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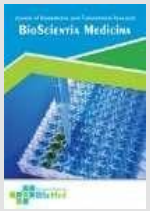

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# The 15-Year Shadow: Borderline Lepromatous Leprosy with Erythema Nodosum Leprosum Following Prolonged Treatment Default

**Rendra Darmasatria[1*], Tutty Ariani[1], Yosse Rizal[2], Rina Gustia[1], Irdawaty Izrul[1]**

[1]Department of Dermatology, Venereology and Aesthetics, Faculty of Medicine, Universitas Andalas/Dr. M. Djamil General Hospital, Padang, Indonesia

[2]Department of Dermatology, Venereology and Aesthetics, Faculty of Medicine, Universitas Andalas/Dr. Achmad Mochtar Regional General Hospital, Bukittinggi, Indonesia

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***Corresponding author:**
Rendra Darmasatria

**E-mail address:**
*dv.rendradarmasatria@gmail.com*

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**A B S T R A C T**

**Background:** Leprosy, caused by *Mycobacterium leprae*, persists as a global health issue where the primary challenges are not merely microbial but are deeply rooted in delayed diagnosis and poor treatment adherence. These delays, often driven by profound social stigma, lead to progressive, irreversible disability and sustain community transmission. Erythema Nodosum Leprosum (ENL), an acute immunological complication, further devastates patients' quality of life and complicates management. **Case presentation:** A 53-year-old Indonesian farmer presented with a 15-year history of untreated leprosy, a journey of neglect initiated by fear of treatment side effects and community ostracism. Clinical examination revealed advanced borderline lepromatous (BL) leprosy with diffuse skin infiltration, multiple anesthetic plaques, and thickened, tender peripheral nerves. He had established WHO Grade 1 disability, characterized by significant sensory loss in his hands and feet and early intrinsic muscle atrophy. A slit-skin smear confirmed a bacteriological index of +3 with a morphological index of 5%, indicating a high load of viable bacilli. Histopathology confirmed BL leprosy with a concurrent mild ENL reaction. A comprehensive, patient-centered management plan was initiated, including a 12-month course of multidrug therapy (MDT-MB), adjunctive care, and intensive counseling. **Conclusion:** This case powerfully illustrates the "shadow effect" of leprosy—how years of untreated disease, fueled by psychosocial barriers, culminate in a complex nexus of advanced infection, immunological reaction, and permanent neurological impairment. The patient's successful re-engagement with the health system underscores that eradicating the burden of leprosy requires a paradigm shift from a purely pharmacological approach to a deeply humanistic one. Effective control hinges on building compassionate health systems that actively dismantle stigma, empower patients with knowledge, and deliver holistic, multidisciplinary care to prevent the profound human cost of neglect.

## 1. Introduction

Leprosy, or Hansen's disease, is a chronic spectral disease caused by the obligate intracellular pathogen *Mycobacterium leprae*.[1] With a unique tropism for Schwann cells of the peripheral nervous system and macrophages of the skin, the bacillus orchestrates a spectrum of disease manifestations dictated entirely by the host's immune response. The global health community has made monumental strides in controlling leprosy since the advent of multidrug therapy (MDT) in the 1980s, rendering the disease curable and dramatically reducing prevalence.[2] Yet, the shadow of leprosy persists. In 2022, the World Health Organization (WHO) reported over 174,000 new

cases, with millions living with the permanent physical disabilities and deformities that are the disease's cruel legacy. Indonesia, alongside India and Brazil, remains a high-burden country, contributing significantly to the global case load and facing persistent challenges in reaching the "last mile" of elimination. The clinical course of leprosy is frequently complicated by lepra reactions, which are acute inflammatory episodes representing abrupt shifts in the host's immune status.[3] These reactions are the primary cause of nerve damage and disability. Type 2 reactions, or erythema nodosum leprosum (ENL), are systemic, humoral-mediated inflammatory events that occur almost exclusively in patients on the lepromatous side of the spectrum who harbor a high bacillary load.[4] Characterized by tender skin nodules, fever, and potential multisystem organ involvement, ENL is a formidable management challenge, often requiring long-term immunomodulatory therapy and carrying a significant risk of morbidity.[5]

Beyond the immunological complexities, the greatest barrier to leprosy elimination is profoundly human: non-adherence to treatment, driven by a deeply entrenched and multifaceted stigma.[6] For millennia, leprosy has been associated with social exclusion, fear, and religious or moral condemnation. This stigma, internalized by patients and enacted by communities, fuels a vicious cycle: fear leads to concealment of symptoms and diagnostic delays; a diagnosis, when made, can lead to treatment default to avoid a stigmatized identity; and the resulting disease progression, with its visible deformities, reinforces the very stigma that initiated the cycle.[7] This treatment gap not only leads to preventable disability but also maintains a reservoir of infection within the community, undermining public health control efforts. The economic impact on individuals, particularly manual laborers who lose function in their hands and feet, and the toll on mental health and quality of life are immense and often unquantified.[8]

This case report presents a detailed, longitudinal account of a patient with borderline lepromatous leprosy who remained outside the healthcare system for 15 years after defaulting on his initial treatment. The novelty of this report lies not in the rarity of the diagnosis but in its comprehensive, multi-faceted analysis of the long-term consequences of this neglect, framed within the CARE guidelines. It offers a unique opportunity to dissect the slow, insidious progression of neuropathy, the immunological tipping point that led to an ENL reaction, and the critical role of stigma as a primary driver of pathology.[9,10] The aim of this study is to meticulously document this 15-year "shadow effect" by integrating the patient's narrative with detailed clinical, histopathological, and immunological analysis. By doing so, we seek to highlight the complex clinical reasoning required in managing such advanced cases and to issue a compelling call for a more integrated, patient-centered public health model that addresses not only the bacillus but the profound psychosocial barriers that allow it to thrive.

