Apical approach to the treatment of peri-implantitis in the esthetic zone: nonincised papillae surgical approach (NIPSA). Case reports.

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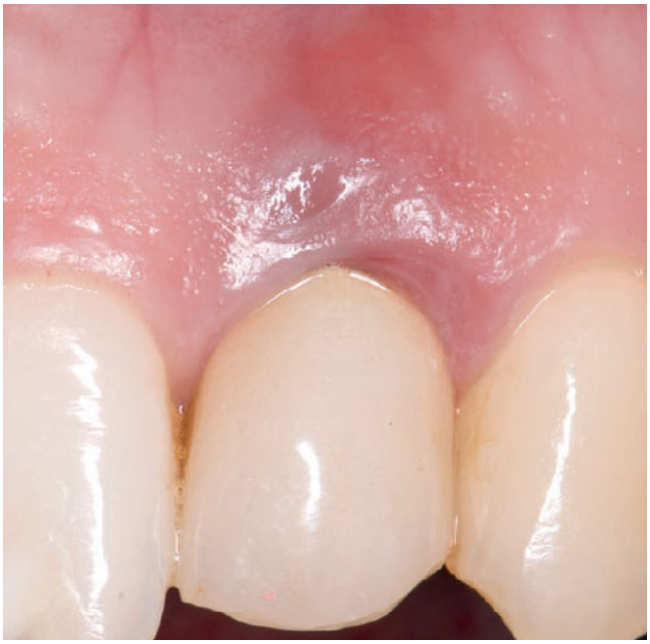

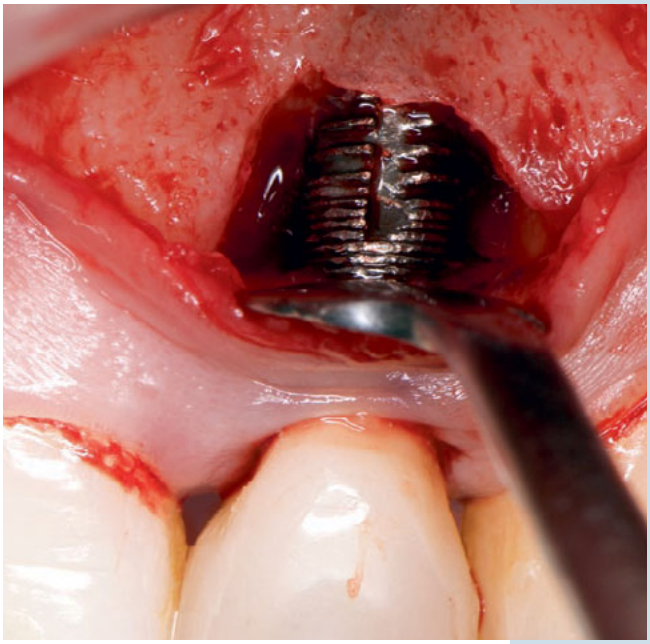

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Abstract

Objective: Advanced peri-implantitis treatment is a clinical challenge. Reconstructive surgery is not suggested in defects with limited bony walls and/or in those with a suprabony defect. All studies of peri-implantitis reconstructive surgery have considered a marginal surgical approach. However, in the present case report, a new apical approach is presented for the reconstruction of an advanced peri-implantitis lesion.

Materials and method: First, a non-surgical phase combines prosthetic, mechanical, and chemical strategies. Second, a surgical phase combines the apical nonincised papillae surgical approach (NIPSA) with biomaterials and a connective tissue graft.

Conclusion: Successful results have been obtained when using a NIPSA for the treatment of peri-implantitis, despite the unfavorable characteristics of the peri-implant defect.

