Surgical Treatment of Periimplantitis With Non–Augmentative Techniques

Philip L. Keeve, DMD, MS,*† Ki Tae Koo, DDS, MS, PhD,‡ Ausra Ramanauskaite, DDS,§ Georgios Romanos, DDS, PhD,¶ Frank Schwarz, DDS, PhD,|| Anton Sculean, DMD, MS, PhD,# and Fouad Khoury, DMD, PhD**††

Objectives: *The aim of this review was to systematically screen the literature on surgical non–regenerative treatments of periimplantitis, especially for radiologic and clinical outcomes, and to determine predictable therapeutic options for the clinical management of periimplantitis lesions.*

Material and Methods: *The potentially relevant literature was assessed independently by 2 reviewers to identify clinical studies, trials, and case series in humans describing the surgical non–regenerative treatment outcomes of periimplantitis with a follow-up of at least 6 months. MEDLINE, EMBASE, and the Cochrane Library were searched for studies reporting changes in probing depth (PD) and/or bleeding on probing (BOP) and/or radiologic marginal bone-level changes.*

Results: *A total of 10 publications were included: 6 prospective randomized controlled trials, 1 prospective cohort study, 2 retrospective controlled studies, and 1 case series. Clinical parameters can be reduced by surgical non–regenerative treatments. Concerning 3 year follow-ups, BOP and PD values decreased more efficiently after implantoplasty than using systematic administration of antibacterials. Adjunctive local chemical irrigations or diode laser have no long-term effects. The non–regenerative surgical approach in combination with implantoplasty also shows improved radiographic parameters.*

Conclusions: *Surgical non–regenerative treatment of periimplantitis can reduce the amount of inflammation in the short-term follow-up. Using implantoplasty may result in the improvement of clinical and radiographic parameters. Because of limited evidence and heterogeneity in study design, there is a need for randomized controlled studies with proper design and powerful sample size in the future. (Implant Dent 2018;28:1–10)*

Key Words: periimplant, dental implants, periodontitis, periimplant disease, CIST, resective, non–augmentative, therapy, implantoplasty

Periimplant diseases are defined as "collective term for inflammatory reactions in the tissues surrounding the implants,"[1] whereas periimplantitis was introduced as an inflammatory process on hard and soft tissue, resulting in pathological pocket formation and loss of supporting bone.[2]

The wide range in prevalence rates of 2.7% to 47.1% of implants[1,3,4] can be attributed to differences in the study population, disease dentition, and implant micro- and macrostructures. Therefore, an effective strategy for treating this disease is required, otherwise a debilitating condition around the affected implants will result in loss of function and esthetics.

The development of an adherent biofilm on the implant surface plays an important role in the etiology of periimplantitis.[2] As a result of this multifactorial, but significant role of bacteria in the initiation and progress of infection of periimplant diseases, elimination of the established biofilm from the implant surface is the main objective in the treatment of periimplant mucositis and periimplantitis.[5]

Several clinical protocols for the treatment of periimplantitis have been proposed, including mechanical debridement, the use of antiseptics and local or systemic antibiotics,[6,7] surgical access,[8–10] and regenerative[11–14] or resective surgical procedures.[15–18]

The aim of this review is to systematically screen the literature on surgical non–regenerative treatments of periimplantitis, especially for radiologic and clinical outcomes, and to determine predictable therapeutic

*Private Practice, Hameln, Germany.
†Head, Department of Periodontology, Private Dental Clinic Schloss Schellenstein, Olsberg, Germany.
‡Professor, Department of Periodontology, Dental Research Institute, Seoul National University School of Dentistry, Seoul, Republic of Korea.
§Research Associate, Department of Oral Surgery, Universitätsklinikum Düsseldorf, Düsseldorf, Germany.
¶Professor, Department of Periodontology, School of Dental Medicine, Stony Brook University, Stony Brook, NY.
||Professor, Chairman, Department of Oral Surgery and Implantology, Carolinum, Johann Wolfgang Goethe-University Frankfurt, Frankfurt, Germany.
#Professor, Chairman, Department of Periodontology, School of Dental Medicine, University of Bern, Bern, Switzerland.
**Director, Private Dental Clinic Schloss Schellenstein, Olsberg, Germany.
††Professor, Department of Oral and Maxillo-Facial-Surgery, University Muenster, Muenster, Germany.

Reprint requests and correspondence to: Philip L. Keeve, DMD, MS, Suentelstrasse 10-12, 31785 Hameln, Germany, Phone: +49515195760, Fax: +495151957611, E-mail: pl.keeve@outlook.com

ISSN 1056-6163/18/02801-001
Implant Dentistry
Volume 28 • Number 1

DOI: 10.1097/ID.0000000000000838

options for the clinical management of periimplantitis lesions.

