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Antiphospholipid antibodies in a patient of Lucio phenomenon presenting with the gangrene of digits

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Sir,

Lucio leprosy is a diffuse form of lepromatous leprosy, commonly seen in Mexico and Costa Rica, not infrequent in the Gulf Coast, but quite rare in the rest of the world.[1] Lucio leprosy presents as a slowly progressive, diffuse infiltration of the skin of the face and most of the body without any previous evidence of discrete lesions. The disease is often unmasked by a specific severe reaction state, the Lucio phenomenon—presenting clinically as multiple, well-defined, angular jagged purpuric lesions evolving into massive ulcerations. The antiphospholipid antibodies were originally detected and used by Wasserman for the diagnosis of syphilis. It was subsequently found that antiphospholipid antibodies are not only specific for syphilis but are also found in autoimmune diseases such as systemic lupus erythematosus and with other infections.[2] Here we present a case of Lucio phenomenon in a patient of Lucio leprosy with positive antiphospholipid antibodies presenting with digital gangrene.

A 45-year-old woman was referred to the dermatology department for sudden blackening of toes and fingers for 6 days along with fever and arthralgia for the last 8 days. There was no clinically relevant significant past, personal and obstetric history. Menstrual history was unremarkable. Systemic examination was normal except for mild splenomegaly. Bilateral pitting edema was present with tense, dry, xerotic skin of both the lower limbs. Blackish discoloration of the left 3rd, 4th, 5th toes, right great toe and right 3rd,4th 5th toes extending from the tip of toes to metatarsophalangeal joints, with similar blackish discoloration of the right ring finger extending from tip of the finger to distal interphalangeal joint on the palmar aspect of the hand were seen. Affected areas were hard, non-tender and cold on palpation, suggesting established gangrene. Multiple livedo racemosa-like lesions were present involving the bilateral lower limb and trunk. Multiple angulated purpuric necrotic lesions with surrounding ill-defined erythema were present on the left thigh with one lesion showing ulcerative changes. The patient had diffuse erythema of face with difficult to pinch tense shiny skin with complete loss of bilateral eyebrows and eyelashes. There was no infiltration of the ear lobes [Figure 1a-c].

Bilateral infraorbital, left ulnar, bilateral radial, left common peroneal and left posterior tibial nerves were thickened and tender. Temperature sensations were impaired on the face and were completely absent in all limbs. Fine touch sensation was lost in the dorsal aspect of bilateral hands and lower limbs. Arterial pulses were palpable and normal in all the limbs.

Relevant laboratory and radiological investigations are mentioned in Table 1.

Slit-skin smear examination from earlobes, eyebrows, normal looking skin of cheek and scraping from nasal mucosa

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Figure 1a: Erythematous shiny facial skin with the complete loss of eyelashes and eyebrows

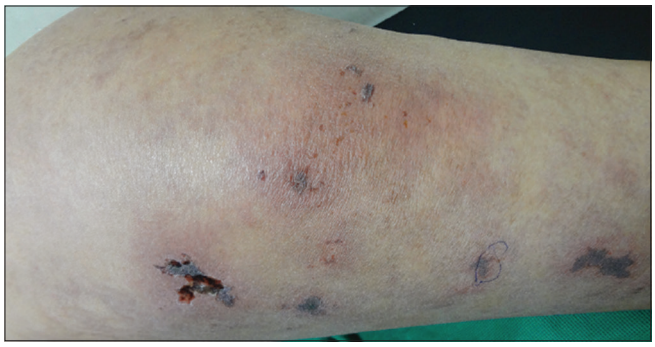

Figure 1b: Multiple angulated necrotic lesions with ulceration

revealed 90% solid staining acid-fast bacilli with globi formation and mean bacterial index of 4+. Polymerase chain reaction study from skin biopsy isolated *Mycobacterium leprae*. Histopathological examination of a skin biopsy from the ulcer and from adjacent normal skin showed perineural and periadnexal foamy macrophages and Virchow's cell with numerous bacilli; multiple superficial vessels showing signs of vasculitis, mild lobular panniculitis, a large subcutaneous vessel invaded by foamy macrophages and the presence of lepra bacilli in endothelial cells as well as in the lumen (characteristic of Lucio phenomenon). Multiple vessels in mid dermis showing the presence of thrombi in the absence of signs of vasculitis typical of occlusive vasculopathy were seen [Figures 2a-c and 3a-c].