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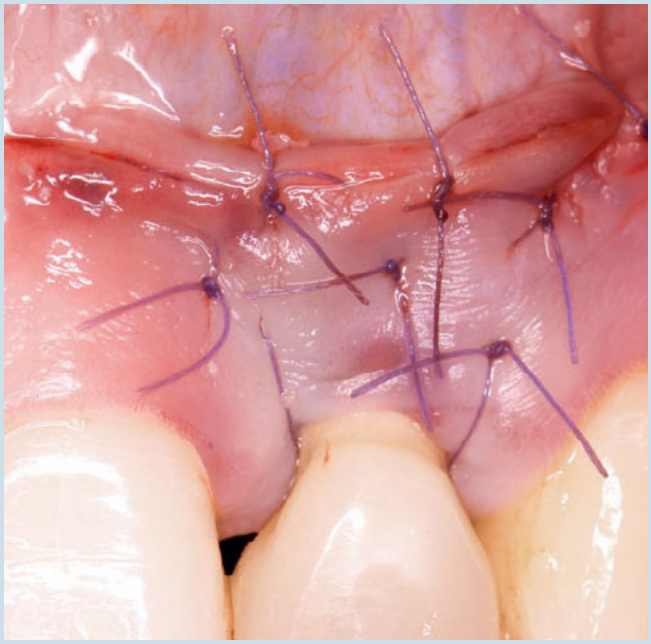
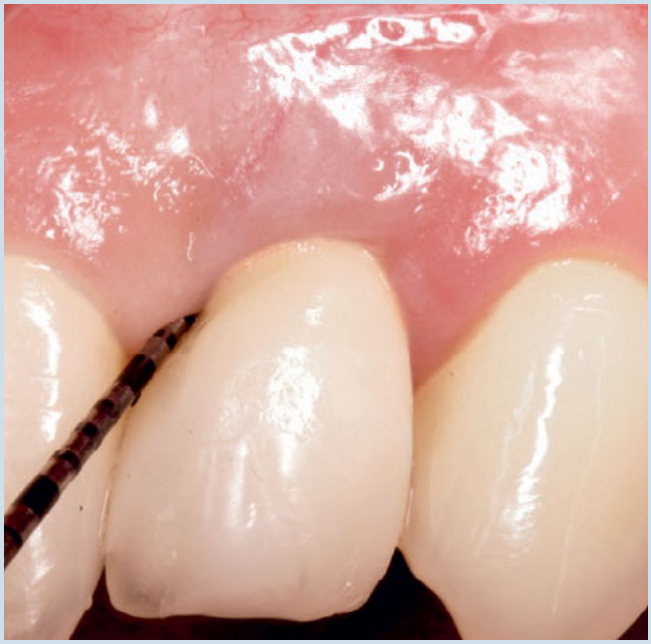




Introduction

Peri-implantitis is characterized by inflammation around an osseointegrated implant with a progressive loss of supporting bone.[1] Peri-implantitis has been reported in 1% to 60% of implants and in 16% to 69% of patients[2-5] and has shown early establishment in the first 3 years postloading, with a nonlinear progression of bone loss.[6] Peri-implantitis occurs as a result of bacterial colonization of the implant surface, which is incompatible with the host.[7] Various risk factors have been implicated in peri-implantitis: a history of periodontal disease and cigarette smoking,[8] poor oral hygiene and inadequate supportive treatment,[9] poor soft tissue quality[5] and/or quantity,[10,11] corrosion of restorative materials, inappropriate restoration and/or placement of an osseointegrated implant,[12] excess cement,[13] and the implant surface.[14]

The progressive nature of the lesions suggests the need for early detection and correction. However, nonsurgical therapy alone is insufficient in deep peri-implantitis lesions, and a surgical approach is indicated.[15] Various approaches have been proposed to treat moderate to advanced peri-implantitis lesions. All surgical approaches access the defect using a marginal incision.[5,16,17] However, a marginal surgical approach may damage the supracrestal soft tissue attachment to the cementum of the adjacent teeth. Furthermore, incising and detaching marginal tissue may impair wound stability and increase postsurgical soft tissue contraction, with negative esthetic consequences due to the dimensional soft tissue changes.[18]

An apical approach has been proposed to treat periodontal lesions with good results, improving soft tissue preservation[19-21] and avoiding an intrasulcular approach and the elevation of the marginal tissue. The aim of the present article is to describe the nonincised papillae surgical approach (NIPSA) to treat peri-implantitis lesions in the esthetic zone.

Case 1

In May 2016, a 26-year-old female in good general health presented with inflammation and suppuration in an implant in position 10 that was placed 8 years previously to treat the agenesis of tooth 10. Four years later, during a routine maintenance visit, the patient complained of a fistula in the area. Clinically, the implant site presented a peri-implant loss of attachment with bleeding on probing (BoP) and suppuration, erythematous gingiva, and a buccal fistula. Over the preceding 4 years, the area had been treated repeatedly by debridement and ozone therapy, without improvement.

The crown was removed to measure the probing from the implant platform. The clinical parameters relating to the implant platform at baseline were: 1) Distal aspect: peri-implant probing pocket depth (PPD) 6 mm, tip of the papillae location (TP) 3 mm; 2) Mesial aspect: PPD 6 mm, TP 5 mm; 3) Buccal soft tissue (BST) location 1 mm; 4) Keratinized tissue (KT) 3 mm; 5) Mesial aspect of adjacent tooth 11 showed attachment loss: PPD 7 mm, clinical attachment level (CAL) 7 mm, recession (REC) 0 mm (Table 1).

