Treatment of peri-implant disease : current knowledge

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Chapter

Treatment of Peri-Implant Disease: Current Knowledge

Alexandre Perez and Tommaso Lombardi

Abstract

Peri-implant diseases represent serious complications after dental implant treatment, affecting both the surrounding soft and hard tissue. Regular, specific check-ups with the assessment and elimination of risk factors are effective precautions, and various conservative and surgical approaches are available for the treatment of peri-implant diseases. Early detection of clinical signs diseases and timely treatment are important for the success of dental implant treatment. Dentists must be familiar with the radiological and clinical characteristics of both conditions to make an accurate diagnosis and determine the appropriate treatment. The purpose of this chapter is to provide advice on the diagnosis and prevention of peri-implant disease for practitioners and to offer an overview of current knowledge on the treatment of peri-implant diseases.

Keywords: peri-implantitis, peri-implant mucositis, treatment, dental implants, periodontal disease

1. Introduction

Clinically, peri-implant mucositis is characterized by bleeding on gentle probing (<0.25 N), erythema, swelling, and/or suppuration [1–3]. As plaque is the primary etiological factor, effective treatment relies on mechanical plaque removal and ongoing plaque control, which can lead to significant improvement or complete resolution of the condition [4, 5]. The primary clinical endpoint in such interventions is the resolution of inflammation, typically assessed by the reduction or elimination of bleeding on probing, evaluated 2–3 months posttreatment [6, 7]. However, it is important to recognize that the development of peri-implant mucositis and its progression to peri-implantitis represent a pathological continuum. This dynamic may influence treatment outcomes and lesion responsiveness, particularly when distinguishing between short- and long-standing lesions [3].

Causative peri-implant mucositis treatment ideally comprises a combination of professionally administered mechanical or physical plaque removal and patient-administered plaque control [8, 9]. Professional nonsurgical treatment of peri-implant mucositis can be provided by dental hygienists, general dentists, and specialists [10]. While various treatment approaches are routinely employed in clinical practice, a universally accepted standard of care, apart from submarginal instrumentation, to treat peri-implant mucositis remains pending [2].

Professionally administered plaque removal (PAPR) aims to reduce soft tissue inflammation by removing inflammatory hard and soft deposits from the implant surface and/or superstructure [9]. Modalities and instruments that prevent damaging the smooth transmucosal components, potentially aggravating future plaque accumulation (e.g., implant collar, abutment), should be chosen [11]. Commonly used instruments and modalities include ultrasonic scalers with plastic or carbon fiber tips, air-polishing, plastic, carbon, or titanium curettes, rotating brushes, and lasers [12]. There is limited evidence to support or refute the effectiveness of repeated PAPR procedures in improving clinical parameters related to peri-implant mucositis [13]. Clinicians may also consider modifying the restorative contours, verifying the absence of excess cement, or motivating patients to cease smoking as part of the overall treatment strategy [10, 14, 15]. Furthermore, systematic reviews evidence low effectiveness of professional adjunctive photodynamic therapy, NaOCl, or chlorhexidine use as part of PAPR, while the administration of systemic antibiotics, whilst proving efficient, remains unjustifiable [10, 16, 17]. Although peri-implant mucositis is generally considered a reversible condition, complete resolution is rarely achieved across treatment modalities. Despite significant reductions in bleeding on probing and clinical signs of inflammation, residual signs of the disease frequently persist, highlighting a recurring limitation of current therapeutic approaches [4, 9, 18].

In addition to professional plaque removal, it is essential to implement and reinforce patient-administered plaque control (PAPC) routines [8]. The importance of PAPC has been illustrated within studies, demonstrating that peri-implant mucositis can be induced by oral hygiene routine suspension. In contrast, re-institution of oral hygiene has been shown to reverse disease after a period of >3 weeks [4, 19]. Individual routines comprise mechanical plaque removal using manual or powered toothbrushes, potentially in combination with oral irrigators or interdental brushes; chemical plaque control with adjunctive delivery of antimicrobials or probiotics, and triclosan-containing toothpaste. The application and combination of individual routines show great variability, without pronounced evidence to support a consensus for a single, specific standard of care, except for brushing [2, 8]. Some of the more recent reviews support the general additional benefit of adjuncts, comprising, for example, probiotics or oral irrigators as part of PAPC [18].

