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Clinical Translation of the 2024 AO/AAP Consensus on Prevention and Management of Peri-implant Diseases and Conditions

As the number of placed implants increases exponentially worldwide, the prevalence of peri-implant diseases, namely mucositis and peri-implantitis, is also in an ascending trajectory. Focusing on prevention and identifying and decreasing risk factors are important aspects of managing the epidemic of peri-implant diseases. However, many clinicians are faced with patients who already have signs of inflammatory disease in varying degrees of severity. Treatment of peri-implant mucositis involves addressing the etiologic factors causing local inflammation, as well as improving self-performed oral hygiene and enrolling the patient in a personalized maintenance program. On the other hand, managing peri-implantitis can be very challenging. Peri-implantitis therapy may involve nonsurgical, nonreconstructive, reconstructive, and—in some instances—explantation procedures. The aim of this report is to provide clinical recommendations and reference flowcharts to manage peri-implant diseases and related complications on the basis of expert opinions and the scientific evidence discussed at the 2024 AO/AAP Consensus on Prevention and Management of Peri-implant Diseases and Conditions. *Int J Periodontics Restorative Dent 2025;45(suppl):s1–s23. doi: 10.11607/prd.7658*

Peri-implant mucositis and peri-implantitis are commonly seen among dental implants.[1] With increases in both the number of implants placed and the length of time in function, their surfaces may become exposed, making them more susceptible to biofilm development.[2] Moreover, implant positioning may create a scenario where the prosthetic design hinders effective oral hygiene, leading to the development of these conditions.[3] Thin tissue phenotypes can exacerbate these issues, resulting in peri-implant soft tissue deficiency and recession, which can lead to further bone loss and inflammation.[4]

Peri-implant mucositis serves as a warning to patients and clinicians, drawing their attention to improving preventative measures and at-home cleaning, decreasing the frequency between dental visits, and reducing modifiable risk factors that may lead to increased pocket depths, suppuration, and hard tissue destruction characteristic of peri-implantitis.[3,5,6] Peri-implant mucositis has been shown to be reversible, and its management should consist of possible clinical intervention with close monitoring and improved implant maintenance. If clinical intervention is chosen, treatment options may include systemic antibiotics or local

antimicrobial delivery, nonsurgical mechanical debridement, or nonsurgical chemotherapeutic irrigation.[6–14] After therapy, closer monitoring is crucial to prevent recurrence or progression of peri-implant mucositis, although 60% of cases have historically shown partial (but not complete) resolution.[15]

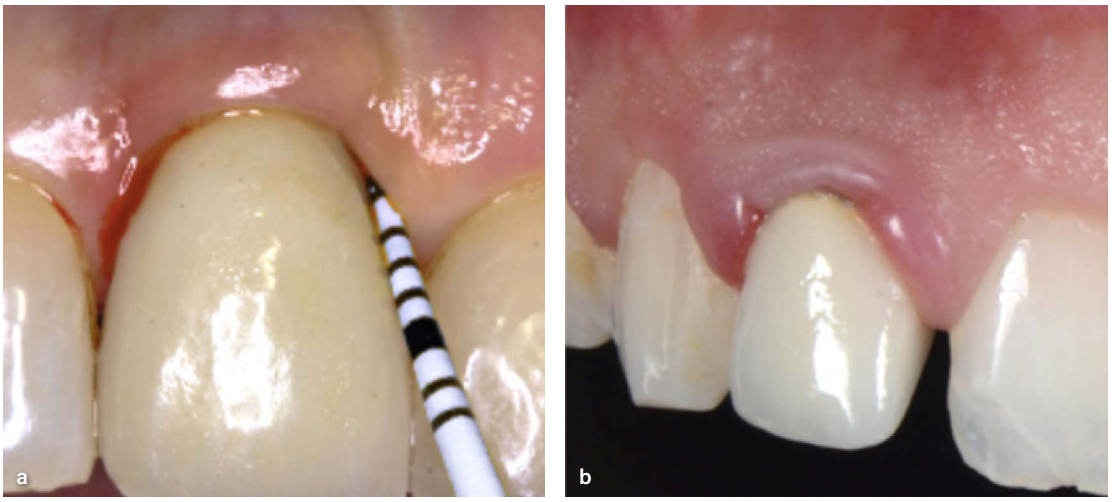
Peri-implantitis is a more devastating form of peri-implant disease, characterized by progressive inflammation and bone loss around implants. Its diagnosis requires the presence of bleeding and/or suppuration on gentle probing, increased probing depths relative to previous clinical measurements, and radiographic evidence of bone loss beyond the initial crestal bone remodeling. In cases where no prior clinical data are available, peri-implantitis can be diagnosed based on the simultaneous presence of bleeding and/or suppuration on probing, probing depths ≥ 6 mm, and bone levels ≥ 3 mm apical to the most coronal portion of the intraosseous implant. In such cases, intervention is indicated to halt disease progression.[7,16–18]

The major challenge in effective therapy, whether surgical or nonsurgical, is the ability to adequately debride and decontaminate the implant surface. This is especially difficult because the majority of implant surfaces are roughened or enhanced at the micro- or nanolevel.[2,19] There is a plethora of suggested mechanical and chemical decontamination methods, including but not limited to curettes, ultrasonic scalers, chlorhexidine, citric acid, abrasive devices, implantoplasty, lasers, and combinations of these.[19] The goal of any of these techniques is to arrest the disease and aid in implant reosseointegration for long-term stability and maintenance of peri-implant health. Despite controversies within the literature, surgical techniques must be considered if nonsurgical means prove ineffective. Once the implant is debrided and decontaminated, the question of resection vs regeneration must be addressed. If the defect is not contained, it is generally treated with flap surgery alone or in combination with osseous resection to facilitate cleansability and implant maintenance.[18] When the defect has a contained infrabony component and adequate keratinized tissue, regenerative therapy can be performed, utilizing various bone replacement grafting materials with or without biologic additions.[20] This report aims to provide clinical recommendations and flowcharts for clinicians to reference to manage peri-implant diseases and related complications on the basis of expert opinion and the scientific evidence discussed at the 2024 AO/AAP Consensus on Prevention and Management of Peri-implant Diseases. The consensus statements are included in the Appendix.

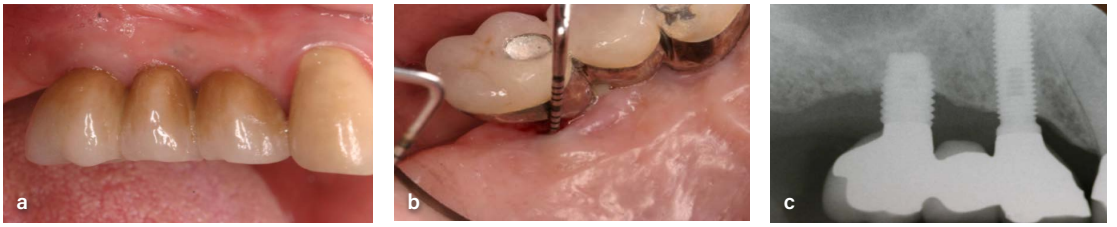
Peri-implant Mucositis and Peri-implantitis

Peri-implant mucositis is characterized by inflammation confined to the soft tissues surrounding an implant without affecting the supporting bone[6] (Fig 1). Clinical measurements for diagnosing peri-implant mucositis include increased pocket probing depths compared to baseline but generally < 5 mm. The presence of bleeding on probing is a critical indicator of inflammation. Unlike more advanced peri-implant conditions, peri-implant mucositis does not necessarily present with suppuration. Clinically, the mucosa surrounding the implant often demonstrates signs of redness indicative of an inflammatory response. Importantly, there is no radiographic evidence of bone loss beyond what may be seen with physiologic bone remodeling, distinguishing it from peri-implantitis.[3]

Peri-implantitis is a more advanced condition involving both soft tissue inflammation and progressive bone loss[3,17,21] (Fig 2). Clinical measurements for diagnosing peri-implantitis include pocket probing depths typically ≥ 5 mm, reflecting the formation of deep peri-implant pockets. Bleeding on probing is consistently present, signaling ongoing inflammation. Additionally, suppuration is usually observed, indicating infection within the peri-implant tissues. Clinically, peri-implantitis is marked by a progressive loss of clinical attachment, with evident bone loss visible on radiographic examinations. This bone loss exceeds what is evident with physiologic remodeling that takes place after implant placement; further, it is generally ≥ 3 mm from the baseline or previous radiographs and may include vertical bone defects



▲ **Fig 1** *(a and b)* Clinical examples of peri-implant mucositis. Note the peri-implant tissue inflammation, erythema, and bleeding on probing.



▲ **Fig 2** *(a to c)* Clinical and radiographic examples of peri-implantitis. Note the increased peri-implant probing depths and radiographic bone loss.

≥ 3 mm as well as significant horizontal bone loss, which may affect implant stability. The surrounding peri-implant soft tissues often exhibit redness, swelling, and other signs of inflammation, compounded by changes in bone levels that progressively worsen over time. This combination of soft tissue inflammation and bone loss necessitates a comprehensive approach to diagnosis and treatment to prevent further deterioration and potential implant failure.[3,21]

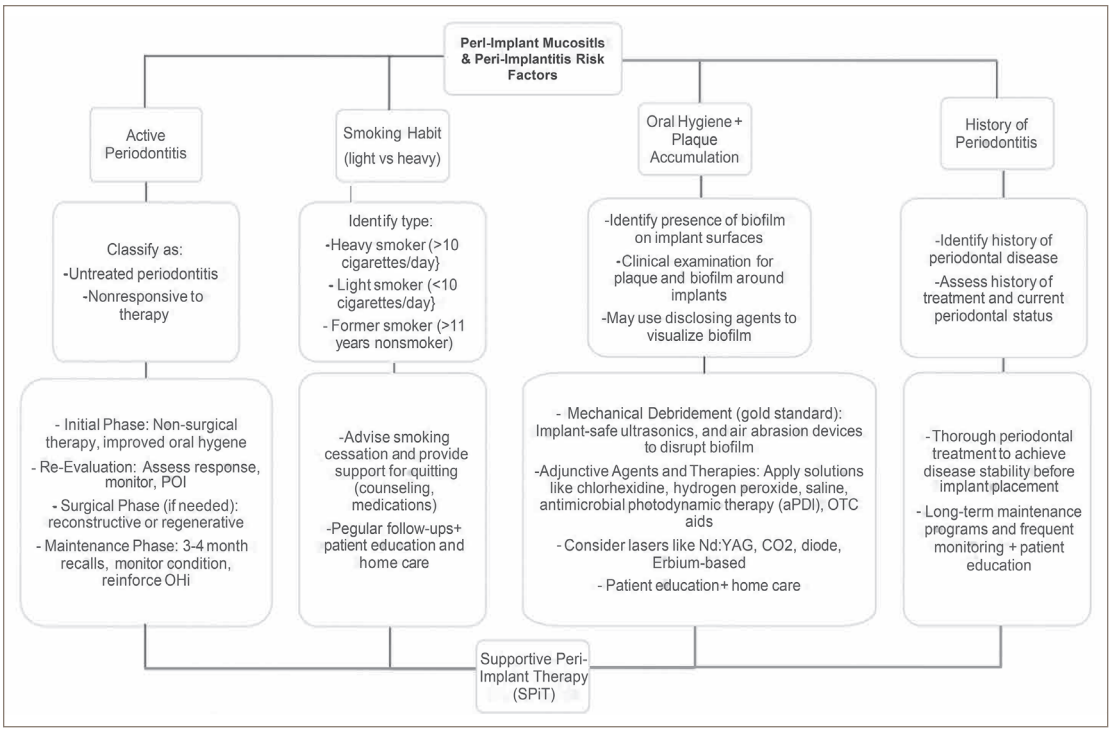
Management of Peri-implant Disease Risk

Peri-implant diseases have been associated with several predisposing and precipitating risk factors (Figs 3 and 4). Research and expert clinician consensus (see Appendix) have explored methods to minimize the possible onset and progression of inflammation and bone changes around implants and are highlighted in the flowcharts within this article.

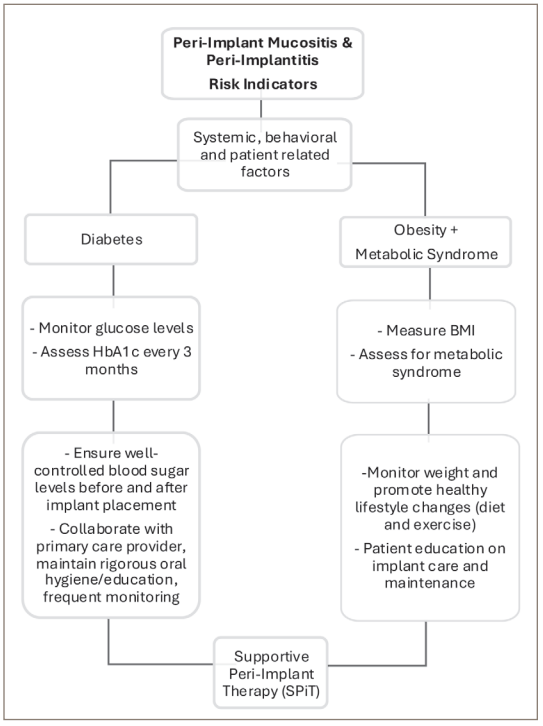
Biologic Factors

Smoking habit

The consensus flowcharts begin with Fig 3, which categorizes patients based on their smoking habits (among other things), distinguishing between active, former, light, and heavy smokers.[22] Active smokers are those currently smoking, while former smokers have quit, with a specific focus on whether they have been smoke-free for > 5 years. Light smokers are defined as those smoking < 10 cigarettes per day, while heavy smokers consume > 10 cigarettes daily. The intensity and duration of smoking are critical factors that exacerbate the risk of peri-implant mucositis. This risk is highest in heavy smokers, making them the most vulnerable group.[1,21]



▲ **Fig 3** Flowchart of clinical recommendations for smoking, plaque accumulation, and history of active periodontitis.



▲ **Fig 4** Flowchart of clinical recommendations for diabetes and obesity/metabolic syndrome.

Recommended management strategy

The flowchart outlines a targeted approach for smokers. Smoking cessation is ideal for heavy smokers. This process may include intensive counseling in severe cases, possibly involving behavioral therapy and pharmacologic aids. Light smokers, while at lower risk than heavy smokers, still require education on the potential impacts of smoking on implant health and are advised to reduce or cease smoking if possible. For former smokers, the treatment strategy shifts towards maintaining the health of the peri-implant tissues through continued oral hygiene practices and regular dental visits.[22]

Biofilm (plaque) accumulation/oral hygiene

Biofilm accumulation is a primary etiologic factor in peri-implant mucositis and peri-implantitis. The Fig 3 flowchart mandates a comprehensive clinical examination to identify the presence of biofilm on implant surfaces. The presence of biofilm leads to an inflammatory response in the peri-implant tissues, characterized by redness, swelling, bleeding on probing, and sometimes exudate. Patients with poor oral hygiene habits or limited manual

Table 1 Management of Different Stages of Periodontitis According to Periodontal Status

Stage	Management strategy
Gingivitis (no attachment loss)	Nonsurgical management: Routine periodontal care with focus on plaque control. Scaling and root planing may be necessary if gingivitis persists. In overweight or obese patients, adjust the frequency of recall visits based on their BMI and other risk factors.
Stage I–II periodontitis (mild to moderate periodontitis)	Nonsurgical and surgical management: Scaling and root planing is recommended. For Stage II, consider adjunctive therapies, especially in obese patients with associated systemic inflammation. Increase periodontal maintenance frequency and provide dietary counseling. Assess the need for systemic antibiotics in severe cases.
Stage III–IV periodontitis (severe periodontitis)	Surgical intervention and rigorous maintenance: Surgical treatments, such as flap surgery, may be required. In obese patients, prioritize controlling inflammation and consider the systemic effects of obesity. Frequent recall and close monitoring are essential. Collaboration with the patient's healthcare team is crucial for managing both periodontal and systemic health.

