

## Updates in diagnosis and management of Melasma

### Abstract:

Melasma is a chronic acquired skin disease that chiefly manifests as bilateral, hyperpigmented, irregularly molded macules on the cheeks, forehead, and .mandible

It is a broad-minded, macular, nonscaling hypermelanosis of sun-exposed skin, primarily on the face and dorsal forearms. It is typically associated with pregnancy or use of oral contraceptives or anticonvulsants (e.g., phenytoin [Dilantin]), or it could .be idiopathic

Melasma can be diagnosed rendering to the patient's typical clinical manifestations. Melasma can be further staged and captured using certain noninvasive detection techniques such as diascopy and Wood's lamp examination, which canister facilitate classification and documentation of the stage of melisma. Management is use of products with strong fragrances or an oily base or use of camouflaging proxies might not be acceptable to some patients. Additionally, certain patients might find the idea of applying skin care products several times a day or smooth once daily unappealing, .and clinicians should be aware of this before emerging their treatment plans

### Introduction

Pigmentary disorders, such as melasma and postinflammatory hyperpigmentation (PIH), involve an increase in melanin production in response to numerous factors. The hyperpigmentation that develops can have a profound influence on the quality of life in these patients, expressly when exposed areas, such as the face, are involved.(1) It is appraised that the disorders of pigmentation are the 11th most common condition seen by dermatologists, with about 24.7 million dermatology [visits made between 1994 and 2010 for the administration of dyschromias.(1,2)

A search of the National Ambulatory Medical Care Survey by Kang et al. establish that photoprotection was the tenth most common treatment decision prescribed to Asians and the sixth for African Americans as associated to the third most common in Caucasians for the treatment of dyschromia.(1) This incongruity highlights the lack of awareness regarding the use of photoprotection in convinced populations who are also more susceptible to these changes. In melanocompetent individuals, the higher melanin content and more responsive melanosomes prime to a greater risk of hyperpigmentation, which is often more prominent and lengthier lasting when compared to lighter-skinned individuals.(3,4) These issues, in combination with others, manifest as varying incidences of melasma in vulnerable individuals around the world: 1.8% in Ethiopia, 3.4% in Beirut, 8.8% in Dallas, TX, USA, and 10.1% in Peru.(5)

Melasma is a common, chronic, and recurrent disorder of hyperpigmentation arising from hyperfunctional melanocytes that deposit extreme sums of melanin in the epidermis and dermis. Melasma is predominantly mutual in women, especially those of reproductive age, and in body areas with high amounts of sun exposure, remarkably the face. Contributing factors involved in the pathogenesis of this condition include genetic influences, sun experience, sensitivity to hormones, pregnancy, and, in some cases, medications.

Melasma is a reformist, macular, nonscaling hypermelanosis of sun-exposed skin, primarily on the face and dorsal forearms. It is typically associated with pregnancy or use of oral contraceptives or anticonvulsants (e.g., phenytoin [Dilantin]), or it could be idiopathic. Melasma disproportionately disturbs women (9:1 ratio), as well as skin types IV to VI.(6) It is usually asymptomatic, but it could be cosmetically distressing to the patient. There are three typical designs of distribution: centrofacial (63%), malar (21%), and mandibular (16%).(6) It is usually, but not continuously, bilateral. Epidermal melasma tends to be light brown, improving under Wood Lamp examination. Dermal melasma seems grayish in color and is nonenhancing. Mixed types of melasma are dark brown with mutable enhancement. Dermal melasma is resistant to topical therapy. Dermatologic referral is suggested in these cases.(6)

Melasma is a chronic acquired skin disease that mainly demonstrates as, irregularly shaped macules on the cheeks, forehead, and mandible. The occurrence of melasma is as high as 30% in Asian women of childbearing age(7) Melasma voluntarily relapses and is difficult to cure. In recent years, much improvement has been made in the pathogenesis, clinical staging and classification, and clinical administration of melasma. Accordingly, the prior version of the agreement on melasma (2015) is herein revised by a panel of specialists from the Pigmentary Disorder Group, Combination of Traditional and Western Medicine Dermatology, Research Center for Vitiligo, Chinese Society of Dermatology, Working Collection on Pigmentary Disorders, and China Dermatologist Suggestion.(8)

Melasma is a common pigmentary disorder that demonstrates as symmetric hyperpigmented macules and patches on the face. It characteristically affects women of reproductive age with Fitzpatrick skin type IV-VI, though the disorder can occur in men also. Genetic predisposition, ultraviolet (UV) radiation experience, hormonal factors such as female sex hormones and thyroid ailment, pregnancy and .drugs like phenytoin are the recognized risk factors(9)

