

Review Article

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Gingival Depigmentation

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ABSTRACT

A pretty smile enhances an individual's self-confidence. A smile express joy, success, affection and or even say hello and reveals self-confidence and kindness. Gingival melanin pigmentation is one of the factors which determine the smile of an individual. Excessive gingival pigmentation is a major esthetic concern for many individuals. Gingival pigmentation occurs in all races of man and it varies from one race to another. It may vary depending on whether it is physiological or pathological. Melanin pigmentation of the gingiva is completely benign and it does not present a medical problem, complaints of 'black gums' are commonly seen in patients having high smile line (gummy smile). Different treatment modalities are reported for depigmentation of gingiva; which includes cryosurgery, electrosurgery, laser and scalpel surgical technique.

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Introduction

A pretty smile enhances an individual's self-confidence. Dark or black coloured gingiva is an esthetic concern especially in patients with high lip line or gummy smile [1]. Gingival colour is generally seen as "coral pink". The colour of the gingiva varies among different individuals and it is thought to be associated with cutaneous pigmentation [2]. Melanin pigmentation of the gingiva occur in all races [3].

Melanin, a brown pigment, is the most common cause of endogenous pigmentation of gingiva and is the most predominant pigmentation of mucosa [4]. Gingival pigmentation exists as a deep purplish discoloration or it is seen as irregularly shaped brown and light brown or black patches. It results due to melanin granules, which are produced by melanoblasts. Melanocytes are highly differentiated cell with round nucleus which produce melanin pigments which is transferred to adjacent keratinocytes and macrophages. Melanin is located in the basal layer.

Gingiva also exhibit some other factors for pigmentations, they may be due to etiological factors. Some common factors are benign and malignant lesions, use of drugs, smoking habit and also due to some systemic problems.

Gingival Depigmentation

Gingival depigmentation can be defined as a periodontal plastic surgical procedure whereby the gingival hyperpigmentation is removed or reduced by various techniques [5]. Melanin

pigmentation in gingiva creates unaesthetic smile which requires depigmentation procedure to enhance the aesthetics.

Physiology of Melanin Pigmentation

The colour of gingiva depends upon-

- The number and size of vasculature
- Epithelial thickness
- Degree of keratinization
- Pigments within the gingival epithelium

Melanin is the end-product of complex multistep transformations of L-tyrosine, are polymorphous and multifunctional biopolymers, represented by [6]

- Eumelanin
- Pheomelanin
- Neuromelanin

Melanosomes

- Melanin is a pigment produced by melanocytes that reside in the basal layer of the epidermis. It is stored in vesicles called melanosomes and it is transferred to adjacent epithelial cells via dendritic processes.
- Melanocytes are cells that are capable of synthesizing within specialized organelles, the melanosomes, initiates events leading to the synthesis and deposition of melanin.

According to the degree of maturation, melanosomes are classified into 4 stages: Nordlund et al 1998

1. Type I melanosomes have intraluminal vesicles and resemble multivesicular bodies.

2. Type II is characterized by an elongated, elliptical shape, with intraluminal fine fibrils giving a striated appearance
3. Type III exhibits pigment deposition along the fibrils
4. Type IV has a dense pigmentation filling the organelle and obscuring the fibrillar structure.

Formation of Melanin

Functions of melanocytes

Melanin, produced by melanocytes determines the colour of skin, hair and eyes, and provides protection from stressors such as UV radiation, reactive oxygen species and free radicals in the environment. Melanin also has the capacity to sequester metal ions and to bind certain drugs and organic molecules [7, 8]. The colour of skin or pigmented part of the oral mucosa is genetically determined by the number and size of the melanosomes and the type of melanin (eumelanin, pheomelanin) that they produce.

Etiology

The gingiva is considered as the most commonly pigmented tissue in the oral cavity [9]. Gingival pigmentation is the discoloration of the gingiva due to different lesions and conditions which are associated with several endogenous and exogenous etiologic features [10]. It may vary 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 source of a mucosal pigmentation before planning a treatment of such type of lesion [11]. There are many local and/ or systemic factors like genetics, tobacco use, antimalarial agents and tricyclic antidepressants drugs.

