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University of Baghdad
College of Dentistry

Assessment Of Gingival Pigmentation (Etiology & Treatment Modalities)

A Project Submitted to
The College of Dentistry, University of Baghdad
Department of Periodontology
In Partial Fulfilment for the Bachelor of Dental Surgery

By

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CERTIFICATION OF THE SUPERVISOR

I certify that this project entitled "**Assessment Of Gingival pigmentation**" was prepared by **Asmaa Basil** and **Asmaa Abdulrahim** under my supervision at the College Of Dentistry/University of Baghdad in partial fulfilment of the graduation requirements for the Bachelor degree in dentistry.

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ABSTRACT

Background : Melanin, a brown pigment, is the most common cause of endogenous pigmentation of gingiva and is the most predominant pigmentation of mucosa. 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 Non esthetic condition. This problem is aggravated in patients with a "gummy smile" or excessive gingival display while smiling.

The Aim Of Study : The Aim OF Study Is To Assist The Gingiva Pigmentation And Knowledge The Causes And Treatment.

Chapter one:
Review of literature

CHAPTER ONE REVIEW OF LITERATURE

A beautiful smile surely enhances the individual's self-confidence. The harmony of smile is attributable to the shape, colour, and position of the teeth in conjunction with the gingival tissue **(Sedeh SA et al.,2014)** Gingival health and appearance are essential components for an attractive smile and removal of unsightly pigmented gingiva is the need for a pleasant and confident smile **(Grover HS et al.,2014)** Gingival colour is generally described as "coral pink". Gingival pigmentation is presented as a diffuse deep purplish discoloration or as irregularly shaped brown and light brown or black patches, striae or strands Melanin, carotene, reduced haemoglobin and oxy-haemoglobin are the prime pigments contributing to the normal colour of the gingiva, out of which melanin shows the maximum incidence rate **(Antony VV and Khan R.2013).** Excessive deposition of melanin located in the basal and supra-basal cell layers of the epithelium will result in gingival hyperpigmentation **(Dummett, 1979)**

Health and appearance of gingiva are important parts of a smile **(verma S et al.,2013)** the color of the gingiva is various among different individuals and it is thought to be associated with cutaneous pigmentation**(prabhuji M et al.,2011)**. It varies from light to dark brown or black.

Gingival hyperpigmentation can be defined as a darker gingival color beyond what is normally expected**(Kumar S et al.,2013).**

The gingiva is considered the most frequently pigmented tissue in the oral cavity **(Westerhof W,2006) .** Gingival pigmentation is a discoloration of the gingiva due to a variety of lesions and conditions associated with several endogenous and exogenous etiologic features **(Sangeevini H et al.,2012).** It may range from physiologic reasons (e.g. racial pigmentation) to manifestations of systemic illnesses

(e.g. Addison's disease) to malignant neoplasms (e.g. melanoma and Kaposi's sarcoma). It is essential to understand the cause of a mucosal pigmentation before planning the treatment of such lesion **(Slominski A et al.,2012)**

1.1 PHYSIOLOGY OF MELANIN PIGMENTATION :

The gingival color depends primarily upon: - The number and size of vasculature - Epithelial thickness - Degree of keratinization - Pigments within the gingival epithelium include:

1.1.1 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. The name “melanin” comes from the Greek word “melanos”, meaning “dark,” and the term was first applied by the Swedish chemist Berzelius in 1840 to call a dark pigment extracted from eye membranes **(Patil KP et al.,2015).** Melanin pigmentation appears as early as 3 h after birth in the oral tissues and in some cases is the only sign of pigmentation on the body.

melanosomes within the keratinocytes disintegrate releasing melanin ‘dust’ that protects the oral mucosa against microbial toxins and other microenvironmental stressors. Various stimuli can result in excessive deposition of melanin located in the basal and supra-basal cell layers of the epithelium will result in gingival hyperpigmentation, such as trauma, hormones ,radiation and medication**(Dummett CO and Barens G,1971).**

1.1.2 Melanocytes

Melanocytes constitute a heterogeneous group of cells. These unicellular dendritic cells reside in the basal cell layer of the epidermis and oral epithelium. Primitive melanocytes originate from neural crest of ectoderm. Melanocytes have a round nucleus with a double nucleus membrane and clear cytoplasm lacking desmosomes or attachment plates, but possess long dendritic processes **(Dummett CO and Barens G,1971)**.

Melanin provides protection from environmental stressors such as ultraviolet radiation and reactive oxygen species; and melanocytes function as stress-sensors having the capacity both to react to and to produce a variety of microenvironmental cytokines and growth factors, modulating immune, inflammatory and antibacterial responses.

The population of melanocytes of the oral epithelium appears to be more or less constant throughout life, despite the fact that some melanocytes are lost owing to the natural process of programmed cell death, and to mechanical, thermal or chemical injury. The mechanism by which the population of oral melanocytes is maintained in a steady state is unknown .

1.1.3 Melanosomes

Melanocytes synthesize melanin in organelles called melanosomes. There are four stages in melanosome development **(Cichorek M et al.,2013)**Stage I Premelanosomes: They are round, small vesicles with an amorphous matrix. Stage II Melanosomes: They have an organized, structured fibrillar matrix and tyrosinase is present but pigment synthesis has not been noted. Stage III: The beginning of melanin production takes place at this stage, where pigment is deposited on protein fibrils. Stage IV: At the last, pigment fills the whole melanosome. Fully melanized

melanosomes lose tyrosinase activity and are transported to surrounding keratinocytes by elements of the cytoskeletal system.

Major determinant of normal human skin color is the melanogenic activity within the melanocytes and the quantity and quality of melanin production, but not melanocyte density. The degree of clinical melanin pigmentation in human epidermis and in the epithelium of oral mucosa is related to the amount of melanin i.e. the maturation of melanosomes, the number of keratinocytes containing melanosomes and the distribution of melanin loaded keratinocytes throughout the epithelium **(Lerner AB and Fitzpatrick TB,1950) .**

1.2 ETIOLOGY OF PIGMENTATION

The causes of pigmentation mainly classified into **endogenous** and **exogenous**. 1.2.1 Endogenous pigmentation

1.2.1.1 Physiologic pigmentation or Racial pigmentation

Physiological pigmentation is common and results from an increase in the production of melanin pigment by the melanocytes **(Prabhuji M et al.,2013).** Darker skinned individuals are more commonly affected. The color of physiological pigmentation can range from light brown to almost black. Phy-siological pigmentation increases with age, and color intensity can be influenced by smoking, hormones and systemic medications **(Kumar S et al.,2013).** The attached gingiva is the most common location, but physiological pigmentation can be noted anywhere in the oral cavity, including the tips of the fungiform papillae on the dorsal tongue and the diagnosis of physiological pigmentation normally is made clinically and do not need any treatment **(Nagati R et aj.,2017)**.

1.2.1.2 Pathological pigmentation

1.2.1.2.1 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 freckle like macules about the hands, perioral skin, and intraorally to include the gingiva, buccal, and labial mucosa. Pigmented spots are particularly found on the lower lip and buccal mucosa but rarely on the upper lip, tongue, palate, and gingiva **(Kopacova M et al.,2009)**

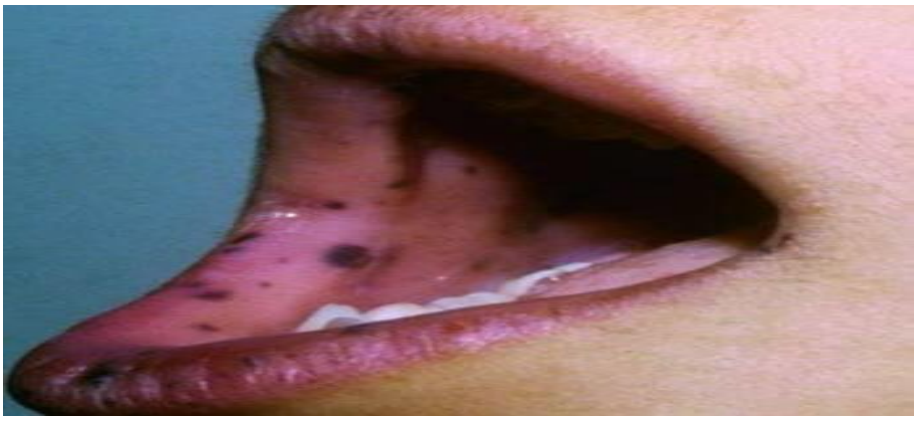

Figure 1 : Peutz-Jeghers syndrome

1.2.1.2.2 Addison's disease

Addison's disease, or primary hypoadrenalism, is due to progressive bilateral destruction of the adrenal cortex by autoimmune disease, infection or malignancy. The lack of adrenocortical hormones in the blood stimulates production of adrenocorticotropic hormone (ACTH) by the anterior pituitary gland. The increased production of ACTH induces melanocyte-stimulating hormone, which results in diffuse pigmentation of the skin and oral mucosa **(Kim HW,1988).** 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 **(Chuong R and Goldberg MH,1983).**

