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# GINGIVAL PIGMENTATION

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## ABSTRACT

Cosmetic expectations have increased with time and current trends in gingival and smile designing. gingival pigmentation especially on the labial aspect of anterior teeth has become an important concept of general aesthetics. Pigmentation is both the normal and abnormal discoloration of oral mucous membrane. It has got multi factorial etiology. Melanin hyper pigmentation is usually does not present a medical problem, but patients complain of dark gums as unaesthetic. The etiology, differential diagnosis, clinical, histopathological and treatment of pigmentation are discussed and current literature is reviewed.

**KEYWORDS:** Gingival Pigmentation, Melanin, Treatment.

## INTRODUCTION

Any feeling of pleasure, happiness, laughter or simply a greeting leads to a smile resulting in the exposure of teeth and gingiva. A smile is not only a method of communication but also a method of socialization. Harmony of smile is based on not only the color of teeth but also depends on the colour of gingival tissues. Dentists may see patients with concern regarding the colour variations of their gingiva. It could be an aesthetic issue for some patients, especially when it is located in the anterior labial gingiva, is combined with a high smile line and is not uniform in appearance.

### Oral Pigmentation

Oral pigmentation is a discolouration of the oral mucosa or gingiva associated with several exogenous and endogenous factors.[1-3] This type of pigmentation is mostly located in the anterior labial gingiva, affecting females more than males.[1,5] (KAUZMAN ET AL 2004). It

have various etiology, including drugs, heavy metals, genetics, endocrine disturbance, and inflammation. The colour of the oral melanin pigmentation may vary from light to dark brown or black, depending on the amount and distribution. Melanin pigmentation is caused by melanin granules in gingival tissue, which are produced in melanosomes of melanocytes.Melanocytes are primarily located in the basal and suprabasal cell layers of the epithelium.[1,3,7] ALEX A FARNOOSH(1990). Also, smoking may stimulate melanin production and cause melanin pigmentation. The intensity of the pigmentation is related to the duration of smoking and number of cigarettes consume. HEDIN ET AL (1991).[3,5]

**Etiology**

**Dum met and Coin (1946)**

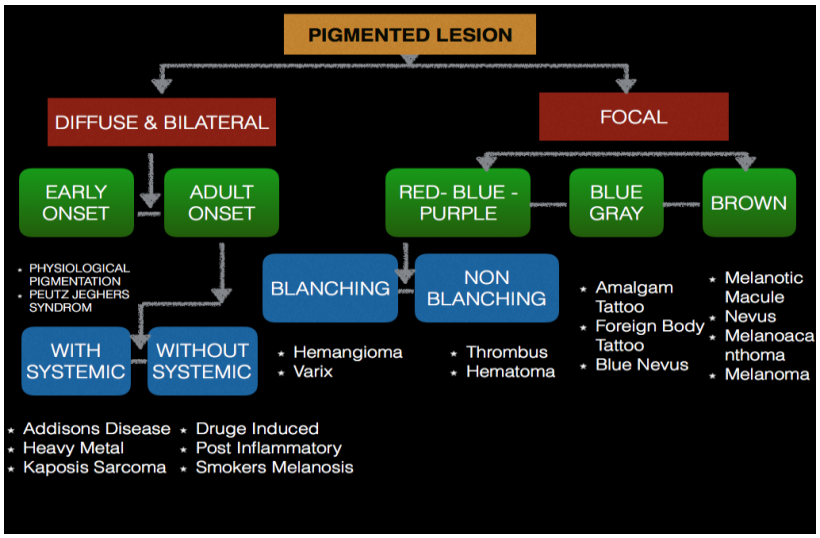
The colour of healthy gingiva is variable ranging from a pale pink to a deep bluish purple hue. Pigmentation is due to: the intensity of melanogenesis, depth of epithelial cornification, arrangement of gingival vascularity. More over colour variation may not be uniform and may exists as unilateral, bilateral, mottled, macular or blotched and may involve gingival papillae alone or extend throughout the gingiva on to other soft tissues.

**Primery Pigments**

- Melanin,
- Melanoid,
- Oxyhemoglobin,
- Reduced hemoglobin,
- Carotene.
- Bilirubin and iron.

Alex A Farnoosh in 1990 said that melanin deposits mainly in basal and suprabasal cell layers of epithelium.[9] The degree of pigmentation is attributed to melanoblastic activity and density of melanophores in gingiva.[8] The gingiva is the most commonly affected intraoral tissue, which is responsible for an unpleasant appearance. Melanin pigmentation often occurs in the gingiva as a result of an abnormal deposition of melanin. Brown or dark pigmentation and discoloration of gingival tissue can be caused by a variety of local and systemic factors.

**Classification**




Endogenous pigmentation and exogenous pigmentation.

**Classification: (Peeran et al 2014)**

CLASS 0: coral pink colored gingiva, no pigmentation.

CLASS 1: mild gingival pigmentation involving anterior gingiva, with or without posterior gingiva.

CLASS 2: moderate to severe pigmentation involving anterior gingiva, with or without posterior gingiva.

CLASS 3: gingival pigmentation only in posterior gingiva.

CLASS 4: tobacco associated pigmentation (smokeres melanosis, chewing tobacco).

CLASS 5: gingival pigmentation due to endogenous pigments (amalgam tattoo, betel nut, drink, food colour, silver, topical medication, idiopathic etc.

CLASS 6: gingival pigmentation due to exogenous pigment (bilirubin, blood breakdown product, ecchymosis, patchiea).

CLASS 7: drug induced gingival pigmentation (antimalarial drug, oral contraceptive).

CLASS 8: gingival pigmentation associated with other causes (addisons disease, albrights syndrome, HIV patients, lichen planus, peutz-jeghers syndrome,).

