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Management of Hyperpigmentation in Darker Racial Ethnic Groups

Pearl E. Grimes, MD

Dyschromias, in particular hyperpigmentation, are major issues of concern for people of color. Pigmentary disorders such as melasma and postinflammatory hyperpigmentation (PIH) can cause psychological and emotional distress and can pose a negative impact on a person's health-related quality of life. The precise etiology of these conditions is unknown. Therapies for melasma and PIH target various points during the cycle of melanin production and degradation. Therapies for these conditions include topical agents and resurfacing procedures. Hydroquinone remains the gold standard of topical agents. Other efficacious agents include kojic acid, azelaic acid, mequinol, and retinoids. Cosmeceutical agents include licorice, arbutin, soy, *N*-acetyl glucosamine, and niacinamide. Resurfacing procedures that have been used to treat melasma and PIH include chemical peels, microdermabrasion, lasers, and intense pulsed light. These procedures are best used in combination with topical bleaching agents. Given the propensity of darker skin to hyperpigment, resurfacing procedures should be used with care and caution. Maximal results are best achieved with repetitive, superficial, resurfacing modalities. In addition, ultraviolet protective measures such as broad-spectrum sunscreens are fundamental to the successful management of these conditions.

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Multiple studies now document the increased frequency of disorders characterized by hyperpigmentation in darker racial ethnic groups.[1-3] The melanocytes of darker-skinned individuals exhibit labile, exaggerated responses to cutaneous injury. In a study assessing the frequency of common dermatoses in 2000 black patients, pigmentary disorders excluding vitiligo were the third most-common reason for seeking dermatologic treatment. Such conditions included postinflammatory hyperpigmentation (PIH) and melasma.[1] Similarly, in a series of Hispanic patients, pigmentary disorders were the third most common-reason for seeking treatment.[2] Alexis et al[3] reported the most common diagnoses for 1412 patient visits at an urban dermatology faculty practice. Dyschromias were the most common diagnosis in black patients. It is indeed evident that dyschromias are major issues of concern for people of color. This article will review the treatment of melasma and postinflammatory hyperpigmentation, the 2 most common disorders of hyperpigmentation in darker racial ethnic groups.

Vitiligo and Pigmentation Institute of Southern California, and Clinical Professor, Division of Dermatology, Department of Medicine, David Geffen School of Medicine, University of California, Los Angeles, Los Angeles, CA.

Address reprint requests to Pearl E. Grimes, MD, 5670 Wilshire Blvd., Suite 650, Los Angeles, CA 90036. E-mail: pegrimesmd@earthlink.net.

Quality of Life

Pigmentary disorders, such as melasma and PIH, can cause psychological and emotional distress and can pose a significant negative impact on a person's health-related quality of life.[4,5] In a prospective cohort study on the prevalence of pigmentary disorders and their impact on quality of life, 47.3% of patients admitted being self-conscious about their skin to some degree, 21.8% thought others focused on their skin, 32.7% reported feeling unattractive because of their skin, 32.7% put effort into hiding pigment changes, and 23.6% thought their skin affected their activities.[5] Disfiguring facial lesions, in particular, can cause decreased social functioning, lowered productivity at work, and reduced self-esteem.[6,7]

Using a specially designed index, the Melasma Quality of Life Scale, to measure health-related quality of life in patients with melasma, Balkrishnan et al[8] found that the 3 domains most affected by the condition were social life, recreation and leisure, and emotional well-being. Cestari et al[9] showed that 65% of patients were bothered all the time or most of the time, 55% were frustrated, and 57% were embarrassed by the condition; 42% said it had an influence on interpersonal relationships. Forty-three percent of patients also felt unattractive as the result of their skin condition. However, after

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treatment, there was a significant reduction of the Melasma Quality of Life Scale. Hence, the need for effective treatments for pigmentary disorders is of importance based on the prevalence and effect on quality of life.

Pathogenesis of Melasma

Melasma is an acquired symmetric hypermelanosis characterized by irregular light-brown to gray-brown patches involving the face, particularly the cheeks, nose, forehead, chin, and upper lip. Lesions may also appear on other sun-exposed areas, such as the back, chest, and arms.

