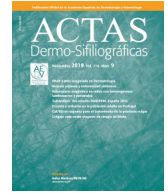




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Practical Dermatology

Clinical and Dermoscopic Characteristics of Facial Hyperpigmentation Disorders in Skin of Color

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ABSTRACT

Facial hyperpigmentation disorders (FHD) are common in dermatology, particularly in people with skin of color (SOC). They include melasma, post-inflammatory hyperpigmentation, and less frequent conditions such as exogenous ochronosis, lichen planus pigmentosus, Riehl's melanosis, acanthosis nigricans, seborrheic melanosis, pigmentary demarcation lines, drug-induced pigmentation, and nevus of Ota, among others. These conditions may significantly affect quality of life. They can present similar clinical and dermoscopic features, making diagnosis challenging. A thorough medical history, physical examination, and careful dermoscopic evaluation are essential to achieve an accurate diagnosis. In this article, we present a narrative review of the clinical and dermoscopic characteristics of FHD in SOC and provide diagnostic clues for the early diagnosis of these conditions.

Introduction

Facial hyperpigmentation disorders (FHD) are a frequent reason for consultation among individuals with skin of color (SOC), with an incidence rate up to seven times higher than in lighter skin types.¹ Individuals with SOC include those with Fitzpatrick phototypes IV, V, and VI, whose population proportion has increased in Spain and Europe in recent years.^{2,3}

FHD in SOC may be related to differences in melanosome structure and increased responsiveness to visible light and UVA radiation.⁴⁻⁶ These disorders include very common conditions such as melasma and post-inflammatory hyperpigmentation (PIH), as well as less common entities such as lichen planus pigmentosus, Riehl's melanosis, ochronosis, or nevus of Ota. These dermatoses may significantly affect quality of life, with an impact even greater than that of vitiligo.⁷ The diagnosis of FHD may be challenging, and dermoscopy can be highly useful.⁸ Unfortunately, dermoscopic studies in SOC remain relatively scarce.

Below, we review the clinical and dermoscopic presentation of FHD in SOC, providing diagnostic clues to facilitate the clinical approach to these conditions in daily practice.

Material and methods

We conducted a narrative literature review. During September 2024, searches were performed in Spanish and English across Medline and Google Scholar using the following terms (alone or in combination): "dermoscopy," "dark skin," "skin of color," "ethnic skin," "black skin," "Fitzpatrick phototypes," "pigmentary disorders," "facial," "pigmentary demarcation lines," "melasma," "post-inflammatory hyperpigmentation," "drug-induced hyperpigmentation," "acanthosis nigricans," "frictional melanosis," "seborrheic melanosis," "nevus of Ota," "lichen planus pigmentosus," "Riehl's melanosis," "ochronosis," "maturational hyperpigmentation," and "erythema dyschromicum perstans."

Articles were initially screened according to their titles and abstracts and subsequently reviewed in full to determine their relevance. Studies specifically addressing the clinical and dermoscopic description of FHD in SOC were included. Given the limited number of publications, some studies also included patients with lighter skin types or described lesions in other body regions. The search and selection process was performed independently by 2 authors (MMP and DMC), who resolved discrepancies by consensus.

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54 **Facial hyperpigmentation disorders (Table 1)**55 *Pigmentary demarcation lines*

56 Pigmentary demarcation lines (PDL), also known as Futcher–Voigt
57 lines, are more easily observed in SOC due to the greater contrast with
58 hypopigmented skin. Approximately 6% of patients with PDL present
59 facial involvement (types F, G, and H) (Fig. 1).⁹

60 A recent article demonstrated that >80% of women with facial PDL
61 present alterations of the sexual hormonal axis.¹⁰ Clinically, they appear
62 as well-defined hyperpigmented lines: type F forms a “V” shape on the
63 lateral face between the temple and malar area; type G forms a “W”
64 shape; and type H extends from the angle of the mouth to the chin.¹¹

65 Dermoscopic findings include exaggeration of the normal facial pseu-
66 doreticular pattern¹² and white dots arranged in a granular pattern.¹³

67 *Melasma*

68 Melasma may reach a prevalence of 10–30% in SOC, or even higher
69 when additional risk factors are present.¹⁴ It can be disabling, with de-
70 pression rates of around 50%.¹⁵ It typically develops between 20 and 40
71 years of age and presents as irregular brown macules with well-defined
72 borders in photoexposed areas, often with areas of “spared” skin. Facial
73 melasma is classified as centrofacial (Fig. 2A–C) or peripheral.^{14,16} The
74 most common dermoscopic finding is accentuation of the facial pseu-
75 doreticular pattern. Brown dots and globules may be observed (although
76 not predominating), along with annular or arcuate structures and te-
77 langiectasias.¹⁷ A recent study demonstrated that the reticuloglobular
78 pattern was more prominent in epidermal melasma, whereas granular or
79 dotted patterns were observed in dermal melasma.¹⁸ Unlike ochronosis,
80 follicular openings are preserved in melasma.^{12,17}

