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Purpose: *As dental implants have become routine therapy, clinicians are more frequently being faced with treating peri-implantitis. To date, no single treatment protocol has been shown to be the preferred means to treat peri-implantitis. The aim of this retrospective case series is to present a novel approach utilizing porcine collagen-coated bovine bone (CBB) to treat peri-implantitis.* ***Materials and Methods:*** *Eleven patients, with no history of periodontitis, presenting with peri-implantitis around a single restored dental implant, were included in the study. At initial and follow-up examinations, bleeding on probing (BOP), probing depth (PD), and gingival margin location (GM) were recorded. Following surgical debridement of the peri-implant defect and treatment of the implant surface with a 0.12% chlorhexidine gluconate solution, bony defects were grafted with CBB. All patients had 12 months of follow-up.* ***Results:*** *Upon presentation, average PD at the deepest site (DS) was 7.6 ± 1.9 mm. At the time of surgery, excess cement was found around nine implants (81%). All patients healed uneventfully without postoperative complications. At 6 and 12 months, all implants showed favorable results with average DS PD reduction of 3.9 ± 1.5 mm and 4.1 ± 1.6 mm, respectively. All implants showed radiographic signs of bone fill, while GM showed no changes from preoperative measurements at either 6 (0.1 ± 0.5 mm) or 12 (0.0 ± 0.6 mm) months.* ***Conclusion:*** *The use of a porcine collagen-coated bovine bone graft to treat peri-implantitis represents a potentially predictable therapeutic modality. Randomized controlled trials are necessary to substantiate the treatment outcomes.*
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Keywords: *alveolar bone loss, bone regeneration, bone substitutes, dental implants, peri-implantitis*

Dental implants have become a predictable means to replace missing teeth and support a wide variety of prostheses.[1] As implant restorations have gradually become routine therapy, and given that peri-implantitis prevalence can be as high as 47% (depending on severity threshold),[2,3] clinicians are now being faced with the challenge of treating peri-implantitis with increasing frequency.[4,5] As of yet, conventional nonsurgical and regenerative surgical procedures that have been successfully used to treat attachment loss around natural teeth have had varying or minimal success around dental implants.[6–9] To date, no single treatment protocol has been shown to be the preferred means to treat peri-implantitis.[7]

Peri-implantitis occurs as an inflammatory process, infective in nature, which leads to bone loss around a dental implant and eventually leads to implant loss.[8,10] Ideally, the treatment of peri-implantitis should remove the offending etiology and regenerate supporting bone around the implant. While reosseointegration is possible,[11] the degree of reosseointegration has been shown to be unpredictable.[12,13] Furthermore, reosseointegration has been shown to depend on factors other than treatment technique, such as implant surface type.[12,14,15] Persson et al[15] reported that osseointegration was 62% greater on a sandblasted, acid-etched (SLA) surface, compared with a smooth machined surface, even though defect fill was not different between the two surfaces (76% vs 72%). Currently, the goals of any peri-implantitis treatment are surface decontamination and defect reduction, either by resective or regenerative techniques, to improve the lifetime of the diseased implant.[6,9,10,12] Since replacing missing teeth with dental implants can be a costly and lengthy process, treatment of peri-implantitis should be predictable, simple, and cost-effective.[16]

