Case Series

A Comparative Study of Root Defect Coverage Using an Acellular Dermal Matrix With and Without a Recombinant Human Platelet-Derived Growth Factor

Christopher M. Carney,* Jeffrey A. Rossmann,* David G. Kerns,* Daisha J. Cipher,† Terry D. Rees,* Eric S. Solomon,‡ Francisco Rivera-Hidalgo,* and M. Miles Beach*

Background: The objective of this case series is to compare root defect coverage results and healing responses of bilateral recession defects treated with acellular dermal matrix (ADM) with and without recombinant human platelet-derived growth factor (rhPDGF).

Methods: Seventeen patients with 40 bilateral gingival recession defects were compared. Each defect was ≥2 mm and treated with ADM and a coronally advanced flap. Using split-mouth design, the control-side ADM was hydrated in sterile saline, whereas the test-side ADM was hydrated in rhPDGF. The patients were evaluated at 1 week, 1 month, 3 months, and 6 months. Standardized measurements were taken pre-operatively at 3 and 6 months. Healing was clinically assessed at 1 week and 1 month post-surgically.

Results: Both test and control groups showed significant gain in root defect coverage over the 6-month period for all individuals, with the test group showing a 69.0% gain and the control group showing a 76.7% gain. Patients divided into Miller Class I and Class III defects were also found to have a significant gain in root defect coverage over 6 months. The test group showed 84.1% gain, and the control group showed 84.7% gain for Miller Class I defects. For Miller Class III defects, the test group showed 51.5% gain, and the control group showed a 60.8% gain. One week after surgery, 35% of the test group showed better healing, whereas 15% of the control group showed better healing. One month after surgery, 20% of the test group showed better healing, whereas 15% of the control group showed better healing.

Conclusion: Based on the results of this case series, there were no statistically or clinically significant differences in root defect coverage, keratinized tissue, clinical attachment level, or clinical healing for treatment of root recession with a coronally advanced flap and ADM with and without rhPDGF. *J Periodontol 2012;83:893-901.*

KEY WORDS

Case series; gingival recession; recombinant platelet-derived growth factor-BB.

* Department of Periodontics, Texas A&M University Health Science Center, Baylor College of Dentistry, Dallas, TX.
† College of Nursing, University of Texas at Arlington, Arlington, TX.
‡ Department of Public Health Services, Texas A&M University Health Science Center, Baylor College of Dentistry.

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Gingival recession is defined as an acquired deformity of the gingival marginal tissue in which the result is loss of attachment and an exposed root surface. This condition can lead to root sensitivity, root caries, difficulty in achieving plaque control, and esthetic concerns. There are several surgical techniques available for correction of this acquired deformity using the patient's own tissue, such as the free gingival graft, lateral pedicle graft, coronally advanced flap, and subepithelial connective tissue graft. Predictability for root coverage is often dependent on having adequate donor tissue available. This is also a limiting factor in covering multiple recession defects.

The subepithelial connective tissue graft technique is currently the gold standard for gingival recession therapy.[1] However, given the reluctance of patients to have additional surgical sites, potentially greater patient discomfort, limitation of adequate donor tissue, and increased surgical time,[2,3] periodontists have turned to allograft substitutes. Treatment of gingival recession using an acellular dermal matrix allograft (ADM) was documented by Harris and Aichelmann-Reidy et al.[4-7] and has become an accepted alternative to autogenous sources. Harris and others have shown that ADM is comparable to connective tissue without a clinically significant difference in mean root coverage.[5,6,8-11] Currently, there are several ADM products for treating gingival recession available. Although the use of one product[§] is well documented,[12-18] very little literature exists demonstrating the utility of other products. Barker et al.[19] compared root coverage using two of the leading ADM products and found no statistical or clinical differences in root coverage, keratinized tissue (KT), probing depth (PD), or clinical healing. One limitation in the use of ADM is the dependency on an adequate blood supply at the recipient site to nourish the allograft material and establish integration of the matrix with the patient's tissues. The application of a growth factor to enhance angiogenesis and accelerate the healing cascade could improve survivability of the ADM and increase a successful outcome.

