

Neuroimaging Analysis of a Case with Left Homonymous Hemianopia and Left Hemispatial Neglect

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Purpose: To correlate the neuro-ophthalmological observations with the magnetic resonance images (MRI) and positron emission tomographic (PET) findings in a case with left homonymous hemianopia and left hemispatial neglect.

Case: A 57-year-old woman underwent surgery for a ruptured anterior communicating artery aneurysm. After she recovered consciousness, it was found that she had left homonymous hemianopia and left hemispatial neglect. Although the hemispatial neglect slowly improved, the homonymous hemianopia persisted. MRI and measurements of cerebral glucose metabolism by 2-fluoro-2-deoxy-D-glucose (FDG)-PET were performed 1 year later.

Results: MRI revealed infarctions on the medial surface of the frontal lobe, on the right medial surface of the occipital lobe, and global atrophy of the right cortical hemisphere. FDG-PET disclosed severe glucose hypometabolism in the entire right hemisphere. Glucose metabolism in the right occipital cortex was 61.1% of that in the homologous region on the left side, 62.8% in the right anterior cingulate gyrus, and 93.8% in the temporal-parietal-occipital junction.

Conclusions: The low glucose metabolism in the right visual cortex explains the persistent left hemianopia, and that in the right anterior cingulate gyrus and the right temporal-parietal-occipital junction may be responsible for the left hemispatial neglect. The relatively mild damage in the right temporal-parietal-occipital junction explained the recovery of the neglect symptom. Measurements of regional cerebral glucose metabolism by PET are useful for determining the cause of cerebral visual dysfunction and its prognosis after a cerebral lesion.
Jpn J Ophthalmol 2003;47:59–63 © 2003 Japanese Ophthalmological Society

Key Words: Glucose metabolism, hemispatial neglect, homonymous hemianopia, positron emission tomography, subarachnoid hemorrhage.

Introduction

Hemispatial neglect is a unique failure in orienting stimuli located in the hemispace contralateral to a cerebral lesion, and is not attributable to either sensory or motor deficits.¹ Thus, hemispatial neglect is a

defect of attention to unilateral space, and visual and/or other types of stimuli in the affected hemi-field are totally unrecognized in cases of severe hemispatial neglect. In cases of moderate hemispatial neglect, an unequal cognition of stimuli in each hemispace is detected. Hemispatial neglect frequently accompanies homonymous hemianopia, however, these two conditions are ascribable to different pathophysiological mechanisms. Patients with homonymous hemianopia do not detect visual stimuli in the unilateral visual hemifield due to postchiasmatal lesions in the vi-

Received: February 12, 2002

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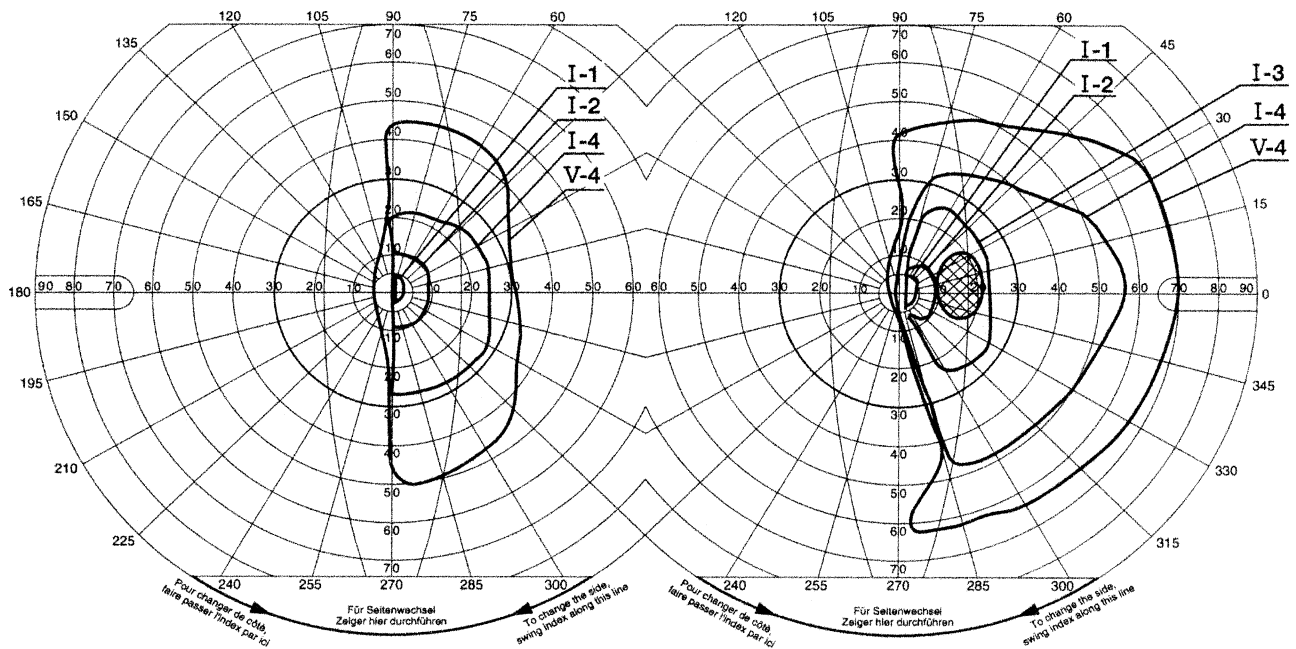