It predominantly affects women, with men comprising only 10% of all cases. The disease affects all racial groups but is most prevalent in darker-skinned individuals (Fitzpatrick's skin types IV–VI), such as Hispanics, East Asians, Southeast Asians, and blacks who live in areas of intense ultraviolet (UV) radiation. The precise cause of melasma is unknown; however, multiple factors have been associated with the etiopathogenesis of this condition.[10] These factors include genetic influences, exposure to UV radiation, pregnancy, use of oral contraceptives, hormonal replacement therapies for menopause, thyroid dysfunction, use of cosmetics, and use of phototoxic and antiseizure drugs. Of these, it is thought that the most important are genetic influences and exposure to UV radiation. Several studies have reported abnormal circulating hormone levels in patients with melasma.[11,12] Data suggest that the increased expression of stem cell factor in the dermis and its receptor c-kit in the epidermis play an important role in the mechanism of hyperpigmentation in melasma.[13,14]

Biopsies of the involved and uninvolved skin of patients suggest that there is no quantitative increase in melanocytes in the hyperpigmented areas. In contrast, the melanocytes in the affected area are larger, intensely staining cells with prominent dendrites compared to normal skin.[15]

Pathogenesis of PIH

PIH is characterized by an acquired increase in pigmentation secondary to an inflammatory process. Excess pigment is deposited in the epidermis or both the epidermis and dermis. In darker skin types, common causes of PIH include acne vulgaris, atopic dermatitis, allergic and/or irritant contact dermatitis, trauma, psoriasis, lichen planus, and drug eruptions.

PIH is caused by an increase in melanin production or an abnormal distribution of melanin pigment. After cutaneous trauma or inflammation, melanocytes can react via production of normal, increased, or decreased melanin. Although the precise pathogenesis is unknown, it is thought that hyperpigmentation results from cytokines, inflammatory mediators, and reactive oxygen species. In vitro studies have shown that LTC_4, LTD_4, PGE_2, and TxB_2 stimulate human melanocyte enlargement and dendrocyte proliferation. LTC_4 also significantly increases tyrosinase activity and mitotic activity in cultured melanocytes.[16] Studies also suggest that direct stimulation of melanocytes by inflammatory mediators, such as il-α, stem cell factor, and endothelin 1 can cause hyperpigmentation. In addition, reactive oxygen species, nitric oxide, and superoxide from damaged skin or from inflammatory cells stimulate hyperpigmentation.[17]

Depigmenting Agents for Melasma and PIH

Multiple pathways and proteins have been identified as targets to control or decrease pigmentation. Ideal hypopigmenting agents have a strong and selected effect on melanocytes and carry minimal or no acute or long-term risks of side effects. Such agents can act at various points during the cycle of melanin production and degradation (Table 1).[18]

These pathways include:

- the transcription and activation of tyrosinase, tyrosinase-related protein-1 (TRP-1), tyrosinase-related protein-2 (TRP-2), and/or peroxidase;
- the uptake and distribution of melanosomes in recipient keratinocytes; and
- melanin and melanosome degradation and turnover of "pigmented" keratinocytes.

Hydroquinone (HQ)

In 1961, Spencer evaluated the efficacy of HQ, 1.5% and 2%, in 98 subjects with hyperpigmentation. Improvement was noted in 45% of subjects and none experienced adverse events. Since this seminal study, HQ has been the gold standard for treatment of hyperpigmentation for almost 50 years.

Table 1 Depigmenting Agents and Their Mechanism of Action

Mechanism of Action	Depigmenting Agent
Tyrosinase transcription and glycosylation	Tretinoin, glucosamine, retinol, *N*-acetyl glucosamine, retinaldehyde
Tyrosinase inhibition	Hydroquinone, 4-hydroxy-anisole, arbutin aloesin, azelaic acid, kojic acid,
Tyrosinase degradation	ellagic acid, resveratrol, Oxyresveratrol
Inhibition of melanosome transfer	Linoleic acid
Anti-inflammatory	Niacinamide, soybean/milk extracts
Reactive oxygen species scavengers	Topical steroids, glycyrrhetinic acid
Increased epidermal turnover	Vitamin C, vitamin E, thioctic acid
	Retinoids, lactic acid, glycolic acid, salicylic acid, Liquirtin