MATERIALS AND METHODS

A literature research was performed in MEDLINE via the PubMed database of the US National Library of Medicine, Ovid MEDLINE, EMBASE, and Dentistry and Oral Sciences Source for articles published between January 2005 and January 2018 using Medical Subject Heading search terms "periimplantitis" OR "peri-implantitis" OR "periimplant" OR "peri-implant" or "implant" AND "failure" AND "surgical" OR "treatment" OR "therapy" OR "non-regenerative" OR "nonregenerative" OR resective OR "laser" OR "lasers" OR "implantoplasty" OR "osteoplasty" OR "flap debridement" + free text terms, and in different combinations. To be included in the study, studies were screened by 2 independent reviewers and had to be

(i) written in the English language;
(ii) published in an international peer-reviewed journal;
(iii) clinical studies or clinical trials in humans;
(iv) prospective or retrospective studies;
(v) case series.

Case reports, letters, editorials, and literature reviews were excluded.

According to disease definition, periimplantitis was defined as a clear radiographic threshold of more than 2 mm of marginal bone loss beyond biological periimplant bone remodeling, presence of bleeding on probing (BOP), and/or suppuration.[19]

Finally, the matching full-text articles were included if they fulfilled the following criteria:

- surgical non–regenerative/resective treatment in patients with at least 1 osseointegrated screw-shaped dental implant affected by "periimplantitis" mentioned above
- minimal sample size of 10 implants
- minimum 6 months of follow-up period
- description of at least 1 surgical non–regenerative treatment method of periimplantitis
- Report on clinical and radiographic periimplant tissue changes, including probing depth (PD) and/or BOP as the primary outcome measure and/or radiographic bone-level (RBL) change as the secondary outcome measure

The exclusion criteria were defined as follows:

- *In vitro* and *in vivo* studies
- Studies with inclusion of patients with severe systemic diseases and uncontrolled metabolic disorder or osteoporosis
- Studies concerning ceramic or coated implant surfaces
- Insufficient information for the review, including unavailability of authors

Risk of Bias Assessment

The following criteria were used according to the randomized clinical trial checklist of the Cochrane Center[20] and the CONSORT statement.[21] The degrees of bias were categorized as follows: low risk, if all the criteria were met; moderate risk, if 1 criterion was missing; and high risk, if 2 or more criteria were missing.

Data Extraction and Method of Analysis

All data from the eligible studies were extracted with a data extraction template. For data analysis, author and year of publication, type of study, sample size, antimicrobial adjunctive agents, detoxification methods, as well as PD change, BOP change, and RBL change before treatment and after respective healing periods including the duration of follow-up were extracted (Table 1).

RESULTS

After initial screening of 863 potentially relevant publications because of data extraction and analysis described above, 724 articles were excluded and 139 studies were included, aiming on non–regenerative surgical treatments of periimplantitis. Among these 139 studies, furthermore 129 were excluded because of lack of information regarding the topic, the criteria for "diagnosis periimplantitis" did not match, or the follow-up period was too short. Finally, 10 articles were included in this review. Six prospective randomized controlled trials,[9,10,16–18,22] 1 prospective cohort study,[8] 2 retrospective controlled studies,[15,23] and 1 case series[24] were designed with follow-up periods of 6 months,[9,24] 1 year,[8,10,15,22,23] and 3 years.[16–18] The risk of bias across studies was classified as unclear risk in 5 cases (1 or more criteria missing),[8,9,17,18,22] as moderate risk in 1 case,[10] and as high risk in 4 studies (2 or more criteria missing).[15,16,23,24]

Clinical periimplant parameters as PD and BOP improved after access apical surgery only,[8,15–17] surface decontamination with chlorhexidine (CHX), and/or cetylpyridinium chloride (CPC)[15,23] and systemic antibiotics.[8,18,22,24] One study showed no clinical benefits using adjunctive systemic azithromycin in combination with open flap debridement[10]; another study stated no potential benefits of systemic antibiotics over 3 years.[18] The clinical parameters improved significantly, especially in studies performing implantoplasty[17] or use of systemic antimicrobials.[8,22] In detail, implantoplasty (diamond/Arkansas burs + silicone polishers) as an adjunct to open flap debridement with bone recontouring and apical flap repositioning resulted in better BOP and PD scores, but higher mean mucosal recessions compared with the control group (PD: 1.64 ± 1.29 vs 2.3 ± 1.45 mm), where persistent active signs of periimplant inflammation recurred in all patients after 24 months. Adjunctive benefits, derived from the addition of resective surgical treatment consisting of apically repositioned flap, bone recontouring, and surface debridement with 0.12% CHX + 0.05% CPC to a placebo solution (without CHX/CPC),[15,23] tend to show greater immediate suppression of anaerobic bacteria on the implant surface than a placebo solution, but do not lead to superior clinical results or differences in mean marginal bone loss at 12 months of follow-up. Similarly, the adjunctive use of 980-nm diode laser to mechanical open flap debridement[9] failed to reveal any significant clinical improvements in mean BOP and PD scores at the 6-month follow-up (Figs. 1–5).

