Post-MDTL Arthritis in Lepromatous Leprosy: A Case Report of Erosive Joint Disease

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ABSTRACT:
The musculoskeletal complications of Hansen's disease frequently mimic autoimmune rheumatic conditions and may emerge or persist despite the completion of antimicrobial regimens. This case report outlines the therapeutic assessment of an 18-year-old male diagnosed with lepromatous leprosy (LL) who developed progressive inflammatory polyarthritis affecting the proximal phalanges. Following a 12-month course of Multi-Drug Therapy for Leprosy (MDTL), the patient achieved Release from Treatment (RFT) status, with post-treatment slit-skin smear evaluation demonstrating a Morphological Index (MI) of 0—confirming eradication of viable Mycobacterium leprae—despite a persistently positive Bacteriological Index (BI) of 1.3+ reflecting residual non-viable bacillary antigens. Despite this confirmed bacteriological response, the articular inflammation persisted, necessitating targeted pharmacological management. This report emphasizes the critical need for diagnostic vigilance within endemic regions and advocates for integrated treatment pathways that combine antimicrobial agents with Disease-Modifying Antirheumatic Drugs (DMARDs) to avert irreversible structural deformities.

Keywords: Lepromatous Leprosy (LL); Progressive Inflammatory Polyarthritis; Multi-Drug Therapy for Leprosy (MDTL); Disease-Modifying Antirheumatic Drugs (DMARDs); Good health

Introduction

Hansen's disease, commonly known as leprosy, is a chronic granulomatous infection triggered by the obligate intracellular bacillus *Mycobacterium leprae*. On a global scale, this disease remains a fundamental public health challenge, with Indonesia consistently ranking third worldwide in new case contributions. The primary pathology of *M. leprae* is driven by its specific tropism for dermal macrophages and peripheral nervous system Schwann cells. Nevertheless, the clinical spectrum of this illness extends beyond neurocutaneous boundaries; musculoskeletal involvement is documented in up to 75% of affected individuals, representing the third most prevalent clinical presentation following dermal lesions and neuropathy.[1]

Articular manifestations in leprosy can present across various disease stages—whether as a prodromal sign, during the active treatment phase, or following microbial eradication. Such occurrences frequently create substantial

diagnostic dilemmas, primarily because their clinical and radiological features present as symmetrical polyarthritis, closely resembling primary autoimmune disorders like Rheumatoid Arthritis (RA) or Systemic Lupus Erythematosus (SLE). Overlapping clinical presentations—such as morning joint stiffness, soft tissue swelling, and eventual bone erosion—often precipitate misdiagnosis. These diagnostic errors carry severe risks; administering misguided immunosuppressive therapy to an undiagnosed leprosy patient can provoke the systemic dissemination of *M. leprae* bacilli.[2] Furthermore, the conventional paradigm in contemporary leprosy management relies heavily on the microbiological elimination of the pathogen. While Multi-Drug Therapy for Leprosy (MDTL) has proven highly effective in achieving clearance of viable *Mycobacterium leprae*—as reflected by a Morphological Index (MI) of 0—the foremost challenge in modern clinical practice has shifted from mere bacterial eradication to managing persistent immunological sequelae. Degraded bacillary fragments, which may continue to be reflected in a slowly declining Bacteriological Index (BI) despite the absence of viable organisms, frequently trigger immune hyperactivation and prolonged pro-inflammatory cytokine cascades, leading to sustained destruction of the articular cartilage infrastructure.[3] This case report illustrates a case of progressive arthritis in leprosy to underscore the critical discrepancy between confirmed antimicrobial success—defined here as eradication of viable bacilli rather than mere completion of therapy—and the continuation of musculoskeletal morbidity driven by sterile inflammation. The present report highlights the urgent need to update post-MDTL pharmacotherapy and drug management strategies—specifically by integrating anti-inflammatory and disease-modifying agents—to prevent irreversible joint deformities among populations in endemic territories..

