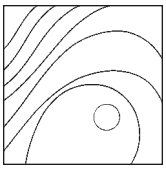
Peri-implantitis Management in the Esthetic Zone in a Periodontally Compromised Patient: Five-Year Results Including Cone Beam Computed Tomography



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A 59-year-old woman was referred for specialist treatment due to peri-implantitis affecting four implants in the anterior maxilla. On presentation, probing depths were up to 12 mm with bleeding on probing/suppuration and extensive peri-implant bone loss. There was a history of treated chronic periodontitis. Nonsurgical management was followed by regenerative peri-implant surgery. Peri-implant probing depths reduced to a maximum of 5 mm and remained stable throughout the 5-year follow-up period. Cone beam computed tomography at 2 years postoperatively confirmed the presence of regenerated peri-implant tissue and integration with the pre-existing peri-implant bone.
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Peri-implantitis is defined as an inflammatory process that affects the tissues around an osseointegrated implant in function and results in loss of supporting bone.[1] Reports of its prevalence vary substantially due to the application of different diagnostic criteria. For example, its prevalence has been reported as 11% to 47%,[2] 12% of patients and 5% of implant sites,[3] 18.8% of patients and 9.6% of implants,[4] and even as high as 28% to 56% of patients and 12% to 43% of implant sites.[5] On the other hand, Albrektsson et al[6] reported significant bone loss requiring surgical intervention or implant removal in < 5% of cases over a period of 10 years. A meta-analysis of marginal bone changes after 5 years in function showed a mean bone loss of −0.24 to 0.75 mm, well below the currently accepted criteria for success.[7] Based on this and similar studies, it has been stated that a small minority of patients are affected by clinically significant bone loss around implants.[8]

Although it seems to be widely accepted that peri-implantitis is caused by the presence of a biofilm on the implant surface,[9,10] a positive correlation also exists between occlusal overloading and peri-implant marginal bone loss.[11] Other authors consider occlusal overload or the bacterial insult from periodontopathogens controversial factors in

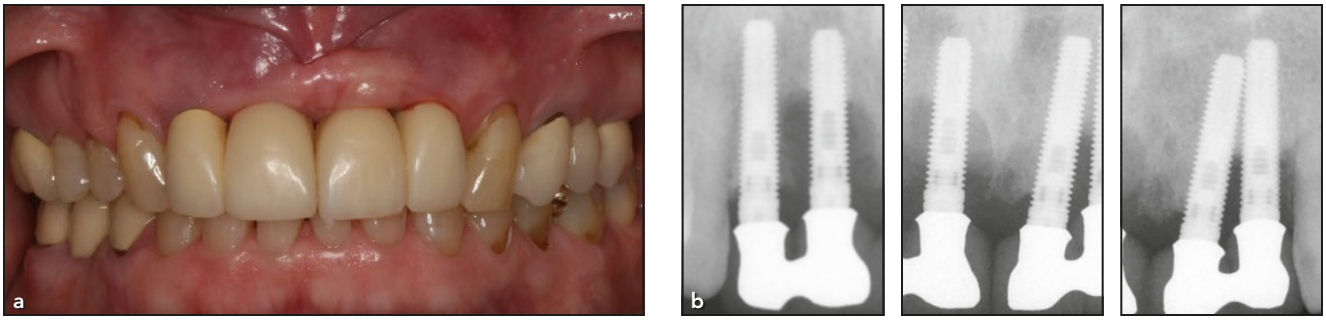


Fig 1 *Case at presentation.* (a) *Frontal view.* (b) *Periapical radiographs of maxillary incisor implants.*

osseoseparation (ie, partial or complete osseointegration failure) and refute an analogy to periodontal disease pathogenesis, suggesting that the term peri-implantitis should not be used routinely.[8] More recently, the concept has emerged that peri-implant crestal bone loss may occur as a biologic response to treatment—not as a disease process but as the result of a foreign body reaction, in which case the establishment of a biofilm is secondary to the peri-implant bone loss.[12]

