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CASE REPORT

# When leprosy mimics tuberculosis: A diagnostic challenge between lepromatous leprosy and pulmonary tuberculosis in an indigenous community – A case report

Sabrina Mokhtar[1,2], MD ; Noor Azimah Muhammad[1], PhD ; Petrick Periyasamy[3], MMed

[1] Department of Family Medicine, Faculty of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur Campus, Kuala Lumpur, Malaysia.
[2] Ministry of Health Malaysia
[3] Department of Medicine (Infectious Disease), Faculty of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur Campus, Kuala Lumpur, Malaysia.

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**Corresponding Author:**

**Sabrina Mokhtar**
Department of Family Medicine, Faculty of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia.
Email: sabrinamokhtar159@gmail.com

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## Abstract

Lepromatous leprosy can pose a significant diagnostic challenge in endemic regions where tuberculosis is also prevalent. We report the case of a 64-year-old Orang Asli Temuan man from Kuala Kubu Bharu, Hulu Selangor, Selangor, initially misdiagnosed with pulmonary tuberculosis following a positive sputum acid-fast bacilli (AFB) smear. The patient presented with grade 2 leprosy-related deformities including nasal cartilage destruction, autoamputation of the digits over the bilateral hands and feet with chronic ulcers and leonine facies. Slit-skin smear demonstrated a high bacteriological index (2.7), while his chest radiograph was inconclusive. Despite initial sputum AFB positivity, GeneXpert MTB/RIF and mycobacterial cultures repeatedly showed negative findings. These findings were attributed to bacillary shedding from the upper airway, a known but under-recognised phenomenon in multibacillary disease. The case highlights the risk of diagnostic confusion between leprosy and tuberculosis when relying solely on smear microscopy. Anti-tuberculous therapy and multidrug therapy (MDT) were temporarily withheld due to drug-induced liver injury. Pulmonary tuberculosis was excluded via negative rapid molecular testing and thoracic CT, and MDT was restarted. This case underscores the importance of confirmatory molecular testing, early diagnosis and culturally sensitive public health measures in indigenous communities affected by ongoing leprosy transmission.

## Introduction

Leprosy (Hansen's disease), caused by *Mycobacterium leprae* and *Mycobacterium lepromatosis*,[1] remains a public health concern in Southeast Asia despite global elimination efforts. It is a chronic granulomatous infection affecting the skin, peripheral nerves, upper respiratory tract mucosa and eyes.[1] Transmission occurs via respiratory droplets during close, prolonged contact with untreated cases, with an incubation period of 5–20 years.[2]

Globally, approximately 180,000–200,000 new cases are diagnosed annually, with high endemicity in Southeast Asia, the Americas, India, Brazil and Indonesia.[2] Although Malaysia achieved the World Health Organization (WHO) elimination target of <1 case per 10,000 population in 1994,[3] recent trends are concerning: The number of new cases increased by 40%, from 183 in 2022 to 256 in 2023.[2] In February 2025, an outbreak among the Orang Asli in Negeri Sembilan resulted in nine new cases and one death, underscoring the vulnerability of marginalised communities.[4]

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The Orang Asli population experiences a higher disease burden than non-indigenous groups, largely due to socioeconomic disparities, limited healthcare access, poor sanitation and inadequate clean water supply.[5] These factors promote ongoing transmission and delayed diagnosis.

Lepromatous leprosy (LL), the most severe form of the disease spectrum, is characterised by high bacterial load and widespread skin and systemic involvement.[6] Diagnostic challenges arise when patients with LL present with positive sputum acid-fast bacilli (AFB) smears. While this may suggest concurrent pulmonary tuberculosis (PTB), it can be misleading, as AFB positivity may result from *M. leprae* or non-tuberculous mycobacteria (NTM) colonising the nasal and upper respiratory mucosae of patients with multibacillary disease.[7,8] Misinterpretation can lead to unnecessary anti-tuberculosis (TB) therapy, with risks of hepatotoxicity and drug interactions with multidrug therapy (MDT) for leprosy. This case highlights the importance of confirmatory molecular diagnostics before initiating anti-TB therapy in patients with leprosy.

