
Lucio Phenomenon Mimicking Necrotizing Myofasciitis: A Case Report Study

Elita Nurhidayati, Putu Dyah Ayu Saraswati Wangaya

General Hospital Denpasar, Indonesia

Email: itsmeelita85@gmail.com

Abstract

Lucio phenomenon is a rare but severe lepra reaction occurring in untreated patients with diffuse lepromatous leprosy, characterized by widespread cutaneous necrosis due to endothelial invasion and vascular thrombosis caused by *Mycobacterium leprae*. Its clinical resemblance to necrotizing soft tissue infections often leads to diagnostic confusion and inappropriate surgical intervention. We report a case of a 47-year-old man presenting with bilateral necrotic ulcers of the lower limbs, initially diagnosed as necrotizing myofasciitis and scheduled for debridement. However, further dermatological evaluation revealed hallmark signs of multibacillary leprosy, including madarosis, multiple anesthetic plaques, and sensory loss, in the absence of peripheral nerve thickening. A diagnosis of Lucio phenomenon was established, and surgical intervention was withheld in favor of initiating multidrug therapy (MDT) and supportive care. This case underscores the critical importance of early dermatological consultation and high clinical suspicion for atypical leprosy reactions in endemic regions, particularly in patients with rapidly progressive ulcers mimicking necrotizing infections.

Keywords: Lucio phenomenon, lepromatous leprosy, necrotizing myofasciitis, misdiagnosis, multidrug therapy, ulcerative skin lesions, leprosy reactions

Corresponding: Elita Nurhidayati
E-mail: itsmeelita85@gmail.com



INTRODUCTION

Leprosy, also known as Hansen's disease, is a chronic infectious disease caused by *Mycobacterium leprae*, primarily affecting the skin, peripheral nerves, and, in some cases, the upper respiratory mucosa and other organs (Chen et al., 2022; Khadilkar et al., 2021; Makhakhe, 2021; Mi et al., 2020; Ramam, 2019). Despite longstanding global elimination programs, Indonesia remains among the top three countries with the highest leprosy burden worldwide, alongside India and Brazil. According to data from the Indonesian Ministry of Health, 13,487 new cases of leprosy were reported in 2023, with a grade 2 disability rate of 6.1%, indicating a persistent delay in diagnosis and treatment (Kementerian Kesehatan Republik Indonesia, 2024).

One of the rare yet serious complications of untreated diffuse lepromatous leprosy is *Lucio phenomenon*, a distinct leprosy reaction characterized by ischemic necrosis resulting from thrombosis of superficial blood vessels due to direct endothelial invasion by *M. leprae*. First described in Mexico by Lucio and Alvarado in the 19th century, this condition was initially believed to be geographically restricted to Central America. However, *Lucio phenomenon* has now been increasingly reported in other endemic regions, including Indonesia, albeit infrequently (Frieda et al., 2022; Kaur et al., 2021; Norman et al., 2022; Rusia et al., 2024).

Globally, *Lucio phenomenon* remains an under-recognized complication of leprosy. A systematic review by Vera-Cabrera et al. (2012), analyzing 98 cases reported worldwide, found that the majority of cases originated from Mexico and Central America, with emerging reports from South Asia and Southeast Asia, including India and Indonesia. The phenomenon typically occurs in patients with long-standing, untreated diffuse lepromatous leprosy, characterized by extensive bacterial infiltration

without nerve thickening. In India, Ramesh and Girdhar (2010) reported three cases of *Lucio phenomenon*, all presenting with necrotizing skin lesions and initially misdiagnosed as vasculitis or infectious processes. Similarly, Sharma et al. (2008) documented a case series demonstrating that over 50% of *Lucio phenomenon* cases were initially misdiagnosed as necrotizing soft tissue infections, leading to unnecessary surgical interventions. These case reports highlight a consistent pattern: diagnostic delay, clinical mimicry of surgical emergencies, and the critical importance of dermatological assessment in endemic settings.

Clinically, *Lucio phenomenon* may mimic necrotizing soft tissue infections such as necrotizing myofasciitis, presenting with rapidly progressive ulcerative lesions, severe pain, systemic symptoms, and tissue necrosis. This similarity often poses a diagnostic challenge, particularly in emergency settings, and may lead to misdiagnosis resulting in unnecessary surgical interventions and delayed initiation of appropriate anti-leprosy therapy (Sharma et al., 2008).

The present case contributes to the limited Indonesian literature on *Lucio phenomenon* by documenting a diagnostically challenging presentation that was initially misinterpreted as necrotizing myofasciitis in an emergency setting. This report emphasizes three key clinical contributions: (1) demonstration of the critical role of multidisciplinary consultation in preventing unnecessary surgical intervention; (2) identification of specific clinical features that should prompt consideration of leprosy in patients presenting with necrotizing skin lesions in endemic areas; and (3) reinforcement of the need for heightened awareness among emergency physicians and surgeons regarding atypical presentations of leprosy reactions. To our knowledge, this is among the first documented cases in Indonesia where surgical debridement for presumed necrotizing myofasciitis was avoided through timely dermatological consultation and recognition of pathognomonic leprosy features.

