Periodontal Conditions of Sites Treated With Gingival Augmentation Surgery Compared With Untreated Contralateral Homologous Sites: An 18- to 35-Year Long-Term Study

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Background: The aim of this split-mouth study is to compare long-term (18 to 35 years) periodontal conditions of sites treated with gingival augmentation procedures (GAPs) and untreated homologous contralateral sites.

Methods: Forty-seven patients with 64 sites (test group), with lack of attached gingiva associated with recessions, were treated with marginal or submarginal free gingival grafts. Sixty-four contralateral homologous sites (control group), with or without gingival recession (GR) and with attached gingiva, were left untreated. Patients were recalled every 4 to 6 months during follow-up period. GR depth, keratinized tissue (KT) width, and probing depth were measured at baseline (T_0), 1 year after surgery (T_1), during follow-up (10 to 27 years, T_2), and at the end of the follow-up period (18 to 35 years, T_3). Multilevel and regression analyses were conducted.

Results: At the end of T_3, 83% of the 64 treated sites showed recession reduction (RecRed), whereas 48% of the 64 untreated sites experienced increase in recession. Treated sites ended with gingival margin (GM) 1.7 mm ($P = 0.01$) more coronal and KT 3.3 mm ($P < 0.001$) wider than untreated sites. In grafted sites, KT at T_3 remained stable compared with T_1 value (4.1 mm, $P < 0.001$).

Conclusions: Sites treated with GAPs resulted in coronal displacement of GM with RecRed up to complete root coverage, whereas contralateral untreated sites showed a tendency to increase in existing recession or develop new recession during the 18- to 35-year follow-up. *J Periodontol 2016;87:1371-1378.*

KEY WORDS

Gingival recession; surgery, plastic; surgery procedures, operative; transplants.

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The role of the amount of keratinized gingiva for maintenance of periodontal health in terms of gingival inflammation and stability of gingival margin (GM) has been debated for many years. Some experimental studies[1-3] support the hypothesis that it is possible to maintain periodontal health in sites with a reduced amount or absence of attached gingiva in the presence of optimal plaque control. Conversely, another study[4] concludes that "all surfaces with <2.0 mm of keratinized gingiva exhibited clinical inflammation, despite the fact that the tooth surfaces were free from plaque." In addition, there is evidence indicating that sites with reduced amounts of keratinized tissue (KT), in particular "thin biotypes," have a tendency to develop more recession defects than sites protected by large and thick amounts of attached gingiva.[5]

Tooth site–related conditions combined with missing/reduced amounts of attached gingiva can contribute to negatively influencing periodontal health and GM stability. These conditions include: 1) gingival recession (GR); 2) thin periodontium; 3) bucco-lingual displacement of teeth; 4) root prominence; 5) shallow depth of vestibulum; 6) frenum pulling; 7) cervical restorations; and 8) orthodontic treatment.[6,7]

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Published proceedings of the 1996 World Workshop in Periodontics recommended considering gingival augmentation procedures (GAPs) to prevent soft tissue damage in the presence of alveolar bone dehiscence during natural or orthodontic tooth eruption, halt progression of GR, improve plaque control and patient comfort around teeth and implants, and increase insufficient dimension of gingiva in conjunction with fixed or removable prosthetic dentistry.[7]

These clinical indications for gingival augmentation have been discussed in a recent systematic review of periodontal soft tissue non-root coverage procedures.[8] Among different surgical techniques, autogenous free gingival graft (FGG) is considered the gold standard surgery as well as the most common approach used for GAPs.[9] However, only a few studies[10-12] report short- or medium-term data on stability of GM after FGG. A 4-year split-mouth study[10] showed significant differences in amount of KT, attached gingiva, and GR between treated and untreated sites. Similar outcomes in favor of grafted sites were reported in 6-[11] and 8-year[12] follow-up studies. A 10- to 25-year long-term study demonstrated that marginal FGG (MFGG) and submarginal FGG (SMFGG) for gingival augmentation in sites without attached gingiva were able to provide significant increase in KT and long-term stability of GM.[13]

Agudio et al.[14] compared periodontal conditions of sites treated with FGG and untreated homologous contralateral sites during 10 to 27 years. Treated sites showed a tendency to coronal displacement of GM with recession reduction (RecRed), whereas untreated sites showed a tendency for apical displacement of GM. A recent systematic review[15] of long-term outcomes of untreated buccal GRs confirmed these findings.