Broadly gingival pigmentation is classified into:

- Physiologic gingival pigmentation
- Pathologic gingival pigmentation

Physiologic Gingival Pigmentation

It is otherwise known as racial pigmentation. Except albinos, all other patients have some degree of physiologic melanin deposition throughout their epidermis. It develops during first two decades of life but it will be observed by the patient later on. The pigmentation is an asymptomatic condition and therefore, treatment is not required for this. Oral gingival pigmentation occurs in all races of man and it varies from one another. There are no specific differences in oral pigmentation between males and females. The intensity and distribution of racial pigmentation of the oral mucosa varies among the races, between different individuals of the same race and also within different areas of the similar mouth. The most common intraoral pigmented area is Attached Gingiva. Other uncommon sites are tongue, hard palate and lips [12]. Color variation in this type may be uniform, unilateral, bilateral, mottled, macular or blotched and may involve the gingival papillae alone or it may extend throughout the gingiva and into other oral tissues [13].

The process of pigmentation consists of three phases

- Activation of melanocytes
 - Synthesis of melanin
 - Expression of melanin
1. The activation phase occurs when the melanocytes are stimulated by factors like stress hormones, sunlight etc. leading to production of chemical messengers like melanocyte stimulating hormone [14].
 2. In synthesis phase, melanocytes make granules known as melanosomes. This process occurs when the enzyme tyrosinase (TYR) converts amino acid tyrosine into a molecule called 4-dihydroxy phenylalanine (DOPA). Tyrosinase then

converts DOPA into secondary chemical DOPA quinone. DOPA quinone in presence of cysteinyl reacts and forms 3- or 5-cysteinyl DOPAs, which then oxidize and polymerize to give rise to a yellow-red soluble melanin called pheomelanin. After a series of reactions, DOPA quinone is converted into either dark melanin (eumelanin) or light melanin (pheomelanin).

3. In expression phase, melanosomes are transferred from the melanocytes to the keratinocytes, which are the skin cells located above melanocytes in the epidermis. After this, melanin color eventually becomes visible on the surface of skin [15].

Pathologic Gingival Pigmentation

- ❖ Endocrine diseases: like Addison's disease, Albright's syndrome, Acromegaly, and Nelson's syndrome [16].

Addison's Disease

It is otherwise known as primary hypoadrenalism. It is due to progressive bilateral destruction of the adrenal cortex caused by autoimmune disease, infection or malignancy. The lack of adrenocortical hormones in the blood stimulates production of adrenocorticotrophic 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 [17]. Oral involvement presents as diffuse brown patches on the gingiva, buccal mucosa, palate and tongue, which may resemble physiologic pigmentation [18].

Figure 6 represents addison's disease

❖ Kaposi's sarcoma

Kaposi's sarcoma is the most common malignancy associated with human immunodeficiency virus infection. It may also affect the whole part of the body. Although, palate is the most common site of AIDS related Kaposi's sarcoma, intraoral lesions may also involve the gingiva and other areas. Gingival lesions may extend into the free gingiva and adjacent mucosa or involve the frenulum [19].

Figure 7 represents kaposi's sarcoma

❖ Smoker's Melanosis

Smoking causes oral pigmentation in light-skinned individuals and, but more prominent in dark-skinned patients. There is increased production of melanin, which may provide a biologic defence against the noxious agents that is present on tobacco smoke. Smoker's melanosis increases with the use of tobacco. Clinically it is represented as a multiple brown pigmented macule. Smoker melanosis has been reported in 22% of smokers [20].

❖ Amalgam tattoo

Sometimes, accidental displacement of metal particles in oral soft tissues during restorative dental procedures using amalgam (tooth restorative material) may result in amalgam tattoo. Metal particles may penetrate into oral tissues and cause discoloration overtime. Amalgam pigmentation is generally called "Amalgam tattoo". Generally, it appears as black, grey or a combination of both. It is an iatrogenic lesion which is caused by traumatic implantation of amalgam into the soft tissues. Amalgam tattoo is the most common localized pigmented lesion in the mouth [21].

Figure 10 represents amalgam tattoo

❖ Post-Inflammatory Pigmentation

Oral post-inflammatory pigmentation (OPP) is the discoloration of the oral mucosa which is caused by the excess production of melanin and its deposition within the basal layer of the

epithelium and the connective tissues of the affected areas by chronic inflammation [22]. Long standing inflammatory mucosal lesions, mainly lichen planus can cause mucosal pigmentation. This phenomenon is more frequently seen in the dark-skinned individuals. Clinically, OPP appears as a localized or diffuse, black to brown pigmentation.

