Understanding, identifying and treating peri-implantitis

Abstract

Peri-implantitis, is a destructive inflammatory process affecting the soft and hard tissues surrounding dental implants. This is a growing concern in implant dentistry with increasing reported incidences related to better understanding of the inflammatory process and identification clinically. Incidence is influenced by factors such as the patient's systemic health, oral hygiene practices, and implant surface characteristics. Early identification of peri-implantitis, through regular clinical and radiographic monitoring correlates with improved treatment outcomes, including stabilization of peri-implant bone levels and reduction in inflammation. Delayed detection often leads to significant bone loss, compromised implant stability, and more complex, less predictable therapeutic interventions and potential for loss of the implant. This article will review the causes of peri-implantitis, how to identify it clinically and how to treat it when identified to improve the long-term prognosis of dental implants.

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INTRODUCTION

Gingival and osseous issues are recognized as an issue associated with implants that may lead to their failure when not identified early enough or treated. Inflammatory conditions affecting the soft and hard tissues adjacent to the implant involve progressive loss of the bone in contact with the implant and is primarily caused by a combination of microbial infection, host immune response, and several risk factors. The primary causative factor is colonization of the implant's surface by bacteria associated with oral biofilm normally found in the gingival sulcus and oral environment. Those bacteria induce an inflammatory response in the tissues in contact with the implant's surface. The most common bacterial pathogens involved include Porphyromonas gingivalis, Fusobacterium nucleatum, Staphylococcus epidermidis and Streptococcus (1-3). The host's immune response also plays a crucial role in determining the extent of the peri-implant tissue inflammation and subsequent hard tissue loss. Some patients may demonstrate an exaggerated immune response leading to tissue destruction and bone loss around the implant (4).

Peri-implantitis is an immune-mediated host response that is associated with the presence of peri-implant biofilm (5, 6). Studies Heyman had published described the immune mechanisms and microbiome around dental implants in peri-implantitis. They demonstrated that dental implants not only dysregulate local immunity but elicit broader effects on remote sites via microbial dysbiosis and alteration of the systemic immune response. This demonstrated that oral microbial dysbiosis facilitates bone loss around implants dysregulating the more delicate immune homeostasis that exists following implant placement (7, 8). Additionally, it has been reported that a higher accumulation of leukocytes and an expression of proinflammatory cytokines are observed around implants as compared with teeth (9). This was supported by another study that reported an aggressive immune response, with quick uncontrolled tissue destruction, and bone loss around implants (10). Studies have reported varying prevalence rates of peri-implantitis. Systematic reviews estimated the prevalence of peri-implantitis at 18.5% at the patient level and 12.8% at the implant level (11, 12). A review reported that the prevalence of peri-implantitis ranged from 1% to 47%, with an estimated weighted mean of 22% (13). Another study with a follow-up period of 17 to 23 years found that 15.0% of implants were diagnosed with peri-implantitis (14).

Risk factors that contribute to the incidence and severity of peri-implantitis include the patient's oral hygiene and homecare as well as systemic conditions. Systemic diseases such as arterial hypertension, diabetes mellitus, osteoporosis, and cardiovascular diseases may show a significant influence on the incidence of peri-implantitis. Management of those risk factors, include improving homecare, treating periodontal issues, and addressing systemic health issues (ie. diabetes), can aid in decreasing the incidence of peri-implantitis and its severity (15).

Inadequate plaque control due to poor homecare around implants can lead to an accumulation of sulcular biofilm, a direct risk factor for the development of inflammation around both teeth and implants. Regular homecare maintenance is essential to prevent soft and hard tissue inflammation and is in the control of the patient via appropriate daily homecare (16). Systemic conditions such as diabetes and immunocompromised states are known risk factors for peri-implantitis (17). Oral bacterial dysbiosis and foreign body stimulation are factors contributing to such dysregulation, which impair host immune cell function and trigger an inflammatory response. These conditions can impair the body's ability to fight infections, contributing to the progression of peri-implantitis (18). Additionally, habits such as smoking can compromise implant health. Smoking is a significant risk factor for peri-implant diseases and hampers the clinical outcomes of peri-implant therapies (19). Smokers have a high incidence of peri-implantitis (72.7%) compared to non-smokers (27.3%), with higher richness of microbiota identified in patients with peri-implantitis who are smokers (20, 21). Thus, peri-implantitis is a multifactorial disease, where bacteria play a central role, but is exacerbated by host factors, oral hygiene, and other systemic or local factors.

Inflammatory changes can affect both the soft and hard tissues adjacent to the implant. Peri-mucositis and peri-implantitis are both inflammatory conditions that affect the tissues surrounding dental implants, but they differ in severity, clinical presentation, and potential outcomes.

Peri-mucositis is confined to the soft tissues and is a reversible inflammatory process primarily of the mucosa, without affecting the underlying bone. It is considered a precursor to peri-implantitis. Peri-implantitis is a more severe condition that involves both soft tissue inflammation and progressive bone loss around the implant. It is considered a pathological state and may lead to implant failure if left untreated (22). The clinical features of peri-mucositis include gingival redness, swelling, and bleeding on probing of the mucosa around the implant, but no radiographic evidence of bone loss is evident (23). Whereas, with peri-implantitis, these same clinical signs are present, but there is bone loss evident radiographically (24). As mentioned both conditions are primarily caused by bacterial biofilm accumulation around the implant, poor oral hygiene, and other risk factors such as smoking and diabetes. However, peri-implantitis' progression is due to sustained inflammation leading to bone

destruction, while peri-mucositis is often associated with less severe inflammatory responses (25). Peri-implantitis was initially recognized as an inflammatory condition associated with dental implants but did not receive widespread attention as a distinct disease until later in the 20th century (26). Early signs of peri-implant disease became more widely recognized in the 1980s. This marked the first significant efforts to differentiate between normal and pathological conditions around dental implants. During that period researchers and clinicians recognized bone loss was occurring around implants in a manner similar to periodontitis around natural teeth. Studies began to highlight the role of bacteria in the development of those inflammatory conditions. Researchers began to recognize that microbial biofilms around implants could trigger an inflammatory response leading to bone loss. The term peri-implantitis was formally introduced in the early 1990's and began to be used to describe an inflammatory process involving both the soft tissues and the surrounding bone, with clinical evidence of bone loss. The International Workshop on Periodontics and Implantology held in 1990 played a significant role in defining the terminology for peri-implant diseases and formally recognized as a distinct disease, separate from general implant complications and other forms of inflammation (27). Research into peri-implantitis became more robust in the early 2000's, with a focus on the microbial causes of the disease, risk factors, and host responses. In recent years an increased focus on prevention and treatment of peri-implantitis has become one of the most significant concerns in implant dentistry due to its high prevalence and potential for implant loss. As implants have become more widely used, the frequency of peri-implantitis has also risen, leading to a greater emphasis on prevention, early detection, and effective treatment strategies. Today, peri-implantitis is a well-known and actively studied condition in implant dentistry, with ongoing efforts to improve its prevention, diagnosis, and management. A history of periodontitis is considered a significant risk factor for peri-implantitis and potential for implant failure (28).

