

Decontamination and Repair Protocol Promotes Positive Outcomes in Implants Affected by Peri-implantitis: A Human Case Series



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This study assessed the effectiveness and predictability of a readily available protocol to treat peri-implantitis utilizing mechanical debridement, chemical antiseptic surface detoxification, and osseous grafting. Nine patients (7 women, 2 men; mean age: 56.5 years) with 15 implants with peri-implantitis were included. Pocket probing depth (PPD), bleeding on probing (BOP), and standardized digital periapical radiographic measurements were taken. Surgical flaps were elevated, and the implant threads were cleaned with a plastic curette. Chemical decontamination was performed by scrubbing solutions of 0.25% sodium hypochlorite (NaClO) and 1.5% hydrogen peroxide (H₂O₂) around the exposed implant using cotton pellets. Bony defects were filled with a 50/50 mixture of bovine hydroxyapatite and nanocrystalline calcium sulfate (CaSO₄). A porcine collagen membrane was placed over the grafted bony defect. Follow-up appointments were scheduled 1 week, 2 weeks, 3 months, 6 months, 9 months, and 1 year posttreatment. Clinical and radiographic parameters were assessed and compared. At baseline, PPD ranged from 5 to 7.5 mm (mean: 6 ± 0.7 mm). At 12 months, PPD ranged from 1.5 to 4.2 mm (mean: 2.5 ± 0.8 mm). The mean PPD reduction of 3.6 mm (59.2%) was statistically significant (P < .001). The number of bleeding sites around each test implant decreased significantly from 4 to 0.4 sites between baseline and 12 months (P < .001). Mean radiographic bone loss decreased from 4.8 ± 1.3 mm to 2.7 ± 1.2 mm (P < .001). The proposed method of mechanical decontamination, chemical detoxification, and bone regeneration is clinically effective and reproducible. Clinical peri-implant parameters and radiographic bone levels were improved and maintained their stability for 1 year using this peri-implantitis treatment protocol. *Int J Periodontics Restorative Dent* 2023;43:699–705. doi: 10.11607/prd.5534

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The search for an effective treatment for peri-implantitis is a prominent topic of research in the field of implant dentistry. A 26-year longitudinal study found peri-implantitis in 34% of patients and around 21% of implants.¹ Due to variations in the definition of peri-implantitis its prevalence has been found to range from 1% to 47%.² Similarly, as the resolution of peri-implantitis following treatment has been inconsistent, numerous therapeutic regimens have been proposed to detoxify a contaminated implant surface, debride the adjacent inflamed soft tissue, and regenerate the surrounding bone and soft tissue attachment.^{3,4}

Multiple histologic animal studies have shown that reosseointegration can be achieved on a cellular level using appropriate decontamination and grafting techniques.^{5–7} While human clinical reports describing bone regeneration following treatment for peri-implantitis are numerous,^{8–12} few show actual histologic evidence of reosseointegration.^{13–15}

The authors recently demonstrated in a proof-of-principle human histologic case report that reosseointegration was feasible following a low-cost, readily available protocol of mechanical debridement, antiseptic chemical detoxification, and osseous grafting.¹³ The objective of this study was to clinically assess the effectiveness and predictability