81 *Post-inflammatory hyperpigmentation*

82 Post-inflammatory hyperpigmentation (PIH) represents pigmen-
83 tation due to a previous inflammatory process.¹⁹ Clinically, it is
84 recognized by the appearance of well-defined brown macules in areas
85 of prior inflammation, with a latency period ranging from several days
86 to several months (Fig. 2D–F).¹⁹ PIH may be particularly prolonged in
87 SOC, sometimes persisting for 2–5 years.²⁰ Dermoscopic findings in PIH
88 are not well described in the literature. An accentuated pseudoreticular
89 pattern with light- or dark-brown globules and dots sparing follicular
90 openings may be observed. Pigmentation appears dark brown when the
91 pigment is located in the epidermis or superficial dermis and grayish
92 when deposited more deeply.²¹

93 *Drug-induced hyperpigmentation*

94 Drug-induced hyperpigmentation is classically associated with
95 tetracyclines, nonsteroidal anti-inflammatory drugs, antimalarials, an-
96 tipsychotics, amiodarone, diltiazem, or certain antiretroviral drugs.²² Its
97 pathophysiology is complex: some drugs induce melanin accumulation
98 (e.g., hydroxychloroquine), others stimulate melanin synthesis (e.g., li-
99 popofuscin), or lead to iron deposition (e.g., minocycline).²³ Certain drugs
100 such as voriconazole may induce phototoxic reactions.²⁴ Some drugs
101 have been particularly associated with facial pigmentation, including
102 sunitinib (yellowish hyperpigmentation), phenytoin, polymyxin B, dil-
103 tiazem, or imipramine.²² Clinically, drug-induced hyperpigmentation
104 appears as hyperpigmented macules with focal or diffuse facial involve-
105 ment, sometimes accompanied by pigmentation in other body areas.^{22,23}
106 Only 1 dermoscopic description has been reported in the literature, sho-
107 wing accentuation of the facial pseudoreticular pattern with light- or
108 dark-brown dots and globules.²⁵

109 The late phase of fixed drug eruption (FDE) may present with isola-
110 ted pigmentation. Dermoscopy in FDE shows pigment dots and globules
111 with different shades ranging from brown to gray.¹²

Acanthosis nigricans

Acanthosis nigricans (AN) presents with grayish-brown hyperpig-
mented plaques with a velvety texture in flexural areas, particularly
the neck, axillae, and groin, and less frequently on the face (Fig. 3).
It is more common in adult SOC populations and is frequently associa-
ted with obesity and insulin resistance. Some authors reserve the term
AN for cases not associated with drugs, neoplasms, or syndromic condi-
tions, referring to obesity-related cases as pseudoacanthosis nigricans.²⁶
Dermoscopically, AN shows a pattern known as “sulci and gyri/cristae,”
characterized by accentuated skin grooves and ridges, sometimes
hyperkeratotic and more pigmented, on a grayish-brown “dirty” back-
ground²⁷ resembling “crocodile skin.”²¹ Occasionally, dark-brown dots
or globules and, more rarely, telangiectasias may also be observed.²⁵

Frictional melanosis

Frictional melanosis (FM) is also more common in SOC (particu-
larly in individuals from India). It appears in areas subjected to chronic
or repeated friction. Clinically, it presents as well-defined dark-brown
macules and plaques in areas exposed to trauma (Fig. 4). It is im-
portant to ask about possible causes of the lesions (e.g., forehead
lesions caused by friction during prayer).²⁸ Mutalik et al. described 6
clinical patterns in facial FM: zygomatic–supraorbital, metomelanosis
(forehead), panfacial, parafacial, perioral, and mixed. These patterns
show a characteristic symmetrical distribution with uniform brown-
black pigmentation without textural changes.²⁹ In contrast to FM,
macular amyloidosis usually does not affect the face, tends to be more
pruritic, and shows superficial amyloid deposition on histopathology.
Dermoscopically, the most frequent finding in FM is exaggeration of the
facial pseudoreticular pattern.^{12,29} Dark-brown or grayish dots and
globules and prominent follicular openings have also been described.^{25,28}

Seborrheic melanosis

Seborrheic melanosis (SM), also known as sebomelanosis, is an en-
tity almost exclusively observed in individuals with skin of color (SOC)
(Fig. 5). For most authors, it is considered a post-inflammatory sequela
of seborrheic dermatitis. However, cases of SM have also been described
in patients without a prior history of seborrheic dermatitis and who are
asymptomatic. SM is characterized by hyperpigmented macules and thin
plaques with very superficial scaling, predominantly affecting the nasal
grooves and nasolabial folds. It also typically involves the inferior nasal
fold or the perioral region. It may or may not be pruritic and can in-
volve other body areas such as the scalp or trunk.^{30,31} Dermoscopically,
a prominent pseudoreticular pattern, brown granular structures, poorly
defined vessels, prominent follicular openings, and yellowish-white pe-
ri-follicular sebaceous scales are observed.^{12,30}

