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Persistent anterograde amnesia due to the artery of Percheron occlusion: a case report

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ABSTRACT

Bilateral thalamic infarction involving the artery of Percheron (AOP) can cause diagnostic difficulties due to the varying clinical presentations. AOP infarcts presented with isolated memory impairment are not common and the factors affecting the persistence of memory disorders are still unknown. A 41-year-old male patient was hospitalized with acute unconsciousness. MRI disclosed bilateral paramedian thalamic infarction. The patient had isolated memory deficit and his anterograde amnesia continued without any change in the past decade. More cases might answer the questions concerning the intra- and extra-thalamic structures responsible for the amnesic syndrome and the factors affecting the persistence of the symptoms.

ARTICLE HISTORY

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KEYWORDS

Percheron; anterograde amnesia; thalamic infarction; amnesia; persistent amnesia

Introduction

Blood supply to the thalamus has many anatomic variations. The artery of Percheron (AOP) is a rare vascular variant supplying both paramedian thalami. Occlusion of this uncommon vessel results in symmetrical bilateral thalamic infarcts with or without mesencephalic infarctions. The complex anatomy, vascular supply and functions of thalamus are responsible for the variable clinical presentations leading the physician to think of an etiology other than stroke. AOP infarcts are mainly characterized by a triad of altered mental status, gaze palsy, and memory impairment (Arauz et al., 2014). The prognosis of AOP infarction is usually good with low mortality and often rapid reversal of the coma, but the outcome of memory deficits after the acute phase of the disease is unknown. All cases with AOP infarction might not pursue a similar long-term course, raising the burning question why some cases have only temporary memory disorder after the incident while others are obliged to live with lifelong memory deficit. We report a case with long-term anterograde amnesia due to AOP occlusion by questioning the factors that contribute to the persistence of the symptoms.

Case report

A 41-year-old male patient with no history of illness, tobacco, or alcohol consumption was hospitalized with acute unconsciousness. There was no focal neurological deficit on admission. His Glasgow Coma Scale was 7/15 (eye 2, verbal 1, motor 4). He was treated at the intensive care unit (ICU) without requiring mechanical ventilation. Although he demonstrated fluctuations in his level of consciousness ranging from coma to somnolence, his pupils were equal and reactive to light bilaterally. MRI disclosed

bilateral paramedian thalamic infarction; MR angiography, blood biochemistry, serological tests, and markers of vasculitis were completely normal. Transthoracic echocardiography revealed ejection fraction to be 35% and diffuse hypokinesia. Etiology was evaluated as cardioembolic and anticoagulant therapy was started. The patient was examined after regaining consciousness, where orientation to person, place and time, reading, writing, copying, and verbal expression were all found insufficient. Within a month, all language functions completely returned to normal, but anterograde amnesia continued without any change. Mini Mental State Examination (MMSE) score was 9/30. He is 50 years old now and has been living for the past decade without being able to turn his life into memories. He has no retrograde amnesia, no memory deficit regarding his past life but he has not been able to record any new memories since the cerebrovascular accident 10 years ago. He is a retired worker, he could chat with his family and old friends, but never remembering what he talked about more than a few minutes. He cannot recognize his now grown-up children, but he knows their names in order. The patient does not show any behavioral disorders but is usually in a depressive mood, probably since he is aware of his incompetencies. We administered MMSE to see if the score stayed the same and it was 10/30 after 10 years. Montreal Cognitive Assessment (MOCA) score was 11/30. The worst part of assessment was the delayed record. The recall of word list after 5 min was impossible. Naming and attention were normal, although visuospatial skills, orientation and abstraction were insufficient in the test. Although he has no stroke recurrence under the adjusted-dose warfarin treatment and no other neurological disabilities, daily life is very distressing for the patient as well as his family.

Discussion

A number of studies have been carried out on thalamic amnesia, with the aim of clarifying the role of thalamic nuclei and bundles in memory processes (Danet et al., 2017). Thalamic abnormalities occur in some of the earliest stages of Alzheimer's disease. Appreciating the importance of the thalamic nuclei for memory may provide the understanding of Alzheimer's disease (Aggleton, Pralus, Nelson, & Hornberger, 2016). To date, cognitive outcome has been assessed in single cases and small case series, some of which have reported residual memory deficits, and others apparent recovery following limbic encephalitis (Butler et al., 2014), but there is no sufficient data on long-term prognosis of memory deficit following thalamic stroke. The prognosis of AOP infarctions is usually good. Life threatening complications have been reported very rarely (Zappella et al., 2014). Recent reviews and case reports on this topic cover the early phase of the disease thoroughly (Lamot, Ribaric, & Popovic, 2015; Zappella et al., 2014) and just temporary memory deficit has been reported in three-month follow-up (Bailey & Khadjooi, 2016).

Only Arauz et al. reported a long-term follow-up (a mean of 55 months) for the case series of 15 patients including 2 patients with anterograde amnesia, but if there was any change in the level of amnesia, it was not mentioned. It was pointed out that the presence of midbrain extension was meaningful in the persistence of clinical findings. Their observations suggested that the non-extension to the midbrain explains the favorable outcome in 67% of the cases mostly presenting with the behavioral amnesic impairment and mental status disturbance (Arauz et al., 2014). Cerebral MRI of our patient on the 10th year demonstrates bilateral thalamic paramedian infarction (Figure 1(a,b)) due to the AOP occlusion without the extension to the brainstem (Figure 1(c, d)) and there are no lesions at the other regions of the brain.