A recurring finding in peri-implant mucositis treatments is that complete disease resolution is unlikely to be achieved [2, 18]. This finding reinforces the importance of strict and periodic recall programs to ensure early detection and treatment to prevent disease progression [9, 18]. Patients may further benefit from individual oral hygiene instructions tailored to the individual patient profile and type of restoration to prevent plaque accumulation.

2. Treatment of peri-implantitis

The treatment of peri-implant pathologies remains a significant clinical challenge. While numerous studies have reported favorable outcomes, there is currently insufficient evidence to determine the most effective therapeutic modality or to define a universally accepted "gold standard" protocol [10, 20, 21]. Treatment approaches, techniques, and outcomes remain highly variable, reflecting the multifactorial and heterogeneous nature of peri-implantitis. This chapter aims to provide clinicians with a structured overview of current treatment strategies, modalities, and diagnostic criteria to support evidence-based decision-making.

2.1 General approach to peri-implantitis treatment

Despite distinct differences in treatment response, the treatment of peri-implantitis typically follows a stepwise approach, mirroring established protocols from periodontal therapy [22–24]. Initial causative, nonsurgical therapy aims at reducing the infection and peri-implant inflammation. This phase is—if indicated—followed by a corrective surgical therapy to reduce probing depths, restore tissue homeostasis, and improve access for maintenance. The third treatment phase consists of supportive Peri-Implant Therapy (SPIT). This phase represents the cornerstone of secondary prevention to ensure regular examination, professional hygiene, and reinforcement of oral hygiene routines to reduce the risk of disease recurrence (**Figure 1**) [10].

2.2 Phase I causative nonsurgical therapy

Initial-phase therapy mainly focuses on preserving the implant-supported restoration when clinically feasible. Its primary aim is to control infection and inflammation, manage potential risks, and ensure patient compliance before potentially escalating into more invasive interventions [22, 25, 26]. Initial nonsurgical therapy should also serve as an opportunity to identify, reassess, and manage existing and newly recognized risk factors. This includes correcting local contributing factors—such as removal of excess cement or modification of plaque-retentive prosthetic features—and reinforcing patient-related behavioral changes, including improved oral hygiene practices and support for smoking cessation. Additionally, systemic risk factors should be addressed, for example, by optimizing glycemic control in diabetic patients or initiating concurrent periodontal treatment in individuals with poorly controlled periodontitis [20, 22, 25–30].

The therapeutic principles of causative nonsurgical therapy are very similar to those applied during peri-implant mucositis treatment. Among available interventions, submucosal debridement using mechanical instrumentation—with or without adjunctive therapies—remains the most evidence-based approach.

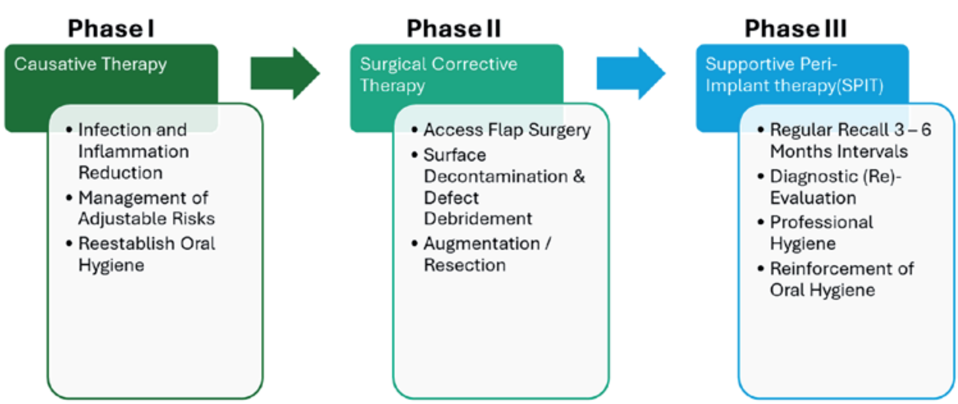

Figure 1.
Schematic overview of the stepwise treatment approach for peri-implantitis, comprising Phase I (causative therapy), Phase II (surgical corrective therapy), and Phase III (supportive peri-implant therapy, SPIT).