## Case presentation

A 64-year-old man from the Orang Asli Temuan community was referred by the Inspectorate and Environmental Health team for clinical evaluation on 21 February 2025. He indicated a history of a long-standing, progressively worsening deformity of the hands with loss of his digits since the age of 30 years. The patient attributed these changes to a work-related injury and reported that the deformity did not interfere with activities of daily living, accounting for the delayed presentation for medical evaluation. He had no active systemic symptoms such as fever, cough or night sweats. There was no dyspnoea, gastrointestinal or urinary symptoms, joint pain or swelling or cutaneous numbness. There was no known exposure to TB or leprosy.

On examination, his temperature was 37°C; blood pressure, 140/87 mmHg; pulse rate, 76 beats per minute; and body mass index, 18.2 kg/m$^2$. There was no lymphadenopathy. He had leonine facies with madarosis, a deformed nose and prominent auricles. He had WHO grade 2 impairments of the hands and feet, characterised by autoamputation of the digits in both hands and feet, claw hands, chronic ulceration, scarring, hypopigmentation and associated sensory loss (**Figure 1**). Other systemic examination findings were unremarkable.




**Figure 1.** Clinical photograph of the patient with lepromatous leprosy. (A) Frontal view of the face showing leonine facies with madarosis, a deformed nose and prominent auricles. (B) Lateral view highlighting the prominent auricles. (C) Hypopigmented, dry patches and autoamputation of the digits of the hands. (D) Multiple hypopigmented patches, scarring and chronic ulceration of the feet.

Blood investigations showed that the patient had normochromic normocytic anaemia with a haemoglobin level of 10 g/dL and a raised erythrocyte sedimentation rate (ESR) of 42 mm/h. His other investigation findings such as renal profile, liver function, G6PD test result, random blood sugar level and urinalysis result were normal. Screening tests for hepatitis B, hepatitis C, HIV and syphilis also yielded negative results. Slit-skin smear microscopy revealed a bacteriological index (BI) of 2.7, indicating a high bacillary load (**Table 1**).

**Table 1.** Slit-skin smear results.

| Site | Density | Solid | Fragmented |
|---|---|---|---|
| 1. Right earlobe | 2+ | 0 | 100 |
| 2. Left earlobe | 3+ | 0 | 100 |
| 3. Right forearm | 3+ | 0 | 100 |
| Date: 19/2/2025<br>Bacteriological index: 2.7<br>Morphological index: 0.0 | | | |

Sputum smear microscopy showed AFB positivity on two occasions, while chest radiography demonstrated non-specific findings, including prominent right perihilar haziness (**Figure 2**).




**Figure 2.** Chest radiograph taken on 21/2/2025.

Based on his clinical presentations, the patient was diagnosed with smear-positive PTB with concurrent LL. On 22 February 2025, first-line anti-TB therapy, consisting of isoniazid 300 mg, rifampicin 600 mg, pyrazinamide 1250 mg, ethambutol 1000 mg and pyridoxine 10 mg daily, was commenced under respiratory physician guidance based on the positive sputum AFB smear. MDT for leprosy was initiated concurrently, comprising monthly clofazimine 300 mg and rifampicin 600 mg, followed by daily clofazimine 50 mg and dapsone 100 mg. However, he developed significant hepatotoxicity within 2 weeks of treatment, with an ALT level of 155 U/L and an AST level of 465 U/L, accompanied by an elevated total bilirubin level of 33.5 µmol/L. Therefore, both anti-TB and MDT regimens were discontinued. Molecular and microbiological tests were arranged to confirm the diagnosis. The combination of negative molecular assays, absence of growth in TB culture and reduction in AFB smear positivity led to diagnostic revision (**Table 2**). Thoracic computed tomography (CT) was performed to increase diagnostic confidence, which showed right middle lobe atelectasis with no features of active pulmonary disease. The final diagnosis was revised to multibacillary LL without PTB. Following normalisation of liver enzymes on 14 March 2025, MDT for LL was cautiously reintroduced using rifampicin 600 mg monthly, clofazimine 300 mg monthly plus 50 mg daily and dapsone 100 mg daily, with close monitoring of liver function. Liver function remained within normal limits, but on 5 May 2025, his haemoglobin levels gradually declined from 9.5 g/dL to 7.9 g/dL over the course of 2 months. Further evaluation revealed Southeast Asian ovalocytosis, and dapsone-induced anaemia was considered. In response, dapsone was discontinued, and an alternative second-line regimen with ofloxacin 400 mg daily was initiated, while rifampicin and clofazimine were continued. Subsequent monitoring showed an improvement in his haemoglobin levels, alongside clinical progress throughout the planned 12-month treatment duration.