Modified from Briganti et al.[18]

HQ blocks the conversion of DOPA to melanin by inhibiting tyrosinase. It may also inhibit RNA and DNA synthesis, degrade melanosomes, and destroy melanocytes.[19,20] HQ is commonly used at concentrations of 2-4%. Clinical studies report good-to-excellent responses induced by 2% HQ preparations in 14-70% of treated patients.[21,22] HQ preparations of 3-5% are also very effective, but some irritation may occur.[23]

Combination formulations, containing HQ and other agents, particularly retinoids and glycolic acid, offer maximal efficacy.[23-27] Such products are used to enhance the effectiveness of a single depigmenting agent and reduce the risk of side effects. The Kligman or modified Kligman formula has emerged as the most popular combination formula.

The original Kligman formula contained 5% HQ, 0.1% tretinoin, and 0.1% dexamethasone. Albeit effective, it had 2 main disadvantages: the use of a high concentration of tretinoin and a potent fluorinated steroid, ie, dexamethasone. To reduce side effects and irritation caused by the steroid, various other combination therapies have been proposed for the treatment of melasma.[9,28] A fixed, triple combination therapy, containing 4% HQ, 0.05% tretinoin, and 0.01% fluocinolone acetonide (Tri-Luma, Galderma Laboratories, Fort Worth, TX), was developed. This combination was shown to be safe and effective in the treatment of melasma for up to 8 weeks.[26] In this multicenter, randomized, investigator-blind study, 26.1% of patients experienced complete clearing of melasma by week 8; 75% reduction in melasma/hyperpigmentation was observed in >70% of patients. Many publications have subsequently described the safety and efficacy of the triple combination cream in >2000 patients with melasma, particularly in darker-skinned racial ethnic groups. Some patients were treated for at least 12 months.[29-31] Overall, the results of these studies indicate that the triple combination cream is efficacious in treating melasma across a range of patients, including whites, Hispanics, blacks, Asians, American Indians, and Pacific Islanders, and exhibits a safe profile with low potential for adverse events.[32]

Other combination formulas include Pathak's and Westerhof's formula. Pathak's formula contains 2% HQ and 0.05-0.1% tretinoin; the use of steroids is avoided and only used if irritation from HQ or tretinoin is observed.[9] Westerhof's formula consists of 4.7% *N*-acetylcysteine, 2% HQ, and 0.1% triamcinolone acetonide. This combination allows significant bleaching within 4-8 weeks.[33]

A new formulation of HQ 4% with retinol, 0.15% entrapped in microsponge reservoirs was developed for the treatment of melasma and PIH (EpiQuin Micro, SkinMedica, Inc., Carlsbad, CA). Microsponges were used to release HQ gradually to prolong exposure to treatment and to minimize skin irritation. The safety and efficacy of this product was evaluated in a 12-week open-label study. A total of 28 patients were enrolled. Study end points included disease severity, pigmentation intensity, lesion area, and colorimetry assessments. The microentrapped HQ 4%/retinol, 0.15% formulation produced improvement at all study end points. Improvement in disease severity and pigmentation intensity was statistically significant at weeks 4, 8, and 12 compared with baseline ($P < 0.001$) (Fig. 1). Lesion area and colorimetry measurements also were significantly improved at each visit ($P < 0.001$).[34] Side effects were minimal throughout the study. A subsquent study showed that this novel nonsteroidal product showed comparable results to triple combination bleaching in a randomized split face investigation.[35]

Figure 1 Patient with melasma (A) before and (B) after 12 weeks of treatment with hydroquinone, 4% and 0.15% retinol.

Complications and Controversies With the Use of HQ

Side effects of HQ include acute and long-term effects. Acute complications include irritant or allergic contact dermatitis, PIH, and postinflammatory hypopigmentation. Of these, irritant reactions are the most common.[9,25,26,30,34,36] Review of the literature suggests that monotherapy hydroquinone agents cause irritant reactions in 0-70% of patients. In combination therapy, the incidence increases to 10-100%. There are infrequent reports of allergic contact sensitization to hydroquinone.

