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# Teeth And Implant Surroundings: Clinical Health Indices And Microbiologic Parameters

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## Abstract

***Background****. Dental implants are a predictable and well-established treatment method in dentistry. However, when considering potential dental implant failures, a distinction must be made between early and late loss. The aim of this study was to investigate the role of implant surface debridement alone and in conjunction with systemic antibiotics on clinical and microbiological variables of peri-implantitis****. Materials and methods****. Fifty-two patients who underwent at least one dental implant with bleeding on the probe (BoP), probe pocket depth (PPD) of more than 5 mm, and radiographic bone loss of more than 3 mm, were retrieved from clinical records. Data on dental implants with the deepest PPD, BoP, and bone loss from each patient wer[1]e recorded. "Group-A" received implant surface debridement alone, while "Group-B" additionally received systemic antibiotics. Clinical and microbiological data of patients before and after treatment were compared.* ***results****. At the transplantation level, a significant reduction in PPD, mucostasis (MR), and BoP was achieved for all patients. Group B achieved significant improvement in MR and BoP compared to Group A at the implant level. PPD, MR, and panel results showed improvement at the implant site level. At the 3-month recall visit, 44% of Group A implants and 52% of Group B implants required surgical treatment. The presence and proportions of the studied bacteria in both groups did not differ significantly at the recall visit compared to the initial visit. However, P. intermedia and P. micros significantly decreased in group A at the recall visit.* ***Conclusions****. Implant surface debridement improved clinical indicators of peri-implantitis. In addition, adjunctive use of systemic antibiotics increased mucosal stasis and improved bleeding when examining peri-implantitis.*

## Introduction

The insertion of dental implants has become a routine and well predictable surgical procedure in the last decades, with high rates of osseointegration and long-term success [1]. Since the success of early implantology, this field has nowadays established itself in the daily treatment.

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There are many causes of tooth loss. Frequent problems are the development of caries at the margins of crowns, gingivitis, pulp infections and mechanical complications, such as tooth fractures [2,3,]. While the first two factors do not affect dental implants and rates of mechanical complications of the implant itself are low, peri-implant infections are a major risk factor for implant failure [6].

The etiology of peri-implantitis is multifactorial in nature. However, bacteria play a vital role in the initiation and progression of the disease [5]. Significant differences in the microbiota associated with diseased dental implants compared to healthy dental implants have been reported [6, 7]. In contrast to healthy implants that contain mainly a biofilm composed of Gram-positive cocci [6, 8], the biofilm associated with peri-implantitis is characterized by a predominance of anaerobic bacteria. Prevotella intermedia/nigrescens, Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans are the most common bacteria associated with peri-implantitis [9, 10]. Multiple similarities can be drawn between peri-implantitis and gingivitis including similar bacterial species associated with both diseases [4]. However, unique microbial species for peri-implantitis have also been reported in the medical literature.

Studies on the treatment outcomes of dental peri-implantitis are scarce, and the evidence of a single effective treatment method for dental peri-implantitis is inconclusive [5]. It has been reported that antibiotics combined with implant surface cleaning/debridement improve clinical and microbiological parameters in peri-implantitis [8].

In the past decades, knowledge of bacterial infections, especially biofilms, has changed fundamentally [5,6]. While the hypothesis that the pathogens of periodontitis and peri-implantitis are similar has been proposed in several previous studies based on genetic analysis methods [7] it is at the same time well established that there are certain differences regarding the microbiota in These diseases using new methods. and more accurate diagnostic methods [8,9].

Therefore, we aimed to study the role of implant surface debridement alone and in conjunction with systemic antibiotics on the clinical and bacteriological variables of peri-implantitis.

## 2. Materials and Methods

In this retrospective study, the private patient database at King Abdulaziz University Dental Hospital, Saudi Arabia was searched for peri-implantitis patients based on the following criteria as suggested by Renvert et al. [10].

In this retrospective study, the private patient database at King Abdulaziz University Dental Hospital, Saudi Arabia was searched for peri-implantitis patients based on the following criteria as suggested by Renvert et al. [10].