**Table 2.** Serial sputum acid-fast bacilli and molecular test results.

| Date | 21/2/2025 | 7/3/2025 | 13/3/2025 | 19/3/2025 |
|---|---|---|---|---|
| Sputum AFB | 2+ | 2+ | 1+ | No AFB seen |
| Sputum GeneXpert MTB-RIF | | MTB not detected | | |
| Sputum MTB C&S | No growth* | | | |
| Sputum MDR molecular | | | MTB complex not detected | |
| Sputum MTB PCR | | | DNA not detected | |
| Sputum *M. leprae* PCR | | | | DNA not detected |

Abbreviations: AFB, acid-fast bacilli; C&S, culture and sensitivity; DNA, deoxyribonucleic acid; MDR, multi-drug resistant; MTB, *mycobacterium tuberculosis*; PCR, polymerase chain reaction; RIF, rifampicin.
* Sputum sample collected on 21/2/2025 showed no growth following standard incubation; final culture confirmation was reported on 23/4/2025.

## Discussion

This case illustrates a critical diagnostic challenge in clinical practice: A positive sputum AFB smear in a patient with leprosy may be misinterpreted as evidence of concurrent active PTB.[8] In patients with LL, particularly those with a high BI, such as in this case (BI: 2.7), the nasal mucosa and upper respiratory tract harbour large amounts of *M. leprae* bacilli.[7] In LL, bacilli proliferate extensively in cooler body areas, including the nasal and upper respiratory mucosae.[6] During coughing or sneezing, these organisms are shed into sputum and bronchial secretions. When examined using conventional AFB microscopy, *M. leprae* bacilli stain as acid-fast organisms, producing results indistinguishable from *M. tuberculosis* on morphology alone.[6] Additionally, NTM may appear as transient colonisers or contaminants in respiratory specimens without causing disease, leading to false-positive AFB smears in patients evaluated for TB.[9]

The patient in this case presented with classical LL features, including grade 2 deformities, leonine facies, claw hands and autoamputation, which are hallmark features of high bacillary burden. The positive sputum AFB smear could have easily been misinterpreted as PTB, particularly with concurrent right perihilar haziness on chest radiography. The diagnosis of PTB was made purely from sputum AFB direct smear in a background of neither PTB symptoms nor a diagnostic chest radiographic change.

Subsequent molecular testing provided diagnostic clarity. The GeneXpert MTB/RIF assay, a WHO-recommended initial diagnostic test for TB diagnosis and rifampicin resistance,[10] yielded negative results, effectively ruling out TB. This was confirmed by negative MTB culture and MTB PCR results, verifying the absence of TB complex DNA. The *M. leprae* PCR from sputum also yielded negative results, suggesting that the bacilli detected earlier were shed from colonised upper respiratory sites rather than representing pulmonary infection.[7] The clinical course supported this interpretation, as the sputum AFB smear grade decreased from 2+ to 1+ and became negative within 2 weeks, consistent with natural clearance of colonising organisms. The thoracic CT findings further supported non-TB infection. The normalisation of liver function after judicious reintroduction of MDT indicated a drug-induced mechanism rather than other causes.

