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Absence of Progressive Bone Loss Following Peri-Implantitis Surgical Therapy with Implantoplasty: A Case Series

Arthur Brincat [1,2,*], Angéline Antezack [1,2,3], Camille Sadowski [1,2], Mathias Faure-Brac [1,2], Romain Ohanessian [1,2] and Virginie Monnet-Corti [1,2,3,4]

[1] Dental School, Aix-Marseille University, 13385 Marseille, France; angeline.antezack@univ-amu.fr (A.A.); camille.sadowski@univ-amu.fr (C.S.); mathias.faure-brac@univ-amu.fr (M.F.-B.); romain.ohanessian@univ-amu.fr (R.O.); virginie.corti@univ-amu.fr (V.M.-C.)
[2] Pole Odontologie Centre Dentaire, Timone Hospital, 13005 Marseille, France
[3] IHU Mediterranee Infection, 13005 Marseille, France
[4] Institut de Recherche Pour le Développement, 13002 Marseille, France
* Correspondence: arthurbrincat@gmail.com

Abstract: Background: Peri-implantitis, a bacteria-associated inflammatory disease, is characterized by inflammation of the peri-implant mucosa and progressive loss of the supporting bone, thereby reducing the chances of dental implant survival. The absence of progressive marginal bone loss is crucial for implant success. The aim of this study is to assess the peri-implantitis resolution by measuring the absence of progressive bone loss rate around the implant over a period of one year to more than three years after surgical reconstructive (REC) treatment, apically repositioned flap (ARP) surgery, or combined (COM) treatment of peri-implantitis with implantoplasty. Methods: Peri-implantitis patients, that underwent surgical therapy with implantoplasty and that enrolled in a regular peri-implant supportive care program with a follow up of ≥12 months, were recruited in this study. ARP, REC, or COM surgical therapy was performed depending on the anatomy of the bone defect. For REC and COM groups, intraosseous defects were filled with a bone substitute. The ARP group consisted of an apically positioned flap without osseous surgery. Absence of progressive marginal bone loss was evaluated on radiographs of the treated implants. Results: A total of 57 patients (91 implants) were included. The study occurred over a follow-up period of 12 to 42 months (mean = 24 months). The surgical treatment with implantoplasty yielded an absence of progressive bone loss rate of 96.7% at implant level (100% REC, 98% COM, 92.9% ARP) and 96.5% at patient level. Three implants had to be removed in two patients due to relapse or progression of peri-implantitis. Conclusions: This case series demonstrated that implantoplasty during surgical treatment of peri-implantitis lesions resulted in favorable biological conditions to maintain functional implants with 96.7% of implants that did not show bone loss over time from one year to more than three years.

Keywords: peri-implantitis; alveolar bone loss; dental implant; dental implantation; implant survival; peri-implant lesions; implantoplasty; flap apicalisation surgery; reconstructive surgery

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1. Introduction

One of the key factors in the success of dental implants is the absence of progressive marginal bone loss, as its presence significantly decreases the chances of implant survival. The loss of peri-implant bone is influenced by multiple factors, with peri-implantitis being a potential contributing cause.

Peri-implantitis is a bacteria-associated inflammatory disease affecting the tissues around implants, characterized by inflammation of the peri-implant mucosa and progressive loss of the supporting bone [1]. Peri-implant microbiota presents a different bacterial ecosystem compared with the microbiota in periodontitis, as it is qualitatively lower in

terms of microbial diversity, but quantitatively higher for some bacterial genera [2]. Epidemiologic studies report a prevalence of peri-implantitis in 2 out of 10 implant holders and 1 out of 10 implants [3]. Three risk factors, namely patients with a history of periodontal disease, poor plaque control, or not following a regular maintenance protocol are identified with a high level of evidence. Implant surface roughness, tobacco, lack of keratinized tissue, and systemic diseases, such as diabetes, also seem to have an adverse effect, though no scientific data are available to this date [4].

One of the objectives of peri-implantitis therapy is to make an effective debridement of all granulation tissue from the area of defect and a decontamination of the exposed surfaces of an implant. Different methods of decontamination of the implant surface, involving mechanical or chemical means, are available. However, at present, no other protocol has demonstrated a higher efficacy and there is a lack of consensus in the literature [5]. The results of non-surgical therapy are inconclusive and surgical procedures demonstrated better outcomes [6].

One critical element to successfully resolve peri-implantitis is to decontaminate the implant surface and this can be affected by implant surface characteristics and geometry [7].

Implantoplasty has been proposed to eliminate the threads and smoothen the implant surface using rotatory instruments. For surgical therapy, a beneficial effect has been reported following implantoplasty associated with flap apicalisation surgery, reconstructive surgery, or a combined surgical approach [8,9].

In addition to being an effective method of decontamination, implantoplasty could limit the risk of relapse of peri-implantitis. Following treatment of peri-implantitis, implant surfaces may become exposed to the oral environment due to bone loss and soft tissue shrinkage during healing [10]. Removal of the rough and affected surface of titanium reduces the potential for biofilm adhesion and thus its maturation on the implant surface [11,12]. In vitro, implantoplasty reduces bacterial recolonization more than other implant surface decontamination techniques [13].

Similarly, the smoothed titanium surface exposed to the oral environment seems easier for the patient to clean and will also be easier to clean for the practitioner during supportive therapy. Albrektsson and Wennerberg suggested that smooth surfaces have an Sa value of <0.5 mm; minimally rough surfaces were identified with an Sa of 0.5–1 mm, moderately rough surfaces with Sa 1–2, and rough surfaces with an Sa of 42 mm [14].

Studies show a similar roughness between a "machined" implant [15] and an implant after an implantoplasty procedure (Ra = 0.39 µm ± 0.13 µm) [16]. In addition, the biocompatibility of these titanium surfaces after implantoplasty seems to be preserved in vitro. Furthermore, obtaining a hydrophilic surface is conducive to bone healing [17].

