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Hepatic Tuberculosis in an Immunosuppressed Patient: Diagnostic Challenge Facing Multiple Hepatic Lesions

Edras Samuel Hernández Musús[1], Katherine Nineth Recinos González[2], Julia Milagro Ovalle Quiñonez[3], Luis Fernando Sandoval Garcia[4], Dania Anaberta Campos García[4], Carlos Paredes Amenabar[4], Nadhikrishna Samayoa González[4]

[1]Gastroenterology Residency, Gastroenterology Unit, Hospital General de Enfermedades, Instituto Guatemalteco de Seguridad Social (IGSS), Guatemala, Guatemala

[2]Physician and Surgeon, Universidad de San Carlos De Guatemala

[3]Pathology, Hospital General de Enfermedades, Instituto Guatemalteco de Seguridad Social (IGSS), Guatemala, Guatemala

[4]Gastroenterology Unit, Hospital General de Enfermedades, Instituto Guatemalteco de Seguridad Social (IGSS), Guatemala, Guatemala

***Corresponding author:**

Edras Samuel Hernández Musús, Gastroenterology Residency, Gastroenterology Unit, Hospital General de Enfermedades, Instituto Guatemalteco de Seguridad Social (IGSS), Guatemala, Guatemala

ORCID https://orcid.org/0009-0003-7049-7059

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1. Abstract

Hepatic tuberculosis is a rare and potentially life-threatening manifestation of extrapulmonary tuberculosis. In tuberculosis-endemic countries such as Guatemala, its recognition is particularly important in profoundly immunocompromised patients, as its clinical and radiologic presentation may mimic invasive fungal infection or malignant infiltration, delaying definitive diagnosis. This is the case of a 33-year-old woman with acute leukemia of ambiguous lineage in early relapse who underwent reinduction chemotherapy with a modified CALGB regimen. During profound neutropenia, she developed persistent fever and multiple hypodense hepatic and splenic lesions. Empiric broad-spectrum antibacterial and antifungal therapy was initiated; blood cultures remained negative, whereas serum galactomannan was positive. Because of persistent fever and hepatosplenic lesions despite antimicrobial therapy, liver biopsy was performed. Histopathologic examination demonstrated necrotizing granulomatous inflammation with caseous necrosis and multinucleated giant cells, while Kinyoun staining revealed acid-fast bacilli, confirming disseminated tuberculosis with hepatic involvement. Standard antituberculous therapy was initiated; however, the clinical course was complicated by septic shock, thrombosis, and gastrointestinal bleeding, resulting in a fatal outcome. This case highlights the importance of considering hepatic tuberculosis in persistent febrile neutropenia with multifocal hepatosplenic lesions, particularly in tuberculosis-endemic settings, where early tissue diagnosis may be essential for timely treatment.

2. Introduction

Tuberculosis remains a major cause of morbidity and mortality worldwide. In immunocompromised patients, particularly those with hematologic malignancies undergoing intensive chemotherapy, a relative increase in extrapulmonary forms of the disease is observed [1,2]. Abdominal tuberculosis accounts for approximately 10% to 15% of extrapulmonary cases, with hepatic involvement representing a rare manifestation and a significant diagnostic challenge [3]. In patients with hematologic malignancies and severe immunosuppression, disseminated tuberculosis is associated with high mortality, particularly when diagnosis and initiation of specific treatment are delayed [2,9]. Hepatic involvement usually presents with prolonged fever, hepatomegaly, and focal lesions on imaging studies, findings that may be confused with bacterial abscesses, invasive fungal infections, or neoplastic infiltration [4]. Diagnostic delay is common due to the nonspecific clinical presentation and the low sensitivity of microbiological cultures. Therefore, tissue biopsy remains the gold standard for etiological confirmation (5).

In patients with hematologic malignancies, the coexistence of active tuberculosis and chemotherapy poses a significant therapeutic challenge due to the potential hepatotoxicity of antituberculous drugs and possible drug interactions with chemotherapeutic agents [6]. A case of disseminated hepatic tuberculosis in a patient with acute leukemia undergoing chemotherapy reinduction at the Hospital General de Enfermedades, Instituto Guatemalteco de Seguridad Social (IGSS), Guatemala, is described.

