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Spontaneous resolution of portal vein thrombosis and pseudoaneurysm secondary to hepatic tuberculosis in a 30-year-old male: A case report

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Abstract

Objectives: To report a case of a patient with a spontaneous resolution of his portal vein thrombosis and pseudoaneurysm following isoniazid (H), rifampicin (R), and ethambutol (E) treatment for hepatic tuberculosis, set in a tertiary government hospital.

Results: This case involves a 30-year-old male from South Cotabato, Philippines, who presented with jaundice, abdominal pain, and fever. He was initially diagnosed with hepatic abscess, nodules, and chronic portal vein thrombosis with cavernous transformation. Due to financial constraints, he delayed follow-ups and treatment, worsening his condition. Upon admission, he had severe jaundice and abdominal pain. Initial tests suggested obstructive jaundice due to common hepatic duct strictures and secondary complications. A whole abdomen CT scan showed multiple hepatic masses with calcifications. He underwent endoscopic retrograde cholangiopancreatography (ERCP), where a stent was placed to relieve bile obstruction. Sputum geneXpert revealed *Mycobacterium tuberculosis*, leading to the initiation of the anti-Koch regimen. His condition improved with antibiotics and hepatoprotective therapy. Later, a repeat CT scan suggested a hepatic pseudoaneurysm, but treatment continued with an HRE regimen. On follow-up, a repeat whole abdomen ultrasound revealed the resolution of the pseudoaneurysm and portal vein thrombosis with cavernous transformation, confirming that tuberculosis was the underlying cause. The patient was discharged in stable condition with an 8-month anti-TB regimen, and regular follow-ups were advised.

Conclusion: This case emphasizes the importance of a high level of suspicion when diagnosing hepatic TB, as tuberculosis is the greatest masquerader of all, particularly in high-burden regions like the Philippines. It also stresses the need for a multidisciplinary approach in managing complex hepatobiliary conditions. Although thrombectomy with anticoagulation is typically necessary for portal vein thrombosis, and hepatic artery pseudoaneurysms are generally considered irreversible, treated through coiling or surgery, this case suggests that careful medical management can lead to significant improvement and even spontaneous resolution. To our knowledge, this is the first documented case in the Philippines demonstrating spontaneous resolution of portal vein thrombosis and a pseudoaneurysm secondary to hepatic tuberculosis following anti-tuberculosis therapy. Further research is needed to understand these rare occurrences and optimize treatment strategies.

Keywords: *hepatic tuberculosis, Mycobacterium tuberculosis, portal vein thrombosis, hepatic pseudoaneurysm, endoscopic retrograde cholangiopancreatography*

Introduction

Hepatic tuberculosis is a rare cause of portal vein thrombosis (PVT) and hepatic pseudoaneurysm, as identified in published studies.[1, 2] PVT is most common in patients with cirrhosis, but can also occur in conditions like malignancy, pancreatitis, abdominal infections, and hypercoagulable states.[3] Hepatic pseudoaneurysms are characterized by abnormal dilation of the hepatic arteries, and patients present with the classic Quincke triad: upper abdominal pain, jaundice, and hemobilia (gastrointestinal bleeding).[4] It is rare, but can develop after trauma, surgery, or disease, posing a significant risk if untreated.[4] Both conditions typically require prompt surgical or endovascular treatment, although a few cases report spontaneous resolution with conservative or medical therapy. [2,5,6,7] This case report discusses a patient with PVT and hepatic pseudoaneurysm due to hepatic tuberculosis, whose condition improved with anti-Koch regimen.

Case Report

A 30-year-old male from South Cotabato, Philippines, presented with a two-week history of low-grade intermittent fever, progressive right upper quadrant abdominal pain, and gradual onset of jaundice. The patient had a documented history of pulmonary tuberculosis diagnosed in 2021, for which treatment was initiated but not completed due to loss to follow-up. He had no known history of diabetes mellitus or hypertension. Occupationally, he was a former seafarer and delivery driver. He reported occasional alcohol consumption, denied smoking, and had no history of illicit drug use. Family history was unremarkable for liver disease, diabetes mellitus, asthma, or allergies. However, a maternal aunt was diagnosed with nasopharyngeal carcinoma, and his mother had a history of hypertension.

