

Concurrent Lepromatous Leprosy and Active Tuberculosis in A Young Male: A Complex Diagnostic and Therapeutic Challenge with Hematologic Manifestations

Juan Fausto Martínez González^{*1}, Ana Laura Ricarte González², Alma Elizabeth Díaz Gamiño³, Leonel Castro López⁴, Francisco Adrián Razo Moreno³, Ana Victoria Pérez García³, Mireya Nolasco Sanchez³, Cristina Montserrat García Montalvo⁵, Katia Gabriela Silva Esquivel¹, Mayra Aréchiga López⁶, Alejandro Calderón Romo¹, Jonathan Guzmán Rodríguez¹, Ilse Nájera Basurto⁷, Sergio Ronaldo Esparza Herrera¹, María José Soto Martínez⁷, Karla Teresa Granados Velasco⁸, Alexa Ximena Márquez Durón¹

¹Universidad Cuauhtémoc Aguascalientes. Aguascalientes, Mexico.

²Universidad Popular Autónoma del Estado de Puebla. Puebla, Mexico.

³Universidad de Celaya Escuela de Medicina. Mexico.

⁴Universidad de Guanajuato. Mexico.

⁵Benemerita Universidad Autónoma de Aguascalientes. Mexico.

⁶Universidad de Guadalajara. Guadalajara, Jalisco, Mexico.

⁷Universidad Autónoma de Aguascalientes. Aguascalientes, Mexico.

⁸Universidad del Valle de Atemajac. Mexico.

ABSTRACT

Introduction: This report details the clinical evolution of a 23-year-old male presenting with the exceptionally rare co-occurrence of active lepromatous leprosy and tuberculous lymphadenitis, complicated by severe immune thrombocytopenia. The patient's presentation—spanning chronic dermatological findings, acute systemic symptoms, and profound hematological disturbance—required a multifaceted diagnostic approach culminating in histopathological and microbiological confirmation of both mycobacterial infections. Management was successfully integrated, utilizing a single rifampin-based antitubercular regimen to simultaneously address both diseases, resulting in resolution of thrombocytopenia and significant clinical improvement. This case highlights the diagnostic complexity in co-endemic regions, the potential for tuberculosis to drive significant parainfectious hematologic complications, and the feasibility of a unified pharmacological strategy for dual mycobacterial disease.

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BACKGROUND

Mycobacterium leprae and *Mycobacterium tuberculosis*, both belonging to the *Mycobacterium* genus, cause chronic granulomatous diseases with significant global health burdens, particularly in overlapping endemic regions of Asia, Africa, and Latin America. Despite shared epidemiological niches, the simultaneous presentation of active disease from both pathogens in a single individual is a documented rarity. This low reported incidence is attributed to divergent host immune responses elicited by each bacterium. Lepromatous leprosy, the multibacillary, anergic pole of the disease spectrum, is characterized by a specific T-cell anergy to *M.*

leprae antigens, high bacterial loads, and a humorally biased (Th2) immune profile. In contrast, effective containment of *M. tuberculosis* typically requires a robust cell-mediated (Th1) immune response to form organized granulomas and limit bacterial dissemination.^{1,2}

The pathophysiological paradox of their co-existence suggests the possibility of compartmentalized or antigen-specific immune dysregulation. Furthermore, tuberculosis is a well-known, though not universally common, cause of secondary immune thrombocytopenia (ITP). The mechanism is thought to involve immune dysregulation, cross-reactive antibodies, or immune complex deposition leading to

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peripheral platelet destruction. This hematological manifestation can sometimes be the presenting feature, overshadowing or complicating the primary infectious diagnosis. The management of such a dual infection presents a unique therapeutic puzzle, primarily due to the shared cornerstone drug—rifampin—used in both the World Health Organization's Multidrug Therapy (MDT) for leprosy and the standard regimen for tuberculosis. Crafting a regimen that is effective, minimizes polypharmacy and toxicity, and ensures adherence requires careful pharmacological integration.^{2,3,4} The patient's history provides several contextual elements, though a direct causal link remains speculative. A 23-year-old male with no prior significant medical history, no known chronic illnesses, and negative for HIV, reported a history of multiple tattoos on the anterior chest, performed approximately five years prior. While tattooing is a known, though uncommon, potential route for transmission of blood-borne pathogens and theoretically for cutaneous inoculation, its role here is unclear. Of greater epidemiological interest is a history of temporary residence in the United States for work seven years prior, during which he reported living in crowded conditions ("hacinamiento") for a four-month period. Crowding is a classic risk factor for the transmission of airborne pathogens, notably *Mycobacterium tuberculosis*. The temporal gap between this exposure and clinical presentation is consistent with the potential for latent tuberculosis infection (LTBI) to reactivate years later, possibly triggered by an unrelated immunological shift or stressor.

