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REVIEW ARTICLE

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An Overview of Diagnosis and Treatment of Melasma

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Abstract

Melasma is a persistent hyperpigmentation condition that is most commonly seen in middle-aged women. The malar, central, and mandibular parts of the face are most affected by this condition, Factors that contribute to melasma's etiology are still a mystery. Multifactorial causes are now widely accepted in these cases. In order to diagnose melasma and its variants, distinguish them from other pigmented illnesses, and keep track of treatment progress and side effects, several diagnostic tools are employed. The limited response and high recurrence rate make treating melasma difficult. Melasma can be treated with a variety of methods, including topical medicines, laser treatment, and injections. The objective of this article is to offer a concise overview of melasma diagnosis and management.Melasma treatment is a cosmetic challenge. Chemical, physical, and laser therapy are all options. (**International Journal of Biomedicine. 2022;12(2):199-203.**)

Key Words: hyperpigmentation • melasma • diagnosis • dermoscopy • treatment

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Introduction

Cosmetically, facial pigmentations are essential, and they have a significant influence on patients' quality of life. Melasma is the most frequently acquired pigmented condition, characterized by homogeneous hyperpigmented patches with asymmetric contours on the face, most typically in middle-aged women with dark skin. The use of oral contraceptives, exposure to the sun, and pregnancy are all aggravating factors.[1-3] Hydroquinone, retinoid, azelaic acid, and glycolic acid are some of the topical medicines now often utilized to treat hyperpigmentation. Irritation is one of these chemicals' most prevalent side effects.[4-6]

Causes of Facial Hyperpigmentation

Frequently, the reasons for facial hyperpigmentation are unclear. Genetic and racial variables both have a role. Increased pigmentation is more common in those with dark skin. UV exposure, endocrine factors, pharmaceuticals (e.g., phenytoin, phototoxic medications), and other diseases (e.g., autoimmune thyroid *diseases*) can all exacerbate melasma, and are also implicated in other melanoses to a lesser extent. Cosmetics and Addisonian pigmentation may contribute to the development of face melanosis.[6]

Hypermelanos is due to genetic and environmental causes:

- •Ephelides (freckles), Peutz–Jeghers syndrome, Naevus of Ota
- •Lentigines, juvenile acanthosis nigricans, café au lait macules, melasma

Acquired hypermelanosis:

- •Metabolic: Liver disease, porphyria and haemochromatosis
- •Endocrine: Melasma and Addison's disease
- •Chemical: Minocycline pigmentation, Berloque dermatitis
- •Post inflammatory hyperpigmentation: Lichen planus, erythema dyschromicum
- •Tumors: Acanthosis nigricans with adenocarcinoma, malignant and metastatic melanoma.[7]

Diagnostic Approach to Facial Hyperpigmentation

History and physical examination

The patient's history and physical examination provide insight into the etiology of hyperpigmentation. The history should include information on the lesion's start, as certain illnesses (e.g., neurofibromatosis) are present from birth, whereas others (e.g., ephelides) emerge during infancy or (e.g., melasma) occur during pregnancy. The presence of systemic symptoms may imply hyperthyroidism or Addison's disease. A study of drug usage, dietary supplements, and exposure to plants and UV light can assist in determining if hyperpigmentation is a side effect of a medicine or a phototoxic response. Neurofibromatosis, ephelides, and lentigines can be diagnosed based on the size and number of lesions. The border, color, and nature of a lesion aid in differentiating melanoma from benign lesions, whereas the pattern of skin changes assists in identifying melasma and acanthosis nigricans[8] (Table 1).

Clinical Features of Melasma

Patches ranging from light to dark brown with uneven boundaries typically occur on the facial area.(Figures 1-7) Melanosis occurs in three typical patterns:

(1) Centrofacial (the most frequent), including the forehead, cheeks, nose, upper lip (save the philtrum and nasolabial creases), and chin.

(2) Malar, which affects the cheekbones and nose.

(3) Mandibular, running parallel to the jaw.

The extensor portion of the forearms and the middle area of upper chest are less often used locations.[6,9]

Wood's light

A Wood's lamp may aid in diagnosing certain conditions. When examined with a Wood's light, epidermal melanosis intensifies and becomes more confined.[10] Lamp evaluations are typically reserved for Fitzpatrick skin types I–IV. They are ineffective on those with skin type VI and are only marginally effective on persons with skin type V. Generally, the Wood's light is incapable of distinguishing between epidermal and dermal pigments in mixed hypermelanosis in all skin types.[3,6,9]

Dermoscopy

Dermoscopy is a systematic approach for evaluating the colors and structural components of the skin.[11] There have been developed scoring methods that evaluate asymmetry, border, color, and dermatoscopic features. The development of computerized image analysis is assisting in differentiating benign melanocytic lesions from melanoma.[12-15] Dermoscopy has exceeded conventional evaluation methods in distinguishing melasma from other sources of face hyperpigmentation, measuring its depth, monitoring treatment efficacy, and detecting early consequences, such as atrophy and telangiectasia.[16,17]

Histopathology

A biopsy can conclusively determine the site of the hypermelanosis; however, it is seldom necessary for

Table 1.

