Influence of vestibular depth on the outcomes of root coverage therapy: A prospective case series study

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One-sentence Summary: Shallow vestibular depth negatively impacts the outcomes of root coverage therapy.

[1]

Abstract

Background: To investigate the influence of vestibular depth (VD) on the outcomes of root coverage therapy.

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Material and methods: Patients presenting gingival recession defects (GRD) with a minimum depth of 2mm underwent root coverage therapy consisting of a coronally advanced flap plus a connective tissue graft (CAF+CTG). Clinical examinations were performed, and intraoral scans were obtained at baseline, 3 and 6 months after surgery to assess changes in probing depth (PD), keratinized tissue width (KTW), recession depth (RD), GRD area, marginal gingiva thickness (MGT), and VD. The influence of VD on % root coverage (%RC) and the likelihood of achieving complete root coverage (CRC) were explored.

Results: Twenty patients were enrolled. A total of 44 teeth were treated. RD decreased and MGT increased in all treated sites. At 6 months, mean %RC was 87.47±18.37 and CRC was observed in 61.4% of sites. Mean baseline VD was 7.33±2.67mm. Mean VD reduction from baseline to 6 months was 1.98±1.27mm. %RC and CRC were significantly correlated with baseline VD. Each additional 1 mm of baseline VD implied a gain of 6.58% for %RC and increased 2.75 times the probability of achieving CRC. Narrow baseline KTW and mandibular arch location were associated with inferior treatment outcomes.

Conclusions: Lower %RC and likelihood of achieving CRC can be expected after root coverage therapy via CAF+CTG in sites presenting a shallow vestibulum.

KEYWORDS

Aesthetics; connective tissue graft; coronally advanced flap; gingival recession; root coverage

INTRODUCTION

A gingival recession defect (GRD) is characterized by partial exposure of the root surface to the oral cavity because of apical migration of the gingival margin respective to the cementoenamel junction (CEJ). Prevalence and incidence of GRDs are high in the general population.[1,2] The etiology of GRDs is multifactorial, including predisposing and precipitating factors such as traumatic tooth brushing technique and other deleterious habits (e.g., finger picking), irritants (e.g., lip or tongue piercing), local inflammation and subsequent periodontal breakdown derived from biofilm accumulation, tooth malposition, and high frenal attachment.[3] If left untreated, the probability of progression of GRDs is high.[4] Successful root coverage outcomes can be achieved with different surgical protocols.[5] Corrective surgical therapy of GRDs primarily aims at shifting the location of the gingival margin (GM) to a more coronal location, achieving complete root coverage (CRC) or partial root coverage if CRC is not feasible, with a shallow probing depth and a pleasant soft tissue integration.[6]

Proper planning of root coverage procedures should include a careful analysis and consideration of local factors that may influence the execution of the surgical intervention and the outcomes of therapy. Among these factors, solid evidence supports the importance of interproximal bone and attachment level, marginal gingiva thickness (MGT), width of attached gingiva (AG), and recession defect depth (RD) as key elements that can be used to design the surgical plan and even predict the therapeutic result.[7-11] Tooth malpositioning and tooth type (molar vs. non-molar) have also been regarded as relevant predictive factors. [12-14]

In a recent publication, Aroca et al. pointed out that labial muscular pull and a shallow vestibular depth (VD) may negatively influence the results of root coverage procedures.[13] Although shallow VD is frequently found in association with GRDs, particularly in mandibular sites, evidence regarding its impact on the outcomes of root coverage procedures is scarce. Therefore, the primary objective of this study was to evaluate the influence of VD on the outcomes of root coverage therapy consisting of a coronally advanced flap plus an autogenous connective tissue graft (CAF+CTG).

MATERIAL AND METHODS

Experimental design and setting

This clinical investigation was designed as a pre-post case series study and is reported in compliance with the Preferred Reporting of Case Series in Surgery (PROCESS) guidelines.[15] This study was registered in clinicaltrials.gov under code NCT04813302. The clinical component of the study was conducted in a private practice setting affiliated with the Universitat Internacional de Catalunya (UIC) in Barcelona (Spain). The study took place between September 2019 to March 2021.

Ethical approval

The study protocol was reviewed and approved by the Ethical Committee of UIC (PER-ECL-2019-04), and was conducted in accordance with the Helsinki Declaration of 1975, as revised in 2013.