Stable radiographic periimplant bone levels were observed after

Table 1. Descriptive Analysis of Included Studies With Study and Patient/Implant Characteristics, Treatment Protocols, Clinical and Radiographic Outcomes, as Well as Complications

Study	Year	Type of Study	Sample Size	Follow-up (m)	Implant Surface	Periimplantitis Definition	Treatment Method Used	Decontamination Method	Outcomes Evaluated	Mean PD Changes (SD)	Mean BOP Changes Mean (SD)	Radiographic Marginal Bone-Level Changes
Heitz-Mayfield et al[8]	2012	Prospective cohort study	24 patients; mean age 56 years	12	36 implants, rough	BOP and/or pus on probing + PD ≥5 mm and bone loss ≥2 mm	Open flap debridement and implant surface decontamination with saline and with adjunctive systemic amoxicillin and metronidazole	Sterile saline	Clinical: PD, recessions, BOP, and pus; radiographic: MBL changes	Baseline: mean PD ≥ 6 mm: 20%; 5 ≤ PD < 6 mm: 25%; 4 ≤ PD < 5 mm: 28%; < 4 mm: 7%. After 12 mo: ≥ 6 mm: 0%; 5 ≤ PD < 6 mm: 0%; 4 ≤ PD < 5 mm: 11%; < 4 mm: 89%. Statistically significant (P < 0.01) reduction in mean PD.	Number of sites with BOP: baseline: 2.5 (1); after 12 mo: 1 (1.2) Statistically signicant (P < 0.01) reduction in BOP.	Three implants in 3 patients had 0.6–1 mm bone loss at 12 mo. Three implants in 3 patients showed bone gain, whereas the remaining implants had stable marginal bone levels.
Papadopoulos et al[9]	2015	Randomized controlled clinical study	16: age 55 years. (40–73) group 1: 8, group 2: 8	6	Not known	BOP and/or pus on probing + PD ≥ 6 mm and bone loss ≥2 mm	Group 1: open flap debridement alone.	Group 1: use of cotton swabs soaked in saline solution	Clinical: PD, recessions, BOP, pus, and plaque index; radiographic: MBL changes	Group 1: baseline mean PD 5.92 mm; after 6 mo 4.44 mm; reduction of 1.38 mm.	Group 1: baseline 93.5%; after 6 mo 31.3%; mean reduction 72.9%.	
							Group 2: open flap debridement with the additional use of diode laser	Group 2: saline solution + diode laser for 2 min, 532 nm;		Group 2: baseline mean PD 5.52 mm; after 6 mo 4.31 mm; reduction of 1.19 mm. No statistically significant difference between the 2 groups.	Group 2: baseline 81.2%; after 6 mo 23.8%; mean reduction 66.7% (P < 0.05) No statistically significant difference between groups	
de Waal et al[15]	2013	Retrospective clinical study	30: group 1: 15, age 61.5 years; group 2: 15, age 59.4 years	12	79 implants, rough	BOP and/or pus on probing + PD ≥5 mm and bone loss ≥2 mm	Resective surgery with bone recontouring and surface decontamination.	Test: 0.12% CHX+0.05% cetylpyridinium chloride (CPC); control: no CHX/CPC	Clinical: BOP, PD, and pus; radiographic: MBL loss after 1 y and comparison between the groups	Mean PD ≥ 5 mm: group 1: baseline 88.2 (18.4)%; after 12 mo 733.9 (39)%.	Group 1: baseline 87.1 (27)%; after 12 mo 25.8 (8)%.	Group 1: baseline 4.3 (2.1) mm; after 12 mo 5 (2.5) mm. Group 2: baseline 3.61 (1.9) mm; after 12 mo 3.9 (2) mm.
							Group 1: 0.12% CHX + 0.05% CPC.			Group 2: baseline 75.2 (26.1)%; after 12 mo 17.1 (24)%	Group 2: baseline 81.3 (39)%; after 12 mo 15.8 (6)%.	

(continued on next page)

Table 1. (Continued)