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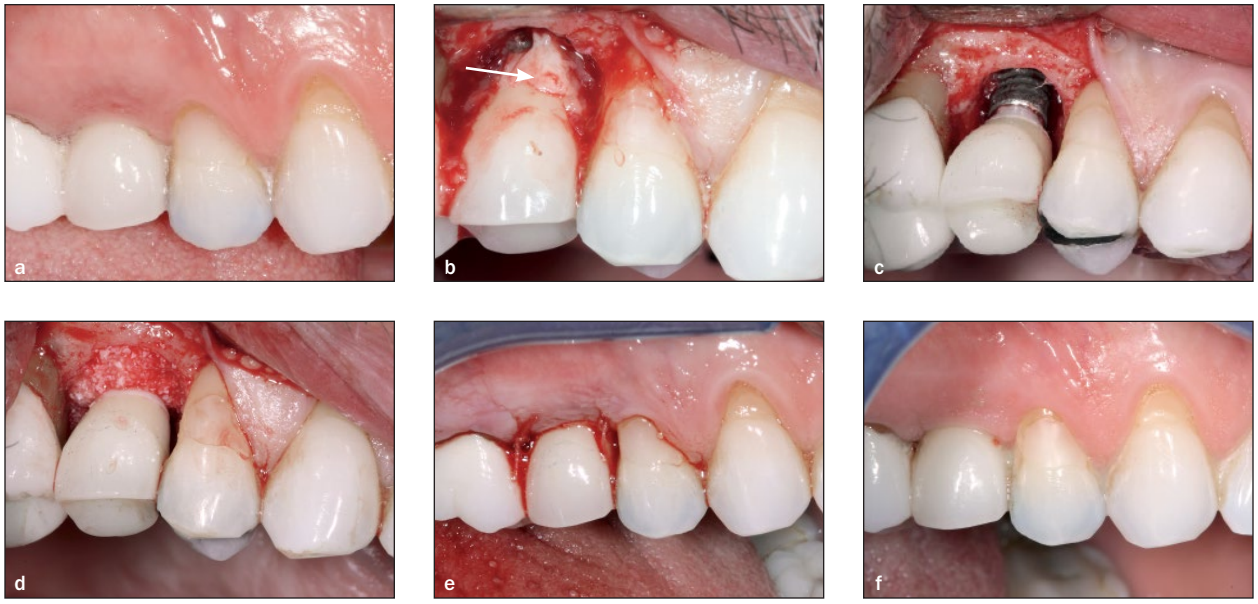

Fig 1 Clinical images of representative case (implant replacing right maxillary second premolar). *(a)* Initial presentation with evident gingival color change. *(b)* Flap elevated; arrow points to cement found. *(c)* Implant and defect debrided. *(d)* Defect grafted with collagen-coated bovine bone. *(e)* Flap sutured. *(f)* Presentation at 12 months postoperatively.

Several bone grafting materials have been used to treat peri-implant defects, including autogenous bone, demineralized freeze-dried bone allograft (DFDBA), bovine bone xenograft (Bio-Oss, Geistlich Pharma), hydroxyapatite (HA), recombinant human bone morphogenetic protein (rhBMP; INFUSE, Medtronic), and titanium particles.[11,17–22] Collagen-coated bovine bone (CBB; Bio-Oss Collagen, Geistlich Pharma) consists of 90% cancellous bovine bone particles combined with 10% porcine collagen. Studies have shown good success in postextraction healing,[23,24] ridge augmentation,[25] and in the treatment of infrabony periodontal defects[26,27] and peri-implant defects[28] using CBB. The purpose of this case series is to present a predictable surgical approach utilizing CBB to treat peri-implantitis.

MATERIALS AND METHODS

Study Design and Study Population

This private practice–based, retrospective chart review study included patients treated for peri-implantitis following a routine protocol used by the practitioner (SAR) and followed for at least 1 year postoperatively. The study protocol was approved by the Ohio State University Institutional Review Board.

Charts of patients treated for peri-implantitis between January 2010 and December 2011 around a single restored dental implant were selected based on the following criteria: nonsmoker, adult patient with no history of periodontal disease treatment or diabetes; endosseous dental implant placed to replace single missing tooth that had been restored with a cement-retained crown and in function for more than 6 months; implant was parallel walled, screw-form, with a sandblasted, large grit, acid-etched surface (SLA);[15,29] peri-implantitis diagnosis, based on the presence of radiographic bone loss in comparison to historic radiographs, ≥ 5 mm of peri-implant probing depths (PD), and presence of bleeding on probing (BOP) or suppuration; no previous treatment for peri-implantitis including nonsurgical mechanical therapy or antibiotics (systemic or locally delivered); post-treatment follow-up of 6 and 12 months.