Similar clinical features and outcomes were recorded in patients with AOP infarction identified in the literature, of whom 13 required ICU management. In stroke patients who required mechanical ventilation, 1-year mortality rates range from 40–70% and cognitive impairments persist in 63–75% of survivors (Zappella et al., 2014). Our patient was treated at the

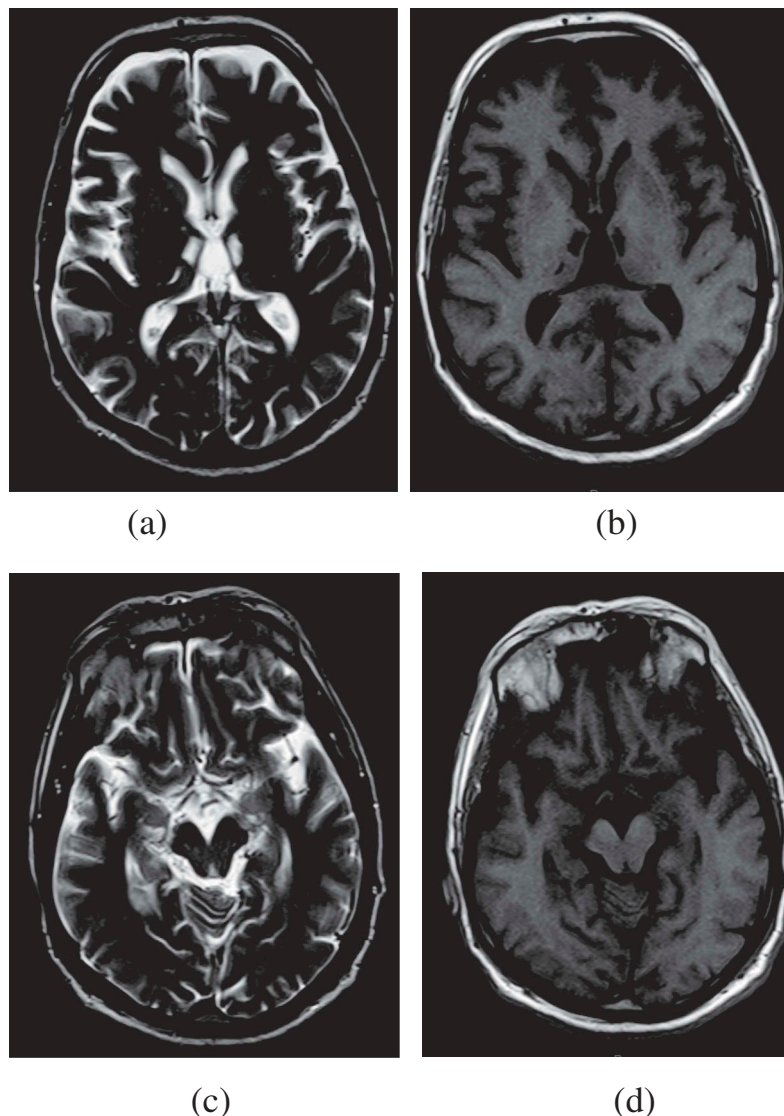


Figure 1. MRI shows bilateral paramedian thalamic infarction (a, b) without midbrain involvement (c, d).

ICU but the need of mechanical ventilation did not arise. Embolic heart disease is one of the most common types of etiology which occludes the AOP leads to bilateral paramedian thalamic infarction (Jumeau et al., 2016). Our patient has a cardioembolic etiology with diffuse hypokinesia, but it is reported that unfavorable outcome is not related to the etiology of stroke (Arauz et al., 2014).

Intra- and extra-thalamic structures primarily responsible for the amnesic syndrome are still unknown. Vascular thalamic amnesics often present dysexecutive and behavioral deficits similar to those observed in patients with frontal damage, but our case showed no behavioral dysfunctions. Data reported in a few single cases provide support for the hypothesis that thalamic regions connected to distinct areas of the mesiotemporal lobe play differential roles in recollection and familiarity processes (Carlesimo, Lombardi, & Caltagirone, 2011). The connections link hippocampal and parahippocampal regions with the mammillary bodies, the anterior thalamic nuclei, and the cingulate gyrus, all structures now strongly implicated in memory functions (Bubb, Kinnavane, & Aggleton, 2017). Unfortunately, findings from the literature review did not allow identifying consistent localization and criteria for the persistence of the memory disorder. The fact that our case did not have any recurrent infarcts, its “single attack-single location” characteristic and the unchanging severity of amnesia for a decade suggests a clarifying effect regarding the location of amnesia.

In conclusion, our patient is the first case with a 10-year history of anterograde amnesia due to AOP occlusion; more cases with long-term follow-up may provide better understanding of the clinical persistence of the symptoms and the thalamic structures primarily responsible for the amnesic syndrome. This case highlights the difficulty in the prediction of the prognosis in the AOP infarction presenting with isolated memory disorder.

Disclosure statement

No potential conflict of interest was reported by the authors.

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