CASE SERIES

Isolated hepatopancreaticobiliary and splenic tuberculosis: a case series and review of the literature

G. S. Dessai, N. K. Soni, S. Pujari, R. Prabhu, C. V. Kantharia

Department of Surgical Gastroenterology, Seth GS Medical College and KEM Hospital, Mumbai, India

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Introduction

Tuberculosis is a preventable and curable disease; however, in 2022, it remained the world's second leading cause of death from a single infectious agent, following coronavirus disease (COVID-19). [1] India accounts for 28% of the global tuberculosis burden. While the lungs are the most commonly affected organ, extra-pulmonary tuberculosis constitutes approximately 16% of cases. Abdominal tuberculosis represents 11–13% of extra-pulmonary cases in India and often poses a diagnostic challenge due to its varied clinical manifestations. [2]

Hepato-Pancreatico-Biliary (HPB) tuberculosis is a rare form of abdominal tuberculosis and typically results from hematogenous spread of pulmonary infection rather than occurring as an isolated entity. [2] Isolated Hepatic tuberculosis accounts for about 1% of all hepatic tuberculosis cases, with just over 100 cases reported. [3] [4]Isolated pancreatic tuberculosis is extremely rare, with around 80 case reports identified in the literature. Although Splenic tuberculosis has been reported in up to 4% of splenectomy specimens and is the third most commonly affected organ in miliary tuberculosis, truly isolated splenic involvement is exceedingly rare, with only 23 cases reported in the most recent review of literature. [5, 6, 7, 8]

This article presents a case series of three patients with Isolated HPB and Splenic tuberculosis managed at our centre. All three cases posed significant diagnostic dilemmas and underwent surgical resection. The diagnosis of tuberculosis was confirmed postoperatively by Histopathological Examination (HPE), revealing granulomatous inflammation with caseous necrosis.

Correspondence: C. V. Kantharia
E-mail: kanthariachetan@gmail.com
https://orcid.org/0000-0003-1679-0548
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Cases

Case 1: Hepatobiliary tuberculosis

A 20-year-old male presented with dull, aching pain in the right upper abdomen for the past 4 months. Abdominal examination was unremarkable, and initial blood investigations were within normal limits. An Abdominal Ultrasonography (USG) with Doppler revealed a 7 × 5 × 4 cm heterogeneous lesion with internal vascularity in the right lobe of the liver. Liver function tests were normal (Aspartate Transaminase-AST/Alanine Transaminase-ALT/Alkaline Phosphatase (ALP): 23/36/156 Units/litre respectively), and viral markers (HBsAg and Anti-HCV) were negative. Chest X-ray did not show any abnormalities.

Contrast-enhanced computed tomography (CECT) of the abdomen and pelvis revealed a multiloculated, peripherally enhancing cystic lesion with internal septations, measuring 7 × 7 × 4.5 cm in segments V and VI of the liver. Mild intrahepatic biliary radicle dilatation was also noted. To further characterise the lesion, Contrast-Enhanced Magnetic Resonance Imaging (CE-MRI) of the abdomen was performed. It demonstrated a 7 × 7 × 4.5 cm lesion in segments V and VI of the liver. The lesion was hypo-intense on T1-weighted images and hyper-intense on T2-weighted images, with no post-contrast enhancement. There was no diffusion restriction; however, communication with intrahepatic biliary radicles was evident. Periportal lymphadenopathy was present, with the largest node measuring 1.5 cm.

Tumour markers were within normal limits:- (Alpha-fetoprotein (AFP) 1.1 nanogram/ml, Carbohydrate Antigen 19-9 (CA 19-9) 6.78 International Units/ml, and Carcinoembryonic Antigen (CEA) 0.99 nanogram/ml.)

Based on imaging findings, differential diagnoses included Intrahepatic Cholangiocarcinoma and Biliary Cystadenoma. Ultrasound-guided liver biopsy was non-diagnostic, and the decision was taken to proceed with resectional surgery. The patient was appropriately counselled, and informed consent was obtained. The patient underwent laparotomy, and a non-anatomical resection of the liver lesion was performed under

intraoperative ultrasonography guidance.

Histopathological examination (HPE) revealed the diagnosis of Hepatobiliary tuberculosis. On gross examination, the biliary radicles were filled with caseous material. Microscopy showed dilated bile ducts containing caseous necrotic debris, along with neutrophils, macrophages, Langhans giant cells, and dense plasmacytic infiltration.

The postoperative course was uneventful. The patient was started on Anti-Tubercular Therapy (ATT) according to the National Tuberculosis Elimination Programme (NTEP): Isoniazid (INH) 200 mg, Rifampicin 750 mg, and Ethambutol 1200 mg for 9 months, with regular liver function tests monitoring. At 2-year follow-up, the patient is asymptomatic, and abdominal USG shows a residual lesion measuring 2 × 2 cm.