Notably, the patient's dermatological symptoms had a much longer incubation. He reported the insidious onset of skin lesions approximately three years before the current acute presentation. This timeline is consistent with the slowly progressive nature of lepromatous leprosy, which can have an incubation period of several years. The progression from

indeterminate early lesions to the classic infiltrative plaques of LL over this period is plausible. Therefore, the clinical scenario suggests a longstanding, smoldering *M. leprae* infection that was present concurrently with what was likely initially a latent *M. tuberculosis* infection. The acute systemic illness and hematological crisis three months prior to admission may represent the point of reactivation of the latent tuberculous infection, superimposing its pathophysiology onto the chronic lepromatous disease. This sequence of events sets the stage for a complex interplay of two chronic infections, one manifesting cutaneously over years and the other erupting systemically, culminating in a rare and diagnostically challenging presentation.

CASE REPORT

A 23-year-old male with a history of multiple chest tattoos (5 years prior) and temporary residence in the USA (7 years prior) presented with a 3-month history of involuntary weight loss (6 kg). In February 2024, he developed left subconjunctival hemorrhage resolving spontaneously within 24 hours, followed by evening fevers, night sweats, progressive asthenia, adynamia, and three episodes of epistaxis. The final epistaxis episode was profuse and refractory to mechanical hemostasis, prompting hospital evaluation.

Physical examination revealed multiple, ill-defined, erythematous-hyperpigmented macules and plaques with bilateral, relatively symmetric distribution on the anterior thorax, without evident scaling. The skin surface was smooth, with noted loss of sensation within the lesions and in distal areas (hands and feet). Additional findings included madarosis (loss of eyebrow hair) and non-tender inguinal lymphadenopathy. The patient reported the skin lesions had been present for approximately 3 years. (Figure 1)



Figure 1. Skin lesions on anterior thorax and face.

Initial laboratory studies (27/03/2024) revealed several critical abnormalities: severe thrombocytopenia (platelets: $11 \times 10^3/\mu\text{L}$; reference: 150-450), absolute lymphopenia ($1.40 \times 10^3/\mu\text{L}$), and an elevated reticulocyte count (2.92%).

Coagulation profile (PT, INR, aPTT) was within normal limits, ruling out consumptive coagulopathy. Chemistry panel was largely unremarkable, with creatinine slightly below normal (0.62 mg/dL) and normal liver enzymes.

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Given the cutaneous findings and lymphadenopathy, biopsies were performed.

1. **Skin biopsy (abdominal region):** Histopathological examination confirmed a

diagnosis of lepromatous leprosy. Special staining (Ziehl-Neelsen) revealed abundant acid-fast bacilli (AFB).Figure 2

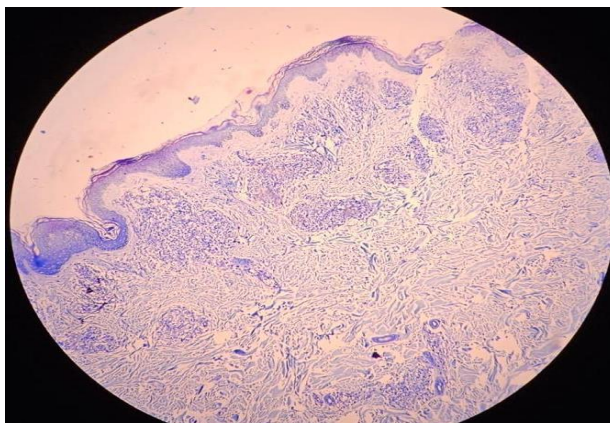


Figure 2. Acid-Fast bacilli on skin.

2. **Lymph node biopsy (inguinal region):** Histopathology indicated tuberculous lymphadenitis. Ziehl-Neelsen staining was also positive for AFB in the lymph nodes. Fungal stains (PAS) were negative.Figure 3

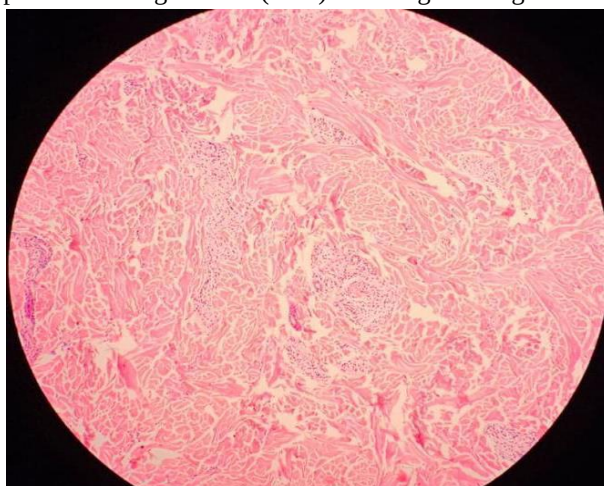


Figure 3. Lymph node PAS stain.