Radiographs showed a deep intrabony defect on the mesial aspect and a deep suprabony defect on the distal aspect with loss of the bone peak. Implant threads were destroyed due to previous treatments. The implant had a rough surface and an external hex prosthetic connection. The platform was positioned at the cementoenamel junction (CEJ) level of the adjacent teeth and was restored with a cement-retained metal-ceramic crown. The patient provided her informed consent after receiving a complete description of the proposed periodontal

Table 1 Case 1: Clinical measurements (millimeters)

	Distal aspect			Mesial aspect			Tooth 11 Mesial aspect		
	Baseline	9-month follow-up	Change	Baseline	9-month follow-up	Change	Baseline	9-month follow-up	Change
PPD	6	0	6	6	0	6	7	2	5
TP#	3	5	-2	5	5	0			
	Baseline		9-month follow-up		Change				
BST*	1		3		-2				
KT	3		5		2				
CAL							7	2	5
REC							0	0	0

PPD: probing pocket depth; TP: tip of the papillae location; BST: buccal soft tissue location; KT: keratinized tissue; CAL: clinical attachment level; REC: recession; #: negative TP value indicating papilla coronal displacement; *: negative BST value indicating tissue coronal displacement

surgery (Fig 1), in full accordance with the guidelines of the World Medical Association Declaration of Helsinki and the Good Clinical Practice Guidelines as revised in 2013.

The treatment goals were: 1) Implant surface detoxification; 2) Elimination of chronic infection; 3) Peri-implant hard tissue reconstruction; 4) Peri-implant soft tissue thickening and marginal soft tissue height preservation; 5) Esthetic improvement.

