

# Lepromatous (Multibacillary) Leprosy Complicated by Type 2 Lepra Reaction (Erythema Nodosum Leprosum) in a Young Adult from Tanzania: A Case Report

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Publication Date: 2026/10/12

**Abstract:** Lepromatous leprosy remains a significant public health challenge in endemic regions of East Africa. Here, we describe a 31-year-old previously healthy male artisan from the Tanga Region of Tanzania who presented with a one-year history of painless papulonodular lesions of the face and extremities, progressive hand edema, and unnoticed sensory loss that culminated in an accidental burn. Examination revealed diffuse facial infiltration, madarosis, bilaterally thickened nodular earlobes, multiple firm papulonodules, peripheral-nerve thickening, and sensory impairment. Ziehl–Neelsen-stained slit-skin smears demonstrated numerous acid-fast bacilli, confirming multibacillary (lepromatous) leprosy by World Health Organization (WHO) operational criteria. Concurrent features of type 2 lepra reaction (erythema nodosum leprosum, ENL) were present. The patient received the standard 12-month WHO multidrug therapy (MDT) regimen for multibacillary disease, together with a tapering course of oral corticosteroids. Marked regression of nodules, softening of earlobes, and resolution of reactional lesions were observed. This case underscores the diagnostic value of simple clinical examination and slit-skin smear microscopy in resource-limited settings and the necessity of simultaneous antimicrobial and anti-inflammatory therapy to prevent irreversible nerve damage.

**Keywords:** *Lepromatous Leprosy; Multibacillary Leprosy; Erythema Nodosum Leprosum; Type 2 Lepra Reaction; Tanzania; Multidrug Therapy; Case Report.*

**How to Cite:** Juliah Wangari Muchunu; Badria Kombe Mushi; Ashraf Yusuph; Twijise Ambah Mwamfupe; Neema Ezekiel Kunyaranyara; Godwin Silas Macheku (2026) Lepromatous (Multibacillary) Leprosy Complicated by Type 2 Lepra Reaction (Erythema Nodosum Leprosum) in a Young Adult from Tanzania: A Case Report.

*International Journal of Innovative Science and Research Technology*, 11(9), 3374-3378.

<https://doi.org/10.38124/ijisrt/26sep1450>

## I. INTRODUCTION

Leprosy (Hansen's disease), caused by the obligate intracellular pathogen *Mycobacterium leprae*, continues to rank among the neglected tropical diseases targeted for elimination by the World Health Organization (WHO). Although the global prevalence has fallen dramatically since the introduction of multidrug therapy (MDT) in the 1980s, more than 200 000 new cases are still reported annually, the majority from a limited number of endemic countries in South Asia, Latin America, and Africa [1].

Tanzania remains one of the 23 WHO global priority countries for leprosy. In 2023, the national prevalence stood at approximately 0.2 per 10 000 populations and the new-case detection rate at 23 per million, yet regional disparities persist; coastal and southern regions, including Tanga, continue to report rates well above the national average [2]. Despite having achieved the formal elimination threshold (prevalence <1 per 10 000) at the national level in 2006, the country still notifies more than 1 000 new cases each year, of which the majority are multibacillary and a substantial proportion present with grade-2 disability [3,4].

The clinical spectrum of leprosy is determined by the host's cell-mediated immune response and ranges from polar tuberculoid (paucibacillary) to polar lepromatous (multibacillary) disease. Lepromatous leprosy is characterized by high bacillary loads, multiple skin lesions ( $\geq 6$ ), diffuse cutaneous infiltration, madarosis, bilaterally thickened earlobes, and progressive peripheral neuropathy [1,5]. Because of profound impairment of Th1-mediated immunity, patients with polar lepromatous or borderline-lepromatous disease are particularly susceptible to type 2 (erythema nodosum leprosum, ENL) reactions. ENL is an immune-complex-mediated (type-III hypersensitivity) phenomenon that may occur before, during, or after MDT and is driven by the release of mycobacterial antigens, complement activation, and a cascade of pro-inflammatory cytokines (TNF- $\alpha$ , IL-6, IL-1) [6,7]. Clinical manifestations include crops of tender erythematous nodules, systemic inflammation, and, most importantly, acute neuritis that can rapidly produce irreversible nerve damage if untreated. Up to 50 % of patients with lepromatous leprosy experience at least one episode of ENL [6,8].

