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Hepato-Pancreato-Biliary Tuberculosis in A Young Patient: A Rare but Fatal Presentation

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Abstract **Case Report**

Background: Hepato-pancreato-biliary tuberculosis is a rare form of extrapulmonary tuberculosis, particularly in young immunocompetent patients. Its clinical and radiological presentation can closely mimic other conditions, most notably pancreatic malignancy, posing significant diagnostic challenges. Complications such as portal vein thrombosis and portal cavernoma further complicate its management, especially in resource-limited settings where advanced interventional procedures remain unavailable. ***Case Presentation:*** We report the case of a 21-year-old immunocompetent male who presented with a two-month history of jaundice, night sweats, and significant weight loss. Clinical examination revealed jaundice, a firm painless right cervical lymphadenopathy, hepatomegaly, and splenomegaly. Laboratory findings demonstrated thrombocytopenia, moderate hepatocellular injury, and a cholestatic pattern. Magnetic Resonance Cholangiopancreatography (MRCP) revealed a pancreatic head mass with peripancreatic fat infiltration, portal vein thrombosis, portal cavernoma, biliary dilation secondary to distal common bile duct stricture, and splenomegaly. An extensive infectious and autoimmune workup was negative. Histopathological examination the cervical lymph node confirmed tuberculosis. Upper gastrointestinal endoscopy identified grade III esophageal varices, managed with endoscopic band ligation and non-selective beta-blockers. Transjugular Intrahepatic Portosystemic Shunt (TIPS) placement was considered but unavailable at our institution. The patient was started on the standard four-drug antituberculous regimen (HRZE) with no subsequent hepatotoxicity. Tragically, one month into treatment, and just prior to his scheduled second endoscopic band ligation session, the patient succumbed to a massive variceal hemorrhage. ***Conclusion:*** This case highlights the diagnostic complexity of hepato-pancreato-biliary tuberculosis and its potentially fatal course when complicated by severe portal hypertension. It underscores the critical importance of early diagnosis, prompt endoscopic management of high-grade varices, and the urgent need for access to advanced interventional procedures such as TIPS in resource-limited settings.

Keywords: tuberculosis; pancreatic tuberculosis; hepatic tuberculosis; portal vein thrombosis; portal cavernoma; esophageal varices; portal hypertension; TIPS; extrapulmonary tuberculosis.

INTRODUCTION

Hepato-pancreato-biliary (HPB) tuberculosis is a rare but clinically severe form of extrapulmonary tuberculosis that poses major diagnostic challenges due to its ability to closely mimic pancreatic and hepatobiliary malignancies. While tuberculosis (TB) remains a major global health burden - particularly in Africa - involvement of the HPB system is uncommon and is often diagnosed late or incidentally through histopathological examination. Early diagnosis and appropriate treatment are crucial for preventing complications and ensuring patient safety. [1] When complicated by vascular involvement such as portal vein thrombosis and portal cavernoma, the condition carries a substantially elevated risk of life-threatening hemorrhage. We report a case of disseminated HPB tuberculosis in a young immunocompetent patient, presenting with obstructive jaundice, portal hypertension, and grade III esophageal varices, highlighting the diagnostic and therapeutic challenges encountered in a resource-limited setting.

CASE PRESENTATION

A 21-year-old male patient with no prior medical history presented to our institution with a two-

month history of progressive jaundice, drenching night sweats, and significant unintentional weight loss.

On physical examination, the patient was icteric. Cervical examination revealed a firm, painless right-sided neck mass. Abdominal examination demonstrated hepatomegaly and splenomegaly without ascites or signs of chronic liver disease.

Laboratory investigations revealed thrombocytopenia at 78 G/L, along with a moderate hepatocellular and cholestatic pattern of liver injury, with aspartate aminotransferase (ASAT) at 144 U/L, alanine aminotransferase (ALAT) at 132 U/L, alkaline phosphatase (ALP) at 432 U/L, gamma-glutamyl transferase (GGT) at 210 U/L, and direct bilirubin at 43 µmol/L.