Figure 11 represents post-inflammatory pigmentation

❖ **Drug associated with oral mucosal pigmentation**
A large variety of medications cause oral mucosal pigmentation. The pathogenesis of this depends upon the causative drug. Some drugs are chloroquine, quinine, minocycline, zidovudine, chlorpromazine, ketoconazole, bleomycin, cyclophosphamide. It can involve accumulation of melanin pigments under the influence of drugs or deposition of iron after damage to dermis [23]. Chloroquine which is an antimalarial drug appears as blue-grey or blue-black due to mucosal discolouration. In most cases, hard palate is involved. According to laboratory studies, these drugs produce a direct stimulatory effect on melanocytes [24].

Figure 9 represents drugs associated with oral mucosal pigmentation.

❖ **Pigmented Nevi**
It is an uncommon condition. The clinical feature of pigmented nevi includes brownish black to blue elevated papules with a well-defined border. They are generally asymptomatic. 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 otherwise called as a mole, are most commonly seen in sun exposed regions. Nevus represents a benign proliferation of melanocytes [25].

Figure 8 represents pigmented nevi

❖ **Mucosal melanomas**
Mucosal melanomas are extremely rare. They have higher prevalence in Japanese people. It has a propensity to occur on the anterior aspect of labial gingiva as well as the anterior aspect of the hard palate. In early stages, it appears as brown or black plaques and eventually it becomes more diffuse, nodular and tumefactive [26, 27].

Figure 12 represents mucosal melanoma.

