



Research Article

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## ***Outcomes of Gingival Depigmentation Among Smokers and Non-Smokers: A Comparative Study***

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### **ABSTRACT**

*Aim: The aim of this study was to compare depigmentation outcomes and recurrence rates among smokers and non-smokers after gingival hyperpigmentation treatment.*

*Materials & Methods: A total of 34 patients (16 smokers and 18 non-smokers) aged 19–45 years who sought treatment for “black gums” were included. A single calibrated examiner conducted all the treatments and assessments. Clinical observations on the distribution and severity of pigmentation according to the Gingival Pigmentation Index (GPI) and Dummatt-Gupta Oral Pigmentation Index (DOPI) were recorded at baseline and at 3 and 6 months after treatment. A rotary diamond abrasion technique was used for the de-epithelialization procedure, which involved abrading the gingiva until all visible pigmentation was removed. The data were analyzed using Mann–Whitney U tests.*

*Results: The mean age of the participants was 29 ( $\pm$  6) years, and 29.4% were male. The baseline GPI and DOPI scores were significantly higher among the smokers than the non-smokers ( $P < 0.05$ ). For example, regarding the baseline GPI scores, 50% of the smokers had scores of 3 whereas only 6.2% of non-smokers had scores of 3. At 3 months after treatment, the GPI and DOPI scores were not significantly different between the two groups ( $P > 0.05$ ). However, at 6 months, they were significantly higher among smokers ( $P < 0.05$ ). The recurrence rate at 6 months was higher among smokers than non-smokers, with 31% of smokers having GPI scores  $\geq 2$  compared to only 7.7% of non-smokers.*

*Conclusion: The abrasion with a rotary round bur resulted in satisfactory outcome, wound healing and tolerable pain. Smokers had more severe gingival hyperpigmentation and faster recurrence rate.*

**Keywords:** *Gingiva, hyperpigmentation, depigmentation, smoker’s melanosis, repigmentation*

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### **INTRODUCTION**

Cosmetic dentistry can be used to create an attractive smile, thereby helping to restore confidence and function to the patients. A person’s smile is a key feature of their appearance, and an esthetically pleasing smile is highly desirable. The attractiveness of an individual’s smile is determined not only by the shape, position, and color of the teeth, but also by the aesthetics of the gingival tissues. Pigmented gingiva can be a major source of embarrassment for patients, particularly those with a very high smile line (i.e., a gummy smile). Individuals often seek treatment for “black gums,” especially those with a very high smile line.

The color of gingival tissues is influenced by multiple variables including vascularity (i.e., the number and size of the blood vessels), epithelial thickness, degree of keratinization, and presence of pigment in the gingival epithelium. The major sources of pigmentation in a normal oral mucosa are melanin, carotene, reduced hemoglobin, and oxyhemoglobin [1].

Melanin is a non-hemoglobin-derived brown pigment that is the most common endogenous pigment in the oral mucosa. It is generated in the epithelial layer of the gingiva by cells known as melanocytes, which are mainly located in the basal

layer. Excessive levels of melanin deposition in the basal layer (produced due to melanoblastic activity of cells situated between the epithelial cells) can lead to gingival hyperpigmentation. The degree of pigmentation varies due to multiple factors, including melanoblastic activity [2].

Oral pigmentation may be due to physiological or pathological causes [3]. Healthy gingiva ranges from pale pink to deep blue/purple [5]. Among individuals of African, East Asian, or Hispanic descent, oral pigmentation due to melanin is often considerable [6,7]. In contrast, despite the fact that fair-skinned individuals have similar numbers of melanocytes in their gingival epithelia, they generally do not have overt oral pigmentation [8].

Pigmented oral lesions are common, and they can represent a localized anomaly of little clinical significance to the effects of a life-threatening systemic condition [4]. Common reasons for pigmented oral lesions include illnesses (e.g., Addison's disease, Albright's syndrome, Peutz-Jeghers syndrome, hemochromatosis, chronic pulmonary disease, endocrine disturbance, and malignant neoplasms such as melanoma and Kaposi's sarcoma), trauma, and antimalarial use.

