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CASE REPORT OPEN ACCESS

Unusual Presentation of Disseminated Tuberculosis in an Incompetent Adult A Case Report and Review of Immunological Implications

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Abstract

Disseminated tuberculosis (TB) is primarily associated with immunocompromised states, yet it may occasionally present in immunocompetent individuals, posing diagnostic challenges. We report the case of a 28-year-old immunocompetent male with disseminated TB involving the lungs, liver, and spleen. The case underscores the need for a high index of suspicion, even in the absence of classic risk factors. We also discuss the immunological mechanisms that may influence atypical TB dissemination.

Introduction

Tuberculosis remains a significant global health challenge, with approximately 10.6 million cases and 1.3 million deaths reported in 2022 [1]. Disseminated TB, involving two or more non-contiguous organ systems, is uncommon in immunocompetent individuals and is often misdiagnosed as malignancy or autoimmune disease [2]. Understanding the immune responses underlying TB dissemination is crucial for early diagnosis and appropriate management.Tuberculosis (TB), caused by Mycobacterium tuberculosis, remains one of the leading infectious causes of morbidity and mortality worldwide. While pulmonary TB is the most common form, extrapulmonary and disseminated TB—characterized by involvement of two or more non- contiguous sites—pose significant diagnostic and therapeutic challenges, particularly when occurring in immunocompetent individuals. Disseminated TB is traditionally associated with immunosuppressive conditions such as HIV infection, malignancy, or immunosuppressive therapy, where the host's immune defenses are compromised [1,2]. However, rare presentations in individuals without apparent immunodeficiency have been increasingly documented, suggesting that subtle or undetected immune alterations may predispose certain patients to severe or atypical forms of TB. These cases often mimic malignancy, systemic autoimmune diseases, or pyogenic infections, resulting in delays in diagnosis and initiation of appropriate therapy [3,4]. The host immune response, particularly the cell-mediated arm, plays a pivotal role in containing TB infection. Key immunological pathways—including the Th1-type response, interferon-gamma (IFN-γ) signaling, and macrophage activation—are critical for granuloma formation and disease control. Impairment in these pathways, even in the absence of overt immunodeficiency, may underlie cases of disseminated TB in seemingly healthy individuals [5]. In this report, we present a rare case of disseminated TB in an immunocompetent young adult. We discuss the clinical presentation, diagnostic workup, and treatment course, along with a focused review on the immunological mechanisms that may contribute to such atypical disease dissemination.

Case Presentation

A 28-year-old previously healthy male presented with a 6-week history of intermittent fever, weight loss (8 kg), and night sweats. He denied any history of HIV, diabetes, or prior TB infection. Physical examination revealed hepatosplenomegaly and mild pallor. Chest auscultation showed bilateral crepitations.

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Investigations:

CBC: Normocytic normochromic anemia

ESR: 85 mm/hr

Chest X-ray: Miliary pattern

Abdominal ultrasound: Multiple hypoechoic lesions in liver and spleen

CT thorax/abdomen: Diffuse nodular infiltrates and splenic microabscesses

HIV ELISA: Negative

Tuberculin Skin Test (TST): 15 mm induration

GeneXpert MTB/RIF: Positive in sputum, rifampicin sensitive

Liver biopsy confirmed caseating granulomas, consistent with TB. The patient was diagnosed with disseminated tuberculosis involving lungs, liver, and spleen.

Immunological Considerations

Although typically observed in immunosuppressed individuals, disseminated TB in immunocompetent hosts suggests nuanced host-pathogen interactions. Possible factors include:

- Defective Th1 response and interferon-γ production, leading to impaired macrophage activation [3]. Dysfunctional pattern recognition receptors (PRRs) such as TLR-2 and TLR-4, affecting initial immune signaling [4]. Genetic polymorphisms in IL-12/IFN-γ axis that reduce resistance to mycobacterial infections [5] [Figure 1].

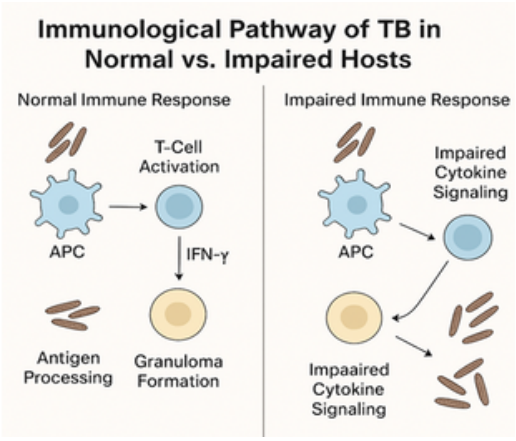

Figure 1: Immunological Pathway of TB in Normal vs. Impaired Hosts

Treatment and Outcome

The patient was started on standard anti-tubercular therapy (isoniazid, rifampicin, pyrazinamide, ethambutol). Fever subsided within two weeks, and follow-up imaging at 3 months showed regression of pulmonary and abdominal lesions. He completed 9 months of therapy with complete clinical resolution.

Discussion

This case emphasizes that disseminated TB can occur in the absence of known immunodeficiency. Delayed diagnosis may arise due to atypical presentations and lack of clinical suspicion [6]. Immunological defects in innate pathways may go undetected in routine testing but significantly contribute to dissemination [7].Timely recognition and histopathological confirmation are vital. Management does not differ significantly, but prolonged therapy and monitoring are recommended [8]. Our report adds to a growing body of literature calling for immunogenetic profiling in unusual TB cases [9].

Conclusion

Disseminated TB in immunocompetent individuals remains rare but important. Awareness of its possibility, combined with knowledge of underlying immunological mechanisms, is essential for prompt diagnosis and effective treatment.

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