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IV. 7. Personality Change and Mental Deterioration Resulting Bilateral Thalamic Infarctions Studied by Positron Emission Tomography

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Introduction

The stroke of bilateral medial thalamus and midbrain is called paramedian mesencephalic syndrome. Patients with paramedian mesencephalic syndrome usually show impairment of consciousness, hypersomnia, neuropsychological deficits and ocular motor disorders affecting especially the vertical gaze[1-3]. Thalamic strokes have in recent years increasingly been shown to produce neuropsychological deficits, such as aphasia, apraxia or attentional disorders, previously thought to occur only subsequent to lesions in the cerebral cortex at specific sites[4-8]. In a review of the older literature, Walker concluded that bilateral lesions of the medial thalamic nuclei produce severe mental impairment[9]. In a recent reports[10,11], it was noted that a bilateral thalamic tumor without hydrocephaly can produce personality change and dementia in the relative absence of motor or sensory deficits. We report a patient with paramedian mesencephalic infarction, who showed dramatic personality change and mental deterioration.

Case report

A 27-year-old right-handed woman suddenly lost her consciousness during her work in a office on August 3, 1992. She has educational background for 12 years and had been healthy. One hour after the onset, she was admitted to the Department of Neurosurgery of Sendai National Hospital. When she was arrived at the hospital, she was semicoma with left-hemiparesis, bilateral mydriasis, sluggish pupillary reflex on both side, and Oculo-cephalic reflex and Cilio-spinal reflex were negative. On the third day after the admission, cerebral infarctions in the right midbrain and bilateral medial thalamus were revealed by the brain computed tomography (CT) scan. Axial, coronal, and sagittal cerebral magnetic resonance (MR) showed in T1-and T2-weighted images lesions in the bilateral medial part of the thalamus and right midbrain (Fig. 1). Angiography of the carotid and vertebral arteries showed a dissecting aneurysm in the 1.5 cm peripheral end of the basilar artery.

Repeat angiogram two month later showed disappearing of the false lumen. Her consciousness had been improved with conservative therapy and became clear about three months later. However, she showed a childish behavior and mental deterioration, she transferred to the Department of Neurology on April 15, 1993 in order to have neuropsychological examination. On the neurological examination in April, she was alert but her orientation was disturbed. She was noted ataxic speech, disturbance of visual acuity (the right was 0.5 and the left was 0.15), anisocoria (right>left), sluggish pupillary reflex on both side, left hyperexophonia, vertical gaze disorder, and severe limb ataxia. There were no obvious paralysis, pathological reflexes or sensory disturbances, and the deep tendon reflexes were normal. A neuropsychological examination revealed memory disturbance and intellectual impairment without aphasia, agnosia, and apraxia. Her ability of calculation was good. Neither unilateral spatial neglect nor extinction were observed. Digit span was 6. Mini-mental state test was 19 scores, and, in Wechsler adult intelligence scale (WAIS), full IQ was 51. In Wechsler memory scale, Memory Quotient (MQ) was 63. The result of Benton's visual retention test showed only one correction and 18 errors. She got 98 points on Bender-Gestalt test, which was in a 4 to 6-years-old level. She was also examined by a psychiatrist and she was revealed abnormality such as making stories with her wishes, a tendency which is from a irrelevant answer to a correct answer, or an indirect tease to an interviewer. Moreover, an abnormality was also found on her general attitude such as childish performance.

