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Long-Term Outcomes of Implantoplasty in the Surgical Treatment of Peri-Implantitis: A Retrospective Case Series

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ABSTRACT

Introduction: Peri-implantitis represents a major issue in implant dentistry, often requiring surgical intervention, as non-surgical treatment does not always achieve clinical endpoints. Implantoplasty is a surgical technique aimed at smoothing the contaminated implant surface to reduce bacterial biofilm adhesion.

Methods: Five patients (eight implants) were included in this retrospective case series. Clinical and radiographical parameters—including probing depth (PD), bleeding on probing (BoP), mucosal recession (REC), visible plaque index (VPI), and marginal bone loss (MBL) were collected at baseline and after a minimum of 12 months of follow-up from medical records. Statistical analysis was conducted according to the results of the normality test.

Results: Improvement was observed in all clinical parameters except REC: mean PD (5.3 ± 1.8 mm to 2.8 ± 0.7 mm, $p = 0.011$), BoP (87.5% to 6.2%, $p = 0.014$), VPI (58.3% to 29.2%, $p = 0.042$), and REC (0.7 ± 1.3 mm to 2.3 ± 1.4 mm, $p = 0.011$), while variation in MBL was not statistically significant. No implant failure or loss was recorded during follow-up.

Conclusion: Implantoplasty provided stable, long-term clinical and radiographic outcomes in the treatment of peri-implantitis.

Keywords: Bone loss; Implantoplasty; Peri-implant disease; Peri-implantitis; Surgical treatment

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INTRODUCTION

Dental implants are widely used to replace missing teeth and for partial or full-mouth rehabilitation. However, despite their high survival rate, peri-implant diseases still represent a debated and unresolved problem in dentistry.[1] Peri-implant mucositis is an inflammation of the soft tissues around dental implants, characterized by bleeding on probing (BoP), erythema, and swelling but without radiographic bone loss.[2,3] It is typically induced by the accumulation of bacterial biofilm at the peri-implant mucosal interface.[2,3]Fare clic o toccare qui per immettere il testo. Treatment of mucositis is mandatory as it may progress to peri-implantitis.[4] While mucositis is limited to the soft tissues, peri-implantitis further progresses, causing progressive bone loss. A probing depth (PD) ≥ 6 mm and radiographic bone loss ≥ 3 mm, together with the presence of BoP and suppuration, are indicative of peri-implantitis.[1]

Etiologically, dental biofilm deposits are the main cause of both mucositis and peri-implantitis, with several additional risk factors identified, such as untreated severe periodontitis, lack of regular maintenance therapy, smoking, uncontrolled diabetes, and residual cement or prosthetic components that impede proper oral hygiene maneuvers.[3,5,6] The prevalence of peri-implantitis ranges from 15% to 22%, depending on factors such as the studied population and diagnostic criteria. Therefore, peri-implantitis represents a relatively common pathology capable of compromising patients' well-being and quality of life.[1]

Treatment is complex and often requires a stepwise approach, starting with non-surgical periodontal therapy to reduce and eliminate both plaque and inflammation. Given that non-surgical periodontal therapy does not always prove effective, the evaluation of new therapeutic protocols involving adjunctive substances and surgical therapies is necessary.Fare clic o toccare qui per immettere il testo.[7,8] In particular, surgical intervention may provide better access for debridement as well as for dental implant surface modification.

Implantoplasty aims to modify the morphology of the contaminated implant surface, in association with a resective approach, to reduce or eliminate the peri-implant pocket and bone defect.[9] The technique involves the removal of implant threads and polishing of the exposed implant surface to create a smooth, plaque-resistant surface. Currently, few studies report long-term (≥5 years) outcomes after implantoplasty, and comparative data remain limited. This procedure is generally considered in non-contained, supracrestal, or horizontal defects where bone regeneration is difficult to achieve.[10] The rationale behind the technique lies in the fact that a smooth implant surface may reduce bacterial adhesion and aid patients in biofilm control.[11]

Therefore, the aim of the present study was to retrospectively assess the long-term efficacy of implantoplasty in the surgical treatment of peri-implantitis by evaluating clinical parameters such as PD, BoP, and radiographic bone stability.