In recent times, there have been studies flinging light on other factors that could be involved in the pathogenesis of melasma. These embrace various vascular growth factors, genetic aspects, and the role of H19, inducible nitric oxide synthase (iNOS), and Wnt pathway modulator genes. Identifying these aspects could facilitate the .development of newer treatment selections for melisma(9)

Melasma is a pattern of acquired hyperpigmentation and one of the most mutual pigmentation disorders involving the face. Three types of melasma happen, as classified according to their distribution on the face, which contain centrofacial, malar, and mandibular patterns (10,11)Another arrangement of melasma according to the site of the pigment as epidermal, dermal, or mixed type

## **Epidemiology**

The exact prevalence of melasma is not known among the over-all population, including men and women. This could be due to underreporting by the exaggerated

patients due to its asymptomatic nature and since many patients choose to treat it with over-the-counter products rather than access with a dermatologist.(12) The global prevalence of melasma differs according to ethnicity, skin type, and intensity of sun exposure. It has been found to be more mutual in Hispanics, Asians, and African Americans than in Caucasian people.(13,14) Further, it is more common in personages with dark skin and Fitzpatrick skin types IV, V and VI.(15,16)

The majority of the studies reporting the occurrence of melasma are based on clinical samples rather than population samples. The occurrence of melasma among the general population is stated to be 1.8 percent in Ethiopia(17), 2.88 percent in Saudi Arabia(18), 3.4 percent in Lebanon(19)and 8.2 percent in United States.(20) South Asian countries have a relatively higher prevalence of melasma than in other countries, as seen in Nepal (6.8%) and China (13.61% )(21,22)

Overall, the prevalence of melasma in the general populace is approximately 1 percent, but it can be as high as 9 to 50 percent in high-risk populaces . Between 4 and 10 percent of individuals presenting to dermatology clinics in Central and South America may have melisma. Melasma usually gifts in women of reproductive age. The mean age of onset in two studies from Brazil was about 28 years . The mean age of onset in 140 cases from India was 37 years [6]. A survey of 324 women existence treated for melasma in nine countries informed a mean age of onset of 34 years . The Fitzpatrick phototype of a patient may influence the stage of onset. In a group of melasma patients from Brazil, the mean age of onset was 27 years in persons with type II skin, 28 years in type III, 30 years in type IV, and 35 years in type V . These results suggest that lighter skin may be a disposing factor due to its susceptibility to .photodamage

## **Etiology**

The three major pathogenic aspects of melasma are genetic susceptibility, sunlight exposure, and variations in sex hormone levels. Excessive melanin synthesis, angiogenesis,(23) skin barrier dysfunction, and inflammation at the lesional position .are also tangled in the pathogenesis of melasma

## Genetic predisposition

Melasma is more mutual in darker-skinned races with Fitzpatrick skin types III to V. About 40% of patients with a family history of melasma are more likely retort poorly to treatment.(24,25)

## Sunlight exposure

Long-wave ultraviolet light [ultraviolet A (UVA)], medium-wave ultraviolet light [ultraviolet B (UVB)], and blue light within sunlight right stimulate melanocytes to synthesize melanin. Moreover, ultraviolet light can harm the basement membrane, resulting in diffusion of melanin into the dermis, degeneration of elastic fibers, and skin photoaging. Ultraviolet light can also rouse fibroblasts, mast cells, and sebaceous gland cells to secrete melanogenesis issues such as hepatocyte growth factor and stem cell factor, which can activate tyrosinase and improve melanocyte function, thus cumulative melanin synthesis(26,27,28)

## Sex hormones

Pregnancy, oral contraceptives, and hormone auxiliary therapy can induce and exaggerate melasma in women.(24,25,29)

## Enlarged melanin synthesis

Various factors directly or indirectly disturb microphthalmia-associated transcription factor in melanocytes to stimulate melanocytes and increase melanin synthesis by up-regulating downstream tyrosinase, tyrosinase-related protein 1, and dopachrome .tautomerase

## Vascular issues

The number and diameter of minor blood vessels in the dermis and the expression of vascular endothelial growth factor and endothelin 1 are meaningfully higher in melasma lesions than in usual skin.(23)

## Inflammation

Up-regulated expression of Toll-like receptors 2 and 4 in melasma lesions encourages the release of prostaglandin E2 and stem cell factor, principal to an increase in melanogenesis. In addition, rises in other inflammatory factors such as interleukin 1 $\beta$ , interleukin 17, c-kit receptor, and cyclooxygenase 2 can also arouse melanogenesis.(30,31)