Inflammatory incidence associated with implants

The incidence of peri-mucositis varies in the literature, but it is generally reported to affect a significant proportion of dental implant patients. A systematic review by Zitzmann and Berglundh in 2008 found that peri-mucositis is present in approximately 50% of patients with dental implants (29). This is supported by more recent publications (30). Although it may range from 19% to 65% depending on the patient population and study methods (31). The general prevalence of peri-mucositis is estimated to be 43%–47% (32-34). Some studies have indicated that the prevalence of peri-mucositis increases with time, suggesting that long-term implant maintenance and effective oral hygiene are key factors in managing this condition. The incidence of peri-mucositis was reported at 37.7% after 5 years and 64.6% after 10 years. Whereas the incidence of peri-implantitis has been reported at 10.4% after 5 years and 19.5% after 10 years. After 10 years, the extent of peri-mucositis and peri-implantitis is reported at 52.8% and 43.8%, respectively (35-38). This underscores the importance of early detection and intervention in preventing the progression of peri-mucositis to more advanced peri-implant diseases and patient homecare being a critical factor to long-term survival.

Identification of peri-implant disease

Increasing focus on peri-implantitis has occurred in recent years, due to an escalation in the reported incidence. This does not relate to a growing case numbers based on worldwide acceptance of dental implant over decades, but to more frequent identification of this pathological process as practitioners come to understand the signs and symptoms associated with the disease (39). Yet, clinical conditions leading to the conversion from gingival inflammation to peri-implantitis are not completely understood. Clinically at the histologic level, sites that have peri-implantitis often have larger inflammatory lesions than sites around natural teeth that have periodontitis. That evidence suggests that progressive crestal bone loss around implants in the absence of clinical signs of soft-tissue inflammation is rare (40). Implants, as with natural teeth require regular homecare to maintain the soft tissue, which acts as a barrier to deeper progression of bacteria and their inflammatory factors. Dental implants need to be routinely monitored as part of a comprehensive periodontal evaluation. As peri-implant diseases affect a significant number of patients who have implants, it is necessary to understand diagnosis of these diseases and the risk factors that can be modified to reduce the potential for disease occurrence or progression. Another significant variable, patients with periodontitis demonstrate a 50% chance of having peri-implantitis (41). Those who have oral inflammatory changes associated with their remaining natural teeth are at a much greater risk of acquiring peri-implantitis. Additionally, oral biofilm in periodontally involved patients may make them more immunologically sensitive to further insult from particles that are present around or on the implant surface. Recently it has been reported that patients who report an allergy to penicillin have double the failure rate of dental implants, and it may also be related to receiving alternative antibiotic therapy (42). As implants do not have periodontal ligaments, patients may not be aware of any pain during function on the implant until the implant is failing. They may report gingival bleeding around the implant when

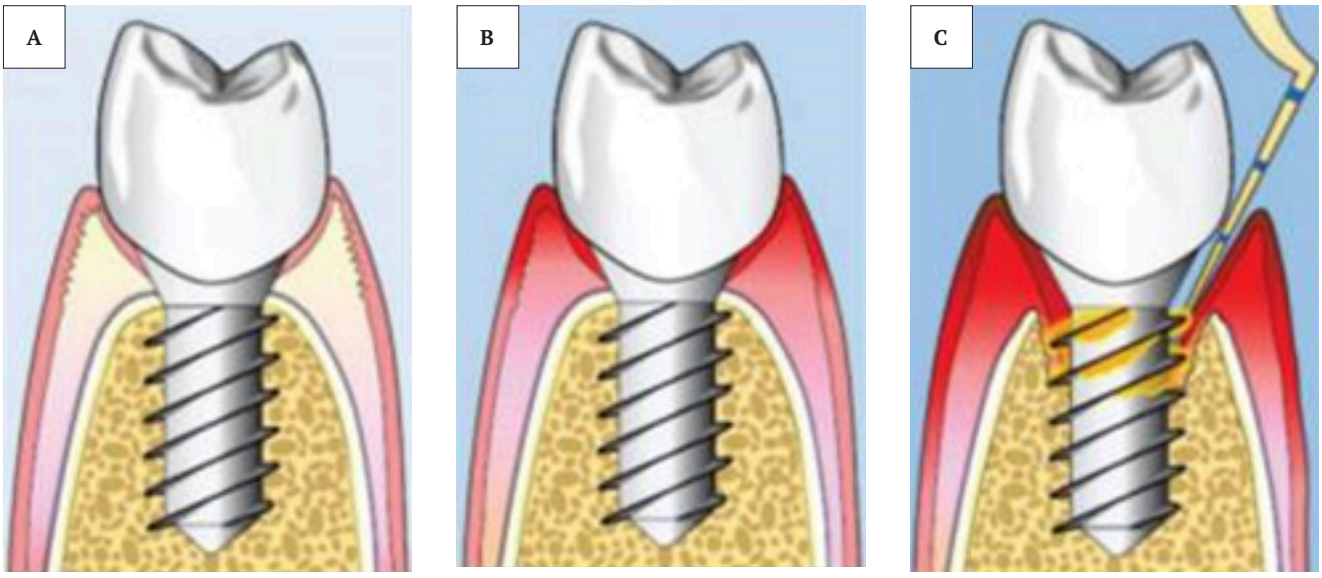

Fig. 1A **Fig. 1B** **Fig. 1C**

Fig. 1 Comparison of healthy hard and soft tissue adjacent to an implant (left), peri-mucositis involving inflammation of the soft tissue only (middle) and peri-implantitis where inflammation has affected the bone adjacent to the implants surface (right).