Case 2: Pancreatic Tuberculosis

A 32-year-old female presented with a low-grade fever and left upper quadrant abdominal pain for over 2 months. She was a known case of chronic Hepatitis B virus (HBV) infection and had been on Entecavir and Tenofovir for the last 10 years. Her recent HBV viral load was <34 copies/mL. General physical and abdominal examinations were unremarkable.

Abdominal USG revealed a 3 × 2 cm hypoechoic lesion in the tail of the pancreas, with associated peripancreatic lymphadenopathy. Blood investigations were within normal limits, including serum amylase and lipase (113/63 Units/L, respectively). Complete blood count showed Haemoglobin of 10 grams/dL, Total leukocyte count (TLC) of 5800 cells/mm^3, neutrophils at 70%, and lymphocytes at 27%. Serum CA 19-9 was normal (7.4 Units/mL), and chest X-ray showed no abnormalities.

CECT of the abdomen showed a well-defined, mildly enhancing hypodense lesion measuring 4 × 3 × 3 cm in the tail of the pancreas, along with calcified peripancreatic lymph nodes.

Endoscopic ultrasonography (EUS) revealed a hypoechoic cystic lesion (3 × 3 cm) in the pancreatic tail and periportal-peripancreatic lymphadenopathy (maximum diameter 2 cm). EUS-guided biopsy from the periportal lymph node was inconclusive. Differential diagnoses considered at this stage included lymphoma, parasitic cyst, or an inflammatory cyst.

As the lesion did not regress after 2 months of observation and the patient continued to be symptomatic, she was counselled for surgical intervention. Informed consent was obtained, and she underwent an open anterior radical antegrade modular pancreatico-splenectomy. The postoperative period was uneventful.

HPE revealed necrotising granulomatous inflammation involving the pancreatic parenchyma and lymph nodes, with Langhans giant cells, lymphocytes, and plasma cells—features consistent with a tuberculous aetiology.

The patient was initiated on Anti-Tubercular Therapy (ATT) with INH, Rifampicin, Ethambutol, and Pyrazinamide for 2 months (intensive phase), followed by INH, Rifampicin, and Ethambutol for 7 months (continuation phase).

At 18 months of follow-up, the patient remains asymptomatic. Apart from a few subcentimetric intra-abdominal lymph nodes, USG of the abdomen shows no significant radiological abnormality.

Case 3: Splenic Tuberculosis

A 47-year-old female presented with left upper abdominal pain for 2 months. On examination, there was tenderness in the left hypochondrium, but no guarding or rigidity was noted. Blood investigations were within normal limits. Chest X-ray was unremarkable.

Abdominal USG revealed a 3 × 4 × 3 cm hypoechoic lesion in the lower pole of the spleen with internal echoes, along with cholelithiasis (1.5 cm stone in the gallbladder neck). Further evaluation with CECT of the abdomen revealed a single 4 × 3.5 × 3 cm hypodense, non-enhancing lesion in the spleen with enhancing internal septations—suggestive of a complex splenic cyst. The pancreas appeared normal, and simple hepatic cysts were also noted.

Magnetic resonance imaging (MRI) of the abdomen revealed a 4 × 3.4 × 3 cm T1- and T2-hypointense lesion in the spleen with internal debris. Cholelithiasis and simple hepatic cysts were also confirmed.

A preoperative diagnosis of a parasitic cyst or granulomatous disease of the spleen was made. The patient was counselled, and informed consent was obtained. She underwent a laparoscopic splenectomy and cholecystectomy. The postoperative course was uneventful, and she was immunised 2 weeks following surgery.

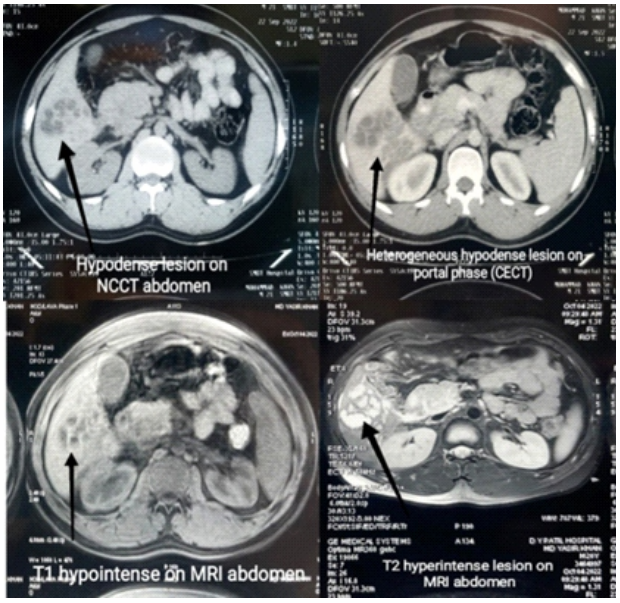

Figure 1: Contrast-enhanced computed tomography and Magnetic resonance imaging pre-operative images of Hepatobiliary Tuberculosis. (A) A hypodense lesion is seen in segments V and VI of the Liver in the non-contrast phase with heterogeneous partial enhancement in the portal phase (top two images). MRI abdomen reveals a T1 hypointense and T2 hyperintense lesion noted in segments V and VI of the liver (lower two images).