The patient was initially seen for fever, abdominal pain, and jaundice in October 2023. A CT scan was suggestive of hepatic abscess vs. tuberculous infection described as multiple, fairly-defined, irregular, solid and cystic hypodense foci, some with mild heterogeneous and minimal peripheral enhancement diffusely seen in both hepatic lobes involving segments I, II, IVA, V, VI, VII and VIII, the largest measuring 10.3 × 5.2 x 5.2 cm (CCWAP) seen in segment 1. A hepatic nodule was also seen in segment 1, measuring 5.9 x 4.7 x 3.9 cm (LWT). A portal venous compression from segment 1 hepatic mass, with secondary cavernous transformation, suggestive of a portal vein thrombosis, was also noted. He was started on Rivaroxaban, Spironolactone, and Carvedilol, but was discharged with jaundice, referred for further evaluation, but could not follow up due to financial constraints.

One month later, he still had jaundice and abdominal discomfort. As claimed, the patient was compliant with his discharge medications. He then sought consult with a private physician, where a hepatic ultrasound and Doppler confirmed the initial findings of intrahepatic and common bile duct ectasia and portal vein thrombosis. He was referred to hematology and hepatobiliary surgery for evaluation. The recommendation was to do endoscopic retrograde cholangiopancreatography (ERCP). However, he could not comply due to economic limitations.

On the day of admission, the patient developed sudden nausea and weakness, unable to perform daily activities, prompting a visit to the emergency room. Upon arrival, vital signs were stable. On physical examination, the patient had jaundice and a distended, soft abdomen. The liver edge was soft and palpable with mild tenderness on deep palpation. No spider angiomata or abdominal wall collateral veins were observed. While the spleen was not clearly palpable, fullness and dullness on deep palpation of the left upper quadrant suggested possible splenic enlargement. Bowel sounds were normoactive. There was no palmar erythema, no bipedal edema, and capillary refill time was less than 2 seconds. Laboratory results showed anemia

(109 g/L), mild thrombocytopenia (125 × 10^3), elevated liver enzymes [Alanine Aminotransferase (ALT) 314.93 U/L (NV <50); Aspartate Aminotransferase (AST) 160.44 U/L (NV <50)], and bilirubin levels [total bilirubin 247 (NV 5-21 umol/L), direct bilirubin 143 (NV <3.4 umol/L), indirect bilirubin 103]. Viral hepatitis panel showed the patient was non-immune to hepatitis B [HBsAg: nonreactive, 0.41 (cutoff: 1.0); anti-HBs: nonreactive, 0.80 mIU/mL (cutoff: 10.0)], and negative for hepatitis C [anti-HCV: nonreactive, 0.22 (cutoff: 1.0)]. HIV screening was also negative. He was started on UDCA 500mg BID, and Rivaroxaban was temporarily discontinued due to consideration of anemia from upper gastrointestinal bleeding. A work-up for primary thrombophilia was requested but not performed due to financial constraints.

A whole abdomen CT scan was performed upon admission, which revealed multiple hepatic masses with calcifications and suspicious hepatic infarcts (Figure 1). Persistence of PVT was noted with secondary portal hypertension and cavernous transformation.

