

Case Report

A case of tuberculosis presented by obstructive jaundice tuberculosis-related mechanical icterus

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Abstract

Obstructive jaundice caused by tuberculosis lymphadenitis is a rare condition. It can mimic clinical and radiological findings of hepatobiliary malignancies. The authors report a 24-year-old male patient who presented with abdominal pain, fever and jaundice for the last two weeks. It was found that cholestasis enzymes were increased by 2-3 fold and direct bilirubin was 6.13 mg/dL. Imaging studies revealed conglomerated lymph nodes with some cavitory lesions and dilated intrahepatic biliary canal secondary to compression by the lymph nodes. Tuberculosis was found to be positive in the polymerase chain reaction analysis of the aspirate that was obtained in the guidance of imaging studies. *M. tuberculosis* complex was isolated from mycobacterial culture. Anti-tuberculosis treatment was initiated. Clinical, laboratory and radiological findings completely resolved by medical therapy alone. Tuberculosis lymphadenitis should be kept in mind in cases presenting with obstructive jaundice in endemic areas and interventional diagnostic techniques should be preferred in eligible patients.

Key words: Tuberculosis lymphadenitis; obstructive jaundice; radiological findings; interventional diagnostic techniques.

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Introduction

Abdominal tuberculosis is rare and comprises 3.5-4% of all extra-pulmonary tuberculosis cases. Abdominal tuberculosis is generally seen in 4 forms: tuberculosis lymphadenitis (TLAP), peritoneal tuberculosis, gastrointestinal tuberculosis and visceral tuberculosis involving solid organs. In general, patients present with a combination of these forms [1-4].

Obstructive jaundice secondary to tuberculosis can occur due to inflammatory stricture of the main biliary duct, compression by TLAP, enlargement of pancreas head due to tuberculosis, and retroperitoneal abscess of tuberculosis [5]. TLAP is most commonly seen form of abdominal tuberculosis and it generally follow drainage pathway of involved organs; however, it may involve any lymph node. Most commonly seen tuberculosis lymphadenopathies are mesenteric nodes, omental nodes, those at porta hepatis, those along celiac axes and those localized at peripancreatic area [6].

Here, we discussed a patient presented with obstructive jaundice caused by TLAP and diagnosed by

interventional radiology methods, resulting in complete recovery with early medical treatment.

Case presentation

A 24-year old man presented to our hospital with new-onset jaundice. He had abdominal pain and night sweating for the past one month. Lasting 13 days fever was occur. The patient had no history of chronic disease. No alcohol consumption or smoking. With no family history. The patient was admitted for further evaluation.

Physical examination

The patient had icteric appearance. There was tenderness at right upper quadrant with negative Murphy sign. There were inguinal and axillary lymphadenopathies < 1cm in size.

Laboratory evaluations

Hemoglobin, 10.5 mg/dL; platelet count, 387,000/ μ L; leukocyte count, 14,600/ μ L; serum total

bilirubin, 4.77 mg/dL; direct bilirubin, 3.81 mg/dL; serum AST, 86 IU/Lt; serum ALT, 112 U/Lt; AST, 137 U/Lt; ALP, 464 U/Lt; and GGT, 534 U/Lt. Other biochemical parameters were within normal range (Table 1). Serology revealed no abnormal finding: anti Hbc IgM (-); anti HIV (-); anti CMV IgM (-); anti HAV IgM (-); and Brucella agglutination (-).

Radiological evaluation

On hepatobiliary sonography, gallbladder appeared as contracted with wall thickness of 4 mm and dilated intrahepatic biliary duct. Common bile duct was measured as 11 mm and there was a stone (9 mm in size at proximal). Multiple lymphadenopathies were detected at porta hepatis as largest being 27×15 mm in size. On abdominal CT scan with contrast enhancement, a lobulated collection area with septa (55×40 mm in size) was detected in hepatic segment 4 and there were fluid and small lymph nodes at hepatic hilus and peripancreatic area (Figure 1). On MRCP, conglomerated lymphadenopathies with some cavitary lesions (largest being 5 cm in size) were detected at portal hilus, celiac axis and para-aortocaval area with dilated intrahepatic biliary duct secondary to compression by lymphadenopathy and collapsed gallbladder. Because malignancy was suspected, 18-fluoro-2-deoxyglucose positron emission tomography (18F-FDG PET) was taken, a mass lesion with necrotic centre and intensive hyper-metabolism at peripheral areas in the junction of hepatic segments 2 and 3 in addition to multiple hyper-metabolic lymphadenopathies at mediastinum, bilateral supraclavicular lymphatic area and abdomen were detected (Figure 2).