Study	Year	Type of Study	Sample Size	Follow-up (m)	Implant Surface	Periimplantitis Definition	Treatment Method Used	Decontamination Method	Outcomes Evaluated	Mean PD Changes (SD)	Mean BOP Changes Mean (SD)	Radiographic Marginal Bone-Level Changes
							Group 2: placebo.			Mean PD ≥ 6 mm: Group 1: baseline 54.5 (33.7)%; after 12 mo 17.7 (34.3)%. Group 2: baseline 46.9 (33.7)%; after 12 mo 17.2 (19.2)%.		
Romeo et al[17]	2005	Randomized clinical study	17: group 1: 10; group 2: 7	36	35 implants, rough	Pus or BOP, PD > 4 mm, no mobility, and radiographic horizontal bone loss	Group 1 (test): resective surgery and modification of surface topography (implantoplasty) Group 2: resective surgery only (control group)	Metronidazole + tetracycline hydrochloride (3 min)	Clinical: survival rate, PD, mBI, and mucosal recession index	Group 1: baseline 5.79 (1.69) mm; after 36 mo 3.21 (0.56) mm Group 2: baseline 6.52 (1.62) mm; after 24 mo 5.5 (1.47) mm.	Group 1: baseline 2.83 (0.47); after 36 mo 0.61 (0.67) Group 2: baseline 2.86 (0.35); after 24 mo 2.33 (0.75)	
Romeo et al[16]	2007	Randomized clinical study	19: group 1: 10; group 2: 9	36	38 implants, rough	Pus or BOP, PD > 4 mm, no mobility, radiographic horizontal bone loss	Group 1 (test): resective surgery and implantoplasty. Group 2 (control): resective surgery alone.	Metronidazole + tetracycline hydrochloride (3 min)	Radiographic: marginal bone loss			Group 1: baseline mesially 3.82 (1.52) mm, distally 3.94 (1.64) mm; after 3 y mesially 3.81 (3.94) mm, distally 1.72 (1.79) mm. Group 2: baseline mesially 3.45 (1.93) mm, distally 3.49 (1.8) mm; after 3 y mesially 5.35 (1.99) mm, distally 5.42 (1.91) mm Significantly higher ($P < 0.05$) mean MBL was recorded in group 2 than in group 1.
de Waal et al[23]	2015	Retrospective clinical study	44: group 1: 22, age 60.5 years; group 2: 22, age 58.6 years	12	108 implants, rough	BOP and/or pus on probing + PD ≥ 5 mm and bone loss ≥2 mm	Resective surgery with bone recontouring and surface decontamination.	Test: 2.0% CHX; control: 0.12% CHX+0.05% CPC	Clinical: BOP, PD, and pus; radiographic: MBL loss after 1 y and comparison between the groups	Mean PD ≥ 5 mm: Group 1: baseline 57.5 (26.6)%; after 12 mo 7.3 (12.6)%.	Group 1: baseline 82.1 (23.9)%; after 12 mo 42.7 (34.2)%.	Group 1: baseline 4 (1.5) mm; after 12 mo 4.3 (1.7) mm.

(continued on next page)

Table 1. (Continued)

Study	Year	Type of Study	Sample Size	Follow-up (m)	Implant Surface	Periimplantitis Definition	Treatment Method Used	Decontamination Method	Outcomes Evaluated	Mean PD Changes (SD)	Mean BOP Changes Mean (SD)	Radiographic Marginal Bone-Level Changes
							Group 1: 0.2% CHX.			Group 2: baseline 60.2 (28.3)%; after 12 mo 5.3 (12.5)%.	Group 2: baseline 74.2 (27.8)%; after 12 mo 37.0 (35.3)%.	Group 2: baseline 4.1 (1.6) mm; after 12 mo 4.1 (1.7) mm.
							Group 2: 0.12% CHX + 0.05% CPC.			Mean PD ≥ 6 mm: Group 1: baseline 29.1 (31.6)%; after 12 mo 2.1 (7)%.	No significant difference between the groups (P = 0.6)	Radiologic bone loss was not significantly different between the groups (P = 0.8)
										Group 2: baseline 34.4 (31.8)%; after 12 mo 1.4 (5.8)%.		
										No significant difference between the groups (P = 0.6)		
Carcuac et al[22]	2016	Randomized controlled clinical study	100/179; group 1: 27/47, nonmodified surface (N) 3, modified (M) 44; group 2: 25/46, N 12, M 34; group 3: 24/49, N 15, M 34; group 4: 24/37, N 13, M 24	12	Different surfaces included	PD ≥ 6 mm + BOP and/or pus bone loss >3 mm	Bone recountouring +: groups 1 and 2 systemic antibiotics (amoxicillin 2*750 mg, 10 d, commenced 3 d before surgery)	0.2% CHX for 2 min in group 1 and 3; saline groups in group 2 and 4	Clinical: PD, BOP, and pus; radiographic: MBL changes	Group 1: −3.03 mm; group 2: −3.44 mm; group 3: −2.16 mm; group 4: −1.69 mm; reduction in PPD occurred in all treatment groups but was significantly larger in group 2 than in groups 3 and 4.	Group 1: 18%; group 2: 16%; group 3: 20%; group 4: 18%; no significant differences between treatment groups	Group 1: 0.18 mm; group 2: 0.51 mm; group 3: −0.69 mm; group 4: −0.96 mm.
												Bone gain was observed in implants in patients of groups 1 and 2, whereas additional bone loss occurred in the other 2 groups

(continued on next page)

Table 1. (Continued)