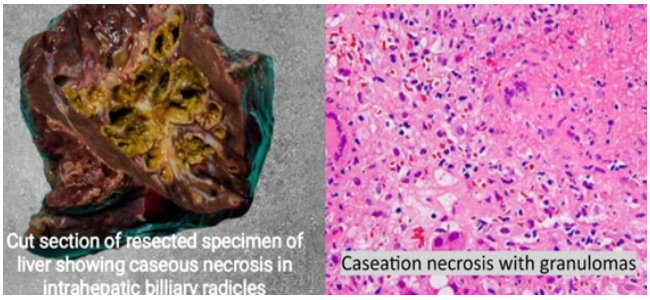

Figure 2: (A) Gross photograph of the resected liver specimen showing caseous necrosis within the intrahepatic biliary radicals, appearing as yellow-white cheesy material replacing the bile duct.
(B) Photomicrograph showing caseation necrosis surrounded by epithelioid cell granulomas and Langhans giant cells, consistent with tuberculous involvement of the liver. (Haematoxylin & Eosin stain, original magnification ×200)

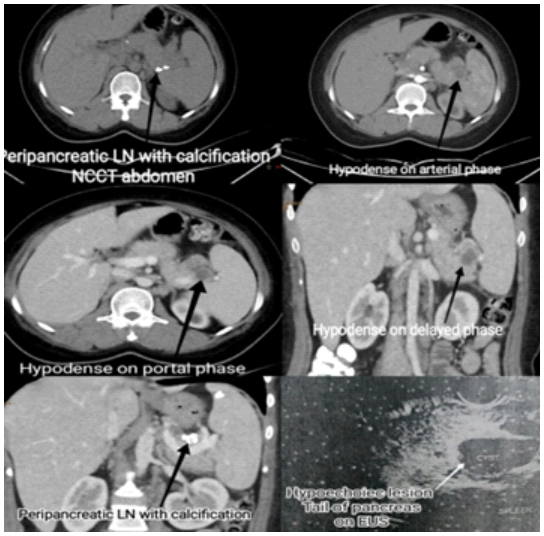

Figure 3: Contrast-enhanced computed Tomography and Endoscopic Ultrasonographic images of Pancreatic tuberculosis mass. (A) Non-contrast images show Peri-pancreatic Lymph node calcification and a hypodense lesion in the tail of the pancreas, which is seen in all the phases (B, C, D). The image shows Peripancreatic Lymph node calcification in a Coronal Section. Image (F) reveals an EUS finding of a hypoechoic lesion in the tail of the pancreas.

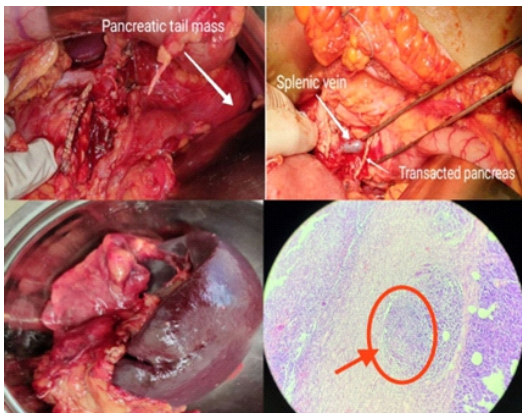

Figure 4: Intra-operative, Gross and Microscopic images of Pancreatic tuberculosis suggestive of granulomatous inflammation within pancreatic parenchyma.
A) Intraoperative photograph showing a mass in the pancreatic tail region.
(B) Dissection of the splenic vein with transection of the pancreas during distal pancreatectomy.
(C) Gross specimen of the resected distal pancreas and spleen.
(D) Microscopic image showing epithelioid cell granuloma with central caseation necrosis, consistent with tuberculous involvement of the pancreas. (Hematoxylin and eosin stain, original magnification ×100)

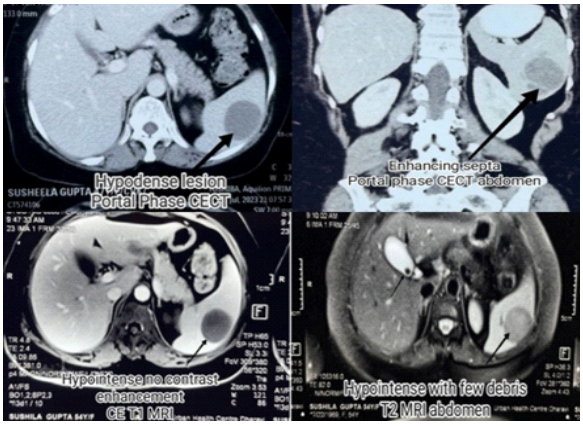

Figure 5: Contrast-enhanced computed tomography and Magnetic resonance imaging pre-operative images of Isolated Splenic Tuberculosis. Images A and B show a CECT abdomen and pelvis showing a hypodense lesion in the lower pole of the spleen with enhancing septa. MRI abdomen showing a T1 and T2 hypointense lesion with a few debris on CE MRI(Image C and D).