MATERIALS AND METHODS

Ethical considerations

The research protocol was reviewed and approved by the Institutional Review Board of the University of Catania (approval number: 2679/2022/PO).

Study design and setting

Clinical data were collected from medical records at the Department of Periodontology, University of Naples Federico II. The study was conducted in full accordance with the Declaration of Helsinki (2013). The Strengthening the Reporting of Observational Studies in Epidemiology checklist for observational research was followed (**Table A1**).

Patient selection

Clinical records of all patients treated between January 2012 and December 2022 were collected based on predefined inclusion and exclusion criteria.

Inclusion criteria:

i Age ≥ 18 years

ii Systemically healthy or medically controlled patients

iii Presence of at least one osseointegrated dental implant with a diagnosis of peri-implantitis

iv Implants in function for at least 12 months before diagnosis

v Underwent surgical treatment including implantoplasty

vi At least 12 months postoperative follow-up

Diagnosis of peri-implantitis (PD ≥ 6 mm and radiographic bone loss ≥ 3 mm apical to the implant shoulder)

Exclusion criteria:

i Incomplete records

ii Uncontrolled systemic diseases (e.g., uncontrolled diabetes)

iii Use of bisphosphonates or medical-related osteonecrosis of the jaws-associated drugs

iv History of head and neck radiotherapy

Clinical and radiographic parameters

The following parameters were collected at baseline (before surgery) and at the final follow-up (minimum 1 year, up to 10 years):

i PD: Measured at six sites per implant using a calibrated periodontal probe (UNC-15, Hu-Friedy)

ii BoP: Recorded dichotomously (present/absent)

iii Mucosal recession (REC)

iv Visible plaque index (VPI)

v Radiographic bone levels: Evaluated on standardized periapical radiographs using the parallel technique; bone loss was measured from the implant shoulder to the first bone-to-implant contact.

Measurements were performed by calibrated examiners. When possible, measurements were taken from digital records and confirmed with radiographic documentation.

Statistical analysis

The implant was considered a statistical unit. For all clinical and radiographical parameters, descriptive statistics were performed. Results were expressed as mean and standard deviation for continuous variables (e.g., PD, bone loss), while categorical variables (e.g., BoP) were reported as percentages. A normality test was performed to assess data distribution. According to the results, changes in clinical parameters between baseline and follow-up were evaluated using paired *t*-tests (e.g., worst PD, marginal bone loss

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[MBL] mesial, mean MBL) or Wilcoxon signed-rank tests (e.g., VPI, BoP, mean PD, mean REC, worst REC, MBL distal). A lambda test for the frequency distribution of BoP-positive (1) and BoP-negative (0) sites at follow-up was also performed. A p-value < 0.05 was considered statistically significant. Statistical analysis was performed using SPSS Statistics version 29.9 (IBM, United States of America).

Surgical procedure

After local anesthesia with 2% mepivacaine with epinephrine 1:100,000, full- or partial-thickness mucoperiosteal flaps were elevated, depending on the amount of residual keratinized mucosa, to expose the implant surface and surrounding defect. Granulation tissue was carefully removed using both ultrasonic and hand instruments (curettes). Implantoplasty was then performed using a sequence of rotary instruments: a diamond bur to remove the exposed implant threads, followed by decreasing-grit silicon polishers to smooth the implant surface. Continuous irrigation with refrigerated sterile saline solution was used throughout the procedure to avoid overheating. Implantoplasty was performed only in the supracrestal, exposed portion of the implant, where regenerative therapy was difficult to achieve due to the defect morphology. After surface decontamination, the surgical site was irrigated, and the flap was repositioned and sutured with resorbable sutures. No bone grafts or regenerative materials were used (**Figures 1** and **2**).

