In June 2002, a right-handed woman was admitted to the hospital at the age of 91 and showed intense aggressiveness. She was diagnosed as having Alzheimer’s senile dementia at the age of 88 and was associated with delusion and hostility towards the family. She often appeared angry and displayed resistance and violence towards caregivers. At the age of 95, a slightly unstable posture was observed when walking or sitting. A brain MRI was performed and diffusion-weighted imaging showed hyperintensity in a small region within the right basal ganglia, adjacent to the right lateral ventricle (fig. 1a). We diagnosed acute brain infarction and administered treatment for 2 weeks. A later MRI ascertained that the infarction caused a restricted lesion in the right anterior thalamic nucleus (ATH) (fig. 1b, c). After the infarction, the patient’s emotional and behavioral aggressiveness disappeared; she talked gently with the care staff and received assistance without refusal or aggression. In an assessment based on the Behavioral Pathology in Alzheimer’s Disease Rating Scale [2], the scores of threat, violence, anxiety and fear decreased by at least 2 points compared to those before the infarction. The Mini-Mental State Examination scores were 5/30 in 2005 before the infarction, 2/30 in July 2006 after the infarction and 17/30 in April 2007. She was discharged in May 2007 and severe aggressiveness has not recurred.

Introduction

Emotional disturbances including aggressiveness, behavioral abnormalities and violence cause difficulty in the treatment and care of patients with Alzheimer’s disease. Although neuroleptics, including atypical antipsychotics, often alleviate these problems [1], the mechanisms underlying the aggressive behavior are still unknown in this disease.

Fig. 1. a Brain MRI diffusion-weighted imaging taken just after the infarction showed a hyperintense lesion within the right basal ganglia. b, c Fluid-attenuated inversion recovery sequences 8 months after the infarction revealed evidence of infarction in the right ATH (arrows).
Discussion

The ATH connects the temporal lobe to the prefrontal cortex and is known as a component of Papez’s circuit [3]. Papez’s circuit includes the mamillothalamic tract, which connects the thalamus to the mammillary body, and is thought to be involved in emotion and memory. Damage in the ATH is associated with memory and motor performance deficits: mamillothalamic tract damage is related to long- and short-term memory deficits [4]; the ATH is the primary region involved in amnesic symptoms in alcoholic Korsakoff’s syndrome [5]. In addition, perseverative behavior [6] or dystonic hand tremors [7] were observed after ATH infarctions. Hence, in the present case, the improvement in emotion and memory after an ATH infarction was a paradoxical outcome.

We found severe atrophy of the temporal lobe including the hippocampus and amygdala in the early stages of dementia. In contrast, the frontal cortex function may have been preserved because the frontal lobe atrophy was not severe. Therefore, we speculate that the temporal lobe degeneration might have disturbed the frontal cortical function and the ATH infarction may have reduced this disturbance, thus normalizing the frontal cortical function rather than causing an impairment. Indeed, the ATH is a recognized therapeutic target in epilepsy treatments [8].

The case presented here suggests that the ATH is a critical intermediary point between the temporal and frontal lobes. Thus, the ATH might be an effective target for the treatment of emotional and memory disturbances in dementia that involves severe temporal lobe pathology.

References


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Intravenous Thrombolysis Cancelled in Acute Right Hemiparesis

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Discussion

Evaluating the indication of i.v. thrombolysis in acute stroke patients requires a careful work-up within a short time frame. Ruling out cerebral hemorrhage by CT is of highest priority. However, there may be more pitfalls in emergency stroke cases, which is illustrated in a patient who presented with acute neurological symptoms and back pain.

Case Description

A 62-year-old retired chemical laboratory worker was referred to the emergency room with numbness and weakness of his right arm and leg with suspected acute stroke. He was seen by a neurologist 1 h and 45 min after symptom onset. Sudden-onset hemiparesis had started immediately after he had used the bathroom, together with severe pain between his shoulder blades radiating to the right arm. On neurological examination the patient was alert and fully orientated, there was no aphasia or apraxia. He showed equivocal right facial weakness (facial asymmetry) and a right MRC grade 3–4 flaccid hemiparesis with a plantar extensor response and hypesthesia on the right side of the body. Reflexes were ++++ and symmetrical, the NIHSS score on admission was counted as 6 points.

An ECG and routine blood tests were within normal limits. The blood pressure was 137/97 mm Hg. I.v. thrombolysis was considered, but due to the initial pain syndrome there was a suspicion of an aortic and carotid artery dissection (and subsequent middle cerebral artery infarction). A cranial CT was negative for signs of hemorrhage or ischemic infarction, or arterial dissection. A CT scan of the aorta showed no arterial dissection. Subsequently, his wife arrived and reported that the facial asymmetry (mimicking right facial weakness) had been present before. A further neurological examination revealed a Brown-Séquard syndrome with reduction of pain and temperature sensation on the left below C4 level in addition to the hemiparesis and hypesthesia of the right arm and leg. The thoracic CT scan was reviewed and the suspicion of focal hemorrhage was confirmed by MRI (fig. 1, 2) demonstrating a cervical epidural hematoma causing compression of the cord affecting the nerve roots at levels C4 to C7 on the right. The patient underwent emergency right hemilaminectomy at levels C5 and C6. He subsequently recovered without sequelae.

Discussion

Spontaneous spinal epidural hematoma (SSEH) is rare, but particularly since the availability of i.v. thrombolysis physicians

1 Both authors contributed equally to this work.