Int J Clin Exp Med 2016;9(7):12457-12468
www.ijcem.com /ISSN:1940-5901/IJCEM0019318

Original Article

Non-AIDS hepatic tuberculosis in adult: delineation using computed tomography

Fan Zhang[1*], Xiao Meng[2*], Yang Li[3]

[1]Department of Radiology, The Hainan Branch of Chinese PLA General Hospital, Haitang Bay, Sanya 572013, Hainan Province, China; [2]Guangdong Provincial Key Laboratory of Food, Nutrition and Health, School of Public Heath, Yat-Sen University, Guangzhou 510080, Guangdong Province, China; [3]Department of Radiology, Chinese PLA General Hospital, Beijing 100853, China. [*]Co-first authors.

Received November 6, 2015; Accepted February 10, 2016; Epub July 15, 2016; Published July 30, 2016

Abstract: Objective: The purpose of this study was to depict the CT manifestations of Hepatic Tuberculosis (HTB) in non-AIDS adult patients. Methods: Our institutional review board has approved the study. 14 patients (8 males and 6 females) were collected from two institutions (Guangzhou and Beijing) retrospectively. The age ranged from 25 to 73 (51±9). Patients' medical histories were obtained from all available medical records in hospital or telephone contact directly. There was no evidence of human immunodeficiency virus (HIV) infection in all patients. All subjects were performed by using a helical CT (Somatom Plus 4, Siemens Medical Solutions), and axial images were evaluated including plain scan images, hepatic arterial phase (HAP) images, portal vein phase (PVP) images. 5 subjects had equilibrium phase (EP) images. Results: Six types of HTB were observed on CT imaging: miliary HTB (3/14), nodular HTB (5/14), Pseudo-tumoral HTB (2/14), cholangitic HTB (1/14), serosal HTB (2/14), and false negative HTB (1/14). The mean size of lesions of miliary HTB was 0.9±0.4 cm. It trended to military distribution in the liver. 2/5 solitary nodular HTB. Conclusions: should be mainly identified from liver metastatic tumor. It is more even and solitary nodular HTB commonly occurs in hepatic periphery. It generally has central powder calcification in parenchyma of lesion. Pseudo-tumoral HTB was predilection left lobe of liver cholangitic HTB serosal HTB false negative HTB Apart from "powder calcification", CT findings of HTB lack the specificity for definitive diagnose.

Keywords: Non-AIDS, liver, tuberculosis, adult, tomography, x-ray computed

Introduction

Tuberculosis in the liver is infrequent as compare to those found in the lungs or GI track [1]. Primary TB of the liver without other organs involved is even rarer [2]. It is possibly due to abundant hepatic blood supply and efficiency mononuclear phagocyte system [3]. Normally the tuberculosis bacilli are likely to be inactivated by the bile before they become predominant in the liver. Hepatic tuberculosis (HTB) spreads mostly under the condition of hypoimmunity of the organism. HTB shows a variety of CT imaging patterns; depending on 1) the pathways the tuberculous bacilli enter the liver, 2) the patient's immune status, and 3) the virulence of the pathogenic bacteria. In western countries, immunosuppression induced by human immunodeficiency virus (HIV) infection increases the opportunity of potential tuberculosis infection which would become active when the patient's mononuclear phagocyte system was depressed [4-6]. Therefore, AIDS is one of the most important causes for the primary hepatic TB in the western countries. In our study, none of the patients are HIV positive. Low income, poor access to healthcare, and presence of a concomitant tumor are main contributing factors. In our study, 7/14 patients were living in the rural areas, and 2/14 suffered from malignant tumors. The others suffered from severe pulmonary TB and weakness. CT signs of HTB documented in literature were mostly in terms of case reports. In this study, a wide variety of CT manifestation of HTB would be presented. The purpose of this article is to describe the CT manifestations of Hepatic Tuberculosis in adult non-AIDS infectious patients, and to discuss corresponding differential diagnosis.

Materials and methods

14 patients (8 males and 6 females) from two institutions were involved in our study. Their

Table 1. Medical information of patients

No./Sex/Age	Living condition	Main complaints/clinical signs and symptoms	The history of TB	Chest X-ray (CXR)	Blood sedimentation rate (mm/min)
1/M/37	Countryside	5 weeks after space-occupying lesion found in the liver by ultrasound in health examination	NO	-	5/30; 12/60
2/M/48	Countryside	17 years after liver tumor excision, 9 months of recurrent fever	NO	+	30/60; 58/120
3/M/30	City	8 days of fatigue and fever	NO	+	25/60
4/F/73	Countryside	More than 1 month of upper abdominal hunger-like pain	NO	-	10/60
5/M/25	Countryside	1 year of recurrent low-grade fever, 10 months of cough and stethocatharsis	YES	+	35/60
6/M/64	Countryside	More than 3 months of recurrent low-grade fever	NO	-	28/60; 55/120
7/M/50	Countryside	20 days of upper abdominal pain and distending pain in the right rib area	YES	+	42/120
8/F/52	Countryside	3 months of recurrent cough accompanied by fever, fatigue, loss of appetite and weight loss	YES	+	30/60
9/M/68	City	2 months of persistent fever, night sweat, gradual weight loss	YES	+	25/60; 54/120
10/M/67	City	More than 6 months of sub-xiphoid dull pain and uncomfortability	YES	+	28/60; 40/120
11/F/40	Countryside	Infection more than 3 months	NO	-	25/80
12/F/45	Countryside	Alp (+)	YES	+	35/85
13/F/63	Countryside	More than 6 months of recurrent low-grade fever	NO	-	30/120
14/F/54	Countryside	More than 3 months of recurrent low-grade fever	NO	-	15/110

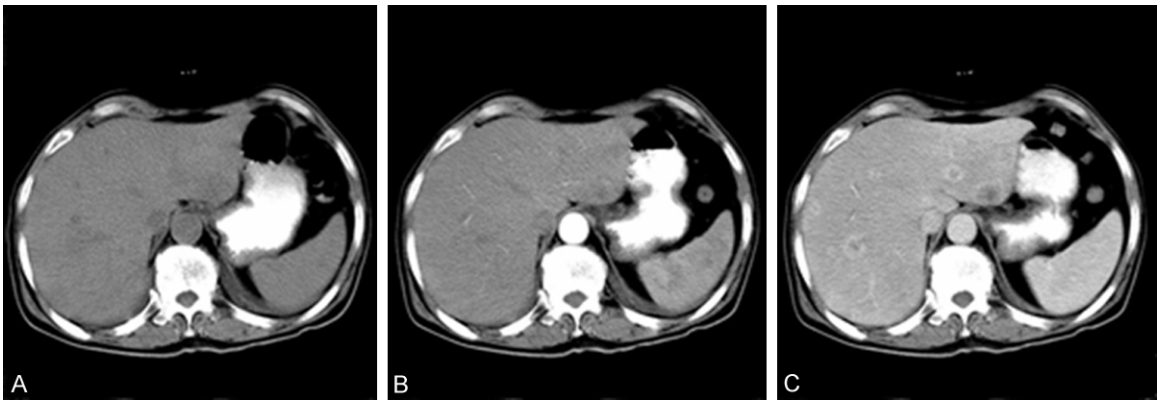

Figure 1. 58-year-old woman with Miliary HTB. (A, B) Liver CT at presentation in plain scanning (A) shows multiple wild low density lesions in liver, and in arterial phase (B) the lesions shows no enhancement. (C) In portal phase shows rim enhancement of the lesions.

