

# World Journal of *Clinical Cases*

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## Contents

Thrice Monthly Volume 9 Number 30 October 26, 2021

### REVIEW

- 8953** Endothelial progenitor cells and coronary artery disease: Current concepts and future research directions  
*Xiao ST, Kuang CY*

### MINIREVIEWS

- 8967** Regulation of bone metabolism mediated by  $\beta$ -adrenergic receptor and its clinical application  
*Zhong XP, Xia WF*
- 8974** Tricuspid valve endocarditis: Cardiovascular imaging evaluation and management  
*Fava AM, Xu B*

### ORIGINAL ARTICLE

#### Case Control Study

- 8985** Novel application of multispectral refraction topography in the observation of myopic control effect by orthokeratology lens in adolescents  
*Ni NJ, Ma FY, Wu XM, Liu X, Zhang HY, Yu YF, Guo MC, Zhu SY*

#### Retrospective Cohort Study

- 8999** Uncertainty in illness and coping styles: Moderating and mediating effects of resilience in stroke patients  
*Han ZT, Zhang HM, Wang YM, Zhu SS, Wang DY*

#### Retrospective Study

- 9011** Development and validation of a prognostic nomogram model for Chinese patients with primary small cell carcinoma of the esophagus  
*Zhang DY, Huang GR, Ku JW, Zhao XK, Song X, Xu RH, Han WL, Zhou FY, Wang R, Wei MX, Wang LD*
- 9023** Preliminary establishment of a spinal stability scoring system for multiple myeloma  
*Yao XC, Shi XJ, Xu ZY, Tan J, Wei YZ, Qi L, Zhou ZH, Du XR*
- 9038** Effect of intrauterine perfusion of granular leukocyte-colony stimulating factor on the outcome of frozen embryo transfer  
*Zhu YC, Sun YX, Shen XY, Jiang Y, Liu JY*
- 9050** "An integrated system, three separated responsibilities", a new fever clinic management model, in prevention and control of novel coronavirus pneumonia  
*Shen J, He Q, Shen T, Wu ZQ, Tan MM, Chen YL, Weng Q, Nie LM, Zhang HF, Zheng B, Zhang J*

**Clinical Trials Study**

- 9059** Single dose dexamethasone prophylaxis of postembolisation syndrome after chemoembolisation in hepatocellular carcinoma patient: A randomised, double-blind, placebo-controlled study

*Sainamthip P, Kongphanich C, Prasongsook N, Chirapongsathorn S*

**Observational Study**

- 9070** Serum calcium, albumin, globulin and matrix metalloproteinase-9 levels in acute cerebral infarction patients

*Zhong TT, Wang G, Wang XQ, Kong WD, Li XY, Xue Q, Zou YA*

**SYSTEMATIC REVIEWS**

- 9077** Neoadjuvant radiotherapy dose escalation for locally advanced rectal cancers in the new era of radiotherapy: A review of literature

*Delishaj D, Fumagalli IC, Ursino S, Cristaudo A, Colangelo F, Stefanelli A, Alghisi A, De Nobili G, D'Amico R, Cocchi A, Ardizzoia A, Soatti CP*

**META-ANALYSIS**

- 9090** Clinical significance of breast cancer susceptibility gene 1 expression in resected non-small cell lung cancer: A meta-analysis

*Gao Y, Luo XD, Yang XL, Tu D*

**CASE REPORT**

- 9101** Particular tumor of the pancreas: A case report

*Zhu MH, Nie CF*

- 9108** Dynamic changes in the radiologic manifestation of a recurrent checkpoint inhibitor related pneumonitis in a non-small cell lung cancer patient: A case report

*Tan PX, Huang W, Liu PP, Pan Y, Cui YH*

- 9114** Spontaneous rupture of a mucinous cystic neoplasm of the liver resulting in a huge biloma in a pregnant woman: A case report

*Kośnik A, Stadnik A, Szczepankiewicz B, Patkowski W, Wójcicki M*

- 9122** Diagnosis and laparoscopic excision of accessory cavitated uterine mass in a young woman: A case report