The simultaneous positivity for AFB in distinct tissues (skin and lymph node), with supportive histology, confirmed the rare diagnosis of concurrent active lepromatous leprosy and active tuberculous lymphadenitis. HIV co-infection was investigated and ruled out. Figure 3 and 4

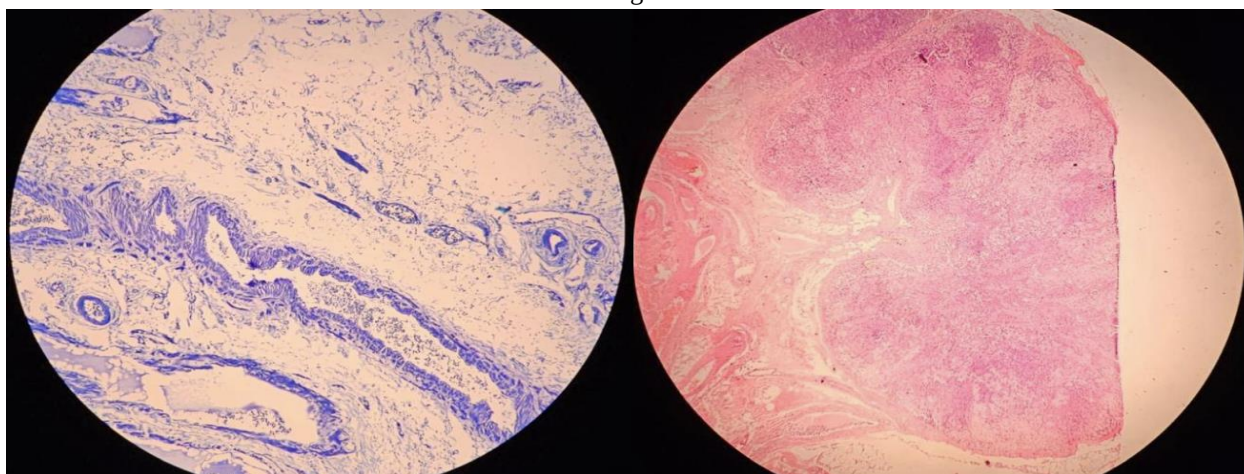


Figure 4. Ziehl-Neelsen staining on lymph node (left) and PAS staining on lymph node.

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The management was intricate due to the overlapping treatment regimens for both diseases, both of which include rifampin. The strategy prioritized the antitubercular regimen, leveraging its daily rifampin to cover the monthly requirement for leprosy treatment.

- **Antitubercular Therapy:** Standard intensive phase with isoniazid, rifampin, pyrazinamide, and ethambutol for 2 months, followed by a continuation phase of isoniazid and rifampin for 4 months.
- **Multidrug Therapy (MDT) for Lepromatous Leprosy:** This consisted of daily dapsone (100 mg), daily clofazimine (50 mg), plus monthly clofazimine (300 mg) for 12 months. The rifampin component was fulfilled by the daily dose from the anti-TB regimen; no additional monthly rifampin was added.
- **Supportive Care:** Pyridoxine prophylaxis was administered to prevent neurotoxicity from isoniazid. Close monitoring included serial complete blood counts (CBC), liver function tests (LFTs), and neurological assessments to watch for dapsone-induced hemolytic anemia, drug-induced hepatotoxicity, and peripheral neuropathy progression.

The profound thrombocytopenia ($11 \times 10^3/\mu\text{L}$) at presentation was a major concern. Given the normal coagulation studies and the subsequent diagnosis of tuberculosis, the most likely etiology was immune thrombocytopenia (ITP) secondary to tuberculosis. Mycobacterial infections can trigger an immune response leading to platelet destruction. The thrombocytopenia showed significant improvement and eventual normalization during the course of anti-tuberculous therapy, supporting this parainfectious immune-mediated mechanism. No leprosy reactions (Type 1 or 2) were observed during treatment.

The patient completed the full 6-month anti-TB regimen and the 12-month MDT for leprosy without requiring an extended phase. He demonstrated marked clinical improvement in systemic symptoms, cutaneous infiltration, and hematologic parameters. Platelet count recovered to normal range, and there was no evidence of hemolytic anemia or significant hepatotoxicity. The patient remained under prolonged follow-up with sustained clinical and hematological remission.

