Isolated cerebellar hypermetabolism on FDG PET in a case of remitted primary breast lymphoma

Arun Kumar Reddy, Sampath Santhosh, Bhagwant Rai Mittal, Anish Bhattacharya, Suresh C Sharma[1]

Departments of Nuclear Medicine and PET and [1]Radiotherapy, Postgraduate Institute of Medical Education and Research, Chandigarh, India

ABSTRACT

We present here a case of primary non-Hodgkin's lymphoma of the breast that succumbed in a sub-acute course to death after three months of initial remission. The fluorodeoxyglucose positron emission tomography imaging during the declining clinical status showed isolated cerebellar hypermetabolism.

Keywords: Fluorodeoxyglucose positron emission tomography, isolated cerebellar hypermetabolism, paraneoplastic cerebellar degeneration, primary breast lymphoma

A 50-year-old female patient presented with a palpable mass in the left breast for the last four months. Histologic and immunocytochemical analyses of biopsy tissue from the breast mass showed high grade-B cell non-Hodgkin's lymphoma (NHL). Pre-chemotherapy evaluation showed additional involvement of left axillary lymph nodes. Bone marrow evaluation for involvement of lymphoma was negative. The patient was subjected to six cycles of chemotherapy with CHOP regimen and a post-chemotherapy evaluation with contrast enhanced computed tomography (CECT) showed remission of disease. Three months following the initial remission, the patient presented with complaints of gait imbalance, dysarthria, headache, and vomiting. Suspecting a relapse with central nervous system (CNS) involvement, PET/CT imaging was performed. Figure 1 anterior (A) and left lateral (B) maximum intensity projection images show hypermetabolic cerebellum with no abnormal FDG localization elsewhere. CT, PET, and fused PET/CT images of the head in axial (C-E) and sagittal (F-H) views show intense diffuse FDG uptake in the cerebellum. The findings were suggestive of relapse of disease vs. paraneoplastic cerebellar degeneration. However, the patient's clinical status declined rapidly and died before further diagnostic workup.

The neurologic complications of the malignant lymphomas are common, serious, but treatable; vast majority of which are seen

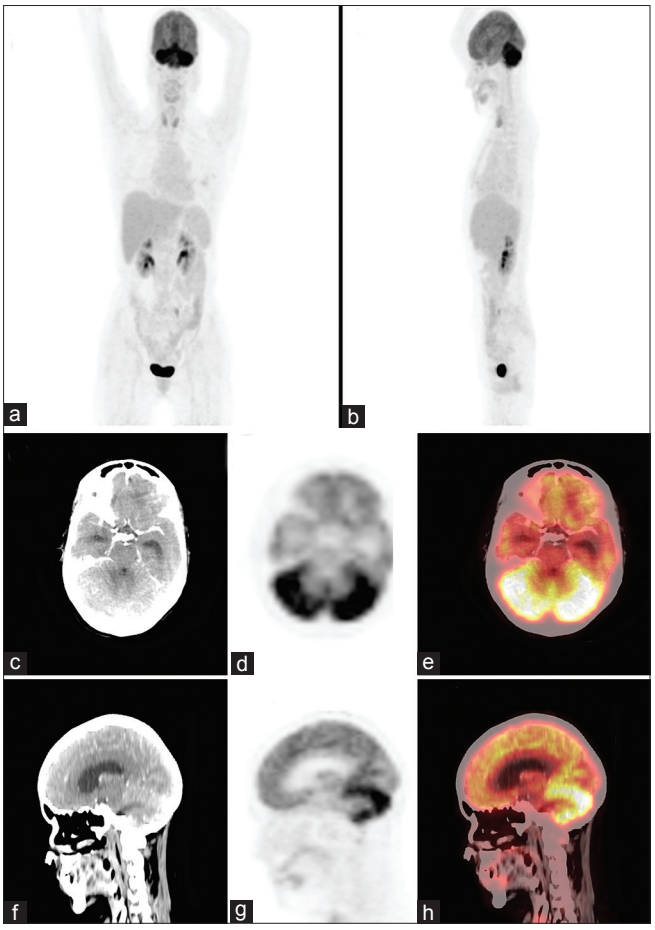

Figure 1: PET/CT images (a) anterior and (b) left lateral maximum intensity projection images show hypermetabolic cerebellum with no abnormal FDG localization elsewhere. CT, PET and fused- PET/CT transaxial (c-e) and sagittal (f-h) images of the head show intense diffuse FDG-uptake in the cerebellum. The findings were suggestive of relapse of disease vs. paraneoplastic cerebellar degeneration

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Address for correspondence: Dr. Bhagwant Rai Mittal, Department of Nuclear Medicine and PET, PGIMER, Chandigarh - 160 012, India. E-mail: brmittal@yahoo.com

Table 1: The various causes of cerebellar injury causing hypermetabolism include

Malignancy
Primary brain tumor arising from the cerebellum
Metastatic involvement from a systemic malignancy
Toxin induced
Drug-induced cerebellar toxicity (Ex: Post-chemotherapy)
Autoimmune
Paraneoplastic cerebellar degeneration (Early stage)
Post-vaccination encephalitis
Infectious
Viral cerebellitis
Prion diseases
Secondary to CNS infections

CNS: Central nervous system

associated with NHLs. There is wide variation in the incidence, frequency, and type of neurologic involvement in the NHLs. They range from direct disease involvement to paraneoplastic pathologies.[1] The differentiation of the direct and paraneoplastic involvement is a challenge for the treating physician as it has a bearing on the choice of further treatment. CNS involvement of NHL has a higher incidence in the high-grade subtypes and presence of extranodal primary at presentation or involvement of two or more extranodal sites are known risk factors for CNS relapse.[2,3] Cerebellum is a common target in such cases, although, leptomeningeal involvement is more common. Increased cerebellar metabolic activity has a broad range of differential diagnosis. The etiology can be an inciting agent causing damage to the cerebellar parenchymal cells (either infectious, autoimmune, toxin etc.) or malignant involvement of the cerebellum [Table 1].[4-6] The current case being a treated case of NHL in remission suggests the possible diagnoses of acute stage of paraneoplastic cerebellar degeneration, CNS relapse of lymphoma and drug-induced cerebellar toxicity. However, as CNS toxicity is not a commonly documented entity with the CHOP regimen and isolated cerebellar relapse of lymphoma is rare, a provisional diagnosis of paraneoplastic cerebellar degeneration was considered. Further workup for confirmation of diagnosis (including MRI, autoantibodies associated with paraneoplastic syndrome for example, Anti-Hu, Anti-Yo)[7,8] could not be performed because of death of the patient. To conclude, isolated cerebellar hypermetabolism is an atypical and uncommon finding on FDG-PET. Our current case emphasizes the need for careful and urgent patient evaluation in view of various possible diagnoses that greatly vary in management and patient outcome. Combined clinical, imaging, and hematological workup to reach a diagnosis is necessity of the situation in such cases.

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