Clinical Parameters

The recorded clinical parameters included: PD, which was measured at six sites (mesiobuccal, mesiolingual, midbuccal, midlingual, distobuccal, and distolingual) around the restored dental implant using a UNC-15 probe; BOP, which was recorded at the same six sites as PD within 15 seconds of probing, and gingival margin position (GM), which was measured (six sites per implant) as the distance from the occlusal surface (posterior teeth) or the incisal edge (anterior teeth) of the implant crown to the gingival margin. Radiographic bone fill was noted as present or absent by comparing the nonstandardized preoperative and postoperative periapical radiographs.

Surgical and Postoperative Protocols

All patients signed a consent form informing them of the current lack of evidence supporting a definitive treatment for peri-implantitis and for the use of CBB. An

Table 1 Subject Demographics, Implant Position, and Peri-implantitis Surgical Defect Description

	Age (y)	Sex	Implant position[a]	Defect type	Defect location
1	51	Male	15	Vertical 2 Wall	M + D
2	59	Male	36	Vertical 2 Wall	M + D
3	60	Male	15	Moat	M, B, D, L
4	61	Male	12	Vertical 2 Wall	D
5	62	Female	24	Vertical 2 Wall	D
6	70	Female	25	Moat	M, B, D, L
7	53	Male	25	Moat	M, B, D, L
8	61	Male	26	Moat	M, B, D, L
9	62	Female	16	Moat	M, B, D, L
10	70	Female	22	Dehiscence	B
11	62	Female	25	Vertical 2 Wall	M + D

M = mesial; B = buccal; D = distal; L = lingual.
[a]FDI tooth-numbering system.

experienced periodontist (SAR) performed all surgical procedures and clinical measurements. On the day of surgery, occlusion of the implant crown was checked for excursive contacts and for heavy contacts during maximal intercuspation using articulating paper. If excursive or heavy contacts were found, the occlusion was adjusted on the implant crown. At no time during active treatment or follow-up were the implant-supported restorations removed or replaced. None of the implants received nonsurgical treatment prior to surgery.

Following local anesthesia, intrasulcular incisions were made on the buccal and lingual of the dental implant and extended one tooth mesial and distal to the implant. A full-thickness envelope flap was created (no vertical releasing incisions), and the presence or absence of dental cement was noted (Fig 1). The implant surface and bony defect were instrumented using titanium-coated curettes.[30] A plastic-tipped ultrasonic instrument was also used on the exposed implant surface. Sterile cotton gauze soaked in a 0.12% chlorhexidine gluconate solution was then used in a scrubbing motion for 2 minutes. The action was repeated again using gauze soaked in saline.[31] CBB was hydrated with sterile saline and used to fill the bony defect and cover the exposed implant surface (Fig 1). Flaps were replaced and sutured using 4.0 polyglycolic acid sutures (Fig 1). Patients were prescribed a 0.12% chlorhexidine gluconate rinse to be used twice per day for 2 weeks following the procedure. Patients were also prescribed systemic antibiotics (500 mg amoxicillin three times a day or 300 mg clindamycin four times a day) and analgesics (hydrocodone 5 mg, acetaminophen 325 mg every 4 to 6 hours as needed) for 1 week.

Patients returned for five postoperative visits: at 2 weeks, 6 weeks, 3 months, 6 months, and 12 months. At the 6- and 12-month evaluations, PD and GM were recorded, and a periapical radiograph was obtained.

Data Analysis

Descriptive statistics are used to report the preoperative and postoperative recorded clinical parameters (PD, GM) and the observed changes between the examination time points. Data were normally distributed (Shapiro-Wilk test). Differences between preoperative and postoperative clinical measurements were analyzed by repeated measures ANOVA. Significance was set at 95% ($P \leq .050$).

RESULTS

Eleven patients (six men, five women), aged 61 ± 5.8 years (range, 51 to 70 years), were identified who met the selection criteria. The 11 treated implants had been inserted into six maxillary premolars, two maxillary lateral incisors, one mandibular first molar, and two maxillary first molar sites (Table 1). Upon initial presentation, average peri-implant PD (all six sites) was 5.0 ± 1.2 mm. The average preoperative PD at the deepest site (DS) was 7.6 ± 1.9 mm; 72.7% of DS were > 5 mm. Upon initial presentation, all implants exhibited BOP at DS, while three of the implants also presented with suppuration at DS. At the time of surgery, excess cement was found around nine implants, and signs of heavy/excursive contacts were found on two implant restorations.

