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Coexistence of Multibacillary Hansen's Disease and Pulmonary Tuberculosis in A Patient with Type 2 Diabetes Mellitus: A Diagnostic and Therapeutic Challenge

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Abstract:
Background: The coexistence of Hansen's disease and pulmonary tuberculosis is uncommon despite both diseases being highly endemic in India. Impaired cell-mediated immunity associated with multibacillary Hansen's disease and diabetes mellitus may increase susceptibility to dual mycobacterial infection, posing significant diagnostic and therapeutic challenges.
Case Presentation: A 50-year-old male with poorly controlled type 2 diabetes mellitus diagnosed case of Hansen's disease on multidrug therapy (MDT) with history of Type I (reversal) lepra reaction presented with low-grade intermittent fever, productive cough, anorexia, generalized weakness, and weight loss. Chest radiography revealed bilateral pulmonary infiltrates. Sputum examination by Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) detected *Mycobacterium tuberculosis* without rifampicin resistance, confirming pulmonary tuberculosis. The patient was started on anti-tubercular therapy under the National Tuberculosis Elimination Programme while continuing MDT for Hansen's disease, with subsequent clinical improvement.
Conclusion: This case underscores the importance of maintaining a high index of suspicion for tuberculosis in patients with Hansen's disease, particularly in the presence of diabetes mellitus and lepra reactions. Early diagnosis using molecular techniques and prompt initiation of appropriate therapy are crucial to prevent treatment delays, inappropriate rifampicin exposure, and adverse clinical outcomes.

Keywords: Hansen's disease; Pulmonary tuberculosis; Type 2 diabetes mellitus; Multibacillary leprosy; Lepra reaction; Dual mycobacterial infection; CBNAAT.

Introduction

Leprosy (Hansen's disease) and tuberculosis are chronic granulomatous infections caused by *Mycobacterium leprae* and *Mycobacterium tuberculosis*, respectively. Both diseases remain important public health concerns in India; however, their coexistence in the same individual is relatively uncommon despite their endemicity.[1,2] Historically, it was believed that infection with one mycobacterial species might confer partial protection against the other.[3] However, subsequent studies have suggested that impaired cell-mediated immunity in multibacillary leprosy may increase susceptibility to tuberculosis.[1]

The risk of coinfection may be further enhanced by comorbid conditions such as diabetes mellitus, malnutrition, smoking, and chronic alcohol consumption.[2]

Diabetes mellitus is a well-established risk factor for active tuberculosis because chronic hyperglycaemia adversely affects macrophage function and T-cell-mediated immunity.[4] Furthermore, lepra reactions are associated with alterations in host immune responses and may contribute to the activation of latent tuberculosis infection.[5] Recent advances in molecular diagnostics, particularly Nucleic Acid Amplification Test (NAAT), have improved the ability to confirm tuberculosis in patients with concomitant acid-fast bacilli positivity.[6]

We report a case of multibacillary Hansen's disease associated with microbiologically confirmed pulmonary tuberculosis and Type 1 lepra reaction in a patient with poorly controlled type 2 diabetes mellitus, highlighting the diagnostic and therapeutic challenges posed by dual mycobacterial infection.

Case Presentation

A 50-year-old male from Puri, Odisha, with a weight of 50 kg, body mass index (BMI) of 19.3 kg/m² presented to chest OPD with history of cough with expectoration, intermittent low-grade fever, generalised weakness, loss of appetite, and significant weight loss for one month. Diagnosed case of Hansen's diseases based on multiple anaesthetic hypopigmented skin lesions of varying sizes over both upper limbs and the back, along with a nodular lesion involving the right ear lobule for the preceding four months. Slit-skin smear obtained from the right ear lobule demonstrated acid-fast bacilli positivity (4+), confirming multibacillary Hansen's disease. No history of TB & none of the family members suffered from PTB. There was no clinical evidence of significant peripheral nerve thickening on examination. There was no ophthalmic involvement. Clinical examination revealed no visible deformities or disabilities, such as claw hand, foot drop, trophic ulcers, or absorption of digits. Furthermore, protective sensation in the hands and feet was preserved. He was on Multidrug therapy (MDT) as per national guidelines (MDT MB Adult) [Rifampicin 600mg once a month, Clofazimine 300 mg once a month followed by 50mg daily, Dapson 100 mg daily. History of Type 1 lepra reaction fifteen days after initiation of MDT, for which oral methylprednisolone(32mg) therapy was initiated but patient took for 2 days, then stopped.