Postoperative therapy and maintenance

The postoperative therapy was standardized for all treated patients:

i Amoxicillin 875 mg + clavulanic acid 125 mg twice daily for 7 days (or clindamycin 300 mg in case of allergy)

ii Chlorhexidine 0.12% mouth rinse twice a day for 2 weeks

iii Analgesics as needed (e.g., ibuprofen 600 mg)

Patients were instructed to avoid mechanical trauma in the surgical area for 1 week. Sutures were removed after 10–14 days. Supportive periodontal therapy (SPT) was initiated and maintained at three- to 6-month intervals, including professional cleaning and reinforcement of oral hygiene instructions.

RESULTS

A total of five patients (three females and two males) with a mean age of 65.2 ± 6.9 years were included in the study. Four patients were non-smokers, and one reported smoking fewer than 10 cigarettes per day. The periodontal history of the patients was variable, and all demographic data are presented in **Table 1**. Eight dental implants were evaluated. The implants were mainly placed in the maxillary anterior region, with lengths ranging from 10 to 12 mm and diameters between 3.3 and 4.8 mm (**Table 2**).

Clinical outcomes

A statistically significant improvement in all clinical parameters was observed between baseline and follow-up. A significant reduction in mean PD from 5.3 ± 1.8 mm at baseline to 2.8 ± 0.7 mm at follow-up ($p = 0.011$) was recorded, while mean REC increased from 0.7 ± 1.3 mm to 2.3 ± 1.4 mm ($p = 0.011$). VPI showed a statistically significant reduction from 58.3 ± 46.3% to 29.2 ± 42.5% ($p = 0.042$), indi-