Delayed diagnosis remains common in rural African settings and is frequently compounded by prior consultation of traditional healers, incomplete BCG coverage (BCG confers approximately 50 % protection against leprosy) [9], and limited recognition of the disease among frontline health workers. The present report describes a young adult male from the Tanga Region with classic untreated lepromatous leprosy complicated by ENL, highlighting diagnostic clues accessible in primary-care facilities and the successful outcome of standard WHO MDT combined with corticosteroids.

## II. CASE PRESENTATION

### ➤ History

A 31-year-old divorced male artisan living alone in the Tanga Region presented with a one-year history of painless, non-itchy skin lesions distributed over both extremities and the face. The lesions had partially improved after a short course of oral prednisolone obtained elsewhere. In the weeks preceding presentation, he noticed progressive non-pitting swelling of the hands and fingers, later involving the right upper limb. He recalled loss of sensation over the right forearm that had gone unnoticed until he sustained an accidental burn injury; the burn healed with a residual hyperpigmented scar. Because of the chronicity of symptoms, he had first sought care from traditional healers. He had never received BCG vaccination, having been born at home outside the national immunization programme. He denied tobacco or alcohol use and there was no family history of similar illness.

### ➤ Physical Examination

Examination revealed diffuse facial skin infiltration with multiple firm, skin-colored to hyperpigmented papules and nodules over the forehead, nose, and cheeks, accompanied by thinning of the lateral eyebrows (madarosis). Both earlobes were markedly thickened and nodular. Multiple discrete, firm, non-tender papulonodular lesions were present on the extensor aspects of the forearms, together with a well-demarcated hyperpigmented burn scar overlying the area of documented sensory loss. Additional nodules were noted on the nostrils and other extremities. Peripheral-nerve thickening and reduced light-touch and pain sensation were elicited over the right upper limb; mild non-pitting edema of the hands and fingers was present. No corneal or ocular involvement was observed.



Fig 1 Pre-Treatment Appearance Showing Diffuse Facial Infiltration, Multiple Firm Papulonodular Lesions, Madarosis, and a Markedly Thickened Nodular Ear Lobe.



Fig 2 Pre-Treatment Forearm Lesions Showing Multiple Discrete, Firm Papulonodular Lesions Along the Extensor Forearm, Consistent with Cutaneous Involvement in Multibacillary (Lepromatous) Leprosy.

➤ *Investigations*

Slit-skin smear samples obtained from both earlobes and a representative skin nodule were stained using the Ziehl–Neelsen method, revealing numerous acid-fast bacilli appearing as red slender rods against a blue background. In

conjunction with the clinical findings ( $\geq 6$  skin lesions and a positive smear), the diagnosis of multibacillary leprosy, according to the WHO operational classification, was established.

