BRIEF COMMUNICATION

A Case of Multiple Sclerosis with Homonymous Hemianopia Examined by Positron Emission Tomography

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Abstract

Background: To demonstrate the efficacy of positron emission tomography (PET) for examining multiple sclerosis (MS) patients with hemianopia.

Case: A 20-year-old man visited us with a complaint of left homonymous hemianopia and headache.

Observations: The patient’s visual acuity was 1.2 (n.c.) OD and 0.9 (1.0) OS. Magnetic resonance imaging (MRI) showed a mass in the temporoparietal lobe. A pathological diagnosis of MS was made by brain biopsy. Low glucose metabolism in the lesion and visual cortex was observed by PET with 18F-fluorodeoxyglucose. PET with 11C-flumazenyl revealed a reduction of 11C-uptake in the demyelinated optic radiation, and only a slight reduction of 11C-uptake in the primary visual cortex. The results of 11C-flumazenyl PET suggested a slight reduction of neuronal density. In 2 years, the visual field recovered to the normal state.

Conclusion: PET can be a useful tool for estimating the visual outcome of patients with hemianopia in MS.

Key Words: glucose metabolism, homonymous hemianopia, multiple sclerosis, positron emission tomography, receptor density

Introduction

Homonymous hemianopia is rarely seen in cases of multiple sclerosis (MS). We report a patient who was diagnosed with MS and whose first neurological sign was homonymous hemianopia. Positron emission tomography (PET) revealed the characteristic changes of cerebral glucose metabolism (CMRGluc) and the neuronal density in optic radiation and in the primary visual cortex (PVC) on the affected side.

Case Report

A 20-year-old man visited us on June 22, 2001, with a complaint of left homonymous hemianopia and headache. His medical and family history was unremarkable. His visual acuity was 1.2 (n.c.) OD and 0.9 (1.0) OS. The pupillary light reflex was normal, and a relative afferent pupillary defect was not present. The anterior segment, ocular media, and fundi were normal. Goldmann perimetry revealed complete left homonymous hemianopia.

Neurological examination revealed no abnormality other than those found by the ophthalmological tests. Lumbar puncture disclosed clear liquid with 14 cells/µl, but myelin basic protein was not present. A lesion in the parietal lobe was well enhanced in T1-weighted magnetic resonance (MR) images and extended into the surrounding area of the right lateral ventricle. Computed tomography (CT)-
Figure 1A–D. Histopathological findings in the masslike lesion. A Infiltration of mononuclear cells into cerebral parenchyma. H&E, bar = 50µm. B Small lymphocytes infiltrating around venules. H&E, bar = 33µm. C Myelin sheaths are not heavily stained. Klüver-Barrera staining, bar = 50µm. D Axons are relatively well preserved. Bodian staining, bar = 25µm.

Figure 2. T1-weighted magnetic resonance image (MRI) (left), 18F-fluorodeoxy glucose positron emission topography (FDG-PET) image (middle), 11C-flumazenil(FMZ)-PET image (right) on 23 December 2001. MRI: There is a demyelinated lesion in the white matter in the parietal lobe (arrow). FDG-PET image: Glucose hypometabolism was detected in the right lesion, which corresponded to the demyelinated white matter (arrow), and there is extreme glucose hypometabolism in right primary visual cortex (arrow). FMZ-PET image: There is a little reduction of benzodiazepine receptor density in the right optic radiation, and there is only a slight reduction of benzodiazepine receptor density in right primary visual cortex (arrow).