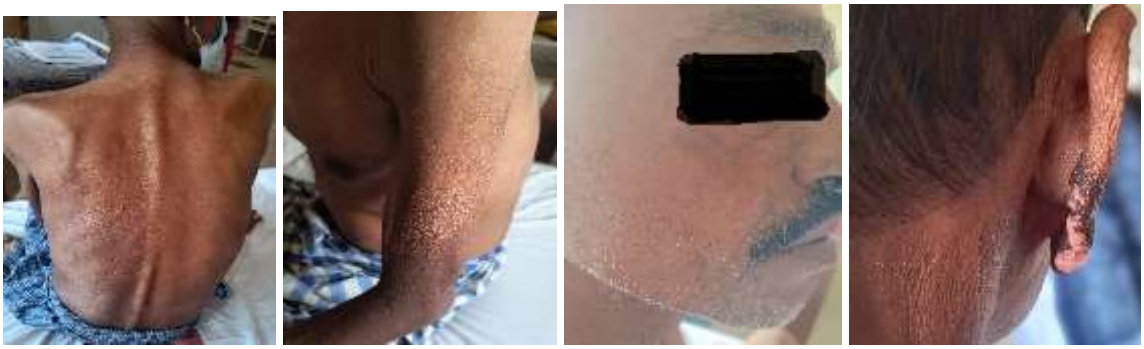


Fig. 1 & 2: Hypopigmented anaesthetic patches over back and left arm

Fig.3: Type 1 lepra reaction

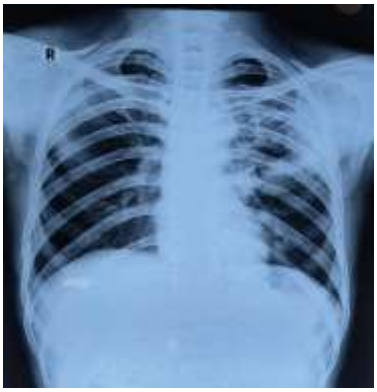
Fig.4: Ear lobule nodular lesion suggestive of multibacillary involvement

The patient was a known case of type 2 diabetes mellitus for the past three years and was receiving metformin 500 mg twice daily and glimepiride 1 mg once daily. He was also a chronic smoker and habitual alcohol consumer.

Investigations

Haematological and Biochemical Investigations: Haemoglobin: 11.4 g/dL (normocytic normochromic anaemia), Random blood sugar: 224 mg/dL, Fasting blood sugar: 170 mg/dL, Postprandial blood sugar: 220 mg/dL, HbA1c: 8.3%, Liver and renal function tests: Within normal limits, Serum albumin: 2.4 g/dL, HIV, HBsAg, and anti-HCV: Negative.

Radiology



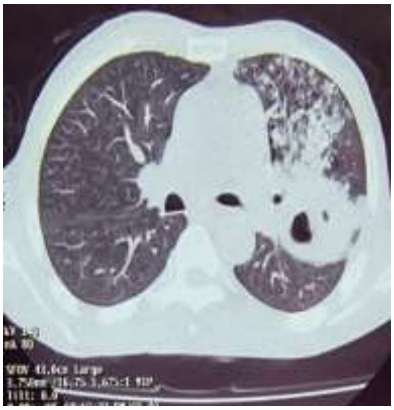


Fig.5 Chest X-ray showing bilateral infiltrative shadows on upper zone.

Fig.6 CT Thorax showing cavitary lesion with surrounding consolidation and patchy nodular opacity in the left upper lobe.

Microbiological Investigations

- Slit-skin smear: AFB positive (4+), Sputum CBNAAT: *Mycobacterium tuberculosis* detected, Rifampicin resistance not detected

Final Diagnosis

Microbiologically confirm Pulmonary tuberculosis with multibacillary Hansen's disease with Type 1 lepra reaction with uncontrol Type 2 diabetes mellitus.

