Diffuse Splenic F-18 FDG Uptake in Visceral Leishmaniasis

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Abstract: A 51-year-old woman had splenomegaly and enlarged multiple splenic hilar lymph nodes. The patient was referred to our department for F-18 fluorodeoxyglucose positron emission tomography/computed tomography (F-18 FDG PET/CT) evaluation to determine the metabolic activity of lymph nodes and define a biopsy site. PET/CT images showed diffuse increased FDG uptake in an enlarged spleen and hypermetabolic splenic hilar lymph nodes. The metabolic activity in bone marrow also seemed diffusely increased. After splenectomy, histopathologic analysis showed the growth of *Leishmania amastigotes* in splenic tissue, and bone marrow biopsy did not reveal any significant pathology but only mild hypercellularity.

Key Words: FDG PET, visceral leishmaniasis, spleen

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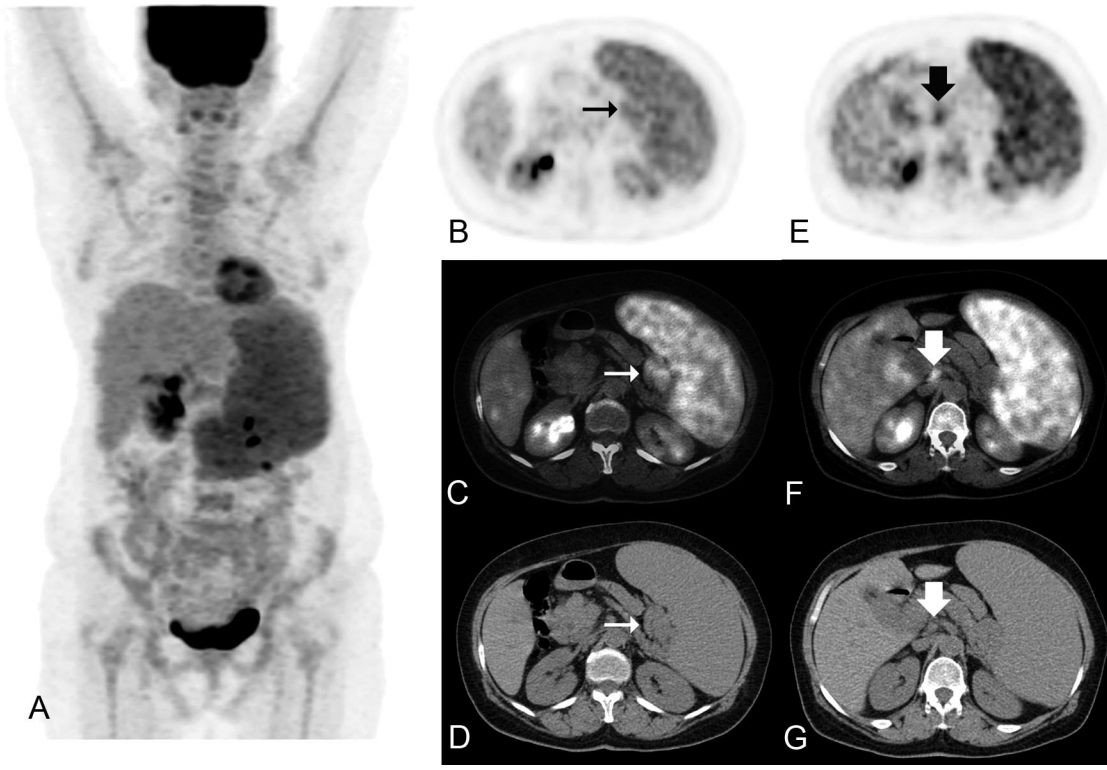