Outcomes of interest

The main outcomes of interest were percentage of root coverage (%RC) and whether complete root coverage (CRC) was achieved.

Sample size calculation

The number of independent teeth that would be required for an estimated linear regression model to reach a power of 80% in detecting r=0.5 as a significant correlation was calculated. This analysis rendered a total of 27 teeth. Assuming that each patient would provide an average of two teeth (n=2), this sample size should be corrected because of intra-subject dependence. A correcting factor D of the sample size was estimated using the formula D = 1 + (m-1) x ICC by Pandis where m is the number of teeth per subject and ICC the intra-class correlation coefficient. Becasue ICC of RC% could not be extracted from previous studies, we assumed a moderate correlation (=0.5).[16,17] Therefore, D=1.5 and the sample size for independent teeth should be increased +50%, obtaining an ideal sample size of at least 40 teeth.

Eligibility criteria and recruitment

Adult patients (≥18 years) presenting at least one single buccal Cairo RT1 GRD with a minimum of 2 mm of RD on single rooted teeth with identifiable CEJ were consecutively enrolled. The exclusion criteria were as follows: 1) Full-mouth plaque and bleeding score >20%; 2) Smoking ≥10 cigarettes a day; 3) Systemic contraindications for periodontal surgery; 4) Taking medications known to affect gingival homeostasis or interfere with wound healing; 5) Pregnancy; 6) Active orthodontic therapy. 7) Previous periodontal surgery, caries, or restorations in the experimental site(s); 8) Malpositioned/crowded teeth.

Prior to enrollment, all patients were informed of the purpose and timeline of the study, and were required to read, understand, and sign an informed consent.

Clinical procedures

Cause-related periodontal therapy was completed one month prior to surgery. Patients received a pre-surgical prophylaxis and oral hygiene instructions, including proper tooth brushing, if necessary.

All surgical interventions were performed by the same operator (GB). Briefly, after local anesthesia was achieved, one intrasulcular and two vertical releasing incisions lateral to the papillae adjacent to each GRD were made. Subsequently, a split-full-split-thickness flap was elevated beyond the mucogingival junction (MGJ). Periosteal scoring was done and blunt dissection into the vestibular lining mucosa was completed to eliminate muscle tension, so that the mucosal flap margin could be coronally positioned and passively stabilized. The papillae were de-epithelialized and EDTA 24% ‖

was applied for 2 minutes over the exposed root surface for conditioning. A CTG of 1mm in thickness was harvested from the palatal mucosa by means of extraoral de-epithelization of a free gingival graft.[18] The graft was trimmed to fit the defect area, placed over the root, sutured to the interdental papillae, ¶ and subsequently covered with the flap, which was advanced to position its margin at approximately 1 to 2 mm coronal to the CEJ with a sling suture, followed by simple interrupted sutures to close both vertical releasing incisions [Figure 1].#

Patients were instructed to avoid any mechanical trauma, including tooth brushing in the surgical site, for 2 weeks. Anti-inflammatory medication (Ibuprofen 600mg oral, three times per day, as required) was prescribed and patients were instructed to rinse with 0.12% Chlorhexidine two times per day for 2 weeks. Sutures were removed after 2 weeks, and patients were allowed to resume their regular oral hygiene routine using a soft

toothbrush. Patients were recalled at 1, 3 and 6 months for intraoral evaluation, supragingival plaque control, and data collection.

[‖]PrefGel®, Straumann, Basel, Switzerland

[¶] 7-0 Polyglactin 910, Vycril, Ethicon, Johnson and Johnson, New Brunswick, NJ, USA

[#]6-0 Polypropylene, Prolene, Ethicon, Johnson and Johnson, New Brunswick, NJ, USA

Data collection

An interview was conducted during the pre-surgical visit to obtain information regarding age, sex, medical and dental history (including history of periodontal surgical therapy), current use of medications, and exposure to tobacco.