brushing or gingival inflammation may be noted during a hygiene appointment. Natural teeth have a gingival fiber orientation perpendicular to the tooth's long axis acting as an effective barrier to bacteria in the biofilm from progressing apically. Whereas the gingival fiber orientation around implants is parallel to the implants long axis which does not provide that same bacterial restrictive factor. Routine periodontal probing of natural teeth can accurately identify periodontal disease related to that physical stop created by the perpendicular fibers at the base of the periodontal sulcus. Routine probing of implants is inaccurate related to the lack of a physical stop allowing the periodontal probe to progress through the tissue past the apical extent of the gingival sulcus until the hard barrier of the crestal bone is encountered. This will give a false sense that periodontal issues are present around the implant and may initiate periodontal issues by introducing bacteria apically into the gingival tissue (43, 44). When gingival inflammation is identified around the implant or bleeding is noted marginally, probing is indicated to aid in diagnosing if the issue is confined to the soft tissue or has an osseous involvement (Figure 1). Periapical radiographs are also indicated to evaluate the crestal bone position in relation to the implant's platform. That will allow the practitioner to determine if peri-mucositis is present or peri-implantitis has resulted. According to the world workshop, the classification of periodontal and peri-implant diseases and conditions would follow these guidelines with regard to what is clinically present (45). When no visual signs of inflammation are noted and no radiographic bone loss from initial remodeling is present, peri-implant health is normal. (Figure 1A). Should visual signs of inflammation and/or BOP with or without suppuration be present with no radiographic evidence of crestal bone loss, peri-mucositis is the diagnosis (Figure 1B). And when visual signs of inflammation are noted, BOP with or without suppuration, with increased probing depth since the previous visit, and increased radiographic bone loss from initial remodeling is noted, peri-implantitis is the appropriate diagnosis (Figure 1C). Treatment success is centered on cleaning the exposed threads of any debris including calculus, and soft tissue that is affixed to the implant's surface. Avoidance of metal instruments (hand or ultrasonic) is recommended to prevent damage to the exposed implant surface

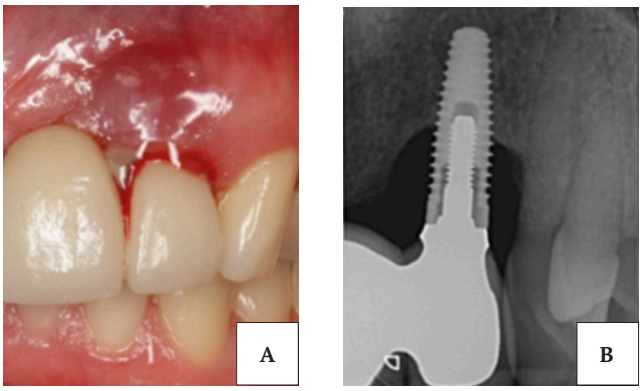

Fig. 2 Clinical appearance demonstrating gingival inflammation on the restored implant at the left maxillary lateral incisor (**Fig. 2A**) and the radiographic appearance demonstrating significant bone loss demonstrating advanced peri-implantitis (**Fig. 2B**).

and plastic instruments are utilized (Figure 4A). The surface is then treated with air abrasion utilizing glycine, erythritol, or sodium bicarbonate powder to remove any residual debris that the plastic instruments were unable to access (Figure 4B). Those suggested air abrasion powders are unable to pit or abrade the implant's surface (metal or zirconia) yet are effective in leaving a clean surface. Should an osseous graft be planned to cover the exposed implant threads, a wire brush may be utilized on those threads (Figure 4C).

Peri-implantitis treatment

Clinical manifestation of peri-implantitis includes visual signs of inflammation (redness, swelling), BOP with suppuration and radiographic evidence of bone loss > 3 mm around implant fixture/threads (Figure 2). Following identification of peri-implantitis which may be noted on routine radiographs or compared to a prior radiograph taken (Figure 3A) in the absence of gingival inflammation (Figure 3B), probing would be performed to check any bleeding on probing (BOP) (Figure 3C). Surgical exposure of the implant fixture is performed to expose any crestal dehiscence or thread exposure (Figure 3D). Treatment is focused on mechanical debridement to remove bacterial biofilm and debris from the implant surface. This can be done

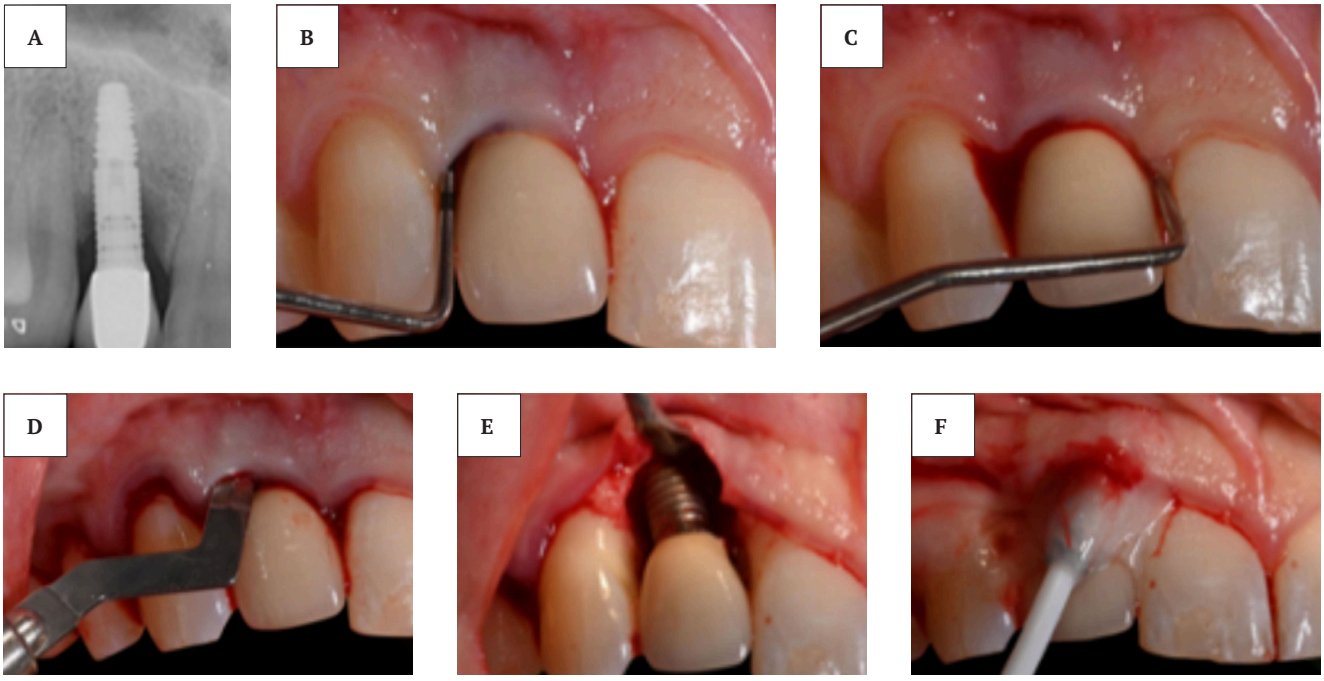

Fig. 3 Radiograph of a restored implant demonstrating crestal bone loss (**Fig. 3A**), probing with gingival bleeding at the sulcus of a restored implant (**Fig. 3B, 3C**), elevation of the soft tissue to evaluate the crestal aspect of the implant (**Fig. 3D**), crestal bone loss resulting in exposed implant threads (**Fig. 3E**) and decontamination of the implants exposed threads as part of peri-implantitis treatment (**Fig. 3F**).