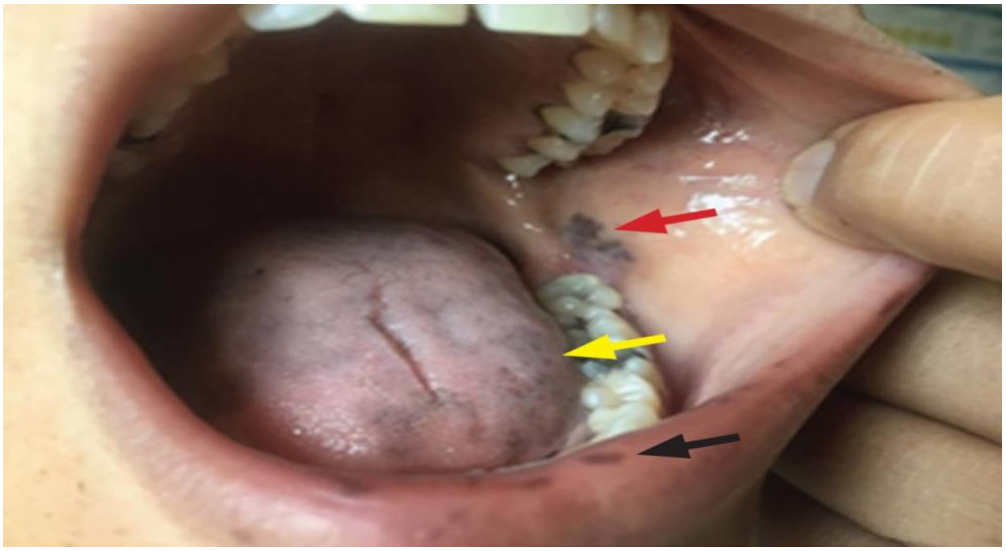

Figure 2: Addison disease

1.2.1.2.3 Kaposi's Sarcoma

Kaposi's sarcoma (KS) is a multifocal vascular malignancy seen predominantly in HIV-infected individuals. KS in the oral mucosa most commonly affects the hard palate, gingiva and tongue **(Mohanna S et al., 2007).** Early lesions appear as flat or slightly elevated brown to purple lesions that are often bilateral. Advanced lesions appear as dark red to purple plaques or nodules that may exhibit ulceration, bleeding and necrosis **(Lager I et al.,2003).**

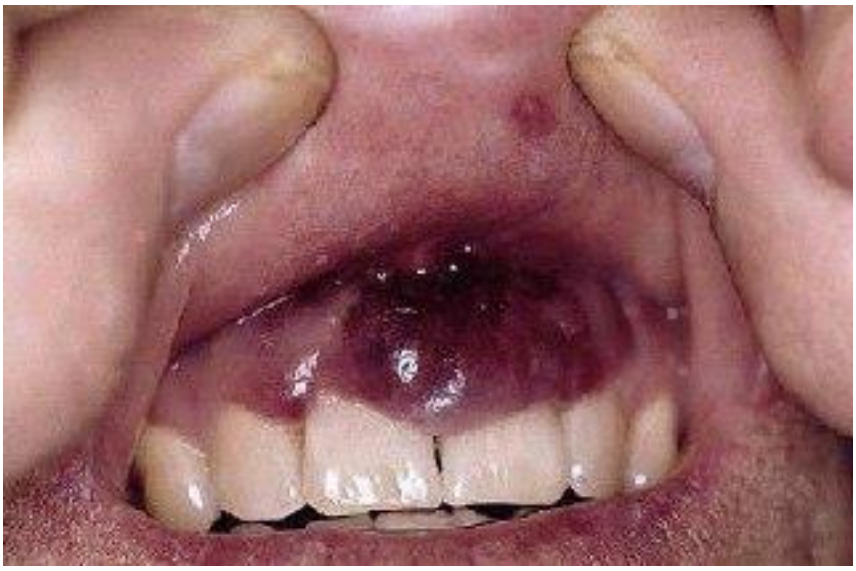

Figure 3: Kaposi sarcoma

1.2.1.2.4 Post inflammatory pigmentation

Oral post-inflammatory pigmentation (OPP) is a discoloration of the oral mucosa caused by an excess of melanin production and deposition within the basal layer of the epithelium and connective tissue of areas affected by chronic inflammation such as oral lichen planus (OLP) and other oral lichenoid lesions (OLLs), pemphigus, pemphigoid, periodontal disease, Steven-Johnson syndrome and graft versus host disease **(Anjum R et al.,2012).** Clinically OPP appears as a localized or diffuse, black to brown pigmentation. OPP may persist for many years even though the disappearing of the pigmentation after the resolution of the inflammatory state has been reported **(Mergoni G et al.,2011)**

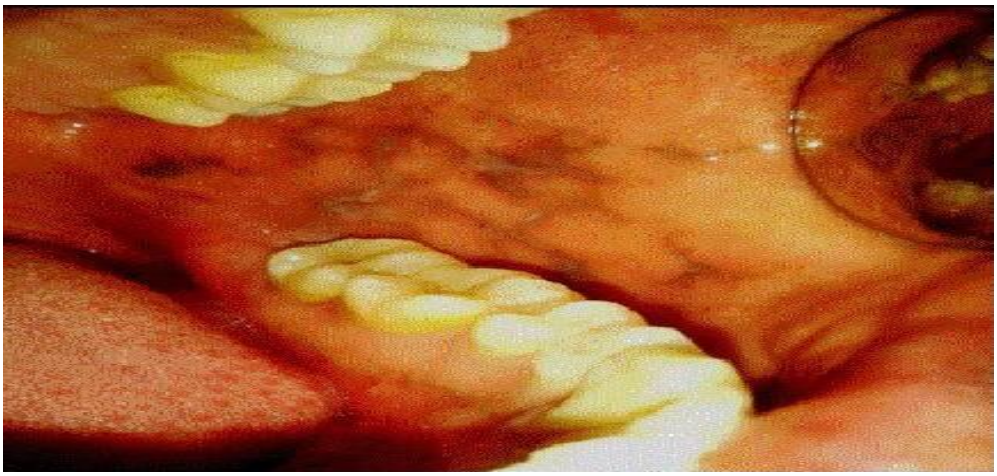

Figure 4: Post inflammatory pigmentation

1.2.1.2.5 Pigmented Nevi

Pigmented nevi of the oral cavity are uncommon. The clinical features include brownish black to blue elevated papules with a well-defined border. Nevi can be classified based on time of occurrence as congenital and acquired. Congenital nevi, can be sub-classified as giant nevus and small nevus. An acquired nevus is also called as a mole, occurs most commonly in the sun exposed regions. Nevus represents a benign proliferation of melanocytes **(Sreeja C et al.,2017)**

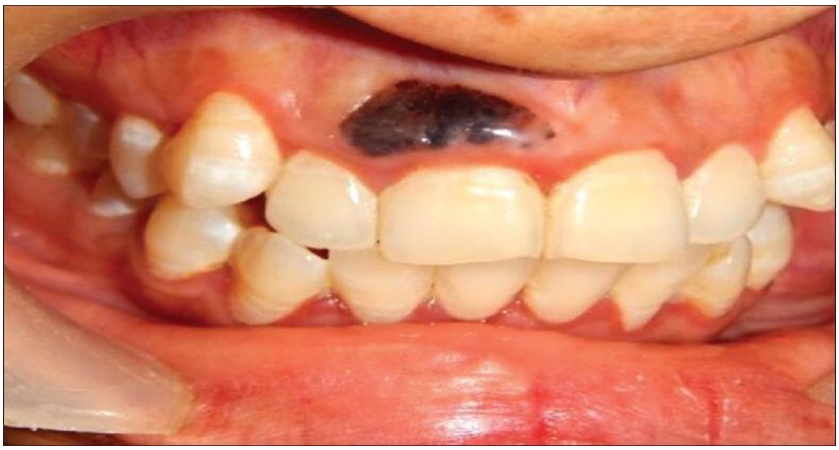

Figure 5: Pigmented nevi

1.2.1.2.6 Oral 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 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 **(Grinspan D et al.,1969).** The most common site is the palate, which accounts for about 40% of cases, followed by the gingiva (30%), which accounts for one third of cases. Other oral mucosal sites may also be affected **(Symvoulakis EK et al.,2006)**