The aim of the present split-mouth study is to compare periodontal conditions of sites treated with GAPs (FGG) and untreated homologous contralateral sites during 18 to 35 years.

MATERIALS AND METHODS

Study Design

This study has been reported according to Strengthening the Reporting of Observational Studies in Epidemiology guidelines for reporting observational studies.[16]

Study Population

The study population consisted of 47 highly motivated and compliant patients (15 males and 32 females, aged 36 to 73 years; mean age: 54.2 ± 9.0 years; 128 sites) with thin biotype, high level of oral hygiene, and no signs of active periodontal diseases. Patients were treated at a private practice in Bergamo, Italy, from 1981 to 1998. This cohort originates from a population of 55 patients (146 sites) described in detail in a previous long-term (10 to 27 years) study[14] and followed up during an 8-year period (18 to 35 years). The present, further follow-up study was approved by the Ethics Committee of the Tuscan Academy of Dental Research, Florence, Italy.

Written consent was obtained from all 47 patients participating in the present study before surgical treatment with agreement to use their data for the clinical trial, in accordance with the Helsinki Declaration of 1975, as revised in 2013.

Each patient contributed at least one pair of sites. Some patients contributed multiple pairs of experimental units.

Entry criteria for the study were: 1) age ≥18 years; 2) good systemic health; 3) no active periodontal diseases; 4) presence of at least one site (test) showing absence of attached gingiva associated with GR at baseline; this site was treated with a GAP consisting of FGG; and 5) presence of contralateral homologous site (control), with or without attached gingiva and with or without GR, that was either judged not to be amenable to mucogingival surgery or considered amenable to mucogingival surgery, but left untreated because of preference of patient for avoiding surgical treatment. For ethical reasons, these patients were clearly informed that recessions could occur during the study follow-up period due to their thin periodontal biotype. Patients with baseline untreated sites, who underwent gingival augmentation during the follow-up period, had been already excluded from a previous study.[14]

Patients presenting teeth with undetectable cementoenamel junction (CEJ), non-cervical carious lesions, crowns and restorations, or requiring orthodontic treatment were excluded from the study.

Measurements

Clinical measurements were always recorded by an expert periodontist (GA) with >30 years of clinical experience[14] using a calibrated offset periodontal probe§ throughout the study period (up to 35 years of follow-up).

Patient-, tooth-, and site-associated variables were recorded for each patient at baseline (T_0), 1 year after surgery (T_1), during the follow-up period (T_2) (10 to 27 years),[14] and at the end of the follow-up period (T_3) (18 to 35 years). Patient-associated variables included age, sex, and tobacco smoking. Tooth-associated variables included survival of experimental unit, tooth position (maxillary or mandibular), and tooth type. Site-associated variables included GR depth (Rec), KT width, and probing depth (PD).

§ Williams probe, Hu-Friedy, Chicago, IL.

Surgical Procedures

Forty-seven patients of the present study underwent surgical augmentation procedures using MFGGs or SMFGGs in test sites, as described in detail in previous studies.[13,14] MFGG was used when existing free gingiva was found to be very thin. In these instances, the coronal part of the graft was positioned at the presurgical level of GM after removing existing free gingiva. SMFGG was used when free gingiva was considered thick. In these cases, the graft was sutured at a submarginal level without removing marginal free gingiva.

Patients were recalled every 4 to 6 months for supportive periodontal maintenance (PM) care during the follow-up period of 18 to 35 years.

Questionnaire

Presence or absence of dental hypersensitivity of test and control sites was investigated through a questionnaire given to patients at baseline, during follow-up, and at the end of the follow-up period. Patients were asked about their subjective feelings of comfort or discomfort during toothbrushing at the end of the follow-up period to compare treated and untreated sites.

Statistical Analyses

Descriptive statistical analyses were performed using mean ± SD for quantitative variables and frequency and percentage for qualitative variables.