In addition, smoking can also cause excessive melanin deposition in the oral mucosal epithelial layer. The polycyclic amines present in tobacco (e.g., nicotine and benzopyrenes) can induce melanin production by melanocytes, potentially as a protective response to the components in tobacco [9]. The term "smoker's melanosis" was coined by Hedin et al. in 1977 to describe the benign and limited melanin pigmentation of the attached gingiva in tobacco smokers [26]. Smoker's melanosis is found in 22% of smokers, and it occurs in a dose-dependent manner [10]. Women who smoke are affected more frequently than men, and they often present with diffuse black or brown macules that tend to mainly involve the gingiva, and to a lesser extent, the buccal mucosa, lips, and hard palate [10].

Multiple cosmetic treatments have been suggested for gingival depigmentation. These techniques include abrasion with a diamond bur, [16] Nd:Yag laser, [17] semiconductor diode laser, [18] and CO<sub>2</sub> laser [19]. Regarding surgical depigmentation, the most common technique involves using a scalpel to carry out gingivectomy involving free gingival autografting (with full- or partial-thickness bed preparation), [7] and other techniques include cellular dermal matrix allografts [11] electrosurgery, [12] cryosurgery, [13,14] and chemical techniques (e.g., using 90% phenol and 95% alcohol) [15]. The choice of a particular treatment should be based on the practitioner's experience, cost and the patient's ability to pay, and other patient preferences [3].

As the melanin is generated by melanocytes in the basal layer of the gingival epithelium, treatment requires the removal of the entire epithelium. The subsequent wound healing involves the proliferation of cells at the edge of the wound, which migrate and thereby help to re-epithelialize the wound [20].

Oral repigmentation refers to the clinical reappearance of the melanin in the gingival mucosa after achieving depigmentation [31]. The aim of this study was to compare the gingival depigmentation outcomes and recurrence rates among smokers and non-smokers after gingival depigmentation using a bur abrasion technique.

## MATERIALS & METHODS

Thirty-four consecutive patients (16 smokers and 18 non-smokers) who sought treatment for gingival melanin pigmentation were included in the study. The inclusion criteria consisted of melanin hyperpigmentation of the upper and lower gingivae and good oral hygiene. The exclusion criteria consisted of having a history of systemic disease linked to pathological hyperpigmentation, delayed wound healing (due to uncontrolled diabetes, autoimmune diseases, etc.), untreated periodontal disease, being aged <16 years, pregnancy, and lactation.

A single calibrated examiner conducted all the treatments and assessments. Clinical observations regarding the distribution and severity of melanin pigmentation were recorded according to the Gingival Pigmentation Index (GPI) [22] and the Dummett-Gupta Oral Pigmentation Index (DOPI) [21] at baseline and at 3 and 6 months after depigmentation. The DOPI is the most commonly used index in this context, due to its ease of use.

The de-epithelialization involved an abrasion technique using a high-speed handpiece and rotary diamond bur under local anesthesia to remove the epithelium in the pigmented areas (Figure 1). The depigmentation treatment was carried out in the anterior region, from the premolar to the premolar. To reduce the risk of recurrence, all of the pigmented gingival layer was removed, along with a thin layer of connective tissue (Figure 2). Saline irrigation was then applied to the exposed surface. The largest bur available was used because the smaller versions cannot be easily used to create smooth surfaces and they tend to cause small pits in the area being treated. Feather-light brush strokes were applied, avoiding holding the bur in a single position. To reduce hemorrhage during treatment, pressure was applied with sterile gauze.

The patients were provided with analgesics after surgery. They were advised to use chlorhexidine mouthwash every 12 h for 2 weeks and to avoid eating hot or spicy food for 24 h. After 1 week, the patients attended a follow-up appointment. For all the patients, healing was uneventful, with no major postoperative complications (such as pain or hemorrhaging).

The data were analyzed using Mann–Whitney U tests.

## RESULTS

The mean age of the participants was 29 ( $\pm$  6) years, and 29.4% were males and 70.6% females. The baseline GPI and DOPI scores were significantly higher among the smokers compared to the non-smokers ( $p < 0.05$ ) (Tables 1 and 2). For example, eight (50%) of the smokers had baseline GPI scores of 3, whereas only two (11.1%) of the non-smokers had baseline scores of 3 (Table 1). In addition, eight (50.0%) of the smokers had baseline DOPI scores of 4, whereas only five (27.8%) of the non-smokers had baseline scores of 4 (Table 2).