Figure 1. Visual fields of the patient at the first visit. Goldmann perimetry shows left complete homonymous hemianopia without macular sparing.

sual pathway contralateral to the visual field defect, eg, lesions in the optic tract, lateral geniculate body, optic radiation, or primary visual cortex.

Although hemispatial neglect is one of the most important vision-related symptoms after cerebral insults, not many ophthalmologists recognize the presence of this sign. Hemispatial neglect and homonymous hemianopia can be observed simultaneously in a patient, making a precise diagnosis difficult.

We report a patient with left hemianopia and left hemispatial neglect who had severe brain damage following a subarachnoid hemorrhage. With the passage of time, the hemispatial neglect improved to some degree, but the homonymous hemianopia persisted. We report the correlation of the neuro-ophthalmological findings with the neuroimaging data including magnetic resonance imaging (MRI) and 2-fluoro-2-deoxy-D-glucose positron emission tomography (FDG-PET).

Case Report

A 57-year-old woman was diagnosed as having a subarachnoid hemorrhage due to a ruptured aneurysm of the anterior communicating artery on March 21, 2000. Intracranial pressure remained high after clipping surgery for the aneurysm and she developed falx herniation causing infarctions in the frontal lobe and the right occipital lobe. A ventriculo-peritoneal

shunt led to gradual recovery of consciousness by 15 days after the procedure, followed by rehabilitation training for several months.

One year later, her husband noticed that she continued to have difficulty in seeing food located on the left side of the table, despite a certain improvement in her motor skills. On March 5, 2001, the patient visited us with a complaint of difficulty in recognizing objects in the left side of the visual field. She was orthophoric and ocular movements were full. Visual acuity was RE: 0.01 (0.4 x –19.00 D); LE: 30 cm/n.d. (0.2 x –20.50 D). The intraocular pressure was 12 mm Hg in both eyes.

Pupillary reaction to light was normal. Incipient cataracts were observed. Fundus examination showed bilateral diffuse myopic chorioretinal atrophy in the macula. Goldmann perimetry revealed left homonymous hemianopia without macular sparing (Figure 1). The Line Cancellation Test showed short lines missing in the left half of the test sheet (Figure 2A). The Line Bisection Test showed a rightward shift of the bisection points. From these findings, a diagnosis of left hemispatial neglect with left homonymous hemianopia was made.

FDG-PET studies were performed to determine the region responsible for the hemispatial neglect and to evaluate the severity of the damage in the lesion. A written informed consent for PET was obtained.

