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# A UNIQUE CASE OF TUBERCULAR LYMPHADENITIS WITH ERYTHEMA NODOSUM LEPROMATOSUS IN A YOUNG IMMUNOCOMPETENT MALE

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**ABSTRACT** It is well known that leprosy and tuberculosis (TB) both have similar regional endemicities. If a co-infection does occur, it can be quite challenging to treat. Despite some degree of cross-immunity between the two species, dual mycobacterial infections still happen although the incidence is quite low.(1) Here we report a rare case of tubercular lymphadenitis with Hansen's disease (leprosy).

**KEYWORDS :** Tuberculosis, Leprosy, Erythema Nodosum Lepromatosus, Lymphadenitis

## INTRODUCTION

Both tuberculosis (TB) and leprosy are prevalent throughout India. Given the size and population of the nation, India has an extremely high prevalence of both of these illnesses. [2] TB is recognized sto manifest across the range of leprosy conditions.[3,4] TB can develop in individuals with leprosy who have underlying predisposing factors such as malnutrition, diabetes, or other conditions that weaken the immune system. Additionally, TB may also occur in patients undergoing corticosteroid treatment for leprosy-related complications. [2] On the other hand, other studies propose the existence of cross-immunity, implying that TB may offer protection against leprosy.[4,5] Dual infections are associated with elevated mortality rates of 37% and morbidity rates of 5.5%. [4] The majority of case reports involving co-infection with mycobacteria typically describe patients with both leprosy and pulmonary tuberculosis. While generalized lymphadenopathy in lepromatous leprosy is frequently observed, simultaneous tuberculous infection of the lymph nodes is uncommon. This report presents one such rare case.

## CASE REPORT

A young immunocompetent male presented with complaints of fever, tender nodules over legs and arms over a course of 2 months, gradual in onset and progressively increasing in size. It initially developed in the inguinal region and later also appeared in the axillary and epitrochlear region. 7 days before presentation the patient developed an erythematous rash over the extensor aspect of his legs and arms. There were no associated sensory deficits. The patient had no known comorbidities and no history of contact with a proven case of tuberculosis or leprosy. A dermatology opinion was sought for the erythematous nodules he was started on steroid treatment, specifically prednisolone, for a duration of 2 weeks, with a gradual tapering plan to be implemented upon review. Fine-needle aspiration cytology (FNAC) of lymph nodes indicated tubercular lymphadenitis, leading to the initiation of anti-tubercular therapy (HRZE) based on FNAC findings. A slit skin smear confirmed the diagnosis of Hansen's disease with erythema nodosum leprosum (ENL), type 2 lepra reaction with a 1+ acid-fast bacilli (AFB) count. Consequently, the patient was started on oral dapsone (100mg daily) and oral clofazimine (300mg once a month and 50mg daily). The overall management of the patient involved the WHO-recommended 1-year multidrug therapy (MDT) for multibacillary leprosy, consisting of three drugs: dapsone (at a dosage of 10mg per kilogram of body weight), clofazimine (at a daily dosage of 1mg per kilogram along with a monthly dosage of 6mg per kilogram), and rifampicin. However, the monthly rifampicin dose was omitted since it was already part of the patient's prescribed antitubercular regimen. The patient underwent the standard 6-month short-course chemotherapy for tuberculous lymphadenitis confirmed by fine-needle aspiration cytology (FNAC). The treatment regimen comprised a 2-month intensive phase with daily administration of four drugs: isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by a 4-month continuation phase with three drugs: isoniazid, ethambutol, and rifampicin. This protocol adhered to the guidelines outlined by both the World Health Organization (WHO) and the National Tuberculosis Elimination Programme for India. Additionally, to manage the type 2 lepra reaction, the patient received steroid therapy in the form of prednisolone at a dosage of 1mg per kilogram of body weight for a duration of 2 weeks, with a tapering plan scheduled for review. Symptoms resolved quickly, and the patient was discharged to continue their prescribed therapy. The management challenge in this case was to prevent the development of rifampicin-resistant tuberculosis, the use of steroids to manage ENL.

## DISCUSSION

Given the endemic nature of both leprosy and tuberculosis in India, a provisional diagnosis of coinfection was made, prompting further investigations conducted in accordance with established protocols. Following consultation with the dermatologist, samples including a slit skin smear and a skin biopsy from the active edge of a lesion were sent for histopathological examination. The conclusive diagnosis revealed tubercular lymphadenitis confirmed by fine-needle aspiration cytology (FNAC) alongside slit skin smear confirming Hansen's disease (borderline lepromatous leprosy) with associated erythema nodosum leprosum (ENL).