Adjuncts may include antiseptics, antibiotics, or laser applications, though current evidence suggests limited additional benefits compared to submarginal instrumentation alone [31]. Current treatment guidelines discourage the use of lasers [10]. While effective in biofilm disruption, air-polishing systems have been associated with a higher incidence of adverse effects, such as pain and subcutaneous emphysema, and should be used with caution [32–35]. Furthermore, the effect of adjunct systemic antibiotics has proven statistically significant and clinically meaningful, and may be carefully considered to improve treatment outcomes of nonsurgical therapy in severe cases [36, 37].

Clinical endpoints for nonsurgical peri-implantitis therapy should be assessed 6–12-weeks posttreatment and comprise the resolution of inflammation (absence of bleeding on probing and suppuration), the reduction in probing depths (ideally to ≤5 mm), improved plaque control and oral hygiene, and satisfactory cleanability of the prosthesis [20, 22, 25, 26]. While the complete resolution of the disease may not be achieved at this stage, stabilization of the condition, risk factor adjustment, and preparation for potential surgical intervention are key objectives of this treatment phase. In general, nonsurgical therapy may be effective in treating peri-implant mucositis and early-stage peri-implantitis while lacking efficiency in treating severe peri-implantitis [10, 24, 38–40].

2.3 Corrective surgical therapy

Refractory sites presenting with an increased probing depth of ≥5 mm, as well as persistent signs of inflammation, that is, BoP and suppuration, are indicated for corrective surgical therapy [10, 30]. This second phase of therapy comprises access flap surgery, potentially followed by reconstructive or resective procedures. Specifically, surgical therapy primarily aims to provide access to the implant surface to facilitate defect debridement, that is, removal of inflamed granulation tissue, as well as cleaning and decontaminating the implant surface from any bacterial deposits and calculus to achieve complete resolution of the inflammatory lesion [30]. Procedures may include tissue resection and/or augmentation of peri-implant osseous defects. They may vary based on the use of techniques and adjuncts for surface decontamination, the use of biomaterials, biologics, and their combination, and local or systemic adjunctive antibiotics and antimicrobials [10, 39] .

A reduction in probing depth to ≤5 mm and the absence of bleeding on probing (BoP) at 6 months post-surgery, along with the absence of progressive bone loss as assessed by radiographs at 12 months postoperatively, represent generally accepted clinical endpoints of surgical therapy [41, 42]. Notably, ensuring adequate compliance with oral hygiene procedures is essential before commencing intervention [24]. Clinicians should also be aware that, despite considerable improvement of clinical and radiographic parameters that may be achieved after surgical therapy, a considerably high rate (32–44%) of disease recurrence and a considerable rate (14–21%) of implant loss within 5 years posttreatment were reported [30, 41, 43, 44].

2.3.1 Anatomic factors affecting treatment outcomes: Peri-implantitis defect classifications

Attempts have been made to classify peri-implant osseous defects to describe disease severity and to estimate peri-implant-treatment effectiveness [44–46]. The most widely accepted peri-implant defect classifications differentiate between purely infraosseous (Class I), purely supracrestal, that is, horizontal (Class II), or combined defects, that is, presenting a horizontal and an infraosseous component (Class III). Furthermore, the infraosseous components are differentiated into buccal dehiscence (a-type), 2–3 defects (b-type), and circumferential saucer-shaped defects [47]. Finally, the defect depth extension below the implant neck, or the ratio of bone loss relative to the total length of the implant, may be used to differentiate between small (S) defects (3–4 mm), medium (M) (4–5 mm) to advanced (A) defect (≥ 6 mm) types (**Figure 2**).