Magnetic resonance cholangiopancreatography (MRCP) demonstrated global enlargement of the pancreatic head measuring 34 mm in anteroposterior diameter. There was infiltration of the hepatic pedicle associated with portal vein thrombosis, development of portosystemic collateral circulation, and splenomegaly measuring 180 mm. The common bile duct was dilated at 9 mm with moderate intrahepatic biliary dilation, secondary to a smooth stricture of the distal common bile duct. A stricture of the cephalic portion of the main pancreatic duct was also noted, while the body and tail of the pancreas appeared normal. The liver was of normal size with homogeneous signal, and no peritoneal effusion was identified. (figure 1,2 and 3)

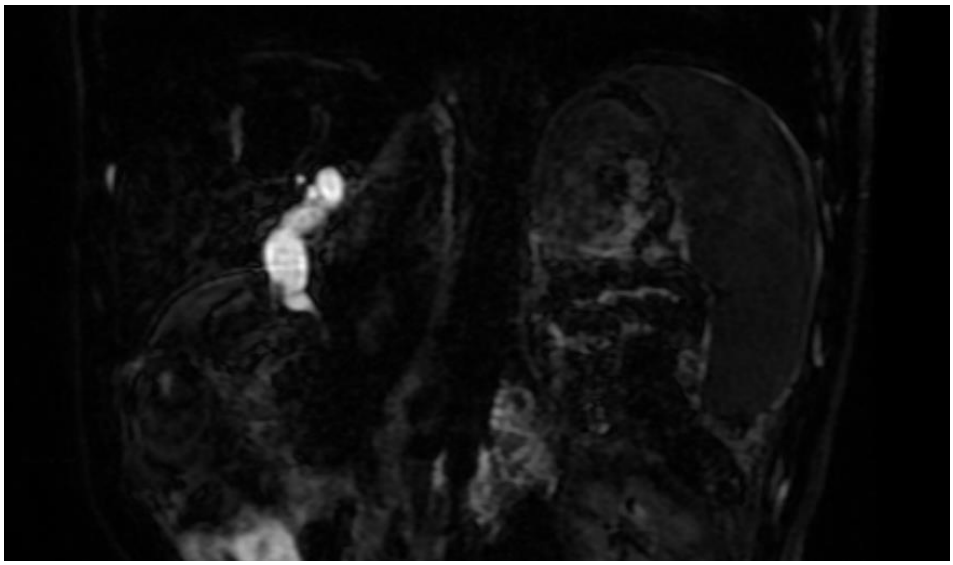

Figure 1: MRCP T2W in 3D reconstruction showing dilation of the common bile duct with a threadlike narrowing of its distal part

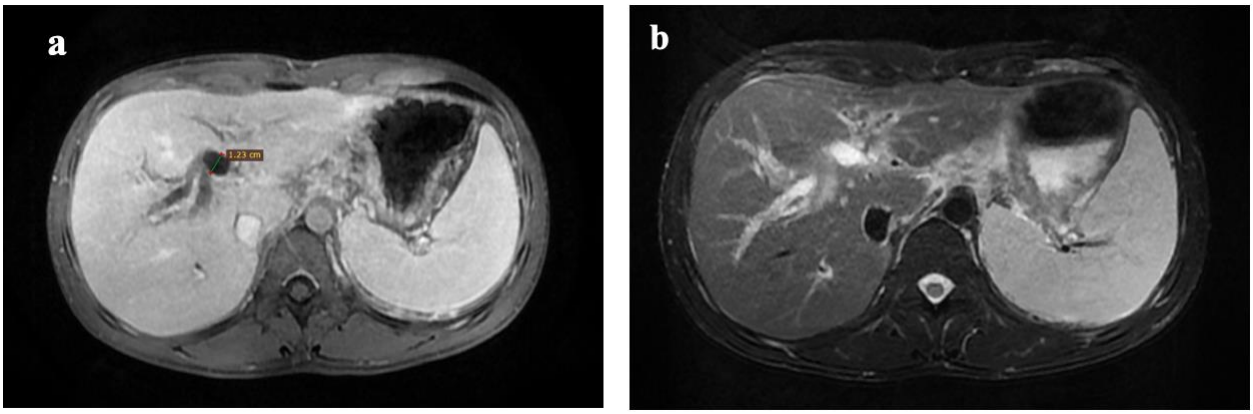

Figure 2: Axial MRCP Contrast-enhanced T1 (a) and T2 FAT SAT (b) demonstrating a dilation of the common bile duct and the proximal intrahepatic bile ducts