Inferential statistics were applied using multilevel linear regression models[17] at two levels (pair and site). These models considered that sites (treated and untreated) are clustered in the same pair, and different pairs of teeth can be clustered in the same patient. Outcome variables were RecRed and KTgain. Covariates were: 1) Rec_{T0} in $RecRed_{T0–T1}$ and $RecRed_{T0–T2}$ models; 2) Rec_{T0} and KT_{T0} in $RecRed_{T0–T3}$ model; and 3) K_{T0} in $KTgain_{T0–T1}$, $KTgain_{T0–T2}$, and $KTgain_{T0–T3}$ models. The explicative variable was surgery (performed or not performed). Interaction terms were explored in analysis and reported only when statistically significant. Linear regression analyses were conducted separately for treated and untreated sites to explore influence of KT_{T1} on outcome variable RecRed at T_2.

RESULTS

Forty-seven patients with thin periodontal biotype contributed 128 sites for this study. There were four (9%) smokers and 43 (91%) non-smokers.

Sixty-four sites (test group) were treated with GAPs, 47 with SMFGGs, and 17 with MFGGs. Sixty-four homologous contralateral untreated sites were used as control units. Of the 64 treated sites, 14 were in maxillary arch and 50 were in mandibular arch.

Mean follow-up period was 23.6 ± 3.9 years (range: 18 to 35 years).

Full-mouth plaque score (FMPS) and full-mouth bleeding score (FMBS) were <20%.

Clinical cases are shown in Figures 1 and 2.

Figure 3 shows trends in variations in GMs and amount of KT between baseline (T_0) and end of follow-up (T_3) in sites treated with FGG and control sites.

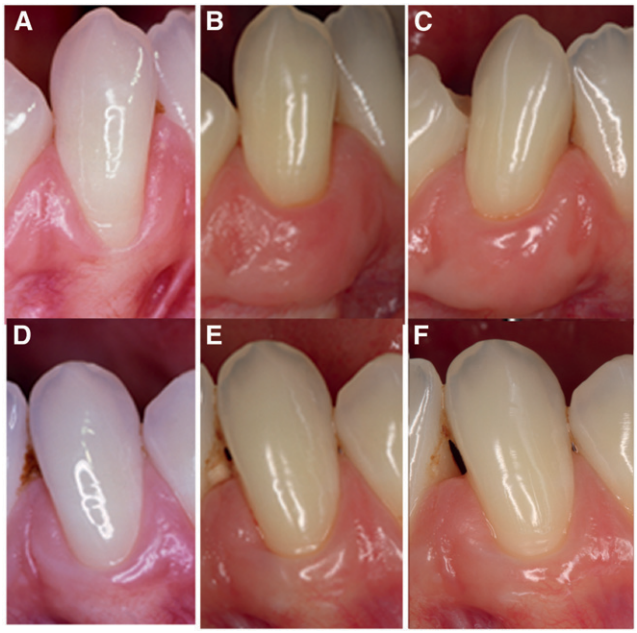

Figure 1.
Test and control sites of one study patient over time. Test site with treated right mandibular canine ***(A through C)*** *and control site with untreated left mandibular canine* ***(D through F)****.* ***A)*** *Test site at baseline (T_0) (year 1984) with 3 mm GR and absence of attached gingiva; this site underwent a GAP using FGG.* ***B)*** *Test site at follow-up 19 years after FGG (T_2) (year 2003) with GM close to CEJ with 7 mm KT.* ***C)*** *Test site at follow-up 31 years after FGG (T_3) (year 2015) with GM stable with adequate amount of KT.* ***D)*** *Control site at baseline (T_0) with 0.5 mm GR and presence of attached gingiva.* ***E)*** *Control site at follow-up (T_2) with 1 mm of GR.* ***F)*** *Control site at follow-up (T_3) with increased recession (2 mm) of GM.*

Figure 2.
Clinical view of treated and untreated sites after follow-up of 31 years (T_3).