Defect classification may help clinicians in guiding treatment decisions and estimating treatment prognosis. Small supracrestal Class II defects, for example, have shown to respond well to nonsurgical approaches or to access flap, resective surgery. Conversely, infraosseous Type Ic defects have responded better to guided bone regenerative approaches than buccal dehiscence defects (Class Ib) [46]. Advanced, i.e., > 7 mm deep defects, have been described as less responsive to surgical therapy (**Figure 3**) [48].

2.3.2 Peri-implant surface detoxification

Given the overwhelming evidence classifying peri-implantitis as a biofilm-associated disease, effective implant surface disinfection and complete decontamination appear crucial to achieving disease resolution [1, 20, 38, 49, 50]. Seminal studies have demonstrated that re-osseointegration and defect fill following regenerative approaches, even after implant surface decontamination, remain incomplete—highlighting the inherent challenge of achieving complete biofilm removal from contaminated implant surfaces [50–52]. Microroughened surface topographies, while established as the gold standard to promote osseointegration, may hinder effective decontamination further [53–55].

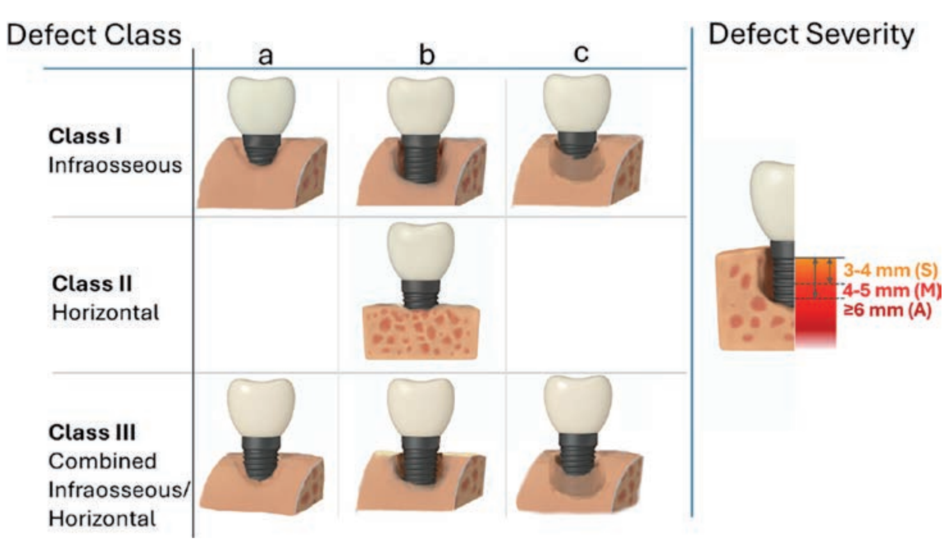

Figure 2.
Peri-implantitis defect type (left) and severity classification (right). Defect types are categorized by defect morphology: buccal dehiscence (a), 2–3-wall (b), and circumferential (contained) (c) defects and by the presence of infraosseous and/or supracrestal components (Class I–III). Defect severity is based on the vertical extension between the implant shoulder and the lesion's apex.