Nevus of Ota

Nevus of Ota, also known as nevus fuscocaeruleus ophthalmoma-
xillaris, is an oculodermal melanocytosis characterized by coalescent
macules distributed along the V1/V2 branches of the trigeminal nerve
(Fig. 6A–C). It is more common in women of Asian or African an-
cestry. In 90% of cases it is unilateral and involves the sclera in 60%
of patients.³² There is a slight increased risk of glaucoma, and rare
cases of transformation into uveal or cutaneous melanoma have been
described.^{32,33} Therefore, patients with nevus of Ota should undergo
ophthalmologic follow-up.³⁴ Dermoscopic features include structureless
blue-gray areas and scattered brown-gray dots.^{12,35}

Lichen planus pigmentosus

Lichen planus pigmentosus (LPP) is characterized by oval brown
to gray-brown macules and plaques, predominantly affecting the pre-

Table 1
Clinical and dermoscopic characteristics of the main facial hyperpigmentation disorders in darker phototypes.

Disorder	Clinical characteristics	Dermoscopic characteristics	Diagnostic clues
Pigmentary demarcation lines	Well-defined brown lines. Bilateral and symmetrical. Three patterns: F, G, and H.	Light or dark brown color. Exaggeration of the facial pseudoreticular pattern. White dots in a granular pattern.	Congenital. Well-defined, bilateral, symmetrical, and often subtle.
Melasma	Irregular, well-defined brown macules. Areas of spared skin.	Light or dark brown color. Exaggeration of the facial pseudoreticular pattern. No predominance of dots or globules. Arcuate structures. Preservation of follicular openings.	Most frequent centofacial distribution (malar area, forehead, upper lip). Islands of spared skin. Young women, often associated with oral contraceptive use.
Post-inflammatory hyperpigmentation	Well-defined hyperpigmented macules. History of prior inflammation.	Brown structureless areas, sometimes with erythema.	Previous inflammatory process. Well-defined margins similar to those of the preceding dermatosis.
Drug-induced hyperpigmentation	Variable depending on the drug. Diffuse or localized lesions. Usually involvement of other body areas.	Not clearly defined. In fixed drug eruption: diffuse brown-gray dots and globules.	History of drug intake. Pigmentation in other areas (e.g., melanonychia with minocycline) or generalized pigmentation with antimalarial use.
Acanthosis nigricans	Dark-brown velvety plaques on cheeks and frontal region. Also present in skin folds (neck, axillae, groin). History of obesity, metabolic syndrome, and acrochordons.	“Sulci and gyri/cristae” pattern (papillomatous appearance). Occasionally dark-brown dots or globules or telangiectasias. “Dirty” background.	Middle-aged adults, obesity, and other signs of insulin resistance. Textural changes of affected skin. “Dirty” appearance in folds, especially neck and axillae.
Frictional melanosis	Lesions in friction areas. Well-defined hyperpigmented lesions. Typical locations (nasal dorsum in allergic rhinitis; central forehead in Muslim individuals).	Exaggeration of the facial pseudoreticular pattern. Diffuse brown or gray dots and globules.	Friction areas: detailed history-taking is essential. Exogenous-appearing pigmentation.
Seborrheic melanosis	Seborrheic areas such as the alar groove. Typical involvement of nasal base and perioral region. May be associated with seborrheic dermatitis elsewhere. Pruritus may be present.	Superficial whitish-yellow scales similar to seborrheic dermatitis, but with a hyperpigmented background and exaggerated pseudoreticular pattern.	Almost exclusive to darker skin types. Typical seborrheic areas plus nasal base and perioral region. Superficial scaling clinically and dermoscopically.
Nevus of Ota	Present during the first years of life. Blue-gray plaques in the V1 and V2 distribution of the trigeminal nerve. Possible ocular involvement (glaucoma, melanoma).	Blue-gray background. Superimposed brown globules.	Appears in early childhood and may progressively enlarge. V1 and V2 involvement. Diffuse blue-gray background.
Lichen planus pigmentosus	Brown-gray macules, sometimes well defined. Possible association with other lichen planus variants. May involve eyelids.	Exaggerated pseudoreticular pattern with predominance of brown-gray dots or globules. Telangiectasias. Occasionally alopecic plaques.	Poorly defined grayish macules. Possible association with lichen planopilaris or frontal fibrosing alopecia.
Riehl’s melanosis	Brown-gray (sometimes bluish) hyperpigmented plaques. Erythematous background, sometimes with scaling. History of exposure to exogenous substances (e.g., perfumes). Pruritus may be present.	Brown/gray/blue reticular pattern on erythematous background. Fine scaling, follicular keratotic plugs, and perifollicular pigmented halos.	History of perfumes or other exogenous substances (detailed investigation in the clinical history).
Exogenous ochronosis	Previous use of depigmenting agents. Brownish or blackish macules and patches with superimposed hypopigmented areas.	Very dark brown/black color. Obliteration of follicular openings. Hypopigmented areas within an exaggerated facial pseudoreticular pattern. Curvilinear or amorphous very dark structures.	History of prolonged depigmenting agent use (especially hydroquinone). “Banana bodies” on histology.
Maturational hyperpigmentation	Brown macules and patches with a central “granular” area. Involves temples, cheeks, and forehead. Often reported in individuals of Indian origin and associated with metabolic syndrome.	Hyperpigmented rings with predominantly follicular distribution.	Indian ethnicity. Cheeks and temples with granular background.
Ashy dermatosis	Blue-gray macules and patches on trunk, extremities, neck, and less frequently on the face.	Blue-gray dots without vascular structures.	Subtle blue-gray background. Involvement of trunk and limbs in addition to the face. In early stages, lesions may have a palpable border.

