REVIEW ARTICLE

GINGIVAL PIGMENTATION: REVISITED

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ABSTRACT:
Cosmetic expectations have increased with time and current trends speak volumes about gingival esthetics and smile designing. Gingival pigmentation especially on the labial aspect of anterior teeth has become an important component of general esthetics. Pigmentation is both the normal and abnormal discoloration of oral mucous membrane. Pigmentation has multifactorial etiology. Most of the pigmentation is physiologic but sometimes it can be a precursor of severe diseases. Gingival hyperpigmentation are major concerns for a large number of patients visiting the dentist. Melanin hyperpigmentation usually does not present a medical problem, but patients usually complain of dark gums as unaesthetic. The differential diagnosis, clinical, etiology, and histopathological features of pigmentation are discussed and the current literature is reviewed.
Key words: Esthetics, gingival pigmentation, melanin.

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INTRODUCTION

The normal physiologic colour attributed to gingiva is coral pink or salmon pink, with physiological variations of melanin pigmentation. Melanin pigmentation of gingiva is common in dark-skinned individuals. Melanin hyperpigmentation may possess a defensive role against progress of gingival inflammation.[1] Melanin is the most common endogenous pigment present in the body. It is a nonhemoglobin-derived brown pigment produced by melanocytes and is also a powerful cation chelator.[2] Melanocytes are dendritic cells of neuroectodermal origin. They work independent of the surrounding epithelial cells and behave as unicellular exocrine gland that convert tyrosine to melanoprotein (melanin), which is transferred to keratinocytes by way of melanosomes. Thus, the melanin is deposited in the basal layer of the oral epithelium.[3] Most of the pigmentation is caused by five primary pigments. These include: melanin, melanoid, oxyhemoglobin, reduced haemoglobin, and carotene[4] (Figure1).

Melanin

Melanin, a non hemoglobin derived brown pigment, is the most common of the endogenous pigments and is produced by melanocytes present in the basal layer of the epithelium. Melancocytes have a round nucleus with a double nucleus membrane and clear cytoplasm lacking desmosomes or attachment plates. Melanin accumulates in the cytoplasm, and the melanosome is transformed into a structureless particle no longer capable of melanogenesis. The number of melanocytes in the mucosa corresponds numerically to that of skin; however, in the mucosa their activity is reduced. Various stimuli can result in an increased production of melanin at the level of mucosa including trauma, hormones, radiation, and medications. Thyrosinase activity is present in premelanosome and melanosomes but absent in melanin granules.[5]

Melanoid

Granules of melanoid pigment are scattered in the stratum lucidum and stratum corneum of the skin. Initially it was assumed melanoid was a degradation product of melanin, but more recently it has been shown that such a relationship is highly improbable. Melanoid imparts a clear yellow shade to the skin.[4]

Oxyhemoglobin and Reduced Hemoglobin

Oxyhaemoglobin and reduced haemoglobin are pigments resulting from haemosiderin deposits. The skin colour is affected by the capillary and venom plexuses shining through the skin.[4]

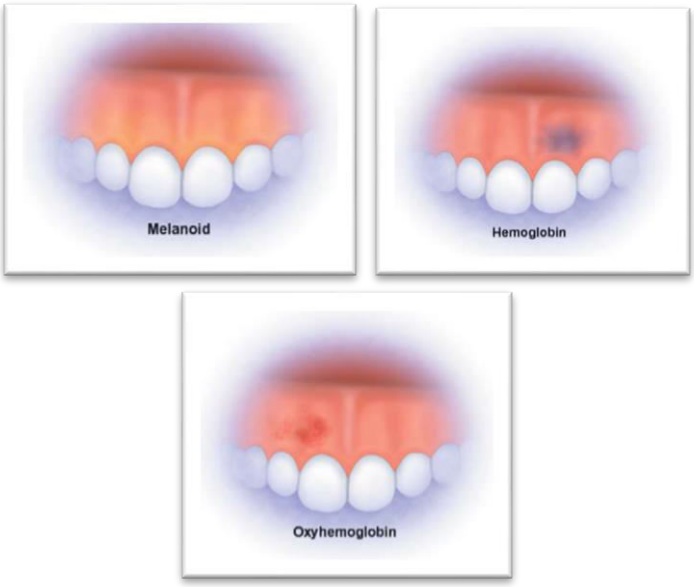

Figure 1: Various pigments causing pigmentation

Carotene

Carotene is distributed in the lipids of the stratum corneum and stratum lucidum and gives a deep yellow colour to the skin. It is found in higher concentrations in more women than in men.[6]

