal or peri-implant treatment in the preceding three months were also included. Individuals with smoking or tobacco chewing habits were excluded from the study. Pregnant and breastfeeding women were excluded. Individuals with uncontrolled diabetic mellitus, autoimmune disorders, malignancies, hepatic disease, renal disease, or any significant systemic illness were excluded. Patients who had consumed antibiotics, anti-inflammatory medications, steroids, or antioxidant supplements within the preceding three months were also excluded. Individuals exhibiting active generalised periodontitis surrounding natural teeth were excluded.

Clinical examination:
A comprehensive case history was documented for each subject. Age, gender, number of implants, implantation site, time since implantation, and type of prosthetic implant were recorded. A clinical examination was conducted utilizing a mouth mirror and periodontal probe. Plaque accumulation, bleeding upon probing, suppuration, peri-implant probing depth, and keratinised mucosa breadth were documented. The peri-implant probing depth was assessed at six locations surrounding each implant: mesiobuccal, mid-buccal, distobuccal, mesiolingual or mesiopalatal, mid-lingual or mid-palatal, and distolingual or distopalatal. A gentle probing force was used to avoid injury to the peri-implant tissues.

Diagnostic criteria:
Peri-implant health was determined by the lack of erythema, oedema, haemorrhage upon probing, suppuration, increased probing depth, and radiographic bone loss above typical initial bone remodelling. Peri-implant mucositis is diagnosed when bleeding occurs upon probing, with or without accompanying redness and swelling, although without radiographic evidence of bone loss exceeding typical early remodelling. Peri-implantitis is diagnosed when there is bleeding upon probing and/or suppuration, increasing peri-implant probing depth, and radiographic evidence of bone loss surrounding the implant.

Radiographic assessment:
Intraoral periapical radiographs were acquired utilising the paralleling approach. Marginal bone loss was quantified from the implant shoulder to the initial bone-to-implant contact on both the mesial and distal aspects. The average of the mesial and distal measures was documented as the marginal bone loss value.

Saliva collection*:*
Unstimulated whole saliva was collected from all patients in the morning, namely between 9:00 AM and 11:00 AM. Patients were instructed to refrain from all food and beverages, with the exception of water, for at least 2 hours before to sample collection. They were directed to abstain from brushing, rinsing, chewing gum, or using mouthwash on the morning of saliva collection. Before collection, patients meticulously washed their mouths with tap water and waited for 10 minutes. Saliva was collected before periodontal probing to prevent blood contamination. The patient was positioned comfortably with the head slightly tilted forward. Saliva was allowed to gather in the sublingual area and then collected into a sterile container utilizing the passive drooling method. Approximately 2-5 cc of saliva was collected from each participant.

Assessment of salivary pH and oxidative stress parameters:
The salivary pH was measured immediately after sample collection using a digital pH meter. The pH meter was calibrated before use using standard buffer solutions. The electrode was put into the saliva sample, and the measurement was recorded once the value stabilized. The saliva samples were centrifuged at 3000 rpm for 10 minutes. The transparent supernatant was extracted and stored in labeled Eppendorf tubes. The samples were preserved at low temperatures until biochemical analysis was performed. Oxidative stress was assessed by quantifying salivary malondialdehyde levels and total antioxidant capacity. Malondialdehyde functioned as a biomarker for lipid peroxidation. Total antioxidant capacity functioned as a measure of antioxidant defense. The analysis was performed using known biochemical techniques or commercially available kits following the manufacturer's instructions. The primary outcome variables comprised salivary pH, salivary malondialdehyde content, and salivary total antioxidant capacity. The secondary outcome variables encompassed plaque score, bleeding on probing, peri-implant probing depth, suppuration, marginal bone loss, and width of keratinized mucosa.

Statistical assessment:
The collected data was analyzed utilizing SPSS. Continuous variables were represented as mean and standard deviation. Categorical variables were expressed as frequencies and percentages. The data's normality was evaluated utilizing the Shapiro–Wilk test. One-way ANOVA was utilized to analyze normally distributed variables among the three groups. Tukey's post hoc test was utilized for pairwise comparisons. The Kruskal–Wallis test was utilized for data that deviated from a normal distribution. The Chi-square test was utilized for categorical variables. Correlations between salivary metrics and clinical peri-implant characteristics were assessed using Pearson correlation coefficients. A p-value under 0.05 was considered statistically significant.