Study	Year	Type of Study	Sample Size	Follow-up (m)	Implant Surface	Periimplantitis Definition	Treatment Method Used	Decontamination Method	Outcomes Evaluated	Mean PD Changes (SD)	Mean BOP Changes Mean (SD)	Radiographic Marginal Bone-Level Changes
												$> P < 0.05$ groups 1 and 2 vs groups 3 and 4
Carcuac et al[18]	2017	Randomized controlled clinical study	67/121 group 1: 68 implants; group 2: 53 implants; group 3: 90 implants; group 4: 31 implants	36	Different surfaces included	PD ≥ 6 mm + BOP and/or pus bone loss >3 mm	Bone recountouring +: Groups 1 and 2 systemic antibiotics (amoxicillin 2*750 mg, 10 d, commenced 3 d before surgery)	0.2% CHX for 2 min in group 1 and 3; saline groups in group 2 and 4	Clinical: PD, BOP, and pus; radiographic: MBL changes	Group 1: –3 mm; group 2: –2.38 mm; group 3: –2.67 mm; group 4: –2.9 mm; PD reduction was more pronounced at the non–modified surface implants and that adjunctive use of systemic antibiotics improved the outcome at implants with modified surfaces	BoP/SoP+ was lower for implants with non–modified surfaces, ranging from 27% to 44%, when compared with modified surface implants (70%) Systemic antibiotics had no effect in terms of BoP/SoP.	Group 1: –0.32 mm; group 2: 0.51 mm; group 3: 0.28 mm; group 4: –0.65 mm.
Hallström et al[10]	2017	Randomized controlled clinical study	Control: 16/16; test: 15/15	12	Different surfaces included, rough	BOP and/or pus on probing + PD ≥ 5 mm and bone loss ≥2 mm	Test group: open flap debridement, cleaning with sterile curettes and saline-soaked cotton gauzes, Zithromax 250 mg × 2 on the day of surgery, and 250 mg × 1 per day during 4 additional d; control group: Open flap debridement, cleaning with sterile curettes and saline-soaked cotton gauzes	saline-soaked cotton gauzes	Clinical: PD, BOP, and pus; radiographic: MBL changes; microbial samples	Successful clinical outcome: PPD ≤ 5 mm, no suppuration, no BOP at the implant sites, and bone loss ≤0.5 mm. Based on the both treatment groups, 11/31 individuals (35.5%) presented with a successful treatment outcome.	Statistical analysis failed to demonstrate differences in BOP scores.	Mean gain of alveolar bone at implants assessed from radiographs was also significant (mean diff: 0.4 mm, *SE* mean diff: 0.2 mm, 95% CI: 0.0, 0.8, $P <$ 0.05)

(continued on next page)

Table 1. (Continued)

Study	Year	Type of Study	Sample Size	Follow-up (m)	Implant Surface	Periimplantitis Definition	Treatment Method Used	Decontamination Method	Outcomes Evaluated	Mean PD Changes (SD)	Mean BOP Changes Mean (SD)	Radiographic Marginal Bone-Level Changes
Koldsland et al[24]	2017	Case series	45 patients	6	143 implants, rough	Bone loss ≥ 2 mm and BoP/ suppuration	Open flap debridement, cleaning with titanium curettes, rubbing of the implant surface with surgical gauze soaked in 3% H_2O_2 systemic antibiotics, amoxicillin (500 mg ×3) + metronidazole (500 mg ×3), for 10 d starting the day before surgery	3% H_2O_2	Clinical: PD, BOP, and pus; radiographic: MBL changes	BSL PPD > 12 mm: mean 5.3 mm postsurgical; BSL PPD 10–11 mm: mean 5.0 mm postsurgical; BSL PPD 8–9 mm: mean 4.3 mm postsurgical; BSL PPD 6–7 mm: mean 4.0 mm postsurgical; BSL PPD 4–5 mm: mean 3.4 mm postsurgical;	BSL PPD > 12 mm: 24% no BOP postsurgical; BSL PPD 10–11 mm: 19% no BOP postsurgical; BSL PPD 8–9 mm: 29% no BOP postsurgical; BSL PPD 6–7 mm: 40% no BOP postsurgical; BSL PPD 4–5 mm: 48% no BOP postsurgical;	

systemic application of antibiotics[8,10,22] or local use of chemical compounds.[15,23] In studies with significant improvement of clinical periimplant parameters after implantoplasty, RBLs and marginal bone loss were significantly lower after 3 years of follow-up,[16] with interproximal bone loss at control sites up to 1.45 to 1.54 mm.

A total number of 35 implants were removed after non–augmentative surgery and progressive recurrence of periimplant inflammation.[15–18,22] Because of implant neck fracture, 1 implant was removed.[23]

Four studies reported on the smoking status of the patients, ranging from 25%[8] to 59.1%[23] Despite 2 studies[8,10] reporting no negative effect of smoking on treatment outcome, smoking was influencing treatment success when adjusted for baseline and follow-up.[15,23]

DISCUSSION

Surgical techniques are used depending on multiple factors including patient general health condition, oral hygiene, type of bony defects, implant surface, postoperative maintenance program, and other factors that cannot be completely assumed in a systematic literature review. The non–augmentative treatment concept is indicated for supracrestal bone defects (horizontal bone loss) with exposed threads in esthetically non–demanding areas based on patient needs and satisfaction and involves reduction or elimination of pathological periimplant pockets, the apical positioning of a mucosal flap, or recontouring bone with or without implant surface modification, called implantoplasty. In case of modifications of implant surfaces, the rough design should be removed and polished. However, a concern of remaining titanium particles should be addressed.