When true TB–leprosy coinfection occurs, management is challenging due to overlapping drug toxicities. Rifampicin, a key component of both MDT and anti-TB therapy,[8] together with isoniazid and pyrazinamide, can induce hepatotoxicity, particularly when used concurrently.[11] WHO and Malaysian guidelines recommend simultaneous treatment of both diseases once TB is microbiologically confirmed, with close liver function monitoring and regimen modification if toxicity develops.[11–13] Additionally, immune activation during TB treatment may precipitate type 2 leprosy reactions such as erythema nodosum leprosum,[8] although this was not observed in our patient.

From a primary care perspective, this case highlights the importance of confirmatory molecular testing before commencing anti-TB therapy in patients suspected to have leprosy. While empirical treatment may be justified in high-burden or resource-limited settings, rapid molecular diagnostic tests for TB are increasingly available across Malaysian healthcare facilities and should be prioritised to prevent unnecessary drug exposure. The case also highlights ongoing transmission risks in marginalised communities, reinforcing the need for contact tracing, evaluation for leprosy signs and administration of single-dose rifampicin prophylaxis (600 mg for adults) once active disease has been excluded.[13,14]

The long-standing nature of the symptoms of our patient without appropriate medical treatment warrants attention in the current health system. The delayed case detection may be due to stigma, limited awareness and restricted healthcare access among indigenous populations. Community education, stigma reduction and active case-finding initiatives by healthcare professionals and community champions are vital for reaching the WHO's zero-leprosy goal by 2030.[14]

Certain limitations in this case warrant mention. Only sputum samples were analysed; bronchoscopic sampling or bronchoalveolar lavage might have yielded more definitive results but was not clinically indicated following negative molecular test findings. Additionally, incomplete epidemiological data and delayed molecular confirmation led to prolonged exposure to potentially hepatotoxic drugs.

## Conclusion

A positive sputum AFB result in a patient with LL does not inherently indicate active TB. The utilisation of rapid molecular diagnostic tests, such as GeneXpert MTB/RIF or PCR, is paramount to preclude unnecessary anti-TB therapy and mitigate potential drug-related toxicities. Particularly within indigenous communities, primary care clinicians must recognise this diagnostic pitfall, reinforce syndromic screening and expedite contact tracing. Ensuring timely access to rapid diagnostic tools is essential to optimise clinical outcomes and curtail inappropriate antibiotic use.

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## Conflicts of interest

None.

## Author contributions

SM identified the case and wrote the manuscript. NAM provided case review, critical revision, and expert advice. PP contributed to case review and expert advice.

## Patient's consent for the use of images and content for publication

Written informed consent was obtained from the patient for the publication of this case report.

**What is new in this case report compared to the previous literature?**

- This case shows that sputum acid-fast bacilli positivity in lepromatous leprosy can be caused by *Mycobacterium leprae* or non-tuberculous mycobacteria, not necessarily active pulmonary tuberculosis, highlighting diagnostic challenges and the importance of molecular confirmation before anti-tuberculosis treatment.
- The patient is an Orang Asli affected during an outbreak, highlighting delayed diagnosis and ongoing burden among indigenous communities.
- Early diagnosis and appropriate therapy minimised risk of further disability, supported rehabilitation and helped preserve quality of life.
- This report offers practical recommendations for rural and primary care providers, including syndromic screening, rapid referral, contact tracing and single-dose rifampicin prophylaxis, relevant in Malaysian settings.

**What is the implication to patients?**

The implementation of rapid molecular diagnostics and multidisciplinary care, as shown in this case report, enables accurate treatment, preserves physical function, reduces psychological stress and stigma and supports social well-being for patients with leprosy, particularly those at risk of disability and isolation in vulnerable communities.

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