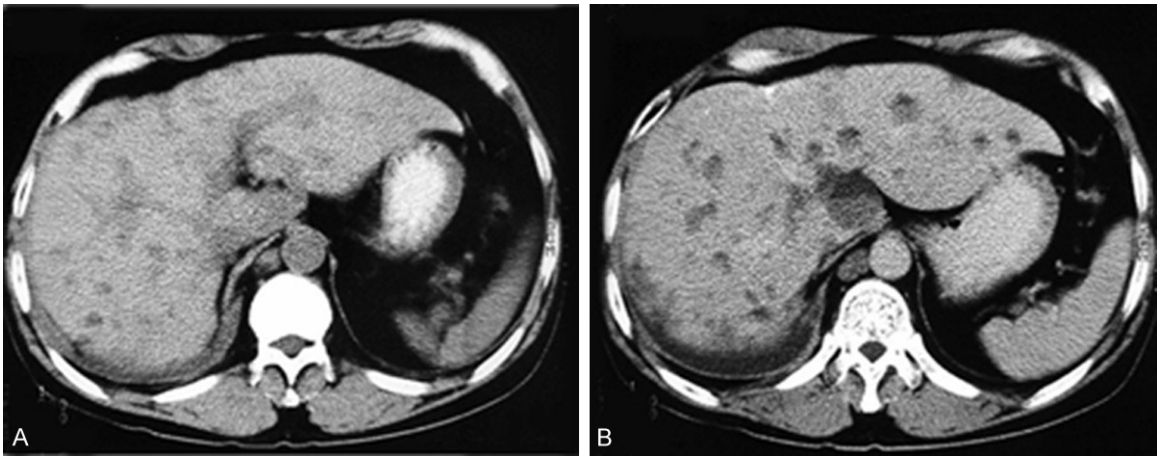

Figure 2. 42-year-old woman with Miliary HTB. A. Liver CT at presentation in plain scanning shows multiple wild low density lesions in liver. B. No enhancement was found after contrast agents were administrated.

complete medical history between 1992 and 2004 were collected. The patients' ages ranged from 25 to 73, and the median age was 51 years. The medical information and history of our collection showed on **Table 1**.

The CT examination evaluated for this study was performed on helical CT scanner (Somatom Plus 4, Siemens Medical Solutions). Oral and i.v. contrast material were administered to all patients. The parameters used in this study were as follows: 5-mm collimation; 5-mm reconstruction interval; 1:1 pitch; slice thickness: 5-10 mm; 120 kV/225 mAs. Non-ionic contrast agents, Iohexol, 300 mg I/ml (Amersham Health, GE Health Care) or Iopromide 300 mgI/ml or 350 mgI/ml (Bayer Schering Pharma Division, Bayer HealthCare Company Limited) were used. With a high pressure syringe (Medrad, Inc. USA); intravenous bolus injection of 75 to 100 ml contrast medium was performed with the FLR 2.0-3.0 ml/s (adjusted according to the patient's body weight and tolerance). Hepatic Artery Phase (HAP): 22-25 s; Portal Venous Phase (PVP): 45-50 s; balanced phase: 90-120 s. Four patients were scanned with two phases, i.e. HAP and PVP; while eight patients with 3 phases.

Results

All HTB cases were categorized into seven types. The CT findings were shown as followed:

Miliary HTB in both cases were secondary; similar-sized nodules were widely spread in the whole liver with no distribution trend. Plain