*Hu YL, Wang A, Chen J*

- 9129** Unusual cervical foreign body - a neglected thermometer for 5 years: A case report

*Yang L, Li W*

- 9134** Long-term survival of a patient with pancreatic cancer and lung metastasis: A case report and review of literature

*Yang WW, Yang L, Lu HZ, Sun YK*

- 9144** Synchronous diagnosis and treatment of acute myeloid leukemia and chronic lymphocytic leukemia: Two case reports

*Chen RR, Zhu LX, Wang LL, Li XY, Sun JN, Xie MX, Zhu JJ, Zhou D, Li JH, Huang X, Xie WZ, Ye XJ*

- 9151** Conversion therapy of hepatic artery ligation combined with transcatheter arterial chemoembolization for treating liver cancer: A case report  
*Feng GY, Cheng Y, Xiong X, Shi ZR*
- 9159** Hemophagocytic lymphohistiocytosis secondary to composite lymphoma: Two case reports  
*Shen J, Wang JS, Xie JL, Nong L, Chen JN, Wang Z*
- 9168** Fatal visceral disseminated varicella-zoster virus infection in a renal transplant recipient: A case report  
*Wang D, Wang JQ, Tao XG*
- 9174** Choriocarcinoma misdiagnosed as cerebral hemangioma: A case report  
*Huang HQ, Gong FM, Yin RT, Lin XJ*
- 9182** Rapid progression of colonic mucinous adenocarcinoma with immunosuppressive condition: A case report and review of literature  
*Koseki Y, Kamimura K, Tanaka Y, Ohkoshi-Yamada M, Zhou Q, Matsumoto Y, Mizusawa T, Sato H, Sakamaki A, Umezu H, Yokoyama J, Terai S*
- 9192** Temporary pacemaker protected transjugular intrahepatic portosystemic shunt in a patient with acute variceal bleeding and bradyarrhythmia: A case report  
*Yao X, Li SH, Fu LR, Tang SH, Qin JP*
- 9198** Recurrent pyogenic liver abscess after pancreatoduodenectomy caused by common hepatic artery injury: A case report  
*Xie F, Wang J, Yang Q*
- 9205** Transient ventricular arrhythmia as a rare cause of dizziness during exercise: A case report  
*Gao LL, Wu CH*
- 9211** Successful management of infected right iliac pseudoaneurysm caused by penetration of migrated inferior vena cava filter: A case report  
*Weng CX, Wang SM, Wang TH, Zhao JC, Yuan D*
- 9218** Anterior abdominal abscess - a rare manifestation of severe acute pancreatitis: A case report  
*Jia YC, Ding YX, Mei WT, Xue ZG, Zheng Z, Qu YX, Li J, Cao F, Li F*
- 9228** Monteggia type-I equivalent fracture in a fourteen-month-old child: A case report  
*Li ML, Zhou WZ, Li LY, Li QW*
- 9236** Diagnosis and treatment of primary pulmonary enteric adenocarcinoma: Report of Six cases  
*Tu LF, Sheng LY, Zhou JY, Wang XF, Wang YH, Shen Q, Shen YH*
- 9244** Choroidal metastatic mucinous abscess caused by *Pseudomonas aeruginosa*: A case report  
*Li Z, Gao W, Tian YM, Xiao Y*
- 9255** Diagnosis and treatment of acute graft-versus-host disease after liver transplantation: Report of six cases  
*Tian M, Lyu Y, Wang B, Liu C, Yu L, Shi JH, Liu XM, Zhang XG, Guo K, Li Y, Hu LS*