The following mid-buccal clinical measurements were recorded at baseline, 3 and 6 months after surgery using a periodontal probe:[**] probing depth (PD), keratinized tissue width (KTW), and clinical attachment loss (CAL). Additionally, a digital dental scan of the whole arch was performed with an intraoral scanner[††] (using a bilateral mouth retractor [‡‡]) to obtain standard tessellation language (STL) files. STL files were transferred into a digital imaging software.[§§] Baseline and corresponding follow-up scans of each clinical case were digitally superimposed by using anatomic landmarks as reference points. Digital linear and volumetric measurements to determine soft tissue dimensional changes were performed by a single, calibrated examiner (JM). The examiner was trained using 15 casts with GRDs that were not included in this study. Two sets of assessments were repeated in an interval of 24 hours; a difference of ≤0.5 mm in at least 90% of the cases was acceptable.

The following measurements were made:

- RD was measured from CEJ to the GM in a mid-buccal cross section. [19]
- Recession area was measured by delineating the denuded root surface area between the GM and the CEJ.
- MGT was measured in an individually defined area at 1 and 2 mm apical to the GM.
- VD was measured from the GM to the point of greatest concavity of the mucosal fold [Figure 2].

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Statistical analysis

Absolute and relative frequencies for categorical variables, and mean, standard deviation, range, and median for continuous variables were calculated.

Simple binary logistic regression models were estimated using generalized estimation equations (GEE) to assess the probability of CRC at each follow-up time as a function of the outcomes of interest at baseline. Unadjusted estimates of Odds Ratio (OR) and 95% confidence intervals (CI) were obtained using Wald's Chi2 statistic. Then, a multiple regression model allowed to adjust the results for all the independent variables simultaneously. For the dependent variable %RC, simple linear regression models were applied, also using a GEE approach, in which estimates were obtained for the beta coefficients of the regression with 95% CI, and later adjusted through a multiple regression model. The GEE analysis methodology applied was justified by the intra-subject correlation typical of a multi-level data structure. The level of significance used in the analysis was 5% ($\alpha = 0.05$).

A linear regression model such as the one previously described to evaluate the influence of each variable of interest on CR% would reach a power of 97.9% to detect a correlation of moderate magnitude (r=0.5) as significant with a confidence of 95%. Due to the multi-level structure of the data (several sites per patient), the power had to be corrected so, assuming a moderate intra-subject correlation ($\rho = 0.5$), 80% power could be reached.

RESULTS

The study population consisted of 20 patients (5 males and 15 females). All patients were non-smokers. No patients were lost to follow-up. A total of 44 GRDs were treated, of which 65.9% were mandibular and 34.1% maxillary. Regarding tooth type, 29.5% were incisors, 27.3% were canines and 43.2% were premolars [Table 1]. No postoperative healing complications were observed.

A statistically significant change between baseline, 3 and 6 months was observed for all variables [Table 2]. Mean RD was 2.74±0.77mm at baseline, 0.42±0.64mm at 3 months, and 0.40±0.62mm at 6 months. Therefore, from baseline to the 3-month follow-up, mean RD reduction was -2.32±0.73mm ($p<0.001$), while this value from baseline to 6 months was -2.35±0.72mm ($p<0.001$), which is reflective of gingival margin stability between 3 and 6 months after surgery. Mean %RC was 86.46±19.31% at 3 months and 87.47±18.37% at 6 months

(p<0.001). CRC was observed in 61.4% of teeth at 6 months (p<0.001). Mean recession area was 6.92±3.99mm^2 at baseline, 1.79±2.70mm^2 after 3 months and 1.53±2.36mm^2 after 6 months (p<0.001). Mean VD was 7.33±2.67mm at baseline, was 2.62±1.28mm at 3 months, and 1.98±1.27mm at 6 months.

Simple linear regression revealed that %RC after 6 months was significantly correlated with baseline VD (p <0.001) [Table 3A]. In fact, each additional mm of VD at baseline implied, on average, an increase of 6.58% in %RC [Figure 3A]. Furthermore, %RC was correlated with baseline KTW. Mean %RC was 98.8% and 80.1% in maxillary and mandibular sites, respectively (p <0.001). After neutralizing confounding factors with a multiple linear regression model, VD was the only variable retaining statistical significance (p<0.01) [Table 3B and 3C].