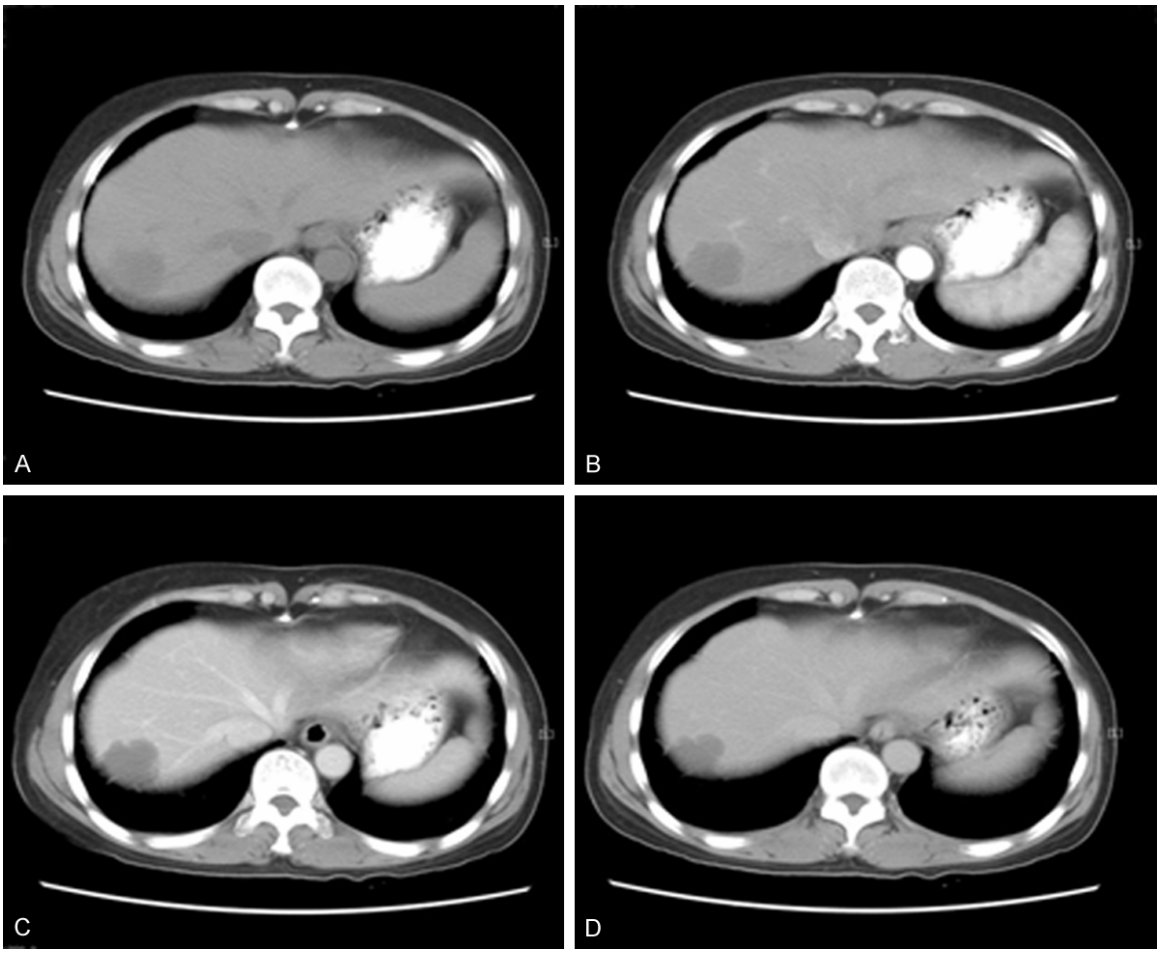

Figure 3. 54-year-old man with Solitary NHTB-Tuberculoma. (A) Liver CT at presentation in plain scanning shows solidary nodular in right lobe of liver with low density. (B-D) Contrast-enhanced scanning shows slight enhancement in HAP (B), and clearer border was presented in PVP (C), No more apparently delayed enhancement in the marginal area of the nodules (D).

scanning CT (CT value 35-48 Hu) demonstrated that multiple miliary low-attenuation lesions were clear-bordered and could be easily differentiated from normal hepatic tissue (**Figure 1**).

In the PVP, Case 1 showed rim enhancement, of which CT value was higher than that of hepatic parenchyma (**Figure 1**). For Case 2, no enhancement was found after contrast agents were administrated in either HAP or PVP (**Figure 2**). CT value of the central necrosis increased by a few units, but it was lower than 35 Hu. An abscess was formed when some of the subcapsular miliary tubercles combined to form clusters and some exchange occurred. "Layerization" phenomena could be seen in the abscess cavity (**Figure 9**), and fluid-fluid levels (FFLs) were seen inside the lesion. No obvious enhancement was observed after dynamic contrast enhanced. Anterior-inferior pericardial lymph nodes showed obvious swelling. Punctate calcification was seen inside the swollen lymph nodes (**Figure 9**). The swollen lymph nodes appeared as circular enhancement after contrast agent was administrated.

Nodular HTB were quite different from miliary HTB, the lesions were differently sized, most of which spread in the peripheral parenchyma of the liver, even part of them located in subcapsular regions of the liver. The lesions were attached on the liver edge with obtuse angle. For most of the nodular HTB, single or multiple focal low-density areas were seen using plain scanning. Multiple nodules combined and the borders became a less defined but could still be distinguished from the normal hepatic tissue. Some lesions of isodensity showed no

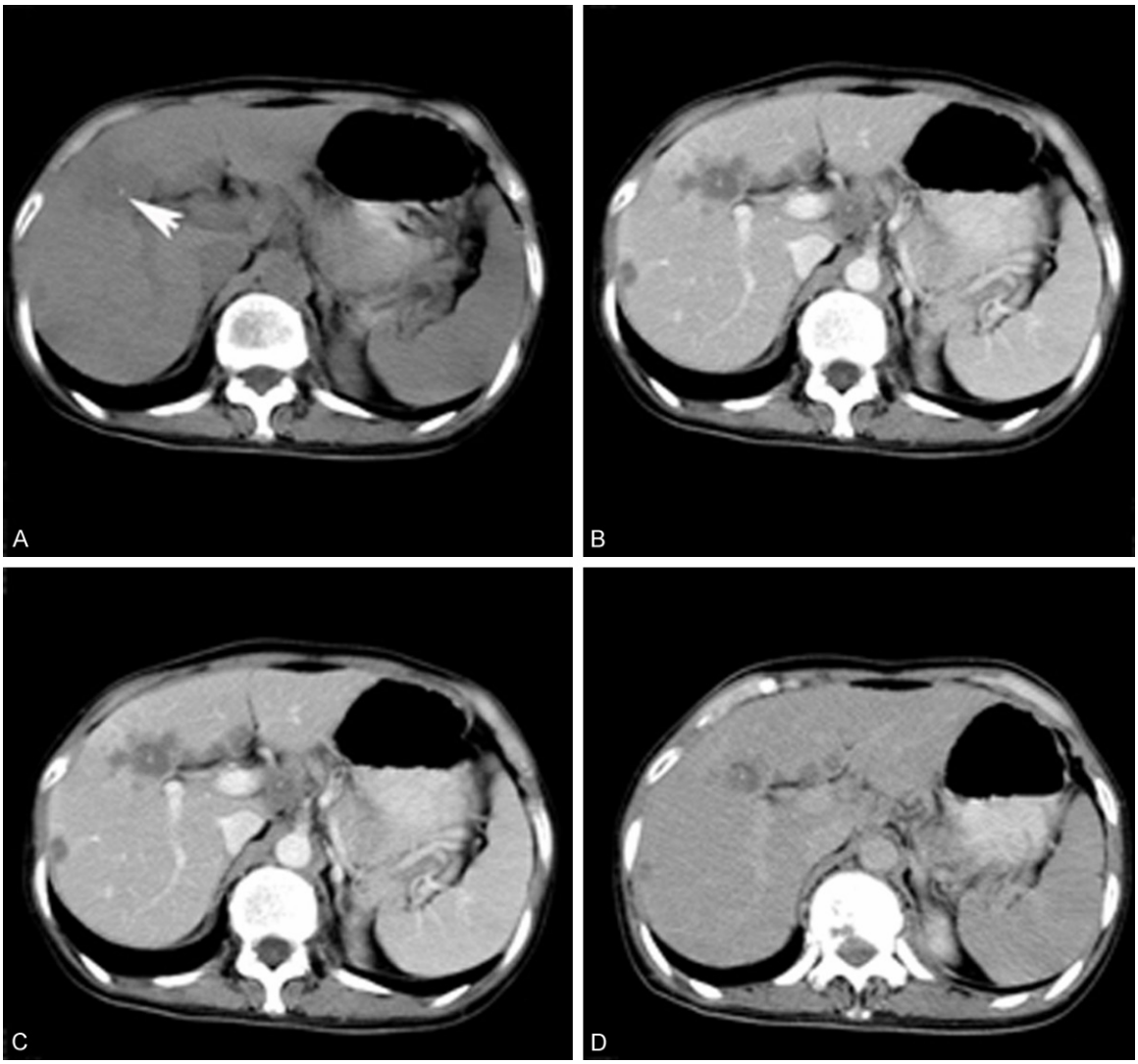

Figure 4. 37-year-old man with Nodular HTB (multiple NHTB). (A) Liver CT at presentation in plain scanning shows multiple wild low density lesions in liver, Note high density powder-like calcification in the central area (arrow head). Contrast-enhanced scanning shows slight enhancement in HAP (B), and clearer border was presented in PVP (C). In the parenchymal phase, a filling-in enhancement was observed (D).