1) Pocket depths (PPD) greater than 5 mm
2) Bleeding/suppuration on probing (BoP)
3) Radial bone loss of more than 3 mm

**General inclusion criteria were:**

1) Patients over 18 years of age
2) Patients who had at least one titanium dental implant and were diagnosed with peri-implantitis
3) Dental implants must be in use for a minimum of one year or more

**Patients were excluded from the study if:**

1) Patients with diabetes and other chronic systemic diseases were excluded
2) NSAIDs have been used in the past 4 weeks
3) Systemic antibiotics were used in the 3 months prior to treatment

**Data Collection**

The following data were obtained at the initial visit (before treatment began): age (in years), sex, presence of chronic systemic disease, dental status, the number of remaining teeth),

history of current or past smoking, and history of gingivitis. The following clinical measurements were obtained for all teeth/implants present at the initial visits and at the recall visits (3 months after the first visit): plaque scores (measured by the modified plaque index proposed by Van der Weijden et al. [11], Bleeding/ suppuration on examination, PPD in mm, Clinical attachment level (CAL).
Data regarding the use and type of systemic antibiotics during treatment of peri-implantitis were retrieved from a patient database. Data from patients who received a standard antibiotic regimen (amoxicillin 500 mg three times daily plus metronidazole 400 mg twice daily for 5 days) were selected for the study. Submucosal plaque samples were previously obtained using sterile paper dots from the deepest implant sites at the initial visit as well as the 3-month reminder visit. Microbiological data were obtained from laboratory records.

The data were fully anonymized by assigning a serial number to each record, and ethical approval was obtained from the Institutional Ethical Committee of King Abdulaziz Hospital, Saudi Arabia.

**Initial visit**
The data of all peri-implantitis patients referred to King Abdulaziz Hospital for treatment of peri-implantitis were evaluated. Previous medical and dental history were recorded at the initial visit. Patients were divided into two groups:
- Group (A) received implant surface debridement combined with a standard antibiotic regimen (amoxicillin 500 mg three times daily plus metronidazole 400 mg twice daily for 5 days).
- Group (B) patients received only implant surface debridement without the use of systemic antibiotics.

**Microbiological analysis**
Sterile paper dots were used to obtain a submucosal plaque from the peri-implant sinus with the largest PPD measurement [12]. Next, the paper dots were transferred into sterile 5 ml tubes with reduced standard transfer fluid (dithiothreitol-balanced mineral salt solution) [13]. Within two hours of collecting samples for microbiological culture. Selected bacterial species were also cultured anaerobically according to standard methods [14] Serial dilutions of previously obtained submucosal plaque samples were cultured on 5% horse blood agar plates supplemented with hemin (5 mg/L). and menadione (1 mg/L). Trypticase-soy serum-bacitracin-vancomycin (TSBV) plates were used as a culture medium for the growth of A. actinomycetemcomitans. Incubation of blood agar plates was performed in an anaerobic environment (80% N2, 10% H2, and 10% CO2) at 37°C. TSBV plates were incubated and carried in 5% CO for up to 2 weeks. Bacterial colonies were counted three times on agar plates using a magnifying glass, and the average was taken to calculate colony forming units per ml (CFU/ml). The presence and relative proportions of target bacteria were noted. Colony morphology, Gram stain, microscopy, anaerobic growth, glucose fermentation, and indole were used to identify bacterial species.

**Implant surface debridement**
Before starting nonsurgical treatment, patients were given a commercially available 0.12% chlorhexidine mouthwash to rinse for 1 minute. Local anesthesia was administered to the affected implant (2% medication, 1: 100,000 epinephrine). Patients suffering from gingivitis or gingivitis were also treated. A generic mouthwash containing 0.12% chlorhexidine was prescribed, and patients were instructed to use it three times daily for 30 days [15]. Standard oral hygiene instructions (OHI) were given to all patients.

**Recall Visit**

After 3 months from the date of the initial medical visit, patients were examined again by the same physician (MI), and clinical measurements were recorded. Patients were referred for peri-transplant surgery if necessary.

**statistical analysis**

GraphPad Prism software (version 5.00 for Windows) was used to analyze the data. To compare continuous and categorical variables, Wilcoxon signed ranks and McNemar tests were used, respectively. Differences were considered significant with a value of ≥5.

**Results**

Table (1) presents the general features of the patients included in the study. Fifty-two (52) patients, aged 53 to 72 years, were included in the study. The number of participants was 37 males and 15 females. Group A included 28 patients who received a standard regimen of systemic antibiotics as previously reported in the Materials and Methods section, while 24 patients received implant surface debridement alone.

| Age (mean ± SD) | 53-72 (56.5±15) | |
|---|---|---|
| Gender | Male | 37 (74) |
| | Female | 15 (26) |
| Dental status | Edentulous | 17 (30) |
| | Dentate | 32 (73) |
| Smoking habits | Smoker | 6 (8) |
| | Nonsmoker | 33 (67) |
| | Past-smoker | 7 (11) |
| Past history of periodontitis | Not known | 7 (13) |
| | Yes | 19 (37) |
| | No | 25 (52) |
| | Unknown | 7 (11) |

