Unusual presentation of more common disease/injury

Memory deficits due to brain injury: unique PET findings and dream alterations

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The authors herein report the case of a young male with memory deficits due to a traumatic head injury, who presented with sleep-related symptoms such as hypersomnia and dream alterations. Although MRI and polysomnography showed no abnormalities, ¹⁸F-fluorodeoxyglucose positron emission tomography (FDG-PET) and ¹¹C flumazenil (FMZ)-PET revealed findings consistent with cerebral damage to the affected temporal region. The memory deficit of the patient gradually improved in parallel with the relief of the sleep-related symptoms. FDG-PET showed considerable improvement in glucose metabolism when he had recovered, however, evidence of neural loss remained in the FMZ-PET findings.

BACKGROUND

Traumatic brain injury may cause various types of memory disturbance, such as amnesia and difficulty of recall. In addition to mnemonic differences, patients also may manifest psychiatric and neurological symptoms. Although neuroimaging techniques have progressed, the neural mechanism of brain injury and the process of recovery still remain to be fully elucidated.

CASE PRESENTATION

In July 2009, an 18-year-old male with memory loss was referred to our hospital. The patient had experienced trauma to the right of his head while playing handball in April 2008. Although his response and reflexes were normal after the accident, the patient repeatedly talked about the same things. The patient was unable to recall incidents that had happened more than three or four days earlier.

Since the beginning of 2009, the patient manifested hypersomnia that lasted half of the day and claimed that the pattern of his dreams had changed. After the accident, the patient only dreamed about what he had actually experienced during the daytime, such as playing baseball or having dinner with his family. The patient's family would confirm that the dreams were just replays of actual past experiences. The patient was diagnosed with idiopathic hypersomnia or dissociative disorder at another hospital.

Upon admission, cognitive tests were performed. The mini mental state examination and Wechsler memory scale-revised demonstrated that the patient had no short-term memory deficits. No physical abnormalities were detected through blood tests, brain MRI or electroencephalography. However, the patient had difficulties visually recalling incidents that had happened more than 3 days earlier.

Over the course of hospitalisation, the patient's hypersomnia remitted without medication. The patient was told to keep a diary everyday to record his dreams. The recorded

contents of the dream were always what the patient had actually seen or experienced previously. Although polysomnography revealed that there was a slight increase in his sleep latency, most of the sleep architecture remained intact, including rapid eye movement (REM) sleep.

INVESTIGATIONS

¹⁸F-Fluorodeoxyglucose (FDG) positron emission tomography (PET) showed a significant decrease in glucose metabolism within the right supramarginal and inferior temporal gyrus. ¹¹C-flumazenil (FMZ) PET was used to test the neuronal viability of the brain. The study showed the FMZ uptake to decrease in the right middle temporal gyrus; the reduction in binding likely represented neuronal damage, thus indicating a loss of neuronal integrity (figure 1A,B).

DIFFERENTIAL DIAGNOSIS

- ▶ Dissociative disorder
- ▶ Idiopathic hypersomnia.

TREATMENT

The patient was started on memory rehabilitation, focusing on memory restoration. For example, writing down the events to memorise in a notebook. A couple of months later, his memory performance gradually improved, accompanied by changes in the contents of the dreams. No medication was prescribed for his treatment.

OUTCOME AND FOLLOW-UP

The patient started to dream again about foreign incidents, such as Hollywood movie stars visiting his home. His memory recall also gradually recovered during the hospitalisation, and the duration of memory recall was prolonged. When the patient was discharged in February 2010, the patient showed a remarkable memory

Figure 1 Initial positron emission tomography (PET). Z-score brain surface maps from ^{18}F -fluorodeoxyglucose (FDG)-PET showed decreased values in the supramarginal and inferior temporal gyrus (A). ^{11}C -flumazenil (FMZ)-PET showed decreased values in the right middle temporal gyrus (B). The spatial preprocessing and the statistical analysis of the images were done using the SPM2 software program (available at www.fil.ion.ucl.ac.uk/spm). Follow-up PET. An FDG-PET examination 6 months later (C) showed improvement in the inferior temporal gyrus, while FMZ-PET 14 months later (D) showed no improvement in the supramarginal gyrus.

improvement, no disturbances in memory recall and had no difficulties in managing daily life. FDG-PET showed a substantial improvement in glucose metabolism within the inferior temporal gyrus, while FMZ-PET demonstrated no significant regional improvement in the FMZ uptake (figure 1C,D).

DISCUSSION

It is well established that sleep facilitates the offline reactivation of formed memories, and that dream experiences reflect this memory processing.¹ Torda reported that subjects with hippocampal lesions had dreams with a stereotyped repetition of past experiences.² Although our case did not have hippocampal lesions, the affected cerebral regions including the inferior temporal lesion that the initial PET findings demonstrated may alter the dream contents, as well as memory loss.

We were unable to find any abnormality of REM sleep that is thought to generate dreams from neural signals in the brainstem,³ that is, different brain regions from that of our case. Previous sleep studies of head trauma patients showed a similar result.⁴ It was previously reported that the cessation of dreaming generally follows damage to the bilateral temporo-parieto-occipital junctions.⁵ Along with the PET measurements, the unilateral lesion in this case coincided with the supramarginal gyrus, which may thus have been responsible for the temporary alterations in his dream contents. As the function of the inferior temporal gyrus improved to compensate for the neural damage to the supramarginal gyrus, the patient's mnemonic function was also improved, accompanied by normalisation of his dream contents. Taken together, the neuronal mechanism underlying the improvement in memory retrieval was

likely associated with the alteration of brain plasticity, and with the physiology of sleep, incorporating sleep-related memory organisation.

Learning points

- ▶ Memory deficit due to traumatic brain injury is a diagnosis of exclusion.
- ▶ If you see a patient with amnesia or sleep-related symptoms, ask about the patient's history of brain trauma.
- ▶ PET is often necessary for a definitive diagnosis.
- ▶ FMZ-PET has the potential to detect neuronal damage.
- ▶ Sleep, including dreams, has an influence on memory process, and alters the underlying brain plasticity.

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