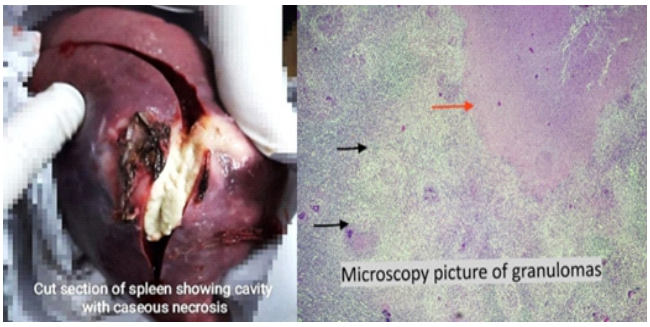

Figure 6: (A) Cut section of the spleen showing a cavity filled with caseous necrotic material.
(B) Microscopic image showing multiple epithelioid cell granulomas with central caseation necrosis, consistent with tuberculous involvement of the spleen. (Hematoxylin and eosin stain, original magnification ×100)

HPE of the spleen revealed caseous necrosis with granulomas composed of Langhans and foreign body-type giant cells, epithelioid cells, lymphocytes, and fibroblasts. The hilar lymph nodes also showed caseating granulomas consistent with tuberculosis. On gross examination, pus was noted in the lesion. It was sent for nucleic acid amplification testing (NAAT), which confirmed *Mycobacterium tuberculosis* (rifampicin-sensitive strain).

The patient was started on ATT with INH, Rifampicin, Ethambutol, and Pyrazinamide for 2 months (intensive phase), followed by INH, Rifampicin, and Ethambutol for 8 months (continuation phase). At 12 months of follow-up, the patient remains asymptomatic.

Table 1: Summary of all 3 cases

	Case 1	Case 2	Case 3
Diagnosis	Hepatobiliary tuberculosis	Pancreatic tuberculosis	Splenic tuberculosis
Age/Sex	20-year-old male	32-year-old female	47-year-old female
Associated medical illness	Nil	HBsAg positivity on treatment	Cholelithiasis
Presenting complaints	Right upper abdominal pain for 4 months	Left upper abdominal pain with low-grade fever for 2 months	Left upper abdominal pain for 2 months
Imaging features	7x7x4.5cm lesion in segments V and VI of the liver	4x3x3 cm lesion in the tail of the pancreas with peripancreatic Lymphadenopathy	3x4x3cm lesion in the lower pole of the spleen
Ultrasonography	Heterogenous	Hypoechoic lesion	Hypoechoic lesion
CECT abdomen and pelvis	Peripherally enhancing lesion with internal septations and intrahepatic biliary radicle dilatation.	Hypodense lesion with mild enhancement with calcified peripancreatic lymphadenopathy	Hypodense non-enhancing with enhancing internal septations
CE MRI of abdomen	T1 hypointense and T2 hyperintense with no contrast enhancement	Not applicable	T1 and T2 hypointense lesions with debris
EUS	Not applicable	Hypoechoic lesion	Not applicable
Surgery performed	Non-anatomical resection of liver lesion	Anterior Retrograde Modular Pancreatico-splenectomy	Laparoscopic splenectomy with cholecystectomy
Final Diagnosis	Hepatobiliary Tuberculosis	Pancreatic Tuberculosis	Splenic Tuberculosis
Duration ATT			
Intensive Phase	Total 9 months of therapy	2 months	2 months
Continuation Phase		7 months	8 months
Total	9 months	9 months	10 months
Follow up	24 months	18 months	12 months

Discussion

Abdominal tuberculosis can be classified into intestinal, peritoneal, hepatobiliary, pancreatic, splenic-lymph nodal, and other rare variants. Rare variants include oesophageal, gastro-duodenal, and perianal tuberculosis, among others.[2] Hepatobiliary tuberculosis is seen in 50-80% cases of disseminated tuberculosis, but primary hepatobiliary tuberculosis occurs in only around 1% of patients. According to Kandasamy et al (2018)[3] Fewer than 100 cases of isolated hepatic tuberculosis have been reported in the literature. Thereafter, the largest two case series have reported 14 [4] and 5 cases each [9].

Various classifications exist but National Tuberculosis Elimination Programme (NTEP) classifies Hepatobiliary TB based on clinicopathological presentation into Miliary TB (Multiple miliary tubercles), Granulomatous hepatitis (Tuberculomas forming aggregates of 1–4 cm.), Nodular (Atypical heterogeneous abscess/solid pseudo-tumour), Ductal (Focal/diffuse lesions affecting ducts), and Nodal (Nodal mass/obstruction at the porta). [2]

The pathogenesis of Hepatobiliary tuberculosis includes hematogenous spread of miliary tuberculosis, in which the pulmonary infection spreads to the liver via the hepatic artery.