**2. Case Presentation**

The patient was a 53-year-old male from a rural farming community in West Sumatra, Indonesia. He was married with two children and had worked his entire life as a rice farmer. His educational background was limited to primary school. The narrative commences at Year 0, a critical starting point where the patient, at 38 years of age, receives an initial diagnosis of multibacillary leprosy and is initiated on a curative multidrug therapy (MDT-MB) regimen. This first event represents a moment of opportunity—a point at which medical science offers a clear path to cure and the prevention of disability. However, this initial optimism is tragically short-lived. The timeline starkly illustrates a catastrophic turning point just two months later: Treatment Default. As the figure notes, this was not a simple act of non-compliance but a decision rooted in fear of side effects and the powerful, coercive force of social stigma. This single event transforms the clinical trajectory from one of healing to one of prolonged, unmitigated disease progression. It is the inciting incident that casts a "15-year shadow" over the patient's life, a period of

disengagement that would allow the underlying pathology to flourish unchecked. The extensive gap between the second and third depicted events visually represents this 15-year period of therapeutic neglect, a silent interlude pregnant with immense biological significance. During this decade and a half, while the patient was absent from the clinic, the *Mycobacterium leprae* bacilli were not dormant. This period was one of insidious, relentless proliferation within the patient's Schwann cells and macrophages. The timeline's quiet expanse signifies a slow, ongoing assault on the peripheral nervous system, leading to progressive demyelination, chronic inflammation within the nerve trunks, and eventual axonal death. This long pathological process is the direct cause of the advanced state of the disease observed upon the patient's return. The functional decline was gradual, a slow erosion of sensation and strength that ultimately became too profound to ignore, leading directly to the next critical event on the timeline. At Year 15, the narrative arc pivots once more with the patient's re-presentation to the clinic. This moment signifies the culmination of 15 years of damage; the neurological symptoms of numbness and weakness had become functionally limiting, compelling the patient to overcome his long-held fears and seek help. This event marks the end of the period of neglect and the beginning of a renewed, more complex therapeutic encounter. The subsequent diagnostic confirmation, occurring just a week later, reveals the true cost of the preceding 15 years. The diagnosis of borderline lepromatous (BL) leprosy with a mild erythema nodosum leprosum (ENL) reaction is a direct reflection of the long-term untreated infection. The BL classification points to a high bacterial load and an unstable immune system, while the ENL reaction represents a systemic inflammatory state triggered by this massive antigenic burden. The bacteriological findings are particularly telling: a Bacteriological Index (BI) of +3 confirms a moderate-to-high bacterial population, and a Morphological Index (MI) of 5% provides definitive proof of viable, actively replicating bacilli. This crucial detail confirms that the patient's condition was not a relapse but a continuous, active infection. The final phase of the journey, as depicted in Figure 1, is one of recovery and rehabilitation. The re-initiation of treatment at Year 15 is critically distinguished from the first attempt by the inclusion of "intensive, patient-centered counseling". This highlights a pivotal shift in the management strategy—one that addresses the psychosocial barriers that led to the initial failure. It acknowledges that healing in leprosy requires treating the patient's fears as diligently as the infection itself. The ultimate outcome, documented at Year 16, is one of success: the patient completes the 12-month MDT-MB regimen, achieves bacteriological clearance with a BI of 0, and attains a "stable neurological status". The term "stable" is scientifically precise and poignantly informative; it signifies that the progressive nerve damage has been halted. While a victory, it also implies the permanence of the deficits acquired during the 15-year shadow, serving as an enduring reminder of the cost of delayed treatment. In its entirety, Figure 1 masterfully condenses a complex, 16-year medical and personal history into a clear, scientific, and profoundly human story, illustrating that the timeline of a chronic disease is not merely a sequence of clinical data points, but a narrative of human experience.

The journey into fifteen years of therapeutic neglect begins with the "Illness Narrative," which the figure identifies as the patient's internal belief system that catalyzed his treatment default. This domain provides the psychological genesis for the entire subsequent ordeal. The patient’s own words are captured, revealing a critical misinterpretation at the very outset of his treatment: "I thought the medicine was poison and was making it worse". This belief was formed when he observed his skin becoming darker, a known and reversible side effect of the drug clofazimine, which is a cornerstone of multidrug therapy. Lacking proper initial counseling, he interpreted this physiological reaction not as a sign of the medication's activity but as evidence of its toxicity.