All surgeries were completed uneventfully, and all patients returned for the postoperative appointments. No postoperative complications were noted. All implants retained the original restoration throughout treatment and the 12-month postoperative observation period. At 6 and 12 months, all implants showed favorable results. The average PD (six sites) was reduced to 3.5 ± 0.6 mm and 3.3 ± 0.4 mm at 6 and 12 months, respectively. The average PD at 6 and 12 months was statistically significantly different from preoperative PD ($P < .002$). The average PD reduction at 6 and 12 months was 1.5 ± 0.8 mm and 1.7 ± 1.0 mm, respectively, when compared with preoperative PD. The average DS PD at 6 and 12 months was 3.7 ± 0.8 mm and 3.5 ± 0.7 mm, respectively ($P < .001$, compared with preoperative DS PD). The mean DS PD reduction was 3.9 ± 1.5 mm and 4.1 ± 1.6 mm at 6 and 12 months, respectively. At 6 months, there were no DS PD > 5 mm, and only 18.2% (n = 2) of DS PD were > 4 mm. At 12 months, only one implant (9.1%) had DS PD > 4 mm. At 6 months, BOP was evident at DS for four


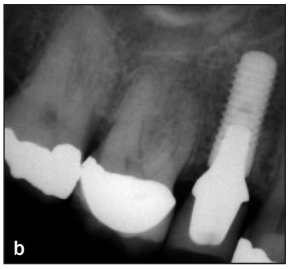

Fig 2 Radiographic images of representative case (same case as in Fig 1). *(a)* Preoperative and *(b)* 12-month postoperative presentation.

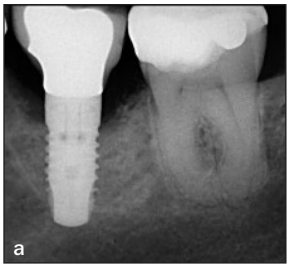
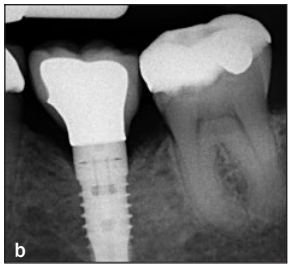

Fig 3 Radiographic images of second representative case. *(a)* Preoperative and *(b)* 12-month postoperative presentation.

implants, two of which remained BOP positive at 12 months. No implant exhibited suppuration, either at 6 or 12 months.

The mean GM (six sites) showed no significant changes at 6 and 12 months (0.1 ± 0.5 mm and 0.0 ± 0.6 mm change from preoperative measurements, respectively; $P = .23$). At 6 months, 17% of sites (in five implants) showed an increase in GM from baseline. At 12 months, 15% of sites (in five implants) showed an increase in GM compared with the preoperative measurements. The greatest GM increase was 2 mm at one site, at 12 months. When analyzing buccal sites alone, GM increased 0.1 ± 0.4 mm and 0.1 ± 0.5 mm, at 6 and 12 months, respectively ($P = .35$). The percentage of buccal sites with GM increase was 12.1% (n = 4 sites in 3 implants) and 15.1% (n = 5 sites in 4 implants) at 6 and 12 months, respectively. At midbuccal sites, 10 of the 11 implants treated (90.1%) showed no apical migration of the gingival margin compared with baseline. All implants showed radiographic signs of defect fill (Fig 2).