Long-term adverse events related to exposure to HQ are of greater concern. These complications include ochronosis, nail discoloration, conjunctival melanosis, and corneal degeneration.[37,38] Ochronosis is the most common long-term complication related to long-term use of HQ (Fig. 2).[39-45] The condition was initially described by Findlay et al[39] among South African Bantu women who applied high concentrations of HQ for many years. Cutaneous ochronosis is a common complication of HQ use in Africa. Most affected individuals are women. Clinically, ochronosis is characterized by asymptomatic hyperpigmentation, erythema, papules, papulonodules, and gray-blue colloid milia on sun-exposed areas of the skin. Despite the extensive use of HQ formulations in the United States, ochronosis is uncommon. Most reported cases in the United States have occurred with the use of 2% HQ.

Factors accounting for the disparity in the frequency of ochronosis in the United States and Africa include the routine use of sunscreens and the absence of resorcinol in formulations in the United States.[43] In addition, in contrast to Africa, there are few hydroethanolic formulations marketed in the United States. Such formulations may permit enhanced absorption of HQ. HQ has been banned in some countries, including over-the-counter dispensed formulations of 2% HQ by the European Cosmetic Product Regulation. In August of 2006 the Food and Drug Administration proposed a ban on over-the-counter HQ and considered requiring new drug applications for 4% formulations as the result of concerns regarding ochronosis and possible carcinogenicity. Some animal studies have shown an increase in cancers which are species and sex specific.[46,47] However, there are no human studies documenting an increased incidence of skin cancer or internal malignancies in users of HQ.

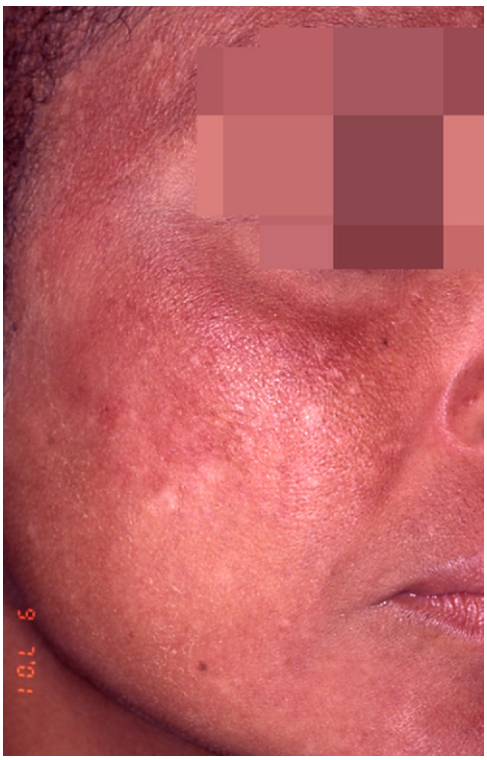

Figure 2 Patient with hyperpigmented patches and papules of the cheeks and forehead. Biopsies were consistent with ochronosis.

Mequinol

Mequinol, 4-hydroxyanisole, a hydroquinone derivative, is an alternative to HQ. In a study by Fleischer et al,[48] mequinol was shown to be effective in improving the appearance of solar lentigines and other hyperpigmented lesions in a 2% formulation in combination with 0.01% tretinoin. A study also suggests that mequinol, 2% and tretinoin, 0.01% solution is a promising alternative for PIH.[49]

Other HQ Derivatives

Other HQ derivatives include mono benzyl ether of HQ, 4-methoxy phenol, 4-isopropylcatechol, 4-hydroxyanisole, and *N*-acetyl-4-S-cystaminylphenol. Mono benzyl ether of HQ should only be used as a depigmenting agent in patients with vitiligo.