Periodontal status as determined by the 2017 AAP Classification.

dexterity are at a higher risk for plaque/biofilm buildup, necessitating early and frequent intervention to prevent the onset of mucositis.[7,19]

Recommended management strategy

The flowchart specifies the use of implant-safe instruments, such as titanium curettes, specialized piezoelectric ultrasonic scalers for implants, and air-abrasion devices. These tools are designed to disrupt and remove biofilm without significantly compromising the implant surface.[19,23,24] Adjunctive chemical agents may be utilized to further reduce the microbial load. These include chlorhexidine (applied as a rinse or in gel form), hydrogen peroxide, saline, and iodine solutions. For cases with significant inflammation or where mechanical debridement alone is insufficient, the flowchart advises that clinicians consider systemic or localized antibiotic therapy. Options may include local delivery of minocycline microspheres, particularly in areas with persistent inflammation.[19,25,26] Regular monitoring and patient education on daily oral hygiene practices are critical to preventing recurrence. In addition, supportive peri-implant therapy (SPiT) every 2 to 3 months is recommended to manage and prevent the progression of mucositis.[23]

Periodontitis patients

A history of periodontal disease (Table 1) is a critical factor predisposing patients to peri-implant mucositis and peri-implantitis.[3] The Fig 3 flowchart underscores the importance of a thorough periodontal assessment. This includes reviewing the patient's history of periodontal treatment, the current status of periodontal health, and any residual effects of the previous disease. Patients with a history of severe or untreated periodontitis are at a heightened risk for peri-implant mucositis leading to peri-implantitis due to the potential for unresolved inflammation.[27]

Active periodontitis is one of the most significant risk factors for peri-implantitis. Patients with ongoing periodontal disease, characterized by clinical signs such as increased probing depths, bleeding on probing, and radiographic evidence of bone loss, are highly susceptible to peri-implant inflammation and subsequent bone loss around implants.[1,3,21] A comprehensive periodontal examination is advised to identify these indicators, as sites with untreated or inadequately managed periodontitis pose a high risk for the development of peri-implantitis, necessitating immediate and targeted intervention.

Recommended management strategy

For patients with a history of periodontitis (on a reduced periodontium), preventing recurrence is crucial to reducing the risk of peri-implant biologic complications after implant placement. This may involve a combination of nonsurgical maintenance and possible reintroduction of phase 1 therapy, which may progress to surgical intervention in cases of advanced periodontal recurrence (ie, stage 3 or 4 periodontitis). Similar to active disease cases, surgical interventions, including flap alone

Table 2 Management of Diabetes

Parameter	Guideline	Management strategy
Fasting glucose level	< 100 mg/dL (normal)	Routine care: Patients with normal glucose levels are at standard risk. Follow the AAP guidelines for maintenance. Reinforce good oral hygiene practices.
	100–125 mg/dL (prediabetes)	Increased monitoring: Patients with prediabetes require more frequent periodontal checkups. Emphasize the importance of glycemic control through diet and exercise, and monitor for early signs of periodontal disease.
	> 125 mg/dL (diabetes)	Intensive treatment: Collaboration with a medical team is essential. Focus on reducing periodontal inflammation through SRP. Consider adjunctive therapies if necessary.
Postprandial glucose level	< 140 mg/dL (normal)	Routine care: Standard periodontal maintenance as per the AAP guidelines. Continue routine exams and prophylaxis at regular intervals.
	140–199 mg/dL (impaired glucose tolerance)	Enhanced preventive care: Increase frequency of periodontal monitoring and consider early intervention strategies. Patient education on controlling blood sugar and maintaining oral hygiene is crucial.
	> 200 mg/dL (diabetes)	Immediate intervention: Address acute periodontal conditions aggressively. SRP should be performed promptly. Assess the need for systemic antibiotics, especially in severe cases.
HbA1c	< 5.7% (normal)	Routine care: Maintain standard periodontal treatment intervals. Reinforce preventive care and regular periodontal assessments.
	5.7%–6.4% (prediabetes)	Preventive and early intervention: Increase the frequency of periodontal visits to catch and manage early signs of disease. Educate patients on the link between glycemic control and periodontal health.
	> 6.5% (diabetes)	Comprehensive management: This includes frequent debridement and possibly surgical interventions. Emphasize the importance of strict glycemic control to prevent further periodontal breakdown.

SRP = scaling and root planing.

(with or without resection or regeneration), seem necessary to restore periodontal health if phase 1 therapy fails to resolve disease. Once stability is achieved, the flowchart recommends returning the patient to a long-term maintenance program with frequent periodontal evaluations and professional cleanings to monitor for any signs of inflammation.[16,28,29]

Diabetes mellitus

Expert opinion from the consensus conference identifies poorly controlled diabetes mellitus as a significant risk factor for peri-implant mucositis and peri-implantitis, as shown in the Fig 4 flowchart.[1] It recommends assessing the patient's glycemic control through fasting blood glucose levels and HbA1c measurements. Patients with fasting glucose levels > 126 mg/dl or HbA1c levels > 7% are considered to have poorly controlled diabetes. Patients with diabetes require close monitoring to identify any early signs of peri-implant disease[30,31] (Table 2).

Recommended management strategy

Managing peri-implant disease in diabetic patients requires a collaborative approach. First, the flowchart emphasizes the importance of achieving and maintaining tight glycemic control.[30] This involves working with the patient's primary care physician or endocrinologist to optimize diabetes management. Second, rigorous oral hygiene practices are essential.[23] Mechanical debridement is recommended to maintain biofilm control and prevent further inflammation; antiseptic agents such as chlorhexidine or phenolic rinses may also be combined as adjunctive treatments, but they have not been shown to have significant effect. Patients should be instructed on the importance of maintaining excellent oral hygiene, with regular dental visits for professional cleanings and monitoring.[23]

Obesity/metabolic syndrome

Obesity and metabolic syndrome are associated with an increased risk of peri-implant mucositis

Table 3 Management of Obesity

Parameter	Guideline	Management strategy
Body mass index (BMI)	Normal weight (18.5–24.9 kg/m^2)	Routine periodontal care: Standard risk for periodontal disease. Follow AAP guidelines for maintenance. Encourage healthy lifestyle choices to maintain normal weight and oral health.
	Overweight (25–29.9 kg/m^2)	Increased monitoring: Patients are at a higher risk for developing periodontal disease. Recommend regular periodontal checkups and emphasize the importance of weight management alongside oral health.
	Obese (≥ 30 kg/m^2)	Intensive periodontal management: Obesity is associated with chronic inflammation, increasing the risk for severe periodontitis. Frequent periodontal evaluations, SRP, and potential surgical interventions may be necessary. Coordinate with healthcare providers to address obesity-related systemic inflammation.
Waist circumference	Men: < 40 in (102 cm) Women: < 35 in (88 cm)	Routine care with lifestyle counseling: No significant increase in periodontal risk, but provide advice on maintaining a healthy diet and regular exercise to prevent obesity.
	Men: ≥ 40 in (102 cm) Women: ≥ 35 in (88 cm)	Enhanced periodontal monitoring: Increased abdominal fat correlates with higher risk of periodontitis. Recommend frequent periodontal assessments and emphasize the importance of reducing central obesity through diet and exercise.
Metabolic syndrome	Absence of metabolic syndrome	Standard periodontal care: Focus on prevention and routine periodontal maintenance. Provide lifestyle counseling to prevent the onset of metabolic syndrome and associated periodontal risks.
	Presence of metabolic syndrome	Comprehensive periodontal management: Metabolic syndrome (characterized by central obesity, hypertension, hyperglycemia, and dyslipidemia) significantly increases the risk of periodontitis. Intensive periodontal care is recommended, including SRP, possible use of systemic antibiotics, and frequent recall visits. Collaborate with the patient's healthcare provider to manage underlying conditions.

SRP = scaling and root planing.

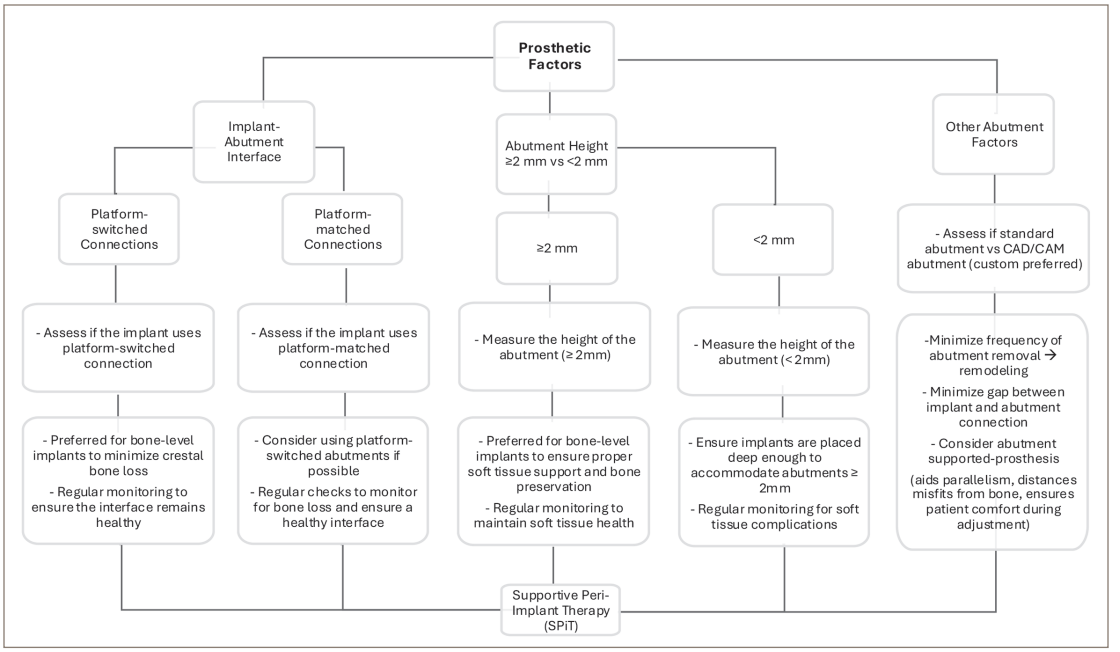
due to their impact on systemic inflammation and immune response. The Fig 4 flowchart advises a comprehensive assessment of the patient's metabolic health, such as measuring body mass index (BMI). Evaluating other markers of metabolic syndrome can include waist circumference (BMR), triglyceride levels, HDL cholesterol, blood pressure, and fasting glucose levels. A BMI of 30 kg/m^2 or higher is indicative of obesity (Table 3), which is linked to chronic low-grade inflammation that can exacerbate peri-implant tissue inflammation.[1,32]

Recommended management strategy

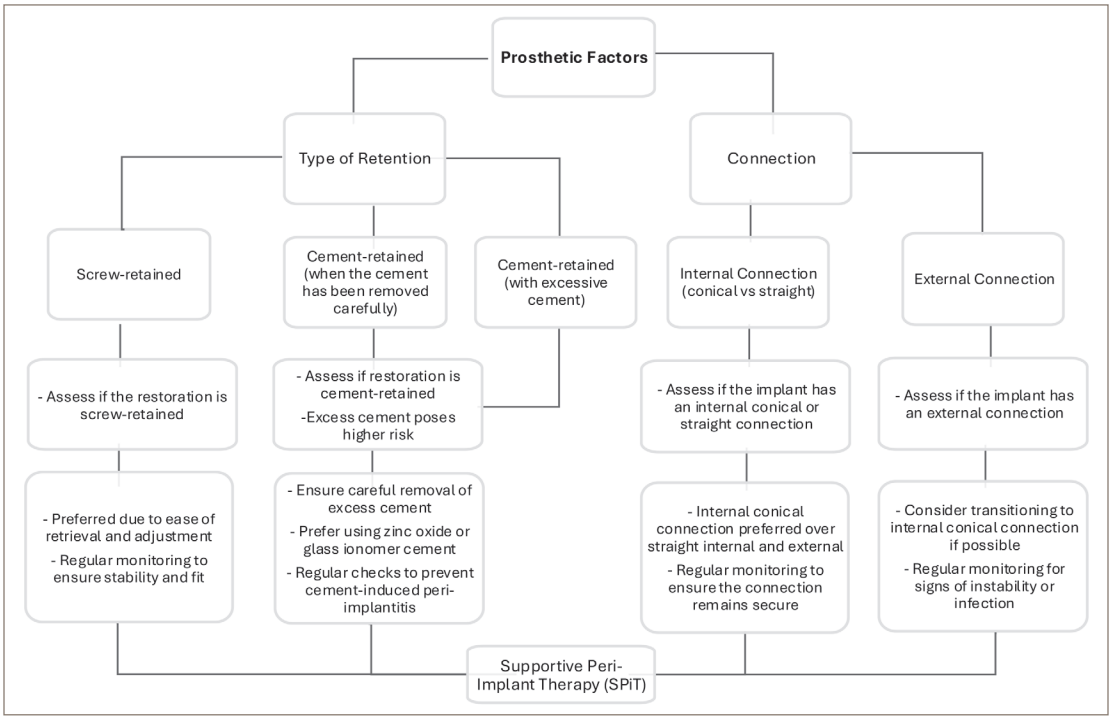
For obese patients or those with metabolic syndrome, the flowchart recommends a multifaceted treatment approach aimed at improving overall metabolic health and reducing inflammation. This includes promoting weight loss through dietary changes, increased physical activity, and, where appropriate, medical interventions to manage metabolic syndrome components such as hyperlipidemia, hypertension, and insulin resistance.[1,21,32] In conjunction with systemic management, close attention to peri-implant maintenance is critical. Regular follow-ups are essential to monitor the patient's progress in managing their metabolic health and to adjust the peri-implant care regimen as needed.[23] By addressing both systemic and local factors, the risk of peri-implant mucositis can be significantly reduced.