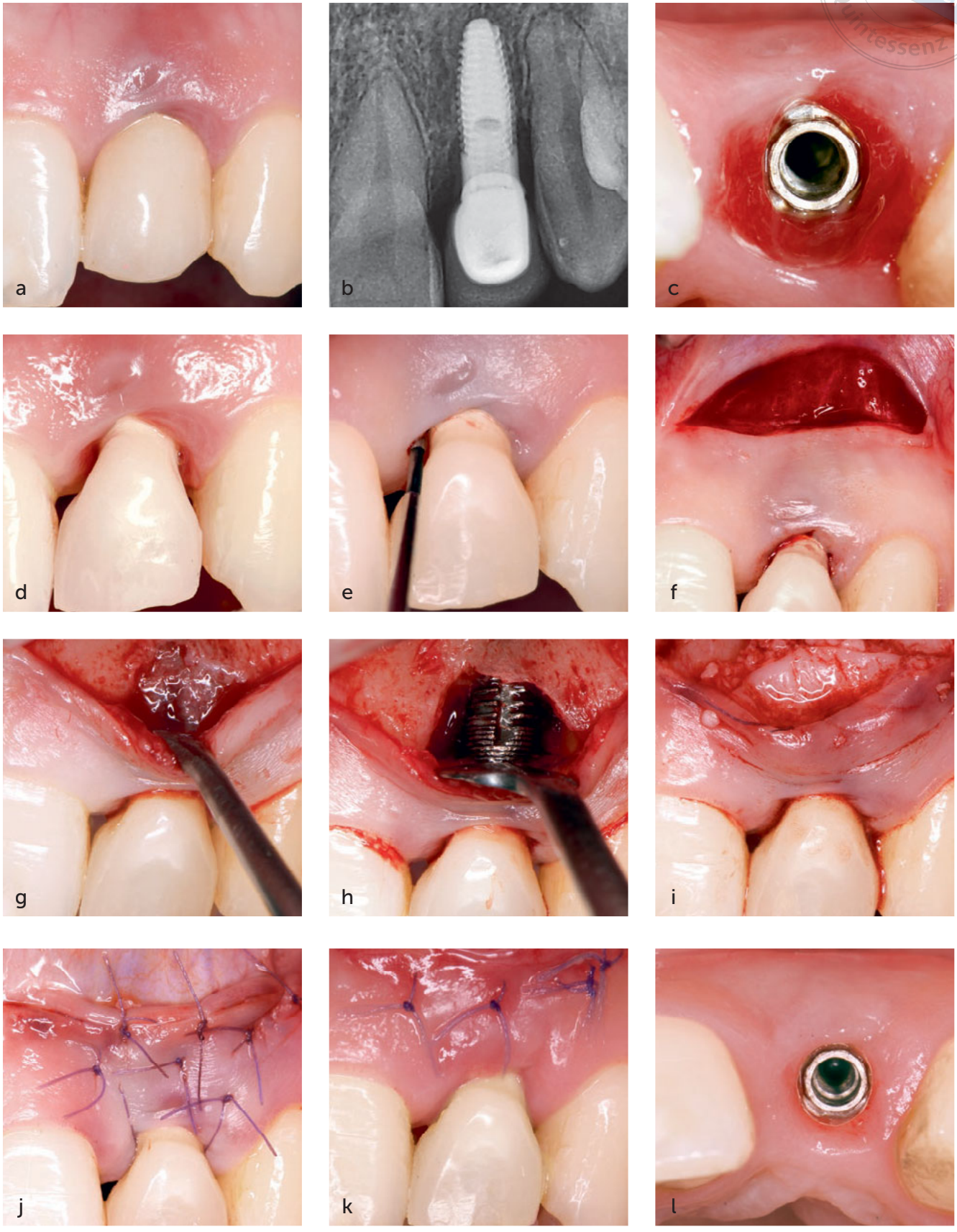
Presurgical procedures

Nonsurgical therapy and provisional restoration

The crown and abutment were removed, showing ulcerated spongy soft tissue around the first thread with suppuration from the peri-implant pocket. The abutment showed evidence of residual cement. The implant was provisionally restored with a titanium abutment and a resin crown. The emergence profile was designed to facilitate daily oral hygiene and interproximal tissue creep. The implant surface was decontaminated by ultrasonics with plastic inserts and abundant 0.2% chlorhexidine irrigation. Tetracycline gel was applied to improve the marginal soft tissue tone. The patient was instructed to brush the area with a soft toothbrush and a roll technique and to use a thin interproximal brush.

Surgical procedure

The principles of the NIPSA procedure described for periodontal regeneration[19-21] were applied 2 weeks later. An apical horizontal/oblique mucosal incision was made on the buccal cortical bone, as apical as possible to the peri-implant defect and away from the marginal tissue, although not excessively apical, avoiding a very extensive incision and allowing access to the defect. The placement, design, and dimension of the incision were determined based on the