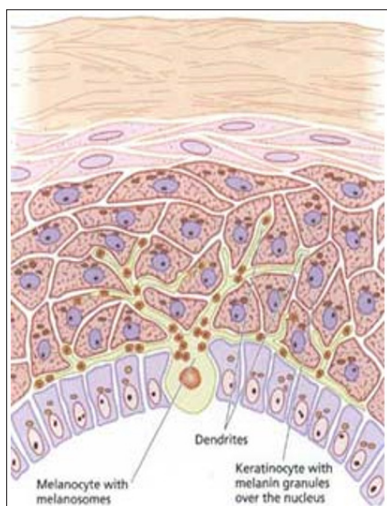


Figure 1: Represents Structures in Melanosomes

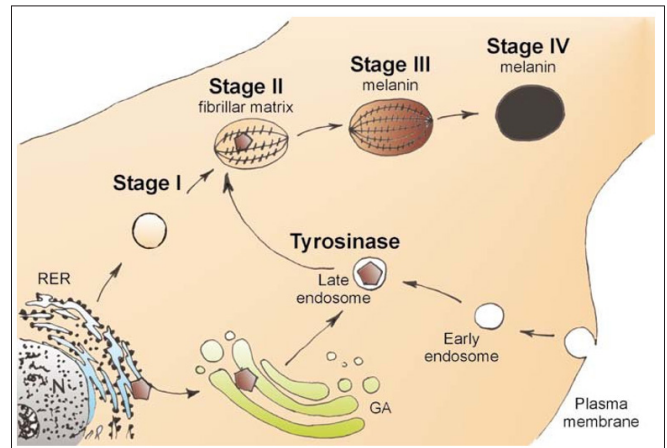


Figure 2: Represents Melanosome Formation & Maturation during Melanin Production

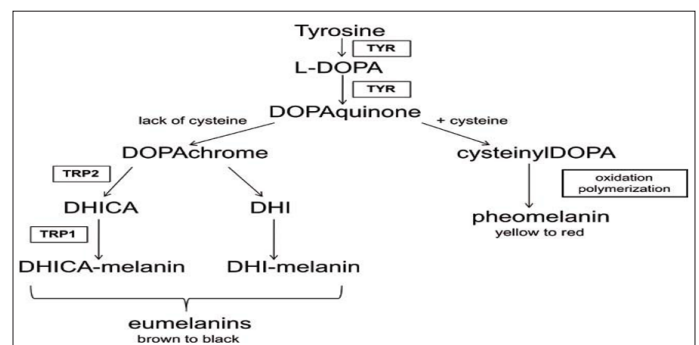


Figure 3: Represents Formation of Melanin

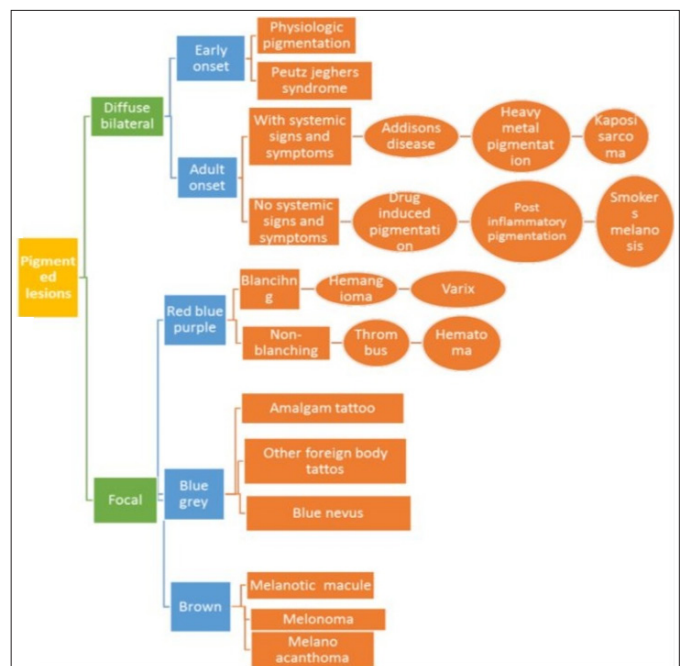


Figure 4: Represents Classification of Pigmented Lesion (Kauzman et al 2004)

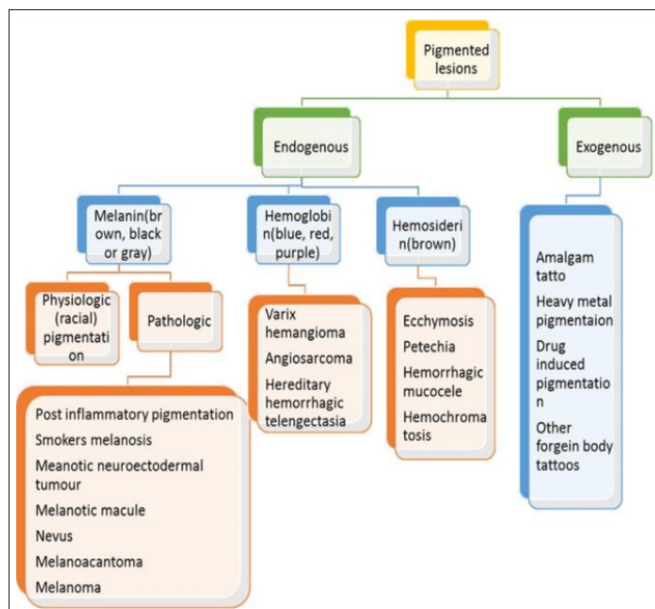


Figure 5: Represents Classification of Pigmented Lesion (Patil S, et al-2015)



Figure 6: Represents Addison's Disease



Figure 7: Represents Kaposi's Sarcoma



Figure 8: Represents Pigmented Nevi



Figure 9: Represents Drugs associated with Oral Mucosal Pigmentation



Figure 10: Represents Amalgam Tattoo



Figure 11: Represents Post-Inflammatory Pigmentation



Figure 12: Represents Mucosal Melanoma

Criteria for Selection of Technique

It depends upon the skin colour of the patient, extent of the gingival pigmentation, lip line, upper lip curvature, esthetic concern and expectation from the treatment, influence the patient regarding each treatment modalities, and selection of the technique [28].

However, the procedure designed should be simple, cost-effective, and comfortable to the patient as well as clinician [29]. Precaution must be taken 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.

Gingival Depigmentation Procedure

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

Methods Used to Remove the Gingival Pigmentation

1. Surgical method
2. Chemical method

Surgical Method

- a. Scalpel surgical technique
- b. Bur abrasion method
- c. Electro-surgery
- d. Cryosurgery
- e. Lasers
- f. Radiosurgery

Scalpel Surgical Technique

It is also called as split thickness epithelial excision and surgical stripping [30]. It was one of the earliest techniques used for gingival depigmentation and it still is the most well practiced treatment modality. The procedure involves surgical removal of the gingival epithelium along with a layer of the underlying connective tissue. After giving adequate local anesthesia, it will be removed surgically using B.P blade no 15 & 11. Compared with other surgical techniques, healing is quite faster in this method. Proper care should be maintained at the time of surgery to make sure no pigmented remnants are left on the denuded area. Complete epithelial healing occurs between 7-14 days. Depigmentation allows the denuded connective tissue to heal by secondary intention. Thus, new epithelium is formed without melanin pigmentation [31].

