

ORIGINAL RESEARCH

Clinical analysis of coronally positioned flap for root coverage in smokers and non-smokers: A comparative study

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ABSTRACT

Background: Gingival recession is significantly more common among smokers, while cigarette smoking has been shown to negatively influence healing following periodontal therapeutic procedures as compared to non-smokers. The objective of this study was to evaluate the influence of cigarette smoking on the outcome of coronally positioned flap (CPF) in the treatment of Miller Class I gingival recession defects.

Materials and Methods: Ten current smokers (≥ 10 cigarettes daily for at least 5 years) and 10 non-smokers (never smokers), each with one 3 to 4-mm Miller Class I recession defect in an upper canine or bicuspid, were treated with CPF. At baseline and 6 months, clinical parameters, probing depth (PD), clinical attachment level (CAL), recession depth (RD), and apico-coronal width of keratinized tissue (KT) were determined.

Results: Intra-group analysis showed that CPF was able to reduce RD and improve CAL in both groups ($p < 0.001$). Intergroup analysis demonstrated that smokers presented greater residual RD at 6 months and lower percentage of root coverage (60.09% versus 76.05%; $p < 0.05$). No smokers obtained complete root coverage compared to 30% of non-smokers ($p < 0.05$).

Conclusion: Within the limits of present study, it can be concluded that cigarette smoking may present negative impact on root coverage outcome by CPF as compared to non-smokers and therefore represent one more challenge to periodontal plastic therapy.

Key words: Gingival recession/therapy, flap, smoking/adverse effects

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INTRODUCTION

Gingival recession that is apical displacement of marginal tissue causes root exposure and esthetic concern to patients. It not only poses one of the major esthetic problems but also creates a functional deformity by destruction of the attached gingiva as well as it causes root hypersensitivity, root surface abrasion, higher incidence of root caries, compromised plaque control and esthetically compromised restorative treatment^[1].

Smokers, who are at greater risk for destructive periodontal disease, have been repeatedly shown to have more gingival recession than non-smokers, due to vasoconstriction and less inflammatory response caused by nicotine.

Therefore, smokers represent a population with potentially great root-coverage treatment needs^[2]. Smoking has been shown to impair revascularization during soft and hard tissue wound healing, which is critical for periodontal plastic, regenerative, and implant procedures. The oral tissues of smokers are exposed to high nicotine concentrations that negatively affect local cell populations. A myriad of periodontal surgical procedures have been developed over the years that demonstrated predictable root coverage like various pedicle grafts as lateral, oblique, coronal, double papillae and semilunar to free gingival graft, subepithelial connective tissue graft and guided tissue regeneration. Coronally positioned flap procedure has

the advantage of average root coverage ranged from 75% to 82.7%, with 24% to 95% of sites achieving complete root coverage^[3]. Here, an attempt has been made to clinically compare and evaluate the predictability of Coronally Positioned Flap root coverage procedure in the treatment of Miller's Class I recession defects in smokers and non-smokers.

MATERIALS & METHODS PATIENT SELECTION

Twenty systemically healthy individuals were selected from patients referred to the Department of Dentistry, SKIMS Medical College and Hospital, Bemina Srinagar, ten of the subjects were smokers (≥ 10 cigarettes daily for at least 5 years) and ten were non-smokers (never smoke) who demonstrated isolated Miller Class I recession defect (3 to 4 mm in depth) involving maxillary canine or premolar. The treatment modality for each gingival recession is Coronally Positioned Flap. The individuals selected were in the age range of 22 to 53 years. Patients were in excellent general medical health with no detectable systemic contraindications to surgical treatment. All the patients agreed to participate in the study and a signed surgical consent form was obtained.

The study protocol involved a screening appointment to verify eligibility, followed by initial therapy to establish optimal plaque control and gingival health conditions, fabrication of stent for clinical parameters, surgical therapy, postoperative professional plaque control, and final evaluation 6 months after the surgical intervention.

Clinical Parameters: At baseline and throughout the study,

- Gingival index (Loe and Sillness 1963).
- Plaque index (Turseky-Gilmore-Glickman modification of Quingley Hein Plaque Index 1970) were used to monitor oral hygiene and gingival health conditions.