Results:
Sixty patients were equally distributed among Group I (peri-implant health), Group II (peri-implant mucositis), and Group III (peri-implantitis), with 20 patients in each group. The groups were comparable regarding age (p=0.690), gender distribution (p=0.825), and duration of implant function (p=0.196). As shown in **Table 1**, plaque index increased progressively from Group I (0.68±0.21) to Group II (1.28±0.32) and Group III (1.82±0.39;

p<0.001). Bleeding on probing was 5.50±4.12%, 42.35±12.24%, and 68.10±15.45%, respectively (p<0.001). Peri-implant probing depth increased from 2.42±0.35 mm in Group I to 3.38±0.52 mm in Group II and 6.28±0.81 mm in Group III (p<0.001). Marginal bone loss was also significantly greater in Group III (3.72±0.84 mm) than in Group II (0.84±0.31 mm) and Group I (0.62±0.28 mm; p<0.001). The width of keratinised mucosa decreased progressively across Groups I, II, and III (3.10±0.76, 2.56±0.68, and 1.94±0.61 mm, respectively; p<0.001). Suppuration was absent in Groups I and II but was observed in six patients (30.0%) in Group III (p=0.001). As presented in **Table 2**, salivary pH decreased significantly from Group I (7.18±0.29) to Group II (6.72±0.34) and Group III (6.28±0.41; p<0.001). Salivary malondialdehyde levels increased progressively across the three groups (2.86±0.64, 4.92±0.88, and 7.38±1.21 nmol/ml, respectively; p<0.001). Conversely, total antioxidant capacity decreased from 1.42±0.26 mmol/L in Group I to 1.04±0.22 mmol/L in Group II and 0.71±0.18 mmol/L in Group III (p<0.001). Post-hoc comparisons demonstrated the following trends: salivary pH and total antioxidant capacity: Group I > Group II > Group III; malondialdehyde: Group III > Group II > Group I. Overall, peri-implantitis showed the most severe clinical deterioration, greater oxidative stress, lower salivary pH, and reduced antioxidant capacity.

Table 1: Comparison of demographic and clinical peri-implant parameters among the study groups

Variable	Group I: Peri-implant health n=20	Group II: Peri-implant mucositis n=20	Group III: Peri-implantitis n=20	p value
Age in years, Mean ± SD	45.20 ± 8.11	46.05 ± 7.60	47.35 ± 8.42	0.690
Male, n (%)	11 (55.0)	10 (50.0)	12 (60.0)	0.825
Female, n (%)	9 (45.0)	10 (50.0)	8 (40.0)	0.825
Duration of implant in function in years, Mean ± SD	3.10 ± 1.12	3.45 ± 1.28	3.85 ± 1.36	0.196
Plaque index, Mean ± SD	0.68 ± 0.21	1.28 ± 0.32	1.82 ± 0.39	<0.001
Bleeding on probing in %, Mean ± SD	5.50 ± 4.12	42.35 ± 12.24	68.10 ± 15.45	<0.001
Peri-implant probing depth in mm, Mean ± SD	2.42 ± 0.35	3.38 ± 0.52	6.28 ± 0.81	<0.001
Marginal bone loss in mm, Mean ± SD	0.62 ± 0.28	0.84 ± 0.31	3.72 ± 0.84	<0.001
Width of keratinised mucosa in mm, Mean ± SD	3.10 ± 0.76	2.56 ± 0.68	1.94 ± 0.61	<0.001
Suppuration present	0 (0.0)	0 (0.0)	6 (30.0)	0.001

Table 2: Comparison of salivary pH and oxidative stress levels among the study groups