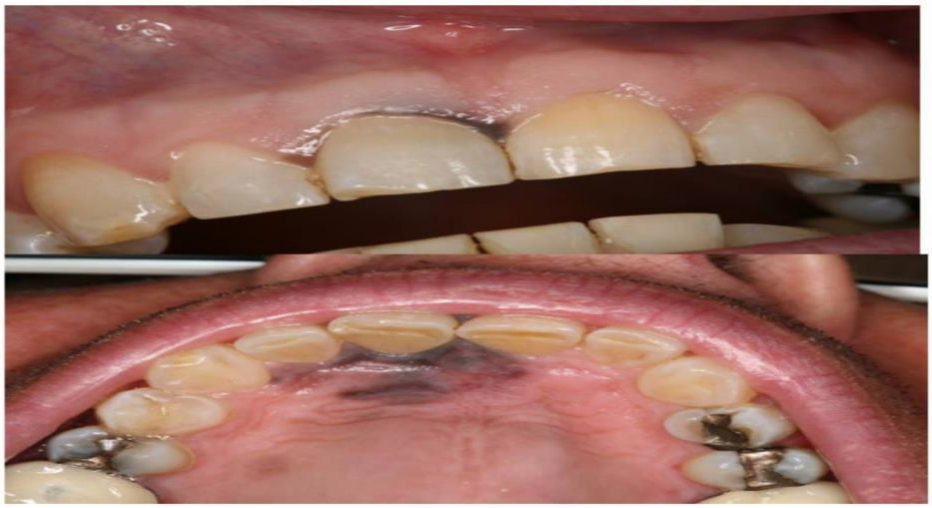

Figure 6 : Oral melanoma

1.2.1.2.7 Ecchymosis

Ecchymosis commonly known as bruises, frequently occur after injury. Traumatic ecchymosis is common on the lips **(Molenda MA etal.,2010)**

1.3.1.2.8 Petechiae

Petechiae are submucous or subcutaneous minute pinpoint hemorrhages. In most cases, the petechiae are identified on the soft palate, although any mucosal site may be affected **(Lynch B et al.,2003)**

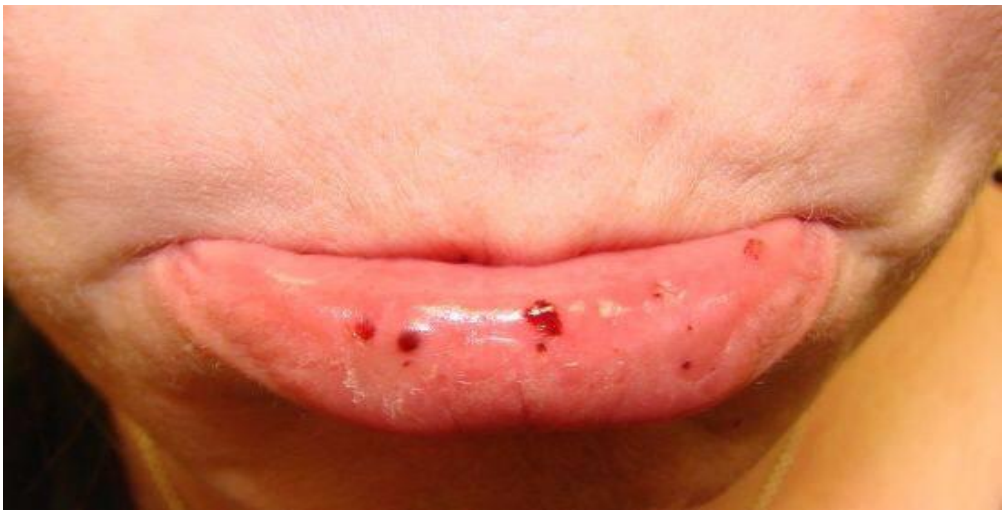

Figure 7: Petechiae

1.2.2 Exogenous pigmentation

1.2.2.1 Heavy Metal Pigmentation Increased levels of heavy metals (e.g., lead, bismuth, mercury, silver, arsenic and gold) in the blood represent a known cause of oral mucosal discolouration. In adults, the most common cause for such increased levels is occupational exposure to heavy metal vapours. In adults, the most common cause for such increased levels is occupational exposure to heavy metal vapours **(Neville BM et al.,2002).** 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 **(Ten Bruggenkate CM et al.,1975)**

1.2.2.2 Drug-Related Discolorations

Numerous systemic medications have been implicated in causing oral mucosal pigmentation by either inducing melanin synthesis or by deposition of the drug or its metabolites. Medications which have been most frequently implicated include antimalarials, hormones, oral contraceptives, phenothiazines, chemotherapeutics, amiodarone, minocycline. Any patient taking these medications long term should be monitored for the development of oral pigmentation . Hard palate, gingiva and buccal mucosa are the most common locations affected **(Sanjeevini H et al.,2012)(Slominski A et al.,2004).**Clinically, the discoloration is flat and can be focal, multifocal or diffuse. The color may be black, gray, blue or brown. Pigmentation restricted to the hard palate is classically seen with antimalarials, which are commonly prescribed in the treatment of rheumatoid arthritis and systemic lupus erythematosus **(Kopacova M et al.,2009)**

1.2.2.3 Amalgam Tattoo/Foreign Body Tattoo

Mucosal pigmentation can occur due to deposition of exogenous foreign materials such as dental amalgam, tattoo pigment, or graphite. This can occur during placement or removal of a restoration where fragments can enter through an abrasion, extraction site or the gingival sulcus. Amalgam tattoo can occur in anyone with a history of amalgam restorations. It presents as a flat discoloration which can be gray, blue, or black . The borders may be well-defined, irregular or diffuse. The

majority of amalgam tattoos are 6 mm or less .The most common location is gingiva or alveolar mucosa however other sites including buccal mucosa and floor of mouth are often affected **(De Melo Filho MR et al.,2012).**

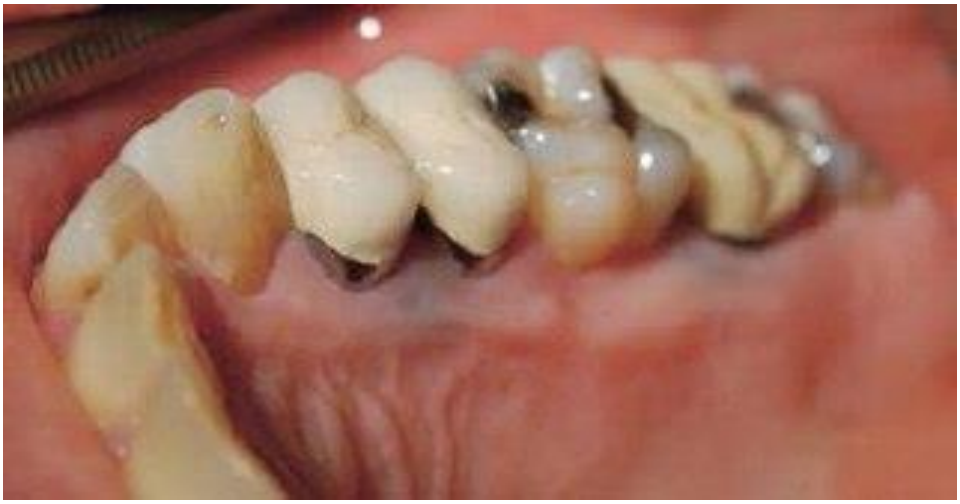

Figure 8: Amalgam tattoo

1.2.2.4 Smoker's Melanosis

Smoker's melanosis is a common, benign, reactive condition resulting in increased pigmentation of the oral mucosa from cigarette or pipe smoking. This process is thought to be a result of either the noxious chemicals in cigarette smoke or heat stimulating melanocytes to protectively produce melanin **(Prabhuji M et al.,2011)(Antony VV and Khan R et al.,2013)(Westerhof W,2006)(Lynch B et al.,2003) .** This condition typically occurs in adults and has been reported in 21.5–30% of smokers . The most common location for smoker's melanosis is the anterior labial mandibular gingiva, however, buccal mucosa, lip, hard palate, and tongue can also be affected .Typically, multiple brown macules are present and may appear light brown to brown–black, depending on duration and amount of tobacco smoking **(Kwon JS et al.,2012)**

