CASE REPORT

Tuberculosis and Leprosy: A Rare Case of Dual Mycobacterial Infection

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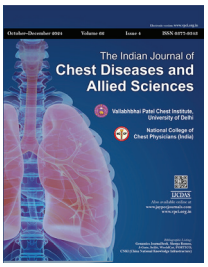

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Abstract

Leprosy and tuberculosis (TB) share geographic endemicity. The degree of cross-immunity of one against the other makes coinfection an uncommon clinical encounter. Here, we report a 35-year-old male patient who presented with a history of fever and cough for a month. He had ulnar claw deformity and multiple hypopigmented lesions since childhood. Chest radiograph showed left middle zone airway opacification and HR-CT revealed left lingular consolidation. Cartridge-based Nucleic Acid Amplification Test (CBNAAT) confirmed *Mycobacterium tuberculosis* and ulnar nerve biopsy confirmed borderline leprosy. The patient had no predisposing factors for TB other than the underlying leprosy. The dual infection was approached with WHO-recommended antitubercular treatment along with dapsone, clofazimine, and prednisolone for leprosy. Prednisolone was gradually tapered and discontinued, while the other medications were maintained. The patient's overall health showed improvement on follow-up. The possibility of concomitant leprosy and TB must be considered by the clinicians to obtain an accurate clinical diagnosis, advise a comprehensive management plan, and avoid treatment-related complications.

Keywords: Case report, Coinfection, Leprosy, Tuberculosis.

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Abbreviations used in this Article

CT = Computed tomography; MDT = Multidrug therapy; TB = Tuberculosis.

Introduction

Leprosy is a chronic granulomatous disease primarily affecting the skin and peripheral nervous system, caused by *Mycobacterium leprae*. It was first identified by Armauer Hansen in Norway in 1873.[1] The rare occurrence of both tuberculosis (TB) and leprosy can be attributed to the transmission dynamics of these infections. The faster replication of TB bacilli compared with leprosy bacilli, along with a certain level of cross-immunity in individuals, typically prevents both diseases from occurring together. However, there have been occasional reports of both TB and leprosy coexisting in the same patient.[2] In this report, we present a case of borderline lepromatous leprosy with a type II reaction, occurring alongside pulmonary TB in a single individual.

Case Description

In November 2023, a 35-year-old male presented to a tertiary care hospital in North India with a 1-month history of fever and cough. The fever had a gradual onset, with an evening rise in temperature, and was responsive to acetaminophen. The cough was associated with sputum production. The patient also reported loss of appetite and weight loss. Upon further inquiry, the patient mentioned a childhood history of sensory loss in the last two digits of his right hand. He had experienced frequent injuries in the same hand over the years and had consulted multiple doctors who prescribed oral prednisolone for neuropathy. The patient did not smoke or consume alcohol. Clinical examination revealed a fever of 101°F, a pulse rate of 112 beats per minute, a blood pressure of 128/72 mm Hg, oxygen saturation of 97% on room air, and a respiratory rate of 24 breaths per minute. Coarse crepitations were heard in the left lung field upon auscultation. A comprehensive physical examination uncovered an ulnar claw hand deformity in the right hand (Fig. 1). The patient also had multiple hypopigmented lesions varying from 2 to 4 cm over his trunk and dorsum of his foot, which he reported having since childhood (Fig. 2). Laboratory investigations, including complete blood counts, liver function tests, and renal function tests, returned normal results. The diagnostic workup for diabetes, which included blood sugar monitoring and HbA1c testing, yielded negative results. Additionally, the patient's Vitamin B12 levels were within the normal range. An electrocardiogram demonstrated sinus rhythm. Further evaluation included a plain chest roentgenogram, which revealed airspace opacification in the left middle zone (Fig. 3). High-resolution chest computed tomography (CT) displayed consolidation in the left lingula (Fig. 4). Sputum analysis using the CBNAAT test confirmed the presence of *Mycobacterium tuberculosis*,

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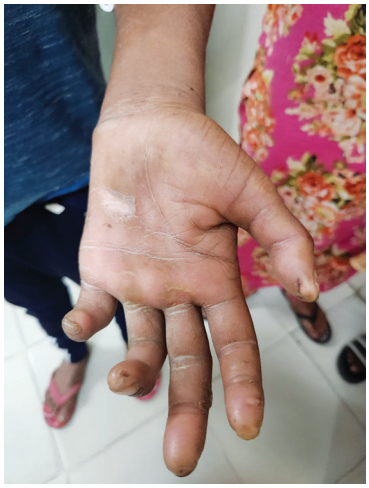