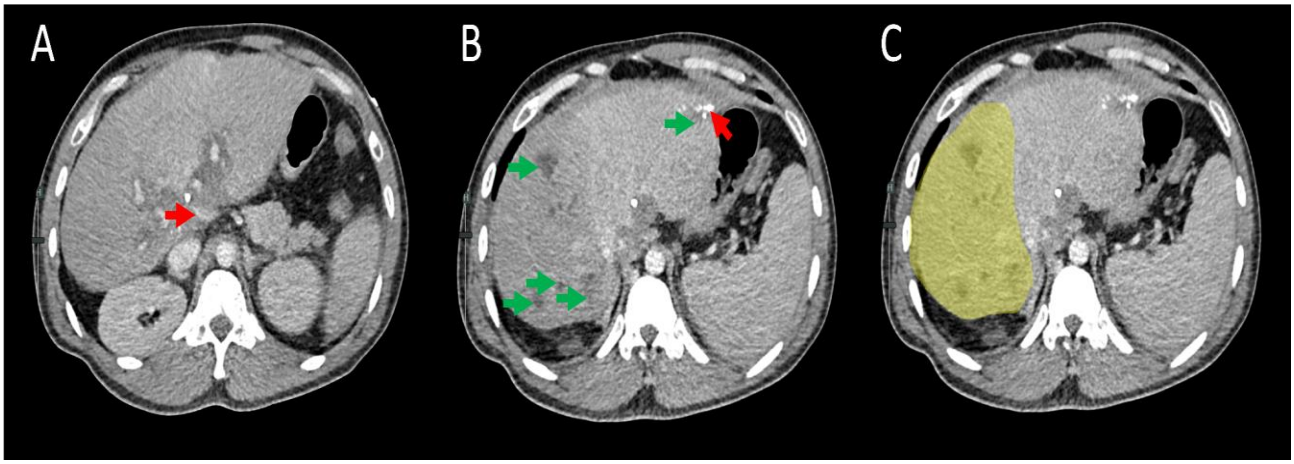

Figure 1. Portovenous phase, axial views. [A] The arrow shows the portal vein's abrupt cut-off at the porta hepatis region. [B] Multiple hepatic masses (green arrows) in segments II, VII, and VIII. Calcifications (red arrow) are also seen in segment II. [C] Hypoattenuating parenchymal areas suggest hepatic infarct (shaded area) involving segments V to VIII.

The patient underwent ERCP, which revealed a stricture in the common hepatic duct, causing ectasia in the right intrahepatic duct and a filling defect extending into the left main intrahepatic duct. A 7-French, 15 cm Duodenal Bend Boston Scientific Stent was successfully deployed in the left intrahepatic duct, with good bile and dye flow post-insertion. The procedure was uneventful, and the patient was transferred to the ward for monitoring.

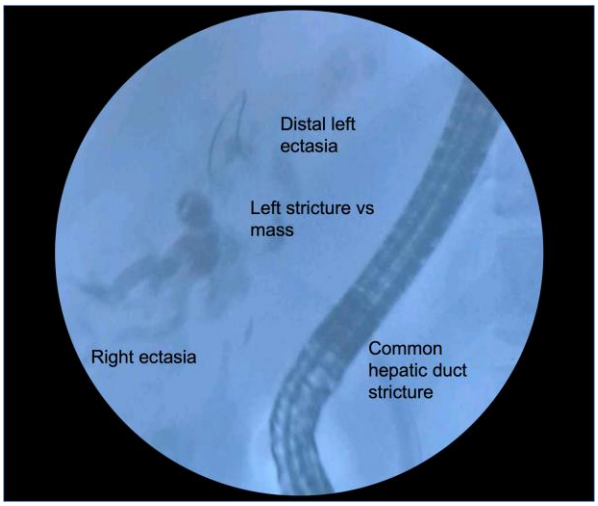

Figure 2. Cholangiography shows a common hepatic duct stricture of 15 mm length, producing upstream ectasia of the right intrahepatic duct. The mass effect or filling defect was seen from the stricture extending 20 mm into the left main intrahepatic duct, producing upstream ectasia of the distal left intrahepatic duct radicles.

The hepatic nodule biopsy showed few atypical cells with mild nuclear enlargement and scant to moderate cytoplasm, which was inconclusive. Given the patient's history of a prior diagnosis of pulmonary tuberculosis with incomplete treatment and subsequent loss to follow-up, a sputum GeneXpert test was performed. The test returned positive for *Mycobacterium tuberculosis*, so the patient was started on an anti-Koch regimen (HRE) for pulmonary and potential hepatic tuberculosis. Pyrazinamide was excluded due to elevated liver enzymes and a hepatic abscess seen on outpatient CT scan. Cefepime and metronidazole, later switched to meropenem, were given to treat the abscesses.

Upper gastrointestinal endoscopy was done after the patient had melena. It showed Grade I esophageal varices. Duodenal bulb varices were also seen, with the largest treated using cyanoacrylate glue injection. A Forrest IIA ulcer (non-bleeding visible vessel) in the second portion of the duodenum was also noted, and a hemostatic clip was applied to prevent further bleeding. Intravenous omeprazole was started.