History of periodontitis is considered a risk indicator for peri-implantitis.[9,13] However, there is no consensus in the current literature regarding the etiology of peri-implantitis and its specific relationship with periodontitis, although a history of periodontitis and smoking may contribute to a higher incidence of peri-implantitis.[14]

Various approaches have been followed in the treatment of peri-implantitis, but there is insufficient evidence as to the most effective therapeutic interventions.[15] Despite the favorable short-term outcomes, recurrence is as high as 100% for some interventions when the follow-up is longer than 1 year, and retreatment may be necessary due to the chronicity of the disease.[15,16]

Cone beam computed tomography (CBCT) is used increasingly in implant therapy to aid diagnosis, treatment planning, and follow-up based on high-resolution three-dimensional evaluation. To the author's knowledge, this is the only long-term study of treated peri-implantitis reported in the literature that includes CBCT findings.

Materials and Methods

A 59-year-old woman was referred by her general dental practitioner for specialist assessment and treatment due to signs of infection and bone loss in relation to four implants in the anterior maxilla. The implants had been placed by a colleague 7 years earlier. There was a history of advanced periodontal disease that had been treated nonsurgically and surgically by a specialist periodontist shortly before and subsequent to implant treatment in the anterior maxilla.

The patient was not aware of symptoms but was concerned about the extent of bone loss and presence of infection (as informed by her dentist). She placed great value on her four anterior teeth, because she felt that they were the essence of her smile. She was aware of her susceptibility to advanced periodontal disease and mentioned that her father had "terrible gum disease." She was a nonsmoker with no relevant medical history otherwise. On presentation (Fig 1), there were few signs of inflammation superficially, apart from slight diffuse swelling and erythema in relation to the maxillary right central incisor. Bleeding on probing (BOP) was 51%, with probing depths (PD) up to 12 mm and purulent exudate in the anterior maxilla. The percentage of PD ≥ 6 mm was 10.1%. Radiographic examination confirmed extensive peri-implant bone loss and close proximity of the implants in the maxillary left central and lateral incisor positions. Despite a thorough check of existing clinical records, it was not possible to identify the implant system that had been used. The implants had been placed in