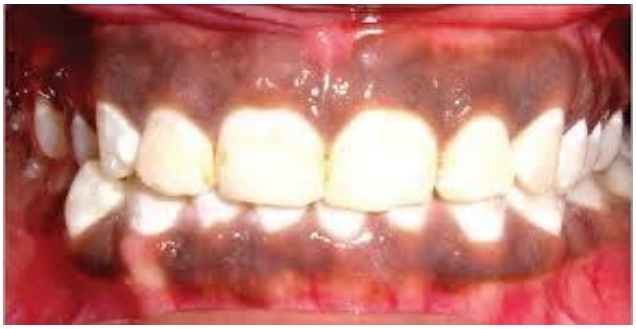
CLASS 9: pigmentation due to bening lesion (hemangioma, melanocytic nevus).

CLASS 10: pigmentation due to malignant lesion (kaposis sarcoma, malignant malanoma).

**Pathological Pigmentation**

Hyperpigmentation

Hypopigmentation



**Hyperpigmentation**

Smoking Assoc Melanosis

Melanoma

Addison's Disease

Peutz - Jegher Syndrom[5]

Albrights Syndrome

Nevus

Ephelis

Lentigo

Hiv Induced Pigmentation

**Hyperpigmentation**

Vertigo

Leukoderma

Albinism

**Distribution and Index for Pigmentation**

DUMMETT – 1946

Gingiva - 60%, Hard palate – 61%, Mucous membrane – 22%, Tongue – 15%.

In gingiva, number of melanophores in the epithelium and the subepithelial connective tissue gradually decreases starting from the free gingival groove area towards the gingival crest in the free gingiva & towards the muco gingival junction in attached gingiva. In addition, the

total number of melanophores in attached gingiva (3230) approximately is 16 times greater than in free gingiva (198).

Dummet Proposed the Dummet Oral Pigmentation Index (Dopi) Assessment): 1964.
Score 0: Pink tissue (No clinical pigmentation).
Score 1: Mild light brown colour (Mild clinical pigmentation).
Score 2: Medium brown or blue black tissue (Heavy clinical pigmentation).
Score 3: Deep brown or blue black tissue (Heavy clinical pigmentation).

Melanin Index: [HEDIN 1997]
No pigmentation.
One or two solitary unit(s) of pigmentation in papillary gingiva without the formation of a continuous ribbon between solitary units.
More than three units of pigmentation in papillary gingiva without the formation of a continuous ribbon.
One or more short continuous ribbons of pigmentation.
One continuous ribbon including the entire area between canines.

Takashi et al 2005.
Score 0: No pigmentation
Score 1: Solitary unit(s) of pigmentation in papillary gingiva without extension between neighboring solitary units
Score 2: Formation of continuous ribbon extending from neighboring solitary units.

Gingival Pigmentation Index: Bradley and Grace
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

Melanin is a pigment produced by melanocytes that reside in the basal layer(stratum basale) of the epidermis. It is stored in vesicles called melanosomes and is transferred to adjacent epithelial cells via dendritic processes. Melanocytes are cells capable of synthesizing tyrosinase, which, when incorporated within specialized organelles, the melanosomes, initiates events leading to the synthesis and deposition of melanin.[8]

According to the Degree of Maturation, Melanosomes are Classified in 4 Stages. Nordlund ET AL 1998

Type I melanosomes have intraluminal vesicles and resemble multivesicular bodies.

Type II is characterized by an elongated, elliptical shape, with intralumenal fine fibrils giving a striated appearance

Type III exhibits pigment deposition along the fibrils,

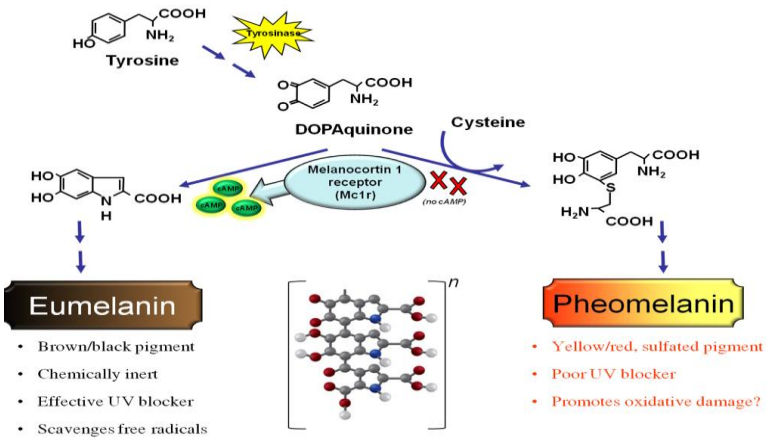
Type IV has dense pigmentation filling the organelle and obscuring the fibrillar structure.

**Melanocytes**

Melanin is a pigment produced by melanocytes that reside in the basal layer (stratum basale) of the epidermis. It is stored in vesicles called melanosomes and is transferred to adjacent epithelial cells via dendritic processes. Melanocytes are cells capable of synthesizing tyrosinase, which, when incorporated within specialized organelles, the melanosomes, initiates events leading to the synthesis and deposition of melanin. Eumelanin Pheomelanin Property Eumelanogenesis Pheomelanogenesis Melanosome Shape Ellipsoidal Spherical Structure Lamellae or filaments Micro vesicles Or Micro granules Melanin Color Dark brown to black Yellow to reddish brown Solubility Insoluble in acid and Soluble in alkali alkali Element Nitrogen 6-9% 8-10% Sulfur 0-1% 9-12% Structure Dihydroxyindole Benzothiazine. 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.[21] While its function remains unknown, its absence is associated with Parkinson's disease. Simon JD 2008, Nicolaus 2003, Mandel 2005

**Function of melanocytes**

Synthesis of melanin pigment granules (melanosomes) and transfer to surrounding keratinocytes. Protective function: Ultraviolet (UV) rays of the sun cause damage of the skin called ultraviolet ray injury. Melanin and the thick stratum corneum together prevents such injury exposure to UV rays stimulate more melanin production and melanin absorbs the light of all wave lengths. Absence of enzyme melanocyte tryosinase leads to failure of melanin formation from tyrosine.




