Review Article

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GINGIVAL PIGMENTATION: A REVIEW AND CASE REPORT

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ABSTRACT:

Gingival pigmentation is seen in all races in varying degrees. It is not a disease entity, however, it is an important component of harmonious esthetics. Many patients find gingival pigmentation esthetically undesirable and seek treatment for the same. While most pigmentation is physiologic, gingival pigmentation may also be indicative of underlying systemic disorders. This review discusses gingival pigmentation, its differential diagnosis, the treatment options available and the advantages and disadvantages of each. It ends with a case report where favorable outcomes were achieved.

Keywords*:* Gingival pigmentation, depigmentation, repigmentation.

INTRODUCTION:

A beautiful smile is most desirable. It is influenced not only by the colour, shape and position of the teeth but also by the characteristics of the gingival tissues. The colour of gingival tissues is an esthetic concern for many patients. The personal preference and attitude towards gingival pigmentation was demonstrated in the survey conducted by Dummett [1] which revealed that "pink gums" was considered most desirable. The colour of healthy gingiva varies from a pale pink to a deep bluish purple. The lighter color is associated with superficial melanin, while the darker blue is associated with more profound melanin, located deeper in the connective tissues. [2] According to Dummett, [3] the colour is influenced by factors such as the intensity of melanogenesis, the degree of epithelial keratinization and the vascularity of gingival tissues. Gingival pigmentation varies from individual to individual. Fair skinned individuals often have nonpigmented, pale or coral pink gingiva contrary to dark skinned individuals, who more often have pigmented gingiva. Several studies in different racial groups have confirmed that the intensity of oral pigmentation appears to be proportional to that of cutaneous pigmentation.[2,4] Raut et al, [5] in a study on Indian individuals whose skin colour varied from light to dark brown, reported an increase in pigmentation with darker complexion. They found that the distribution in the maxilla and mandible were similar, while the anterior regions were more pigmented than the posteriors.

Pigmentation may be localized to specific areas or may extend to involve the entire gingiva and the other oral tissues. It may be seen uniformly, unilaterally or bilaterally, or even have a mottled, macular or blotched appearance. [3]

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The earliest appearance of gingival pigmentation has been reported to be as early as three hours after birth, as the only sign of pigmentation in the body. [2] Monash [6] indicated that oral pigmentation may be complete by the second decade of life. Brown [7] described that gingival pigmentation decreased with age, while pigmentation of the lips, cheeks and palate increased with age. No sex differences were noted.

Gingival colour is produced due to the presence of pigments, mainly melanin, which has been described as a yellow to black pigment. Other pigments such as melanoid, carotene, oxyhaemoglobin and hemoglobin also influence it. [2] Melanocytes are mature melanin forming, dendritic cells present in the basal and spinous layer of the gingival epithelium. These synthesize melanin in organelles called melanosomes. They contain tyrosinase, which hydroxylates tyrosine to dihydroxyphenylalanine, which is then coverted to melanine. These melanin granules are phagocytosed and contained in melanophages. Increased melanin production can be stimulated by stimuli such as trauma, hormones, radiation and some medications. [8] Scattered granules of melanoid and the presence of carotene impart a yellow colour to the tissues. The gingival colour is also influenced by hemosiderin deposits and the underlying tissue vasculature. [9]

CLASSIFICATION AND DIFFERENTIAL DIAGNOSIS:

Dummett [2] proposed a classification of oral pigmentation. This included primary and secondary oral melanin pigmentations, oral non-melanin pigmentations and oral melanoclasias.

Broadly gingival pigmentation can be classified as exogenous and endogenous:[10]

Exogenous:

i. **Occupational**: Exposure to heavy metals may lead to their systemic absorption and subsequent perivascular sulfide precipitation in the subepithelial connective tissue. [11] Lead results in deep blue linear pigmentation of the gingival margin (Burtonian line); bismuth, arsenic and mercury produces a black line which follows the contours of the margin; while silver produces a violet line along the margin, accompanied by a diffuse bluish – grey discoloration of the oral mucosa.

ii. **Habits:** The use of tobacco may cause substantial increase in keratinization and melanin pigmentation. [11] Smoker's melanosis presents with discrete or coalescing multiple brown macules on the attached gingiva labially as well as palatally and bucally. Improvement can be expected with smoking cessation.

