Therapy for Peri-Implantitis: Significant Radiographic Bone Fill After Keratinized Mucosa Augmentation Surgery With Supportive Implant Therapy: A Novel Approach

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Introduction

In dental implant therapy, peri-implant health can be defined by the absence of inflammation[1] and unchanged soft and hard tissue dimensions.[2] This outcome represents homeostasis between the intraoral microbial biofilm and the host response in the peri-implant tissue.[3]

Scientific research aims to identify potential risk factors for the onset of peri-implant diseases.[1,4,5] One potential risk factor is insufficient soft tissue quality, as indicated by the width of the keratinized mucosa (KM)[3,4] and the mucosal thickness.[2]

Nonsurgical therapy of peri-implantitis comprises intensive mechanical debridement and biofilm removal of all accessible surfaces of the implant/abutment/denture, often followed by the application of antimicrobial agents and permanent biofilm control via supportive implant therapy (SIT).[3] Several studies have reported that this approach can reduce the clinical signs of peri-implant inflammation (bleeding on probing [BOP]) and the peri-implant pocket depths (PDs).[6–9]

However, in most cases of peri-implantitis, surgical treatment must be performed.[10]

This approach may lead to a significant decrease in inflammation, a reduction in PD and stabilization of the peri-implant bone level, which provides adequate support for the overlying soft tissues.[11]

Currently, the possible effect of peri-implant soft tissue dimensions on the onset of peri-implant diseases remains controversial. Some studies have not revealed any correlation between a lack of KM and increased disease rates.[12–14] However, it must be stated that these data have mostly resulted from well-maintained patients under SIT conditions. On the other hand, 2 systematic reviews concluded that a peri-implant KM width of <2 mm is associated with more plaque and higher inflammation scores.[15,16]

Recommendations for the treatment of existing peri-implantitis with soft tissue surgery are very rare. This case series presents four patients with unexpected results regarding the amount of peri-implant radiographic bone fill (RBF) after treatment consisting exclusively of soft tissue augmentation plus biofilm control via SIT.

Materials, Methods, Treatment Course, and Results

This retrospective clinical case series was conducted at a private practice specializing in dental implant therapy. This study was reviewed and authorized by the Ethics Commission of the Albert-Ludwigs-University of Freiburg, Germany (application No. 10007/19). Our study was conducted in compliance with the appropriate EQUATOR guidelines (STROBE). All patients diagnosed with peri-implantitis initially underwent nonsurgical therapy consisting of repeated removal of the peri-implant biofilm and repeated installation of chlorhexidine gel (1%) during a 3-month period. Thereafter, patients with keratinized mucosa widths (KMWs) <2 mm were recommended to undergo peri-implant KM augmentation surgery (with a free gingival graft [FGG] or partially epithelialized free connective tissue graft [PECTG][12]).

Three patients received an FGG, and 1 patient (HD) received a PECTG. After wound healing, all patients were enrolled in an SIT program with 3-monthly follow-up (remotivation, professional biofilm removal, and measurement of PD and BOP values). All patients were compliant with SIT (minimum 2 times/y).

No true endpoints have been identified to diagnose peri-implantitis.[4,17–19] Therefore, the following surrogate endpoints were used: clinical signs of inflammation (redness, swelling, positive BOP, and suppuration), increase in PD $\geq$ 2 mm and increase in bone loss $\geq$ 1 mm along the mesial or distal contour of the implant (on radiographs).[1]

Peri-implantitis causes peri-implant bone loss, which usually presents with a conical (funnel-like) shape. Therefore, on radiographic examination, one can find an upper bone line representing the upper bone margin of the funnel. Moreover, the second important structure to examine is the bottom boundary of bone loss (Figure 1), which represents the most apical point of the radiographically visible bone resorption process. We assessed bone level changes along the upper bone contour lines (UBLs) and the deepest bone resorption lines (DBLs). UBL values were defined as the distance between the implant shoulder and the first radiographic bone-to-implant structure in an apical direction. DBL values were defined as the