Prosthetic Factors

Peri-implantitis prosthetic risk factors are critical for the prevention of disease (Figs 5 to 9). The implant-abutment interface is an area where peri-implantitis can develop.[17,24,33,34] The risk is greatly influenced by whether the implant system uses a platform-switched or platform-matched design. Platform-switched abutments (where the abutment has a smaller diameter than the implant platform) have demonstrated a reduced risk of peri-implant bone loss by shifting the

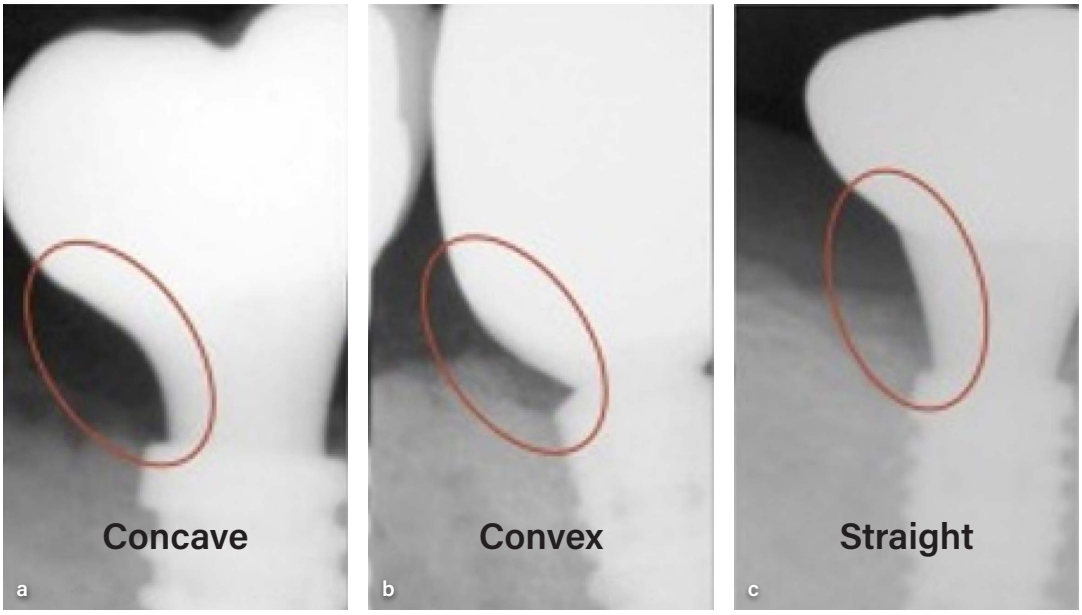


▲ **Fig 5** Flowchart of clinical recommendations for abutment-related prosthetic risk factors.



▲ **Fig 6** Flowchart of clinical recommendations for the retention and connection types as prosthetic risk factors.

inflammatory response away from the implant shoulder.[24] Abutment height is another key determinant in peri-implant health, particularly in relation to soft tissue support and plaque control. Abutment heights < 2 mm may lead to crestal bone remodeling, as shorter abutments do not provide sufficient vertical space for healthy attachment to form.[4] In contrast, abutments with heights ≥ 2 mm are typically preferred, as they allow for adequate soft tissue adhesion, which



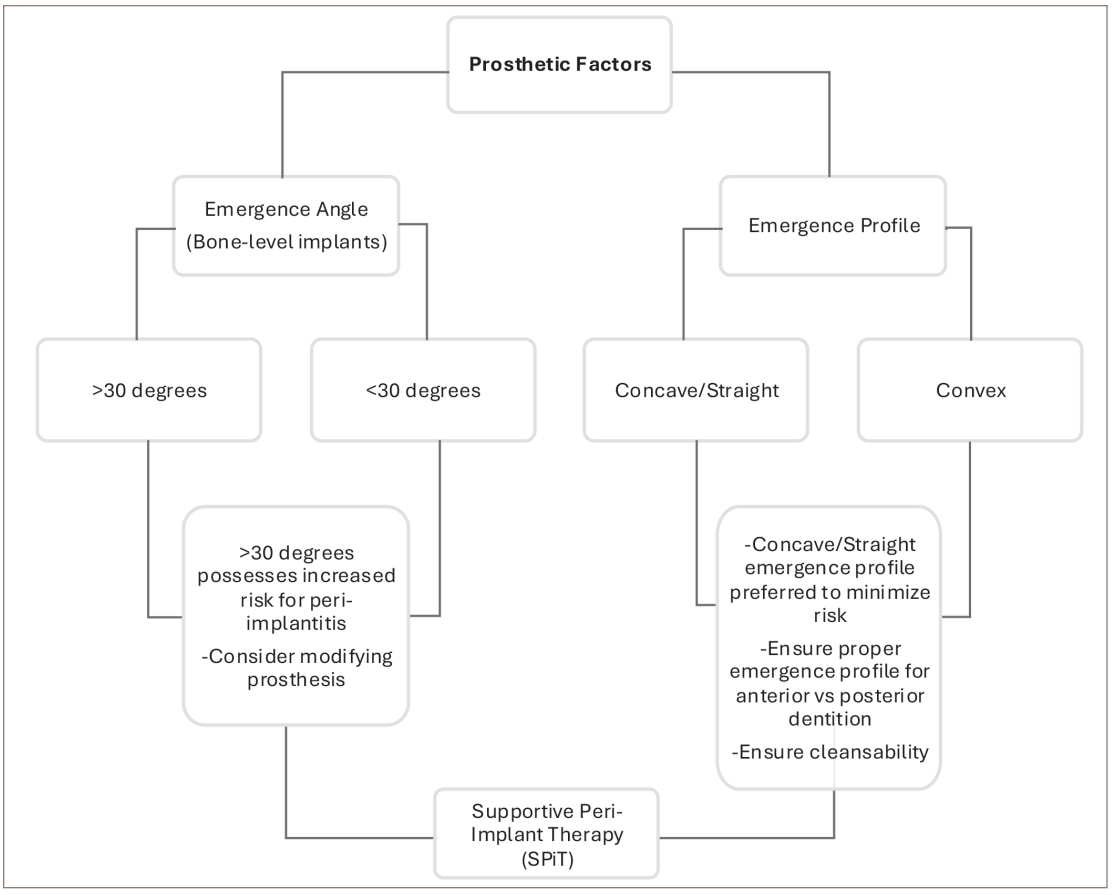
▲ **Fig 7** Radiographic examples of different implant emergence profiles: *(a)* concave, *(b)* convex, and *(c)* straight.

helps protect the peri-implant area from bacterial penetration.[24] Additionally, shorter abutments can lead to severe crown emergence profiles, making it harder for patients to maintain good hygiene around the implant. See Fig 5 for the flowchart on the abutment interface, height, and other factors.

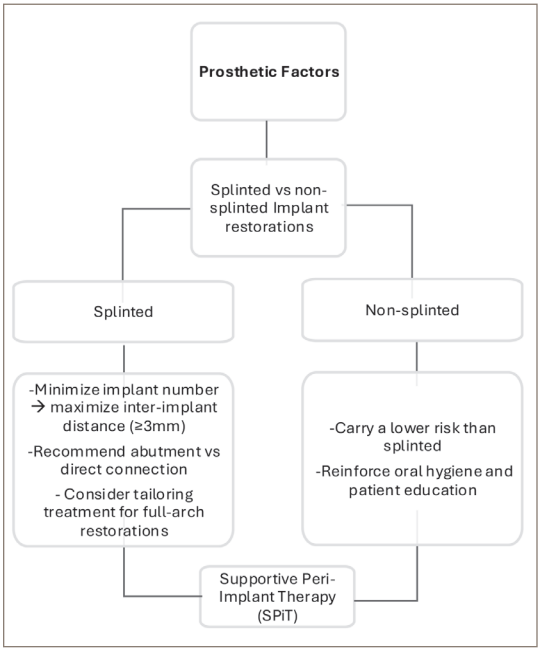
The emergence profile (see Fig 7) of an implant restoration is a critical determinant of peri-implant health, as it affects the ability of patients to maintain effective oral hygiene around the implant.[24] The Fig 8 flowchart identifies emergence angles > 30 degrees as high risk due to the difficulty in accessing and cleaning these areas, which can lead to increased plaque accumulation. A convex emergence profile is particularly problematic, as it creates undercuts where plaque can easily accumulate, exacerbating the risk of peri-implant inflammation and bone loss.[35] The flowchart advises a thorough assessment of the emergence profile during prosthesis planning and fabrication stages to minimize these risks.

The implant-abutment connection type also plays a critical role in peri-implant health (see Fig 6).[24] Internal connections are generally indicated due to their favorable stability and reduced micromovements compared to external connections. Internal connections tend to have tighter seals at the implant-abutment interface, which reduces the risk of bacterial penetration and subsequent peri-implantitis.[24,33,35] External connections are more prone to mechanical complications, such as screw loosening, and they create larger microgaps where bacteria can accumulate, leading to a higher risk of peri-implantitis. External connections may still be used in specific cases where the implant system requires them, but they necessitate more careful monitoring and maintenance.[35]

Several other factors related to the abutment can increase the risk of peri-implantitis, including the frequency of abutment removal, which can disrupt the soft tissue seal around the implant, leading to bacterial colonization and inflammation.[24,35,36] The use of CAD/CAM custom abutments is preferred, as they provide a more precise fit and better tissue support compared to standard/stock abutments.[37–39] Additionally, large gaps between the implant and abutment connections, or misalignment during abutment placement, can create niches for bacterial growth, increasing the risk of peri-implantitis. Splinted vs nonsplinted restorations also factor into risk (see Fig 9), with splinted restorations offering better stability in full-arch restorations but potentially increasing the risk of biofilm buildup if not designed with adequate spacing to facilitate oral hygiene efforts.[24]



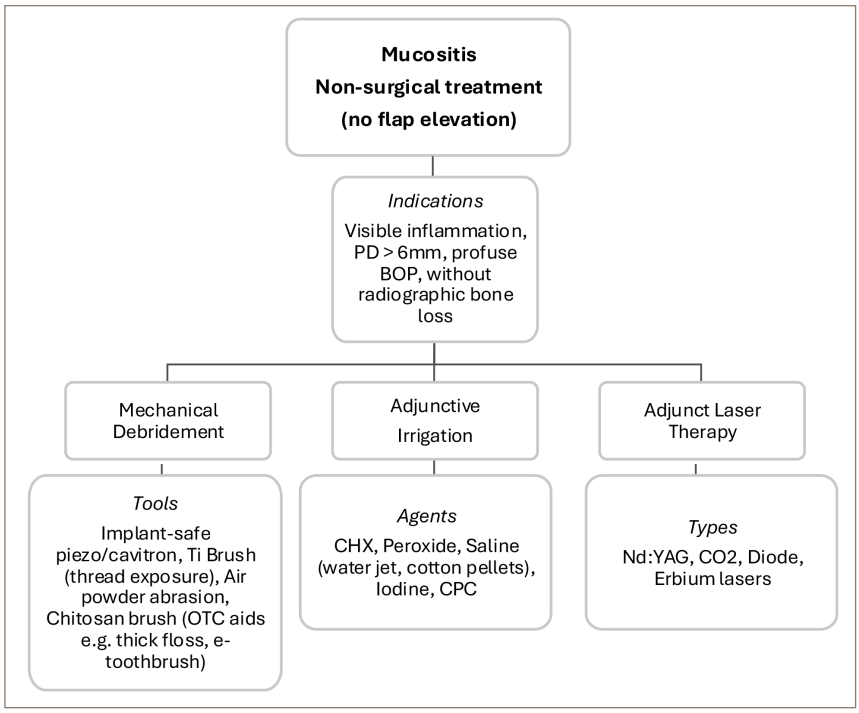
▲ **Fig 8** Flowchart of clinical recommendations for emergence angle and emergence profile as prosthetic risk factors.



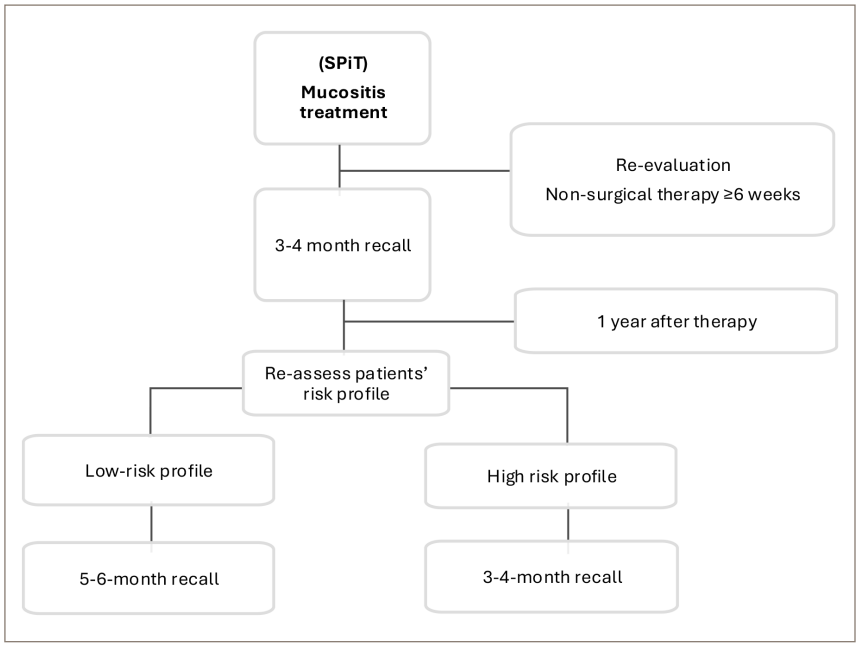
▲ **Fig 9** Flowchart of clinical recommendations for splinted vs nonsplinted restorations as prosthetic risk factors.

Comprehensive Management of Peri-implant Mucositis

Clinical management of peri-implant mucositis involves a comprehensive approach aimed at eliminating inflammation, preventing progression to peri-implantitis, and maintaining long-term peri-implant health[3,6] Figures 10 to 12 show the treatment approach flowcharts for mucositis. The initial step is nonsurgical mechanical debridement using implant-safe instruments, such as titanium curettes, piezoelectric ultrasonic scalers, and air-abrasion devices to effectively disrupt and remove biofilm without damaging the implant surface[6,23] (Fig 13). Disclosing agents can be employed to visualize plaque and biofilm buildup in difficult-to-see areas. If mechanical debridement alone is insufficient, adjunctive chemical irrigation with agents like chlorhexidine gluconate (0.12% to 0.2%), hydrogen peroxide (H_2O_2), or saline is

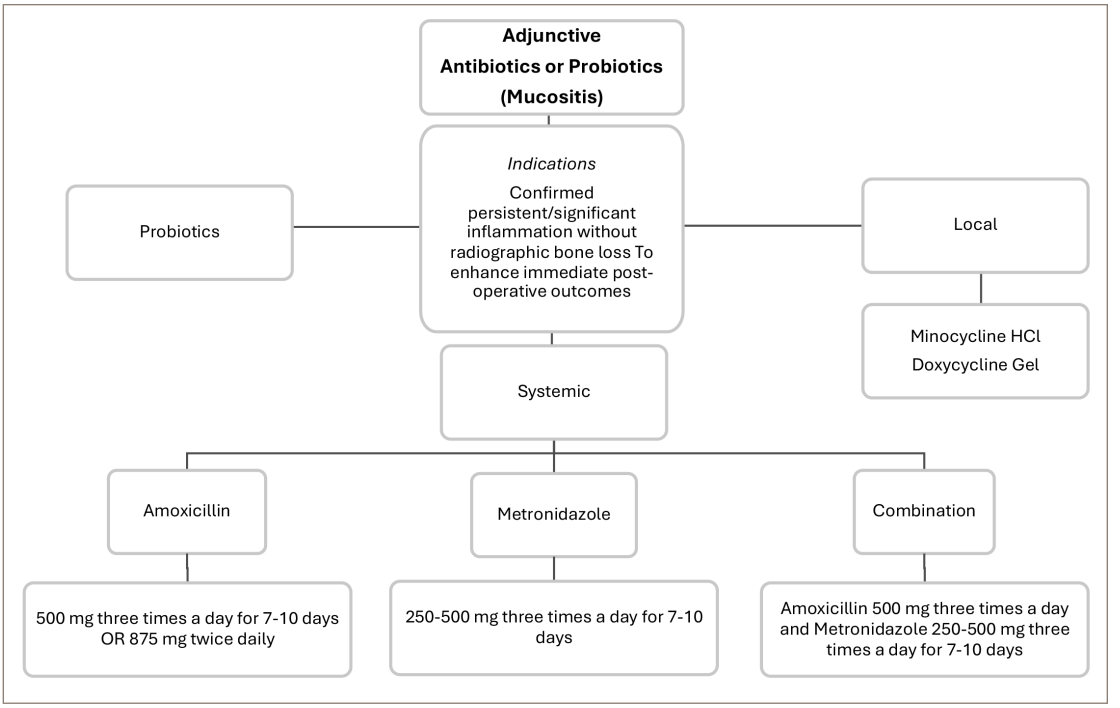


▶ **Fig 10** Flowchart of clinical recommendations for peri-implant mucositis therapy. Note that sites with 4- to 5-mm pocket probing depths and profuse bleeding, though not listed, may also warrant nonsurgical therapy to prevent disease progression in the absence of bone loss. This figure is meant to emphasize the current definition for peri-implant mucositis per the 2017 AAP World Workshop Classification.