The portal vein is another point of access, and lymphatic spread has also been proposed. [10] However, in its isolated form, it begins as primary intestinal tuberculosis, i.e., an ulcer in the small bowel wall. Mycobacteria in the bowel wall gain access to the portal vein and seed into the hepatic parenchyma, while the bowel lesion heals. The primary ulcer has completely resolved and manifests as isolated hepatobiliary tuberculosis. [9]

The hepatic parenchyma has a rich, extensive blood supply, making it an ideal substrate for mycobacteria. The presence of abundant reticuloendothelial cells leads to the formation of granulomas, which are usually seen in the vicinity of portal triads. [10] However, the low oxygen tension in the liver negatively affects the mycobacterial growth. [4]

Hepatobiliary tuberculosis is twice as common in men. [10] It presents with abdominal pain (the most common symptom), jaundice, fever, anorexia, weight loss, and hepatomegaly in around 70% of patients. [2] Laboratory investigations are usually non-specific; however, an increase in Serum ALP and gamma-glutamyl transferase (GGT) may be reported. [4] Low serum albumin levels and hypergammaglobulinemia have also been described. [3] A chest x-ray is normal in isolated Hepatobiliary tuberculosis, as was the case in our patient.

Hepatobiliary involvement in tuberculosis can be milliary, tuberculoma, abscess or biliary type. Each of these types will have different radiological appearances. [10] A combination of USG and CECT of the abdomen should be used for initial evaluation. [11] [12]. Our patient presented with a tuberculoma.

On USG of the abdomen, they can appear as hypo- or hyperechoic lesions. On CECT abdomen, tuberculomas appear as hypodense lesions with central low density corresponding to necrosis with occasional peripheral rim enhancement (which corresponds to granulation tissue) [3], [10]. Yu et al. [13] have classified Hepatobiliary tuberculosis based on CECT findings into Parenchymal, Tubercular cholangitis (associated with duct wall thickening), and Serohepatic type (thickened subcapsule, known as frosted liver). The parenchymal type has been further classified into miliary (multiple nodules <2cm), nodular (multiple nodules >2cm), and mixed (miliary macronodular type). MRI has higher diagnostic accuracy for Hepatobiliary tuberculosis than CECT. Nodular lesions are typically T1 hypointense and T2 hyperintense. In the dynamic phase, they tend to exhibit rim or heterogeneous enhancement. The nodules may show slight diffusion restriction on diffusion-weighted images. [13]

Biopsy can be considered with USG or CECT guidance, but only 23% of patients with isolated HPB tuberculosis are diagnosed preoperatively. Hence, a high index of suspicion is essential, especially in young patients in high-prevalence areas. [2], [11], [12]

The 18 FDG-PET CT (Fluoro-2-deoxy-D-Glucose Positron Emission Tomography Computed Tomography) scan is not useful for distinguishing tuberculomas from malignancies, as both lesions are FDG avid; however, the miliary type of involvement doesn't show any uptake. [4], [10]

In isolated Hepatobiliary tuberculosis, diagnosis is based on demonstrating caseating granulomas on HPE, most often postoperatively. Only 68% of cases demonstrate typical histopathological features of tuberculosis. However, granulomas may also be seen in histoplasmosis and sarcoidosis. [2] [12], [14] . The methods for diagnosing Hepatobiliary tuberculosis preoperatively include USG, CECT, or EUS-guided liver biopsy. Diagnostic Laparoscopy and biopsy of the lesion can also be considered if there is a strong suspicion of granulomatous disease. The biopsied tissue or aspirated pus may be sent for ZN (Ziehl-Neelsen) staining and microscopy, Mycobacterial culture, and NAAT (Nucleic Acid Amplification Test). However, sensitivity varies across studies. [2] ZN staining is positive in 40% of tissue samples. [9] The AFB (Acid Fast Bacilli) culture can grow mycobacteria from tissue samples in only 0-45% of cases, and results are available only after 4-8 weeks due to mycobacteria's long doubling time. Although NAAT has a sensitivity of 88% in tissue samples, its sensitivity in liver biopsies falls to 53%. [2] The MTB-PCR (Mycobacterium Tuberculosis- Polymerase chain reaction) has sensitivity of 82%[9]but this facility was unavailable at our centre. Saluja et al. reported 11 cases of isolated hepatobiliary tuberculosis [12]. In this series, which also included pancreatic tuberculosis, 80% of patients were diagnosed post-operatively as the preoperative working diagnosis was malignancy.

