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REVIEW ARTICLE

DEPIGMENTATION: A NOVEL CLASSIFICATION AND REVIEW

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ABSTRACT

A healthy smile provides a boost of confidence to an individual. Gingival hyperpigmentation is a common complaint among individuals when esthetics is a prime concern. Gingival depigmentation is a periodontal plastic surgical procedure whereby the epithelium is denuded of pigment cells so as to produce a more desirable pink colour of the gums. The current review aims at providing a wholesome knowledge of gingival pigmentation, its physiology, pigments in the human body, classification and treatment modalities available for gingival depigmentation. The authors have introduced a novel classification system taking into account both the etiology and the region affected.

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INTRODUCTION

A winning smile can boost an individual's confidence to have successful social interactions. Dark pigmentation of the gums unlike the esthetics provided by pink gingiva can lay down heavily on an individual's day to day interactions. Presence of physiologic pigmentation in the oral cavity is usually seen due to deposition of melanin. Such pigmentation can be treated to provide a patient with an esthetic smile line.

Pigments in the oral mucosa: Dummet in 1967 stated that six sources of pigment contributed to the normal color of the gingiva namely, melanin, melanoid, carotene, reduced hemoglobin, soft keratin and oxyhemoglobin (Dummett and Barens, 1967). More recent research has concluded that gingival pigmentation can be attributed to five primary pigments which are: melanin, melanoid, oxyhemoglobin, reduced haemoglobin, and carotene (Cicek and Ertas, 2003). Melanin: It is the most common pigment produced by melanocytes in the basal layer of the epithelium. The number of melanocytes present in the epithelium usually corresponds with those in the skin. However, in mucosa their activity is reduced. Various stimuli such as trauma, body hormones, radiation and medications may increase formation of melanin within the mucosa.

Melanoid: This pigment is known to impart a clear yellow shade and its granules are scattered through the stratum lucidum and stratum corneum of the skin.

Oxyhemoglobin and Reduced Hemoglobin: These pigments are products of hemosiderin deposits.

Carotene: This pigment imparts a deep yellow colour to the skin and is frequently found in higher concentrations in females.

Melanin synthesis and physiologic functions: Melanin is the primary determinant of colour of human skin, mucosa, hair, etc. The process of pigmentation consists of a phase of activation of melanocytes, synthesis of melanin and its expression. Environmental factors like sunlight and stress hormones trigger release of melanocyte stimulating hormone which in turn activates melanocytes. Melanocytes are dendritic cells located in the basal and spinous layers of the gingival epithelium. Upon stimulation, melanocytes contain granules called melanosomes. They are formed as a result of conversion of tyrosine to dihydrophenylalanine (DOPA) in the presence of tyrosinase. DOPA is then converted to dopaquinone. After a series of reactions dopaquinone is converted to either eumelanin or pheomelanin. Once melanin is synthesized within the melanosome, melanosomes are transferred to keratinocytes. Melanin is then transferred to adjacent epithelial cells through its dendritic processes (Newman *et al.,* 2018; Gulati *et al.,* 2016; Moneim *et al.,* 2017). Melanin prevents UV radiations from damaging DNA by absorbing the UV radiation and transforming it to heat. It regulates the amount of UV radiation penetrating the skin. The requirement for vitamin D3 synthesis depending on environmental factors such as geographic

location where an individual stays governs the pigmentation pattern. This explains why individuals staying close to the equator have darker skin.

Depending on the chemical composition melanin can be of three types: ***eumelanin, pheomelanin and neuromelanin*** Eumelanin is the most abundant form of melanin found in the human body and determines the colour of skin and hair. It may be black or brown and is responsible for imparting black, grey, brown and yellow colours. Reduced amounts of black eumelanin pigment in absence of other pigments results in grey hair colour. Whereas small amounts of brown eumelanin results in a blonde appearance of hair. Pheomelanin is known to impart a reddish hue to skin and hair. Neuromelanin is a dark pigment which increases with age and reaches its peak around 20 years of age (Gulati *et al.,* 2016).

Types of gingival pigmentation (Classification): Gingival pigmentation may be physiologic or pathologic (Moneim *et al.,* 2017).

I Physiologic gingival pigmentation: This kind of pigmentation develops upto 20 years of age. It is asymptomatic and does not require any kind of treatment. The pigmentation may be uniformly distributed throughout the gingiva and oral tissues or may be diffuse. The intensity of pigmentation differs among different ethnic groups. Attached gingiva is the most commonly pigmented as compared to other areas.
II Pathologic gingival pigmentation: Various systemic diseases may contribute to abnormal discolouration of the gingiva. Some of these are listed below:
a. Endocrine disorders b. Heavy metal toxicity caused by lead, bismuth, silver, arsenic and gold. c. Kaposi's Sarcoma d. Drugs such as chloroquine, quinine, minocycline, zidovudine, chlorpromazine, ketoconazole, bleomycin, cyclophosphamide can cause accumulation of melanin pigments. Minocycline is also association pigmentation of the gingiva and lips. e. Post-inflammatory pigmentation: long standing inflammatory mucosal lesions, mainly lichen planus can cause mucosal pigmentation f. Smoking associated melanosis g. Hemangioma: Lesions that are superficially placed appear reddish in colour while deeper lesions have a bluish tinge. h. Amalgam tattoo i. Graphite tattoo j. Nevocellular nevus and blue nevus k. Oral melanoacanthoma: the term was first used to describe a benign mixed skin tumor composed of basal and prickle cell keratinocytes and pigment laden dendritic melanocytes l. Mucosal melanomas m. HIV oral melanosis