#### Skin barricade dysfunction

The abnormal appearance of keratin, cornified envelope protein, and acid ceramidase in melasma lesions can cause epidermal penetrability barrier dysfunction, resulting in improvement of ultraviolet light-induced melanin production via stimulation of the p53/pro-opiomelanocortin/tyrosinase-related protein 1 motioning pathway.(32,33)

Sleep disorders, use of cosmetics encompassing excessive mercury, heat radiation (eg, cooking),(34) and other sicknesses such as thyroid disease, female reproductive .system disease, and liver disease can also induce or aggravate melasma

#### Pathophysiology

The most important factor is exposure to sunlight. UV radioactivity induces production of alpha-melanocyte–stimulating hormone and corticotropin as well as interleukin 1 and endothelin 1, which donates to increased melanin production by intraepidermal melanocytes. Protracted UV exposure-induced dermal inflammation and fibroblast activation upregulate stem cell issues in the melasma dermis, resulting .in improved melanogenesis(35)

#### HISTOPATHOLOGY

The diagnosis of melasma is commonly made clinically. However, skin biopsy with histopathological inspection (HPE) can be done. The histopathology of melasma in men is similar to that in women.(36) In epidermal melasma, increased melanin seen in the basal and suprabasal layers, where dendritic melanocytes and melanophages are current in the dermal type. In an Indian study, HPE was done in 48.8 percent (20/41) of the male patients, and the findings exposed that epidermal melasma was the most common pattern (50%), trailed by mixed melasma (45%). Dermal melasma

was the least common (5%) Other topographies included solar elastosis in 17 subjects (85%), flattening of rete ridges in nine (45%) and continuing inflammatory penetrate in six (30%) male patients with no signal of basal layer degeneration.<sup>5</sup> In concordance with this study, two other studies also described epidermal melasma as the predominant histopathological type seen in men. The education by Jang et al<sup>43</sup> also found epidermal melasma and enlarged elastotic material in the lesional dermis compared to the nonlesional dermis was more mutual among male patients, however, the difference was not statistically important<sup>(35)</sup>.

## **Melasma in males**

Melasma is a mutual skin condition characterized by the presence of symmetrical, irregular, light to dark brown hyperpigmentation connecting sun-exposed areas, especially on the face. Although melasma can touch all races and both sexes, it is more frequently seen in women of child-bearing age and in dark-skinned individuals living in areas with powerful ultraviolet (UV) radiation.<sup>(13,44)</sup>Hyperpigmentation on exposed areas such as the face can be a cradle of cosmetic concern for patients, that can negatively impact quality of life (QOL). Melasma in women has been intentional in detail, but despite several similarities, there are convinced differences in clinical, etiological, and treatment aspects of melasma in men that still essential to be studied. Understanding characteristics of melasma exact to men will allow for better management of the disorder among male patients.Melasma classically presents as well-defined, symmetrical, brown-black hyperpigmented spots on sun-exposed areas of the skin. The clinical presentation of melasma in men is like to the presentation in women, except for a few subtle differences. The age of commencement of melasma in male patients is variable, ranging from 18 to 72 years, with the typical age of onset (.being 30.7 years.<sup>(45,46,47</sup>

## **DIAGNOSIS**

While the diagnosis of melasma is typically an important differential and might be confused with melasma.<sup>(37)</sup> A detailed medical history, comprehensive clinical examination of the skin, dermoscopy, and histopathology are cooperative in arriving .at the correct diagnosis

Melasma can be diagnosed else conferring to the patient's typical clinical manifestations. Melasma can be further theatrical and typed using certain noninvasive recognition techniques such as diascopy and Wood's lamp examination, which can facilitate classification and documentation of the stage of melisma

### **Differential diagnosis**

#### **Post-inflammatory pigmentation**

The skin lesions are light brown, purple-brown, or dark brown rendering to whether the inflammatory skin disease is acute or chronic. The lesions are limited to the inflamed site and have clear borders. They can be distinguished from melasma .according to the patient's history of inflammatory skin complaints

#### **Hori nevus**

In most patients, the age at onset is 20 to 30 years old. The clinical appearance is predominantly characterized by yellow-brown to slate-gray round, dispersed, and isolated spots that are bilaterally dispersed over the zygomatic and temporal districts. Under RCM, elongated, highly refractive dendritic melanocytes are scattered among the collagen fiber bundles in the insincere and middle layers of the dermis. Though, the melanin content is normal in the basal layer of the epidermis.(38)