**Melanin Synthesis**

**Physiologic Pigmentation**

Etiology

Normal melanocyte activity

**Clinical Presentation**

Seen in all ages

Symmetric distribution over many sites, gingiva most commonly

Surface architecture, texture unchanged Diagnosis

History • Distribution

**Differential Diagnosis**

Mucosal melanotic macule

Smoking-associated melanosis

Superficial malignant melanoma

**Treatment**

None Prognosis

Excellent.

**Pathological pigmentation**

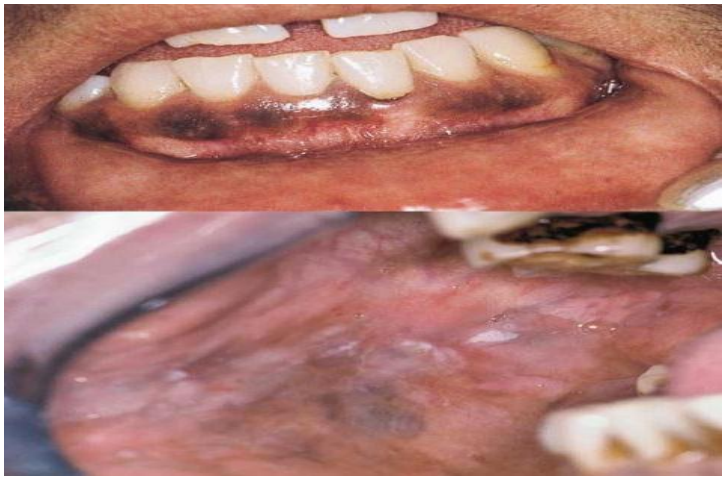
**Smokers Melanosis**

Etiology

Melanin pigmentation of oral mucosa in heavy smokers

May occur in up to 1 of 5 smokers.

Melanocytes stimulated by a component in tobacco smoke.



**Clinical Presentation**

Brownish discoloration of alveolar and attached labial gingiva, buccal mucosa. Pigmentation is diffuse and uniformly distributed; symmetric gingival pigmentation occurs most often. pigmented areas are brown flat and irregular.

**Microscopic Findings**

Increased melanin in basal cell layer

Increased melanin production by normal numbers of melanocytes •

Melanin incontinence

**Diagnosis**

History of chronic, heavy smoking

Biopsy

**Differential Diagnosis**

Physiologic pigmentation

Addison's disease

Medication-related pigmentation (drug- induced pigmentation by chloroquine, chlorpromazine, quinidine, or zidovudine)

Malignant melanoma

**Treatment**

Reversible, if smoking is discontinued.

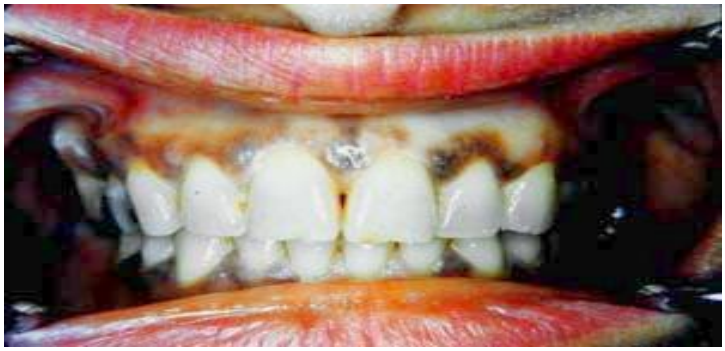
**Prognosis**

Good, with smoking cessation.

**Addisons Disease**

**Etiology**

Results from atrophy of the adrenal cortices and failure of secretion of cortisol and aldosterone. Oral manifestations due to secondary melanocyte stimulation by increased levels of adrenocorticotropic hormone (ACTH).



**Clinical Presentation**

Brown macular pigmentation of local or diffuse quality.

Pigmentation usually seen in association with cutaneous bronzing, weakness, weight loss, salt craving, nausea, vomiting, hypotension.

**Diagnosis**

Confirmation of hypoadrenocorticism by plasma ACTH levels

Biopsy of mucosa shows melanosis Differential Diagnosis

Smoker's melanosis • Physiologic/ethnic pigmentation

Heavy metal deposition/argyrosis

Medication-related pigmentation

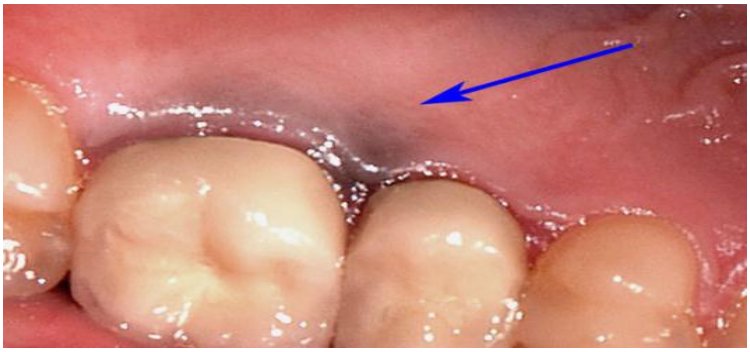
Peutz-Jeghers syndrome

**Treatment**

Management of underlying adrenal insufficiency by corticosteroid replacement therapy.

**Prognosis**

Good with replacement therapy



**Amalgum Tattoo**

**Etiology**

Implantation or passive/frictional transfer of dental silver amalgam into mucosa

**Clinical Presentation**

Gray to black focal macules, usually well defined, but may be diffuse with no associated signs of inflammation. Typically in attached gingiva, alveolar mucosa, buccal mucosa. Occasionally may be visible radiographically.