FDE, fixed drug eruption; FHD, facial hyperpigmentation disorders; V1 and V2, 1st and 2nd branches of the trigeminal nerve.

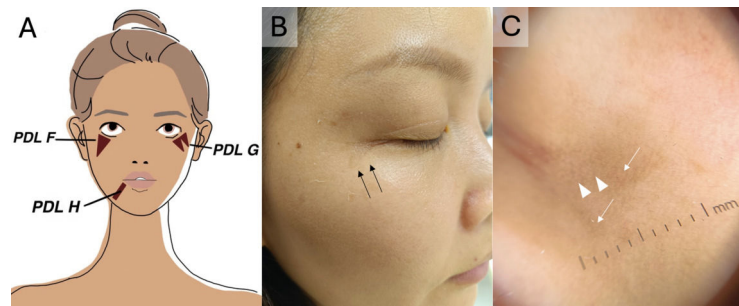


Fig. 1. Facial pigmentary demarcation lines. (A) Diagram illustrating the location of the lines. (B, C) Asian patient with type G lines (black arrows), forming a “W” shape at the lateral canthus of the right eye. Dermoscopy showed accentuation of the facial pseudoreticular pattern (white arrows) and a granular white pattern (white arrowheads).



Fig. 2. (A, B) Melasma in a 42-year-old Latin American man. (A) Irregular brown macules with areas of spared skin, predominantly on the cheeks. (B) Dermoscopy shows a prominent pseudoreticular pattern (black arrows), arcuate structures (black arrowheads), and lighter areas of spared skin (white arrows). Note the preservation of follicular units (white arrowheads). (C) Melasma on the cheek of a Latin American patient. (D) Post-inflammatory hyperpigmentation in a woman of African ancestry, with brown macules on the forehead and eyelids secondary to atopic dermatitis. (E) Post-inflammatory hyperpigmentation in a young man due to beard folliculitis. (F) Frontal post-inflammatory hyperpigmentation secondary to prayer in a Muslim man.

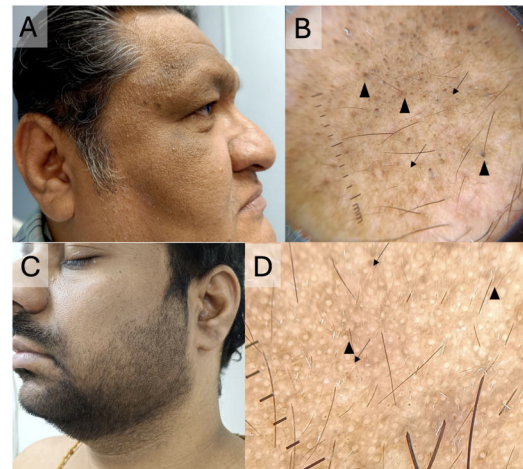


Fig. 3. (A, B) Acanthosis nigricans in a Central American man. (A) Brownish macules with a velvety appearance predominantly on both cheeks and the supraciliary areas. (B) Dermoscopy shows a “sulci and gyri/cristae” pattern (black arrows) along with follicular plugs (black arrowheads). (C, D) Acanthosis nigricans in an Indian man. (C) Brownish velvety macules on the cheek. Note the acanthosis nigricans on the neck. (D) Dermoscopy shows a “sulci and gyri/cristae” pattern (black arrows).

hyperpigmented macules or plaques on the face, neck, and upper chest (Fig. 7A–C). It may occasionally present with pruritus and superficial scaling. It is considered a type IV hypersensitivity reaction triggered by contact allergens such as fragrances, textiles, and cosmetics, although genetic factors, autoimmune alterations, and sun exposure may also contribute.⁴⁰ Dermoscopically, a marked pseudoreticular pattern may be observed, with predominance of gray dots and globules, telangiectatic vessels, and an erythematous background.^{17,21,12} These findings are therefore very similar to those seen in LPP. Clinical history is useful to differentiate RM from LPP (history of allergen exposure in RM). Dermoscopic findings more specific to RM include fine scaling, follicular keratotic plugs, perifollicular hypopigmented halos,⁴¹ and obliteration of follicular openings.²⁵