One month post-ERCP, a repeat CT scan was done since the patient developed new-onset GI symptoms (abdominal discomfort, bloating, and nausea), which revealed new findings, including portal venous compression, cavernous transformation, and a pseudoaneurysm in the hepatic mass (Figure 3). Although an adverse drug reaction to the HRE anti-tuberculosis regimen was initially considered, this was deemed less likely given the exclusion of pyrazinamide. Instead, disease progression was favored, with imaging suggesting increased intrahepatic mass effect and vascular involvement as likely contributors to the symptoms, possibly through mechanisms such as portal hypertension and local compression. Gastroenterology and infectious disease recommended continuing the HRE regimen, and the patient was started on hepatoprotective therapy (carnitine orotate, silymarin, essential phospholipids, B vitamins, zinc) and anticoagulation (Rivaroxaban 10 mg tablet once a day). The patient showed clinical improvement and was discharged in stable condition, with strict follow-up advised for ongoing treatment.

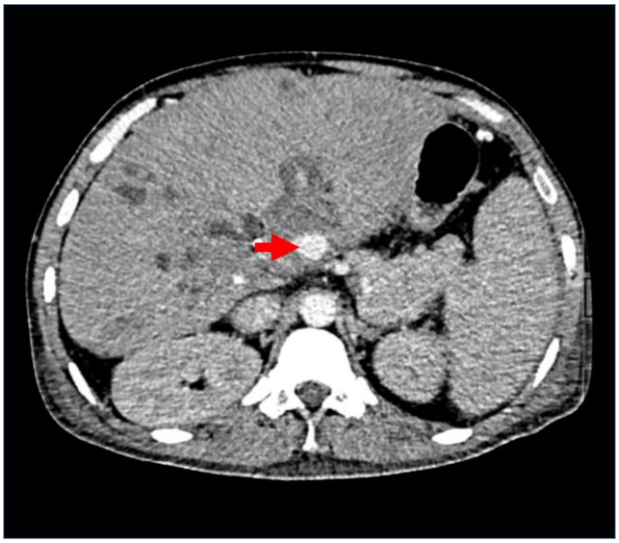

Figure 3. Portovenous phase, axial view. A mass in segment I exhibits a centrally dilated vascular structure with strong enhancement measuring 3.1 x 1.8 x 1.7 cm. A feeding vessel is noted from the left branch of the common hepatic artery and vein.

Three months post-discharge, the patient was readmitted for ERCP and stent removal. Vital signs were stable, and jaundice had resolved. A repeat abdominal CT scan showed the disappearance of the pseudoaneurysm in segment one and resolution of portal vein thrombosis with cavernous transformation (Figure 4).

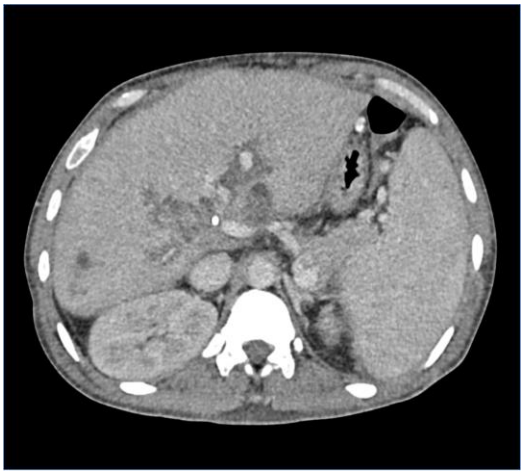

Figure 4. Portovenous phase, axial view. A comparative study was done, which showed spontaneous resolution of the pseudoaneurysm previously seen in segment I.

During the ERCP, the plastic stent was removed using a snare. Cholangiography showed a non-dilated biliary tree with no filling defects, and the previous common hepatic duct stricture was no longer present. Biliary balloon dilation was performed, and satisfactory bile and dye flow were observed. The patient was discharged in a stable and improved condition. He was advised to complete his anti-Koch regimen for a total of 8 months and to maintain regular follow-up.