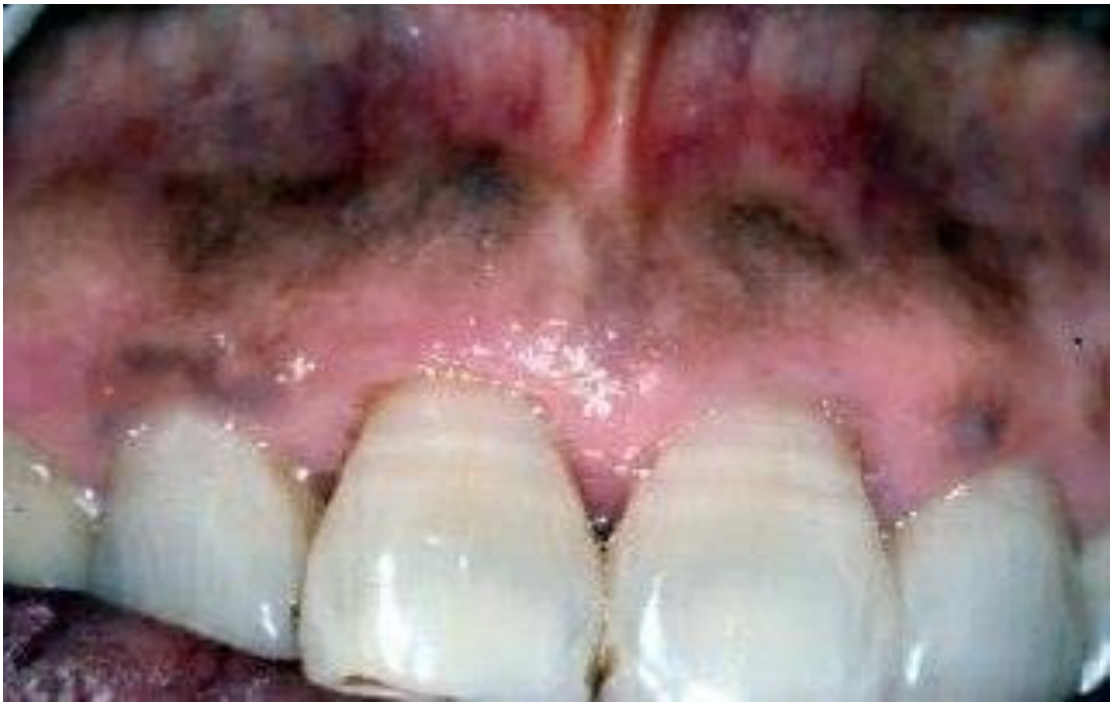

Figure 9: Smoker melanosis

1.3 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 **(Dummett CO,1945)**

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 **(Ozbayrak S et al.,2000)** 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 **(Dummett CO,1960).**

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 **(Fry L and Almeyda JR,1968)** .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 **(Kathariya R and Pradeep AR,2011).**

1.4 CLASSIFICATION:

Pigmented lesions of the oral cavity are of multiple origin.

Different classifications are used at this time:

1.4.1 Dummet et al. (1967)

- Primary oral melanin pigmentations
- Secondary oral melanin pigmentations

- Oral non-melanin pigmentations
- Oral melanoclasias.

(**Dummett CO, Barens G. Pigmentation 1967)**

1.4.2 Patil S et al. (2015) classified pigmented lesions into different groups

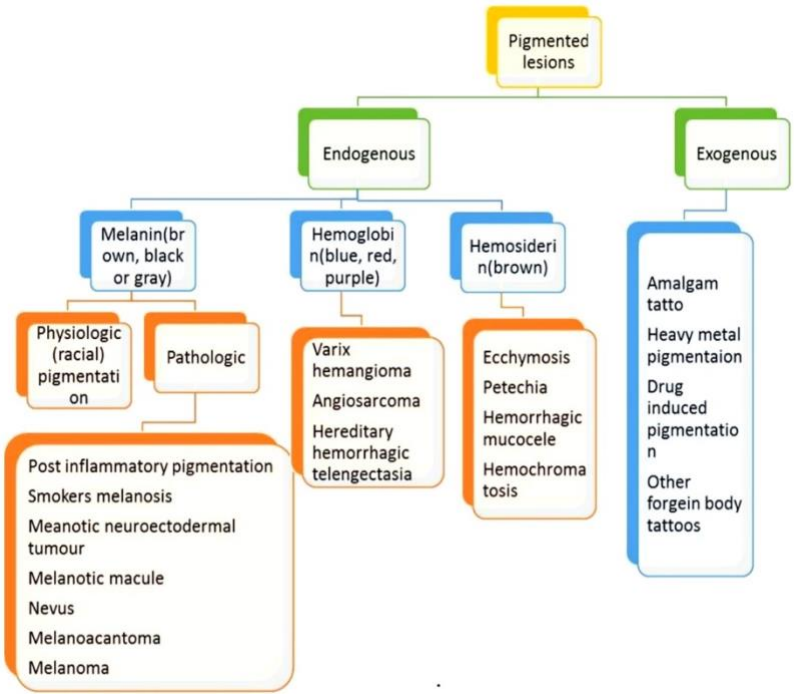

Figure 10: Classification of pigmented lesion (Patil S, et al -2015)

(Patil S, Raj T, Rao RS, Warnakulasuriya 2015)

1.5 REVIEW OF CURRENT INDICES :

Gingival pigmentation has three dimensions: etiology, distribution, and severity. The existing indices on gingival pigmentation are as follows:

1.5.1 Dummet proposed the oral pigmentation index (DOPI) :

Dummet proposed the oral pigmentation index (DOPI) assessment) in 1964. This index of oral pigmentation is the commonly used index due to its simplicity and ease of use. The gingivae of the maxillary and mandibular arches are each divided into 32 unit spaces, sixteen on the lingual aspect and sixteen on the buccal and labial surfaces. Each unit space approximates the area of the marginal gingiva, and extends from the gingival crest apically about 4 or 5 mm up to the level of the attached gingiva. The unit spaces correspond to the buccal and lingual gingival areas which norm ally invest the human adult dentition. In cases in which there are either partially or completely edentulous areas, this division into 32 unit spaces is still maintained since the oral pigmentation is independent of the presence or absence of teeth.The method consists of assigning a numerical oral pigmentation estimate to each one of these 32 unit spaces.

The assigned estimate is based upon the following scale: -

The scores are as follows:

Score 0 - No clinical pigmentation (pink-colored gingiva)

Score 1 - Mild clinical pigmentation (mild light brown color)

Score 2 - Moderate clinical pigmentation (medium brown or mixed pink and brown color)

Score 3 - Heavy clinical pigmentation (deep brown or bluish black color)

$$\text{DOPI assessment} = \frac{\text{Sum of assigned estimates of components}}{\text{32 unit spaces}}$$

The DOPI assessment is scaled according to following designations

0	No clinical pigmentation of the gingiva
0.031-0.97	Mild gingival pigmentation
1.0-1.9	Medium gingival pigmentation
2.0-3.0	Heavy gingival pigmentation

(Dummett CO, Gupta OP 1964.)(Raghu Raaman A, Pratebha B, Jananni M, Saravanakumar R. 2015)

1.5.2 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 gingiva.

(Singh V, Giliyar SB, Kumar S, Bhat M. 2012)

1.6 GINGIVAL DEPIGMENTATION :

Gingival depigmentation can be defined as a periodontal plastic surgical procedure whereby the gingival hyperpigmentation is removed by various techniques. **Malhotra S, Sharma N, Basavaraj P. 2014** Depigmentation isn't a clinical indication treatment of choice where esthetics is a concern and is desired by the patients. **Grover HS, Dadlani H, Bhardwaj A, Yadav A, Lal S.2014**

1.6.1 Gingival depigmentation techniques

Different procedures have been proposed for gingival depigmentation. Roshni & Nandakumar in 2005 classified different gingival depigmentation Methods

1.6.1.1 Methods used to remove the gingival pigmentation:

1.6.1.1.1 Scalpel surgical technique :

It is also called as split thickness epithelial excision **Kumar S, Bhat GS, Bhat KM. 2012** and surgical stripping. **El-Shenawy H, Fahd A, Ellabban M, Dahaba M, Khalifa M. 2017.**Conventional scalpel method involves the surgical excision of gingival epithelium using a scalpel and allowing the denuded connective tissue to heal by secondary intention. **Dummett CO.1946** The new epithelium that forms is devoid of melanin pigmentation. **Roshna T, Nandakumar K. 2005** Care should be taken not to leave any pigmented remnants over the denuded area. **Prasad S, Agrawal N, Reddy N. 2010 .**The scalpel method is one of the most economic techniques and also does not require extensive armamentarium. **Sanjeevini H ;Pudakalkatti P, Soumya B, AaratiN. 2012** Healing with this technique is faster in comparison to other surgical techniques. However, scalpel surgery causes bleeding during and after the procedure and it is necessary to cover the surgical site with periodontal dressing for 7e10 days. Though the initial results of depigmentation procedure are highly encouraging, repigmentationis a possibility. This process may be attributed to the fact that active melanocytes from the adjacent pigmented tissues migrate to the treated areas. Thinner gingival biotype and narrow papillary areas contraindicate the use of this technique. As seen Figure11 **Bergamaschi O, Kon S, Doine AI, Ruben MP.1993**