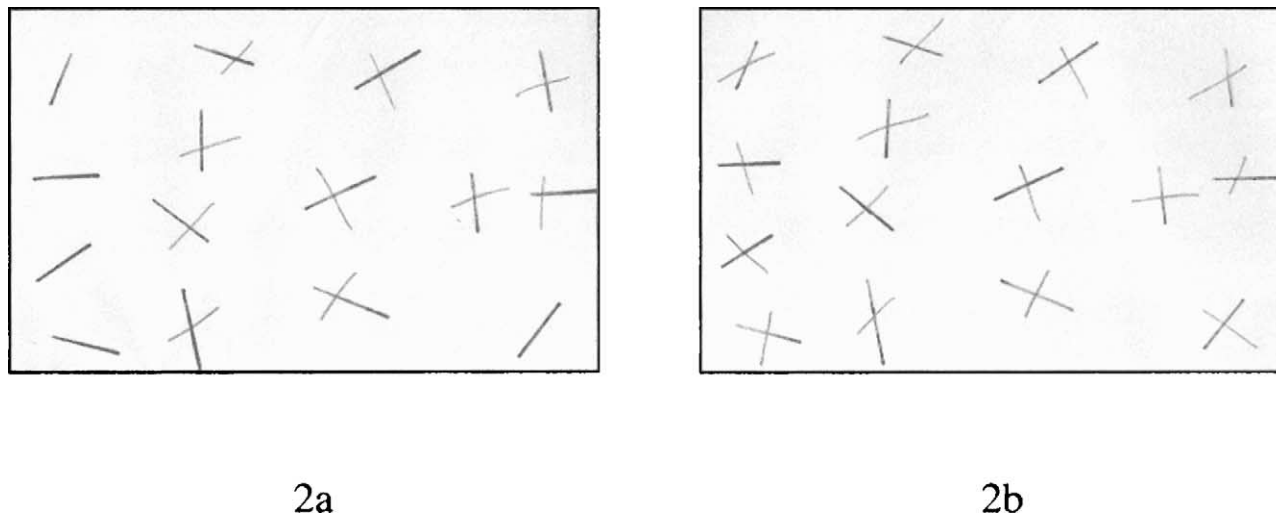


Figure 2. Line Cancellation Test. On the first visit (a), the Line Cancellation Test shows left hemispatial neglect. Test on the day of positron emission tomography study 1 year later shows recovery of the left hemispatial neglect symptom (b).

During the 2-month follow up, the left hemianopia persisted and the left hemispatial neglect improved. The results of the Line Cancellation Test on April 13, 2001, were almost normal (Figure 2b).

Results

MRI and FDG-PET images were obtained on May 17, 2001, after a partial recovery of the hemispatial neglect was noticed (Figure 3). MRI showed atrophy of the entire right-hemispheric cortex and cerebral infarctions bilaterally in the medial orbito-frontal region, in the right medial surface of the frontal lobe, and in the right medial surface of the occipital lobe. FDG-PET revealed global hypometabolism in the right hemisphere, more prominently in the right frontal lobe and right occipital lobe (Figure 3, right). A summary of the cerebral glucose metabolism evaluated by Standardized Uptake Values (SUV)² is presented in Table 1. The glucose metabolism in the right occipital lobe was 61.1% of that in the left side. In the right anterior cingulate gyrus, it was 62.8% of that in the left. In the right temporal-parietal-occipital junction, it was 93.8% of that in the left.

Discussion

This patient developed brain edema and falx herniation of the brain following a subarachnoid hemorrhage resulting in cerebral damage. Left homonymous hemianopia and left hemispatial neglect were detected after the recovery of consciousness. These deficits are ascribable to the damage in the right hemisphere found by MRI and PET.

Hemispatial neglect is considered to be caused by disturbances of hemispatial attention. The parietal lobe, the frontal eye fields (Brodmann area 8), the superior colliculus, the anterior cingulate gyrus, and the pulvinar nucleus of the thalamus have been observed to be responsible for a shifting attention task.³ In a PET study of 8 right-handed, normal volunteers, Gitelman et al⁴ reported activation of the right cingulate, the premotor, posterior parietal cortex, and caudate nucleus during a spatial exploration task without visual input. Leibovitch et al⁵ investigated 81 acute stroke patients with unispatial neglect associated with lesions in the right hemisphere by means of single photon emission computed tomography and demonstrated that the most influential region was the cortical area surrounding the right temporal-parietal-occipital junction, and that accompanying hypoperfusion in the medial frontal cortex, including the anterior cingulate gyrus, caused more severe hemispatial neglect.

The site responsible for the hemispatial neglect in our case is considered to be a combination of the right temporal-parietal-occipital junction and the medial frontal region. Its partial recovery is ascribable to the mild damage of the right temporal-parietal-occipital junction. The hemispatial neglect improved in our case during the 2-month follow-up period; however, the reason for the delayed recovery was not clear. The rehabilitation training to direct attention to the left hemifield in the repeated Line Cancellation Test may have brought about a slow improvement. We can point out two additional explanations. First, as Perani et al⁶ suggested, based on the results of an FDG-PET