Another classification proposed by Peeran *et al.* (2014) is described as follows:

Class	Criteria of classification
I.	Coral pink/salmon pink colored gingival
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: 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: Bilirubin, Blood breakdown products: Ecchymosis, Petechiae, Hemochromatosis, Hemosiderin
VIII.	Drug-induced gingival pigmentation: ACTH, Antimalarial drugs, Chemotherapeutic agent-busulfan and doxorubicin, Minocycline, Oral contraceptives, Phenothiazines
IX.	Gingival pigmentation associated with systemic diseases and syndromes: 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 gingiva: Angiosarcoma, Hemangioma, Kaposi's sarcoma, Malignant melanoma, Melanocytic nevus, Pigmented macule

Causes of gingival pigmentation (Farid *et al.,* 2017)

Causes of gingival hyperpigmentation may be exogenous or endogenous as listed below:

Exogenous causes	
a.	Heavy metals: gold, bismuth, arsenic, mercury, silver, lead, copper
b.	Tattoos: Amalgam, graphite
c.	Smoking
d.	Medications: Antimalarials, minocycline, ketoconazole, oral contraceptives.
Endogenous causes	
a.	Genetic: Peutz-Jegher's Syndrome, Von Reckling Hausen's disease, Gaucher's disease, etc
b.	Physiological: Pregnancy
c.	Endocrine disturbances: Addison's disease, Albright's syndrome.
d.	Infection: HIV
e.	Blood dyscrasias: Thalassemia
f.	Liver disorder: Jaundice
g.	Benign and malignant neoplasms
h.	Trauma.

Indices for gingival pigmentation (Gulati *et al.,* 2016; Peeran *et al.,* 2014)

Current literature provides five indices for gingival pigmentation which are described in brief as follows:

1.Dummet and Gupta (1964) gave the Oral pigmentation index (DOPI) scored as:

No clinical pigmentation (pink-colored gingiva)
Mild clinical pigmentation (mild light brown color)
Moderate clinical pigmentation (medium brown or mixed pink and brown color)
Heavy clinical pigmentation (deep brown or bluish black color)

2. Hedin CA (1977) proposed Melanin index described as follows:

No pigmentation
One or two solitary unit(s) of pigmentation in papillary gingiva
More than three units of pigmentation in papillary gingiva
One or more short continuous ribbons of pigmentation
One continuous ribbon including the entire area between canines

3. Hanioka T (2005) proposed Melanin pigmentation index:

Score 0	No pigmentation
Score 1	Solitary unit(s) of pigmentation in papillary gingiva
Score 2	Formation of continuous ribbon extending from neighboring solitary units

4.Kumar S (2012) proposed gingival pigmentation index:

Score 0	Absence of pigmentation
Score 1	Spots of brown to black color or pigments.
Score 2	Brown to black patches but not diffuse pigmentation
Score 3	Diffuse brown to black pigmentation, marginal, and attached

5.Peeran *et al.* (2014)

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 agentbusulfan 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 mucocutaneous 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.

6.Gulati *et al.* (2016)

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 gingiva
Score 2	Moderate to severe, solitary or diffuse, gingival melanin pigmentation involving anterior gingiva with or without the involvement of posterior gingiva
Score 3	Gingival melanin pigmentation only in posterior gingiva
Score 4	Tobacco-associated pigmentation
Score 5	Exogenous pigments
Score 6	Endogenous pigments
Score 7	Drug-associated gingival pigmentation
Score 8	Systemic causes
Score 9	Pigmented benign lesions
Score 10	Pigmented malignant lesions

Gulati *et al.* elaborated that 0-3 was the range available to record the gingival color and its variation within physiological limits. A depigmentation procedure was indicated when the patient scored 1-2 in the index and had up to class 2 of Liebart and Deruelle Smile line classification.

NEW PROPOSED INDEX

While the afore-mentioned authors have used varied techniques for classification and treatment of gingival hyperpigmentation, there is not enough homogeneity in terms of classifying lesions based on their extent – generalized or localised, anterior or posterior, solitary or full arch.

