

diagnosis.⁵ Although these findings are not seen frequently, but when present, they are highly suggestive of melanoma.⁷ Milky-red areas correlate with a positive predictive value of 77.8%.⁷ However, they can also be seen in basal cell carcinoma, Spitz nevus, pyogenic granuloma and even in inflammatory lesions. Cutaneous melanoma metastasis and non-melanoma skin cancer should be considered in the differential diagnosis of polymorphous and corkscrew vessels.⁷ In our opinion and experience, dermoscopic features described in amelanotic melanoma at other locations are noted to be present in amelanotic subungual melanoma also. However, more reports are necessary to substantiate this finding. Dermoscopic examination is of utmost importance in diagnosing early stages of subungual melanoma.

Onychomycosis, trauma, subungual hematoma, onychodystrophy and pyogenic granuloma are other differentials that can cause delay in the diagnosis and worsening of the prognosis. In the present case, the patient consulted for a contact dermatitis which masked the subjacent subungual melanoma hidden by the use of artificial nails. In recent years, there has been an increase in the use of artificial nails resulting in a higher incidence of contact dermatitis.⁸

In conclusion, subungual melanoma is a diagnostic challenge with crucial prognostic implications. A considerable proportion of cases are amelanotic which makes the diagnosis even more difficult. Dermoscopic examination, showing polymorphic and irregular vessels, may be extremely helpful. Contact dermatitis mask its presentation, particularly in users of artificial nails and it is of vital importance to make the general population aware that any destruction of the nail plate should be evaluated by a dermatologist before covering it with an artificial nail.

Declaration of patient consent

The patient's consent is not required as the patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

**Ignasi Marti-Marti, Ramon Pigem,
Maria Margarita Narvaez, Llúcia Alós¹,
Susana Puig**

Departments of Dermatology, ¹Pathology, Melanoma Unit, Hospital Clínic, Universitat de Barcelona, Barcelona, Spain

Corresponding author:

Prof. Susana Puig,
Department of Dermatology, Hospital Clínic de Barcelona, Carrer de
Villarroel, Barcelona, Spain.
spuig@clinic.cat/susipuig@gmail.com

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Dermoscopic features of clofazimine-induced pigmentation in a borderline tuberculoid leprosy plaque

Sir,

A 30-year-old male diagnosed with borderline tuberculoid Hansen disease presented with a new-onset redness over the preexisting lesion since one month. It was not associated with

pain or any other systemic complaints. He was on multibacillary multidrug therapy, consisting of rifampicin, dapsone and clofazimine, for the past three months. Cutaneous examination showed a solitary, well-defined, dusky erythematous non-tender

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plaque on the right side of the forehead extending up to the upper eyelid [Figure 1]. Other mucocutaneous, neuromuscular, general and systemic examinations were within normal limits. Dermoscopy under polarized mode (Dermlite, DL4, ×10) demonstrated a blanchable erythema and blue-gray peppering in a reticular or hexagonal pattern [Figure 2]. The surrounding skin did not have any pigmented structures. The diagnoses of clofazimine-induced pigmentation and type I lepra reaction were considered. Histology demonstrated a normal looking epidermis with fine and coarse brown granular pigment within macrophages, including occasional foamy ones. In addition, freely lying pigment was also noticed in the dermis, along with loose epithelioid granulomas and lymphohistiocytic infiltration [Figures 3a and b]. Fite stain was negative for acid-fast bacilli. Special stains for melanin and iron did not stain the dermal deposits. The diagnosis of clofazimine-induced pigmentation was made. The patient was counseled about the benign nature of the condition.

Clofazimine, a weakly basic lipophilic phenazine antibiotic, is a common cause of drug-induced pigmentation, especially in leprosy patients. It produces a pink to red discoloration of skin during the initial period, followed by a typical dark brown pigmentation after a few months. The pigmentation persists throughout the period of drug intake and fades gradually after the discontinuation of clofazimine.¹ The exact nature of the material responsible for the discoloration is still a topic of debate. The initial reddish to reddish-blue color is thought to be due to the clofazimine (a reddish-blue aniline dye) accumulation. The dark brown color is due to drug-induced ceroid lipofuscinosis.² It stains negative for melanin and iron.¹ Although clofazimine can produce a generalized pigmentation, it usually involves leprosy-affected skin.¹ The

selective discoloration of the borderline tuberculoid leprosy plaque and sparing of other body parts is due to the selective uptake of clofazimine in the borderline tuberculoid plaque.³

The initial red color imparted by clofazimine can have cosmetic implications, especially in light-skinned individuals, and can mimic type I lepra reaction, as in the index case. Clinically, the absence of pain and tenderness over the plaque and neuritis can help rule out type I lepra reaction. However, the subjective nature of pain and self-administration of over-the-counter analgesics or oral corticosteroids can suppress the signs and symptoms, making the diagnosis difficult.