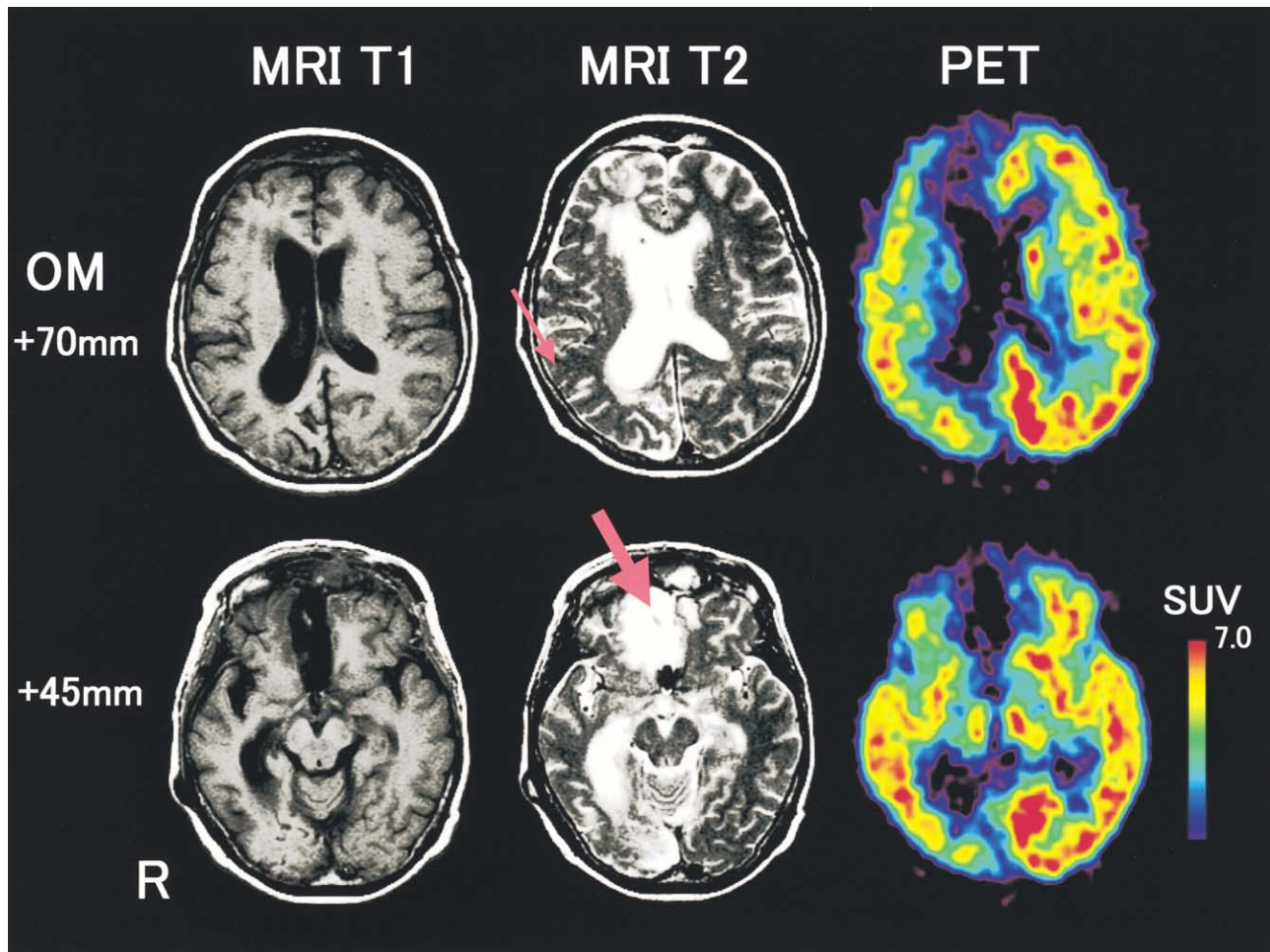


Figure 3. Magnetic resonance imaging (MRI) and fluorodeoxyglucose(FDG)-positron emission tomography (PET) images after incomplete recovery of hemispatial neglect. T1-weighted MRI (left), T2-weighted MRI (middle), and FDG-PET image (right). Images in the upper and lower rows are slices 70 and 45 mm above the orbito-meatal line. MRI shows cortical atrophy of the entire right hemisphere, and cerebral infarction in the right medial surface of the frontal lobe and in the right medial surface of the occipital lobe. The small arrow points to the right temporal-parietal-occipital junction. The large arrow points to the right anterior cingulate gyrus. The PET images reveal glucose hypometabolism in all of the right hemisphere.

study of two cases of hemispatial neglect, remission of hemispatial neglect might be associated with a functional metabolic recovery in both the undamaged left hemisphere and in the unaffected regions of the right hemisphere. A second possible explanation is a reorganization of cerebral function. Pizzamiglio et al⁷ reported on the results of $H_2^{15}O$ -PET activation studies on three chronic phase patients with hemispatial neglect who showed symptomatic recovery. They suggested that the more active regions were found in the cortical areas of the right hemisphere after a 2-month rehabilitation program. In our case, the functional recovery should be considered in

terms of reorganization in both the nonaffected left hemisphere and the damaged right hemisphere.

Hemispatial neglect is one of the most important neuropsychological symptoms related to vision. Although the temporal-parietal-occipital junction is the most common site for a causative lesion, the medial surface of the frontal cortex is also a possible site. Low glucose metabolism in the medial frontal cortex with mild reduced glucose metabolism in the temporal-parietal-occipital junction was observed in our patient. We considered that there was remission of hemispatial neglect because of the mild reduced hypometabolism in the temporal-parietal-occipital junction.

Conclusions

A patient presented with left homonymous hemianopia with left hemispatial neglect after a subarachnoid hemorrhage. MRI revealed infarction in the frontal lobe and in the right medial surface of the occipital lobe. The low glucose metabolism in the right visual cortex, confirmed by FDG-PET, explains the persistent left hemianopia, and that in the right anterior cingulate gyrus and the right temporal-parietal-occipital junction may be responsible for the left hemispatial neglect.

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