Ochronosis exogena

Exogenous ochronosis (EO) is characterized by dark-brown or even blackish macules and plaques.⁴² Papules resembling “caviar” (colloid milium) or interspersed atrophic hyperpigmented patches may be observed. Dermoscopically, EO presents with exaggerated facial pseudoreticular pattern, hypopigmented areas, telangiectasias, and dark-brown curvilinear or amorphous structures with obliteration of follicular openings.^{17,21,12} Histological examination is particularly useful, showing the characteristic ochre-colored “banana bodies” in the upper dermis.¹²

auricular and temporal regions, neck, cheeks, and/or intertriginous areas (Fig. 6D–F). Some authors classify LPP within acquired dermal macular hyperpigmentation (ADMH), which includes several dermal facial hyperpigmentation disorders such as pigmented contact dermatitis (Riehl’s melanosis) and erythema dyschromicum perstans.¹² LPP may sometimes be associated with other variants of lichen planus, such as lichen planopilaris or frontal fibrosing alopecia.³⁶ Dermoscopically, there is accentuation of the facial pseudoreticular pattern with predominant blue-gray and brown dots and globules arranged in arcuate, perifollicular, and mottled patterns (or without a clear pattern),³⁷ without obliteration of follicular openings.²⁵ Other findings include diffuse erythema, telangiectasias, rhomboidal structures, follicular asymmetry, and reduction of vellus hair.³⁸ Mulinari-Brenner et al. recently described additional dermoscopic features including decreased follicular openings, follicular hyperkeratosis, keratotic plugs, perifollicular erythema, simple loop interfollicular vessels, branching vessels, and white dots. Areas of reduced hair density or alopecia may also be observed.³⁹

Pigmented contact dermatitis (Riehl’s melanosis)

Riehl’s melanosis (RM) predominantly affects SOC, particularly older women. It is characterized by reticulated to diffuse grayish-brown

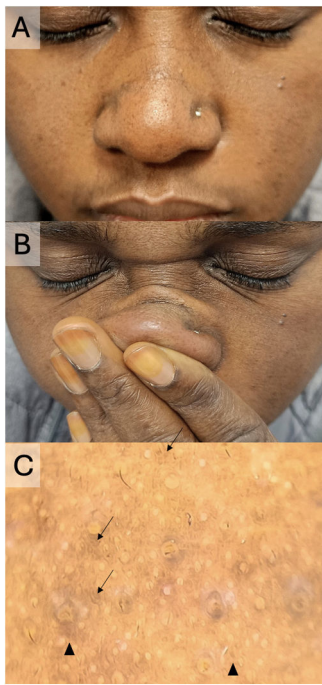


Fig. 4. (A–C) Frictional melanosis secondary to allergic rhinitis in a 32-year-old African woman. (A) Hyperpigmented macules in the nasal region. (B) Repetitive gesture performed by the patient (frequent nasal rubbing). (C) Dermoscopy showing exaggeration of the facial pseudoreticular pattern (black arrows) and diffuse brown-gray dots/globules (black arrowheads).

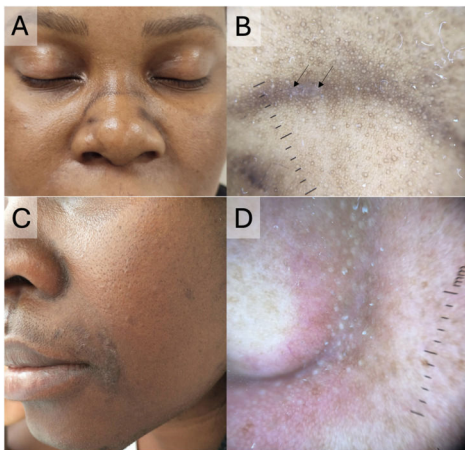


Fig. 5. (A, B) Thirty-year-old African woman with hyperpigmented lesions in the nasolabial folds and alar grooves. (A) Dark-brown macules in the alar grooves and nasolabial folds. (B) Dermoscopy: whitish scales (black arrows), predominantly in the alar groove, with background hyperpigmentation. (C, D) Latin American woman with pigmentation in the nasal groove and perioral region due to seborrheic dermatitis. Dermoscopy of the nasal groove showed prominent follicular openings, mild scaling, telangiectasias, and diffuse pigment.

212 *Maturational hyperpigmentation*

213 Maturational hyperpigmentation (MH) is a recently described entity,
214 almost exclusive to SOC (especially individuals from India), character-
215 ized by dark-brown macules predominantly on the cheeks, temples, and
216 forehead. Macroscopically, the lesions often show a “granular” sur-
217 face and poorly defined borders. It is considered a marker of metabolic
218 syndrome.⁴³ Dermoscopically, it shows a nonspecific pattern with a
219 light-brown background overlaid by multiple dark-brown globules and