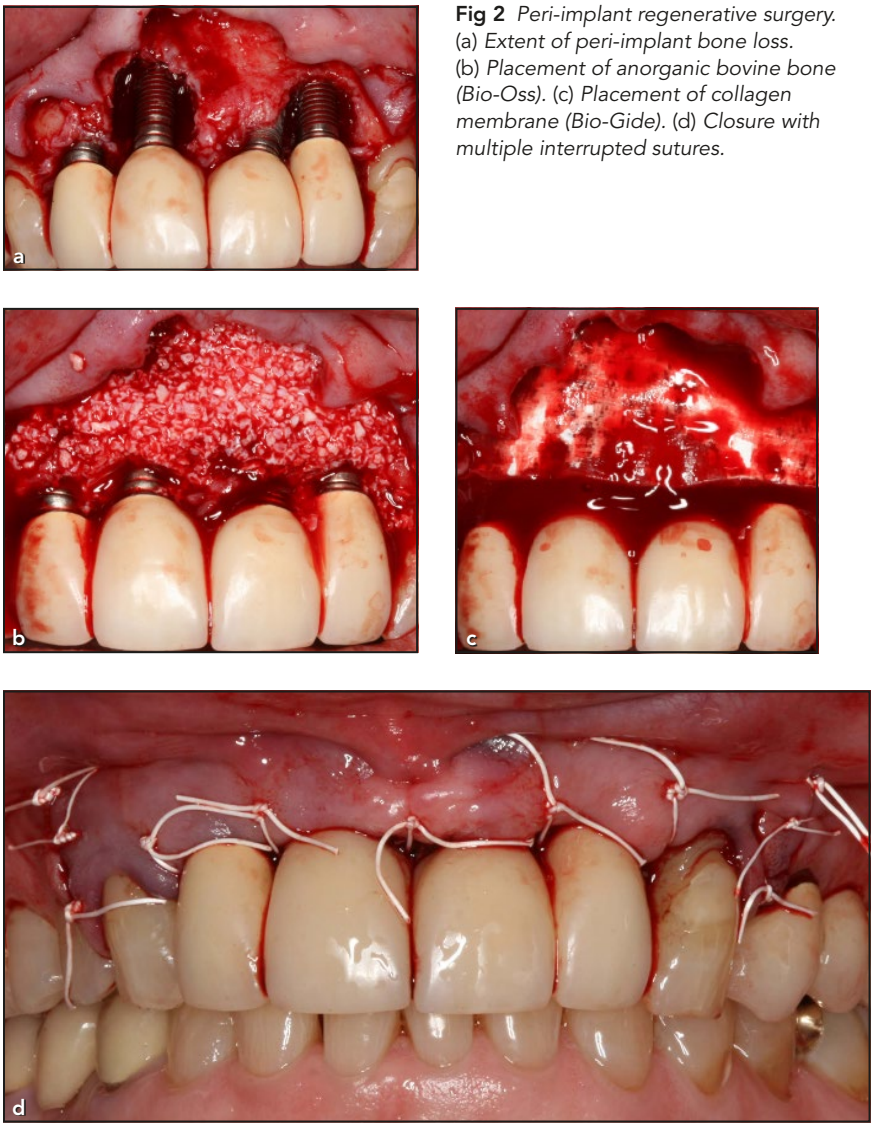


Fig 2 *Peri-implant regenerative surgery.* (a) *Extent of peri-implant bone loss.* (b) *Placement of anorganic bovine bone (Bio-Oss).* (c) *Placement of collagen membrane (Bio-Gide).* (d) *Closure with multiple interrupted sutures.*

native bone without grafting, and radiographic bone levels initially appeared normal.

Nonsurgical therapy consisted of oral hygiene instructions/reinforcement and full-mouth supra- and subgingival/submucosal debridement under local anesthesia with the adjunctive use of systemic antimicrobials (amoxicillin 500 mg and metronidazole 400 mg every 8 hours for 7 days). Re-evaluation after 6 weeks showed significant improvement in BOP (5%), a reduction of PD ≥ 6 mm to 2.9%, but PD up to 11 mm and persistent suppuration in relation to the maxillary right central incisor, the implant that was associated with the most severe bone loss on radiographs. A decision was made to proceed with exploratory/regenerative peri-implant surgery under local anesthesia. The patient was placed on a further course of antimicrobials immediately preoperatively in view of the persistent suppuration (amoxicillin 500 mg and metronidazole 400 mg every 8 hours for 7 days). A buccal mucoperiosteal flap with vertical releasing incisions was raised from the distal side of the right maxillary canine to the distal side of the left maxillary canine (Fig 2). Granulation tissue was removed using carbon fiber and periodontal surgical curettes on the implant and bone surfaces, respectively. The peri-implant bone defects were to an extent circumferential and were approached from the buccal aspect. The exposed implant threads (machined surface) were debrided as thoroughly as possible using an ultrasonic device (EMS Piezon/EMS implant tip, EMS Electro Medical Systems) with distilled water and subsequent copious saline irrigation. The cortical plate was perforated with a small round bur to open the marrow spaces. An anorganic bovine bone substitute was placed into the bony defects and over the buccal surface and was covered with a double layer of a porcine collagen membrane (Bio-Oss/Bio-Gide, Geistlich). The flap was repositioned following incision of the periosteum to achieve tension-free closure and secured with multiple interrupted nonresorbable sutures (Gore-Tex CV-5/CV-6, W.L. Gore & Associates). Postoperative follow-up showed uneventful healing without any flap dehiscence or membrane exposure (Figs 3 and 4). The patient remained compliant and regularly attended her recall appointments at 3- to 4-month intervals, as determined at the annual assessment visits. All treatment, including maintenance, was carried out by the same specialist periodontist (S.K.).