Unique pigmentation associated with the habitual chewing of plants was reported by Ashri and Gazi.[12] A brown pigmentation was caused by the bark of *Juglans regia*, a bright yellow

pigmentation from chewing of *Cola nitida* seeds, and a mousy brown pigmentation from chewing of the leaves of *Catha edulis*.

iii. Therapeutic: Antimalarial drugs such as quinacrine, chloroquine and hydroxychloroquine have been found to induce at times a slate grey pigmentation of the oral mucosa. [13] The prolonged use of antimicrobial drug minocycline may result in increased brown melanin pigmentation on the gingiva, lips, and tongue.[14]

iv. Others: The amalgam tattoo arises due to the accidental implantation of amalgam or other metal fragments into the gingival tissues. It manifest as asymptomatic, isolated bluish - black macules in the gingiva and alveolar mucosa, more commonly in the mandibular region. [15] Graphite tattoos [16] may be seen palatally due to traumatic lodging of graphite into the soft tissues from a pencil. Rawal et al [17] observed gingival tattooing as a cultural practice in three different ethnic groups in West African females. They presented with diffuse pigmentation of the maxillary attached gingiva with no underlying radiographic defects. Biopsies revealed foreign material in the connective tissues. The women were found to have undergone one or more traditional gingival tattooing sessions.

Endogenous:

i. Physiologic pigmentation: All patients, except albinos, have some degree of physiologic pigmentation ranging from pale or coral pink to a deep brownish black colour. As mentioned above, this pigmentation varies from person to person, has a racial or genetic predilection, varies in its distribution in oral tissues and at different ages. This does not present as a medical problem and requires no treatment. However, dark coloured gingiva may not be favoured by some individuals, who cosmetically seek to alter the colour of their gingiva. Pregnancy may also clinically manifest as oral pigmentation due to increased adrenocorticotropic hormone (ACTH) levels. Diffuse browning may occur on the skin and oral mucosa, which slowly disappears post delivery. Postmenopausal changes in sex hormones and the use of oral contraceptive pills containing high doses of sex hormones can also induce pigmented changes of the oral mucosa.

ii. Pathologic pigmentation:

a. Pigmented nevi: These are flat to slightly elevated grey, smooth, brown, grey or bluish macules occurring on the lips and gingiva produced by a group of four or more melanocytes in contact with the basal layer of the epithelium. The clinically blue colour is due to the Tyndall effect, where there is preferential absorption of long wavelengths of light by melanin and scattering of shorter wavelengths of light by the collagen bundles. On reaching a certain size, growth ceases and the nevus remains stable. [18, 19]

b. Oral melanotic macules: These grey, brown, blue or black, flat and smooth macules occur due to increased melanin pigmentation by basal cells melanocytes, with no increase in the number of melanocytes. They are common on the vermilion border of the lower lip, and less commonly seen on the buccal mucosa, palate and gingiva. These are analogous to skin freckles, and are more common in the fair individual. [19]

c. Oral melanoacanthoma: This is a rare, benign pigmented lesion characterized by proliferation of dendritic melanocytes in acanthotic and hypekeratotic epithelium. These present as flat or slightly raised, black to brown macules, with an increased tendency to affect areas exposed to trauma or irritation. The lesion may resolve on removal of the offending stimulus. They more frequently affect black females.[19]

d. Hemochromatosis: It involves a tetrad of liver cirrhosis, diabetes, cardiac failure and bronze skin. Patients frequently also have diffuse brown - black pigmentation at the junction of the hard and soft palate, gingiva and buccal mucosa. The increased tanning is due to deposition of iron containing pigments - ferritin and hemosiderin and enhanced cutaneous and oral mucosal melanin production.[19]

e. Addison's disease: Pigmentation occurs because the dysfunctional adrenal cortex is unable to produce sufficient glucocorticoid to cease production of ACTH by the pituitary gland. Hence ACTH production continues to increase. Because ACTH and melanocyte stimulating hormone are similar in structure, ACTH has some melanocyte stimulating activity, thus producing the colour changes characteristic of Addison's disease – a smoky tan or chestnut brown. Isolated bluish black to pale or deep chocolate brown patches, spreading over the buccal mucosa, gingiva, tongue, angle of the mouth and lips may be the first evidence of this disease.[19]

f. Peutz-Jegher's syndrome: Intestinal polyposis is the major manifestation. It is also characterized by asymptomatic, pigmented, freckle - like macules on the skin and intraorally, measuring between 1 to 10 mm. These usually appear at birth, but while the cutaneous lesions may gradually disappear, the lesions on the buccal mucosa remain.[19]

g. Albright's syndrome and neurofibromatosis: Both these diseases produce irregular, light brown pigmented spots, also known as café – au – lait spots, intraorally and cutaneously.[19]

h. Jaundice: Here the oral mucosa may be stained a yellowish colour by the bile pigments.