- 9269** Hepatic portal venous gas without definite clinical manifestations of necrotizing enterocolitis in a 3-day-old full-term neonate: A case report  
*Yuan K, Chen QQ, Zhu YL, Luo F*
- 9276** Emergence of lesions outside of the basal ganglia and irreversible damage to the basal ganglia with severe  $\beta$ -ketothiolase deficiency: A case report  
*Guo J, Ren D, Guo ZJ, Yu J, Liu F, Zhao RX, Wang Y*
- 9285** Skeletal muscle metastasis with bone metaplasia from colon cancer: A case report and review of the literature  
*Guo Y, Wang S, Zhao ZY, Li JN, Shang A, Li DL, Wang M*
- 9295** Biopsy-confirmed fenofibrate-induced severe jaundice: A case report  
*Lee HY, Lee AR, Yoo JJ, Chin S, Kim SG, Kim YS*
- 9302** Missense mutation in *DYNC1H1* gene caused psychomotor developmental delay and muscle weakness: A case report  
*Ding FJ, Lyu GZ, Zhang VW, Jin H*
- 9310** Isolated hepatic tuberculosis associated with portal vein thrombosis and hepatitis B virus coinfection: A case report and review of the literature  
*Zheng SM, Lin N, Tang SH, Yang JY, Wang HQ, Luo SL, Zhang Y, Mu D*

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# Isolated hepatic tuberculosis associated with portal vein thrombosis and hepatitis B virus coinfection: A case report and review of the literature

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**Author contributions:** Zheng SM designed the report; Mu D, Zhang Y, Tang SH, and Luo SL collected the patient's clinical data; Wang HQ provided and analyzed the microscopy images; Yang JY and Wang HQ analyzed the ultrasound and CT images; Zheng SM and Lin N wrote the paper; and all authors have read and approved the final version to be published.

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## Abstract

### BACKGROUND

While tuberculosis (TB) itself is a common disease, isolated TB of the liver is a rare entity. Tubercular involvement of the liver is more commonly a part of a disseminated disease of the hepatic parenchyma. In contrast, isolated hepatic TB spread through the portal vein from the gastrointestinal tract is seldom encountered in clinical practice, with only a few sporadic cases and short series available in the current literature. Vascular complications, such as portal vein thrombosis (PVT), have rarely been reported previously.

### CASE SUMMARY

A 22-year-old man was hospitalized with complaints of a 3-mo history of fever and weight loss of approximately 10 kg. He had a 10-year hepatitis B virus (HBV) infection in his medical history. Contrast-enhanced computed tomography (CECT) confirmed hepatosplenomegaly, with hypodensity of the right lobe of the liver and 2.1 cm thrombosis of the right branch of the portal vein. A liver biopsy showed epithelioid granulomas with a background of caseating necrosis. Ziehl-Nelson staining showed acid-fast bacilli within the granulomas. The patient was diagnosed with isolated hepatic TB with PVT. Anti-TB therapy (ATT), including isoniazid, rifampentine, ethambutol, and pyrazinamide, was administered. Along with ATT, the patient was treated with entecavir as an antiviral medication



Checklist (2016).

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against HBV and dabigatran as an anticoagulant. He remained asymptomatic, and follow-up sonography of the abdomen at 4 mo showed complete resolution of the PVT.

## CONCLUSION

Upon diagnosis of hepatic TB associated with PVT and HBV coinfection, ATT and anticoagulants should be initiated to prevent subsequent portal hypertension. Antiviral therapy against HBV should also be administered to prevent severe hepatic injury.

**Key Words:** Hepatic tuberculosis; Portal vein thrombosis; Hepatitis B virus; Case report

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**Core Tip:** Tubercular involvement of the liver is more commonly a part of a disseminated disease of the hepatic parenchyma. In contrast, isolated hepatic tuberculosis (TB) spread through the portal vein from the gastrointestinal tract is seldom encountered in clinical practice. Vascular complications, such as portal vein thrombosis, have rarely been reported previously. Patients with hepatitis B virus (HBV) and TB coinfection needing anti-TB therapy may have more risks for hepatic injury. We hereby describe a case with an unusual appearance of local hepatic TB associated with portal vein thrombosis and HBV coinfection who was successfully treated with anti-TB therapy, anti-coagulants, and antiviral treatment against HBV.