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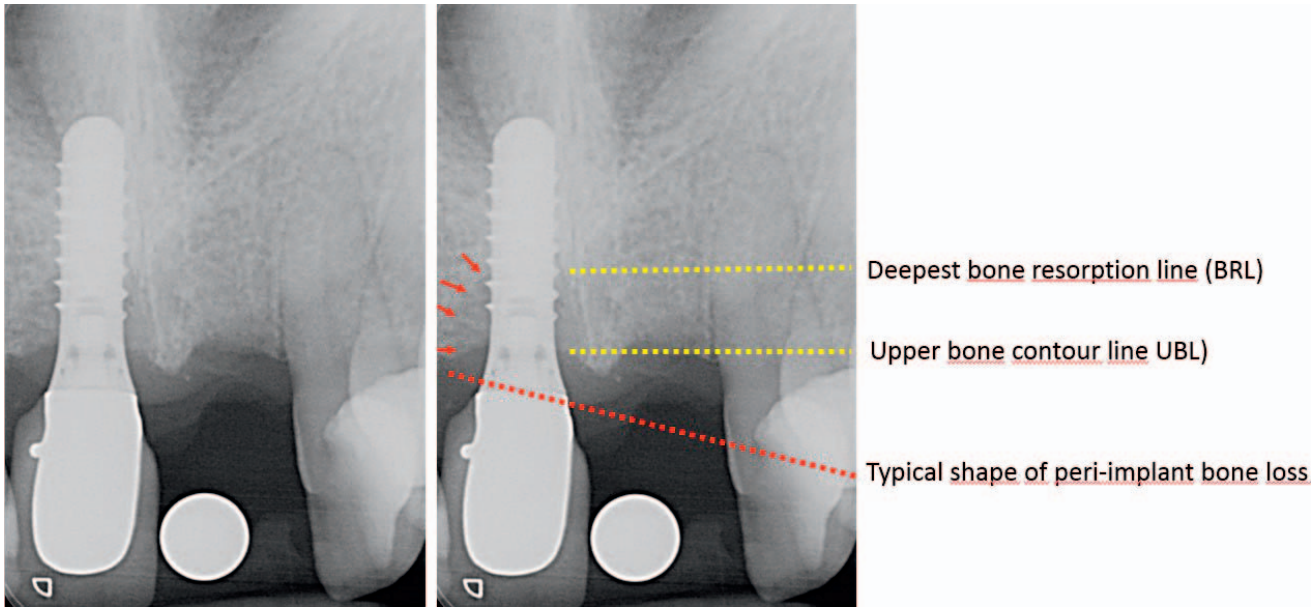


Figure 1. (a) Demonstration of the definitions of the upper bone contour line (UBL) and deepest bone resorption line (DBL) in a patient with peri-implantitis (ITI Bonefit, length 12 mm) before surgical therapy. (b) Radiographically, the UBL values were 2.5 mm mesially and distally, while the DBL values were 7 mm mesially and 6.5 mm distally.

distance between the implant shoulder and the most apical resorption that was visible (Figure 1).

In this case series, the examination comprised 5 implants in 4 patients (Figures 2 through 5). In total, 7 measurement points (MPs) for radiographic bone loss along the mesial and/or distal contour of an affected implant were identified.

Before surgery, the affected implants had been under intraoral treatment for 1.42 to 8 years. The observation period of this study (time from KM augmentation to last examination) ranged between 1.17 and 18.41 years. In all patients, peri-implantitis was treated successfully and did not reoccur (Table 1). All implants showed a significant increase in KM width. Compared to baseline (peri-implantitis), the peri-implant radiolucency findings (bone loss) decreased to an unexpected extent (up to 100%) in the final radiographic examination (Table 2).

UBL

The UBL changes varied from -0.5 mm to +3 mm (mean: 0.86; median: 0.5; SD: 1.21).