DISCUSSION

This case series reports on the use of CBB as a treatment for peri-implantitis around SLA implants following a protocol that excludes nonsurgical treatment or removal of the implant-supported single-tooth restoration. Data were collected up to 12 months post-treatment, a time point that is considered to represent adequate follow-up to determine treatment success of peri-implantitis.[32,33] The reported treatment approach resulted in significant PD and BOP reduction with minimal postoperative recession in implants inserted to replace single missing anterior and posterior teeth. While PD alone is not a diagnostic factor for peri-implantitis, change in PD from baseline serves as the best method to measure treatment outcomes.[34] All implants showed radiographic signs of defect fill. Since this study did not standardize the radiographic technique to ensure repeatable digital sensor positioning, the extent of radiographic defect fill (Fig 2, Fig 3) was not examined in further detail. Moreover, neither surgical reentry nor histologic sampling was performed; thus, the vital bone-to-residual graft ratio following treatment was unavailable. Previous studies on bovine bone xenograft have shown that a significant residual amount of graft particles remain in the defect site up to 24 months.[35] To date, no studies have shown a relationship between radiographic defect fill, or reosseointegration, and improvements in long-term prognosis of dental implants with peri-implantitis.[7,8,10,14]

Upon flap elevation, dental cement was observed around 9 of the 11 implants treated. Linkevicius et al reported peri-implant disease in 85% of implants with retained cement,[36] suggesting that retained cement around implant crowns does not always lead to peri-implantitis. In a scanning electron microscopy study of human biopsy specimens around failing implants, Wilson et al discovered that dental cement was a common finding.[37] Dental cement has been found to initially induce a foreign body reaction leading to marginal bone loss and biofilm formation on the implant surface.[38] Once a bacterial biofilm has contaminated the implant surface, subsequent bone loss is due to bacterial infection.[39] Moreover, the type of dental cement may contribute to an increased risk of peri-implantitis.[40–42] Korsch and Walther showed that glass ionomer cement not only led to greater cement excess after restoration placement, but had a higher occurrence of peri-implant bone loss in comparison to zinc oxide eugenol cement.[41] Although the findings of the current study and previous studies appear to highlight a significant role of dental cement in peri-implantitis, meta-analyses have revealed no evidence to suggest a difference in marginal bone loss between cement-retained and screw-retained implant restorations.[43,44]

In a previous study utilizing CBB to treat peri-implantitis, Roccuzzo et al also showed favorable results.[28] In their study, CBB was used as graft material to compare treatment of peri-implantitis around two different dental implant surfaces, titanium plasma-sprayed (TPS) and SLA, on subjects with a history of periodontal disease. GM was

not measured, and it was not reported whether restorations were retained during treatment; however, based on the clinical photos presented, restorations may have been removed.[28] Implants with SLA surfaces showed better success, with a mean PD reduction of 3.4 ± 1.7 mm at 12 months,[28] an outcome comparable to the present report (mean PD reduction = 3.3 ± 0.4 mm at 1 year). A major difference in treatment protocol between Roccuzzo et al[28] and the protocol outlined in the present report is that nonsurgical therapy was deliberately not performed prior to surgery on any patient included in this case series. The exclusion of nonsurgical therapy in this report and the comparable treatment outcomes suggest that nonsurgical therapy is not absolutely necessary prior to surgical treatment of peri-implantitis. In this context, it should be noted that both nonsurgical and surgical therapy have shown great outcome variability in the treatment of peri-implantitis.[7] Roccuzzo et al[28] also utilized ethylenediaminetetraacetic acid (EDTA) 24% gel and chlorhexidine 1% gel for 2 minutes each to decontaminate the implant surface prior to CBB placement. The current report utilized chlorhexidine gluconate 0.12% solution and sterile saline for surface decontamination, with results similar to the EDTA and chlorhexidine gel combination. The choice of chlorhexidine 0.12% solution and sterile saline was made because they are commonplace and routinely used in a private practice setting in this country.

Wiltfang et al combined autogenous bone and xenograft to treat peri-implantitis defects, without removal of the restorative crown during treatment and follow-up.[45] At 1 year postoperatively, PD reduction was found to be 4.0 mm.[45] Bone substitutes have also produced comparable results in PD reduction in peri-implantitis defects.[20,46] Use of algae-derived hydroxyapatite resulted in 3.4 ± 1.6 mm PD reduction at 1 year[33]; as part of the treatment protocol, the restorative components of the implants were removed at the time of surgery and replaced immediately postoperatively.[46] Porous titanium granules used to correct peri-implantitis defects resulted in an average PD reduction of 1.7 ± 1.7 mm and DS PD reduction of 1.7 ± 2.7 mm at 1 year postoperatively.[20] The present study data, using CBB, showed DS PD reduction at 1 year to be 4 mm. Of the 11 treated implants, only one still had DS PD > 4 mm at 12 months; PD at this site was only 5 mm (9 mm at baseline). These results suggest that the treatment protocol reported herein merits further evaluation.