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## INTRODUCTION

*Mycobacterium tuberculosis* usually infects the lungs, resulting in pulmonary tuberculosis (TB), but it can infect almost any organ in the body, causing extrapulmonary infection. Hepatic TB is an extrapulmonary manifestation of an active infection. Hepatic TB can be broadly divided into two types, the more common miliary type and the less common local type, each of which can be further divided into diffuse and nodular subtypes. The miliary or disseminated type characterized by diffuse hepatic involvement is caused by the hematogenous spread of the *Mycobacterium tuberculosis* by the hepatic artery, generally from the lungs. Such patients usually show evidence of miliary pulmonary TB, as well[1]. Isolated or local hepatic TB is the rarest form of local hepatic TB, which occurs through *Mycobacterium tuberculosis* spread through the portal vein from the gastrointestinal tract[2,3]. Among reported hepatic TB cases, the miliary form accounts for 79%, while local hepatic TB accounts for 21% [4]. Biliary TB is another form of TB in the liver and is considered rare[2,5].

Local hepatic TB associated with portal vein thrombosis (PVT) and hepatitis B virus (HBV) infection poses challenges in both diagnosis and treatment and has not, to our knowledge, been reported previously. We hereby describe a case with an unusual appearance of local hepatic TB associated with PVT and HBV coinfection.

## CASE PRESENTATION

### Chief complaints

A 22-year-old man was admitted to our hospital with a 3-mo history of fever and weight loss of approximately 10 kg.



### **History of present illness**

The patient's symptoms started 3 mo ago with chronic fever and progressive weight loss.

### **History of past illness**

The patient had a 10-year HBV infection in his medical history. He had never taken antiviral treatment against HBV.

### **Personal and family history**

This patient had no special personal and family history.

### **Physical examination**

On examination, the patient's temperature was 39.5 °C, heart rate was 94 beats per minute, respiratory rate was 17 breaths per minute, and blood pressure was 100/70 mmHg, without any other pathological signs.

### **Laboratory examinations**

A laboratory workup revealed a hepatic injury pattern, with alanine aminotransferase of 137 IU/L (normal range, 9-60 IU/L), aspartate aminotransferase of 95 IU/L (15-45 IU/L), alkaline phosphatase of 290 IU/L (45-125 IU/L), and  $\gamma$ -glutamyltransferase of 172 IU/L (10-60 IU/L). The hepatitis panel showed positivity for hepatitis B surface antigen (HBsAg), hepatitis B e antigen (HBeAg), and hepatitis B core antibody. HBV-DNA level was  $3.496 \times 10^7$  IU/mL ( $< 2.0 \times 10^2$  IU/mL). C-reactive protein was 46 mg/L (0-3 mg/L). Procalcitonin was 2.37 ng/mL (0-0.05 ng/mL). The purified protein derivative skin test, autoimmune, HIV, and ESR tests were within normal limits.

### **Imaging examinations**

Abdominal ultrasound showed that the liver was enlarged, with a span of 15.2 cm with no other focal lesions. The spleen was enlarged with a span of 7.3 cm. The right branch of the portal vein showed evidence of a 2.1 cm thrombosis (Figure 1). All other abdominal viscera appeared normal with no free fluid.

Contrast-enhanced computed tomography (CECT) of the thorax and abdomen was then performed to further assess the burden of the disease in the chest and to evaluate the portal vein. CECT confirmed hepatosplenomegaly, with hypodensity of the right lobe of the liver; thrombosis of the right branch of the portal vein (Figure 2); and enlargement of several hepatic portal, retroperitoneal, and para-aortic lymph nodes.

### **Histological examinations**

A bone marrow smear and biopsy were attempted with the result of slightly decreased whole cell counts. The subsequent esophagogastroduodenoscopy and colonoscopy results were normal. Although the possibility of viral, bacterial, and parasitic infestations was clinically considered, it was kept at a low priority due to the lack of significant results of laboratory examinations. Ultrasound-guided percutaneous liver biopsy was then performed. Histology of the liver biopsy showed entirely effaced architecture of hepatic parenchymal and portal areas by inflammation and necrosis. Central caseating necrosis was surrounded by lymphocytes, multinucleate giant cells, and epithelioid macrophages (Figure 3A). This revealed epithelioid granulomas with a background of caseating necrosis. Ziehl-Nelson staining showed acid-fast bacilli within the granulomas (Figure 3B).