DBL

All MPs showed significantly reduced UBL values from 1.5 mm to 5.5 mm (mean: 3.86; median: 4.5; SD: 1.41).

RBF

Values ranged from 1.5 mm (60%) to 5.5 mm (100%) (mean: 3.9 [91%]; median 4.5 [100%]). Five MPs at 3 implants showed 100% RBF.

KMW

At baseline, only 1 implant showed a KM (0.5 mm), and no others had keratinized tissue at the buccal aspect. After therapy, the KMW was established for all implants, with a range of 2–6 mm (mean: 4.2; median: 4; SD: 1.48).

PD

One to 18 years after KM augmentation surgery, the mean PD values ranged from 3 to 3.83 mm (mean: 3.36; median: 3.33; SD: 0.38), while the deepest PD values ranged from 3 to 5 mm (mean: 4.29; median: 5; SD: 0.95).

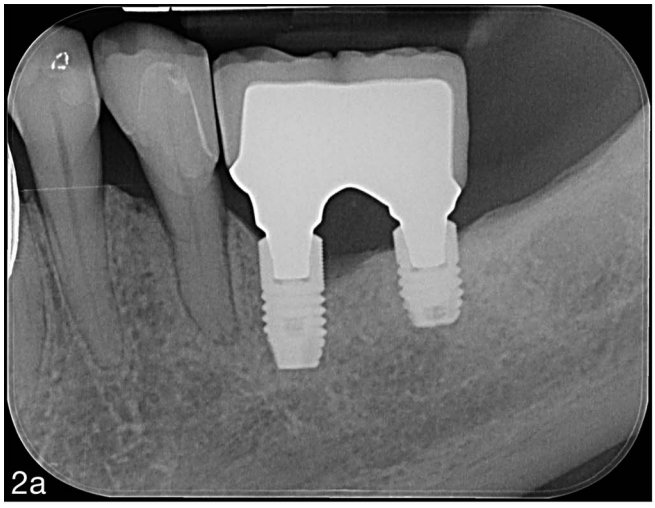
BOP

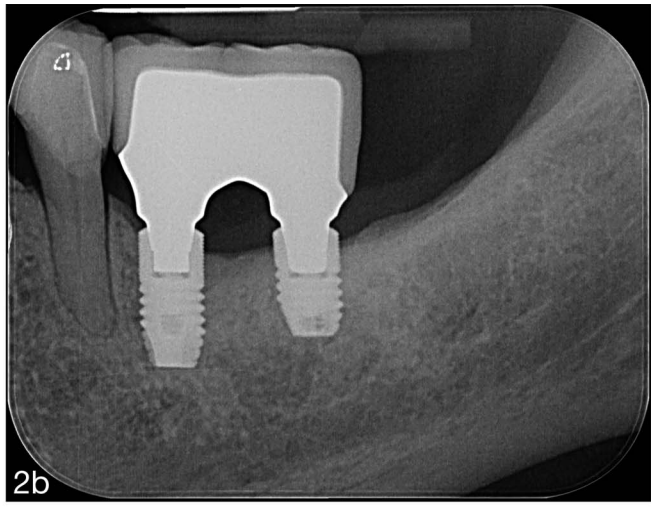
Two of 7 MPs (28.6%) showed a positive BOP measurement.