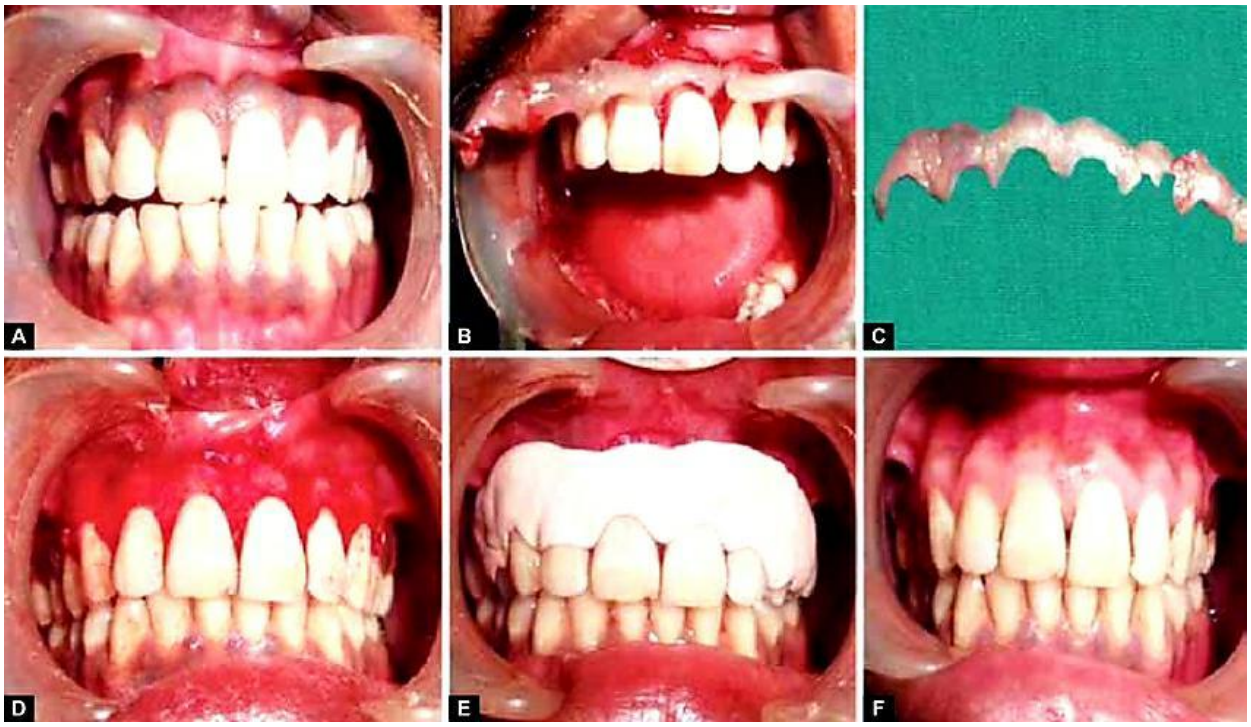

Figure 11 : Pre-operative view, b. Maxillary pigmentation removal using scalpel surgical technique, c. Immediately after depigmentation, d. Mandibular pigmentation removal, e. Immediately after depigmentation, f. After 3 month.

1.6.1.1.2 Gingival Abrasion :

The first case using this technique was documented by Ginwalla et al. in 1966. **Ginwalla TM, Gomes BC, Varma BR. 1966** It involves the denuding of pigmented gingival epithelium by superficial abrasion using grit football shaped or doughnut-shaped coarse diamond burs in a low-speed handpiece. **Kumar S, Bhat GS, Bhat KM. 2012** Extra care should be taken to control the speed and pressure of handpiece, so as not to cause unwanted abrasions or pitting of the tissue. **Kathariya, Pradeep 2011** It is a relatively a non-invasive and cost effective technique and does not require any specific instruments. However, it is associated with various drawbacks

such as technique sensitivity, increased treatment duration, post-treatment pain, placement of periodontal dressing, and high recurrence rate. **Kumar S, Bhat GS, Bhat KM. 2012** Exposure of underlying alveolar bone can occur with high speed and/or increased pressure So minimum pressure with feather light brushing strokes with copious saline irrigation should be used without holding the bur in one place to perceive Good results. As we seen in Figure 12 **Deepak P, Sunil S, Mishra R.Sheshdri 2005**

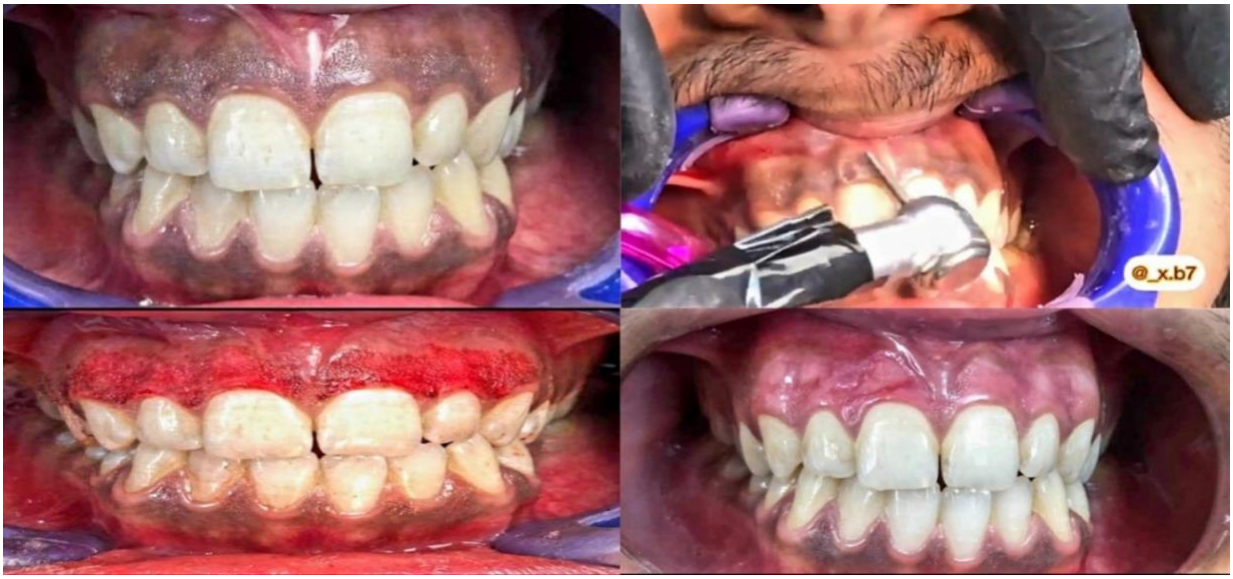

Figure 12: a. Pre-operative, b. Gingiva depigmentation by bur abrasion, c. 3 Months post-operative view

1.6.1.1.3 Electro-surgery :

Electro-surgery is the use of high frequency electrical energy in the radio transmission frequency band, which is applied directly to tissue to induce histological effects. As the current passes, the impedance to the passage of current though the tissue generates heat, which boils the tissue water, creating steam,

resulting in either cutting or coagulation of tissue. **Prasad S, Agrawal N, Reddy N. 2010** It was found that this method controls hemorrhage, permits adequate contouring of tissues, causes less discomfort to patient, less scar formation and lesser chair time. **Sanjeevini H, Pudakalkatti P, Soumya B, Aarati N. 2012** However, pain and patient discomfort during the initial healing period is more with this technique. **Bergamaschi, Kon , Doine, Ruben 1993** Electro-surgery requires more expertise than scalpel surgery. Prolonged or repeated application of current to tissue induces heat accumulation and undesired tissue destruction. Contact with periosteum or alveolar bone and vital teeth should be avoided As we seen in Figure 13 **Ginwalla, Gomes, Varma 1966**