i. Post inflammatory pigmentation: This is most commonly seen in relation to long standing mucosal lesions observed as brown - black pigmented

areas adjacent to reticular or erosive lichen planus lesions.[19]

j. HIV melanosis: The etiology of theses lesions remains uncertain though it may be attributed to adrenocortical involvement or medication such as systemic clofazimine or ketoconazole therapy.[20, 21]

k. Kaposi's sarcoma: It often affects the oral cavity as a manifestation of infection with HIV. This multifocal, angioproliferative neoplasm presents as multiple red or purple macules or nodules, which progress to darken and coalesce, forming clusters of single nodules. These lesions result in pain, dysphagia, difficulty in chewing, frequent traumatization, bleeding and a cosmetically displeasing appearance.[19]

l. Melanoma: Also known as melanocarcinoma, these malignant neoplasms arise from benign melanocytic lesions or *de novo* from melanocytes. They exhibit a definite predilection for the maxillary gingiva and palate, appearing initially as brown or black macules with irregular borders which later enlarges to an exophytic mass once vertical growth is initiated. These lesions may further undergo ulceration.[19]

CLINICAL ESTIMATION OF PIGMENTATION:

Dummett and Gupta [22] described a clinical index for the quantitative estimation of gingival pigmentation known as the Dummett-Gupta Oral Pigmentation Index (DOPI). The gingiva of the maxillary and mandibular arch are each divided into 32 units spaces, 16 lingually/palatally and 16 bucally/labially. Each unit space extends from the marginal gingiva upto the level of the attached gingiva. The units are individually scored based on the following scale:

0: No clinical pigmentation (pink tissue)

1: Mild clinical pigmentation (mild light brown colour)

2: Moderate clinical pigmentation (medium brown or mixed pink and brown colouration)

3: Heavy clinical pigmentation (deep brown or blue black tissue)

The numerical estimates of each arch are totaled and divided by the total number of units (32) to give the DOPI assessment of that arch.

The DOPI assessment is scaled as follows:

0: No clinical pigmentation of the gingiva

0.031 – 0.97: Mild gingival pigmentation

1.0 – 1.9: Medium pigmentation

2.0 – 3.0: Heavy pigmentation

TREATMENT OPTIONS FOR GINGIVAL DEPIGMENTATION:

Gingival depigmentation is a periodontal plastic surgical procedure for the removal of hyperpigmented gingiva. The foremost indication of gingival depigmentation is the request for improved esthetics by the patient. The procedures available today as are follows:

i. **Scalpel technique:** This is one of the oldest, most economical, and most popular techniques in use today, which is simple and effective. The gingival epithelium along with a layer of underlying connective tissue is carefully excised from the gingival margin to the mucogingival junction, using a number 15 surgical blade or a Kirkland knife. The denuded connective tissue then heals by secondary intention with the establishment of new epithelium free from melanin pigmentation. Alternatively the pigmented tissue layers may also be gently scraped away. With the scalpel technique, healing is faster than other techniques. However it may result in unpleasant bleeding during and after the surgery, necessitating the use of periodontal dressing for 7 to 10 days.[23]

ii. **Rotary abrasive burs:** This involves the removal of pigmented areas using a high speed handpiece with larger diamond abrasive burs. Light feather, brushing strokes with copious irrigation are to be used, without keeping the bur at a single place for prolonged periods, as that could result in thermal trauma. The procedure should be done with caution to avoid damage to the underlying periosteum and bone which could result in gingival recession. Care should be taken to avoid accidental loss of enamel. The use of smaller burs may result in undesirable pitting in the tissues. [24] Healing with this method is similar to that seen in the scalpel method. Both the scalpel technique and the bur technique have the advantage of not requiring any sophisticated equipment making these procedures economical.

iii. **Cryosurgery:** This technique makes use of local freezing for the controlled removal of vital tissues with cryogens such as salt ice (-20°C), slush (-20°C), fluorocarbons (-30°C), nitrous oxide (-75°C), CO_2 snow (-79°C) and liquid nitrogen (-196°C).[25]