Bur Abrasion Method

Comparatively, bur abrasion method is simple and non-aggressive. This method involves de-epithelisation of pigmented areas of

the gingiva by using a high-speed rotary instrument after giving adequate amount of local anesthesia. In this technique, a diamond bur is used at high speeds to denude the epithelium with copious irrigation. Medium sized round bur is used instead of small burs because small burs might produce small pits rather than surface abrasion. Minimum pressure with feather light brushing strokes accompanied with copious saline irrigation should be used without holding the bur in one place to perceive excellent results. The speed of the instrument should always be controlled [32].

Electro-Surgery

According to oringer's exploding cell theory, it is forecasted that electrical energy leads to the molecular disintegration of melanin cells of the operated and adjacent sites [33]. In this technique, heat generated by transmission of high-frequency of electrical energy to the tissues leads to either cutting or coagulation of the tissue [34]. After giving adequate amount of local anesthesia to the patient, the desired diamond loop electrode is attached the the hand piece. The hand-piece is held in a pen-like fashion and the tip of the electrode is moved over to the pigmented area. By using this technique for gingival depigmentation, it favours bleeding control, tissue contouring and less scar tissue formation, lesser time in chair, haemorrhage and less discomfort to the patient [35]. Prolonged or repeated usage of current to tissues result in heat accumulation and undesired tissue destruction. Contact with periosteum or alveolar bone and vital teeth should be avoided while during the procedure [36].

Cryosurgery

It is the most accepted method for gingival depigmentation [37]. The tissue destruction is done by freezing with different materials such as liquid nitrogen at very low temperature. The cytoplasm of cells freezes, leading to denaturation of proteins and cell death. Local anesthesia or periodontal dressing is not required and no scar will be seen in this method. This technique is comparatively painless and has a quick healing [38]. The disadvantage of this technique is post-operative swelling and difficulty in controlling the penetration depth [39]. The after effects of cryosurgery include considerable swelling, accompanied by increased soft tissue deterioration. There is difficulty in depth control, and optimal duration of freezing is unknown, but prolonged freezing amplifies tissue destruction [40].

Lazery

It is considered as one of the effective techniques and as an optimal effect in gingival depigmentation. It is considered as safest treatment. Most commonly used lasers for gingival depigmentation are carbon dioxide lasers, neodymium: Yttrium, aluminum, and garnet and diode lasers [41]. Although it has better esthetic results, it requires larger space, it is quite expensive and also it requires sophisticated equipment. A one step laser treatment is required to remove all pigmented gingiva. In this technique, periodontal dressing is not required. The advantage of this technique is less pain, it is easy to handle, hemostasias, short treatment time, dry surgical field, decreased swelling, edema and scarring, minimal mechanical trauma, etc. [42]. The disadvantage of laser treatment is delayed wound healing, thermal damage, deep penetration and comparatively it cost high, harmful to eyes and skin, requires special trainings.

Radiosurgery

It is considered as the advanced form of electrosurgery. In this technique the soft tissues are removed with the help 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 [43]. Electrically generated thermal energy from the radiofrequency apparatus influences the molecular disintegration of melanin cells which is presented on the basal and suprabasal layer of the gingival epithelium. The latent heat of radiosurgery delays the development and migration of melanocytes, which makes more efficient method than conventional methods of depigmentation [44].

Chemical Method

Chemical gingival peeling

This method is done by destroying the overlying gingival epithelium by using chemical peeling agents such as, phenols, salicylic acid, glycolic acid, and trichloroacetic acid [45]. In this the most commonly used agents are phenols and alcohols. Based on their ability of penetration, chemical agents are classified into: Very superficial, superficial, medium depth and deep.

90% phenol and 95%alcohol

According to the study of 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 [46]. The growth of new gingiva was prolonged, and repigmentation developed in 3 patients. The rest 9 subjects to met with the same result within a short while. In addition to pain, it causes tissue necrosis. This treatment was not adapted to either clinician or the patient.

Ascorbic Acid

It has been used in the treatment of melanin pigmentation. Shimada et al. in 2009 examinations, the effects of ascorbic acid on melanin formation in B-16 mouse melanoma cells and 3-dimensional human skin models [47]. It was found that ascorbic acid remarkably inhibits the activity of tyrosinase in both cases as mentioned above. Moreover, a significant relative change in pigmentation was seen after four weeks with the application of ascorbic acid gel compared to the placebo [48].