3. Case Description

This is the case of a 33-year-old woman with acute leukemia of ambiguous lineage, initially treated with a modified CALGB regimen, achieving complete bone marrow remission after induction. She subsequently developed an early relapse and underwent reinduction chemotherapy. During the course of treatment, persistent fever developed in the setting of profound neutropenia, and computed tomography demonstrated multiple hepatic and splenic lesions (Figure 1). Given the suspicion of an opportunistic infection, empiric broad-spectrum antibacterial and antifungal therapy was initiated. Serum galactomannan was positive, whereas repeated cultures remained negative. Liver biopsy revealed chronic granulomatous inflammation with caseous necrosis and multinucleated giant cells, together with a positive Kinyoun stain for acid-fast bacilli (see Figure No. 2). The Infectious Diseases Department recommended immediate initiation of standard antituberculous therapy with isoniazid, rifampin, pyrazinamide, and ethambutol.

The hospital course was complicated by episodes of febrile neutropenia, septic distributive shock requiring norepinephrine, upper extremity thrombosis, and anthracycline-associated sinus bradycardia. She required transfusion support, correction of electrolyte imbalances, and close monitoring of liver function. Subsequently, upper gastrointestinal bleeding developed in the setting of worsening cytopenia, followed by a fatal outcome.

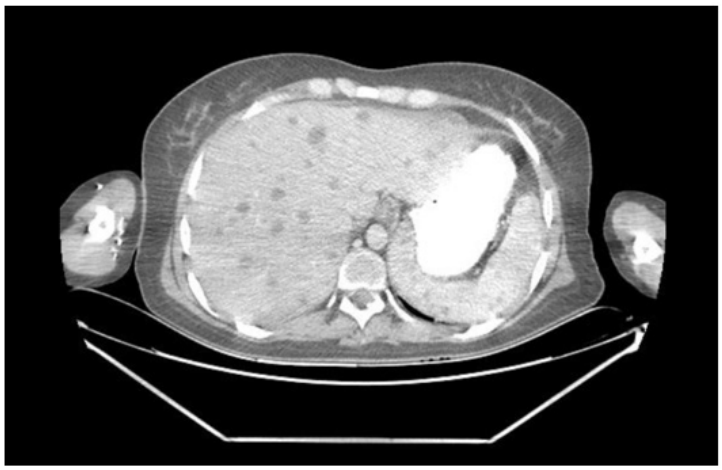

Figure 1: Abdominal computed tomography with multiple hepatosplenic lesions.
Contrast-enhanced abdominal computed tomography showed multiple hypodense lesions disseminated throughout the hepatic parenchyma and spleen, some with a cystic appearance. The findings raised the differential diagnosis of metastatic involvement versus a multifocal infectious process.