Discussion

Hepatic tuberculosis is an extrapulmonary manifestation of *Mycobacterium tuberculosis* infection, commonly presenting as infectious hepatic granulomas.[8] Hepatic tuberculosis can occur independently of pulmonary disease and is classified into three forms: local hepatic TB which involves focal liver lesions with caseating hilar lymph nodes; miliary TB, the most common, features numerous clustered tubercles in the liver; and tuberculomas which are larger granulomas, formed from miliary foci or tertiary nodular development, typically appearing as 1–4 cm nodules with calcifications. [9, 10]

Hepatic involvement occurs in about 1% of patients with active TB.[11] Certain racial and ethnic groups have higher TB rates due to factors such as origin from high-prevalence countries like the Philippines, HIV infection, low socio-economic status, and exposure to TB in high-risk environments.[12]

Zheng et al. suggest that thromboembolic complications like portal vein thrombosis in TB may result from all components of Virchow's triad (hypercoagulability, venous stasis, and endothelial dysfunction). Inflammation, granuloma formation, and subintimal fibrosis may also contribute to PVT.[5] While commonly seen in liver cirrhosis, it can also be linked to cancer, abdominal infections, pancreatitis, autoimmune disorders, and hematologic conditions that increase blood clotting.[13] It is a rare (1.5 - 3.4%) complication of pulmonary and extrapulmonary tuberculosis.[14]

PVT is often asymptomatic but can present acutely or chronically, with no apparent time distinction. Chronic PVT may cause portal hypertension, leading to left upper quadrant fullness from splenomegaly or upper GI bleeding from varices. Acute PVT typically presents with abdominal pain, fever, and ascites.[17] Severe cases of thrombus extension into the superior mesenteric vein can cause intestinal ischemia, bowel infarction, and ileus, leading to hematochezia, fever, and sepsis, with high mortality.[13] This case of a 30-year-old male reflects typical portal vein thrombosis features. Chronic obstruction leads to portal hypertension, causing splenomegaly, abdominal discomfort, and variceal bleeding, which aligns with the patient's symptoms of jaundice, recurrent abdominal pain, and esophageal varices confirmed during endoscopy.

Doppler ultrasound was the primary diagnostic tool for portal vein thrombosis, with sensitivity and specificity ranging from 80% to 100%, and 88% to 98%, respectively. It detected material that partially or fully obstructs the portal vein without reducing venous flow.[13, 16] CT and MRI further evaluated PVT, providing detailed information on thrombus extension, bowel infarction, and surrounding organ condition.[13] Doppler ultrasound confirmed persistent portal vein thrombosis and cavernous transformation. A contrast-enhanced CT scan revealed hepatic nodules, calcifications, and chronic PVT with cavernous transformation, consistent with the described chronic PVT features.

Portal cholangiopathy could occur in chronic PVT due to bile duct compression by venous collaterals, presenting as obstructive jaundice and cholangitis.[17] This case displayed typical portal cholangiopathy features, including bile duct strictures and intrahepatic duct ectasia, confirmed by ERCP and imaging. Successful biliary stent placement and resolution of obstruction supported the diagnosis of external bile duct compression from portal cavernoma.

The patient's progression involved complications of portal vein thrombosis, including esophageal varices and biliary obstruction. The treatment plan included ERCP, stenting, an anti-Koch regimen for hepatic tuberculosis, and hepatoprotective therapy, which aligned with the recommended multidisciplinary approach. As noted by Zheng et al., early initiation of anti-TB treatment with isoniazid, rifapentine, ethambutol, and pyrazinamide was crucial.[5] In this case, anticoagulation therapy was initially withheld due to melena but was resumed after upper gastrointestinal endoscopy revealed a duodenal ulcer that was successfully treated, and no further episodes of bleeding were observed. While spontaneous

thrombosis resolution had occurred, anticoagulant patients typically showed higher recanalization rates in acute and chronic cases.[13, 18] In this patient, it was plausible that the eventual administration of anticoagulants contributed to the resolution of the portal vein thrombosis, potentially enhancing the natural recanalization process once bleeding risks were controlled. The patient was started on isoniazid, rifampicin, and ethambutol (excluding pyrazinamide due to elevated liver enzymes and a hepatic abscess). Despite this, symptoms resolved, similar to Zheng et al.'s case, where symptoms improved one week after starting the HRZE regimen.[5] A follow-up ultrasound three months post-discharge showed resolution of portal vein thrombosis. ERCP revealed a non-dilated biliary tree with no defects, and the previous common hepatic duct stricture had resolved. Biliary balloon dilation was performed, with satisfactory bile and dye flow.