Indeed, Carcuac et al. showed that at 5 years, there were five times more relapse of peri-implantitis on implants with a rough surface compared with a machined surface [18]. Re-osseointegration on animal studies are contradictory, with some indicating that it would be better with a rough surface [19], while others concluded that it would be better with a smooth surface [20], or even others who did not find any significant differences [21]. These data argue in favour of the realisation of an implantoplasty on the entire implant surface, regardless of the anatomy of the corresponding bone defect, but to our knowledge, there are no animal studies on re-osseointegration after implantoplasty.

Complementary techniques for disrupting the biofilm must therefore be used [22]. Among these techniques, rotating titanium brushes show improvements in the elimination of the biofilm in hard-to-reach areas, with significant results in terms of reducing the depth [23,24].

After the decontamination of the implant surface, several surgical approaches are available, depending on the anatomy of the bone defect: apically repositioned flap (ARP), reconstructive (REC), or combined (COM).

The purpose of this retrospective case series was to assess the peri-implant absence of progressive marginal bone loss after implantoplasty associated with surgical therapy of peri-implant osseous lesions followed by supportive care for 12 months or more.

2. Materials and Methods

Patients were recruited at Dr Arthur Brincat's (AB) private practice in Toulon, France, from April 2018 to October 2020.

Peri-implantitis was defined according to the 2017 World Workshop of Periodontal and Peri-implant diseases: presence of bleeding and/or suppuration on gentle probing (~0.2 N), probing pocket depths of ≥6 mm, bone levels ≥3 mm apical of the most coronal portion of the intraosseous part of the implant based on standardized periapical X-ray [1].

The following inclusion criteria were applied: all patients of 18 years or more with peri-implantitis, without implant mobility, without occlusal overload (checked by occlusion paper in static and dynamic), without active periodontitis nor infectious diseases, without antibiotics treatment in the previous two months, without any systemic disease nor medication known to alter bone metabolism and participating in a recall program with a follow up of ≥12 months. Subjects were excluded because of pregnancy or lactation, and former (<10 years) or current smoking.

The primary or positive outcome was the absence of progressive bone loss. It was evaluated 6 months after the surgery and at one year and once yearly afterwards.

Failure was defined as relapse or progression of the peri-implant lesion, which indicated the dropping out of the implant.

Data Collection

The peri-implant marginal radiographic bone loss (MBL) was determined at TB (baseline) and at latest follow-up examination TF (final) by taking linear measurements (DBSWIN, DÜRR DENTAL®) from the most mesial and distal point of each implant platform to the crestal bone on each intraoral periapical radiograph positioned on a Rinn angulator using a long-cone parallel technique. Characterization of the peri-implantitis defect morphology was classified on radiographs as follows: Class I: infra-osseous defect, Class II: supra-crestal /horizontal defect, and Class III: combined defect [25].

Our collected radiographic data were analysed as a percentage rate at implant and patient levels.

Our study was conducted in accordance with the Helsinki declaration of studies on humans. Before participation, each patient was given a detailed description of the procedure and was required to sign an informed consent form.

Once the diagnosis of peri-implantitis had been made, all patients received a de novo personalized oral hygiene instruction to change hygiene, and behavioural and professional prophylactic supragingival cleaning (Figure 1).

Non-surgical therapy phase consisted in sub-gingival debridement of the peri-implant pocket using specific titanium ultrasonic device (Implant Protect, Satelec Acteon® Merignac, France) 4 to 6 weeks before the surgical procedure.

If necessary, modification of the prosthesis design was made until it was satisfactory.

When possible, screw-retained prostheses were removed to improve access to the contaminated surfaces and repositioned at the end of the surgery, allowing non-submerged healing of all the treated implants.

All surgical procedures were performed under local anaesthesia (Articadent Dentsply-Sirona® Charlotte, NC, USA 4% articaine with 1/100,000 adrenaline) by the same surgeon (AB).

Intrasulcular incisions were made using a microsurgical blade (MJK Instrument®, Marseille, France) and extended one tooth on both sides of the treated implant(s). When necessary, vertical releasing incisions were made to allow better direct access to the peri-implant defect. Full-thickness flaps were raised, and granulation tissue and hard deposits were removed using titanium curettes (IMEDIMPLM, Hu-Friedy®, Chicago, IL, USA) and an ultrasonic titanium device (Implant Protect, Satelec Acteon®).

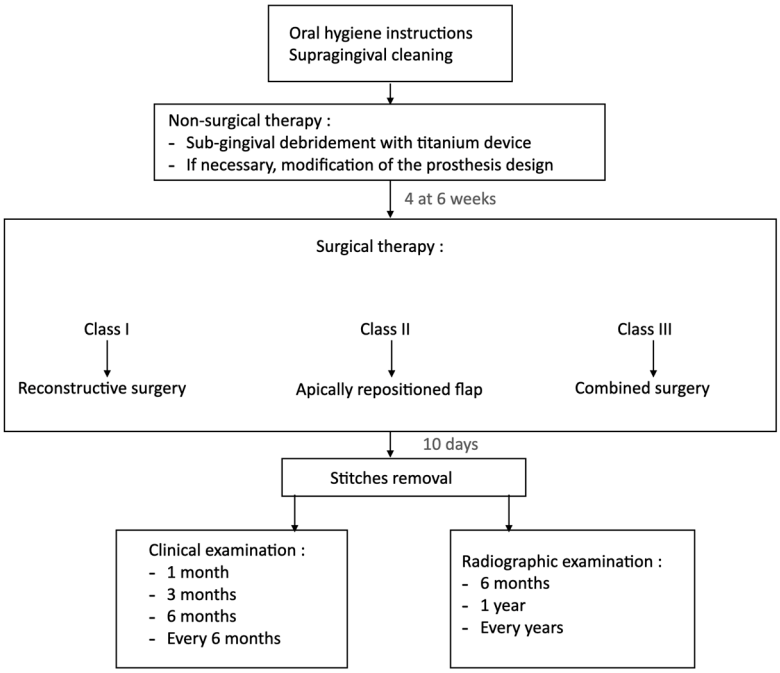


Figure 1. Therapeutic flow-chart.