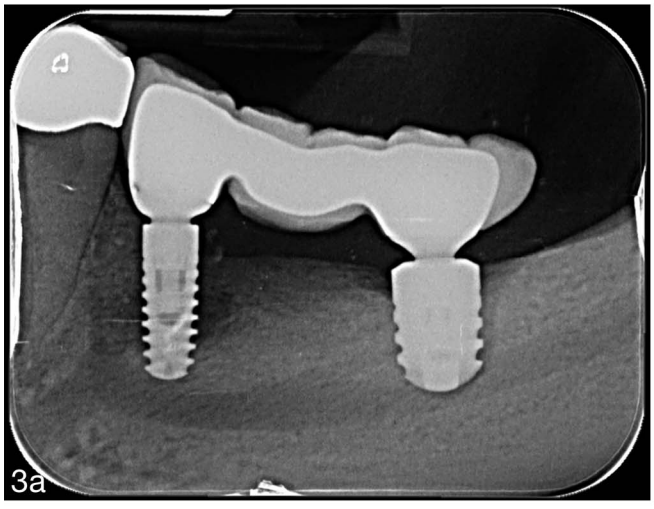
Discussion

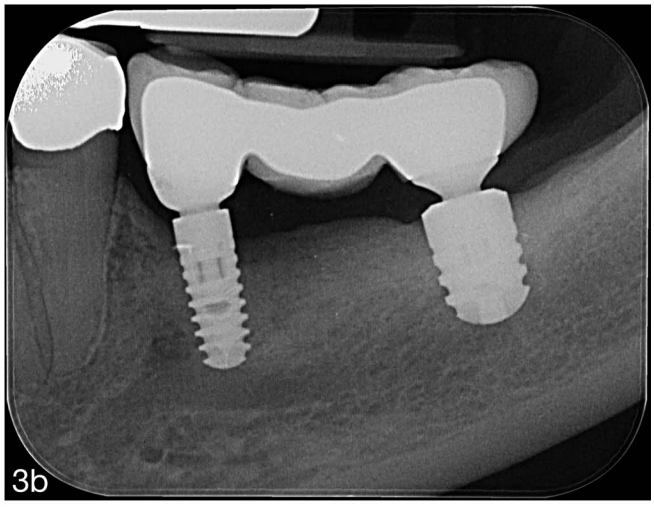
Nonsurgical therapy for peri-implantitis was shown to reduce BOP at the implant site to values of 19% to 84%.[20] Moreover, in several cases, a nonsurgical approach was sufficient for successful treatment of peri-implantitis.[21] The clinician may then assess the peri-implant tissue response and the patient's oral hygiene results.[10] Nonetheless, in many cases of peri-implantitis, nonsurgical therapy is not sufficient.[22] Most cases require surgical therapy.[10]

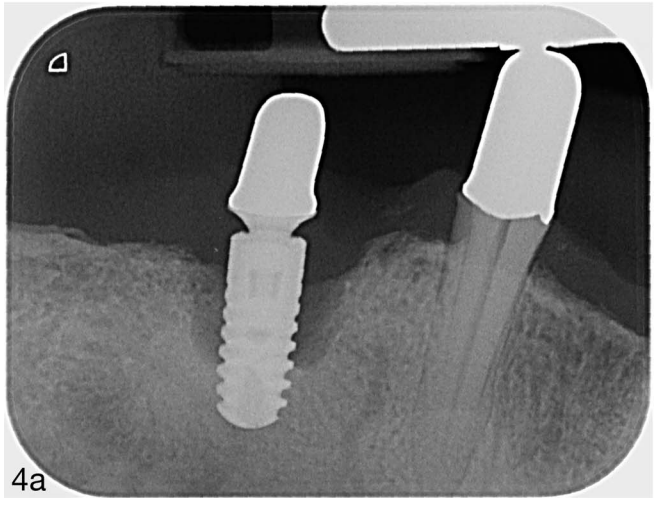
Currently, surgical approaches for the treatment of peri-implantitis comprise nearly exclusively hard tissue manipulation procedures. These approaches aim to reduce PD and stabilize the peri-implant bone level.[11] A systematic review and meta-analysis reporting regenerative treatment approaches included 5 case series and 1 controlled trial. After a minimum follow-up of 36 months, a meta-analysis of the amount of RBF revealed a mean of 2.41 mm (range, 0.66–3.50 mm), with a 95% confidence interval of 1.46 mm to 2.87 mm and an upper limit of 4.28 mm. A mean PD reduction of 3.06 mm was found.[23] A second

