

Bullous lesion in lepromatous leprosy -A rare case with review of literature

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Introduction

Hansen's disease (or leprosy) is a chronic granulomatous infection caused by *Mycobacterium leprae*. The incidence is higher in developing countries. ¹ It runs a chronic, indolent course that can become complicated by acute, immune-mediated phenomena called lepra reactions.² The clinical severity of leprosy is dependent on the affected individual's degree of immunity to *Mycobacterium leprae*, as it is hypothesised that most immunocompetent people in endemic areas have mounted an immune response against it.³

Type 2 leprosy reaction is a severe multisystem immune-mediated complication of lepromatous leprosy and borderline leprosy that causes patients to become acutely ill with fever, malaise, and tender erythematous skin lesions.⁴ Erythema nodosum leprosum (ENL) typically occurs in lepromatous patients with abundant bacilli in cutaneous and peripheral nerve lesions within a few months of

initiating antibiotic treatment for leprosy.⁵ The pathophysiology is not clear.

Case Report

A 20-year-old married, malnourished female presented with fever, joint pain, multiple reddish skin lesions, pustules, and bullas, with extensive ulceration mainly affecting the extremities. On physical examination, the patient had pallor with bilateral pitting pedal oedema and huge tender bilateral submandibular, submental, and inguinal lymphnodes. A cutaneous examination revealed numerous bullous lesions, pustules with symmetrical purpuric macules and plaques, the majority with ulceration and necrotic slough in the center, mostly on the extensor of extremities, thigh, and gluteal region, at the time of admission (Figure 1). A fine needle aspiration cytology (FNAC) of the lymph node and pus for Gram stain and AFB were sent. FNAC from lymph node aspirates shows clumps of solid and broken bacteria. The pus from

the skin lesion was negative for Gram stain and showed acid-fast positive globi in the ZN stain. Then a diagnosis of LL with severe Type II reactions was made. The patient was admitted to the dermatology ward. On examination, we noticed two hypopigmented anaesthetic patches over the bilateral knee, symmetrical nerve thickening and tenderness, partial clawing of the right hand with atrophy of the small muscles of the hand, extensor of the foot with weakness, glove and stocking type hypoesthesia. A biopsy of skin lesions and a slit skin smear (SSS) for AFB were sent. The diagnosis was BT downgrading to LLs with a severe type 2 reaction.

Routine blood investigations reveal leucocytosis with elevated ESR. Histopathological examination showed multiple granulomas in the dermis and dermal vessels, showing ischemic necrotizing vasculitis with swelling of the endothelium and intraluminal accumulation of neutrophils and fibrin, or panniculitis. ZN stain showed clumps of acid-fast bacilli (AFB) periadenexally and within the macrophages, but not in the endothelium. A slit skin smear revealed a morphological index of 80% and a bacteriological index of 6+. Based on clinico-histopathological findings, a diagnosis of sub-polar lepromatous leprosy with type II reaction with bullous lesions was made. The patient was started on a multibacillary multidrug therapy (MB-MDT) regimen with the addition of prednisolone (1 mg/kg per day). After 4 weeks of treatment, the ulcers healed with hypertrophic scars (Figure 2). Due to the re-occurrence of type II reactions on tapering steroids, the patient was started on thalidomide

200mg twice daily for 7days, tapered by 100mg every four weeks until 100 mg, and continued on 100 mg every alternate day for 4 months. There was scarring with no recurrence of lesions during thalidomide treatment and during the subsequent 6-month follow-up. (Figure 3).

Figure 1: Photomicrograph: Before starting of treatment



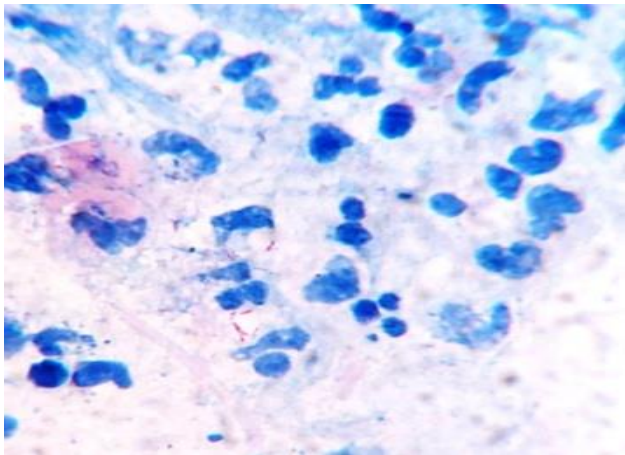
Figure 2: Photomicrograph: healed older lesion with new pustular lesions after 3 months (Recurrent Type 2 reaction)



Figure 3: Photomicrograph: lesions healed with scarring after treatment with MDT and thalidomide (no new lesion)



Figure 4: Photomicrograph: ZN stain of aspirate from lymphnode showing Acid fast bacilli



Discussion

Bullous lesions in LL are extremely rare and may occur during reactions to the Lucio phenomenon, lazarine leprosy, necrotizing ENL, autoimmune bullous disorder, and other causes of necrotizing vasculitis.⁶

Our patient showed multiple bullae and pustules with extensive ulceration but without any skin nodules. ZN staining of lymph node aspirate and skin bullae showed a high number of acid-fast bacilli with globi. The skin punch biopsy showed the classical histopathology of lepromatous leprosy. So our patient's presentation was atypical in that her skin lesions had bullae and pustules, but no nodules

were seen. Therefore, we diagnosed LL leprosy with severe type II reactions and bullous lesions.

The Lucio phenomenon is excluded in this case as it is encountered in non-treated cases.⁷ It begins as painful purpuric lesions that evolve into jagged ulcerations, heals with an atrophic scar, and has no constitutional symptoms.⁸⁻⁹ Vasculitis was excluded because of negative p-ANCA and c-ANCA.

In our case, the patient presented with necrotic oval ulcers, a bulla with constitutional symptoms, and neuritis, prompting the diagnosis of BT downgrading to LL with a severe type II reaction and a bullous skin lesion with vasculitis. After 4 weeks of treatment, the patient developed a hypertrophic scar and was placed on an antileprosy drug and steroid.

Conclusion

A bullous lesion is very rare in leprosy patients, and there is a high chance of a wrong diagnosis, thus delaying proper treatment. A high index of suspicion is very important for early diagnosis and prompt treatment, which can improve the disease outcome in areas not endemic for leprosy.

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Conflict of Interest

None.

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