

World Journal of *Clinical Cases*

World J Clin Cases 2021 November 6; 9(31): 9320-9698



FRONTIER

- 9320 Gut-liver axis in cirrhosis: Are hemodynamic changes a missing link?
Maslennikov R, Ivashkin V, Efremova I, Poluektova E, Shirokova E

REVIEW

- 9333 Pharmaconutrition strategy to resolve SARS-CoV-2-induced inflammatory cytokine storm in non-alcoholic fatty liver disease: Omega-3 long-chain polyunsaturated fatty acids
Jeyakumar SM, Vajreswari A
- 9350 Major depressive disorder: Validated treatments and future challenges
Karrouri R, Hammani Z, Benjelloun R, Otheman Y

MINIREVIEWS

- 9368 Gene × environment interaction in major depressive disorder
Zhao MZ, Song XS, Ma JS
- 9376 Deep learning driven colorectal lesion detection in gastrointestinal endoscopic and pathological imaging
Cai YW, Dong FF, Shi YH, Lu LY, Chen C, Lin P, Xue YS, Chen JH, Chen SY, Luo XB

ORIGINAL ARTICLE**Case Control Study**

- 9386 Cognitive behavioral therapy on personality characteristics of cancer patients
Yuan XH, Peng J, Hu SW, Yang Y, Bai YJ

Retrospective Cohort Study

- 9395 Extrapneumonic necrosis volume: A new tool in acute pancreatitis severity assessment?
Cucuteanu B, Negru D, Gavrilescu O, Popa IV, Floria M, Mihai C, Cijevschi Prelipcean C, Dranga M
- 9406 Establishment of a risk assessment score for deep vein thrombosis after artificial liver support system treatment
Ye Y, Li X, Zhu L, Yang C, Tan YW

Retrospective Study

- 9417 Clinical management and susceptibility of primary hepatic lymphoma: A cases-based retrospective study
Hai T, Zou LQ
- 9431 Association of serum pepsinogen with degree of gastric mucosal atrophy in an asymptomatic population
Cai HL, Tong YL

- 9440** Risk factors for relapse and nomogram for relapse probability prediction in patients with minor ischemic stroke

Yu XF, Yin WW, Huang CJ, Yuan X, Xia Y, Zhang W, Zhou X, Sun ZW

- 9452** Incidence, prognosis, and risk factors of sepsis-induced cardiomyopathy

Liang YW, Zhu YF, Zhang R, Zhang M, Ye XL, Wei JR

- 9469** Associations with pancreatic exocrine insufficiency: An United Kingdom single-centre study

Shandro BM, Chen J, Ritehnia J, Poullis A

- 9481** Retrospective analysis of influencing factors on the efficacy of mechanical ventilation in severe and critical COVID-19 patients

Zeng J, Qi XX, Cai WW, Pan YP, Xie Y

Observational Study

- 9491** Vitamin D deficiency, functional status, and balance in older adults with osteoarthritis

Montemor CN, Fernandes MTP, Marquez AS, Poli-Frederico RC, da Silva RA, Fernandes KBP

- 9500** Psychological impact of the COVID-19 pandemic on Chinese population: An online survey

Shah T, Shah Z, Yasmeen N, Ma ZR

- 9509** Outcomes of different minimally invasive surgical treatments for vertebral compression fractures: An observational study

Yeh KL, Wu SH, Liaw CK, Hou SM, Wu SS

META-ANALYSIS

- 9520** Glycated albumin as a biomarker for diagnosis of diabetes mellitus: A systematic review and meta-analysis

Xiong JY, Wang JM, Zhao XL, Yang C, Jiang XS, Chen YM, Chen CQ, Li ZY

CASE REPORT

- 9535** Rapid response to radiotherapy in unresectable tracheal adenoid cystic carcinoma: A case report

Wu Q, Xu F

- 9542** Clinical observation of pediatric-type follicular lymphomas in adult: Two case reports

Liu Y, Xing H, Liu YP

- 9549** Malignant adenomyoepithelioma of the breast: Two case reports and review of the literature

Zhai DY, Zhen TT, Zhang XL, Luo J, Shi HJ, Shi YW, Shao N

- 9557** Validation of diagnostic strategies of autoimmune atrophic gastritis: A case report