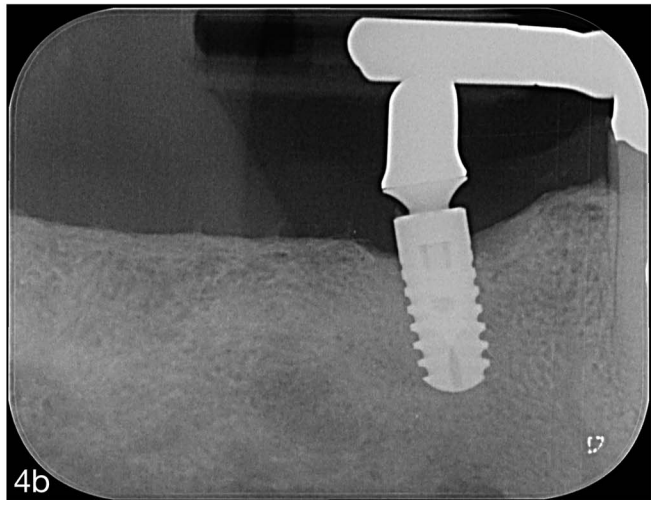




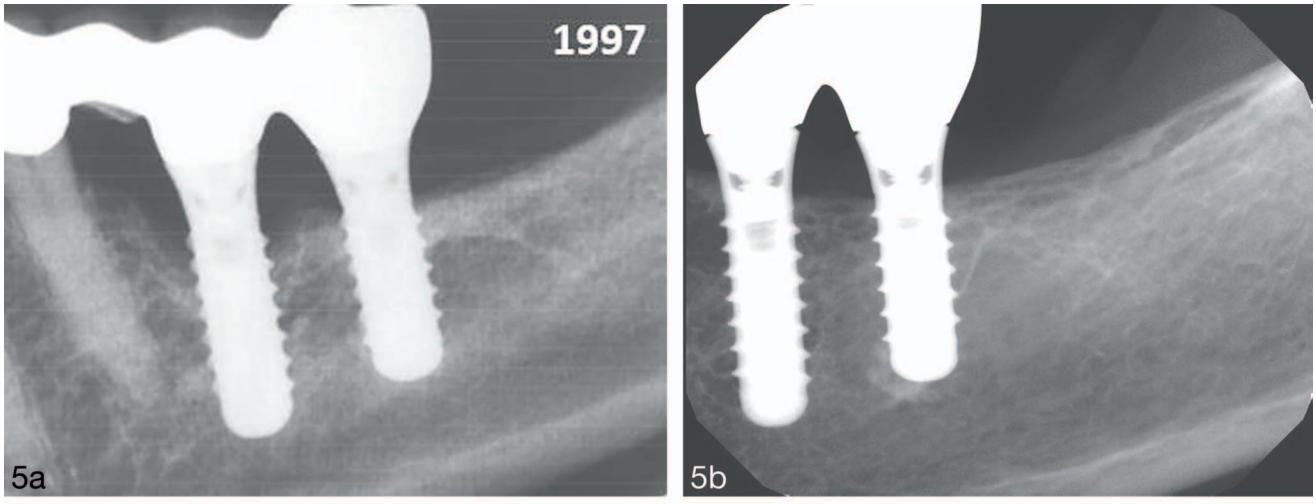


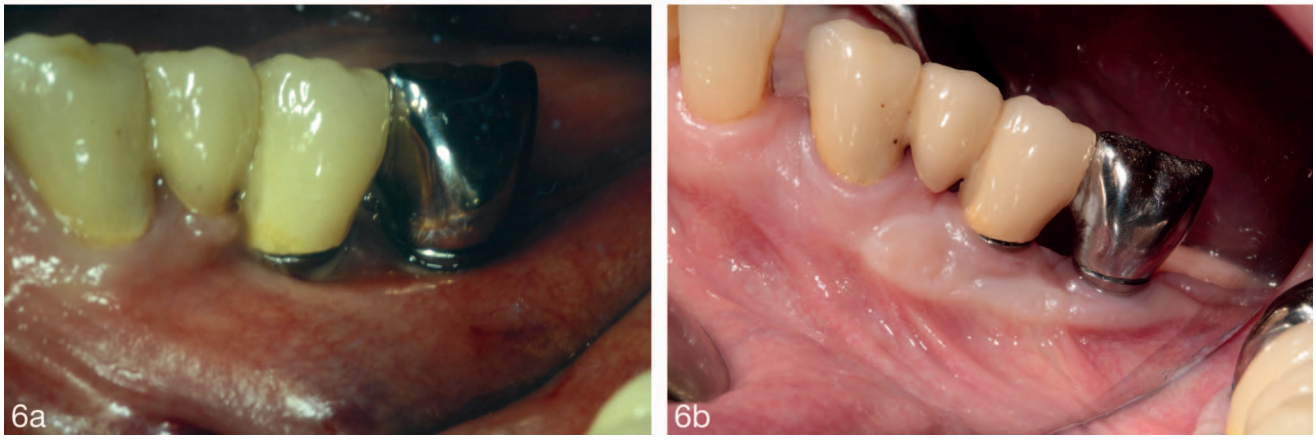






Figures 2–4. Figure 2. Patient No. 1: female, age 66 years (at the time of keratinized mucosa [KM] surgery). KM augmentation surgery: free gingival graft; implant system: AstraTech TX (L, 8 mm; D, 4 mm); inserted September 2012; implant position: 036; affected region: distal; observation period: 1.17 years. **Figure 3.** Patient No. 2: female, age 44 years (at the time of KM surgery). KM augmentation surgery: free gingival graft; implant system: Ankylos (L, 11 mm; D, 3.5 mm and L, 8 mm; D, 5.5 mm); inserted April 2012; implant position: 035 and 037; affected regions: 035 distal and 037 mesial; observation period: 3.08 years. **Figure 4.** Patient No. 3: male, age 67 years (at the time of KM surgery). KM augmentation surgery: partially epithelialized connective tissue graft; implant system: Ankylos (L, 11 mm; D, 4.5 mm); inserted June 2003; implant position: 045; affected regions: mesial + distal; observation period: 5.01 years.





