

CosmoDerma





Letter to the Editor

Lucio phenomenon – An enigma

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Received: 09 August 2023 Accepted: 28 August 2023 Published: 14 September 2023

DOI 10.25259/CSDM_137_2023

Quick Response Code:



Dear Sir,

Hansen's disease is a chronic infection caused by Mycobacterium leprae, characterized by a prolonged course with intermittent acute immune-mediated processes called as lepra reactions. Lucio leprosy is a naïve and diffuse form of lepromatous leprosy and the Lucio phenomenon (LP) is one of the reactional states seen in Lucio leprosy; first defined by Lucio and Alvarado in Mexico in 1852. Lucio leprosy was initially considered an endemic process in Mexico and Central America until few sporadic cases were reported from non-endemic areas of the world, including countries in Southeast Asia.[1] LP is difficult to diagnose, mainly in non-endemic areas which can lead to a delay in its diagnosis and management, so hereby, we present a case of LP from North India with classical clinical presentation and histopathology.

A young male presented with multiple cutaneous ulcers of 2 weeks duration which started as purpuric macules over forearms and legs followed by buttocks, trunk, and knees with subsequent ulceration over the next 2-3 days. There was no history of associated systemic features or treatment for Hansen's disease. Examination revealed diffuse infiltration of face and ear lobules with ciliary and supraciliary madarosis and extensive deep, bizarrely shaped, and punchedout ulcers with polycyclic margins, predominantly over extensor aspects of extremities and buttocks [Figure 1a and b]. Superficial depigmented atrophic scarring at sites of healing of prior ulcers was noted. Bilateral ulnar and radial cutaneous nerves were thickened and non-tender. Based on the clinical presentation, differential diagnoses of LP and vasculonecrotic erythema nodosum leprosum (ENL) were considered. Slit-skin smear showed bacteriological index of 5+ and morphological index of 80%. Histopathology examination from the ulcer margin over the right thigh revealed multiple foamy macrophages containing clusters of lepra bacilli (H and E stain ×40) [Figure 2a]. Special staining (Fite-Faraco) showed pinkish clusters of lepra bacilli clogging dermal vessels and seen inside endothelial cells (×1000) [Figure 2b]. Based on clinical, microbiological, and histopathological findings, a diagnosis of lepromatous leprosy with LP was made. The patient was started on multibacillary multidrug therapy with partial healing of ulcers with hypochromic scars on the next follow-up after 1 month.

Vasculonecrotic reactions in leprosy include LP and vasculonecrotic ENL.[2] LP is seen in untreated or incompletely treated, diffuse, and infiltrative non-nodular form of leprosy known as Lucio leprosy or Lepra bonita (Spanish for "pretty leprosy" due to the shiny infiltrated appearance of the skin seen in this form).[1] Although, LP can also be seen in classic nodular form of lepromatous and borderline leprosy.^[3] LP is characterized by painful purpuric lesions that develop into welldefined, multiangulated, and jagged ulcerations with a geometric shape involving - in descending order of frequency - the feet, legs, hands, forearms, thighs, arms, and rarely, the trunk and face.

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Figure 1: (a) Clinical image shows multiple serpiginous well-defined ulcers over bilateral knees, (b) Clinical image shows multiple well-defined deep geographical ulcers over bilateral thighs and knees.

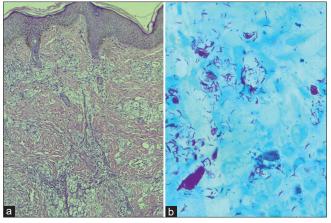


Figure 2: (a) Hematoxylin and eosin staining showing multiple foamy macrophages containing clusters of lepra bacilli (×40). (b) Fite-Faraco staining showing pinkish clusters of lepra bacilli clogging dermal vessels and seen inside endothelial cells (×1000).

Ulcers usually heal in about 2-8 weeks, leaving curvilinear jagged atrophic hypopigmented scars. Associated fever, constitutional symptoms, systemic involvement, and neuritis are usually absent as opposed to ENL. Histological features are colonization of the endothelial cells by acid-fast bacilli; endothelial proliferation of vessels of the mid-dermis and neutrophilic infiltration, ischemic epidermal necrosis, and necrotizing vasculitis of the small vessels of the superficial dermis.[3] LP might be confused with vasculonecrotic ENL, which presents as painful necrotic ulcers with constitutional symptoms, neuritis, and commonly visceral involvement.[4] Hence, based on distinctive clinical and histopathological features, these two can be differentiated. For management, systemic steroids are usually not indicated for LP and it is unresponsive to thalidomide as opposed to ENL.[1]

Declaration of patient consent

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

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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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How to cite this article: Sangwan P, Yadav P, Bagri M, Hazarika N. Lucio phenomenon - An enigma. CosmoDerma 2023;3:121.