**Diagnosis:** Radiographs may be useful (intraoral filmplacement) Biopsy may be necessary if clinical diagnosis is in doubt or to rule out lesions of melanocytic origin

**Differential Diagnosis**

Vascular malformation •

Mucosal nevus

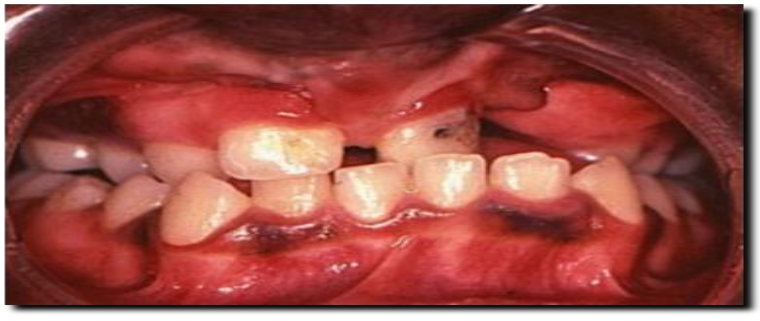
Melanoma

Mucosal melanotic macule

Melanoacanthoma

**Treatment:** Biopsy or observation only

**Prognosis:** Little clinical significance if untreated.



**Melanoacanthoma**

**Etiology**

A reactive and reversible alteration of oral mucosal melanocytes and keratinocytes

Usually associated with local trauma

**Clinical Presentation**

Unilateral dark plaque; rarely multiple, bilateral. Most often noted among Blacks and other non-Caucasians. Occurs more often in women than men by a ratio of 3:1. History of trauma and local irritation Forms rapidly, most often on buccal/labial mucosa. Asymptomatic melanotic pigmentation.

**Diagnosis**

Clinical history of rapid onset

Histologic evaluation Scattered dendritic melanocytes within spongiotic and acanthotic epithelium Increased number of melanocytes along basal layer as single units

**Differential Diagnosis**

Melanoma

Drug-induced pigmentation

Smoker's melanosis

Mucosal melanotic macule

Mucosal nevus

Amalgam tattoo

**Treatment**

None after establishing the diagnosis

Often resolves spontaneously.

**Prognosis**

Excellent

**Mucosal Malignant Melanoma**

Etiology

Unknown

Cutaneous malignant melanoma with relation to sun exposure or familial-dysplastic melanocytic lesions.

**Clinical Presentation**

Rare in oral cavity (< 1% of all melanomas). Most cases occur in those older than 50 years of age.Usually arises on maxillary gingiva and hard palate. May exhibit early in situ phase: a macular, pigmented patch with irregular borders. Progression to deeply pigmented, nodular quality with ulceration. May arise de novo as a pigmented or amelanotic nodule. Rarely may be metastatic to the oral cavity as a nodular, usually pigmented mass.

**Microscopic Findings**

Early stage: atypical melanocytes at epithelialconnective tissue interface, occasionally with intraepithelial spread Later infiltration into lamina propria and muscle. Strict correlation to cutaneous malignant melanoma is not well established, although, as in skin, a similar horizontal or in situ growth phase often precedes the vertical invasive phase. Amelanotic forms may require use of immunohistochemical identification: S-100 protein, HMB-45, Melan-A expression.

**Diagnosis**

Biopsy

**Differential Diagnosis**

Mucosal nevus

Extrinsic pigmentation

Melanoacanthoma

Kaposi’s sarcoma

Vascular malformation

Amalgam tattoo

Mucosal melanotic macule

**Treatment**

Surgical excision. Marginal parameters related to depth of invasion and presence of lateral growth, Wide surgical margins; resection for large, deeper lesions

**Prognosis**

poor

**Mucosal Melanotic Macule**

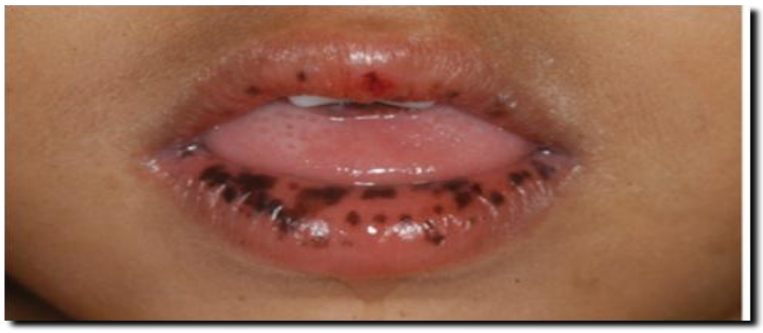
Etiology

Most idiopathic, some postinflammatory, some drug-induced

Multiple lesions suggest syndrome association, as follows:

Peutz-Jeghers syndrome

Carney's syndrome "LEOPARD syndrome"



**Clinical Presentation**

Most in adulthood (fourth decade and beyond). Most are solitary and well circumscribed. Lower lip vermilion border most common site, mostly in young women (labial melanotic macule). Buccal mucosa, palate, and attached gingiva also involved (mucosal melanotic macule). Usually brown, uniformly pigmented, round to ovoid shape with slightly irregular border. Usually < 1 mm in diameter

**Microscopic Findings**

Normal melanocyte density and morphology

Increased melanin in basal cells and subjacent macrophages (mucosal melanotic macule)

Increased melanin in basal cells with elongated rete pegs

Diagnosis

Biopsy

Differential Diagnosis

Melanoacanthoma

Mucosal melanotic macule

Congenital syndromes (Carney's, Peutz-Jeghers, LEOPARD)

**Treatment**

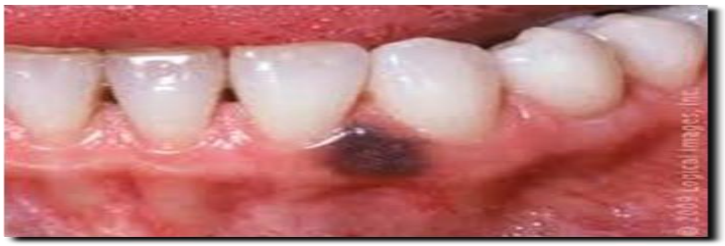
Observation

Biopsy for esthetics

If increase in size or development of atypical signs occurs, macule should be removed to rule out malignant melanoma, particularly if on palate or alveolar mucosa.