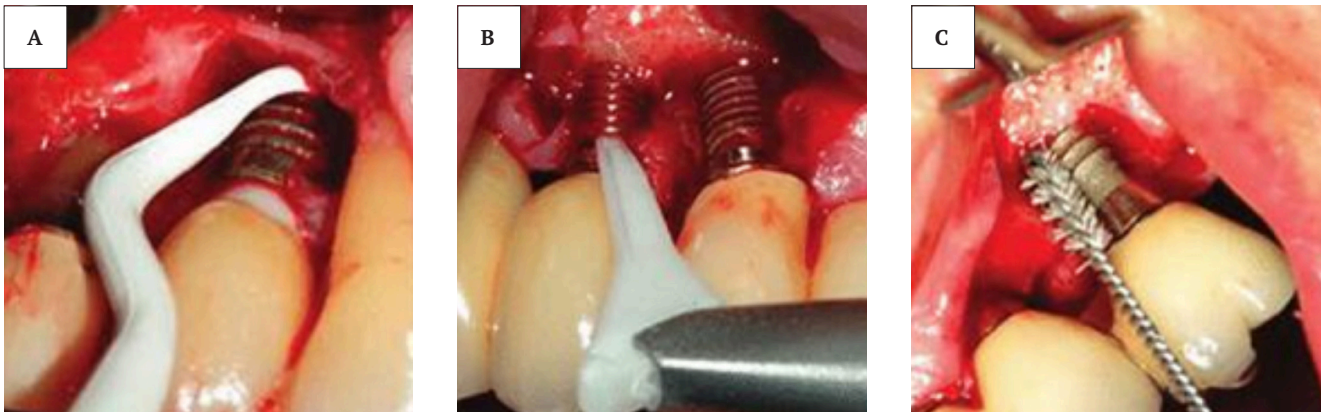

Fig. 4 Surgical intervention to treat peri-implantitis with debris removal from the exposed threads with a plastic curette (**Fig. 4A**), air abrasion to remove residual biofilm and bacteria from the exposed threads (**Fig. 4B**) and final preparation of the exposed threads with a metallic brush (**Fig. 4C**) prior to osseous graft placement.

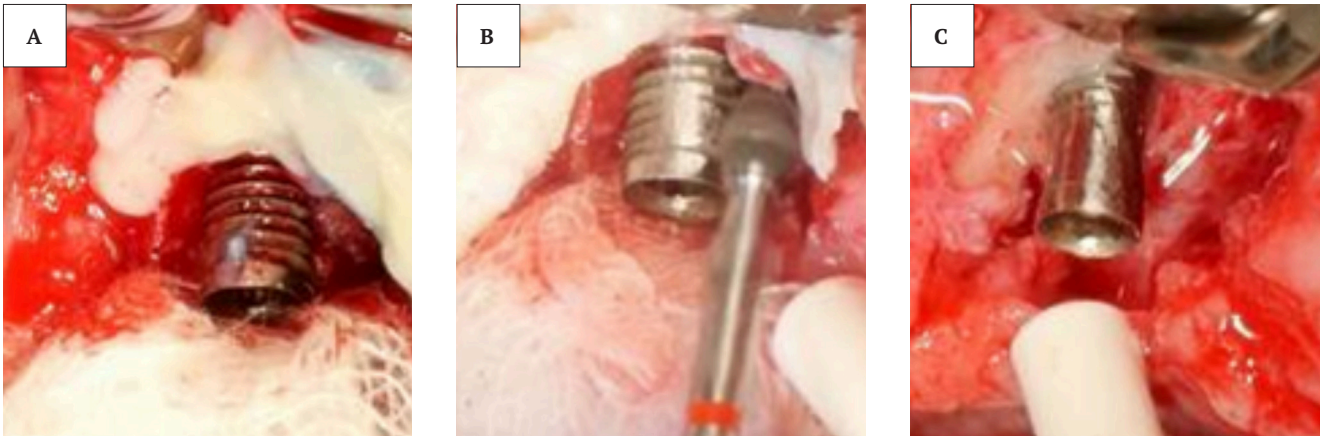

Fig. 5 Implantoplasty being performed to remove the exposed threads and provide a surface that the patient can easier maintain via homecare to remove oral biofilm and decrease further peri-implantitis occurrence.

using ultrasonic scalers, hand instruments, or specially designed brushes. The goal is to decontaminate the implant surface without causing damage to the titanium or other implant materials. An ultrasonic scaler with a plastic tip is utilized to remove any calculus and soft tissue from the exposed threads to yield a debrided surface on the exposed implant threads (Figure 3E). After mechanical debridement, antiseptic agents such as chlorhexidine, hydrogen peroxide, or antiseptic irrigation are utilized to further reduce bacterial load and inflammation. The author's preference is irrigation of the exposed threads with 0.2% chlorhexidine solution, rinsed with saline then irrigated 2% hydrogen peroxide mixed with saline in a 50:50 mixture, then again rinsed with saline to decontaminate any exposed implant surface. When available, a laser (diode, Er:YAG, Nd:YAG or CO2) is utilized to further ensure the implant surface is decontaminated of any bacteria (46). Additionally, laser irradiation has been shown to stimulate healing and host regeneration (47). After mechanical debridement with Labrida BioClean™ (Straumann, Basel, Switzerland), the implant surface is treated with a 24% EDTA gel (Straumann PrefGel®) for chemical surface decontamination (Figure 3F). At this stage there are three treatment options dictated by how much thread exposure is noted.

When minimal thread exposure is present, the gingival tissue can be approximated to the implant and ridge then secured with sutures. The patient should be instructed to avoid smoking, alcohol usage (including alcohol-containing mouth rinses) and avoid brushing the area for the 1st week. Gentle warm salt water rinses can be used starting the next day after surgery and routine homecare can be started with a toothbrush on the area after the 2nd week post-surgically.

Should the amount of thread exposure be more than minimal and contraindications are present that prevents osseous grafting, implantoplasty is indicated to improve homecare. Implantoplasty involves physical removal of the exposed threads to eliminate areas that would trap biofilm and be difficult for the patient to maintain via homecare. Should this approach be indicated, flap elevation requires exposure of the site with full visualization of the exposed threads to the crestal bone (Figure 5). A bur in a high-speed handpiece with irrigation is utilized to physically remove the exposed threads to the crestal level. The result when finished should be a smooth polished surface that is easy to keep clean by the patient.