Peripheral lymphadenopathy constitutes approximately 30% of all cases of extrapulmonary tuberculosis (TB) and remains the predominant cause among various extrapulmonary manifestations. [6] Typically, tuberculous lymphadenopathy primarily affects the cervical lymph nodes, presenting commonly in pediatric populations. [7] Research indicates that the cervical lymph node group is the most frequently involved site, with incidence rates ranging from 74% to 90%, followed by the axillary group in 14–20% of cases, and the inguinal group in 4–8% of cases.[8] Fine-needle aspiration cytology (FNAC) serves as a dependable diagnostic tool for the identification of TB lymphadenitis.[9]The National Tuberculosis Elimination Programme recommends a standard 6-month tuberculosis (TB) treatment regimen as the primary therapy.

Leprosy, also referred to as Hansen's disease, is relatively uncommon in many regions worldwide. However, in India, the incidence rate has been reported at 0.45 per 10,000 population for the year 2021-22.([10] Leprosy presents with various clinical manifestations, categorized by Ridley and Jopling into five types:

1. Polar tuberculoid leprosy: Characterized by a robust cell-mediated immune response against Mycobacterium leprae.
2. Borderline tuberculoid leprosy.
3. Borderline borderline leprosy.
4. Borderline lepromatous leprosy (BL).
5. Polar lepromatous leprosy (LL): Associated with a diminished or absent cell-mediated immune response against Mycobacterium leprae.

The progression from the tuberculoid end to the lepromatous end of the spectrum is marked by an increase in bacterial burden, more frequent skin and nerve involvement, and a gradual decline in M. leprae-specific cell-mediated immunity.[2] For clinical purposes, the World Health Organization (WHO) classifies leprosy differently, dividing it into paucibacillary leprosy (PB) and multibacillary leprosy (MB).[11] This classification is based on the number of skin lesions and the presence of bacilli observed on skin smear examinations. Paucibacillary leprosy is characterized by five or fewer skin lesions and the absence of organisms on skin smears. Conversely, the multibacillary form of leprosy is defined by six or more lesions and/or the visualization of bacilli on skin smears. According to the World Health Organization (WHO), the presence of any of the following cardinal signs is considered diagnostic of leprosy:

1. Hypopigmented or reddish patches accompanied by definite sensory loss.
2. Thickened peripheral nerves.
3. Positive skin smears or biopsy material indicating the presence of acid-fast bacilli. [12]

The immune response to Mycobacterium leprae in an individual can vary, leading to fluctuations in the clinical state, which are termed lepra reactions. These reactions can pose medical emergencies, characterized by inflammatory episodes affecting the skin, nerves, and other organs, potentially resulting in irreversible nerve damage. Although they can occur at any point during the course of leprosy, they are most commonly observed after initiating treatment.

There are two primary types of reactions:

1. Type 1, or reversal reaction: These reactions occur in approximately one-third of patients with borderline forms of the disease and are triggered by a spontaneous increase in cell-mediated immunity to M. leprae. Initiation of multidrug therapy (MDT) is considered a significant risk factor for these reactions.
2. Type 2, or erythema nodosum leprosum (ENL): These reactions may manifest among patients with multibacillary (MB) leprosy, including borderline (BL) and lepromatous (LL) forms. The risk of type 2 reactions increases with higher bacterial loads.[12,13]

In the context of India, the treatment of leprosy adheres to guidelines set forth by both the World Health Organization (WHO) and India's National Leprosy Elimination Programme. Presently, two finite duration multidrug therapy (MDT) regimens are commonly utilized, incorporating dapsone, clofazimine, and rifampicin. [14,15]

The precise relationship between tuberculosis (TB) and leprosy remains unsettled. Both diseases are chronic granulomatous conditions caused by mycobacteria that are acid-fast bacilli (AFB), and they are primarily transmitted through the aerosol route. However, leprosy, caused by Mycobacterium leprae, predominantly affects the skin and peripheral nervous system, while TB, caused by Mycobacterium tuberculosis, can manifest as pulmonary or extrapulmonary forms, affecting various organs.