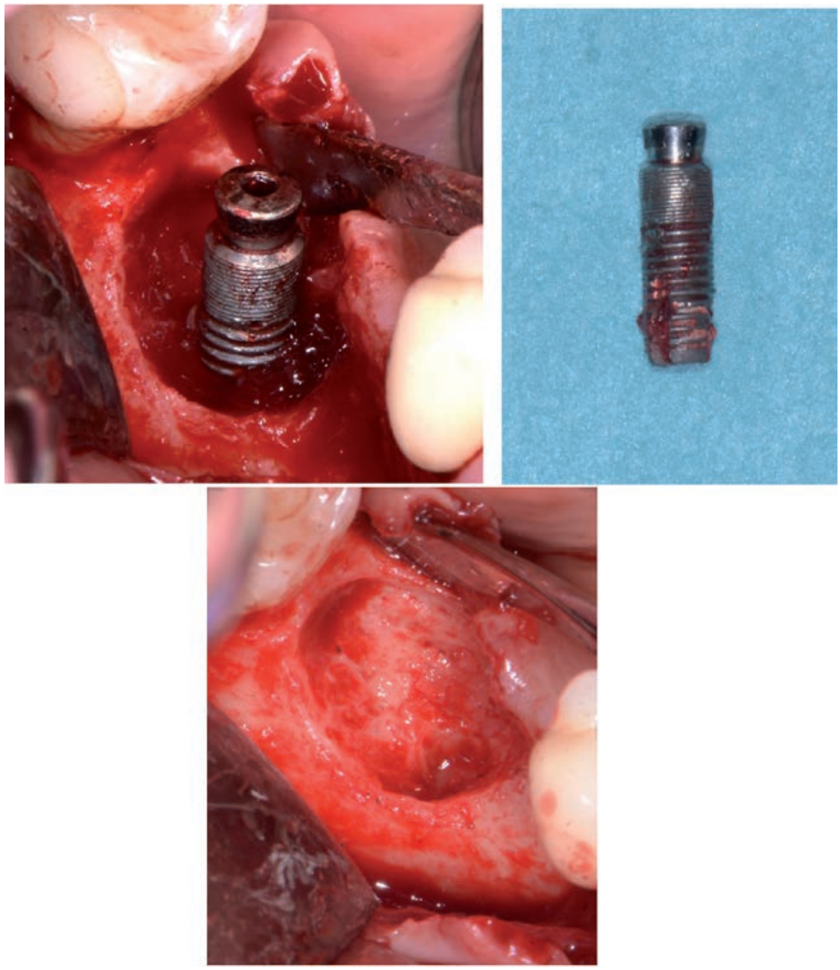

Figure 3.
Peri-implantitis defect type III (> 7 mm deep defects) and treatment (explantation).

A multitude of mechanical, chemical, and physical methods, with or without adjunctive local application of antiseptics or antibiotics, have been proposed [10, 39, 56, 57]. In contrast to nonsurgical approaches with limited access and visibility, open-flap mechanical instrumentation has been demonstrated as effective in removing plaque and calculus deposits [58]. Individual methods include scaling with metallic and nonmetallic scalers and rotating brushes or air powder abrasion. Implantoplasty using rotary instruments may be applied to remove the coronal implant threads and smooth the implant surfaces to prevent future plaque accumulation as part of apically repositioned flaps (APF) or guided bone regenerative (GBR) procedures [38, 59–61].

Residual biofilm following instrumentation has been described as common after mechanical debridement. The release of titanium particles remains a general concern [58, 62, 63]. Clinicians often complement mechanical methods with a second chemical or physical technique to improve surface decontamination [57, 58]. Physical methods include, for example, photodynamic therapy, laser-assisted techniques, or electrolytic methods. In contrast, chemical strategies may include rinsing with saline, application of hydrogen peroxide, ethylenediamine tetra acetate (EDTA) or local antibiotics or antiseptics, for example, tetracycline or chlorhexidine [57, 58, 64]. Despite considerable efforts to enhance implant surface decontamination as a prerequisite for re-osseointegration, evidence supporting the efficacy of individual techniques—or their combinations—in improving key clinical outcomes of surgical regenerative or resective approaches remains limited, except for modest support for the use of titanium brushes [39, 50, 57, 65, 66]. Notably, this finding may partly reflect the complex and heterogeneous nature of peri-implantitis, involving a broad spectrum of contributing factors, notably treatment site characteristics, implant design, defect morphology, and patient-specific risk profiles, including genetic predisposition (**Figure 4**) [3, 27, 67].

2.3.3 Regenerative peri-implantitis treatment

While some evidence supports using apically positioned flaps (APF) in combination with implantoplasty to restore peri-implant health, this approach may be unsuitable in esthetically sensitive areas [60, 61]. In such cases, regenerative surgical techniques are preferred to augment peri-implant lesions to support soft tissue contours [31, 68–70].