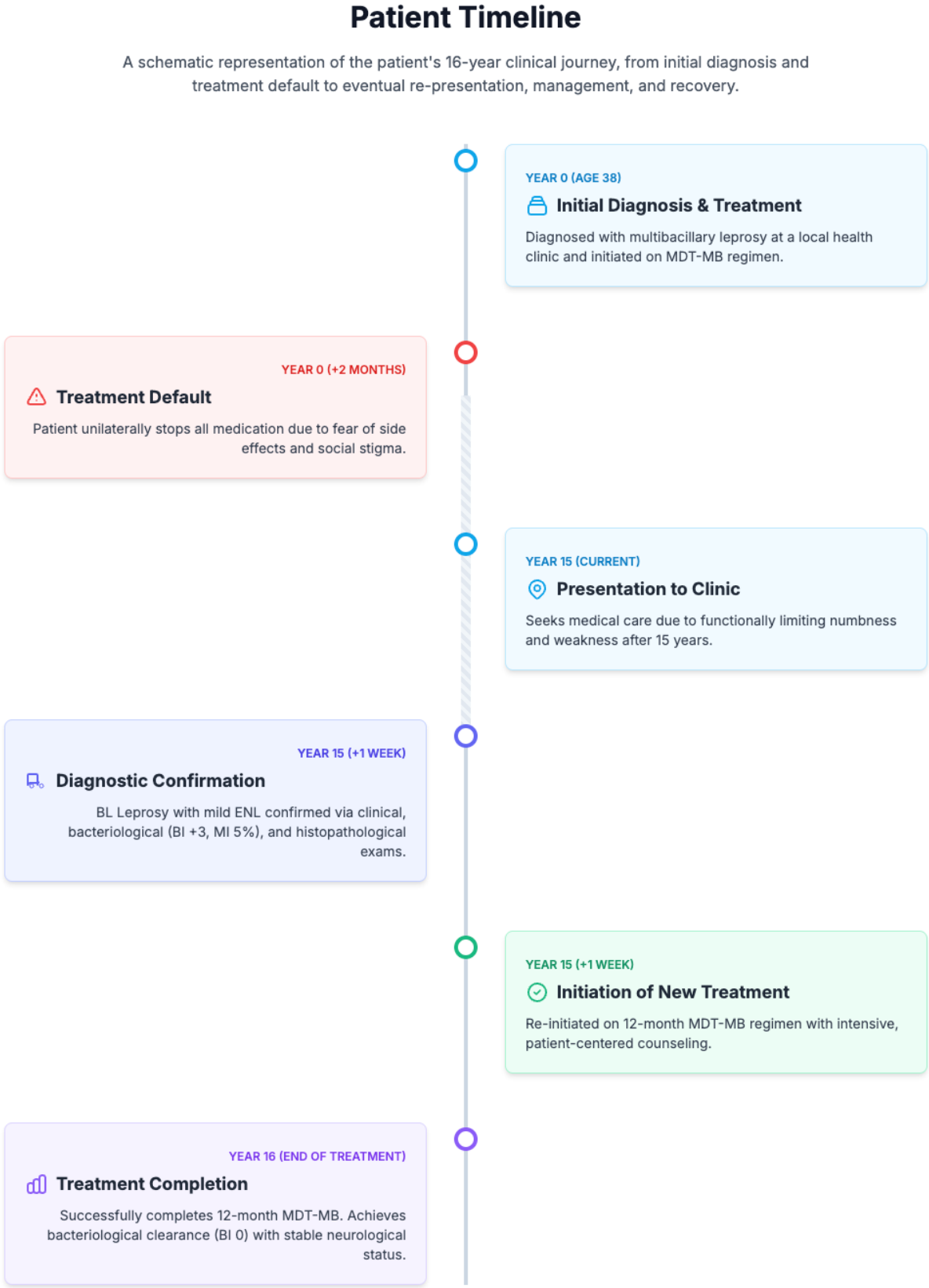


Figure 1. Patient timeline.

This misunderstanding created a powerful and deeply personal narrative in which the prescribed cure was reframed as a greater threat than the disease itself. This narrative became the logical foundation for his decision to abandon treatment, a decision that would have devastating consequences but which, from his perspective, was a rational act of self-preservation against a perceived poison. This deeply held illness narrative directly precipitated the cascade of negative outcomes detailed in the "Psychosocial Impact" domain. The fear of having a "poisonous" and disfiguring disease synergized with the pre-existing community stigma surrounding leprosy, leading to a state of profound fear of ostracism. As Figure 2 highlights, this was not a passive anxiety but one that translated into active behavioral changes. The patient engaged in deliberate social isolation, consciously avoiding community gatherings and social events where his condition might be discovered. This self-imposed exile, lasting over a decade, resulted in a state of chronic anxiety, which he aptly described as a "heavy burden to carry alone". This illustrates how the internal narrative of disease and the external pressure of stigma combine to dismantle a person's social identity, creating a feedback loop of fear and isolation that makes seeking help increasingly difficult. The "heavy burden" was the daily psychological weight of his secret, a weight that compounded year after year. While the patient contended with this immense psychosocial burden, the untreated *M. leprae* infection was waging a silent war on his body, leading to the severe outcomes detailed in the "Functional Impact" domain. This section of the figure demonstrates the physical manifestation of his fifteen years of neglect. The progressive decline in physical capacity began to affect the very core of his identity and livelihood. As a farmer, his hands were his most essential tools, and he reported increasing difficulty gripping his *cangkul* (hoe), which led to a direct reduction in his work capacity. This occupational decline was not an isolated symptom; it had direct and severe consequences, leading to lower crop yields and subsequent economic strain for his family. Furthermore, the insidious nature of leprous neuropathy is captured in the report of "recurrent, unnoticed minor injuries". The loss of protective sensation meant his hands and feet were being damaged without his brain registering the alarm of pain, a terrifying state of bodily alienation. This functional decay represents the point at which the consequences of his initial decision became unavoidably physical, eroding his ability to work, provide, and safely navigate his environment. The final domain in the schematic, "Motivation for Seeking Care," represents the dramatic climax of this 16-year narrative—the critical tipping point where the patient's long-standing pattern of avoidance was finally broken. For fifteen years, the fear of stigma had been the dominant force in his life. However, Figure 2 clearly outlines the factors that ultimately shifted this balance. The escalating symptoms of numbness and weakness became "functionally limiting and impossible to ignore," representing a daily, undeniable crisis that could no longer be intellectually suppressed. This chronic crisis was amplified by a new, more potent fear: the "catalyzing fear" of the complete and permanent loss of hand function. This was a specific, visceral terror that threatened his autonomy and future, a fear more immediate and personal than the social fear of ostracism. The culmination of this process was a state of "desperation," a point at which, as the figure powerfully states, the fear of disability finally outweighed the fear of stigma. It was this desperate calculus that finally propelled him to overcome the immense psychological barriers he had lived behind for more than a decade and re-engage with the healthcare system. In essence, At presentation, a thorough physical examination revealed advanced signs of multibacillary leprosy. The dermatological findings were extensive, with diffuse infiltration of the facial skin creating a subtle leonine facies, accompanied by madarosis and thickened earlobes. Symmetrically distributed, ill-defined, coppery-red plaques with shiny, anhidrotic surfaces covered his trunk and extremities.