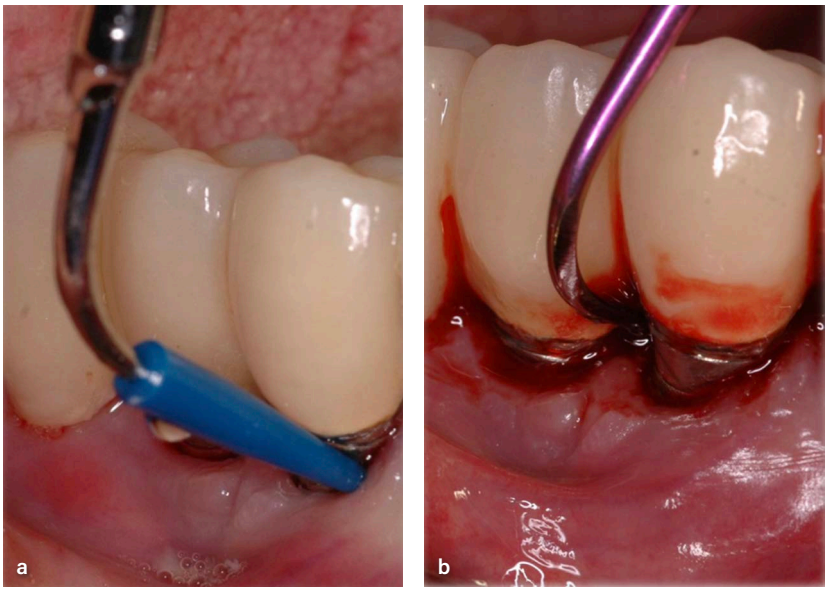


▶ **Fig 11** Flowchart of clinical recommendations for supportive peri-implant mucositis therapy.

recommended to further reduce bacterial load and inflammation.[9] In some localized deep probing depths, localized antimicrobial delivery systems, such as minocycline microspheres (eg, Arestin [Bausch Health]), can be applied directly into the peri-implant sulcus to target resistant bacterial colonies (Fig 14); however, this data remains inconclusive.[6,11,40] If pocket probing depths approach 6 mm without evidence of bone loss, laser therapy (eg, Nd:YAG, CO_2, diode, or Er:YAG lasers) may be considered as an adjunctive treatment to decontaminate peri-implant tissues and promote



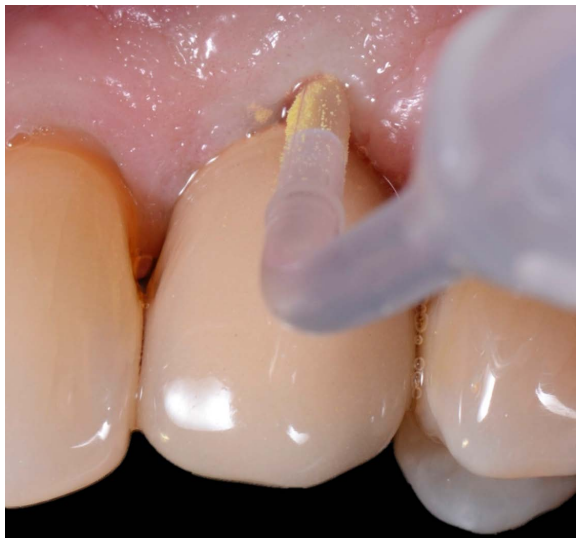
▲ **Fig 12** Flowchart of clinical recommendations for adjunctive mucositis therapy.

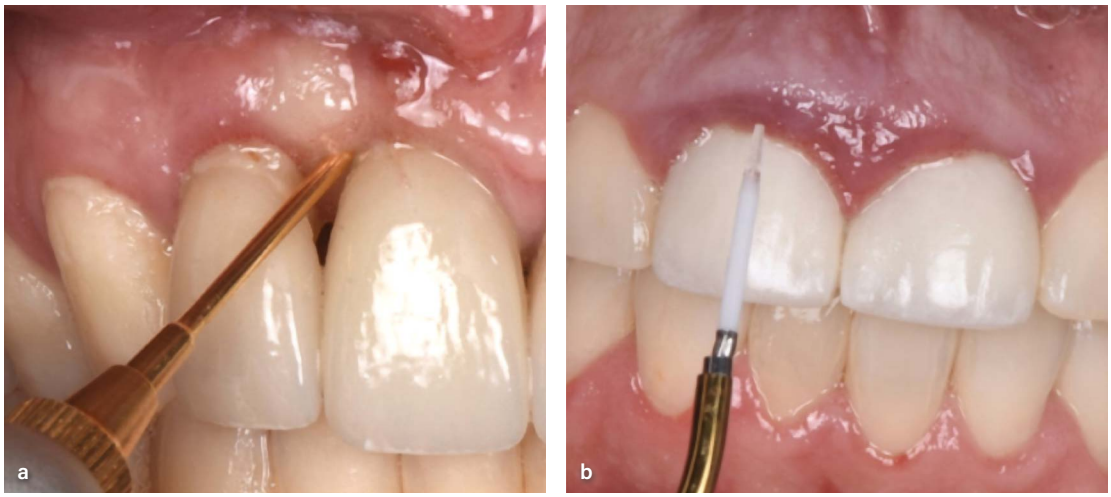


◀ **Fig 13** *(a and b)* Example case of mechanical debridement of peri-implant mucositis.

healing[8,12,25] (Fig 15). Lasers may reduce bacterial load and stimulate soft tissue regeneration in areas that are challenging to mechanically debride, but insignificant data exists. Probiotics have emerged as another adjunct in the management of peri-implantitis, offering a novel approach to rebalancing the microbial environment around implants. The theory behind probiotic use in peri-implantitis is that introducing beneficial bacteria can outcompete pathogenic microorganisms, thereby reducing inflammation and promoting a healthier oral microbiome.[13] Probiotics typically comprise strains of *Lactobacillus* and *Bifidobacterium*, which are known for their antimicrobial and anti-inflammatory properties following these interventions; however, this data is solely preliminary.[13]

▶ **Fig 14** Example of adjunctive antibiotic therapy for peri-implant mucositis.





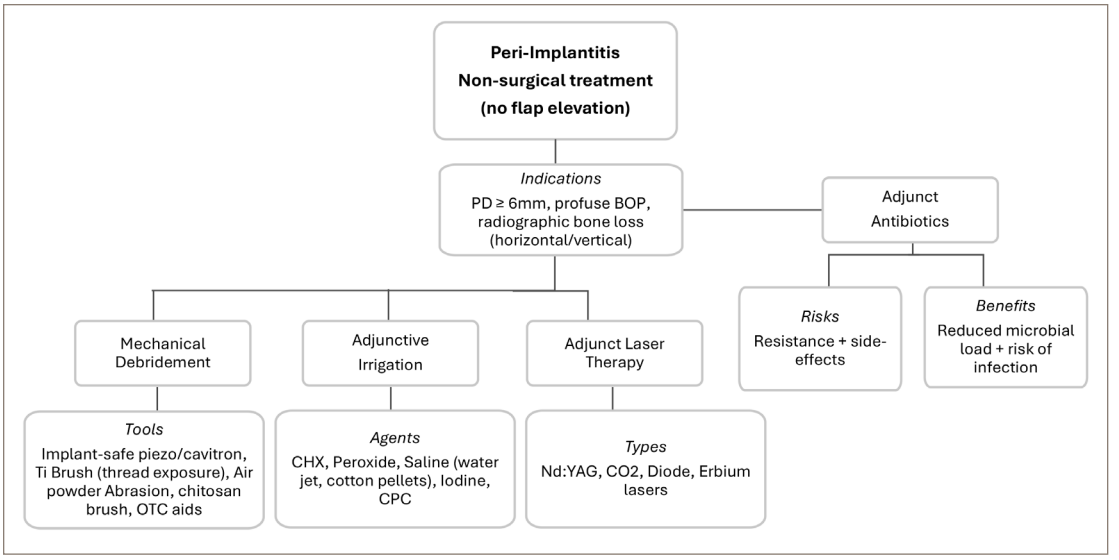
▲ **Fig 15** *(a and b)* Clinical examples of laser therapy for peri-implant mucositis.

Patients should be placed on a structured SPiT regimen with recall visits scheduled every 3 to 4 months.[23] At each visit, professional mechanical debridement should be repeated as necessary, and additional adjunctive treatments should be applied if biofilm accumulation is detected. Patient education is crucial, emphasizing daily oral hygiene practices such as using soft-bristled toothbrushes, interdental brushes, and antimicrobial rinses to prevent plaque buildup.[6] This combination of professional intervention and rigorous at-home care is essential to reverse the inflammatory process, prevent progression to peri-implantitis, and ensure long-term peri-implant health.

Management of Peri-implantitis

Nonsurgical Management of Peri-implantitis

Nonsurgical management of peri-implantitis is the first line of treatment when addressing early to moderate cases of inflammation and minimal bone loss around implants.[6] The primary goal of nonsurgical therapy is to reduce bacterial biofilm and inflammation while preserving the implant and surrounding bone. Figure 16 shows the treatment approach flowchart for nonsurgical peri-implantitis management. Mechanical debridement is the cornerstone of this treatment, involving the use

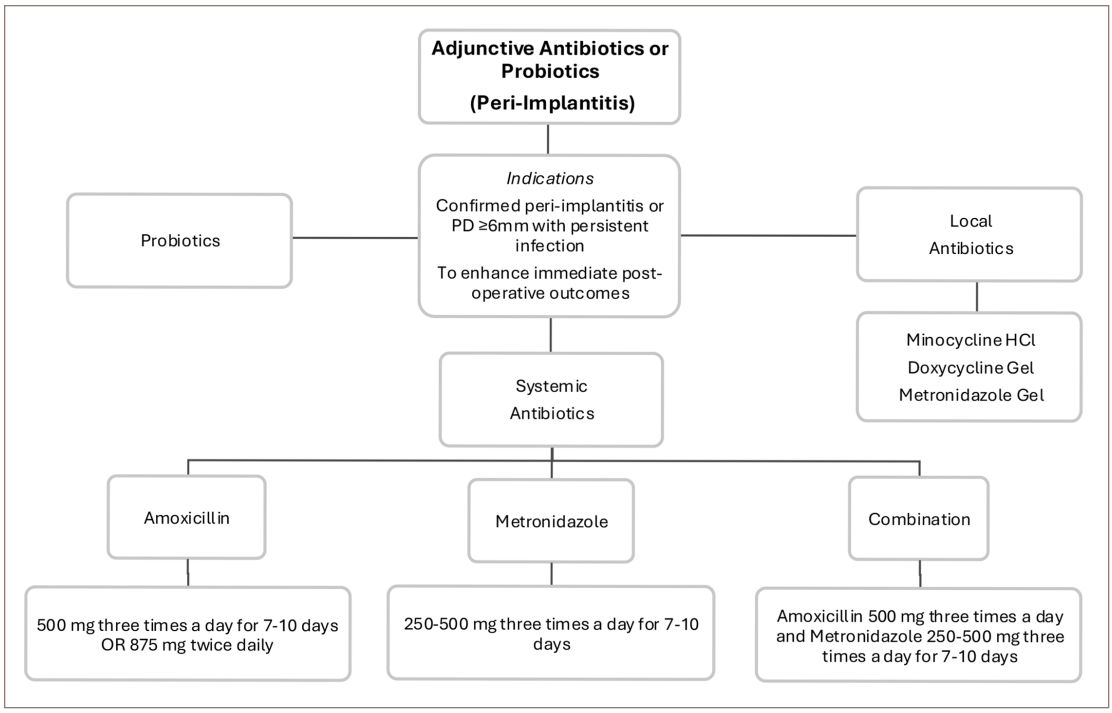


▲ **Fig 16** Flowchart of clinical recommendations for nonsurgical peri-implantitis therapy.

of implant-safe instruments like titanium curettes, ultrasonic scalers with plastic or carbon tips, or air-powder abrasion devices to disrupt biofilm on implant surfaces without significantly altering the implant surface.[6,25] Special attention is given to using implant-specific tools, such as the TiBrush (Straumann) for exposed threads, which is designed to thoroughly clean implant surfaces.[18,26,41] Alongside mechanical debridement, adjunctive therapies may also play a role in enhancing outcomes, as shown in the Fig 17 flowchart. However, variable conclusions have been presented in the literature.

