Practice and Educational Gaps in Abnormal Pigmentation

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KEYWORDS

• Dyschromia • Postinflammatory hyperpigmentation • Melasma • Vitiligo • Treatment • Hyperpigmentation • Hypopigmentation • Education

KEY POINTS

- Dyschromia is one of the most common diagnoses in dermatology, yet there are significant educational and practice gaps in this area.
- The main educational and practice gaps in melasma and postinflammatory hyperpigmentation include alternative or adjunct therapies to hydroquinone and lack of exposure to a variety of skin tones.
- The main educational and practice gaps in vitiligo include underutilization and customization of phototherapy, use of agents for stabilization of progressive disease, and surgical treatment of vitiligo.
- Residency programs need to address educational gaps in disorders of pigmentation to prevent educational gaps from becoming practice gaps.

INTRODUCTION

Dyschromia refers to abnormal pigmentation of the skin or nails, including numerous conditions that cause both hyperpigmentation and hypopigmentation. It is one of the most common diagnoses in dermatology, especially in individuals with skin of color. From 1993 to 2010, there were approximately 24.7 million office visits made to dermatology practices for the management of dyschromia.[1] Despite the high prevalence of dyschromia in the general population, there is a disconnect between the occurrence of this condition and the resources directed toward its management. As such, it is important to define the standard of care for abnormal pigmentation, more specifically, postinflammatory hyperpigmentation (PIH), melasma, and vitiligo, as well as address clinical and educational gaps that may lead to substandard practices.

GOLD STANDARD

Melasma/Postinflammatory Hyperpigmentation

Melasma is a condition characterized by irregularly shaped hyperpigmented patches most commonly located in a centrofacial, malar, or mandibular pattern. Depending on the location of pigment deposition, melasma can be characterized as epidermal, dermal, or mixed type, with worse prognosis associated with dermal pigmentation. Pigment location can be determined by examination under a Wood lamp, with epidermal pigment

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becoming accentuated under illumination. Melasma is often associated with UV exposure, genetic predisposition, and hormonal changes, such as pregnancy or the initiation of oral contraceptive pills (OCPs). PIH occurs when cutaneous inflammation leads to increased pigmentation in the dermis or epidermis. Several conditions are capable of inducing PIH, such as atopic dermatitis, acne, medication reactions, and procedure-related inflammation. Hyperpigmentation develops in the same area as preceding inflammation and occurs more commonly in skin types III–VI.[2]

Evaluation of melasma is performed using the Melasma Area and Severity Index, or MASI, score. This evaluation involves adding the severity ratings for darkness and homogeneity, which are then multiplied by the numerical value given for 4 facial areas, including the forehead, chin, and left and right malar regions, and the percentage involvement of each area. A modified MASI score, which removes the homogeneity parameter, has subsequently been created. Clinical photography is also important in following this condition over time.[3] The MelasQoL is a scale that assesses the impact of melasma on quality of life (QoL).[4] The only validated outcome measure for evaluation of PIH is the Postacne Hyperpigmentation Index. Scoring is based on median lesion size, intensity, and number of lesions.[5] A PIH Investigator's Global Assessment scale has also been developed at Henry Ford Hospital to characterize the intensity of hyperpigmentation.[6] Serial photography is also important in monitoring progression and treatment response. Currently, no validated quality-of-life measure specific to PIH exists.

The first step in treatment of both melasma and PIH is the avoidance of causative factors. In patients with melasma, those currently taking OCPs should switch to an alternate form of birth control.[7] With regards to PIH, treatment of the underlying condition causing cutaneous inflammation is imperative. In both instances, photoprotection is critical because UV exposure can exacerbate hyperpigmentation.[3] Although most broad-spectrum chemical sunscreens provide protection against both UV-A and UV-B wavelengths, they are not effective against visible light. Visible light has been shown to induce production of reactive oxygen species (ROS) in addition to proinflammatory cytokines, and matrix metalloproteinase 1.[8] A study performed by Mahmoud and colleagues[9] showed that exposure to visible light induced pigmentation in skin types IV–VI, which was darker and more sustained than pigmentation caused by UV exposure. Physical sunscreens, such as iron oxide, provide coverage against the visible light spectrum and are often present in commercially available make-up products. Use of tinted sunscreen containing iron oxide in patients with melasma showed a smaller increase in MASI scores over time compared with patients using the same sunscreen without iron oxide.[10] Another study comparing the use of a visible light-UV sunscreen and hydroquinone to UV-only sunscreen with hydroquinone showed greater increases in MASI scores with the UV-only sunscreen and hydroquinone combination.[11] There is a paucity of literature regarding the effect of physical sunscreens in PIH, which is an area that needs to be addressed in future studies.

With respect to topical therapies, hydroquinone is the mainstay of treatment. Inhibition of tyrosinase, the enzyme responsible for converting dihydrophenylalanine to melanin, is its main mechanism of action. It can be used alone, but is more effective when used in combination with a retinoid and corticosteroid, which is known as Kligman's formula.[7] However, caution is advised because use of hydroquinone has been associated with ochronosis, a blackish discoloration of the skin that is difficult to reverse.[12] Currently, there are several other lightening agents that can be used as an alternative or adjunct to hydroquinone. Examples include soy, ellagic acid, niacinamide, licorice extract, kojic acid, vitamin C, and arbutin (**Table 1**). These compounds have been shown to cause statistically significant reductions in pigmentation. Most of these alternative therapies are available as over-the-counter products.[13,14]

Currently, there are few oral therapies being used for the treatment of melasma or PIH. Most commonly used in Southeast Asia, tranexamic acid is a plasmin inhibitor that has been used to treat melasma successfully. Multiple studies have shown that oral tranexamic acid decreases MASI scores when used in both monotherapy and combination therapy. Nausea, diarrhea, menstrual irregularities, headaches, and back pain are the most commonly reported side effects. No increased risk of thromboembolic events has been noted.[4,15] This option is possible for the patient with recalcitrant melasma who has not responded to conventional therapies. Studies on the use of polypodium leucotomos extract (PLE), an oral antioxidant, for melasma have also shown improvement in MASI scores when used in conjunction with photoprotection. It has been postulated that PLE would be beneficial in treatment of PIH due to its anti-inflammatory effects, but no specific studies have been performed to investigate this theory.[16]

Chemical peels as well as light-based and laser therapy have been used in the management of melasma and PIH with varying success. Extreme caution must be used when using these modalities