At 3 months after depigmentation, the GPI scores were not significantly different between the two groups ( $P > 0.05$ ) (Table 1). At 6 months after depigmentation, the GPI scores were significantly higher among the smokers ( $P < 0.05$ ) (Table 1) and the DOPI scores were marginally significantly higher among the smokers ( $P = 0.07$ ) (Table 2). The recurrence at 6 months was lower among non-smokers (Figure 3) than the smokers (Figure 4), with only 7.7% of the non-smokers having GPI scores of  $\geq 2$  compared to 50.0% of the smokers (Table 1).

**Table 1.** Gingival Pigmentation Index (GPI) scores among smokers and non-smokers

GPI score	Pre-operative		3 months		6 months	
	Smokers (%)	Non-smokers (%)	Smokers (%)	Non-smokers (%)	Smokers (%)	Non-smokers (%)
0	0	0	87.5	94.4	30.0	69.2
1	16.7	18.7	12.5	5.6	20.0	23.1
2	33.3	75.0	0	0	50.0	7.7
3	50.0	6.2	0	0	0	0
P-value	0.049		0.48		0.03	

**Table 2.** Dummett-Gupta Oral Pigmentation Index (DOPI) scores among smokers and non-smokers

DOPI score	Pre-operative		3 months		6 months	
	Smokers (%)	Non-smokers (%)	Smokers (%)	Non-smokers (%)	Smokers (%)	Non-smokers (%)
1	0	5.6	87.5	100.0	30.0	69.2
2	6.2	16.7	12.5	0	70.0	30.8
3	37.5	55.6	0	0	0	0
4	56.2	22.2	0	0	0	0
P-value	0.033		0.128		0.068	



**Figure 1.** Rotary diamond bur abrasion technique

Figure 2: Photographs of two examples of (A) preoperative gingival hyperpigmentation and (B) result immediately after depigmentation