One of the most commonly used adjuncts is chemotherapeutic agents (such as chlorhexidine, hydrogen peroxide, saline, or iodine), which are applied topically or used in subgingival irrigation to reduce bacterial load.[9,40,41] The use of antibiotics, whether systemically or locally delivered, can also be considered, especially in cases with significant inflammation or infection.[23,40] Antibiotics can be delivered locally, like minocycline microspheres, or placed directly in the peri-implant pocket to target the infection site.[11,40] Systemic antibiotics (such as a combination of amoxicillin and metronidazole) may be prescribed for more widespread infection control.[5,41,42] In addition to chemical agents, lasers like Nd:YAG, CO_2, diode, and Er:YAG have been explored for their ability to decontaminate the implant surface and reduce inflammation, often with the possible benefit of tissue healing and biostimulation.[43] Another emerging treatment modality is antimicrobial photodynamic therapy, which utilizes a photosensitizing agent activated by light to kill bacteria in the peri-implant pocket.[7,40]

Patient education and oral hygiene improvement are integral to case management.[3,5,23] Because biofilm control is key to preventing the progression of peri-implantitis, patients must be trained in proper home care techniques using tools such as interdental brushes, floss (preferably thick types) or superfloss, and electric toothbrushes. Regular maintenance visits are essential for monitoring the health of peri-implant tissues and ensuring that plaque and biofilm control remain effective. Typically, patients are recalled at least every 3 to 4 months during active surveillance, with adjustments made based on risk profiles.[23] While nonsurgical therapy can effectively manage mild to moderate peri-implantitis, it often requires close follow-up and may need to be combined with surgical interventions if the condition does not respond adequately. Nonsurgical approaches are most successful in cases without deep bone defects or extensive inflammation, focusing on preserving peri-implant health through meticulous biofilm removal, patient adherence, and adjunctive therapies.[41]



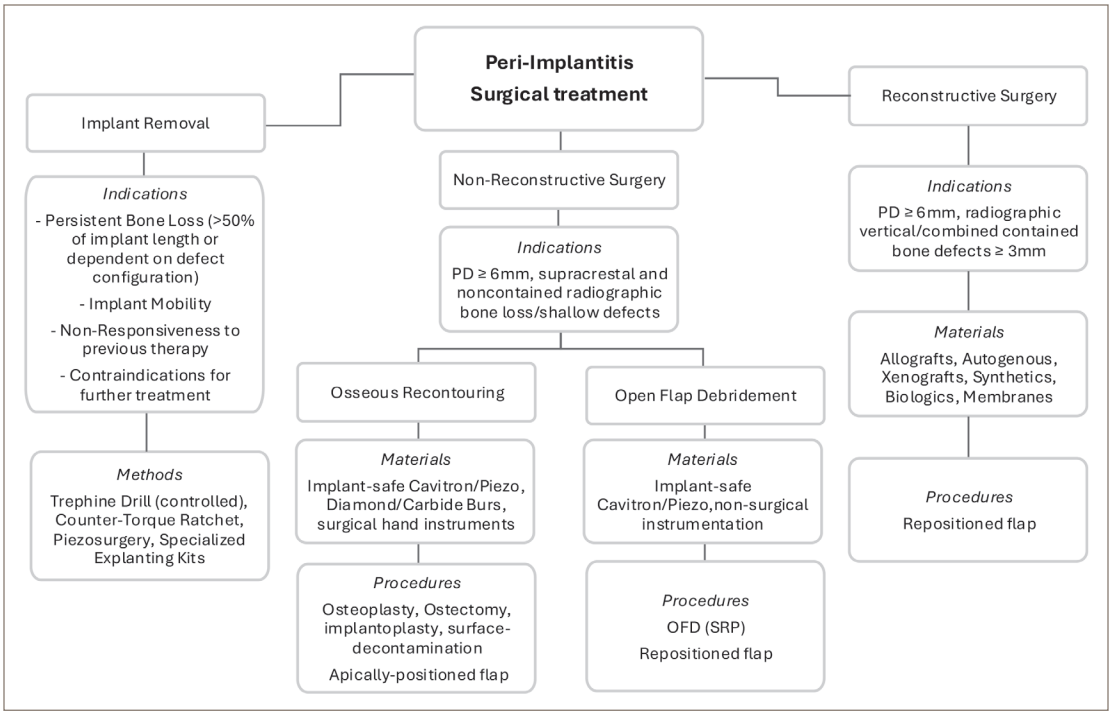
▲ **Fig 17** Flowchart of clinical recommendations for adjunctive antibiotics or probiotics in peri-implantitis treatment.

Surgical Management of Peri-implantitis

Surgical management of peri-implantitis aims to preserve the implant by not only preventing its failure and avoiding its removal whenever possible but also to restore it to health, if possible.[16,18,44,45] The objectives include providing access to the implant surface for professional and at-home care, reconstructing lost bone, and implementing procedures to prevent further disease progression. The Fig 18 flowchart shows treatment approaches for the surgical management of peri-implantitis.

Implant removal

When peri-implantitis results in severe bone loss > 50% of the implant length, nonregenerable defects, recurrent infections unresponsive to treatment, or compromised implant stability, implant removal may become a necessary intervention.[18,45,46] The decision to remove an implant should be based on a comprehensive clinical and radiographic assessment, considering factors such as the extent of bone loss, implant mobility, and patient-specific risk factors like systemic health conditions, poor oral hygiene, and/or compromising adjacent teeth or implants. The primary goal of implant explanation is to remove the implant while preserving as much surrounding bone and soft tissue as possible to facilitate future rehabilitation. Various techniques are available for implant removal.[47] One method involves the use of counter-torque ratchets and explantation devices, which engage the internal connection of the implant, allowing it to be unscrewed in a reverse manner.[3,21] This minimally invasive approach aims to preserve bone but is only effective if the implant threads are not extensively fused to the bone due to osseointegration. Another technique employs trephine burs—hollow, cylindrical drills that cut around the implant, removing it along with a small amount of surrounding bone.[18,21] While effective, this method results in more significant bone loss and may require bone grafting. Piezoelectric surgery may also be utilized; these instruments use ultrasonic vibrations to disrupt the bone-implant interface, facilitating implant removal while minimizing damage to the bone.[48] Additionally, laser-assisted removal has been reported to aid explantation; however, this technique



▲ **Fig 18** Flowchart of clinical recommendations for surgical peri-implantitis therapy.

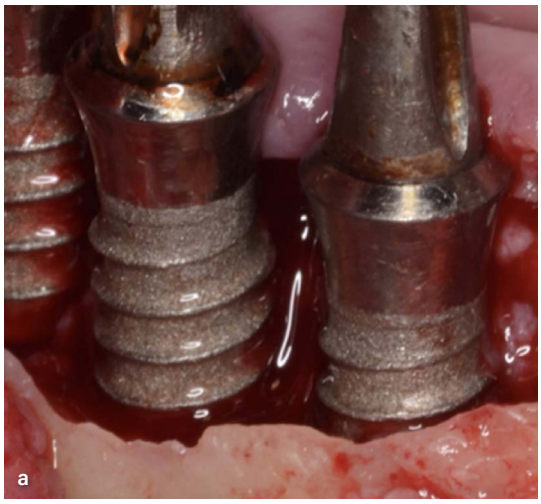
requires specialized equipment and expertise.[49] After removal, thorough debridement of the site is essential to eliminate any residual infected tissue. Depending on the extent of the defect, bone grafting procedures may be performed immediately to regenerate lost bone and prepare the site for possible future implant placement.[16,47]

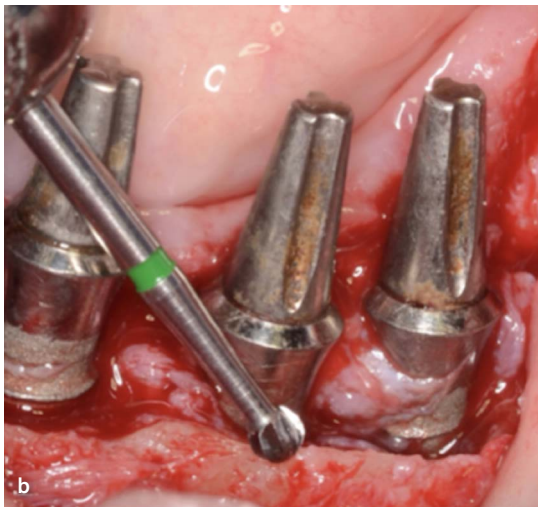
Access flap with nonreconstructive procedures

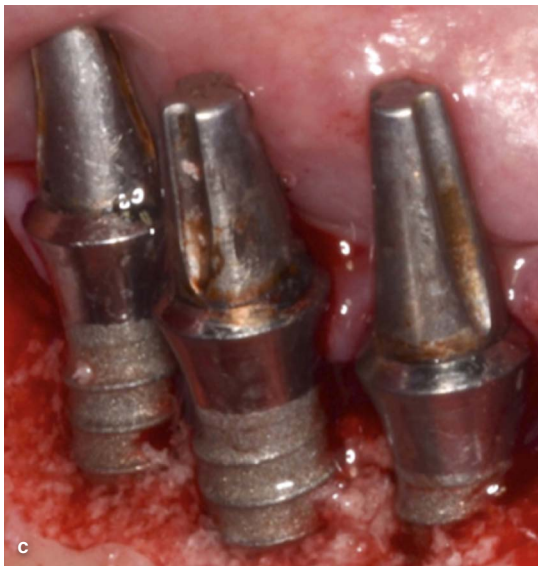
Flap procedures focus on providing access to the contaminated implant surface for debridement and decontamination.[18,28] A full-thickness flap with or without vertical releasing incisions, which may extend to adjacent teeth or implants, is often necessary to ensure comprehensive access. Granulomatous tissue and inflamed soft tissue are meticulously removed using curettes, rotary instruments, or other methods, followed by thorough decontamination of the implant surface. Once completed, the flap is repositioned and sutured back in place using suturing techniques that ensure primary closure and minimize pocket recurrence. Minimally invasive flap elevation techniques can be employed in cases with minimal soft tissue involvement, employing small, precise incisions to access the peri-implant defect while minimizing tissue trauma.[5,28]

Access flap with nonreconstructive procedures

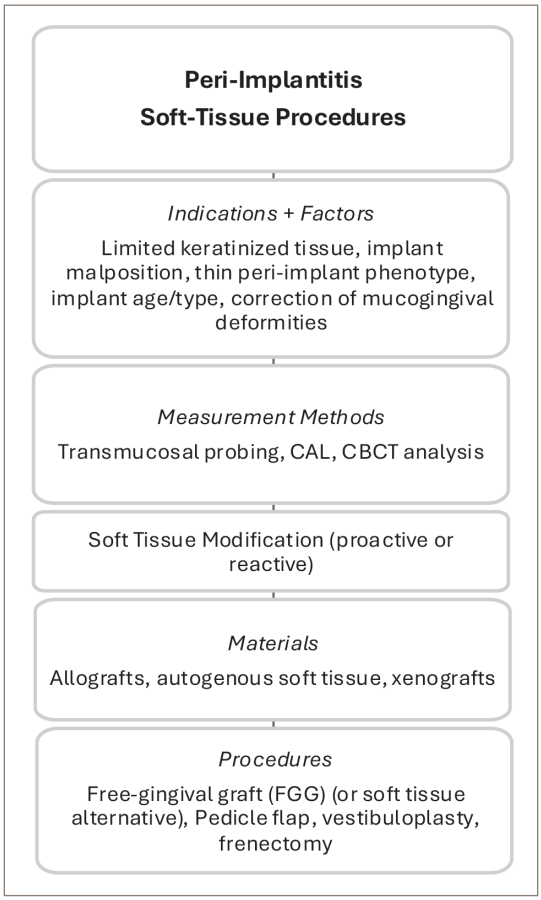
When combining access flap debridement with resective procedures, the incision design may include vertical releasing incisions for better access and visibility.[50] This design allows the flap to be apically positioned, facilitating pocket reduction while maintaining as much keratinized tissue as possible to facilitate oral hygiene efforts. Osseous resective surgery, such as bone recontouring or removal, may need to be performed to reshape the nonsupporting bone around the implant, creating a more favorable contour for soft tissue healing[51] (Fig 19). Granulomatous tissue is removed, and the implant surface is thoroughly debrided as described above. Finally, the flap is repositioned and secured with sutures to reduce pocket depth and promote stable soft tissue healing. Apical positioning of the flap can be employed in cases where significant pocket reduction is required after osseous resection to ensure reduced pocket depths and better soft tissue adaptation.[18,50,51]







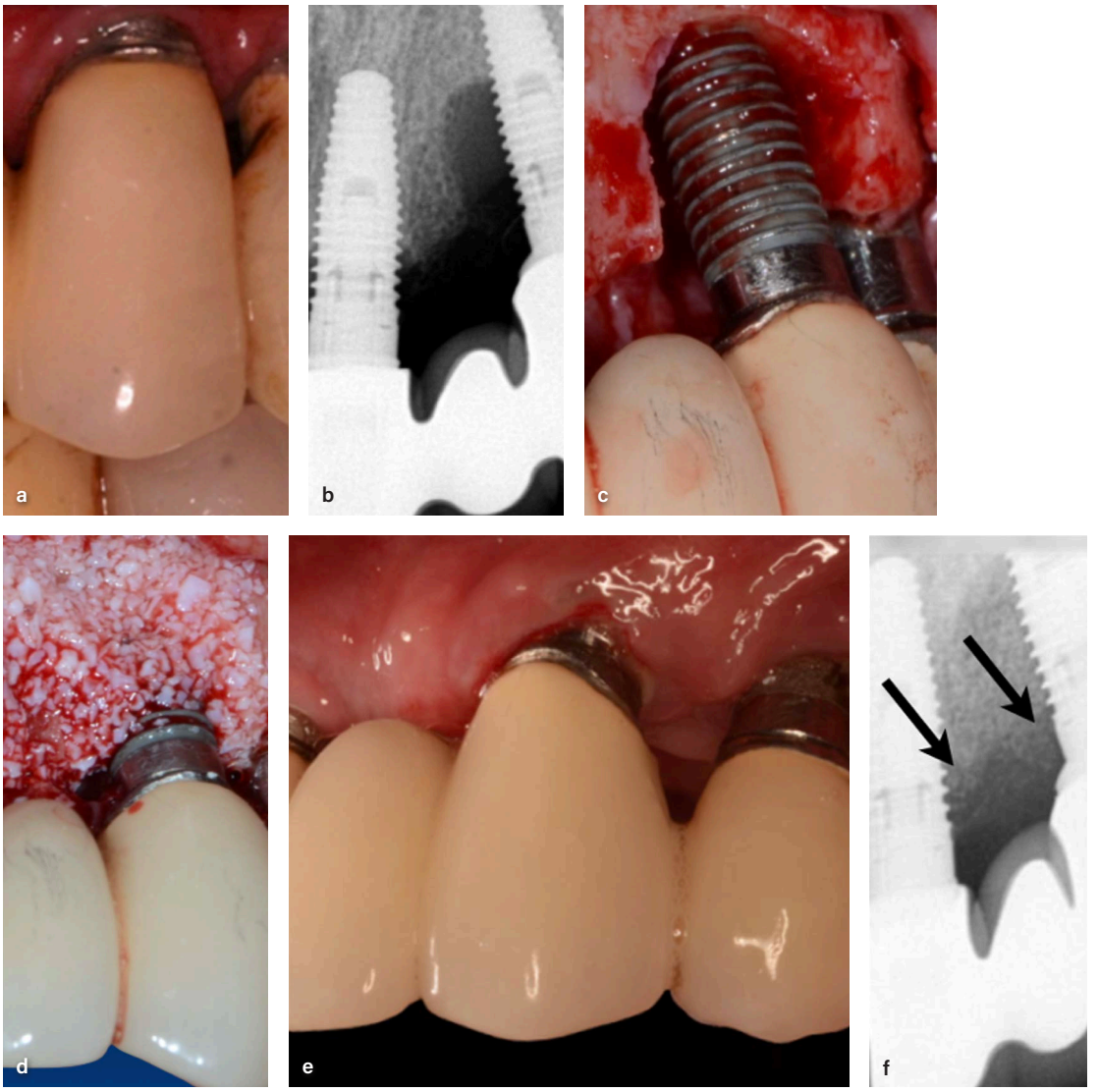
▲ **Fig 19** Resective therapy for the treatment of peri-implantitis. *(a)* Implant site after debridement. *(b)* Osseous resective procedure with a rotary bur. *(c)* Recontoured osseous with grafting of the remaining defect.



▲ **Fig 20** Flowchart of clinical recommendations for soft tissue reconstruction procedures in the treatment of peri-implantitis.