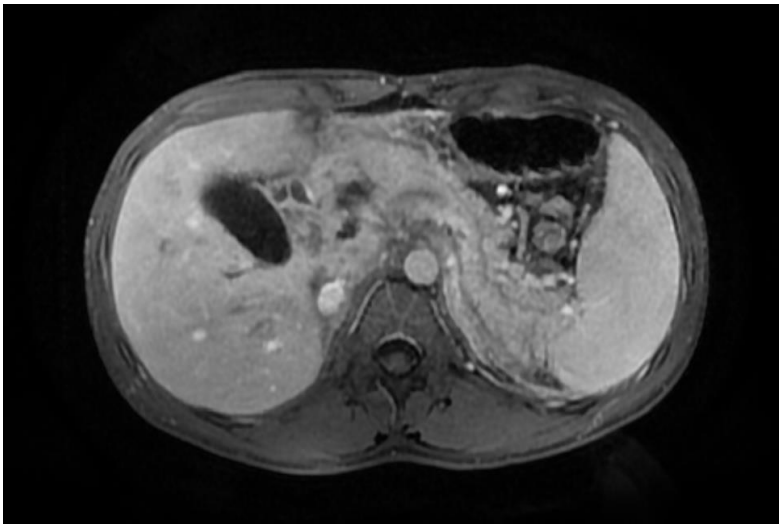

Figure 3: Axial T1-weighted sequence after contrast injection showing perihilar collateral circulation consistent with early portal cavernoma formation

An extensive infectious and autoimmune workup was performed. Serologies for hepatitis B, hepatitis C, human immunodeficiency virus (HIV), Epstein-Barr virus and cytomegalovirus were all negative. Evaluation for autoimmune etiologies, including serum IgG4 levels, was similarly unremarkable.

Upper gastrointestinal endoscopy revealed grade III oesophageal varices, consistent with significant portal hypertension (Figure 4). Given the severity of the varices, endoscopic band ligation was performed as primary prophylaxis, with a second session of religation scheduled as part of the standard eradication protocol.

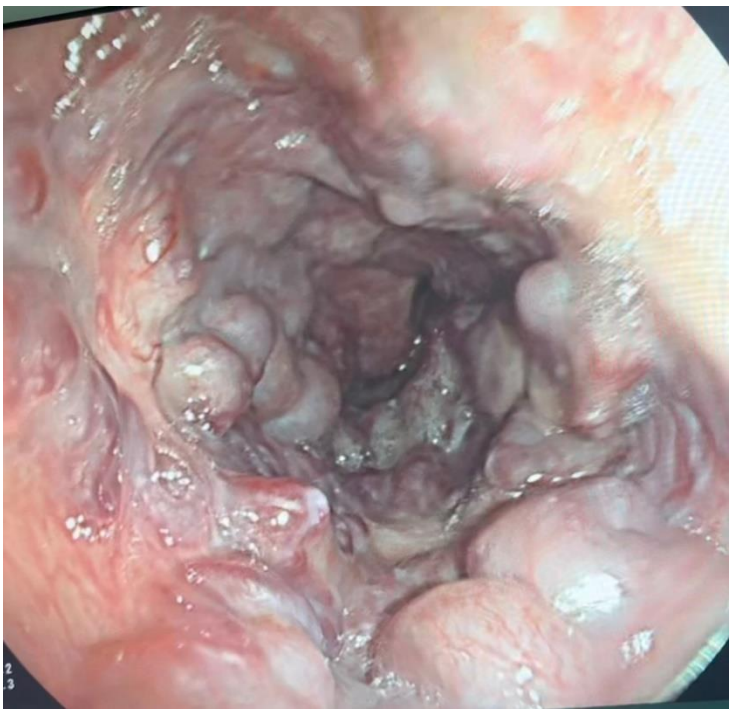

Figure 4: Upper gastrointestinal endoscopy showing esophageal varices grade III

Following the procedure, and after careful exclusion of contraindications including hemodynamic instability, significant bradycardia, and obstructive airway disease the patient was initiated on non-selective beta-blocker therapy for secondary prevention of variceal bleeding. Given the extent of portal vein thrombosis, portal cavernoma, and the severity of portal hypertension, transjugular intrahepatic portosystemic shunt (TIPS) placement was discussed and considered by the multidisciplinary team as a potential therapeutic option. However, this procedure was unavailable at our institution, and is not currently performed in Morocco, precluding its use in this patient.

Histopathological examination of the right cervical lymph node biopsy demonstrated granulomatous inflammation with caseous necrosis, consistent with tuberculosis. Liver biopsy similarly couldn't be performed due to an important dilation of the intrahepatic bile ducts at the moment of the procedure.