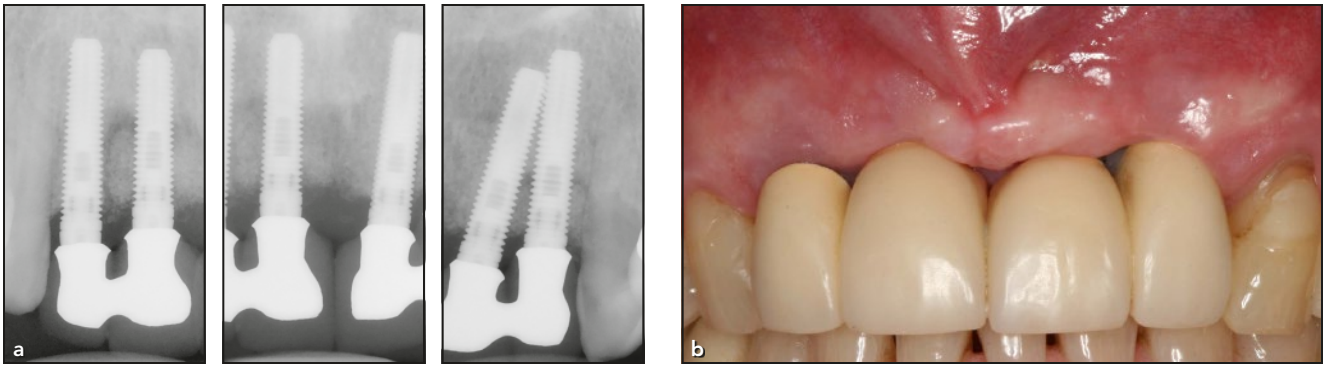


Fig 3 *Radiographic* (a) *and clinical appearance* (b) *at 6 weeks postoperative.*

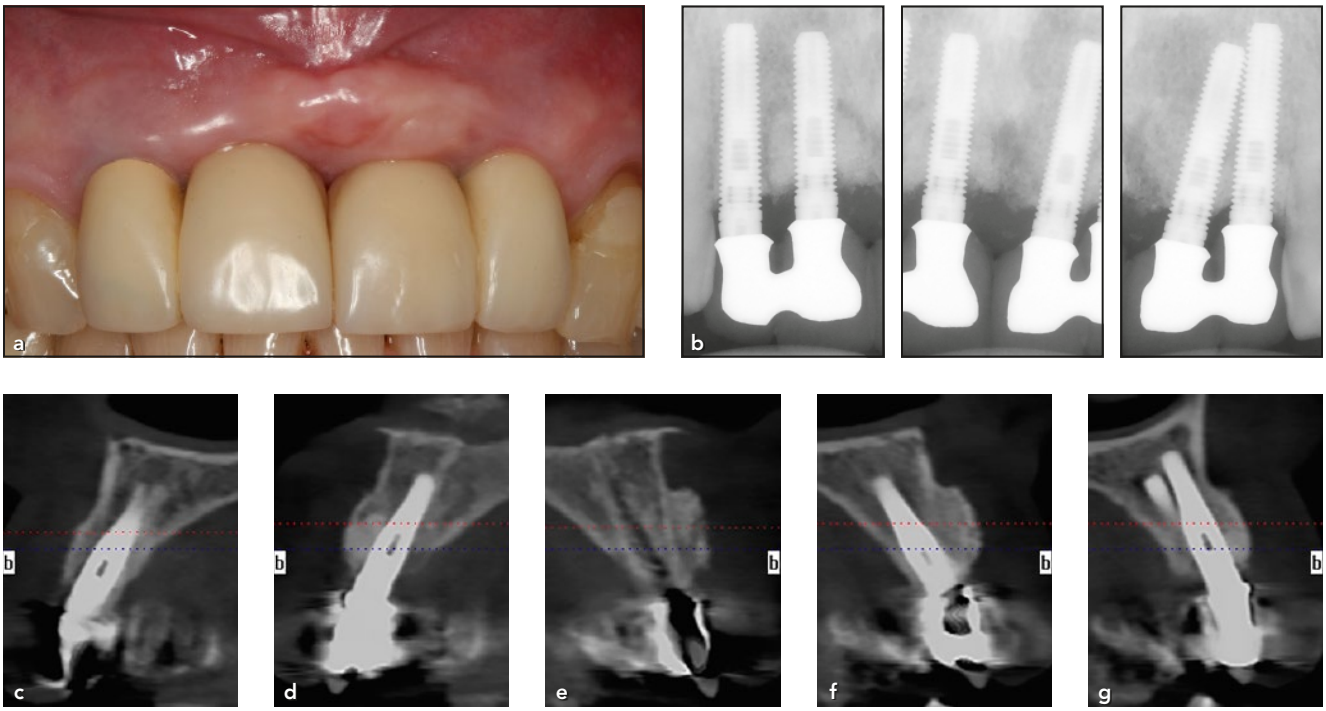
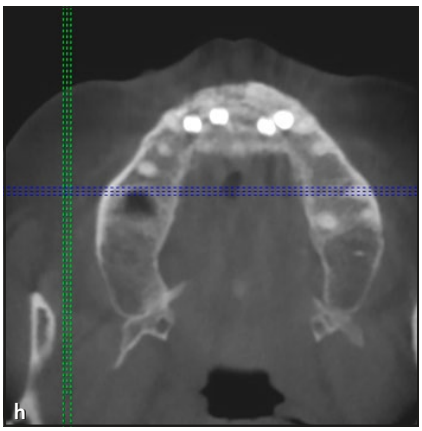


