Clinical note

Chronic myeloid leukemia detected on FDG PET/CT imaging in a patient with renal cell carcinoma

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Abstract

It is well known that hematopoietic cytokine stimulation can cause increased fluorodeoxyglucose (FDG) accumulation in bone marrow on PET/CT imaging, which simulates that seen in patients with bone marrow metastases. However, increased bone marrow FDG uptake can be caused by other etiologies. We report a patient with operated renal cell carcinoma had no history of hematopoietic cytokine stimulation. The FDG PET/CT images showed increased bone marrow FDG uptake, and the patient was diagnosed as chronic myeloid leukemia. This case revealed that increased FDG uptake on bone marrow may be related to neoplastic disease of the hematopoietic tissues.

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Introduction

Chronic myeloid leukemia (CML) is a clonal myeloproliferative disorder of pluripotent hematopoietic stem cells, characterized by increased proliferation, and decreased apoptosis of myeloid progenitor with peripheral leukocytosis.1 CML is a malignant disorder of the stem cell due to reciprocal balanced translocation of genetic material between the long arms of chromosomes 9 and 22.2

Leukemias are characterized by diffuse replacement of bone marrow with proliferating leukemic cells, abnormal numbers and forms of immature white blood cells in the blood and widespread infiltrates in the liver, spleen, lymph nodes, and other sites throughout the body. Leukemias are classified as lymphocytic or myelocytic, according to the type of white blood cells involved.3 CML is a pluripotential stem cell disease characterized by anemia, extreme blood granulocytosis, granulocytic immaturity, basophilia, often thrombocytosis, and splenomegaly.4

Whole-body fluorine-18 (18F) FDG positron emission tomography (PET) has been useful in the management of a variety of malignancies. In patients with chemotherapy followed by bone marrow stimulants such as granulocyte colony-stimulating factor and granulocyte-macrophage colony-stimulating factor, the bone marrow will have diffuse increased FDG accumulation. Therefore, diffuse bone marrow FDG uptake is commonly attributable to the effect of hematopoietic cytokines. However, diffuse bone marrow FDG uptake can also be caused by bone marrow involvement by malignancy, inflammatory conditions, after chemotherapy and idiopathic myelofibrosis.5–8

Case report

A 60-year-old male patient was referred for FDG PET/CT imaging for detecting recurrence or metastasis of renal cell cancer (RCC). He was diagnosed having RCC 5 years ago and he never had chemotherapy after first diagnoses of renal malignancy and before PET/CT examination. The patient underwent FDG PET/CT scan 60 min after intravenous injection of 370 MBq FDG. Disseminated infiltration and increased FDG uptake in bone marrow prominent in the
vertebral bodies, pelvic bones and proximal parts of the upper and lower extremities (Fig. 1) were demonstrated with FDG PET/CT imaging. Findings on FDG PET/CT were suspicious for bone marrow involvement or using of granulocyte colony-stimulating factor. However the patient had no history of using granulocyte colony-stimulating factor. Hemogram analysis showed white blood cell count 207 k/ul (normal ranges: 4–10 k/ul), hemoglobin 13.2 g/dl (normal: 12.1–17.2 g/dl), mean corpuscular volume 90 Fl (normal: 82.2–99 Fl), and platelet count 214,000 u/l (normal: 150–400) with neutrophils 91.7%, lymphocytes 2.62%, and monocytes 7.11%, LDH 763 u/l (normal ranges: 98–192). For hyperleukocytosis, he was admitted to the hematology clinic and bone marrow examination. Bone marrow aspiration and biopsy revealed hypercellular bone marrow with prominent myeloid hyperplasia. He was given hydroxyurea, and allopurinol was also started for potential tumor lysis syndrome.

Discussion

The diffuse increase in FDG uptake in the bone marrow not only may be caused by malignancy or hematopoietic disease, but also may be due to an inflammatory reaction, recent chemotherapy, or administration of hematopoietic growth factors. A diffuse increase in FDG uptake has also been reported in hyperplastic bone marrow as seen an idiopathic myelofibrosis. To determine the effect of granulocyte colony stimulating factor and granulocyte-macrophage colony stimulating factor on bone marrow glucose metabolism, the specific uptake of FDG in bone marrow has been evaluated. It was shown that a substantial increase in bone marrow FDG uptake is rapidly induced by colony stimulating factor treatments and should not be misinterpreted as diffuse bone metastases or bone marrow disease. FDG PET can also be used for the evaluation of CML. Nakajo et al. have reported in their study that patients with CML in the chronic phase that showed increased FDG uptake in the bone marrow in pretreatment period and follow-up FDG PET scan findings after termination of treatment had demonstrated reduced FDG uptake in the bone marrow. Chiang et al. reported a patient with diffuse bone marrow involvement of Hodgkin’s disease that appears indistinguishable from hematopoietic cytokine-mediated FDG bone marrow uptake. Inoue et al. reported diffuse bone marrow uptake on 18F FDG PET in two patients with myelodysplastic syndromes. FDG PET/CT is useful in the diagnosis, initial staging, follow-up, and restaging of patients with bone marrow malignancy. It is also well known that increased bone marrow uptake of FDG after administration of colony stimulating factor is one of the most important cause of visualization of bone marrow in FDG PET/CT imaging. This case suggests that the various pathologic and idiopathic clinical conditions should be kept in mind to avoid mis-interpretation of the images for reporting patients with increased bone marrow involvement in FDG PET/CT.

Conflict of interest

The authors declare not to have any conflict of interests.

References