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Case Report

A Rare Case of De Novo Ulcerative Lepromatous Leprosy

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Leprosy is a systemic chronic infectious disease caused by bacteria *Mycobacterium leprae* and *Mycobacterium lepromatosis*, mainly affecting the skin and peripheral nerves, and has a long incubation period. Leprosy can sometimes have uncommon clinical presentations, one of them is ulcerative manifestation. In such scenarios, a keen clinical suspicion of leprosy, especially in endemic areas, helps in early diagnosis and treatment, thereby reducing further complications. We hereby report a case of lepromatous leprosy with spontaneous ulceration, with high bacillary load and malnutrition as predisposing factors, which manifested clinically as ulcerative presentation of leprosy.

Keywords : Ulcerative Leprosy, Lepromatous Leprosy, Trophic Ulcer

Introduction

Leprosy, one of the ancient diseases of mankind, also known as Hansen's disease, is a chronic granulomatous infectious disease caused by bacteria, *Mycobacterium leprae* and *Mycobacterium lepromatosis*, primarily affecting the skin and nerves. The clinical spectrum of leprosy is wide and depending upon infected individual's immune status and genetics it has varied presentations (Sardana et al 2020). A keen clinical suspicion along with examination and laboratory investigations to confirm leprosy are of utmost importance, especially in patients with uncommon presentations. Cutaneous lesions of leprosy usually present as hypoesthetic, hypochromic macules, papules, plaques, and diffuse infiltration of the skin with alopecia and xerosis. Less frequent manifestations include blisters, hyperpigmented patches, verrucous lesions, zosteriform pattern, macrocheilia, anetoderma, histoid leprosy, lucio leprosy, pure neural leprosy, long-standing leg ulcers, non-trophic ulcers, or spontaneous skin ulcerations. Unfortunately, delayed diagnosis and treatment are still an issue in endemic poor resource settings and in non-endemic countries due to global migration (Trindade et al 2009). Ulcer is not a common feature in leprosy patients, except during reactional states, lucio's phenomenon or secondary to neuropathies (Miyashiro et al 2019). We hereby report a case of lepromatous leprosy with spontaneous ulceration, with high bacillary load, manifesting clinically as ulcerative leprosy.

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Fig 1 : Multiple ulcers with black colored crusts over dorsal aspect of hands and fingers.

Fig 2 : Multiple ulcers with black colored crusts over dorsal aspect of feet and toes, with bilateral pedal edema.

Fig 3 : Diffuse hyperpigmentation with xerosis of lower third of bilateral legs.

Fig 4 : Cachexic built of the patient indicating malnutrition.

Case Report

A 72-year-old male, farmer, presented to the emergency department, with complaints of multiple raw areas over his hands, legs and feet, associated with swelling for the past five days. These raw areas were developed following the spontaneous shedding of black-colored flakes, which were asymptomatic. He also reported asymptomatic patches with reduced sensation over both the feet for the past two years, along with a history of epistaxis 15 days prior to presentation. There was no history of crops of any red raised lesions, with constitutional symptoms, neither he had any sudden onset pain or weakness of the limbs.

Patient was examined in detail and important findings are shown in Figs. 1 to 5.

Fig 5 : Diffuse infiltration of eye-brows with furrowing, madarosis, saddle nose and thickened ear lobes.

On general examination, the patient appeared cachectic (Fig. 4), with a BMI of 15.2, classified as underweight. He had pallor and enlarged, non-tender, rubbery bilateral inguinal lymph nodes (six in number, measuring approximately 2cm x 1cm). Pitting pedal edema extending up to ankle joint was also noted.

Dermatological examination revealed multiple (15-20) painless, well-defined, ulcers ranging from 2-6 cm in diameter, with necrotic slough, overlying scab formation and surrounding hyperpigmentation present over dorsal aspect of both hands (Fig. 1), feet & lower legs (Fig. 2). Additionally, diffuse hyperpigmentation with xerosis of the lower limbs was observed (Fig. 3). Characteristic leprosy findings, including loss of lateral half of eyebrows (madarosis) and enlarged hanging ear lobes (Buddha ears) were present (Fig. 5). Dystrophic changes were noted in all nails.

