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When tuberculosis hides behind cancer: A challenging case of hepatopancreatic involvement and post-treatment fibrotic sequelae

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Abstract

Hepatopancreatic tuberculosis (HPTB) is a rare and often underdiagnosed form of extrapulmonary tuberculosis that presents with clinical and radiological features mimicking malignancy. We present the case of a 36-year-old male with no history of alcohol or tobacco use, who presented with elevated gamma-glutamyl transferase (GGT) levels during a routine check-up. Initial imaging, including abdominal ultrasound, CT scan, and MRI, revealed hepatic and pancreatic lesions that raised suspicion for metastatic disease. However, a liver biopsy confirmed the presence of granulomatous inflammation with caseous necrosis, consistent with tuberculosis. The patient was treated with a 12-month course of anti-tubercular therapy and anticoagulants for portal vein thrombosis. Follow-up imaging demonstrated partial regression of hepatic lesions and resolution of the pancreatic mass. However, the regression of the lesions led to retraction and fibrosis at the hepatic hilum, resulting in jaundice, which resolved after the patient underwent right hepatectomy. This case underscores the importance of considering HPTB in the differential diagnosis of hepatic and pancreatic masses, particularly in regions with high tuberculosis prevalence.

Keywords: Hepatopancreatic tuberculosis; Cancer; Challenge diagnosis; Fibrotic sequelae; Rare

1. Introduction

Hepatopancreatic tuberculosis (HPTB) is a rare manifestation of extrapulmonary tuberculosis, characterized by involvement of the liver and pancreas, often presenting with features that mimic malignancy both clinically and radiologically. While tuberculosis primarily affects the lungs, hematogenous dissemination can lead to the involvement of distant organs, including the liver and pancreas (1,2). The clinical presentation of HPTB can be nonspecific, with no symptoms or symptoms such as fever, weight loss, and jaundice, which may be attributed to various other conditions, including malignancy (3,4). This case emphasizes the diagnostic challenges associated with HPTB, particularly the need to differentiate it from malignancy, and highlights the importance of considering tuberculosis in the differential diagnosis of unusual hepatic and pancreatic lesions, especially in endemic regions (5,6).

2. Case Presentation

A 36-year-old male with a medical history significant for paracetamol-induced hepatitis in 2015, presented asymptomatically for a routine check-up. Laboratory investigations revealed elevated gamma-glutamyl transferase (GGT) levels at twice the normal limit. The patient had no history of alcohol or tobacco consumption. There was no family history of hepatic or pancreatic neoplasms, and the patient had never undergone any surgery. On physical examination, the patient was in good general condition, with no abnormalities on clinical examination, including no palpable mass, no abdominal tenderness, no dullness, and the lymph node areas were free. Laboratory results revealed mild liver enzyme elevation: GGT was 135 U/L (2x normal), with normal liver function parameters. The

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patient's complete blood count (CBC) was within normal limits, with a white blood cell count of 6710/mm^3, lymphocytes at 2013/mm^3, and a normal platelet count (284,000/mm^3). An initial abdominal ultrasound revealed heterogeneous hepatic steatosis. A contrast-enhanced abdominal CT scan performed revealed a normal-sized liver with heterogeneous density and signs of perfusion abnormalities. Two hepatic nodules were identified in segment 7 and 2, measuring 35x32 mm and 15x11 mm, both displaying peripheral enhancement suggestive of a possible metastatic process. Several hypodense, poorly defined, heterogeneous lesions were also noted in segments 4, 5, and 8, which showed mild peripheral enhancement with contrast. Notable findings also included a mass in the tail of the pancreas, measuring 27x16 mm, infiltrating the splenic pedicle, and several enlarged lymph nodes in the celiac chain, infiltrating the left gastric artery. The primary differential diagnosis was metastatic pancreatic neoplasm.
An abdominal MRI was conducted to better characterize these lesions. The liver showed some dysmorphia with hypertrophy of the left lobe and atrophy of the right lobe. The MRI confirmed multiple hepatic nodes, some of which coalesced in segment 4, creating an infiltrative mass with peripheral annular enhancement and invasion of the right portal branch. These findings were consistent with secondary liver lesions, but an infectious etiology, particularly tuberculosis, could not be excluded. The thoracic CT scan was normal, with no lesions suggestive of pulmonary tuberculosis. A liver biopsy revealed hepatic parenchyma remodeled by a granulomatous reaction composed of epithelioid giant cells, some with central caseous necrosis, suggestive of a granulomatous infection. No evidence of malignancy was found. The patient was initially started on a 9-month course of anti-tubercular therapy, along with anticoagulants due to the presence of portal vein thrombosis. However, given the persistent hepatic lesions observed at the 9-month evaluation, the anti-tubercular treatment was extended to 12 months. Follow-up imaging showed partial regression of the hepatic lesions, with complete resolution of the pancreatic lesion. One year later, the patient developed jaundice, attributed to hepatic fibrosis at the hilum, and underwent right hepatectomy. The surgical specimen confirmed the diagnosis of hepatic tuberculosis. Postoperative recovery was uneventful, and the patient is currently in good health. Four months after surgery, the patient remains clinically stable and asymptomatic.