Fig 4 *Clinical and radiographic appearance at 2 years postoperative.* (a) *Frontal view.* (b) *Periapical radiographs of maxillary fixtures.* (c) *Maxillary right lateral incisor (CBCT).* (d) *Maxillary right central incisor (CBCT).* (e) *Maxillary midline (CBCT).* (f) *Maxillary left central incisor (CBCT).* (g) *Maxillary left lateral incisor (CBCT).* (h) *CBCT of maxilla showing the facial contour of the anterior maxilla.*



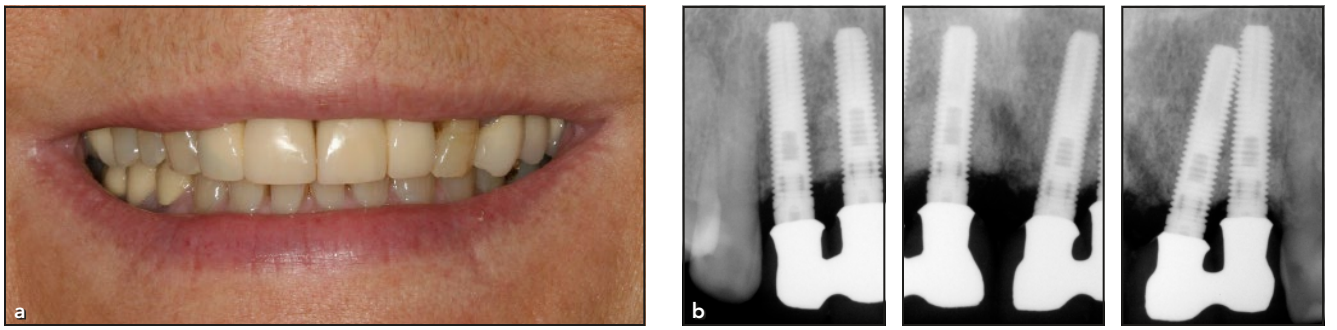


Fig 5 *Outcome at 5-year recall.* (a) *Smile.* (b) *Radiographic appearance.*

Table 1 Six-Point Baseline Chart for Probing Depth (PD), Bleeding on Probing (BOP), and Suppuration (S)

S (B)				*	*	*				*		
BOP (B)	*	*	*	*	*	*	*	*		*	*	
PD (B) (mm)	4	4	4	9	12	9	5	5	5	10	10	4
Implant location (FDI)		12			11			21			22	
PD (P) (mm)	4	2	2	5	10	3	6	4	4	8	7	7
BOP (P)	*		*	*	*		*		*	*	*	*
S (P)												

BOP and S are denoted with an asterisk. B = buccal; P = palatal.