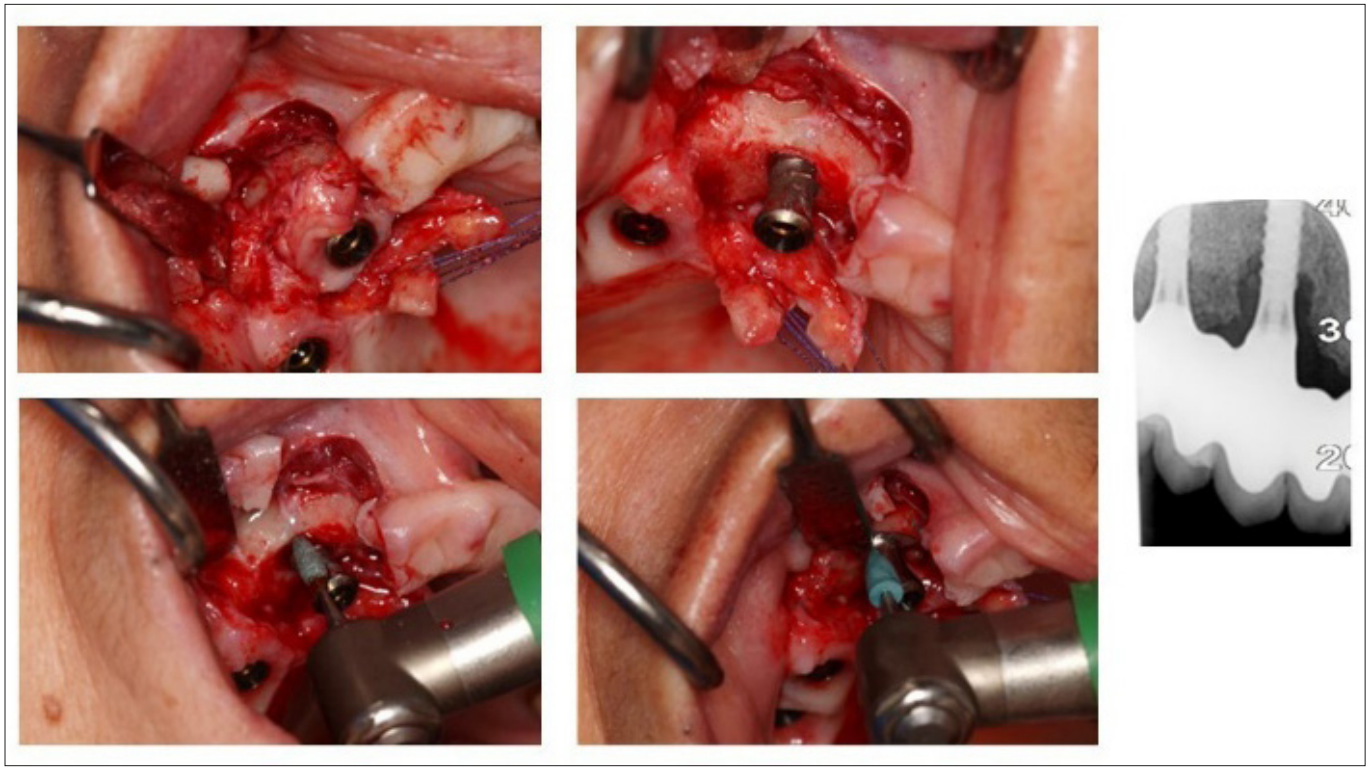

Figure 1. Elevated flap showing the granulation tissue surrounding the implant (A) and the exposed implant surface (B) after removal of granulation tissue and initial implantoplasty. Implant surface is polished with with decreasing grit silicon burs (C, D). Radiographic aspect at baseline (E).

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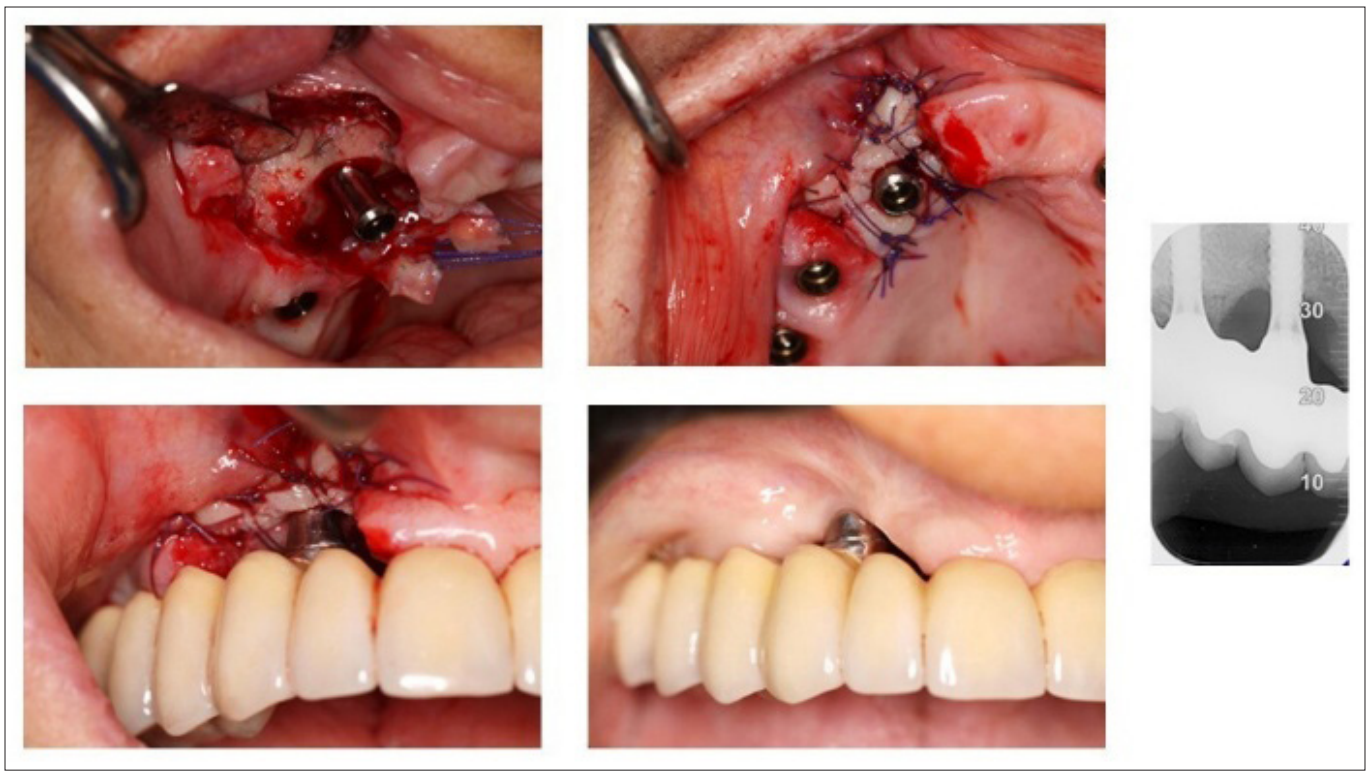

