Anti-Leucine-Rich Glioma-Inactivated 1 Limbic Encephalitis with Normal Magnetic Resonance Imaging Detected on Fluordeoxygluose Positron Emission Tomography/Computed Tomography

Abstract
Leucine-rich glioma-inactivated 1 (LGI1) antibody encephalitis is an emerging autoimmune disorder with antibodies to the voltage-gated potassium channel complex. Here, we report clinico-imaging findings of a 77-year-old female presenting with acute-onset seizures, normal magnetic resonance imaging and with abnormal fluordeoxyglucose positron-emission tomography-computed tomography and positive anti-LGI1 antibodies on immunofluorescence assay.

Keywords: Autoimmune, encephalitis, fluordeoxyglucose positron-emission tomography, leucine-rich glioma-inactivated 1, limbic, magnetic resonance imaging, positron-emission tomography-computed tomography

Seventy-seven-year-old female, with a history of hypertension, presented to the emergency department with two episodes of generalized tonic-clonic seizures. Routine laboratory investigations only revealed low serum sodium (116 mEq/L, Normal 136–142 mEq/L) and raised C-reactive protein (CRP) (22 mg/L, Normal <10 mg/L). Cerebrospinal fluid (CSF) analysis was normal, and CSF microarray test was negative for any viral encephalitis. An electroencephalogram (EEG) was suggestive of low amplitude polyphasic delta slow waves in the left anterior temporal lobe region Figure 1. Brain magnetic resonance imaging (MRI) and magnetic resonance (MR)-angiography done on day 3 of admission was unremarkable. In view of this EEG picture and high CRP, fluordeoxyglucose (FDG) positron-emission tomography-computed tomography (PET/CT) was ordered to evaluate for limbic encephalitis. FDG PET/CT done on day 4 showed intense FDG uptake in the entire left mesial temporal lobe (Cortex ID Z score + 30.27) Figure 2. Rest of the whole-body scan was unremarkable for any malignancy. Cell-based indirect immunofluorescence assay revealed positive leucine-rich glioma-inactivated 1 (LGI1)-antibody in the serum sample.

Autoimmune encephalitis is subclassified on the type of antibodies identified on various assays. These include autoantibodies to intracellular antigens (Hu, Ma2, glutamic acid decarboxylase (GAD)), to synaptic receptors (N-methyl-D-aspartate, Gamma amino-butyric acid (GABA), amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid, mGluR5, D2 receptor) or against ion channels (LGI1, CASPR2, dipeptidyl-peptidase-like protein 6). Most patients with autoimmune encephalitis have a delay in clinical diagnosis due to the lack of well-defined clinical syndrome or normal or nonspecific MR findings, especially early in the disease course. Anti-LGI1 antibody-related encephalitis are rarely associated with tumors and tends to respond...
well to treatment with more favorable outcomes seen in those treated with combined steroid and immunotherapy than those with monotherapy.[2] Hyponatremia seen in about 60% of these patients is presumed to be a result of inappropriate anti-diuretic hormone secretion, which is probably related to the co-expression of LGI1 in hypothalamus/kidneys.[3] In the acute phase of the disease, it is not unusual to have a normal MRI and abnormal FDG PET is seen in about three-fourth of the patients.[3,4] FDG PET/CT done in acute phase of this disease frequently shows hyper-metabolism in bilateral basal ganglia and/or bilateral mesial temporal lobes, with unilateral involvement of mesio-temporal lobe without basal ganglia involvement (seen in case reported above) appears to be relatively uncommon.[4-7]

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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