Neurological examination revealed thickened, non-tender, bilateral ulnar and common peroneal nerves (Grade 2 thickening). Sensory examination showed loss of touch, pain and temperature sensations in both hands and feet. Motor examination revealed wasting of muscles over the thenar and hypothenar eminences, with weakness in finger adduction and abduction. The card test, pen test and book test were positive for both hands. There was clawing of the right little finger.

Laboratory investigations showed hemoglobin level of 7.4g/dL and a total leukocyte count of 11,500 cu.mm with neutrophilia. Slit skin smear analysis demonstrated a bacillary index of 5+ at all tested sites (eyebrows and earlobes). A histopathological examination of a biopsy from left lower leg lesion, showed foamy macrophages surrounding nerves and adnexa with globi of acid fast bacilli (Virchow cells) (Figs. 6, 7A, 7B, 8).

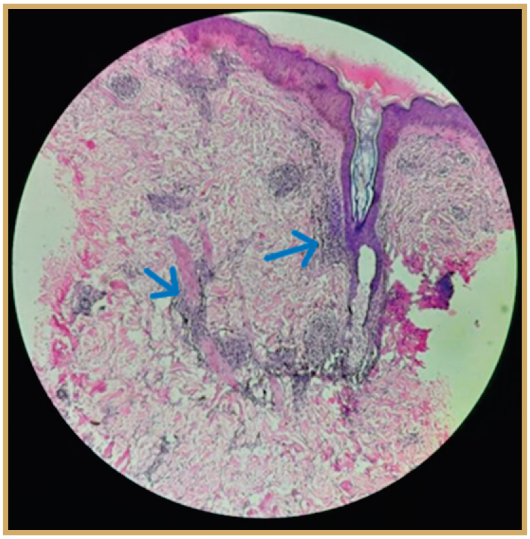

Fig. 6 : Low power (10 X)) magnification showing granulomas in dermis surrounding nerves and adnexa.

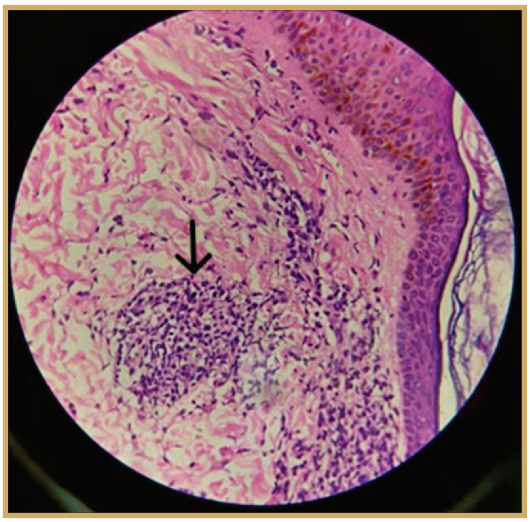

Fig. 7A : High power (40 X) showing granuloma in superficial dermis.

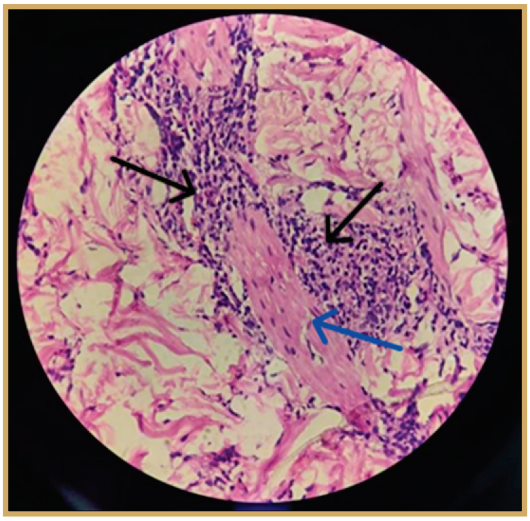

Fig 7B : High power (40X)) showing perineural location of granulomas.