Figure 2. Once the implant surface is thoroughly polished (A), the flap is sutured (B). Post-operative aspect (C) and at follow-up (D). Radiographic aspect at follow-up (E).

Table 1. Demographic characteristics of the included samples

Patient #	Age (years)	Sex (M/F)	Smoking habit (S/NS)	Periodontal status (staging/grading and extension)
Patient 1	68	F	NS	III-b generalized
Patient 2	74	F	NS	IV-c generalized
Patient 3	63	M	S	III-c generalized
Patient 4	77	M	NS	I-a generalized
Patient 5	60	F	NS	IV-c generalized
	65.2 ± 6.9	3F/2M	4 NS/1S	

Abbreviations: F: Female; M: Male; NS: Non-smoker; S: Smoker.

cating improved plaque control. BoP decreased from 87.5 ± 35.3% to 6.2 ± 12.4% at follow-up, showing a statistically significant change ($p = 0.014$) (**Table 3**). When evaluating the worst site per implant, mean PD showed a statistically significant reduction from 6.3 ± 1.7 mm to 2.75 ± 0.7 mm ($p < 0.001$). On the contrary, recession at the worst sites increased from 1.3 ± 2.3 mm to 2.0 ± 1.7 mm; however, this change was not statistically significant ($p = 0.234$; **Table 4**). The lambda test for frequency distribution of BoP-positive (1) and BoP-negative (0) sites at follow-up was not statistically significant ($p = 0.149$); the outcome is presented in **Table 5**.

Radiographic outcomes

Radiographic assessment of marginal bone levels revealed stable conditions over time, with only minor changes in mesial and distal measurements. Mean mesial bone loss increased slightly from 3.1 ± 2.2 mm to 3.5 ± 1.8 mm ($\Delta = 0.4 \pm 1.2$ mm, $p = 0.402$), and distal bone loss from 3.8 ± 1.2 mm to 3.9 ± 1.1 mm ($\Delta = 0.1 \pm 0.3$ mm, $p = 0.371$), with neither change being statistically significant. However, the overall mean MBL between baseline and follow-up was 1.6 ± 1.5 mm, which was statistically significant ($p = 0.02$; **Table 6**). Despite this, the final bone levels remained within functional thresholds, and no implant failures or signs of peri-implantitis progression were recorded during the follow-up period.

Implant survival and complications

All implants remained in function throughout the entire follow-up period (minimum 1 year, maximum 10 years) with an average follow-up of approximately 7 years. No biological complications, such as recurrence

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Table 2. Dental implant characteristics			
Implant #	**Location**	**Length (mm)**	**Diameter (mm)**
1	1.3	10	4.1
2	1.4	10	3.5
3	3.3	10	4.1
4	3.6	12	4.8
5	4.1	12	3.3
6	1.3	12	3.3
7	1.2	12	3.3
8	1.1	12	3.3

Table 3. Mean (mm) of clinical parameters			
Clinical parameter	**Baseline**	**Follow-up**	**Significance**
PD (mm)	5.3 ± 1.8	2.8 ± 07	$p = 0.011$*
REC (mm)	0.7 ± 1.3	2.3 ± 1.4	$p = 0.011$*
VPI (%)	58.3 ± 46.3	29.2 ± 42.5	$p = 0.042$*
BoP (%)	87.5 ± 35.3	6.2 ± 12.4	$p = 0.014$*
Note: *Statistically significant value ($p < 0.05$). Abbreviations: BoP: Bleeding on probing; PD: Probing depth; REC: Mucosal recession; VPI: Visible plaque index.			