All the contaminated implant surface accessible to the burs was treated by implantoplasty with a tungsten carbide bur (red H379 and white H379UF, biconvex and conical form, implantoplasty kit, Komet® Paris, France), using abundant sterile saline solution irrigation with a red contra-angle.

Mechanical decontamination was completed using titanium NiTibrushes (ICT Micro and ICT Nano, Hans Korea® Gyeonggi-do, South Korea) and airflow with glycine powder (Air-N-Go® PERIO Acteon® Merignac, France) [26,27].

Chemical decontamination was carried out with 2 min rinses of chlorhexidine (0.12%) [28,29]. After these decontamination procedures, the surgical sites were profusely irrigated with sterile saline solution to remove residual titanium particles (Figure 2).

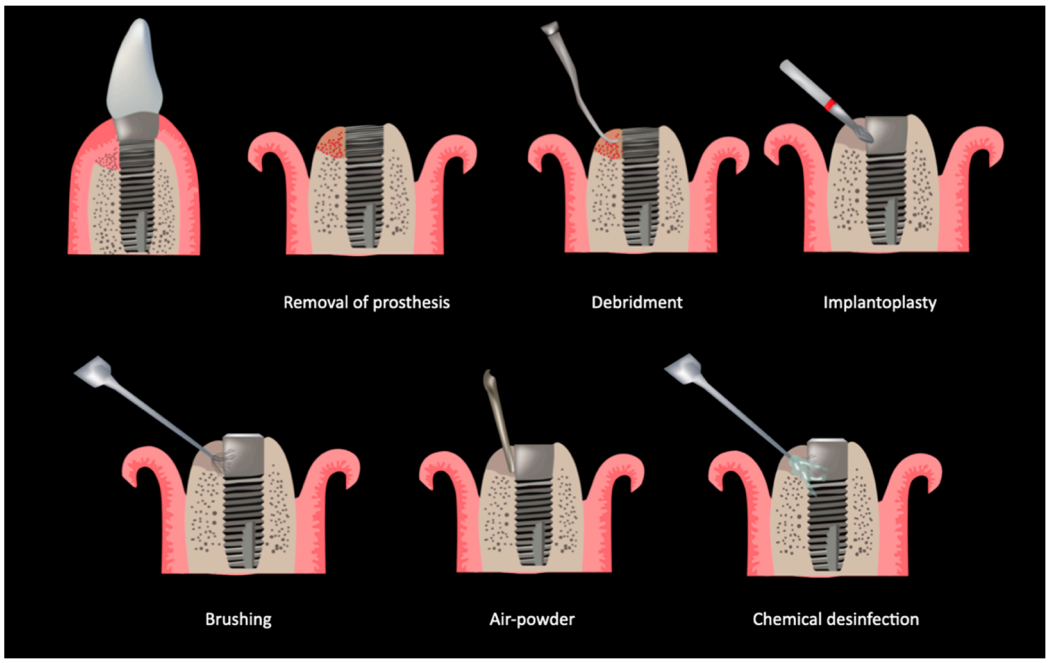


Figure 2. Surgical steps for decontamination of implant surface affected by peri-implantitis.

Depending on the defect morphology, the surgical design/approach was chosen among three different modalities (Figures 3–5) before beginning the surgical procedure.

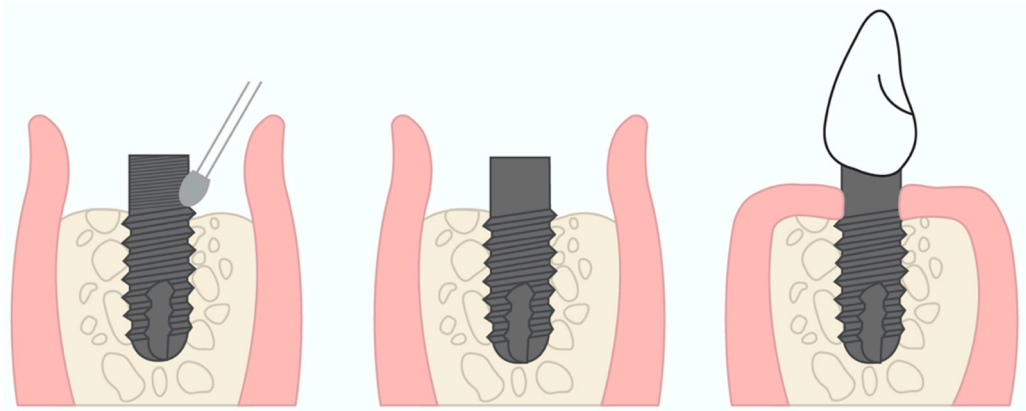


Figure 3. Implantoplasty for non-osseous treatment of peri-implantitis.

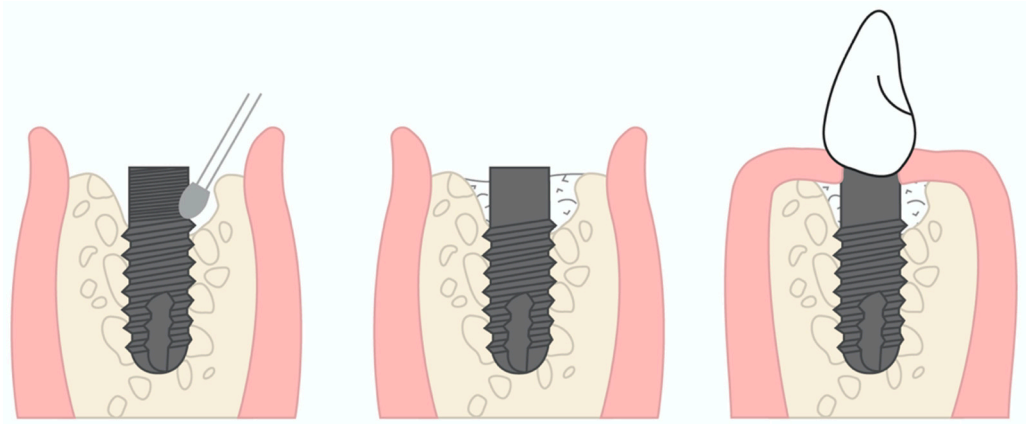


Figure 4. Implantoplasty for reconstructive treatment of peri-implantitis.