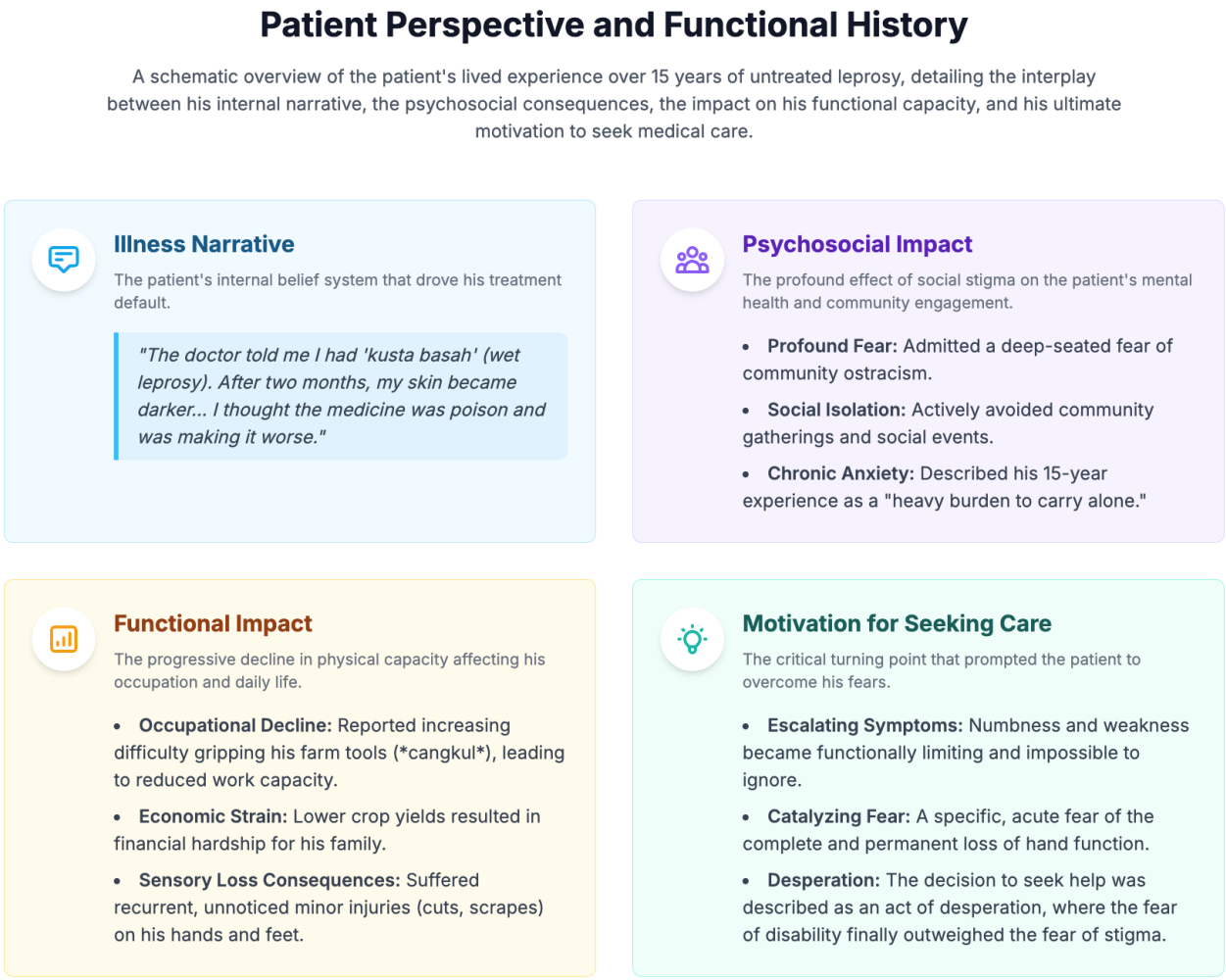


Figure 2. Patient perspective and functional history.

Palpation revealed a few tender, subcutaneous nodules on his shins and forearms, characteristic of ENL. Figure 3 presents a masterful and comprehensive synthesis of the patient's clinical status at the time of his re-engagement with the healthcare system. Acting as a detailed clinical map, the schematic elegantly breaks down the complex presentation of advanced leprosy into its distinct yet interconnected domains: dermatological, neurological, motor, and sensory. By integrating clinical photographs with concise, scientific descriptions, the figure provides a powerful, multi-layered view of the cumulative damage inflicted by fifteen years of untreated disease. It moves beyond a simple diagnostic label to quantify the extent of the pathology, offering a clear and objective foundation for understanding the patient's functional limitations and justifying his overall disability classification. The visual and textual evidence begins with the dermatological findings, which represent the most outwardly visible signs of the patient's high bacillary load. The figure details diffuse facial infiltration resulting in a subtle leonine facies, accompanied by madarosis (the characteristic loss of the outer third of the eyebrows) and thickened earlobes. These features are hallmarks of lepromatous-spectrum leprosy, indicating widespread infiltration of the facial dermis by *Mycobacterium leprae*-laden macrophages. Beyond the face, the schematic describes multiple, symmetrical, coppery-red plaques covering the trunk and extremities. The note that these surfaces are "anhidrotic" is a crucial detail, signifying damage to