Melanin Synthesis and Physiologic Functions

Melanin is a pigment produced by melanocytes that reside in the basal layer (stratum básale) of the epidermis. It is stored in vesicles called melanosomes and is transferred to adjacent epithelial cells via dendritic processes. Melanin protects DNA from the ionizing, damaging effect of UV radiation. It absorbs the UV radiation and transforms it to heat through a process described as "ultrafast internal conversion".[7] While melanin is present in all individuals, with the exception of albinos, pigmentation may not always be clinically detectable. Melanin is the primary determinant for human tissue coloration, including the skin, mucosa, hair, iris and parts of the brain.

While the melanocyte concentration and distribution in the human skin is similar between individuals, the expression pattern of melanin may vary significantly, explaining the variation in skin pigmentation among ethnic groups. Human skin pigmentation is a highly variable trait among human populations.[8]

The intensity of melanin pigmentation in the skin is considered an environmental adaptation that regulates the level of UV radiation penetration into the epidermis. Melanin pigmentation pattern is determined by the requirement for vitamin D3 synthesis and by the need for photoprotection, and presents with a characteristic geographic distribution. Human populations closer to the equator with higher annual UV radiation have increased need for protection from UV induced injury and therefore have darker skin, while populations in the poles with limited annual sunlight exposure have lighter skin.[9]

The importance of highly melanized skin has been established as it offers greater protection both against the deleterious effect of the UV radiation of sunlight (skin burns, dermal malignancies) and prevents nutrient photolysis. However, melanin's role in the pigmentation of the oral tissues remains unclear. Melanin, ubiquitously expressed in most organisms, is a polymer composed of polyacetylene, polypyrrole and polyalanine. It is a derivative of tyrosine and is comprised of different proportions of smaller component molecules that give rise to three types of melanin: eumelanin, pheomelanin and neuromelanin.[10]

Eumelanin comprises of numerous crosslinked 5, 6-dihydroxyindole (DHI) and 5, 6-dihydroxyindole-2-carboxylic acid (DHICA) polymers. Eumelanin is the most abundant form in humans. It is the main determinant for the skin and hair coloration. The two types of eumelanin, black and brown, are responsible for colours black, grey, brown and yellow. Small amounts of black eumelanin in the absence of other pigments result in grey (hair), while small amounts of brown eumelanin in the absence of other pigments result in blond. Brown eumelanin is usually present in young Europeans while older Europeans and non-Europeans express mostly black eumelanin (Figure 2).

Figure 2:

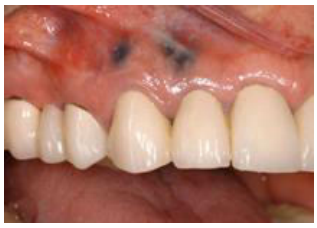

Figure 3a: Amalgam tattoo

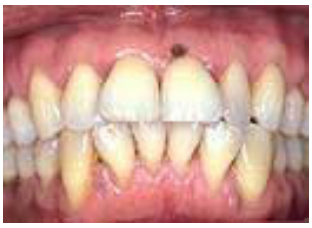

Figure 3b: Pigmented naevi

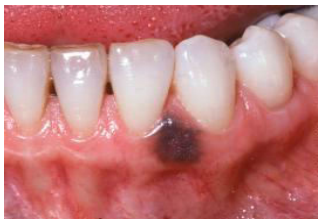

Figure 3c: Melanotic macule

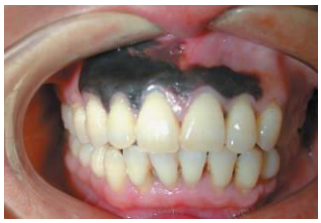

Figure 3d: Melanoma

Pheomelanin is also found in the skin and hair and attributes a reddish hue. Its polymeric structure contains benzothiazine and requires L-cysteine instead of DHI and DHICA in eumelanin.