Hepatobiliary tuberculosis can present with multiple complications, such as tubercular cholangitis and biliary stricture due to erosion into the biliary tree. Due to caseous necrosis, hepatic abscesses or liver pseudo-tumours can form. Compression of the portal vein by tubercular lymph nodes may result in portal hypertension, and pseudo-cirrhosis of the liver can occur due to scarring of multiple tubercles. Miliary form may lead to acute liver failure. These complications are

more commonly seen in the miliary form than in the isolated form. [15]

The diagnostic dilemma in isolated Hepatobiliary TB is such that most often it is misdiagnosed pre-operatively as malignancy, like Hepatocellular Carcinoma or Intrahepatic Cholangiocarcinoma. [16] The other differentials to consider are Sarcoidosis, Non-Hodgkin's lymphoma, Brucellosis, and Histoplasmosis. [4] The differentials that need to be mentioned in the current era are Post-COVID-19 Cholangiopathy, CMV (Cytomegalovirus) infection, and hemophagocytic lymphohistiocytosis. [16]

Treatment is with Anti-Tubercular Therapy (ATT), which is usually prescribed for 6 months but can be extended to 1 year. Selection of Anti-tubercular drugs should be based on the Child-Turcot-Pugh (CTP) and Model for End-Stage Liver Disease(MELD) scores. Pyrazinamide is usually avoided. [2] (Treatment details in Table 2). This therapy should be instituted irrespective of the timing of diagnosis, i.e., whether the diagnosis is established by biopsy or after post-resectional surgery.

Although a diagnostic dilemma, tuberculous hepatic abscess and failed endoscopic therapy for biliary strictures are the indications for surgery in these patients [2], Xing et al have established the role of hepatic segmentectomy in hepatic tubercular pseudotumours [17]. In this series, of the total 8 patients who underwent segmentectomy for tuberculosis, 3 patients were diagnosed pre-operatively based on CECT-guided biopsy. There has been a case report of spontaneous rupture of isolated hepatic tuberculosis requiring urgent right hepatectomy. [18] If an isolated hepatic lesion, as reported in our case, with the largest dimension of 7cm, is diagnosed preoperatively or intraoperatively by biopsy, whether Anti-Tubercular therapy alone would suffice without the need for surgical resection is a research question in itself.

Pancreatic tuberculosis was first reported by Harles in 1912. [19] Pancreatic tuberculosis is a rare entity, as the pancreas is considered a hostile environment for mycobacteria due to the anti-mycobacterial activity of pancreatic lipase and deoxyribonucleases. [20].Therefore, Pancreatic involvement even in miliary tuberculosis is 2.1-4.7% [2]. Pancreatic tuberculosis can be classified as a part of miliary tuberculosis, secondary involvement in tuberculosis of retroperitoneal lymph nodes and isolated pancreatic tuberculosis. [19].There have been only 73 cases of isolated pancreatic tuberculosis reported in the literature until 2002 [7] An electronic search reported 9 more cases to date.

Table 2: Treatment strategy in Hepatobiliary Tuberculosis based on CTP and MELD scores according to the National Tuberculosis Elimination Programme in India

CTP score	MELD score	Treatment strategy
Child A	Less than 18	9 months of Isoniazid, Ethambutol and Rifampicin OR 2 months of Isoniazid, Ethambutol, Rifampicin and Streptomycin followed by 6 months of Isoniazid, Ethambutol and Rifampicin The therapy can include only two potential hepatotoxic drugs, as compared to standard therapy, which includes 3 of them.
Child B	18-25	2 months of Rifampicin, Ethambutol and Streptomycin and 10 months of Rifampicin and Ethambutol. OR 9-12 months of Rifampicin, Levofloxacin and Ethambutol with or without Streptomycin Regimen containing only one hepatotoxic drug, where Rifampicin is preferred over Isoniazid
Child C	More than 25	18-24 months of Streptomycin, Amikacin and Levofloxacin. No potential hepatotoxic drugs should be given.

Each of the three forms of pancreatic tuberculosis has a different pathogenesis. In miliary tuberculosis there is haematogenous spread from active pulmonary tuberculosis. The pancreas can also be involved secondarily due to erosion by infected, contiguous retroperitoneal lymph nodes. An isolated form of pancreatic tuberculosis is secondary to intestinal infection, with resolution of the primary lesion. [21] Reactivation of a tubercular focus in the pancreas can occur from steroid therapy, alcoholism, acute pancreatitis or surgery. [2] Pancreatic tuberculosis most likely involves the head and uncinate due to its relatively rich blood supply. [19]

According to a systematic review of 166 patients, which involved all 3 forms, pancreatic tuberculosis is more commonly seen in males (62%), with an average age of 41 years. The most common risk factor was HIV (Human Immunodeficiency Virus) positivity, followed by alcohol abuse, and a previous history of tuberculosis was seen in only 7% of patients. [22]

Patients present with abdominal pain (66%), fever and night sweats (52%), anorexia and weight loss (46%), malaise (28%), back pain (20%) and jaundice (15%).On examination, there may be epigastric tenderness or a palpable lump. [21] There may be various complications, the most common being gastrointestinal bleeding due to splenic vein thrombosis, which may be the initial presentation. Other complications like arterial pseudo-aneurysm with duodenal bleeding, secondary diabetes mellitus, severe iron deficiency anaemia, acute or chronic pancreatitis, etc., may also be seen in these

patients. [19] A pancreatic head mass may lead to obstructive jaundice or portal vein thrombosis. [21] Pancreatic exocrine insufficiency has been reported in only one patient in a 19th-century case report, but the relationship between pancreatic tuberculosis and endocrine insufficiency is two-fold. Endocrine insufficiency may be considered a risk factor for pancreatic tuberculosis, and pancreatic parenchymal destruction due to tuberculosis can lead to diabetes mellitus. [22]

Serum Amylase (26%) and lipase (31%) are of no diagnostic value and are elevated in only a few cases. Anaemia may indicate vascular complications, and conjugated hyperbilirubinemia with elevated ALP suggests obstructive jaundice. This results from a pancreatic head mass compressing the intrapancreatic common bile duct or from extrinsic compression by hilar or peri-choledochal enlarged lymph nodes. [4] [23]. In isolated cases, the chest X-ray can be normal.