The procedures may be performed via the dipstick technique, the spray technique or the cryoprobe technique. In the dipstick technique, a small cotton bud or swab is dipped into liquid nitrogen. The liquid nitrogen is then applied to the pigmented tissues and allowed 20-30 seconds of contact.[26] In the spray technique, the table top or hand held cryosurgical unit is filled with liquid nitrogen and sprayed within the border of the lesion, holding the spray tip 1 cm away from the lesion. Once solid ice has formed over the area, freeze time begins. The lesion is allowed to thaw slowly for a time period that is usually double that of the freeze time. In the cryoprobe technique, the tip is cooled by circulation liquid nitrogen and

is the applied to the lesion resulting in freezing by conduction. [25]

Following cryosurgery, thawing occurs within 15-20 seconds, progressing from the periphery to the center of the lesion. By 12 hours a fluid filled blister occurs which may increase in size in the first 24 hours. The roof of the blister then ruptures, exposing the smooth underlying connective tissue. This is followed by surface repair and reepithelization. [27, 28]

This technique has the advantage in that it is relatively painless, hence does not require the use of local anesthesia or periodontal dressing. Long lasting results have been reported. [26] Depigmentation with cryosurgery also has several disadvantages. Cryosurgery requires the use of special containers for storing liquid nitrogen. Liquid nitrogen does not have a long shelf life. Dispensing the cryogen is often difficult, and care is to be taken to prevent accidental contact and spillage, as this could be injurious when in contact with skin. Depth of penetration and maintenance of accurate freeze time is also difficult to control, which may result in increased tissue destruction. It is difficult to assess the success of therapy during the procedure, which may necessitate multiple applications since immediate clinical changes cannot be appreciated. [29]

iv. Electrosurgery: Oringer explained with the "exploding cell theory" that molecular disintegration will occur of melanin cells present in the basal and suprabasal layers at the operated and surrounding sites when electrical energy is applied to it. [30] Caution should be exercised while using electrosurgery. The tip should always be kept in motion, with light brushing strokes, avoiding repeated application of current to the tissues which would result in undesirable heat buildup and destruction of tissues. Tip contact with the periosteum and tooth root should be avoided to prevent bone loss and cemental burns.

v. Lasers: Lasers used in the treatment of depigmentation include the Nd:YAG laser, CO_2 laser, argon laser and the semiconductor diode lasers. The Nd-YAG laser has an added affinity for pigmented tissues. Laser energy is converted to ablation energy which results in cell rupture and vaporization with minimal heat production. Treatment with lasers have the advantages of producing a bloodless field of surgery while also maintaining a sterile working field. It also eliminates the use of a periodontal dressing on the wound. Minimum damage is caused to the periosteum and underlying bone which facilitates healing. There is ease of handling and shorter treatment time. [31] Repigmentation is reduced since the laser beam destroys the epithelial cells including those at the basal layer. However it is associated with disadvantages such as loss of tactile feedback, delayed reepithelialization and a delayed type of inflammatory

reaction producing mild postoperative discomfort. [32] The use of sophisticated equipment makes treatment expensive.

vi. **Masking the pigmented lesions with free gingival grafts and acellular dermal matrix allografts:** These procedures involve the replacement of pigmented gingiva with nonpigmented free gingival autografts. Tamizi and Taheri [33] reported favorable results with no evidence of repigmentation for 4 and a half years postoperatively. The disadvantage of this technique is that a second surgical donor site is required which causes additional discomfort to the patient. Healing is prolonged. Also sufficient donor tissue may not be available. Tissue color matching and the presence of a well demarcated line between the grafted tissues and the surrounding sites post operatively is another issue which hampers esthetics.

An alternative to using free gingival autografts is the use of acellular dermal matrix allografts (ADMA). This technique has advantages such as being acellular and non-immunogenic, requiring reduced surgical time when compared to the free gingival autograft procedures. Unlimited donor tissue is available, postoperative pain and complications are minimal and excellent esthetics can be achieved. However this procedure is also expensive.

vii. **Chemical agents:** Chemical peeling agents that destroy the epidermis or dermis have been used for depigmentation such as phenol, salicylic acid, glycolic acid, trichloroacetic acid. [29] However these agents may cause damage to the underlying tissues as their depth of penetration is difficult to control. In addition, due care has to be taken to control the area of application to prevent accidental damage to surrounding tissues. These agents are not popular today due to more reliable techniques being available.