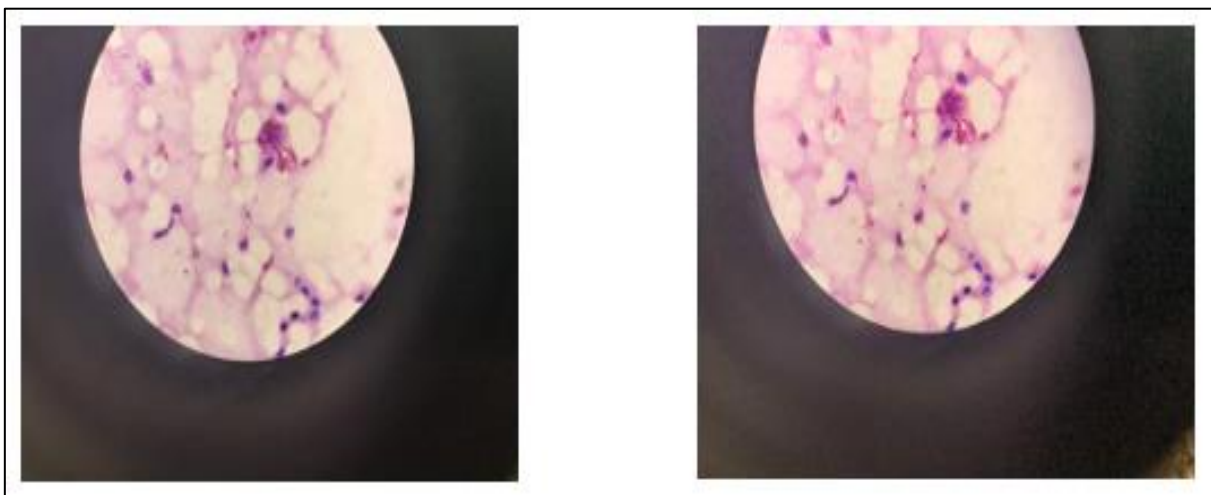


Fig 3 Skin Smear Showing Acid-Fast Bacilli Clusters on ZN Stain.

➤ *Diagnosis*

Lepromatous (multibacillary) leprosy complicated by type 2 lepra reaction (erythema nodosum leprosum) was diagnosed on the basis of the characteristic dermatoneurological picture—diffuse facial infiltration, madarosis, nodular earlobes, symmetrical papulonodules, peripheral-nerve involvement with sensory loss, and corticosteroid-responsive lesions—and the demonstration of numerous acid-fast bacilli.

➤ *Treatment*

The patient was commenced on the WHO-recommended multidrug therapy regimen for multibacillary leprosy: monthly supervised rifampicin 600 mg and clofazimine 300 mg, together with daily self-administered clofazimine 50 mg and dapsone 100 mg, for a planned

duration of 12 months [1]. Because of concurrent ENL, he also received a tapering course of oral prednisolone to control the reaction and to minimize the risk of reaction-associated nerve damage [10,11]. Counselling covered the chronic yet curable nature of the disease, the critical importance of treatment adherence, self-care of the anesthetic limb to prevent further injury, and the psychosocial dimensions of leprosy stigma.

➤ *Outcome and Follow-up*

Follow-up clinical photographs obtained after several months of combined multidrug therapy and corticosteroid treatment demonstrated substantial regression in the number and size of facial and forearm papulonodular lesions, softening and reduction of previously thickened earlobes, and resolution of acute reactional lesions. Residual findings

consisted of post-inflammatory hyperpigmented macules and a pre-existing burn scar. The patient tolerated multidrug therapy well and remained under follow-up to complete the

12-month course and to monitor for recurrent reactional episodes.