More extensive thread exposure that has resulted related to peri-implantitis with no mobility evident on the implant, then osseous grafting is indicated as part of treatment to aid in preservation of the implant. The preliminary steps as outlined for yielding a clean surface are followed. Demineralized cortico-cancellous bone is mixed with PRF derived from the patient's blood to form sticky bone. This is placed to cover the exposed threads and the host's bone at the ridge's crestal area. Other osseous graft options may be used depending on the practitioner's preference. A resorbable membrane may be indicated depending on the graft material utilized. The soft tissue is approximated to position the flap margin coronal to the grafted area and sutures are placed. Post-surgical instructions follow those recommended previously in the article.

Case examples

A 76-year-old male patient presented for a prophylaxis and routine exam. Radiographs were taken to evaluate the implants present in the lower right posterior quadrant (Figure 6). Implants were restored at the 1st premolar and 1st molar with a three-unit bridge. Crestal bone loss was noted on the implant at the 1st molar with slight recession. Bleeding was noted on probing on the buccal and lingual of the implant. Periodontal probing was performed and a probing

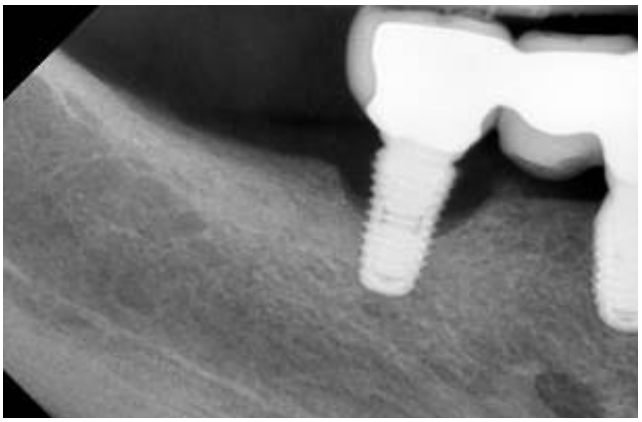

Fig. 6 Periapical radiograph demonstrating crestal bone loss related to peri-implantitis on the distal implant with no bone loss on the mesial implant (August 2023).

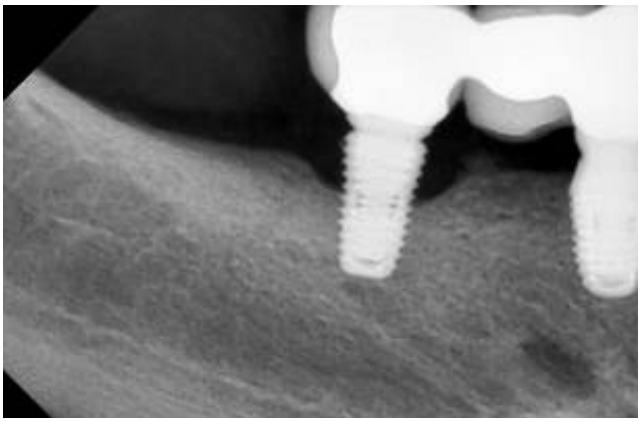

Fig. 7 Periapical radiograph at 10-month follow-up after peri-implantitis treatment demonstrating crestal bone stability on the implant (June 2024).

depth of 5 mm was noted on the buccal and lingual sides at the mesial and distal positions with 4 mm mid-tooth. Exudate was also noted at each probing site on the implant. No mobility was noted (Table 1). Treatment was recommended to the patient to address the peri-implantitis and aid in preventing its continuation and potential loss of the implant. The soft tissue was flapped, and the exposed threads were treated as previously outlined to decontaminate the implant superior to the crestal bone.

The patient was seen for regular recall prophy appointments and at 10 months post-surgery, and a periapical radiograph was taken (Figure 7). The bone level matched the initial radiograph indicating no further bone loss had occurred since treatment. Periodontal recharting was performed of the area. Probing depths had reduced and were within normal limits and no BOP was noted nor exudate in the sulcus (Table 1). Clinically the soft tissue demonstrated a lack of inflammation (Figure 8). Recharting was again performed at 13 months post treatment demonstrating further reduction in probing depths around the implant (Table 1). A periapical radiograph taken at that appointment again confirmed crestal bone stability (Figure 9) and clinically the soft tissue was stable with no inflammation noted (Figure 10).

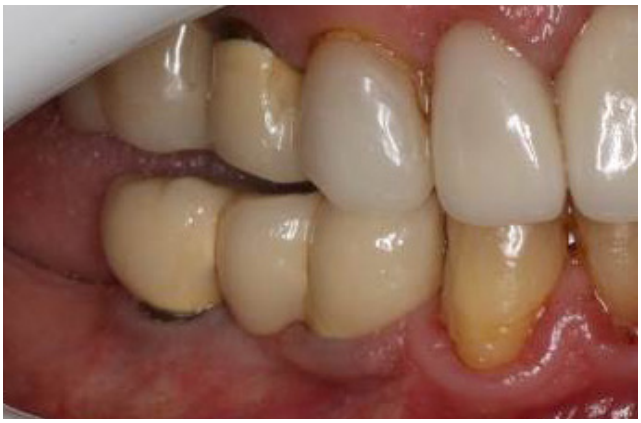

Fig. 8 Clinical appearance at 10-month recall demonstrating an absence of inflammation of the soft tissue associated with the implant that was treated previously for peri-implantitis (June 2024).

Date	Sept 13, 2023			June 15, 2024			Sept 25, 2024		
Area	Distal	Mid	Mesial	Distal	Mid	Mesial	Distal	Mid	Mesial
Buccal	5	4	5	3	2	2	1	2	2
Recession	0	1	0	0	1	0	0	1	0
BOP	*	*	*	-	-	-	-	-	-
Suppuration	*	*	*	-	-	-	-	-	-
Mobility	-	-	-	-	-	-	-	-	-
Lingual	5	4	5	1	2	2	1	2	2
Recession	0	1	0	0	1	0	0	1	0
BOP	*	*	*	-	-	-	-	-	-
Suppuration	*	*	*	-	-	-	-	-	-
Mobility	-	-	-	-	-	-	-	-	-

Tab. 1 Table of periodontal charting of the patient at initial presentation and at 10-month and 13-month follow-up appointments.