In this case report, we present a patient with *Lucio phenomenon* who was initially diagnosed with necrotizing myofasciitis in the emergency department. This case underscores the need for heightened clinical awareness of severe leprosy reactions in patients presenting with progressive ulcerations, especially in endemic regions. It also highlights the critical role of multidisciplinary collaboration and accurate differential diagnosis when managing cases with clinical features resembling necrotizing soft tissue infections.

METHOD

This study employed a qualitative research design in the form of a case report. The data population conceptually encompasses all documented cases of *Lucio phenomenon*, particularly those presenting with diagnostic challenges. The data sample for this specific research is a single, purposively selected case: a 47-year-old male patient who presented to the emergency department with bilateral lower limb ulcers initially diagnosed as necrotizing myofasciitis. The sampling technique used was purposive sampling, as the case was specifically chosen for its rarity, its value in illustrating a critical diagnostic dilemma, and its relevance to the endemic setting in Indonesia.

The primary research instrument was the researcher, who conducted a comprehensive review of the patient's medical records, including clinical history, physical examination findings, laboratory results, and consultation notes. A structured data extraction approach was used to gather information systematically. The data analysis technique involved descriptive analysis and thematic analysis of the clinical data. The patient's presentation, diagnostic journey, and outcome were meticulously described. Furthermore, the findings were analyzed and discussed in relation to existing literature on *Lucio phenomenon* and necrotizing soft tissue infections to identify key learning points and clinical implications, thereby generating in-depth, context-specific insights from the single case.

RESULT AND DISCUSSION

Case Report

A 47-year-old man was brought to the emergency department with a two month history of bilateral leg ulcers and associated pain. The lesions began as swelling, followed by purulent discharge and subsequent rupture. The patient also reported swelling of both hands after consuming medication from a local clinic, the name of which he could not recall. No systemic symptoms such as fever, chills, or altered consciousness were reported.

On examination, the patient appeared clinically stable and well-nourished. Vital signs were within normal limits: GCS E4V5M6, respiratory rate 18 breaths per minute, oxygen saturation 98% on room air, and axillary temperature 36.3°C. Local examination of the lower extremities revealed necrotic ulcers on both crura, accompanied by hyperemia, edema, tenderness, and warmth. Head, ENT, and neck examinations were unremarkable.

Laboratory studies showed leukocytosis (WBC: $20.64 \times 10^9/L$), severe anemia (Hb: 7.1 g/dL; Hct: 21.7%), thrombocytosis (Plt: $469 \times 10^9/L$), and hypoalbuminemia (albumin: 1.9 g/dL). Liver enzymes were elevated (SGOT: 104 U/L; SGPT: 92 U/L), with normal renal function (urea: 19 mg/dL; creatinine: 0.7 mg/dL). Electrolyte analysis revealed hyponatremia (Na: 130 mmol/L) with normal potassium and chloride levels. Coagulation profile showed prolonged clotting time (14 minutes) and normal bleeding time (2 minutes). Chest radiograph and electrocardiography showed no abnormalities. HBsAg and anti-HCV testing were performed as part of preoperative screening.

The initial clinical impression was necrotizing myofasciitis of both lower limbs, and the patient was scheduled for urgent debridement. However, due to marked anemia and hypoalbuminemia, he was first referred to internal medicine for stabilization and transfusion.

Dermatology consultation was subsequently requested for further evaluation of the skin lesions. The patient reported a one-year history of numb patches on the lower limbs and back, along with eyebrow loss (madarosis). Over the past six months, the ulcers on the legs had progressed and remained insensitive to pain. The patient had never received leprosy treatment nor sought medical care for these symptoms.

Dermatologic examination revealed madarosis, multiple anesthetic erythematous plaques on the back and arms with associated edema, and hyperpigmented, xerotic plaques on the lower limbs covered with dressings. Sensory testing demonstrated reduced to absent superficial touch, pain, and thermal perception across the affected areas, confirmed by pinprick and temperature discrimination tests. Motor strength was generally preserved, though mild weakness (MMT 4/5) was observed in dorsiflexion of both feet. Mild atrophy of the hand interosseous muscles was noted, but no deformities such as claw hand or foot drop were present. Palpation of the ulnar and peroneal nerves revealed no thickening or tenderness.

Based on clinical findings, a working diagnosis of Lucio phenomenon was established. The planned surgical debridement was cancelled, and management was refocused on improving the patient's general condition and initiating multidrug therapy (MDT) for multibacillary leprosy according to national guidelines. Patient education and counseling were provided regarding the chronic and treatable nature of the disease.



Figure 1. Leonine facies and Lucio phenomenon lesions on both forearms.



Figure 2. No visible thickening of bilateral auricular nerves.



Figure 3. Diffuse purpuric lesions on the back and flank regions.



Figure 4. Multiple necrotic ulcerative lesions on bilateral lower limbs and dorsum of the feet..

Discussion

Lucio phenomenon is a rare form of leprosy reaction and represents a severe complication observed in untreated cases of diffuse lepromatous leprosy. First described in Mexico by Lucio and Alvarado in the 19th century, this reaction was initially believed to be endemic to Central America. However, over the past two decades, similar cases have been increasingly reported in other endemic countries, including India and Indonesia.