**Prognosis**

Excellent



**Mucosal Pigmentation Excintric**

**(DRUG OR METAL INDUCED)**

Etiology.

**Occupational Exposure—Metals Vapors (Lead, Mercury)**

Therapeutic—metal salt deposits (bismuth, platinum, silver); also nonmetal agents, such as chloroquine, minocycline, zidovudine, chlorpromazine.

**Clinical Presentation**

Focal to diffuse areas of pigmentary change.

If heavy metals are the cause, a typical gray to black color is seen along the gingival margin or areas of inflammation.Palatal changes characteristic with antimalarial drugs and minocycline. Most medications cause color alteration of buccal-labial mucosa and attached gingiva. Darkened alveolar bone with minocycline therapy

**Diagnosis**

History of exposure to, or ingestion of, heavy metals or drugs

Differentiation from melanocyte-related pigmentation by biopsy if necessary

**Differential Diagnosis**

When localized: amalgam tattoo, mucosal melanotic macule, melanoacanthoma, mucosal nevus, ephelides, Kaposi's sarcoma, purpura, malignant melanoma, ecchymosis

When generalized: ethnic pigmentation, Addison's disease

If asymmetric, in situ melanoma must be ruled out by biopsy.

**Treatment**

Investigation of cause and elimination if possible

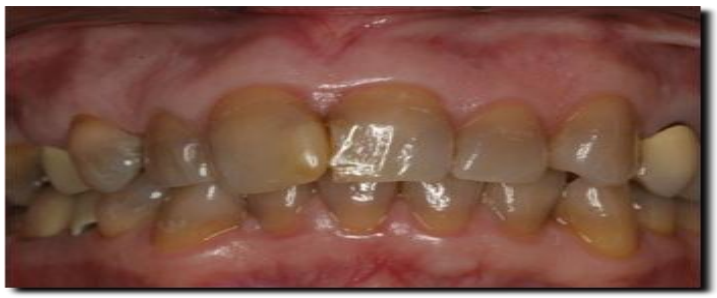
**Prognosis**

Excellent

**Tetracycline**

**Etiology**

Prolonged ingestion of tetracycline during tooth development. Less commonly, tetracycline ingestion causes staining after tooth formation is complete: reparative (secondary) dentin and cementum may be stained.



**Clinical Presentation**

Yellowish to gray (oxidized tetracycline) color of enamel and dentin. May be generalized or horizontally banded depending on duration of tetracycline exposure Alveolar bone may also be stained bluish red (particularly with minocyline use, 10% after 1 year and 20% after 4 years of therapy).

**Diagnosis**

Clinical appearance and history

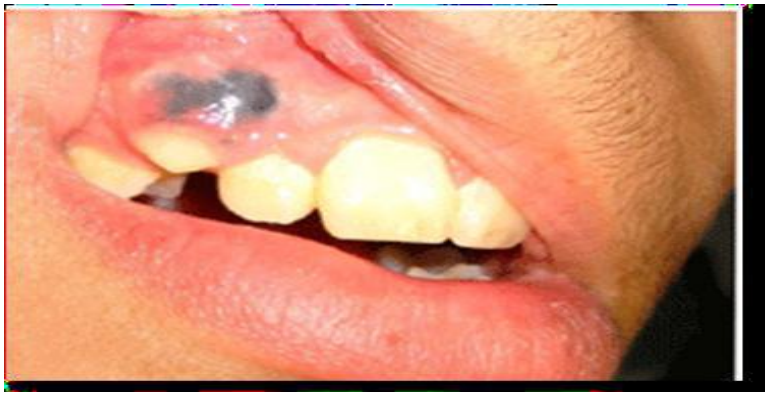
Fluorescence of teeth may be noted with ultraviolet illumination.

**Treatment**

Restorative/cosmetic dental techniques

**Prognosis**

Good



**NEVUS**

Etiology

Unknown

Lesion of melanocytic origin within mucosa and skin

**Clinical Presentation**

Usually elevated, symmetric papule<1cm,solitary brown or blue.Pigmentation usually uniformly distributed, Common on skin; unusual intraorally, Palate and gingiva most often involved

**Microscopic Findings**

Most are intramucosal

Blue nevi are deeply situated and are composed of spindled nevus cells.

Other variants are rare; junctional and compound nevi

Nevus cells are oval/round and are found in unencapsulated nests

Melanin production is variable.

**Diagnosis**

Clinical features

Biopsy

**Differential Diagnosis**

Melanoma

Varix

Amalgam tattoo/foreign body

Mucosal melanotic macule

Kaposi's sarcoma

Ecchymosis •

Melanoacanthoma

**Treatment**

Excision of all pigmented oral lesions to rule out malignant melanoma is advised.

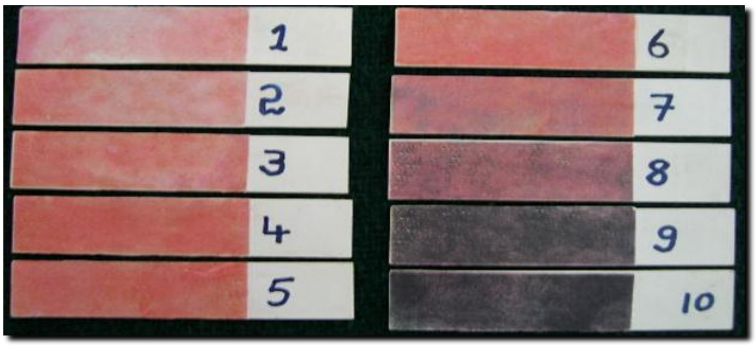
Malignant transformation of oral nevi probably does not occur.

