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Purpuric Hypoesthetic Plaques: Localization of Pigmented Purpuric Dermatosis within Plaques of Borderline Tuberculoid Leprosy

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Dear Editor,

Atypical presentations of leprosy can delay diagnosis. We report a case of coexistence of borderline tuberculoid (BT) leprosy with pigmented purpuric dermatoses (PPD). The coexistence may be due to locus minoris resistentiae resulting from autonomic nerve damage leading to vasodilation and red blood cell extravasation.

A 42-year-old male presented with three asymptomatic erythematous skin lesions on his left ankle for 8 months. These lesions gradually changed color from bright red to dusky red and brown and slowly spread to involve the left ankle and adjacent lower leg. There was no history of pedal edema or varicose veins. Upon further questioning, he reported patchy, partial to complete sensory loss over the left ankle and lower leg, accompanied by xerosis and decreased sweating for 3 years, with no motor weakness. Cutaneous examination revealed three well-defined dusky erythematous plaques on the left lower leg and ankle. The largest plaque encircled the left ankle over a length of 10 cm, extending to the dorsum of the midfoot. Two smaller, circular plaques measuring 4 cm × 4 cm each were present proximally on the anteromedial aspect of the lower leg. These plaques had closely aggregated non-blanchable pinpoint dusky erythematous macules interspersed with apparently normal skin (FIG. 1). Dermoscopy showed multiple red dots, brown globules, and yellow-orange structureless areas (FIG. 2). The plaques were slightly xerotic without any palpable feeder nerve. There was over 90% sensory loss to pinprick and temperature sensations over the three macules, but no sensory loss over the sole and distal forefoot. The left common peroneal nerve was thickened with a firm, cord-like consistency, without any motor deficit. Differential diagnoses of pigmented purpuric dermatosis (PPD)

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and borderline tuberculoid (BT) leprosy were considered. Skin biopsy showed an oblong perivascular moderately dense infiltrate composed of tuberculoid granulomas with epithelioid cells, histiocytes, lymphocytes, and giant cells along with dilation of blood vessels, erythrocyte extravasation, and perivascular lymphohistiocytic infiltrate (FIG. 3). Acid-fast bacilli were not detected on Ziehl-Neelson staining from the biopsy or slit skin smear from the earlobes. Nerve conduction studies indicated pure sensory neuropathy involving the left sural and left superficial peroneal nerves. A venous Doppler of both lower limbs was unremarkable. A final diagnosis of coexistent borderline tuberculoid leprosy with pigmented purpuric dermatosis was made, and the patient was started on monthly rifampicin 600 mg and clofazimine 300 mg, along with daily dapsone 50 mg and clofazimine 50 mg for 6 months which resulted in disease resolution.

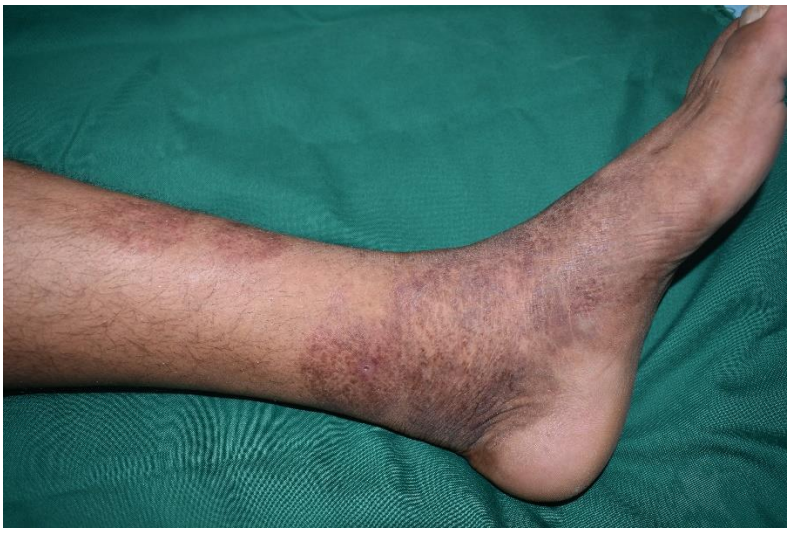

FIG. 1. **Well-defined dusky erythematous to dark brown plaques composed of multiple closely aggregated tiny purpuric papules over the left ankle and lower one-third of the left leg.**

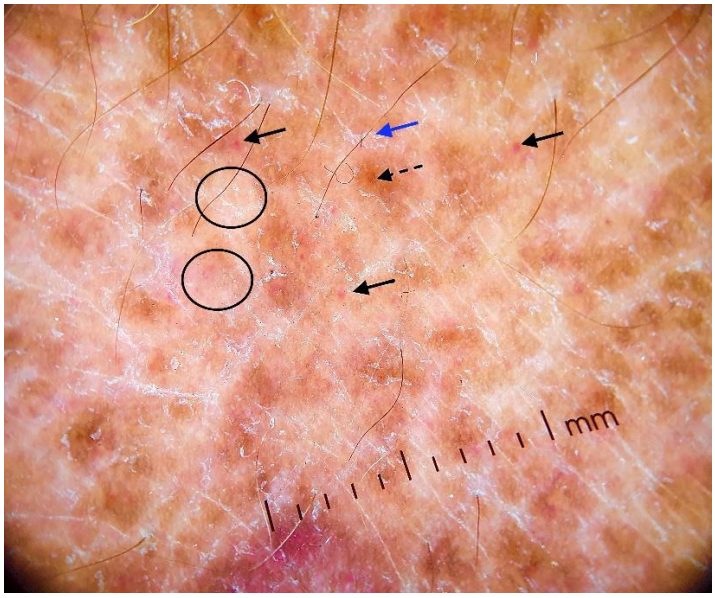

FIG. 2. **Dermoscopy [Heine DELTAone] showing red dots (thick solid arrow), brown globules (dotted arrow), white scales (blue arrow), yellow-orange structureless areas (10x, polarized).**