Similar correlations were observed for CRC after 6 months. Simple logistic regression revealed that VD is the variable that most influenced the likelihood of achieving CRC [Table 4A]. The estimate indicated that each additional 1mm of VD increased 2.75 times the probability of achieving CRC (p=0.009) [Figure 3B]. CRC was not achieved in any site with a baseline VD <6mm. Tooth location was also a determining factor. Moreover, each additional mm of baseline KT multiplied by 3 the probability of achieving CRC (p=0.037). After neutralizing confounding factors with a multiple linear regression model, CRC was not statistically correlated with any of the included variables [Table 4B]. Nevertheless, VD displayed a strong tendency towards significance (p<0.067).

DISCUSSION

To the best of our knowledge, this is the first clinical study designed to evaluate the effect of VD upon the outcomes of a root coverage procedure consisting of CAF+CTG. Digital methods enabled the reliable assessment of variables that have not been frequently reported in the existing literature on root coverage procedures, such as changes in VD, gingival thickness, or recession area. Recently published studies support the reliability of data collection and analysis of the anatomical baseline features of GRD and subsequent outcomes of therapy using STL files obtained with intraoral scanners.[20-22]

In the present study, mean %RC was 87.47% at 6 months, and CRC was achieved in 27 out of 44 sites. These findings are comparable to those reported by other authors after the treatment of GRDs with CAF+CTG.[23-25] The main purpose of the study was to evaluate whether VD was correlated with %RC and CRC. Our results showed that, among all variables analyzed, VD is the most relevant and influential factor for both %RC and CRC. In both adjusted models, the %RC increases almost 7% and the probability of achieving CRC is multiplied almost 3 times for each additional mm of VD.

Current evidence indicates that CAF+CTG is the gold standard procedure for the treatment of GRDs.[26-28] Nevertheless, it should be acknowledged that most studies on root coverage therapy via CAF+CTG refer to the treatment of maxillary GRDs and significantly higher %RC and CRC after treating GRDs with CAF+CTG have been observed in the maxilla compared to mandibular sites.[14,26,30] The use of CAF alone or in combination with CTG has rarely been reported in the mandible, while gingival augmentation in mandibular sites using a free gingival graft has been extensively studied.[28,29] Possibly, the difficulty of effectively displacing and stabilizing a flap in a coronal position in mandibular sites influences the clinical decision-making process for many clinicians.[13] A passive flap is of utmost importance to achieve a favorable outcome after a CAF procedure. In fact, it has been demonstrated that higher flap tension generally leads to lower %RC, while lower flap tension is associated with higher recession depth reduction.[31] Likewise, the extent of coronal advancement over the CEJ is of paramount importance.[32] According to Pini-Prato et al., passive flap advancement 2mm coronal to the CEJ results in 100% root coverage.[33] A shallow VD contributes to increase the amount of flap tension and restrains the coronal advancement of a flap, thus limiting the amount of root coverage than can be predictably achieved, which explains the observations hereby reported.

Another relevant finding of our study is that CRC was not observed in any site with a baseline VD <6 mm. Interestingly, all sites presenting VD <6 mm were in the mandible. Different techniques have been proposed to overcome the unfavorable anatomical conditions that are frequently found in mandibular sites, particularly in the anterior region, such as thin gingival phenotype, lack of KT and shallow VD.[34-36] However, clinical evidence is scarce. Much of the limited data available on the outcomes of root coverage procedures in the mandible pertains to tunneling techniques.[36-40] Interestingly, all these studies are case series and the mean %RC ranged from 83.25% to 100% in Miller Class I defects.[7] The main advantage of the tunnel approach is that, by leaving the CTG partially exposed or by closing the tunnel laterally, minimal to no coronal advancement is required, and therefore minimal tension is applied to the flap. Additionally, KTW augmentation can be achieved when the CTG is left partially uncovered.[38,41,42] Zucchelli et al. conducted a randomized clinical trial aimed at evaluating the outcomes of CAF with or without removal of labial submucosal tissue (LST) for the treatment of GRDs at mandibular incisors. The addition of LST removal to CAF+CTG resulted in a tension-free flap leading to a significantly higher chance of achieving CRC as compared to CAF+CTG alone (88% vs 48%). Surprisingly, limited post-operative morbidity was reported in both groups.[35] These results are difficult to compare to those obtained in our study, in which maxillary and mandibular sites were included and no LST was performed. Regardless, both studies point out the critical importance of minimal of flap tension to achieve adequate coronal flap mobilization and obtain predictable RC, which is more difficult to achieve in the presence of shallow VD. A

recent study that evaluated the influence of VD on RC showed that the addition of LST to CAF not only may improve %RC but also increase VD.[43] One of the shortcomings of this study was the method used to measure VD. This assessment was made intraorally using a periodontal probe while the lip was pulled until the muscles were almost perpendicularly oriented toward the buccal surface of the alveolar bone. As a result of this manual pulling, the VD can change since the position and the force applied with the fingers can vary. In our study the amount of pressure and force applied was standardized by using a bilateral retractor with the teeth in occlusion while taking the intraoral scan.