**Prognosis**

excellent

**Treatment**

The first and foremost indication for depigmentation is patient demand for improved esthetics.




**Weinman Scale**

**Methods for Gingival Depigmentation**

1. Gingival abrasion technique
2. Scalpel surgical technique.

3. Cryosurgery
4. Electro surgery.
5. Lasers Neodymium;Aluminum-Yitrium-Garnet (Nd- YAG) lasers. Erbium-YAG lasers. Carbon-di-oxide CO2 laser
6. Chemical method
7. Free gingival grafts
8. Acellular dermal matrix allograft

**Gingival Abrasion**

In this technique a medium grit football shaped diamond bur is used at high speeds to denude the epithelium. The procedure requires 45 min to 1 h for completion. Healing by re epithelialisation occurs within 7-10 days. It is relatively non invasive, a cost effective technique and it doesn't require specific instrument. Procedural discomfort, placement of periodontal pack, duration of procedure, recurrence of pigmentation and technique sensitivity are few of the drawbacks

**Scalpel Technic**

This procedure essentially involves surgical removal of gingival epithelium along with a layer of the underlying connective tissue and allowing the denuded connective tissue to heal by secondary intention. Dummett and Bolden (1963) reported in a study that Scalpel surgery can cause unpleasant bleeding during and after the procedure. It is essential to cover the exposed lamina propria with a periodontal pack for 7-10 days. Delicate scarring, exposure of the alveolar bone at areas where the gingiva is thin and repigmentation can be few of the disadvantages of the procedure.

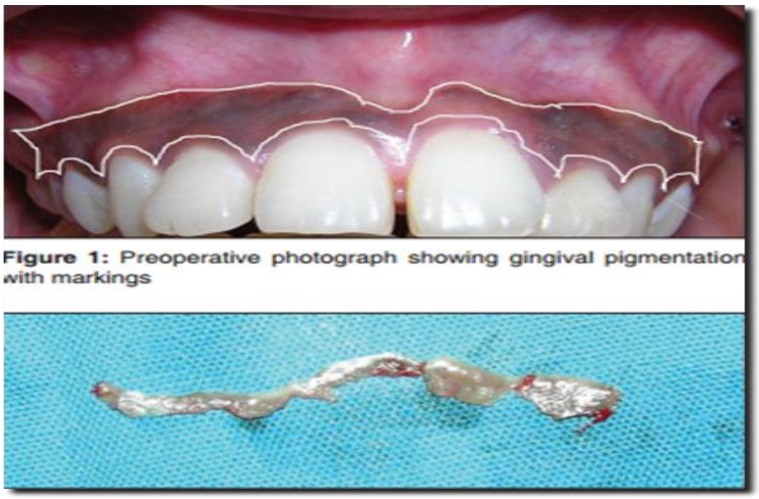
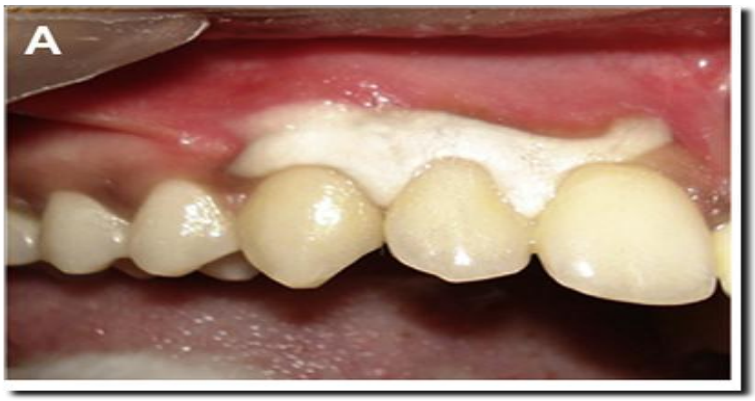


**Figure 1:** Preoperative photograph showing gingival pigmentation with markings

**Cryosurgery Method**

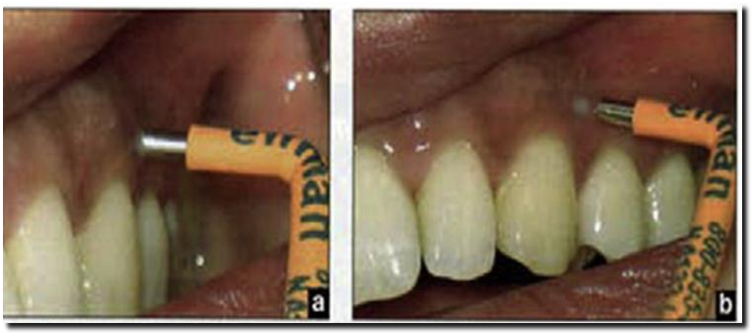
- Salt ice -20 C,
- CO2 slush -20 C,
- Fluorocarbons (Freons) -30 C,
- Nitrous oxide -75 C,
- CO2 snow - 79 C,
- Liquid nitrogen -20 C (swab)and - 196 C (spray),
- Tetrafluoroethane -20 C to- 40 C




**Technique**

Cryosurgical apparatus are used in 2 basic technique. Open system were, cryogen is directly sprayed on the tissues. Closed system using cryoprobe for application on the tissue. Open involves the direct application to superficial lesions of usually either carbon dioxide snow – 79 degree C or liquid nitrogen -196 degrees C applied on cotton pellets or as an open spray. Closed system offers a great degree of control, but instrumentation is more complex, the depth of freezing being in general less profound than with open system.[21]