A wide range of biomaterials has been utilized to regenerate peri-implantitis defects across different healing modalities, including autologous bone, allografts, xenografts, and alloplasts, either alone or in combination with membranes and/or biologic or chemical agents [50, 68, 71]. High variability in outcome and high risk of bias have been reported throughout systematic reviews analyzing and comparing the effectiveness of regenerative procedures for peri-implantitis defects. This finding may be attributable to the complex and multifactorial nature of peri-implantitis, contributing to methodological heterogeneity, rendering it difficult to design low-bias, standardized comparative clinical trials [10, 31, 50, 68, 70]. There is currently no evidence suggesting that a specific material or

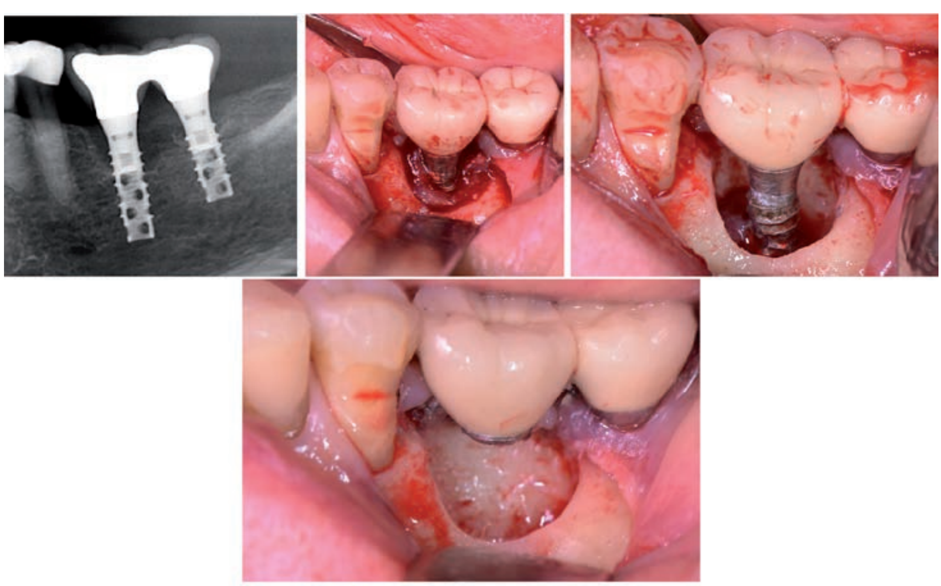

Figure 4.
Peri-implantitis at site 36, complex treatment: mechanical debridement, complex due to the hollow implant design, sectioning, and explantation of the implant.

material combination might be superior to others [50]. For example, there is no conclusion on the effectiveness of membranes in conjunction with bone grafts in augmenting peri-implantitis treatment. Experiments failed to show an increase in radiographic bone fill when using bone grafts with membranes compared to bone grafts alone [70, 72]. Also, there is currently no conclusion on the superiority of submerged vs. non-submerged healing as part of regenerative peri-implant therapy [68].

Compared to nonsurgical treatment, guided bone regeneration of peri-implantitis defects has been reported to result in 3.52 mm greater periodontal probing depth (PPD) reduction compared to nonsurgical therapy [31]. When comparing relative pocket depth (PD) reduction of individual approaches, values of 37.9% were reported for access flap and debridement, 33.4% for resective approach, 37.1% for augmenting with bone grafting materials, and 48.2% for GBR, suggesting marginal advantages of regenerative compared to non-regenerative approaches [70].

A mean PD reduction ranging from 2.0 to 4.5 mm and a mean reduction in BoP from 44.8 to 86% at 12 months post-therapy was recently reported for the regenerative peri-implant therapy using bone grafts [68, 69]. The mean radiographic bone fill was reported to be 2 mm [70]. Despite encouraging results superiority of regenerative therapy over access flap surgery without regenerative therapy in terms of clinical and esthetic outcomes needs to be established [10, 68, 69]. Finally, defect classification may affect treatment outcomes, with circumferential contained defects showing a better prognosis [46]. Regenerative techniques may be preferably adopted at intraosseous defects with a depth of ≥3 mm [68].