Salivary parameter	Group I: Peri-implant health n=20	Group II: Peri-implant mucositis n=20	Group III: Peri-implantitis n=20	p value	Post-hoc interpretation
Salivary pH	7.18 ± 0.29	6.72 ± 0.34	6.28 ± 0.41	<0.001	Group I > Group II > Group III
Malondialdehyde in nmol/ml	2.86 ± 0.64	4.92 ± 0.88	7.38 ± 1.21	<0.001	Group III > Group II > Group I
Total antioxidant capacity in mmol/L	1.42 ± 0.26	1.04 ± 0.22	0.71 ± 0.18	<0.001	Group I > Group II > Group III

Discussion:

This study assessed salivary pH and oxidative stress levels in individuals with peri-implant health, peri-implant mucositis, and peri-implantitis. The findings revealed a clear decline from health to illness. Patients with peri-implantitis had increased plaque scores, heightened bleeding on probing, greater probing depths, and more severe radiographic bone loss. Alongside these clinical changes, salivary pH decreased, and oxidative stress increased in the illness group. This suggests that peri-implant disease is not merely a localized inflammatory condition around the implant, but may also present as measurable changes in saliva. The study revealed higher malondialdehyde levels in patients with peri-implantitis. Malondialdehyde serves as a prevalent marker for lipid peroxidation and oxidative tissue injury. This discovery is consistent with Karasu *et al.*, who reported increased oxidative damage biomarkers in the saliva of individuals with peri-implant problems **[11]**. Sánchez-Siles *et al.* also reported altered salivary oxidative stress markers in individuals with peri-implantitis **[12]**. The data support the idea that peri-implant inflammation is associated with an imbalance between free radical production and antioxidant defence systems. The peri-implantitis group had reduced overall antioxidant capacity compared with the healthy implant group. This may suggest that the antioxidant defence system is diminished as inflammation progresses. Mousavi Jazi *et al.* examined oxidative stress markers in peri-implant crevicular fluid and identified associations between these markers and clinical peri-implant features **[13]**. The present study demonstrated an inverse relationship between total antioxidant capacity and both probing depth and bleeding on probing. This indicates that reduced antioxidant defense may be associated with heightened clinical inflammation severity. The gradual progression from peri-implant health to peri-implant mucositis and peri-implantitis is noteworthy. Peri-implant mucositis demonstrated values that are between those of health and peri-implantitis. This suggests that oxidative stress may begin before substantial bone loss. Liskmann *et al.* discerned differences in the antioxidant profile of saliva between peri-implant health and disease **[14]**. Thus, salivary oxidative stress markers may facilitate the early detection of biological changes surrounding implants, preceding the clinical onset of substantial tissue deterioration. The present study revealed that salivary pH was lowest in the peri-implantitis group. A reduction in pH may promote conditions favorable for pathogenic bacterial growth

and reduce the natural buffering ability of saliva. Karpavicius *et al.* examined pH values in peri-implant crevicular fluid and established that the pH of this fluid can vary around implants **[15]**. Notwithstanding disparities between their work and the present study, the current research supports the idea that pH variations may be linked to the peri-implant environment. Lăzureanu *et al.* further indicated that diminished salivary pH and decreased salivary flow correlated with more severe periodontal disease **[16]**. Given that peri-implantitis and periodontitis exhibit distinct inflammatory and microbial characteristics, similar pH changes around implants are biologically plausible. The increased malondialdehyde levels in peri-implantitis may be due to enhanced neutrophil activity and persistent bacterial invasion. In the course of inflammation, immune cells produce reactive oxygen species as a component of host defense. Overproduction of these reactive compounds can harm lipids, proteins, and extracellular matrix components. Akalin *et al.* demonstrated increased lipid peroxidation and oxidant concentrations in the saliva and gingival crevicular fluid of patients with chronic periodontitis **[17]**. The findings of the present study align with this process and suggest that oxidative damage may contribute to the degeneration of peri-implant tissue. The reduction in overall antioxidant capacity merits attention. Saliva contains several antioxidants that protect oral tissues from oxidative damage. In the context of chronic inflammation, these antioxidants may be consumed at an accelerated rate. Brock *et al.* established that antioxidant capacity differs between periodontal health and disease **[18]**. The present investigation indicates that reduced antioxidant capacity in peri-implantitis may signify an insufficient salivary protective response to the inflammatory burden surrounding compromised implants. The correlation analysis substantiates the conclusions. This study identified a positive correlation between malondialdehyde and probing depth, bleeding on probing, and radiographic bone loss. This indicates that as the severity of clinical disease increased, oxidative damage increased as well. Total antioxidant capacity and salivary pH demonstrated adverse correlations with disease severity. Ahmadi-Motamayel *et al.* reported alterations in oxidative stress levels in saliva and serum in chronic periodontitis **[19]**. Baltacioglu *et al.* suggested that the oxidant–antioxidant balance may function as a diagnostic indicator for periodontal disease **[20]**. These results support the prevailing concept that oxidative stress markers may signify inflammatory tissue damage. This study emphasises the prospective value of saliva as a simple diagnostic fluid. Saliva collection is straightforward, painless, and non-invasive. It can be reiterated during maintenance visits without causing discomfort to the patient. The study included specific limitations. This cross-sectional study can indicate a correlation but cannot determine causation. Salivary pH and oxidative stress markers may be influenced by nutrition, hydration, systemic diseases, medications, tobacco consumption, dental hygiene, and the timing of sample collection. Only certain biomarkers were assessed. Future studies should include larger sample sizes, longitudinal studies, microbiological analyses, and additional inflammatory markers, such as interleukins, matrix metalloproteinases, and biomarkers from peri-implant crevicular fluid.