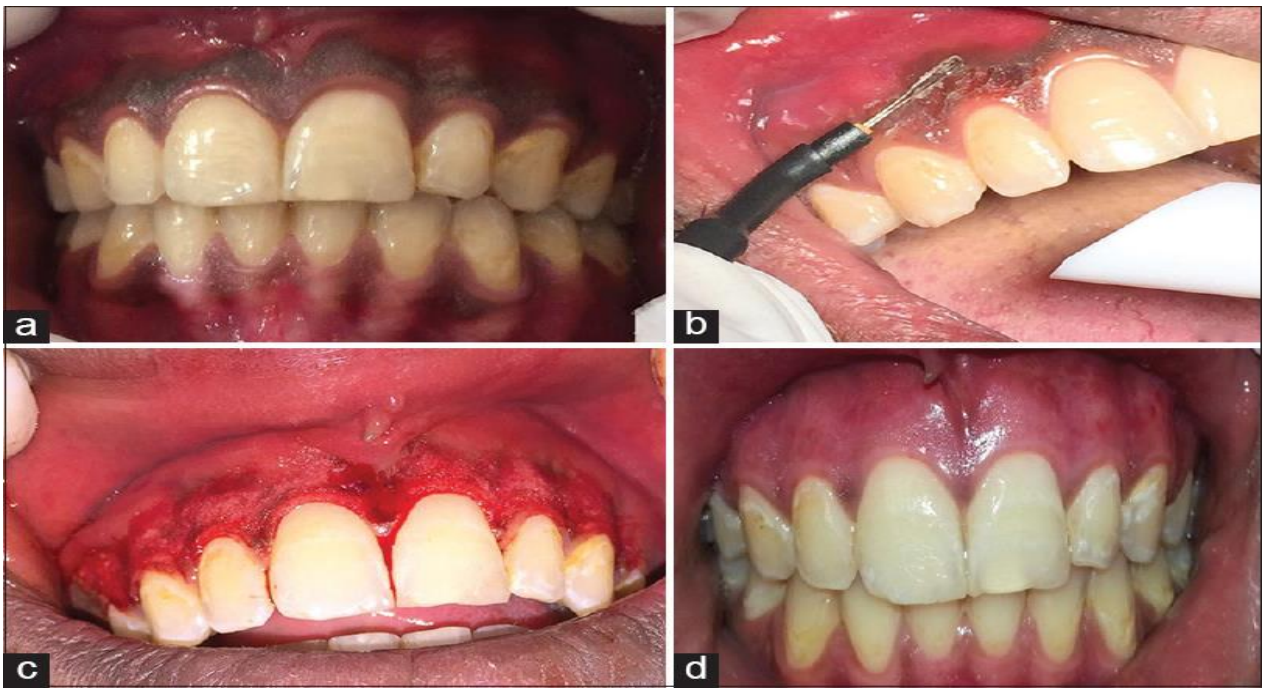

Fig 13 : Gingival depigmentation by electrocautery. (a) Preoperative; (b) use of loop electrode; (c) after de-epithelialization; (d) postoperative - 6 months

1.6.1.1.4 Cryosurgery :

Cryosurgery is most widely accepted method of gingival depigmentation. **Kumar S, Bhat GS, Bhat KM. 2013** It involves freezing of gingiva with the application of different materials, i.e. cryogen such as liquid nitrogen at very low temperatures. **Moneim RA, El Deeb M, Rabea AA.2017** The effect of ultralow temperature of cryogen on gingival tissue causes the epithelium to undergo cryonecrosis, which helps to eliminate gingival pigmentation. It is an inexpensive method with long-term superior esthetic results, rapid healing, and low recurrence rate. Lack of bleeding, pain and scar formation, application without regional anaesthesia, sutures or drugs, ease of application of cryogen at papillary areas and need of no complicated instruments, and prioritizes the cryosurgery over other depigmentation methods. **Kumar S, Bhat GS, Bhat KM. 2013** Post-operative swelling and difficulty in controlling the penetration depth constitute disadvantages of this technique. **Prasad, Agrawal, Reddy. 2010** The cryotherapy has some direct effects including cell dehydration, enzyme inhibition, protein denaturation, and cell death due to thermal shock. It has also some indirect effects such as changes in vasculature and immune response of the tissue, which leads to cell death. As we seen in Figure 14 **Shirazi ARS, Taghavi AM, Khorakian F. 2010**

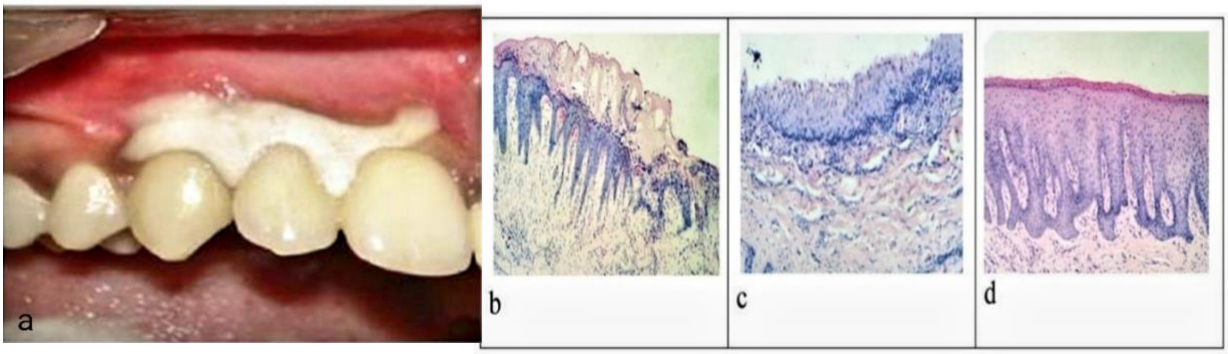

Figure14:Depigmentation by cryosurgical technique. b. 8 hours following freezing showing epithelial degeneration. c. Specimen after 24 h showing loss of rete pegs. d. Clinical resemblance after a week of application of cryogen.

1.6.1.1.5 Lasers :

Laser ablation of gingival depigmentation has been recognized as one of the effective, pleasant and reliable techniques **Prabhuji M, Madhupreetha S, Archana V. 2011**. It's usually sufficient to eliminate the pigmented areas and do not require any periodontal dressing **Javali M, Tapashetti R, Deshmukh J. 2011** It also shows reduced pain and discomfort due to formation of protein coagulum. Meanwhile, it allows clean and dry operating field and stable results **Simsek Kaya G, Yapici Yavuz G, Sümbüllü MA, Dayi E. 2012**. Laser light may also seal free nerve endings **Nagati RR, Ragul M, Al-Qahtani NA,Pasupuleti MK. 2017**. But it also has its own disadvantages of delayed wound healing, thermal damage, deep penetration and the comparably high costs of the procedure **Kumar S, Bhat S, Bhat M. 2012**. Different lasers have been used for gingival depigmentation including carbon dioxide (10.600 nm), diode (810 nm), neodymium: Yttrium, aluminium, and garnet (Nd: YAG, 1,064 nm) lasers. The diode laser has been introduced in dentistry few years back. It is a solid-state semiconductor laser that typically uses a combination of elements to change electrical energy into light energy. It also can be delivered through a flexible quartz fiber optic handpiece. This energy level is absorbed by pigmentation in the soft tissues and makes the diode laser an excellent hemostatic agent **Prabhuji M, Madhupreetha, Archana 2011**. It also allows good visibility at the surgical site. The post-operative patient comfort is better at the

surgical sites treated with diode laser than surgical scrapping method **Nagati RR, Ragul M, Al-Qahtani NA, Ravi KS, Tikare S, Pasupuleti 2017** The CO2 laser causes minimal damage to the periosteum and bone under the gingiva being treated, and it has the unique characteristic of being able to remove a thin layer of epithelium cleanly **Prasad, N. Agrawal, N. 2010**. YAG laser has demonstrated the best application of laser use, leaving the least thermal damage. Shown Figure15