Fig. 1: Ulnar claw hand deformity in the right hand

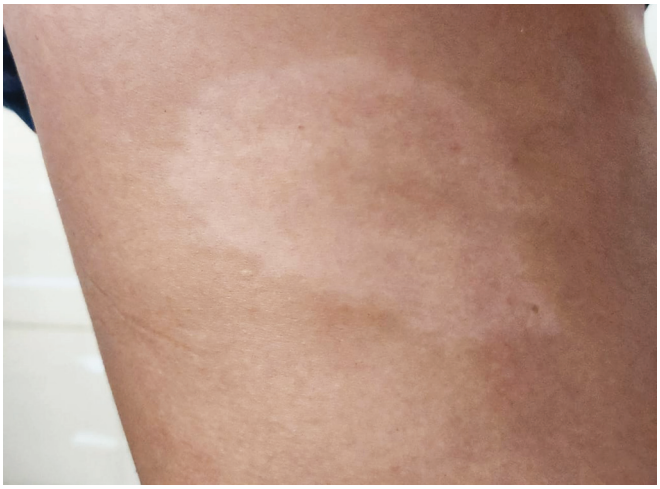

Fig. 2: The patient also had multiple hypopigmented lesions varying from 2 to 4 cm over his trunk and dorsum of foot, which he reported having since childhood

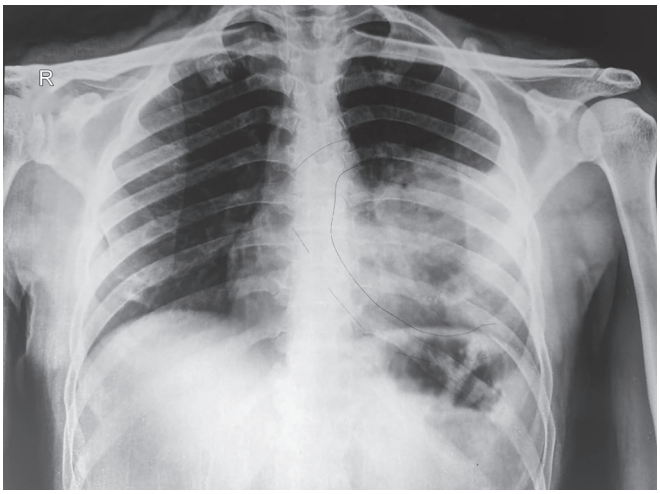

Fig. 3: Plain chest roentgenogram, which revealed airspace opacification in the left middle zone

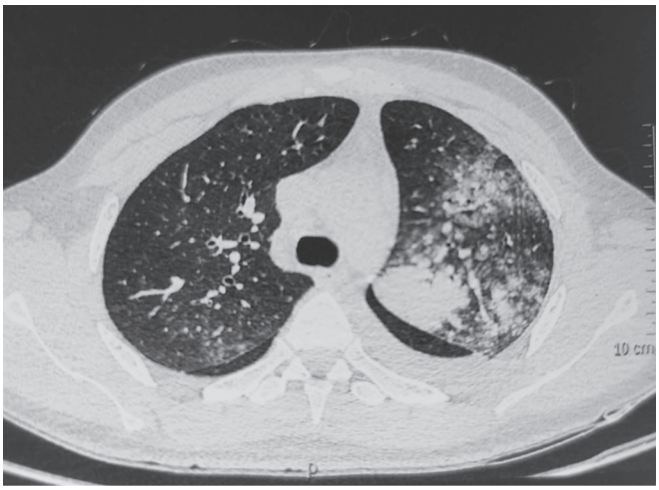

Fig. 4: High-resolution chest CT displayed consolidation in the left lingula

and the strain was sensitive to rifampicin. An ulnar nerve biopsy revealed findings consistent with borderline tuberculoid leprosy progressing to mid-borderline leprosy, providing an explanation for the sensory loss and hand deformity. A conclusive diagnosis was reached, indicating borderline tuberculoid leprosy in conjunction with lower lobe pulmonary TB. The patient was subsequently referred to a DOTS clinic and initiated antitubercular treatment. Additionally, a regimen of anti-leprosy medications, comprising dapsone, clofazimine, and oral prednisolone was prescribed. Prednisolone was gradually reduced and eventually discontinued, while the other medications were maintained. The patient's overall health showed improvement, and regular follow-up appointments were scheduled.

Discussion

Leprosy, caused by *Mycobacterium leprae*, primarily affects the skin and peripheral nervous system. Although its prevalence has decreased from 5 million cases in the mid-1980s to under 1,75,000 cases by the end of 2022, it remains a concern in over 120 countries, especially in Southeast Asia with India bearing a significant burden. Multidrug therapy (MDT) introduced in 1982 has been pivotal in this reduction. India was declared to have eliminated leprosy as a public health problem by the end of 2005.[1]

Tuberculosis, caused by *Mycobacterium tuberculosis*, is a major global health issue and can present in various forms, from miliary TB to lupus vulgaris. Both TB and leprosy are granulomatous infections with aerosol transmission. Leprosy has a long incubation period of 6 months to 40 years, while TB's incubation period is about 4 weeks.