Positron emission tomography (PET)

PET study was performed on a scanner, PT-931 (CTI Inc, USA), at the Cyclotron Radioisotope Center, Tohoku University, Sendai, Japan. This study was approved by the Research Ethics Committee of the Tohoku University, School of Medicine. For all studies, the patient was positioned in the scanner with the orbitomeatal (OM) line parallel to the detector rings. Using the bed and gantry coordinates, we tried to position the head in exactly the same position for each study. Before scanning, a short 21-gauge cannula was inserted to a brachial artery for arterial blood sampling. All the procedures were performed in a semidarkened room and she was put to sleep by a sedative. A 15-min transmission scan was collected using a retractable germanium 68-gallium 68 ring source. To determine cerebral blood flow (CBF) and cerebral metabolic ratio of oxygen ($CMRO_2$), steady-state emission data were collected for two scans each of 10 min duration, during inhalation of $C^{15}O_2$ and $^5O_2$ (370 MBq/min), respectively, with sufficient washout time following each study. Each scan was reconstructed into 14 planes with an 8mm axial and transaxial resolution. Regional CBF and $CMRO_2$ were calculated according to Frakowiak et al. [12] from the emission scans using arterial oxygen content and whole blood and plasma radioactivity counts measured in triplicate during each scan. To determine cerebral metabolic ratio of glucose (CMRGlc), a series of three emission scans each of 10 min

duration was commenced 30 min after an intravenous bolus injection of 185 MBq (% ml) 2[^{18}F]-fluoro-2-deoxy-D-glucose (^{18}FDG). Twenty blood samples were collected to determine the plasma radioactivities and glucose concentrations, taken every 10 min during the study[13]. Regional CMRGlc was calculated from the emission scans using plasma radioactivity counts and the operational equation derived by Phelps et al. and Huang et al from Sokoloff et al [14-16]. Representative appearances of brain images obtained by MR and those of CMRGlc, CBF, and $CMRO_2$ are shown in Fig. 2. Regional CMRGlc, CBF and $CMRO_2$ were decreased in the bilateral thalamus and right midbrain. On the other hand, regional CMRGlc, CBF and $CMRO_2$ were preserved in the cerebral cortex.

Discussion

Our patient suffered from cerebral infarctions in bilateral medial thalamus and midbrain by ischemia of perforating arteries caused by a dissecting aneurysm in the basilar artery. The infarction in bilateral medial thalamus and mid-brain is known as paramedian mesencephalic syndrome, which shows characteristic CT findings, transient coma of sudden onset, sleeping tendency, vertical gaze paralysis, conversion paralysis and dementia. Our patient also revealed these symptoms. There are about forty reports of this syndrome and it is comparatively rare.

When the patient was transferred to our department eight months after the onset, her main symptom was a dramatic personality change. According to her mother, the patient's character was totally different as compared with before. She showed childish tease for questions, for example, answering a popular singer's name when asked her name, or clapping her hands every time when she got a right answer, moreover, when she was told to go to rehabilitation, she behaved like a child saying "You are mean!". Generally, she answered irrelevantly for questions teasingly to an interviewer, then her answer were tend to be correct. From these attitudes, it might be possible to get lower points in WAIS than her real intelligence. However, when she stayed overnight at her house, she took care of her little sons' meals or giving warning to them about their mischiefs. In the hospital, there were not troubled behavior at all on making mistake to go to her room, incontinence, wandering or excitement. Eating, urination, defecation and dressing were all independent. The patient had been thought that she had dementia with acting, therefore, the psychiatrist diagnosed her as pseudodementia due to hysteria. These symptoms, however, still has continued for one year and seven months after her onset, it is not seemed to be only a conversion reaction. On her PET study, CBF, $CMRO_2$, and CMRGlc were decreased only in the thalamus and midbrain and those in the cortex were not decreased. Consequently, the patient's personality change and the symptoms of dementia were caused by the bilateral thalamic lesions. Partlow et al.[11] reported eight cases of bilateral thalamic glioma and noticed a personality change and a dementia in all cases. In all of their cases, the lesions included medial thalamus. Lesions of the medial thalamic nuclei destroy important relay

stations between cerebral cortices, and should, therefore, theoretically have the potential to produce similar symptoms as those observed with hemispheric lesions. In fact, thalamic lesions have produced a number of symptoms commonly associated with cortical lesions, such as aphasia, apraxia and attentional disorders. In our patient, the lesions of bilateral thalamic structures may have led to acute personality change and mental deterioration in a manner similar to the diaschisis phenomena of another cognitive disturbances.