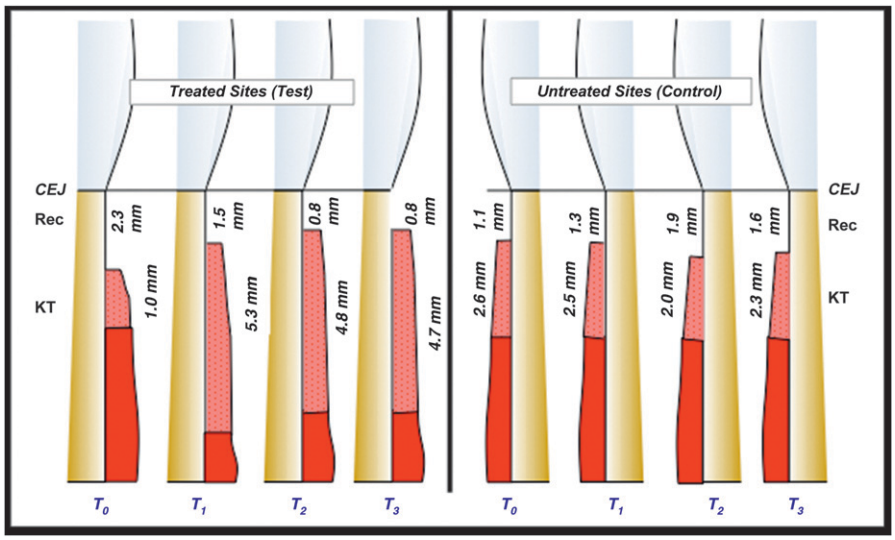

Figure 3.
Mean values of GR depth (Rec) and KT in treated and untreated sites at baseline (T_0) and during follow-up (T_1, T_2, and T_3) for all participants.

GR and Keratinized Gingiva

Baseline T0. Treated sites: At baseline, all 64 treated units presented with recession of GM (Rec_{T0}: 2.3 ± 0.9 mm on average) associated with 1 mm of residual KT (Table 1).

Untreated sites: In the control group, only 40 (62.5%) sites presented with GR, whereas others had normally positioned GMs (Rec_{T0}: 1.1 ± 1.1 mm). In this group KT averaged 2.6 ± 0.8 mm (1 to 4 mm).

Follow-up T_1 (1 year after surgery). Treated sites: Thirty-seven (58%) test units experienced RecRed, including six sites with complete root coverage (CRC), whereas GR was unchanged in 27 sites. No site showed increase of recession. Overall RecRed was associated with consistent increase in KT (5.3 ± 1.1 mm).

Untreated sites: Thirteen control sites showed increase in GR, two units with no GR at baseline developed recession, and in 49 sites GM remained stable. Overall, recession increase was associated with substantial stability in amount of KT.

Comparison between treated and untreated sites: Result of multilevel model adjusted for Rec_{T0} showed statistically significant difference in RecRed between test and control groups of 0.9 mm (95% confidence interval [CI]: 0.6 to 1.1 mm; P <0.001) in favor of grafted sites. Similarly, multilevel model adjusted for KT_{T0} showed statistically significant difference in KTgain between test and control groups of 4.1 mm (95% CI: 3.6 to 4.5 mm; P <0.001) in favor of grafted sites.

Follow-up T_2 (mean = 15.6 ± 3.9 years, 10 to 27 years). Treated sites: At the end of the mid-follow-up period (T_2), 84% of treated units experienced RecRed in comparison with baseline (T_0). In particular, RecRed of 1 mm was observed in 23 sites, 2 mm in 23, 3 mm in seven, and 4 mm in one site; overall, 37 units achieved CRC. Nine sites (14%) were unchanged compared with baseline. One site only showed 1-mm increase in GR. GR (Rec_{T2}) was 0.8 ± 1.2 mm for test sites. These outcomes were associated with slight reduction of amount of KT.

Regression analysis of RecRed adjusted for Rec_{T1} and KT_{T1} showed that amount of KT measured 1 year after surgery was significantly correlated with RecRed recorded at T_2. In particular, every millimeter of KT measured at T_1 accounted for 0.2 mm of coronal advancement of GM during the period between T_1 and T_2 (95% CI: 0 to 0.3 mm; P = 0.03) (Table 2).

