

Malaria masquerading as relapse of Hodgkin's lymphoma on contrast enhanced ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography: A diagnostic dilemma

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ABSTRACT

¹⁸Fluorodeoxyglucose (¹⁸F-FDG) positron emission tomography/computed tomography (PET/CT) is nowadays routinely used in management of lymphoma patients. We here present a case of Hodgkin's lymphoma which showed ¹⁸F-FDG avid splenomegaly on PET/CT done for clinically suspected relapse. Further evaluation by peripheral smear examination revealed malaria. The patient was then started on anti-malarial medications and follow-up PET/CT revealed resolution of hypermetabolic splenomegaly. This report highlights that in endemic regions malaria can cause ¹⁸F-FDG avid splenomegaly and might mimic relapse of lymphoma.

Keywords: ¹⁸F-Fluorodeoxyglucose, lymphoma, malaria, positron emission tomography/computed tomography, spleen

INTRODUCTION

¹⁸Fluorodeoxyglucose (¹⁸F-FDG) positron emission tomography/computed tomography (PET/CT) has become the imaging modality of choice for initial staging, follow-up and treatment response assessment in patients with Hodgkin's lymphoma and has proved superior to contrast enhanced CT (CECT) in these settings. ¹⁸F-FDG PET/CT has accuracy of almost 100% in diagnosing primary splenic involvement during initial staging of lymphoma. However, in the post-therapy setting its role for evaluation of secondary splenic involvement is limited. One of the pattern of splenic activity that help to detect splenic involvement on PET/CT is diffusely increased ¹⁸F-FDG uptake greater than that in the liver and bone marrow with or without corresponding CT lesions. In this context, we report a case of a patient with Hodgkin's lymphoma in remission presenting with ¹⁸F-FDG avid splenomegaly.

CASE REPORT

The present case report is about a 22-year-old male patient who presented with enlarged right cervical lymph node. Biopsy revealed-Hodgkin's lymphoma (mixed cellularity). ¹⁸F-FDG PET/CECT performed for staging revealed metabolically active lymph nodes on either side of the diaphragm [Figure 1a, broken arrows], enlarged spleen with multiple hypodense hypermetabolic, lesions [Figure 1a, arrow] (SUV_{max} = 9.4; Spleen SUV_{max}/liver SUV_{max} ratio = 3.76) and bone lesion. He was then given 6 cycles of chemotherapy and ¹⁸F-FDG PET/CECT was done for response evaluation. PET/CT showed complete metabolic response, with normal spleen uptake [Figure 1b, arrow] (Spleen SUV_{max} = 2.5, Liver SUV_{max} = 2.6 S/L ratio = 0.96). At 1-year later routine follow-up the patient complained of mild fever, lethargy and listlessness. In view of previous history of Hodgkin's lymphoma, relapse was suspected and ¹⁸F-FDG PET/CECT was advised. PET/CT revealed enlarged spleen with diffusely increased FDG uptake [Figure 2a-c, arrow] (SUV_{max} = 5.3; Liver SUV_{max} = 2.3 S/L ratio = 2.30). The first differential in the given clinical scenario was splenic relapse of lymphoma, however, a second differential diagnosis of some infective/inflammatory process was considered. On further evaluation, peripheral smear showed evidence of malaria parasite infection (*Plasmodium vivax*). The patient was then started on anti-malarials with complete clinical improvement. Follow-up

Access this article online

Quick Response Code:



Website:
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DOI:
10.4103/0972-3919.130299

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PET/CT after 7 months revealed normalization of size and ¹⁸F-FDG uptake of spleen [Figure 2d, arrow] (SUV_{max} = 3.0; Liver SUV_{max} = 2.7 S/L ratio = 1.1). This clinical case can be easily misinterpreted as lymphoma relapse. Hence, malaria and other relevant (endemic) infective possibilities (Kala-Azar etc.) should be considered and further investigation, if warranted, should be advised.