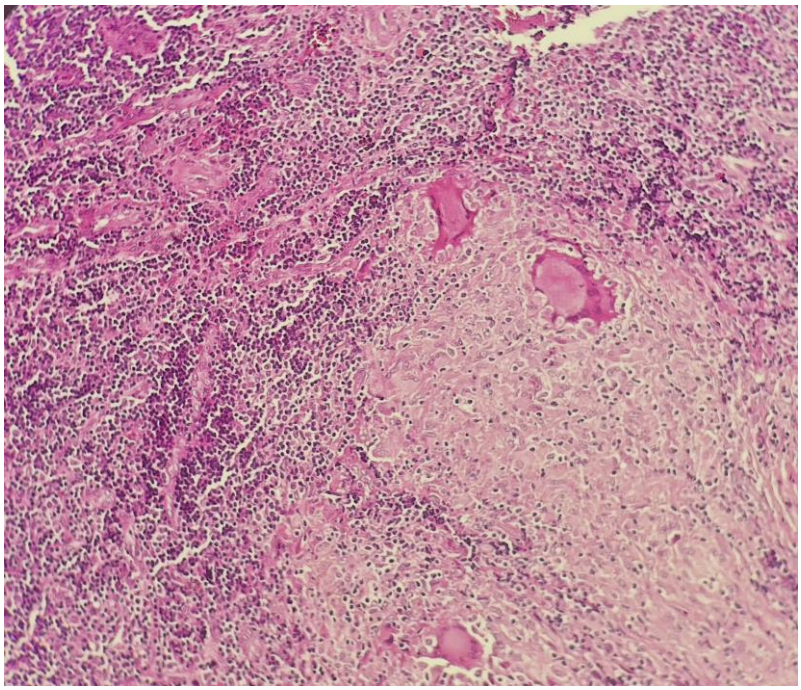

Figure 5: Lymphoid tissue showing a vaguely granulomatous epithelioid and giant cell lesion with foci of caseous necrosis (HE ×20)

Based on these findings, a diagnosis of disseminated tuberculosis with pancreatic, hepatic, and lymph node involvement, complicated by portal vein thrombosis, portal cavernoma, and severe portal hypertension was suspected. The patient was initiated on a standard four-drug antituberculous regimen consisting of isoniazid (H), rifampicin (R), pyrazinamide (Z), and ethambutol (E). Hepatic function was closely monitored throughout the course of treatment. Notably, serial liver function tests following initiation of antituberculous therapy demonstrated a significant improvement during the follow-up period.

Unfortunately, approximately two months after treatment initiation, and critically just prior to the patient's scheduled second endoscopic band ligation session, the patient developed a sudden and massive variceal hemorrhage secondary to rupture of his oesophageal varices. The hemorrhage occurred before the planned religation could be performed, highlighting the narrow and unforgiving window between endoscopic sessions in patients with high-grade varices and severe underlying portal hypertension. Despite prompt resuscitative measures, the hemorrhage proved fatal.

DISCUSSION

Hepato-pancreato-biliary (HPB) tuberculosis is a rare form of extrapulmonary tuberculosis, (<1% of abdominal TB) that poses a diagnostic challenge and is a great masquerader of malignancy, both clinically and radiologically. It is almost always curable but requires a high degree of suspicion and corroborating evidence to establish the diagnosis. [2]

Tuberculosis continues to be prevalent in African and Asian countries, where extrapulmonary forms account for approximately 15% of all cases, with abdominal tuberculosis being one of the most common localizations. This could possibly be due to increased number of immunocompromised individuals, particularly those affected by (HIV). [3]

Mycobacteria may reach the liver and biliary tract via the hepatic artery from the lungs, or via the portal vein from the gastrointestinal tract, where intestinal ulcers may heal with time, leaving the patient with apparently isolated HPB tuberculosis. Pancreatic involvement is thought to occur through contiguous spread from adjacent retroperitoneal and peripancreatic lymph nodes. [1,3]

The clinical presentation of HPB tuberculosis is nonspecific. Common symptoms include jaundice, weight loss, abdominal pain, and other constitutional symptoms that render the condition clinically indistinguishable from malignancy. Pancreatic tuberculosis may manifest as a mass resembling malignancy in up to 80% of patients, with concomitant peripancreatic lymph nodal involvement seen in approximately half of cases. Biliary strictures may occur at the distal common bile duct with dilation of the

intrahepatic ducts, and the overall picture can closely mimic cholangiocarcinoma, as was the case in our patient. The diagnosis was only established through histopathological examination of the cervical lymph node, underscoring the importance of tissue sampling in atypical presentations. Imaging modalities including MRI may be used in the evaluation and can reveal dilated intrahepatic biliary radicles, mass lesions, biliary strictures, and portal vein thrombosis, but histopathological confirmation remains the gold standard. [4]