Neuromelanin is a dark pigment found in the dopamine and noradrenergic neurons in the substantia nigra pars compacta and locus coeruleus of the human brain, increasing with age and reaching its peak around the age of 20 years.[11]

EPIDEMIOLOGY

Oral pigmentation occurs in all races of man though there range varies from one race to another. There were no significant differences in oral pigmentation between males and females. The intensity and distribution of racial pigmentation of the oral mucosa is variable, not only between races, but also between different individuals of the same race and within different areas of the same mouth. Physiologic pigmentation is probably genetically determined, but as **Dummett** suggested, the degree of pigmentation is partially related to mechanical, chemical, and physical stimulation.[12] In darker skinned people oral pigmentation increases, but there is no difference in the number of melanocytes between fair-skinned and dark-skinned individuals. The variation is related to differences in the activity of melanocytes.[13]

Physiological pigmentation of the oral mucosa (mostly gingiva), is clinically manifested as multifocal or diffuse melanin pigmentation with variable amounts in different ethnic groups worldwide and it occurs in all races.[14] In Caucasians, most melanocytes have striated granules that are incompletely melanized and vary in size from 0.1 to 0.3 mm. But, the amount is insufficient to cause pigmentation (less than 10%

demonstrate pigmentation). A high amount of melanin granules is found in individuals of African and East Asian ethinicity.[15] In dark-skinned and black individuals, an increased melanin production has long been known to be the result of genetically determined hyperactivity of melanocytes. Melanocytes of dark skinned and black individuals are uniformly highly reactive, whereas in light skinned individuals, melanocytes are highly variable in reactivity.[16]

ETIOLOGY:-

Systemic and Local Causes of Pigmentation

1. **Amalgam Tattoo**:-

The pigmentation of the oral mucous membrane by tooth restoration material (amalgam) is a common finding in dental practice. Amalgam pigmentation is generally called amalgam tattoo (Figure3a). The lesion represents embedded amalgam particles and usually manifests itself as an isolated bluish or black macule in various areas of the mucosa. The colour is usually described as black, blue, grey, or a combination of these. Almost half were located on the gingiva and alveolar mucosa, the mandibular region being affected more than the maxillary region.[17]

2. Pigmented Nevi:-

Pigmented nevi of the oral cavity are uncommon. The pigmented nevi are classified as intramucosal, junctional, compound, or blue according to their histological features. Nevi are seen particularly on the vermillion border of the lips and the gingivae (Figure3b). They are usually grey, brown, or bluish macules and are typically asymptomatic. Melanocytes are pigment producing cells characterized by the ability to syntesize via the enzyme dihydroxyphenylalanine (DOPA).[18]

3. **Oral Melanotic Macules** :-

Oral melanotic macules are relatively rare oral mucosal lesions, analogous to skin freckles, due to the focal increase of melanin production. These melanotic macules have been variously termed ephelis, melonosis, lentigo, solitary labial lentigo, labial melanotic macule, and oral melanotic macule (Figure 3c). The vermillion border of the lower lip is most commonly involved. The buccal mucosa, palate, and gingiva are less commonly affected. The color is usually described as grey, brown, blue, black, or a combination of these.[17]

4. Melanoma:-

Melanoma is a cancerous condition of the melanocyte. Special corpusles in this cell, known as melanosomes, contain the necessary enzyme (tyrosine) to transform amino acids into melanin. Melanocytes are found among the basal cells of the epidermis (Figure 3d). Histopathogically, the mucosal epithelium is abnormal with large atypical melanocytes and excessive melanin. Malignant melanoma of the oral mucosa affects both sexes equally usually after 40 years of age. The great majority of the lesions (about 70-80%) occur on the palate, upper gingival, and alveolar mucosa. Initially there usually is a solitary small asymptomatic brown or black macule.[19]