Growth factors are generally accepted to be essential mediators of tissue repair via established mechanisms of action that include stimulatory effects on angiogenesis and cellular proliferation, growth, differentiation, and matrix biosynthesis.[20] Specifically, platelet-derived growth factor (PDGF) is a natural protein that regulates cell division and growth via increased angiogenesis. It acts early in the wound-healing cascade by initially attracting and activating neutrophils and macrophages, which are key cell mediators of early tissue repair.[21] Vascular endothelial growth factor (VEGF) as related to PDGF possesses a powerful angiogenic action. PDGF can directly stimulate angiogenesis as well as increase the action of VEGF, further developing the vasculature in the area.[22] Applied locally, recombinant human PDGF (rhPDGF) has been shown to destabilize blood vessels, probably as a result of the action of pericytes after the rhPDGF chemotactic gradient. Because of this effect, blood vessels adjacent to the healing wound will bud capillaries and filamentous webs of neovasculature into the site.[23]

PDGF-BB is the only dimeric form that can bind to all three PDGF receptor combinations with high affinity.[24] In the case of PDGF-BB, the protein is naturally released from the blood platelets, as well as macrophages, fibroblasts, and osteoblasts after an injury in which the protein recruits cells from the surrounding matrix. The positive effects of rhPDGF have been shown on regeneration of Class II furcation defects when used with bone allograft material[25,26] as well as on the periodontal attachment apparatus.[27-30] In a study by Nevins et al.,[20] rhPDGF-BB stimulated significant increases in bone fill and clinical attachment level gain in intraosseous defects with minimal gingival recession after 3 months. The usage of recombinant protein products has been shown to provide ≈1,000 times greater concentration of PDGF-BB than that typically found in platelet concentration.[31,32] The ability of rhPDGF to stimulate cellular chemotaxis and mitogenesis with its effect on angiogenesis by increasing levels of VEGF makes this biologic mediator attractive as a wound-healing agent for periodontal surgery[33] and possibly treatment of gingival recession.

This study is designed to evaluate the root defect coverage and healing of bilateral recession defects using ADM[‖] with and without rhPDGF[¶] over a 6-month healing period. To date, no randomized, controlled, clinical trial has analyzed the use of rhPDGF in terms of root defect coverage and clinical healing with an ADM. This adjunct should in theory promote faster revascularization of the ADM and improve root defect coverage in the treatment of gingival recession defects.

MATERIALS AND METHODS

The study protocol was approved by the Baylor College of Dentistry–Texas A&M Health Science Center (BCD) institutional review board and conducted in accordance with the Helsinki Declaration of 1975, as revised in 2000. Seventy-eight individuals were screened from February 2009 to June 2010 from either the BCD general patient pool or by request from the individual patients outside of BCD. Individuals selected were ≥18 years old with bilateral, maxillary, or mandibular, single or multiple, buccal, vertical recession (VR) defects of ≥2 mm. The defects were measured from the

§ AlloDerm, LifeCell, The Woodlands, TX.
‖ Puros Dermis, Zimmer Dental, Carlsbad, CA.
¶ GEM 21S, Osteohealth, Shirley, NY.

cemento-enamel junction (CEJ) to the midfacial gingival margin and were limited to incisors, canines, and premolars. Each participant was required to maintain good plaque control as defined by a modified O'Leary index[34] of ≥85% after initial therapy. Each defect site was required to be Miller Class I, II, or III,[35] to have no bleeding on probing at the surgical sites, and to have a PD ≤3 mm. The participants were also required to be in good general health and non-smokers without uncontrolled systemic conditions that may have compromised surgery.

Seventeen patients (5 males and 12 females, aged 30 to 69 years; mean age: 49.4 years) with a total of 40 defects were recruited for the study. Each patient received verbal and written instructions and signed an informed consent document before participating. There were a total of 20 test and 20 control teeth, which all tested vital to thermal evaluation. Each group had 12 Miller Class I or II defects and eight Miller Class III defects, which consisted of two maxillary incisors, 12 maxillary canines, eight maxillary premolars, four mandibular canines, and 14 mandibular premolars. Three of the 17 patients had contralateral recession defects in both the maxillary and mandibular arches, which were treated separately. The study was performed in a split-mouth design in which each of the bilateral defects were treated with an ADM hydrated in either an rhPDGF (test) or sterile saline (control). Before surgery, photographs and radiographs were taken of the selected teeth to evaluate the interproximal bone levels. The clinical variables used to identify preoperative findings and evaluate the results included the following: 1) gingival index (GI)[36] scored as 0, 1, 2, or 3; 2) VR defined as the distance measured from the CEJ to the free gingival margin (FGM); 3) horizontal recession (HR) defined as the width of recession on the midfacial surface of the tooth at the level of the CEJ; 4) PD defined as the distance from the FGM to the base of the sulcus on the midfacial surface; 5) clinical attachment level (CAL) defined as the distance from the CEJ to the measurable base of the gingival sulcus on the midfacial surface of the tooth; 6) width of KT defined as the distance from the FGM to the mucogingival junction (MGJ) on the midfacial surface of the tooth; 7) papillary height (PH) defined as the distance from the base of the papilla at the level of the CEJ to the tip of the papilla; and 8) papillary width (PW) defined as the horizontal measurement from the adjacent CEJs at the base of the papilla. To measure the width of the KT, Lugol solution was applied to the alveolar mucosa with a cotton tip applicator that temporarily stained the KT to the level of the MGJ. The stained tissue was measured from the FGM to the MGJ as defined by the clear demarcation line. All measurements were made with a periodontal probe[#] and estimated to the nearest 0.5 mm using a customized acrylic surgical stent with a groove cut at the midfacial of the selected teeth. For each patient, two masked examiners performed the measurements immediately before surgery and repeated them at the 3- and 6-month follow-up appointments. At the 1-week and 1-month follow-up appointments, the healing status of each bilateral site was subjectively evaluated by each of the two masked examiners (JAR, DGK) as "same" or "better" based on the presence or absence of inflammation and healing appearance. Finally, each patient was asked whether he/she was experiencing any sensitivity on either the test or control teeth at the baseline, 1-month, 3-month, and 6-month appointments, with the response categorized as "not sensitive," "slightly sensitive," "generally sensitive," or "very sensitive."