Azelaic Acid

Azelaic acid is a naturally occurring dicarboxylic acid obtained from cultures of *Pityrosporum ovale*. In vitro studies show that it reversibly inhibits tyrosinase activity and may also interfere with DNA synthesis.[50] Azelaic acid has an antiproliferative and cytotoxic effect on abnormal melanocytes but does not affect normal melanocytes.[51] At concentrations of 15-20%, azelaic acid shows equivalent efficacy to HQ 4% in the treatment of melasma and PIH.[52] After a 24-week treatment period for facial hyperpigmentation in dark-skinned patients (phototypes IV to VI), Lowe et al[53] showed that azelaic acid, 20% cream produced significantly greater decreases in pigmentary intensity than vehicle (investigator's subjective scale; $P = 0.021$). Verallo-Rowell et al[22] also demonstrated that azelaic acid, 20% cream was significantly more effective than HQ 2% cream in decreasing pigmentary intensity due to melasma. After 24 weeks of treatment, 95.4% azelaic acid vs 73.1% HQ-treated patients had decreases in pigmentary intensity of 1 to 3 levels.[22] Combinations with topical tretinoin, 0.05% and glycolic acid 15-20% also enhance its efficacy.[54] Other alternatives include the use of topical potent steroids, such as clobetasol propionate, and 20% azelaic acid, as demonstrated by Sarkar et al.[55] At the end of this study, 96.7% and 90% of patients (sequential therapy and azelaic acid) had good-to-excellent responses to treatment. Adverse effects included pruritus, transient erythema, and scaling.

Kojic Acid

Kojic acid is a hydrophilic fungal derivative obtained from *Aspergillus* and Penicillium species. It inhibits tyrosine kinase and has demonstrated the ability to reduce pigmentation. In a study by Lim,[56] 40 Chinese women with epidermal melasma were treated with 2% kojic acid in a gel containing 10% glycolic acid and 2% HQ on one side of the face. The other side was treated with the same application but without kojic acid. At the end of the study, all patients showed improvement in melasma on both sides of the face. However, there was more than 50% improvement of melasma in 24 of 40 patients (60%) treated with kojic acid compared with 19 of 40 patients (47.5%) patients in the control group. The most frequent side effect of kojic acid is irritant contact dermatitis. It is frequently used as an ingredient in cosmeceutical formulations marketed in the United States and parts of Asia.

Licorice

Licorice extract is the most commonly used ingredient in cosmetics for lightening the skin.[57] The active agents in licorice are glabridin, licochalcone A, and liquiritin. Licorice extracts have antiinflammatory and anticarcinogenic effects. They inhibit melanogenesis and can be used for the treatment of sensitive and irritated skin.[58-60] A study performed on 20 subjects with melasma who applied a liquiritin cream at 1 g/d for 4 weeks showed satisfactory to excellent results: pigmentation intensity was markedly reduced in 70% of liquiritin-treated subjects compared with control patients.[61]

Arbutin

Arbutin, which is the β-D-glucopyranoside derivative of HQ, is a naturally occurring plant-derived product that causes decreased tyrosinase activity without affecting RNA expression. It also inhibits melanosome maturation. The action of arbutin is dependent on its concentration. Greater concentrations are more efficacious than lower ones, but they may result in a paradoxical hyperpigmentation.[62] Arbutin has been used in a variety of pigment-lightening preparations in Japan at concentrations of 3%. A synthetic form, deoxyarbutin, is available with greater inhibition of tyrosinase than the naturally occurring form.[57] Arbutin is also a component of a myriad of cosmeceutical lightening formulations marketed in the United States.

Retinoids

Tretinoin acts by inhibiting tyrosinase transcription and by inhibiting the dispersion of keratinocyte pigment granules. It induces desquamation, enhances epidermal cell turnover, and reduces the contact time between keratinocytes and melanocytes, hence promoting the rapid loss of pigment through epidermopoiesis.[17] Retinoids are used in concentrations ranging from 0.01% to 0.1% and have been associated with irritant dermatitis characterized by erythema, dryness, and scaling. Tretinoin, 0.1% was used to treat melasma in a randomized placebo controlled study of 28 black patients. After 40 weeks of treatment, the tretinoin group showed a statistically significant reduction in the Melasma Area Severity Index score. Side effects were reported in 67% of the tretinoin group, which were mild in most cases.