The case was diagnosed as Lucio leprosy with Lucio phenomena having antiphospholipid antibodies. Standard anti-leprosy treatment recommended by the World Health

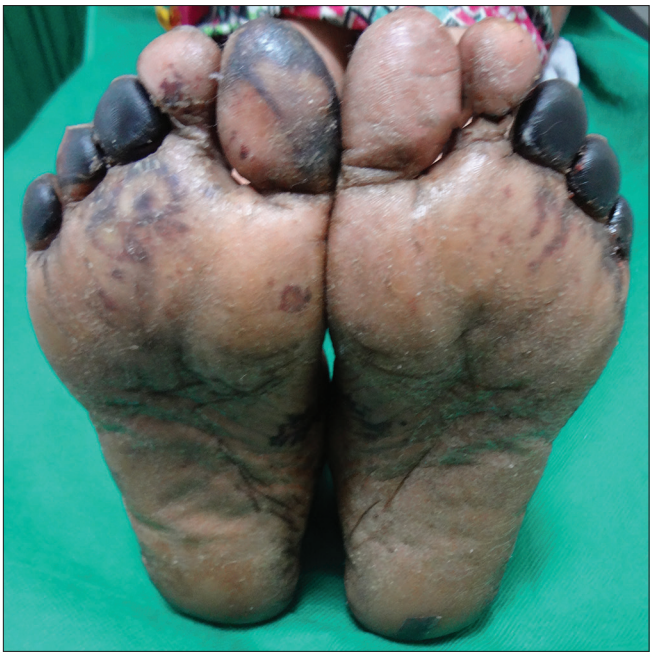

Figure 1c: Gangrene involving multiple toes

Table 1: Relevant findings of investigations

Investigation	Patient value
Hemoglobin	11.5 g/dL
Total leukocyte count	14400/mm^3
Erythrocyte sedimentation rate	32 mm/h
Prothrombin time	*P*=14.8, C-12.8 INR-1.16
Activated partial thromboplastin time	*P*=31.5, C-29.6 s
Random blood sugar	120 mg/dL
Serological screening for HIV, hepatitis B, hepatitis C, syphilis, ANA, ANCA, RA factor	Negative
24-h urine protein excretion	841 mg/24 h
D-dimer	800 mg/mL
aPL	IgG=48.3 GPL U/mL (0-10) IgM=156 MPL U/mL (0-10)
aCL	IgG=56.6 GPL (>15 positive) IgM=>255 MPL (>15 positive)
ß2GPI	IgG=3.0 U/mL (>16 positive) IgM=>100 U/mL (>16 positive)
DRVVT screen (LA1)	85.9 s (31-33 s)
DRVVT confirm (LA2)	48.6 s (31-33 s)
DRVVT mixing (1:1 with PNP)	51.3 s (31-33 s)
DRVVT confirm (LA1/LA2)	1.77 s (0.8-1.2 s)
APTT screen	54.4 S (31.4-43.4 s)
APTT mixing	40.1 s (31.4-43.4 s)
X-ray chest, ECG, echocardiography, arterial and venous Doppler study of extremities	No abnormality detected
Pulmonary and brain CT angiography study	No abnormality detected

ANA=antinuclear antibodies, ANCA=antinuclear cytoplasmic antibodies, DRVVT=diluted Russel viper venom test, APTT=activated partial thromboplastin time, ECG=electrocardiogram, CT=computed tomography, RA=rheumatoid arthritis, aPL=antiphospholipid antibodies, aCL=anticardiolipn, ß$_2$GPI=beta-2 glycoprotein 1, IgG=immunoglobulin G, IgM=immunoglobulin M,