Reconstructive procedures

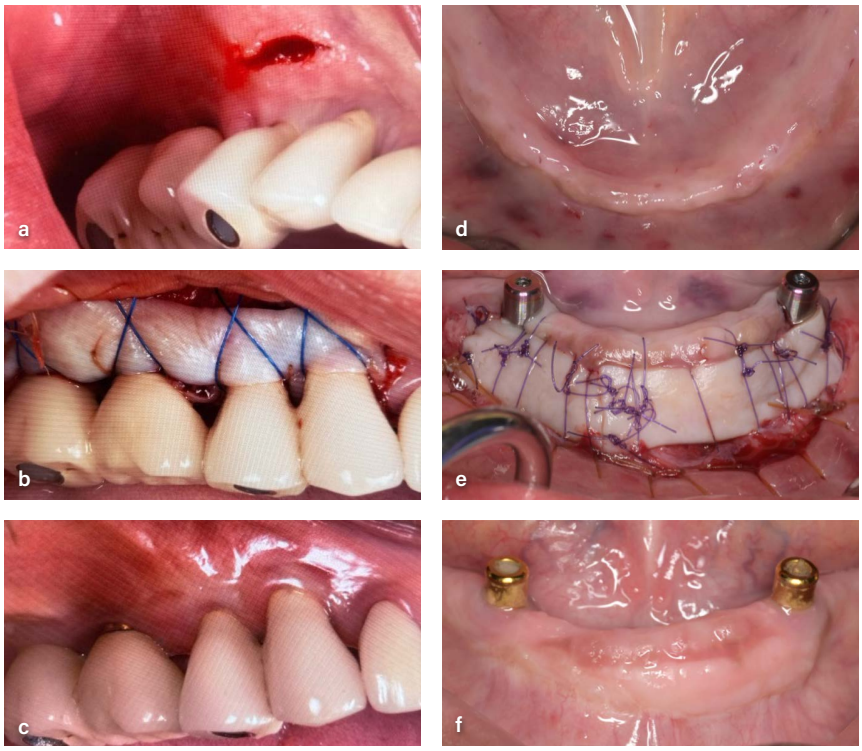
Reconstructive procedures aim to restore lost bone and soft tissue around the implant.[16,29] Figure 20 shows the flowchart of soft tissue procedures, and Figs 21 and 22 show clinical examples of reconstruction and augmentation therapies, respectively. A full-thickness flap with or without vertical releasing incisions, which may extend to adjacent teeth or implants, is often necessary to ensure adequate access. After elevating the flap to expose the peri-implant defect, granulomatous tissue is removed, and the exposed implant surface is decontaminated. Bone replacement graft materials (such as autografts, allografts, xenografts, or alloplasts) are placed into the defect, often combined with barrier membranes to promote guided bone regeneration.[16] The flap is adapted to ensure tension-free closure, using appropriate



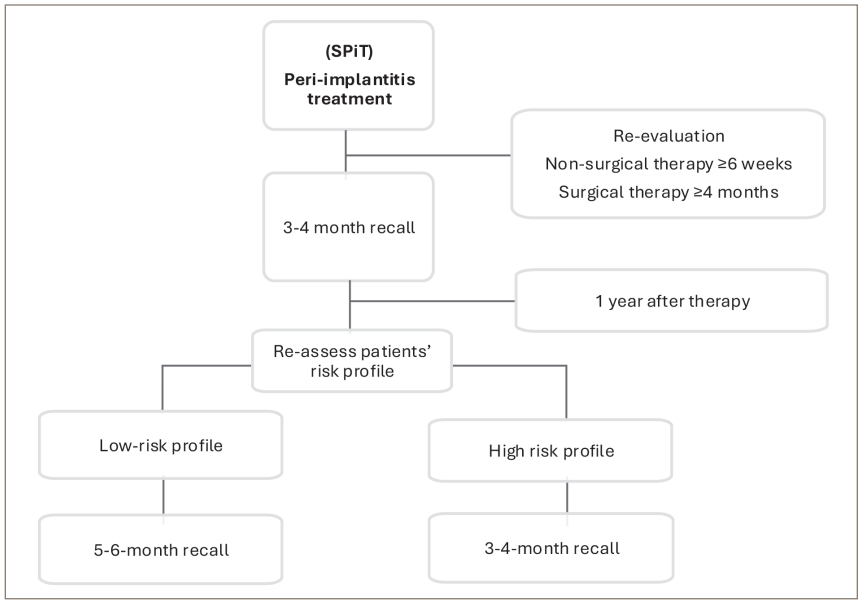
▲ **Fig 21** Reconstructive therapy for the treatment of peri-implantitis. *(a)* Clinical appearance of peri-implantitis with soft tissue inflammation. *(b)* Radiograph showing peri-implant bone loss. *(c)* View of the implant following debridement and surface decontamination. *(d)* A bone graft was placed for osseous reconstruction. *(e)* Soft tissue after reconstructive surgery. *(f)* Radiograph showing bone fill *(arrows).*

techniques that secure the flap and promote stable healing.

Reconstructive procedures take into account the amount of bone loss, categorized as < 25%, 25% to 50%, or > 50% bone loss.[52] The types of considered defects include supraosseous, infraosseous (which can be 1-wall, 2-wall, or 3-wall defects), and combination.[21] Soft tissue considerations include phenotypes that may be lacking in apicocoronal width or thickness and potentially require additional soft tissue grafting to enhance the quantity and quality.[4] Flap techniques should consider both lesion access and soft tissue retention as much as possible and should, in certain instances, submerge the implant to protect the implant and graft materials and allow clotting during the healing process. Implant removal should be considered in cases of severe bone loss (> 50%), nonregenerable defects, recurrent peri-implantitis, or when the implant stability or support of adjacent teeth or implants is compromised.[47] The range of treatment techniques include soft tissue augmentation, hard tissue augmentation, and combinations of both.



▶ **Fig 22** Soft tissue augmentation therapy for the prevention of peri-implantitis. *(a and b)* Peri-implant tissues lacking attached and keratinized tissue. *(c and d)* A free gingival graft was sutured in place. *(e and f)* Peri-implant tissues after healing, showing improved tissue quality.



▶ **Fig 23** Flowchart of clinical recommendations for supportive peri-implant therapy.

Supportive Peri-implant Therapy

SPiT is a critical component of long-term peri-implant health maintenance and disease prevention, particularly following the treatment of peri-implant mucositis and peri-implantitis.[23] The Fig 23 flowchart emphasizes the need for tailoring SPiT intervals based on the patient's overall risk profile. High-risk patients—including those with a history of periodontitis, poorly controlled diabetes, complex prosthetic restorations, or significant plaque accumulation—require more frequent SPiT visits, typically every 3 to 4 months. These intervals are necessary to ensure early detection and management of any signs of peri-implant disease.

In contrast, low-risk patients may be scheduled for SPiT visits every 5 to 6 months, provided that their peri-implant tissues remain stable. Table 4 shows the management strategy and SPiT intervals dependent on the peri-implant status, present risk factors, implant age and condition, and patient compliance.

SPiT involves a combination of professional cleanings to remove plaque and biofilm, monitoring of peri-implant tissue health, and reinforcement of oral hygiene practices.[23] Specifically, patients should receive detailed guidance on home-care prevention methods, including the use of soft-bristled or implant-specific toothbrushes, daily interdental cleaning with implant-safe floss or interdental brushes, and adjunctive oral irrigation devices when appropriate. Education should highlight lifestyle adjustments such as smoking cessation and glycemic control in diabetic patients, alongside recognizing early symptoms of peri-implant disease, such as bleeding, swelling, or discomfort, to facilitate prompt professional intervention. During SPiT visits, clinicians should use implant-safe instruments for debridement and assess the condition of both the soft tissues and the implant-abutment interface. Any signs of inflammation or tissue deterioration should prompt immediate intervention, which may include additional mechanical debridement, application of antiseptic agents (such as chlorhexidine), or localized antibiotic therapy, and perhaps even surgical intervention.[5,9,16,40] Ongoing patient education is of high importance, as is ensuring that patients understand the role of daily oral hygiene in preventing peri-implant disease. The patient risk profile should be regularly reassessed, particularly after the first year of treatment, to determine whether adjustments to the SPiT interval are necessary. By adhering to these guidelines, clinicians can effectively prevent the recurrence of peri-implant diseases and ensure the long-term success of dental implants.

Conclusions

With the exponential increase in implant placement, managing complications such as peri-implant mucositis and peri-implantitis has become increasingly important. This publication presents comprehensive flowcharts for these conditions, highlighting the crucial role of prevention and early intervention. The importance of proper treatment planning, risk analysis, appropriate therapy, and long-term maintenance cannot be understated. The findings from the conference confirm that SPiT and proper oral hygiene instruction effectively minimize the risk of these diseases, underscoring the significance of patient education and regular maintenance. However, as the clinicians' understanding of peri-implant diseases continues to evolve, there are still gaps in knowledge regarding their complex causes and optimal treatments. Continued research is essential to deepen this understanding and improve management strategies. By integrating current best practices with ongoing studies, these implant-related complications can be better addressed, and the long-term success of implants can be enhanced.

Acknowledgments

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Author contributions: J.P.F. conceptualized the study framework, oversaw the design and execution of the project, and led the manuscript drafting and coordinated revisions. S.M. assisted in drafting key sections of the manuscript and provided critical revisions and images. H.S. provided substantive feedback, significant revisions, and supplemented images. T.A. assisted in drafting the manuscript and contributed significantly to the critical review and revision of the content.

References

1. Galarraga-Vinueza ME, Pagni S, Finkelman M, Schoenbaum T, Chambrone L. Prevalence, incidence, systemic, behavioral, and patient-related risk factors and indicators for peri-implant diseases: An AO/AAP systematic review and meta-analysis. J Periodontol 2025;96. doi: 10.1002/jper.24-0154.
2. Wojtowicz A. From rough surface to peri-implantitis, hybrid implants and subcrustal implant placement: A mini review of last decade research and clinical findings in implant dentistry. Mod Res Dent 2020;5:549–551.

Table 4 Supportive Peri-implant Therapy Protocols

Parameter	Guideline	Management strategy
Peri-implant status	Healthy implant (no clinical signs of inflammation)	Routine SPiT schedule (every 6–12 mo): Focus on regular monitoring, professional cleaning, and patient education. Emphasize maintaining excellent oral hygiene and controlling systemic conditions, especially in patients with obesity or diabetes. Consider radiographic evaluation based on clinical findings.
	Peri-implant mucositis (inflammation w/out bone loss)	Increased SPiT frequency (every 3–6 mo): Increase frequency of SPiT visits to manage inflammation. Implement mechanical debridement of the implant surface, possibly with adjunctive antiseptic agents (eg, chlorhexidine). Reinforce oral hygiene practices and evaluate patient compliance. For patients with obesity or other systemic conditions, consider modifying the treatment plan to address these factors. Regular monitoring for progression to peri-implantitis is essential.
	Peri-implantitis (inflammation w/ bone loss)	Frequent SPiT (every 1–3 mo): Aggressive management is required. Perform thorough debridement of the implant surface, possibly including surgical intervention, depending on the severity of bone loss. Use adjunctive therapies such as systemic or local antibiotics, antiseptics, or laser treatment. Radiographic monitoring is crucial to assess bone levels and the effectiveness of treatment. Collaboration with medical providers may be necessary, especially if systemic factors like obesity are contributing to the inflammation.
Systemic risk factors	Low risk (no significant systemic health issues)	Routine SPiT (6–12 mo): Standard maintenance is generally sufficient. However, consider more frequent visits if the patient has a history of periodontal disease or other risk factors. Focus on preventive care and education.
	Moderate risk (overweight, controlled systemic conditions)	Increased SPiT (3–6 mo): Patients with risk factors like being overweight or having controlled systemic conditions (eg, well-managed diabetes) should have more frequent SPiT visits. This helps in early detection of peri-implant diseases and effective management. Emphasize the importance of controlling these systemic conditions to prevent peri-implant disease progression.
	High risk (obesity, uncontrolled systemic conditions)	Frequent SPiT (1–3 mo): Patients with obesity or poorly controlled systemic conditions are at high risk for peri-implantitis. Intensive monitoring and treatment are required. Consider adjunctive therapies (eg, antibiotics, antiseptics) and coordinate care with the patient's healthcare provider. Aggressive inflammation control is necessary, and surgical intervention may be needed if nonsurgical approaches are insufficient. Radiographic evaluations should be frequent to monitor bone loss.
Implant age and condition	Recent implant placement (< 1 y)	Frequent SPiT (3 mo): Early detection of peri-implant diseases is critical in the first year after implant placement. Regular professional cleanings, patient education on oral hygiene, and close monitoring for signs of mucositis or peri-implantitis are necessary. Adjust frequency based on the patient's risk profile, especially if systemic conditions are present. Consider using adjunctive diagnostic tools like peri-implant probing and radiographs to monitor implant health.
	Older implant (> 5 ys)	Increased SPiT (3–6 mo): Implants in place for over 5 years require close monitoring, especially in patients with risk factors such as a history of periodontitis or systemic conditions like obesity or diabetes. Regular debridement, possibly with adjunctive antiseptic treatments, is recommended. Be vigilant for signs of peri-implantitis, and manage any inflammation promptly. Radiographic monitoring of bone levels should be conducted periodically to assess for any progressive bone loss.
Patient compliance	High compliance (good oral hygiene, regular SPiT visits)	Routine SPiT (6–12 mo): Continue a standard SPiT protocol with regular monitoring and professional cleanings. Reinforce positive behaviors and adjust frequency only if new risk factors emerge or if there are changes in systemic health.
	Low compliance (poor oral hygiene, irregular SPiT visits)	Frequent SPiT (1–3 mo): Patients with poor compliance are at higher risk for peri-implant disease. Increase the frequency of SPiT visits to ensure early detection and intervention. Intensive patient education is critical, focusing on the importance of oral hygiene and regular follow-up. Consider using adjunctive therapies or motivational interviewing techniques to improve compliance. Monitor for disease progression with regular probing and radiographs.