Adapalene is a synthetic retinoid. It has greater selectivity than tretinoin for certain retinoic acid receptors. The efficacy and safety of adapalene gel 0.1% was assessed in a 12-week open-label study of 65 African patients. All had PIH associated with acne. At baseline, 20% of patients had severe PIH. There was significant improvement in the degree of PIH at weeks 4, 8, and 12 compared with baseline ($P < 0.01$). Less than 5% of patients reported moderate or severe skin irritation during treatment.[63]

Another synthetic retinoid, tazarotene cream, 0.1%, has also been shown to significantly reduce overall disease severity and intensity and hyperpigmentation within 18 weeks compared with vehicle ($P \leq 0.05$). In this double-blind, randomized, vehicle-controlled study of 74 acne patients from darker racial ethnic groups, the only adverse events encountered were trace levels of erythema, burning and peeling, and mild levels of dryness.[64]

Ascorbic Acid

Ascorbic acid is an antioxidant that affects melanogenesis by reducing *o*-dopaquinone to DOPA. It also changes melanin from jet black to light tan. Ascorbic acid is highly unstable and is quickly oxidized in aqueous solution. Some ascorbate esters, such as magnesium ascorbyl-2-phosphate, have been synthesized to prevent such disadvantages.[65] Ascorbic acid is often used in combination with hydroquinone in bleach formulations. Such combination products are tolerated well in darker racial ethnic groups.

Soy

Soy inhibits the protease-activated receptor (PAR)-2 pathway, expressed on keratinocytes.[66] PAR-2 regulates the ingestion of melanosomes by keratinocytes. Studies on dark-skinned animals showed that inhibition of PAR-2 caused depigmentation. After 12 weeks of use, a soy formulation was effective in lightening mottled pigmentation and solar lentigines.[67,68]

N-Acetyl Glucosamine

N-acetyl glucosamine, an amino-monosaccharide produced by the body through the addition of an amino group to glucose, is a new hypopigmenting agent.[69] It has several functions within the body, including acting as a substrate for the production of hyaluronic acid, heparin sulfite, and proteoglycans; important substances that maintain the water content of the dermis. *N*-acetyl glucosamine also inhibits the glycosylation of tyrosinase, an enzyme that is necessary for melanin production, accounting for its pigment-lightening ability.[70] *N*-acetyl glucosamine is now contained in several over-the-counter products for skin lightening.

Niacinamide

Niacinamide is the physiologically active form of niacin. It is a precursor of NADH and NADPH. Niacinamide inhibits the transfer of melanosomes to epidermal keratinocytes. It has been shown to improve photodamage in Asian subjects. In a study of 18 Asian subjects with hyperpigmentation, a 5% niacinamide moisturizer caused a significant decrease in facial hyperpigmentation.[71] Multiple over-the-counter cosmetic formulations now contain niacinamide.

Emerging Products

New and emerging skin lightening products include oral and topical formulations. Oral agents include grape seed extract and pycnogenol. New topical agents include ellagic acid, resveratrol, linoleic acid, aloesin, green tea extracts, and paper mulberry tree extracts.[18,50,72]

Resurfacing Procedures

Resurfacing procedures that have been used to treat melasma and PIH in darker racial ethnic groups include chemical peels, microdermabrasion, lasers, and intense pulsed light. These procedures facilitate absorption of topical bleaching agents, enhance epidermal turnover, and decrease epidermal melanin. Laser procedures may also cause disruption of melanin granules.

Multiple chemical peeling agents have been used to treat melasma and PIH in darker racial ethnic groups.[73] Some of these peeling agents include glycolic acid, trichloroacetic acid (TCA), Jessner's solution, salicylic acid, and tretinoin (Figs. 3 and 4). Given the propensity of darker skin to hyperpigment, peeling agents should be used with care and caution. The very best results are achieved with superficial peeling agents. To minimize complications, retinoids should be discontinued 1 to 2 weeks before peeling. Excessive peeling and exfoliation may result in post inflammatory hyperpigmentation.