the autonomic nerves supplying the sweat glands within the skin—an early sign of the pervasive neuropathy. Critically, Figure 3 also identifies the presence of a reactional state, Erythema Nodosum Leprosum (ENL), characterized by scattered, tender, erythematous subcutaneous nodules. This finding confirms that the patient's immune system was actively, albeit dysfunctionally, responding to the immense antigenic burden, creating a systemic inflammatory state on top of the chronic infection. The accompanying clinical photographs visually anchor these descriptions, providing incontestable evidence of the disease's extensive cutaneous impact. Transitioning from the skin to the underlying nervous system, the figure delves into the core pathology of leprosy. The "Neurological Examination" section details the palpable sequelae of chronic nerve inflammation. The bilateral thickening of the ulnar, posterior tibial, and peroneal nerves into firm, "rope-like" structures is a pathognomonic sign, representing years of granulomatous infiltration, edema, and eventual fibrosis within the nerve trunks. A particularly vital clinical sign highlighted in Figure 3 is the tenderness upon palpation of the ulnar nerves. This tenderness is the clinical correlate of active neuritis, an acute inflammation within the nerve that often accompanies ENL. It signals an ongoing, damaging process that puts the remaining nerve function at immediate risk. The non-tender nature of the other thickened nerves suggests a more chronic, "burnt-out" state of fibrotic damage. The direct consequences of this profound nerve damage are meticulously cataloged in the "Motor Function Assessment" and "Sensory Function Assessment" domains. The motor assessment reveals the visible atrophy of the intrinsic muscles of both hands, specifically the hypothenar and first dorsal interosseous muscles, which are primarily innervated by the ulnar nerve. The integrated photograph of the patient’s hands provides clear visual proof of this muscle wasting. This atrophy is quantified by the Medical Research Council (MRC) scale, which grades the patient's muscle strength as 4/5—indicating moderate weakness against resistance. Importantly, Figure 3 notes the absence of definitive claw hand or foot drop deformities. This is a crucial distinction, as it signifies that while muscle weakness and atrophy are present, fixed contractures have not yet developed. The "Sensory Function Assessment" details what is arguably the most insidious and dangerous aspect of leprous neuropathy. The classic "glove-and-stocking" pattern of hypoesthesia indicates a length-dependent peripheral neuropathy affecting the distal extremities first. The most critical finding presented here is the absence of "Protective Sensation" on the plantar surfaces of the feet and the ulnar aspects of the palms, as determined by the 10g monofilament test. This is not merely a symptom; it is a catastrophic functional loss. It means the patient's natural alarm system is broken; he can no longer detect the pressure and trauma that lead to tissue injury, placing him at an exceptionally high risk for developing painless ulcers, subsequent infections, and the eventual destruction of bone and tissue that leads to severe deformities. The additional finding of impaired pain and temperature discrimination further confirms damage to the small, unmyelinated nerve fibers, a process that is characteristic of the disease. Finally, Figure 3 synthesizes all these disparate findings into a single, internationally recognized metric in the "Overall Disability Grading" banner. The patient is classified as WHO Disability Grade 1. This grade is precisely justified by the evidence presented throughout the figure: the presence of sensory loss (loss of protective sensation) and mild weakness (MRC Grade 4/5), but crucially, the absence of Grade 2 deformities like ulcers or fixed clawing. Thus, the figure tells a complete story. It illustrates a patient standing on a precipice—a man whose body bears the deep scars of a 15-year infection but who has not yet fallen into the abyss of severe, irreversible deformity. It provides a comprehensive, baseline assessment that is essential for formulating a targeted rehabilitation plan aimed at preserving function and preventing any further progression of his disability


Figure 3. Clinical findings and functional assessment.

The primary diagnosis of leprosy was strongly suggested by the pathognomonic combination of skin lesions and thickened peripheral nerves. However, a differential diagnosis for the progressive sensorimotor neuropathy was considered and excluded. Diabetic neuropathy was ruled out by a normal random blood glucose level. The patient's social history lacked significant alcohol consumption, and his nutritional

status was adequate, making alcoholic or nutritional neuropathies unlikely. The highly specific clinical signs made other immune-mediated neuropathies like CIDP improbable. The diagnosis was definitively confirmed and classified through a series of investigations, summarized in Figure 4. A slit-skin smear was crucial, revealing a Bacteriological Index (BI) of +3 and a Morphological Index (MI) of 5%. The MI of 5% was particularly significant, as it confirmed a substantial population of viable, active bacilli, clearly distinguishing his condition from a late-stage, sterile reaction. A skin biopsy from a plaque on his back provided the final piece of the puzzle. Histopathology was pathognomonic for borderline lepromatous (BL) Leprosy and also captured the features of a mild ENL reaction.


Figure 4. Diagnostic reasoning and investigation.

A comprehensive, multidisciplinary management plan was instituted, detailed in Figure 5. The patient was re-initiated on the standard 12-month WHO MDT-MB regimen. A critical decision was made to manage his mild ENL conservatively, withholding systemic corticosteroids and relying on the anti-inflammatory properties of clofazimine. The cornerstone of the intervention was intensive, empathetic counseling to rebuild trust and empower the patient with knowledge. Adherence was closely monitored and found to be excellent. The patient responded well to treatment, with resolution of the ENL and bacteriological clearance at 12 months.