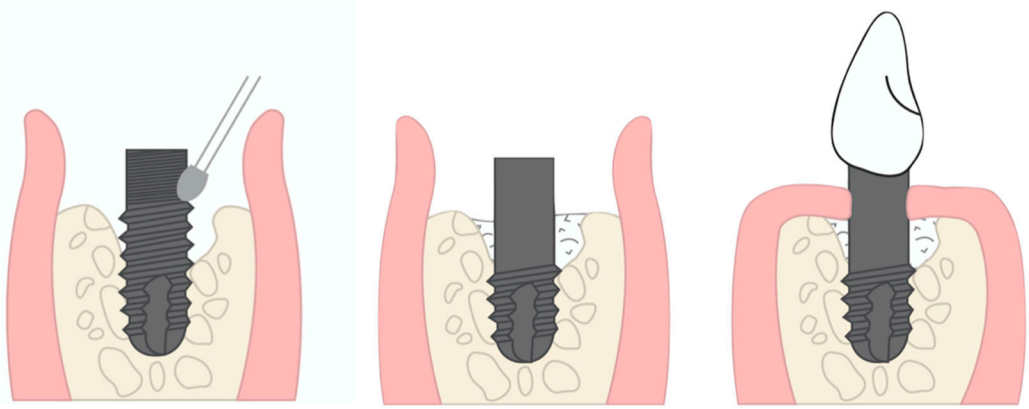


Figure 5. Implantoplasty during combined treatment of peri-implantitis.

All the infra-osseous defects were filled with xenograft (Symbios Xenograft, Dentsply-Sirona® Charlotte, NC, USA) or alloplastic (Symbios Biphasic, Dentsply-Sirona®), when the patient refused the animal origin's graft and when it was necessary to stabilize bone-filling materials, covered by a resorbable membrane (Creos Xenoprotect, Nobel Biocare® Kloten, Switzerland or Ossix Plus, Datum Dental® Lod, Israël). Flaps were repositioned and closed with horizontal mattress and single interrupted sutures (Arago PRED® Arcueil, France non-resorbable monofilament 5.0 nylon) (Figures 6 and 7).

