

F-18 Fluorodeoxyglucose Positron Emission Tomography Metabolic Phenotype in Myelin Oligodendrocyte Glycoprotein Antibody-Positive Autoimmune Epilepsy

Abstract

We describe the metabolic phenotype on F-18 fluorodeoxyglucose positron emission tomography (PET) in a 13-year-old female with myelin oligodendrocyte glycoprotein (MOG) antibody-positive encephalitis. Unilateral hemispheric hypometabolism on PET may be the metabolic phenotype of autoimmune epilepsy associated with MOG antibody.

Keywords: Autoimmune encephalitis, fluorodeoxyglucose positron emission tomography, myelin oligodendrocyte glycoprotein, myelin oligodendrocyte glycoprotein

A 13-year-old female presented with recent-onset left focal seizures and painful vision reduction in both eyes. Contrast-enhanced magnetic resonance imaging (MRI) was performed. Axial T1 [Figure 1a], T2 [Figure 1b], and fluid attenuated inversion recovery (FLAIR) [Figure 1c] images at the level of upper midbrain showed multifocal subcortical white matter lesions, iso-hypointense on T1, hyperintense in T2 and FLAIR, in the right perisylvian region [arrows in Figure 1b and c]. Coronal T2-weighted image (WI) [Figure 1d] showed focal hyperintense lesions in the right inferior frontal (long arrow in d) and superior temporal [short arrow in [Figure 1d] subcortical white matter; these lesions [long arrow and short arrows in Figure 1f] enhanced in postgadolinium coronal T1-WI [Figure 1f]. Postgadolinium axial T1-WIs [Figure 1e] showed patchy subcortical enhancement (arrow in e) in the right cerebral hemisphere. She was referred for ¹⁸F-fluorodeoxyglucose positron emission tomography (¹⁸F-FDG PET) to rule out paraneoplastic etiology. Brain axial and coronal PET-MR fusion images revealed hypometabolism in the right cerebral hemisphere including both visual cortices [Figure 1g and h]. No metabolically active lesion to suggest a primary was identified on whole body PET. Cerebrospinal

fluid did not reveal any significant abnormality, and screening for AQP4, NMDAR, AMPA, LGI1, CASPR2, and TPO antibodies was negative, while myelin oligodendrocyte glycoprotein (MOG)-IgG was strongly positive. She was started on methylprednisolone pulse therapy following which there was improvement in vision.

MOG antibodies are directed against a myelin protein MOG expressed at the outermost lamellae of the myelin sheath in the central nervous system (CNS).^[1,2] The phenotypic presentation of inflammatory diseases with MOG antibodies can be similar to that of neuromyelitis optica spectrum disease with unilateral or bilateral optic neuritis.^[3,4] The other presentation that has been described in adults is a unilateral hemispheric cortical encephalitis, wherein hyperperfusion was noted on single photon emission computed tomography.^[5] The MRI and PET findings in this 13-year-old girl supported a unilateral hemispheric involvement. Different metabolic patterns have been reported on ¹⁸F-FDG PET in autoimmune encephalitis with hypometabolism and hypermetabolism; however, so far, hemispheric involvement has not been reported.^[6-9] Inflammatory demyelinating diseases with overlapping clinical features, as well as similarities in MRI, often make diagnosis at onset difficult,^[10] and ¹⁸F-FDG PET/CT metabolic phenotypes could be useful in specific antibody subtypes such as anti-MOG.

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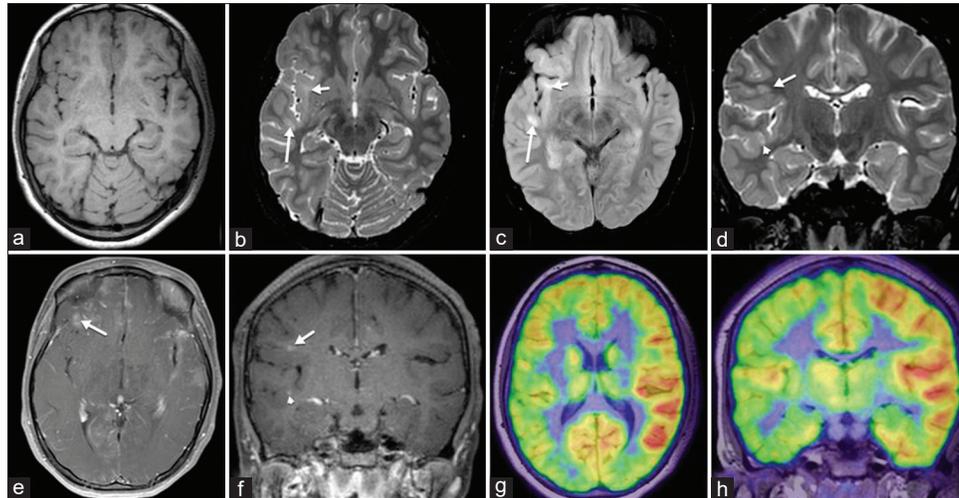


Figure 1: Contrast-enhanced magnetic resonance axial T1 (a), T2 (b), and fluid attenuated inversion recovery (c) images showing multifocal subcortical white matter lesions, iso-hypointense on T1, hyperintense in T2 and fluid attenuated inversion recovery (arrows). Coronal T1-weighted image (d) showing focal hyperintense lesions in the right inferior frontal (long arrow) and superior temporal (short arrow) subcortical white matter, which (long and short arrows) enhanced in postgadolinium coronal T1-weighted image (f). Postgadolinium axial T1-weighted (e) images showing patchy subcortical enhancement (arrow) in the right cerebral hemisphere. Axial and coronal positron emission tomography/magnetic resonance fusion images revealed hypometabolism in the right cerebral hemisphere (g and h)

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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