DISCUSSION

The presented case of a 23-year-old male with concomitant, histologically confirmed active tuberculous lymphadenitis and lepromatous leprosy represents an extraordinarily rare clinical entity. This concurrence transcends mere epidemiological coincidence and ventures into the complex interplay of host immunity, mycobacterial pathogenicity, and diagnostic challenge. The case is further complicated by a

significant hematologic manifestation—severe thrombocytopenia—adding a critical layer to the clinical management.^{4,5}

The simultaneous active presentation of these two mycobacterial infections is pathophysiologically intriguing. Lepromatous leprosy (LL) is characterized by a state of specific cellular anergy towards *Mycobacterium leprae*, with a dominant Th2 cytokine profile and poor granuloma formation, allowing for widespread bacterial dissemination. In contrast, containment of *Mycobacterium tuberculosis* typically requires a robust Th1-mediated cellular immune response, leading to well-formed granulomas. The presence of active tuberculosis in this patient suggests that the anergy in LL is highly specific to *M. leprae* antigens and does not represent a global failure of cell-mediated immunity. This indicates compartmentalized or divergent immune responses within the same host, a phenomenon rarely documented. The estimated probability of such a dual active infection is exceedingly low, making this case a notable exception that challenges simplified models of host-mycobacterium interaction.^{5,6,7}

The profound thrombocytopenia ($11 \times 10^3/\mu\text{L}$) observed at presentation was a striking finding. The normal coagulation studies (PT, INR, aPTT) effectively ruled out disseminated intravascular coagulation (DIC). In the context of the subsequent diagnosis, the most plausible etiology is immune thrombocytopenia (ITP) secondary to tuberculosis. Mycobacterial infections, particularly tuberculosis, are known to induce a state of immune dysregulation that can lead to the production of anti-platelet antibodies or immune complex deposition, resulting in peripheral platelet destruction. The temporal correlation—significant improvement and normalization of platelet counts coinciding with the initiation of anti-tuberculous therapy—strongly supports this parainfectious, immune-mediated mechanism. While leprosy reactions (e.g., Type 2 reaction or Erythema Nodosum Leprosum) can have systemic components, the absence of other reactional symptoms and the direct response to TB treatment pinpoint tuberculosis as the primary driver of the hematologic abnormality in this instance.^{6,7}

This case underscores the imperative for comprehensive diagnostic evaluation in endemic regions. The initial presentation with classic hypopigmented, anesthetic macules could have easily led to a singular focus on leprosy. However, the presence of significant systemic symptoms (fever, night sweats, weight loss) and profound thrombocytopenia were "red flags" prompting a broader investigation. The discovery of inguinal lymphadenopathy became the critical clue leading to the lymph node biopsy, which unveiled the concurrent tuberculosis. It highlights that in patients with leprosy, especially the lepromatous type, new or severe systemic symptoms should not be automatically attributed to leprosy reactions but must trigger a search for co-infections or complications.^{6,7,8}

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The management strategy employed was a model of pharmacological efficiency and rational design. Rifampin, a cornerstone bactericidal agent for both diseases, served as the therapeutic bridge. By utilizing the daily rifampin from the intensive and continuation phases of the standard anti-tuberculosis regimen (2HRZE/4HR), the requirement for the monthly supervised dose of rifampin in the Multidrug Therapy (MDT) for leprosy was inherently fulfilled. This ingenious approach prevented unnecessary polypharmacy, reduced the risk of additive toxicity (particularly hepatotoxicity), and likely improved treatment adherence. The completion of both regimens (6 months for TB, 12 months for LL) without the need for an extended leprosy treatment phase and without the occurrence of leprosy reactions signifies a highly successful therapeutic outcome. Close monitoring for dapsone-induced hemolysis and drug-induced hepatotoxicity was appropriately vigilant and contributed to the safe administration of this combined regimen.^{7,8}

CONCLUSIONS

Extreme Rarity with Profound Implications: The concurrent active infection with *Mycobacterium tuberculosis* and *Mycobacterium leprae* is an exceptionally rare event that illustrates the nuanced and compartmentalized nature of the human immune response to different mycobacterial species. **Hematologic Clue to Co-infection:** Severe thrombocytopenia in a patient with leprosy should not be overlooked. In this case, it acted as a major clinical clue pointing towards an underlying systemic co-infection—specifically tuberculosis—and its resolution with anti-TB therapy confirmed the diagnosis and mechanism. **Imperative for Exhaustive Diagnosis:** In endemic areas, a diagnosis of leprosy does not preclude the presence of other serious infections. Systemic symptoms and unexplained laboratory abnormalities, especially cytopenias, mandate a thorough investigation for comorbidities, including tuberculosis.

Feasibility of Integrated Treatment: A carefully designed, synergistic treatment plan that leverages common drugs (e.g., rifampin) from one regimen to cover the needs of another is not only feasible but also optimal. This strategy can effectively cure both complex infections while minimizing complexity and potential toxicity. **Successful Outcome:** This case demonstrates that even faced with a rare and complex dual mycobacterial disease accompanied by significant hematologic complications, a logical, evidence-based, and integrated therapeutic approach can yield excellent clinical, microbiological, and hematological outcomes with diligent monitoring and follow-up.

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