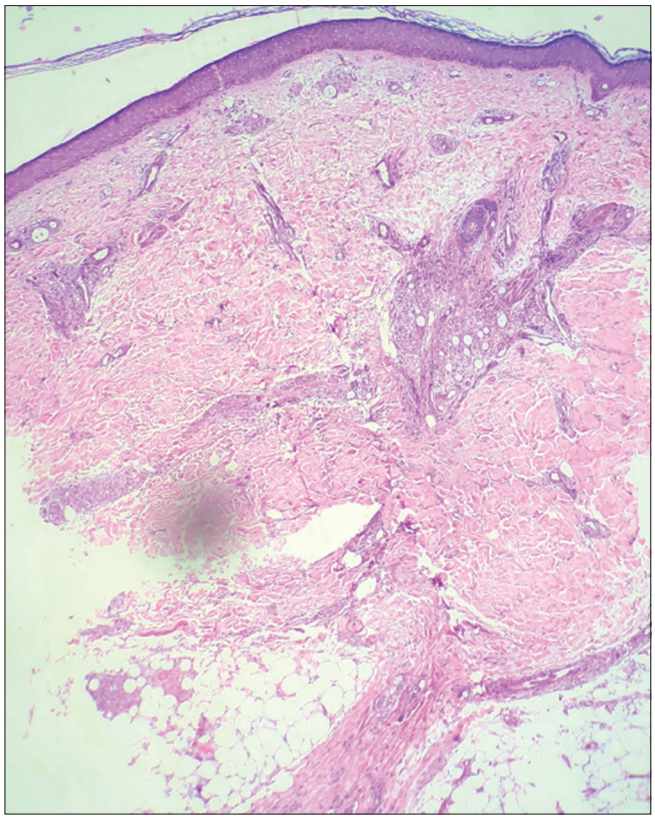

Figure 2a: Biopsy from normal-appearing skin on thigh showing atrophic epidermis with periappendageal and perineural infiltrate (H&E, ×40)

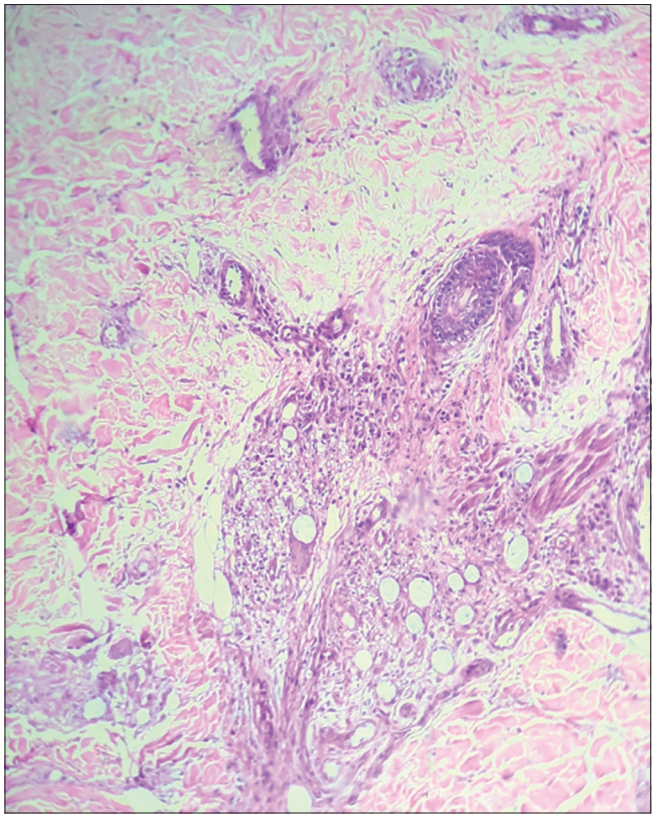

Figure 2b: Periappendageal and perineural infiltrate of foamy macrophages and Virchow's cells. (H&E, ×100)