Sun WJ, Ma Q, Liang RZ, Ran YM, Zhang L, Xiao J, Peng YM, Zhan B

- 9564** Characteristics of primary giant cell tumor in soft tissue on magnetic resonance imaging: A case report

Kang JY, Zhang K, Liu AL, Wang HL, Zhang LN, Liu WV

- 9571** Acute esophageal necrosis as a complication of diabetic ketoacidosis: A case report
Moss K, Mahmood T, Spaziani R
- 9577** Simultaneous embolization of a spontaneous porto-systemic shunt and intrahepatic arterioportal fistula: A case report
Liu GF, Wang XZ, Luo XF
- 9584** Ureteroscopic holmium laser to transect the greater omentum to remove an abdominal drain: Four case reports
Liu HM, Luo GH, Yang XF, Chu ZG, Ye T, Su ZY, Kai L, Yang XS, Wang Z
- 9592** Forearm compartment syndrome due to acquired hemophilia that required massive blood transfusions after fasciotomy: A case report
Kameda T, Yokota T, Ejiri S, Konno SI
- 9598** Transforaminal endoscopic excision of bi-segmental non-communicating spinal extradural arachnoid cysts: A case report and literature review
Yun ZH, Zhang J, Wu JP, Yu T, Liu QY
- 9607** T-cell lymphoblastic lymphoma with extensive thrombi and cardiac thrombosis: A case report and review of literature
Ma YY, Zhang QC, Tan X, Zhang X, Zhang C
- 9617** Perfect pair, scopes unite – laparoscopic-assisted transumbilical gastroscopy for gallbladder-preserving polypectomy: A case report
Zheng Q, Zhang G, Yu XH, Zhao ZF, Lu L, Han J, Zhang JZ, Zhang JK, Xiong Y
- 9623** Bilateral hematoma after tubeless percutaneous nephrolithotomy for unilateral horseshoe kidney stones: A case report
Zhou C, Yan ZJ, Cheng Y, Jiang JH
- 9629** Atypical endometrial hyperplasia in a 35-year-old woman: A case report and literature review
Wu X, Luo J, Wu F, Li N, Tang AQ, Li A, Tang XL, Chen M
- 9635** Clinical features and literature review related to the material differences in thread rhinoplasty: Two case reports
Lee DW, Ryu H, Jang SH, Kim JH
- 9645** Concurrent tuberculous transverse myelitis and asymptomatic neurosyphilis: A case report
Gu LY, Tian J, Yan YP
- 9652** Diagnostic value of contrast-enhanced ultrasonography in mediastinal leiomyosarcoma mimicking aortic hematoma: A case report and review of literature
Xie XJ, Jiang TA, Zhao QY
- 9662** Misidentification of hepatic tuberculosis as cholangiocarcinoma: A case report
Li W, Tang YF, Yang XF, Huang XY

- 9670** Brunner's gland hyperplasia associated with lipomatous pseudohypertrophy of the pancreas presenting with gastrointestinal bleeding: A case report
Nguyen LC, Vu KT, Vo TTT, Trinh CH, Do TD, Pham NTV, Pham TV, Nguyen TT, Nguyen HC, Byeon JS
- 9680** Metachronous squamous cell carcinoma of pancreas and stomach in an elderly female patient: A case report
Kim JH, Kang CD, Lee K, Lim KH
- 9686** Iatrogenic giant pseudomeningocele of the cervical spine: A case report
Kim KW, Cho JH
- 9691** Traditional Chinese medicine for gait disturbance in adrenoleukodystrophy: A case report and review of literature
Kim H, Kim T, Cho W, Chang H, Chung WS

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Misidentification of hepatic tuberculosis as cholangiocarcinoma: A case report

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Abstract

BACKGROUND

Hepatic tuberculosis (TB) is uncommon clinically. Because of a lack of specific signs, characteristic symptoms and clinical manifestations and because pathological samples are difficult to obtain, hepatic TB is easily missed or misdiagnosed.