Table 4. Worst clinical parameters (mm)			
Clinical parameter	**Baseline**	**Follow-up**	**Significance**
PD (mm)	6.3 ± 1.7	2.75 ± 0.7	$p < 0.001$*
REC (mm)	1.3 ± 2.3	2.0 ± 1.7	$p = 0.234$
Note: *Statistically significant value ($p < 0.05$). Abbreviations: PD: Probing depth; REC: Mucosal recession.			

Table 5. Frequency distribution of bleeding on probing (BoP)-positive (1) and BoP-negative (0) sites at follow-up			
Implant #	**BoP sites**		***p*-value**
	0	**1**	
Implant 1	6	0	0.149
Implant 2	6	0	
Implant 3	6	0	
Implant 4	6	0	
Implant 5	6	0	
Implant 6	6	0	
Implant 7	5	1	
Implant 8	4	2	
Total	45	3	–

Table 6. Marginal bone loss (MBL)				
	Baseline (mm)	**Follow-up (mm)**	**Δ-value (mm)**	**Significance**
MBL (mesial)	3.1 ± 2.2	3.5 ± 1.8	0.4 ± 1.2	$p = 0.402$
MBL (distal)	3.8 ± 1.2	3.9 ± 1.1	0.1 ± 0.3	$p = 0.371$
MBL (mean)	2.1 ± 1.1	3.7 ± 1.4	1.6 ± 1.5	$p = 0.02$*
Note: *Statistically significant value ($p < 0.05$).				

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of peri-implantitis, suppuration, or severe inflammation, were noted. Similarly, no mechanical failures, such as implant fracture or prosthetic loosening, were observed.

DISCUSSION

The main aim of the present study was to evaluate the efficacy of implantoplasty in the treatment of peri-implantitis. All implants in our study demonstrated marked reductions in PD, BoP, and VPI, while radiographic bone levels remained stable, with only modest and clinically irrelevant MBL. Based on the obtained data, it appears that implantoplasty can achieve stable clinical and radiographical results.

The recorded PD reduction is in line with data available in the literature. In fact, Lima *et al.*,[12] in a meta-analysis, confirmed a mean PD reduction of approximately 3.37 mm and implant success probabilities of 97.5% at 6 months and 94.7% at 24 months. Furthermore, both BoP and VPI reductions were statistically significant. Clinical studies have demonstrated that implantoplasty can significantly reduce PD, BoP, and peri-implant inflammation. This may be due not only to improved oral hygiene procedures but also to the smoother implant surface obtained, which appears less prone to plaque retention.[11,13]Fare clic o toccare qui per immettere il testo.

In addition, the recorded REC is consistent with the resective nature of the surgical procedure, whose objective is to control inflammation and remove plaque through soft-tissue excision. Nevertheless, the PD reduction (2.5 mm) and near-elimination of BoP represent relevant clinical improvements that fulfil the criteria for treatment success (PD ≤ 5 mm, absence of BoP/suppuration, and stable bone levels) proposed in the *Prevention and Treatment of Peri-Implant Diseases* clinical guidelines of 2023.[1] Although esthetic outcomes were not evaluated, no patient reported dissatisfaction with the postoperative results. However, implantoplasty should be carefully evaluated when performed in the anterior region.

No significant mechanical failures, such as implant fractures or prosthetic complications, were observed when the procedure was correctly performed.[14]Fare clic o toccare qui per immettere il testo. In the present study, treatment success was defined according to the *Treatment of Stage I–III Periodontitis—The European Federation of Periodontology S3 Level Clinical Practice Guideline* (≤1 point of BoP, absence of suppuration, PD ≤ 5 mm, and absence of progressive bone loss compared to pre-treatment bone levels). All implants in the present study met these criteria at follow-up, supporting the long-term clinical stability of implantoplasty in the surgical management of peri-implantitis.[15]Fare clic o toccare qui per immettere il testo.