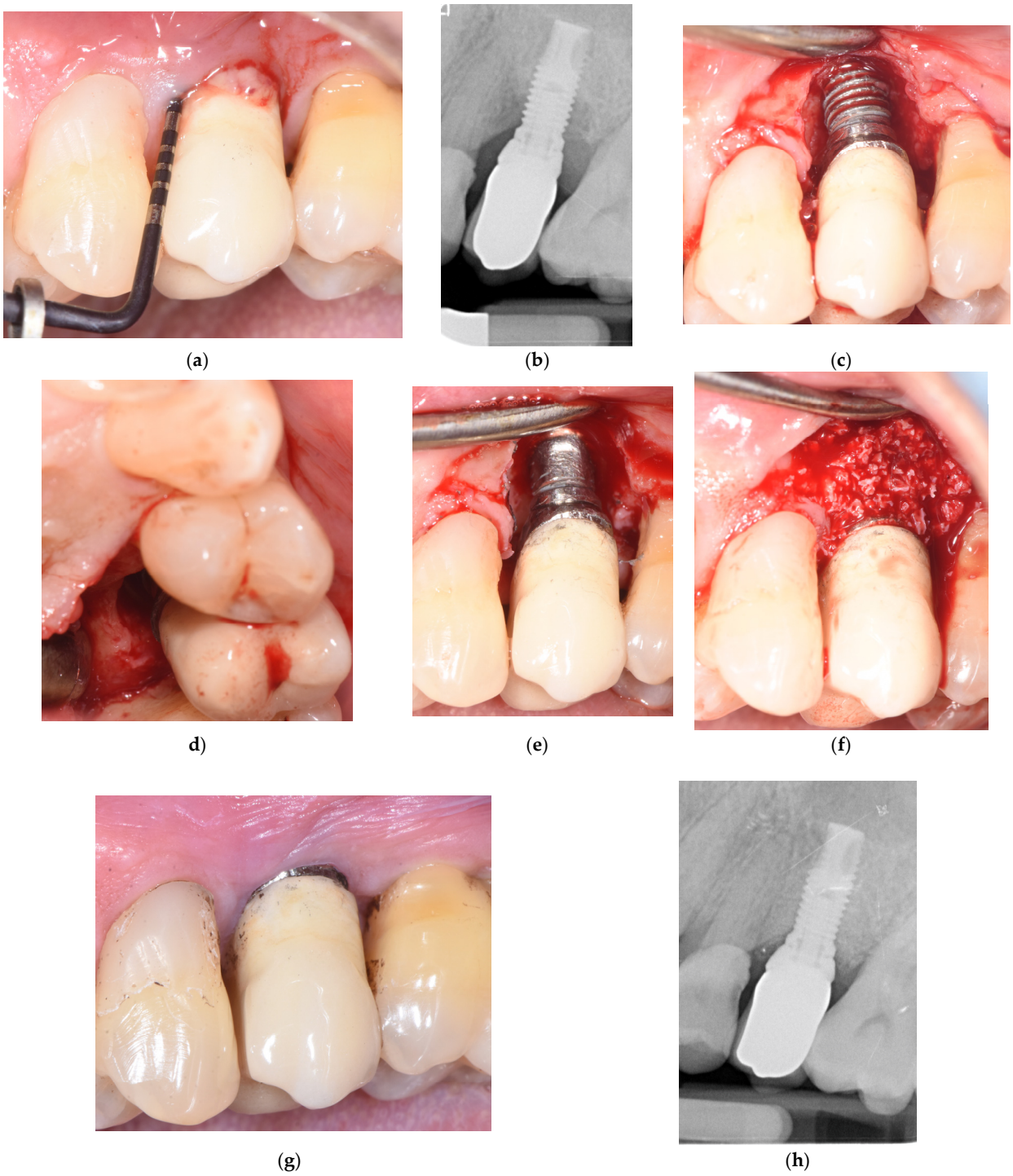


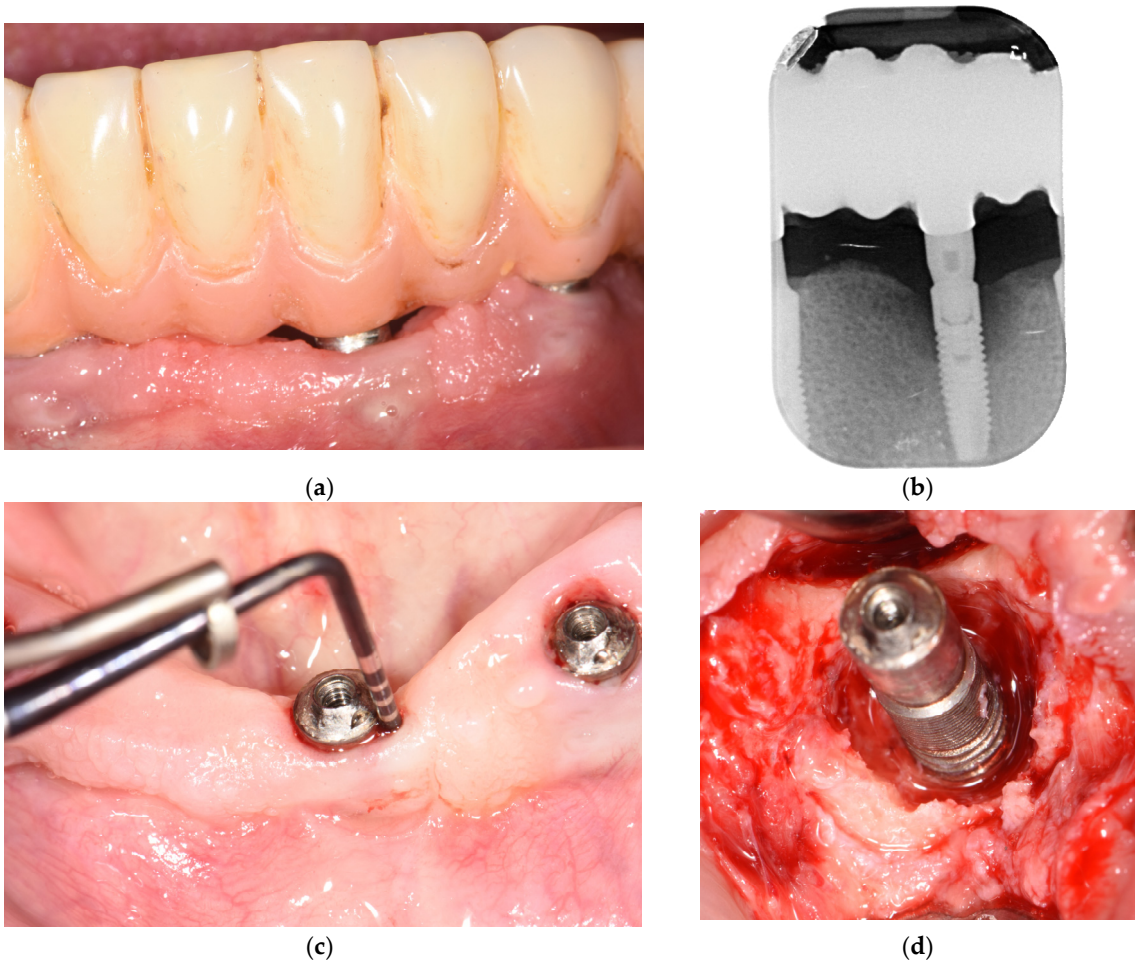
Figure 6. Peri-implantitis on implant n°25: initial situation with suppuration (**a**) and radiographic bone loss (**b**), intra-operative view showing peri-implant defect (class I) (**c**,**d**), implantoplasty procedure on all the accessible implant surIes (**e**), Symbios Xenograft (Dentsply-Sirona® Charlotte, NC, USA) is used in intra-bony defect (**f**), and the clinical (**g**) and radiographic (**h**) result at 12 months.

Non-osseous surgery was carried out neither in the vestibular nor in the lingual areas. The flap was positioned apically to the residual bone level in the palatal area. A gingivectomy was performed when necessary. The mucosal flap was apically repositioned with vertical mattress and single interrupted sutures.

In the presence of both supra- and infra-bony lesions, a combined REC and ARP approach has been performed at the same surgical time. All implants underwent a non-submerged approach during the healing process.

All patients received postoperative care instructions. Antibiotics (2 g of amoxicillin in two doses starting the day before the surgery and for 7 days), prednisolone (1 mg/kg/day from the morning of the surgery and lasting for 3 days), and paracetamol (1 g every 6 h for 3 days) were prescribed. From the day of the surgery, rinsing with chlorhexidine for 1 min twice daily during 7 days with a 0.2% chlorhexidine solution followed by 7 more days with a 0.12% chlorhexidine solution was also prescribed. Patients were asked to apply chlorhexidine gel (0.2%) on the treated zone for 10 days with an extra-soft toothbrush instead of the mechanical brushing of this region. At 10 days, sutures were removed.

Clinical examinations were assessed at 1 month, 3 months, 6 months, and every 6 months onwards. At the recall visits, oral hygiene instructions were reinforced, and careful professional supragingival cleaning was performed. A probing and a radiographic examination were performed at 6 months, 1 year, and then on a yearly basis.