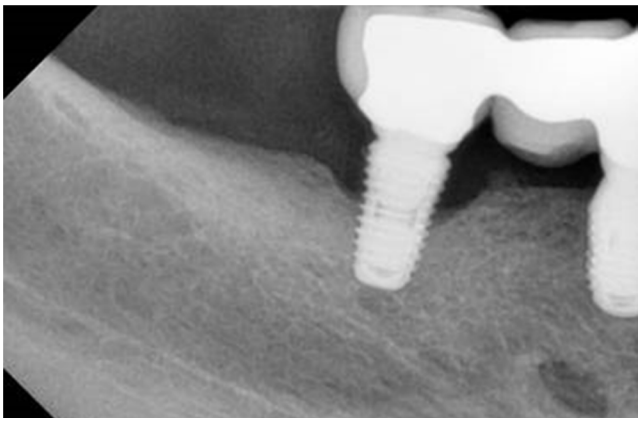

Fig. 9 Periapical radiograph at 13-month follow-up after peri-implantitis treatment demonstrating crestal bone stability on the implant (Sept 2024).

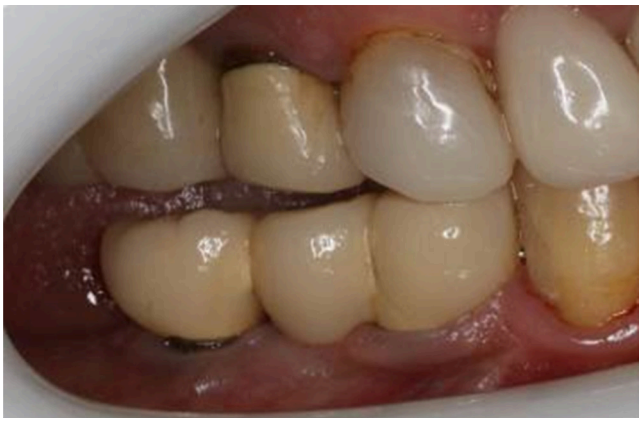

Fig. 10 Clinical appearance at 13-month recall demonstrating an absence of inflammation of the soft tissue associated with the implant that was treated previously for peri-implantitis (Sept 2024).

A 67-year-old female patient was seen on routine prophy appointment and inflammation and slight recession was noted on the posterior implants in the mandibular right quadrant that were splinted together restoratively (Figure 11). Recession was also present on the natural 1st premolar that had been present at previous recall appointments without any noted soft tissue inflammation on that tooth. A periapical radiograph was taken to evaluate the bone level in relation to the implants (Figure 12). Crestal bone loss was noted on the 2nd molar implant on all sides with slight bone loss on the distal of the 1st molar implant. The bone on the distal of the mesial implant was noted to not be clinically significant. Clinically, greater recession was noted on the distal implant lingually than was observable on the buccal (Figure 13). Periodontal charting was performed on the affected implant at the 2nd molar demonstrating significant probing depths on the buccal and lingual at all sites on the implant (Table 3). BOP was noted at all sites with exudate also present at each site. Treatment was performed following the protocol previously described.

The patient was seen at 2 months post treatment and a periapical radiograph was taken (Figure 14). Bone levels were comparable to the initial radiograph and appeared stable. Periodontal charting was performed and probing depths had reduced in each site on the

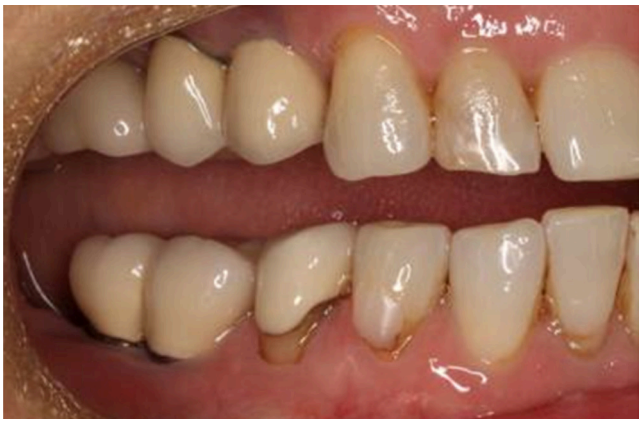

Fig. 11 Initial clinical buccal view of the implants in the mandibular posterior right of the splinted implant restorations (Dec 2023).

Date	December 11, 2023			January 11, 2024			August 19, 2024		
Area	Distal	Mid	Mesial	Distal	Mid	Mesial	Distal	Mid	Mesial
Buccal	6	4	9	3	4	5	4	4	6
Recession	0	0	0	0	0	0	0	0	0
BOP	*	*	*	-	-	-	*	*	*
Suppuration	*	*	*	-	-	-	-	-	-
Mobility	-	-	-	-	-	-	-	-	-
Lingual	6	8	8	6	3	4	2	2	5
Recession	0	0	0	0	0	0	0	0	0
BOP	*	*	*	*	*	*	*	*	*
Suppuration	*	*	*	*	*	*	-	-	-
Mobility	-	-	-	-	-	-	-	-	-

Tab. 2 Table of periodontal charting of the patient at initial presentation and at 1-month and 9-month follow-up appointments.

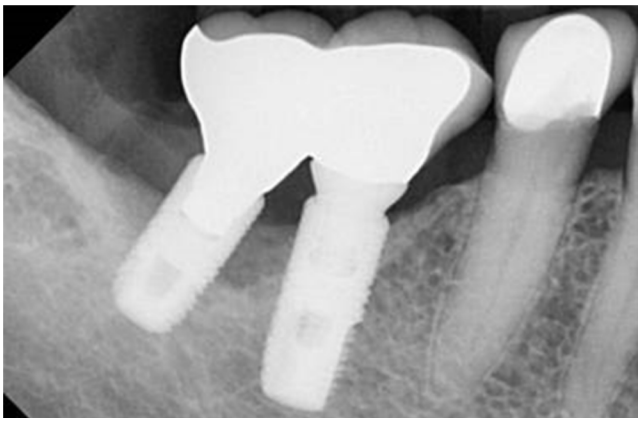

Fig. 12 Periapical radiograph demonstrating crestal bone loss related to peri-implantitis on the two implants with no bone loss (Dec 2023).

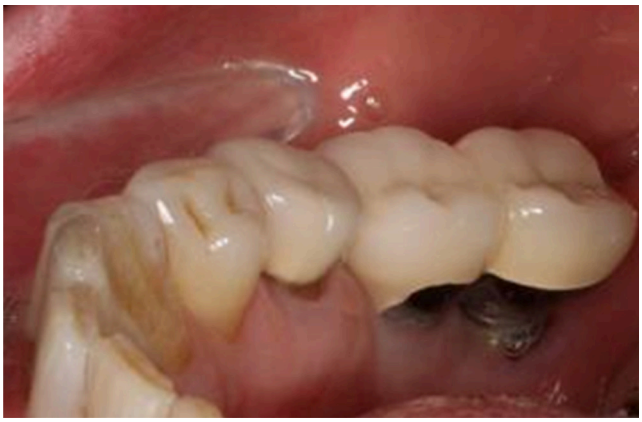

Fig. 13 Initial clinical lingual view of the implants in the mandibular posterior right of the splinted implant restorations (Dec 2023).