CASE SUMMARY

A 62-year-old Chinese man presented with jaundice for 1 wk and no abnormal laboratory tests other than elevated bilirubin, aminotransferases and C-reactive protein. Computed tomography (CT) of the abdomen showed a mass in the left lobe of the liver and hepatic hilum with striped calcified foci. Mild enhancement was visible at the edges, along with extensive intrahepatic biliary ductal dilatation in the right lobe of the liver. In the arterial phase of both CT and magnetic resonance imaging, the main trunk and right branch of the portal artery were partially visualized. Magnetic resonance cholangiopancreatography (MRCP) indicated that the left lobe of the liver and most of the bile ducts in the hilum were not visible. Pathological examination revealed coagulative necrosis, and granulomatous nodules were seen around areas of necrosis; therefore, TB was considered.

CONCLUSION

Hepatic tuberculosis is easily misdiagnosed or missed on imaging. Percutaneous puncture biopsy is the most useful tool for definitive diagnosis.

Key Words: Hepatic tuberculosis; Pathology; Differential diagnosis; Clinical Management; Case report

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Core Tip: Hepatic tuberculosis (TB) is rare in clinical practice and can also be easily missed or misdiagnosed. The clinical symptoms of hepatic TB reported in this case were atypical and a series of investigations were completed in our hospital, including computed-tomography-enhanced scans, magnetic resonance imaging multiparametric scans and pathological examination. We have also conducted a series of discussions on the case, which provide more reference for the diagnosis of hepatic TB in the future.

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INTRODUCTION

Tuberculosis (TB) can affect any system or organ throughout the body[1]. TB infection of the liver, also known as hepatic TB, is a manifestation of extrapulmonary infection with *Mycobacterium tuberculosis*[2]. Hepatic TB is uncommon clinically, and the main clinical manifestations include fever, cyanosis, jaundice, shortness of breath, cough, pulmonary moist rales, hepatomegaly, splenomegaly and abdominal distention[3]. Because of a lack of specific signs, characteristic symptoms and clinical manifestations and because pathological samples are difficult to acquire, hepatic TB is easily missed or misdiagnosed. The radiological manifestations and clinical features of hepatic TB can be mistaken for malignant lesions, leading to unnecessary surgical intervention and erroneous clinical diagnosis[4]. Among the cases of hepatic TB reported in the literature, the miliary form was common (79% of cases), with local hepatic TB accounting for only 21% of cases[5]. We report a case of local hepatic TB that was misdiagnosed as intrahepatic cholangiocarcinoma by magnetic resonance imaging (MRI). The patient underwent left hemihepatectomy, and postoperative pathology confirmed hepatic TB. The imaging findings, histopathology, differential diagnosis and treatment strategies are analyzed and summarized to provide a reference for future clinical treatment.

CASE PRESENTATION

Chief complaints

A 62-year-old Chinese man presented with jaundice for 1 wk.

History of present illness

The patient complained of yellowing of the sclera, darkened urine, fatigue, loss of appetite without significant weight loss, abdominal pain and bloating for 1 wk.

History of past illness

The patient had a history of lower limb deformity due to trauma at an early age, head and lower limb fracture caused by a car accident 4 years ago and surgically treated; details unknown.

Personal and family history

The patient's family members have no history of specific genetic or infectious diseases.

Physical examination

Physical examination showed double lower limb deformities. The patient's temperature was 36.7 °C, the pulse was 65 beats/min and the blood pressure was 135/92 mmHg.

Laboratory examinations

The laboratory test findings were not specific (Table 1). Biochemical tests showed that total bilirubin (380.6 $\mu\text{mol/L}$), direct bilirubin (347.9 $\mu\text{mol/L}$) and indirect bilirubin (30.9 $\mu\text{mol/L}$) were elevated, with normal electrolyte profiles and renal function. Elevated glutamic oxaloacetic transaminase and glutamic pyruvic transaminase levels indicated impaired liver function. Tumor markers such as α -fetoprotein, carcinoembryonic antigen, and carbohydrate antigen 199 were negative, and there was no evidence of hepatitis A, B, C or E virus infection.