Figure 2A. Preoperative clinical appearance with hyperpigmentation



Figure 2B. Clinical appearance immediately after depigmentation



Figure 2A. Preoperative clinical appearance with hyperpigmentation



Figure 2B. Clinical appearance immediately after depigmentation



Figure 3. Photographs of the gingivae of non-smokers

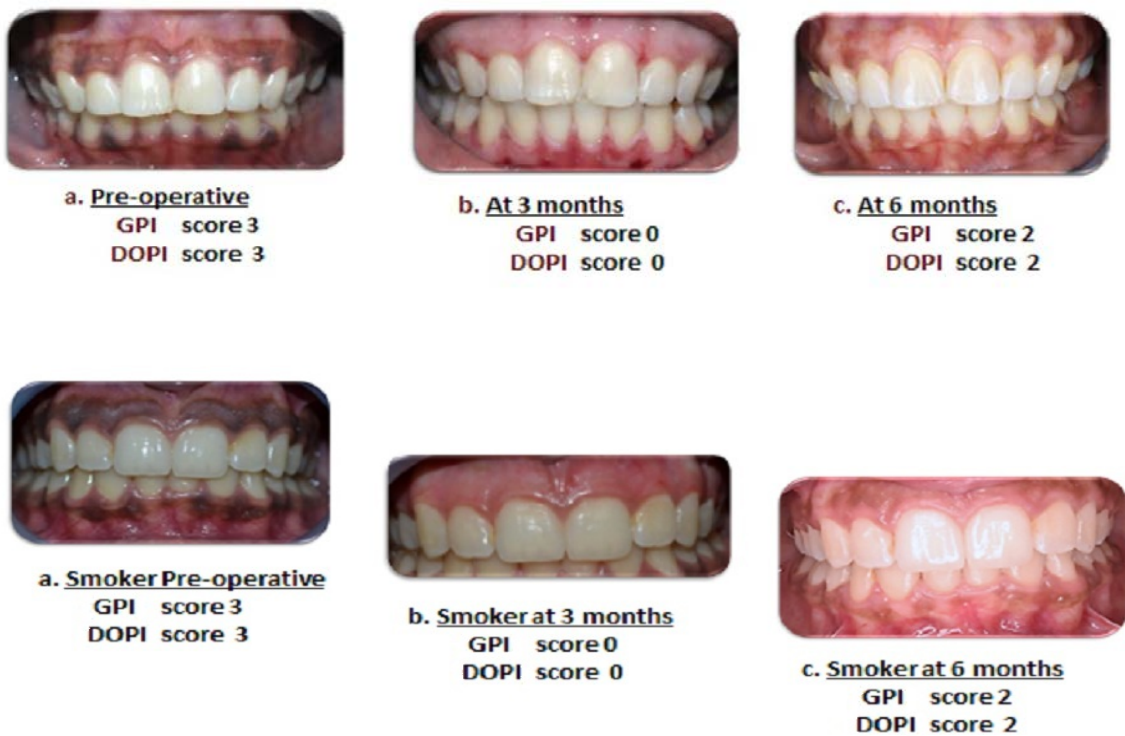


Figure 4. Photographs of the gingivae of smokers

## DISCUSSION

Individuals often seek cosmetic treatment for pigmented gingival tissues. [23] Multiple types of treatment have been reported in the literature, from simple scalpel-based techniques to hi-tech laser-based techniques. [24]

The first cosmetic treatment for gingival depigmentation involved an exfoliative technique using 90% phenol conducted in 1946. However, the technique is associated with multiple limitations, including rapid drainage of the phenol solution and decreased mucosal penetration due to rapid epidermal coagulation. [15] Several other techniques were later developed, including gingival abrasion (which commonly involves removing the gingival epithelium with a high-speed handpiece and diamond bur) [27, 28], cryosurgery (involving rapid freezing-induced local necrosis), gingivectomy (using a scalpel to remove the gingival epithelium along with a layer of connective tissue, and subsequently allowing secondary intention healing) [27, 29], and gingival grafts [14, 30]. However, these techniques have multiple limitations, including lack of precise control over the tissue being eliminated (which particularly applies to the chemical and cryosurgery techniques), bleeding, and, in the case of gingivectomy, the requirement for surgical dressings [14, 30, 29]. In addition, lasers can be used to treat hyperpigmentation. However, although a one-step laser procedure generally results in the elimination of the pigmented gingiva, this approach is very expensive and relies on hi-tech lasers that are not widely accessible [17]. In contrast, the present study involved a simple but effective depigmentation procedure that does not rely on hi-tech devices. The results of this study indicated that preoperative GPI and DOPI scores were significantly higher among the smokers than the non-smokers, highlighting that the gingival pigmentation is heavier and more diffuse in smokers compared to non-smokers. Eight (50%) of smokers had baseline GPI scores of 3, which indicates the presence of diffuse black or brown pigments (affecting the marginal and attached gingiva) whereas only two (6.2%) of the non-smokers had baseline scores of 3 (Table 1).

At 3 months after treatment, gingival pigmentation for all the patients was  $\leq 1$  in terms of the GPI scores and  $\leq 2$  in terms of the DOPI scores. However, at 6 months, in terms of GPI scores, the smokers exhibited more gingival repigmentation than the non-smokers. Spontaneous repigmentation is thought to be due to melanocytes that migrate from surrounding areas to the initially depigmented areas. In the present study, the risk of hyperpigmentation recurrence was clarified for all the patients. Only one patient was dissatisfied with their treatment outcomes. This was likely due to the fact that all the cases of repigmentation were very mild, involving miniscule spots or stripes that did not cause any esthetic issues, which compared favorably with the moderate to heavy pigmentation (manifested as continuous bands) that was present at baseline.

