Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography Findings in Nodular Hepatic and Splenic Sarcoidosis

Dear Editor,

A 62-year-old male patient presented with the chief complaint of loss of weight of 1 year duration. There was no history of associated fever. On evaluation, patient had liver parenchymal disease with raised serum alkaline phosphatase. Computed tomography (CT) of abdomen revealed multiple hypo-attenuating lesions in the liver and spleen. The patient underwent F-18 fluorodeoxyglucose (FDG) positron emission tomography (PET)/CT scan [Figure 1a] for characterization of the hepato-splenic lesions. PET/CT scan showed heterogeneous pattern of tracer uptake in the enlarged liver measuring approximately 18 cm in cranio-caudal direction with increased tracer uptake (SUVmax 9.7) in the hypo-attenuating lesions [arrow in the Figure 1b and c]. Increased FDG uptake with SUVmax 7.3 was also noted in the multiple well-defined hypo-dense nodules of variable sizes (largest measuring 1.8 cm) in the normal sized spleen [Figure 1b and c]. Increased tracer uptake was seen in right level III cervical, multiple enlarged mediastinal [Figure 1d and e] and abdominal lymph nodes. There was no abnormal lung uptake in the scan. In the given clinical context, differential diagnosis of lymphoma or granulomatous disease was made. The biopsy from the liver showed granulomas. Serum angiotensin converting enzyme (ACE) was also raised on further evaluation, thus indicative of sarcoidosis.

Sarcoidosis is a systemic disease of unknown etiology, which is characterized by noncaseating epitheloid granulomas. Although it primarily affects lungs and hilar lymph nodes; liver, spleen, heart, and bone marrow are common extra pulmonary sites of disease manifestations.[1] The patients with liver and spleen involvement usually are asymptomatic and show normal liver enzyme tests. The commonest clinical finding is hepatosplenomegaly in 15-40% of patients. In advanced disease, jaundice, pruritus, liver failure, ascites, and hepatic encephalopathy may rarely occur. Serum ACE is usually elevated. Furthermore, the radiographic findings of abdominal sarcoidosis are less characteristic. The hepatic sarcoidosis usually manifests with minimal organomegaly in radiographic imaging such as CT and magnetic resonance. Coalescing

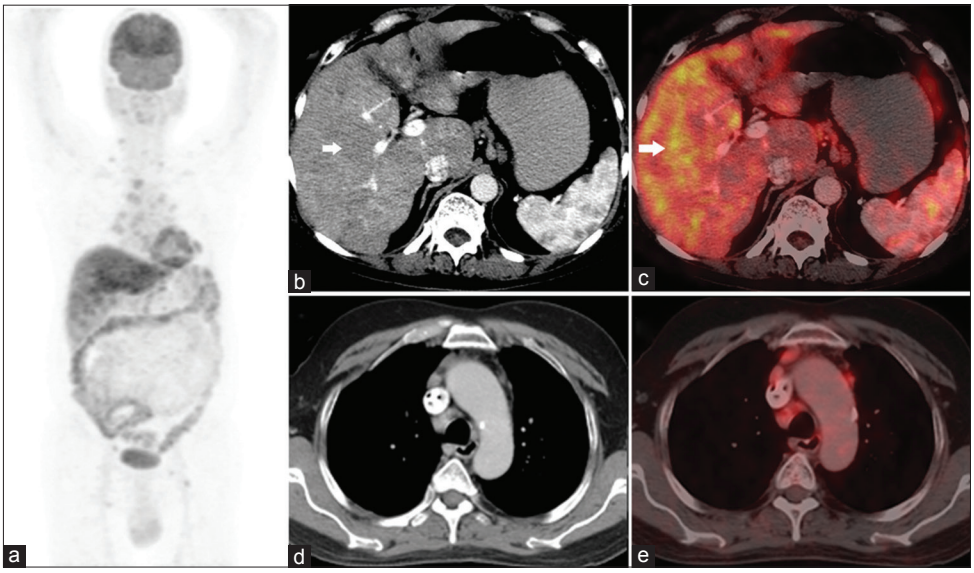

Figure 1: Fluorodeoxyglucose (FDG) positron emission tomography/computed tomography maximum intensity projection image (a) heterogeneous tracer uptake in the enlarged liver with increased tracer uptake (SUVmax 9.7) in the hypo-attenuating lesions (arrow in b and c). Increased FDG uptake with SUVmax 7.3 is also noted in the multiple well defined hypo-dense nodules of variable sizes in the normal sized spleen (b and c). Multiple enlarged FDG avid mediastinal (d and e) lymph nodes are also seen

granulomas become apparent as multiple hypointense or hypo-attenuating nodules relative to adjacent normal parenchyma in only 5-15% of patients. Peripheral enhancement is not seen in these lesions.[2] Although calcification is uncommon, it may be present in long standing disease.[3] Generally, splenic nodules are larger and more common than hepatic lesions.[4] The hypointense and hypo-attenuating liver and spleen nodules of variable sizes ranging from 5 to 20 mm are present in only 10–15% of patients.[2] It is sometimes difficult to differentiate between sarcoidosis and more common diseases, such as metastases and lymphoma. However, in lymphoma lymph node enlargement is more pronounced and the retrocrural lymph nodes are more frequently involved, which were not present in this case. Furthermore, simultaneous hepato-splenic involvement and no other abnormal tracer activity anywhere, except liver, spleen and lymphadenopathy makes diagnosis of metastatic disease less likely. Increased FDG accumulation has been reported in literature in sarcoidosis. Increased metabolism in more unusual sites like liver and spleen, may mimic lymphoma or metastatic disease.[4-6] However, this unique case may serve as template for pattern recognition in hepatic and splenic sarcoidosis. Thus in patients with hypermetabolic hypo-attenuating lesions in the liver and spleen with associated hypermetabolic lymphadenopathy, possibility of sarcoidosis may be considered, even if pulmonary involvement is absent.

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DOI: 10.4103/1450-1147.139150

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