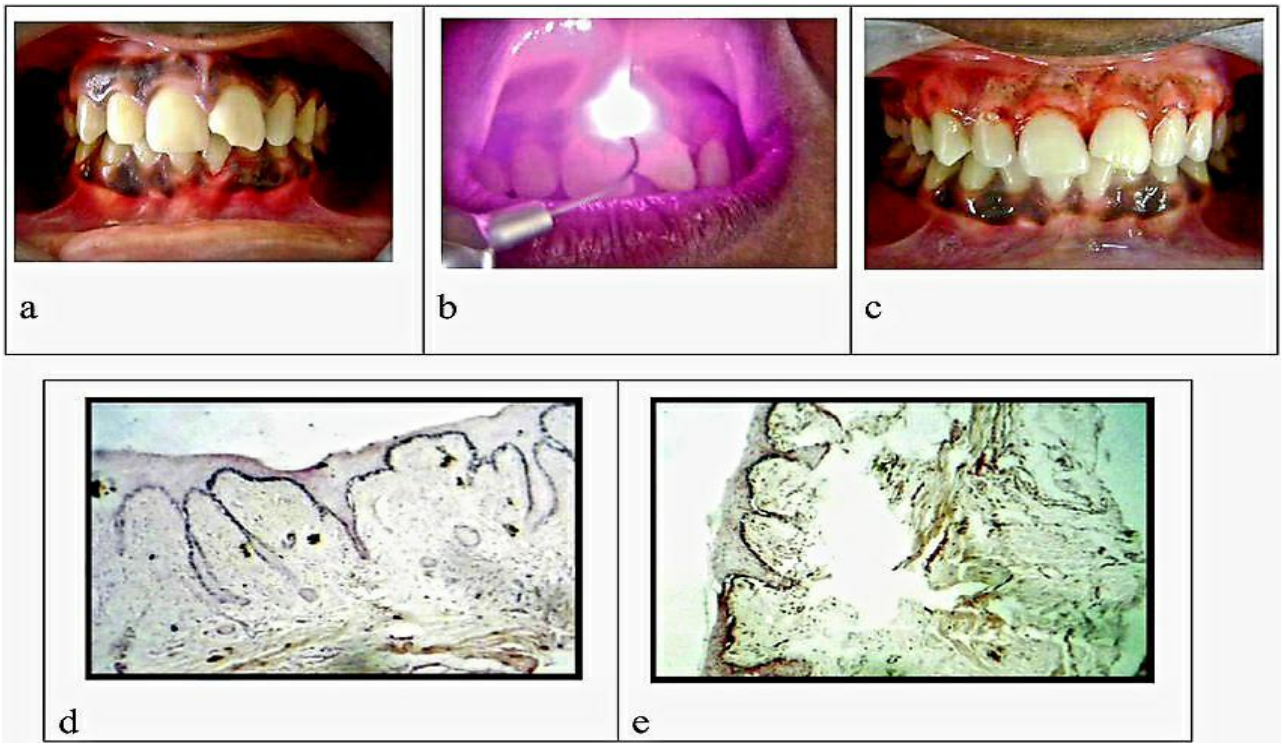

Figure 15 : a. Pre-operative situation, b. Use of the FOX diode laser to treat gingival pigmentation, c. Immediate post-operative view. d:Postoperative biopsy specimen form Er: YAG treated site showed basal cells with moderate staining positivity (50–75%), whereas (Fig. 5e) showed biopsy from CO2 treated sites showed mild to moderate staining (<50%) positivity

1.6.1.1.6 Radiosurgery :

Describes the most advanced form of electro-surgery. It is the removal of soft tissue with the aid of radio frequency energy. This electromagnetic energy operates between the frequencies of 3.0 MHz (MHz) to 4.0 MHz, with 4.0 MHz being the optimal frequency. The main advantage of radiosurgery can be found in its ability to produce coagulation in the operative area which would often have extensive bleeding. Also, some studies reported less thermal damage and faster healing with the 4 MHz radio wave technology over the scalpel and lasers **Sherman J, Gürkan A, Arikan F. 2009**. On the other hand, the main disadvantage of this method is that it requires at least two sittings for completion within 2 weeks of treatment **Herschfeld I, Herschfeld L. 1951.** Papillary areas can be easily depigmented with radiosurgery. As shown in Figure 16 **Kumar S, Bhat GS, Bhat KM. 2012**

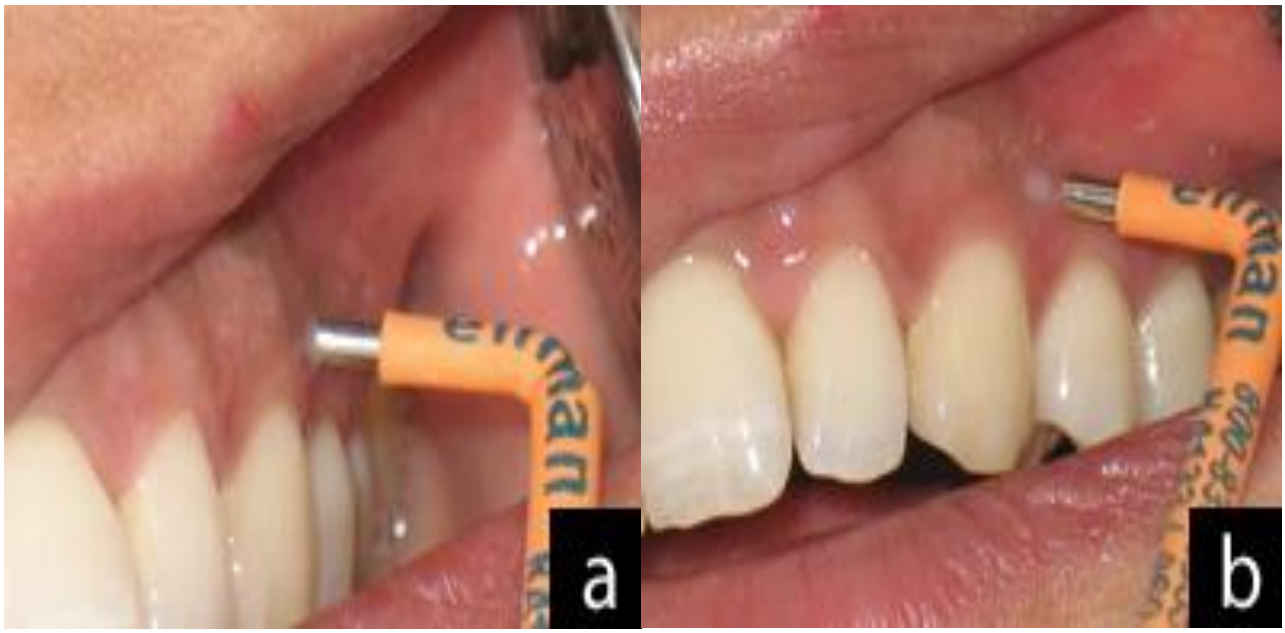

Fig 16 : a , b Application of the tapping electrode to the pigmented gingiva

1.6.1.1.7 Chemical Gingival Peeling :

It is a treatment method used to destroy the overlying gingival epithelium using a chemical peeling agent. A variety of chemical agents are available such as phenols, salicylic acid, glycolic acid, and trichloroacetic acid **Dummett CO. 1946**. The most commonly used are phenols and alcohols.[1475] In a study by Hirschfield and Hirschfield in 1951, pigmented gingiva was burnt out by destroying tissue down to and slightly below the basal layer of mucous membranes using a mixture of 90% phenol and 95% alcohol. However, repigmentation and relapse occurred in all cases shortly after the application of either agent **Herschfeld L. 1951**. As phenols may induce cardiac arrhythmias, cardiac monitoring is necessary **Kathariya R, Pradeep AR.2011**.The inability to control the depth of penetration and amount of destruction are the main drawbacks of this method. Thereby, these methods are no longer in use and are unacceptable to the clinicians as well as patients. As seen in Figure 17

Fig 17 : Chemical Gingival peeling

1.6.1.1.8 Ascorbic Acid :

Ascorbic acid/Vitamin C has potential in the treatment of gingival melanin pigmentation. It inhibits the melanin formation by suppressing the tyrosine activity which is essential for melanin biosynthesis **Shimada Y, Tai H, Tanaka A, Ikezawa-Suzuki I, TakagiK, Yoshida Y, et al. 2009**. Furthermore, ascorbic acid directly down regulates dopaquinone formation, a precursor in melanin synthesis, thus inhibiting the melanin formation. A study by Sheel et al. have reported the delay in repigmentation of gingiva with the local application of ascorbic acid following the depigmentation procedure. As Seen in Figure **18 Kathariya** ,**Pradeep 2011**.

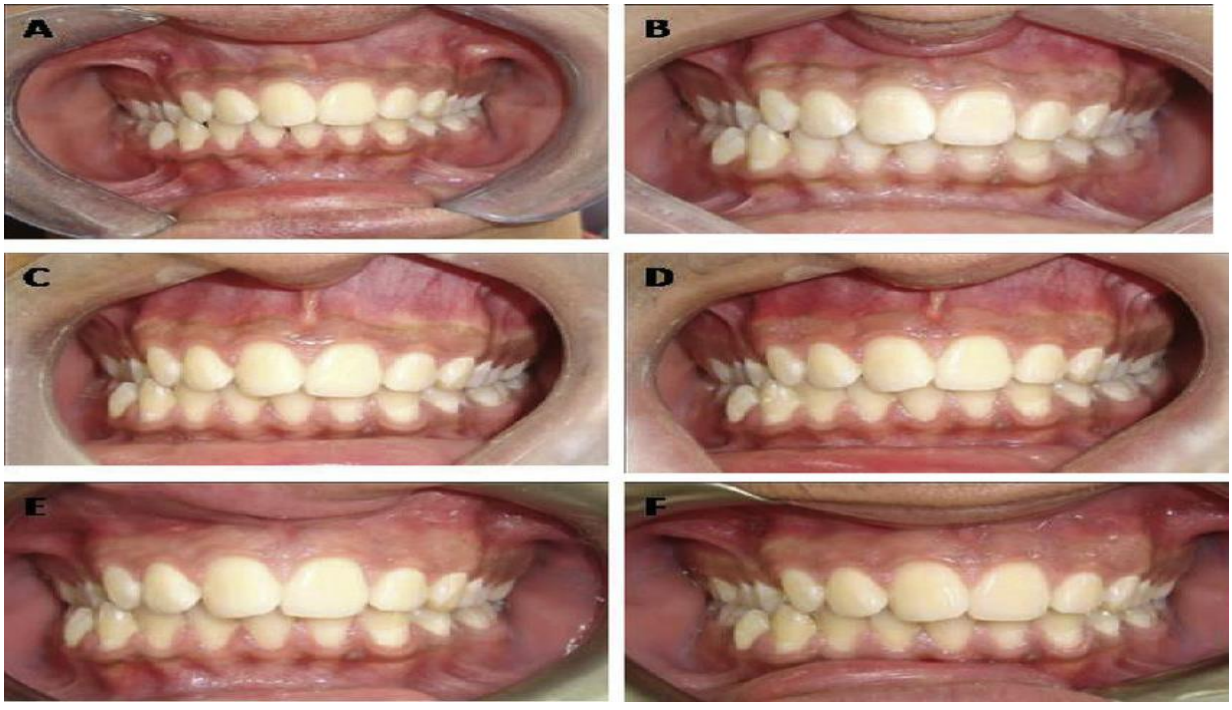

Fig 18 : The stages of vitamin C depigmentation; A: pre-operative, B: after 1st visit, C: after 2nd visit, D: after 3rd visit, E: after 1 month and F: after 9 months follow up