Glycolic acid peels at concentrations ranging from 10% to 70% are popular and can be used in dark-skinned patients with significant improvements.[74] A 91% improvement in melasma (reduction in Melasma Area Severity Index score) was found in a study involving 25 Indian women treated with a series of 3 50% glycolic acid peels.[75] In addition, a series of serial glycolic acid peels (68% maximum) was performed in 19 black patients with PIH. Results were compared with a control group treated with 2% hydroquinone/10% glycolic acid twice daily and tretinoin, 0.05% hs. The peel group used the same topical regimen. Although not statistically significant, greater improvement was noted in the chemical peel group.[76]

Nanda et al[77] assessed the efficacy of 10-30% TCA peels after priming with either 2% hydroquinone or 0.025% tretinoin in 50 melasma patients, 92% of whom were Fitzpatrick type IV-V. "Fair" or "good" responses were achieved in 76-80% of patients in both groups.

Salicylic acid is one of the oldest chemical peels, its use having first been described by the German dermatologist Unna in 1882. Grimes evaluated the efficacy and safety of this agent in patients with darker skin.[78] In a study involving 25 patients of Fitzpatrick types V–VI: 9 patients had acne, 5 had PIH, 6 had melasma, and 5 had rough, oily skin with enlarged pores. All patients were treated with 4% hydroquinone for 2 weeks before undergoing a series of 5 20% or 30% salicylic acid peels at 2-week intervals. Efficacy was assessed by biweekly photographs. Moderate to significant improvement occurred in 22 patients (88%), and the remainder showed mild improvement. One hundred percent of patients with PIH, and 66% of those with melasma, showed moderate or significant improvement (Fig. 4). Mild adverse events occurred in 4 patients (16%): one patient experienced temporary crusting and hyperpigmentation, and 3 had transient dryness and hyperpigmentation, all of which resolved within 14 days. Complications of chemical peels in darker-skinned patients include PIH, postinflammatory

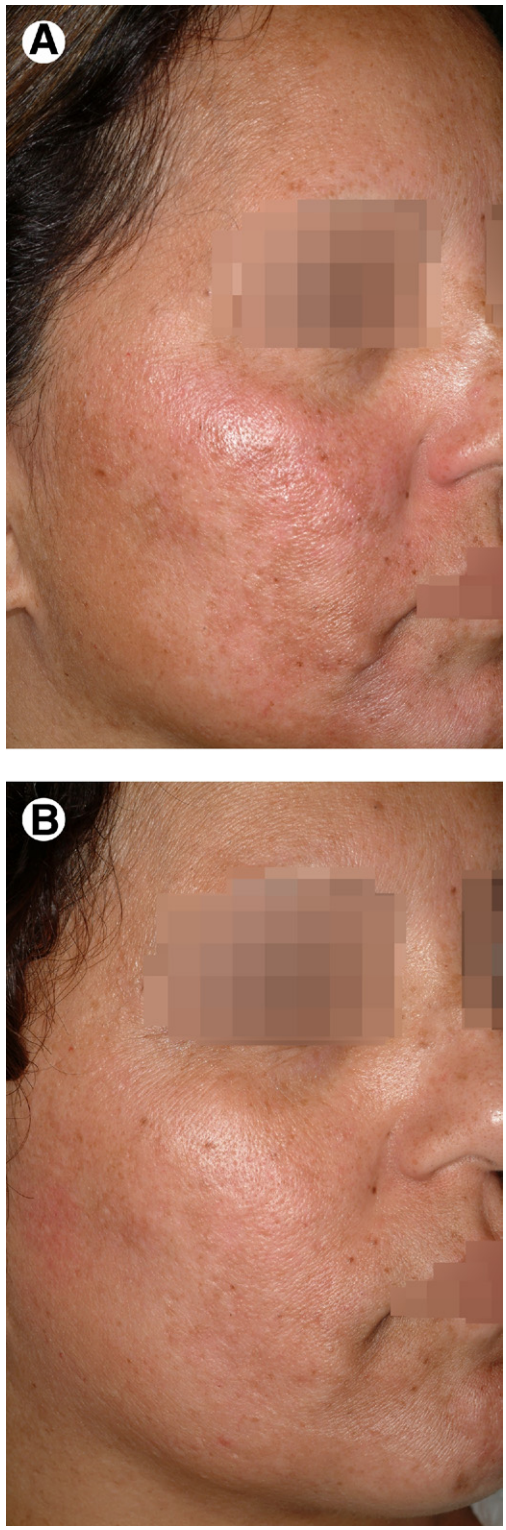

Figure 3 Patient with melasma (A) before and (B) after treatment with triple combination bleach and a series of 3 10% TCA peels.