(**a**) (**b**)

(**c**) (**d**)

Figure 7. *Cont.*

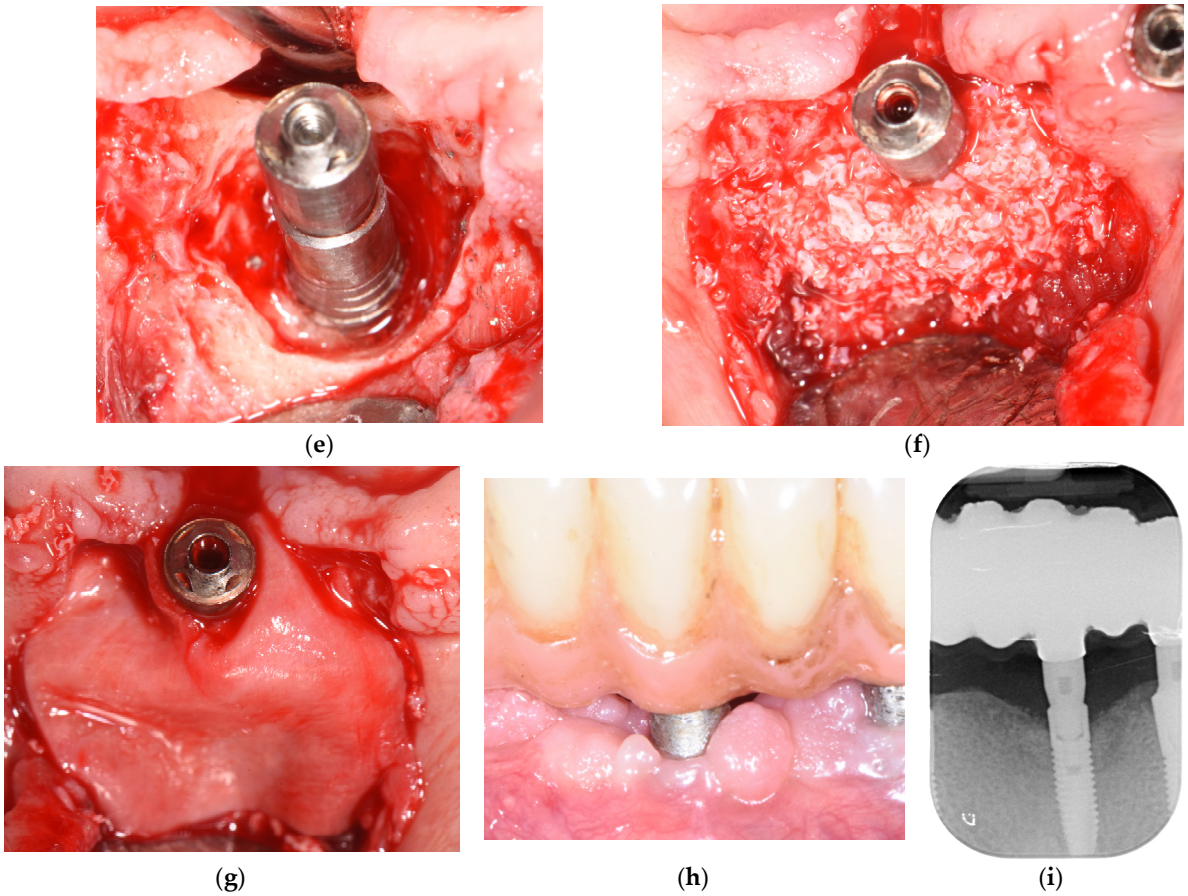


Figure 7. Peri-implantitis on implant supporting full-arch rehabilitation, initial clinical (**a**) and radiographic (**b**) situation, removal of the screw retained prosthesis and probing (**c**), intra-operative view showing a class I defect (**d**), implantoplasty on all the accessible implaIsurfaces (**e**), xenograft (Symbios Xenograft Dentsply-Sirona® Charlotte, NC, USA) used for bone grafting (**f**), resorbable membrane (Creos Xenoprotect, Nobel Biocare® Kloten, Switzerland) (**g**), and clinical (**h**) and radiographic (**i**) result at 12 months.

3. Results

3.1. Demographic Data

Fifty-seven patients (mean age = 66.2, SD = 10.5) and ninety-one implants were assessed with a mean ratio of 1.6 implant/patient.

Mean follow up was 24 months (range = 12 to 42 months).

A total of 14% of peri-implantitis defects were class I treated by REC, 55% of peri-implantitis defects were class II treated by ARP, and 31% were class III treated by COM (Table 1).

Table 1. Distribution of peri-implant osseous defects and type of procedure.

	Class I	Class II	Class III
N (implants) = 91	13	50	28
%	14	55	31
Procedure	Reconstructive	Apically repositioned flap	Combined

3.2. Absence of Progressive Marginal Bone Loss (MBL) at Implant Level

A total of 96.7% of the overall treated implants did not have progressive bone loss (Table 2).

Table 2. Peri-implant failure and absence of marginal bone loss rate.

	Reconstructive	Apically Repositioned Flap	Combined	Total
Failure (implant)	0	2	1	3
Failure (patient)	0	1	1	2
Absence of marginal bone loss rate (implant level)	100%	92.9%	98%	96.7%
Absence of marginal bone loss rate (patient level-)	100%	98.2%	98.2%	96.5%

The rate was 100% in the class I lesions sub-group treated by REC. The rate was 92.9% in the class II lesion sub-group treated by ARP. The rate was 98% in the class III lesion sub-group treated by COM.

Two failures occurred in class II and one in class III but none in class I. The three implants were dropped out.

3.3. Absence of Progressive Marginal Bone Loss (MBL) at Patient Level

A total of 96.5% of the included patients did not have progressive bone loss. Three failures occurred in two different patients and had to be dropped out (Table 2) (Figure 8).