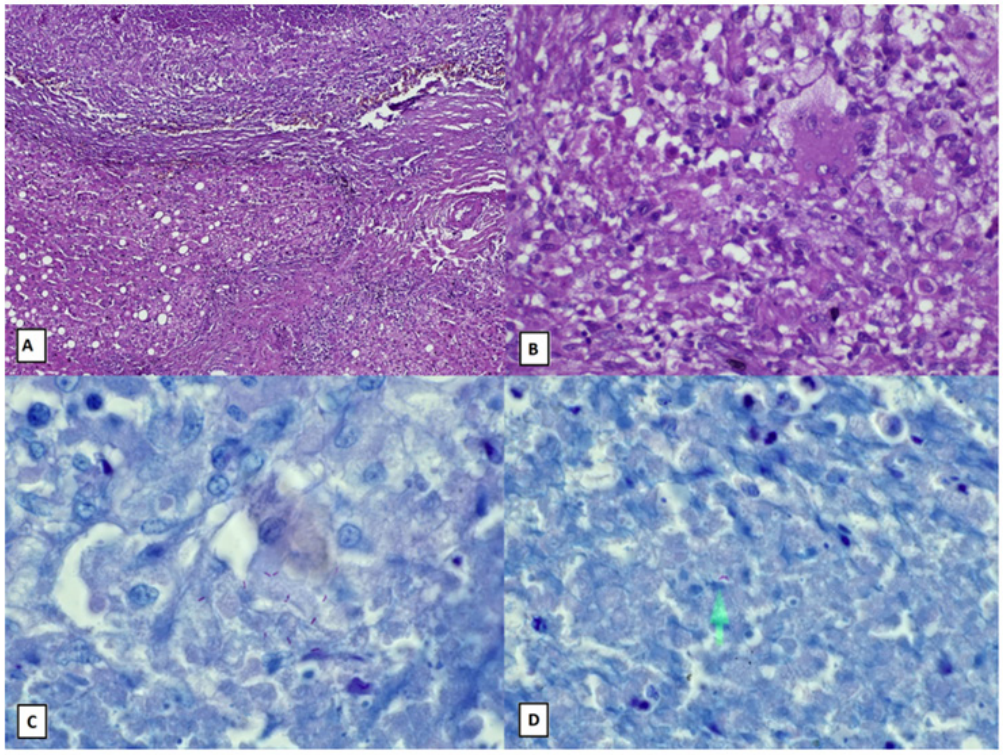

Figure 2: Histopathological findings compatible with hepatic tuberculosis.
(A) Hepatic histological section with hematoxylin-eosin (low magnification) showing extensive areas of granulomatous inflammation with distorted architecture and foci of necrosis.(B) At higher magnification, epithelioid granulomas with central caseous necrosis and multinucleated giant cells are identified, characteristic of tuberculous infection.(C) Special Kinyoun stain demonstrating acid-fast bacilli inside the inflammatory infiltrate (reddish-purple linear structures).(D) Detail of acid-fast bacilli indicated by arrow, confirming active Mycobacterium tuberculosis infection in hepatic parenchyma.

4. Discussion

Extrapulmonary tuberculosis is significantly more common in patients with hematologic cancer, particularly during episodes of chemotherapy-induced neutropenia, where clinical manifestations are often atypical and diagnosis poses a challenge [2,6]. In the present case, altered liver function tests were initially attributed to probable L-asparaginase-induced liver injury, a well-recognized complication during the treatment of acute lymphoblastic leukemia. However, the persistence of fever and the subsequent identification of multiple cystic liver lesions forced the differential diagnosis to expand beyond drug toxicity. The main clinical challenge consisted of differentiating between chemotherapy hepatotoxicity, invasive fungal infection, leukemic infiltration, bacterial abscesses, and hepatic tuberculosis, entities that can present similar clinical and imaging findings [3,4]. This scenario highlights the risk of attributing liver alterations solely to the adverse effects of treatment, which can delay the diagnosis of potentially treatable opportunistic infections. Liver biopsy was decisive in establishing the definitive diagnosis by demonstrating granulomas with caseous necrosis and acid-fast bacilli. It remains the diagnostic standard in these cases, while complementary tests such as urine TB-LAM can provide additional information in patients with severe immunosuppression [5,7]. Simultaneous treatment of active tuberculosis and chemotherapy represents another major challenge due to drug interactions, especially with rifampin, and the increased risk of hepatotoxicity, requiring a multidisciplinary approach and close monitoring of liver function [6,8]. Previous studies have documented high mortality in patients with leukemia and disseminated tuberculosis when there is a delay in recognition and initiation of specific treatment [2,9]. This case emphasizes the importance of maintaining a high index of diagnostic suspicion in patients with leukemia under chemotherapy who present persistent fever and multiple liver lesions, particularly in countries with a high burden of tuberculosis such as Guatemala. Furthermore, it highlights that not all liver test alterations during L-asparaginase treatment correspond to drug hepatotoxicity, and clinical evolution should guide early diagnostic re-evaluation.

5. Conclusion

Hepatic tuberculosis should be included in the differential diagnosis of patients with leukemia undergoing chemotherapy treatment who present persistent liver function test alterations, fever, and multiple hepatic lesions. This case demonstrates that L-asparaginase hepatotoxicity, although common, should not be assumed as the sole explanation for hepatic abnormalities when the clinical course is atypical. Early histological confirmation via liver biopsy and timely initiation of antituberculous treatment, alongside a multidisciplinary approach, are essential to improve the prognosis in these patients.

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