Clinical features of often occurring face hyperpigmented disorders.[2,8]

	Color	Age of onset	Medication related	Sun exposure
Melasma	Brown cheek, forehead and upper lip macular lesions	Adulthood	Pregnancy, oral contraceptive pills	Enhanced by exposure to sunlight
Post inflammatory hyperpigmentation	Macular brown discoloration at the location of inflammation	Any age	Irrelevant	Injuries caused by physical or chemical agents or dermatoses
Ephelides	Multiple small red, tan, or brown macules, on sun-exposed regions	Early life	Irrelevant	Increased number and pigmentation
Lentigines	Multiple small tan, brown, or black macules on sun-exposed parts	Early life	Irrelevant	Enhanced in sun exposed regions
Photoallergic/phototoxic reaction	Diffuse inflammation followed by hyperpigmentation in sun-exposed regions	Any age	Irrelevant	Sun exposure coupled with the problematic medications
Café au lait macules	Multiple small macules with smooth or asymmetrical, but distinct edges	Congenital or during childhood	Irrelevant	Uninvolved
Hemochromatosis	Hyperpigmentation in a diffuse slate-gray or bronze hue	Adulthood	Irrelevant	Uninvolved
Poikiloderma of Civatte	Reticulate dark pigmentation Lateral and low neck	Adulthood	cosmetics, hormones	Increased by sun exposure
Erythromelanosis follicularis faciei et colli	Affecting follicles Pre auricular, maxillary areas. Symmetric pigmentation	Adulthood	Irrelevant	Uninvolved

this purpose; rather, it is used to ascertain an origin that is unknown.[18-20] Three types of hypermelanosis are defined based on the melanin distribution (as determined by lesion color, its enhancement under Wood's light, and pathology):

1. Brown hypermelanosis: A condition in which the basal and suprabasal layers have an excess of melanin, and the pigmentation is exacerbated when exposed to the Wood's lamp.
2. Blue hypermelanosis: A condition in which the dermis has an excess of melanin and the pigmentation is not exacerbated by Wood's light.
3. Mixed hypermelanosis: Melanin levels in the epidermis and dermis are elevated.[21]

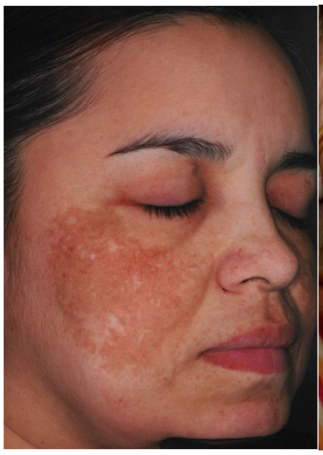

Fig.1. *Melasma on the cheek.* [6]

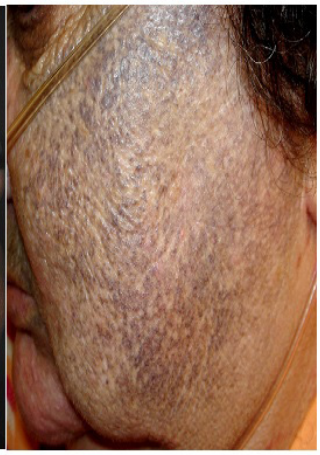

Fig.2. *Minocycline hyperpigmentation* [6]

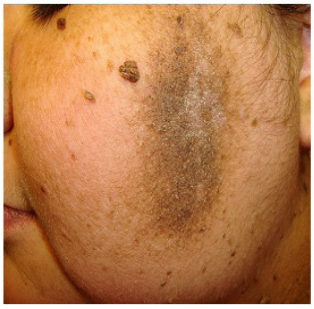

Fig.3. *Acanthosis nigricans*[6]

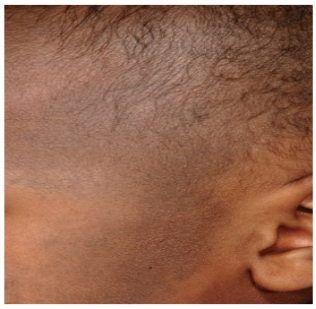

Fig.4. *Nevus of Ota*[22]