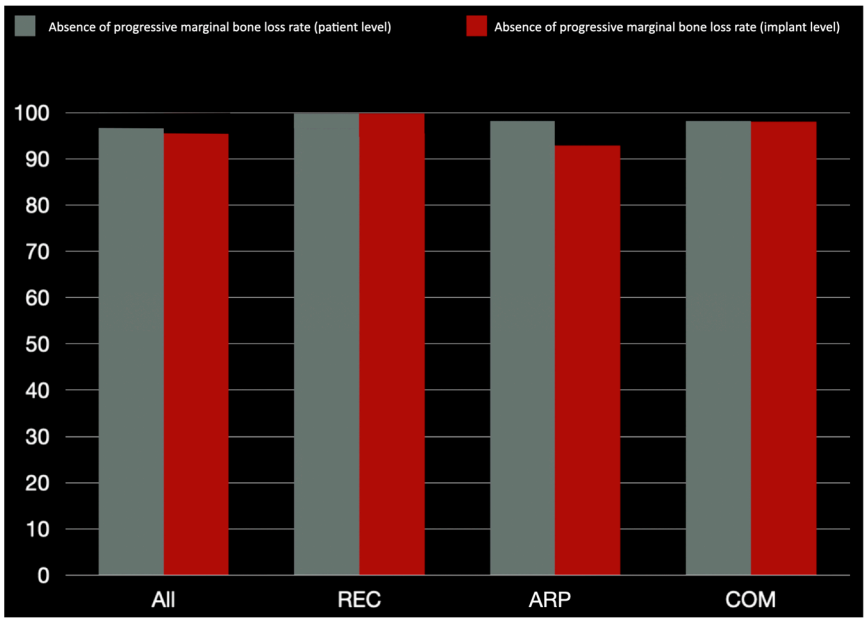


Figure 8. Percentage of implant or patient showing absence of progressive bone loss. REC: reconstructive, ARP: apically repositioned flap, COM: combined.

4. Discussion

In our study, which involved 91 implants and had a mean follow-up period of 24 months, the absence of progressive bone loss around the implants was observed at a rate of 96.7% at the implant level and 96.5% at the patient level when considering all types of surgical treatment combined with implantoplasty.

In the subgroup that underwent reconstructive surgical modalities, a 100% absence of progressive bone loss rate was achieved at both the implant level and patient level.

For the subgroup treated with apically repositioned flap, a rate of 92.9% absence of progressive bone loss was observed at the implant level, while it reached 98.2% at the patient level.

In the subgroup that underwent a combination of flap apicalisation and reconstructive surgery, an absence of progressive bone loss rate of 98% was achieved at the implant level and 98.2% at the patient level.

The main criticism of our study is that the implantoplasty procedure can diffuse titanium particles into the tissue surrounding the peri-implant lesion. However, in biopsies of peri-implant inflammatory lesions, titanium particles are frequently found [30]. According to Mombelli et al., titanium, in small quantities, has no influence on the tissue but can cause inflammation if its concentration increases [31]. To minimise these side effects caused by titanium particles, we rinsed the surgical site thoroughly and used a strong surgical suction close to the drill.

The advantages of our study lie in the large number of subjects (57), which to our knowledge is the largest number of patients evaluated in this respect. The large number of implants (91) evaluated is also an advantage in a case series. Only one study (Monje et al.) [32] evaluated more implants (135) but on a smaller number of patients (43). All other studies evaluated fewer patients and fewer implants (Lasserre et al. [33], Romeo et al. [34,35], Dalago et al. [36], Ravida et al. [37], Englezos et al. [38], Matarasso et al. [39], Bianchini et al. [40,41], Nart et al. [42], Suh et al. [43], Austoni et al. [44], Sapata et al. [45], Pommer et al. [46]).

Our study was based on routine clinical practice and gives an insight into the results that can be expected in a routine peri-implantitis treatment activity.

Our mean follow up was 2 years, the same as Englezos et al. [38] and Monje et al. [32], whereas in many studies the follow up was only one year (Ravida et al. [37], Nart et al. [42], Austoni et al. [44], Matarasso et al. [39]) or less (6 months, Lasserre et al. [33]). This may be a disadvantage of our study as this follow up should be increased to more than 3 years, as did Romeo et al. [34,35], Dalago et al. [36], and Bianchini et al. [40], or even more (9 years, Pommer et al. [46]) to know the long-term effects of the treatment.

Due to heterogeneity in study designs, patient characteristics, materials used (the use or not of membranes and different types of bone grafts/bone substitutes), evaluation methods, and follow-up duration, comparisons with previous studies were difficult.

First, some studies have used other associated criteria to assess the success rate after surgical treatment of a peri-implant lesion. Among the criteria evaluated, all these studies assessed the absence of increased peri-implant bone loss called “bone stability” (Bianchini et al., 2020) [40], the ”absence of progression of decrease in peri-implant crestal bone level” (Dalago et al.) [36], “no ongoing bone loss” (Englezos et al.) [38], “peri-implant bone levels remained stable over time” (Sapata et al.) [45], or “no further bone loss” (Lasserre et al.) [33]. Only one study measured “bone regeneration” (Suh et al.) [43].

For the studies that used the survival rate criterion (Romeo et al. [34,35], Ravida et al. [37], Nart et al. [42], Pommer et al. [46], Matarasso et al. [39]), it corresponded to implants that were not removed/lost.

In our study, 96.7% of the overall treated implants did not have progressive bone loss.

Compared with our results, some studies (Romeo et al. [34,35], Dalago et al. [36], Matarasso et al. [39], Austoni et al. [44], Suh et al. [43], Sapata et al. [45], Nart el al. [42], Bianchini et al. [40]) described a 100% implant survival rate. These higher results can be explained by the fewer number of implants treated (19, 9, 11, 2, 6, 2, 17, 4, respectively). On the other hand, some studies showed lower results, 70% (Lasserre et al.) [33], 95.3% (Monje et al.) [32], 90% (Ravida et al.) [37], 92.5% (Englezos et al.) [38], 87% (Pommer et al.) [46], and 87% (Bianchini et al.) [41], which can be explained by the fact that they did not use the same assessment criteria or the difference could be explained by the lower number of implants, 30 (Bianchini et al.) [41] and 70 (Pommer et al.) [46], compared with our 91 treated implants.

