Non-surgical peri-implantitis therapy: what now?

Non-surgical therapy can control peri-implantitis and delay the need for surgery when supported by maintenance care. Future management emphasises minimally invasive, regenerative, and technology-assisted approaches to optimise clinical outcomes.

Learning outcomes

1. Describe the main non–surgical treatments for peri–implantitis and their clinical effectiveness.
2. Recognise the role of maintenance care in improving treatment outcomes.

Introduction

Peri-implantitis is a plaque-induced condition that causes inflammation and progressive bone loss around implants.[1] It is clinically significant because it represents one of the primary causes of implant failure. Its reversible precursor, peri-implant mucositis, presents with bleeding on probing (BOP), erythema, swelling, and/or suppuration without bone loss. Peri-implantitis, by contrast, shows BOP and/or suppuration, increased probing depths (PDs), and radiographic bone loss (**Figures 1-3**). Histologically, lesions extend beyond the junctional epithelium and contain more plasma cells, macrophages, and neutrophils than mucositis.[1]

Diagnosis relies on detecting disease progression, greater PDs, BOP and/or suppuration on probing (SOP), and bone loss beyond initial remodelling. Without baseline data, indirect criteria apply, including BOP and/or suppuration on gentle probing, PD of ≥6mm and "bone levels ≥3mm apical of the most coronal portion of the intra-osseous part of the implant".[1]

Peri-implantitis is influenced by both subject- and implant-level risk indicators. At the subject level, a history of severe periodontitis, diabetes, and smoking increase susceptibility. Implant-level indicators include implant surface characteristics, suboptimal restorations, residual cement, improper positioning, insufficient keratinised mucosa, and occlusal overload.[2] Overall, disease severity strongly correlates with plaque accumulation, underscoring the importance of plaque control.[2]

Microbiological studies show that peri-implantitis lesions are dominated by *Treponema* spp. and *Synergistes* cluster A, with *Campylobacter*, *Fusobacterium*, *Gemella*, *Porphyromonas*, *Parvimonas*, and *Treponema* spp. present in all samples, and *Prevotella*, *Staphylococcus*, and *Streptococcus* spp. in 60-80% of samples. Higher levels of *T. forsythia*, *P. micra*, *F. nucleatum*, *F. necrophorum*, and *C. rectus* distinguish peri-implantitis from periodontitis.[3]

Prosthesis design significantly affects peri-implant health (**Figures 4** and **5**). Different design features influence plaque accumulation, cleanability, and the surrounding peri-implant soft tissue response. Factors such as emergence profile, angulation, and cervical margin influence plaque retention and soft tissue stability, reinforcing the importance of prosthetic-driven implant placement.[4] A weak soft tissue seal may predispose to disease, while poor prosthetic design can exacerbate inflammation through plaque accumulation and mechanical stress. Effective management requires identifying and addressing prosthetic, local, and systemic risk factors. Strict adherence to personalised oral hygiene and supportive implant maintenance is essential, as regular post-restorative care can prevent or delay disease progression.[4]

The potential for true re-osseointegration following peri-implantitis treatment remains controversial, with most supporting evidence derived from animal

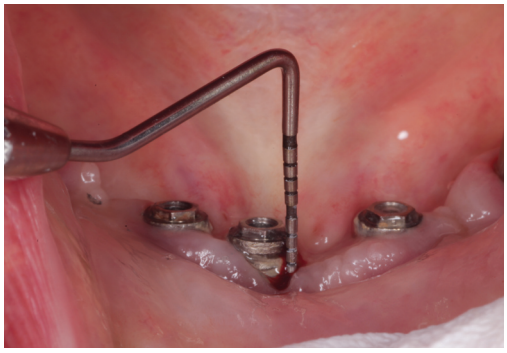

FIGURE 1: Increased probing depths and bleeding on probing around implants supporting a full-arch prosthesis.

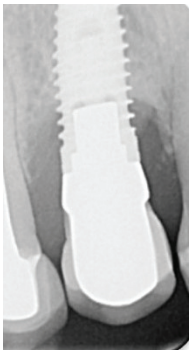

FIGURE 2: Radiographic bone loss around a threaded implant design.