1.6.1.2 Methods Used To Mask The Gingival Pigmentation:

1.6.1.2.1 Free Gingival Graft :

In this technique, an unpigmented free gingival autograft harvested from the patient's palate is placed on the prepared recipient site **Tamizi M, Taheri M.1996**. This technique masks the pigmented gingival area rather than eliminating it **Malhotra S, Sharma N, Basavaraj P. 2014**. Two surgical sites, post-operative discomfort due to pain, technique sensitivity, and ghost-like appearance of the treated site due to hypopigmentation are the drawbacks of this technique. As Shown in Figure 19 **Sanjeevini H, Pudakalkatti P,2012.**

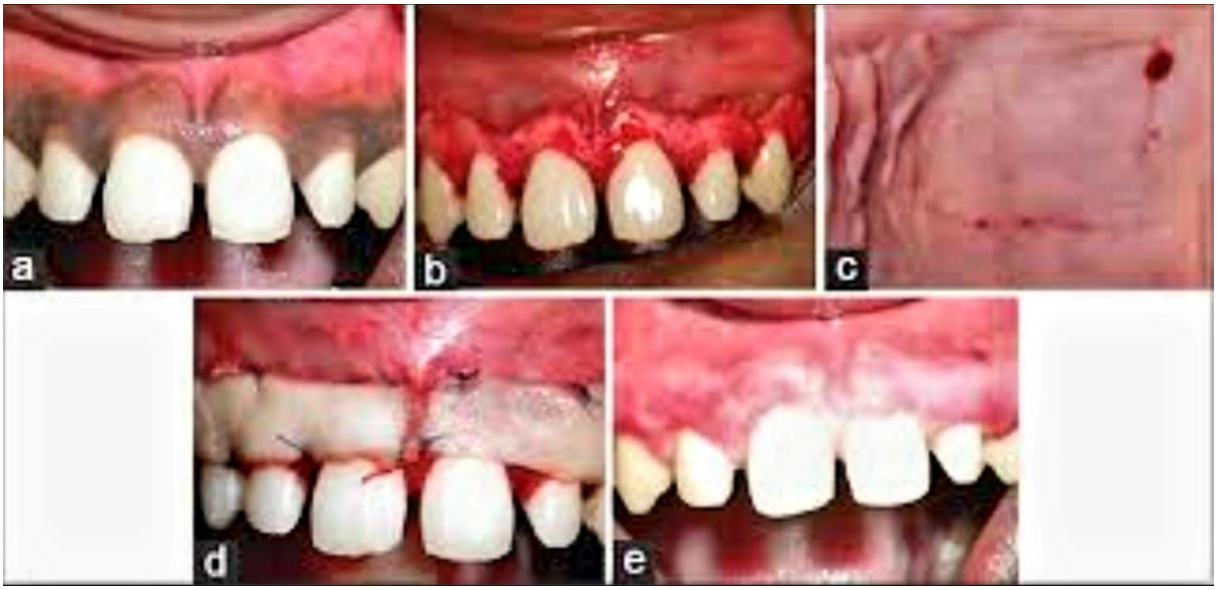

Fig 19 : a: pre-operative b: Immediate post-operative view c: autograft from patient's palate d: sutured with recipient site e: after 6 months follow up

1.6.1.2.2 Acellular dermal matrix allograft : (ADMA)

It can be used as a safe substitute for free gingival autograft in the treatment of gingival hyperpigmentation **Novaes AB, Jr, Pontes CC, Souza SL, Grisi MF, Taba M.2002**. ADMA has benefits of elimination of second surgical procedure for donor site, decreased post-operative complications, availability of unlimited amount of graft material, and satisfactory esthetic results than the FGG. However, it is technique sensitive, expensive and requires clinical expertise as we can seen in Figure 20 **Kathariya R, Pradeep AR. 2011**.

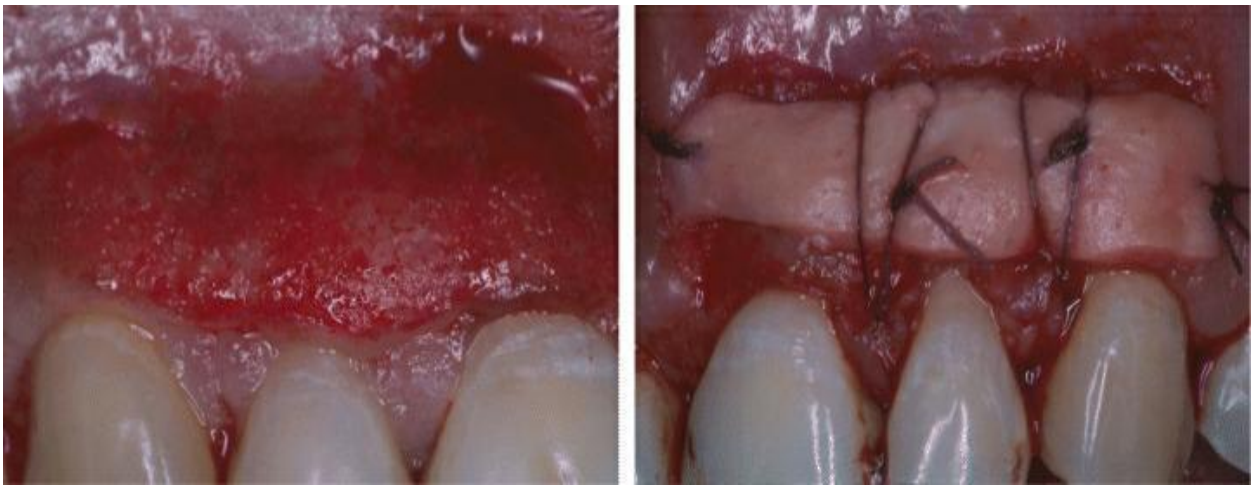

Fig 20 : Acellular dermal matrix allograft (ADMA)

1.6.2 Criteria for Selection of Technique

Patient's skin color, extent of gingival pigmentation, lip line, upper lip curvature, esthetic concern and expectation from the treatment, influence the orchestration of treatment plan, and selection of technique. **Malhotra, Sharma, Basavaraj 2014**. However, the procedure adopted should be simple, cost-effective, and comfortable to the clinician as well as patient with less pain and minimal tissue loss **Dummett CO. 1946**. Caution must be employed to avoid injury to soft tissues and adjacent

teeth. Inappropriate technique or inadvertent application can result in a gingival recession, damage to attachment apparatus, underlying bone, as well as enamel**.**

1.7 Repigmentation :

Oral repigmentation refers to the clinical reappearance of melanin pigment following a period during which clinically pigmented oral tissues were depigmented Dummet et al. noticed repigmentation as early as 33 days. They noticed 100% repigmentation in dark complexioned individuals **Dummett CO, Bolden TE 1963**. Kaur et al. noted that the melanin pigmentation score decreased from 2.40 to 0.93. The mechanism of repigmentation is not understood; but according to the migration theory, active melanocytes from the adjacent pigmented tissues migrate to the treated areas, causing repigmentation. Another reason for repigmentation may be the melanocytes which are left during surgery. These may have become activated and started synthesizing melanin wherever appeared, was of very mild intensity in the form of very small spots, dots or streaks without creating any esthetic problem, as compared to dark continuous bands of heavy or moderate intensity area and cause repigmentation **Perlmutter S, Tal H. 1986**. It has been hypothesized that the rate of melanin formation is higher in dark complexioned people than fair complexioned **Billingham RE. 1949**. The gingival pigmentation is more in the anterior than posteriors and have been attributed to sunlight exposure. **Raut, Baretto 195**

Conclusions :

1.Health and appearance of gingiva are important parts of a smile

2.The gingiva is considered the most frequently pigmented tissue in the oral cavity.

3.Gingival pigmentation is a discoloration of the gingiva due to a variety of conditions associated with several Endogenous and exogenous etiologic feature

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