

Case Report

Delayed Diagnosis of Leprosy in a Patient with Pulmonary Tuberculosis: Lessons for Clinical Practice

Novianti R Reza^{1,3}, Margaretha Nathania^{1,2,3}, Karunia R Dewi^{1,2,3}, Cita RS Prakoeswa^{2,3}

¹ Department of Dermatology, Venereology and Aesthetic, Universitas Airlangga Hospital, Surabaya, Indonesia

² Department of Dermatology, Venereology and Aesthetic, Dr Soetomo General Academic Hospital, Surabaya, Indonesia

³ Department of Dermatology, Venereology and Aesthetic, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia

Abstract

Introduction: Tuberculosis and leprosy are infections caused by the *Mycobacterium sp.* Both are significant health problems in Indonesia. Delayed diagnosis is still common in leprosy, causing disability.

Case Presentation: A 51-year-old with low socioeconomic status complained of cough, weight loss, and decreased appetite for more than a month. Anamnesis, physical examination, sputum, and Chest X-ray were consistent with pulmonary tuberculosis. The 10-month-old skin lesions became more inflamed one week after antituberculosis treatment. Dermatological examination revealed hyperesthetic lesions, enlargement of peripheral nerves, and negative Acid-Fast bacilli (AFB) results. He was diagnosed with multibacillary leprosy, type 1 reaction, grade 2 disability, and pulmonary tuberculosis. Treatment includes antituberculosis therapy, Multidrug Therapy, Multibasilar and corticosteroids.

Discussion: A complex relationship existed between socioeconomic factors, environment, nutritional status, access to health services, and the intricate host immune system in co-infection with *Mycobacterium leprae* and *M. tuberculosis*, which affected the occurrence of delays in our case. The host immune response to TB treatment likely triggered recognition of leprosy through an immune-modulatory mechanism, manifesting as a type 1 reaction. This complexity also presented a challenge in the diagnosis and treatment process, especially in developing countries.

Conclusions: This case illustrated the complexity of tuberculosis and leprosy infections, particularly in terms of risk factors, immune system interactions, and therapeutic interactions. Comprehensive management is important to prevent delays in diagnosis prevent complications such as disability, and reduce the risk of transmission of both diseases in the community.

Key words: coinfection; delayed diagnosis; leprosy; tuberculosis; case report.

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Introduction

Leprosy and tuberculosis are chronic infectious diseases caused by *Mycobacterium leprae* and *Mycobacterium tuberculosis*, respectively [1,2]. *Mycobacterium* infection is a granulomatous infection influenced by the host's immune system, which will affect clinical symptoms and the patient's clinical course. Leprosy, an infection primarily affecting the skin and peripheral nervous system, while tuberculosis predominantly involves the lungs, although extrapulmonary manifestations can occur in some cases. Co-infection of both diseases presents challenges due to the complex interplay of various factors, including environmental, genetic, and immune system influences, which can lead to overlapping clinical symptoms and treatment considerations [3].

Leprosy and tuberculosis often occur in the same geographical regions, particularly in developing countries like Indonesia. Co-infection of leprosy and tuberculosis is not rare and has been documented since

ancient times. Globally, the prevalence of co-infection is 0.02 cases per 100,000 population, ranging from 2.5 to 13.5 cases in endemic areas [4]. Indonesia achieved national elimination status for leprosy in 2020, with a prevalence of new cases reaching 0.7 per 10,000 people [5]. However, new cases with disabilities are still found and are a problem that must be addressed. Meanwhile, tuberculosis is still a health problem in Indonesia, with a prevalence in adults reaching 753 cases per 100,000 population [6].

Several factors influence co-infection in leprosy and tuberculosis. As neglected tropical diseases, leprosy and tuberculosis share common risk factors. Both often occur in individuals with low socioeconomic status, poor nutrition, and limited access to healthcare facilities, disproportionately affecting vulnerable populations [2,7,8]. One theory of co-infection suggests that the deficiency of cellular immunity in leprosy may increase susceptibility to tuberculosis. The intricate interplay between risk factors and the host immune

system can impact clinical symptoms, disease progression, and treatment outcomes in patients, including reactions and disabilities [9,10].