5. Physiologic Pigmentation:-

Physiologic pigmentation of the oral mucosa is clinically manifested as multifocal or diffuse melanin pigmentation with variable prevalence in different ethnic groups. Melanin is normally found in the skin of all people. In dark skinned persons the gingiva may contain melanin pigment to a greater extent than the adjacent alveolar mucosa. If pigmented gingiva is surgically resected, it will often heal with little or no pigmentation; therefore, surgical procedures should be designed so as to preserve the pigmented tissues.[20]

6. Peutz-Jeghers Syndrome :-

Peutz-Jeghers syndrome (intestinal polyposis) is a genetic disorder characterized by mucocutaneous pigmentation and hamartomas of the intestine. It manifests itself as frecklelike macules about the hands, perioral skin, and intraorally to include the gingiva, buccal, and labial mucosa. Pigmented spots are 1 to l0 mm in diameter. Pigmented spots are particularly found on the lower lip and buccal mucosa but rarely on the upper lip, tongue, palate, and gingiva.[21]

Smoker's Melanosis:- Smoker's melanosis is a benign focal pigmentation of the oral mucosa. It tends to increase significantly with tobacco consumption. Tobacco smokers have significantly more oral surfaces pigmented than non-tobacco users. Clinically, the lesion usually presents as multiple brown pigmented macules less than 1 cm in diameter, localized mainly at the attached labial anterior gingival and the interdental papillae of the mandible.[22]

7. **Antimalaria Drug Use** :-

Several antimalarial drugs are known to be capable of inducing intraoral melanin pigmentation. These drugs include: quinacrine, chloroquine, and hydroxychloroquine. Long term use may cause pigmentation of the oral mucosa. The pigmentation of the oral mucosa is described as slate-grey in color, bearing some resemblance to pigmentation caused by silver arsplenamine.[23]

8. Minocycline Use:-

Minocycline is a synthetic tetracycline that is commonly used in the treatment of acne vulgaris.

Although tetracycline causes pigmentation of bones and teeth, minocycline alone is also responsible for soft tissue pigmentation. It is usually seen as brown melanin deposits on the hard palate, gingiva, mucous membranes, and the tongue.[24]

9. Heavy Metals:-

Heavy metals absorbed systemically from therapeutic use or occupational environments may discolor the gingiva and other areas of the oral mucosa. Bismuth, arsenic, and mercury produce a black line in the gingival which follows the contour of the margin. Lead results in a bluish red or deep blue linear pigmentation of the gingival margin (Burtonian line). Exposure to silver causes a violet marginal line, often accompanied by a diffuse bluish-grey discoloration throughout the oral mucosa.[25]

10. Addison's Disease:-

Addison's disease or primary adrenocortical hypofunction is due to adrenocortical damage and hypofunction. Bronzing of the skin and increased pigmentation of the lips, gingivae, buccal mucosa, and tongue may be seen. Oral pigmentation may be the first sign of the disease. A biopsy of the oral lesions shows acanthosis with silver-positive granules in the cells of the stratum germinativum. Melanin is seen in the basal layer.[26]

11. Periodontal Diseases :-

Periodontal diseases often produce discolorations of the oral mucosa. The pigmentation is worsened by gingivitis, which increases vascular permeability and allows the heavy metals access to the soft tissues. Melanin re-pigmentation is related to after surgical injury.[27]

12. Hemachromatosis :-

Hemachromatosis (bronze diabetes) is a chronic disease characterized by the deposition of excess iron (ferritin and hemosiderin) in the body tissues, resulting in fibrosis and functional insufficiency of the involved organs. Hyperpigmentation may appear both in skin and mucous membranes (oral and conjunctiva). Gingival or mucosal pigmentation is reported to occur in 15 to 25% of patients with hemachromatosis. The oral mucosa shows diffuse homogeneous pigmentation of gray-brown or deep brown in about 20% of the cases. The buccal mucosa and the attached gingiva are the most frequently involved sites.[22]