**Timed Spot Freeze Technique**

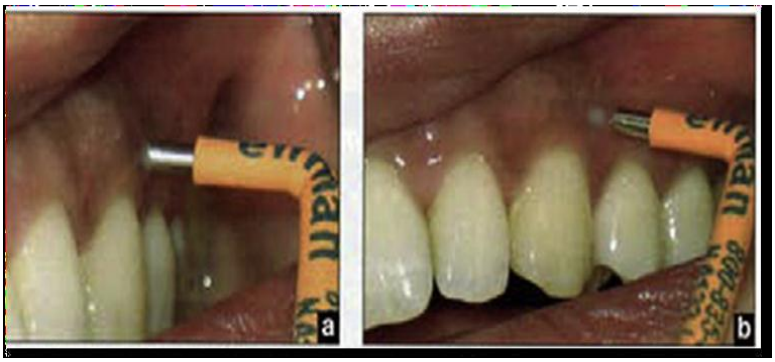


The timed spot freeze technique allows greater standardization of liquid nitrogen delivery. Use of this technique maximizes the ability to destroy a lesion with minimal morbidity. The freezing time is adjusted according to variables such as skin thickness, vascularity, tissue type, and lesion characteristics. Timed spot freezing is performed with a small spray gun that typically holds 300 to 500 mL of liquid nitrogen. Nozzle sizes range from A through F, with F representing the smallest aperture

**Cryoprobesz**

While the open spray technique can be used for the most easily accessible lesions, a cryoprobe attached to the liquid nitrogen spray gun can provide added versatility, depending on the site and type of the lesion. Various sizes and types of cryoprobes are available. The cryoprobe is applied directly to the lesions. A gel interface medium often is used between the probe and the skin surface. The tissue freezes solid taking on the appearance of a "snowball" Thawing occurs in 15-20 s with progression from the periphery to the center of the snow ball.

At 12 hrs an elevated white fluid filled blister appears which increases in size slightly up to 24 h. The roof of the blister area consists of a white membrane, outlined by an indistinct red halo. At 48 h the blister ruptures, exposing a smooth underlying surface. Repair and reepithelization takes place deep to the slough, which separates off after leaving a clean surface.




**Laser**

Light Amplification By Stimulation Of Emission Radiation

Lasers are of soft tissue and hared tissue laser.

- Gas lasers
- chemical lasers
- metal vapor lasers

- solid state lasers
- Co2 laser[21]
- semiconductor diode laser
- Nd:YAG Laser[15]
- argon laser
- ErYg Laser has been used in gingival depigmentation.[16]

Dental laser has affinity for different tissue components. Lasers have wavelength characteristics that specially target soft tissues. It has affinity for hemoglobin and melanin. In the case of laser depigmentation, the ability of melanin- containing Melanocytes to absorb the laser light is dependent on the wavelength of the laser and its ability to penetrate tissue. Melanin has an absorption spectrum range between 351 and 1064 nm.[19,20]

**Electrosurgery**

Electro surgery or radio surgery is currently used to identify surgical techniques performed on soft tissue using controlled high-frequency electric currents in the range of 1.5 to 7.5 million cycles per second or megahertz.

**Types of Electrode**

Needle electrode for incising or excising

Loop electrodes for planing tissue

Heavy, bulkier electrodes for coagulation procedures.

**Principles of Electrosurgery**

Electrosurgery- alternating current.

**Techniques**

Electrosection

Electrocoagulation

Electrofulguration

Electrodessication

Electrosection also referred to as electrotomy or acusection, is used for incisions, excisions, and tissue planing. Incisions and excisions are performed with single-wire active electrodes that can be bent or adapted to accomplish any type of cutting procedure.

Electrocoagulation provides wide range of coagulation or hemorrhage control obtained by using the electrocoagulation current. Electrocoagulation can prevent bleeding or hemorrhage at the initial entry into soft tissue, but it cannot stop bleeding after blood is present. All forms of bleeding should be stopped first. After bleeding has momentarily stopped, final sealing of the capillaries can be done with the application of electrocoagulation current.

Basic rule of electrosurgery is to keep the tip moving. Prolonged or repeated application of current to tissue induces heat accumulation and tissue destruction. Interrupted applications eliminates heat build up. It is a means of modifying soft tissue with little discomfort or hemorrhage for the patient.

**Chemical Method**

- 5% paraformaldehyde
- Potassium hydroxide
- Phenol…etc.

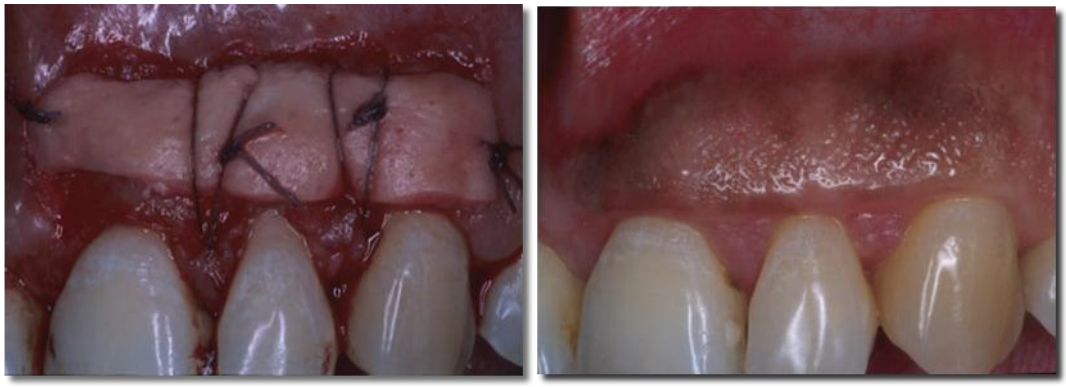
Hirschfeld I and Hirschfeld L in 1951, used a mixture of phenol (90%) and alcohol (95%) to burn out pigmented gingiva in 12 patients with gingival pigmentation. The growth of new gingiva was prolonged, and repigmentation soon developed in three patients; the rest of the 9 subjects met with the same result a short while later. These substances caused tissue necrosis in addition to pain, which was the result of burning both during and especially after the treatment. The treatment was not acceptable to the clinician or the patient.

