CASE REPORT

Recovery of Visual Field Defect via Corpus Callosum in a Patient with Cerebral Infarct

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ABSTRACT

Recovery mechanism of visual field defect in stroke patients has not been clearly elucidated. In this study, we report on a patient with a cerebral infarct who showed recovery of visual field defect via the corpus callosum, using diffusion tensor tractography (DTT) for optic radiation (OR). A 57-year-old male patient underwent conservative management for a cerebral infarct in the subcortical white matter of the right temporal lobe. Left homonymous hemianopsia was detected on the 2-week Humphrey visual field test. However, the patient showed improvement of hemianopsia with time; consequently, a left lower peripheral visual defect with the resolution of the upper and medial portions of the initial left hemianopia was observed on the 11-month Humphrey visual field test. Both 2-week and 11-month DTTs for the right OR showed a discontinuation between the right lateral geniculate nucleus (LGN) and the right OR. On 2-week DTT, the left OR was connected to the transcallosal fibres, and on 11-month DTT, these transcallosal fibres were elongated to the right primary visual cortex via the right posterior OR. The visual field defect in this patient appears to have recovered by the neural pathway originating from the left OR and terminating in the primary visual cortex via the transcallosal fibres and right distal OR. We believe that the results of this study may suggest one of the mechanisms for recovery of visual field defect following injury of OR in stroke patients.

Keywords: Brain plasticity, diffusion tensor tractography, hemianopia, optic radiation, visual field defect

INTRODUCTION

Injury of optic radiation (OR) can occur in stroke, and visual field defect is a typical clinical manifestation of injury of the OR.^{1,2} Although the incidence of visual field defect in stroke patients is high, a considerable portion of these patients show recovery of visual field defect: in detail, Gray et al. reported that among 99 patients who developed homonymous hemianopia within 72 hours of stroke onset, complete visual field recovery was observed within 28 days in 14 (17%) of 81 patients with complete homonymous hemianopia and in 13 (72%) of 18 patients with partial homonymous hemianopia.^{2–4} However, the recovery mechanism of visual field defect in stroke patients has not been clearly elucidated, although a few studies have reported on recovery of injured OR.^{5–7} On the other hand, many studies have reported that the corpus

callosum connects both occipital lobes and callosal connections represent a major pathway in induction of plastic rearrangements in the visual cortex.^{8–10} However, the recovery mechanism of visual field defect related to plasticity via the corpus callosum has not been reported.

In this study, we report on a patient with a cerebral infarct who showed recovery of visual field defect via the corpus callosum, using diffusion tensor tractography (DTT) for OR.

METHODS

Case Description

A 57-year-old male patient underwent conservative management for a cerebral infarct in the right

Received 5 November 2014; revised 11 December 2014; accepted 11 December 2014; published online 2 February 2015

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temporal lobe. Brain magnetic resonance images (MRIs) taken at 2 weeks and 11 months after onset showed a cerebral infarct in the subcortical white matter of the right temporal lobe (Figure 1A). He complained of left homonymous hemianopsia since the onset of cerebral infarct. The left homonymous hemianopsia was detected on the 2-week

Humphrey visual field test (Figure 1C). However, the patient showed improvement of hemianopsia with time; consequently, the left lower peripheral visual defect with recovery of the upper and medial portions of the initial left hemianopia was observed on the 11-month Humphrey visual field test.

FIGURE 1 (A) T2-weighted brain MR images taken at 2 weeks and 11 months after onset show a cerebral infarct in the subcortical white matter of the right temporal lobe. (B) Diffusion tensor tractography (DTT) for the optic radiation (OR) in the patient. Both 2-week and 11-month DTTs show discontinuities (sky blue arrows) of the right anterior OR, and the right posterior ORs are connected to the left OR via the transcallosal fibres. On 2-week and 11-month DTTs, the left OR is connected to the transcallosal fibres, and on 11-month DTT, these transcallosal fibres are elongated to the right primary visual cortex via the distal right OR (yellow arrows). (C) The 2-week Humphrey visual field test shows left homonymous hemianopsia, and the left lower peripheral visual defect with recovery of the upper and medial portions of initial left hemianopia is observed on the 11-month Humphrey visual field test. (D) DTI for the OR in the control subjects. The transcallosal fibres of the control subjects were similar with the left OR on 2-week DTT.