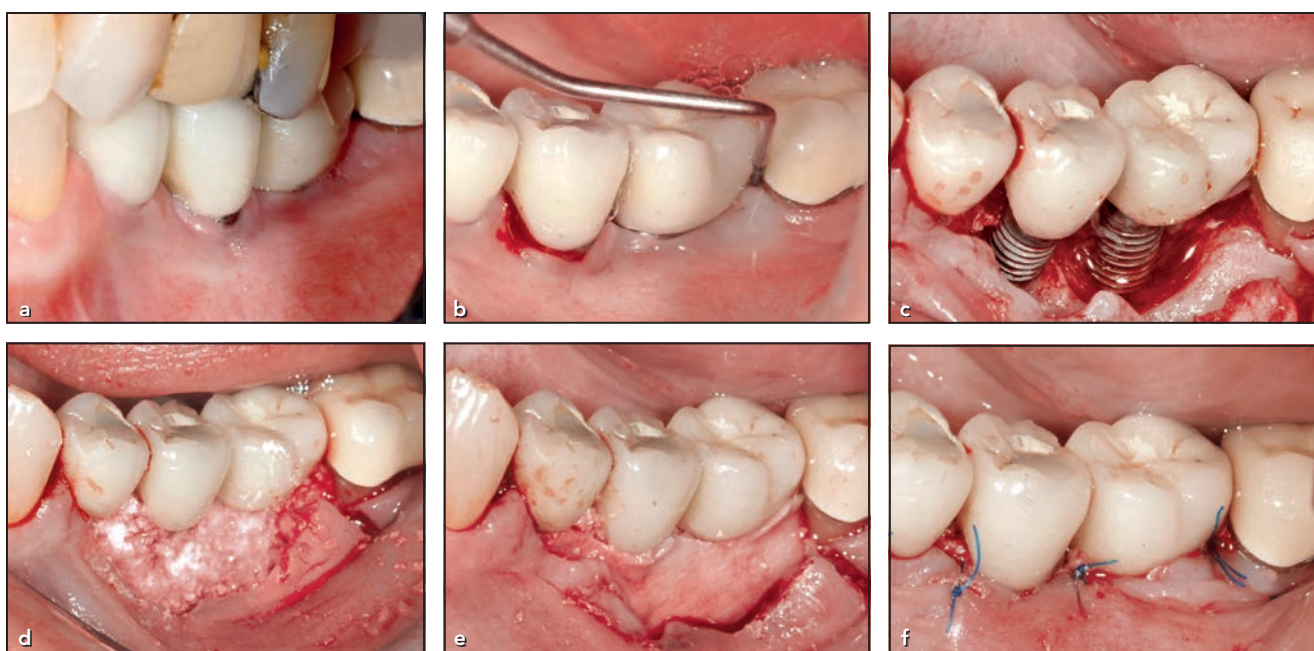


Fig 1 (a) Initial aspect. (b) Peri-implant pocket probing. (c) Surgical access showing the bone defect. (d) Biomaterial filling. (e) Graft covered by bovine collagen barrier. (f) Repositioned sutured flap.

of this therapy in a larger number of treated patients and implants.

Materials and Methods

The research protocol was approved by the Ethics Committee of University Hospital Pedro Ernesto under protocol no. CAEE 82773317.5.0000.5259. All eligible patients signed informed consent forms. All surgical treatments were performed by a single investigator (M.R.S.) who had comprehensive experience in oral surgery.

Patients from the dental clinic of the Department of Periodontics and Implantology at the State University of Rio de Janeiro, as well as from the author's private office, having bleeding on probing, swollen mucosa, purulence, or pain in the soft tissue around an implant were screened.

To further confirm a patient's suitability for the study, comprehensive anamnesis, a clinical examination including probing of the affected implants, and radiographs were used to diagnose the presence of peri-implantitis. Inclusion criteria included: pocket probing depths (PPD) > 3 mm, bleeding on probing (BOP) and/or suppuration on probing (SOP) assessed 1 minute after probing, and radiographic evidence of progressive vertical bone loss around an implant in function for > 1 year. Exclusion criteria included: implant mobility, an implant with less than one third of its length remaining in bone, heavy smokers (≥ 25 cigarettes per day),¹⁶ evidence of current systemic disease, a history of bisphosphonate therapy, head and neck irradiation during the past 5 years, contraindications to undergoing surgical dental treatment,

and noncompliance with the study protocol.

Nine consecutive patients (7 women, 2 men) with a mean age of 56.5 years (range: 52 to 65 years) who fulfilled the above selection criteria and had 15 implants with peri-implantitis (7 premolars and 8 molars), were selected. PPD and BOP were measured at 4 points around the implant (mesiobuccal [MB], mesiolingual [ML], distobuccal [DB], and distolingual [DL]) (Figs 1a and 1b) with a periodontal probe (PCPUNC 156, Hu-Friedy). All initial baseline and subsequent PPD were taken and confirmed by one author (M.R.S.). The first consultation (screening appointment) was considered as baseline.

Digital radiographs were taken at baseline and postoperatively by attaching an index to a radiographic

positioner. This index ensured a reproducible x-ray incidence angle, allowing for standardized pre- and posttreatment radiographic comparisons.

All surgical procedures followed the protocol described in our prior study.¹³ Patients were prescribed a medication regimen of 250 mg of amoxicillin and 250 mg of metronidazole tid for 10 days, and instructed to begin 1 day prior to the procedure. Following local infiltrative anesthesia with 4% articaine, intrasulcular and full-thickness buccal and lingual surgical flaps were elevated to gain access to the exposed implant surface and its associated infrabony defect (Fig 1c). A large curette (Prichard, Hu-Friedy) was used to debride the granulosomatous tissue from the bony defect around the implant. The implant threads were cleaned using a plastic curette (Implacare II, Hu-Friedy) in an effort to disrupt or remove all visible plaque and calculus. Once the bony defect and the implant were thoroughly debrided mechanically, the entire surgical site was rinsed with copious amounts of sterile saline.