13. HIV Infection :-

In patients infected with human immunodeficiency virus (HIV), progessive hyperpigmentation of the skin, oral mucosa, fingernails, and toenails have been reported being related to primary adrenocortical deficiency and to zidovudine (azidothymidine) therapy in some cases. Clinically, oral pigmentation appears as irregular macules with brown or dark brown color. The tongue, buccal mucosa, and palate are the most commonly affected sites.[28]

INDICES ON GINGIVAL PIGMENTATION:

GP has three dimensions: etiology, distribution, and severity. The existing indices on GP are as follows:- Dummet CO, Gupta OP (1964) proposed Oral pigmentation index (DOPI). Hedin CA (1977) proposed Melanin index. Hanioka T (2005) proposed Melanin pigmentation index. Kumar S (2012) proposed Gingival pigmentation index.[29]

The recent indices on GP is given by Peeran et al (2014)[29] which stated that, all the before mentioned indices seem to lack the capacity to relate various aspects of GP. They are also not determining the patient's treatment need. Moreover, other gingival-pigmented lesions are beyond their scope, as they were intended only for racial pigmentation.

Therefore the authors proposed **gingival melanin pigmentation and pigmented lesions index:-**

Score 0	Coral pink-colored gingiva, no gingival pigmentation, and/or pigmented lesions.
Score 1	Mild, solitary/diffuse, gingival melanin pigmentation involving anterior gingiva, with or without the involvement of posterior gingival
Score 2	Moderate to severe, solitary or diffuse, gingival melanin pigmentation involving anterior gingiva with or without the involvement of posterior gingival.
Score 3	Gingival melanin pigmentation only in posterior gingival
Score 4	Tobacco-associated pigmentation: Smoker's melanosis, chewing tobacco
Score 5	Gingival pigmentation due to exogenous pigments-Amalgam tattoos, arsenic, bismuth, chewing betel nut, cultural gingival tattooing, drinks, food colors, lead-burtonian line, mercury, silver, topical medications, idiopathic etc
Score 6	Gingival pigmentation due to other endogenous pigments: Bilirubin, blood breakdown products, ecchymosis, hemochromatosis, hemosiderin, petechiae etc
Score 7	Drug-associated gingival pigmentation: Antimalarial drugs, minocycline, oral contraceptives etc
Score 8	Gingival pigmentation associated with other causes: Addison's disease, albright's syndrome, basilar melanosis with incontinence, hereditary hemorrhagic telangiectasia, HIV patients, lichen planus, neurofibromatosis, Peutz-Jeghers syndrome, granulomatous epulis etc
Score 9	Pigmented benign lesions: Hemangioma, melanocytic nevus, pigmented macule.
Score 10	Pigmented malignant lesions: Angiosarcoma, Kaposi's sarcoma, malignant melanoma'

In this proposed index, 0-3 is the range available to record the gingival color and its variation within physiological limits. A clinician may recommend a depigmentation procedure when the patient scores 1-2 of our index and has up to class 2 of **Liebart and Deruelle "Smile line classification",** [30]which is as follows:

Class 1: Very high smile line - more than 2 mm of the marginal gingiva visible.
Class 2: High smile line - between 0 and 2 mm of the marginal gingiva visible.
Class 3: Average smile line - only gingival embrasures visible.
Class 4: Low smile line - gingival embrasures and cemento-enamel junction not visible.[30]

CLASSIFICATION:-
Pigmented lesions of the oral cavity are of multiple origin. Different classifications are used at this time. Some researchers divide the lesions into two main groups as either endogenous or exogenous lesions.