Designation	*Region Affected*
UA	**U**pper **A**nteriors
UP	**U**pper **P**osteriors
U	**U**pper **A**rch
LA	**L**ower **A**nteriors
LP	**L**ower **P**osteriors
L	**L**ower **A**rch
(l)	Left
(r)	Right

Grade	*Etiology of Hyperpigmentation*
1	Physiologic
2	Tobacco Associated
3	Exogenous Pigmentation
4	Endogenous Pigmentation
5	Drug Induced
6	Systemic Diseases
7	Benign Lesions
8	Malignant Lesions

Also, etiology of the resultant hyperpigmentation was not taken into consideration in earlier classifications. Although recent classifications have taken these drawbacks into account, they failed to address the presence of two or more etiologic factors in different regions of the oral cavity in the same individual. This novel classification aims to overcome the shortcomings of these conventional classifications and indices proposed since 1964 till date. The new proposed index takes into consideration the region affected and the etiology for the hyperpigmentation (Table 1 and Table 2). The alphabetic representation (Designation) denotes the region affected, in case of the unilaterally affected posterior regions the side is denoted by (l) for left side and (r) for right side respectively and lastly the numerical value (Grade) denotes the etiology of the clinical presentation in the form of hyperpigmentation.

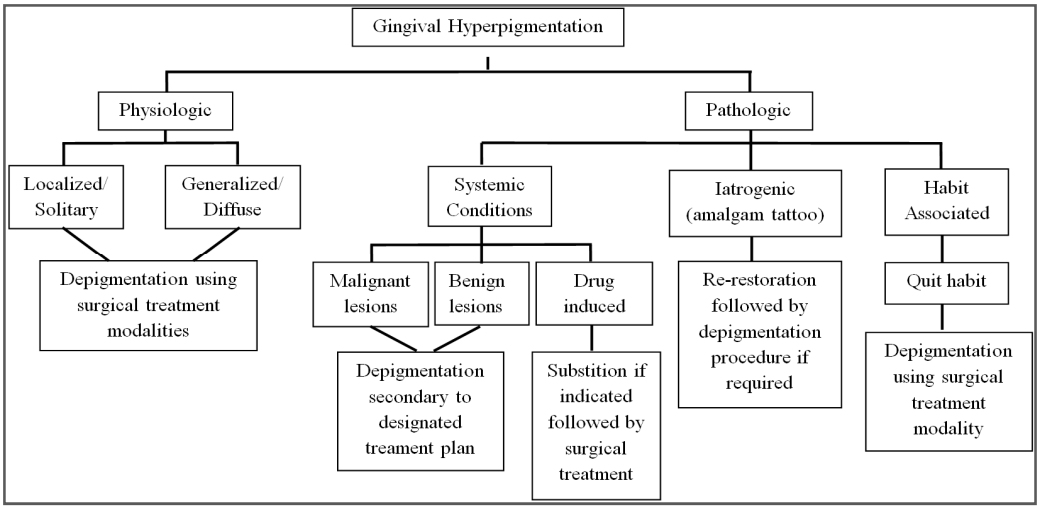
For easier understanding examples have been explained following the tables.

Examples:

- If the patient presents with tobacco associated pigmentation of the maxillary anterior gingiva, it will be classified as UA-2.
- If the patient presents with physiologic melanin pigmentation in the maxillary and mandibular anterior gingiva, it will be classified as UA-LA-1.
- If the patient presents with physiologic hyperpigmentation in the maxillary anterior region and exogenous pigmentation in mandibular posterior region it is denoted by UA-1; LP-3
- If the patient presents with tobacco associated pigmentation of the mandibular right posterior gingiva, it will be classified as LP(r)-2.

Advantages and Clinical Indications of this novel classification system and index for the gingival pigmentations include:

- ✓ Ease of application



Decision making tree for treatment of gingival hyper pigmentation

- ✓ Easy to remember
- ✓ Easy to communicate
- ✓ Due consideration given to the different etiologies and their clinical manifestations in different areas of the oral cavity
- ✓ Due consideration given to the region affected

Treatment: The various treatment modalities available are (Gulati *et al.,* 2016; Farid *et al.,* 2017):

1. Non surgical methods: Use of ascorbic acid
2. Surgical methods:
   a. Gingivectomy
   b. De-epithelialization by bur abrasion, laser, electrocautery
   c. Free gingival autografts
   d. Acellular dermal matrix grafts

Roshna *et al.* (2005) classified treatment modalities based on whether the pigment layer was removed or masked as follows:

I. Methods aimed at removing the pigment layer

a. Surgical methods of depigmentation

- Scalpel surgical technique.
- Cryosurgery.
- Electro surgery.
- Lasers: Neodymium: Aluminium-Yttrium-Garnet (Nd:YAG) lasers, Erbium:YAG (Er:YAG) lasers, Carbon dioxide (CO2) lasers.

b. Chemical methods of depigmentation using caustic chemicals: This method is not used nowadays.

II. Methods aimed at making the pigmented gingival with grafts from the less pigmented areas.

a. Free gingival grafts
b. Acellular dermal matrix allografts.

Taking these treatment modalities into account, a decision making tree has been charted above

Conclusion

Oral screening by the dentist should mandate due importance to the gingival pigmentation as it provides a window to diagnosis of various systemic conditions, benign or malignant lesions within or outside the oral cavity and underlying habit history such as tobacco consumption which may affect overall treatment plan and prognosis of the planned dental procedure. The current review aims at providing a classification index which is comprehensive and easy to communicate among clinicians. A wholesome approach towards characterizing gingival hyperpigmentation along with its intended treatment plan can help to provide a long term optimal esthetic result.

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