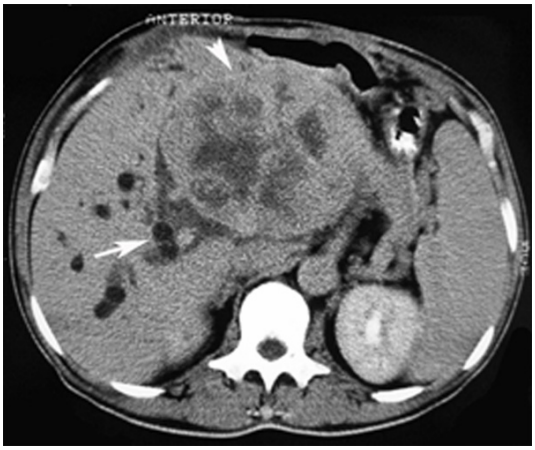

Figure 5. 48-year-old man with Pseudo-tumoral HTB. Liver CT at presentation in arterial phase shows iso-density lesion with massive low-density necrosis in the centre in left lobe of liver, and Intrahepatic bile ducts were greatly dilated due to the compression against the bile ducts in the portal area (arrow). Note mild-moderate enhancement of the pseudo capsule (arrow head).

apparent difference compared to the normal hepatic tissue. Therefore, it was difficult to identify them using only plain scanning. Contrast-enhanced scanning showed no enhancement or slight enhancement in HAP, gradually enhanced further in PVP, and more

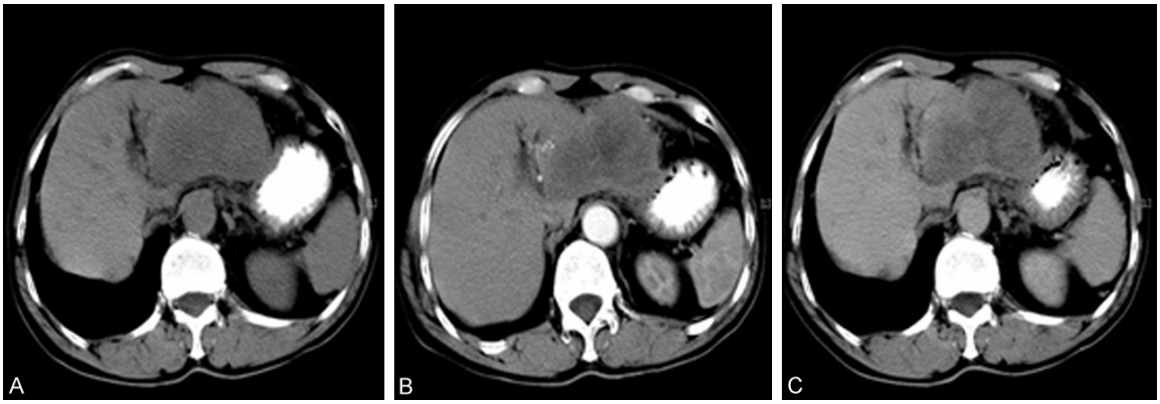

Figure 6. 30-year-old man with Pseudo-tumoral HTB. (A) The plain CT shows the lesion in left lobe of liver with homogeneous low-density. (B, C) The tumor body presented slight continuous enhancement during both contrast-enhanced in HAP (B) and PVP.

apparently delayed enhancement in the marginal area of the nodules (**Figure 3**). During HAP, there was abnormal blood perfusion showing hyperdensity in the shape of a wedge or triangle. Some nodules were mixed-density lesions: higher density in the central areas associated with powder-like calcification with CT Value about 90 Hu (**Figure 4**). In the parenchymal phase, a filling-in enhancement was observed.

Pseudo-tumoral HTB (2 cases). In each of cases, a large solitary mass was observed in the left lobe of liver (**Figures 5**, **6**). The enhanced CT showed the lesion was iso-density with massive low-density necrosis in the centre in one case (**Figure 5**). The nodule demonstrated significant mass effect and it caused the deformation of the liver. In another case, the plain CT showed the lesion was low-density, and during both contrast-enhanced HAP and PVP, the tumor body presented slight continuous enhancement. In terms of pseudo capsule, there was one case with mild-moderate enhancement of the pseudo capsule (**Figure 5**), and the other without (**Figure 6**). In **Figure 5**, intrahepatic bile ducts were greatly dilated due to the compression against the bile ducts in the portal area.

Cholangitic HTB. The patient previously had a cholecystectomy combined with choledochojejunotomy. Plain scanning revealed great dilation of intrahepatic bile ducts, which were filled with materials of soft-tissue density, particularly in the central bile ducts. There was no obvious dilation in distal bile ducts. In HAP, the mass filling in the bile ducts was enhanced slightly and further enhanced during PVP. In delayed phase, its density was quite similar to that of the normal hepatic tissue (**Figure 7**).

Serosal HTB. On plain CT, the local serosa around left lobe was greatly thickened with calcified shell pressing against the surface of the liver in a curvilinear pattern, leading to an impression. The lesions showed no enhancement after contrast agent administrated (**Figure 8**).

Mixed-type HTB. One case suffered from serositis associated with a large pseudo-tumoral nodule in the left lobe of liver. The other case had serositis combined with intrahepatic miliary HTB (**Figure 9**).

False negative HTB. This type is very rare. Abnormal density was not found in either plain or contrast-enhanced scanning (**Figure 10**). However, on the image of F^{18}-FDG-PET-CT, there were hypermetabolic lesions with high SUV value, distributing in the whole liver, which was identified as HTB by liver biopsy.

Discussion

Etiology: HTB could be classified as primary and secondary HTB. The primary HTB was defined as no TB found in any other part of the body except the liver or only slightly inactive tuberculosis with systematic and/or local symptoms of tuberculosis such as fever, shiver, night sweats, hepatic pain, hepatomegaly and jaundice etc [7-9]. The secondary HTB defined as