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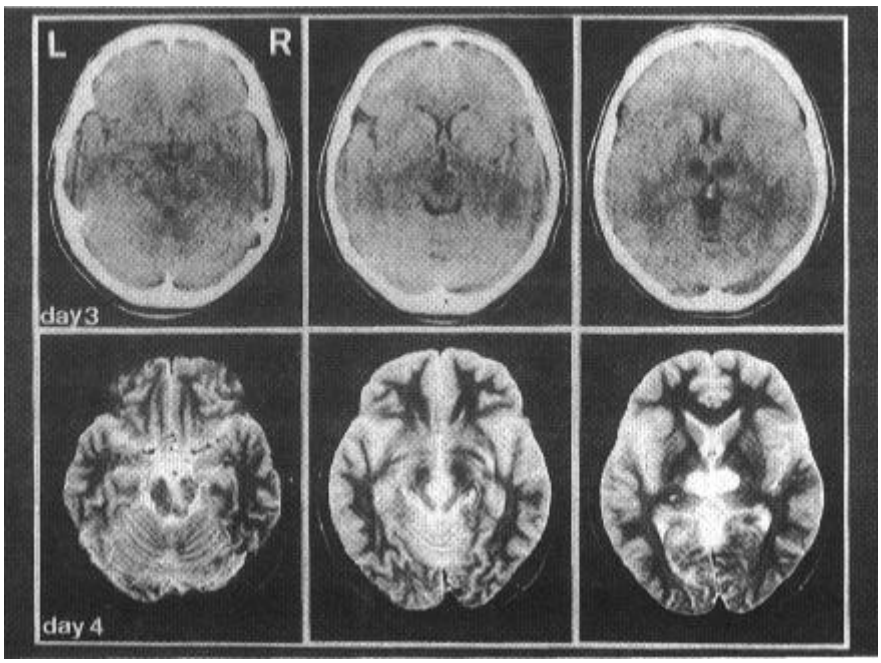

Fig. 1. Axial cerebral CT 3 days after and MR showed in T2-weighted images 4 days after the onset.

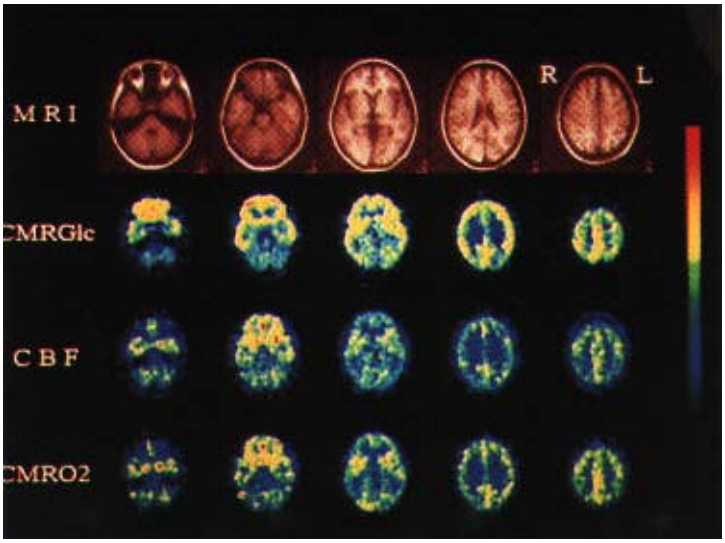

Fig. 2. Representative appearances of brain imagings obtained by MR and those of CMRGlc (color scale ranged from 0 to 10 mg/100gr/min), CBF (color scale ranged from 0 to 50 ml/100gr/min), and $CMRO_2$(color scale ranged from 0 to 5 mg/100gr/min) in the same brain slices.