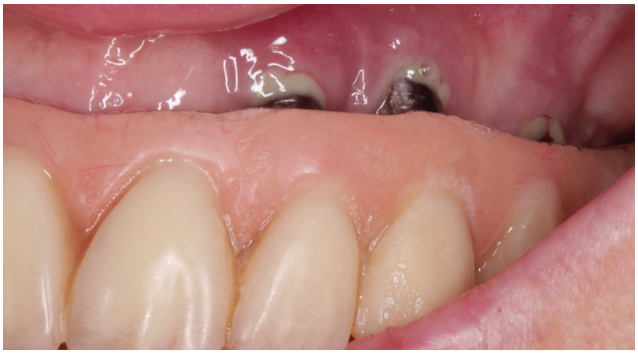

FIGURE 3: Implant-retained full-arch prosthesis with early peri-implantitis.

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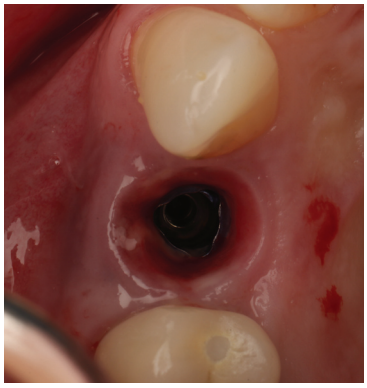

FIGURE 4: Inflamed peri-implant soft tissue.

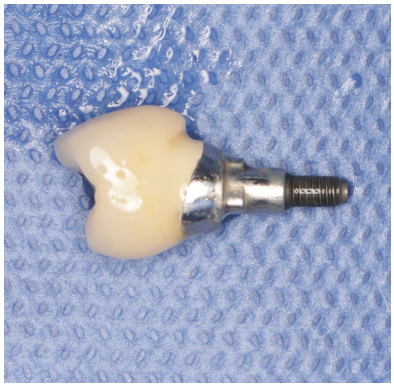

FIGURE 5: Implant-supported crown with poor cleansability due to contour/design.

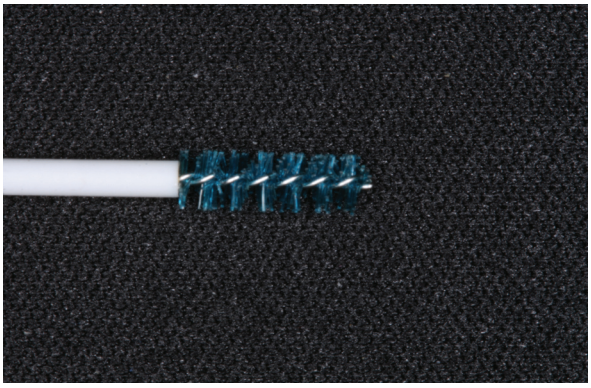

FIGURE 6: Labrida BioClean chitosan brush.

histological studies.[5] Initial management should begin with non-surgical therapy aimed at reducing biofilm and inflammation via sub-marginal mechanical decontamination, often supplemented with adjunctive measures.[6] Surgical interventions are typically recommended when non-surgical therapies fail, as they provide superior access for thorough decontamination and defect management.[1,6] A notable drawback, however, is the frequent occurrence of postoperative gingival recession, which restricts the use of these procedures in aesthetically sensitive areas. Non-surgical debridement remains a low-cost treatment option, but its efficacy is limited in advanced peri-implant disease. Regenerative surgical approaches, although more expensive, may offer long-term cost effectiveness by preserving compromised implants and reducing the need for future replacement. Nevertheless, both non-surgical and conventional surgical therapies often yield unpredictable outcomes.[6] These limitations have prompted a growing interest in minimally invasive, flapless surgical techniques, which aim to enhance therapeutic predictability while reducing morbidity.

Successful treatment protocols can yield clinical improvements, including reduced probing pocket depth (PPD) and BOP, as well as stabilisation or regeneration of marginal bone levels. Re-evaluation is recommended approximately six months post treatment to assess healing and determine the need for further surgical intervention. However, relying solely on PPD and BOP may not accurately reflect treatment success. Current evidence supports the use of composite outcome measures, incorporating PPD, BOP, SOP, peri-implant recession, patient-reported outcomes, and marginal bone level changes, to provide a more comprehensive assessment of peri-implantitis therapy.[7-9]

The aim of this article is to comprehensively evaluate current non-surgical treatment modalities for peri-implantitis, focusing on their clinical efficacy, limitations, and the evolving role of adjunctive and future therapies in optimising non-invasive management. This article explores mechanical decontamination (laser and air-abrasive powder), adjunctive chemical decontamination, subgingival drugs, and systemic antibiotics. The majority of the studies discussed are randomised controlled trials (RCTs), in which the control group received conventional scaling and root planing using titanium curettes alone, as this remains the most routinely employed non-surgical periodontal treatment modality.