Untreated sites: Among control sites, 70% showed increase in recession (1 mm in 38 units and 2 mm in seven units). In the residual 19 control sites, GM remained stable. All 24 sites without recession at baseline showed an apical shift of GM at T_2. Average GR (Rec_{T2}) was 1.9 ± 1.3 mm for control sites. Slight reduction in amount of KT was observed in this group.

Regression analysis of RecRed adjusted for Rec_{T1} and KT_{T1} showed that variation in position of GM between T_1 and T_2 was not correlated with preexisting amount of KT at control sites (P = 0.96; Table 2).

Comparison between treated and untreated sites: Result of multilevel model adjusted for Rec_{T0} showed statistically significant difference in RecRed (T_0–T_2) between test and control groups of 2.2 mm (95% CI: 1.9 to 2.5 mm; P <0.001) in favor of grafted sites. Similarly, multilevel model adjusted for KT_{T0} showed statistically significant difference in KTgain at T_2 between test and control groups of 4.2 mm (95% CI: 3.6 to 4.8 mm; P <0.001) in favor of grafted sites.

Follow-up T_3 (mean = 23.6 ± 3.9 years, 18 to 35 years). Treated sites: At the end of the follow-up period (T_3), 83% of test units showed reduction in GR. Twenty-two units showed 1 mm, 18 showed 2 mm, 12 showed 3 mm, and 1 showed 4 mm of RecRed; 34 sites achieved CRC. Ten sites (15%) showed same GR depth as at baseline. The single site that showed increase of 1 mm of GR at T_1 did not show further progression at T_2. Average GR depth (Rec_{T3}) was 0.8 ± 1.0 mm. Amount of KT remained stable compared with T_2.

Table 1.

Rec, KT, and PD in Treated and Untreated Sites at Baseline (T_0), 1 Year After Surgery (T_1), During Follow-Up (T_2), and at End of Follow-Up (T_3)

Time Point	Treated Sites (n = 64) (mm)	Untreated Sites (n = 64) (mm)
Baseline T_0		
Rec_{T0}	2.3 ± 0.9 (1 to 5)	1.1 ± 1.1 (0 to 5)
KT_{T0}	1.0 ± 0.0 (1 to 1)	2.6 ± 0.8 (1 to 4)
PD_{T0}	1.0 ± 0.0 (1 to 1)	1.0 ± 0.0 (1 to 1)
Follow-up T_1		
Rec_{T1}	1.5 ± 1.1 (0 to 5)	1.3 ± 1.2 (0 to 5)
KT_{T1}	5.3 ± 1.1 (2 to 7)	2.5 ± 0.8 (1 to 4)
Difference T_0–T_1		
$RecRed_{T0–T1}$	0.8 ± 0.8 (0 to 2)	–0.2 ± 0.4 (–1 to 0)
$KTgain_{T0–T1}$	4.3 ± 1.1 (1 to 6)	–0.1 ± 0.2 (–1 to 0)
Follow-up T_2		
Rec_{T2}	0.8 ± 1.2 (0 to 5)	1.9 ± 1.3 (0 to 5)
KT_{T2}	4.8 ± 1.2 (2 to 7)	2.0 ± 0.9 (1 to 4)
PD_{T2}	1.0 ± 0.0 (1 to 1)	1.0 ± 0.0 (1 to 1)
Difference T_1–T_2		
$RecRed_{T1–T2}$	0.7 ± 0.6 (–1 to 2)	–0.6 ± 0.5 (–1 to 0)
$KTgain_{T1–T2}$	–0.4 ± 0.7 (–2 to 1)	–0.5 ± 0.5 (–1 to 0)
Difference T_0–T_2		
$RecRed_{T0–T2}$	1.5 ± 1.0 (–1 to 4)	–0.8 ± 0.6 (–2 to 0)
$KTgain_{T0–T2}$	3.8 ± 1.2 (1 to 6)	–0.6 ± 0.6 (–2 to 0)
Follow-up T_3		
Rec_{T3}	0.8 ± 1.0 (0 to 5)	1.6 ± 1.3 (0 to 5)
KT_{T3}	4.7 ± 1.5 (2 to 8)	2.3 ± 0.9 (1 to 5)
PD_{T3}	1.0 ± 0.2 (1 to 2)	1.0 ± 0.1 (1 to 2)
Difference T_2–T_3		
$RecRed_{T2–T3}$	0.1 ± 0.9 (–2 to 3)	0.3 ± 0.9 (–1 to 3)
$KTgain_{T2–T3}$	–0.2 ± 1.0 (–3 to 2)	0.2 ± 0.8 (–2 to 2)
Difference T_1–T_3		
$RecRed_{T1–T3}$	0.8 ± 0.9 (–1 to 3)	–0.2 ± 0.9 (–2 to 2)
$KTgain_{T1–T3}$	–0.6 ± 1.0 (–3 to 1)	–0.3 ± 0.8 (–2 to 1)
Difference T_0–T_3		
$RecRed_{T0–T3}$	1.5 ± 1.1 (–1 to 4)	–0.5 ± 0.9 (–3 to 2)
$KTgain_{T0–T3}$	3.7 ± 1.5 (1 to 7)	–0.3 ± 0.8 (–2 to 1)