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## **FINAL DIAGNOSIS**

Based on the histological, bacteriological, and laboratory findings, a final diagnosis of the present case was isolated hepatic TB associated with PVT and HBV coinfection.

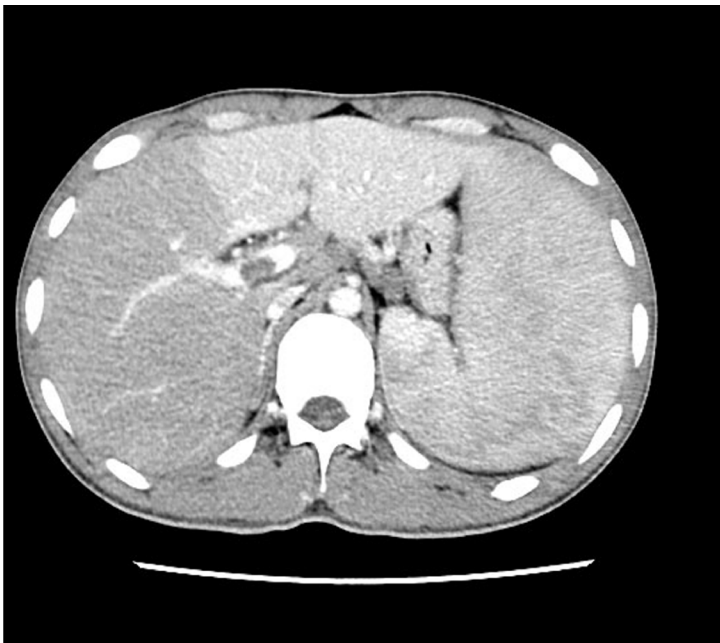
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## **TREATMENT**

Anti-TB medication, including isoniazid, rifapentine, ethambutol, and pyrazinamide, was administered. Along with ATT, the patient was treated with entecavir as an antiviral medication and dabigatran as an anticoagulant. One week after ATT, he was discharged from the hospital free of symptoms.



**Figure 1 Abdominal ultrasound findings before treatment.** The right branch of the portal vein showed evidence of a 2.1 cm thrombosis.



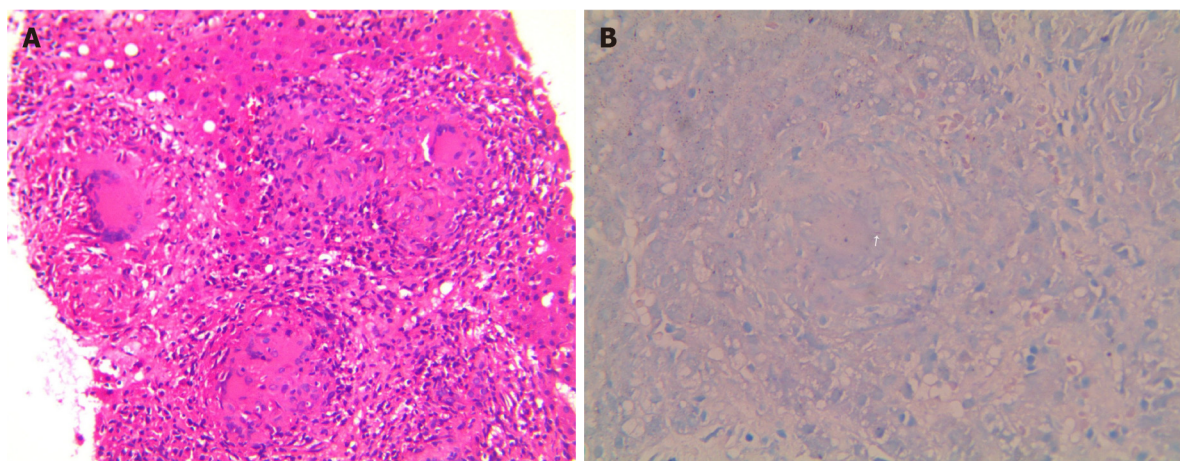
**Figure 2 Abdominal computed tomography findings.** Abdominal contrast-enhanced computed tomography confirmed hepatosplenomegaly, with hypodensity of the right lobe of the liver, and thrombosis of the right branch of the portal vein.