Our findings revealed that tooth location was a determining factor that influenced %RC. On average, almost 19% less root coverage was observed in mandibular teeth compared to maxillary sites ($p < 0.001$). From these results, it can be inferred that CAF is less predictable in mandibular sites. In accordance with this observation, a recent systematic review evaluating the effectiveness of different approaches to treat GRDs in the anterior mandible showed that laterally positioned flap + CTG and tunnel + CTG achieved a higher mean %RC (91.2% and 89.4%, respectively) compared to CAF+CTG (78.9%).[44] It must be acknowledged that VD is typically lower in mandibular sites compared to maxillary sites. Therefore, it can be argued that the observed effect of arch location in the outcomes of root coverage therapy is directly related to VD.

Another relevant finding from our study is the VD reduction after 3 and 6 months, which was 2.62±1.28mm and 1.98±1.27mm, respectively. A reduction in VD can be detrimental for plaque control by impeding proper oral hygiene.[45,46]

Several limitations of the present study should be noted. The low sample size could be a source of bias. Nevertheless, an appropriate statistical power and a strong significance level support drawing sufficiently reliable statements on the influence of VD on treatment outcome. Moreover, pre-operative MGT measurements were not recorded and their effect on %RC could not be assessed. A short follow-up period is another potential limitation. Futures studies with longer follow-ups are warranted to assess the effect of time on the stability of the clinical outcomes. Although internal validity is supported by strict eligibility criteria and a single operator executing a specific technique, external validity should be confirmed with multicenter clinical trials including a range of other surgical procedures. Finally, digital assessment of baseline VD involved certain degree of uncertainty due to the absence of reliable anatomical landmarks. An attempt to attenuate the impact of this factor on the quality of the data was made by training and calibrating the examiner to increase the probability of making reproducible measurements.

CONCLUSIONS

The findings of this study indicate that VD is a significant predictor for the outcomes of root coverage therapy via CAF+CTG. Other anatomical factors such as mandibular arch location and reduced keratinized tissue width negatively affected treatment outcomes, as well. The effect of these factors on the outcomes of other surgical interventions for root coverage should be further explored in future clinical studies.

Conflict of interest and source of funding statement

The authors declare that they have no conflict of interest. The study was self-funded.

Author contribution

G.Blasi, contributed to conception, design, surgery, data interpretation, drafted and critically revised the manuscript. A.Monje and J.Muñoz-Peñalver have been involved in data collection, data interpretation and data analysis. T.W.Oates, G.Avila-Ortiz and J.Nart have been involved in data interpretation, drafting the manuscript and revising it critically and have given final approval of the version to be published. All authors have made substantial contributions to conception and design of the study.

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FIGURE 1. Treatment sequence. A. Baseline, B. Flap elevation and positioning of CTG, C. Flap stabilization with sutures, and D. 6-month follow-up.