Table 2 Six-Point Chart at Reevaluation

S (B)				*	*							
BOP (B)				*						*		
PD (B) (mm)	2	3	2	11	5	3	2	4	3	8	2	3
Implant location (FDI)		12			11			21			22	
PD (P) (mm)	3	2	2	3	3	3	6	3	3	9	4	5
BOP (P)						*			*	*		
S (P)												

Bleeding on probing (BOP) and suppuration (S) are denoted with an asterisk. B = buccal; P = palatal.

Results

Clinical and radiographic assessment at 5 years postoperatively confirmed a stable outcome (Fig 5). BOP was 7%, and maximum PD on the four fixtures was ≤ 5 mm, representing a clinical attachment gain of up to 8 mm (Tables 1 to 4). The soft tissue architecture improved over time with re-establishment of the interimplant papillae (Fig 6). Plaque score reduced from 26% at the start of treatment to as low as 3% and remained well below 20%, and was 8% at the 5-year recall. PD ≥ 6 mm remained at zero initially, but by year 5 there was a 6-mm PD on the distobuccal aspect of the maxillary left first premolar (this site was treated with regenerative surgery at the time of implant placement). Intraoral radiographs showed periimplant bone fill (Figs 3a, 4b, and 5b), although a zone of relative radiolucency was visible in relation to the maxillary right central incisor. CBCT (i-CAT, voxel dimension 0.25 mm, Imaging Sciences International) at 2 years postoperatively showed the presence of regenerated tissue buccally and interproximally (Figs 4c to 4h). The tissue appeared integrated, but more radiopaque than the adjacent bone, especially on the buccal aspect of the maxillary right central incisor.

Discussion

In this case of regenerative peri-implant surgery in the esthetic zone in a periodontally compromised patient, a stable outcome was observed after 5 years of follow-up. Although nonsurgical and surgical periodontal treatment had been carried out in part prior to implant placement, adequate periodontal stability and appropriate supportive periodontal/