All values presented as mean ± SD (range).

Regression analysis of RecRed adjusted for Rec_{T2} and KT_{T2} showed that variation in position of GM between T_2 and T_3 was not correlated with amount of KT_{T2} at treated sites ($P = 0.11$).

Untreated sites: Control units (48%) showed increase in recession compared with T_0. Twenty-four units had 1 mm, six had 2 mm, and one unit had 3 mm of Rec increase. Twenty-six (41%) units showed same GR depth as measured at baseline and seven showed RecRed. Only four of 24 sites with no baseline recession did not develop any recession between baseline and T_3. On average, GR depth (Rec_{T3}) was 1.6 ± 1.3 mm for control sites.

Regression analysis of RecRed adjusted for Rec_{T2} and KT_{T2} showed that variation in position of GM between T_2 and T_3 was not correlated with amount of KT_{T2} at control sites ($P = 0.62$).

Comparison between treated and untreated sites: Result of multilevel model adjusted for Rec_{T0} and KT_{T0} showed statistically significant difference in RecRed (T_0–T_3) between test and control groups of 1.7 mm (95% CI: 1.2 to 2.3 mm; $P = 0.011$) in favor of grafted sites. Baseline recession (Rec_{T0}) was statistically significant (mean difference = 0.4 mm; 95% CI: 0.2 to 0.5 mm; $P < 0.001$), whereas baseline KT (KT_{T0}) was not (mean difference = 0.1 mm; 95% CI: –0.2 to 0.4 mm; $P = 0.46$) (Table 3). No statistically significant interactions were observed between surgery and covariates Rec_{T0} and KT_{T0}.

For KT, result of multilevel model adjusted for KT_{T0} showed statistically significant difference of 3.3 mm in KTgain at T_3 between test and control groups (95% CI: 2.6 to 4 mm; $P < 0.001$) in favor of grafted sites.

PD, Plaque, and Bleeding on Probing (BOP)

PD remained stable from baseline to the end of the follow-up period in test and control sites. Overall, FMPS and FMBS remained very low (<20%) in all patients, and no clinical signs of periodontitis were detected in any part of mouth during entire period.

Table 2.

Linear Regression Analysis of RecRed at T_2 Adjusted for Rec T_1 and KT T_1 in Treated and Control Sites

Term	Estimate (SE)	95% CI	t Ratio	*P* Value
Treated sites				
Intercept	–2 (0.4)	–1 to 0.7	–0.50	0.62
Rec T_1	0 (0.1)	–0.1 to 0.2	0.24	0.81
KT T_1	0.2 (0.1)	0 to 0.3	2.31	0.03*
Control sites				
Intercept	–0.5 (0.3)	–1 to –0.01	–2.1	0.04*
Rec T_1	–0 (0.1)	–0.1 to 0.1	–0.4	0.71
KT T_1	–0 (0.1)	–0.2 to 0.2	–0.1	0.96

SE = standard error.
* Statistically significant difference.

Table 3.