Most people infected with TB remain asymptomatic, but 5–10% develop active disease, presenting symptoms like persistent cough, fever, night sweats, and weight loss.[2] Reactivation disease often follows an immunological trigger, though in this case, the patient had no common triggers, such as malnutrition, HIV infection, chronic hemodialysis, immunosuppressive therapy, diabetes, or silicosis, but displayed sensory loss and hypopigmented skin patches, suggesting the need to investigate leprosy.

Pulmonary TB often affects the upper lobes due to higher oxygen tension and delayed lymphatic drainage, but lower lung field TB can

present challenges in diagnosis, sometimes mimicking pneumonia, bronchiectasis, or bronchogenic carcinoma. This can delay accurate diagnosis, particularly in regions with high TB burden. [3]

In this patient, conditions, such as diabetes, HIV, end-stage renal disease, and corticosteroid use—common factors associated with lower lung field TB—were ruled out through appropriate laboratory tests. To address the patient's peripheral neuropathy, vitamin B12 deficiency and diabetes were excluded. An ulnar nerve biopsy, conducted to investigate the sensory deficits in the fingers, revealed borderline tuberculoid leprosy. This finding suggested that leprosy might be an underlying immunological trigger for pulmonary TB in the absence of other risk factors.

There are no specific guidelines for treating the co-occurrence of leprosy and TB. Verma et al. reported that the management of leprosy with concurrent TB follows the same WHO treatment protocols for each disease separately.[4] Other studies have suggested using rifampicin daily, but this poses a risk of developing rifampicin-resistant TB if used monthly in leprosy patients with concurrent TB. Fortunately, in this case, the patient's leprosy was undiagnosed, so no treatment for it was initiated.

The patient received a simultaneous regimen for both TB and leprosy, including prednisone, which was later tapered and discontinued. A recent review of 156 patients with dual infections highlighted a high mortality rate of 37.2%.[5] However, our patient showed improvement in overall health during regular follow-up appointments.

Leprosy and TB are linked by social, ecological, and biological factors, such as poverty, poor healthcare access, malnutrition, urban living, and weakened immune systems. Studies in endemic areas show that simultaneous occurrence of these two mycobacterial infections ranges from 2.5 to 13.5%, although global detection rates are only 0.02 per 1,00,000 people per year.[6]

Historically, the Chaussinand theory proposed that cross-immunity between leprosy and TB might explain declining leprosy rates in TB-endemic regions and why the BCG vaccine for TB provides partial protection against leprosy.[7] However, the occurrence of simultaneous infections challenges this theory. Subsequent research introduced the "Coinfection" theory, which suggests that dual infections are not rare. Individuals with multibacillary leprosy are particularly vulnerable to TB due to a compromised immune response, and postmortem studies have shown TB as a frequent cause of death in leprosy patients. Overall, these findings indicate that leprosy, especially in its anergic form, can predispose individuals to TB.[8]

Donoghue HD et al.'s work explains that there are more severe disease manifestations with simultaneous infections. The compromised immune system in multibacillary leprosy can reactivate latent TB or trigger new infections, contributing to faster mortality rates and a perceived decline in global leprosy cases. In our patient, the dampening of the immune system due to underlying, undiagnosed, untreated leprosy is the only understandable cause for activation of pulmonary TB. Tuberculosis can occur throughout the spectrum of leprosy. The symptom of leprosy precedes those of TB in most of the cases as seen in the current case report as well.[9]

Shetty S et al. observed that most of the cases of TB were associated with lepromatous leprosy (52.5%) followed by borderline leprosy (20.5%), while the association of tuberculoid form of leprosy with TB was found to be uncommon.[10] Interestingly, our patient exhibited borderline tuberculoid leprosy on nerve biopsy that was uncovered upon evaluation of his pulmonary TB symptoms, which adds to the peculiarity of this case.

In the plot of dual infection, it is also worth reflecting upon the impact of coinfection on clinical outcomes. According to Luciana Cavalcante et al.'s systematic review, dual infections are associated with 37.2% mortality and 5.5% major morbidity.[6] This underscores the importance of screening leprosy patients for TB to prevent serious, unforeseen outcomes. In coinfected individuals, undiagnosed TB poses a significant risk, particularly due to the potential development of rifampicin resistance from monotherapy used in leprosy treatment.

Clinical Implications

The possibility of concomitant leprosy and TB must be known to the clinicians and ruling out one in the presence of the other must be considered for accurate clinical diagnosis and a comprehensive plan. If treatment begins despite a missed diagnosis of coinfection, it can develop rifampicin resistance in mycobacterial strains which can be challenging. Management of coinfection requires a multidisciplinary approach and adequate social support for favorable outcomes.

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