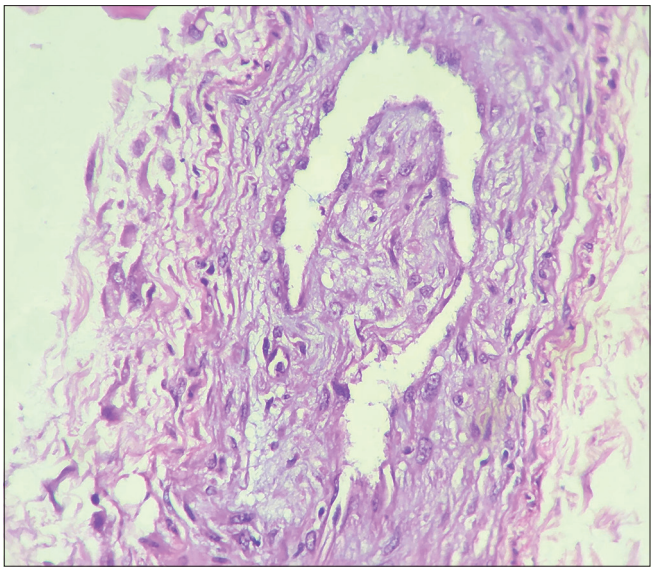

Figure 2c: Infiltrate of foamy macrophages invading medium-size vessel (H&E, ×400)

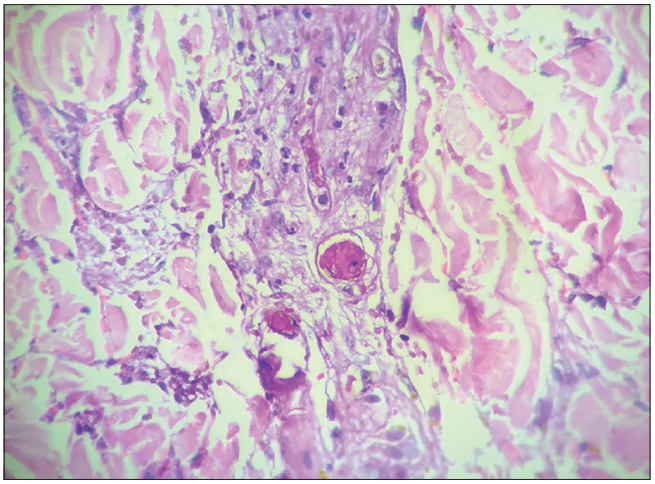

Figure 3a: Vessels in mid dermis showing thrombosis without signs of vasculitis (H&E, ×400)

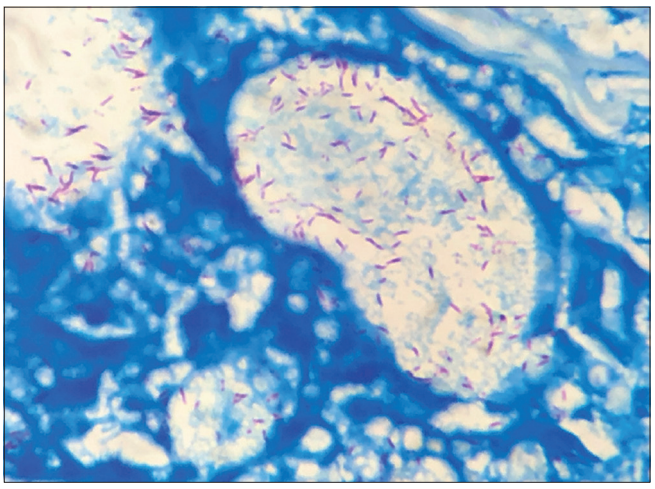

Figure 3b: Presence of lepra bacilli in Virchow's Cell (Fite Faraco stain, ×1000)