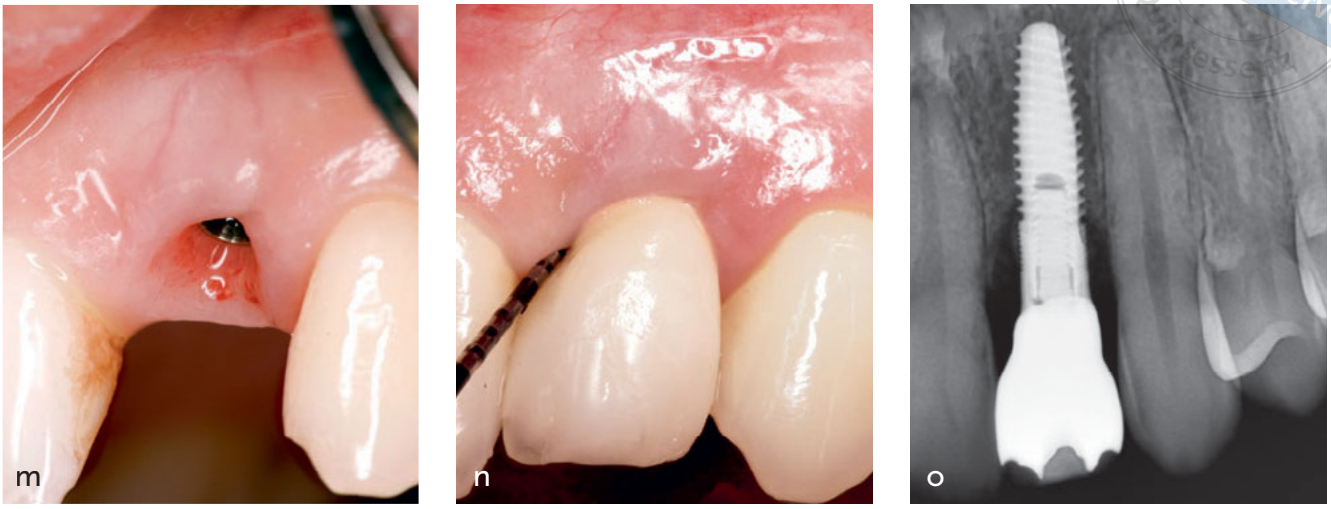


Fig 1 Case 1: baseline, surgical procedure, and follow-up. (a). Frontal view of the initial clinical situation. Inflamed soft tissue and fistula. (b) Initial radiograph showing deep intrabony defects with bone peak loss on the distal side, affecting the adjacent tooth. (c) Occlusal view without crown–abutment structure. Inflamed peri-implant tissue with screw exposed. Spongy and ulcerated surface with suppuration between the implant and the soft tissue. (d) Presurgery treatment. Provisional restoration with space at the papillae to facilitate daily oral hygiene procedures and interproximal tissue creep. Note the marginal fistula. (e) Peri-implant soft tissue status on the day of surgery after presurgery conditioning. Reduction in marginal inflammation and creep of interproximal tissue, filling interproximally. Peri-implant probing pockets on the mesial side. Note the healed fistula and implant translucency through the thin marginal soft tissue. (f) NIPSA: apical incision. (g) Coronally elevated full-thickness flap. Integrity of the suprabony soft tissue complex preserved. Granulation tissue filling the peri-implant defect. (h) Peri-implant defect after defect debridement. Absence of the buccal bony wall. Distal suprabony defect affecting the adjacent tooth. (i) Application of enamel matrix derivative (EMD) protein (Emdogain; Straumann). Composite xenograft plus EMD application. Sutured connective tissue graft (CTG) acting as a buccal barrier protecting the grafted peri-implant defect. (j) Complete closure of the incision by double-line suturing. (k) Complete wound closure 1 week after surgery. Marginal tissue maintaining the structural integrity, with the papillae totally preserved. (l and m) Occlusal and frontal views at 9 months. Fibrous, firm, and healthy peri-implant tissue with marginal creep. (n and o) 3-year follow-up: clinical attachment and radiograph.

preservation of the blood supply and the location of the buccal bony wall, and was performed outside the smile line. From the incision, a coronal full-thickness flap was elevated to access the peri-implant-bony defect, with every attempt being made to maintain the preoperative marginal tissue and the papillae architecture intact. The granulation tissue was detached from the base of the papillae with micro scissors and from the bony walls with titanium curettes, and the detached granulation tissue and peri-implant pocket epithelium were removed. The implant surface was decontaminated using a soft-tip implant ultrasonic insert, but no implantoplasty was performed. Surface detoxification was carried out with 0.25% sodium hypochlorite solution (SHS), meticulously applied with embedded cotton pellets, followed by the application of 24% ethylenediaminetetraacetic acid (EDTA) gel (PrefGel; Straumann) for 2 min. The area was carefully rinsed with saline twice after the SHS and EDTA applications. The root surface of the tooth adjacent to the one involved in the defect was treated by scaling and root planing with micro curettes and ultrasound tips, and EDTA

was applied for 2 min. Enamel matrix derivative (EMD) protein (Emdogain; Straumann) was applied to the surface of the implant and the affected root surface, followed by a composite graft of corticocancellous porcine bone xenograft (OsteoBiol, GenOs; Tecnoss) and EMD, filling the intrabony component. A connective tissue graft (CTG) from the palate at the level of the first molar was harvested as a free gingival graft and deepithelialized extraorally. The mesiodistal length was equal to the distance between the two papillae neighboring the implant. The apicocoronal dimension was 5 mm and the thickness was 1 mm. The CTG was sutured to the inside palatal mucosal surface of the papillae by two vertical mattress 6-0 polyglycolic acid (PGA) sutures at each side of the implant. A primary incision line closure between the two connective tissue sides was achieved with horizontal internal mattress 6-0 PGA sutures along the incision line.

Postsurgical procedure

Postoperative medication included antibiotics (250 mg amoxicillin and 250 mg metronidazole three times a day for 7 days) and ibuprofen 600 mg every 8 hours, as needed. The patient was instructed to rinse with 0.2% chlorhexidine digluconate twice a day for 4 weeks. The sutures were removed after 7 days. Complete wound closure was maintained, and no alteration in the marginal tissue was observed. After 4 weeks, the patient was instructed to start brushing with a soft toothbrush and a roll technique, and to use a thin interproximal brush during the first months so as not to damage the healing tissue during the maturation period. The patient was recalled for control and prophylaxis at 1, 2, 3, and 4 weeks, and at 3, 6, and 9 months.