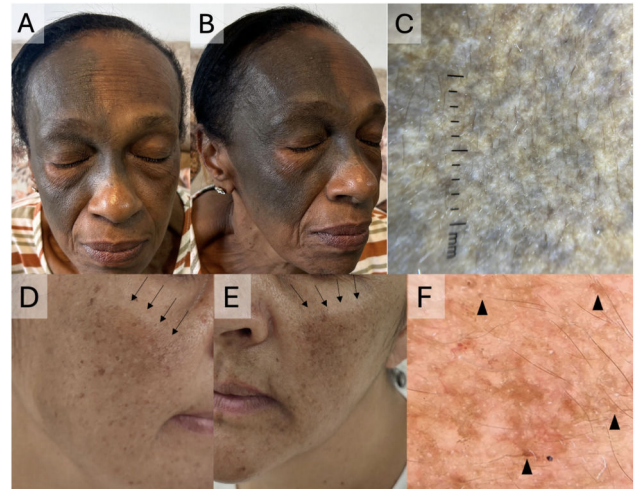


Fig. 6. (A–C) Nevus of Ota. (A) Blue-gray pigmented macule on the right hemiface, predominantly in the V1 and V2 territories. (B) Dermoscopy shows blue-gray coloration with light-brown and some lighter areas within the nevus. Photographs courtesy of Dr. Verónica Echeverry. (D–F) Lichen planus pigmentosus in a Latin American woman. (D–E) Grayish-brown macules affecting the entire face, predominantly on the cheeks (black arrows). (F) Dermoscopy shows perifollicular brown-gray dots and globules (black arrowheads) without obliteration of follicular openings.

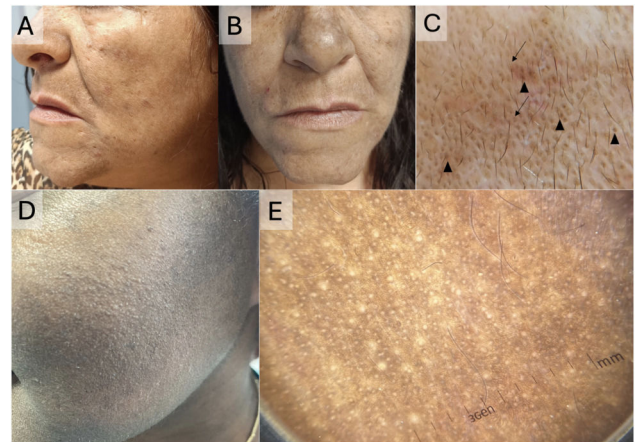


Fig. 7. (A–C) Latin American woman with Riehl's melanosis. (A, B) Dark brown-gray macules and patches diffusely affecting the face, especially the nose, cheeks, and perioral region. (C) Dermoscopy highlights follicular keratotic plugs (black arrows) and perifollicular pigmented halos (black arrowheads). (D, E) Latin American woman with ashy dermatosis. (D) Blue-gray macules on the left cheek. (E) Dermoscopy shows a brown reticular pattern without evident vascular structures.

220 clods. The most distinctive feature is circular to oval dark-brown struc-
221 tures with perifollicular accentuation. Unlike acanthosis nigricans, it does
222 not show sulci or ridges.^{43,44}

223 *Ashy dermatosis (erythema dyschromicum perstans)*

224 Ashy dermatosis (erythema dyschromicum perstans) is a condition
225 of unknown etiology, more common in SOC (Latino, Afro-descendant,
226 and Asian populations). It is characterized by grayish or bluish hyper-
227 pigmented macules that appear on the trunk, extremities, neck, and less
228 frequently on the face (Fig. 7D–E). Dermoscopy helps differentiate it
229 from other conditions such as melasma, PIH, fixed drug eruption, and
230 LPP. Dermoscopy typically shows a reticulated pattern with gray and

231 brown networks, grayish or black pigment globules in more advanced
232 lesions, and absence of vascular structures.^{21,45}

233 Discussion

234 Given globalization and migratory flows, improving dermatologic
235 training in diseases that more frequently affect individuals with skin of
236 color (SOC) is necessary.⁴⁶ FHD are very common in this population,
237 and certain conditions such as seborrheic melanosis or maturational
238 hyperpigmentation are almost exclusive to SOC. Although studies on
239 dermoscopy in SOC have increased over the last decade, few specifi-
240 cally evaluate FHD,^{21,25,12} and we did not find systematic reviews or
241 meta-analyses addressing this topic.

242 The diagnosis of FHD may be complex, and histological examination
243 is not always particularly useful (except in certain conditions such as
244 exogenous ochronosis or lichen planus pigmentosus). A thorough medi-
245 cal history (for example, to rule out the use of topical products that
246 may favor Riehl's melanosis, a history of inflammatory dermatoses in
247 post-inflammatory hyperpigmentation, among others), physical exami-
248 nation, and dermoscopy will often allow an accurate diagnosis of FHD
249 in SOC or at least a very close diagnostic approximation. Dermoscopy
250 can provide important diagnostic clues, although no pathognomonic
251 findings have been identified.

252 Limitations

253 The present review has the limitation of being narrative rather than
254 a systematic review or meta-analysis, implying that study selection may
255 be subject to bias. In addition, many of the included studies did not spe-
256 cifically address FHD but extrapolated facial findings from observations
257 made in other body areas. Most of the reviewed studies correspond to
258 small case series or retrospective studies, which limits the generaliza-
259 bility of the findings and the ability to establish causal relationships.
260 Furthermore, it should be noted that not all the reviewed publications
261 were strictly focused on higher phenotypes.

262 Conclusions

263 FHD are very common in individuals with skin of color and may
264 significantly affect quality of life. A thorough medical history, physical
265 examination, and dermoscopic evaluation can assist in establishing the
266 diagnosis.

267 Conflict of interest

268 The authors declare that they have no conflict of interest.

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272 patient with nevus of Ota.

