Primary Biliary Tuberculosis Masquerading Cholangiocarcinoma in ¹⁸F-Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography

Abstract

Hepatobiliary involvement is a less common manifestation of abdominal tuberculosis. We present the case of a 42-year-old female who presented with fever, abdominal pain, and jaundice of 2 months duration. ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography done for disease evaluation suggested the likely possibility of cholangiocarcinoma but excision biopsy from periportal lymph node later confirmed a granulomatous etiology and she was successfully treated with antitubercular therapy.

Keywords: ¹⁸*F*-fluorodeoxyglucose positron emission tomography/computed tomography, biliary tuberculosis, cholangiocarcinoma

Introduction

Hepatobiliary involvement is an uncommon manifestation of abdominal tuberculosis representing 1% of extrapulmonary location. Isolated biliary involvement without involvement of the liver is an exceedingly rare manifestation.^[1] Biliary tuberculosis can mimic cholangiocarcinoma as both of them can present with the same clinical picture of fever, abdominal pain, and jaundice. Although tissue diagnosis is difficult from the location, early histopathological differentiation can often change the management from surgical to medical therapy.^[2]

Case Report

A 42-year-old female came to our hospital with complaints of fever and abdominal pain of 2 months duration. Clinical examination revealed jaundice and hepatomegaly. Blood investigations showed raised total and direct bilirubin values (total bilirubin - 3.42 mg/dl, direct bilirubin - 3.15 mg/dl, and indirect bilirubin - 0.27 mg/dl). CA 19-9 and CEA levels were 59 U/ml and 18.66 ng/ml, respectively. Ultrasonography (USG) whole abdomen showed a focal hypoechoic lesion in segment IVb of the liver. Contrast-enhanced computed tomography (CT) abdomen conglomerated lymph revealed nodal mass in the pancreaticoduodenal groove, peripancreatic region, and porta hepatis with compression of the portal vein and common bile duct (CBD). Magnetic abdomen resonance imaging (MRI) suspicious malignant suggested а CBD stricture at terminal CBD with lymphadenopathy. peripancreatic The patient was sent to our department for ¹⁸F-fluorodeoxyglucose positron emission tomography/CT (18F-FDG PET/CT) scan to evaluate the disease. After keeping the blood sugar levels under control levels, the patient was administered 210 MBq of ¹⁸F-FDG intravenously and scan was done 45 min following radiotracer administration. Whole-body [Figure 1a] images revealed multiple intense areas of FDG uptake in the mid-abdomen. Axial CT and fused PET/CT images reveal FDG avid (maximum standardized uptake value-7.3) soft-tissue thickening along the biliary tract from the dilated left hepatic duct up to CBD [Figure 1c and d] with necrotic abdominal retroperitoneal lymphadenopathy [Figure 1g and h]. Findings of the scan suggested the possibility of cholangiocarcinoma in view of elevated CA 19-9 values. Excisional biopsy from the periportal lymph node confirmed necrotizing granulomatous lymphadenitis with Koch's etiology [Figure 1m-o]. The patient was given a complete course of

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Sarin Krishna, Ajit Kumar Mishra¹, Mudalsha Ravina, Yashwanth Kashyap², Himanshu Bansal, Subhajit Dasgupta

Departments of Nuclear Medicine and ²Medical Oncology, All India Institute of Medical Sciences, ¹Department of General Surgery, DKS Hospital, Raipur, Chhattisgarh, India

Address for correspondence: Dr. Ajit Kumar Mishra, Assistant Professor, DKS PGI and Research Centre, Raipur, Chhattisgarh, India. E-mail: ajitm85@gmail.com

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Figure 1: (a) Black arrows – Maximum intensity projection (MIP) image showing fluorodeoxyglucose (FDG) avid lesion in the mid-abdomen, (b) MIP image following antitubercular therapy (ATT) showing complete resolution of lesions, (c and d) White arrowhead - Axial computed tomography (CT) and fused positron emission tomography/CT (PET/CT) images showing FDG avid soft-tissue thickening along the biliary tract, (e and f) Axial CT and fused PET/CT images showing complete resolution of biliary tract lesion following ATT, (g and h) White right-angled arrow - Axial CT and fused PET/CT images showing peripheral FDG avid necrotic peripancreatic lymph node, (I and J) Axial CT and fused PET/CT images showing complete resolution of biliary tract lesion and necrotic retroperitoneal lymphadenopathy, (I) Coronal fused PET/CT showing FDG avid biliary tract lesion and necrotic retroperitoneal lymphadenopathy, (I) Coronal fused PET/CT infigure and necrosis, (n) H and E (40×) – section showing Langerhans type giant cells (black arrow heads) with necrosis and dense inflammatory infiltrate composed of epithelioid histiocytes and lymphocytes, (0) H and E (100×) - section showing well defined granuloma & necrosis

6 months antitubercular therapy (ATT). The patient was symptomatically better after completion of ATT. Follow-up ¹⁸F-FDG PET/CT scan after completion of 6 months of ATT revealed complete resolution of lesion along biliary tract with a significant reduction in size and complete metabolic resolution of retroperitoneal lymph nodes. The patient responded well to ATT.