Figure 1. The mass lesion with necrotic centre and intensive hyper-metabolism at peripheral areas in the junction of hepatic segments 2 and 3.

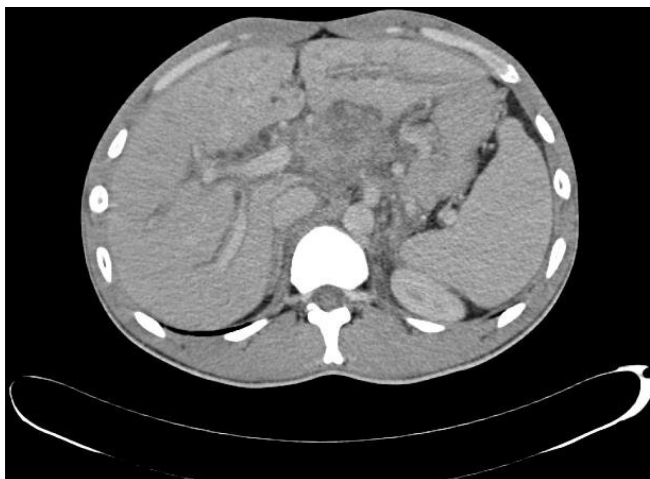


Figure 2. Multiple, hyper-metabolic lymphadenopathies at mediastinum, bilateral supra-clavicular lymph node areas and abdomen.

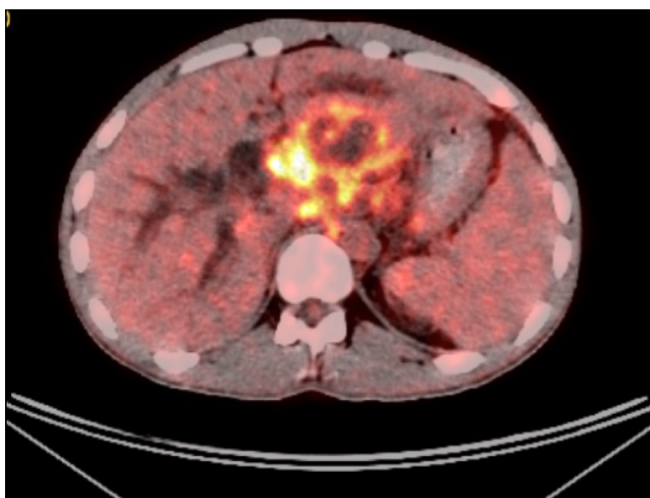


Table 1. Laboratory findings.

Following days	1.	5.	8.	15.	20.	30.	55.	90.	Normal range
AST	86	137	82	42	28	24	23	25	< 37 IU/Lt
ALT	112	157	110	57	39	31	23	27	< 42 IU/Lt
GGT	441	534	725	534	166	75	22	23	12-64 U/Lt
ALP	369	464	506	410	178	85	45	46	< 150 IU/Lt
T.Bil.	4.77	6.54	7.71	3.68	1.91	1.08	0.64	0.89	0.2-1.2 mg/dL
D.Bil.	3.81	4.69	6.13	2.85	1.39	0.74	0.21	0.32	0-0.5 mg/dL
WBC	14.60	12.2	14.80	16.70	9.96	12.30	6.48	5.63	3.7-9.7 K/UL
NEU	11.60	9.72	12.8	14.2	7.74	9.45	4.21	3.23	2-6.90
HGB	12.6	12.4	12	11	11.9	14	17	15,8	13-18 g/dL
ALB	3.1	3.1	2.8	2.8	3.2	-	2.7	4.5	3.5-5g/dL
ESR	63	-	-	-	59	10	-	2	0-15 mm/h
CRP	6.8	8	14.5	13.5	2.5	0.2	0.2	0.5	0-0.5 mg/dL
PT	16.5				15.2			14.3	11-16 sn

Alanine aminotransferase (ALT); aspartate aminotransferase (AST); gamma-glutamyl transpeptidase (GGT); alkaline phosphatase (ALP); total bilirubin (TBIL), direct bilirubin (DBIL), white blood cells (WBC); neutrophil (NEU); Hemoglobin(HBG); albumin (ALB); erythrocyte sedimentation rate (ESR), C-reactive protein (CRP); prothrombin time (PT);

Microbiological and histopathological examinations

An aspirate sample was obtained from hepatic lesion under imaging guidance. The aspirate sample was positive for tuberculosis PCR and *M. tuberculosis* complex growth was detected in mycobacterium culture.

