

Case report

Detection of Diffuse Infiltrative Primary Hepatic Lymphoma on FDG PET-CT: Hallmarks of Hepatic Superscan

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Abstract

Primary hepatic lymphoma (PHL) is an extremely rare entity with scarce information in evidence-based literature. Few case reports have described the role of 18F-fluorodeoxyglucose positron emission tomography-computed tomography (FDG PET-CT) in the diagnosing and staging of PHL. We report the case of a 78-year-old man with PHL who initially presented with weight loss and nonspecific fatigue. FDG PET-CT proved to be a beneficial tool in arriving at the diagnosis of this patient with nonspecific clinical presentation and also in the staging of PHL. Physiological uptake of FDG in the liver can be a potential cause of misinterpretation in such cases. Hence, knowing the imaging hallmarks can increase the accuracy in PET image interpretation.

Keywords: Diffuse infiltrative, flourodeoxyglucose positron emission tomography-computed tomography, nonspecific fatigue, primary hepatic lymphoma

Introduction

Primary hepatic lymphoma (PHL) are relatively rare with non specific clinical findings, often presenting as a diagnostic challenge. Computed tomography (CT) and ultrasound may be non diagnostic. We report a case of primary hepatic lymphoma where CT scan was non specific and revealed splenic infarcts. Fluorodeoxyglucose (FDG) positron emission tomography (PET)-CT showed diffuse uptake in the liver which raised the possibility of primary tumor and needed further evaluation.

Case Report

A 78-year-old man with past medical history of diabetes mellitus type II, hypertension, hypothyroidism, and recurrent urinary tract infection had presented

with fatigue, pain in the left upper abdomen, and urinary frequency for the past 1 week and weight loss of approximately 10 kg in the past 3 months. Laboratory analysis was notable for severe anemia [hemoglobin (Hb) - 7.2 g/dL, peripheral smear showing anisocytosis with normochromic normocytic anemia], normal white blood cell (WBC) count, high lactate dehydrogenase (LDH) (1266 U/L), and high alkaline phosphatase levels (434 IU/L). Contrast-enhanced computed tomography (CT) scan of the abdomen revealed a mildly enlarged liver with normal attenuation pattern and hypodense wedge-shaped splenic lesions, likely infarcts. Whole body PET-CT scan was done [Figure 1] for the screening of any associated neoplastic etiology in view of nonspecific weight loss, high LDH, and deteriorating coagulation profile. Notably, the liver showed diffusely increased fluorodeoxyglucose (FDG) uptake [standardized uptake value (SUV) max: 4.5]. The hypodense lesion in the spleen did not show any significant FDG uptake [Figure 2]. The rest of the scan did not show any abnormal FDG avid disease. Transjugular liver biopsy revealed hepatic parenchyma with focal/interstitial/sinusoidal prominence of lymphoid cells with modest portalobular portocytic infiltrates. The lymphoid cells in sinusoids and the interstitial location were diffusely CD20-positive with a Ki-67 index of

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Figure 1: 18F- FDG PET maximal intensity projection image showing diffusely increased abnormal uptake of FDG in the liver. Physiological tracer uptake is seen in the brain, right kidney, and urinary bladder

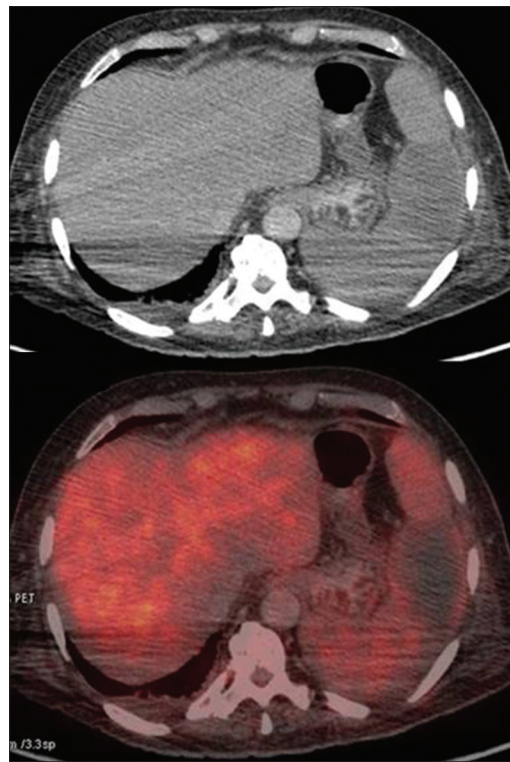


Figure 2: Axial section of the liver on CT and fused PET-CT showing normal attenuation pattern of liver parenchyma with increased FDG uptake and a large non-FDG avid hypodense wedge-shaped infarct in the spleen

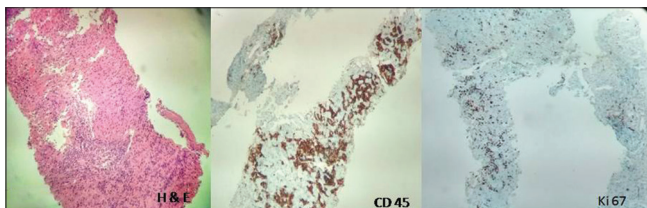


Figure 3: Micrograph showing hematoxylin and eosin staining of tissue obtained from the liver, positive staining for CD 45 and Ki 67 positive tumor cells (80%)

80% (approximately) and were leukocyte common antigen (LCA)-positive/CD5-positive/CD38-negative/CK20-negative [Figure 3]. No expression of terminal deoxynucleotidyl transferase (Tdt), CD23, or cyclin D₁ was noted. The diagnosis of high-grade B-cell lymphoma was confirmed. The patient was started on rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone (R-CHOP) regimen of chemotherapy.

Discussion

Primary hepatic lymphoma (PHL) is extremely rare and constitutes less than 0.4% of all extranodal non-Hodgkin lymphomas.^[1] It is defined as lymphoma confined to the liver with no evidence of lymphomatous involvement in the spleen, lymph nodes, bone marrow, or other lymphoid structures.^[2] There are three main

morphological patterns known including the solitary nodule (60%), multiple focal nodules (35%), and diffuse infiltrating without nodular formation (5%).^[3] The clinical presentation is variable and atypical with common features including fever, weight loss, night sweats, and right upper abdominal pain. Imaging studies like CT and magnetic resonance imaging (MRI) reveal nonspecific findings in diffuse infiltrating variants. Some cases, however, may show hepatomegaly and diffusely hypovascular lesions with occasional ascites. The data on the role of FDG PET-CT in PHL is limited. Infiltrating PHL shows diffuse intense FDG uptake in the liver (much higher than the physiological uptake in the brain), sometimes referred to as hepatic superscan. In this case, FDG PET-CT proved beneficial in diagnosis and staging. Management, restaging, and follow-up in such a case is challenging as the known PET response criteria in solid tumors (PERCIST) and Deauville scoring method used in the National Comprehensive Cancer Network (NCCN) recommendations have limited reliability since the liver itself is the primary site of involvement. A recent case report, however, describes the use of FDG PET-CT in showing complete remission of PHL.^[4] Few studies in the literature have, however, correlated the level of FDG uptake with disease activity and tumor proliferation.^[5] Other differentials for hepatic superscan on FDG PET-CT include chronic myeloid

leukemia,^[6] tuberculosis,^[7] metastases, and Richter's transformation of chronic lymphocytic leukemia.^[8] In conclusion, solitary intense uptake in the liver on FDG PET-CT needs thorough evaluation and a liver biopsy should be carried out for further management.

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