Surgical Procedure

Immediately before surgery, the test site was determined by using a randomized table. Each surgical site was anesthetized with ≈51 mg 2% lidocaine HCl with 1:100,000 epinephrine. The surgery began with treating the test site using incisions described by Zucchelli and De Sanctis,[37] followed by de-epithelialization of the papillae. A full-thickness flap was reflected and extended ≈3 mm apical to the alveolar bone crest using a microsurgical elevator,[**] followed by split-thickness flap reflection. The split-thickness flap was prepared mesially, distally, and apically to provide adequate mobility and passive coronal positioning of the flap using a modified periodontal knife.[††] After flap reflection, the exposed root surfaces were planed thoroughly with a curet[‡‡] followed by conditioning with neutral pH and 24% EDTA[§§] for 2 minutes and irrigated thoroughly with sterile saline. The defect site was measured horizontally from the distal root line angle of the most mesial tooth to the mesial line angle of the most distal tooth in the incision design. The site was also measured vertically from the CEJ to ≈3 mm beyond the bony dehiscence. The ADM graft was cut to the appropriate size to fit the defect and hydrated in ≈2 mL of rhPDGF for ≥3 minutes. The graft was then placed over the defect site with the connective tissue side down and the coronal aspect at the level of the CEJ. The ADM was sutured using 5-0 chromic gut suture using a double-sling technique. The flap was then coronally advanced to cover the entire ADM graft and sutured at the level of the CEJ using a 5-0 polypropylene, non-resorbable monofilament suture[||||] using a double-sling suture technique. Interrupted sutures were also used to secure all the papillae and provided for interproximal primary closure (Fig. 1). The contralateral (control) site was prepared and treated

PCP-UNC probe, Hu-Friedy, Chicago, IL.
** Allen Periosteal Elevator, Hu-Friedy.
†† Modified Orban Knife, Hu-Friedy.
‡‡ 7/8 Younger-Good Curet, Hu-Friedy.
§§ PrefGel, Straumann, Andover, MA.
|||| 8698 Prolene, Ethicon, Somerville, NJ.

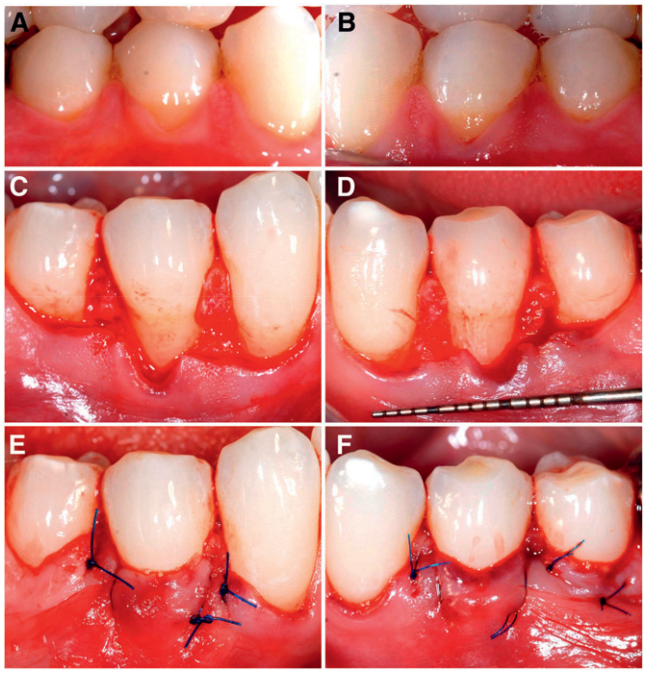

Figure 1.
A) *Baseline, test.* ***B)*** *Baseline, control.* ***C)*** *Initial incisions, test.* ***D)*** *Initial incisions, control.* ***E)*** *Graft sutured, test.* ***F)*** *Graft sutured, control.*

using the same surgical protocol with the exception that the ADM was hydrated in sterile saline, rather than rhPDGF, for 3 minutes. In every case, both defect sites were surgically treated during the same appointment.