At the 9-month postsurgery follow-up, no alteration in the soft tissue was observed. The crown was removed to measure the probing from the implant platform. The clinical parameters relating to the implant platform at 9 months were: 1) Distal aspect: PPD 0 mm, TP 5 mm; 2) Mesial aspect: PPD 0 mm, TP 5 mm; 3) BST 3 mm; 4) KT 5 mm; 5) Mesial aspect of tooth 11: PPD 2 mm, CAL 2 mm, REC 0 mm (see Table 1). The clinical examination revealed no BoP and healthy soft tissue.

Restorative procedures

The provisional crown was not removed or modified for emergence profile modulation for 9 months, when a final restoration was placed according to standard protocol. The emergence profile was straight, corresponding to the part of the crown emerging from the soft tissue, and the cervical contour was convex, corresponding to the CEJ located in the sulcus. Thirty months after the final restoration, the peri-implant tissue remained healthy and stable under routine maintenance. Radiographic examination showed a complete fill of the intrabony defect.

Case 2

In June 2017, a 50-year-old female non-smoker in good general health presented with inflammation in implants in positions 12 and 13. The area had been restored with screw-retained metal-ceramic crowns 20 years previously. Clinically, the implant site presented a peri-implant pocket and loss of attachment with BoP. After removal of the superstructure, the clinical parameters relating to the implant platform at baseline were: 1) Mesial aspect (implant 12): PPD 8 mm; 2) Interimplant aspect: PPD (taking the highest value) 9 mm, interimplant papillae height (Ph) 2 mm; 3) Distal aspect (implant 13): PPD 7 mm; 4) BST 3 mm (implant 12), 2 mm (implant 13); 5) KT 2 mm for both implants (Table 2).

Radiographs showed an intrabony defect plus supraalveolar-type defects affecting both implants.

The treatment goals and procedures were the same as for case 1, except that provisional crowns were not used. As the crowns that the patient already had were correctly adapted, they were unscrewed, deep cleaned, and replaced.

Two years postsurgery, the clinical parameters were: 1) Mesial and distal aspects: PPD 2 mm; 2) Interimplant aspect: PPD 3 mm; 3) Ph 2 mm; 4) BST 3 mm for both implants; 5) KT 4 mm (implant), 3 mm (implant) (see Table 2). The clinical examination revealed no BoP and healthy soft tissue. Radiographic images showed a situation compatible with complete reconstruction of the intrabony and supraalveolar defect (Fig 2).

Discussion

The present report describes the treatment of a peri-implant pocket in the esthetic zone using NIPSA.

Case 1 reports an early onset of the peri-implantitis lesion, as has been reported previously.[4] The peri-implant lesion affected the coronal half of the alveolar bone surrounding the implant and the interproximal periodontal support of an adjacent tooth. Several factors may be related to this peri-implantitis lesion: 1) The affected implant was placed 8 years previously to treat the absence of a maxillary lateral incisor. The lack of permanent tooth eruption is often associated with hard and soft tissue deficiencies;[4] 2) Excess cement, as found on the surface of the abutment, has previously been correlated with peri-implantitis[13] and may facilitate biofilm retention, acting as a potential initiating factor in peri-implant disease;[22] 3) Poor implant abutment quality; 4) Inappropriate implant placement;[11] 5) Inadequate maintenance and supervision.

Case 2 showed peri-implant disease in a long-term osseointegrated dental implant in a patient with periodontal disease. A close relationship between periodontal disease and the risk of peri-implantitis has been demonstrated.[23] In this case, although the patient maintained good dental hygiene and attended all control visits and periodontal maintenance revisions, and there were no risk factors such as smoking or systemic disease, she developed peri-implantitis.

The treatment of peri-implantitis requires a combined restorative–surgical effort to achieve the biologic and esthetic goals, especially in iatrogenic restorations where 1) the prosthetic structure and the transmucosal components may be a source of bacterial contamination,[24] and/or 2) the restoration does not respect the space required to stabilize good biologic sealing by the peri-implant soft tissue above the alveolar bone.[24,25] The first treatment step in case 1 was to modify the transmucosal component to allow cleaning of the prosthetic component exposed to the environment. These modifications corrected the overcontouring of the crown that violated the space for peri-implant soft tissue stabilization (peri-implant biologic width).[25-27] The modifications may also have provided space for soft tissue growth and thickening as well as improvement in the delicate interproximal tissue quality and vascularity for further surgical manipulation.[28]

No presurgical mechanical debridement was carried out in either case. It is difficult to achieve complete biofilm removal from the implant surface compatible with healthy peri-implant tissue, thus leading to a tendency to disease recurrence.[29] Furthermore, uncontrolled curettage of the marginal soft tissue may occur in deep areas of the peri-implant pocket, resulting in marginal soft tissue shrinkage. Presurgical local antibiotic[30] was applied as a chemical agent to reduce bacterial levels and peri-implant