**Clinical Parameters at the Target Implant Site**

Table (2) provides a comparison of clinical parameters between initial and recall visits. The clinical parameters studied did not differ significantly between the two groups at the initial visit. PPD of target sites decreased significantly (ρ>0.001) in both groups at the recall visit compared to the initial visit. The mean PPD of group B was significantly lower than that of group A (ρ=0.003), when the two groups were compared at the recall visit. CAL measurements changed significantly only in group B (ρ=0.002), while they were not significant in group A (ρ=0.12). For both groups, MR values were significantly higher at the recall visit than at the initial visit (Group B, ρ=0.002; Group, A ρ=0.01). In addition, mean MR values were significantly greater in group A (ρ=0.005), compared to group B at the recall visit. A significant decrease in BOP was also observed for both groups at the recall visit compared to the initial visit (Group B, ρ=0.003; Group A, ρ=0.011). The deeper peri-implant pockets showed the greatest changes in PPD and MR in both groups.

| | Initial visit | Recall visit | value initial vs. recall visit | value evaluation group B vs. group A |
|---|---|---|---|---|
| A. Target implant site | | | | |
| PPD (mm±SD) | | | | |
| Total | 7.1 (1.6) | 5.4 (1.3) | <0.001 | 0.003 |
| Group B | 7.4 (1.7) | 4.9 (1.2) | <0.001 | |
| Group A | 7.3 (1.2) | 5.6 (1.5) | <0.001 | |
| CAL (mm±SD) | | | | |

| | | | | |
|---|---|---|---|---|
| Total | 11.7 (2.2) | 10.2 (1.3) | 0.001 | 0.3 |
| Group B | 12.3 (1.9) | 10.1 (1.3) | 0.003 | |
| Group A | 11.2 (1.6) | 10.9 (1.6) | 0.12 | |
| BoP (mm±SD) | | | | |
| Total | 100 | 87 | 0.004 | 0.4 |
| Group B | 100 | 90 | 0.03 | |
| Group A | 100 | 93 | 0.011 | |
| Suppuration on probing (mm±SD) | | | | |
| Total | 24 | 7 | 0.20 | 0.2 |
| Group B | 26 | 6 | 0.09 | |
| Group A | 20 | 8 | 0.33 | |
| B. Target implant | | | | |
| PPD (mm±SD) | | | | |
| Total | 5.1 (1.3) | 4.7 (1.2) | <0.001 | 0.07 |
| Group B | 5.1 (0.8) | 4.1 (0.7) | 0.003 | |
| Group A | 5.3 (1.7) | 4. (1.1) | 0.04 | |
| CAL (mm±SD) | | | | |
| Total | 11.3 (2.1) | 10.5 (2.4) | 0.34 | 0.32 |
| Group B | 12.5 (2.6) | 10.3 (2.1) | 0.2 | |
| Group A | 9.6 (2.3) | 9.7 (2.2) | 0.6 | |
| BoP (mm±SD) | | | | |
| Total | 5.1 (1.2) | 3.7 (1.9) | <0.001 | 0.02 |
| Group B | 5.3 (1.3) | 3.3 (1.7) | 0.001 | |
| Group A | 5.1 (1.3) | 4.3 (1.7) | 0.08 | |
| Suppuration on probing (mm±SD) | | | | |
| Total | 1.1 (1.2) | 0.4 (1.4) | 0.05 | 0.8 |
| Group B | 0.7 (1.1) | 0.3 (1.7) | 0.23 | |
| Group A | 0.9 (1.8) | 0.3 (1.1) | 0.17 | |

**Microbiological Parameters**

Table 3 presents the microbiological data of the cultures. The differences between the mean proportions and prevalence of bacterial species studied for the two groups at the first visit were not significant. Likewise, group A did not show significant changes in the prevalence or proportions of bacterial species between initial and recall visits. Interestingly, the prevalence of P. intermedia and P. micros in group A was significantly lower at the recall visit ($\rho$=0.002) and, ($\rho$=0.001) respectively) compared to the initial visit. Moreover, a decrease in the proportions of P. intermedia ($\rho$=0.04) was observed in group A at the recall visit.