Even in cases of periimplant mucositis, it can be advisable to perform an access flap for proper mechanical and chemical decontamination (ie, additional removal of cement remnants) of the implant surface.

The resolution of infection can be achieved by a proper method of implant surface decontamination. Clinical studies comparing different decontamination

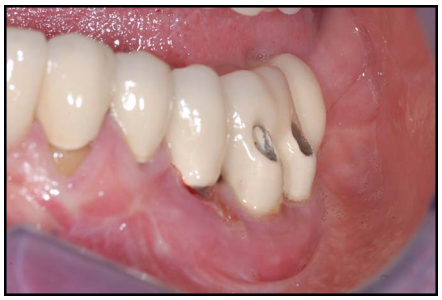

Fig. 1. Clinical situation (baseline) of a 53-year-old patient suffering from periimplantitis in the left lower jaw, with massive soft-tissue swelling, redness, ulceration, and BOP. Implants were inserted 14 years ago, but there was no adherence to a supportive periodontal treatment for the past 6 years. Nevertheless, his oral hygiene was acceptable and implants were placed too buccally, but surrounded by adequate keratinized mucosa.

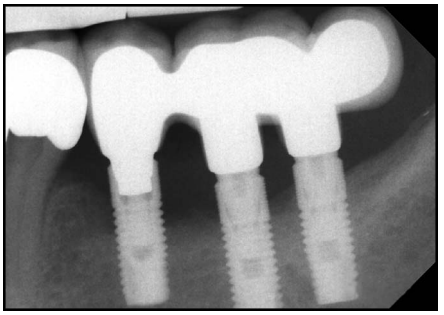

Fig. 3. Baseline radiographs showing horizontal bone loss with a supracrestal defect height of 3 to 5 mm. In addition, vertical defects could be detected around the implants 35 and 36 with a depth of 3 mm. The combination of the clinical and radiographic diagnostics stated a clear indication for surgical, non–augmentative treatment of the periimplantitis.

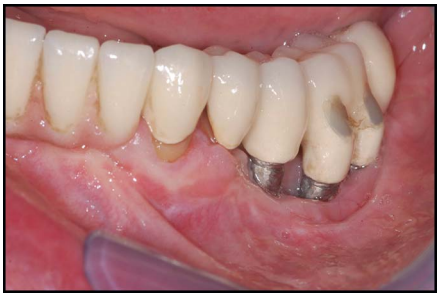

Fig. 5. After a 3-year-follow-up, no signs of inflammation, proper oral hygiene, and sufficient keratinized mucosa were present. Implant recessions of 2 to 3 mm resulted from the non–augmentative surgical periimplantitis. Supportive periodontal treatment took place every 3 months after the diagnosis of periimplantitis.

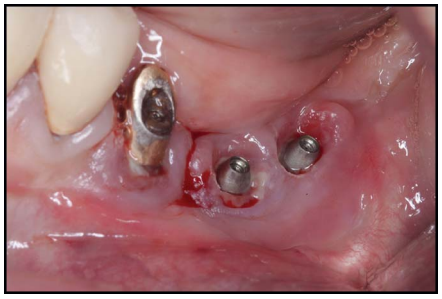

Fig. 2. Eight weeks after nonsurgical treatment with mechanical (plastic ultrasonic device) and chemical debridement (hydrogen peroxide 3% and chlorhexidine digluconate solution 0.2%), as well as adjunctive local antibiotics (tetracycline derivate), the clinical situation showed furthermore signs of massive inflammation. For a proper surgical treatment, including implantoplasty, it was absolutely necessary to remove the suprastructure, especially in the lower jaw.

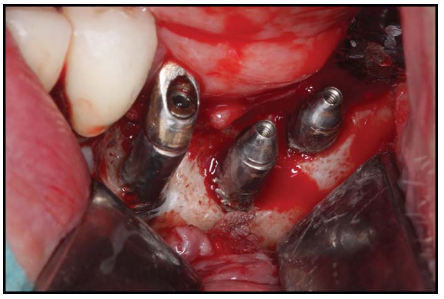

Fig. 4. A surgical, non–augmentative treatment of periimplantitis was performed, including an open flap debridement, after implantoplasty of infected and exposed implant surfaces, chemical surface decontamination and disinfection of the implants, and minimally invasive osteoplasty concerning only small intrabony defects. After minimal-invasive access flap, implant surfaces were treated with diamond burs and finally polished with greenies and supergreenies. Internal gingivectomy was completed before minimal-invasive osteoplasty and apical reposition flap with periosteal sutures.

