CASE REPORT

Homonymous Visual Field Loss without Structural Lesion on Magnetic Resonance Imaging: Documented with Positron Emission Tomography and Diffusion Tensor Imaging

Tae-Ho Yang¹, Sun-Young Oh^{1,2}, Kichang Kwak³, and Jong-Min Lee³

¹Department of Neurology, Chonbuk National University College of Medicine, and Research Institute of Clinical Medicine, Chonbuk National University, Jeonju, Korea, ²Biomedical Research Institute of Chonbuk National University Hospital, Jeonju, Korea, ³Department of Biomedical Engineering, Hanyang University, Seoul, Korea

ABSTRACT

The authors describe a 35-year-old man suffering from homonymous hemianopia after head trauma 4 years before but with negative magnetic resonance imaging (MRI) findings. Brain fluorine-18 fluorodeoxyglucose positron emission tomography (¹⁸FDG-PET) showed hypometabolism at the unilateral occipital lobe and crossed cerebellar hemisphere, and diffusion tensor imaging (DTI) revealed that the ipsilateral optic radiations were completely interrupted. The crossed cerebellar diaschisis (CCD) observed in the chronic stage of brain damage was caused by cerebellar suppression of the cerebral blood flow due to an involvement of the corticopontocerebellar tract. PET and DTI provide objective means for determining the relationship of functional deficits to head trauma, even in cases where the injury was sustained years prior to the evaluation.

Keywords: Crossed cerebellar diaschisis, diffusion tensor imaging, homonymous hemianopia, traumatic brain injury

INTRODUCTION

Retrochiasmal visual pathways anywhere from the optic tract to the visual cortex can cause homonymous hemianopia (HH) in contralateral visual field and which can be usually demonstrated by neuroimaging. However, HH can occur rarely in patients with no definite structural lesion, such as carbon monoxide poisoning, global hypoxia, migraine or seizure, subtle occipital ischaemia, anaemia, Creutzfeld-Jacob disease, Alzheimer disease, nonketotic hyperglycemia, head trauma, or functional illness.¹

Crossed cerebellar diaschisis (CCD) is defined as metabolic depression on the cerebellar hemisphere contralateral to supratentorial lesions. CCD has been noted to accompany symptoms of hemiparesis and

ataxia with pathological conditions such as infarcts, haemorrhages, trauma, arteriovenous malformation, encephalitis, epilepsy, hydrocephalus, dementia, Moyamoya disease, and tumours.² It has been usually observed in cases of frontal or parietal lobe lesions; however, there have been also reports of CCD in other hemispheres, including the occipital visual cortex.³

We report a young man who presented homonymous hemianopia and mild ataxia after head trauma, but without lesion on the brain magnetic resonance imaging (MRI). However, brain fluorine-18 fluorodeoxyglucose positron emission tomography (¹⁸FDG-PET) revealed crossed hypometabolism of the occipital cortex and cerebellum, suggestive of CCD. We also use diffusion tensor imaging and fibre tractography

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Correspondence: Sun-Young Oh, MD, PhD, Department of Neurology, Chonbuk National University Hospital, Research Institute of Clinical Medicine, Chonbuk National University, 20 Geonji-ro, Deokjin-gu, Jeonju-city, Chonbuk, 561-712, South Korea. E-mail: ohsun@jbnu.ac.kr

(DTI-FT) to identify the most likely pathway between the lateral geniculate nucleus (LGN) and the calcarine cortex in our patient, which revealed completely interrupted ipsilateral optic radiations. This case showed that CCD after head injury is associated with HH without structural lesion on the conventional MRI and that HH associated with CCD can be documented as cortical hypometabolism of visual cortex and the prominent structural lesion of fibre tract on the visual pathway with PET and DTI.

CASE REPORT

A 35-year-old man visited our department for an evaluation of a gradual change in his vision and subtle left upper extremity clumsiness since 4 years ago following an autobike accident. Examinations revealed mild dysmetria on the left limbs by finger-to-nose and heel-to-shin tests, left HH by a

confrontation method, and a tendency to fall toward the left side on tandem gait. Humphrey visual field measurement demonstrated a complete left homonymous hemianopia (Figure 1A). Other ophthalmic findings were normal, including visual acuity, intraocular pressure, and fundus photography. Visual evoked potential responses were also normal. Laboratory findings did not show abnormal result on complete blood count, chemistry, glucose, thyroid function test, erythrocyte sedimentation rate and C-reactive protein, vitamin B12, rheumatoid factor, antinuclear antibody, antibody for syphilis, and human immunodeficiency virus. Previous personal histories including seizure or developmental anomaly were negative.