3. Discussion

Hepatopancreatic tuberculosis (HPTB) is a rare form of extrapulmonary tuberculosis that affects the liver and pancreas, often mimicking malignancy in clinical and radiological presentations. The pathogenesis of HPTB involves the hematogenous spread of *Mycobacterium tuberculosis* from a primary focus, most commonly in the lungs, but it can also be a result of direct spread from nearby structures, such as the gastrointestinal tract (7). This case highlights the diagnostic challenges in distinguishing between HPTB and malignancy, particularly pancreatic neoplasms, due to overlapping clinical and imaging features. Several cases of hepatopancreatic tuberculosis have been described in the literature. Although rare, this form of abdominal tuberculosis can involve both the liver and pancreas, presenting with nonspecific symptoms such as fever, weight loss, jaundice, or right upper quadrant pain—clinical features that often mimic more common hepatobiliary or pancreatic malignancies (8). In our case, the absence of respiratory symptoms and the normal findings on thoracic CT scan excluded pulmonary tuberculosis, which is typically the primary focus for extrapulmonary dissemination. Hepatic involvement was confirmed by liver biopsy, which revealed granulomatous inflammation with central caseous necrosis—hallmark features of tuberculosis (9). Imaging holds a central role in the diagnosis of HPTB, although its findings are often nonspecific. On CT and MRI, hepatic lesions may appear as hypodense or infiltrative masses with peripheral enhancement, often indistinguishable from metastatic tumors (10). In our patient, these radiologic features initially suggested a diagnosis of metastatic cancer. However, the infiltrative appearance observed on MRI, along with histological confirmation, ultimately led to the correct diagnosis of tuberculosis. Although pancreatic involvement is uncommon, it may occur, particularly through direct extension from nearby infected structures (11). Treatment of HPTB typically involves a combination of standard anti-tubercular therapy, including rifampicin, isoniazid, pyrazinamide, and ethambutol, for six to nine months (12). In our case, a 9-month regimen was initially proposed, but persistent hepatic lesions at the 9-month evaluation prompted the extension of treatment to 12 months. The addition of anticoagulation therapy in this patient was warranted due to the presence of portal vein thrombosis, a complication that can occur in cases with severe hepatic involvement (13). Follow-up imaging showed partial regression of the hepatic lesions, which is consistent with the known response of tuberculous lesions to anti-tubercular treatment (14). While fibrotic evolution is not commonly reported in hepatopancreatic tuberculosis (HPTB), cases of fibrosing sequelae have been described in extrapulmonary tuberculosis, particularly in abdominal forms. A case from Japan describes a 33-year-old female patient diagnosed with hepatobiliary tuberculosis. Despite successful anti-tuberculosis treatment and microbiological cure, the patient developed progressive cicatricial stenosis of the hepatic bile ducts 14 months later. Endoscopic management was initially limited by the tight hilar stricture, necessitating percutaneous transhepatic biliary drainage (PTBD). The fibrotic stenosis gradually improved over several years with repeated interventions. However, the case illustrates how chronic inflammation in biliary TB can lead to irreversible fibrotic remodeling and stricture formation, emphasizing the need for prolonged surveillance even after microbiological resolution (15). A 2024 literature review on hepatic-pancreatic tuberculosis from India highlights the fibrotic healing process after anti-tubercular therapy (ATT). This fibrosis, often a consequence of inflammatory scarring, can lead to

strictures in the biliary ducts or pancreas. When fibrosis persists or causes complications, surgical intervention may be necessary. The review emphasizes the importance of monitoring for these fibrotic changes post-treatment to prevent long-term complications (16). In our case, the evolution toward hilar fibrosis following partial regression of lesions—despite a favorable microbiological response—resulted in obstructive jaundice, necessitating a right hepatectomy. This highlights the unpredictable progression of certain HPTB cases and underscores the need for close imaging follow-up and early consideration of structural complications, particularly in endemic regions. This fibrosing progression has been described in other case reports, but its occurrence is rare. In the context of current knowledge, this is the first case of such fibrosing evolution following hepatopancreatic tuberculosis described in Morocco.

4. Conclusion

Hepatopancreatic tuberculosis presents significant diagnostic challenges due to its ability to mimic malignancy, particularly pancreatic cancer, in both clinical and radiological assessments. This case underscores the importance of maintaining a high level of suspicion for tuberculosis in the differential diagnosis of unusual hepatic and pancreatic masses, especially in regions with high tuberculosis prevalence. While anti-tubercular therapy is usually effective in resolving active infection, the fibrosing cicatricial evolution that may follow treatment can complicate management, potentially leading to structural changes that hinder further therapeutic interventions. This case represents a rare instance of this fibrosing complication in hepatopancreatic tuberculosis highlighting the need for ongoing surveillance and early intervention in these cases.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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