One of the most striking and life-threatening features of this case was the presence of portal vein thrombosis with portal cavernoma formation. Portal hypertension secondary to portal vein compression or thrombosis is a rare but recognized complication of pancreatic tuberculosis, particularly when the head of the pancreas is involved. [5] Cavernous transformation of the portal vein may occur as early as 6 to 20 days after portal vein obstruction, with an average of approximately 5 weeks, and often manifests insidiously with gastro-oesophageal variceal bleeding, splenomegaly, and thrombocytopenia. [7] The biliary tree may also undergo morphological changes due to portal cavernoma, resulting in obstructive jaundice a phenomenon referred to as portal biliopathy which likely contributed to the biliary obstruction observed in our patient in addition to the direct compressive effect of the pancreatic head mass. [7] The association between abdominal tuberculosis and portal vein thrombosis, while rare, has been described in the literature. Ruttenberg *et al.,* reported cases of abdominal tuberculosis complicated by portal vein thrombosis and portal hypertension, emphasizing the importance of considering tuberculosis in the differential diagnosis of non-cirrhotic portal hypertension in endemic regions. [8]

The management of portal hypertension in this context is particularly challenging. Current guidelines recommend screening for varices in patients with non-cirrhotic portal vein thrombosis who do not achieve recanalization, and treating high-risk varices with either non-selective beta-blockers or endoscopic variceal ligation. For secondary prophylaxis, a combination of both approaches is recommended. [9] In our patient, endoscopic band ligation was performed, followed by initiation of non-selective beta-blockade after elimination of contraindications, in keeping with these recommendations.

Tragically, the fatal hemorrhage occurred just before the scheduled second ligation session. It has been emphasized that one of the most common errors in clinical management of patients with variceal bleeding is treating the acute episode but failing to ensure the patient returns for subsequent band ligation sessions, which must be performed every one to two weeks over multiple sessions to achieve complete variceal obliteration. In patients with incompletely obliterated varices, the risk of rebleeding has been estimated at as high as 80%. [10] The timing of hemorrhage in our patient raises the important question of whether a shorter interval between ligation sessions should be considered in patients with grade III varices and established portal cavernoma.

Given the severity of portal hypertension and portal cavernoma, TIPS placement was discussed and considered by our multidisciplinary team. TIPS is an important intervention for managing refractory complications of portal hypertension in patients with chronic non-cirrhotic portal vein thrombosis, with reported technical success rates of approximately 95% and a 12-month portal vein recanalization rate of 79%. [11] Studies have demonstrated that successful TIPS insertion in patients with portal cavernoma significantly reduces portosystemic pressure gradients and lowers the incidence of variceal rebleeding. [12] Unfortunately, this procedure is unavailable in Morocco, representing a critical gap in the therapeutic armamentarium available in resource-limited settings and highlighting the broader issue of inequitable access to advanced interventional hepatology procedures.

The patient was initiated on the standard WHO-recommended four-drug intensive phase regimen: isoniazid (H), rifampicin (R), pyrazinamide (Z), and ethambutol (E). Drug-induced hepatotoxicity during standard multidrug tuberculosis treatment has been reported with an incidence ranging from 2% to 28%, depending on the definition used and the population studied.[13] Among the first-line agents, isoniazid, rifampicin, and pyrazinamide are recognized as potentially hepatotoxic, while ethambutol is generally considered hepatically safe.[14] Given our patient's pre-existing hepatic involvement from tuberculosis, close biochemical monitoring was undertaken throughout treatment. Serial liver function showed a considerable improvement which comforts the diagnosis giving the lack of histological confirmation.

CONCLUSION

This case illustrates the exceptional diagnostic complexity of disseminated hepato-pancreato-biliary tuberculosis, whose clinical, laboratory, and radiological features closely mimic pancreatic malignancy. The concurrent development of portal vein thrombosis, portal cavernoma, and high-grade esophageal varices transformed a treatable infectious disease into a fatal condition. The absence of TIPS availability at our institution and more broadly across Morocco represented a decisive therapeutic limitation. Clinicians in endemic regions should maintain a high index of suspicion for tuberculosis in young patients presenting with hepato-pancreato-biliary involvement, even in the absence of classical pulmonary features. Furthermore, this case calls for urgent efforts to expand access to advanced interventional procedures in resource-limited healthcare systems, where their absence can directly translate into preventable mortality.