	Maxillary left 2nd premolar									Maxillary left 1st premolar								
	Aug 18, 2024			Oct 17, 2024			Dec 19, 2024			Aug 18, 2024			Oct 17, 2024			Dec 19, 2024		
Area	Dis	Mid	Mes	Dis	Mid	Mes	Dis	Mid	Mes	Dis	Mid	Mes	Dis	Mid	Mes	Dis	Mid	Mes
Buccal	7	2	1	6	5	2	3	3	1	6	4	7	6	3	6	3	3	4
Recession	0	0	0	1	1	0	2	3	0	0	0	0	0	1	1	2	3	0
BOP	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Suppuration	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mobility	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lingual	8	4	2	6	2	4	5	6	1	2	6	2	6	1	8	0	0	0
Recession	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0
BOP	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Suppuration	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mobility	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Tab. 3 Table of periodontal charting of the patient at initial presentation for the implants in the maxillary posterior left and mandibular posterior right and at the 2-month and 4-month follow-up appointments.

implant (Table 3). BOP was not noted on the buccal but was still present on the lingual. Clinically the soft tissue had improved on the buccal and lingual (Figures 15 and 16). The patient was next seen at 9 months post treatment and periodontal charting was again performed to check if the area was stable or peri-implantitis was continuing (Table 3). Buccal probing has increased slightly, and BOP was again noted on the side of the implant. Lingual probing has continued to improve from pre-treatment depths. The patient was scheduled for 3-month recall to further evaluate the soft and hard tissue condition and determine if additional treatment may be needed.

The same patient, when seen at a recall appointment in August 2024, it was noted loss of the papilla between the implants at the maxillary 2nd premolar and 1st molar that had been restored in the past with splinted crowns. A periapical radiograph was taken and demonstrated crestal bone loss on both implants

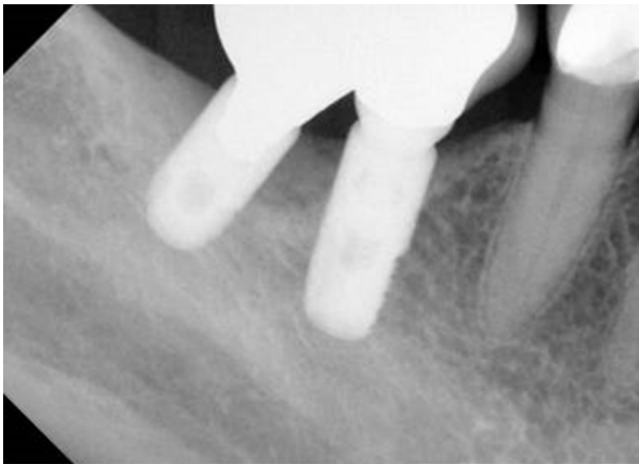

Fig. 14 Table of periodontal charting of the patient at initial presentation for the implants in the maxillary posterior left and mandibular posterior right and at the 2-month and 4-month follow-up appointments.

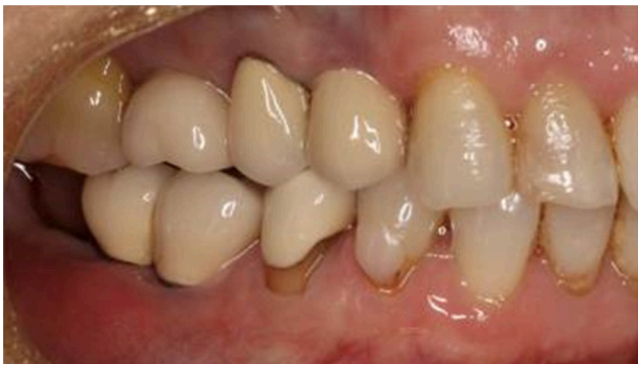

Fig. 15 2-month post peri-implantitis treatment clinical buccal view of the implants in the mandibular posterior right of the splinted implant restorations demonstrating a lack of gingival inflammation (January 2024).

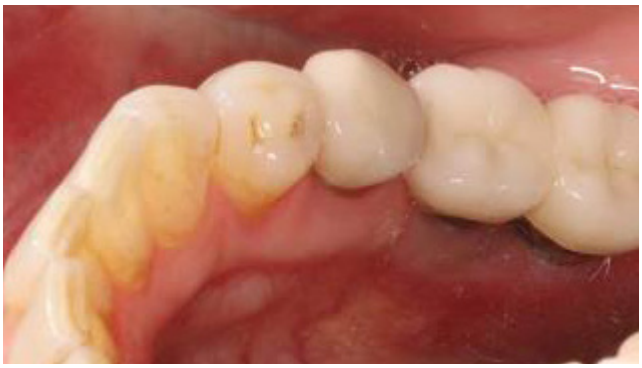

Fig. 16 2-month post peri-implantitis treatment clinical lingual view of the implants in the mandibular posterior right of the splinted implant restorations demonstrating a lack of gingival inflammation (January 2024).

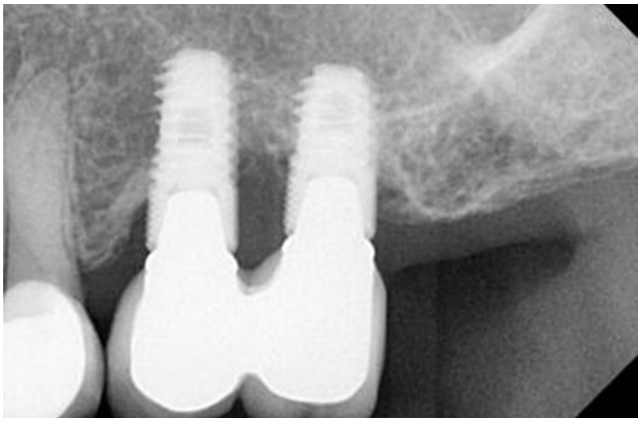

Fig. 17 Periapical radiograph demonstrating crestal bone loss related to peri-implantitis on the two implants with no bone loss (August 2024).