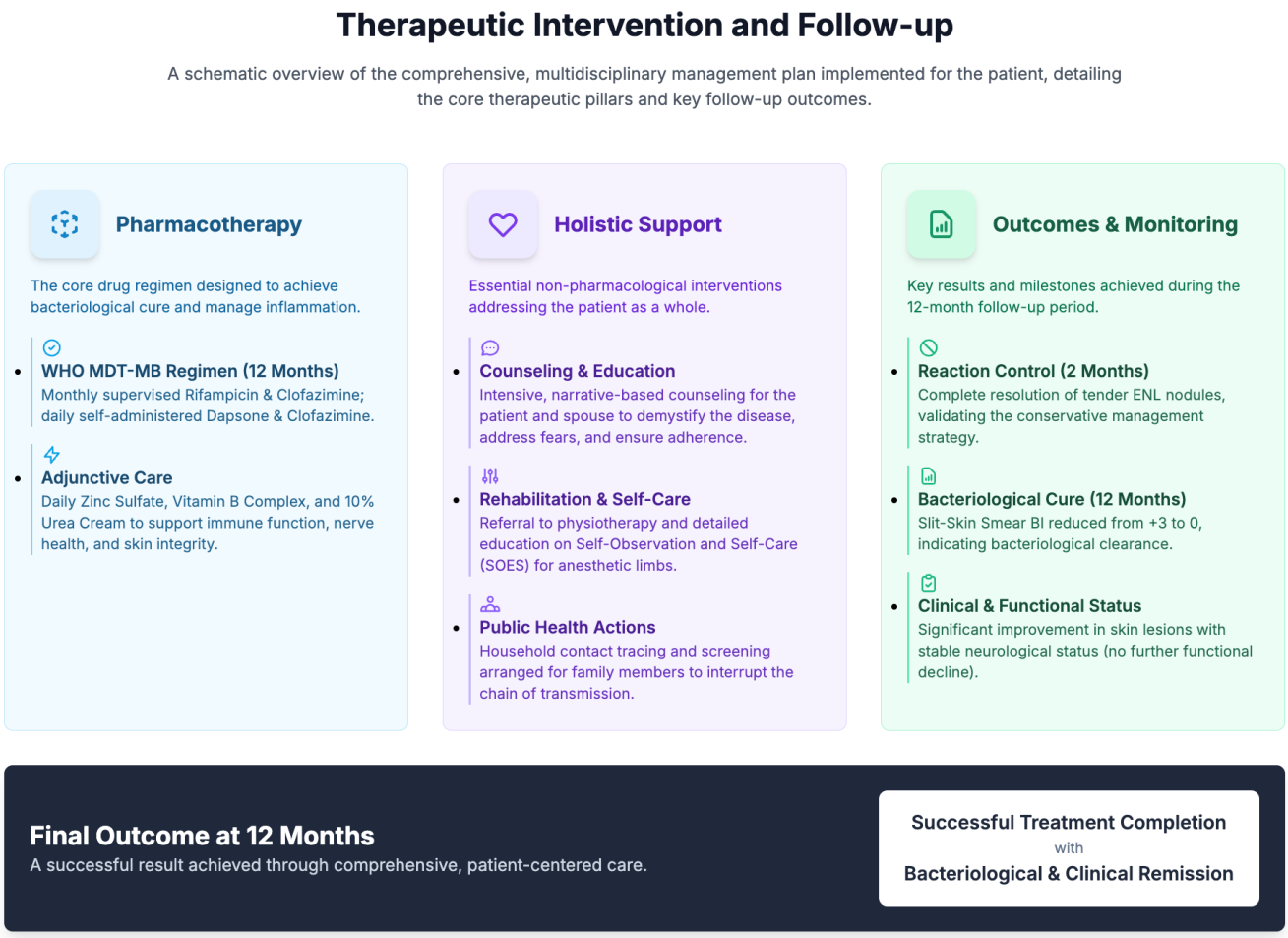


Figure 5. Therapeutic intervention and follow-up.

Written informed consent was obtained from the patient for the publication of this case report and any accompanying clinical images. The patient's identity has been kept confidential. All procedures were conducted in accordance with the ethical standards of the institutional research committee and with the Helsinki Declaration of 1975, as revised in 2013.

## 3. Discussion

This case report provides a sobering, long-term view into the consequences of untreated multibacillary leprosy, serving as a powerful nexus for discussing the intricate dance between host immunology, microbial pathogenesis, clinical management, and the overwhelming influence of social determinants of

health.[11] The 15-year journey of this patient from a frightened defaulter to a man with established disability is not an anomaly but a sentinel event that reflects systemic challenges in leprosy control. Our discussion dissects this case through multiple lenses: the diagnostic challenge in a long-term defaulter, the deep pathophysiology of the borderline immune state and its neurological sequelae, the nuanced clinical reasoning behind the management of his lepra reaction, and the overarching role of stigma as a driver of pathology and a target for public health intervention. The presentation of a patient with a history of leprosy and new symptoms after a long interval raises a critical diagnostic question: is this a relapse of a previously cured infection, or is it the progression of a continuously active, untreated disease? The distinction is vital, as it has implications for understanding treatment efficacy and pathogen resistance. A true relapse is defined as the re-emergence of disease after the successful completion of a full course of MDT.[12] In contrast, this patient only received two months of therapy. Therefore, his condition does not represent relapse but rather the predictable, unmitigated progression of his original infection. The key laboratory finding that confirms this is the Morphological Index (MI) of 5%. The MI is a microscopic assessment of the percentage of bacilli that are solidly stained, indicating an intact cell wall and, by inference, viability and metabolic activity.[13] An MI of 0% suggests that all visible bacilli are dead or dying. An MI of 5% in this patient, after 15 years, is definitive proof of a substantial population of actively replicating *M. leprae*. It confirms that the initial two months of therapy were insufficient to clear the infection and that he has remained a reservoir of active disease for a decade and a half. This finding is crucial for public health, as it underscores his potential to transmit the infection to close contacts throughout this period. This case highlights the importance of the MI in clinical decision-making, particularly in complex scenarios like treatment default, as it provides a direct, albeit imperfect, window into the bacteriological activity of the disease.[14]