A longer follow up of 9 years (Pommer et al.) [46] and 2 to 6 years (Bianchini et al.) [41] could also have an impact on the results.

In our study, 96.5% of the included patients did not have progressive bone loss.

Compared with our results, Monje et al. [32] showed a patient-level implant survival rate of 95.3% and Bianchini et al. [41] showed a patient-level “disease resolution” of 83%. These lower results could be explained by the fact that they did not use the same evaluation

criteria (implant survival rate; bleeding/suppuration on probing and pain on peri-implant palpation, respectively).

In our study, a 100% no-marginal-implant bone loss rate was obtained for the reconstructive procedure, whereas this rate was 90% in the study of Monje et al. [32] This lower result could be explained by a difference in the location of the implantoplasty, which they performed only on the supraosseous surface of the implant, whereas we also performed it in the intraosseous part.

In our study, a 92.9% of no-marginal-implant bone loss rate was obtained for the flap apicalisation procedure. Some studies showed a 100% implant survival rate (Monje et al. [32], Romeo et al. [34,35] Dalago et al. [36] Bianchini et al. [40]). These higher results could be explained because in our study two implants failed in the resective surgery group and probably because of the higher number of implants treated by Monje et al. (104 implants) than our study (50 implants).

Two studies (Pommer et al. [46] Bianchini et al. [41]) showed an implant survival rate of 87%. These lower results could be explained by the longer follow up of 9 years (Pommer et al.) [46] or because the use of clinical parameters for establish disease resolution rate (Bianchini et al., 2019) [41].

In our study, a 98% of no-marginal-implant bone loss rate was obtained for combined procedure which is different from the results obtained by Matarasso et al. [39] who obtained 100% probably because their follow up was shorter (12 months) than ours. The results obtained in our study should be interpreted with caution due to the short follow-up time (mean $\approx$ 2 years), and the bias consisted in the duration difference which varies from 12 to 42 months depending on the patient. The efficacy of surgical treatment in combination with implantoplasty of peri-implant lesions in the long term is still unknown. Studies with longer-term follow up should be undertaken in the future. It is known that the case series is not the study design that yields the most powerful scientific results; however, our large case series is representative of current practice in the treatment of peri-implant osseous lesions.

Bias inherent to the treatment protocol must be disclosed: removing prosthesis was not always possible and therefore sometimes surgery was performed with the prosthesis in place. Bone-filling graft and biomaterials are not always the same for philosophical, religious, or ethical considerations related to the patient. Membranes were not systematically used to stabilize bone-filling materials according to Monje et al. [47]. It is important to mention that the chemical decontamination included in the surgical protocol of this study was empirical since, so far (January, 2023), no study has been able to show the efficacy of the tested antibacterial agents in decontaminating the implant surfaces. Implantoplasty, by mechanically and macroscopically removing a layer of the implant surface, effectively removes the adhering biofilm. This method of decontamination, although imperfect and rudimentary, could only be indicated until a chemical that is non-toxic to the peri-implant tissues but sufficiently disinfectant to remove biofilm from implant surfaces is found/discovered.

Comparison of different methods of implant surface decontamination versus implantoplasty in randomized trials would allow us to propose, ideally in the very near future, a therapeutic gold standard for peri-implant lesions or at least reliable evidence-based clinical recommendations.

Progressive bone loss rate based on radiographic analysis criteria alone have limitations due to its two-dimensional nature which restricts analysis to mesial and distal sites and does not allow assessment of the buccal or lingual/palatal marginal bone. For future research, it would be necessary to perform a multifactorial clinical assessment of parameters that may reveal signs of inflammation, infection, or healing, such as mean PPD reduction $\geq$ 0.5 mm, absence of a peri-implant site with PD $\geq$ 5 mm with concomitant bleeding or suppuration on probing, recession height, percentage of bone fill, and keratinized mucos height.

Comparative statistical analysis of the three groups was not carried out because, firstly, the groups were not comparable in terms of anatomy of bone loss and, secondly, the

anatomy of bone loss was an indication for the choice among the three surgical protocols used.

In our study, 96.7% of treated implants showed marginal bone stability. To refine these results, it would be necessary to compare two groups of surgically treated peri-implant bone lesions with or without associated implantoplasty. In addition, other criteria such as depth of probing, clinical attachment level, BOP, recession height, keratinized mucosa height, and percentage of bone fill, should be measured in addition to the absence of progressive bone loss.

Larger prospective case series with longer-term follow up on comparable populations (plaque score, compliance, age, general health status, non-smokers) should be conducted to evaluate the therapeutic efficacy of this surgical protocol on peri-implant bone lesions.

5. Conclusions

The present case series demonstrates that 96.7% of implants show absence of progressive bone loss following peri-implantitis surgical therapy with implantoplasty over a follow-up period of 12 to 42 months (mean = 24 months).

The decontamination of the implant surface poses a significant therapeutic challenge in the management of peri-implantitis. However, our surgical treatment protocol has demonstrated promising results, enabling our patients to retain their implants. The success of our approach can be attributed to several factors. Firstly, our protocol takes into account the morphology of the defects, allowing us to tailor the treatment accordingly. By considering the specific characteristics of each individual case, we can optimize the surgical procedures to effectively address the peri-implantitis condition. Additionally, long-term follow up plays a crucial role in assessing the therapeutic success of our protocol. By closely monitoring patients over an extended period, we can evaluate the stability and health of the treated implants, providing valuable insights into the long-term outcomes. Furthermore, the combination of meticulous decontamination techniques and appropriate treatment procedures contributes to the positive outcomes observed in our study. Our protocol emphasizes the thorough decontamination of the implant surface, minimizing the bacterial load and promoting the regeneration of peri-implant tissues.

Within the limits of our current study, we believe that implantoplasty during debridement surgery for peri-implant bone lesion can maintain marginal bone stability for at least one year.

Further studies are needed and should be conducted to evaluate the effect of implantoplasty combined with surgical debridement of the peri-implant bone lesion on the long-term absence of progressive peri-implant bone loss.

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