Malignant Portal Vein Thrombosis with No Obvious Liver Parenchymal Mass on Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography

Abstract

The presence of portal vein thrombosis (PVTT) in hepatocellular carcinoma (HCC) is associated with adverse prognosis with dismal survival. Malignant portal vein thrombosis usually develops as a contiguous extension of the liver tumour into portal vein or its branches. Here we present an interesting FDG PET-CT image of a patient with chronic hepatitis B infection having isolated malignant portal vein thrombosis without any obvious liver mass.

Keywords: *Fluorodeoxyglucose, hepatocellular carcinoma, portal, positron emission tomography, thrombosis, tumor in vein, vein*

Piyush Chandra, Ashish Sethi[1], Sheela Chinnappan[2]

Departments of Nuclear Medicine and [2]Radiodiagnosis, Zydus Hospitals, [1]International Gastro Institute, Vadodara, Gujarat, India

A 56-year-old male, a known case of hepatitis B-related chronic liver disease since 2012, presented with abdominal pain and jaundice. Liver function tests revealed serum glutamic-oxaloacetic transaminase: 109 U/L, serum glutamate-pyruvate transaminase: 40 U/L, total bilirubin: 2.7 mg/dL, alkaline phosphatase: 286 U/L, serum albumin: 2.7 g/dl, and alpha-fetoprotein (AFP): 62 ng/mL. Triphasic computed tomography (CT) scan showed an arterially enhancing mass within the dilated main portal vein with contiguous extension along its branches [yellow arrows, Figure 1b] with washout on the venous phase [Figure 1c]. No obvious/dominant parenchymal mass was noted. Gross splenomegaly with multiple dilated varices was seen in the lower end of the esophagus and perigastric and perisplenic regions – possibly secondary to the occlusion of the portal vein by tumor with mild ascites. Fluorodeoxyglucose (FDG) positron emission tomography/CT (PET/CT) revealed an FDG-avid mass within the expanded main portal vein and its branches [Figure 1d]. No obvious FDG-avid mass was noted in the liver or elsewhere in the whole body to suggest any distant metastasis [Figure 1a]. In view of liver-limited disease, a transarterial radioembolization (TARE) was contemplated. Pre-TARE planning scintigraphy with technetium-99-labeled macroaggregated albumin injected after selective cannulation of the left proximal hepatic artery, however, showed exaggerated liver–lung to shunt of 62%, further procedure was abandoned, and the patient was subsequently managed with tyrosine kinase inhibitors and immunotherapy. The patient died 3 months later.

The presence of portal vein tumor thrombosis (PVTT) in hepatocellular carcinoma (HCC) is associated with adverse prognosis, with a median survival in untreated cases of 2–4 months. The only established treatment options for this are multikinase inhibitors such as sorafenib and regorafenib or Y-90 transarterial radioembolization.[1] The occurrence of HCC within the portal vein without any obvious liver mass, to our knowledge, is exceedingly rare and reported only twice previously, first by Poddar *et al.* in a 60-year-old male with hepatitis C infection[2] and later by Saito *et al.* in a 43-year-old man with chronic hepatic B infection.[3] In the latter case report, the authors confirmed the absence of liver mass on autopsy, and H/E staining of portal vein tumor thrombosis (PVTT) showed a poorly differentiated HCC. Immunostaining of PVTT was found to be negative for AFP

Address for correspondence:
Dr. Piyush Chandra, Zydus Hospitals, Vadodara, Gujarat, India.
E-mail: drpiyushchandrak@gmail.com