Figures 5 and 6. Figure 5. Patient No. 4: female, age 66 years (at the time of keratinized mucosa [KM] surgery). KM augmentation surgery: free gingival graft [FGG]; implant system: ITI Bonefit (L, 12 mm; D, 4.1 mm); inserted October 1991; implant position: 036; affected regions: mesial + distal; observation period: 18.41 years. **Figure 6.** Patient No. 4. Implants 36 and 37 before KM augmentation surgery via FGG (a) and a 5-year control (b).

systematic review verified these values. That review comprised 18 articles and found a mean defect fill of 1.97 mm and a mean PD reduction of 2.78 mm.[24] Based on the current available literature, RBF values of ~2–2.5 mm with an upper limit of ~4.5 mm can be expected after regenerative bone surgery for the treatment of peri-implantitis. Considering this estimate, the RBF values of the 4 presented KM augmentation cases, namely, 2–5.5 mm, seem comparable to these results, even though no hard tissue surgery was performed. The biological principles underlying this extensive RBF remain unclear at present.

Table 1

Final assessment of the peri-implant soft tissue status*

Patient	Localization	Mean PD	Deepest PD	KMW	BOP	Suppuration
1	36 d	3.5	5	6	1	no
2	35 d	3.33	5	5	1	no
2	37 mo	3	4	2	0	no
3	45 mo	3.83	5	4	0	no
3	45 d	3.83	5		0	no
4	46 mo	3	3	4	0	no
4	46 d	3	3		0	no
Range		3–3.83	3–5	2–6		
Mean		3.36	4.29	0.4		
Median		3.33	5	4		

*PD, probing depth; KMW, keratinized mucosa; BOP, bleeding on probing.