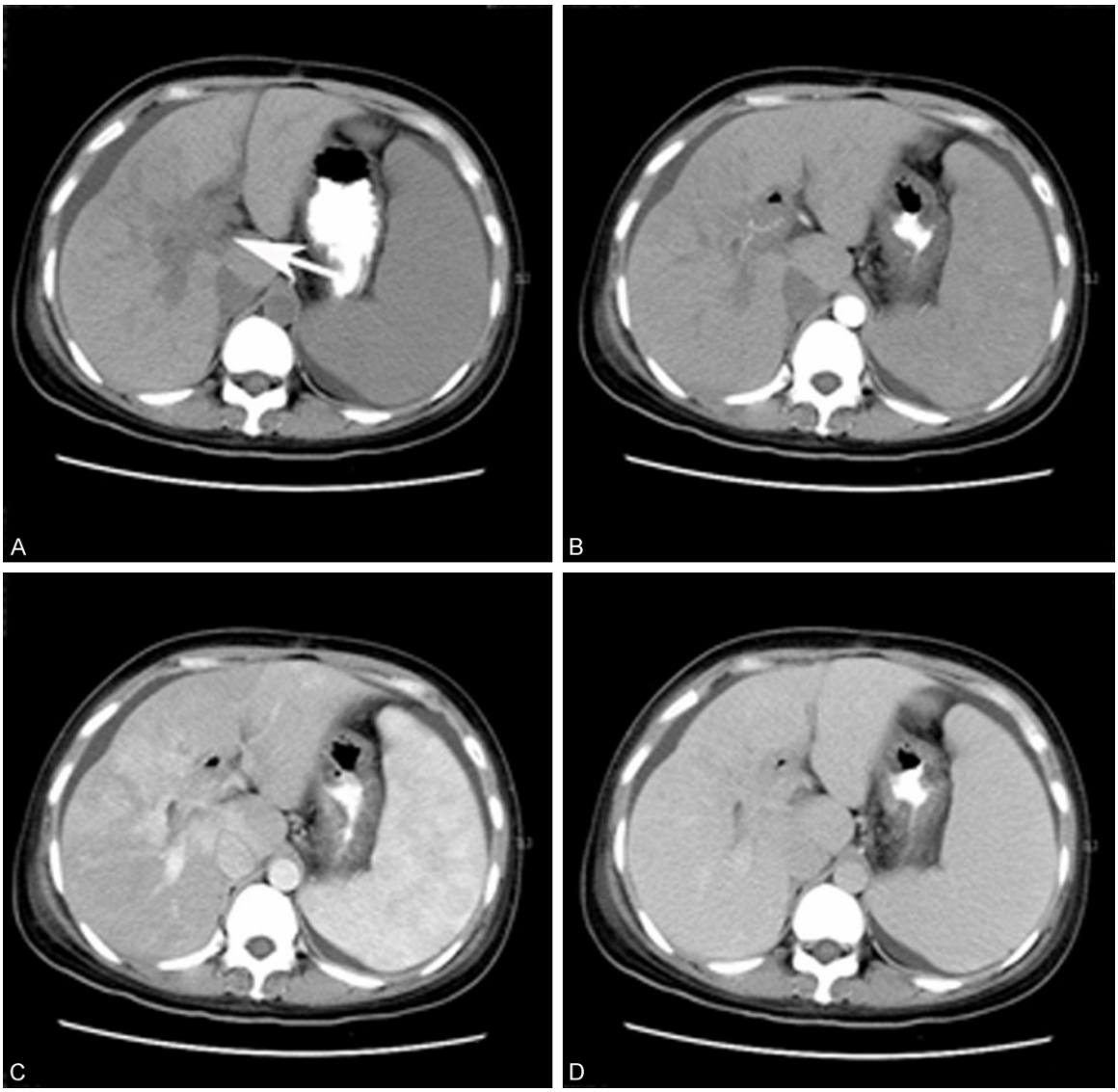

Figure 7. 73-year-old woman with Cholangitic HTB. A. Plain CT shows great dilation of intrahepatic bile ducts which were filled with materials of soft-tissue density (arrow) in the central bile ducts. B, C. The mass filling in the bile ducts shows heterogeneous enhancement slightly in HAP, and further enhanced in PVP. D. In delayed phase, its density was quite similar to that of the normal hepatic tissue.

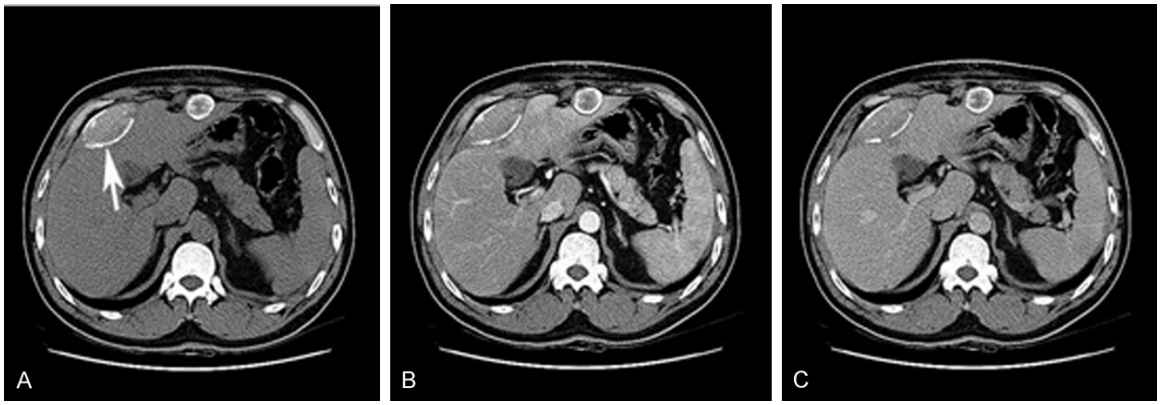

Figure 8. 25-year-old man with Serosal HTB. A. Plain CT shows the thickened serosa around local left lobe of liver with calcified shell (arrow). B, C. The lesions showed no enhancement after contrast agent administrated.

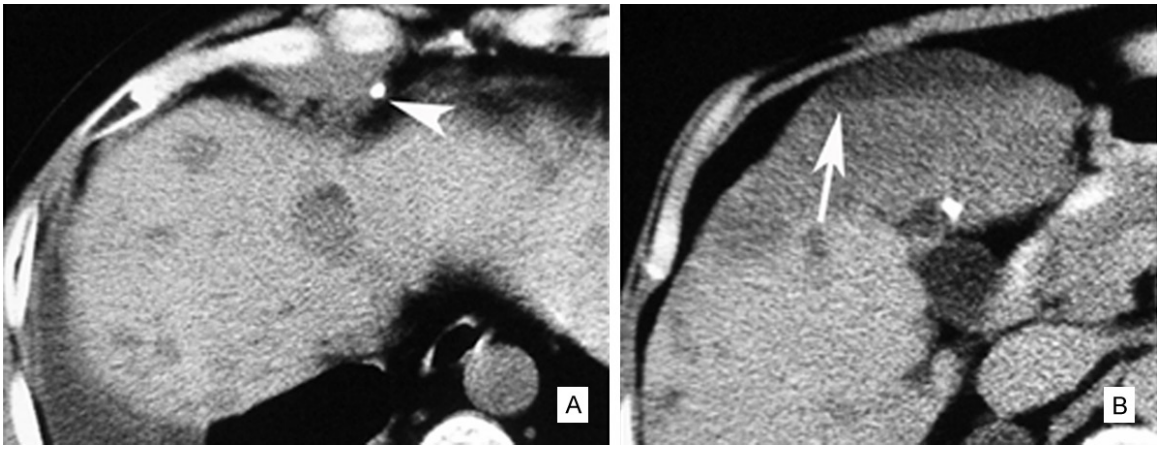

Figure 9. 52-year-old woman with Mixed-type HTB. A. Anterior-inferior pericardial lymph nodes showed obvious swelling. Punctate calcification (arrow head) was seen inside the swollen lymph nodes. B. "Layerization" phenomena could be seen in the abscess cavity (arrow).