Treatment

The patient was initiated on weight-band-based anti-tubercular treatment (ATT) fixed-dose combination of Rifampicin, Isoniazid, Ethambutol & Pyrazinamide;(4FDC 4 tab) under the National Tuberculosis Elimination Programme (NTEP), while multidrug therapy for Hansen's disease was continued concurrently. Glycaemic control was optimized with metformin, glimepiride & Insulin. Monthly rifampicin skipped due part of a regimen in TB. During follow-up after 2 months, the patient showed significant clinical improvement with reduction in respiratory symptoms, improvement in appetite, weight gain, and stabilization of skin lesions.

Discussion

Hansen's disease was diagnosed based on WHO guidelines, which include the presence of at least one of the three cardinal signs: (i) hypopigmented or erythematous patch or plaque with definite loss of sensation (ii) thickened peripheral nerve with sensory/motor/autonomic deficit in its distribution, and (iii) presence of acid-fast bacilli in slit skin smear.[7] The diagnosis was supported by the presence of two of the three

WHO cardinal signs of Hansen's disease. Furthermore, the detection of acid-fast bacilli (AFB) on slit-skin smear fulfilled the WHO criterion for classification as multibacillary (MB) leprosy.[7] WHO disability grade 0 as no loss of protective sensation in the hands or feet, no visible deformity, and no eye involvement. Since our case had a positive slit-skin smear (AFB 4+), nodular ear involvement, and multiple lesions, it likely falls toward the lepromatous end of the spectrum (Border line Lepromatous)based on Ridley–Jopling classification.[8]

Coinfection of tuberculosis and Hansen's disease, though rare, has been documented in endemic regions like India.[1,2 & 3] They occur due to impaired cell-mediated immune response, and a common genetic predisposition leading to impaired immunity has been postulated as the reason behind dual infections.[9]

Immunological vulnerability

Diabetes mellitus impairs cell-mediated immune responses and macrophage function, thereby increasing susceptibility to active tuberculosis. [4]

Lepra reactions reflect alterations in host immune responses and may facilitate the activation of latent infections, including tuberculosis.[10] The lepromatous pole of the spectrum is more vulnerable to TB due to decreased cell-mediated immunity.

Diagnostic challenge

The presence of acid-fast bacilli may create diagnostic uncertainty regarding the causative mycobacterial species; however, CBNAAT provides rapid and reliable confirmation of pulmonary tuberculosis. [6]

Therapeutic concern

As rifampicin is a key component of treatment regimens for both tuberculosis and Hansen's disease, failure to recognize coexisting tuberculosis may result in inadvertent rifampicin monotherapy, potentially leading to the development of drug resistance. [2]

Clinical Significance

This case represents a rare coexistence of multibacillary Hansen's disease, pulmonary tuberculosis, and type 2 diabetes mellitus. The emergence of tuberculosis shortly after initiation of MDT and during a Type 1 lepra reaction raises the possibility of immune-mediated unmasking of latent tuberculosis infection. Patient was triple association: Hansen's disease + tuberculosis + diabetes**,** TB developed after MDT initiation and lepra reaction, suggesting immune-mediated unmasking.

Public health relevance: The coexistence of chronic infectious diseases with diabetes mellitus, compounded by behavioural risk factors such as smoking and alcohol consumption, presents significant diagnostic, therapeutic, and public health challenges in endemic settings.

Conclusion

This case highlights the importance of active tuberculosis screening in patients with Hansen's disease, particularly in the presence of diabetes mellitus, malnutrition, and lepra reactions. Early diagnosis and prompt initiation of appropriate therapy are essential to prevent treatment failure, disease progression, and the emergence of drug resistance. Such cases underscore the need for integrated management strategies for chronic infectious diseases in endemic regions.

Learning Points

- Concomitant tuberculosis and Hansen's disease, although uncommon, should be considered in endemic regions.
- Diabetes mellitus significantly increases susceptibility to active tuberculosis.

- Lepra reactions may unmask latent tuberculosis through alterations in host immune responses.
- CBNAAT is valuable in confirming pulmonary tuberculosis in patients with concomitant acid-fast bacilli positivity.
- Tuberculosis should be actively excluded before or during MDT to prevent inadvertent rifampicin monotherapy and the emergence of drug resistance.

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