What is the state of play now?

Mechanical decontamination

A variety of instruments can be used for sub-marginal instrumentation, including plastic or titanium curettes, sonic and ultrasonic devices, air-abrasive systems, and lasers, with no single method demonstrating clear superiority.[7] Of the nine RCTs analysed in a systematic review by Cosgarea *et al.*, five investigated laser-assisted decontamination (Nd:YAG, diode, Er:YAG, Er,Cr:YSGG), while four examined air-abrasive systems.[7] Lasers provided limited short-term advantages: Nd:YAG produced improvements in PD and BOP at three months but not at six; Er,Cr:YSGG achieved greater PD reductions at six months; and, Er:YAG significantly decreased BOP. Nd:YAG also lowered inflammatory markers, although diode laser outcomes were inconsistent. Across devices, mean PD reductions ranged from 0.8 to 1.5mm, with minimal influence on suppuration or bone levels. Rough implant surfaces and cement-retained prostheses complicated decontamination, and only limited data were available on patient-reported outcomes or implant survival. Laser systems, especially Er:YAG and Er,Cr:YSGG, provide effective decontamination with limited disruption to surface topography. Additionally, the Er:YAG-activated electromagnetic device (EM) appears promising for biofilm disruption and facilitating re-osseointegration, although strong independent clinical evidence is still lacking.[10]

Low-abrasive air-abrasive (AA) powders, such as glycine, erythritol, calcium sodium phosphosilicate, calcium carbonate, and tricalcium phosphate, have demonstrated encouraging results. Glycine powder achieved significantly greater reductions in PD and BOP at three and six months than Gracey curettes used alongside chlorhexidine (CHX) irrigation.[11] Erythritol air polishing provided clinical and microbiological outcomes comparable to ultrasonic debridement, with minimal discomfort.[12] Combinational approaches, such as glycine mixed with tricalcium phosphate, have shown superior biofilm removal compared with glycine or sodium bicarbonate alone on titanium and zirconia surfaces, while also reducing treatment time.[12] Low-abrasive powders like glycine and erythritol preserve implant surface topography, in contrast with more abrasive powders like sodium bicarbonate, which can roughen the surface and potentially hinder re-osseointegration. Thus, low-abrasive AA powders offer an effective, minimally invasive, and surface-preserving non-surgical decontamination option. Despite their overall safety, AA devices can occasionally lead to complications, such as subcutaneous emphysema when pressurised air is forced into soft tissues, emphasising the importance of accurate technique.

Clinicians can use implant specific sonic or ultrasonic inserts (e.g., plastic, carbon fibre, or silicone coated tips) for the non-surgical treatment of peri implantitis, as these effectively disrupt biofilm while minimising titanium surface damage. Evidence also supports the use of chitosan brushes (**Figure 6**) as adjunctive decontamination tools. Chitosan brushes seem to improve PD and bleeding in the short term.[13] Finally, where safe and practical, removing a screw retained superstructure can also enhance access for implant decontamination.

Adjunctive chemical decontamination

Adjunctive chemical decontamination should be used cautiously due to limited benefits and potential implant surface damage. Agents like CHX, hydrogen peroxide, citric acid, and sodium hypochlorite (NaOCl), as well as photodynamic systems with photosensitisers, provide only modest reductions in PD and BOP, with no consistent superiority.[6] Across agents and devices and in combination with a variety of instruments, mean PD reductions ranged from 0.5 to 1.87mm. Furthermore, agents like CHX may impair osteoblast function, while saline, citric acid, and NaOCl-EDTA are more biocompatible. Mechanical-chemical combinations may help in select cases, but evidence is limited.

Recently explored compounds like curcumin and xanthohumol show strong antibiofilm activity against *P. gingivalis* and *A. actinomycetemcomitans* in 60-second treatments, making them promising biocompatible alternatives.[14]

The electrolytic cleaning process generates hydrogen bubbles on the implant surface, effectively detaching and flushing away bacterial biofilm without damaging the titanium. It is primarily used as a stand-alone decontamination method during surgical treatment of peri-implantitis, but it can also be combined with mechanical instrumentation and regenerative procedures. Clinical evidence has demonstrated that sites treated with electrolysis were inflammation free and showed significant bone fill, supporting re-osseointegration.[15] The downside is that it is costly, requiring a power unit and single-use tips.