Conclusion:

We show that patients with peri-implant disease displayed reduced salivary pH, elevated malondialdehyde levels, and diminished overall antioxidant capacity in comparison to patients with peri-implant health. Salivary changes were more significant in peri-implantitis and linked with worsened clinical indicators, including increased plaque deposition, bleeding on probing, probing depth, and marginal bone loss. Salivary pH and oxidative stress markers may function as significant supplementary indicators for evaluating peri-implant inflammation and disease severity.

References:

[1] Berglundh T *et al. J Clin Periodontol.* 2018 **20**:S286. [PMID: 29926491]
[2] Schwarz F *et al. J Periodontol*. 2018 **1**:S267. [PMID: 29926957]
[3] Derks J &Tomasi C. *J Clin Periodontol.* 2015 **16**:S158. [PMID: 25495683]
[4] Salvi GE *et al. J Dent Res.* 2017 **96**:31. [PMID: 27680028]
[5] Gomes AM *et al. Arch Oral Biol.* 2018 **96**:216. [PMID: 30300769]
[6] Lumbikananda S *et al. Dent J.* 2024 **74**:387. [PMID: 38065782]
[7] Baliga S. *J Indian Soc Periodontol.* 2013 **17**:461.[PMID: 24174725]
[8] Chapple IL & Matthews JB. *Periodontol* 2000. 2007 **43**:160. [PMID: 17214840]
[9] Tóthová *L et al. Front Cell Infect Microbiol*. 2015 **5**:73. [PMID: 26539412]
[10] Popa PȘ *et al. Int J Mol Sci*. 2025 **26**:11269 [PMID: 41373430]
[11] Özkan Karasu Y *et al. Int J Implant Dent*. 2024 **10**:43. [PMID: 39400614]
[12] Sánchez-Siles M *et al. Clin Implant Dent Relat Res.* 2016 **18**:1015 [PMID: 26230313]
[13] Mousavi Jazi M *et al. J Dent (Tehran).* 2015 **12**:340. [PMID: 26877729]
[14] Liskmann S *et al. Clin Oral Implants Res*. 2007 **18**:27. [PMID: 17224020]
[15] Karpavicius D *et al. Clin Exp Dent Res*. 2019 **5**:236. [PMID: 31249704]
[16] Lăzureanu PC *et al. Med Sci Monit.* 2021 **27**:e931362. [PMID: 34305133]
[17] Akalin FA *et al. J Clin Period.ontol*. 2007 **34**:558. [PMID: 17555410]
[18] Brock GR *et al. J Clin Periodontol*. 2004 **31**:515. [PMID: 15191586]
[19] Ahmadi-Motamayel F *et al. Front Physiol*. 2017 **8**:189. [PMID: 28408887]
[20] Baltacıoğlu E *et al. J Periodontol.* 2014 **85**:1432. [PMID: 24635543]

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