Post-Surgical Care

Immediately after surgery, the patients were prescribed 500 mg amoxicillin, three times daily for 7 days, or 300 mg clindamycin, three times daily for 7 days, if the patient was allergic to penicillin. In addition, 5 mg hydrocodone with 500 mg acetaminophen was also prescribed to be taken if needed for pain control. Each patient was instructed to use an ice pack as needed over the first 8 hours, alternating every 20 minutes at each surgical site. A liquid diet was recommended for the first 2 post-surgical days with a gradual change to soft diet for the following 2 weeks. Oral hygiene instructions were given to the patients recommending the discontinuance of mechanical toothbrushing at the surgical sites, substituting a cotton tip applicator saturated with 0.12% chlorhexidine gluconate for the first 2 weeks. The patients were instructed to gently use strokes in the apical–coronal direction to minimize any tension on the flaps. After 2 weeks, the patients were advised to begin gentle toothbrushing using a roll technique until the 1-month follow-up appointment, at which time hygiene control was returned to regular toothbrushing and flossing. At each follow-up visit (1 week, 1 month, 3 months, and 6 months), photographs were taken (Fig. 2). Professional plaque control was given, and oral hygiene instructions were reinforced. At 1 month, the polypropylene sutures were removed, and at the 3- and 6-month appointments, the clinical measurements were taken again by the same two masked examiners using the customized acrylic stent.

Statistical Analysis

Calculations with a P value of <0.05 significance show that 20 paired defects will show a difference in each of the variables with 98.9% power. The unit of analysis for this study was the patient, with teeth nested within patients. Means ± SDs were calculated for all clinical measurements to 0.5 mm (Table 1). Linear mixed models were constructed to compare the two procedures on changes over time in VR, KT, CAL, PDs, GI, HR, mesial PH, distal PH, mesial PW, distal PW, and sensitivity. The fixed-effects portion of each model was "procedure" (ADM hydrated in rhPDGF or sterile saline), and the random effects portion of each model was the "patient," with teeth nested within each patient. "Time" was specified as the repeated effect, with three levels (baseline, 3 months, and 6 months), with a first-order autoregressive covariance structure. The distance from CEJ to bone at baseline was analyzed with a linear mixed model, with procedure as the fixed effect and patient and tooth nested within patient as the random effects. Friedman tests were computed to test change over time in root defect coverage from baseline and from 3 to 6 months.

RESULTS

Each of the 17 participants recruited for this study had single or multiple bilateral recession defects, and three had bilateral recession defects in both arches. There were 20 control and 20 test teeth, with each group having 12 Miller Class I or II defects and eight Miller Class III defects. These teeth consisted of two maxillary incisors, 12 maxillary canines, eight maxillary premolars, four mandibular canines, and 14 mandibular premolars. All teeth selected tested vital to thermal evaluation.

The linear mixed model for VR indicated that there was no significant effect for procedure ($F_{(1,28.3)} = 0.07$, $P = 0.80$). However, all patients significantly improved over time ($F_{(2,24.7)} = 141.2$, $P<0.001$). VR averaged 2.98 ± 1.00 mm at baseline and improved to 0.96 ± 0.88 mm at 3 months and 0.65 ± 0.76 mm at 6 months for test patients. For control participants, VR averaged 3.04 ± 1.10 mm at baseline and improved to 0.95 ± 0.98 mm at 3 months and 0.76 ± 0.84 mm at 6 months (Fig. 3). Therefore, all patients showed improved root defect coverage (VR) regardless of the procedure received (Table 1). Patients significantly improved from baseline to 6 months ($t_{(42.7)} = 14.1$, $P<0.001$), but not from 3 to 6 months ($t_{(22.3)} = 0.71$, $P = 0.48$). Moreover, the random-effects variance

estimate (the effect for patient) was not significant (Wald $z = 1.76$, $P = 0.08$), indicating that the variability in VR not accounted for by time and procedure was small.