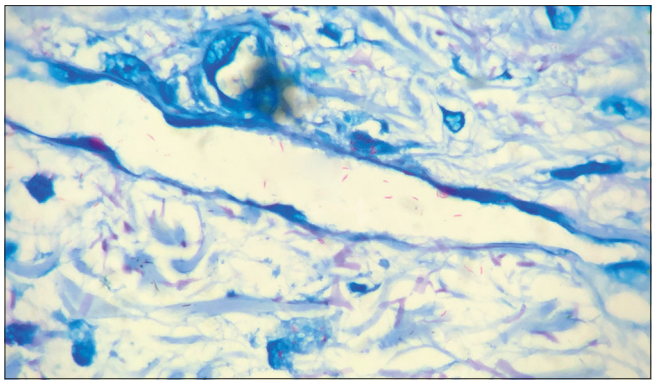

Figure 3c: Lepra bacilli invading vessel wall and vascular lumen (Fite Faraco stain, ×1000)

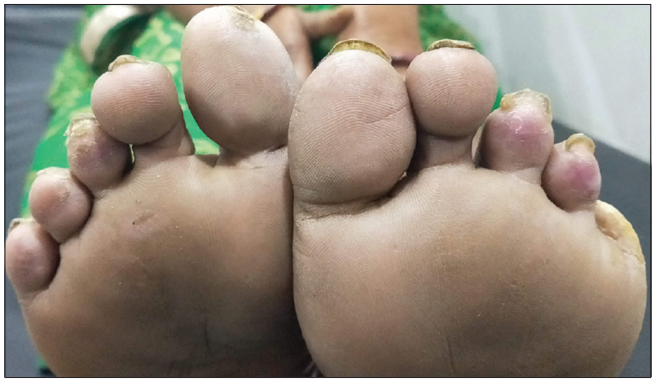

Figure 4: Complete resolution of gangrenous changes except left fifth toe (autoamputation) at 4-month follow-up

Organization (WHO) containing multidrug therapy of rifampicin, clofazimine and dapsone was initiated with oral prednisolone (1 mg/kg) for Lucio phenomenon. Low-molecular-weight heparin 60 mg subcutaneous once a day for 7 days was added and transitioned to oral warfarin keeping international normalization ratio between 2 and 3. At the time of submission of this report, the patient was on regular follow-up with complete resolution of ulcer and gangrene in all digits except the left fifth toe [Figure 4].

Our patient had non-nodular diffuse infiltration of skin with complete loss of eyebrows and eyelashes and shiny skin of face characteristic of Lucio-Latapi leprosy. Lucio phenomenon can be the initial presentation of the disease, presenting as widespread necrotic ulcerations.[3] Necrotic lesions of Lucio phenomenon can mimic cutaneous small or mixed vessel vasculitis clinically, which was true for our patient too. Livedo racemosa-like lesions and gangrene involving multiple digits, in this case, can be attributed to the thrombotic vasculopathy of antiphospholipid syndrome. Histologically, the Lucio phenomenon shows necrotizing panvasculitis with vascular proliferation and the presence of angio-invasion by lepra bacilli as was observed in our patient. A study from Mexico where Lucio leprosy is more prevalent, reported *Mycobacterium Lepromatosis* being isolated from such patients.[4] Polymerase chain reaction study from our patient isolated *M. Leprae* from the skin specimen. Previously, it was postulated that antiphospholipid antibodies associated with infections do not possess *anti*-beta-2 glycoprotein I (anti-beta-2 glycoprotein I) activity, therefore, are not associated with thrombosis.[5] de Larrañaga *et al* found 56.8% patients of leprosy have beta-2 glycoprotein I (majority of immunoglobulin M class) activity without any symptoms, whereas another study found increased levels of anti-beta-2 glycoprotein I antibodies in significant proportion in patients of leprosy and its association with thrombosis.[6,7] Our patient, too, had an immunoglobulin M antibody to beta-2 glycoprotein with symptomatic thrombosis of digital arteries. Nunzie *et al.* and Wallin *et al* have reported similar cases of Lucio phenomenon with antiphospholipid syndrome.[8,9] Noteworthy is the fact that thromboembolic phenomenon is also a well-documented adverse event of thalidomide.[10] Our patient was not on any medication at the time of the presentation. Our case highlights the fact that Lucio leprosy and Lucio phenomenon are possible clinical presentations in Indian patients too.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal the identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Nayankumar H. Patel, Jignaben Krunal Padhiyar, Tejas Patel, Ani Patel, Aseem Chhibber, Ranjan Raval, Bhagirath Patel