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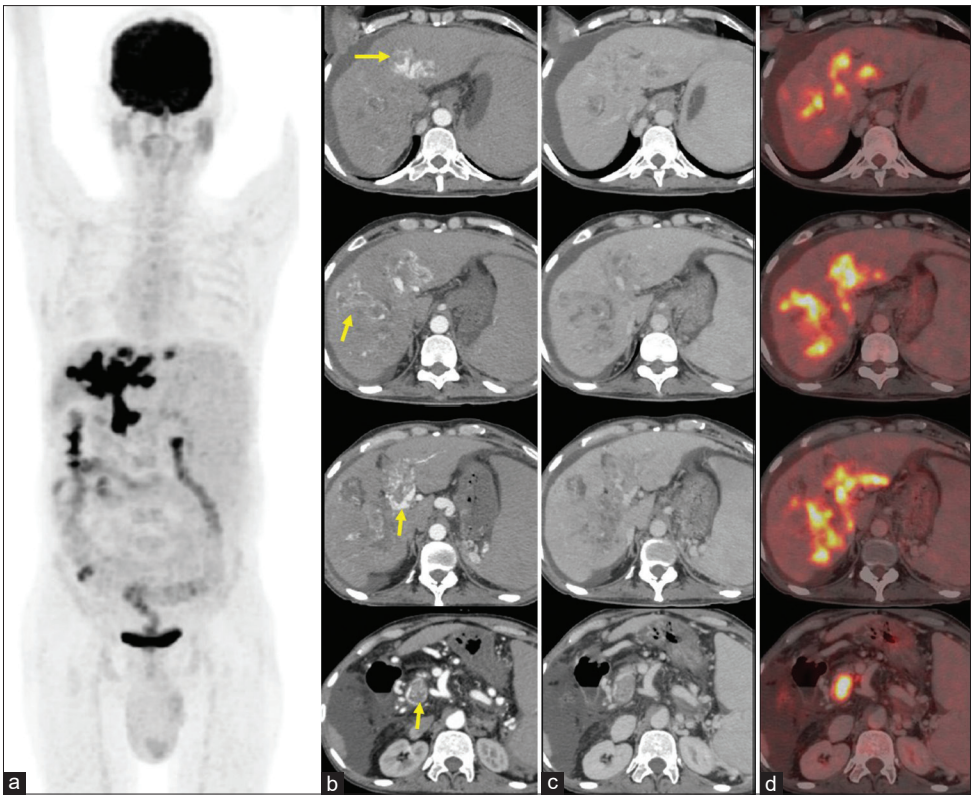

Figure 1: Whole-body fluorodeoxyglucose (FDG) positron emission tomography (PET) maximum intensity projection image. (a) FDG-avid mass within the portal vein and its branches. No evidence of any active extrahepatic disease. Arterial phase computed tomography (CT) (b) enhancing mass within the dilated main portal vein with contiguous extension along its branches (yellow arrows) with washout on venous phase CT, (c) fused PET/CT images, (d) intense FDG avidity in the mass within the portal vein and its divisions without any FDG-avid dominant liver parenchyma mass

and positive c-kit, a liver stem cell marker. In another interesting study, Liu *et al*. studied the clonal relationship of PVTTs with corresponding primary liver tumor (PT), where DNA copy number variation (CNV) profiles of 19 paired PVTTs and PT were analyzed.[4] They found 1 out of 19 PVTTs had no clonal relationship with its corresponding PT, showing independent clonal origin, different gene expressions, and enrichment in biological processes compared to PT. The authors hypothesized that these CNVs may have been caused by gene translocation or genomic instability in the tumor cells. One of the known causes of genomic instability in HCC is hepatitis B infection.[5] We could not do biopsy in our case to confirm the presence of HCC in view of advanced liver disease, no obvious liver mass, and the patient's poor performance status. It is well known that the presence of enhancing, expansible thrombus within portal vein thrombosis is suggestive of hepatocellular carcinoma and not very common with cholangiocarcinoma or metastatic disease.[6]

Enhanced FDG uptake in HCC is associated with aggressive tumor biology and microvascular invasion, supporting its prognostic clinical utility in the patient selection for resection/transplant.[7] As demonstrated above, FDG PET/CT also can play an important role in the noninvasive diagnosis of extremely rare instances of HCC being limited to the portal vein, which may be difficult to characterize on conventional imaging alone.

Declaration of patient consent

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

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Conflicts of interest

There are no conflicts of interest.

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