Imaging examinations

Abdominal CT showed a large patchy hypodense shadow in the left lobe of the liver and hepatic hilum with striped calcified foci (Figure 1A). This mass measured 9.2 cm \times 6.0 cm \times 5.8 cm, and mild enhancement was visible at the edges with extensive intrahepatic biliary ductal dilatation in the right lobe of the liver (Figure 1B, C). Chest CT showed only a few areas of fibrosis and hard nodules, with no obvious signs of active TB. We considered hepatic schistosomiasis or intrahepatic cholangiocarcinoma based on the CT findings. Abdominal MRI demonstrated that the left lobe and caudate lobe of the liver were atrophied, with a lesion measuring 8.7 cm \times 6.8 cm \times 5.2 cm. A slightly hypointense signal on T1-weighted and mixed hyperintense and hypointense signals on T2-weighted fat suppressed images were observed (Figure 2A, B), with mild restricted diffusion (Figure 2F) and a low apparent diffusion coefficient value of $0.764 \times 10^3 \text{ mm}^2/\text{s}$ (Figure 2G). The left branch of the portal vein and the left hepatic vein could not be visualized in the portal venous phase (Figure 1C, 2D). In the arterial phase of both CT and MRI scans, the main trunk of the portal vein and the right branch of the portal vein were partially visualized (Figure 1D, 2E). The bile ducts in the hilar region and most of the intrahepatic bile ducts in the left lobe of the liver could not be observed, and the above intrahepatic bile ducts showed varying degrees of dilatation on magnetic resonance cholangiopancreatography (MRCP) (Figure 2H). This was the main clue that led to our misdiagnosis of cholangiocarcinoma. Enlarged lymph nodes were seen in the retroperitoneum, and the larger lymph node was approximately 1.3 cm \times 1.2 cm, with uniform strengthening after enhancement.

Pathological examination

Intraoperative exploration of the abdominal cavity showed multiple space-occupying masses of variable size in the left liver that protruded from the surface of the liver by approximately 1 cm and were yellowish-white in color and hard in texture; there were multiple scattered small nodules in the hepatic round ligament, small omental sac and other sites, and these nodules were yellowish-white in color. Two yellow nodules were partially removed from the left liver by an ultrasonic knife and then sent for rapid frozen biopsy. Liver tissue was seen, with focal visible necrosis and granulomatous nodules around the necrosis, which were considered to indicate a microbial infection (Figure 3). The nodules in the lesser omentum and the liver nodules were again sampled and sent for rapid analysis: necrosis and granulomatous nodule formation were seen, microbial infection was considered, and TB was not excluded. The left lobe of the liver, the lymph nodes of the duodenal ligament and the omentum were granulomatous.

FINAL DIAGNOSIS

The final pathological diagnosis after biopsy in this case was hepatic TB.

TREATMENT

The patient underwent laparoscopic biliary tissue biopsy, resection of the left half of the liver and portal bile duct and internal bile-intestinal drainage of the right portal bile duct.

OUTCOME AND FOLLOW-UP

The patient was then transferred to the intensive care unit for treatment and was

Table 1 Blood test results

	Patient's results	Normal range
Neutrophil (%)	79.3	50.0–70.0
Lymphocyte (%)	9.40	20.0–40.0
Prothrombin time activity (%)	102	70–130
Fibrinogen (g/L)	4.05	2.0–4.0
ESR (mm/h)	19	0–10
Total bilirubin (μmol/L)	380.6	3.4–17.1
Direct bilirubin (μmol/L)	347.9	0–8
Indirect bilirubin (μmol/L)	30.9	0–20
ALT (U/L)	120	9–50
AST (U/L)	132	15–40
C-reactive protein (mg/L)	31.72	0–6
AFP (IU/mL)	1.77	0–20
CEA (ng/mL)	2.68	0–5
CA199 (U/mL)	18.86	0–34

ESR: erythrocyte sedimentation rate; ALT: glutamic pyruvic transaminase; AST: glutamic oxaloacetic transaminase; AFP: α -fetoprotein; CEA: carcinoembryonic antigen; CA199: carbohydrate antigen 199.