Diffusion Tensor Imaging

Diffusion tensor imaging (DTI) data were acquired twice (2 weeks and 11 months after onset) using a sensitivity-encoding head coil on a 1.5-T Philips Gyroscan Intera (Hoffman-LaRoche, Best, The Netherlands) with single-shot echo-planar imaging and navigator echo. For each of the 32 non-collinear diffusion sensitizing gradients, we acquired 67 contiguous slices parallel to the anterior commissure–posterior commissure line. Imaging parameters were as follows: acquisition matrix = 96×96 ; reconstructed matrix = 192×192 ; field of view = $240 \times 240 \text{ mm}^2$; TR = 10,726 ms; TE = 76 ms; parallel imaging reduction factor (SENSE factor) = 2; EPI factor = 49; $b = 1000 \text{ s/mm}^2$; NEX = 1; and a slice thickness of 2.5 mm with no gap. The Oxford Centre for Functional Magnetic Resonance Imaging of the Brain (FMRIB) Software Library (FSL; www.fmrib.ox.ac.uk/fsl) was used for analysis of diffusion-weighted imaging data, and affine multi-scale two-dimensional registration was used to correct for head motion effects and image distortions due to eddy currents. Fibre tracking was performed using a probabilistic tractography method based on a multi-fibre model and applied using tractography routines implemented in FMRIB Diffusion (5000 streamline samples, 0.5 mm step length, curvature threshold = 0.2). Due to cerebral infarct, we were not able to reconstruct the right OR when given the region of interest (ROI) to the right lateral geniculate nucleus (LGN). Instead, two ROIs were placed on the middle and posterior portions of the OR area at the coronal images on the colour map, and the ORs were reconstructed using fibres passing through two ROIs on the colour map.¹¹ Out of 5000 samples generated from each seed voxel, results for each contact were visualised threshold at a minimum of 100 streamlines through each voxel for analysis.

Both 2-week and 11-month diffusion tensor tractography (DTT) for the right OR showed a discontinuation between the right LGN and the right OR; instead, the right posterior OR was connected to the left OR via the transcallosal fibres, which passed through the splenium of the corpus callosum. On 2-week and 11-month DTTs, the left OR showed preserved integrity between the left LGN and the left OR. In addition, on 2-week DTT, the left OR was connected to the transcallosal fibres, and on 11-month DTT, these transcallosal fibres were elongated to the right primary visual cortex via the right posterior OR.

DISCUSSION

In the current study, we attempted to demonstrate the recovery mechanism of visual field defect in a patient with a cerebral infarct. The anterior portion of the

right OR in this patient appeared to be injured completely by the infarct, which involved the area around the right LGN, because the anterior portion of the right OR showed discontinuation around the right LGN on both 2-week and 11-month DTTs. The patient showed left homonymous hemianopia on the 2-week visual field test. However, this hemianopia had shown recovery in the upper and medial portions of hemianopia; consequently, only the left lower peripheral visual field defect was observed on 11-month visual field test. On the other hand, on 11-month DTT, the discontinuation of the right OR had not been restored; thus, it is suggested that the recovery of hemianopia could be attributed to a visual pathway other than the completely injured right OR. Instead of recovery of the injured right OR, 11-month DTT showed a new neural pathway originating from the left OR and terminating in the right primary visual cortex through the transcallosal fibres and the right distal OR. Consequently, the visual field defect in this patient appears to have recovered by the neural pathway originating from the left OR and terminating in the primary visual cortex via the transcallosal fibres and right distal OR.

Since introduction of DTI, several studies have suggested recovery mechanisms of injured OR in patients with brain injury: in detail, recovery of an injured OR and change of an unaffected OR.^{5–7,12,13} Regarding stroke patients, a few studies have reported on recovery of an injured OR.^{5–7} In 2006, Yoshida et al. reported on a patient who developed right homonymous hemianopic paracentral scotoma following acute infarction in the left extrastriate area.⁵ Initial DTI showed that the affected posterior OR was completely discontinued. However, this discontinuation diminished with time and had disappeared by 1 year after onset. In 2010, using functional MRI (fMRI) and DTI, Chen et al. evaluated changes of visual pathway lesions after hyperbaric oxygen treatment for 10 days in 16 patients with visual pathway lesions, including 4 patients with an occipital lobe infarct.⁶ Both numbers of activated voxels on fMRI and FA (FA value indicates the degree of directionality of water diffusion, represents the white matter organization) of the OR showed an increase after hyperbaric oxygen treatment. Recently, using fMRI and DTI, Polonara et al. demonstrated recovery of left hemianopia in a patient with a right calcarine cortex infarct.⁷ Activation of the right visual cortex was observed on acute-phase fMRI; however, activation of the right visual cortex was observed on 1-month fMRI. In addition, the decreased FA value of the right OR on acute-phase DTT showed resolution on 1-month DTT. As a result, to the best of our knowledge, this is the first study that demonstrated the recovery mechanism of the visual field defect via the transcallosal fibres of the splenium of the corpus callosum. However, limitations of DTI should be considered.¹⁴ DTI is a

powerful anatomic imaging tool for demonstration of gross fibre architecture; however, it can produce false-negative results due to crossing fibre or partial volume effect, or false-positive results; in particular, probabilistic fibre tracking can cause false fibre trajectory.¹⁴

In conclusion, using follow-up DTT, we demonstrated the recovery mechanism of visual field defect in a patient with a cerebral infarct. We believe that the results of this study may suggest one of the mechanisms for recovery of a visual field defect following injury of the OR in stroke patients. However, because it is a case report, this study is limited. Conduct of further complementary studies involving larger numbers of patients is warranted.

ACKNOWLEDGEMENTS

This research was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education, Science and Technology (2012 R1A1A4A01001873).

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Note: Figure 1 of this article is available in colour online at <http://informahealthcare.com/oph>.

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