Case Report

Hepatic tubercular abscess due to paradoxical TB-IRIS during anti-tubercular therapy with concurrent drug-induced liver injury: a case report

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ABSTRACT:

Background: *Anti-tubercular therapy (ATT) is essential for treating tuberculosis but can sometimes cause liver injury and immune-mediated inflammation. The simultaneous occurrence of severe drug-induced liver injury (DILI) and a paradoxical immune reaction like immune reconstitution inflammatory syndrome (IRIS) is very rare, especially in patients without HIV.*

Case Presentation: *A 55-year-old man with sputum AFB and GeneX pert-confirmed pulmonary tuberculosis presented seven weeks after initiation of anti-tubercular therapy (ATT) with high-grade fever, jaundice, and abdominal pain. Laboratory evaluation revealed marked hyperbilirubinemia (total bilirubin 12.2 mg/dL) with moderately elevated liver enzymes. Imaging studies demonstrated a large hepatic abscess accompanied by ascites and pleural effusion. ATT was discontinued due to suspected drug-induced hepatotoxicity. The patient was managed with supportive care and broad-spectrum antibiotics. A subsequent liver biopsy revealed granulomatous hepatitis consistent with tuberculosis. After normalization of liver function tests, ATT was cautiously reintroduced in a stepwise manner, beginning with Ethambutol, followed by gradual addition of other first-line anti-tubercular agents. Oral Prednisolone was also administered as adjunctive therapy.*

Management and Outcome: *Following reintroduction of anti-tubercular therapy, the patient showed marked clinical improvement. The fever subsided, appetite improved, and body weight increased from 36 kg to 39 kg. Liver function tests demonstrated significant recovery, with total bilirubin decreasing to 2.8 mg/dL. Follow-up imaging revealed resolution of the hepatic abscess with residual fibrotic changes.*

Conclusion: *This case illustrates a rare coexistence of anti-tubercular therapy (ATT)–induced liver injury and a paradoxical inflammatory reaction manifesting as a tuberculous hepatic abscess. Early recognition of this dual pathology is essential to avoid misinterpretation as treatment failure or isolated drug-induced hepatotoxicity. Careful monitoring, gradual reintroduction of ATT, and the use of corticosteroids can lead to full recovery and prevent unnecessary interruption of therapy*

Key Words: Tuberculosis, Anti Tubercular Therapy (ATT), Drug-Induced Liver Injury (DILI), Paradoxical Reaction, Tuberculosis-Associated Immune Reconstitution Inflammatory Syndrome (TB-IRIS), Hepatic Tuberculosis, Corticosteroid.

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Introduction:

Paradoxical reactions during anti-tubercular therapy (ATT), also known as tuberculosis-associated immune reconstitution inflammatory syndrome (TB-IRIS), is defined as clinical or radiological worsening of pre-existing tuberculosis lesions or the appearance of new lesions after initiation of effective therapy, despite microbiological response and absence of alternative explanations such as drug resistance or secondary infection.1,2. These reactions arise from an exaggerated immune response to mycobacterial antigens released during effective therapy rather than treatment failure. While TB-IRIS is commonly reported in HIV-positive populations, paradoxical reactions are increasingly recognized in HIV-negative and immunocompetent patients, particularly in extrapulmonary tuberculosis.2,3.

Concurrently, drug-induced liver injury (DILI) remains a significant complication of ATT. Isoniazid, rifampicin, and pyrazinamide are well-established hepatotoxic agents, and DILI may range from mild transaminase elevation to acute liver failure.4,5,6. The simultaneous occurrence of paradoxical reactions and ATT-induced hepatotoxicity poses a diagnostic challenge, as clinical and radiological findings may overlap, potentially leading to inappropriate treatment modification or delay.7,8. We present a rare case of pulmonary tuberculosis complicated by both severe ATT-induced hepatotoxicity and a paradoxical hepatic TB-IRIS reaction. Recognition of this dual pathology is crucial, as timely differentiation influences management decisions, including temporary withdrawal of hepatotoxic agents, corticosteroid therapy, and safe reintroduction of ATT.4,5,7.

Case Presentation:

A 55-year-old man with newly diagnosed pulmonary tuberculosis (sputum smear positive for acid-fast bacilli and Gene Xpert confirmation) was started on standard first-line Anti Tubercular Therapy (isoniazid, rifampicin, pyrazinamide, ethambutol in fixed-dose combination). After 1 month and 24 days of therapy he presented with 7 days of high-grade intermittent fever, progressive jaundice, right upper-quadrant pain, dyspnea, and malaise.