The linear mixed model for horizontal recession indicated that there was no significant effect for procedure ($F_{(1,32.8)} = 0.12$, $P = 0.73$) (Fig. 4, Table 1). However, all patients significantly improved over time ($F_{(2,27.2)} = 39.6$, $P < 0.001$). HR averaged 3.88 ± 0.82 mm at baseline and improved to 2.31 ± 1.79 mm at 3 months and 2.13 ± 1.48 mm at 6 months for test participants. For control individuals, HR averaged 3.85 ± 0.78 mm at baseline and improved to 2.15 ± 1.73 mm at 3 months and 1.93 ± 1.59 mm at 6 months. Therefore, all patients showed improved horizontal coverage regardless of the procedure received (Table 1). Patients significantly improved from baseline to 6 months ($t_{(52.6)} = 7.7$, $P < 0.001$) but not from 3 to 6 months ($t_{(24.9)} = 1.03$, $P = 0.31$). Moreover, the random-effects variance estimate (the effect for patient) was not significant (Wald $z = 1.3$, $P = 0.19$), indicating that the variability in VR not accounted for by time and procedure was small.

The linear mixed model for CAL indicated that there was no significant effect for procedure ($F_{(1,16.5)} = 0.16$, $P = 0.70$) (Table 1). However, all patients significantly improved over time ($F_{(2,19.9)} = 77.8$, $P < 0.001$). CAL averaged 4.44 ± 1.28 mm at baseline and improved to 2.33 ± 0.87 mm at 3 months and 2.38 ± 1.03 mm at 6 months for test participants. For control participants, CAL averaged 4.50 ± 1.04 mm at baseline and improved to 2.30 ± 1.11 mm at 3 months and 2.10 ± 1.07 mm at 6 months. Therefore, all patients showed improved probing attachment regardless of the procedure received (Table 1). Patients significantly improved from baseline to 6 months ($t_{(29.6)} = 10.6$, $P < 0.001$) but not from 3 to 6 months ($t_{(17.9)} = 0.40$, $P = 0.70$). Moreover, the random-effects variance estimate (the effect for

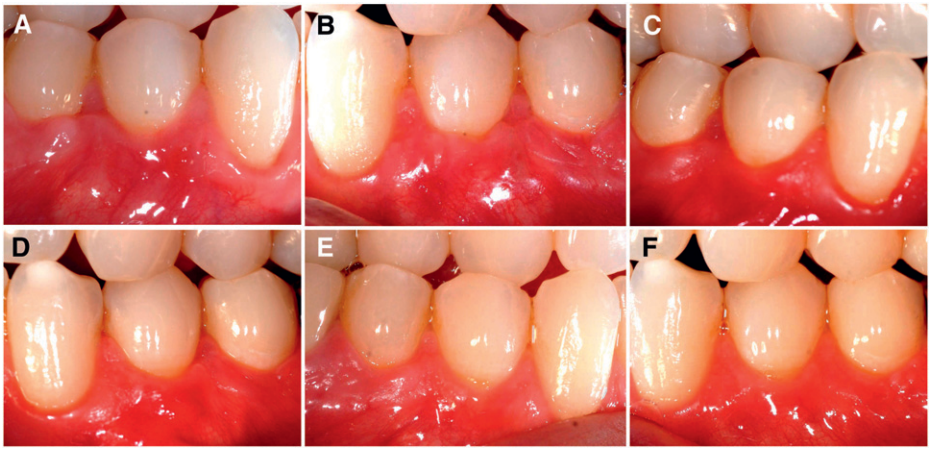

Figure 2.
A) *One month, test.* ***B)*** *One month, control.* ***C)*** *Three months, test.* ***D)*** *Three months, control.* ***E)*** *Six months, test.* ***F)*** *Six months, control.*

Table 1.

Mean (± SD) of Variables at Baseline and at 3 and 6 Months

	Baseline (mm)		3 Months (mm)		6 Months (mm)	
Variable	Test	Control	Test	Control	Test	Control
VR	2.98 ± 1.00	3.04 ± 1.10	0.96 ± 0.88	0.95 ± 0.98	0.65 ± 0.76	0.76 ± 0.84
HR	3.88 ± 0.82	3.85 ± 0.78	2.31 ± 1.79	2.15 ± 1.73	2.13 ± 1.48	1.93 ± 1.59
PD	1.50 ± 0.37	1.60 ± 0.25	1.40 ± 0.33	1.36 ± 0.40	1.41 ± 0.40	1.35 ± 0.40
CAL	4.44 ± 1.28	4.50 ± 1.04	2.33 ± 0.87	2.30 ± 1.11	2.38 ± 1.03	2.10 ± 1.07
KT	2.16 ± 1.23	2.19 ± 1.07	2.00 ± 1.21	2.06 ± 1.23	2.16 ± 1.06	2.05 ± 1.02
PW-M	3.78 ± 0.88	3.80 ± 0.64	3.74 ± 0.92	3.61 ± 0.93	3.71 ± 0.77	3.53 ± 0.84
PW-D	3.38 ± 1.11	3.73 ± 0.91	3.40 ± 1.17	3.70 ± 0.90	3.38 ± 0.91	3.58 ± 0.69
PH-M	3.36 ± 0.61	3.50 ± 0.58	3.28 ± 0.83	3.44 ± 0.69	3.30 ± 0.60	3.34 ± 0.57
PH-D	3.25 ± 1.36	3.36 ± 0.81	2.99 ± 0.96	3.39 ± 0.71	3.18 ± 0.95	3.23 ± 0.64