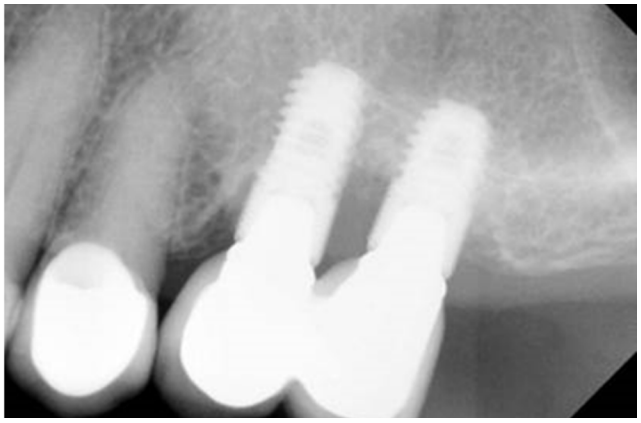

Fig. 18 Periapical radiograph demonstrating crestal bone loss related to peri-implantitis on the two implants with no further bone loss at the 2-month follow-up appointment (Oct 2024).

(Figure 17). Periodontal charting was performed, and significant probing was noted on the buccal and lingual of both implants (Table 3). BOP was also noted on both implants with no exudate noted nor implant mobility. Treatment was performed in a similar manner to the other example case.

The patient was seen 2 months post treatment and a periapical radiograph was taken (Figure 18). Periodontal charting was performed demonstrating slight improvement in pocket depth on both implants but not a significant improvement at that stage (Table 3). Soft tissue inflammation had improved clinically, and the papilla remained flat with the gingival margins of the implants (Figure 19). The patient was next seen at 4 months post treatment to further evaluate treatment progress and tissue stability. Periodontal charting was repeated, and probing depths had significantly improved since pre-treatment depths (Table 3). Clinically, further soft tissue recession had resulted as the peri-implantitis soft tissue inflammation has resolved (Figure 20). The patient was able to maintain homecare, so no further treatment was recommended and she was continued on 3-month recall appointments.

DISCUSSION

When peri-implantitis is not identified and treated, the prognosis is generally poor and may lead to significant complications. As the condition involves both soft tissue inflammation and progressive bone loss around the implant, when left untreated, bone loss can continue, potentially resulting in implant failure. Several key consequences may arise from untreated peri-implantitis.

As peri-implantitis progresses, the infection leads to inflammatory issues with continuing alveolar bone loss crestally at the implant. This ongoing bone loss can eventually compromise the stability of the implant, potentially causing the implant to fail (48). Studies have shown that untreated peri-implantitis is one of the leading causes of implant failure (49). Additionally, when peri-implantitis is left untreated, there is also a risk of spreading the infection to surrounding

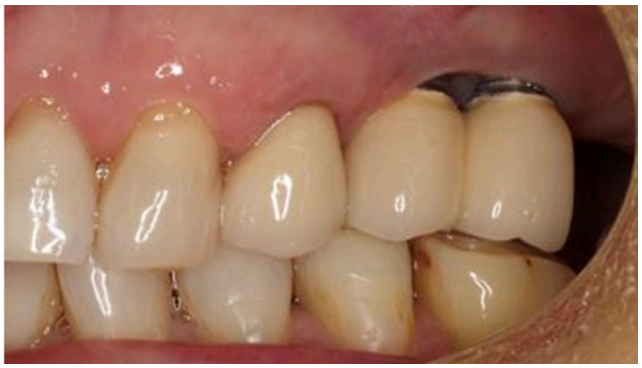

Fig. 15 Clinical appearance at 2-months post peri-implantitis treatment demonstrating an absence of gingival inflammation but loss of the papilla between the two implants (Oct 2024).

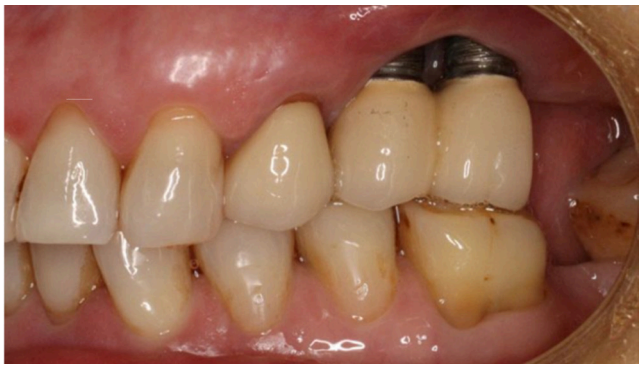

Fig. 15 Clinical appearance at 4-months post peri-implantitis treatment demonstrating an absence of gingival inflammation with further soft tissue recession between the two implants (Dec 2024).

tissues, teeth or implants compromising those areas. Also there is the potential of systemic complications, especially in immunocompromised individuals (50).

When peri-implantitis is treated in a timely manner and appropriately, the prognosis can be significantly improved. Early intervention is key to halting disease progression, preserving the implant. As outlined, the peri-implantitis treatment options include mechanical debridement to remove any debris, calculus and adherent soft tissue to the exposed implant threads, disinfection of the implant's surface, and in some cases, osseous graft placement to eliminate the exposed implant threads. Treatment prognosis depends on several factors, including the severity of the condition (amount of bone loss and soft tissue involvement), the patient's overall health, and their adherence to proper homecare practices following treatment. The key to treatment is resolution of the inflammation and stabilization of the bone levels. With appropriate treatment, peri-implant inflammation of the soft tissue is controllable and further bone loss can be prevented. A study suggests that non-surgical treatment, such as scaling and root planing or the use of antiseptic solutions, can effectively reduce inflammation and stabilize the condition in many cases, especially in the early stages of peri-implantitis (51).

Long-term success of peri-implantitis affected implants depends on the degree of bone loss. Patients with moderate to severe peri-implantitis will require bone grafting with guided tissue regeneration to regenerate lost bone and stabilize the implant (48). When these interventions are successful, the implant can remain functional and stable for many years. But clinical success is dependent on the patient's homecare. Peri-implantitis treatment not only halts bone loss but also improves the health of the surrounding soft tissues. This results in less bleeding on probing, reduced inflammation, and a more favorable aesthetic outcome (52). When treatment is successful and the patient maintains good oral hygiene and regular follow-up care, the prognosis can be quite favorable. A study suggests that with proper treatment and maintenance, the 5-year survival rate of implants affected by peri-implantitis can be high, although implants with more advanced stages of the disease may have a lower long-term success rate (53).

CONCLUSION

In summary, untreated peri-implantitis may lead to severe consequences such as continuous bone loss and potential implant failure. Additionally, failure to treat adds to potential patient discomfort and can contribute to systemic complications via spread of the bacteria in the oral biofilm related to the peri-inflammation. Early detection and intervention are crucial to improving outcomes and preventing the progression of the inflammatory disease. Successful treatment can prevent further bone loss, preserve implant function, and improve both soft tissue health and the patient's overall quality of life. Improved patient homecare is important both to aid in preventing peri-inflammation and following treatment to preventing its recurrence and further worsening of peri-implantitis.

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