The exact mechanism underlying repigmentation is unknown, but the "migration theory" posits that it occurs when activated melanocytes migrate from surrounding pigmented areas to the initially depigmented areas. Several reports of repigmentation have been published in the literature. For example, Perlmutter and Tal reported gingival repigmentation after the removal of gingival tissue from a single patient. [32] In addition, Ginwalla et al. [33] investigated an abrasion technique and found that repigmentation occurred after 24–56 d 50% of the time. [Moreover, Pal et al. [34] investigated gingival depigmentation using an abrasive bur and found that 19% of the patients experienced recurrence. However, Mokeem [35] used an abrasion technique with a high-speed handpiece and diamond bur and found that none of the three treated patients (who all had physiological gingival hyperpigmentation) experienced repigmentation after 18 months. In contrast, Farnoosh [36] investigated a similar technique and reported minor recurrence for two patients after 20 months. However, repigmentation has been reported by others [29, 19]. Smoking appears to influence the success of the treatment. Esenet al. [8] treated 10 patients for gingival melanin pigmentation and found that two experienced recurrence, both of whom were smokers. Thus, it appears that quitting tobacco smoking may be required to reduce the risk of repigmentation.

## CONCLUSIONS

Abrasion with a rotary round bur resulted in satisfactory depigmentation outcomes, good wound healing, and tolerable pain. Compared to the non-smokers, the smokers had more severe baseline gingival hyperpigmentation and a higher recurrence rate at 6 months.

## CLINICAL SIGNIFICANCES

Complaints of "black gums" are common, especially among patients with high smile lines. In addition to genetic factors, tobacco smoking is a major cause of oral melanin pigmentation.

Tobacco smoking cessation may reduce the risk of repigmentation after gingival hyperpigmentation treatment using an abrasion technique.

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**REFERENCES**

1. Mahesh HV, Harish MR, Shashikumar BM, Ramya KS. Gingival pigmentation reduction: a novel therapeutic modality. *J CutanAesthetSurg* 2012 Aug;5:137-40.
2. Cockings JM, Savage NW. Minocycline and oral pigmentation. *Aust Dent J* 1998 Feb;43(1):14-16.
3. Cicek Y, Ertas U. The normal and pathological pigmentation of oral mucous membrane: a review. *J Contemp Dent Pract* 2003 Aug;15;4(3):76-86.
4. Dummett, CO. Oral pigmentation. First symposium of oral pigmentation. *J Periodontol* 1960 Oct;31:356
5. Shafer WG, Hine MK, Levy BM. A textbook of oral pathology. Philadelphia, PA: WB Saunders Co., 1984;p. 89-136
6. Fry L, Almeyda JR. The incidence of buccal pigmentation in caucasoids and negroids in Britain. *Br J Dermatol* 1968 Apr; 80(4):244-247.
7. Tamizi M, Taheri M. Treatment of severe physiologic gingival pigmentation with free gingival autograft. *Quintessence Int* 1996 Aug;27(8):555-558.
8. Esen E, Haytac MC, Oz IA, Erdogan O, Karsli ED. Gingival melanin pigmentation and its treatment with the CO2 laser. *Oral Surg Oral Med Oral Pathol Oral RadiolEndod* 2004 Nov;98(5):522-527.
9. Hedin CA, Pindborg JJ, Axell. T. Disappearance of smoker's melanosis after reducing smoking. *J Oral Pathol Med* 1993 May;22(5);228-230.
10. Axell T, Hedin CA. Epidemiologic study of excessive oral melanin pigmentation with special reference to the influence of tobacco habits. *Scand J Dent Res*1982 Dec;90(6);434-442.
11. Dello Russo NM. Esthetic use of a free gingival autograft to cover an amalgam tattoo: report of case. *J Am Dent Assoc* 1981 Mar;102(3):334-335.
12. Gnanasekhar JD, Al-Duwairi YS. Electrosurgery in dentistry. *Quintessence Int* 1998 Oct;29(10):649-654.
13. Tal H, Landsberg J, Kozlovsky A. Cryosurgical depigmentation of the gingiva. A case report. *J ClinPeriodontol* 1987 Nov;14(10):614-617.
14. Yeh CJ. Cryosurgical treatment of melanin-pigmented gingiva. *Oral Surg Oral Med Oral Pathol Oral RadiolEndod* 1998 Dec;86(6):660-663.
15. Hasegawa A, Okagi H. Removing melagenous pigmentation using 90 percent phenol with 95 percent alcohol. *Dent Outlook* 1973;42:673-676.
16. Bishop K. Treatment of unsightly oral pigmentation: a case report. *Dent Update* 1994 Aug;21(6):236-237.
17. Atsawasuwan P, Greethong K, Nimmanon V. Treatment of gingival hyperpigmentation for esthetic purposes by Nd:YAG laser: report of 4 cases. *J Periodontol* 2000 Feb;71(2):315-321.
18. Yousuf A, Hossain M, Nakamura Y, Yamada Y, Kinoshita J, Matsumoto K. Removal of gingival melanin pigmentation with the semiconductor diode laser: a case report. *J Clin Laser Med Surg* 2000 Oct;18(5):263-266.
19. Nakamura Y, Hossain M, Hirayama K, Matsumoto K. A clinical study on the removal of gingival melanin pigmentation with the CO2 laser. *Lasers Surg Med* 1999 Aug;25(2):140-147.
20. Fiorelline JP, Kim DM, Uzel NG. Anatomy of the periodontium. In: Newmann MG, Takei H, Klokkevold OR, Carranza FA. Carranza's Clinical Periodontology. 11th ed. St.Louis; Saunders Elsevier 2011Feb;p. 12-27.
21. Dumme CO, Gupta OP. Estimating the epidemiology of gingival pigmentation. *J Natl Med Assoc* 1964 Sep;56:419-20.
22. Kumar S, Bhat SG, Bhat MK. Development in techniques for gingival depigmentation - an update. *Indian J Dent* 2012 Oct; 3:213-21.
23. Deepak P, Sunil S, Mishra R, Sheshadri. Treatment of gingival pigmentation: a case series. *Indian J Dent Res* 2005 Oct-Dec;16(4):171-176
24. Prasad SS, Neeraj A, Reddy NR. Gingival depigmentation: a case report. *People's. J Science Research* 2010 Jan;3:27-29.
25. Kathariya R, Pradeep AR. Split mouth de-epithelization techniques for gingival depigmentation: a case series and review of literature. *J Indian SocPeriodontol* 2011 Apr-Jun;15:161-168.
26. Hedin CA. Smokers' melanosis. Occurrence and localization in the attached gingiva. *Arch Dermatol*1977 Nov;113(11):1533-1538.
27. Parwani S, Parwani R. Achieving better esthetics by gingival de-pigmentation: report of three cases with a review of the literature. *J Mich Dent Assoc*2013 Feb;95(2):52-78.