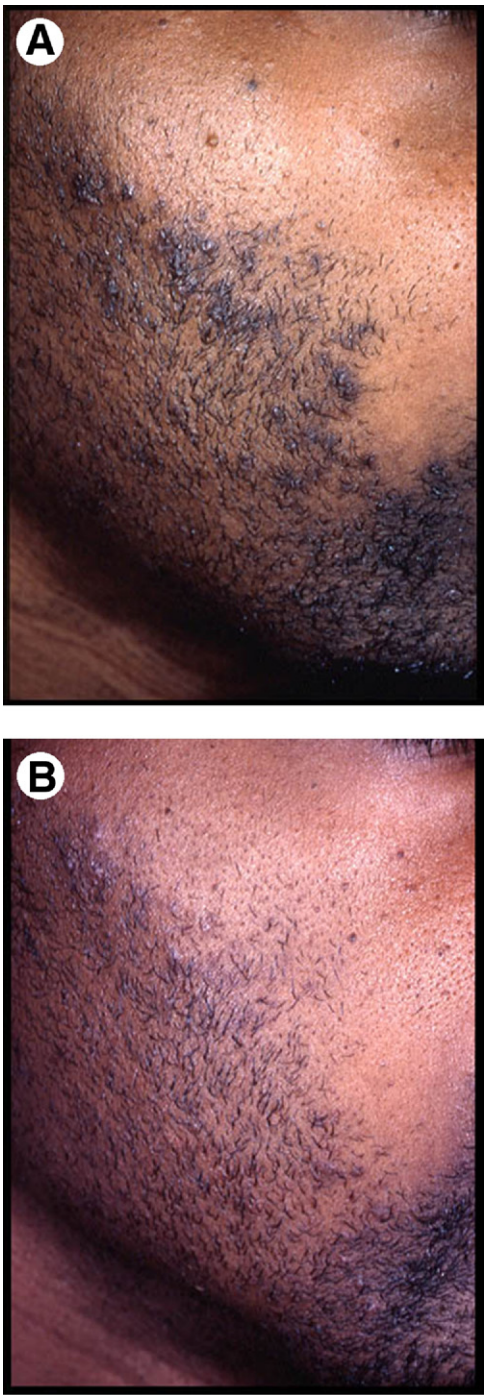

Figure 4 Patient with pseudofolliculitis barbae and PIH (A) before and (B) after a series of 5 salicylic acid peels.

hypopigmentation, depigmentation, hypertrophic scars, keloids, and milia formation.

Several different types of lasers and intense pulsed light sources have been used to treat melasma and PIH, with the most commonly used being the Q-switched pigment-specific lasers. Laser treatment is often complicated by hyperpigmentation, particularly in patients with darker skin types. This has been shown in patients undergoing therapy with the Q-switched ruby laser (694 nm),[79,80] CO_2 laser alone or in combination with Q-switched alexandrite laser (755 nm),[81] erbium:YAG laser (2940 nm),[82] and pigmented lesion dye laser (500-520 nm).[83] Recently, a relatively new treatment modality, fractional photothermolysis (Fraxel laser, Reliant Technologies, Palo Alto, CA), was introduced as an efficacious treatment for melasma.[84] The precise mechanism leading to clinical improvement is, however, unclear. This laser technology produces multiple distinctive microscopic columns of thermal damage. In one study, 10 patients with melasma were treated with 4 fractional photothermolysis treatments. Biopsies post treatment revealed a decrease in melanocytes and melanin.[79] Fraxel is currently the only laser that is approved by the Food and Drug Administration for treatment of melasma. However, there are now increasing concerns regarding the increased occurrence of PIH and/or worsening of melasma in patients treated with this modality.

Conclusions

Melasma and PIH are devastating conditions for many patients. Current data suggest that they have major negative impacts on quality of life. There are indeed several effective treatment strategies, including topical lightening agents and resurfacing procedures, that can improve these disorders. Additionally, UV-protective measures, such as the use of sunscreens, sun-protective clothing, and sun avoidance, are fundamental to the successful management of these conditions.

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