273 References

- 274 1. Dlova NC, Akintilo LO, Taylor SC. Prevalence of pigmentary disorders: a cross-
275 sectional study in public hospitals in Durban, South Africa. *Int J Womens Dermatol.*
276 2019;5:345–348.
- 277 2. Brown-Korsah JB, McKenzie S, Omar D, Syder NC, Elbuluk N, Taylor SC. Variations
278 in genetics, biology, and phenotype of cutaneous disorders in skin of color – Part
279 I: Genetic, biologic, and structural differences in skin of color. *J Am Acad Dermatol.*
280 2022;87:1239–1258.
- 281 3. Estadística de Migraciones y Cambios de Residencia – Inmigración exterior. INE. s.
282 f.
- 283 4. Montagna W, Carlisle K. The architecture of black and white facial skin. *J Am Acad*
284 *Dermatol.* 1991;24:929–937.

5. Thong H-Y, Jee S-H, Sun C-C, Boissy RE. The patterns of melanosome distribution in
285 keratinocytes of human skin as one determining factor of skin colour. *Br J Dermatol.*
286 2003;149:498–505.
6. Tsai J, Chien AL. Photoprotection for skin of color. *Am J Clin Dermatol.*
287 2022;23:195–205.
7. Davis EC, Callender VD. Postinflammatory hyperpigmentation: a review of the epi-
288 demiology, clinical features, and treatment options in skin of color. *J Clin Aesthet*
289 *Dermatol.* 2010;3:20–31.
8. Costa-Silva M, Calistru A, Barros AM, Lopes S, Esteves M, Azevedo F. Dermatoscopy
290 of flat pigmented facial lesions—evolution of lentigo maligna diagnostic criteria.
291 *Dermatol Pract Concept.* 2018;8:198–203.
9. Zieleniewski L, Schwartz RA, Goldberg DJ, Handler MZ. Voigt–Futcher pigmentary
292 demarcation lines. *J Cosmet Dermatol.* 2019;18:700–702.
10. Kathuria S, Khunger N, Ramesh V. Clinical and hormonal evaluation in facial
293 pigmentary demarcation lines: a pilot study. *Indian J Dermatol Venereol Leprol.*
294 2012;78:742–744.
11. Malakar S, Dhar S. Pigmentary demarcation lines over the face. *Dermatology.*
295 2000;200:85–86.
12. Vinay K, Ankad BS. Dermatoscopic features of pigmentary diseases in ethnic skin.
296 *Indian Dermatol Online J.* 2021;12:24–33.
13. Puri N, Gill SK, Brar BK, Goyal S. Facial pigmentary demarcation lines – a new
297 dermoscopic finding. *Indian Dermatol Online J.* 2024;15:329–330.
14. Ogbechie-Godec OA, Elbuluk N. Melasma: an up-to-date comprehensive review. *Der-*
298 *matol Ther (Heidelb).* 2017;7:305–318.
15. Chen W, Wan Y, Sun Y, Gao C, Li J. Prevalence of depression in melasma: a systematic
299 review and meta-analysis. *Front Psychiatry.* 2023;14:1276906.
16. Handel AC, Miot LDB, Miot HA. Melasma: a clinical and epidemiological review. *Am*
300 *Bras Dermatol.* 2014;89:771–782.
17. Chatterjee M, Neema S. Dermoscopy of pigmentary disorders in brown skin. *Dermatol*
301 *Clin.* 2018;36:473–485.
18. Agamia N, Apalla Z, Salem W, Abdallah W. A comparative study between oral
302 tranexamic acid versus oral tranexamic acid and Q-switched Nd-YAG laser in
303 melasma treatment: a clinical and dermoscopic evaluation. *J Dermatolog Treat.*
304 2021;32:819–826.
19. Kaufman BP, Aman T, Alexis AF. Postinflammatory hyperpigmentation: epi-
305 demiology, clinical presentation, pathogenesis and treatment. *Am J Clin Dermatol.*
306 2018;19:489–503.
20. Markiewicz E, Karaman-Jurukovska N, Mammone T, Idowu OC. Post-inflammatory
307 hyperpigmentation in dark skin: molecular mechanism and skincare implications.
308 *Clin Cosmet Investig Dermatol.* 2022;15:2555–2565.
21. Krueger L, Saizan A, Stein JA, Elbuluk N. Dermoscopy of acquired pigmentary disor-
309 ders: a comprehensive review. *Int J Dermatol.* 2022;61:7–19.
22. Nahhas AF, Braunberger TL, Hamzavi IH. An update on drug-induced pigmentation.
310 *Am J Clin Dermatol.* 2019;20:75–96.
23. Elbuluk N, Grimes P, Chien A, et al. The pathogenesis and management
311 of acne-induced post-inflammatory hyperpigmentation. *Am J Clin Dermatol.*
312 2021;22:829–836.
24. Kuklinski LF, Li S, Karagas MR, Weng W-K, Kwong BY. Effect of voriconazole on risk
313 of nonmelanoma skin cancer after hematopoietic cell transplantation. *J Am Acad*
314 *Dermatol.* 2017;77:706–712.
25. Solanki V, Dongre A, Nayak C. A clinico-epidemiological study of different dermos-
315 copic patterns in hyperpigmented facial lesions in a tertiary care centre. *J Cutan*
316 *Aesthet Surg.* 2024;17:112–123.
26. Leung AKC, Lam JM, Barankin B, Leong KF, Hon KL. Acanthosis nigricans: an upda-
317 ted review. *Curr Pediatr Rev.* 2022;19:68–82.
27. Elmas ÖF, Demirbaş A, Kutlu Ö, Kilitçi A, Atasoy M. Utility of dermoscopy in the
318 diagnosis of acanthosis nigricans. *J Cosmet Dermatol.* 2020;19:3426–3427.
28. Arora G, Khandpur S, Bansal A, et al. Current understanding of frictional dermatoses:
319 a review. *Indian J Dermatol Venereol Leprol.* 2023;89:170–188.
29. Mutalik SD, Pethe SV, Nikam BP, Rasal YD. Facial frictional melanosis in indian
320 patients: defining the entity. *Clin Dermatol Rev.* 2019;3:78.
30. Verma SB, Vasani RJ, Chandrashekar L, Thomas M. Seborrheic melanosis: an entity
321 worthy of mention in dermatological literature. *Indian J Dermatol Venereol Leprol.*
322 2017;83:285–289.
31. Vashisht KR, Garg S. Seborrheic melanosis: a unique under-recognized entity in
323 ethnic skin. *J Clin Aesthet Dermatol.* 2022;15:15–16.
32. Patrocínio J, de Sousa D, Frade JV. Nevus of Ota. *J Gen Intern Med.* 2023;38:1302.
33. Shaffer D, Walker K, Weiss GR. Malignant melanoma in a Hispanic male with nevus
324 of Ota. *Dermatology.* 1992;185:146–150.
34. Abdolrahimzadeh S, Pugi DM, Manni P, et al. An update on ophthalmological
325 perspectives in oculodermal melanocytosis (Nevus of Ota). *Graefes Arch Clin Exp*
326 *Ophthalmol.* 2023;261:291–301.
35. Elmas ÖF, Kilitçi A. Dermoscopic findings of Nevus of Ota. *Balkan Med J.*
327 2020;37:116–118.
36. Robles-Méndez JC, Rizo-Frías P, Herz-Ruelas ME, Pandya AG, Ocampo Candiani
328 J. Lichen planus pigmentosus and its variants: review and update. *Int J Dermatol.*
329 2018;57:505–514.
37. Pirmez R, Duque-Estrada B, Donati A, et al. Clinical and dermoscopic features of li-
330 chen planus pigmentosus in 37 patients with frontal fibrosing alopecia. *Br J Dermatol.*
331 2016;175:1387–1390.
38. Sharma VK, Gupta V, Pahadiya P, Vedi KK, Arava S, Ramam M. Dermoscopy
332 and patch testing in patients with lichen planus pigmentosus on face: a cross-
333 sectional observational study in fifty indian patients. *Indian J Dermatol Venereol*
334 *Leprol.* 2017;83:656–662.