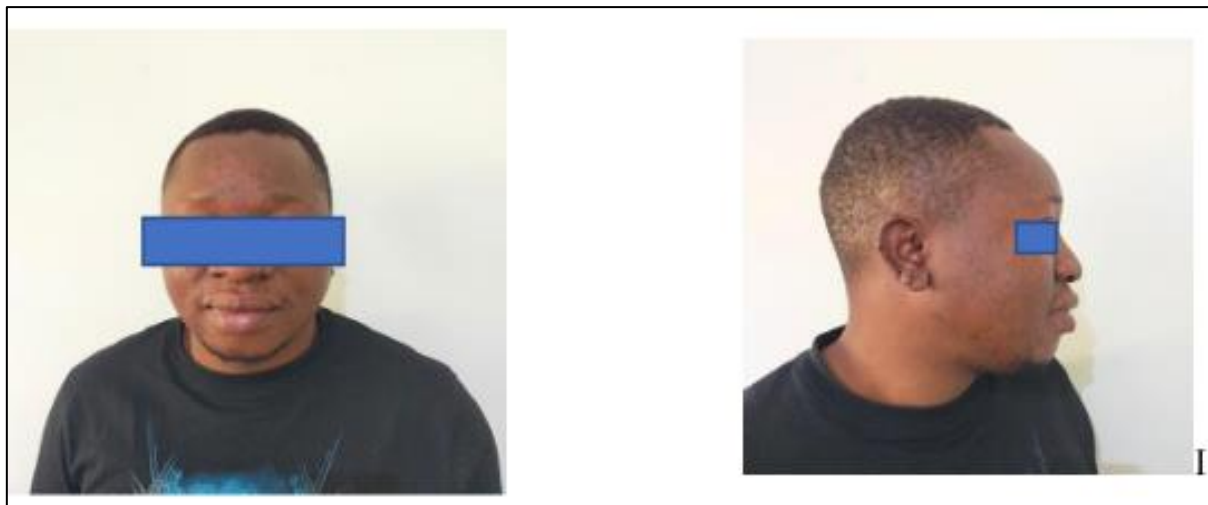


Fig 4 Post-Treatment Appearance Showing Marked Regression of Facial Infiltration And Nodularity, with Softening of the Previously Thickened Earlobe and Residual Post-Inflammatory Hyperpigmentation.



Fig 5 Post-Treatment Forearm Appearance Showing Resolution of the Papulonodular Lesions, with the Residual Hyperpigmented Scar Corresponding to the Earlier Insensate-Limb Burn Injury.

### III. DISCUSSION

This case illustrates the classic clinical phenotype of untreated polar lepromatous leprosy, diffuse cutaneous infiltration, madarosis, bilaterally thickened nodular earlobes, and widespread papulonodules, along with clear evidence of peripheral nerve involvement that had already produced anesthetic injury. The demonstration of numerous acid-fast bacilli on simple slit-skin smear microscopy provided definitive bacteriological confirmation and allowed rapid operational classification as multibacillary disease, avoiding the need for more sophisticated laboratory support that is often unavailable in rural Tanzanian facilities. WHO guidelines continue to endorse clinical diagnosis supplemented by slit-skin smear microscopy as the cornerstone of case detection in endemic settings [1].

Type 2 (ENL) reactions occur in up to half of patients with lepromatous leprosy and are driven by immune-complex deposition and a cascade of pro-inflammatory cytokines [6,7]. Although ENL classically appears after the initiation of MDT, it may also present de novo or be unmasked by intermittent corticosteroid exposure, as appears to have occurred in this patient [8,12]. Prompt recognition and concurrent anti-inflammatory therapy are essential; corticosteroids remain the first-line treatment in most endemic settings, while thalidomide (where available and not contraindicated) or higher-dose clofazimine may be required for severe or recurrent disease [10,11,13]. Recent reviews emphasize that the principal goal of ENL management is the rapid control of neuritis to prevent permanent disability [7,13].

The absence of BCG vaccination is noteworthy. Meta-analyses have consistently shown that BCG confers

approximately 50 % protection against leprosy [9], and the lack of neonatal vaccination in home deliveries continues to leave rural Tanzanian cohorts at elevated risk. Delayed care-seeking, prior traditional-healer consultation, and the social isolation of a divorced artisan living alone further highlight the persistent barriers of stigma and limited health literacy that perpetuate late presentation and disability factors repeatedly documented in East African studies [3,4].

Management followed current WHO guidelines: a uniform three-drug MDT regimen (rifampicin, clofazimine, dapsone) for 12 months in multibacillary disease, together with corticosteroids for reactional states [1]. The observed clinical improvement regression of nodules, reduction of earlobe thickening, and resolution of ENL demonstrate the efficacy of this approach, even in advanced disease. Continued surveillance for recurrent ENL and meticulous self-care of anesthetic extremities remain essential to prevent secondary disability.