Peeran et al (2014)[29] proposed a new improved classification for gingival pigmentation and pigmented lesions. The authors concluded that Due to clarity and simplicity of the proposed index, this classification can be applied even by naïve professionals. The broad implementation of this classification and index may facilitate the comparison of GP across the globe and help esthetic management of such presentations. The classification is as follows:

CLASS	CRITERIA OF CLASSIFICATION
I	Coral pink/salmon pink colored gingiva
II	Localized/Isolated spots/areas of gingival melanin pigmentation which does not involve all the three parts of gingiva, that is, attached, free, and papillary gingiva • Mild to moderate pigmentation • Severe/intense pigmentation
III	Localized/Isolated unit/s of melanin pigmentation which involve all the three parts of gingiva, that is, attached, free, and papillary gingiva • Mild to moderate pigmentation • Severe/intense pigmentation
IV	Generalized diffuse pigmentation which involve all the three parts of gingiva that is, attached, free, and papillary gingiva. • Mild to moderate pigmentation • Severe/intense pigmentation
V	Tobacco associated pigmentation like smoker's melanosis and chewing tobacco
VI	Gingival pigmentation due to exogenous pigments eg:-Amalgam tattoos, Cultural gingival tattooing, Drinks, Food colors, Habitual betelnut/khat chewing, Lead-Burtonian line, Mercury, Silver, Arsenic, Bismuth, Graphite, Other foreign bodies, Topical medications, Idiopathic.
VII	Gingival pigmentation due to endogenous pigments like Bilirubin, Blood breakdown products, Ecchymosis, Petechiae, Hemochromatosis, Hemosiderin.
VIII	Drug-induced gingival pigmentation like ACTH, Antimalarial drugs, Chemotherapeutic agent-busulfan and doxorubicin, Minocycline, Oral contraceptives, Phenothiazines.
IX	Gingival pigmentation associated with systemic diseases and syndromes like Addison's disease, Albright's syndrome, Basilar melanosis with incontinence, Beta thalassemia; Healed muco-cutaneous lesions-Lichen planus, Pemphigus, Pemphigoid; Hereditary hemorrhagic telangiectasia; HIV-associated melanosis, Neurofibromatosis, Peutz-Jeghers and other familial hamartoma syndromes, Pyogenic granuloma/Granulomatous epulis.
X	Pigmented benign and malignant lesions involving the gingival like Angiosarcoma, Hemangioma, Kaposi's sarcoma, Malignant melanoma, Melanocytic nevus, Pigmented macule.

MANAGEMENT
Roshna T et al (2005) enumerated Techniques Employed For Gingival Depigmentation

I. Methods aimed at removing the pigment layer
- **A.** Surgical methods of depigmentation
  - **a.** Scalpel surgical technique.
  - **b.** Cryosurgery.
  - **c.** Electro surgery.
  - **d.** Lasers.
    - Neodymium: Aluminium-Yttrium-Garnet (Nd:YAG) lasers.
    - Erbium:YAG (Er:YAG) lasers
    - Carbon dioxide (CO_2) lasers.
- **B.** Chemical methods of depigmentation using caustic chemicals:- this method is not used nowadays.

II. Methods aimed at masking the pigmentad gingival with grafts from the less pigmented areas.
- A. Free gingival grafts
- B. Acellular dermal matrix allografts.[31]

Non-surgical approaches as well as surgical intervention have been suggested for the management of melanin pigmentation.

Non-surgical approaches:
1. The use of pharmacological agents (monobenzone, mequinol or hydroquinone) has been applied in cases where skin de-pigmentation is required, as in the treatment of vitiligo.[32] Hydroquinone and its derivatives, monobenzone and mequinol, inhibit the production of melanin and have been used for whitening of the skin.

Hydroquinone is not used in the US in over-the-counte preparations, and the FDA has included the drug as potentially carcinogenic (US FDA 2006).[33]

2. The use of 90% phenol or 95% ethanol solutions to reduce oral pigmentation by inducing chemical burn and sloughing of the epithelium. However repigmentation and relapse occurred in all cases shortly after the application of either agent. [34]

Alternative surgical approaches have been reported for the elimination of melanin gingival pigmentation, including free gingival grafts, gingivectomy, deepithelialization by bur abrasion, scalpel, laser and cryosurgery.