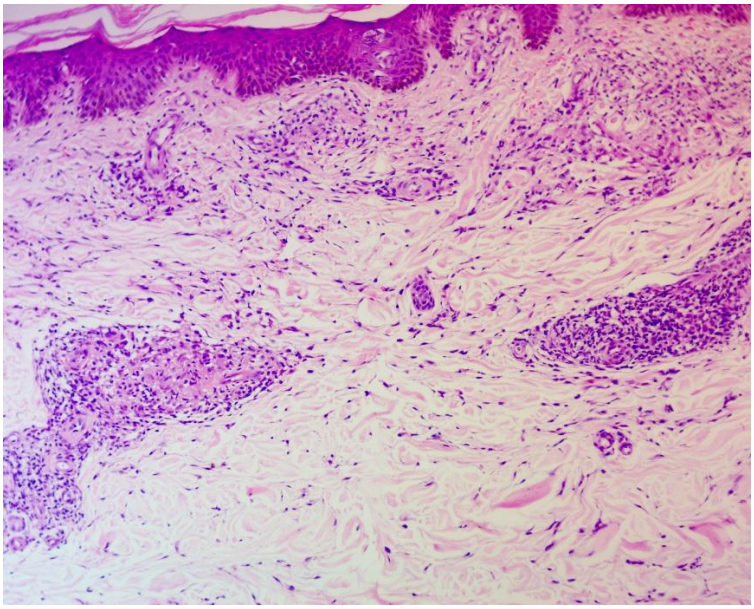

FIG. 3. **Histological section showing oblong perivascular moderately dense infiltrate composed of tuberculoid granulomas with epitheloid cells, histiocytes, lymphocytes, giant cells along with perivascular lymphohistiocytic infiltrate, erythrocyte extravasation, and hemosiderin deposition (x100, hematoxylin & eosin).**

Borderline tuberculoid leprosy is characterized by hypoesthetic, hypopigmented, xerotic patches with loss of appendages and decreased sweating, often associated with a palpable nerve known as the feeder nerve. It can become edematous and erythematous due to type 1 reaction. However, purpura is not typically a feature. Chronic use of topical steroids can cause atrophy, telangiectasia, and hypopigmentation, but the patient denied using any over-the-counter topical preparations and had no history of clofazimine intake. Atypical manifestations of leprosy are not uncommon and can often delay diagnosis [1]. We describe a rare occurrence of PPD within lesions of BT leprosy. PPD is characterized by asymptomatic purpuric macules on the lower legs and feet, which typically start insidiously and progress gradually. This condition is not associated with sensory loss, xerosis, or hypopigmentation [2]. Chronic venous hypertension can lead to pedal edema, extravasation of red blood cells, stasis dermatitis, and in advanced stages, venous ulcers and lipodermatosclerosis on the lower legs, but it is not associated with sensory loss [3]. Our patient had no history of prolonged standing or signs of varicose veins, chronic stasis, or venous hypertension. Our case presented with three hypoesthetic patches on the leg associated with purpuric macules, histological changes of both PPD and BT leprosy, and local sensory loss on nerve conduction studies. Dermoscopy was helpful in arriving at a diagnosis. The red globules correspond to erythrocyte extravasation, the brown globules correspond to hemosiderin deposition and the yellow-orange areas correspond to granulomas on histology [4,5]. Notably, the purpuric macules co-localized with the hypoesthetic patches, a phenomenon that is challenging to explain. Locus minoris resistentiae refers to a specific area of the body that is more susceptible to disease compared to other regions. The preferential co-localization of skin lesions on previously damaged skin, highlights a classic example of locus minoris resistentiae. The Köbner phenomenon, which involves the emergence of new lesions associated with an existing skin condition at sites of trauma or irritation, serves as a prominent illustration of this concept in dermatology. The underlying causes of this phenomenon are varied and complex [4]. We hypothesize that autonomic nerve involvement in the patch may lead to vasodilation and subsequent extravasation of red blood cells, especially in the legs, resulting in purpuric macules [5]. The granulomatous variant of pigmented purpuric

dermatoses is characterized by a lymphohistiocytic infiltrate obscuring the dermoepidermal junction, loose granuloma formation in the superficial dermis, and extravasated erythrocytes on histopathological examination [6]. However, lesional sensory loss and xerosis are difficult to explain. The patient had ignored the hypoesthetic patches for 3 years, but the new onset of purpuric macules prompted him to seek medical attention. A routine examination could have led to a dismissal as pigmented purpuric dermatoses. However, detailed clinical history and examination were crucial for the diagnosis. Dermoscopy, skin biopsy and nerve conduction studies confirmed the diagnosis of borderline tuberculoid leprosy [7,8].

1. Declaration of Patient Consent

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

2. Financial Support And Sponsorship

Nil.

3. Conflicts of Interest

There are no conflicts of interest.

4. Use of Artificial Intelligence (AI)–Assisted Technology for Manuscript Preparation

The authors confirm that there was no use of artificial intelligence (AI)–assisted technology for assisting in the writing or editing of the manuscript, and no images were manipulated using AI.

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