Clinical Examination:

On admission, the patient appeared ill-looking, tachypneic, cachectic, and markedly icteric. His temperature was 103°F, and oxygen saturation (SpO_2) was 92% on room air. His body weight was 36 kg. Abdominal examination revealed fullness of the right upper quadrant, with marked tenderness and guarding over the right hypochondrium. The liver was tender and palpable just below the right costal margin, consistent with hepatomegaly. The right flank appeared full, and the umbilicus was slightly everted. Ascites was present, as evidenced by positive shifting dullness.

Respiratory system examination showed tracheal deviation to the left, reduced chest expansion on the right side, and diminished breath sounds from the right 5th intercostal space downward. Examination of other systems was unremarkable.

Laboratory and Imaging Findings:

Initial laboratory investigations demonstrated a mixed cholestatic–hepatocellular pattern of liver injury. Total serum bilirubin was 12.2 mg/dL. Alanine aminotransferase (ALT) was 198 U/L, aspartate aminotransferase (AST) 71 U/L, alkaline phosphatase (ALP) 196 U/L, and gamma-glutamyl transferase (GGT) 229 U/L. Serum albumin was markedly reduced at 1.83 g/dL. Complete blood count (CBC) and renal function tests were monitored and managed according to standard care. Viral hepatitis and autoimmune screening were performed. HBsAg, anti-HCV, HEV IgM, HAV IgM, and ANA were negative. EBV/CMV serology and extended autoimmune markers (AMA, ASMA) were not performed.

Ultrasonography of the whole abdomen revealed a complex lesion measuring 10.7 × 9.8 cm in the right hepatic lobe, with an internal fluid component measuring 7.6 × 4.9 cm containing internal echoes, consistent with a hepatic abscess. Marked ascites was also noted.

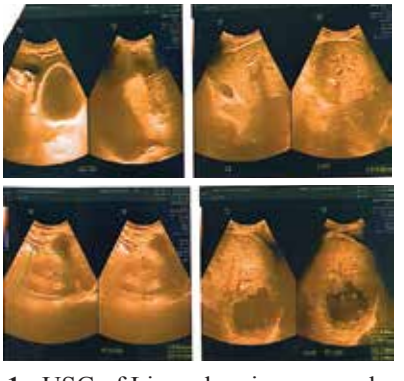

Figure 1 : USG of Liver showing a complex mass lesion in right hepatic lobe

Figure 2. Chest X-ray (posteroanterior view erect posture) demonstrates a large right-sided pleural effusion with homogeneous opacity in the right lower lung zone and obliteration of the right costophrenic angle. The mediastinum appears slightly shifted to the left, with partial compression of the underlying right lung.

Pleural and Ascitic Fluid Analysis:

Pleural fluid analysis revealed a total protein level of 3.31 g/dL (33.1 g/L) and LDH of 158 U/L (serum LDH 379 U/L). Adenosine deaminase (ADA) was 24.1 U/L. GeneXpert and PCR for Mycobacterium tuberculosis (MTB) and non-tuberculous mycobacteria (NTM) were negative. Approximately 3 liters of pleural fluid were initially drained; however, rapid reaccumulation occurred within a few days.

Ascitic fluid was yellow in appearance, with a total leukocyte count of 22,000/mm³ (67% neutrophils) and red blood cells 85/mm³. ADA was 3.68 U/L. Total protein was 1.09 g/dL, and ascitic fluid albumin was 0.86 g/dL. With a corresponding serum albumin of 1.83 g/dL, the serum–ascites albumin gradient (SAAG) was 0.97 g/dL, consistent with low-gradient ascites.

Hepatic Abscess Drainage and Microbiology:

Ultrasound-guided percutaneous pigtail catheter drainage yielded approximately 1,800 mL of purulent material. Gram stain, routine aerobic and anaerobic cultures, testing for Burkholderia pseudomallei, acid-fast bacilli (AFB) staining, and GeneXpert performed on the pus were all negative.

Histopathology:

Ultrasound-guided liver biopsy demonstrated multiple ill-defined epithelioid granulomata with surrounding chronic inflammatory cell infiltrates, consistent with granulomatous hepatitis suggestive of tuberculosis. Caseation necrosis was not prominent, and Ziehl–Neelsen staining was negative for acid-fast bacilli. In the clinical context of confirmed pulmonary tuberculosis and absence of alternative etiologies, hepatic tuberculosis was considered the most likely diagnosis.