Decontamination solutions of 0.25% sodium hypochlorite (NaClO), and 1.5% hydrogen peroxide, (H₂O₂), were prepared by dilution in H₂O and stored in 10-mL syringes. Small cotton pellets saturated with the NaClO solution were used to thoroughly scrub around the implant collar and between the implant threads. After burnishing for approximately 1 minute, the solution was rinsed away using sterile saline. The H₂O₂ solution was then applied using the same protocol. A final thorough ir-



Fig 2 (a) Clinical aspect 14 days postoperative. (b) Clinical aspect 12 months postoperative. (c) Initial periapical radiograph. (d) Final periapical radiograph.

rigation with sterile saline was accomplished subsequent to completing the chemical decontamination procedures.

Bony defects were filled with a 50/50 mixture of bovine hydroxyapatite (Bio-Oss, Geistlich) and nanocrystalline calcium sulfate hemihydrate (CaSO₄; Lumina-Set, Criteria). The bone graft material was incrementally placed into the defect and packed with moderate density to obtain close graft approximation to the implant surface. A porcine collagen membrane (BioGide, Geistlich) was trimmed to fit over the grafted bony defect (Figs 1d and 1e). The flaps were positioned in a tension-free manner to cover the membrane and were sutured with 5-0 nylon sutures (Ethicon) (Fig 1f). Ibuprofen (600 mg) was prescribed to minimize discomfort, taken every 12 hours as needed for 3 days. Patients were

instructed to rinse with chlorhexidine 0.12% bid for 1 minute for 10 days. A periapical radiograph was taken immediately following the surgery.

Follow-up appointments were scheduled 1 week, 2 weeks, 3 months, 6 months, 9 months, and 1 year posttreatment. At these visits, the peri-implant area was debrided and oral hygiene instructions were reinforced. PPD, BOP, and SOP measurements were recorded at postoperative visits, except at the 1- and 2-week follow-ups (Fig 2). Periapical radiographs were taken.

PPD changes were assessed by calculating the difference in mean probing depths between baseline and the 12-month follow-up. Additionally, the differences from baseline to 3 months and 3 months to 12 months were compared to assess the stability of the outcomes.

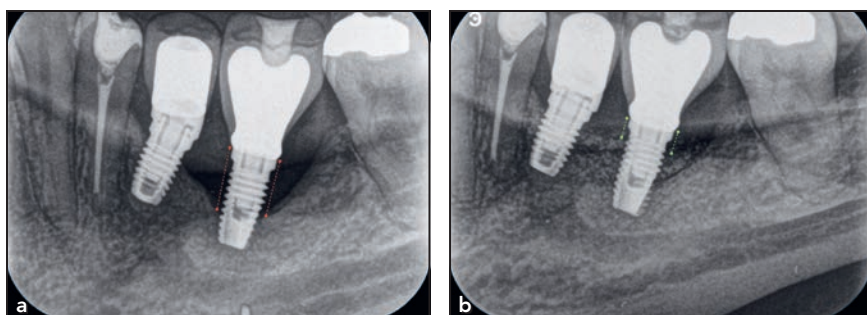


Fig 3 Radiographic measurements of an example case at (a) baseline and (b) 12 months postoperative.

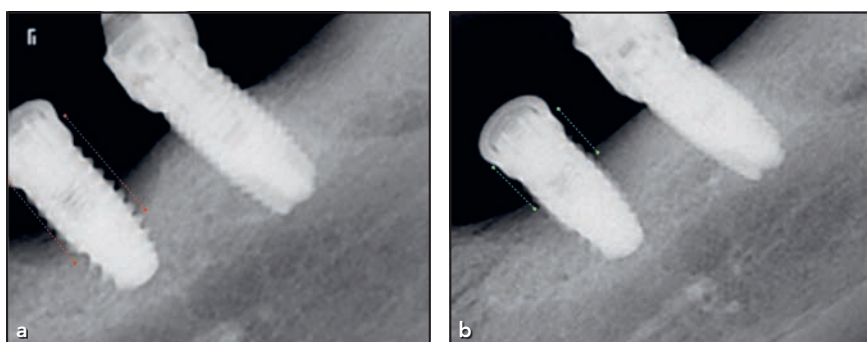


Fig 4 Radiographic measurements of an example case at (a) baseline and (b) 12 months postoperative.