The usual presentation is as a pancreatic mass diagnosed on cross-sectional imaging, most commonly in the head (59% of cases) and in the tail (13% of cases). Peripancreatic lymphadenopathy is a consistent feature, seen in around 70% of cases. [22]

In addition to a mass, it can present as a cystic lesion or an abscess. [21] USG of the abdomen may demonstrate a bulky pancreas or a hypoechoic solid mass or cystic lesion. [19] CECT abdomen is the preferred modality for diagnosis. On CECT, a pancreatic tubercular mass may present as either solid or cystic. Calcifications are associated with 50–70% of cases. Pancreatic tuberculosis often masquerades as malignancy when there is vascular involvement. However, an undilated pancreatic duct and large peripancreatic lymph nodal masses with calcification point towards tuberculosis. [2] [24] MRI of the abdomen shows T1 iso- to hypointense and T2-heterogeneous signal-intensity lesions with post-contrast rim enhancement. EUS plays a role in both diagnosis and as a guide for tissue sampling. EUS-FNA (Fine Needle Aspiration) has a diagnostic yield of up to 76% in pancreatic tuberculosis. However, it was non-diagnostic in our patient. [2], [19] Hemanta Kumar et al [19] have reported two cases of isolated pancreatic tuberculosis, which were diagnosed based on EUS-FNA showing epitheloid granuloma and Langhans giant cells. They could avoid surgical resection, and patients completely recovered within 6 months of ATT.

On 18-FDG PET/CT, pancreatic TB can mimic malignancy, and hence it is not a good diagnostic tool; however, a decrease SUVmax (Maximum Standardised Uptake Value) can be considered a positive response to treatment. [19]

Although Serum amylase and lipase have limited diagnostic value in pancreatic tuberculosis, the tuberculin test and gamma interferon assay have shown significant value in diagnostic dilemmas. The tuberculin skin test, a cheap investigation, is positive in 70% of individuals and can therefore be of great significance in suspected pancreatic tuberculosis. [22]

In a systematic review [22] which included all forms of pancreatic tuberculosis 55% patients were subjected to resectional surgery in view of misdiagnosis as malignancy or due to diagnostic dilemma. In a diagnostic dilemma, patients are often subjected to resection, such as a distal pancreatectomy or a Whipple's procedure, and the diagnosis is established only after histopathological examination, as was the case in our patient. [2]

All patients should undergo ATT with an intensive phase of 2 months (INH, Rifampicin, Ethambutol and Pyrazinamide) and a continuation phase of 4 months (INH, Rifampicin and Ethambutol). Patients should be reassessed at 2 months and after 6 months of therapy. The ATT can be continued for up to 1 year, and this decision should be made on a case-by-case basis. [2]

Splenic Tuberculosis:

Although the spleen is commonly affected in disseminated intra-abdominal tuberculosis, isolated splenic tuberculosis is rarely seen. Coley first described TB of the spleen in 1846, referring to an “enlarged spleen secondary to tuberculosis with absent or limited involvement of other organs.” [25] Around 13 cases of isolated splenic tuberculosis have been reported in 2021 by Grover et al. [26] However, a recent review reported 23 cases. [8]

Splenic tuberculosis is usually seen in immunocompromised patients, with HIV infection being the most common risk factor. [27] In the largest series of isolated Splenic tuberculosis, only 3 out of 23 patients were immuno compromised. [8] The patient with isolated splenic tuberculosis reported by us was immunocompetent and had no history of tuberculosis.

Splenic tuberculosis can present with a wide spectrum of symptoms, including left upper abdominal pain, fever, anorexia, nausea, vomiting and weight loss. On physical examination, splenomegaly is noted in around 50% of

patients with isolated splenic tuberculosis. It can also present with complications like hypersplenism, portal hypertension, and splenic rupture, as the first manifestation, which adds to the complexity of diagnosis. [8],[27]

Abdominal USG is the first radiological investigation, and splenic tubercular involvement may be seen in the form of a miliary pattern, nodular pattern, abscess, calcification or a combination of these findings. [25]In isolated cases, around 50% patients present with multiple hypoechoic lesions. [8].