In our case, the demonstration of blanchable erythema and blue-gray peppering in a reticular or hexagonal pattern provided a valuable clue to the diagnosis of clofazimine-induced pigmentation. Dermoscopically, clofazimine-induced pigmentation shows a black background, honeycomb pattern and yellow to white globules.⁴ The dermoscopic features described for the type I reaction are yellowish-orange area, erythematous background and out-of-focus short linear vessels.⁵ Other dermoscopic differentials are fixed drug eruption (grouped brown, gray to steel blue dots) and lichen planus pigmentosus (diffuse brown color, pseudopigment network, slate gray to blue dots and globules, hem-like pigment pattern and periadnexal brown to blue-gray pigment).^{6,7}

On histopathology, a type I reaction will show loose epithelioid granulomas, dermal edema, dilated vessels and increased lymphocyte infiltration with or without necrosis within the granuloma. Clofazimine-induced pigmentation shows increased epidermal melanin along with a selective accumulation of lipophilic clofazimine within the histiocytes along with ceroid lipofuscin.^{2,8}



Figure 1: Solitary, well-defined dusky erythematous plaque on the right side of the forehead

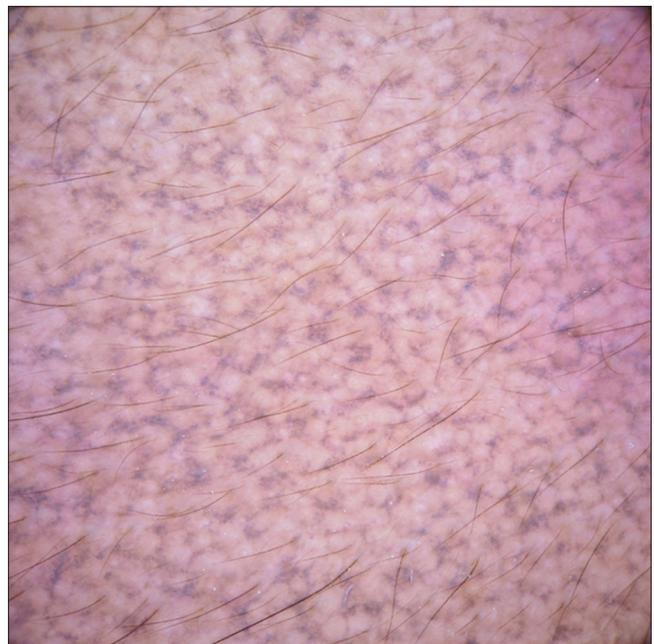


Figure 2: Dermoscopy (Dermlite, DL4, ×10, polarized mode) demonstrates blue-gray peppering in a reticular or hexagonal pattern

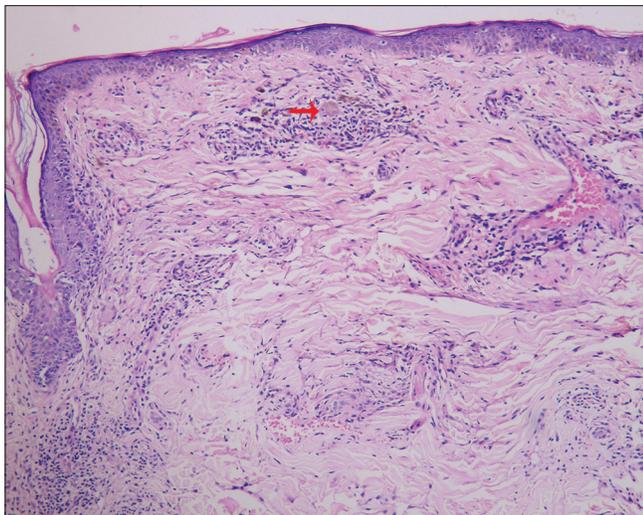


Figure 3a: Histology shows the presence of brown granular pigment within macrophages, including foamy one (arrow). (H and E, ×100)

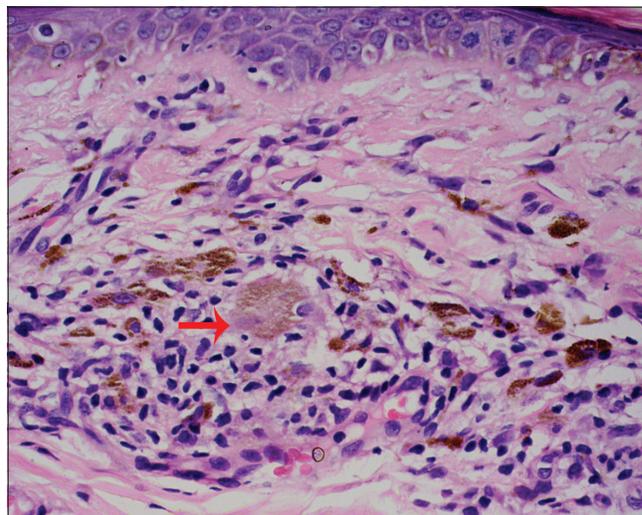


Figure 3b: Histology shows perivascular lymphohistiocytic infiltration and fine and coarse brown granular pigment within macrophages. Note the free-lying dermal pigment and fine granular pigment within the foamy macrophage (arrow) (H & E, ×400)

In conclusion, we report the dermoscopic features of clofazimine-induced pigmentation of a leprosy plaque. In the future, a comparative dermoscopic study may be able to differentiate between clofazimine-induced pigmentation and its clinical mimics.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

**Biswanath Behera, Aparna Palit¹,
Madhusmita Sethy², Ashish Kumar Nayak,
Siddhartha Dash, Pavithra Ayyanar¹**

Department of Dermatology and Venereology, All India Institute of Medical Sciences, Bhubaneswar, Odisha, India, ¹Department of Dermatology, AIIMS, Kalyani, Basantapur, West Bengal, ²Department of Pathology, All India Institute of Medical Sciences, Bhubaneswar, Odisha India

Corresponding author:

Prof. (Dr.) Aparna Palit,
Department of Dermatology, AIIMS, Kalyani, West Bengal, India.
apalit2011@gmail.com

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