methods or placebo failed to reveal significant differences between different, locally applied decontamination methods on clinical treatment outcomes.[15,23] Nevertheless, a combined mechanical and chemical removal of biofilm is further recommended.[5] Furthermore, adjunctive systemic antibiotics had no impact on the treatment's success for implants with a machined surface, and a positive effect was observed for the treatment success of implants with rough surfaces only in the first year.[22] Local antibiotic delivery in addition to mechanical debridement and irrigation with an antimicrobial agent may be an effective option for treating periimplantitis lesions.[6,7] Moreover, the development of bacterial resistance seems to be an unlikely even in the event of repeated applications.[25]

Resective periimplantitis therapy combined with implantoplasty leads to superior clinical and radiographic treatment outcomes compared with resective treatment alone. In particular, implantoplasty, which can be performed with diamond or carbide burs and metal polishing instruments with irrigation, positively influenced implant survival rates (100% test, 87.5% control group), significantly reduced periimplant pocket depths, and reduced bleeding scores.[17] Implantoplasty was associated with stabile interproximal bone levels at the 3-year follow-up, although significantly radiographic bone loss at control sites was detected.[16] However, the gingival recession index was significantly higher when implant surface modification was performed.[17] Therefore, this type of surgical technique can be of benefit primarily in the non–esthetic areas, and alternative treatments should be developed for the esthetic zone.[5]

The resective therapy outcomes were influenced by the experience of the surgical team, the amount of periimplant bone loss, maximum probing pocket depth at baseline, as well as a patient's smoking habits, and the presence of plaque during the follow-up.[23]

Analysis of studies and achieved reliability of treatments that revealed similarities in treatment approaches between protocols included (1) pretreatment phase, (2) cause-related therapy, and (3) maintenance phase. Oral hygiene instructions and its importance must be stressed to patients before and after the treatment.[26] Nonsurgical subgingival mechanical debridement in conjunction with local antibacterials, such as chlorhexidine digluconate or locally delivered antibiotics, is effective in reducing soft-tissue inflammation[7,27] and should be the first step in successful

treatment.[26] The supportive treatment phase is mandatory for the success of periimplantitis treatment.[26] In a 5-year follow-up observational study, healthy periimplant tissue conditions in patients with high oral hygiene standard (maintenance every 6 months) could be maintained for most patients after resective periimplant surgery.[28] Further disease progression occurred only in 10% of the treated implants, revealing that risk factors including the presence of residual pockets after surgery, smoking, poor oral hygiene, untreated periodontal disease, and diabetes may modify both the initial and long-term outcome of the treatment.[26]

All studies included implants with both cemented or screw-retained suprastructures. Hence, some surgeries were performed with suprastructures in place and some after removal of the suprastructures. All clinical assessments were performed with suprastructures in place. The suprastructure probably might affect quality of implant decontamination and modification and flap design and possibly the measurement of clinical parameters. If possible, it is strongly recommended to remove the suprastructure during surgical intervention and might be adapted for better cleansing ability.

Limitations of the revised studies are relatively small sample sizes and short follow-up periods. Furthermore, there were inconsistencies in methodology with various treatment modalities, limited control groups, different implant systems, and follow-up results from the same research groups. Unclear or high risk of bias of included studies may have an effect on the conclusions.[20] The evidence of the review is limited due to significant variations observed in the included studies. Therefore, there is a substantial need for randomized controlled studies with a proper design and powerful sample size to provide strong and comparable evidence of benefits of non–augmentative procedures for treating periimplantitis.

Conclusions

The present systematic review showed that surgical non–regenerative modalities treating periimplantitis can reduce the amount of inflammation in the short-term follow-up, but seem less effective in the long-term perspective. Using implantoplasty in surgical non–regenerative treatment leads to a significant decrease in BOP and PD and may result in improvement of clinical and radiographic parameters up to 3 years after surgery compared with mechanical debridement alone. Application of systemic antibiotics, chemical compounds, or diode laser did not result in significant clinical or radiographic long-term improvements.

Disclosure

The authors claim to have no financial interest, either directly or indirectly, in the products or information listed in the manuscript.

Roles/Contributions by Authors

P. L. Keeve: main author, corresponding author, and involved in writing/editing the manuscript. K. T. Koo: input to discussion. A. Ramanauskaite: input to material & methods and editing the manuscript. G. Romanos: input to discussion. F. Schwarz: proofreading and revision of the manuscript. A. Sculean: input to introduction and results. F. Khoury: main proofreading and leading input to the manuscript.

References

1. Zitzmann NU, Berglundh T. Definition and prevalence of peri-implant diseases. *J Clin Periodontol.* 2008;35:286–291.

2. Lang NP, Berglundh T. Periimplant diseases: Where are we now?—Consensus of the seventh European Workshop on Periodontology. *J Clin Periodontol.* 2011;38: 178–181.

3. Mir-Mari J, Mir-Orfila P, Figueiredo R, et al. Prevalence of peri-implant diseases. A cross-sectional study based on a private practice environment. *J Clin Periodontol.* 2012;39:490–494.

4. Koldsland OC, Scheie AA, Aass AM. Prevalence of peri-implantitis related to severity of the disease with different degrees of bone loss. *J Periodontol.* 2010; 81:231–238.