REPIGMENTATION:

Following clinical depigmentation procedures, repigmentation, which is the reappearance of clinical pigmentation, often occurs at varying times after healing, from immediately after healing to several years later. The mechanism by which this occurs can be explained by the “migration theory”, where in active melanocytes from the surrounding areas proliferate and migrate into the depigmented areas. Perlmutter and Tal [34] speculated that in the postoperative period where depigmentation was maintained, the migration of melanocytes did not occur, or the migrated melanocytes were not active. Repigmentation could also occur due to left out melanocytes which later may become active.[35] The different behavior of these migrating melanocytes in different patients and at different times would require further investigation, though it does appear that the rate and intensity of repigmentation may be related to the complexion of the individual.[36]

CASE REPORT:

The present case describes depigmentation of gingiva, using a combination of the scalpel and bur technique, in a young female patient, for cosmetic reasons.

A 24 year old female patient reported with the chief complaint of 'black gums'. She expressed extreme displeasure with the color of her gingiva and said that she did not feel confident while smiling and even talking. The patient wanted to enquire if anything could be done to change the color of her gingiva in both the upper as well as the lower arch. As far as she could remember, her gingiva had always been dark in colour. The patient had a dusky complexion. Clinical examination revealed generalized, diffuse, deep brownish-black pigmentation, extending from the marginal gingiva till the mucogingival junction. The patient also had a very high smile line, with more than 8 to 9 mm of the maxillary labial gingiva and 4 to 5 mm of mandibular labial gingiva visible on smiling. The patient was otherwise in good health, both dentally and medically. No known systemic disease was present. She did not report recent intake of any medication. No history of smoking was present. The patient was informed about treatment available for depigmentation and that repigmentation frequently occurred. The patient chose to undergo gingival depigmentation. She was scheduled for the procedure after initial therapy.

Local anesthesia was administered via local infiltration. The pigmented areas were delineated using a number 15 scalpel blade. A split thickness flap within the delineated margins was raised while maintaining the architecture of the tissues. It was then excised. Finishing touches were made with a straight rotary diamond bur under copious saline irrigation to remove any pigmented spots or patches still present. Bleeding was controlled by applying gentle, firm pressure with cold saline - soaked gauze swabs. The depigmented surfaces were covered with periodontal dressing (CoePak). The patient was prescribed Ibuprofen, 400 mg, thrice daily for three days, and thereafter, as and when required. The patient healed well. She reported minimal discomfort and had taken analgesics only on the first two postoperative days. At 10 days postoperatively, tiny pale brown pin-point pigmented spots were seen to have appeared against the pale pink depigmented attached gingiva. One month postoperatively light brown pigmentation was seen extending apically from the marginal gingiva halfway towards the mucogingival junction. At two months postoperatively the light brown pigmentation extended to cover almost the entire gingiva. By the third month, diffuse light brown repigmentation was present. Six months later the patient was reviewed. Repigmentation had uniformly occurred but was much lighter than what it initially was. The pale pink gingival colour obtained soon after

depigmentation stood out with respect to the patients complexion. The final repigmented result provided better esthetics as the gingiva was a light brown colour which blended well with the patient's complexion, especially considering her high smile line.

CONCLUSION:

The colour of gingiva in health may vary from a pale pink to a deep bluish purple, differing from one individual to the next. Local, systemic, environmental and genetic factors play a role in its presentation. While physiologic pigmentation is normal and does not pose a medical problem, the practitioner should be aware of the varied differential diagnosis which may necessitate diagnostic tests. Various techniques are available today for depigmentation of gingiva, each with their own advantages and disadvantages. The choice of technique would depend on patient affordability and individual preference and expertise. Repigmentation is a frequent sequelae to depigmentation, and has several factors that affect it that are yet to be understood thoroughly.

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FIGURES:

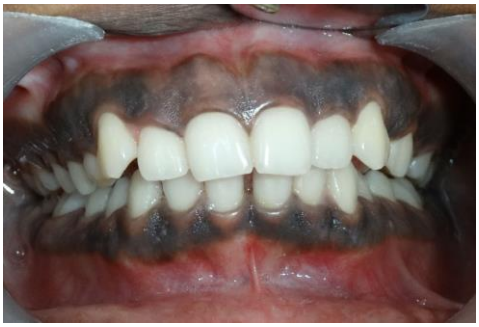

Figure 1: Preoperative view.