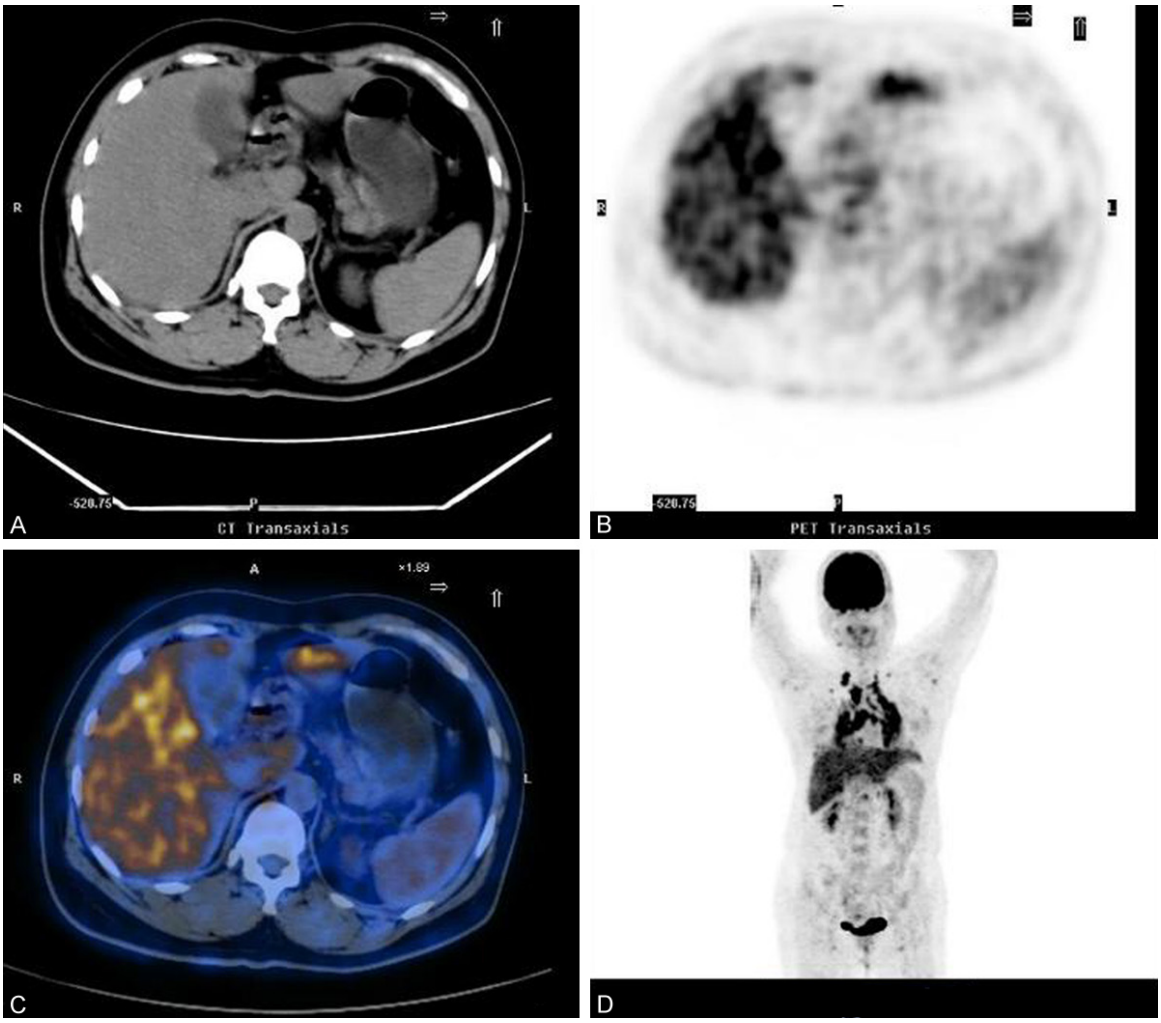

Figure 10. 50-year-old man with Pseudo-negative HTB. (A) CT scan shows no abnormal density in liver. (B-D) F^{18}-FDG-PET-CT shows hypermetabolic lesions with high SUV value. Note yellow represents high SUV value on merge PET-CT image (C).

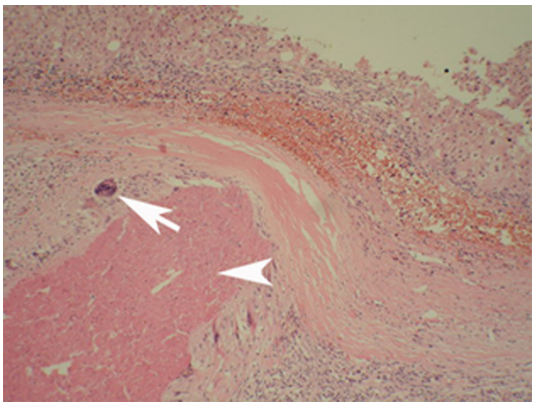

Figure 11. Histologic picture of HTB shows Langhans cell (arrow head) in FOV and caseous necrosis (arrow). HE × 40.

Liver involvement in tuberculosis, though both in pulmonary and/or extra-pulmonary tuberculosis are to see frequently relatively. The primary cause of TB, mycobacterium tuberculosis, is an acid-fast aerobic bacterium and classified as a Gram-positive bacterium [10]. Due to several factors such as the mononuclear phagocyte systems in the liver, the excellent capability of regenerating and repairing of the hepatic tissues, the bile which can inhibit the growth of bacteria, the sufficient blood flow and fatty acids, it is not common for tubercle bacilli to accumulate and multiply. However, the HTB could occur due to immune-suppression [11]. Therefore, HTB should be taken into consideration if there are one or more of the following clinical symptoms or biochemical findings [5]: Fever of unknown origin (FUO); Upper abdominal pain; Weight loss; Night sweats; Fatigue; Anemia; With/without hepatomegaly and hepatic dysfunction; Increased blood sedimentation rate (BSR); Increased alkaline phosphatase (AKP); Purified protein derivative positive (PPD+); Space-occupying lesions in the liver found by image logical methods.

Recently, it has been reported that the incidence of HTB significantly increased in patients with HIV, which suggests that the cell-mediated immunity would be of great importance to the onset of HTB. However, there was no evidence of HIV infection in all of our patients. The reason for that was not very clear. Low income, poor healthcare in the rural areas and the presence of a tumor might be vital etiological factors.