This report adds to the limited published literature on lepromatous leprosy with ENL from East Africa. It reinforces several practical messages for clinicians working in endemic areas: (1) the diagnostic value of simple clinical examination and slit-skin smear microscopy; (2) the need for concurrent management of reactional states to protect nerve function; and (3) the importance of patient education and stigma reduction to improve treatment adherence and long-term outcomes. Strengthening community awareness, ensuring BCG coverage, and integrating leprosy services into primary care are essential if Tanzania is to achieve the WHO goal of zero transmission by 2030 [2,4].

#### IV. CONCLUSION

Lepromatous leprosy with type 2 reaction remains a curable yet potentially disabling condition when diagnosis is delayed. Early recognition of the characteristic dermatoneurological signs, confirmation by slit-skin smear, and prompt initiation of WHO multidrug therapy plus corticosteroids can produce excellent clinical responses and prevent irreversible nerve damage. Strengthening community awareness, ensuring BCG coverage, and integrating leprosy services into primary care are essential if Tanzania is to achieve the WHO goal of zero transmission by 2030.

#### DECLARATIONS

##### ➤ *Ethics Approval and Consent to participate*

In this case report, written informed consent was obtained from the patient. A copy of the consent form is available on file. Approval for publication was obtained from the relevant authority.

##### ➤ *Consent for Publication:*

Written informed consent (in Swahili) was obtained from the patient for the publication of this case report and the accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

- **Competing Interests:** The authors declare no conflicts of interest.
- **Funding:** This paper was funded by Tanga College of Health and Allied Sciences research project which aimed at stimulating health care workers to conduct research and publications
- **Authors' Contributions:** JWM was responsible for conceptualization, patient work-up, acquisition of clinical data and images, literature review, and manuscript writing; BKM and TAM performed the literature review and patient follow-up. AYM and NEK reviewed the literature and wrote the manuscript. JWM and GSM critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.
- **Abbreviations;** MD-Medical Doctor, MMED-Masters of Medicine.

#### REFERENCES

- [1]. World Health Organization. Guidelines for the diagnosis, treatment and prevention of leprosy. Geneva: WHO; 2018.
- [2]. National Tuberculosis and Leprosy Programme, United Republic of Tanzania. Annual tuberculosis and leprosy epidemiological report 2023. Dar es Salaam: Ministry of Health; 2025.
- [3]. Mwageni N, et al. Burden of leprosy and associated risk factors for disabilities in Tanzania from 2017 to 2020. PLoS One. 2024;19(10):e0311676.
- [4]. World Health Organization. Global leprosy (Hansen's disease) update, 2023. Wkly Epidemiol Rec. 2024.
- [5]. Scollard DM, Adams LB, Gillis TP, et al. The continuing challenges of leprosy. Clin Microbiol Rev. 2006;19(2):338–381
- [6]. Kahawita IP, Lockwood DNJ. Towards understanding the pathology of erythema nodosum leprosum. Trans R Soc Trop Med Hyg. 2008;102(4):329–337.
- [7]. Gupta V, et al. Reactions in leprosy: updated insights into pathophysiology, clinical spectrum and therapeutic approaches—a narrative review. Health Sci Rep. 2026.
- [8]. Pocaterra L, Jain S, Reddy R, et al. Clinical course of erythema nodosum leprosum: an 11-year cohort study in Hyderabad, India. Am J Trop Med Hyg. 2006;74(5):868–879.
- [9]. Merle CS, Cunha SS, Rodrigues LC. BCG vaccination and leprosy protection: review of current evidence. Expert Rev Vaccines. 2010;9(2):209–222.
- [10]. World Health Organization. Multidrug therapy (MDT) regimens for leprosy. Geneva: WHO; updated 2018.
- [11]. Walker SL, Lockwood DNJ. Leprosy type 1 (reversal) reactions and their management. Lepr Rev. 2008;79(4):372–386.
- [12]. Prabhu S, Shenoi SD, Pai SB, et al. Erythema nodosum leprosum as the presenting feature in multibacillary leprosy. Dermatol Online J. 2009;15(6):15.
- [13]. Indian Journal of Dermatology, Venereology and Leprology. Leprosy reactions: new knowledge on pathophysiology, diagnosis, treatment and prevention. 2025..