Multilevel Model

Term	Estimate (SE)	*P* Value	95% CI
RecRed (T_0–T_3)			
Intercept	−1.2 (0.4)		
Pair level			
Surgery (1 = yes)	1.7 (0.3)	<0.001*	1.2 to 2.3
KT_{T0}	0.1 (0.2)	0.46	−0.2 to 0.4
Rec_{T0}	0.4 (0.1)	<0.001*	0.2 to 0.5
Variances			
σ_u^2	0 (0.1)		
σ_e^2	0.8 (0.1)		

Theoretical model: RecRed (T_0–T_3) = β_{0ij} + β_{1ij} Surgery + β_{2ij} KT_{T0} + β_{3ij} Rec_{T0}. σ_u^2 and σ_e^2 indicate variances at patient and pair level, respectively. In the theoretical model formula, subscript j refers to patient level and subscript i refers to pair level. β_{0ij} is the intercept.
* Statistically significant difference.

Dental Hypersensitivity

Treated sites: Twelve (19%) test sites had dental hypersensitivity at baseline (T_0). Number of sites experiencing dental hypersensitivity was reduced to eight (13%) at 1 year after surgery (T_1) and to four (6%) at T_2. At the end of the follow-up period (T_3), two (3%) sites continued to show dental hypersensitivity, whereas the problem was resolved in the other two sites.

Untreated sites: Nine (14%) control sites had dental hypersensitivity at baseline (T_0) and at 1 year after surgery (T_1). Number of sites experiencing dental hypersensitivity increased to 11 (17%) at T_2 and to 14 (22%) at the end of the follow-up period (T_3).

Comfort/Discomfort

With regard to comfort/discomfort perceived by patients during toothbrushing, 39 (83%) patients reported greater level of comfort in treated sites; eight (13%) individuals did not notice any significant difference between treated and untreated sites.

Survival of Experimental Units

None of the test and control units showed signs of gingivitis/periodontitis or were lost during the follow-up period and all experimental teeth were still properly functional.

DISCUSSION

This intraindividual study compares periodontal conditions of sites treated with GAPs and untreated homologous contralateral sites during a long period of time (18 to 35 years).

At baseline, test sites presented with 1 mm of free gingiva and absence of attached gingiva associated with 1 to 5 mm of GR (2.3 mm, average).

This is a condition that, reportedly,[1-4] might impair patient performance in terms of home care favoring gingival inflammation and progression of GR with time.[5-8] These sites were all treated with MFGGs or SMFGGs to increase KT width. Clinically, no differences in amount of KT were detected between the two approaches during the entire follow-up period. As expected, at 1 year, KT increased by 4.3 mm on average and was associated with 0.8-mm coronal shift of GM (Table 1). Coronal shift of GM increased significantly between T_1 and T_2, then remained stable up to T_3. Thus, this group experienced significant reduction of GR, thereby reversing baseline tendency for gingiva to recede. A significant percentage of units obtained CRC, even if root coverage was not the primary outcome of this study. RecRed and CRC could be explained by the mechanism of "creeping attachment." According to a previous study, this occurred between 1 month and 1 year after surgery, without any further coronal migration up to 5 years.[18] Outcomes from the present study show creeping attachment ongoing for periods ranging from 10 to 27 years (T_1–T_2), with no further coronal migration of GM in the time span of 18 to 35 years (T_3). Linear regression analysis showed that every millimeter of KT provided by FGG and measured at T_1 accounted for 0.2 mm of coronal advancement of GM during time period T_1–T_2 (Table 2). No further influence of KT on RecRed was noted during the last observation period T_2–T_3, showing that creeping attachment seemed to become exhausted during final 8 years of observation.

These data could help determine the amount of expected creeping attachment when using FGG, thereby providing guidelines for the apico-coronal extent of graft to be placed. Ample variability in the apico-coronal dimension of FGG used in this clinical study (mean KT at 1 year: 5.3 ± 1.1; range 2 to 7 mm) is explained by lack of established guidelines on amount of KT needed to achieve expected outcome and, especially, lack of knowledge on healing dynamics of FGG, in particular on average apico-coronal contraction of FGG. This is relevant because reduced extension of FGG could be insufficient to achieve a result, but excessive extension could cause apical misalignment of alveolar mucosa with functional and esthetic problems as demonstrated by Cortellini et al.[19]

Findings of this study support the hypothesis that modification from thin to thick periodontal biotype induced by placement of FGG can favor long-term stability and healthy condition of gingiva, confirming observations from other investigations[4,10,20] about the beneficial role of attached gingiva on stability of periodontal tissues.