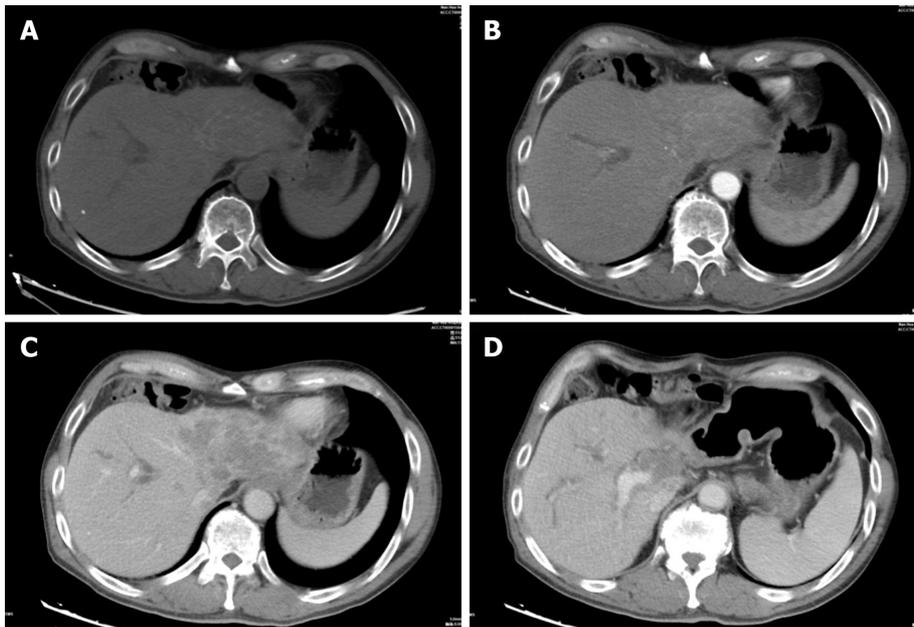


Figure 1 Computed tomography (CT) images of hepatic tuberculosis. A: CT revealed a slightly hypodense mass in the left lobe of the liver and the hilar region, with a few foci of calcification; B: Mild enhancement was observed at the edge in the arterial phase after enhancement; C: The portal vein lesion remained relatively slightly hypodense; D: The main trunk of the portal vein and the right branch of the portal vein were partially visualized in the arterial phase after enhancement.

subsequently discharged 1 wk after the operation.

DISCUSSION

The clinical manifestations of TB are mainly related to the character, range and type of the lesion and the reactivity of the organism. Levine classified TB as follows: (1)

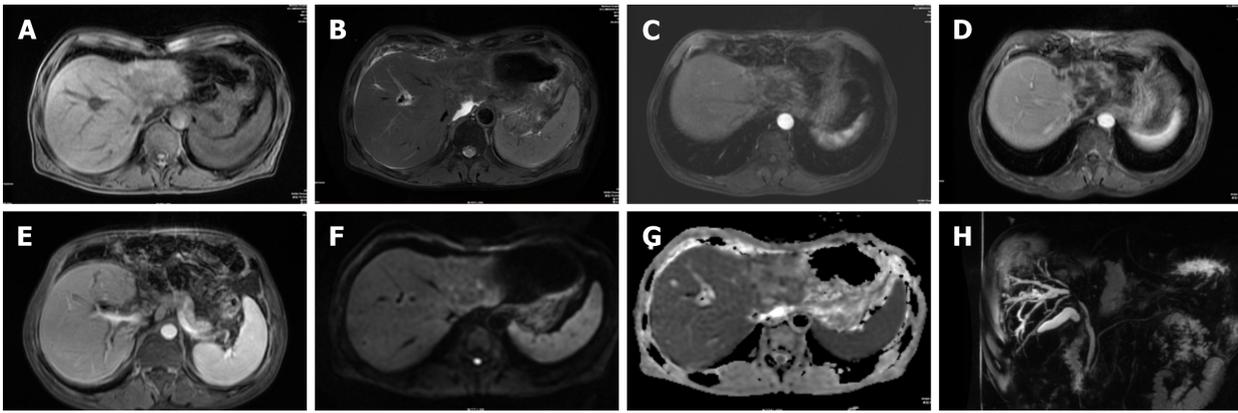


Figure 2 Magnetic resonance cholangiopancreatography scans of hepatic tuberculosis. A: The lesion showed slightly hypointense signals in T1-weighted images; B: The lesion showed mixed hyperintense and hypointense signals in T2-weighted fat suppressed images; C: The lesion was mildly enhanced at the edge of the arterial phase; D: The margins of the lesion were still mildly enhanced in the portal vein, and no enhancement was seen in the main part of the lesion; E: Premature portal manifestation of the disease was seen in the liver in the arterial phase; F: Mild restricted diffusion was observed; G: The apparent diffusion coefficient value was decreased in the central part of the lesion; H: The left lobe of the liver and the bile ducts in the hilar region were not visualized.