Test = graft hydrated in rhPDGF; Control = graft hydrated in sterile saline; M = mesial; D = distal.

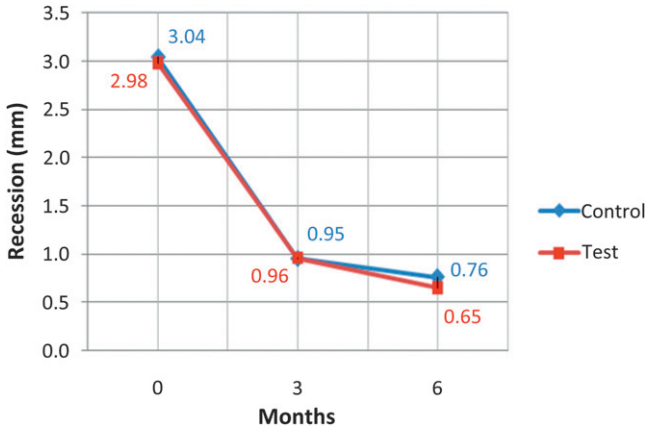

Figure 3.
Comparison of VR at 0, 3, and 6 months.

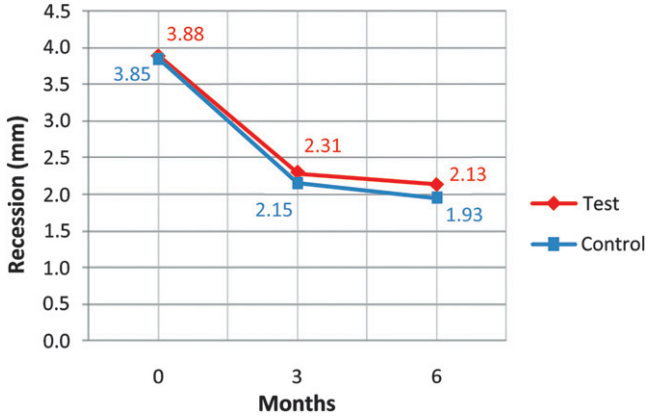

Figure 4.
Comparison of HR at 0, 3, and 6 months.

patient) was not significant (Wald $z = 1.94$, $P = 0.052$), indicating that the variability in VR not accounted for by time and procedure was small but approached significance.

Linear mixed-models analyses with repeated measures resulted in no significant effects for procedure or time for GI, KT, distal PH, mesial PH, distal PW, mesial PW, and sensitivity (Table 2). The time effect for PD also approached significance ($F_{(2,32.8)} = 2.6$, $P = 0.09$) (Table 2). Patients showed a trend toward improvement in PD from baseline to 6 months ($t_{(45.9)} = 1.9$, $P = 0.061$) but not from 3 to 6 months ($t_{(29.3)} = 0.0$, $P = 1.0$).

Both test and control groups showed significant gain in root defect coverage over the 6-month period for all participants, with the test group showing a 69.0% gain and the control group showing a 76.7% gain. Participants divided into Miller Class I and Miller Class III defects were also found to have a significant gain in root defect coverage over 6 months. The test group showed

Table 2.

Comparison of Mean Variables at 0 to 3 Months and 0 to 6 Months

	0 to 3 Months (mm)		0 to 6 Months (mm)		
Variable	Test	Control	Test	Control	$P^{\dagger}$
VR	2.01	2.09	2.33	2.28	0.8
HR	1.56	1.7	1.75	1.93	0.73
PD	0.1	0.24	0.09	0.25	0.95
CAL	2.11	2.2	2.06	2.4	0.7
KT	0.16	0.13	0	0.14	0.92
PW-M	0.04	0.19	0.06	0.28	0.67
PW-D	–0.03*	0.03	0	0.15	0.3
PH-M	0.09	0.06	0.06	0.16	0.46
PH-D	0.26	–0.03*	0.08	0.14	0.51

Test = graft hydrated in rhPDGF; Control = graft hydrated in sterile saline; M = mesial; D = distal.
* Negative values indicate an increase in the measured parameter.
† *P* values from linear mixed-models analysis.

