Bilateral injury of the superior longitudinal fasciculus in a patient with Balint syndrome

Figure Brain MRI and results of diffusion tensor tractography

(A) T2-weighted brain MRI at 4 weeks after onset shows leukomalactic lesions in both fronto-parieto-temporo-occipital lobes. (B) Diffusion tensor tractography (DTT) of the patient. On 4-week DTT, both ends of the superior longitudinal fasciculus are discontinued in both hemispheres. (C) DTT of a normal control (50-year-old woman).

A 51-year-old, right-handed woman had a hemorrhage involving the right putamen with intraventricular spread, and underwent extraventricular drainage (right frontal approach) and decompressive craniectomy. She had the triad of Balint syndrome (simultanagnosia, ocular apraxia, and optic ataxia).^{1,2} She also showed quadriparesis and severe cognitive impairment.

Diffusion tensor imaging data were acquired at 4 weeks after onset; tractography showed that both ends of the superior longitudinal fasciculus (SLF) were interrupted in both hemispheres (figure).

We hypothesize that bilateral injury of the SLF in our patient was the main mechanism of Balint syndrome.¹

Sung Ho Jang, MD, Chul Hoon Chang, MD, Young Jin Jung, MD, Jeong Pyo Seo, PhD

From the Department of Physical Medicine and Rehabilitation (S.H.J., J.P.S.) and Neurosurgery (C.H.C., Y.J.J.), College of Medicine, Yeungnam University, Daegu, South Korea.

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Correspondence to Dr. Seo: raphael0905@hanmail.net

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