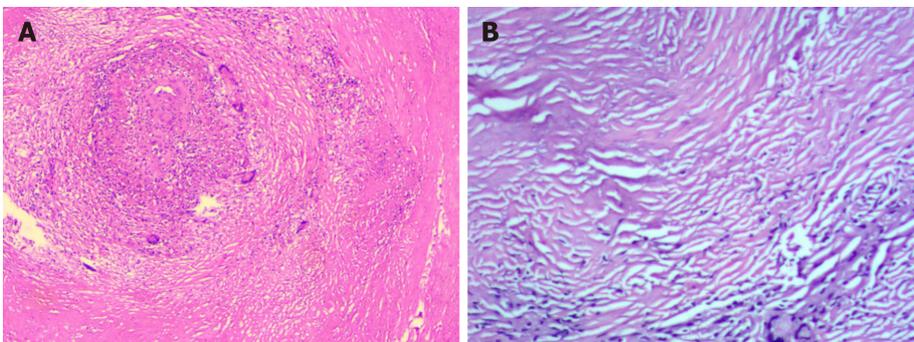


Figure 3 Pathological findings. A: Large coagulative necrosis and granulomatous nodules were seen in the left half of the liver, and the lesions covered almost the entire submitted liver; B: Most of the tissue examined displayed coagulative necrosis, including one piece of liver tissue with focal necrosis and granulomatous nodules around the necrosis, which was considered to indicate a pathogenic microbial infection.

miliary TB; (2) pulmonary TB with hepatic involvement; (3) primary liver TB; (4) focal tuberculoma or abscess; and (5) TB cholangitis. Miliary TB of the liver is more common than other types of hepatic TB[6], and it has been reported to occur in 50%–80% of all patients with terminal pulmonary TB. During the healing stage of hepatic TB, CT scans might present diffuse hepatic calcifications (approximately 50% of these cases)[7]. In our case of hepatic TB, there was a central nonenhancing hypodense lesion due to caseous necrosis, with enhancement at the edges in contrast to the surrounding granulation tissue. Streaks of calcification were visible on CT, indicating that the imaging characteristics of hepatic TB may be multilayered lesions of various densities and demonstrating that lesions of different pathological stages can coexist in liver TB, including tuberculous granuloma, liquefied necrosis, fibrosis and calcification[8]. The heterogeneous, hypodense nodule (central caseous necrosis) with peripheral enhancement on CT after the administration of an intravenous contrast agent (surrounding granulation tissue) can easily lead to a misdiagnosis of hepatocellular carcinoma [9]. A retrospective analysis of 96 patients with hepatic TB showed that they presented with the histological manifestations of granulomas (95.8%), caseation (83.3%) and acid-fast bacilli with granulomas (9%)[10]. Granuloma formation is the histopathological substrate of the miliary pattern. However, the lesions are too small to be observed by conventional imaging[11].

MRI manifestations are associated with nodules at different pathological stages. In the early and middle stages of sarcoidosis with or without caseous or liquefied necrosis, the lesions exhibit a low T1-weighted and a high T2-weighted signal. Similar lesions with low T1-weighted and T2-weighted signals correspond to the fibrous stage of TB and might have slight or no peripheral enhancement. Early- or intermediate-stage tuberculous granulomas and fibroproliferative lesions appear as hypointense areas on CT and with various signal intensities on T2-weighted imaging, which is the

major advantage of MRI for the diagnosis of hepatic TB[12]. Thick peripheral enhancement and/or enhanced irregular thickened septa with restricted diffusivity on diffusion-weighted imaging can be observed according to the protein content (thickened septa and debris) after the use of contrast media[13]. These characteristics are basically consistent with the cases we have reported.

Hepatic TB is commonly imaged with CT, and the pathological manifestations vary in different periods, so this disease needs to be differentiated from metastases, cholangiocarcinoma, bacterial liver abscess, and hepatic schistosomiasis. The misdiagnosis of hepatic TB as metastasis has also been reported in the literature, and sometimes it is hard to distinguish between hepatic TB and metastasis because of the simultaneous involvement of the liver and spleen[14]. Patients with metastases usually have a history of a primary tumor, and these metastases are not limited to the liver but may include other organs or enlarged lymph nodes.