The patient's diagnosis of borderline lepromatous (BL) leprosy places him in the most immunologically volatile region of the leprosy spectrum. This state is not a simple midpoint between tuberculoid (TT) and lepromatous (LL) leprosy but a dynamic, unstable battleground where competing immune pathways create a state of perpetual, ineffective inflammation.[15] At a molecular level, the BL state is characterized by a dysregulated cytokine milieu. Unlike the robust Th1 response (high IFN-γ, IL-2, TNF-α) of TT leprosy that effectively activates macrophages for bacterial killing, or the profoundly suppressed Th2 response (high IL-4, IL-10) of LL leprosy, the BL state features a chaotic mix of both. This immunological confusion is driven by several factors. Firstly, there is a functional deficiency in regulatory T cells (Tregs). While Tregs are essential for preventing autoimmunity, in lepromatous leprosy, their over-activity, driven by cytokines like IL-10 and TGF-β, suppresses the necessary Th1 response. In the BL state, this regulation is imperfect, leading to pockets of Th1 activity amidst a predominantly Th2 environment. This explains the presence of both foamy, bacillus-laden macrophages (an LL feature due to failed M1 polarization) and a significant lymphocytic infiltrate (a TT feature) in the patient's biopsy. Secondly, the innate immune response is compromised. Toll-like receptors (TLRs), particularly the TLR2/1 heterodimer, are crucial for recognizing *M. leprae* lipoproteins. In healthy individuals, this recognition triggers a MyD88-dependent signaling cascade that results in pro-inflammatory cytokine production and macrophage activation. In lepromatous patients, this pathway is dysregulated. Chronic exposure to the high bacillary load in our patient likely led to TLR tolerance and the induction of inhibitory molecules that dampen the inflammatory response, allowing the bacilli to persist.[16] This chronic, smoldering infection within macrophages provides the sustained antigenic stimulus that is the prerequisite for the development of ENL. The BL classification, therefore, is a prognostic marker for a lifetime risk of both Type 1 and Type 2

reactions, representing a state of immunological indecision that is ultimately disastrous for the host's nerves.[16]

The most devastating consequence of the patient's 15-year treatment gap is his permanent neurological disability. The pathogenesis of leprous neuropathy is a multi-step process of microbial invasion and immunologically-mediated collateral damage. *M. leprae*'s unique tropism for Schwann cells is the initiating event. The bacillus binds to the laminin-α2 receptor on the Schwann cell surface via its PGL-1 antigen, gaining entry into the one cell type responsible for maintaining the health of peripheral nerve axons.[17] Once inside, the bacillus reprograms the Schwann cell's biology. It induces a process of dedifferentiation, causing the cell to lose its myelinating phenotype and revert to a progenitor-like state. This has two consequences: first, it leads to segmental demyelination, slowing nerve conduction and causing the early symptoms of paresthesia; second, these dedifferentiated cells may acquire migratory properties, potentially helping to spread the infection along the nerve trunk. However, the majority of nerve destruction in a BL patient is not caused by the bacillus directly but by the host's own inflammatory response—an "outside-in" assault. The endoneurial space, normally protected by the blood-nerve barrier, becomes a battlefield. Infiltrating macrophages and lymphocytes release a toxic soup of inflammatory mediators, including TNF-α, nitric oxide, and reactive oxygen species. This inflammatory exudate creates pressure within the confined nerve fascicles, leading to ischemia, and is directly toxic to both Schwann cells and axons. Over 15 years, this chronic process led to widespread axonal death and replacement of functional neural tissue with fibrotic scar tissue, resulting in the thickened, non-functional nerves palpated on examination. The clinical findings in our patient perfectly map onto this pathophysiology: the loss of pain and temperature sensation reflects damage to small, unmyelinated C-fibers, while the loss of protective sensation (10g monofilament) and motor atrophy reflect the later, more severe destruction of large myelinated A-fibers. His Grade 1 disability, therefore, represents the endpoint of a 15-year-long war within his nerves.

The onset of ENL in this patient marks a critical juncture in his management. Our decision to classify the reaction as "mild" was based on a strict set of criteria: the absence of systemic symptoms (fever, malaise), the limited number of skin lesions, and, most importantly, the absence of signs of acute, severe neuritis (defined as nerve pain rated <5/10 on a visual analog scale, and no new or worsening motor or sensory deficit compared to his baseline). This classification justified our management strategy: a vigilant conservatism that withheld systemic corticosteroids. While corticosteroids are the undisputed treatment of choice for severe ENL with neuritis, their long-term use is fraught with peril, especially in a rural setting. The risks of iatrogenic Cushing's syndrome, uncontrolled hyperglycemia, severe osteoporosis, and reactivation of latent tuberculosis are substantial. Initiating steroids in this patient would have committed him to a complex tapering regimen requiring frequent monitoring, which would have been logistically challenging and costly.[18] Instead, we relied on the known anti-inflammatory properties of clofazimine, a core component of his MDT-MB regimen. Clofazimine is thought to exert its effect by stabilizing lysosomal membranes and inhibiting macrophage function and lymphocyte proliferation, thereby dampening the ENL cascade. This approach required a robust safety net. The patient was seen weekly for the first month, with explicit instructions to return immediately if he experienced increased nerve pain or weakness. This active monitoring was essential to ensure we could intervene rapidly with steroids if his "mild" reaction escalated into sight- or function-threatening neuritis. This case, therefore, demonstrates a key principle of clinical leprology: the importance of tailoring treatment not only to the disease activity but also to the patient's socioeconomic context and the realities of the local health system.