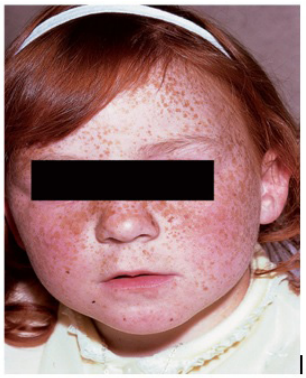

Fig.5. *Freckles* [22]

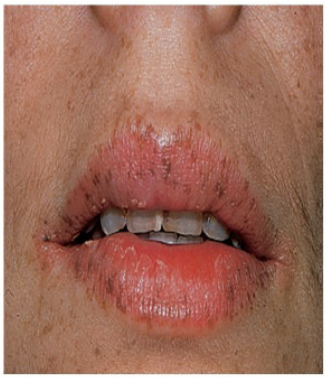

Fig.6. *Multiple lentigines (Peutz-Jeghers syndrome)*[9]

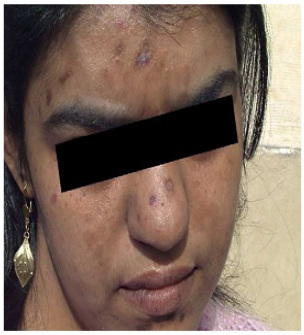

Fig.7. *Post-inflammatory hyperpigmentation* [23]

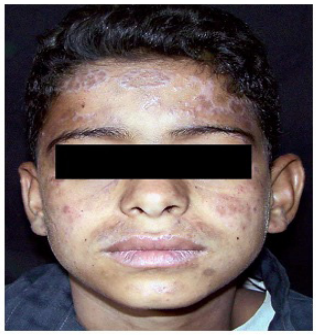

Fig.8. *Actinic lichen planus* [1]

Treatment of Facial Hyperpigmentation

Topical medications that influence pigment synthesis, such as broad-spectrum sunblock and camouflage, are typically used as first-line treatment. Chemical peels are widely used as a second-line treatment; however, they should be used with caution in those with darker skin. While laser and light therapy are possibly beneficial for individuals who have failed to respond to conventional treatment methods, they also entail a high risk of aggravating the condition.[6]

General Instructions

All patients should avoid prolonged exposure to the sun. They should dress appropriately and protect themselves with broad-spectrum sunscreen. If the use of drugs or cosmetics has resulted in facial hyperpigmentation, they must be discontinued. If melasma develops during pregnancy, it is important to apply sunscreen, with the condition improving by the end of gestation.[2]

Sunscreens

Patients with facial hyperpigmentation should apply a broad-spectrum UVA and UVB-protective sunscreen with an SPF not less than 30 and a physical block, such as titanium dioxide or zinc oxide, and should repeat regularly. Additionally, patients should be advised to wear protective caps and clothes when outside and to avoid sunlight if feasible.[18]

Treatment of Melasma

Hydroquinone 3% or 4%, glycolic acid 10% peel, azelaic acid 20% cream, and retinoids (e.g., tretinoin 0.05% or 0.1 % cream; adapalene 0.1% or 0.3 % gel) all exhibit some efficacy.[24,25] Combination treatments are more successful at bleaching than monotherapies, and usually physicians begin therapy with one of these formulae applied once daily (at night), followed by maintenance with 2% hydroquinone[26] (Table 2).

Numerous modest studies indicate that laser treatment or a combination of strong pulsed-light therapy and hydroquinone with sunscreen may be useful in treating dermal or refractory/mixed-type melasmas.[27] Melasma caused by pregnancy or oral contraceptive usage often resolves several months after birth or discontinuation of medication; therefore, cautious waiting should be urged wherever possible.[28]

Table 2.

The most often used depigmenting formulas in melisma.[26]

Name of Formula	Active ingredients
Kligman's formula	Hydroquinone 5% Tretinoin 0.05%-0.1% Dexamethasone or betamethasone valerate 0.1% in hydroalcoholic base in the form a cream or an ointment
Pathak's formula	Hydroquinone 2% Tretinoin 0.05%-0.1% in hydroalcoholic base in a cream or ointment base
Westhorf's formula	N-acetylcysteine 3% Hydroquinone 2% Hydrocortisone 1% in ointment base

According to Lee et al.,[29] topical trans-4-tranexamic acid, a plasmin inhibitor, effectively inhibits UV-induced pigmentation. This procedure involves injecting 0.05ml–0.1ml of highly diluted tranexamic acid or a single product intradermally or subcutaneously into areas of the body with medical or cosmetic issues. It may be able to treat dermal-type melasma in addition to mixed-type melasma by injecting tranexamic acid intradermally.[29,30]

Conclusion

Melasma treatment is a cosmetic challenge. Chemical, physical, and laser therapy are all options. Melasma due to pregnancy or contraception usage may disappear spontaneously over time in patients with light skin.

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Competing Interests

The authors declare that they have no competing interests.

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