Case Report

Fluorodeoxyglucose Positron Emission Tomography-Computerized Tomography Scan in LGI A1-Positive Limbic Encephalitis and Concordance with MRI in a Known Case of Carcinoma Breast

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Abstract

The limbic encephalitis is an autoimmune disorder which characterized by inflammation of the brain with rapidly progressing dementia either due to paraneoplastic or nonparaneoplastic etiology which requires definitive neurological and whole-body evaluation. We describe both clinical and nuclear medicine imaging and radiological findings in a case of limbic encephalitis using positron emission tomography and magnetic resonance imaging.

Keywords: *Fluorodeoxyglucose positron emission tomography-computerized tomography scan (18-fluorine fluoro-deoxyglucose positron emission tomography computerized tomography), LGI A1 antibody, limbic encephalitis, magnetic resonance imaging*

Introduction

The limbic encephalitis is an autoimmune-mediated inflammation typically affecting the mesial temporal cortex and limbic structures (as striatum, cingulate gyrus, and orbitofrontal cortex).[1]

Patients affected usually show a short-term memory deficit with rapid progression and psychiatric or seizures like disorder. Final diagnosis always required either pathological or radiological interventions for the involvement of the limbic structures apart from the precise clinical symptoms. We here present a clinical case of limbic encephalitis with findings of fluorodeoxyglucose (FDG) positron emission tomography-computerized tomography (PET-CT) and magnetic resonance imaging (MRI) neuroimaging.

Case Report

A 67-year-old female is a known case of carcinoma breast diagnosed in 2008, postsurgery modified radical mastectomy, postchemotherapy, and posthormonal therapy for 5 years, presented with two episodes of generalized tonic-clonic seizures on a single day (January 8, 2021) accompanied by progressive worsening behavioral disturbances. Routine blood profile including complete blood count, biochemistry tests, viral loads, thyroid function tests, electrolytes, and routine cerebrospinal fluid (CSF) examination was fairly normal. MRI brain examination showed asymmetric signal abnormality diffusely involving the right medial temporal lobe predominantly right amygdale and hippocampus which appearing hyerintense on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images with no evidence of restricted diffusion or significant enhancement suggestive of limbic encephalitis [Figure 1]. The patient tested positive for LGI A1 auto-antibodies. The patient also tested and negative for Voltage-Gated Potassium-Channel antibodies and n-methyl-daspartate antibodies. Electroencephalography (EEG) record demonstrated right temporal sharp wave discharge. A clinical suspicion of neurodegenerative disorder versus nonparaneoplastic encephalitis versus limbic encephalitis secondary to known carcinoma breast was raised and was referred for further evaluation with FDG PET-CT scan on discussion. She underwent whole-body 18F-FDG PET-CT scan, 60 min after intraveneous injection of 8 mCi of FDG. The PET-CT is acquired and reconstructed, the images demonstrated

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marked asymmetrical FDG hypermetabolism in right medial temporal lobes predominantly in right hippocampus and amygdala region [Figure 2] and in right basal ganglia involving the caudate nucleus and putamen [Figure 3]. Rest of the whole-body FDG PET-CT scan was unremarkable with no abnormal metabolism or lesion anywhere else in the body. Therefore, on behalf of the clinical, MRI, and PET-CT scan results, a diagnosis of nonparaneoplastic autoimmune limbic encephalitis (AE) was rendered.

Discussion

AE is categorized in 2 main groups–paraneoplastic and nonneoplastic on basis of whether the antibodies are due to the presence of an underlying tumor or without tumor. The common tumors are squamous cell carcinoma lung, testicular germ cell tumor, and thymic, breast, renal, colonic, esophageal, ureteric bud, and ovarian carcinomas.[1]

The nonneoplastic group of patients usually responds favorably to immunotherapy.[2] Diagnosis of AE requires to meet following five criteria:[1] subacute onset,[2] T2 FLAIR MRI brain abnormalities restricted to mesial temporal lobes,[3] abnormal routine CSF analysis and detectable antibodies in CSF,[4] abnormal epileptic and slow wave EEG activity in mesial temporal cortex, and[5] keeping out of other possible etiology of AE.[3]

Definitive diagnosis is characterized by the appearance of specific antibody in CSF or blood and response to targeted immunotherapy.[4] On MRI, T2-weighted FLAIR images how hyperintensities with swelling usually restricted to the mesial-temporal cortex. Asymmetric bilateral involvement being most common (60%). Basal ganglia are also frequently involved with less common involvement of the lateral temporal lobe and insula. Contrast enhancements rare with no diffusion restriction and hemorrhage. Many studies have demonstrated hypermetabolism on PET even in MRI-negative or inconclusive cases.[5]