We report a case of co-infection with tuberculosis and leprosy, characterized by undetected skin complaints and grade II disability. Our case highlights the diagnostic challenges, therapeutic interactions, and immune system complexities associated with these conditions.

Case Presentation

A 51-year-old male patient complained of coughing, weight loss of more than 5 kg, and decreased appetite for 3 months. A smear test was carried out, and the result was positive. Chest X-ray shows old bilateral pulmonary tuberculosis, still seen as an active process. The patient was diagnosed with pulmonary tuberculosis and given a fixed-dose combination of anti-tuberculosis drugs.

The patient had experienced hypoesthetic red spots on various parts of his body ten months prior to the onset of cough and weight loss. These lesions initially appeared on the face and later spread to the hands, feet, and back. The patient did not seek medical evaluation

for these skin lesions due to the absence of pain. Six months after the initial skin lesions appeared, the patient began experiencing pain in both legs and stiffness in the fingers of both hands, which impaired his ability to walk and use his hands.

The patient worked as a bus driver and had a 10-year smoking history. He had received the BCG vaccine, as evidenced by scars on his arms. He denied any prior history of tuberculosis or leprosy. The patient lived with his 70-year-old mother and two children in a densely populated area. His living conditions were suboptimal, with a small, damp, and poorly ventilated house lacking adequate sunlight.

A week after the patient received the anti-tuberculosis drug, the prior skin became more inflamed and wider. The skin lesion appeared 1st 10 months before the symptoms of tuberculosis appeared. However, the patient did not experience any complaints when the skin lesions first appeared and, therefore, did not seek medical treatment. Anamnesis and physical examination of the face showed madarosis and saddle-nose, visual disturbances, and watery eyes in the left eye. On the patient's face, back, upper and lower extremities, there are erythematous and

Figure 1. Clinical Manifestation (a) X-ray of the thorax shows old bilateral pulmonary TB, still appears to be an active process. (b, c, d) madarosis, enlargement of the left auricular nerve, necrotic ulcerative lesions with punch-out lesion. (e,f,i) erythema/ hyperpigmentation plaques with firm round and oval borders throughout the body. (f, g) claw-hand digiti 4 and 5 palmar sinistra, atrophy of tenar and hypotenar muscles, xerosis palmar dextra et sinistra. (h) xerosis and ulcers on dorsum pedis dextra et sinistra.



hyperpigmented plaques with well-defined round and oval borders. In some lesions, there is an ulcerative wound with a punch-out lesion appearance (Figure 1). Sensory examination of the lesion revealed hypesthesia. There was enlargement of the left magnus auricular nerve with a soft consistency, accompanied by tenderness. Claw-hand deformity was observed in digits 4 and 5 of the left hand, indicative of ulnar nerve weakness. Atrophy of the thenar and hypothenar muscles was present in both hands. The patient also walked with a limp due to leg pain, particularly when ambulating. Slit-skin smears from skin lesions and earlobes stained with Ziehl-Neelsen were negative for acid-fast bacilli (AFB).

The patient was diagnosed with multibacillary type leprosy with type 1 reaction as well as degree 2 disability and pulmonary tuberculosis. Patients were given a fixed dose combination of anti-tuberculosis drugs consisting of isoniazid, rifampicin, pyrazinamide, and ethambutol (intensive phase for 2 months) followed by isoniazid and rifampicin (continued phase for 4 months) and given multidrug therapy for multibacillary leprosy, consisting of: dapsone 100 mg/day and clofazimine 50 mg/day and 300 mg/once a month and prednisone added for the reaction therapy. Prednisone was started at a dose of 40 mg and gradually tapered according to the guidelines to the lowest dose of 5 mg by week 12. In leprosy with tuberculosis co-infection, the dose of rifampicin follows anti-tuberculosis drugs. Anti-tuberculosis drugs would be given for 6 months, and multidrug drugs for leprosy will be given for 12 months. At the follow-up 6 months later, the clinical condition of the patient's both leprosy skin condition and the tuberculosis manifestation improved after receiving therapy.