Despite these favorable outcomes, implantoplasty remains a debated procedure, as there are potential risks that need to be considered. The process of removing implant threads produces titanium particles that may deposit in soft and hard tissues, potentially leading to cytotoxic or immunogenic effects. *In vitro* studies have shown toxic effects on both fibroblast and osteoblast in the presence of Ti-6Al-4V particles released during polishing procedures[16]; however, constant irrigation appears to reduce the risk of particle deposits. *In vivo* studies on an animal model (rats) have demonstrated that, after implantoplasty, granulomatous reaction can occur in the surrounding tissues, with the presence of histiocytes and multinucleated giant cells (MNGCs) around the metal particles. In addition, small metal particles were found within the cytoplasm of the MNGCs. These histological findings suggest that metal particles may elicit a granulomatous reaction characterized by histiocytes and MNGCs, typical of a foreign body response.[17]

Thread removal may also reduce the structural integrity of dental implants, decreasing resistance to both fatigue and mechanical fracture. Gehrke *et al.*[18]Fare clic o toccare qui per immettere il testo. showed that after implantoplasty, titanium implants exhibited up to 32% loss of structural strength depending on implant design and material. Similarly, Costa-Berenguer *et al.*[19] observed a reduced internal thread diameter and loss of fatigue resistance in narrow-diameter implants following polishing. These findings suggest that the reduction in implant diameter may compromise the structural integrity of the implant itself, especially in narrow-diameter implants or those subjected to high occlusal forces.

Thermal injuries must also be considered. If burs are not adequately cooled, dangerous heating of the surrounding hard and soft tissues may occur, potentially reaching temperatures above 47°C and causing bone necrosis.[20] However, Sharon *et al.* demonstrated that when the proper bur is used under adequate irrigation with refrigerated saline solution, the temperature increase is modest (approximately 1.5°C), making implantoplasty a safe procedure from this standpoint.[21]

A recent randomized clinical trial reported results that contrast with our findings. Cruz *et al.*[22]Fare clic o toccare qui per immettere il testo. found no statistically significant difference in PD reduction between implantoplasty and mechanical debridement alone in the test and control groups. However, this may be attributed to the relatively low baseline PD values (3.17 and 2.78 mm, respectively), suggesting that under such conditions, mechanical plaque removal alone may be equally effective. Similarly, Ravidà *et al.*,[23] in a retrospective study, showed no difference in implant survival rates between resective treatment with implantoplasty (90%) and without implantoplasty (81.6%; $p > 0.05$), suggesting that supportive SPT is the key factor ensuring implant survival.

The lack of difference in the cited studies may be related to adherence to proper SPT protocols. Nevertheless, the rationale behind implantoplasty is to make the exposed implant surface more biocompatible, facilitating effective oral hygiene and potentially reducing the need for frequent SPT. Indeed, the most favorable outcomes were observed in patients who received more than two SPT sessions per year. However, patient adherence to SPT remains essential for long-term implant survival.[23]

Finally, esthetic considerations must be taken into account when implantoplasty is performed in the anterior zone, as it may lead to soft-tissue discoloration and implant exposure. Despite these potential risks, current clinical evidence suggests that implantoplasty, when carefully indicated and executed with proper technique, does not result in significant complications in the short to medium term and may serve as a viable adjunct in the surgical management of peri-implantitis.[14]

To sum up, the benefits of implantoplasty may be most evident in selected clinical cases, particularly when regeneration is not achievable. The decision to perform implantoplasty should be guided by defect morphology, implant design, occlusal loading, and patient risk

doi: 10.36922/sti.0300

factors. In our study, the high success rate and absence of complications may be partly attributed to appropriate case selection (e.g., non-contained defects, adequate implant diameters), proper surgical technique (e.g., controlled irrigation, sequential polishing), and patient adherence to maintenance protocols.