84.1% gain and the control group showed 84.7% gain for Miller Class I defects. The test group showed 51.5% gain and the control group showed a 60.8% gain for Miller Class III defects.

Evaluation of Healing

Patients were evaluated by two masked, experienced, clinical examiners (JAR, DGK) at the 1-week and 1-month postoperative appointments. The healing status of each site was evaluated subjectively for each patient independently. Comparisons were considered strictly based on the clinical opinion of the examiners as same or better based on the presence or absence of inflammation and the healing appearance, including color changes, tissue contour, swelling, etc. One week after surgery, 35% of the test group showed better healing than the control group, and 15% of the control group showed better healing than the test group. One month after surgery, 20% of the test group showed better healing than the control group, and 15% of the control group showed better healing than the test group. The remaining test and control participants appeared to have healed equally at both 1 week and 1 month.

DISCUSSION

Both sterile saline and rhPDGF were effective as hydrating agents and provided equal manipulation of the ADM for placement and suturing when attempting root defect coverage via an envelope flap incision, as described by Zucchelli and De Sanctis,[37] combined

with a coronally advanced flap. Specifically, VR, HR, and CAL each showed significant improvement from baseline over the 6-month study period. Each of the overall treatment effects led to root defect coverage, as described by Greenwell and Bissada.[38] The designation of "root defect coverage" was used to eliminate the confusion regarding what might be viewed as a successful or unsuccessful result and so that accurate conclusions could be drawn. The test treatment group yielded 69% root defect coverage, and the control treatment group yielded 76.7% root defect coverage. However, after categorizing the participants by Miller Class I and Miller Class III (Fig. 5), the Miller Class I participants' data appeared to be more consistent with previous studies.[35] Miller Class I patients achieved 84.1% coverage for the test group and 84.7% for the control, whereas Miller Class III patients achieved 51.5% coverage for the test group and 60.8% for the control. During evaluation of the individual teeth in the Miller Class I group, 11 of 24 teeth achieved 100% root defect coverage regardless of treatment group. Of the remaining 13 teeth, five of the teeth were within 0.5 mm of obtaining 100% root defect coverage. The masked examiners were compromised in their measurements of the mentioned five teeth at all three time periods as a result of the inability to accurately determine the location of the midfacial CEJ because of cervical abrasion. An estimate had to be made based on the CEJs of the adjacent teeth, similar to the technique used to reconstruct an abraded CEJ for treatment of recession, as described by Cairo and Pini-Prato.[39] In their study, they obtained vertical and horizontal measurements to the CEJ of the contralateral tooth. If the contralateral tooth was also abraded at the CEJ, the adjacent tooth was measured instead. The measurements were transferred to identify where the CEJ should be located. The new CEJ was then constructed with composite resin, providing a quantifiable demarcation line for measurement. In future studies, the authors recommend that VR should also be measured from the intact facial cusp tip of the tooth being treated to the gingival margin to eliminate the error caused by the inability to distinguish the CEJ on the midfacial surface of some tooth surfaces attributable to cervical abrasion.

Although not studied here, the benefit of using rhPDGF with ADM may be the improved ability to produce new attachment to include cementum and functional PDL, which is worthy of additional histologic study. In a study by McGuire and Scheyer,[40] rhPDGF with β-tricalcium phosphate bone mineral and a collagen membrane were used to treat gingival recession defects. Their clinical results for this combination were comparable to connective tissue grafting in a sample of seven patients with contralateral defects. Although the clinical appearance was the same, we do not know whether the rhPDGF altered the histologic healing to any significant effect in either the McGuire and Scheyer study or in this study. However, any benefit of increased angiogenesis could not be demonstrated within the limits of this study based on