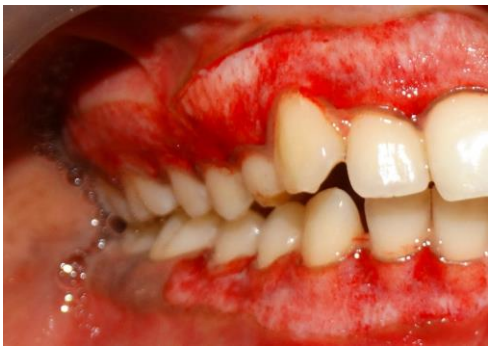

Figure 2: Depigmentation completed on the right side.

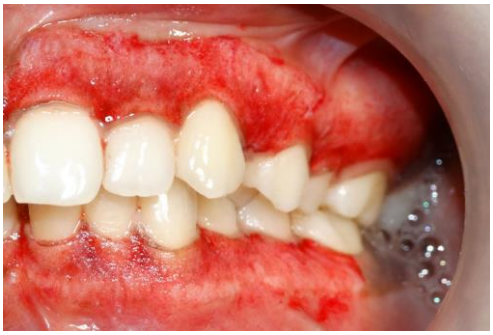

Figure 3. Depigmentation completed on the left side.

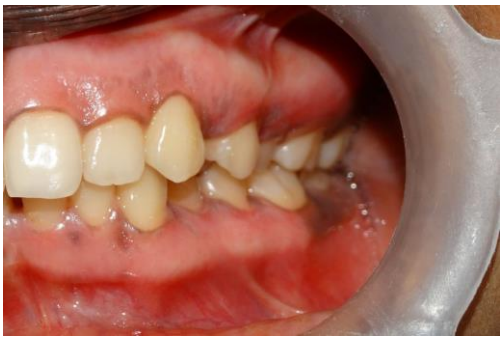

Figure 4: Ten days postoperatively

Figure 5: One month postoperatively.

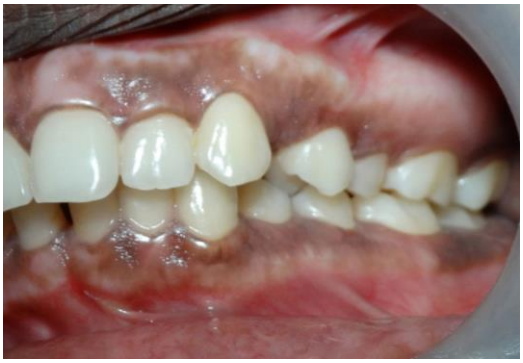

Figure 6: Two months postoperatively.

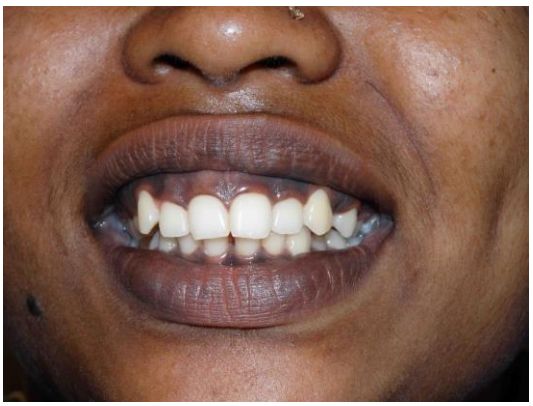

Figure 7: Before depigmentation.

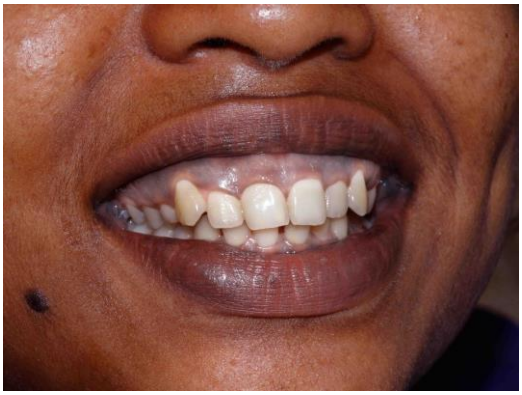

Figure 8: Six months after depigmentation. Notice that repigmentation has occurred, but is lighter than that before depigmentation. In this case, repigmentation provided better esthetics especially in relation to the complexion of the patient. The gingival colour at 10 days, if maintained, would have been “too pink”, resulting in an unesthetic appearance.