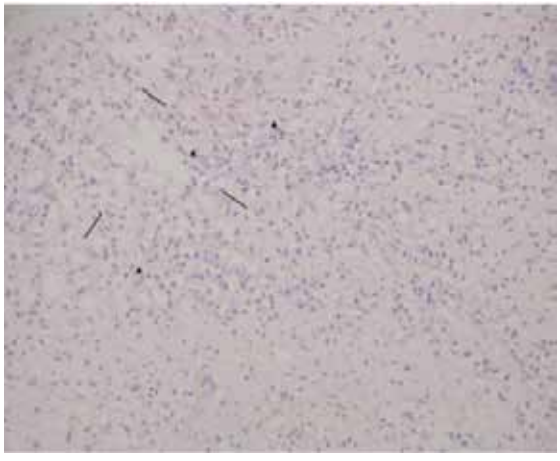

Figure 3: Photomicrograph (H&E stain, ×100) showed multiple ill-defined epithelioid cell granulomata. Black arrows highlight aggregates of epithelioid histiocytes, while arrowheads denote surrounding chronic inflammatory infiltrates.

The patient was non-diabetic and tested negative for HIV infection

Management and Clinical Course:

In view of marked hyperbilirubinemia and a mixed cholestatic–hepatocellular pattern suggestive of significant drug-induced liver injury (DILI), all potentially hepatotoxic anti-tubercular drugs were temporarily withheld. The patient was managed with supportive care, broad-spectrum intravenous antibiotics (empirical coverage for possible pyogenic infection, including meropenem, piperacillin–tazobactam or ceftriaxone, and metronidazole), and image-guided percutaneous drainage of the hepatic abscess and pleural effusion. However, the clinical response to antibiotics alone was limited.

After four weeks, when serum bilirubin decreased to approximately 5 mg/dL and transaminase levels improved toward near-normal values, anti-tubercular therapy (ATT) was cautiously reintroduced in a stepwise manner under close biochemical monitoring.

Ethambutol was initiated as monotherapy at 400 mg daily for three days, followed by 600 mg daily for four days. As no biochemical deterioration occurred, a two-drug fixed-dose combination (isoniazid and rifampicin) was subsequently introduced and carefully observed. After confirming tolerability, the standard four-drug fixed-dose combination (isoniazid, rifampicin, pyrazinamide, and ethambutol) was resumed. Given the suspicion of a paradoxical inflammatory reaction consistent with tuberculosis-associated immune reconstitution inflammatory syndrome (TB-IRIS), adjunctive oral prednisolone was initiated at 40 mg/day for two weeks, followed by gradual tapering over four weeks.

Within two weeks of restarting ATT and corticosteroid therapy, the patient became afebrile, his appetite improved, and body weight increased to 39 kg. Follow-up laboratory evaluation showed a reduction in serum bilirubin to 2.8 mg/dL, with normalization of liver function tests Serial laboratory and imaging assessments demonstrated steady and progressive improvement.

Outcome and follow up:

A follow-up ultrasonography of the whole abdomen performed two months later revealed a marked reduction in the size of the hepatic abscess, with decreased internal echoes, thinning of the abscess wall, and a small residual fibrotic or calcified focus. There was no evidence of fluid re-accumulation, consistent with resolving hepatic abscess.

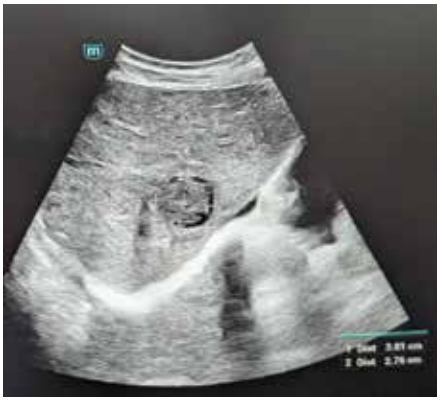

Figure 4: USG of Liver showing resolving hepatic abscess.

Subsequent posteroanterior chest radiography showed complete resolution of the previously documented right-sided pleural effusion.