Case Presentation and Therapeutic Intervention

In September 2024, an 18-year-old male patient was referred to the Rheumatology Outpatient Clinic presenting with chronic swelling and digital deformities that had progressed over a two-year period. The disease trajectory began as monoarticular swelling that evolved into a polyarticular pattern within six months, accompanied by sensory neuropathy (numbness), moderate articular pain (Visual Analogue Scale [VAS] scores of 3-4), and a hallmark rheumatological complaint of morning stiffness lasting between 30 and 45 minutes. An initial dermatological evaluation conducted in October 2022 confirmed a diagnosis of lepromatous leprosy (LL), characterized by persistent erythematous macular lesions, skin thickening, and a bacteriological index of 3+ for *M. leprae* on earlobe slit-skin smears.

The patient was initiated on a standard first-line Multi-Drug Therapy for Leprosy (MDTL) regimen consisting of monthly Rifampicin (600 mg), monthly (300 mg) and daily (50 mg) Clofazimine, and daily Dapsone (100 mg) for a duration of 12 months. By October 2023, he attained Release from Treatment (RFT) status. Post-treatment slit-skin smear evaluation demonstrated a Morphological Index (MI) of 0, confirming the absence of viable, solid-staining bacilli, despite a persistently positive Bacteriological Index (BI) of 1.3+ reflecting residual non-viable bacillary debris—a finding consistent with successful eradication of live *M. leprae* organisms and comprehensive dermatological recovery. Despite this bacteriological response, subsequent rheumatological assessment eleven months post-MDTL revealed an unabated progression of digital joint deformities, even though the active joint pain had subsided.

General physical examination revealed stable hemodynamics alongside characteristic residual leprosy signs, notably facial madarosis. Musculoskeletal evaluation utilizing the Gait, Arms, Legs, and Spine (GALS) diagnostic framework yielded normal parameters for gait, the lower extremities (hip and knee joints), and the vertebral column. In the upper extremities, the range of motion (ROM) for the shoulder, elbow, and wrist joints remained within normal limits. Specific articular provocation

assessments—including Tinel's, Phalen's, Finkelstein's, and the MCP Squeeze tests—were entirely negative, effectively ruling out differential diagnoses of nerve entrapment (carpal tunnel syndrome) or metacarpophalangeal inflammation. Pathological findings were exclusively localized to the proximal interphalangeal (PIP) joints of digits I-V on both the right and left hands, exhibiting soft tissue edema, erythema, warm acral temperatures, and pronounced structural deformities (Figure 1).

Figure 1. Clinical photograph of the hands

A comprehensive ancillary investigation encompassing hematological profiles, renal function (BUN/SCr), hepatic panels (SGOT/SGPT), and urinalysis yielded parameters within normal reference ranges. Viral co-infections were excluded (HBsAg and Anti-HCV were non-reactive). However, a systemic inflammatory marker elevation was detected, with an Erythrocyte Sedimentation Rate (ESR) peaking at 50 mm/hour. Radiological evaluation of the right hand unveiled marginal erosions on the distal aspects of the proximal phalanges of digits II, III, and IV. This was accompanied by soft tissue swelling while maintaining preserved joint spaces. These radiographic findings confirmed the presence of inflammatory arthritis (Figure 2).

Figure 2. Radiograph of the hands

Synthesizing the clinical findings, underlying medical history, and radiographic evidence, the patient was diagnosed with leprosy-related arthritis. Symptomatic therapeutic management was initiated using Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), specifically Diclofenac sodium 50 mg administered every 12 hours on a *pro re nata* basis. A long-term clinical projection has been established to escalate interventions utilizing Disease-Modifying Antirheumatic Drugs (DMARDs) should pain manifestations recur or functional deterioration emerge due to ongoing articular erosion.