3. Berglundh T, Armitage G, Araujo MG, et al. Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions. J Periodontol 2018;89(suppl 1):s313–s318.
4. Tavelli L, Barootchi S. Prevalence, incidence, risk, and protective factors for soft tissue dehiscences at implant sites in the absence of disease: An AO/AAP systematic review and meta-regression analysis. J Periodontol 2025;96. doi: 10.1002/jper.24-0119.
5. Figuero E, Graziani F, Sanz I, Herrera D, Sanz M. Management of peri-implant mucositis and peri-implantitis. Periodontology 2000 2014;66:255–273.
6. Lin GH, Chambrone L, Rajendran Y, Avila-Ortiz G. Treatment of peri-implant mucositis: An AAP/AO systematic review and meta-analysis. Int J Oral Maxillofac Implants 2025;40(suppl):s49–s72.
7. Zeza B, Farina R, Pilloni A, Mongardini C. Clinical outcomes of experimental gingivitis and peri-implant mucositis treatment with professionally administered plaque removal and photodynamic therapy. Int J Dent Hyg 2018;16:e58–e64.
8. Aimetti M, Mariani GM, Ferrarotti F, Ercoli E, Liu CC, Romano F. Adjunctive efficacy of diode laser in the treatment of peri-implant mucositis with mechanical therapy: A randomized clinical trial. Clin Oral Implants Res 2019;30:429–438.
9. Tütüncüoğlu S, Cetinkaya BO, Pamuk F, et al. Clinical and biochemical evaluation of oral irrigation in patients with peri-implant mucositis: A randomized clinical trial. Clin Oral Investig 2022;26:659–671.
10. Sanchez-Perez A, Nicolas-Silvente AI, Sanchez-Matas C, Cascales-Pina E, Macia-Manresa V, Romanos GE. Control of peri-implant mucous inflammation by using chlorhexidine or ultraviolet C radiation for cleaning healing abutments. Double-blind randomized clinical trial. Materials (Basel) 2020;13:1124.
11. Schenk G, Flemmig TF, Betz T, Reuther J, Klaiber B. Controlled local delivery of tetracycline HCl in the treatment of periimplant mucosal hyperplasia and mucositis. A controlled case series. Clin Oral Implants Res 1997;8:427–433.
12. Tenore G, Montori A, Mohsen A, Mattarelli G, Palaia G, Romeo U. Evaluation of adjunctive efficacy of diode laser in the treatment of peri-implant mucositis: A randomized clinical trial. Lasers Med Sci 2020;35:1411–1417.
13. Sargolzaei N, Arab H, Gerayeli M, Ivani F. Evaluation of the topical effect of probiotic mouthwash in the treatment of patients with peri-implant mucositis. J Long Term Eff Med Implants 2022;32:85–91.
14. Herrera D, Berglundh T, Schwarz F, et al. Prevention and treatment of peri-implant diseases—The EFP S3 level clinical practice guideline. J Clin Periodontol 2023;50(suppl 26):4–76.
15. Garaicoa-Pazmino C, Couso-Queiruga E, Monje A, Avila-Ortiz G, Castilho RM, Amo FSLD. Disease resolution following the treatment of peri-implant diseases: A systematic review. Int J Periodontics Restorative Dent 2025;45:115–133.
16. Barootchi S, Monje A, Sabri H, Rosen PS, Wang HL. Surgical reconstructive therapy for the management of peri-implantitis: An AAP/AO systematic review and network meta-analysis. Int J Oral Maxillofac Implants 2025;40(suppl):s1–s48.
17. Mameno T, Wada M, Onodera Y, Fujita D, Sato H, Ikebe K. Longitudinal study on risk indicators for peri-implantitis using survival-time analysis. J Prosthodont Res 2019;63:216–220.
18. Saleh MHA, Misch C, Alrmali A, Neiva R. Efficacy of nonreconstructive surgical treatment of peri-implantitis: An AAP/AO systematic review and meta-analysis of access flap versus osseous surgery procedures. Int J Oral Maxillofac Implants 2025;40(suppl):s73–s90.
19. Ravidà A, Dias DR, Lemke R, Rosen PS, Bertolini MM. Efficacy of decontamination methods for biofilm removal from dental implant surfaces and reosseointegration: An AAP/AO systematic review on peri-implant diseases and conditions. Int J Oral Maxillofac Implants 2025;40(suppl):s91–s160.
20. Schwarz F, Sahm N, Bieling K, Becker J. Surgical regenerative treatment of peri-implantitis lesions using a nanocrystalline hydroxyapatite or a natural bone mineral in combination with a collagen membrane: A four-year clinical follow-up report. J Clin Periodontol 2009;36:807–814.
21. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. J Clin Periodontol 2018;45(suppl 20):s246–s266.
22. Duarte PM, Nogueira CFP, Silva SM, Pannuti CM, Schey KC, Miranda TS. Impact of smoking cessation on periodontal tissues. Int Dent J 2022;72:31–36.
23. Heitz-Mayfield LJA, Salvi GE, Mombelli A, et al. Supportive peri-implant therapy following anti-infective surgical peri-implantitis treatment: 5-year survival and success. Clin Oral Implants Res 2018;29:1–6.
24. Lin GH, Lee E, Barootchi S, et al. The influence of prosthetic designs on peri-implant bone loss: An AO/AAP systematic review and meta-analysis. J Periodontol 2025;96. doi: 10.1002/jper.24-0144.
25. Cha J, Paeng K, Jung U, Choi S, Sanz M, Sanz-Martín I. The effect of five mechanical instrumentation protocols on implant surface topography and roughness: A scanning electron microscope and confocal laser scanning microscope analysis. Clin Oral Implants Res 2019;30:578–587.
26. Al-Hashedi AA, Laurenti M, Benhamou V, Tamimi F. Decontamination of titanium implants using physical methods. Clin Oral Implants Res 2017;28:1013–1021.
27. Renvert S, Aghazadeh A, Hallström H, Persson GR. Factors related to peri-implantitis—A retrospective study. Clin Oral Implants Res 2014;25:522–529.
28. Karlsson K, Trullenque-Eriksson A, Tomasi C, Derks J. Efficacy of access flap and pocket elimination procedures in the management of peri-implantitis: A systematic review and meta-analysis. J Clin Periodontol 2023;50(suppl 26):244–284.
29. Donos N, Calciolari E, Ghuman M, Baccini M, Sousa V, Nibali L. The efficacy of bone reconstructive therapies in the management of peri-implantitis. A systematic review and meta-analysis. J Clin Periodontol 2023;50(suppl 26):285–316.
30. Li H, Wang Y, Zhang D, Chen T, Hu A, Han X. Glycemic fluctuation exacerbates inflammation and bone loss and alters microbiota profile around implants in diabetic mice with experimental peri-implantitis. Int J Implant Dent 2021;7:79.
31. Lin Y, Bai J, Chen C, Zhou Y, Guan X. Application of information-motivation-behavioral model–based continuity of care on the peri-implantitis recovery in diabetic implant overdenture patients: A randomised controlled trial. Research Square, 2023.
32. Vohra F, Alkhudhairy F, Al-Kheraif AA, Akram Z, Javed F. Peri-implant parameters and C-reactive protein levels among patients with different obesity levels. Clin Implant Dent Relat Res 2018;20:130–136.
33. Datte CE, Rodrigues VA, Datte FB, da Rocha Scalzer Lopes G, Borges ALS, Nishioka RS. The effect of different bone level and prosthetic connection on the biomechanical response of unitary implants: Strain gauge and finite element analyses. Int J Adv Eng Res Sci 2021;8:218–224.
34. Luo Q, Ding Q, Zhang L, Xie Q. Analyzing the occlusion variation of single posterior implant–supported fixed prostheses by using the T-scan system: A prospective 3-year follow-up study. J Prosthet Dent 2020;123:79–84.
35. Mombelli A, Müller N, Cionca N. The epidemiology of peri-implantitis. Clin Oral Implants Res 2012;23(suppl 6):67–76.

36. Rathe F, Junker R, Blumenröhr J, et al. The impact of implant abutment angle and height on peri-implant tissue health: Retrospective analyses from a randomized controlled clinical trial. Int J Prosthodont 2024;37:16–26.
37. Târtea DA, Ionescu M, Manolea HO, et al. Comparative study of dental custom CAD-CAM implant abutments and dental implant stock abutments. J Clin Med 2023;12:2128.
38. Apicella D, Veltri M, Chieffi N, Polimeni A, Giovannetti A, Ferrari M. Implant adaptation of stock abutments versus CAD/CAM abutments: A radiographic and scanning electron microscopy study. Ann Stomatol (Roma) 2010;1:9–13.
39. Gallo S, Pascadopoli M, Pellegrini M, et al. CAD/CAM abutments versus stock abutments: An update review. Prosthesis 2022;4:468–479.
40. Bassetti M, Schär D, Wicki B, et al. Anti-infective therapy of peri-implantitis with adjunctive local drug delivery or photodynamic therapy: 12-month outcomes of a randomized controlled clinical trial. Clin Oral Implants Res 2014;25:279–287.
41. Suárez-López Del Amo F, Yu SH, Wang HL. Non-surgical therapy for peri-implant diseases: A systematic review. J Oral Maxillofac Res 2016;7:e13.
42. Toledano-Osorio M, Vallecillo C, Toledano R, et al. A systematic review and meta-analysis of systemic antibiotic therapy in the treatment of peri-implantitis. Int J Environ Res Public Health 2022;19:6502.
43. Fragkioudakis I, Kallis A, Kesidou E, Damianidou O, Sakellari D, Vouros I. Surgical Treatment of peri-implantitis using a combined Nd:YAG and Er:YAG laser approach: Investigation of clinical and bone loss biomarkers. Dent J 2023;11:61.
44. Dalago HR, Perrotti V, Torres de Freitas SF, et al. Prospective longitudinal comparison study of surgical therapies for peri-implantitis: 3-year follow-up. Aust Dent J 2019;64:237–245.
45. Thierbach R, Eger T. Clinical outcome of a nonsurgical and surgical treatment protocol in different types of peri-implantitis: A case series. Quintessence Int 2013;44:137–148.
46. Ravidà A, Siqueira R, Di Gianfilippo R, et al. Prognostic factors associated with implant loss, disease progression or favorable outcomes after peri-implantitis surgical therapy. Clin Implant Dent Rel Res 2022;24:222–232.
47. Monje A, Nart J. Management and sequelae of dental implant removal. Periodontol 2000 2022;88:182–200.
48. Messina AM, Marini L, Marini E. A step-by-step technique for the piezosurgical removal of fractured implants. J Craniofac Surg 2018;29:2116–2118.
49. Smith LP, Rose T. Laser explantation of a failing endosseous dental implant. Aust Dent J 2010;55:219–222.
50. Schwarz F, Schmucker A, Becker J. Efficacy of alternative or adjunctive measures to conventional treatment of peri-implant mucositis and peri-implantitis: A systematic review and meta-analysis. Int J Implant Dent 2015;1:22.
51. Renvert S, Polyzois I. Treatment of pathologic peri-implant pockets. Periodontology 2000 2018;76:180–190.
52. Tomasi C, Regidor E, Ortiz-Vigón A, Derks J. Efficacy of reconstructive surgical therapy at peri-implantitis-related bone defects. A systematic review and meta-analysis. J Clin Periodontol 2019;46(suppl 21):340–356.

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Appendix Consensus Statements

Clinical and basic research have evolved the field of modern implant dentistry. Fundamentally, the placement of a dental implant, its restoration, and long-term care have been practiced with some degree of predictability. However, the clinician is continuously challenged with areas of knowledge that have not been well established. The participants in this conference voted on questions that focus on gaps in current knowledge. In many cases, these responses relied on expert opinions of the group when definitive evidence was not available in the literature. To clarify the level of agreement for each statement, a consensus scale was established as follows: unanimous at 100% agreement among the plenary panel; strong consensus at ≥ 95% agreement; consensus agreement at 75% to 95%; simple majority agreement at 50% to 74%; and no consensus agreement at < 50%.

1. Use of Hydrogen Peroxide Mouth Rinses as an Adjunct in Peri-implant Mucositis Treatment

Statement: Antimicrobial mouth rinses containing hydrogen peroxide fail to provide added benefit as an adjunct in the treatment of peri-implant mucositis.

Strength of Consensus: Simple majority (54% agreed, 18% disagreed, 28% neutral)

Rationale: Hydrogen peroxide has antimicrobial properties and can reduce bacterial load through the release of oxygen radicals. However, its efficacy, specifically for peri-implant mucositis is not well-established. Potential drawbacks include mucosal irritation and alterations in the normal oral microbiome. The high percentage of neutrality and disagreement reflects uncertainty and the need for more robust clinical evidence before it can be routinely recommended.

2. Use of Iodine Mouth Rinses as an Adjunct in Peri-implant Mucositis Treatment

Statement: Antimicrobial mouth rinses containing iodine fail to provide added benefit as an adjunct in the treatment of peri-implant mucositis.

Strength of Consensus: Simple majority (65% agreed, 11% disagreed, 24% neutral)

Rationale: Iodine has antiseptic properties that are effective against a wide range of microorganisms. However, concerns such as allergic reactions, staining, and taste alterations limit its acceptance. The significant agreement with this statement indicates that, without strong clinical evidence supporting its benefits in peri-implant mucositis, iodine mouth rinses are not widely endorsed.

3. Use of Chlorhexidine for Subgingival Irrigation in Peri-implant Mucositis

Statement: Subgingival irrigation with chlorhexidine is recommended as an adjunct to nonsurgical mechanical debridement for treating peri-implant mucositis.

Strength of Consensus: No consensus (48% agreed, 36% disagreed, 16% neutral)

Rationale: Chlorhexidine is a gold-standard antiseptic in dentistry due to its broad-spectrum efficacy and substantivity. While it can reduce microbial load, its penetration into subgingival areas is limited. Side effects such as tooth staining and taste alteration also impact patient compliance. The mixed responses suggest that while some clinicians find it beneficial, others are hesitant due to these limitations.

4. Use of Hydrogen Peroxide for Subgingival Irrigation in Peri-implant Mucositis

Statement: Subgingival irrigation with hydrogen peroxide fails to provide added benefit as an adjunct to nonsurgical mechanical debridement for treating peri-implant mucositis.

Strength of Consensus: Simple majority (53% agreed, 17% disagreed, 30% neutral)

Rationale: Subgingival irrigation with hydrogen peroxide may offer antimicrobial benefits, but its efficacy is questionable due to rapid decomposition and limited tissue penetration. Additionally,

overuse may harm the surrounding tissues. The high percentages of neutrality and disagreement indicate that clinicians are cautious and require more evidence to support its routine use.

5. Use of Iodine for Subgingival Irrigation in Peri-implant Mucositis

Statement: Subgingival irrigation with iodine fails to provide added benefit as an adjunct to nonsurgical mechanical debridement.
Strength of Consensus: Consensus (76% agreed, 27% disagreed, 10% neutral)
Rationale: The substantial agreement reflects skepticism due to limited clinical data supporting iodine's effectiveness in subgingival irrigation for peri-implant mucositis. Potential side effects and patient acceptance are also considerations that may influence clinicians' reluctance to recommend iodine in this context.

6. Use of Local Delivery Antibiotics in Peri-implant Mucositis Treatment

Statement: Clinical evidence fails to support the adjunctive use of local-delivery antibiotics to treat peri-implant mucositis.
Strength of Consensus: Simple majority (63% agreed, 27% disagreed, 10% neutral)
Rationale: Local antibiotics can target specific pathogens, but their use in peri-implant mucositis is debated due to concerns about antibiotic resistance, cost, and inconsistent clinical outcomes. The high percentage of agreement suggests that many clinicians prefer mechanical debridement and improved oral hygiene practices over antibiotic use.

7. Use of Systemic Antibiotics in Peri-implant Mucositis Treatment

Statement: Clinical evidence fails to support the adjunctive use of systemic antibiotics to treat peri-implant mucositis.
Strength of Consensus: Consensus (80% agreed, 6% disagreed, 16% neutral)
Rationale: Systemic antibiotics are generally reserved for severe infections with systemic involvement. Peri-implant mucositis is a localized condition, and systemic antibiotic use may not be justified due to potential side effects and contribution to antibiotic resistance. The predominant agreement aligns with current guidelines that emphasize mechanical debridement and local therapies.

8. Use of Probiotics as Adjunctive Treatment for Peri-implant Mucositis

Statement: Clinical evidence supports the use of probiotics as an adjunctive treatment for peri-implant mucositis.
Strength of Consensus: Simple majority (53% agreed, 28% disagreed, 19% neutral)
Rationale: Probiotics may help modulate the oral microbiome, but evidence supporting their efficacy in peri-implant mucositis is limited and inconsistent. The diverse responses indicate that while some clinicians are open to their potential benefits, the lack of strong evidence leads to general skepticism.

9. Use of Lasers in Nonsurgical Therapy for Peri-implant Mucositis

Statement: Lasers should be used as adjuncts in nonsurgical therapy to manage peri-implant mucositis.
Strength of Consensus: No consensus (25% agreed, 45% disagreed, 30% neutral)
Rationale: Laser therapy offers potential benefits, such as bacterial reduction and enhanced healing. However, the evidence is mixed regarding its effectiveness over traditional therapies. High costs and the need for specialized training may also limit its use. The varied responses reflect the ongoing debate and the necessity for more conclusive studies.