LIMITATIONS

The current study has limitations that need to be addressed: the retrospective design and absence of a control group; the small sample size; and the variability in follow-up duration, which may have influenced the clinical and radiographical outcomes. Furthermore, it must be stated that variability due to patient compliance with oral hygiene maintenance may have affected the results, although all surgeries were performed by the same operator. In addition, the small sample size (five patients, eight implants) limited the generalizability of the results; however, the large effect size recorded for the clinical parameters (e.g., PD reduction) seems to indicate that the clinical improvements were meaningful. Moreover, the study evaluated a heterogeneous follow-up period ranging from 1 to 10 years; however, no time-dependent data were available in the analyzed data. Finally, the lack of a control group prevents direct comparison with other peri-implant treatment modalities. Despite these limitations, the study still provides valuable long-term insights supporting the stability and safety of the described procedure.

CONCLUSION

Despite the limitations of the present study, implantoplasty may be considered as a treatment option for peri-implantitis, as it appears to provide stable, long-term clinical and radiographic outcomes. Therefore, further randomized clinical studies with larger samples are needed to better understand the clinical efficacy of implantoplasty.

AUTHORS' DISCLOSURE

The authors would like to thank the staff of the Department of Periodontology, University of Naples Federico II, for their assistance. The authors declare no conflicts of interest related to this work. This study received no external funding. The research protocol was reviewed and approved by the Institutional Review Board of the University of Catania (approval number: 2679/2022/PO). The study was conducted in full accordance with the Declaration of Helsinki (2013). The work was not presented at any conference. Informed consent was obtained from all subjects involved in the study. Data were collected from the medical records of the Department of Periodontology, University of Naples Federico II. Due to the retrospective nature of the study, consent for publication could not be obtained. Nevertheless, all efforts were made to conceal identifying participant data.

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APPENDIX

Table A1. Strengthening the Reporting of Observational Studies in Epidemiology checklist for observational research

	Item no.	Recommendation	Page no.
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract	1
		(b) Provide in the abstract an informative and balanced summary of what was done and what was found	1
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	2
Objectives	3	State specific objectives, including any prespecified hypotheses	2
Methods			
Study design	4	Present key elements of study design early in the paper	2
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	2
Participants	6	(a) Cohort study—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up Case-control study—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls Cross-sectional study—Give the eligibility criteria, and the sources and methods of selection of participants	2
		(b) Cohort study—For matched studies, give matching criteria and number of exposed and unexposed Case-control study—For matched studies, give matching criteria and the number of controls per case	N.A.
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	3
Data sources/measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	3
Bias	9	Describe any efforts to address potential sources of bias	N.A.
Study size	10	Explain how the study size was arrived at	2
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	3
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding	4
		(b) Describe any methods used to examine subgroups and interactions	N.A.
		(c) Explain how missing data were addressed	N.A.
		(d) Cohort study—If applicable, explain how loss to follow-up was addressed Case-control study—If applicable, explain how matching of cases and controls was addressed Cross-sectional study—If applicable, describe analytical methods taking account of sampling strategy	N.A.
		(e) Describe any sensitivity analyses	N.A.

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Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—e.g., numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	4
		(b) Give reasons for non-participation at each stage	N.A.
		(c) Consider use of a flow diagram	N.A.
Descriptive data	14*	(a) Give characteristics of study participants (e.g., demographic, clinical, social) and information on exposures and potential confounders	4
		(b) Indicate number of participants with missing data for each variable of interest	N.A.
		(c) Cohort study—Summarise follow-up time (e.g., average and total amount)	4
Outcome data	15*	Cohort study—Report numbers of outcome events or summary measures over time	4
		Case-control study—Report numbers in each exposure category, or summary measures of exposure	N.A.
		Cross-sectional study—Report numbers of outcome events or summary measures	N.A.
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (e.g., 95% confidence interval). Make clear which confounders were adjusted for and why they were included	N.A.
		(b) Report category boundaries when continuous variables were categorized	N.A.
		(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N.A.
Other analyses	17	Report other analyses done—e.g., analyses of subgroups and interactions, and sensitivity analyses	N.A.
Discussion			
Key results	18	Summarise key results with reference to study objectives	5
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	6
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	5,6
Generalizability	21	Discuss the generalisability (external validity) of the study results	5,6
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	6

Abbreviation: N.A., not applicable.

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