## OUTCOME AND FOLLOW-UP

The patient had an uneventful clinical course. At 4 wk of therapy, repeat sonography of the abdomen showed regression of hepatosplenomegaly. At 4 mo of therapy, the sonography showed complete resolution of the PVT (Figure 4), so dabigatran was discontinued. ATT and antiviral therapies were administered for a total of 9 mo. The HBV-DNA level was under detection with normal liver function at the 2-year follow-up after therapy.

## DISCUSSION

Although the prevalence of TB decreased quickly worldwide after the widespread use of anti-TB drugs in the 1940s, there has been a global resurgence of TB since the acquired immune deficiency syndrome epidemic of the 1980s, the development of



**Figure 3 Histopathological examination.** A: Histology of the liver biopsy showed entirely effaced architecture of hepatic parenchymal and portal areas by inflammation and necrosis. Central caseating necrosis was surrounded by lymphocytes, multinucleated giant cells, and epithelioid macrophages (Hematoxylin and eosin staining, 40 ×); B: Ziehl-Nelson staining showed acid-fast bacilli (arrow) within the granulomas (Ziehl-Nelson staining, 40 ×).



**Figure 4 Abdominal ultrasound findings at 4 mo of treatment.** At 4 mo of therapy, repeat sonography of the abdomen showed regression of hepatosplenomegaly and complete resolution of the portal vein thrombosis.

multidrug-resistant TB strains, and an increased number of immunocompromised patients[6-8]. The global distribution of reported hepatic TB cases is concentrated in Sub-Saharan Africa and Southeast Asia, which is similar to the distribution of pulmonary TB[4]. A conservative estimate of the incidence of hepatic TB can be made using data from studies conducted by Essop *et al*[9] and Tai *et al*[10], who found hepatic TB in approximately 1% of all active TB cases.

Hematogenous dissemination *via* the hepatic artery from a pulmonary focus is the most common etiology of hepatic TB[2,5]. Other extrapulmonary sites, such as abdominal lymph nodes, may be a rare source of miliary dissemination. In local hepatic TB, dissemination primarily occurs *via* the portal vein from a focus in the gastrointestinal tract. Miliary hepatic TB is characterized by diffuse seeding of the liver, with tubercles less than 2 mm in size situated in lobules of the liver. Local hepatic TB is typically characterized by tubercles greater than 2 mm in diameter situated near the portal triad region[2]. Local hepatic TB tends to cause more hepatocellular damage than miliary hepatic TB[2].

Hepatic TB most commonly affects people in the 11 to 50-year-old age group, with the peak incidence of disease in the second decade of life. Isolated hepatic TB is more common in the fourth to sixth decades of life[12-14]. The disease has a 2:1 male preponderance[15]. The clinical features of hepatic TB are nonspecific, which often delays diagnosis. Analysis of hepatic TB case series revealed that the most common

signs or symptoms were hepatomegaly (80%), fever (67%), respiratory symptoms (66%), abdominal pain (59.5%), and weight loss (57.5%)[4]. The less common signs included splenomegaly (30%), ascites (23%), and jaundice (20%)[4]. Local hepatic TB and miliary TB may differ in clinical presentation. Miliary hepatic TB may present with acute respiratory symptoms, such as a cough, with or without sputum production[2,10,15], while local hepatic TB may present primarily as diffuse abdominal pain. Jaundice is also more common in cases of local hepatic TB and biliary TB.