DISCUSSION

PET/CT is a useful modality for staging and restaging of Hodgkin's lymphoma with high sensitivity and specificity.^[1] Furthermore, it is a sensitive modality for early detection of relapse in asymptomatic

patients making surveillance ¹⁸F-FDG PET/CT clinically important.^[2] Splenic uptake on ¹⁸F-FDG PET can be due to a wide variety of causes such as lymphoma, anemia, granulocyte colony stimulating factor treatment, beta-thalassemia, inflammation and infections.^[3-8] Splenic uptake, greater than hepatic uptake, is a relatively reliable indicator of lymphomatous involvement of the spleen, in the absence of recent cytokine administration. In early stage HIV infection, diffusely increased splenic uptake is noted due to reactive stimulation of B-cells in the spleen. It can also be noted in sarcoidosis, malaria and many other inflammatory diseases. Post-therapeutic reactive splenic uptake is also noted after administration of granulocyte colony-stimulating factor for myelosuppression or high-dose interferon-alpha-2b adjuvant therapy for melanoma. Until date, only a single report by Liu *et al.* have demonstrated ¹⁸F-FDG uptake in spleen in case of malaria.^[9]

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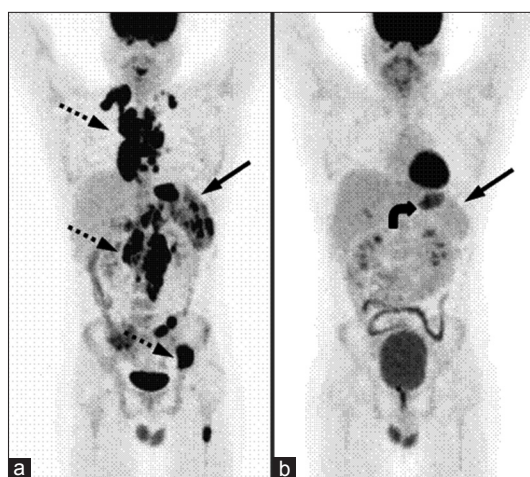


Figure 1: ¹⁸F-fluorodeoxyglucose (¹⁸F-FDG) positron emission tomography/computed tomography (PET/CT) done for staging and response evaluation. (a) Staging maximum intensity projection (MIP) PET image showing multiple metabolically active lymph nodes in thorax, abdomen and pelvis (broken arrows) with splenic (arrow) and bone lesion (left femur). (b) MIP PET image done for response evaluation demonstrates complete metabolic response with normal splenic FDG uptake (arrow). Physiological gastric FDG uptake noted (bent arrow)

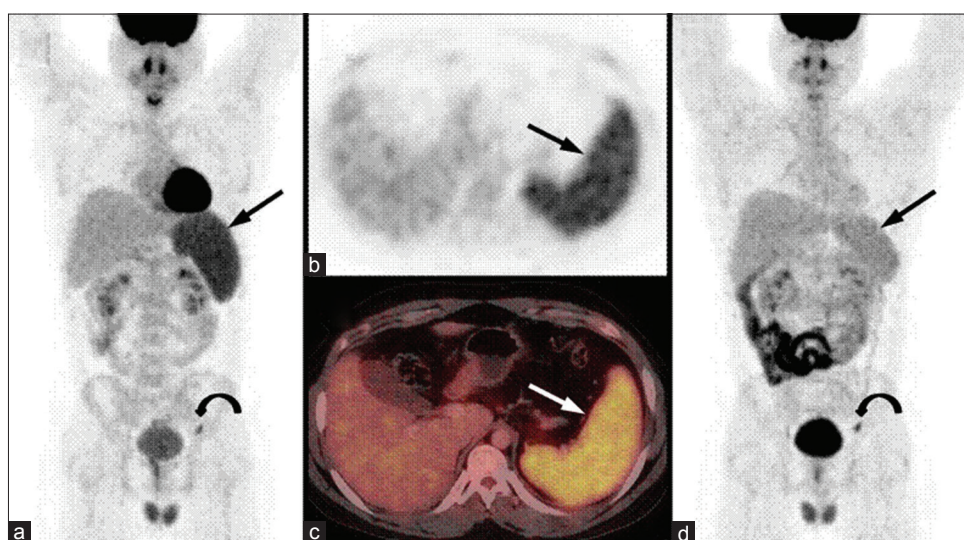


Figure 2: ¹⁸F-fluorodeoxyglucose (¹⁸F-FDG) positron emission tomography/computed tomography (PET/CT) done for recurrence detection and follow-up. (a-c) Follow-up PET/CT performed 1 year after chemotherapy showing enlarged spleen with diffusely increased FDG uptake (arrow). (d) Maximum intensity projection PET image performed after anti-malarial therapy, showing normalization of size and FDG uptake of spleen (arrow). (a and d) Focal FDG uptake noted in the pelvis is due to urinary FDG activity in the left ureter (curved arrows)

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How to cite this article: Jeph S, Thakur K, Shamim SA, Aggarwal A. Malaria masquerading as relapse of Hodgkin's lymphoma on contrast enhanced ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography: A diagnostic dilemma. *Indian J Nucl Med* 2014;29:102-4.

Source of Support: Nil. **Conflict of Interest:** None declared.