5. Renvert S, Polyzois IN. Clinical approaches to treat peri-implant mucositis and peri-implantitis. *Periodontol 2000.* 2015;68:369–404.

6. Mombelli A, Feloutzis A, Bragger U, et al. Treatment of peri-implantitis by local delivery of tetracycline. Clinical, microbiological and radiological results. *Clin Oral Implants Res.* 2001;12:287–294.

7. Renvert S, Lessem J, Dahlen G, et al. 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.

8. Heitz-Mayfield LJA, Salvi GE, Mombelli A, et al. Anti-infective surgical therapy of peri-implantitis. A 12-month prospective clinical study. *Clin Oral Implants Res.* 2012;23: 205–210.

9. Papadopoulos CA, Vouros I, Menexes G, et al. The utilization of a diode laser in the surgical treatment of peri-implantitis. A randomized clinical trial. *Clin Oral Investig.* 2015;19:1851–1860.

10. Hallstrom H, Persson GR, Lindgren S, et al. Open flap debridement of peri-implantitis with or without adjunctive systemic antibiotics: A randomized clinical trial. *J Clin Periodontol.* 2017;44: 1285–1293.

11. Jepsen K, Jepsen S, Laine ML, et al. Reconstruction of peri-implant osseous defects: A multicenter randomized trial. *J Dent Res.* 2016;95:58–66.

12. Roccuzzo M, Gaudioso L, Lungo M, et al. Surgical therapy of single periimplantitis intrabony defects, by means of deproteinized bovine bone mineral with 10% collagen. *J Clin Periodontol.* 2016; 43:311–318.

13. Schwarz F, John G, Schmucker A, et al. Combined surgical therapy of advanced peri-implantitis evaluating two methods of surface decontamination: A 7-year follow-up observation. *J Clin Periodontol.* 2017;44:337–342.

14. Khoury F, Buchmann R. Surgical therapy of peri-implant disease: A 3-year follow-up study of cases treated with 3 different techniques of bone regeneration. *J Periodontol.* 2001;72:1498–1508.

15. de Waal YC, Raghoebar GM, Huddleston Slater JJ, et al. Implant decontamination during surgical periimplantitis treatment: A randomized, double-blind, placebo-controlled trial. *J Clin Periodontol.* 2013;40:186–195.

16. Romeo E, Lops D, Chiapasco M, et al. Therapy of peri-implantitis with resective surgery. A 3-year clinical trial on rough screw-shaped oral implants. Part II: Radiographic outcome. *Clin Oral Implants Res.* 2007;18:179–187.

17. Romeo E, Ghisolfi M, Murgolo N, et al. Therapy of peri-implantitis with resective surgery. A 3-year clinical trial on rough screw-shaped oral implants. Part I: Clinical

outcome. *Clin Oral Implants Res.* 2005;16:9–18.

18. Carcuac O, Derks J, Abrahamsson I, et al. Surgical treatment of peri-implantitis: 3-year results from a randomized controlled clinical trial. *J Clin Periodontol.* 2017;44:1294–1303.

19. Clem D, Rosen P, Cochran D, et al. Peri-implant mucositis and peri-implantitis: A current understanding of their diagnoses and clinical implications. *J Periodontol.* 2013;84:436–443.

20. Higgins JP, Altman DG, Gotzsche PC, et al. The Cochrane collaboration's tool for assessing risk of bias in randomised trials. *BMJ.* 2011;343:d5928.

21. Schulz KF, Altman DG, Moher D. CONSORT 2010 statement: Updated guidelines for reporting parallel group randomised trials. *J Clin Epidemiol.* 2010;63:834–840.

22. Carcuac O, Derks J, Charalampakis G, et al. Adjunctive systemic and local antimicrobial therapy in the surgical treatment of peri-implantitis: A randomized controlled clinical trial. *J Dent Res.* 2016;95:50–57.

23. de Waal YC, Raghoebar GM, Meijer HJ, et al. Implant decontamination with 2% chlorhexidine during surgical peri-implantitis treatment: A randomized, double-blind, controlled trial. *Clin Oral Implants Res.* 2015;26:1015–1023.

24. Koldsland OC, Wohlfahrt JC, Aass AM. Surgical treatment of peri-implantitis: Prognostic indicators of short-term results. *J Clin Periodontol.* 2018;45:100–113.

25. Raghavendra M, Koregol A, Bhola S. Photodynamic therapy: A targeted therapy in periodontics. *Aust Dent J.* 2009;54(suppl 1):S102–S109.

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

27. Sahm N, Becker J, Santel T, et al. Non-surgical treatment of peri-implantitis using an air-abrasive device or mechanical debridement and local application of chlorhexidine: A prospective, randomized, controlled clinical study. *J Clin Periodontol.* 2011;38:872–878.

28. Serino G, Turri A, Lang NP. Maintenance therapy in patients following the surgical treatment of peri-implantitis: A 5-year follow-up study. *Clin Oral Implants Res.* 2015;26:950–956.