Clinically, Lucio phenomenon is characterized by rapidly spreading purpuric or necrotic skin lesions, resulting from thrombosis of superficial blood vessels due to direct endothelial invasion by *Mycobacterium leprae*. Histopathological findings typically reveal vascular thrombosis, diffuse lepromatous infiltrates, and abundant acid-fast bacilli (Rea & Jerskey, 2005). Unlike reversal reaction (Type 1) or erythema nodosum leprosum (Type 2), Lucio phenomenon does not always present with a distinct systemic immune response and may appear insidiously as the initial manifestation of previously undiagnosed leprosy (Ramesh & Girdhar, 2010).

In the present case, the patient presented with bilateral necrotic ulcers on the lower limbs, accompanied by local tenderness, hyperemia, and edema, yet without fever or hemodynamic instability. Laboratory evaluation revealed leukocytosis, severe anemia, and hypoalbuminemia, supporting the

initial suspicion of necrotizing myofasciitis, a diagnosis often associated with severe soft tissue infections and requiring emergent surgical debridement. This highlights the diagnostic challenge posed by Lucio phenomenon, particularly as it can mimic surgical emergencies. Therefore, it is essential to maintain a high index of suspicion and carefully consider differential diagnoses, especially in endemic regions.

Necrotizing myofasciitis is a rapidly progressive and life-threatening soft tissue infection involving the muscle fascia and subcutaneous tissues, frequently accompanied by systemic sepsis. The most common causative organisms include *Streptococcus pyogenes*, *Clostridium* spp., and *Staphylococcus aureus* (Bonne & Kadri, 2017; Holly et al., 2023; Hua et al., 2023; Stevens et al., 2021; Urbina et al., 2021). Clinical features often include severe pain disproportionate to skin findings, progressive edema, skin discoloration, and systemic signs such as fever, tachycardia, and hypotension. Diagnosis is often made based on clinical judgment, imaging studies, or surgical exploration, and treatment requires immediate aggressive debridement and broad-spectrum antibiotics. Delay in surgical intervention significantly increases patient mortality.

Several key clinical features in this case should have prompted consideration of leprosy as a differential diagnosis. These included madarosis, hypopigmented anesthetic patches, multiple lesions on the back and extremities, and marked sensory loss to light touch, pain, and temperature—all classic features of multibacillary leprosy. The absence of palpable peripheral nerve thickening does not exclude leprosy, especially in diffuse lepromatous forms, where *M. leprae* may infiltrate skin and vasculature without producing overt nerve hypertrophy.

A multidisciplinary approach enabled a thorough dermatological assessment, leading to a revised diagnosis before invasive surgery could be performed. Identification of hypopigmented anesthetic patches, madarosis, and sensory deficits served as hallmark features of Hansen's disease, ultimately confirming the diagnosis of Lucio phenomenon. Thus, unnecessary surgical debridement was avoided, and therapeutic focus was redirected toward stabilizing the patient's systemic condition and initiating multidrug therapy (MDT).

The laboratory findings in this patient—leukocytosis, normochromic normocytic anemia, and severe hypoalbuminemia—are common in patients with severe leprosy reactions or chronic infections. Elevated liver enzymes may reflect systemic inflammation or prior drug induced hepatotoxicity. Normal electrolyte levels and renal function suggested no major organ dysfunction at initial evaluation.

Management of Lucio phenomenon primarily involves prompt initiation of WHO-recommended MDT for multibacillary leprosy, alongside supportive care for tissue necrosis and systemic complications such as anemia or hypoalbuminemia (World Health Organization, 2018). Corticosteroids are generally ineffective in managing Lucio reactions unless accompanied by systemic vasculitis or organ involvement.

In a retrospective study by Sharma et al. (2008), Lucio phenomenon was misdiagnosed as vasculitis or soft tissue infection in over 50% of cases, resulting in delayed anti-leprosy treatment and unnecessary surgical procedures. Hence, clinicians in endemic settings must remain vigilant for severe leprosy reactions in patients with progressive ulcerations, even when the presentation mimics surgical emergencies.

Literature reviews have shown that delayed diagnosis in Lucio phenomenon is associated with high mortality, especially in cases progressing to sepsis or multi-organ failure due to extensive necrosis. Improving clinician awareness of atypical leprosy reactions is therefore crucial, particularly in high-burden countries like Indonesia.

CONCLUSION

Lucio phenomenon is a rare but severe complication of lepromatous leprosy that often mimics necrotizing myofasciitis, posing a significant diagnostic challenge in endemic regions where patients frequently present rapidly progressing ulcerative skin lesions. Maintaining high clinical vigilance, supported by early dermatological consultation and a multidisciplinary approach, is essential to avoid unnecessary surgical interventions and ensure timely initiation of multidrug therapy. Future research should focus on developing clearer diagnostic criteria and point-of-care diagnostic tools to distinguish

Lucio phenomenon from necrotizing soft tissue infections, particularly in resource-limited endemic settings.

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