Whole body MIP image [Figure 1b] revealed complete resolution of FDG avid areas in the mid-abdomen. Axial CT and fused PET/CT images reveal complete resolution of FDG avid soft tissue thickening along biliary tract [Figure 1e and f] and complete resolution of retroperitoneal lymph nodes [Figure 1i and j]. Coronal fused PET/CT images before [Figure 1k] and after therapy [Figure 11] shows complete resolution of FDG avid retroperitoneal lymph nodes.

Discussion

Biliary involvement in tuberculosis is exceedingly rare and poses a diagnostic challenge to differentiate from cholangiocarcinoma as both of them are strong mimickers. Biliary tuberculosis is an extremely rare condition which involves bile duct causing obstructive jaundice either due to extrinsic compression by enlarged lymph nodes or by direct involvement of biliary epithelium.^[3,4] Direct involvement can lead to biliary stricture which is often rare and can involve any segment of the extrahepatic bile duct.^[3] Biliary tuberculosis is caused by either spread of caseous material from portal tracts to bile ducts or by dissemination from periportal lymphadenitis or by the ascending spread of caseous material through the ampulla of Vater.^[5] Clinical manifestations of biliary tuberculosis are indistinguishable from cholangiocarcinoma as both of them can present with fever, abdominal pain, jaundice, weakness, loss of appetite, and weight.^[6] Liver function tests can be deranged in biliary tuberculosis often simulating a picture of cholestasis due to obstruction. The probable differential diagnosis which can produce obstructive cholestasis include biliary calculi, malignant cholangiocarcinoma, and primary sclerosing cholangitis. Other than malignancy, CA 19-9 levels can also be raised in benign biliary conditions such as choledocholithiasis, cholangitis, and Mirizzi syndrome.[7,8] Interpretation based on CA 19-9 levels should be made only after the resolution of cholangitis.

USG abdomen, CT, and MRI can help in the diagnosis of biliary tuberculosis. None of the imaging findings can give a definite diagnosis of tuberculosis.^[9,10] USG and CT scans can demonstrate biliary tract dilatation, CBD stricture, and abdominal lymphadenopathy.^[9,10] Magnetic resonance cholangiopancreatography can also help to assess the site and extent of bile duct involvement. Endoscopic retrograde cholangiopancreatography (ERCP) or percutaneous transhepatic cholangiogram can show some characteristic findings pathognomonic of biliary tuberculosis. These include pruning of distal intrahepatic duct, hilar strictures with dilated intrahepatic biliary radicles, long smooth stricture of the distal biliary duct,

and sclerosing cholangitis-like changes.[11] Preoperative histopathological diagnosis is quite difficult because of its location. In majority of the cases, a definite diagnosis is often made by postoperative histopathological examination. Demonstration of caseating granulomas along with tubercle bacilli provides a definite diagnosis during the histopathological examination. Preoperative differentiation of biliary tuberculosis from cholangiocarcinoma can avoid unnecessary surgical intervention. Hence, where ever possible tissue diagnosis should always be attempted before proceeding with any interventions. Sample for histopathological examination can be obtained by brush cytology or bile aspiration during ERCP, USG-guided needle aspiration, and laparoscopic biopsy.[12,13] Early diagnosis and prompt treatment can completely cure biliary tuberculosis and can avoid further complications such as biliary stricture, cirrhosis, and portal hypertension. Definite diagnosis of granulomatous etiology can further avoid unnecessary surgery in case it is misdiagnosed as malignant etiology. Complete course of ATT can completely cure the disease.

Conclusion

Biliary tuberculosis can closely mimic cholangiocarcinoma and should always be considered as a differential diagnosis. ¹⁸F-FDG PET/CT can show FDG avid lesions in the biliary tract with necrotic regional lymphadenopathy in biliary tuberculosis similar to cholangiocarcinoma. It is difficult to distinguish biliary tuberculosis from cholangiocarcinoma solely based on PET/CT findings as both of them show similar findings. Raised CA 19-9 levels should not always be considered as a marker of malignancy as it can be elevated in many benign biliary conditions. A definite diagnosis should only be made after histopathological examination. A proper tissue diagnosis can avoid unnecessary surgery due to misdiagnosed malignant etiology.

Declaration of patient consent

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

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Conflicts of interest

There are no conflicts of interest.

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