The potential of autogenous epithelialized gingival grafts has been established for the management of physiologic gingival pigmentation or amalgam tattoos.[20,35]

Additionally, **Tamizi and Taheri,**[20] **and Fowler et al.**[36]**,** also reported the use of free gingival grafts for the elimination of gingival melanin pigmentation.

Interestingly, **Fowler et al.** used the free gingival graft technique to eliminate an aberrant maxillary labial frenum and to increase melanin pigmentation at the surgical site. For that purpose, they utilized a graft from a donor site rich in melanin pigmentation. As an alternative to the free gingival graft, the use of an acellular dermal matrix (Alloderm, LifeCell Corporation, Woodlands, Texas) has been advocated for the management of gingival melanin pigmentation and amalgam tattoos. [36]

Pontes et al. compared the use of alloderm vs de-epithelialization of the gingiva in fifteen patients." In the alloderm sites, partial thickness flaps were reflected and excised at the base, and the alloderm was sutured over the periosteum. In the contralateral sites, the connective tissue was denuded with a diamond bur. The authors reported superior results and minimal repigmentation in sites treated with alloderm 12 months post-operatively compared to deepithelialization.[37]

Connective tissue (CT) grafts may also be used for the management of gingival pigmentation with simultaneous or subsequent removal of the overlying epithelium in one or two phases. While CT grafts have not been reported for the management of melanin gingival pigmentation, their use has been documented for the management of amalgam or graphite tattoos.[38]

De-epithelialization may be achieved by various techniques including the use of a scalpel in gingivectomy procedures (Figure 4). The pigmented epithelium and the underlying connective tissue support are excised. However, this may not offer permanent results as pigmentation relapsed in all cases in 36 months.[39]

The depigmentation procedure by scalpel technique is simple, easy to perform, noninvasive, and above all, cost effective. According to Almas and Sadiq (2002), the scalpel wound heals faster than that in other techniques. But scalpel surgery causes unpleasant bleeding during and after the operation. It is also necessary to cover the exposed lamina propria with periodontal dressing for 7–10 days.[40]

De-epithelialization with a highspeed handpiece and a diamond bur (2 mm or 2.5 mm of diameter) has been proposed by Farnoosh (Figure 5). He described the use of feather-like brushing strokes under copious water lavage using large burs, since smaller burs may not provide a smooth surface and may create small pits and irregularities in the gingival contour. The removal of all the remnants of the melanin-containing epithelium was recommended to prevent relapse.[41]

Farnoosh also proposed that de-epithelialization may be combined with a flap procedure if the patient has periodontitis and the tissue is thick enough so that flap survival would not be compromised. The technique requires "shaving" of the epithelial layer with a surgical blade under local anesthesia with epinephrine for control of the bleeding. The surgical wound was covered with a periodontal dressing. Residual pigmentation was observed two weeks post-operatively and was removed at a later visit.[41]

Elecrosurgery uses electric energy to cause molecular disintegration of melanin cells of operated and surrounding sites, as explained by Olinger's "Exploding cell theory". Contact of the electrode with periosteum and vital teeth may cause damage to the tissues; hence it is technique sensitive and requires expertise (Figure 6a and 6b). **Cicek (2003)** reported that there is no bleeding and there is minimal patient discomfort while using electrocautery. But electrosurgery also has its own limitations in that its repeated and prolonged use induces heat accumulation and undesired tissue destruction.[42]

The use of **LASERS** has also been proposed for the management of oral melanin pigmentation. The **$C0_2$**, **Er.Cr:YSGG** and **Nd:YAG LASERs** have been used(Figure 7). The Nd:YAG LASER with an invisible, near-infra-red light (wavelength of 1,064 nm) has a high affinity for dark pigments, making it particularly suited for depigmentation. Here, radiation energy is transformed into ablation energy, resulting in cellular rupture and vaporization with minimal heating of the surrounding tissue.[40]