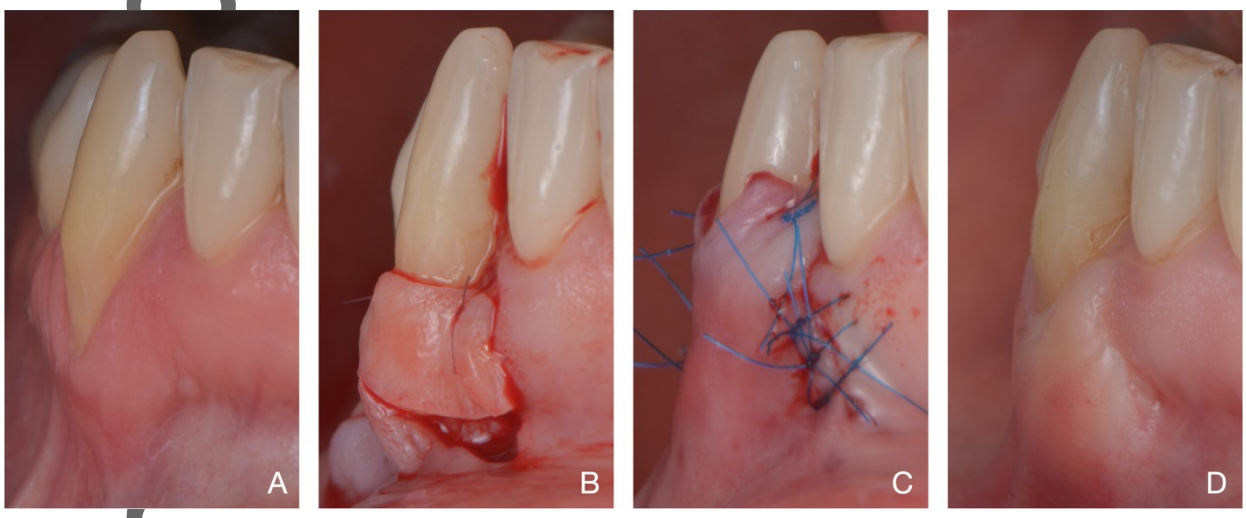

FIGURE 2. Digital superimposition of STL files and linear measurements

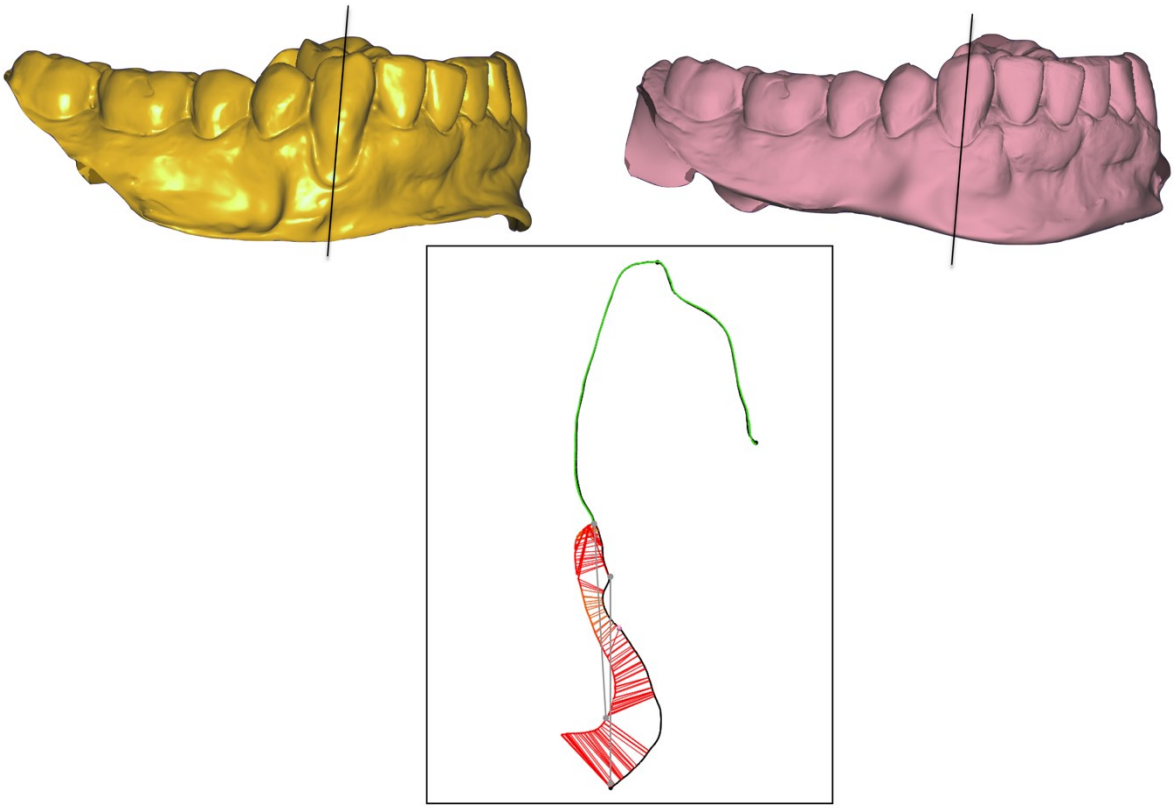

FIGURE 3.A. Correlation between baseline VD and %RC at 6 months.