Results of the present study showed significant reductions in PD with concomitant minimal gingival recession, especially on the buccal aspect of the treated implants. The number of previous studies that measured gingival recession following surgical treatment of peri-implantitis is limited, with no mention of recession in the esthetic zone. In the surgical treatment of peri-implantitis without use of any graft materials, Heitz-Mayfield[34] encountered 1.0 ± 0.9 mm buccal recession following flap access and debridement. Similarly, following treatment with algae-derived hydroxyapatite, mucosal recession of 1.6 ± 1.6 mm was reported.[46] In another study using a bone substitute, hydroxyapatite, gingival recession at 48 months was found to be 0.4 ± 0.5 mm.[47] In the present study, only 1 of the 11 implants followed showed an increase in GM of 1 mm at 12 months. The recession was observed at the mesiobuccal of an implant in the maxillary lateral incisor position. Ten of the 11 implants showed no apical migration of the gingival margin at the midbuccal position, a desirable outcome in the esthetic zone.

A recent meta-analysis by Chan et al examined clinical outcomes of various surgical techniques to treat peri-implantitis.[48] They reported PD reductions of 2.4 ± 0.5 mm, 2.0 ± 0.15 mm, 2.3 ± 0.6 mm, and 3.16 ± 0.6 mm for flap debridement, resective surgery, grafting alone, and grafts + membrane, respectively.[48] In comparison, the present study showed an average of 1.7 ± 1.0 mm PD reduction when analyzing all six sites per implant, and 4.0 ± 1.8 mm PD reduction at the deepest site, at 1 year postoperatively. In terms of gingival recession, the meta-analysis reported results of 1.3 ± 0.61 mm, 1.4 ± 0.4 mm, 0.9 ± 0.9 mm, and 0.4 ± 0.3 mm for flap debridement, resective surgery, grafting alone, and grafts + membrane, respectively.[48] In the present study, using CBB to treat peri-implant defects, the results were more favorable with respect to postoperative recession.

Several studies have examined the use of traditional periodontal surgery techniques for peri-implantitis, with variable outcomes.[6,9,19,49,50] For example, resective surgery combined with implantoplasty has been shown to be a predictable means to treat peri-implant disease; however, exposure of the implant surface may result in an undesirable esthetic outcome.[51,52] In comparison with traditional periodontal regeneration, the use of resorbable or nonresorbable membranes in the treatment of peri-implantitis does not influence, and may actually hinder, treatment success.[6,9,46] Especially if a submerged approach cannot be performed, membrane adaptation can be difficult, resulting in unwanted membrane exposure.[46,53] A recent study by Roos-Jansåker et al showed that the use of a membrane in conjunction with a bone substitute did not provide any additional benefit in the treatment of peri-implantitis after 5 years.[54] Without the use of a barrier membrane, particulate graft containment within the bony defect can be a challenge. When moistened with sterile saline, the graft material used in the present study is easy to handle, pack, and adapt into the bony defect (Fig 1), which likely provides a less technique-sensitive option for clinicians. Results from the current study showed no postoperative complications such as healing by secondary intention or graft exposure.

CONCLUSIONS

The use of a porcine collagen-coated bovine bone graft to treat the hard tissue loss associated with peri-implantitis around SLA implants replacing single teeth represents a potentially predictable therapeutic modality. The ability to leave the restoration in place and the minimal postoperative recession make this protocol particularly appealing for the treatment of peri-implantitis defects in the esthetic area. Randomized controlled trials are necessary to substantiate the short- and long-term outcomes of this treatment modality, while histologic studies are needed to demonstrate bone regeneration and reosseointegration following application of this surgical protocol.

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