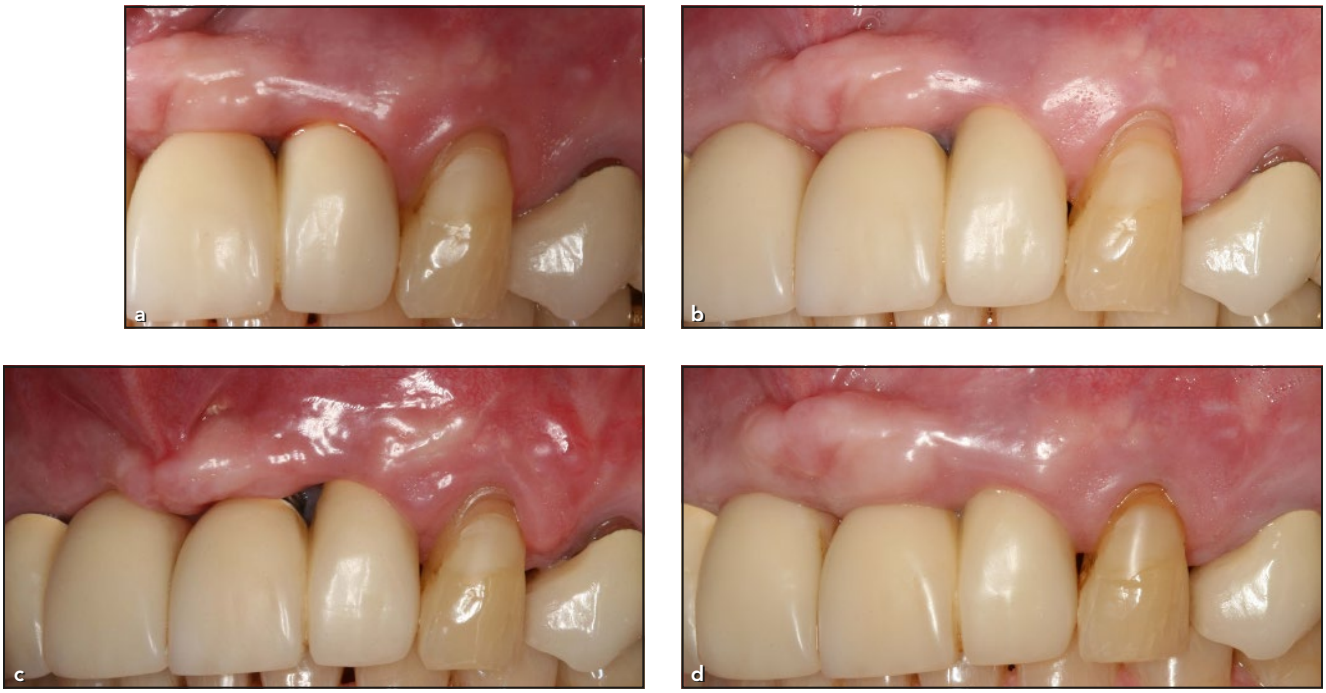


Fig 6 *Improvement in soft tissue contour and papilla formation over time (left lateral view).* (a) *Case at presentation.* (b) *Immediate preoperative view.* (c) *View at 6 weeks postoperative.* (d) *View at 5 years postoperative.*

peri-implant therapy were not emphasized after fixture placement and restoration to ensure long-term stability. A systematic review on the outcome of implant therapy in patients with previous tooth loss due to periodontitis[17] concluded that although implant survival did not differ in patients with and without a history of periodontitis, the incidence of peri-implantitis and peri-implant marginal bone loss increased in the group of patients who had lost teeth due to past periodontal disease. Furthermore, in periodontally susceptible patients the presence of residual periodontal pockets ≥ 5 mm at the end of active periodontal therapy represents a significant risk for development of peri-implantitis and implant loss.[18] In addition, patients with recurrence of periodontitis during

Table 3 Six-Point Chart at 2 Years Postoperative

S (B)												
BOP (B)			*	*						*	*	
PD (B) (mm)	2	2	4	4	4	3	2	4	3	4	4	3
Implant location (FDI)		12			11			21			22	
PD (P) (mm)	2	1	1	4	3	2	5	3	2	3	3	3
BOP (P)					*		*					*
S (P)												

Bleeding on probing (BOP) and suppuration (S) are denoted with an asterisk. B = buccal; P = palatal.

Table 4 Six-Point Chart at 5 Years Postoperative

S (B)												
BOP (B)		*		*				*				
PD (B) (mm)	3	3	3	4	4	3	3	4	4	4	4	4
Implant location (FDI)		12			11			21			22	
PD (P) (mm)	3	2	2	5	3	3	3	3	3	4	3	3
BOP (P)				*					*			
S (P)												

Bleeding on probing (BOP) and suppuration (S) are denoted with an asterisk. B = buccal; P = palatal.

maintenance are at greater risk for peri-implantitis and implant loss than periodontally stable individuals, whereas supportive periodontal therapy reduces the prevalence of peri-implantitis.[18]

Although similarities exist between periodontitis and peri-implantitis lesions, there are also important differences. An experimental study in dogs showed that the peri-implant lesions extended into the bone marrow.[19] Biofilm formation on implant surfaces may be influenced by surface roughness, and the inflammatory cell infiltrate in peri-implant tissues extends to the bone crest. In periodontitis lesions, on the other hand, a connective tissue capsule separates the inflammatory cell infiltrate from the bone.[20] The use of antibiotics is necessary to eliminate or significantly reduce pathogens in peri-implant pockets; metronidazole in particular is directed against Gram-negative anaerobic bacteria.[21] This is part of the Cumulative Interceptive Supportive Therapy (CIST) protocol,[21] which was essentially followed as the treatment approach in this case, also in accordance with the guidelines of the 6th European Workshop on Periodontology.[9] A literature review of peri-implant diseases concluded that in peri-implantitis lesions nonsurgical therapy was not effective, although the use of adjunctive antibiotics reduced BOP and PDs.[22] Further surgical therapy is required following the nonsurgical phase in peri-implantitis cases where suppuration is present.[23]