There are five pathways of how tubercle bacilli became widespread and disseminate [12, 13]: (1) hematogenous dissemination: If TB spreads through the hepatic artery, it is more likely to result in intrahepatic miliary HTB [14]. The hepatic artery is the major route for TB to reach the liver because usually HTB is secondary to the hematogenous dissemination of TB. However, if TB enter the liver through the portal vein, it is more likely to distribute as local lesions in the liver. In that case, tubercle bacilli coming from GI tract (like intestinal TB, mesenteric TB) often take this route, resulting in HTB; (2) maternal-neonatal transmission: The major cause of congenital HTB; (3) lymphatic spread: In some cases in our study, the enlarged lymph nodes were seen in the portal area or cardiophrenic angle, associated with calcification, indicating TB had spread via lymphatic system; (4) direct invasion: It is very rare. In Cases 11, nodular HTB was the result of direct invasion from TB of abdominal wall via peritoneum; (5) bile ducts spread: In cholangitic HTB, the patient who suffered from tuberculous cholangitis had a history of intestinal TB for many years before cholangio-colic fistula occurred, where the TB bacilli invaded the bile ducts, leading to tuberculous cholangitis.

Classification of HTB: there are no standard criteria for the classification of HTB according to current literature. Combining pathological classification with the imageological characteristics, it would be reasonable to classify HTB into 7 types: miliary HTB [15], nodular HTB [16], Pseudo-tumoral HTB [17], cholangitic HTB, serosal HTB, mixed-type HTB [8], and false negative HTB [18]. But it is debatable whether tuberculous peritonitis could be classified as another type of HTB.

The comparison between CT manifestations and pathological findings [13, 19, 20]: HTB is not a common disease so that it has been rarely reported, especially in recent year. Histological diagnostic criteria of HTB are at least one Langhans cell found in every field (**Figure 11**) examined under the SEM accompanied by caseous necrosis. The collected information of miliary HTB, nodular HTB and pseudotoumor HTB in this study showed different pathological findings. It is the individual immunity and the toxicity of pathogenic bacteria that different types of HTB show the various imageological features. Under the SEM, the foci of miliary HTB were mainly distributed in hepatic lobules, revealing micro-focal liquation and

necrosis. Due to the low attenuation coefficient (μ) of necrotic materials, plain scanning often shows a predominant well-defined diffuse hypodense lesion. No enhancement was found after enhanced scanning because there were no apparent vessel structures. Small miliary tubercles combined to form clusters and some exchange occurred.

Caseous necrosis and liquation were seen to form tuberculous hepatic abscesses [1]. Fluid-fluid levels were observed inside of the abscess cavity and no significant enhancement was found with contrast-enhanced dynamic CT scans [21]. However, once the peripheral inflammatory granuloma occurred, the blood supply would cause rim enhancement of lesions.

In terms of nodular HTB, caseous necroses were found in the centre of lesions under the SEM, surrounded by inflammatory granuloma. Caseous materials are low in water and high in protein, therefore the μ value of caseous materials is comparable to that of granuloma, which is only slightly lower than that of the normal hepatic tissue. Moreover, the μ value of caseous materials is close to that of the hepatic tissues with diffuse fatty degeneration. Consequently plain scanning often shows that the lesions are not well-defined, slightly low-dense or iso-dense so that sometimes it is difficult to identify the iso-dense lesions. Nevertheless, after contrast agents were injected, many unobserved lesions could be seen and the calcification would be one of the most remarkable findings of HTB. Caseous materials are acidic and not easily liquefied or absorbed, so they can last a long time. After immunity is improved or medical treatment is given, the metabolic ability of tubercle bacilli decreases and reproductive capacity is weakened, the lesions lose water and become dry, calcium carbonate and calcium phosphate deposit resulting calcification. In the early stages, calcification appears in the centre of the lesions. The typical CT finding is "central powder-like calcification", and the calcification is a marker that shows the inflammation starts to disappear. In the HAP of contrast-enhanced scanning, there was abnormal blood perfusion in the shape of wedge or triangle, indicating the blood supply in those areas came from hepatic artery instead of portal vein as would normally be expected. There might be a slight enhancement in the central area, probably because of the central veins of lobes encased or partial volume effect.

Additionally, it has been reported that the lesions of HTB are usually located in the marginal areas of the liver and the features of nodular HTB observed in this study were consistent with the literature. Pseudo-tumoral HTB is a very rare clinical disease. It depicts that huge solitary intrahepatic mass, surrounded by fibro-pseudocapsule and disseminated satellite lesions. Under the SEM, a large area of tuberculous granuloma was seen accompanied by central caseous necrosis which was iso-dense with large area of low density in the centre as revealed by CT plain scanning. Contrast-enhanced CT scan showed slight enhancement of the tumor body in both HAP and PVP, and slight to moderate enhancement of the capsule. 2 miliary HTB were associated with multiple intra-mediastinal swollen lymphonodes, suggesting that tubercular bacilli transferred via lymphatic pathway to reach the liver as the same time. Spot-like calcification were seen in some lymphonodes, and the swollen lymphonodes showed ring-shaped enhancement by contrast- enhanced scanning.

Differentiation Diagnosis: (1) differentiating features of HTB with primary hepatocellular carcinoma: solitary hepatic tuberculoma is very rare and easily misdiagnosed as primary hepatocellular carcinoma. Main difference between these two diseases: ① in HAP of contrast-enhanced scanning, hepatic tuberculoma more likely showed no significant enhancement, while apparent enhancement was found with primary hepatocellular cancers, characterized by "fast wash-in and fast wash-out, because most blood supply of the tumors comes from the hepatic artery [22]; ② the capsule of hepatic solitary tuberculoma were always very thin, smooth and glossy, while the pseudo-capsule of hepatocellular cancers are uneven and thick without clear borders, as the pseudo-capsule were formed by the proliferation and fibrosis of the surrounding hepatic tissues pressed by the tumor; ③ α-fetoprotein (AFP) tests were positive in approx. 70%-90% of patients suffering from primary hepatocellular cancers, and always accompanied by portal onco-epistom. In terms of hepatic tuberculoma, AFP tests

were negative and no carcinoma epistom found. (2) Differentiating features of HTB with metastasis in the liver: miliary and nodular HTB were likely to be misdiagnosed as hepatic metastasis as the 5 cases in this study. The medical history of primary cancers is one of the key points in differentiation diagnosis, though "central power-like calcification" of nodular HTB could assist to identify HTB. (3) Differentiating features of HTB with multiple angiomatosis in the liver: multiple angiomatosis are featured by "fast wash-in and slow wash-out" when dynamic contrast-enhanced scanned. However, nodular HTB showed apparent ring-shaped enhancement in PVP. (4) Differentiating features of HTB with hepatic abscess: the CT values of tuberculous and those of bacterial hepatic abscess were too close to differentiate. Nevertheless, the borders of bacterial hepatic abscess were always smooth and glossy. As tuberculous hepatic abscess were often formed by nodes/nodules merging, the borders were more likely irregular and cluster-shaped nodular shadow could be seen. More apparent peripheral enhancements were seen in bacterial hepatic abscess. Moreover, clinically, bacterial hepatic abscess were always interactive with systematic bacteremia, resulting in severe clinical symptoms. Patients suffering from tuberculous hepatic abscess were more likely hypo-immunity so that no typical clinical symptoms were found.