#### **Nevus of Ota**

This nevus often occurs at birth or soon after birth and clinically manifests as a dark blue patch unilaterally dispersed over the zygomatic region, temporal area, and conjunctiva. Under RCM, the melanin gratified in the basal layer of the epidermis seems normal, and moderate numbers of highly refractive dendritic melanocytes or highly refractive melanocyte lumps of different figures are present in the middle dermis.(38)

#### **Melanosis**



Patients with melanosia have a history of long-term exposure to tar, long-term use of poor-quality cosmetics, or inflammatory skin disease. The scientific manifestations in the early stage are dermatitis-like variations such as erythema and desquamation. Reticulated or diffuse pigmentation accompanied by telangiectasia happens in the later stage. The lesion appears gray in color and often includes the face and neck; it may also have a widespread distribution. Under RCM, the epidermis–dermis junction is blurry. Other features include loss of some pigmented rings in the basal layer and the attendance of highly refractive pigmentophages and moderately refractive monocytes in the superficial dermis.(39)

**The differential diagnoses include:**

Actinic Lichen Planus

Acanthosis Nigricans

Discoid Lupus Erythematosus

Drug-Induced Photosensitivity

Exogenous Ochronosis

Frictional Melanosis

Mastocytosis

Nevi of Ito and Ota

Pigmented Contact Dermatitis

Poikiloderma of Civatte

Postinflammatory Hyperpigmentation(35)

**Prognosis**

Melasma has no related mortality or morbidity. No cases of malignant transformation or connotation with the increased risk of melanoma or other

malignancies have been informed. Patients with melasma actually are considered to have less threat for melanoma

The dermal pigment may take longer to resolution than the epidermal pigment because no effective therapy is capable of eliminating dermal pigment. However, treatment should not be withheld simply because of a preponderance of dermal pigment. The basis of the dermal pigment is the epidermis, and, if epidermal melanogenesis can be repressed for long periods, the dermal pigment will not replenish and will slowly resolve. Resistant cases or reappearances of melasma occur often and are certain if strict avoidance of sunlight is not severely heeded(35)

## **MANAGEMENT**

Many clinicians might undertake that their male patients are not as concerned about the appearance of their skin as women are and that they will be unwilling to follow a stringent skin care plan. However, clinicians should tolerate in mind that if a male patient is seeking management for a dermatological condition, such as melasma, then cosmesis might at least be of incomplete concern to him, and thus the patient might be very motivated to adhere to a prescribed treatment regimen. To inspire the greatest gradation of treatment adherence, clinicians should take into careful consideration each patient's individual needs when generating treatment regimens, as preferences and expectations might differ significantly among men and their female counterparts. For example, use of crops with strong fragrances or an oily base or use of camouflaging agents might not be suitable to some patients. Additionally, some patients might bargain the idea of applying skin care products several times a day or even once daily unappealing, and clinicians would be aware of this before developing their treatment plans. Additionally, patient therapy is an integral component of melasma management, and clinicians should instruct their patients on its causes, prevention and treatment methods, and reappearance rates(40)

Sun avoidance is the most significant part of melasma treatment, both for current improvement and future prevention of reappearance. The use of broad spectrum sunscreens (UVA and UVB) along with an inorganic sunscreen (physical block) like

zinc oxide or titanium dioxide with a minimum sun protection aspect of 15 should be encouraged.(41) Regardless of sex, physicians should guidance all patients regarding protection from sun exposure, with emphasis on optimum and regular application of sunscreen and use of hats and clothing that lump the sun. Physicians must be extra attentive to male patients, who have exposed to be less successful in adhering to sunscreen submission guidelines.(42)

Melasma can be slow to retort to treatment, particularly if the condition has been :present for many years.(43) Poor prognostic issues for melasma treatment include

Fitzpatrick skin types III–V

genetic and familial predisposition

long-standing disease  $\geq 2$  years persisting despite treatment

(history of procedural interventions (eg lasers, microneedling

history of management by  $\geq 2$  physicians (possibly suggesting long duration and (recalcitrant disease

long-term self-treatment with topical steroids

ochronosis from hydroquinone use – either long term or great strength

.mixed-type melasma

## **CONCLUSION**

Melasma is a complex disorder and various aspects are involved in its pathogenesis, identification of which will help us in developing better management options with more efficacy, less side effects and longer periods of reduction. Newer compounds, especially botanical extracts and device-based managements are being advanced and add to the list of options accessible for management. However, more randomized controlled trials are desirable to evaluate their efficacy compared to the well-known treatments obtainable(9)

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