The misdiagnosis of hepatic TB as intrahepatic cholangiocarcinoma is not uncommon[4,6,15,16]. Of the lesions misdiagnosed as intrahepatic cholangiocarcinoma, only one was located in the porta hepatis, and the rest were located in the left lobe of the liver; all of these lesions caused symptoms of biliary obstruction, including the cases we reported. These misdiagnosed cases typically presented on CT images as mild enhancement and central, hypodense lesions due to caseation necrosis, with mild enhancement at the edges corresponding to the surrounding granulation tissue. MRI of hepatic TB generally shows a hypointense nodule with a hypointense rim on T1-weighted imaging. T2-weighted imaging presents a hypointense, isointense or hyperintense nodule with a less intense margin. These signs are not truly distinguishable from those of cholangiocarcinoma. However, the difference between the case we reported and typical cholangiocarcinoma is that CT usually shows stripes of calcification in hepatic TB, while cholangiocarcinoma rarely exhibits a high number of calcifications; thus, we speculate that the presence of such stripes of calcification may be an important point in differentiating hepatic TB from cholangiocarcinoma.

Simple plain ultrasound cannot differentiate between bacterial and tubercular liver abscesses[17]. Abscesses mostly occur in elderly male patients and patients with diabetes mellitus[18]. A high incidence of TB can be found in regions where HIV is most prevalent, and HIV-induced immunosuppression results in the reactivation of latent TB[19]. *Klebsiella pneumoniae* is one of the microorganisms most commonly isolated from pyogenic liver abscesses in Asians, and 89.7% of patients who have liver abscesses due to *K. pneumoniae* may present with internal gas bubbles[20]. According to the imaging findings, the patient in this case had a hepatic venous obstruction, which needed to be differentiated from hepatic schistosomiasis, as many of the clinical and pathological characteristics of hepatic TB and hepatic schistosomiasis are similar. Pathologically, the patient had periportal inflammation and granuloma formation, which are most commonly found in schistosomiasis[21]. Ultimately, a needle biopsy is needed to identify the disease. Bacteriological surveillance in patients with extrapulmonary TB is difficult and not always feasible. Ziehl-Neelsen staining is positive in only 40% of cases[22]. According to the literature, the sensitivity of polymerase chain reaction for TB is 82%[23], and *M. tuberculosis* infection has also been rapidly identified by analyzing liver tissue by next-generation sequencing[2]. The Xpert Mtb/Rif test is a nucleic acid amplification test that provides a rapid diagnosis of the *M. tuberculosis* complex, and the Xpert Mtb/Rif test has been known to detect some extrapulmonary forms of TB[24].

Anti-TB therapy is the key to treating primary hepatic TB. Owing to the increasing incidence of drug-resistant TB, quadruple therapy (isoniazid, rifampicin, pyrazinamide and ethambutol) is recommended[25]. Recent publications advocate that treatment options for almost all forms of drug-sensitive TB in adults include an initial 2-mo period of isoniazid, rifampin, pyrazinamide, and ethambutol, followed by 4 mo of isoniazid and rifampin[22,26,27]. This regimen cures more than 90% of patients with TB[22]. The optimal duration of time to treat hepatic TB is controversial, but 6–12 mo seems to be effective for most patients[5]. Patients generally do not require any further surgical intervention after the completion of anti-TB therapy, but surgical treatment of hepatic TB is usually required in cases of TB-related biliary compression leading to jaundice, portal hypertension, or biliary bleeding, or when the diagnosis is uncertain [28]. In conclusion, there are insufficient controlled data to recommend the use of corticosteroids in all cases of hepatic TB, either in isolated hepatic TB or in complications of cornified/spread TB. Glucocorticoids may have a role in the treatment of hepatic TB that does not respond appropriately to standard anti-TB therapy[29].

CONCLUSION

Hepatic TB is rare and lacks typical symptoms, signs and laboratory findings. Diagnosing hepatic TB with imaging is also difficult due to its diverse and nonspecific imaging features, which can easily lead to misdiagnosis and underdiagnosis. For hepatic masses with calcified foci visible on CT, biliary obstruction with the progressive loss of bile ducts on MRCP, and mildly enhanced edges after enhancement, the possibility of hepatic TB needs to be considered. Aside from considering the patient's clinical symptoms and relevant examination findings, a percutaneous puncture biopsy would be the most useful diagnostic tool for the definitive diagnosis of hepatic TB.

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