In conclusion, the mixed and often inconclusive outcomes of regenerative therapy compared to open-flap debridement alone underscore the current limitations of regenerative approaches and emphasize the need for individualized, clinically driven, and patient-centered treatment planning.

2.4 Implant removal and reimplantation

In cases where surgical peri-implant therapy is deemed ineffective or the prognosis is unfavorable, clinicians may consider implant removal and reimplantation as viable alternatives. The decision to remove an implant should be based on defect severity and overall treatment prognosis, considering both functional, esthetic, and patient-centered aspects.

Advanced peri-implantitis lesions affecting ≥50% of the implant length are relatively common, occurring in over 40% of cases, and have been associated with poor outcomes following surgical therapy [45, 73, 74]. After explantation, two primary strategies exist [1]: Immediate placement of a wider or longer implant when anatomical and prosthetic conditions allow, or [2] delayed placement following alveolar ridge augmentation and healing.

Immediate reimplantation has shown success rates of approximately 94%, with acceptable marginal bone loss (~0.9 mm) in selected cases [75]. However, in moderate to advanced defects involving ≥25% of the implant length, this approach carries a higher risk due to advanced alveolar bone loss and is generally not recommended. In such cases, bone grafting at the time of implant removal may be considered to preserve and restore ridge volume, with delayed implant placement typically planned for 3–6 months following augmentation [76]. Implant removal may also offer an opportunity to address additional site deficiencies, such as insufficient keratinized mucosa or compromised esthetic soft tissue contours [77–80]. In esthetically demanding regions, staged augmentation procedures and delayed implant placement, allowing for soft tissue conditioning, may be favored to achieve optimal esthetic outcomes. Systematic reviews report an implant survival rate of approximately 88% following delayed reimplantation over a mean follow-up of 40 months, with a ~ 15% reduction in survival rates observed in cases involving multiple reimplantation attempts [81].

From a procedural standpoint, the least invasive removal technique involves applying counter-clockwise torque with a removal tool compatible with the implant's internal or external connection [82, 83]. In selected cases, resectioning 1–2 mm of crestal cortical bone may facilitate explantation. Trephine burs and elevators remain effective but are more invasive alternatives, typically reserved for cases in which conservative approaches fail. Corticotomies may enhance healing potential, as bone at previously infected implant sites often exhibits reduced vascularity.

In summary, implant removal followed by reimplantation offers a viable alternative to regenerative or resective peri-implant therapy. Treatment strategies should be tailored to the individual case, considering defect morphology and severity, anatomical limitations, soft and hard tissue conditions, and esthetic demands following explantation (**Figure 5**).