There are conflicting theories regarding the rare instances of coinfection with leprosy and TB. Some researchers suggest that the two diseases may exhibit antagonistic interactions, offering relative protection against simultaneous infection through cross-immunity.[16-18]

Rifampicin, a key frontline antitubercular medication, is also employed in the treatment of leprosy. It serves as a highly potent bactericidal agent against both Mycobacterium leprae and Mycobacterium tuberculosis. Recognizing tuberculosis in individuals with leprosy is crucial, particularly in developed regions where leprosy treatment protocols include daily rifampicin doses. This approach helps prevent the risk of monotherapy with rifampicin, which could potentially lead to the emergence of rifampicin-resistant tuberculosis. Screening for TB in leprosy patients is advised despite the dearth of evidence of coinfection.[2]

According to the World Health Organization's (WHO) treatment guidelines for erythema nodosum leprosum (ENL), severe cases of ENL should initially be managed with low dosages of prednisolone.[19] However, it remains a subject of debate whether steroid medication for ENL in leprosy affects the prognosis of tuberculosis (TB). More case reports are needed to substantiate the role of steroids in influencing the course of TB.

In conclusion, managing co-infected patients presents unique challenges regarding medication therapy selection, treatment duration, the role of steroids in erythema nodosum leprosum (ENL), and its impact on tuberculosis (TB) and patient response to therapy.

## CONCLUSION

In cases of leprosy infection, it is crucial to emphasize the necessity of ruling out tuberculosis before initiating rifampicin-based therapy to mitigate the risk of developing rifampicin-resistant TB. In conclusion, managing co-infected patients poses unique challenges related to medication therapy selection, treatment duration, the use of steroids in erythema nodosum leprosum (ENL), and its potential impact on tuberculosis (TB) and patient response.

## REFERENCES

1. Sami C, Hassan S, Khan A, et al. (April 06, 2022) A Young Female With Borderline Lepromatous Leprosy and Tuberculous Lymphadenitis: A Rare Coinfection. Cureus 14(4): e23892. doi:10.7759/cureus.23892
2. Shetty S, Umakanth S, Manandhar B, Nepali PB. Coinfection of leprosy and tuberculosis. BMJ Case Rep. 2018 Mar 15;2018:bcr2017222352.
3. 4 Rawson TM, Anjum V, Hodgson J, et al. Leprosy and tuberculosis concomitant infection: a poorly understood, age-old relationship. Lepr Rev 2014;85:288–95
4. Rajagopala S, Devaraj U, D'Souza G, et al. Co-Infection with M. tuberculosis and M. leprae—case report and systematic review. J Mycobac Dis 2012;2:118.
5. Donoghue HD, Marcsik A, Matheson C, et al. Co-infection of Mycobacterium tuberculosis and Mycobacterium leprae in human archaeological samples: a possible explanation for the historical decline of leprosy. Proceedings of the Royal Society B: Biological Sciences 2005;272:389–94
6. Loukeris D, Zormpala A, Chatzikonstantinou K, Androulaki A, Sipsas NV.

Primary unilateral tuberculous inguinal lymphadenitis. Eur J Intern Med 2005;16:531-3.
7. Dayal A, Pai S, Shenoy KV, Kansakar P, Kannan A, Sharma Y,et al. Isolated primary tuberculosis of inguinal lymph nodes: An acute presentation. Internet J Surg 2008;1:4.
8. Seth V,Kabra SK,Jain Y, Semwal OP,Mukhopadhyaya S,JensenRL Tubercular lymphadenitis: Clinical manifestations. Indian J Pediatr 1995;62:565-70.
9. Palanisamy AP, Samuel S, Vadivel S, Kothandapany S. Isolated tuberculous lymphadenitis presenting as bilateral buboes. Indian J Sex Transm Dis 2015;36:80-2.
10. Government of India. (n.d.). Copyright Policy. Press Information Bureau. Retrieved from https://pib.gov.in/Content/102_2_Copyright-Policy.aspx
11. World Health Organization (WHO). WHO: Weekly epidemiological record Relevé épidémiologique hebdomadaire. . Geneva: World Health Organization, 2016:21. 421–8.
12. Britton WJ, Lockwood DNJ. Leprosy. The Lancet 2004;363:1209–19
13. Graham A, Furlong S, Margoles LM, et al. Clinical management of leprosy reactions. Infectious Diseases in Clinical Practice 2010;18:235–8.
14. World Health Organization. WHO recommended MDT regimens [Internet]. 2017 http://www.who.int/lep/mdt/regimens/en/
15. National Leprosy Eradication Programme (NLEE). Training Manual for Medical Officers: Central Leprosy Division, Directorate General of Health Services, Nirman Bhawan. New Delhi, 2013.
16. Tuberculosis CR. and leprosy-antagonistic illnesses. Int J Lepr 1984;16:431–8.
17. Leprosy FJM. and tuberculosis. Arch Dermatol 1957;75:101–6.
18. Lietman T, Porco T, Blower S. Leprosy and tuberculosis: the epidemiological consequences of cross-immunity. Am J Public Health 1997;87:1923–7.