Acute Amnesia with Confabulation in Left Caudate Head Hemorrhage: A Case Report

Ching-Feng Huang, Jing-Jane Tsai, Nan-Tsing Chiu, and Ming-Chyi Pai

Abstract- A 61-year-old man developed transient anterograde amnesia after waking from a nap. Vomiting, slow response and queer behaviors followed. A cerebral MRI showed a left caudate head hemorrhage and a SPECT showed hypoperfusion at left frontal, temporal, parietal, and basal ganglion regions. The verbal memory impairment persisted at one-month follow-up, while the perfusion deficit had improved. This case demonstrates that acute amnesia and confabulation can be the clinical presentation after left caudate head hemorrhage.

Key Words: Transient amnesia, Caudate head hemorrhage, Fronto-subcortical circuit

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INTRODUCTION

Caudate head hemorrhage is uncommon, accounting for about 2% of all intracranial hemorrhages[1]. Clinical manifestations of caudate head hemorrhage are either the features of subarachniod hemorrhage, including headache, nuchal rigidity and vomiting, or the features of cerebral dysfunction, such as confusion-disorientation, dysarthria, abulia, nonfluent aphasia, and visuospatial neglect[1-2]. Its presentation as acute amnesia and confabulation is rare in the literature. We will report a patient with such presentation.

CASE REPORT

Patient J.P. is a 61-year-old right-handed man with 6 years of education. On 22 Oct. 2001, he was found to repeat query at 3:30 PM after waking up from a nap. Several hours earlier on the same day he had enjoyed the family reunion. However, in spite of having been told, J.P. asked three times whether his son had gone or not. Afterwards, J.P. repeatedly asked where was the book on herbal wine. According to his wife, there was no such a book. He was left alone. One hour later, J.P. was found mopping the floor. When being asked, he answered that he was cleaning the table as usual. After vomiting once, his wife urged him to take a shower. Although he replied that he had done that the previous night, J.P. went to bathroom. A few minutes later, the bathroom remained silent. When his wife broke in, she found J.P. standing by the bathtub naked, expressionless and mute. He had passed stool but did not flush the toilet. He also did not

From the Department of Neurology, and Department of Nuclear Medicine, National Cheng Kung University Hospital, Tainan, Taiwan.
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Reprint requests and correspondence to: Ming-Chyi Pai, MD, PhD. Division of Behavioral Neurology, Department of Neurology, National Cheng Kung University Hospital, 138 Sheng Li Road, Tainan 704, Taiwan.
E-mail: pair@mail.ncku.edu.tw

take a shower. He responded that he forgot how to do it. Afterward, J.P. wandered naked in the house. He was brought to our ER at 6 PM. On arrival, he was fully oriented, able to identify his relatives, and had relevant and coherent speech. The blood pressure and blood tests were normal. He was discharged at midnight; and no special treatment was given. The next day J.P. visited our neurology clinic and was admitted under the diagnosis of transient amnesia.

J.P. received an operation for sigmoid tubular adenoma 3 years ago. He had cataracts, glaucoma, and chronic hepatitis B. His visual acuity of left eye was diminished several years ago for unknown reason. Otherwise, he had no history of stroke, transient ischemic attack, seizure, head injury, central nervous system infection, or psychiatric disorders. He had no experience of recent overseas trips, or use of tobacco, alcohol and illicit drugs.

On admission, the blood pressure was 120/76 mmHg and the vital signs were normal. He was fully oriented and attentive. The language function was normal, including spontaneous speech, naming, repetition, comprehension, reading and writing. He could recall 3 objects after a 5-minute delay. However, he could not remember what had happened to him the previous day, with a memory gap from 3:30 P.M. of 22 Oct. 2001 to 9:00 A.M. of 23 Oct. 2001. He would make up a story when being asked what he had done the previous night. Neurological examination was normal except for a profound visual acuity loss and a Marcus-Gunn pupil on the left side. He was discharged on the 7th post-attack day (PAD), without recurrence of an attack during three months of follow-up.

The score of the Mini-mental State Examination (MMSE)[3] on the second PAD was 23/30 and the Chinese version of Cognitive Ability Screening Instrument (C-CASI 2.0)[4] 82.5/100 with a deficit in recent memory (7.5, full score:12). A cerebral MRI on the third PAD showed a hyperintensity at left caudate head on T1-weighted images (Fig. 1). The scalp EEG was normal. A single photon emission computed tomography (SPECT) scan on the fourth PAD showed perfusion deficits at left frontal, temporal, parietal mistyping, and also at right cerebellum (Fig. 2A).

One month later, the score of a follow-up MMSE

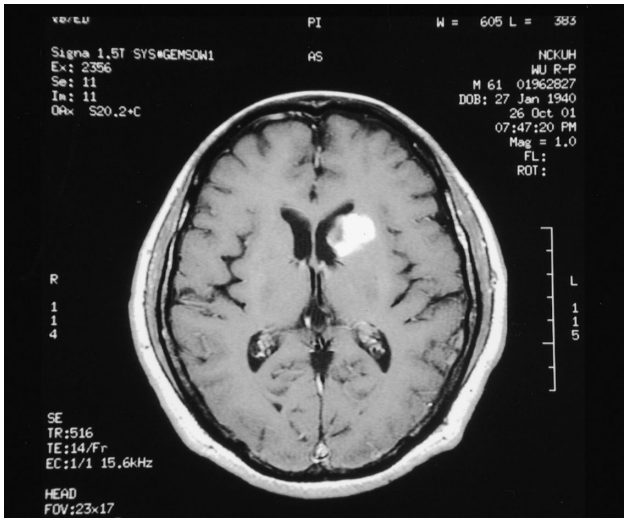

Figure 1. An axial cerebral MRI (TR=516 ms; TE=14 ms/ Fr) on the third post-attack day, showing a hyperintensity at left ventromedial caudate head.