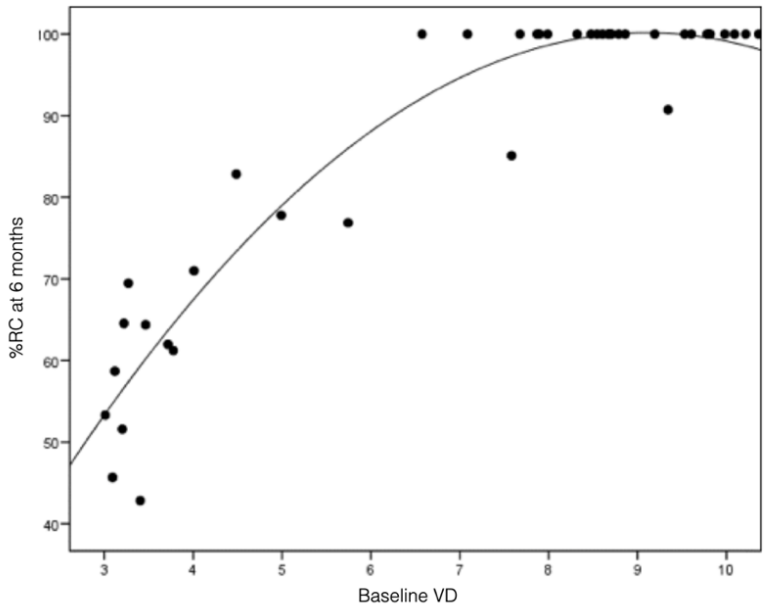

FIGURE 3B. Correlation between baseline VD and CRC at 6 months.

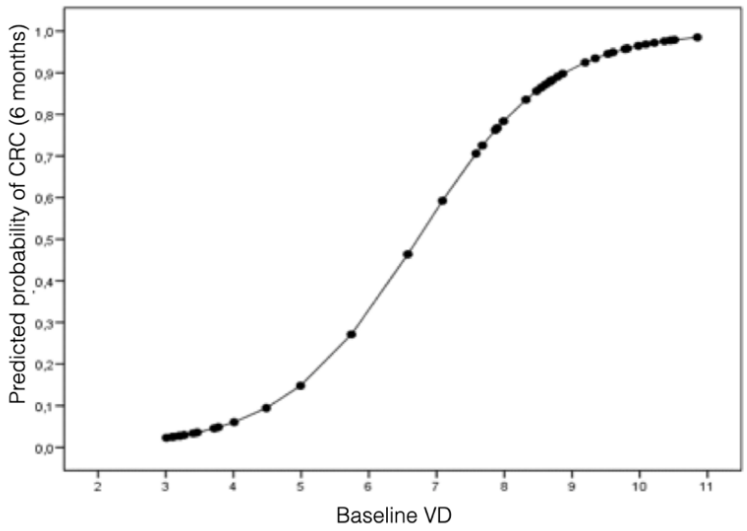

TABLE 1. Number of GRD per patient

	N	%
Total	20	100
1	4	20,00
2	10	50,00
3	5	25,00
5	1	5,00

TABLE 2. Mean ± SD, relative frequencies and timepoint differences. Multiple comparisons were adjusted using Bonferroni from GEE models (p-values).