The following clinical parameters were assessed on the mid-buccal aspect of the study teeth using a periodontal probe and a custom stent for probe positioning:

- **Gingival recession depth (RD):** The score was measured vertically as the distance from the cemento-enamel junction (CEJ) to the gingival margin (GM).
- **Probing depth (PD):** The score was measured vertically as the distance from gingival margin (GM) to the bottom of the gingival sulcus.
- **Clinical attachment level (CAL):** The score was measured vertically as the distance from cemento-enamel junction (CEJ) to the bottom of the gingival sulcus.
- **Apico coronal width of keratinized Gingiva (KG):** The score was measured vertically from the gingival margin (GM) to the mucogingival junction.

- **Percentage of root coverage:** The score was obtained according to the following formula at 6 months post operatively.

$$\frac{\text{Pre operative recession depth} - \text{Post operative recession depth}}{\text{Pre operative recession depth}} \times 100$$

Surgical Technique

All recession defects were treated with Coronally Positioned Flap technique with releasing incisions. Prior to flap elevation, the exposed root surface was instrumented with hand instruments to minimize root convexity. After adequate anesthesia at the recipient site, the flap design started with an intrasulcular incision at the vestibular aspect of the involved teeth and extended horizontally up to interdental gingiva, then giving horizontal incision at the base of papilla up to line angle of adjacent tooth, mesial and distal to the defect. Two oblique apically divergent relaxing incisions extending beyond the mucogingival junction complete the flap design. A full thickness trapezoidal mucoperiosteal flap was elevated until the crest of marginal bone was reached on the mid-buccal aspect of the tooth under treatment, then a split thickness flap was extended further apically to allow the flap to be positioned coronally at CEJ without tension. The deepithelium of the interdental papillae was done to provide a proper wound bed for healing. Then the flap was coronally advanced so that the tissue margin slightly covered the CEJ. Flap was now sutured passively into position using 5.0 silk sutures (Ethicon). A piece of dry foil was placed over the sutures. Area was covered with a non-eugenol periodontal dressing (Coe-Pak).

Post-Operative Protocol

Routine post-operative instructions were given.

- Systemic antibiotics, Amoxicillin 500mg TID daily for five days. Anti-inflammatory analgesic, (Ibuprofen 400 mg) was prescribed 8 hourly.
- 0.2% chlorhexidine mouth rinses twice a day for 4 weeks.
- Patients were advised to refrain from brushing at the surgical site till the periodontal dressing was in place.
- After removing periodontal dressing, brushing was avoided at the treated site. Instead cotton pellet was used to clean and slightly comb the area in an apical to incisal direction for the next 4 weeks.
- No specific instructions were given to smokers to avoid or reduce smoking after surgery.
- Sutures were removed 7 to 10 days after surgery.
- Patients were seen at 1 week, 1 month, 3 months, and 6 months.

Statistical Analysis

- The information gathered from present study was tabulated and analyzed using suitable techniques.
- Descriptive statistics were expressed as Mean \pm Standard deviation for all parametric variables.

- Repeated measures ANOVA was used for examination of mean differences between baseline and 6 months within groups, and factorial ANOVA was used for examination of mean differences between groups at each time point.

RESULTS

Throughout the study period all patients maintained a good standard of supragingival plaque control. No adverse events were recorded during the postoperative period. Descriptive statistics for the clinical parameters at baseline and 6 months after surgery, for both groups, are presented in Table 1. At baseline, no statistically significant differences were found between the two groups for any of the parameters evaluated (Table 1).

At 6 months, RD in the smokers group was significantly greater than the non-smokers group (Table 1). When within group changes in PD, CAL, and KT were compared between groups, the KT change in non-smokers was significantly different than the KT change in smokers ($p < 0.05$).

When the average root coverage percentage was compared, smokers had a significantly lower percentage than non-smokers ($p < 0.05$). The frequency

of complete root coverage was significantly greater in the non-smokers group ($P = 0.014$).

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In the smokers group, statistically significant changes from baseline were found for RD, PD CAL, and RW (Table 1). RD decreased by 1.90 ± 0.173 mm, which represents average root coverage of 60.09%. Complete root coverage was not obtained in any case. CAL increased by 2.40 ± 0.286 mm, KT increased by 0.1 ± 0.391 mm. In the non-smokers group, statistically significant changes from baseline were found for RD, PD CAL and KT (Table 1). RD decreased by 2.30 ± 0.243 mm, which represents average root coverage of 76.05%. Complete root coverage was achieved in three cases. CAL increased by 3.05 ± 0.292 mm, KT increased by 0.6 ± 0.265 mm.