Table 2

Changes in radiologic bone level measurements from baseline to the last examination

Patient	Localization	UBL Changes, mm	DBL Changes, mm	RBF %	Period Year
1	36 d	0	3	100	1.17
2	35 d	−0.5	3	60	3.08
2	37 mo	1.5	1.5	75	
3	45 mo	0	5	100	5.01
3	45 d	0.5	5.5	100	
4	46 mo	1.5	4.5	100	18.41
4	46 d	3	4.5	100	
Range		−0.5 to 3	1.5 to 5.5	60 to 100	
Mean		0.86	3.86	90.71	
Median		0.5	4.5	100	

*UBL, upper bone contour line; DBL, deepest bone resorption line; RBF, radiographic bone fill.

In patients with a thin peri-implant mucosa, increasing the KM width before performing bone regenerative surgery was proposed.[10] Currently, data concerning the potential effects of KM augmentation on peri-implant diseases are rare. A recent review comprised 4 studies on KM widening and 6 studies on thickening of the peri-implant tissue layer,[2] but no study reported on the treatment of peri-implantitis via KM augmentation surgery.

More than 25 years ago, we hypothesized that peri-implant soft tissue quality (as indicated by the mucosal thickness, attachment to the underlying periosteum, and the presence of an adequate KM width) might have a serious impact on peri-implant health. Therefore, in patients diagnosed with peri-implantitis and inadequate tissues, KM augmentation surgery was performed. Later, some of these cases showed an unexpected amount of peri-implant RBF, as described already. One explanation may be that the reconstruction of an adequate peri-implant soft tissue architecture might contribute considerably to re-establishing the equilibrium between bacterial challenge and the host response.

Because this report is based on only 4 clinical cases, no concrete conclusions should be drawn. However, this paper indicates that after the treatment of peri-implantitis via KM augmentation and a subsequent SIT program complete remission from peri-implantitis might be achieved for up to 18 years and significant peri-implant RBF might be achieved.

Therefore, biofilm control and subsequent KM surgery should be discussed as an additional surgical approach for treating peri-implantitis in patients with insufficient peri-implant soft tissue architecture. To the best of our knowledge, this phenomenon has not previously been described. The biological mechanisms underlying this recovery of peri-implant hard tissue remain unclear.

Abbreviations

APF: apical-positioned flap
BOP: bleeding on probing
CM: collagen matrix
DBL: deepest bone resorption line
FGG: free gingival graft
KM: keratinized mucosa
KMW: keratinized mucosa width
MP: measurement point
PD: probing depth
PECTG: partially epithelialized connective tissue graft
RBF: radiographic bone fill
SCTG: subepithelial connective tissue graft
SIT: supportive implant therapy
UBL: upper bone contour line

Note

The authors declare no conflicts of interest.

References

1. 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 Clin Periodontol*. 2018;45 Suppl 20:S286–S291.

2. Thoma DS, Naenni N, Figuero E, et al. Effects of soft tissue augmentation procedures on peri-implant health or disease: a systematic review and meta-analysis. *Clin Oral Implants Res*. 2018;29 Suppl 15:32–49.

3. Heitz-Mayfield LJA, Salvi GE. Peri-implant mucositis. *J Clin Periodontol*. 2018;45 Suppl 20:S237–S245.

4. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. *J Clin Periodontol*. 2018;45 Suppl 20:S246–S266.

5. Dreyer H, Grischke J, Tiede C, et al. Epidemiology and risk factors of peri-implantitis: a systematic review. *J Periodontal Res*. 2018;53:657–681.

6. Nobre MDA, Capelas C, Alves A, et al. Non-surgical treatment of peri-implant pathology. *Int J Dent Hyg*. 2006;4:84–90.