Department of Dermatology, Venereology and Leprosy, GCS Medical College Hospital and Research Institute, Ahmedabad, Gujarat, India

Corresponding author:
Dr. Jignaben Krunal Padhiyar,
Room No. 35, Department of Dermatology, Venereology and Leprosy, GCS Medical College Hospital and Research Institute, Ahmedabad - 25, Gujarat, India.
dr.jignapadhiyar@gmail.com

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Targetoid bullous tinea corporis: Unusual presentation of a dermatophyte infection

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Sir,

Bullous lesions in tinea are uncommon and are usually seen in the setting of tinea pedis. Commonly reported causative fungal pathogens are *Trichophyton rubrum* and *Trichophyton mentagrophytes*.[1] We report a case of *Microsporum canis* causing targetoid bullous tinea corporis.

A 35-year-old woman gave a 6-day history of a generalised pruritic eruption involving the trunk and limbs. She had travelled to Kuantan, Malaysia and Bintan Island, Indonesia, in the past few weeks. One week prior to the onset of the rash, whilst in Kuantan, she had carried a stray kitten which scratched her on the neck. She reported no other contactants or medications prior to the eruption. She had no fever or mucosal involvement and was otherwise systemically well. Her past medical history was significant only for female pattern hair loss and alopecia areata, which was quiescent. On examination, multiple umbilicated targetoid papulovesicular lesions were seen over the neck, trunk, upper and lower limbs [Figure 1a-c]. The palms, soles, oral and conjunctival mucosae were not involved. She did not have any lymphadenopathy.

The differential diagnoses considered were erythema multiforme, syphilis, ecthyma, orf and cat scratch disease. A full blood count was unremarkable and rapid plasma reagin was negative.

Histology from the edge of a left forearm blister showed subcorneal neutrophils, spongiosis of the epidermis with neutrophilic exocytosis and prominent upper dermal oedema resulting in subepidermal pseudovesiculation, as well as septate fungal hyphae within the stratum corneum, which were seen on periodic acid–Schiff and Gomori methenamine silver stain [Figures 2a and b].

She was diagnosed with bullous tinea corporis and commenced on oral terbinafine 250mg daily. Her lesions completely cleared in 3 weeks [Figures 3a and b]. Cultures from the biopsy later returned positive for *M. canis*. *M. canis* is a zoophilic fungus, which usually causes

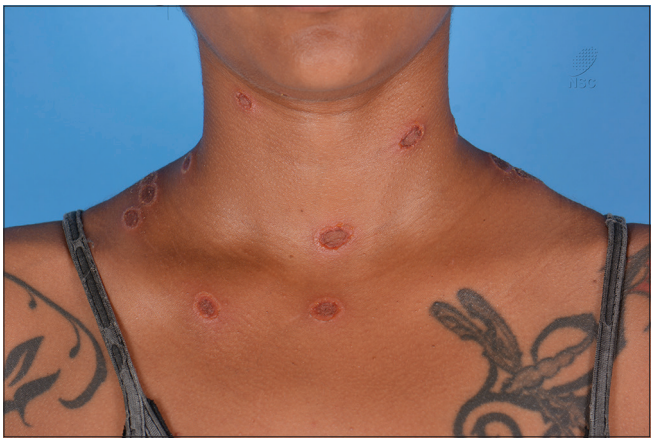

Figure 1a: Multiple umbilicated targetoid papulovesicular lesions over the neck and trunk

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