		Smokers		Nonsmokers	
		Baseline	6 months	Baseline	6 months
RD	3.20 ± 0.349	$1.30 \pm 0.421^{*†}$	3.05 ± 0.438	$0.75 \pm 0.634^{*}$	3.20 ± 0.349
PD	1.70 ± 0.483	$1.20 \pm 0.422^{*}$	1.90 ± 0.568	$1 \pm 0.00^{*}$	1.70 ± 0.483
CAL	4.90 ± 0.614	$2.50 \pm 0.666^{*†}$	4.80 ± 0.675	$1.75 \pm 0.634^{*}$	4.90 ± 0.614
KT	3.65 ± 0.914	3.75 ± 0.956	3.35 ± 0.669	$4.00 \pm 0.577^{*}$	3.65 ± 0.914

* Significantly different from baseline (intragroup comparison; $p < 0.05$)

† Significantly different from non-smoker group at same time point

DISCUSSION

The objective of this prospective clinical trial was to compare the results of root coverage using the CPF in smokers and non-smokers. The results indicated that, under the high oral hygiene standards maintained throughout the study period in both groups, smokers exhibited poorer outcomes at 6 months, when RD at 6 months, percent root coverage, and frequency of complete root coverage were the outcomes considered.

In the present study the percentage of root coverage obtained at 6 month post-operatively is 60.09% in smokers group. This found to be similar to Cleverson O. Silva, (2006)^[4], the average root coverage was 69.3% at 6 months in smokers. The percentage of root coverage obtained at 6 month post-operatively in non-smoker group is 76.05%. The results are similar to those obtained by Antonieta De Queiroz Cortes (2004)^[5] (71%); Robert Carvalho da Silva (2004)^[6] (69%). In this study complete root coverage was obtained in 30% of non-smoker group while no smoker experienced complete root coverage in the study. The frequency of complete root coverage is significantly greater in the non-smoker group.

In a similar study Cleverson Oliveira Silva (2006)^[4], complete root coverage obtained in 50% of non-smokers and no smoker experienced complete root coverage. The results of the present study indicated that use of CPF for root coverage of Miller Class I gingival recession defects provided benefits for smokers and non-smokers alike. However, cigarette smoking negatively impacted the clinical outcome of root coverage. The results of the present study are expanding the list of periodontal plastic surgery procedures for which smokers have been shown to experience poorer outcomes. Significant differences between smokers and non-smokers were found in the treatment of gingival recession with subepithelial connective tissue graft (SCTG)^[7,8] and guided tissue regeneration (GTR)^[9]. Martins *et al.* (2004)^[7] treated 15 patients by subepithelial connective tissue graft technique, seven smokers and eight non-smokers, with Miller Class I or II recession defects ($RD \geq 3$), and observed the mean root coverage of 74.7% for non-smokers and 58.8% for smokers at four months. Complete root coverage was observed in 35% of the non-smokers and apparently not in the smokers.

Kenneth Erly (2006)^[8] treated 22 Miller's Class I and II recession defects, in smokers and non-smokers by subepithelial connective tissue graft technique, the mean root coverage gain was 98.3% in non-smokers and 82.3% in smokers at six months. Complete root coverage was observed in 50% of the non-smokers and no smokers experienced complete root coverage. Trombelli and Scabbia^[9] analyzed the results of 22 patients presenting with Miller Class I or II recession defects (RD \geq 4 mm), nine smokers and 13 non-smokers, treated by GTR. At 6 months, mean root coverage was 57% for smokers and 78% for non-smokers, while the estimated residual recession was 2 mm for smokers and 1.1 mm for non-smokers. Complete root coverage was observed in one smoker (11%) and five non-smokers (38%).

The present study on CPF, SCTG study^[8], and GTR study^[9] found similar differences between smokers and non-smokers. The fact that smokers have poorer root coverage outcomes under such different treatment and defect circumstances strengthens the association between smoking and poor results. In contrast to the above studies, Harris and Harris^[10], Tolmie^{et al.}^[11] and Amarante^{et al.}^[12] did not find an association between smoking and poor root coverage outcomes using a CTG procedure, free gingival graft, and CPF, respectively. More specifically, Harris and Harris^[10] found no difference between non-smokers, light smokers (<10 cigarettes daily), and heavy smokers (>10 cigarettes daily), while Tolmie^{et al.}^[11] obtained complete root coverage in almost 92% of the smokers, they treated. Amarante^{et al.}^[12] found that 62% of heavy smokers (\geq 20 cigarettes per day) had complete root coverage compared to 42% of non-smokers. It should be noted that none of these three studies was specifically designed to test the effect of smoking on root coverage outcomes.