Discussion

Leprosy and tuberculosis are chronic infectious diseases caused by *Mycobacterium Leprae* and *Mycobacterium lepromatosis*. *M. leprae* attacks the skin, peripheral nervous system, and upper respiratory tract. The main problem in leprosy today is related to disability and new cases that persist even though Multi Drug Therapy (MDT) has been found. *Mycobacterium tuberculosis* can cause pulmonary and extrapulmonary symptoms, is still a leading cause of death in the world, and is a global and national health problem [1,2].

The diagnosis of pulmonary tuberculosis in patients is based on clinical symptoms of prolonged cough, weight loss, and appetite loss for more than 1 month, positive AFB sputum examination, and chest X-ray showing active tuberculosis. The diagnosis of leprosy is

based on hypoesthetic erythematous skin patches and enlargement of the peripheral nerves in the nervus auricular Magnus sinistra with muscle weakness and clawed hand consistent with grade II disability. In our patient, the leprosy skin lesion appeared 10 months prior to the clinical manifestation of tuberculosis. At the time of diagnosis, the patient showed a degree 2 disability. Skin lesions were evaluated and examined after an inflamed skin rash appeared after tuberculosis treatment.

The pathogenesis of leprosy reactions in tuberculosis co-infection in our case was likely related to type 1 reactions. Type 1 reactions are acute immune responses triggered by the recovery of the host's immune system, typically in response to anti-tuberculosis drugs consisting of Rifampicin, Isoniazid, Pyrazinamide, and Ethambutol. *Rifampicin* is a bactericidal antibiotic used in the treatment of both leprosy and tuberculosis, with a daily dose in tuberculosis and a monthly dose in leprosy. Treatment with rifampicin causes bacterial death and cell damage that can trigger an immune response of inflammatory syndrome reconstruction. In our case, the skin symptoms of leprosy had occurred 10 months before tuberculosis treatment, but he did not feel any complaints, so he did not undergo an examination at a health facility. Leprosy reaction is very common in co-infection case [7,11]. The administration of OAT tuberculosis causes bacterial destruction, which can modulate the host's immune system and induce leprosy reactions. In our case, we observed a type 1 reaction with a pre-existing lesion that became increasingly red and inflamed. In co-infection cases, administering anti-tuberculosis and anti-leprosy drugs simultaneously will improve both conditions above, with a dose of rifampicin according to the tuberculosis program [12]. The patient was given a combination of OAT tuberculosis and MDT leprosy. The tuberculosis therapy given is Anti-tuberculosis treatment category 1, which consists isoniazid, rifampicin, ethambutol, and pyrazinamide. At the same time, Multibacillary MDT consists of a combination of rifampicin, clofazimine, and dapsone for 12 months.

The pathogenesis of leprosy and tuberculosis co-infection is currently poorly understood. Several hypotheses suggested that cross-immunity occurs in tuberculosis, protecting individuals with leprosy and vice versa, as seen in the protection from leprosy offered by BCG vaccination. BCG vaccination alters the innate immune system through a mechanism referred to as trained immunity [5–7], which leads to protection against TB and also against heterologous

pathogens. BCG vaccination reduces the risk and severity of tuberculosis and leprosy infections but does not provide 100% protection. Studies suggested that BCG vaccination reduces the risk of leprosy by 26%-56% and tuberculosis by 50%, but the protective effect decreases over time. Nevertheless, BCG vaccination still plays an important role in controlling tuberculosis and leprosy [13–15]. The second hypothesis is related to a low cellular immune response, which causes an increased risk of tuberculosis infection, which causes faster disease progression and higher mortality rates [1,16].

In this case, the delayed diagnosis of leprosy resulted in grade 2 disability at the time of diagnosis. However, tuberculosis was diagnosed earlier and served as the entry point for diagnosing *M. leprae* infection. Clinical symptoms of tuberculosis, such as persistent coughing, weight loss, and decreased appetite, are more overt and disruptive to daily activities, prompting earlier medical attention [17]. Leprosy, on the other hand, is often overlooked due to its asymptomatic and painless nature, leading to diagnostic delays. Economic factors also play a role in the decision to delay seeking medical care. These factors collectively contribute to the complexity of delayed diagnosis in leprosy [18]. In addition, the tuberculosis program is still a national health program, so active case-finding programs and screening of populations at risk for tuberculosis are routinely carried out in Indonesia, which leads to faster diagnosis in patients even though leprosy symptoms appear earlier [6].