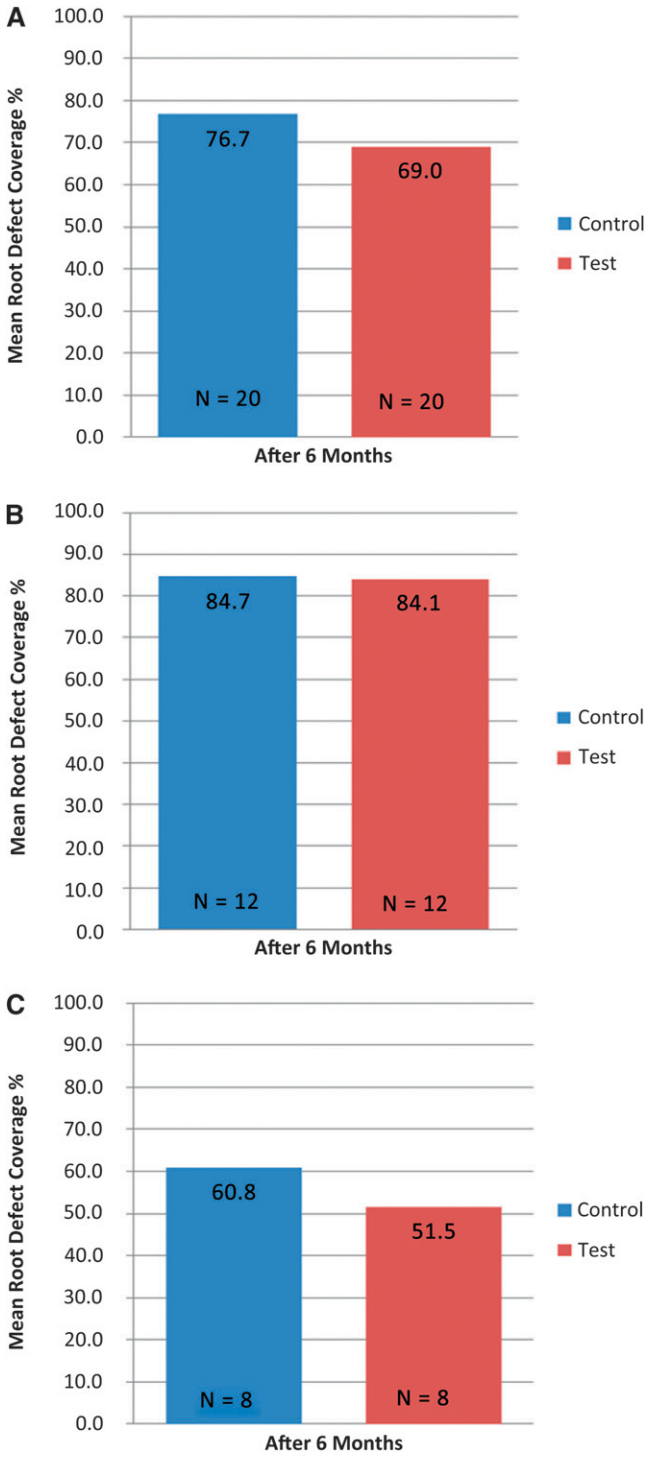

Figure 5.
Mean percentage root defect coverage. ***A)*** *All defects.* ***B)*** *Miller I defects.* ***C)*** *Miller III defects.*

clinical appearance during the first 30 days of healing. This may be attributable to the inability to have the rhPDGF retained on the matrix long enough to show clinical benefit. The ADM used in this study hydrates rapidly and may have been a factor in limiting the uptake of the rhPDGF and its ability to have a positive influence on healing. The ADM hydrated to saturation within 2 minutes and may not have been substantive. For the rhPDGF to have effect, a better transport vehicle may be needed to maintain contact between the rhPDGF and the root surface for a longer time period, which has been demonstrated with bone graft materials such as β-tricalcium phosphate.[25,26]

The use of tissue engineering in combination with a proven technique for correction of gingival recession defects would seem prudent in accomplishing a true regeneration result. The use of rhPDGF has been shown in both animal and human studies to promote regeneration, including the formation of cellular cementum and functional periodontal ligament on a previously diseased root surface.[25,26,41] This study is designed to explore the feasibility of combining the use of the growth factor (rhPDGF) with ADM to achieve root defect coverage. To our knowledge, this combination has not been tested previously. It appears that a human histologic analysis would be warranted to better understand the results of this combined therapy in achieving regeneration. The potential benefit for the patient would be a clinical result that may have improved long-term correction of the gingival recession similar to that shown for connective tissue grafting, without the need for the second surgical donor site.[42]

CONCLUSIONS

Based on the results of this study and the products used, there were no statistically significant differences in root defect coverage, KT, CAL, or clinical healing for treatment of root recession with a coronally advanced flap and the selected ADM with and without rhPDGF. Within the limits of this study, there was no additional benefit derived from adding rhPDGF to the treatment modality for root coverage procedures using this specific ADM product. Because only one ADM product was tested and not all ADM products are processed in the same manner, the conclusions drawn can only be applied to the use of this specific product. Future studies should be conducted to evaluate the histologic differences in root attachment with and without the addition of rhPDGF to ADM.

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Correspondence: Dr. Jeffrey A. Rossmann, Department of Periodontics, Baylor College of Dentistry, 3302 Gaston Ave., Dallas, TX 75246. E-mail: jrossmann@bcd.tamhsc.edu.

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