A conventional brain MRI was normal except for mild atrophy and focal hyperintensity in the right upper midbrain and pons, as shown in the T2-weighted axial imaging. No structural lesions of the posterior visual pathways are identified (Figure 1b–d). However, ^{18}F FDG-PET scans showed

FIGURE 1 (a) Visual fields over the central 30 degrees (Humphrey Visual Field Analyzer; Humphrey Instruments, Dublin, California) revealed a dense left homonymous hemianopia. (b) The T2-weighted axial MRI revealed mild atrophy and focal hyperintensity in the right upper midbrain and pons. No definite lesions of the posterior visual pathways are identified in the T2-weighted axial imaging (c) and diffusion-weighted imaging (d).

severe glucose hypometabolism both in the right occipital lobe and in the left cerebellum (Figure 2a). We also analysed the reconstructed diffusion tensor fibre tractography with the following parameters: echo time 100 ms, repetition time 4000 ms; matrix 128×128 interpolated 256×256 ; field of view 220×220 mm; slice thickness 2 mm; and diffusion number 32 directions with the b 1000 s/mm². To visualise the optic radiations and the middle cerebellar peduncle, we drew a region of interest as a seed on lateral geniculate body and medial cerebellar peduncle. To trace axonal projections, the tensor was tracked and lines were drawn in both anterograde and retrograde directions in three-dimensional space from seeds along the major eigenvector. The bilateral middle cerebellar peduncles were shown symmetrically (Figure 2b), and the left optic radiations from the lateral geniculate body

were only shown without revealing the right one (Figure 2c).

DISCUSSION

Each cerebellar hemisphere is intimately connected to the contralateral cerebral cortex through feedback circuits for smooth execution of motor function. The main pathophysiological mechanism of CCD may be due to functional suppression of the corticopontocerebellar tract, which is the largest pathway derived from extensive areas of the cerebral cortex, projects to the ipsilateral pontine nuclei, which send axons to the contralateral cerebellar cortex via the superior and middle cerebellar peduncles. The topography of cerebral lesion and its relationship to frequency and severity of CCD may be controversial; however, CCD

FIGURE 2 (a) The ¹⁸F-DG-PET (fludeoxyglucose positron emission topography) scans revealed significant hypometabolism in the right visual cortex (occipital lobe; arrowhead) with associated crossed cerebellar diaschisis demonstrated by hypometabolism in the left cerebellum (arrows). The reconstructed diffusion tensor fibre tractography of the middle cerebellar peduncle (b) and the optic radiation (c) using color mapping. There is no significant difference in the shape of nerve tract at the bilateral middle cerebellar peduncle (b), but the left optic radiations from the lateral geniculate body (arrow) were only shown without revealing the right one (arrowhead) (c).

may occur more frequently in patients with frontal lobe lesions, single or multiple, and in patients with motor impairment because the bulk of cortico-cerebellar connections to the cerebellar hemisphere arise from the motor and premotor cortices.^{4,5} Therefore, CCD was present usually in patients with hemiparesis.⁴ The isolated complete homonymous hemianopia (HH) and mild ataxia in CCD without prominent hemiplegia is rare.

CCD could be potentially reversible in cases of supratentorial reperfusion seen within hours of a stroke and lesions such as moyamoya disease or transient ischaemic attack.⁵ However, CCD could be persistent in association with irreversible supratentorial tissue damage, such as cerebral infarction, haematoma, and brain tumour,⁶ and usually showed poor clinical outcome.⁷ There are reports on the delayed onset of CCD after brain injury in childhood, and this could be related to maturation of the corticopontocerebellar tract in the developing brain. CCD of our case associated with head trauma is detected after 4 years after the injury and it can be persistent.

The diffuse axonal injury (DAI) after head trauma can split the optic chiasm and produce a bitemporal hemianopia and may damage the white matter tracts of the optic radiations.⁸ More posterior injuries including the level of the occipital cortex produce homonymous field defects, which have been reported to cause diffusely decreased metabolism in the parieto-occipital cortex.⁹ These structural disruptions, however, have been usually observed in radiological imaging, including brain MRI or computed tomographic (CT) scan. The present case did not reveal definite structural lesion on the visual pathways on conventional MRI. However, crossed hypometabolism in the right occipital lobe and the left cerebellum on ¹⁸FDG-PET and disrupted right optic radiation on the DTI fibre tract map were demonstrated. Cerebral contusion causes reduced diffusion anisotropy,^{10,11} which can produce a fibre-tracking defect. DTI allows for the specific examination of the integrity of white matter tracts, which are especially vulnerable to the mechanical trauma of brain. Standard MR structural imaging itself may be not sensitive enough in identifying impairment in mild injury.¹² In chronic traumatic brain injury, reduced fractional anisotropy (FA) has been reported,^{13,14} even in the absence of observable lesions in standard structural MRI.¹³ Because DTI is more sensitive to changes in the microstructure of white matter, it shows considerable promise in the assessment of traumatic brain injury. In our patient, the metabolic defected area located on the main trajectory of the left optic radiation was morphologically destroyed 4 years after onset on fibre tract maps.

Although standard anatomic MR imaging shows promise for quantifying pathophysiology in traumatic brain injury, it may not be as sensitive to the

neuropathology of mild cerebral dysfunction.¹² Our study suggests that brain PET and DTI are very promising methodology in this regard, and useful for investigating cerebral visual field disorders, including those in the chronic phase.

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Note: Figure 2 of this article is available in colour online at <http://www.informahealthcare.com/oph>.

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