This single case should be viewed as a sentinel public health event, a tracer condition that illuminates multiple fractures in the local health system. The patient's 15-year journey of neglect represents a series of missed opportunities. First, the initial patient counseling was clearly insufficient. The failure to explain common side effects like clofazimine-induced pigmentation and to address the patient's fears directly led to his default. This points to a need for improved training for primary healthcare workers, focusing on patient-centered communication skills. Second, the health system lacked a mechanism for tracking and retrieving a high-risk defaulter. A patient with multibacillary leprosy who stops treatment is a public health priority, yet he was lost to the system for 15 years. This highlights the need for robust community-based tracking systems, potentially utilizing community health volunteers or mobile health (mHealth) technologies. Third, and most profoundly, this case is a stark indictment of the system's failure to address community-level stigma. Stigma is the root cause of this patient's disability. An effective leprosy control program cannot be confined to the clinic; it must actively engage in community destigmatization efforts. This involves partnering with community leaders, religious figures, and former patients to disseminate accurate information and promote social inclusion. The rehabilitation plan for this patient must therefore extend beyond physiotherapy; it must involve vocational counseling and support from patient self-help groups to help him reintegrate socially and economically. It is crucial to frame stigma not as a mere social issue but as a direct contributor to pathophysiology. The chronic stress induced by the fear of ostracism can be analyzed through the lens of psychoneuroimmunology. Prolonged psychological stress is known to dysregulate the hypothalamic-pituitary-adrenal (HPA) axis, leading to altered cortisol rhythms that can paradoxically suppress effective cell-mediated (Th1) immunity while promoting humoral (Th2) responses.[19] It is plausible that the patient's 15 years of chronic stress contributed biologically to the skewing of his immune response towards the ineffective lepromatous pole, thereby facilitating bacillary proliferation. Furthermore, the concept of the "illness narrative" is central. For 15 years, the patient's narrative was one of fear, poison, and isolation. The intensive counseling provided upon his re-presentation was a therapeutic intervention aimed at replacing this destructive narrative with a new one of understanding, empowerment, and cure.[20] By explaining the biological basis of his disease and treatment, the clinical team gave him the tools to reframe his experience. His subsequent excellent adherence is a testament to the power of this narrative-based medicine. Understanding the specific cultural idioms of distress related to skin disease in his community in West Sumatra would provide an even deeper layer of understanding and could inform the design of more culturally competent public health messaging.

The entire pathological cascade, as depicted in Figure 6, is set in motion by a single, critical event: the Initial Trigger of Treatment Default at Year 0. The figure correctly frames this not as a simple act of non-compliance, but as a decision explicitly "driven by stigma and misinformation". This initial step is paramount because it establishes the psychosocial roots of the subsequent biological devastation. It was this fear-based decision that opened the door for the "15-Year Shadow," a prolonged period during which the host's defense mechanisms were left to battle the persistent mycobacterial infection alone, without the aid of curative multidrug therapy. This initial trigger is the starting point from which all subsequent pathology flows. Following the trigger, Figure 6 bifurcates into two concurrent pathological processes that unfolded over the next 15 years. The first of these is the Immunological Dysregulation pathway. The journey begins with the patient's underlying "dysfunctional Th1/Th2 response of the borderline lepromatous (BL) state". This signifies an immune system in a state of chaos, incapable of mounting the effective cell-mediated (Th1) response required to kill the intracellular bacilli, yet not completely anergic like in polar lepromatous disease.


Figure 6. Pathophysiology related case findings.

This immunological indecisiveness is the perfect environment for the next stage in the cascade: Unchecked Bacterial Proliferation. Without an effective immune response to contain it, *M. leprae* replicated freely within the patient's macrophages and Schwann cells. As the figure notes, this led directly to a "high Bacillary Index (BI +3) and accumulation of mycobacterial antigens". This massive and sustained antigenic load saturated the patient's system, leading to the final step in this immunological pathway: Immune Complex Formation. The body produced a large quantity of antibodies against the bacterial

antigens, which then formed antigen-antibody complexes. These complexes, too numerous to be cleared efficiently, deposited in tissues and triggered a "Type III hypersensitivity reaction," the classical mechanism of erythema nodosum leprosum (ENL). This pathway provides a clear, linear explanation for how the patient's underlying immune status led directly to the inflammatory reactional state observed at presentation. Running in parallel to this immunological decline was the second pathway detailed in Figure 6: the insidious process of Neurological Damage. This cascade begins with the foundational event of leprosy neuropathy: *M. leprae* invasion of nerves. The schematic correctly identifies that the bacilli specifically "target and replicate within Schwann cells," the very cells responsible for maintaining the health and function of peripheral nerves. This initial invasion and colonization triggered the next stage: Chronic Nerve Inflammation. For 15 years, the presence of the bacilli within the nerves provoked a persistent, low-grade inflammatory response. The figure explains that this "inflammatory infiltrate causes demyelination, axonal damage, and fibrosis". This is a crucial summary of the destructive process: demyelination strips the nerves of their protective coating, axonal damage destroys the nerve fibers themselves, and fibrosis replaces functional neural tissue with non-functional scar tissue. The inevitable clinical result of this long-term destructive process is progressive neuropathy. The figure explicitly links this pathology to the patient's key symptoms, noting that it "manifests as sensory loss, muscle atrophy, and nerve thickening". The two destructive pathways converge at the end of the 15-year period, resulting in the Clinical Culmination graphically summarized at the bottom of Figure 6. The patient's presentation at year 15 is the logical sum of these parallel cascades. The immunological pathway produced the "mild erythema nodosum leprosum (ENL)," while the neurological pathway produced the profound nerve damage that resulted in "WHO Grade 1 Disability". The final diagnosis of "Advanced Borderline Lepromatous (BL) Leprosy" is the umbrella term that encompasses this entire complex state. Thus, the figure powerfully illustrates that the patient's condition was not a single problem, but a composite of pathologies that had evolved simultaneously over a decade and a half.

## 4. Conclusion

This case report chronicles the 15-year shadow that untreated leprosy cast over one man's life, a shadow woven from threads of microbial persistence, immunological chaos, and profound social fear. The patient's journey from fearful defaulter to a man with permanent disability, and finally to a successfully treated individual, is a microcosm of the enduring challenges and triumphs in global leprosy control. It powerfully demonstrates that the pathway to elimination is not paved with pharmacology alone. It must be built on a foundation of compassionate, patient-centered care that actively seeks to understand and dismantle the psychosocial barriers that prevent individuals from seeking and completing treatment. This case is a clear and urgent call to action for health systems worldwide: to invest in education, to fight stigma as aggressively as we fight the bacillus, and to build holistic, multidisciplinary programs that can prevent such preventable tragedies and finally bring all those who live in the shadow of leprosy into the light of a cure.

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