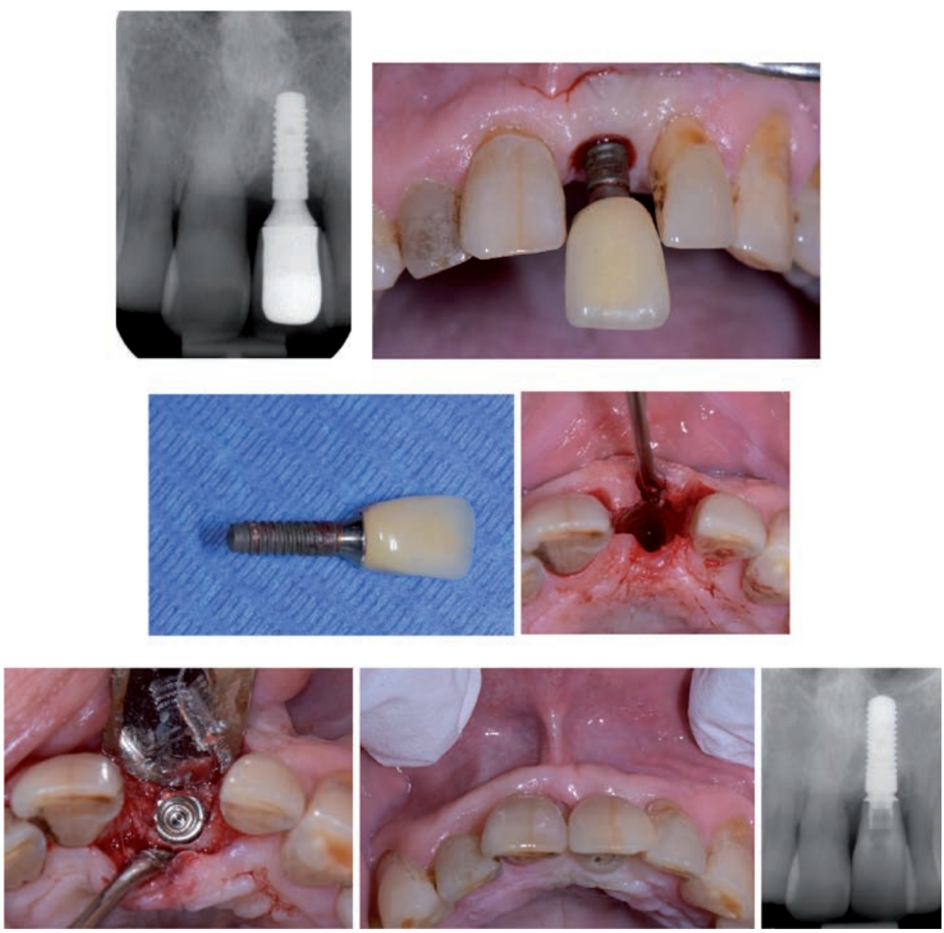

Figure 5.
Implant removal of unstable implant 21, mechanical debridement, reimplantation with simultaneous guide bone regeneration.

3. Prevention of peri-implantitis: Supportive peri-implant therapy

Due to the severity, rapid progression, and lack of consolidated treatment strategies, effective prevention of peri-implant diseases remains a cornerstone of implant therapy. Clinicians should recognize that prevention begins well before implant placement and extends throughout the lifetime of a prosthetic restoration [10, 38]. Prevention strategies target risk reduction at multiple levels—patient selection, treatment planning, surgical and prosthetic execution, and postoperative care. Once osseointegrated, supportive peri-implant care and therapy (SPIC, SPIT) play a critical role in maintaining peri-implant health. At the same time, secondary prevention aims at reducing the risk of disease recurrence after peri-implant treatment [10].

Primordial and primary prevention strategies aim to identify and manage modifiable risk factors and prevent risk factor development before and after implant placement, respectively [84]. Preventive measures include early patient risk profiling but also patient education on the importance of supportive peri-implant care (SPIC) recalls and oral health routines [85]. Furthermore, modifiable risk factors, comprising glycaemic control status, smoking but also the identification of parafunctional habits and bruxisms, are important at this stage [84]. From a treatment perspective, clinicians are advised to carefully plan and control ideal placement and deliver prosthetic designs that enable optimal hygiene routines and reduce the risk for plaque accumulation [86]. Finally, primordial prevention should also include the detection and treatment of any periodontal condition prior to implant placement [87].

SPIC and SPIT are regarded as crucial to prevent the onset or recurrence of peri-implant disease following successful implant integration and active or completed therapy, respectively [88–91]. Corresponding protocols should be individualized and regularly re-evaluated, with risk assessment tools available to guide patient-specific preventive strategies [92]. These protocols should include professional mechanical plaque removal at 3- to 6-month intervals, monitoring peri-implant health using gentle probing (0.2 N) and any signs of plaque accumulation, and ongoing assessment and reinforcement of patient compliance with oral hygiene instructions [10, 27, 28]. Early detection of peri-implant disease to enable timely intervention and prevent progression to peri-implantitis is a key objective of SPIC [2, 90]. Baseline periapical radiographs obtained after initial physiological bone remodeling serve as essential references for confirming disease presence and monitoring its progression over time [1].

Finally, secondary and tertiary prevention aim to reduce the risk of peri-implant disease recurrence and prevent complications in patients who have completed or are undergoing active treatment. Preventive measures are of paramount importance and remain integral to the long-term success and sustainability of implant therapy.

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