Our study is limited by a small sample size of HTB and there might be some other unusual HTB such as tuberculous pylephlebitis reported in some literature, which have not been included in this study. It's also necessary to use low dose CT scanning protocol in our future study [23].

In summary, we described seven distinct CT manisfestations of HTB. It is difficult to prospectively diagnose HTB due to its various CT presentation and potential mimics. "Central power-like calcification" of HTB could assist to identify nodular HTB.

Acknowledgements

2014 Hainan Province of Social Development of Science and Technology Special Projects (No. SF201428). 2014 Sanya Medical and Health of Science and Technology Innovation Projects (2014W37).

Disclosure of conflict of interest

None.

Address correspondence to: Dr. Yang Li, Department of Radiology, Chinese People's Liberation Army General Hospital, 28 Fuxing Road, Haidian District, Beijing 100853, China. Tel: +86 10 66939564; Fax: +86 10 66939564; E-mail: YangLidoc@126.com

References

[1] Limaiem F, Gargouri F, Bouraoui S, Lahmar A, Mzabi S. Coexistence of Hepatocellular Carcinoma and Hepatic Tuberculosis. Surg Infect (Larchmt) 2014; 15: 178-85.

[2] Wu Z, Wang WL, Zhu Y, Cheng JW, Dong J, Li MX, Yu L, Lv Y, Wang B. Diagnosis and treatment of hepatic tuberculosis: report of five cases and review of literature. Int J Clin Exp Med 2013; 6: 845-50.

[3] Wong SS, Yuen HY, Ahuja AT. Hepatic tuberculosis: a rare cause of fluorodeoxyglucose hepatic superscan with background suppression on positron emission tomography. Singapore Med J 2013; 3: 223-7.

[4] Dursun I, Yikilmaz A, Poyrazoglu H, Soyuer I, Gunduz Z. Radiological findings of isolated hepatic tuberculosis in a child with tubulointerstitial nephritis-uveitis syndrome. Pediatr Radiol 2009; 39: 302-5.

[5] Fradet V, Gaudreau C, Perrotte P, Cote J, Paquin JM. Management of hepatic granulomatous tuberculosis complicating intravesical BCG for superficial bladder cancer. Can Urol Assoc J 2007; 1: 269-72.

[6] Lawn SD, Wood R. Hepatic involvement with tuberculosis-associated immune reconstitution disease. AIDS 2007; 21: 2362-63.

[7] Gastrad J. Hepatic tuberculosis. Rev Med Fr 1960; 41: 125-29.

[8] Pia Iglesias G, Garrido Sanjuan JA, Fandino Orgeira JL, Grandes Ibanez J, Gonzalez Moraleja J. Hepatic granulomas, giant-cell arteritis and miliary tuberculosis. An Med Interna 1999; 16: 47-53.

[9] Sheen-Chen SM, Chen MC, Hu TH, Eng HL, Chen WJ, Cheng YF, Lee TY. Computed tomography and angiography in hepatic tuberculosis mimicking liver tumor. Int J Tuberc Lung Dis 2001; 5: 876-8.

[10] de Souza AF, de Oliveira e Silva A, Baldi J, de Souza TN, Rizzo PM. Hepatic functional changes induced by the combined use of isoniazid, pyrazinamide and rifampicin in the treatment of pulmonary tuberculosis. Arq Gastroenterol 1996; 33: 194-200.

[11] Gupta S, Meena HS, Chopra R. Hepatic involvement in tuberculosis. J Assoc Physicians India 1993; 41: 20-2.

[12] Kielhofner MA, Hamill RJ. Focal hepatic tuberculosis in a patient with acquired immunodeficiency syndrome. South Med J 1991; 84: 401-4.
[13] Farooq Sheikh AS, Qureshi IH, Saba K, Bukhari MH. Primary isolated hepatic tuberculosis. J Coll Physicians Surg Pak 2013; 23: 359-61.
[14] Asada Y, Hayashi T, Sumiyoshi A, Aburaya M, Shishime E. Miliary tuberculosis presenting as fever and jaundice with hepatic failure. Hum Pathol 1991; 22: 92-4.
[15] Tritou I, Prassopoulos P, Daskalogiannaki M, Charoulakis N, Papakonstantinou O, Gourtsoyiannis N. Miliary hepatic tuberculosis not associated with splenic or lung involvement. A case report. Acta Radiol 2000; 41: 479-81.
[16] Kok KY, Yapp SK. Isolated hepatic tuberculosis: report of five cases and review of the literature. J Hepatobiliary Pancreat Surg 1999; 6: 195-8.
[17] Diouf ML, Dia D, Ka EH, Diouf B, Mbengue M, Moreira-Diop T. Hepatic tuberculosis of the pseudotumoral form. Dakar Med 1999; 44: 123-5.
[18] Wang X, Shi X, Yi C, Chen Z, Zhang B, Zhang X. Hepatic tuberculosis mimics metastasis revealed by 18F-FDG PET/CT. Clin Nucl Med 2014; 39: e325-7.
[19] Musumba C, Dey D, Richardson P. Atypical presentation of miliary tuberculosis with hepatic involvement early after renal transplantation. Clin Gastroenterol Hepatol 2013; 11: e52-3.
[20] Singh S, Jain P, Aggarwal G, Dhiman P, Sen R. Primary hepatic tuberculosis: a rare but fatal clinical entity if undiagnosed. Asian Pac J Trop Med 2012; 5: 498-9.
[21] Turkel Kucukmetin N, Ince U, Cicek B, Akman H, Boztas G, Tozun N. Isolated hepatic tuberculosis: A rare cause of hepatic mass lesions. Turk J Gastroenterol 2014; 25: 110-2.
[22] Rabhi M, Ennibi K, Berady S, Chaari J, Toloune F. Acquired ichthyosis revealing hepatic and lymph node tuberculosis. Presse Med 2006; 35: 1839-41.
[23] Zhang F, Yang L, Song X, Li YN, Jiang Y, Zhang XH, Ju HY, Wu J, Chang RP. Feasibility study of low tube voltage (80 kVp) coronary CT angiography combined with contrast medium reduction using iterative model reconstruction (IMR) on standard BMI patients. Br J Radiol 2016; 89: 20150766.