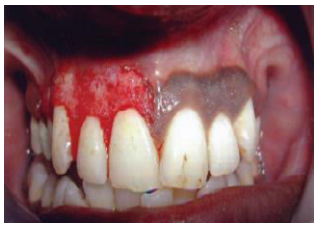

Figure 4

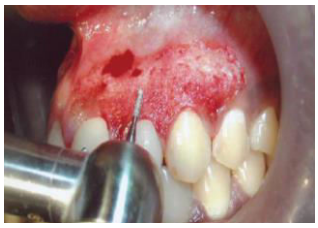

Figure 5

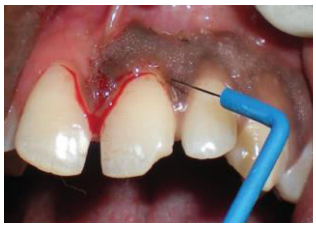

Figure 6a

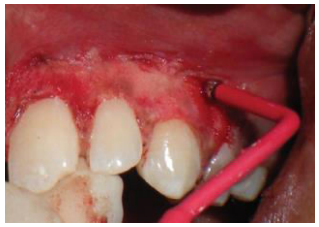

Figure 6b

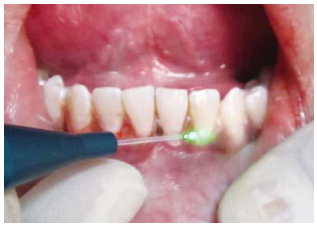

Figure 7

Figures: 4) Scalpel de-epithelization procedure; **5)** Bur abrasion; **6a**) Needle electrode to give incision; **6b**) Ball electrodes used for coagulation; **7**) LASER depigmentation.

According to Atsawasuwan and Greethong (1999), laser beam produces bloodless field for surgery, causes minimum damage to the periosteum and underlying bone, and the treated gingiva and mucosa do not need any dressing. This has the advantages of easy handling, short treatment time, hemostasis, and decontamination and sterilization effects. But this approach needs expensive and sophisticated equipment, which makes the treatment very expensive. Laser beam even destroys the epithelial cells including those at the basal layer, and hence reduces repigmentation. Thus, repigmentation was minimal and patient compliance was much better in this case series while using lasers with than other techniques.[43]

However, ablation should be performed with caution in areas of thin tissue and prominent roots, as gingival fenestration and bone exposure may occur. The advantages of this technique include minimum damage to the underlying tissues when used cautiously, speed of the procedure and minimal bleeding. However, more time is required for the healing of the periodontal tissues.[42]

Cryosurgery has also been proposed for the management of melanin gingival pigmentation. Tal et al. reported the use of a gas expansion cryoprobe cooled to -81^{o}C and applied to the pigmented gingiva for 10 seconds. Gingiva was thawed spontaneously within 1 minute, and necrosis became apparent within 1 week. Healing and keratinization was complete within 3-4 weeks and depigmentation was successful 20 months post-operatively. The use of liquid nitrogen has also been tested in patients with melanin pigmented gingival. The liquid nitrogen (-196^{o}C) was applied directly to the gingiva with a cotton swab in one or two visits. No relapse was reported in 20 patients followed for 3-24 months. Cryosurgery requires the use of additional materials, and depth control is difficult. The risk of increased tissue destruction needs to be considered. The gaseous fluorocarbon tetrafiuoroethane (TFE), used in the field of endodontics for cold pulp testing, is readily available and has also been tested for gingival melanin depigmentation.[44,45]

CONCLUSION

Aesthetic concern requires removal of pigmented gingival areas to create a pleasant and confident smile. Oral melanin pigmentation can be eliminated by a variety of surgical techniques, including free gingival grafts and soft tissue allografts and de-epithelialization by bur abrasion, scalpel, laser and cryosurgery. However, randomized controlled longitudinal studies are needed to establish the efficacy and applicability of these techniques.

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