	Baseline	T1-T0	3 months	T2-T1	6 months	T2-T0
PD	1.14 ± 0.35	0.25 ± 0.49 p<0.001‡	1.39 ± 0.49	0.02 ± 0.26 p=1.000	1.41 ± 0.50	0.27 ± 0.54 p<0.001‡
RD	2.74 ± 0.77	-2.32 ± 0.73 p<0.001‡	0.42 ± 0.64	-0.03 ± 0.08 p=0.052	0.40 ± 0.62	-2.35 ± 0.72 p<0.001‡
CAL	3.87 ± 0.85	-2.03 ± 0.98 p<0.001‡	1.85 ± 0.77	-0.04 ± 0.29 p=1.000	1.81 ± 0.70	-2.06 ± 0.99 p<0.001‡
KTW	2.11 ± 0.78	0.91 ± 0.80 p<0.001‡	3.02 ± 1.11	0.07 ± 0.25 p=0.167	3.09 ± 1.05	0.98 ± 0.76 p<0.001‡
RA	6.92 ± 3.99	-5.13 ± 3.07 p<0.001‡	1.79 ± 2.70	-0.26 ± 0.60 p=0.021*	1.53 ± 2.36	-5.38 ± 3.05 p<0.001‡
%RC	--	--	86.5 ± 19.3	1.01 ± 2.87 p=1.000	87.5 ± 18.4	--
CRC	--	--	61.4%	p=1.000	61.4%	--
VD	7.33 ± 2.67	p<0.001‡	2.62 ± 1.28	p<0.001‡	1.98 ± 1.27	p<0.001‡
MGT 1 mm	--	p<0.001‡	1.18 ± 0.35	p<0.001‡	1.29 ± 0.35	
MGT 2 mm	--	p<0.001‡	1.48 ± 0.41	p<0.001‡	1.53 ± 0.44	

*p<0.05; †p<0.01; ‡p<0.001

PD (Probing Depth), RD (Recession Depth), CAL (Clinical Attachment Level), KTW(Keratinized Tissue Width), RA (Recession Area), %RC (% Root Coverage), CRC (Complete Root Coverage), VD (Vestibular Depth), MGT (Marginal Gingiva Thickness)

TABLE 3A. GEE simple linear regression model for %RC at T2 respective to T0 parameters

	Beta	IC 95%		Significance
VD	6.20	4.78	7.61	**<0.001**‡
KTW	8.30	1.66	14.9	**0.014***
Arch				
Maxilla	0.00			
Mandible	-17.50	-27.74	-7.28	**0.001**†
Tooth type				0.988
I	0.00			
C	0.28	-14.3	14.9	0.970
PM	0.87	-14.1	15.8	0.910

*p<0.05; †p<0.01; ‡p<0.001

Abbreviation: VD (Vestibular Depth), KTW (Keratinized Tissue Width) I (Incisors), C (Canines), P (Premolars)

TABLE 3B. GEE multiple linear regression model for %RC at T2 respective to T0 parameters in maxillary sites

		Beta	IC 95%		Significance
VD		3.17	1.67	4.67	**<0.001**‡
KTW		0.08	-0.56	0.72	0.802
Tooth type					**0.024***
	I	0.00			
	C	4.52	1.04	8.00	**0.011***
	PM	1.15	-0.05	2.35	0.061

*p<0.05; †p<0.01; ‡p<0.001

Abbreviation: VD (Vestibular Depth), KTW (Keratinized Tissue Width) I (Incisors), C (Canines), P (Premolars)

TABLE 3C. GEE multiple linear regression model for %RC at T2 respective to T0 parameters in mandibular sites

		Beta	IC 95%		Significance
VD		7.17	5.44	8.90	**<0.001**‡
KTW		-0.43	-3.82	2.96	0.803
Tooth type					**0.013***
	I	0.00			
	C	5.78	0.42	11.1	**0.034***
	PM	0.91	-7.65	9.47	0.835

*p<0.05; †p<0.01; ‡p<0.001

Abbreviation: VD (Vestibular Depth), KTW (Keratinized Tissue Width) I (Incisors), C (Canines), P (Premolars)

TABLE 4A. GEE simple binary logistic regression for CRC at T2 respective to T0 parameters

	OR	IC 95%		Significance
VD	2.75	1.29	5.88	**0.009**†
KTW	3.08	1.07	8.82	**0.037***
Arch				
Maxilla	0.00			
Mandible	0.06	0.01	0.57	**0.014***
Tooth type				0.842
I	0.00			
C	1.71	0.28	10.4	0.669
PM	1.47	0.25	8.56	0.669

*p<0.05; †p<0.01; ‡p<0.001

Abbreviation: VD (Vestibular Depth), KTW (Keratinized Tissue Width) I (Incisors), C (Canines), P (Premolars)

TABLE 4B. GEE multiple binary logistic regression for CRC at T2 respective to T0 parameters

	OR	IC 95%		Significance
VD	2.73	0.93	8.02	0.067
KTW	1.13	0.43	3.00	0.804
Arch				
Maxilla	0.00			
Mandible	0.75	0.03	21.2	0.866

*p<0.05; †p<0.01; ‡p<0.001