In this study, of 64 treated sites, 14 were in the maxillary arch and 50 in the mandibular arch. This

choice can be explained by the fact that recession defects in the mandibular arch are often associated with reduced amount of KT and, therefore, gingival augmentation is considered the primary goal of surgical treatment. Conversely, different surgical approaches, such as root coverage procedures, are performed to treat maxillary recessions mainly to achieve esthetic outcomes, which was not the objective of this study.

Untreated homologous contralateral sites experienced deterioration of marginal soft tissues that consistently shifted apically, resulting in increment of GR prevalence and GR depth. Eighteen sites with no recession at baseline ended this study with GR, and 33 sites with baseline recession experienced progression of apical shift of GM. One third of control sites presented at baseline with normal position of GM, and average KT was 2.6 ± 0.8 mm (1 to 4 mm). Control teeth presented at baseline with gingival conditions at lesser risk for development or increase of GR defects than test sites.

Variation in position of GM between T_1 and T_2 was not correlated with preexisting amount of KT at control sites (Table 2). During the last observation period, seven (11%) sites showed slight RecRed that could be partially explained by the strict PM program with time and improved patient ability to brush efficiently, minimizing local traumatic impact on marginal gingiva that resulted in high standards of plaque control and absence of marginal gingival inflammation at treated and untreated sites.

The significant difference between test and control sites in terms of GM stability can be explained with the clinical decision to place FGGs to modify gingival biotype in test sites as discussed above (Table 3). It is also important to emphasize that slow development of GR in untreated sites with time was observed in highly compliant, mainly non-smoking patients (91%) with optimal standards of oral hygiene (FMPS/FMBS <20%). All patients were enrolled in a stringent PM program for 4 to 6 months. At each recall visit, patients were informed of their periodontal conditions with particular care devoted to monitoring untreated sites. As absence of marginal inflammation was assessed for these sites, patients opted to maintain their initial decision and avoid surgical treatment throughout the observation period.

During the study period, all new scientific information on professional and home care and new sophisticated devices developed to improve quality of toothbrushing were implemented to further reduce risk of developing GRs. Amount of apical displacement of GM observed in untreated sites during such a long time period was small in size and did not alter prognosis of teeth; survival was 100% in both study arms and periodontal conditions were equally stable in terms of PD and BOP.

Patient-perceived benefit in terms of absence of hypersensitivity was noted in 10 of 12 test sites, whereas in control sites dental sensitivity increased slightly with time. Increased KT thickness in grafted sites positively affected oral hygiene practices of patients; of 47 patients, 39 (83%) reported improved comfort levels during toothbrushing on grafted sites.

CONCLUSIONS

The following conclusions can be drawn from this 18- to 35-year long-term controlled study on well-maintained patients with thin gingival biotype: 1) use of GAPs (FGGs) on sites presenting with recession defects associated with absence or reduced amount of attached gingiva was effective in providing consistent increase of KT and reduction of GR up to CRC; 2) position of augmented and coronally migrated gingiva remained stable for up to 35 years and was associated with reduction of number of sites experiencing dental hypersensitivity as well as improved comfort in toothbrushing; 3) contralateral untreated sites presenting with variable amounts of KT at baseline showed a tendency for apical migration of GM with development of new GRs or progression of existing recession defects; 4) increment of GR depth of untreated sites was clinically and statistically significant but did not impact either periodontal health or tooth survival; 5) GAPs should be considered in clinical conditions in which stability of GM is perceived of primary relevance; and 6) external validity of these outcomes should be considered with caution because the experimental population consisted of patients selected for their motivation and compliance, and experimental teeth were all natural teeth, without restorations or crowns, and had not undergone orthodontic treatments.

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