FIGURE 1. A 51-year-old woman had splenomegaly and enlarged multiple splenic hilar lymph nodes. The patient was referred to our department for F-18 fluorodeoxyglucose positron emission tomography/computed tomography (F-18 FDG PET/CT) evaluation to determine the metabolic activity of lymph nodes and define a biopsy site. She fasted for 12 hours before intravenous injection of 12.74 mCi (471.38 MBq) of FDG. PET imaging was performed 75 minutes after injection on a Discovery STE PET/CT scanner (General Electric). Maximum intensity projection (MIP) (**A**), transaxial PET (**B**), fusion (**C**), and CT (**D**) images showed diffuse increased FDG uptake in an enlarged spleen (maximum standardized uptake value [SUV_{max}] = 5.3) and hypermetabolic splenic hilar lymph nodes (SUV_{max} = 4) (thin arrows). Also, maximum intensity projection image demonstrated diffusely increased F-18 FDG uptake in the bone marrow. Any finding regarding bone marrow could not be recognized on CT images. Transaxial PET (**E**), fusion (**F**), and CT (**G**) images at the level of upper poles of kidneys show mildly hypermetabolic lymph node (SUV_{max} = 3.8) in precaval area (bold arrows). F-18 FDG PET/CT is an important imaging tool for diagnosis, staging, assessment of response to therapy, and detection of recurrence with high accuracy in lymphoma.[1] Causes of splenic FDG uptake on PET imaging reported in the literature include lymphoma, granulocyte colony stimulating factor treatment, beta-thalassemia, and infections.[2–4] Diffuse increased FDG uptake in the bone marrow on PET scan is frequently observed due to hematopoietic stimulation after chemotherapy but can also be noted in a variety of pathologic conditions, including leukemia and lymphoma.[5–11] In the present case, histopathologic analyses of bone marrow did not reveal any significant pathology but only mildly hypercellularity. Increased bone marrow FDG uptake is probably due to bone marrow stimulation secondary to systemic parasitic infection.

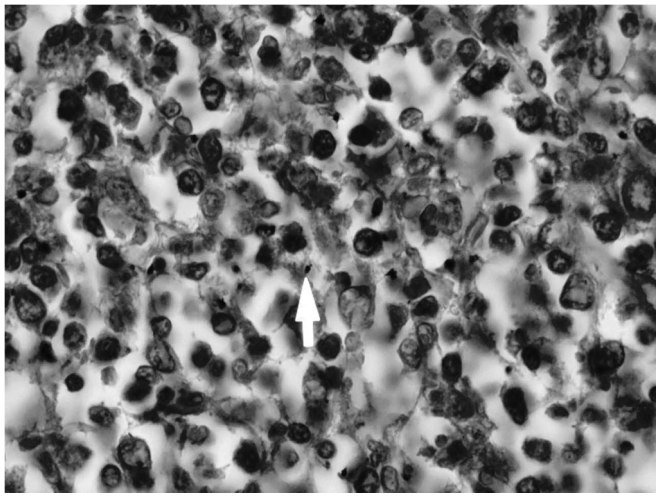

FIGURE 2. Because any biopsy site could not be identified, the patient underwent splenectomy, and splenic tissue was histopathologically analyzed. Microphotograph reveals "signet ring" like *Leishmania amastigote* adjacent to a lymphocyte (HE ×1000). In light microscopic examination, white pulp was hyperplastic. There was grade 2 iron accumulation in the spleen. Cytoplasm of most of the histiocytes was full of hemosiderin pigment. There were *L. amastigotes* both in sinusoids, in white pulp and in the cytoplasm of some histiocytes in which the vision was not fully obscured by hemosiderin pigment. Lymphoid follicles showed bitypical zonal staining with CD3 and CD20 antibodies. BCL-2 stained marginal zones, mante zones, and t lymphocytes, while cyclin D1 was negative. With earlier mentioned histopathological and immunohistochemical features, the splenectomy material was diagnosed as "leishmaniasis." Visceral leishmaniasis (VL) is characterized by multiple infection loci of the protozoan. The etiological agent is a protozoon of genus *Leishmania*. The organs of the reticuloendothelial system, especially liver, bone marrow, and spleen, are most heavily infected. Lymphadenopathy, infection of the gastrointestinal tract, and glomerulonephritis are very rare manifestations.[12,13] The clinical signs and symptoms of VL are not pathognomic and the disease may be confused with other similar conditions such as malaria, tropical splenomegaly, schistosomiasis, brucellosis, malnutrition, lymphoma, and leukemia.[14] This case highlights VL showing increased splenic metabolism and hypermetabolic lymphadenomegaly mimicking lymphoma.