- 369 39. Mulinari-Brenner FA, Guilherme MR, Peretti MC, Werner B. Frontal fibrosing alopecia and lichen planus pigmentosus: diagnosis and therapeutic challenge. *An Bras Dermatol.* 2017;92:79–81. 381
- 370 40. Wang L, Xu A-E. Four views of Riehl's melanosis: clinical appearance, dermoscopy, confocal microscopy and histopathology. *J Eur Acad Dermatol Venereol.* 2014;28:1199–1206. 382
- 371 41. Sitohang IBS, Prayogo RL, Rihatmadja R, Sirait SP. The diagnostic conundrum of Riehl melanosis and other facial pigmentary disorders: a case report with overlapping clinical, dermoscopic, and histopathological features. *Acta Dermatovenereol Alp Pannonica Adriat.* 2020;29:81–83. 383
- 372 42. Gil I, Segura S, Martínez-Escala E, et al. Dermoscopic and reflectance confocal microscopic features of exogenous ochronosis. *Arch Dermatol.* 2010;146:1021–1025. 384
- 373 43. Sonthalia S, Sarkar R, Neema S. Maturational hyperpigmentation: clinico-dermoscopic and histopathological profile of a new cutaneous marker of metabolic syndrome. *Pigment Int.* 2018;5:54. 385
- 374 44. Sonthalia S, Agrawal M, Sharma P, Pandey A. Maturational hyperpigmentation: cutaneous marker of metabolic syndrome. *Dermatol Pract Concept.* 2020;10:e2020046. 386
- 375 45. Elmas ÖF, Acar EM, Kilitçi A. Dermoscopic diagnosis of ashy dermatosis: a retrospective study. *Indian Dermatol Online J.* 2019;10:639–643. 387
- 376 46. Alchorne MMda, Conceição KdC, Barraza LL, Milanez Morgado de Abreu MA. Dermatology in black skin. *An Bras Dermatol.* 2024;99:327–341. 388
- 377 389