Hepatic pseudoaneurysms are rare vascular abnormalities caused by abnormal dilation of the hepatic arteries. The most common cause is iatrogenic injury, although factors like abdominal trauma, tumors, infections, atherosclerotic ulcers, and vasculitis (e.g., juvenile polyarteritis nodosa, Bechet's disease, rheumatoid arthritis) can also contribute.[19] Tuberculosis is a rare cause, typically resulting from an infection that spreads from adjacent lymph nodes.[20]

In this case, the patient presented with jaundice, abdominal pain, fever, and gastrointestinal bleeding. Similarly, Khan et al. described hepatic pseudoaneurysm as presenting with the Quincke triad: upper abdominal pain, jaundice, and hemobilia.[19] Zhao et al. reported that hemobilia was the most common symptom of hepatic pseudoaneurysm in up to one-third of patients. It could lead to gastrointestinal bleeding and symptoms like jaundice, as observed in this case.[4] This was typically caused by a pseudoaneurysm compressing or creating a fistula with the biliary system, although this was not the case here. In this report, jaundice was caused by a bile duct stricture, while gastrointestinal bleeding resulted from varices due to portal hypertension from PVT.

Zhao et al. identified selective hepatic arteriography as the most accurate diagnostic tool for hepatic pseudoaneurysms, providing detailed imaging of arterial damage and enabling targeted interventions.[4] The case report used CT scans, Doppler ultrasound, and ERCP to evaluate hepatic masses, bile duct strictures, and vascular complications. A hepatic artery pseudoaneurysm was incidentally found during follow-up imaging. Although ERCP was used for biliary decompression and biopsy of hepatic nodules, arteriography was not performed, limiting confirmation of the hepatic pseudoaneurysm.

The treatment strategy in this case was conservative, including antibiotics, an anti-Koch regimen, and hepatoprotective therapy. A plastic stent was placed via ERCP to relieve bile duct obstruction, and Rivaroxaban was initially prescribed for portal vein thrombosis but later discontinued. Transarterial embolization (TAE), the preferred treatment for hepatic pseudoaneurysm, might be used urgently to prevent rupture.[4, 21] Jha et al. highlighted the risks of delayed treatment, noting that untreated hepatic pseudoaneurysms could lead to life-threatening hemorrhage.[21] Pandey et al. reported a tuberculous hepatic pseudoaneurysm resolution after embolization and the initiation of an anti-Koch regimen.[22] In our case, although embolization was not performed, the initiation of anti-TB medications led to the eventual resolution of the hepatic pseudoaneurysm, as seen in follow-up imaging.

Conclusion

This case highlights the importance of a high index of suspicion approach for hepatic TB in high TB-burden areas like the Philippines. It emphasizes a multidisciplinary perspective for complex hepatobiliary conditions. While surgery is typically needed for portal vein thrombosis and hepatic artery pseudoaneurysms, this case suggests that careful medical management can lead to significant improvement and even spontaneous resolution. This appears to be the first reported case in the Philippines showing radiologic resolution of both portal vein thrombosis and pseudoaneurysm attributed to hepatic tuberculosis, highlighting the potential reversibility of these rare complications with timely anti-TB treatment. Further studies are needed to understand these rare occurrences and optimize treatment for patients with co-existing portal vein thrombosis, pseudoaneurysm, and hepatic TB.

Declarations and Disclosures

The authors declared that this case report represented original material that had not been published or accepted for publication elsewhere, in full or in part, in print or electronic media; that the manuscript had been read and approved by all authors, that each author had met the requirements for authorship, and that the authors believed that the manuscript represented honest work.

The authors signed disclosures that there were no financial or other, including personal, relationships, intellectual passion, political or religious beliefs, or institutional affiliations that might lead to a conflict of interest.

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