FDG PET Scan in AE usually demonstrates hyper-metabolism in medial temporal cortex, orbitofrontal cortex, occipital hypo-metabolism with symmetrical hyper-metabolism in striatum and amygdala and may even resemble neurodegenerative disease in older patients.[6,7] Although PET scan is helpful in identifying an underlying disease to some extent, diagnosis of AE is still challenging due to the varied presentation and nonspecific initial laboratory as well as imaging findings. Simultaneous PET/MRI combines high-resolution structural and functional information from multiparametric MRI along with metabolic information from PET to localize the abnormality, to rule out other primary abnormalities in the brain, and to identify any other malignancy in the body to rule out paraneoplastic limbic encephalitis.[8] Dedicated FDG PET of brain is always remarkable in patients with AE, and FDG PET findings are more specific for

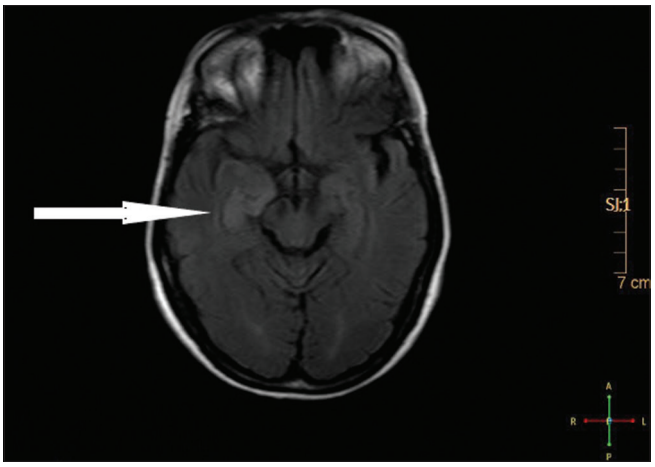

Figure 1: Magnetic resonance imaging T2 fluid-attenuated inversion recovery image of brain showing increased signal intensity in the right medial temporal cortex in the region of amygdala and hippocampus (white arrow)

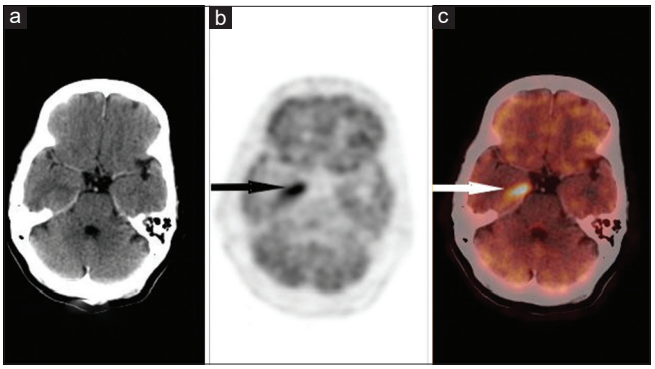

Figure 2: Fluorodeoxyglucose brain positron emission tomography-computerized tomography images in consecutive computerized tomography (a), Positron emission tomography (b) and fused Positron emission tomography/computerized tomography (c) views showing increased metabolic activity in right medial temporal cortex (black and white arrows) with no significant morphological changes on computerized tomography images

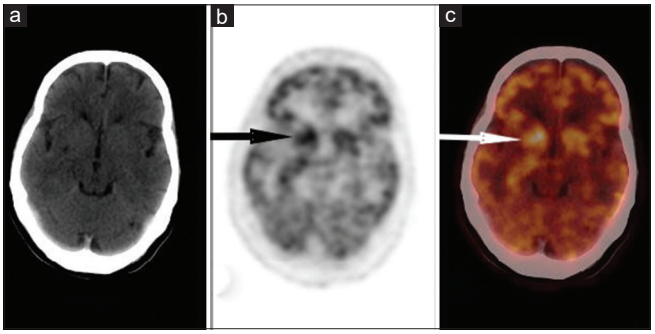

Figure 3: Fluorodeoxyglucose brain positron emission tomography-computerized tomography images in consecutive Computerized tomography (a), Positron emission tomography (b) and fused Positron emission tomography/computerized tomography (c) views showing increased metabolic activity in right basal ganglia (black and white arrows) with no significant morphological changes on computerized tomography images

AE as compared with other EEG, brain-MRI, and CSF analysis. The brain region hypometabolism is seen and

documented in most of the studies, however many studies demonstrated hyper and hypometabolism both and a few with hypermetabolism alone on brain FDG PET scan. The group of findings seen on dedicated brain FDG PET CT scan as abnormal metabolism have a fair agreement with 2 of the 3 paraclinical tests (EEG, MRI brain, and routine CSF examination).[9] In our study, the results suggested that whole-body FDG PET-CT scan with dedicated brain PET is providing evidence to support brain dysfunction in patients with suspected AE and also helping us to rule out the paraneoplastic cause as the patient is already a treated case of carcinoma breast with no evidence of hypermetabolic or active metastasis on present scan. Brain region hypo and hypermetabolism simulate the damage of neuronal activity in AE. This abnormal metabolism is result of either a functional or a structural abnormality, or the combination of two is still questionable. Longitudinal studies are, however, further required to clarify the observed hypo or hypermetabolism is reversible in AE posttreatment.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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