Methods used to mask the gingival pigmentation

- a. Free gingival grafting
- b. Acellular dermal matrix allograft

Free Gingival Grafting

The free gingival grafting is done on the denuded bone for the treatment of the esthetic problem in patients suffering from severe gingival melanin pigmentation. In this technique, an unpigmented free gingival autograft harvested from the patient's palate is placed on the prepared recipient site [49]. The potential of autogenous epithelialized gingival grafts has been established for the management of physiologic gingival pigmentation. 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 [50]. Advantage of this technique is it has More esthetic results and Less recurrence rate.

Acellular Dermal Matrix Allograft

Novaes et al. reported the use of ADMA for elimination of the gingival pigmentation. This type of method is used to treat burn patients and patients with soft tissue defects. ADMA is used as a safe substitute for free gingival autograft in the treatment of gingival hyperpigmentation. DMA 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 [51]. Acellular

dermal matrix allograft is a sensitive technique it is expensive too. Advantage of this technique is it requires less surgical time as compared to free gingival graft.

Future Advancement

Gingival Repigmentation

In hyperpigmented gingiva the primary concern is a relapse or gingival repigmentation [52]. The reappearance of melanin pigment after a period of clinical depigmentation is called as repigmentation. As it depends on methodology and follow-up period, the duration of repigmentation mentioned in literature remains controversial from one technique to other. The factors that influence the duration of relapse are smoking, sun exposure and genetic determination of skin colour. However, the majority of the available literature has shown lower recurrence rate for cryosurgery and lasers [53].

Healing Factors after Depigmentation of Gingiva

• Healing after surgical depigmentation

After surgery it is necessary to cover the exposed lamina propria with periodontal packs for 7 to 10 days. After 6 weeks, the attached gingiva is regenerated by a scar. The newly formed gingiva will be clinically non-pigmented [54].

• Healing following cryosurgical depigmentation

Within two to three days, superficial necrosis becomes apparent and a whitish slough would be separated from the underlying tissue, which leaves a clean pink surface. In one to two weeks, the normal gingiva is formed. In 3-4 weeks, the keratinization process will be completed. In this treatment, no postoperative pain, haemorrhage, infection or scarring is seen in patients [55].

• Healing following depigmentation by laser

During laser treatment, the gingiva gets covered by a yellowish layer which can be easily removed by using a wet gauze. After 1-2 weeks re-epithelization will be completed. At fourth week, gingiva is seen similar to the normal untreated gingiva i.e., there will be complete absence of melanin pigmentation [56].

• Healing after electro surgery

After the surgery, clot formation occurs and the underlying tissue becomes acutely inflamed with some necrosis. Then this clot is replaced by granulation tissue. After 24 hours, there is an increase in the new connective tissue cells mainly angioblasts. By third day, numerous young fibroblasts also reach in the area. Highly vascular granulation tissue grows coronally creating a new marginal gingiva. Simultaneously, after 12-24 hours epithelial cells within the margins start to migrate over the granulation tissues which separate it from clot. Surface epithelialization is generally completed after 5-14 days [57].

• Healing following depigmentation by free gingival grafts

Immediately after the placement of the free gingival graft, a fibrin clot is formed between the graft and the underlying tissue. This medium transports nutrients from the recipient area to the connective tissue of the graft. Re-epithelization occurs during the second half of the first post-operative week with the cells that are originating from the lateral wound margins of the epithelial ridges within the graft itself. By third day, connective tissue proliferation begins and by the end of the first week a tenuous fibrous attachment occurs between the graft and the recipient. By 14th day, the epithelium is seen as a normal histologic thickness [58].

Conclusion

Facial appearance depends on many intraoral and extraoral factors. Gingal pigmentation is not a major problem but it affects facial

appearance. Aesthetics has become a very important and significant aspect of dentistry. Most importantly gingival pigmentation in the anterior aspect of the teeth is considered as significant esthetic problem. Most of the pigmentation is physiologic or pathologic but sometimes it can be a precursor of systemic diseases. Melanin hyperpigmentation commonly does not present a medical problem, but patients usually complain of dark gums as unesthetic. A wide range of treatment modality are founded for gingival pigmentation. Treatment of the pigmentation is determined either surgically or chemically.

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