Subgingival drugs in the non-surgical therapy of peri-implantitis

Subgingival drugs are used adjunctively with mechanical debridement in non-surgical peri-implantitis management to reduce bacterial load, inflammation, and PD. Local antimicrobials delivered via gels, chips, or controlled-release systems provide high site-specific concentrations with minimal systemic exposure, but may cause discomfort and require multiple applications. Controlled-release systems, such as biodegradable polymers, hydrogels, and films, maintain therapeutic levels and can release ions (e.g., calcium, phosphate) to create an alkaline, antibacterial environment supporting tissue regeneration.[16]

CHX chips achieved greater PD reduction (1.76mm vs 1.54mm, p=0.01) and attachment gain (1.47mm vs 1.39mm, p=0.0017) than controls, with more patients showing ≥2mm PD reduction (59% vs 47%).[17] Local antibiotics, including minocycline (chitosan-alginate microspheres), doxycycline hyclate (Atridox), and topical metronidazole, significantly reduced PD, BOP, and inflammation in clinical and animal studies.[18,19]

Overall, subgingival drugs offer modest benefits with no standardised protocol. Implant surface roughness and material influence bacterial colonisation, limiting local antimicrobial efficacy. Advanced systems (hydrogels, microspheres, nanoparticles) and combinations with lasers or photodynamic therapy may enhance biofilm disruption, but high costs, repeated applications, and surface variability restrict routine use. Effective management still depends on early detection, mechanical debridement, patient compliance, and maintenance, with subgingival drugs only as supportive adjuncts.[20]

Systemic antibiotics as adjuncts to non-surgical therapy

Mechanical decontamination remains the cornerstone of non-surgical peri-implantitis management, and systemic antibiotics have been explored as adjuncts to improve outcomes. RCTs show that while amoxicillin and metronidazole may yield short-term PPD and BOP reductions, differences versus mechanical therapy alone are not statistically significant. De Waal *et al.* found no added benefit from antibiotics after full-mouth debridement with CHX, and Polymeri *et al.* similarly reported nonsignificant PPD improvements, with only two implants achieving full resolution.[21,22] Systematic reviews confirm inconsistent and short-lived benefits, limited mainly to deep pockets.[8]

Accordingly, the European Federation of Periodontology (EFP) S3-level guidelines do not recommend routine systemic antibiotics in non-surgical peri-implantitis therapy, advising their use only in severe, high-risk cases. Standard care should prioritise mechanical debridement, patient self-care, and supportive maintenance, with surgery indicated when non-surgical measures fail to restore peri-implant health.[23]

Future directions

The evolution of peri-implantitis management is moving towards minimally invasive surgical approaches, combining the benefits of non-surgical and traditional surgical techniques. These methods aim to reduce procedural morbidity while maintaining efficacy, preserve soft and hard tissue architecture, and promote regeneration using advanced instruments and autologous biomaterials.

Chiang *et al.* reported that microscope-assisted flap surgery with surface decontamination, bone grafting, and membrane placement achieved stable radiographic bone fill and disease resolution over two to four years.[24] Microscope-enhanced and laser-assisted regenerative approaches, alongside autologous products like leukocyte- and platelet-rich fibrin (L-PRF), have also shown promise in improving outcomes. Clinical studies demonstrate that L-PRF enhances attachment gains, PD reduction, and implant stability, compared to conventional treatment.[25]

As with conventional surgical regeneration, defect depth and wall configuration remain key determinants of success in minimally invasive peri-implantitis treatment.

Conclusion

Non-surgical therapy is the first-line treatment for peri-implantitis, focusing on biofilm removal, inflammation control, and implant preservation. Mechanical debridement is central, while chemical agents and systemic antibiotics provide minimal additional benefit and should be used selectively.

Success relies on individualised maintenance, meticulous oral hygiene, and regular supportive care, as recurrence is linked to poor plaque control. Daily home plaque control is essential for managing peri-implantitis, and adjunctive measures such as oral irrigators or short-term CHX rinses can further support biofilm control. If non-surgical measures fail, surgical or minimally invasive regenerative approaches, using biocompatible biomaterials, microscope-assisted precision, and advanced decontamination can offer more predictable long-term outcomes.

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