10. Use of Lasers in Medically Compromised Patients

Statement: Lasers should be used as adjuncts in treating medically compromised groups where other means of nonsurgical therapy may not be possible.
Strength of Consensus: No consensus (39% agreed, 44% disagreed, 17% neutral)
Rationale: In medically compromised patients, lasers might provide a less-invasive alternative. However, concerns about safety, efficacy, and cost persist. The mixed responses suggest that while some clinicians see potential in specific cases, others remain cautious due to insufficient evidence.

11. Increased Frequency of Supportive Peri-Implant Therapy (SPiT) Postintervention

Statement: SPiT intervals should be more frequent for patients who have undergone interventions for peri-implant mucositis.

Strength of Consensus: Consensus (91% agreed, 6% disagreed, 3% neutral)

Rationale: Following intervention, increased monitoring is essential to prevent recurrence and ensure optimal healing. Frequent SPiT allows for professional cleaning, reinforcement of oral hygiene practices, and early detection of complications. The strong consensus underscores the importance of proactive maintenance in peri-implant care.

12. 3- to 4-Month Intervals for SPiT in Active Surveillance

Statement: 3- to 4-month intervals should be utilized for SPiT in patients with peri-implant mucositis who are under active surveillance.

Strength of Consensus: Consensus (88% agreed, 3% disagreed, 9% neutral)

Rationale: Regular 3- to 4-month intervals align with periodontal maintenance recommendations, allowing for effective biofilm disruption and monitoring. This frequency is especially important for patients with a history of peri-implant mucositis, as they are at higher risk for disease progression.

13. 5- to 6-Month Intervals for SPiT in Less Active Surveillance

Statement: 5- to 6-month SPiT intervals should be utilized for patients with peri-implant mucositis who require less active surveillance.

Strength of Consensus: Consensus (88% agreed, 9% disagreed, 3% neutral)

Rationale: For patients demonstrating stability and good oral hygiene, extending SPiT intervals to 5 or 6 months may be appropriate. However, careful assessment of individual risk factors is necessary to avoid missing early signs of disease recurrence.

14. Biofilm as the Etiologic Factor in Peri-implant Disease

Statement: Biofilm is the etiologic factor in peri-implant disease.

Strength of Consensus: Consensus (91% agreed, 3% disagreed, 6% neutral)

Rationale: The accumulation of microbial biofilm is recognized as the primary cause of peri-implant mucositis and peri-implantitis. It triggers an inflammatory response in peri-implant tissues. The overwhelming agreement reflects the well-established role of biofilm in peri-implant disease etiology.

15. The Role of Titanium Biomaterial Degradation in Peri-implant Disease

Statement: Titanium biomaterial degradation may play a role in peri-implant disease initiation and/or progression?

Strength of Consensus: Simple majority (61% agreed, 28% disagreed, 11% neutral)

Rationale: Titanium particles released due to corrosion or wear may induce local inflammation, potentially contributing to peri-implant disease. While some studies suggest a link, the clinical evidence is not definitive. The divided opinions highlight the need for further research to clarify the role of titanium corrosion.

16. No Standard of Practice for Implant Surface Decontamination

Statement: Does evidence support that there is no standard of practice treatment for surface decontamination of dental implants with peri-implantitis?

Strength of Consensus: Consensus (85% agreed, 9% disagreed, 3% neutral)

Rationale: Currently, there is no universally accepted method for implant surface decontamination. Various mechanical, chemical, and laser techniques are used with varying degrees of success. The consensus reflects the lack of standardized protocols and the need for more research to establish evidence-based guidelines.

17. Preference for Reconstructive vs Nonreconstructive Therapy

Statement: Reconstructive therapy is generally preferable to nonreconstructive therapy when possible.

Strength of Consensus: Consensus (79% agreed, 0% disagreed, 21% neutral)

Rationale: Reconstructive therapy aims to regenerate lost bone and soft tissue, potentially restoring implant function and esthetics. When feasible, it may offer better long-term outcomes compared to nonreconstructive approaches, which focus on disease control without tissue regeneration.

18. Performing Reconstructive Therapy Based on Lesion Morphology

Statement: Clinicians should perform reconstructive therapy wherever the lesion morphology favors this (eg, multiple bony walls for containment, circumferential infraosseous defects).
Strength of Consensus: Strong consensus (97% agreed, 0% disagreed, 3% neutral)
Rationale: Lesion morphology significantly influences the potential success of regenerative procedures. Defects with favorable configurations (eg, deep, narrow, and with multiple walls) are more conducive to regeneration. The strong agreement indicates that clinicians recognize the importance of assessing lesion characteristics when planning treatment.

19. Submerging Implants During Reconstructive Therapy

Statement: Submerging the implant should be pursued for reconstructive therapies when possible.
Strength of Consensus: Consensus (82% agreed, 9% disagreed, 9% neutral)
Rationale: Submerging implants during reconstructive procedures can protect the surgical site from microbial contamination and mechanical disruption, potentially enhancing healing and regeneration. However, this approach may not be practical in all cases due to prosthetic and functional considerations.

20. Use of Membranes Over Bone Replacement Grafts

Statement: Should the use of a membrane over the bone replacement graft be performed?
Strength of Consensus: Consensus (79% agreed, 9% disagreed, 13% neutral)
Rationale: Barrier membranes are used in guided bone regeneration to prevent soft tissue invasion into the graft site, promote bone growth, and provide graft containment. The general agreement suggests that many clinicians consider membranes beneficial, though factors like cost and surgical complexity may influence their use.

21. Preference for Open-Flap Nonreconstructive Therapy When Reconstructive Therapy Is Not Possible

Statement: Open-flap nonreconstructive therapy is the preferable treatment in cases of peri-implantitis where reconstructive therapy is not possible.
Strength of Consensus: Consensus (93% agreed, 0% disagreed, 7% neutral)
Rationale: Open-flap nonreconstructive therapy involves surgically accessing the implant surface to debride and decontaminate the area without attempting to regenerate lost bone. It is considered preferable when reconstructive therapy is contraindicated due to factors like unfavorable defect morphology or patient-specific considerations.

22. Effectiveness of Implantoplasty in Managing Supracrestal or Subcrestal Defects

Statement: Implantoplasty is an effective adjunct in managing supracrestal or subcrestal defects.
Strength of Consensus: Simple majority (53% agreed, 25% disagreed, 22% neutral)
Rationale: Implantoplasty involves smoothing the exposed implant threads to reduce plaque accumulation and facilitate soft tissue healing. While some studies support its effectiveness, concerns exist regarding the potential weakening of the implant and the release of titanium particles. The mixed responses reflect varying clinical experiences and the need for more robust evidence to support its routine use.

23. Timing of Soft Tissue Modification in Peri-implantitis Management

Statement: Soft tissue modification should be completed simultaneously with or following peri-implantitis management, depending on the case scenario.
Strength of Consensus: Consensus (94% agreed, 3% disagreed, 3% neutral)
Rationale: Soft tissue modification can enhance peri-implant health by improving mucosal thickness and keratinized tissue width. Timing may

vary based on individual patient needs, defect morphology, and the extent of peri-implantitis. The strong agreement suggests that clinicians value flexibility in integrating soft tissue procedures to optimize treatment outcomes.

24. Indications for Soft Tissue Modification Differ Between Reconstructive and Nonreconstructive Surgery

Statement: Indications for soft tissue modification differ between reconstructive and nonreconstructive peri-implantitis surgery.
Strength of Consensus: Simple majority (65% agreed, 16% disagreed, 19% neutral)
Rationale: The need for soft tissue modification may vary depending on the surgical approach. In reconstructive surgery, soft tissue management aims to support bone regeneration, whereas in nonreconstructive surgery, it may focus on reducing pocket depths and improving hygiene access. The varied responses highlight that clinicians consider case-specific factors when deciding on soft tissue interventions.

25. Consideration of Free Gingival Grafting to Increase Keratinized Mucosa

Statement: The primary therapeutic purpose of free gingival grafting is to increase the band of keratinized mucosa.
Strength of Consensus: Consensus (94% agreed, 0% disagreed, 6% neutral)
Rationale: Free gingival grafts effectively increase the width of keratinized mucosa, enhancing peri-implant tissue health and patient comfort during oral hygiene practices. The overwhelming agreement reflects clinical confidence in this procedure as a reliable method for augmenting keratinized tissue.

26. Use of Soft Tissue Substitutes as an Alternative to Free Gingival Grafting

Statement: The primary therapeutic purpose of soft tissue substitutes as an alternative is to increase the band of keratinized mucosa.
Strength of Consensus: Simple majority (67% agreed, 16% disagreed, 17% neutral)
Rationale: Soft tissue substitutes offer a less-invasive option by eliminating the need for a donor site. However, their effectiveness compared to autogenous grafts may vary. The mixed responses indicate cautious consideration, with some clinicians adopting these alternatives while others prefer traditional methods.

27. Consideration of Autogenous Soft Tissue for Tissue Volume Augmentation

Statement: The primary therapeutic purpose of autogenous soft tissue is to augment the tissue volume.
Strength of Consensus: Strong consensus (97% agreed, 0% disagreed, 3% neutral)
Rationale: Autogenous soft tissue grafts, such as connective tissue grafts, are the gold standard for volume augmentation due to their biocompatibility and predictable outcomes. The strong agreement underscores its preferred status among clinicians for enhancing peri-implant soft tissue volume.

28. Use of Soft Tissue Substitutes as an Alternative for Tissue Volume Augmentation

Statement: The primary therapeutic purpose of soft tissue substitutes is to augment the tissue volume.
Strength of Consensus: Consensus (93% agreed, 7% disagreed, 0% neutral)
Rationale: The majority agree that soft tissue substitutes can be considered for volume augmentation. Advances in biomaterials have improved their effectiveness, though they may not always match the results of autogenous grafts. Clinicians may opt for substitutes in cases where patient factors preclude autogenous grafting.

29. Consideration of Autogenous Soft Tissue for Vestibular Deepening

Statement: The primary therapeutic purpose of autogenous soft tissue is to deepen the vestibule.
Strength of Consensus: Consensus (92% agreed, 4% disagreed, 4% neutral)
Rationale: Deepening the vestibule can improve prosthesis stability and facilitate oral hygiene. Autogenous grafts are effective for this purpose due to their integration and durability. The agreement indicates clinician preference for autogenous tissue in vestibular deepening procedures.

30. Use of Soft Tissue Substitutes as an Alternative for Vestibular Deepening

Statement: The primary therapeutic purpose of soft tissue substitutes is to deepen the vestibule.
Strength of Consensus: Simple majority (66% agreed, 21% disagreed, 13% neutral)
Rationale: Soft tissue substitutes may be used for vestibular deepening, but their efficacy compared to autogenous grafts is variable. The diverse responses suggest that while some clinicians are adopting these materials, others remain skeptical due to concerns about long-term outcomes.

31. Autogenous Connective Tissue as Standard of Practice for All Soft Tissue Grafting Around Implants

Statement: Autogenous connective tissue modification is the standard of practice for all soft tissue grafting procedures around dental implants.
Strength of Consensus: Consensus (80% agreed, 13% disagreed, 7% neutral)
Rationale: Autogenous connective tissue grafts are widely regarded as the gold standard due to their proven success. However, not all cases may necessitate or allow for autogenous tissue use. The majority agreement indicates that while autogenous tissue is preferred, alternatives may be acceptable in certain situations.

32. Use of Connective Tissue Grafting in the Esthetic Zone During Peri-implantitis Treatment

Statement: Connective tissue grafting should be considered when treating peri-implantitis in the esthetic zone.
Strength of Consensus: Consensus (83% agreed, 10% disagreed, 7% neutral)
Rationale: In the esthetic zone, achieving optimal soft tissue contours is crucial. Connective tissue grafting can improve tissue thickness and support, enhancing both functional and esthetic outcomes. The strong agreement reflects the clinical value placed on soft tissue augmentation in these cases.

33. Recommendation of a 3-Month Interval for SPiT After Peri-implantitis Treatment

Statement: 3-month interval for SPiT is recommended in patients who have undergone treatment for peri-implantitis.
Strength of Consensus: Consensus (94% agreed, 6% disagreed, 0% neutral)
Rationale: A 3-month maintenance interval allows for close monitoring and management, reducing the risk of disease recurrence. This frequency aligns with periodontal maintenance recommendations for patients with a history of periodontal disease or peri-implantitis.

34. Reassessment of SPiT Interval Following the First Year of Treatment

Statement: The interval for SPiT should be reassessed following the first year of treatment.
Strength of Consensus: Consensus (89% agreed, 7% disagreed, 4% neutral)
Rationale: Reassessing the SPiT interval allows for adjustments based on the patient's response to treatment and adherence to oral hygiene. This individualized approach helps maintain peri-implant health over the long term.

35. A Minimum 5- to 6-Month SPiT Interval is Essential for Maintaining Treatment Stability

Statement: A minimum 5- to 6-month interval for SPiT is essential for maintaining peri-implantitis treatment stability.
Strength of Consensus: Consensus (94% agreed, 6% disagreed, 0% neutral)
Rationale: While some patients may require more frequent visits, a 5- to 6-month interval may suffice for maintaining stability in patients with good compliance and low-risk factors. The agreement indicates that clinicians consider this interval acceptable in appropriate cases.

36. Tailoring SPiT to the Specific Patient Risk Profile

Statement: All SPiT should be tailored to the specific patient risk profile to maintain peri-implant health.
Strength of Consensus: Consensus (93% agreed, 7% disagreed, 0% neutral)
Rationale: Individualizing SPiT intervals based on patient risk factors such as systemic health, smoking status, and oral hygiene practices enhances the effectiveness of maintenance programs. The overwhelming agreement reflects the importance

of personalized care in preventing peri-implant disease.

37. Importance of Effective Patient Oral Home Care in SPiT

Statement: Regular and effective patient-delivered biofilm control through oral home care is a critical component of ongoing SPiT for prevention of peri-implant disease and maintenance of implant health.

Strength of Consensus: Consensus (93% agreed, 3% disagreed, 4% neutral)

Rationale: Effective oral hygiene is paramount in controlling biofilm accumulation, the primary cause of peri-implant disease. Patient education and compliance are critical components of successful SPiT programs. The strong agreement underscores the necessity of patient involvement in maintaining implant health.

38. Peri-implant Mucositis as a Predisposition for Peri-implantitis

Statement: Peri-implant mucositis is a predisposition for the development of peri-implantitis.

Strength of Consensus: Strong consensus (97% agreed, 0% disagreed, 3% neutral)

Rationale: Peri-implant mucositis is an inflammatory lesion confined to the mucosa surrounding an implant. If left untreated, it can progress to peri-implantitis, which involves bone loss. The strong agreement reflects the understanding that early intervention is critical to prevent disease progression.

39. The Role of Soft Tissue Phenotype in Risk for Peri-implant Mucositis

Statement: Soft tissue phenotype plays a role in the risk for peri-implant mucositis.

Strength of Consensus: Consensus (94% agreed, 0% disagreed, 6% neutral)

Rationale: Soft tissue phenotype, including mucosal thickness and keratinized tissue width, can influence the patient's susceptibility to inflammation and the ease of maintaining oral hygiene. Thinner mucosa may be more prone to inflammation due to less resistance to bacterial insult. The agreement indicates a recognition of the importance of soft tissue characteristics in implant success.