7. Persson GR, Salvi GE, Heitz-Mayfield LJ, Lang NP. Antimicrobial therapy using a local drug delivery system (Arestin) in the treatment of peri-implantitis. I: microbiological outcomes. *Clin Oral Implants Res*. 2006;17:386–393.

8. Renvert S, Lessem J, Dahlen G, Lindahl C, Svensson M. Topical minocycline microspheres versus topical chlorhexidine gel as an adjunct to mechanical debridement of incipient peri-implant infections: a randomized clinical trial. *J Clin Periodontol*. 2006;33:362–369.

9. Salvi GE, Persson GR, Heitz-Mayfield LJ, Frei M, Lang NP. Adjunctive local antibiotic therapy in the treatment of peri-implantitis II: clinical and radiographic outcomes. *Clin Oral Implants Res*. 2007;18:281–285.

10. Renvert S, Polyzois I. Treatment of pathologic peri-implant pockets. *Periodontol 2000*. 2018;76:180–190.

11. Sanz M, Chapple IL. Clinical research on peri-implant diseases: consensus report of working group 4. *J Clin Periodontol*. 2012;39 Suppl 12: 202–206.

12. Frisch E, Ratka-Kruger P, Ziebolz D. A new technique for increasing keratinized tissue around dental implants: the partially epithelialized free connective tissue graft. Retrospective analysis of a case series. *J Oral Implantol*. 2015;41:467–472.

13. Wennstrom JL, Derks J. Is there a need for keratinized mucosa around implants to maintain health and tissue stability? *Clin Oral Implants Res*. 2012;23 Suppl 6:136–146.

14. Zigdon H, Machtei EE. The dimensions of keratinized mucosa around implants affect clinical and immunological parameters. *Clin Oral Implants Res*. 2008;19:387–392.

15. Gobbato L, Avila-Ortiz G, Sohrabi K, Wang CW, Karimbux N. The effect of keratinized mucosa width on peri-implant health: a systematic review. *Int J Oral Maxillofac Implants*. 2013;28:1536–1545.

16. Lin GH, Chan HL, Wang HL. The significance of keratinized mucosa on implant health: a systematic review. *J Periodontol*. 2013;84:1755–1767.

17. Lee DW. Validated surrogate endpoints needed for peri-implantitis. *Evid Based Dent*. 2011;12:7.

18. Klinge B, Meyle J. Peri-implant tissue destruction. The third EAO consensus conference 2012. *Clin Oral Implants Res*. 2012;23 Suppl 6:108–110.

19. Mombelli A, Muller N, Cionca N. The epidemiology of peri-implantitis. *Clin Oral Implants Res*. 2012;23(Suppl 6):67–76.

20. Heitz-Mayfield LJ, Mombelli A. The therapy of peri-implantitis: a systematic review. *Int J Oral Maxillofac Implants*. 2014;29(Suppl):325–345.

21. Renvert S, Lindahl C, Jansaker AMR, Persson GR. Treatment of peri-implantitis using an Er:YAG laser or an air-abrasive device: a randomized clinical trial. *J Clin Periodontol*. 2011;38:65–73.

22. Schwarz F, Bieling K, Bonsmann M, Latz T, Becker J. Nonsurgical treatment of moderate and advanced periimplantitis lesions: a controlled clinical study. *Clin Oral Investig*. 2006;10:279–288.

23. Khoshkam V, Del Amo FS-L, Monje A, Lin GH, Chan HL, Wang HL. Long-term radiographic and clinical outcomes of regenerative approach for treating peri-implantitis: a systematic review and meta-analysis. *Int J Oral Maxillofac Implants*. 2016;31:1303–1310.

24. Daugela P, Cicciu M, Saulacic N. Surgical regenerative treatments for peri-implantitis: meta-analysis of recent findings in a systematic literature review. *J Oral Maxillofac Res*. 2016;7:e15.