| | | Group B | Group A | Recall visit group B vs. |
|---|---|---|---|---|

| | | | | | | | | group A |
|---|---|---|---|---|---|---|---|---|
| | | Initial visit | Recall visit | | Initial visit | Recall visit | | |
| A. actinomycetemcomitans | Prevalence N (%) | 0 (0) | 0 (0) | NS | 0 (0) | 0 (0) | NS | 0 (0) |
| | Mean (±SD) proportion | 0 (0) | 0 (0) | NS | 0 (0) | 0 (0) | NS | 0 (0) |
| P. intermedia | Prevalence N (%) | 9 (36) | 6 (24) | NS | 7 (33) | 5 (24) | NS | NS |
| | Mean (±SD) proportion | 2.3 (4.4) | 3. (5.1) | NS | 1.4 (3.4) | 3.6 (3.4) | NS | NS |
| P. gingivalis | Prevalence N (%) | 4 (16) | 0 (0) | NS | 6 (28.4) | 5 (24) | NS | 0.06 |
| | Mean (±SD) proportion | 1.6 (4.3) | 0 (0) | 0 (0) | NS | 36 (17.5) | NS | NS |
| P. micros | Prevalence N (%) | 19.2 (22) | 12 (14.3) | NS | 19.8 (24) | 8.2 (9.5) | NS | NS |
| | Mean (±SD) proportion | 15 (60) | 14 (56) | NS | 15 (71) | 13 (62) | NS | NS |
| C. rectus | Prevalence N (%) | 2 (8) | 2 (8) | NS | 1 (4.8) | 1 (4.8) | NS | NS |
| | Mean (±SD) proportion | 4.4 (2.2) | 2.28 (0) | NS | 2.0 (0) | 1.5 (2.6) | NS | NS |
| Total CFU count | | $5.4\times10^6$ ($5.9\times10^6$) | | | $3.8\times10^6$ ($2.8\times10^6$) | | NS | |

No statistically significant differences could be detected in bacterial loads (mean CFU/ml) for the two groups at the target culture level. Growth of A. actinomycetemcomitans could not be confirmed in any of the patient samples.

**Discussion**

The present study evaluated the effects of adjuvant systemic antibiotics and implant surface debridement on clinical and microbiological parameters of peri-implantitis. The use of antibiotics improved the mean PPD, MR, and BoP in peri-implantitis. Furthermore, significant improvements in PPD and MR were observed with implant surface debridement with systemic antibiotics at implant sites with greater PPD, MR and BoP measurements at

the implant level compared with implant surface debridement alone. Limited studies are available on the effectiveness of implant surface debridement alone or in combination with systemic antibiotics; Therefore, more research is needed to clarify its role in the evidence-based management of peri-implantitis [16]. One uncontrolled cohort study reported improvement in clinical indicators of peri-implantitis with implant surface debridement with systemic antibiotics [17]. A literature review including 16 studies indicated that non-surgical treatment alone has no or minimal effects on improving clinical parameters of peri-implantitis [13]. However, they observed improvement in BoP and PPD with mechanical debridement combined with systemic antibiotics. These results are in line with the current study.

It has previously been suggested that the absence of pus correlates with the success of treatment of peri-implantitis [19]. Implants that contained pus at the first visit consistently needed surgical management after three months of debridement as described by Thierbach et al. [19], while those who did not show any pus on examination initially did not require surgery. This result cannot be verified in our results.

Furthermore, P. gingivalis was completely eradicated in group B (with antibiotics) at the recall visit in contrast to group A (no antibiotics) where the prevalence and proportions of P. gingivalis were not affected. Previous reports indicate that the combined effects of amoxicillin and metronidazole are effective against P. gingivalis, which confirms our findings [14]. Interestingly, a lower frequency was found for P. intermedia and P. micros only in group A. The effectiveness of implant surface debridement alone in reducing the prevalence and incidence of P. intermedia and P. micros in periodontal diseases [18].

Multiple aspects of peri-implantitis resemble chronic periodontitis, and both are opportunistic infections, caused by the presence of bacteria and an aberrant response of the host immune system [2]. Due to their close similarities, peri-implantitis is usually treated in a similar manner to periodontitis, consisting of mechanical debridement and the use of topical and systemic antibacterial agents [2]. However, recent studies suggest that there may be important differences between the microorganisms associated with peri-implantitis compared with periodontitis [16, 17]. Large-scale microbiological studies using open microbial detection techniques are needed to further elucidate the role of specific microbial species in the etiology and pathogenesis of peri-implantitis. In addition, the behavior of the biofilm on the implant surface and its interaction with the host immune system in the presence of the implant biomaterial also needs further investigation [18].

## Conclusions

In the current study, adjuvant use of systemic antibiotics did not show an additional advantage in reducing peri-implant bacterial species and total bacterial loads. Implant surface debridement alone is effective in improving clinical indicators of peri-implantitis. In addition, the adjunctive use of systemic antibiotics significantly reduced pocket probing depths, increased mucosal stasis, and decreased bleeding when investigating peri-implantitis.

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