Table 2 Case 2. Clinical measurements relating to the implant platform (millimeters)

	Mesial aspect (implant 12)			Interimplant aspect			Distal aspect (implant 13)		
	Baseline	2-year follow-up	Change	Baseline	2-year follow-up	Change	Baseline	2-year follow-up	Change
PPD	8	2	6	9	3	6	7	2	5
	Baseline			2-year follow-up			Change		
Ph	2			2			0		
	Implant 12			Implant 13					
	Baseline	2-year follow-up	Change	Baseline	2-year follow-up	Change			
BST*	3	3	0	2	3	-1			
KT#	2	4	-2	2	3	-1			

PPD: probing pocket depth; Ph: interimplant papillae high; BST buccal soft tissue location; KT: keratinized tissue; *: negative BST value indicating tissue coronal displacement; #: negative KT value indicating KT gain

tissue inflammation. Intrasurgically, a combination of mechanical and antimicrobial treatment was carried out. No single method seems superior, and evidence suggests clinical improvements following combination therapy.[7] In addition, there is evidence that systemic antibiotics as an adjunct to mechanical debridement may potentiate the antibacterial effect by achieving effective levels in the peri-implant crevicular fluid.[31]

It has been suggested that for less than two-wall defect configurations, regenerative procedures are not indicated in peri-implantitis, since the defect may not hold the grafting biomaterial.[17,32] Case 1 showed a two-wall configuration apically, which extended coronally with the loss of the buccal bone, and distally with the absence of the interproximal bone peak, while case 2 showed a supraalveolar-type defect. The combined use of an apical approach, biomaterials, and a CTG appears to be a desirable strategy.

Maintaining the integrity of a firm suprabony soft tissue complex using NIPSA may have the following benefits: 1) It favors the conditions needed for tissue regeneration, which are space provision, wound stability, and primary intention healing;[33] 2) Optimal soft tissue preservation;[19,20] 3) A reduction in the influence of flap micro-movement, preserving the architecture of the interproximal soft tissue, which is one of the main factors influencing the vertical stability of the clot.[18] In addition, the application of EMD may stimulate an increase in blood vessels in the soft tissue, improving wound healing[34] and inhibiting epithelial cell migration,[35] and it may have a bactericidal effect.[36] Corticocancellous porcine bone xenograft was applied to augment the ridge dimension, providing long-term stability[37,38] and masking dark tones from the restorative materials.[39] Finally, a CTG was applied to increase soft tissue thickness,[37] improve crestal bone remodeling,[10,11] delay epithelial