The mechanisms by which cigarette smoking negatively influences the long-term or short-term outcomes of root coverage procedures such as CPF are not clear, although smoking can interfere with several physiologic and cellular functions that could all contribute to poorer outcomes^[13]. Smoking can negatively impact the gingival blood supply, whereas nicotine decreases human gingival fibroblast (HGF) proliferation and collagen production as it increases HGF collagenase activity^[14], and it inhibits HGF migration^[15]. Besides nicotine, volatile fractions of cigarette smoke are cytotoxic for HGF and inhibit HGF adhesion^[16].

CONCLUSION

Within the limits of the present investigation, CPF provided benefits for both smokers and non-smokers in terms of root coverage of shallow Miller Class I recession defects. However, cigarette smoking negatively impacted the clinical outcomes, specifically residual recession, percent root coverage, and frequency of complete root coverage.

REFERENCES

1. Jonson GK, Hill M. Cigarette smoking and the periodontal patient. State of the Art Review. J Periodontol. 2004;75:196-209.
2. Haber J, Watters J, Rowley C, Mandell R, Joslipura K. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol. 1993;64:16-23.
3. Huang LH, Neiva RE, Wang HL. Factors affecting the outcomes of coronally advanced flap root coverage procedure. J Periodontol. 2005;76:1729-1734.
4. Silva CO, Fernando AF. Coronally positioned flap for root coverage in smokers and non-smokers: stability of outcomes between 6 months and 2 years. J Periodontol. 2007;78:1702-1707.
5. Côrtes AD. Coronally positioned flap with or without acellular dermal matrix graft in the treatment of Class I gingival recessions: a randomized controlled clinical study. J Periodontol. 2004;75:1137-1144.
6. Silva RC, Joly JC, Martorelli de Lima AF. Root coverage using the coronally positioned flap with or without a subepithelial connective tissue graft. J Periodontol. 2004;75:413-419.
7. Martins AG, Andia DC, Sallum AW. Smoking may affect root coverage outcome: A prospective clinical study in humans. J Periodontol. 2004;75:586-591.
8. Erley KJ, Swiec GD, Herold R, Bisch FC, Peacock ME. Gingival recession treatment with connective tissue grafts in smokers and non-smokers. J Periodontol. 2006;77:1148-1155.
9. Trombelli L, Scabbia A. Healing response of gingival recession defects following guided tissue regeneration procedures in smokers and non-smokers. J Clin. Periodontol. 1997;24:529-533.
10. Harris RJ. The connective tissue with partial thickness double pedicle graft: The results of 100 consecutively treated defects. J Periodontol. 1994;65:448-461.
11. Tolmie PN, Rubins RP, Buck GS, Vagianos V, Lanz JC. The predictability of root coverage by way of free gingival autografts and citric acid application: an evaluation by multiple clinicians. Int J Periodontics Restorative Dent. 1991;11:261-271.
12. Amarante ES, Leknes KN. Coronally positioned flap procedures with or without a bioabsorbable membrane in the treatment of human gingival recession. J Periodontol. 2000;71:989-998.
13. Palmer RM, Wilson RF, Hasan AS, Scott DA. Mechanisms of action of environmental factors – tobacco smoking. J Clin. Periodontol. 2005;32:180-195.
14. Tipton DA, Dabbous MK. Effects of nicotine on proliferation and extracellular matrix production of human gingival fibroblasts in vitro. J Periodontol. 1995;66:1056-1064.

DOI: 10.69605/ijlbpr_13.6.2024.100

15. Fang Y, Svoboda KK. Nicotine inhibits human gingival fibroblast migration via modulation of Rac signaling pathways. *J Clin.Periodontol.* 2005;32:1200-1207.
16. Rota MT, Poggi P, Boratto R. Human gingival fibroblast cytoskeleton is a target for volatile smoke components. *J Periodontol.* 2001;72:709-713.