was 29/30 (normal) and C-CASI-2.0 88.5/100 (normal). A follow-up SPECT scan showed partial resolution over the previously hypoperfused areas (Figure 2B). His IQ is 96 (VIQ=99, PIQ=95) [Wechsler Adult Intelligence Scale-Revised (WAIS-R)][5]. On the Wechsler Memory Scale-Revised (WMS-R)[6], he scored 99, 71, 93, 71, and 78, respectively, on the attention-concentration, verbal memory, visual memory, general memory, and delayed memory subtests. Each of these subtests yields mean scores of 100 in the normal population with a S.D. of 15. A sphenoidal EEG one month later was normal.

DISCUSSION

Acute amnesia as a manifestation of caudate head hemorrhage is rare[2]. His manifestations of repeated queries, bewilderment, and a preserved ability to contact with the surroundings may raise the diagnosis of transient global amnesia (TGA). However, symptoms of vomiting, queer behavior, a delayed response to questions and confabulation made the diagnosis of TGA unlikely. During the attack of TGA, patients usually have a normal behavior, high-level intellectual activity and language function except for incessant, repetitive questioning about his immediate circumstances. Several hours after the attack, no abnormality of mental function

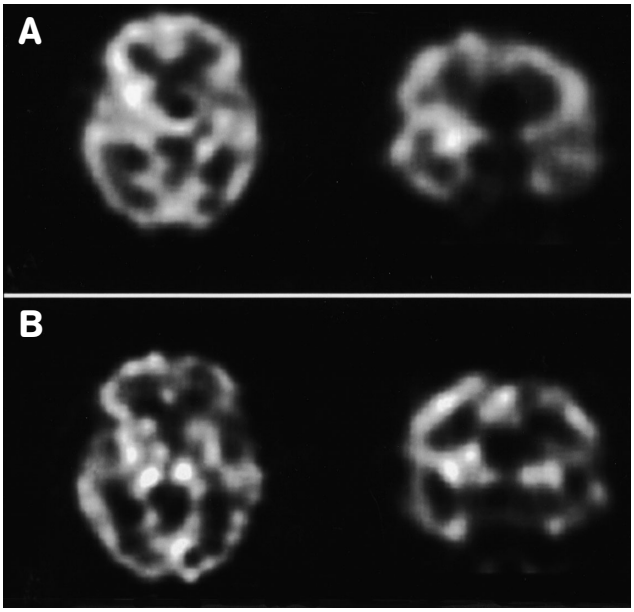

Figure 2. (A) cerebral SPECT, performed on the fourth post-attack day, showing decreased radioactivity at left frontal, temporal, parietal, and basal ganglion; (B) A follow-up SPECT at one month showing partial resolution at the previously hypoperfused areas.

is apparent, except for a permanent memory gap for the period[7]. The persistent verbal amnesia of J.P., as evidenced from the follow-up WMS-R assessment, implies an organic amnesic syndrome.

At the first glance, the clinical feature of J.P. could easily be categorized as confusion. However, acute confusional state was not synonymous with amnesia[8]. The cardinal features of confusional state include disturbance of vigilance, heightened distractibility, impaired working memory, inability to maintain a coherent stream of thought, inability to carry out sequence of goal-directed movements, rapid fluctuation of mental state, and typical nocturnal exacerbation. With retained intellectual function, J.P.'s decreased spontaneous activity as well as speech output and the prolonged latency in responding to questions, which are characteristic of abulia, could not be labeled as confusion.

Caudate nucleus receives projections mainly from dorsolateral prefrontal circuit and lateral orbitofrontal circuit[9], and is the principal area of basal ganglia-thalamocortical and cortico-pallido-nigra-thalamo-cortical loops[2]. Patients with caudate lesion will have symptoms of frontal lobe dysfunction; a dorsolateral caudate lesion may cause decreased spontaneous verbal and motor activities, while ventromedial lesion may cause disinhibited, inappropriate and impulsive behaviors[9]. These circuits may explain some aberrant behaviors of J.P. Moreover, damage to prefrontal cortex may undermine the effectiveness of encoding and retrieval of memory. This causes an impoverishment of associative linkages that are necessary for reconstructing context and temporal order, decreases the speed with which internal data stores are searched, and thus increases the tendency to confabulate[10]. Studies had shown that a measure of the caudate head atrophy, noted frequently in the patients of Huntington's disease, correlated with the performance on Wechsler Memory Scale[8]. From previous reports, patients with left caudate lesion may have verbal amnesia, patients with right caudate lesion visual amnesia[2,9], and patients with bilateral lesions global dementia[11].

Whether the clinical manifestation of J.P. was derived anatomically from the left caudate head hemorrhage alone or hemodynamically from hypoperfusion over left frontal, temporal, parietal, and basal ganglion is unknown. From J.P.'s transient anterograde amnesia with confabulation, we may infer that left caudate nucleus is involved in the functional network of a normal verbal memory.

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