The change in the number of deep peri-implant pockets (≥ 5 mm) was also assessed between time points. BOP was analyzed by assessing the difference in the mean number of bleeding sites for each implant at each time period. Radiographically, the linear distance from the base of the bony defect to the implant platform was recorded using computer software (Carestream Image Suite, Carestream Health) (Figs 3 and 4). These measurements were compared between baseline and 12 months.

The normality of the data was assessed using Shapiro-Wilk test. Student paired *t* test was used to assess normal distribution data, while Wilcoxon paired test was used

to compare non-normal distribution parameters. Statistical analysis was performed using R computer software (Foundation for Statistical Computing).

Results

All surgeries healed uneventfully. All patients were conscientious as they appeared for all follow-up appointments and maintained a high level of oral hygiene.

Pocket Probing Depth

At baseline, PPD at 4 points (MB, DB, ML, and DL) around the 15 test

implants ranged from 5 to 7.5 mm (mean PPD: 6 ± 0.7 mm). At the 12-month follow-up, PPD ranged from 1.5 to 4.2 mm (mean PPD: 2.5 ± 0.8 mm). The mean PPD reduction of 3.6 mm (59.2%) was statistically significant ($P < .001$). Pocket depth measurements at 3, 6, and 9 months were also recorded. Table 1 illustrates the decrease in PPD for each of the 15 implants over time.

Table 2 compares the reduction in mean PPD around the test implants at different time points in the study. Although 3.6 mm (59.2%) of mean pocket depth reduction was achieved between baseline and 12 months, most of this decrease occurred between baseline and 3 months (3.38 mm). The decrease in PPD was found to be statistically significant between baseline and 3 months and between baseline and 12 months, but not between 3 months and 12 months ($P < .01$).

Bleeding on Probing

The number of bleeding sites around each test implant decreased significantly ($P < .001$) between baseline and 12 months (Table 3). The two bleeding sites were found at the 12-month follow-up in one patient.

Radiographic Measurements

The decrease in mean radiographic bone loss from 4.8 ± 1.3 mm at baseline to 2.7 ± 1.2 mm at the 12-month

Table 1 Descriptive Statistics of PPD at the Implant Sites Over Time

	Mean \pm SD	Minimum	1st quartile	Median	3rd quartile	Maximum
Baseline	6 \pm 0.7	5	5.6	6	6.5	7.5
3 mo	2.7 \pm 0.8	1.5	2.2	2.5	3	4.2
6 mo	2.7 \pm 0.7	1.5	2.2	2.5	3.1	4
9 mo	2.7 \pm 0.8	1.2	2.2	2.5	3.1	4
12 mo	2.5 \pm 0.8	1.5	1.9	2.2	2.8	4.2

Data are presented in millimeters.

Table 2 Comparison of Mean PPD Between Time Points

	Mean difference	Average variation	P
3 mo / baseline	-3.383 mm	-55.917%	.000
12 mo / baseline	-3.600 mm	-59.236%	.000
12 mo / 3 mo	-0.217 mm	-3.318%	.346

Table 3 Descriptive Statistics of BOP Sites Over Time

	Mean \pm SD	Frequency/ implant, %	Minimum	1st quartile	Median	3rd quartile	Maximum
Baseline	4 \pm 0	100	4	4	4	4	4
3 mo	0 \pm 0	0	0	0	0	0	0
6 mo	0 \pm 0	0	0	0	0	0	0
9 mo	0.2 \pm 0.56	6	0	0	0	0	2
12 mo	0.4 \pm 0.83	20	0	0	0	0	2

Data are presented as the number of BOP sites.

Table 4 Descriptive Statistics of the Mean Radiographic Bone Level by Time Point

	Mean \pm SD	Minimum	1st quartile	Median	3rd quartile	Maximum
Baseline	4.8 \pm 1.3	1.3	4.4	4.8	5.7	6.4
12 mo	2.7 \pm 1.2	0.9	1.7	2.8	3.4	5.5

Data are presented in millimeters.

follow-up was statistically significant ($P < .001$) (Table 4). This 2.16 mm of radiographic bone fill is equivalent to a 43.5% increase in visible calcified material abutting the implant surface.

