Lucio’s phenomenon in Lucio’s leprosy: A rare case report
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ABSTRACT
Lucio’s leprosy, a non-nodular form of lepromatous leprosy, appears as uniform diffuse shiny infiltration of the entire skin. Lucio’s Phenomenon, described by Lucio and Alvarado and reported mainly in Mexico and South America, is an rare unusual ulcerative immune complex mediated vasculitis occurring in patients of non-nodular form of leprosy. Here we report a case of Lucio’s phenomenon in a 65 year old female patient having Lucio’s leprosy.

Keywords: Lucio, phenomenon, leprosy

INTRODUCTION
Lucio’s phenomenon was described by Lucio and Alvarado in 1852 in Mexico, and was so named in 1948 by Latapi and Zamora. It represents a variant of lepromatous reaction type 2, which forms pure and primitive lepromatosis and manifesting clinically as multiple, irregular-shaped, angulated, deep ulcers covered with necrotic eschar and histopathologically is characterized by acute necrotic vasculitis, being synonymous with the expression "necrotizing erythema". Its main characteristic is a diffuse cutaneous infiltration, without nodule formation, generating a brilliant, moist and myxedematous complexion, imparting a healthy aspect to the patient. As a result of these characteristics it was also called "Pretty Leprosy". It is a relatively rare, peculiar reaction pattern occurring in untreated lepromatous (LL) or borderline lepromatous (BL) leprosy cases. We herewith present a case of Lucio Phenomenon in a 65 year old female.

CASE REPORT
A 65 year old female, housewife, came to our dermatology OPD with multiple large ulceration and thick crusting over both arms, forearms, hands, thighs, legs and feet, trunk and face with duration of 15 days. Initially erythematous, elevated, painful lesion started over the left great toe, followed by involvement of lower extremities, upper extremities, trunk which later rapidly developed into painless necrotic ulcers within two days. Diffuse cutaneous infiltration of facial skin was present [Figure 1]. Oedema feet was present. There was no history of fever, arthralgia or any other constitutional symptoms in the past few days. Past history of hypertension was present since eight years. Diabetes mellitus was diagnosed on admission. On cutaneous examination, there were multiple, symmetrical, irregular shaped, angulated, deep vasculitic necrotic ulcers covered with eschar over both the extremities, face, earlobes, palms and soles [Figure 2]. Besides this, there was loss of lateral one-third of eyebrows. The peripheral nerves were thickened and non-tender. Bilateral glove and stocking anaesthesia was present. Sput skin smear revealed abundant acid fast bacilli [Figure 3 (A)]. FF stain was positive showing abundant acid fast bacilli [Figure 3 (B)]. With the above findings, a provisional diagnosis of Lucio leprosy was made. A differential diagnosis of Lucio Phenomenon, vasculitis, ecthymagangrenosum and calciphylaxis was considered. Initially hemogram was normal. Erythrocyte sedimentation rate was 19 in the first hour. Liver function test, renal functions test and urine examination were normal. Serum HIV and RPR were non-reactive. Fasting and post prandial blood sugar was raised. Smears and cultures for fungus, and mycobacterium tuberculosis obtained from the ulcers were negative. Bacterial culture of pus from lesions revealed a growth of E.coli. Chest X-ray was normal. USG abdomen showed fatty liver grade III. Biopsy showed focal ischaemic necrosis in epidermis with neutrophilic infiltrate. Dermis showed mixed inflammatory infiltrate consisting of lymphocytes, foamy macrophages, few plasma cells and neutrophils in periadnexal and perivascular regions. Few blood vessels showed leukocytoclastic vasculitis [Figure 4]. On the basis of typical clinical presentation of diffuse non-nodular leprosy presenting with multiple, irregular-shaped, angulated, deep vasculitic ulcers covered with necrotic eschar along with absence of fever or constitutional symptoms, the diagnosis of Lucio Phenomenon was considered as the most likely diagnosis and confirmed by histopathology. Patient was started on WHO MDT-MB anti-leprotic drugs consisting of rifampicin 600 mg and clofazimine 300 mg once a month, dapsone 100 mg daily and clofazimine 50 mg daily was initiated along with systemic antibiotics and prednisolone 1 mg/kg/day.

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Daily dressings of ulcers were done. There were no new lesions after the start of therapy.

**Figure 1:** (A) Diffuse thickened facial skin

**Figure 2:** Angulated superficial necrotic ulcers with eschars over legs.

**Figure 3:** (A) ZiehlNeelson stain showing acid fast bacilli in clusters; (B) FiteFaraco staining showing abundant acid fast bacilli.

**Figure 4:** Vasculitic changes within dermal blood vessels.

**DISCUSSION**

Lucio phenomenon (LP) is described as a necrotizing reactional picture that is present in patients with the pure and primitive form of diffuse Lucio leprosy that never evolves with papules, plaques or leprotic nodules. The necrotizing lesions that developed over this diffuse lepromatosis were called Lucio phenomenon eponymically and erythema necroticans (ENe) descriptively.\[^{4,5}\] It is a reactional state, different from erythema nodosum leprosum, affecting LL or BL patients. It is endemic in Mexico. Lucio phenomenon has been reported in the USA, Spain, South and Central America, including Brazil, and East and West Asia.\[^{4,6}\] Lucio phenomenon manifests three to four years after onset of the disease and is more common in untreated patients or in those receiving inadequate treatment.\[^{4,5,6}\] In a series of 10 patients, Lucio's phenomenon occurred before the diagnosis of leprosy was made.\[^{7}\] The lesions start off as painful purpuric macules or plaques, progress to ulcers, affecting in ascending order of frequency: feet, legs, hands, forearms, thighs, arms and, rarely, the trunk and face, leaving atrophic and stellar scars.\[^{2,6}\] Some lesions may begin as blisters. Features of the underlying lucio leprosy commonly described include diffuse thickening of facial skin, alopecia of eyebrows and eyelashes and distal anaesthesia. Cutaneous nodules are not seen. Patients are mostly afebrile. This phenomenon tends to disappear 6-8 weeks after the beginning of the treatment; subsequent reactional episodes present with erythema nodosum leprosum type lesions.\[^{7}\] The main differences between Lucio's phenomenon and erythema nodosum leprosum are that the former is an ulcerative reaction occurring in the absence of cutaneous nodules whereas the latter may occur in any type of lepromatous leprosy and usually present as tender cutaneous nodules and may be followed by ulceration. It is not known why nodules do not form in the diffuse type of lepromatous leprosy, but it may be due to absence of specific cell mediated immune responses. Lepra bacilli are much more in the lesions of Lucio’s phenomenon but constitutional signs are less severe.\[^{7}\] Our patient had a diffuse non-nodular form of lucio leprosy with glove and stocking anesthesia and madarosis. Patient had never been treated for Hansen’s disease in the past. Patient presented with necrotic angular infarcts without any constitutional symptoms, which evolved into deep ulcers with angulated margins. Histopathological changes were consistent with the diagnosis of Luciophenomenon. Treatment is as for other type 2 reactions in leprosy along with supportive care. The response to treatment has been reported as poor and associated with severe morbidity and occasionally be fatal.\[^{7}\] Our patient died of cardiogenic shock due to septicaemia. Lucio Phenomenon is very rarely reported from India despite the huge load of multibacillary cases. This could be due to lack of clinical suspicion or due to inconsistent features on histopathology.

**REFERENCES**