The imaging manifestation of tubercular hepatic disease can be wide ranging but can be broadly categorized into miliary patterns, nodular tuberculosis with serohepatic variants, and tubercular cholangitis. Ultrasound and CT scans are generally the most widely available and first imaging tests to be obtained, but they both lack diagnostic specificity. On CT, miliary lesions appear as microabscesses in the form of multiple small foci and may exhibit minimal peripheral enhancement following intravenous contrast administration[16,17]. In contrast, local hepatic TB generally appears on CT as one large solitary nodule or multiple variable-sized hepatic nodules. Hepatic TB may also reveal hepatomegaly without nodular intrahepatic lesions, or it may reveal abdominal lymphadenopathy with peripheral lymph node enhancement and/or calcifications[18-20]. Sonographically, hepatic TB lesions may appear hypoechoic to isoechoic relative to the background parenchyma; however, in rare instances, a hyperechoic pattern may be demonstrated[12,21-25]. Since the imaging appearances can be quite variable depending on the stage of the hepatic disease, it may often be difficult to distinguish hepatic TB from the more common neoplasm, other granulomatous diseases, vascular disorders, viral hepatitis, and systemic infections[26,27]. Isolated TB of the liver is a rare entity, in which vascular complications, such as PVT and subsequent portal hypertension, have rarely been reported[28]. The present case demonstrates the difficulty of diagnosis despite a complete investigation due to its nonspecific clinical and imaging findings.

The fact that isolated liver TB does not produce characteristic symptoms, clinical signs, or imaging appearances, makes diagnosis challenging. Histopathological or bacteriological confirmation is often required to reach diagnosis. A liver biopsy is recommended in any person with clinical, laboratory, and radiographic suspicion of hepatic TB. These may include but are not limited to hepatosplenomegaly of unknown origin, fever of unknown origin, and abnormal liver enzymes[29,30]. Liver biopsies should be sent for both histological and microbiological evaluation in such cases. The characteristic histological feature of both miliary and local forms of hepatic TB is the granuloma. Histological evidence of caseating granulomas had a median sensitivity of 68% among hepatic TB case series[4]. In TB endemic regions, the presence of a hepatic caseating granuloma on liver biopsy should suggest a TB etiology and may warrant a course of ATT. Liver biopsies should be sent for acid-fast bacilli (AFB) smear testing as well as mycobacterial culture. AFB smears have a median sensitivity of 25% among hepatic TB case series[4]. Mycobacterial culture provides specific evidence for hepatic TB, but the sensitivity has been reported to be less than 10%[31]. Since AFB smears and cultures have a low sensitivity, PCR assays have been recommended for diagnosing hepatic TB[31,32]. In a systemic review of 14 hepatic TB case series, Hickey *et al*[4] reported that PCR had a median sensitivity of 86% among hepatic TB cases. In another series of 43 liver biopsies with granulomas, Diaz *et al*[31] used PCR to amplify the IS6110 insertion sequence to detect TB and showed that PCR had a sensitivity of 53%, specificity of 96%, and positive and negative predictive values of 90% and 76%, respectively. These preliminary studies suggest that PCR may be more sensitive and specific for the diagnosis of TB than AFB smear or culture. This faster method for the diagnosis of hepatic TB may promote early detection and treatment.

It has been reported that the lifetime risk of PVT in the general population is 1% [33]. Venous thrombosis is a known but rare complication of pulmonary and extrapulmonary tuberculosis, occurring in 1.5%–3.4% of patients[34]. Intraabdominal TB, such as peritoneum, bowel, lymph node, and solid organ involvements, may be associated with PVT (Table 1)[28,35-41]. All the three parts of Virchow's triad (hypercoagulability, venous stasis, and endothelial dysfunction) may play a role in the pathogenesis of thromboembolic complications in TB. It has been postulated that contiguous spread of inflammation and granulomas in the vessel wall with subintimal fibrosis may be other risk factors that contribute to PVT. Spontaneous resolution of the thrombosis did occur in some cases, but the frequency of partial or complete recanalization seemed to be higher in patients treated with anticoagulants. Based on a previous study, anticoagulants may be associated with recanalization of both acute and chronic thrombosis[42]. Low molecular weight heparins and vitamin K antagonists have long been the preferred anticoagulants in noncirrhotic PVT; however,