There was a complex relationship between socioeconomic factors, environment, nutritional status, access to health services, and the intricate host immune system in co-infection with *Mycobacterium leprae* and *M. tuberculosis*, which affected the occurrence of delays in our case. The host immune responded to TB treatment likely triggered the recognition of leprosy through an immune modulation mechanism, manifesting as a type 1 reaction. This complexity also became a challenge in the process of diagnosis and therapy, especially in developing countries.

When managing co-infections like tuberculosis and leprosy, it is crucial to consider potential drug interactions. Rifampicin, a key component of both anti-tuberculosis and multidrug leprosy therapy, is a potent inducer of cytochrome P450 enzymes. This can increase the metabolism of corticosteroids, such as prednisone, potentially reducing their efficacy [19]. To mitigate this interaction, the prednisone dose was carefully adjusted and monitored. Regular follow-up

assessments were conducted to monitor for any adverse effects or signs of subtherapeutic corticosteroid levels. Additionally, close monitoring of liver function tests was implemented due to the hepatotoxic potential of rifampicin and other anti-tuberculosis drugs. The patient was also monitored for signs of tuberculosis or leprosy treatment failure, with sputum smear conversion and skin lesion improvement serving as key indicators of therapeutic response.

The diagnosis of leprosy in our case was based on clinical and neurological findings despite negative slit-skin smear results. The patient presented with hypoesthetic erythematous skin patches, enlargement of the left auricular nerve, muscle weakness, and claw-hand deformity consistent with grade II disability. These clinical features strongly supported a diagnosis of multibacillary leprosy. Additionally, the development of a type 1 reaction, characterized by the inflammation and widening of pre-existing skin lesions following the initiation of anti-tuberculosis treatment, further corroborated the leprosy diagnosis. It is important to note that negative slit-skin smears do not necessarily exclude leprosy, particularly in cases of multibacillary leprosy where bacillary loads may vary and smear negativity can occur despite significant disease manifestation.

The uniqueness of this case lies in the co-infection of leprosy and tuberculosis, which presents with its own set of challenges. Despite the early appearance of skin lesions indicative of leprosy, the diagnosis was delayed until after the initiation of anti-tuberculosis treatment. This delay resulted in the progression of leprosy to grade 2 disability. Additionally, the administration of anti-tuberculosis drugs may have modulated the host's immune system, leading to a type 1 reaction that brought attention to the previously overlooked leprosy symptoms. The interplay between these two infections and their impact on the host's immune response highlights the importance of considering co-infections in clinical practice, particularly in endemic areas.

Our case report underscored the challenges of delayed diagnosis and treatment in leprosy patients co-infected with tuberculosis, accompanied by disability. A comprehensive approach, including community education on leprosy symptoms and strengthening national health programs, particularly those focused on leprosy, is essential to prevent disability and achieve optimal treatment outcomes for both diseases.

Conclusions

Co-infection of leprosy with tuberculosis shows overlapping complexity, especially in endemic

countries of leprosy and tuberculosis both in risk factors, immune system, and also interaction in treatment. Comprehensive management of both tuberculosis and leprosy infection is an important factor in reducing transmission of both diseases in the community and preventing late diagnosis, severe disease progression, and disability

Recommendation

Re-introducing and reinforcing the signs and symptoms of leprosy within communities is essential to enable early identification of symptoms and prompt healthcare seeking, thereby reducing the incidence of disabilities.

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Corresponding author

Prof. Dr. dr. Cita Rosita Sigit Prakoeswa, Sp.DVE (K), FINSADV, FAADV

Department of Dermatology Venereology and Aesthetic,
Faculty of Medicine, Universitas Airlangga, Surabaya,
Indonesia. Kampus A UNAIR – Jl. Mayjen Prof. Dr
Moestopo 47,
Surabaya, Indonesia 60131
Tel: +62-31-5020251
Fax: +62-31-5915551
Email: cita-rosita@fk.unair.ac.id

Conflict of interest

No conflict of interest is declared.

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