CECT of the abdomen helps better delineate these lesions. [26] On CECT, these lesions appear as splenic hypodense areas with clear boundaries and heterogeneous enhancement. [27]Splenic involvement can be micronodular or macronodular. Small nodules are usually missed on USG but are seen as multiple hypodense lesions on CECT. Large nodules are hypoechoic on USG and hypodense on CECT. [28] Calcifications may be present. In our patient, the presence of enhancing septa led to a diagnostic dilemma. Malignant lymphoma, metastatic tumour, acute leukaemia, haemangioma, or even infectious diseases may present as splenic hypodense lesions on CECT and hypoechoic lesions on USG. [27] On MRI, a solitary splenic tuberculoma is described as a hypointense or isointense lesion on T1-weighted images, with a hypointense lesion along with a few hyperintense areas in T2-weighted images. On administering contrast, there may be peripheral rim enhancement. The focal splenic lesions exhibit variability in intensity and enhancement patterns on contrast administration. The hypointensity on T2-weighted images rules out neoplastic and other inflammatory lesions. [29] The role of 18 FDG-PET is not well established, but it is usually considered when a patient presents with pyrexia of unknown origin. Rao et al have reported a case of isolated splenic tuberculosis where the lesions were missed on CECT as well as on MRI and detected only on FDG-PET as multiple FDG-avid focal lesions. [30]

Erythrocyte sedimentation rate may be elevated, and bone marrow may demonstrate hypercellularity in 25% of patients. [28] A chest X-ray should be done to rule out pulmonary tuberculosis.

Preoperative diagnosis may provide an opportunity for organ preservation; this can be achieved by USG-guided aspiration of pus and demonstration of acid-fast bacilli, or by demonstrating granulomatous inflammation on core needle biopsy. [28]. The utility of Fine Needle Aspiration (FNA) versus Core Needle Biopsy (CNB) has been debated in the literature, with FNA having a lower risk of bleeding and CNB yielding higher rates. [8]

The aspirated pus can be subjected to ZN staining, MTB (Mycobacterium Tuberculosis) culture, and MTB gene testing. The core biopsy undergoes HPE to assess for granulomatous disease. A wide range of sensitivity and specificity values for each of these tests has been documented in the literature; however, performing multiple tests improves yield. [25]

About 40% of patients with isolated disease are diagnosed after splenectomy based on HPE. Splenectomy is indicated in a diagnostic dilemma, complication of splenic tubercular abscess or inadequate response to ATT. [8]

ATT is advised in both scenarios, irrespective of whether the patient was diagnosed on aspiration of pus/guided biopsy or on HPE post-splenectomy. ATT should be continued for 6 months and may be extended to 9 months or 1 year on a case-by-case basis. A 6-month therapy for abdominal tuberculosis includes 2 months of intensive phase with INH, Rifampicin, Ethambutol and Pyrazinamide, followed by continuation phase with INH, Rifampicin and Ethambutol. [2]

At the initiation of ATT, some patients may experience worsening of symptoms, known as Paradoxical Reaction (PR). This presents with fever, lymphadenitis and pulmonary

Table 3: Dosage of Drugs used in Antitubercular Therapy: [32]

Drug	Dosage according to weight for adults	Dose for 60kg according to fixed dose combinations	Maximum dose for the paediatric age group per day
Isoniazid	4-6 mg/kg	300 mg	300 mg
Rifampicin	8-12 mg/kg	600 mg	600 mg
Pyrizinamide	20-30 mg/kg	1600 mg	2000 mg
Ethambutol	12-18 mg/kg	1100 mg	1500 mg
Streptomycin	15-20 mg/kg	NA	
Pyridoxine is to be given at 10 mg per day			

symptoms. The incidence is 2.4% in HIV negative individuals. There are two hypotheses: the reconstitution of the host immune response after the initiation of ATT and a hypersensitivity reaction to antigens released by dying tubercular bacilli. PR is fortunately self-limiting and usually does not require steroids or discontinuation of ATT. [25] [31]

Table 4: Dosage of second-line drug therapy commonly used in HPB and Splenic tuberculosis: [33]

Drug	16-29 kg	30-45 kg	46-70 kg	>70 kg
Amikacin	500 mg	750 mg	750 mg	1000 mg
Levofloxacin	250 mg	750 mg	1000 mg	1000 mg
Linezolid	300 mg	600 mg	600 mg	600 mg

Conclusion

While evaluating intra-abdominal mass lesions in high-prevalence areas, one should consider isolated HPB or splenic tuberculosis among the differential diagnoses. A high index of suspicion and imaging-guided FNA/biopsy will help in resolving the diagnostic puzzle. Pre-operative diagnosis offers a good chance of organ preservation, as otherwise the patient may undergo surgery, with the diagnosis established only on postoperative histopathological examination.

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Learning Points:

- Isolated HPB and splenic tuberculosis should be considered in patients with mass forming lesions in areas of high prevalence
- Preoperative diagnosis may provide opportunity in organ preservation
- Whether the patient undergoes resectional surgeries or not, they have to complete the course of anti-tubercular therapy
- The guideline of Anti-tubercular therapy in India are regularly updated under NTEP and with current guidelines broadly classifies the patients into drug sensitive and drug resistant