Decontamination of the implant surfaces was performed with carbon fiber curettes and ultrasonic instrumentation with the use of an implant tip and distilled water, followed by irrigation with sterile saline. A 4-year follow-up of combined resective and regenerative therapy in advanced peri-implantitis cases compared two methods of surface decontamination, Er:YAG laser versus plastic curettes plus cotton pellets and sterile saline, and found no difference in clinical outcomes.[24] Implant surface decontamination during peri-implant surgery with application of 0.12% chlorhexidine and 0.05% cetylpyridinium chloride led to greater immediate suppression of anaerobic bacteria on the implant surfaces than a placebo solution, but there was no effect on clinical and radiographic outcomes over time.[25]

A bone substitute was used in this case mainly as a space filler to support the overlying membrane in such an extensive bone defect, but also as a scaffold to provide osteoconductive properties for new bone formation. A systematic review of surgical management of peri-implantitis suggested that the application of grafting materials and barrier membranes resulted in greater PD reduction and radiographic bone fill.[26] A double layer of Bio-Gide results in a barrier of increased collagen area and thickness and may reduce graft resorption and enhance bone augmentation compared to a single layer.[27]

The presence of regenerated tissue buccally and interproximally in areas of total absence of bone at the time of the surgical intervention was confirmed by CBCT 2 years postoperatively. The case was followed up with intraoral radiographs after that time, and no further CBCT exposure was deemed justifiable based on clinical and radiographic stability. The newly formed tissue was more radiopaque and denser compared to the adjacent bone, presumably due to persistence of the xenograft, but integration with the adjacent bone structure and adaptation to the fixture surfaces seemed to be adequate overall. The zone of relative radiolucency in relation to the right maxillary incisor, the implant with the most severe bone loss, might be associated with the increased presence of fibrous connective tissue and/or lower graft density but had no obvious clinical significance. Without histologic examination it is impossible to know what percentage of the bone substitute particles may remain and their association with the adjacent host bone or connective tissue encapsulation, or whether reosseointegration may have occurred and to what extent. Froum and Rosen[28] performed reentry surgery at 6 months to 8 years postoperatively in five patients (12 implants) and showed bone fill around all implants that ranged from 2 to 9 mm, representing 40% to 100% of the original defect depth. In the present case, normal bone contours were present on CBCT at the crestal level and even a facial excess of regenerated tissue, which appeared more pronounced at the midline and in the region of the left central incisor. With regard to the accuracy of radiologic bone levels, it has been shown that CBCT bone levels correlated closely with histologic bone levels in a ligature-induced peri-implantitis model in dogs.[29]

During the 5-year follow-up there was no recurrence of peri-implantitis. The patient was extremely satisfied with the treatment and outcome. No recession was observed, and she maintained her smile. No palatal flap was raised in this case, and that potentially provided coronal stability for the buccal flap during healing. The soft tissue contour improved considerably over time, presumably due to the favorable underlying tissue contour. The esthetic appearance of the implant prosthesis was far from ideal, but the patient was content and did not wish for replacement (in part for financial reasons). She remained highly motivated and attended her recall appointments regularly, primarily at 3-month intervals. A systematic review and meta-analysis of the frequency of peri-implant diseases indicated that supportive periodontal therapy appeared to reduce the rate of occurrence of peri-implant diseases and concluded that long-term maintenance care for high-risk patients is essential to reduce the risk of peri-implantitis. Informed consent for implant treatment also must include the need for regular maintenance therapy.[4] A retrospective study on treatment outcomes of peri-implantitis cases showed that the effectiveness of peri-implantitis treatment was impaired by severe periodontitis, severe peri-implant bone loss, poor oral hygiene, and low compliance.[30]

Conclusions

Management of peri-implantitis in the esthetic zone in this periodontally compromised patient consisted of nonsurgical treatment, peri-implant regenerative surgery, and appropriate supportive periodontal/peri-implant therapy. CBCT at 2 years postoperatively confirmed the presence of regenerated tissue and integration with the pre-existing peri-implant bone. Clinical and radiographic examination after 5 years of follow-up showed a stable outcome without relapse of peri-implant disease.

Acknowledgments

The author reported no conflicts of interest related to this study.

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