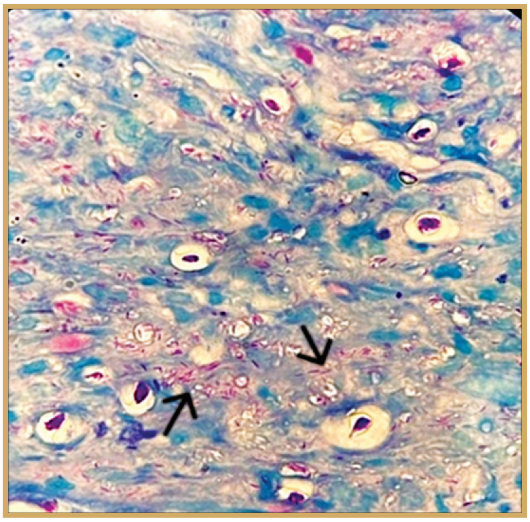

Fig 8 : ZN staining showing acid fast bacilli within macrophages.

Based on clinical, microbiological, and histopathological findings, a diagnosis of lepromatous leprosy with spontaneous ulceration was made.

The patient was initiated on multibacillary multidrug therapy (MB-MDT) without dapsone, along with systemic and topical antibiotics, leading to gradual ulcer healing. Dapsone was

omitted in view of anaemia (haemoglobin less than 10 g/dL). A physician referral was done to investigate and treat anaemia. After about 4 months of treatment with haematinics, dapsone was introduced once haemoglobin rose to 10 g/dL.

Discussion

In our case, the patient had lepromatous leprosy with spontaneous ulceration, along with high bacillary index and malnutrition as predisposing factor, and no features suggestive of type 1 or 2 lepra reaction, manifesting as ulcerative presentation of leprosy.

Two types of ulcers are seen in Hansen's disease, namely, trophic ulcers and leprous ulcers. Trophic ulcers are seen due to sensory and autonomic impairment, whereas leprous ulcers are seen due to alteration in the immune status of these patients or the excessive load of bacilli in these tissues. In tuberculoid pole, the major cause of lesional ulceration is Type 1 lepra reaction. In lepromatous pole, lesional ulceration is seen in erythema nodosum leprosum necroticans (Type 2 lepra reaction). In diffuse lepromatous leprosy, Lucio phenomenon (Type 3 Lepra reaction) can manifest with ulceration as acute, severe necrotizing vasculitis and thrombosis. Such findings were not seen in histopathology of our case, also there were no symptoms or signs suggestive of lepra reaction.

A study conducted at a single center in São Paulo, Brazil, reported eight patients of leprosy, who had specific cutaneous ulcers that were part of their main leprosy manifestation, but were not associated with reactional states, Lucio phenomenon or neuropathies. All patients had delayed diagnosis and seven patients had grade 2 of disability by the time of the diagnosis. Skin ulcers had improved rapidly after administration of MDT-MB, which explains the importance of timely detection of leprosy (Miyashiro et al 2019).

In lepromatous leprosy, there is a cellular infiltration with predominance of macrophages, which are associated with production of tumor necrosis factor (Miyashiro et al 2019). In addition, multibacillary patients, have enhanced oxidative stress (Reddy et al 2003), where reactive oxygen species may contribute to necrosis and ulceration. It remains to be established, why this occurs in only a subset of patients. In addition to *M. leprae, M. lepromatosis* has also been recognized as etiologic agent for leprosy. This species was initially associated with diffuse lepromatous leprosy as well as lepromatous leprosy (Han et al 2008). Whether the infection with *M. lepromatosis* is associated with specific clinical manifestations, including development of specific skin ulcers, is not yet known. Non-trophic ulcers in leprosy could also be caused by cutaneous vasculitis and small fiber involvement by lepra bacilli (Kesav et al 2013). A case of borderline lepromatous leprosy presenting with de-novo ulceration over bilateral lower extremities - a rare Lazarine-like presentation, has already been reported from India (Belgaumkar et al 2018). Awareness of such rare ulcerative manifestations is thus important for leprologists/ dermatologists involved in diagnosis and management of leprosy (Nanda et al 2004, Kesav et al 2013).

Conclusion

Leprosy has varied clinical presentations. This case highlights the importance of recognizing ulcers as a specific cutaneous manifestation of leprosy. Vigilant approach is crucial, especially in endemic countries like India, to identify the diverse manifestations of leprosy. It should be always confirmed with the help of microbiological and histopathological examinations. This is essential for ensuring timely diagnosis and treatment, avoiding development of disabilities and transmission of leprosy, particularly in cases where effective treatment is possible.

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