**Table 1 Clinical studies on intraabdominal tuberculosis associated with portal vein thrombosis**

Ref.	Number	Age/Sex	Location of TB involvement	Site of thrombosis	Therapy	Recanalization
Venkatesh <i>et al</i> [28], 2005	1	31/F	Liver; ileo-cecum	PVT; PH	ATT	Not mentioned
Bhalla <i>et al</i> [35], 2010	6/183	27/M	Peri-pancreatic, coeliac axis adenopathy; cecum	PVT; SVT; SMVT	ATT	Persistent thrombosis
		36/M	Peri-pancreatic adenopathy; duodenum; lung	PVT	ATT	Lost to follow-up
		32/M	Duodenum	PVT	ATT	Recanalization
		25/F	Peri-pancreatic adenopathy; peritoneum; lung	PVT	ATT	Died of disseminated disease
		35/F	Peri-pancreatic, porta, left para-aortic adenopathy	PVT; PC	ATT	Persistent PC
		8/F	Peri-pancreatic, porta, left para-aortic adenopathy; mediastinum	PVT	ATT	Persistent PC
Ozşeker <i>et al</i> [36], 2012	1	43/M	Peritoneum	PVT; PC	ATT	Not mentioned
Ruttenberg <i>et al</i> [37], 1991	1/2	42/F	Pancreas	PVT; SVT; PH	ATT	Not mentioned
Liew <i>et al</i> [38], 2011	1	26/F	Periporta TB	PVT; PC; PH	ATT	Not mentioned
Wariyapperuma <i>et al</i> [39], 2015	1	35/F	Peritoneum	PVT; PC	ATT; VKA	Not mentioned
Kumar <i>et al</i> [40], 2013	1	2/Not mentioned	Abdominal lymph nodes	PVT; PC	ATT	Persistent PC
Elleuch <i>et al</i> [41], 2020	1	60/M	Peritoneum	PVT; PC	ATT; VKA	Recanalization

VKA: Vitamin K antagonist; PVT: Portal vein thrombosis; PH: Portal hypertension; PC: Portal cavernoma; SVT: Splenic vein thrombosis; SMVT: Superior mesenteric vein thrombosis.

direct oral anticoagulants are coming into increasing clinical use and have been shown to be safe and effective in retrospective studies[43,44]. Timely initiation of anticoagulants and ATT may lead to complete resolution of the PVT and thus prevent subsequent portal hypertension and complications, including ascites and life-threatening bleeding from varices.

HBV infection is another serious global public health problem. It is estimated that 240 million people worldwide are chronic carriers of HBsAg[45]. The prevalence of HBV infection is highly heterogeneous throughout the world, with an intermediate to high prevalence in the Asia-Pacific region, representing three-quarters of chronic HBV-positive subjects worldwide. HBV infection has been reported to be a significant risk factor for hepatotoxicity related to ATT[46]. A higher proportion of inactive HBsAg carriers who received ATT evidenced moderate to severe drug-induced liver injury when compared with control subjects (8% *vs* 2%)[47]. Liver injury was also more severe by histological assessment in HBV carriers than in non-carriers[46]. Further research indicated that hepatotoxicity related to ATT was more common in HBV-positive patients who were seropositive for HBeAg than among those who were seronegative for HBeAg[48]. Most episodes of liver dysfunction are usually preceded by an increase in HBV-DNA levels[46]. To date, there have not been guidelines for antiviral therapy against HBV in active TB and inactive HBV coinfecting patients needing ATT. Antiviral agents active against HBV have been shown to suppress HBV replication, prevent progression to cirrhosis, and reduce the risk of hepatocellular carcinoma and liver-related deaths[45]. Therefore, a case can be made for decreasing viral load using antiviral therapy against HBV with high potency, high genetic barrier drugs, such as entecavir or tenofovir, in patients with HBV infection needing ATT to prevent the development of liver dysfunction.

## CONCLUSION

The incidence of TB has likely increased with the development of multidrug-resistant TB strains and an increased number of immunocompromised patients. Isolated hepatic TB poses diagnostic challenges due to its largely nonspecific clinical and imaging presentations. Liver biopsy is recommended for a definite diagnosis. Timely ATT and anticoagulants may be effective in preventing portal hypertension and serious complications in patients with hepatic TB associated with PVT. Since HBV and TB coinfecting patients needing ATT may have a higher risk of hepatic injury, antiviral medication against HBV with high potency and high genetic barrier drugs should be initiated upon diagnosis to prevent liver dysfunction.

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