**Graft**

**Free Gingival Graft[10]**

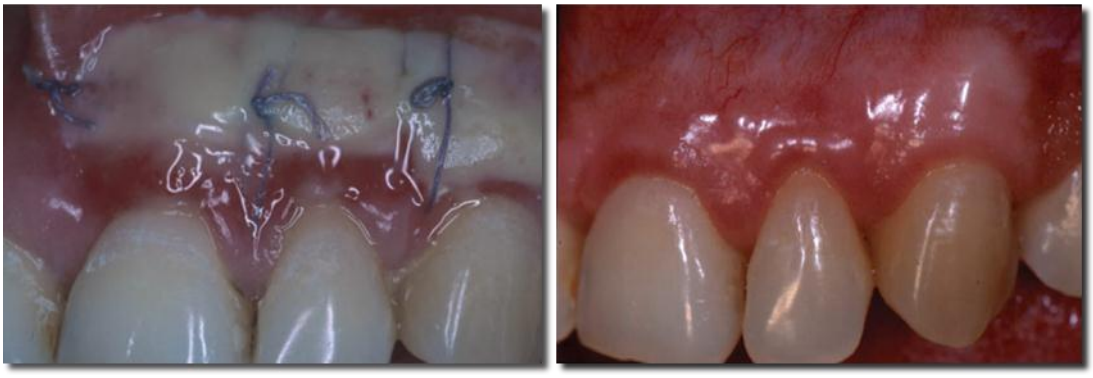
The potential of autogenous epithelialized gingival grafts has been established for the management of physiologic gingival pigmentation. A free gingival graft, as described by Sullivan and Atkins(1968) was harvested from the palate to eliminate the high frenum attachment and to augment the width of the ketatinized gingiva. Although not the primary objective, a local elimination of the melanin pigmentation could be observed one year post-operatively. Additionally, Tamizi and Taheri (1996), and Fowler et al.(2000), also reported the use of free gingival grafts for the elimination of gingival melanin pigmentation. Tamizi and Taheri treated 10 patients with severe melanin pigmentation in at least two areas. Removal of melanin pigmentation was successful in all patients treated, 4.5 years post-operatively, with the exception of one failure in a site treated with the free gingival graft.[8]

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. The pigmentation of the graft increased with time, resulting in gingival pigmentation consistent with the adjacent areas at 6 and 13 months post-operatively.



**Acellular dermal matrix (ADM)**

Pontes et al 2006….Fifteen patients presenting bilateral gingival melanin pigmentation were selected for the study. The sites of the experimental group had a partial thickness flap raised, excised, followed by adaptation and suture of the ADM. On the opposite side (ie, sites from the control group), the oral epithelium was removed with a diamond bur. The healing process was evaluated at 1 and 2 weeks, and 1, 3, 6, 9, and 12 months postoperatively. After 12 months, minimal repigmentation (mean 3.14 ± 7.45%) was noted in 8 of 15 sites from the ADM group, while significant repigmentation (mean 55.84 ± 27.25%) was seen in 15 of 15 abrasion sites. According to the results, it can be concluded that ADM may be successfully used in the elimination or greater reduction of gingival melanic pigmentations, and is more efficient than abrasion.



**Repigmentation**

Oral repigmentation refers to the clinical reappearance of melanin pigment following a period of clinical depigmentation of oral mucosa as the result of chemical, thermal, surgical, pharmacological or idiopathic factors. The exact mechanism of skin repigmentation is unclear, but the 'migration theory' seems to be favored.[6] According to this theory active melanocytes from normal skin proliferate and migrate into the depigmented areas. Clinical repigmentation of depigmented areas may occur spontaneously also after subtotal gingivectomy, or exposure to ultraviolet light or dermabrasion. Information on the repigmentation of oral tissues after surgical procedure is extremely limited. Studies have shown gingival pigmentation to be retained for the first week when periodontal flaps were displaced or replaced. The melanocytes had transiently lost their ability to produce and discard pigmented material to the surrounding keratinocytes but because they were still participants of the epidermal melanin unit, they return to normal much faster than do melanocytes observed after a gingivectomy procedure.

**Perlmutter S. et al. (1986)[6]**

conducted a study on repigmentation of the gingiva following surgical injury. Two patients who had moderate to heavily pigmented gingiva were treated. Surgically treated areas in both patients remained de-pigmented over the first 2 years. After 32 months some pigmentation was found in one of the patients and with the exception of two limited sites. They found different degree of repigmentation after 7 years. The other subject revealed no repigmentation over an 8 years follow up period. The authors concluded that these observations agreed with previous reports that described gingival pigmentation as spontaneous andsuggested that further controlled experimental studies be undertaken to explore the biologic basic for repigmentation.[6] In 1993, Bergamaschi et al studied melanin repigmentation after gingivectomy. Five white patients with comparable gingival pigmentation underwent gingivectomy two reached baseline coloration 1.5 years post surgery, while three returned to baseline coloration by 3 years post surgery. The authors concluded that gingival resective procedures, if performed solely for cosmetic reasons, offer no permanent results.

**CONCLUSION**

Gingival melanin pigmentations can occur as a consequence of local, systemic, environmental or genetic factors. To ensure treatment success, the potential causative or aggravating agent of the pigmentation should be identified and eliminated, if possible, before

the surgical treatment. Various techniques are available with some advantages and some drawbacks. However, choice of the technique should be dependent on individual preference, clinical expertise and patient affordability. More data is required on comparative techniques to ensure the long-term predictability and success.

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