Discussion

Following implant surface decontamination and osseous grafting, multiple human clinical studies have demonstrated postoperative radio-

graphic evidence of bone fill with close bone-to-implant approximation upon clinical reentry.^{11,12} Histologic evidence of reosseointegration is still considered to be a challenging goal.¹⁰

As new direct bone-to-implant contact can only be ascertained by histologic section, it is not possible to confirm its occurrence in clinical trials. A human histologic case report was recently published demonstrating reosseointegration was achievable utilizing a specific systematic protocol.¹³ The results of this case series study, consisting of 15 consecutively treated implants with peri-implantitis, illustrate the clinical and radiographic reproducibility of this surgical-regenerative technique.

Pretreatment pocket depths were measured, but initial attachment and recession levels were not assessed, given the **tenuous** nature of soft tissue adherence to an implant surface and the variable amount of recession that might occur following surgery, depending on implant surface topography. Consequently, total posttreatment pocket reduction, reduction in BOP, and radiographic bone fill were evaluated as measures of success. All implants showed improvements in clinical and radiographic metrics at 3, 6, 9, and 12 months following treatment. Mean total pocket reduction, consisting of **soft tissue readherence and bone fill minus any recession that might have occurred, was 3.6 mm**. This compared with a reduction of 2.8 mm in a recent systematic review.⁷ The mean number of BOP sites was reduced from 4 ± 0 to 0.4 ± 0.83 , which is similar to the results achieved by Dalago et al¹⁷ (0.5 ± 0.27) using open flap debridement, citric acid decontamination, and implantoplasty. Bassetti et al¹⁸ have also demonstrated significant reductions in BOP using adjunct local drug delivery or photodynamic

therapy, even though their 1-year follow-up results show higher BOP rates (1.74 ± 1.37 and 1.55 ± 1.26 , respectively). Mean radiographic bone loss was reduced by 2.1 mm. The reductions in BOP and PPD were viewed as clinical **signs of reduced inflammation in the peri-implant mucosa as well as an indicator of treatment efficacy**.¹⁹

While plastic curettes have been shown to be unreliable at completely removing plaque,²⁰ they cause minimal damage to a smooth implant surface²¹ as they disrupt surface biofilm colonies. Although there have been reports of **plastic residue left on an implant surface following the use of plastic curettes**,^{22,23} there was **no residual inflammation or evidence of clinical healing impairment** of the peri-implant tissues following the decontamination protocol utilized in this study.

The antimicrobial effectiveness of NaClO is based on its high pH, as it interferes with bacteria cytoplasmic membrane integrity by irreversible enzymatic inhibition and alterations in cellular metabolism and **phospholipid degradation**. H₂O₂ has a potent oxidative effect on microorganisms and reinforces the activity of NaClO as it has been shown to promote cell wall **lipopolysaccharide endotoxin removal**.^{13,24} The effectiveness of both antiseptics, along with copious amounts of sterile saline irrigation, combined with thorough mechanical debridement and decontamination, are all thought to have contributed to the favorable results demonstrated in this trial.

Calcium sulfate hemihydrate porosity and **hygroscopic properties**

promote the adsorption and infiltration of platelets and growth factors. **Calcium ions released during its re-sorption activate platelets to release bone morphogenetic proteins (BMPs) and platelet-derived growth factors (PDGFs) that stimulate angiogenesis, osteogenic proliferation, and differentiation of mesenchymal stem cells.**²⁵ Additionally, CaSO₄ retards epithelial and connective tissue ingrowth.²⁶ The bovine hydroxyapatite xenograft is osteoconductive and similar in structure and chemical composition to the inorganic component of human bone. **Its porosity encourages vascular infiltration, diffusion of nutrients from surrounding tissues, osteoblastic cellular ingrowth, and cellular adhesion to its surface.**²⁷

The outcomes obtained in this trial were stable to its 1-year end point. Notwithstanding the outcomes of this case series study, if this protocol is to be identified as a treatment of choice for the management of peri-implantitis, the efficacy of this previously histologically validated regenerative approach needs to be confirmed in a larger, multi-armed clinical trial, having longer-term follow-ups, and the inclusion of control cases.

Conclusions

The proposed method of mechanical decontamination, chemical detoxification, and bone regeneration is clinically effective and reproducible. Clinical peri-implant parameters, as well as radiographic bone levels, were improved and maintained their stability for 1 year using this peri-implantitis treatment protocol.

Acknowledgments

The authors declare no conflicts of interest.

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