Treatment

The patient was diagnosed with obstructive jaundice secondary to TLAP. Thus, anti-tuberculosis treatment was initiated. In the immunohistochemical evaluation of aspirate, no pathological finding was detected other than neutrophil and lymphocyte over the necrotic ground. Within 2 months, total bilirubin, direct bilirubin, ALP and GGT were regressed to 0.6 mg/dL, 0,4 mg/dL, 46 IU/Lt and 23 IU/Lt, respectively. The anti-tuberculosis treatment was maintained for 9 months and abscess formation disappeared on sonography. The patient is being followed without treatment.

Discussion

Obstructive jaundice caused by TLAP, a subtype of abdominal tuberculosis, mostly results from mechanical obstruction of the biliary duct by lymph nodes or mass lesions [2]. Here, we discussed a patient with abdominal tuberculosis who presented with malignancy-like clinical and radiological findings and found to have multiple tuberculosis lymphadenopathies at portal areas and diagnosed by interventional radiology methods. In the assessment for other subtypes of abdominal tuberculosis, no finding of intestinal tuberculosis was detected in upper and lower gastrointestinal endoscopic examination in our case. In this case, multiple lymphadenopathies were observed in all above-mentioned areas as largest being at portal area.

Obstructive jaundice secondary to tuberculosis can occur due to inflammatory stricture of main biliary duct, compression by TLAP, enlargement of pancreas head due to tuberculosis and retroperitoneal abscess of tuberculosis. Pericholedocal tuberculous lymphadenitis in Korea showed a 81.3% male preponderance [7]. It rarely occurs due to rupture of tuberculosis granuloma to biliary ducts, direct involvement of biliary epithelium and pericholangitis [7]. In addition, there are reports of cases presented with obstructive jaundice caused by paradoxical reaction during or after treatment in the literature [8]. In our case, no findings of stricture in biliary duct, abnormality in pancreas and retroperitoneal abscess on imaging studies. Some of the reported cases were treated by surgery with anti-TB medication and some other cases were treated by anti-

TB medication alone or anti-TB medication with endoscopic nasobiliary drainage or prednisolone [5,9-12].

Although clinical, laboratory, endoscopic and radiological sings as well as bacteriological findings are not gold standard for diagnosis of abdominal tuberculosis, the diagnosis is generally made by radiological and histopathological studies [3]. The value of radiological studies is limited in the diagnosis of abdominal tuberculosis. Although sonography is not efficient for evaluation of lymph nodes, it can effectively identify dilatation in intrahepatic and extrahepatic biliary ducts. The CT scan can identify ascites and lymphadenopathy but these are not specific. The CT scan can be useful to assess treatment response as rated by lymph node sizes. Additional MRI findings can be an alternative modality in the differential diagnosis of TLAP. The role of (18F)-fluoro-2-deoxyglucose (18F-FDG) positron emission tomography (PET) has not been clarified yet in tuberculosis and other inflammatory diseases. The 18F-FDG PET has been used to detect tuberculosis granuloma and assess disease activity and the extent of disease. There are ongoing efforts to distinguish benign from malignant lesions by 18F-FDG PET without encouraging results. Active tuberculosis is associated with 18-F-FDG uptake in both pulmonary and extra-pulmonary lesions. Thus, 18F-FDG PET is particularly useful in the detection of extra-pulmonary lesions since tissue or fluid sampling can be impossible or requires invasive procedures [13,14].

In our case, 18F-FDG PET scan was ordered as patient had presentation compatible with malignancy. However, 18F-FDG PET scan did not exclude malignancy in our case. The mass lesion with necrotic centre and intensive hyper-metabolism at peripheral areas in the junction of hepatic segments 2 and 3 was striking in our case. The foci was guiding for biopsy.

TLAP often causes clinical and radiological finding which may be confused with hepatobiliary malignancies [15]. In most cases, diagnosis cannot be made before diagnostic laparotomy; thus, patients generally undergo unnecessary evaluations and explorative laparotomy [7,16]. In recent years, interventional diagnostic techniques have become more accessible; thus, rather than laparotomy, such methods are being used in eligible patients. US- or CT-guided fine-needle aspiration is useful to establish diagnosis [17]. In the literature, a similar case was presented by Al Umari R *et al.* [18]. However, in that case, the diagnosis was made by observing granulomatous inflammatory process in the histopathological

examination of lymph node samples from porta hepatis via laparoscopy. Culture tests and PCR did not provide definitive evidence. In our case, the definitive diagnosis was made by culture test and PCR analysis of aspirate obtained from hepatic lesion under CT guidance.

Conclusion

Obstructive jaundice secondary to TLAP should be kept in mind in endemic areas for tuberculosis although it is a rare entity. Tuberculosis should also be kept in mind in radiological studies. Interventional diagnostic techniques in appropriate conditions may prevent unnecessary burden of surgery and timely treatment can be provided by early diagnosis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initial will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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