List of abbreviations
HPB: Hepato-pancreato-biliary
MRCP: Magnetic Resonance Cholangiopancreatography
TIPS: Transjugular Intrahepatic Portosystemic Shunt
TB: Tuberculosis
ALAT: Alanine aminotransferase
ALP: Alkaline phosphatase
GGT: Gamma-glutamyl transferase
HIV: Human immunodeficiency virus

Declarations
Ethics approval and consent to participate
The study adhered to all applicable ethical guidelines and principles, fully aligning with the Declaration of Helsinki, while ensuring respect for the rights and well-being of the patient.

Informed consent
Informed consent was obtained from the patient involved in the study.

Data availability: No datasets were generated or analysed during the current study.

Competing interests: The authors declare no competing interests

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Author contributions
YY drafted and wrote the manuscript.
SS and JE arranged figures.
HA, ON, FEL, AAE, SO, ZS, and KK provided critical supervision, clinical oversight, and intellectual input throughout the case report.
All authors reviewed and approved the final version of the manuscript.

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REFERENCES

1. Esguerra-Paculan MJA, Soldera J. Hepatobiliary tuberculosis in the developing world. World J Gastrointest Surg. 2023;15(10):2305-2319. doi:10.4240/wjgs. v15.i10.2305
2. Chaudhary P. Hepato-pancreato-biliary tuberculosis: A review. Turkish Journal of Surgery. 2024. doi:10.23581/turkjsurg.2024.6338
3. Arun Sampath, Saravanan Mani, Diagnostic evaluation and management of abdominal tuberculosis, Indian Journal of Tuberculosis,Volume 72, Supplement 1,2025, Pages S7-S11, ISSN 0019-5707, 10.1016/j.ijtb.2025.02.008.
4. Panic, N., Maetzel, H., Bulajic, M., Radovanovic, M. and Löhr, J.-M. (2020), Pancreatic tuberculosis: A systematic review of symptoms, diagnosis and treatment. UEG Journal, 8: 396-402 doi:10.1177/2050640620902353
5. Koea JB, Shaw JH. Tuberculosis of the liver, biliary tract, and pancreas. Microbiology Spectrum. 2016. doi: 10.1128/microbiolspec.TNMI7-0025-2016
6. Heller T, Gabe SM. Hepatobiliary tuberculosis: imaging findings. American Journal of Roentgenology. 2016. doi:10.2214/AJR.15.15926
7. Wu Q, Li L, Feng X, *et al.,* Optimal treatment for patients with cavernous transformation of the portal vein. Frontiers in Medicine. 2022; 9:853138. doi:10.3389/fmed.2022.853138
8. Ruttenberg D, Graham S, Burns D, *et al.,* Abdominal tuberculosis a cause of portal vein thrombosis and portal hypertension. Digestive Diseases and Sciences. 1991; 36:112–115. doi:10.1007/BF01300098
9. Sieger B, Wiest R, Kamath PS, *et al.,* Current concepts in the management of non-cirrhotic non-malignant portal vein thrombosis. World Journal of Gastroenterology. 2024. doi:10.3748/wjg. v30.i19
10. Tasci I. Management of esophageal varices. Gastroenterology and Hepatology. 2017;13(3):166–173. PMCID: PMC5345213
11. Rodrigues SG, Becuwe C, Wiest R, *et al.,* Portal vein thrombosis in patients without cirrhosis : current practical approaches and treatment strategies. Diagnostics. 2025;15(6):721. doi:10.3390/diagnostics15060721
12. Qi X, Han G, He C, *et al.,* Transjugular intrahepatic portosystemic shunt for portal cavernoma with symptomatic portal hypertension in non-cirrhotic patients. Digestive Diseases and Sciences. 2012; 57:1071–1082. doi:10.1007/s10620-011-1975-5
13. Tostmann A, Boeree MJ, Aarnoutse RE, *et al.,* Antituberculosis drug-induced hepatotoxicity: concise up-to-date review. Journal of Gastroenterology and Hepatology. 2008; 23:192–202. doi:10.1111/j.1440-1746.2007. 05207.x
14. Saukkonen JJ, Cohn DL, Jasmer RM, *et al.,* An official ATS statement : hepatotoxicity of antituberculosis therapy. American Journal of Respiratory and Critical Care Medicine. 2006; 174:935–952. doi:10.1164/rccm.200510-1666ST