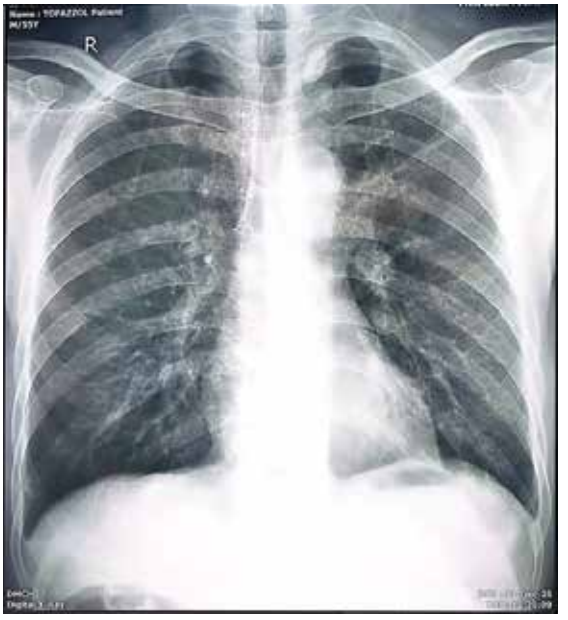

Figure 5: CXR P/ A View showing There is no evidence of active pleural effusion on the right side

Liver function tests normalized on follow-up evaluation. Serum bilirubin decreased to 0.58 mg/dL, and alanine aminotransferase (ALT) declined to 30 U/L, indicating complete biochemical recovery.

Discussion:

The coexistence of anti-tubercular therapy (ATT)-induced liver injury and paradoxical hepatic tuberculosis poses a significant diagnostic and therapeutic challenge. Anti-tubercular drugs—particularly isoniazid, rifampicin, and pyrazinamide—are well-recognized for their hepatotoxic potential, often presenting with elevated serum bilirubin and transaminases that improve upon withdrawal of the offending agents.4-6 In contrast, paradoxical reactions may manifest as new or enlarging inflammatory lesions, complicating clinical interpretation. Distinguishing between drug-induced liver injury (DILI), progressive infection, drug resistance, and immune-mediated paradoxical reactions is essential, as laboratory and imaging findings often overlap.3,7

In this case, the patient developed significant hyperbilirubinemia and elevated transaminases, consistent with ATT-induced hepatotoxicity. Imaging simultaneously revealed a large hepatic abscess with ascites and pleural

effusion. Microbiological tests, including GeneXpert and routine cultures of the abscess and pleural fluid, were negative, effectively excluding active infection or drug-resistant tuberculosis. Liver biopsy showed ill-defined epithelioid granulomas with chronic inflammatory infiltration, confirming granulomatous hepatitis compatible with tuberculosis. The development of a new hepatic abscess during ongoing therapy, in the absence of microbiological progression and with subsequent resolution on continued ATT, supported a diagnosis of paradoxical hepatic involvement. 1-3

This case highlights several key features of dual pathology—ATT-induced hepatotoxicity with paradoxical TB-IRIS:

1.Significant hyperbilirubinemia and transaminase elevation shortly after initiating ATT.4-6

2. Emergence of a large hepatic abscess during ongoing therapy despite absence of microbiological evidence of active infection or resistance.1-3

3. Histopathological confirmation of granulomatous hepatitis.5

4. Negative bacterial, melioidosis, acid-fast bacilli staining, and molecular tests from drained pus.5

5. Complete clinical, biochemical, and radiological resolution following appropriate management.

Management in such scenarios requires careful balancing. Potentially hepatotoxic drugs should be temporarily withheld in suspected DILI, with close monitoring of liver function and gradual reintroduction once biochemical parameters stabilize.4,5 Corticosteroids may be considered in significant paradoxical reactions to reduce excessive immune activation and limit tissue damage.2,3,7 Early recognition and multidisciplinary coordination are crucial to prevent unnecessary interruption of life-saving ATT.

Conclusion:

This case highlights the need for a high index of suspicion for dual pathology, especially in atypical hepatic presentations and HIV-negative patients. In regions with a high burden of tuberculosis, recognizing such scenarios is critical to avoid diagnostic delays, inappropriate interventions, and increased morbidity. Managing tuberculosis becomes particularly challenging when drug-induced liver injury and paradoxical immune reactions occur simultaneously, requiring careful clinical judgment to determine when to withhold, modify, or reintroduce therapy without compromising infection control. Clinicians must remain vigilant, especially when a patient who was initially improving suddenly deteriorates after starting treatment. Early recognition and timely intervention—including judicious corticosteroid use to modulate the immune response and gradual reintroduction of anti-tubercular drugs—can be life-saving. Ultimately, this case underscores the importance of patience, close monitoring, and individualized care. With a thoughtful, multidisciplinary approach, even complex presentations can be guided toward recovery and favorable outcomes.

Ethical Approval:

Ethical approval was not required for this case report according to institutional policy.

Patient Consent:

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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