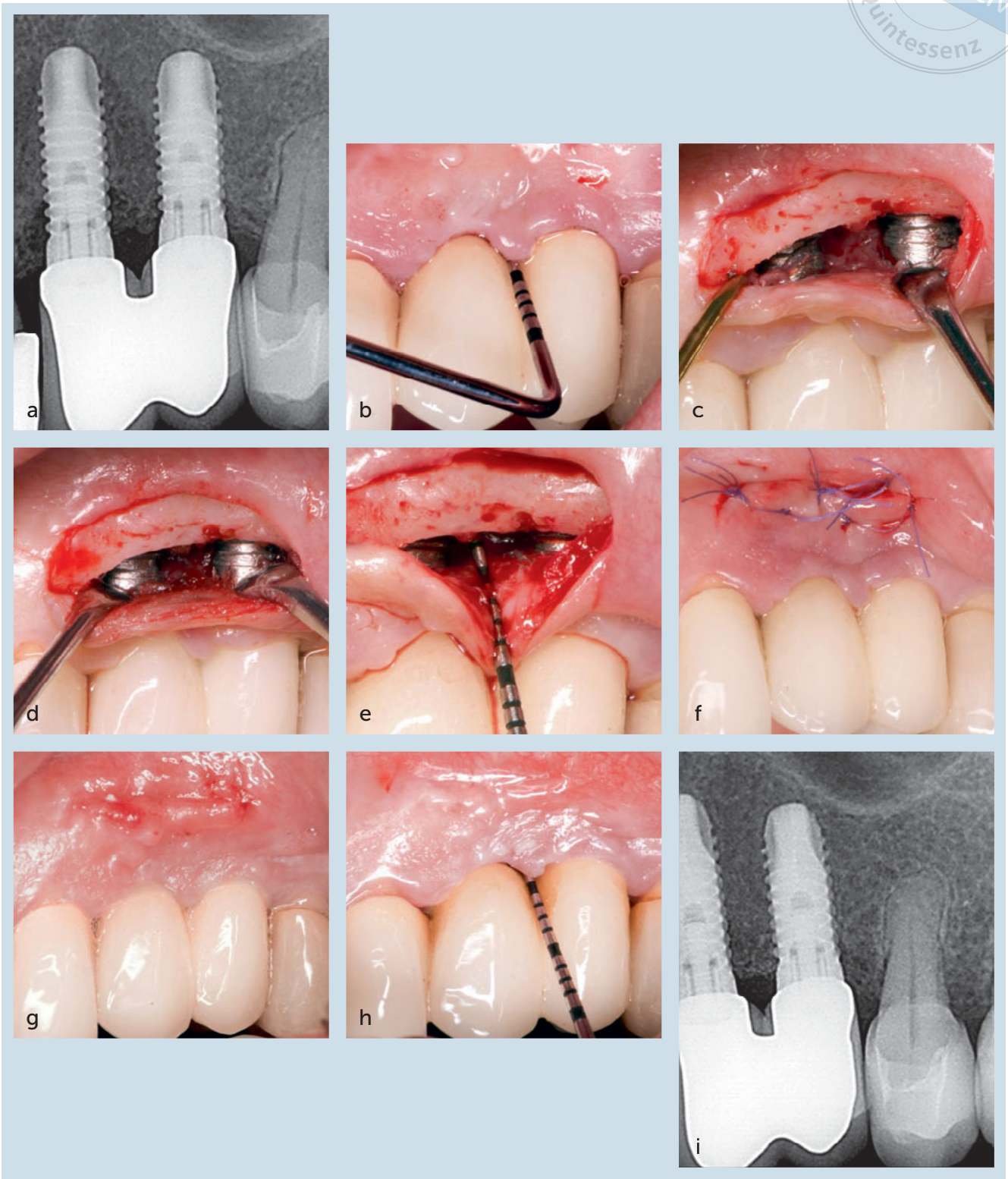


Fig 2 Case 2: baseline, surgical procedure, and follow-up. (a and b) Baseline radiograph and interimplant probing depth. (c to e) Apical incision and defect after granulation tissue debridement and implant surface decontamination. Supraalveolar-type lesion plus 4 mm intrabony lesion. (f) View immediately after surgery showing interimplant papilla preservation. (g) View after suture removal 5 days post-surgery. (h and i) Follow-up: interimplant probing depth and radiograph.

cell downgrowth as a result of clot stabilization maintenance in the defect,[40,41] mask dark tones from implant–restoration structures, provide a shield to compensate for the absence of the buccal bone plate,[42] and increase wound stability in the horizontal aspect.[18]

NIPSA is a blind technique for the lingual aspect of peri-implantitis defects.[20] However, intrabony defects with two- or three-wall configurations (55%)[43] and buccal bone in the lost wall in most situations[43] is the most prevalent peri-implant defect morphology. Therefore, the most frequent peri-implant defect morphology may favor access to the defect, to decontaminate the implant surfaces and control the complete removal of possible hard stains or cement remnants attached to the implant surfaces through a direct view when using NIPSA.

Maintaining the implant restoration connection during the healing phase seems to improve the peri-implant tissue response and maturation.[26,27] The final restoration emergence profile and contour need to reproduce those of the replaced natural tooth.[44]

NIPSA has been tested in the treatment of different types of periodontal lesions, even those that are unfavorable.[19-21] Recently, NIPSA has been applied in peri-implantitis lesions in combination with laser therapy, with good results.[45] Reports show the early beginning of the peri-implant lesion within the first 3 years postloading.[6] In the present cases, the load has remained since the beginning of treatment, and a healthy and esthetic result has been maintained for 2 and 3 years postsurgery, respectively. Although the present report shows good results, further clinical trials are needed.

Conclusion

The preliminary results after the use of NIPSA plus a CTG in peri-implantitis lesions showed considerable improvements in clinical parameters, with complete elimination of the peri-implant pocket and a gain in periodontal attachment and soft tissue preservation.

Clinical significance

Soft tissue preservation and defect resolution may be achieved through an apical approach plus a CTG in the treatment of peri-implantitis lesions.

Consent for participation and publication

The patients gave their written informed consent after receiving a complete description of the surgical procedure as well as their consent for the publication of their intraoral and radiographic images.

Disclaimer

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