28. Lee KM, Lee DY, Shin SI, Kwon YH, Chung JH, Herr Y. A comparison of different gingival depigmentation techniques: ablation by erbium:yttrium-aluminum-garnet laser and abrasion by rotary instruments. *J Periodontal Implant Sci* 2011 Aug;41(4):201-207.
29. Bergamaschi O, Kon S, Doine AI, Ruben MP. Melanin repigmentation after gingivectomy: a 5-year clinical and transmission electron microscopic study in humans. *Int J Periodontics Restorative Dent* 1993 Feb;13(1): 85-92.
30. Talebi M, Farmanbar N, Abolfazli S, SarrafShirazi A. Management of physiological hyperpigmentation of oral mucosa by cryosurgical treatment: a case report. *J Dent Res Dent Clin Dent Prospects* 2012 Nov;6(4):148-151.
31. Doshi Y, Khandge N, Byakod G, Patil P. Management of gingival pigmentation with diode laser: Is it a predictive tool? *Int J Laser Dent* 2012 Jan-Apr;2:29-32.
32. Perlmutter S, Tal H. Repigmentation of the gingiva following surgical injury. *J Periodontol* 1986 Jan;57:48-50.
33. Ginwalla TM, Gomes BC, Varma BR. Surgical removal of gingival pigmentation. *J Indian Dent Assoc* 1966 Jun;38:147-150
34. Pal TK, Kapoor KK, Parel CC, Mukharjee K. Gingival melanin pigmentation: a study on its removal for esthetics. *J Indian SocPeriodontol* 1994;3:52-54.
35. Mokeem SA. Management of gingival hyperpigmentation by surgical abrasion: report of three cases. *Saudi Dental Journal* 2006 Sep-Dec;18:161-166
36. Farnoosh AA. Treatment of gingival pigmentation and discoloration for esthetic purpose. *Int J Periodont Rest Dent* 1990;10:313-318.