Therapeutic Discussion

The comprehensive management of arthritis in leprosy dictates a strict clinical paradigm separation between the eradication of the infectious agent and the suppression of persistent immune responses.[4] Microbiologically, the MDTL regimen exhibits profound efficacy in drastically reducing the *Mycobacterium leprae* bacillary load, as evidenced in this patient by a post-treatment Morphological Index of 0.[5] However, the body's clearance of viable bacilli does not inherently align with immunological resolution within the joint spaces.[6] Antigenic fragments originating from deceased bacteria (such as lipoarabinomannan) can persist within macrophages and synovial tissues, continuously inciting cross-reactive immunological responses.[7] This phenomenon activates the complement cascade and stimulates the relentless release of pro-inflammatory cytokines (including TNF-α and IL-6), manifesting as chronic synovitis.[8] This mechanism is highly analogous to the immune complex-mediated reactions observed in Type II leprosy reactions, or Erythema Nodosum Leprosum (ENL).[3,9]

At this clinical juncture, the primary diagnostic hurdle involves distinguishing reactive arthritis induced by leprosy from definitive autoimmune disorders like Rheumatoid Arthritis (RA), owing to their virtually identical symmetrical polyarticular manifestations.[10] Individuals with Hansen's disease frequently present with false-positive Rheumatoid Factor (RF) serology, a consequence of persistent polyclonal B-cell overstimulation.[11] Therefore, relying on highly specific rheumatological autoantibodies—most notably the anti-cyclic citrullinated peptide (anti-CCP)—is an indispensable step in the serological workup. Anti-CCP carries profound diagnostic specificity for RA and has robust clinical evidence demonstrating minimal positivity rates among leprosy populations.[11] In this patient, the negative anti-CCP result provided definitive confirmation that the joint inflammation was exclusively an immunological sequela of leprosy, effectively ruling out a concurrent RA diagnosis.

Although the antimicrobial regimen involving Rifampicin, Clofazimine, and Dapsone had successfully achieved eradication of viable *M. leprae*—confirmed biochemically rather than by RFT status alone—the radiologically confirmed marginal erosions signaled the presence of active, "sterile" destructive inflammation.[12] Such findings corroborate that pathological bone remodeling and structural joint deterioration can advance independently of an active *M. leprae* infection, fueled entirely by lingering immunological memory within the synovial cavity.[13]

Consequently, the pharmacological strategy following MDTL completion must transition its focus toward the enduring preservation of the patient's articular architecture.[14] Prescribing NSAIDs, including Diclofenac sodium, remains a highly sensible first-line intervention to mitigate acute pain and control inflammatory exacerbations.[15] However, the emergence of osseous erosions indicates that the inflammation has advanced into a tissue-destructive stage. Strict clinical monitoring is critical; should the joint inflammation become refractory or the physical deformities worsen, escalating the therapy to include immunosuppressants like Disease-Modifying Antirheumatic Drugs (DMARDs)—such as Methotrexate—or targeted immunomodulators like Thalidomide becomes a clinical necessity to avert irreversible joint disability induced by chronic leprosy reactions.[3,16,17]

Conclusion

Confirmed eradication of viable *Mycobacterium leprae*—as demonstrated by a Morphological Index of 0—does not universally guarantee the cessation of progressive musculoskeletal complications. This case report reinforces the necessity for clinical vigilance regarding arthritis in leprosy among patients from endemic regions who present with polyarthritis, even if they possess a successful Release from Treatment (RFT) history confirmed by bacteriological evaluation. A combination treatment strategy that simultaneously targets the pathogen and controls immunological synovitis is critically essential within modern leprosy therapeutic algorithms.

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Declarations

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Availability of data and material: All data generated or analysed during this study are included in this published article.

Competing interests: The authors declare that they have no competing interests.

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Authors' contributions: ARK diagnosed the patient and was a major contributor in writing the manuscript. CWN supervised the clinical management and revised the manuscript for intellectual content. All authors read and approved the final manuscript.

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