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Case Report

Acute amnesia caused by left fornix infarction: A case report of an unusual entity ☆

Sunny Qi-Huang, MD[1,], Joshua Barnaby, MD[1], Samaan Mahmoudzadeh, MD[2], Steven Lev, MD[1], Saurabh Patel, MD[1]*

[1] *Department of Radiology, Nassau University Medical Center, East Meadow, NY, USA*
[2] *American University of the Caribbean, School of Medicine, Cupecoy, St. Maarten*

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ABSTRACT

Cerebral infarction is an uncommon and unusual cause of acute amnesia. The fornix is a white matter tract bundle that plays an important function in memory. We present the case of a 60-year-old male presenting with altered mental status and acute onset amnesia with CT and MR imaging demonstrating an acute left fornix infarct. This case serves to further illuminate the findings associated with this uncommon clinical event. In addition, it highlights the importance for physicians across multiple subspecialities to maintain an index of suspicion for fornix infarct in the evaluation of acute onset amnesia.

Introduction

Transient global amnesia (TGA) is an uncommon clinical event and a relatively rare complication of cerebral infarction. It is defined by the transient loss of anterograde and recent retrograde memory without residual cognitive impairment, which can last up to 24 hours [1]. The exact pathogenesis of TGA remains unclear; however, multiple etiologies have been described and associated with this entity such as metabolism, physical or emotional stressors, and cerebral infarction [2]. Amnesia due to fornix infarct, such as in our case, remains an uncommon entity with only several cases reported in the literature.

Case summary

A 60-year-old right-handed male from out-of-town presented to the emergency department for evaluation of new onset memory disturbances after being found by his relatives in the morning soiled with his own feces and urine. The evening prior to presentation, the patient's family reported he was

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* Corresponding author.
E-mail address: sqihuang@numc.edu (S. Qi-Huang).

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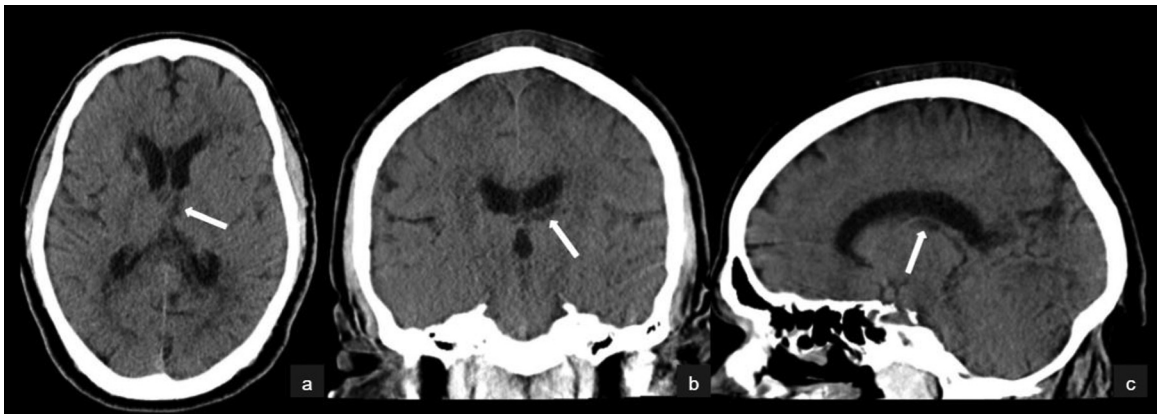

Fig. 1 – Sixty-year-old male with a history of substance abuse presenting with altered mental status and acute onset of amnesia. Findings: Noncontrast CT head in the axial (a), coronal (b), and sagittal (c) planes demonstrates a region of low attenuation in the expected location of the left fornix (white arrows) located adjacent to the left periventricular region and the inferior medial margin of the left posterior lateral ventricle.

found wandering in the backyard of a stranger's home after which he was brought by local law enforcement to his cousin's house. His family stated he was seen one week prior and exhibited normal behavior. On initial examination he was unable to recall immediate short term events, including why he presented to the hospital. His past medical history was significant for hypertension, substance abuse, depression, and a questionable history of prior stroke of unknown location and symptomatology.

He had a 15-year history of cocaine sobriety and recently relapsed secondary to an altercation with his significant other; he rarely consumes alcohol. Prior to this time, he performed all activities of daily living without assistance. He had no history of trauma or recent systemic or constitutional symptoms. There was no family history of cerebrovascular disease or dementia. He denied any headache, vision changes, weakness, numbness, paresthesia, dysphagia, speech changes, gait difficulties, or hallucinations.

On physical examination, the patient was confused, and not oriented to person, place or time. His vital signs were as follows: blood pressure 160/102 mm Hg, heart rate 107 bpm, temperature 99.1 °F, and respiratory rate of 18 bpm. His initial EKG showed normal sinus rhythm with no signs of ischemia.

His blood work, including complete blood count, basic metabolic panel, thyroid stimulating hormone, vitamin B12, liver enzymes, and inflammatory markers (erythrocyte sedimentation rate (ESR) and C-reactive protein) were within normal limits. Urine toxicology was positive for cocaine and benzodiazepines. A repeat EKG showed QT prolongation and repeat laboratory tests showed elevated troponins likely due to rhabdomyolysis following cocaine-induced vasospasm. Carotid doppler ultrasound demonstrated mild atheromatous plaque at the bilateral carotid bifurcations without hemodynamically significant stenosis. Echocardiogram showed mild aortic stenosis with normal left ventricular function.

The patient was admitted to the hospital and the neurology service was consulted to rule out seizure and perform a neuropsychological evaluation for assessment of cognitive status. At this time, the patient again could not recall the circumstances that brought him to the hospital. He also could not remember if he was using any drugs prior to his admission. He did state he had recent onset memory problems that began approximately one month prior but could not recall an eliciting event. He scored 4/22 on the Montreal Objective Cognitive Assessment (MOCA-Blind, due to COVID precautions), in which visuospatial/executive function was not evaluated. He exhibited prominent deficits in delayed recall (0/5) that were not improved by cueing, language (0/5), abstraction (1/2), orientation (1/6), and attention (2/6). His affect was normal with mild disinhibition. There was no dysarthria or ideomotor apraxia. Primitive reflexes were absent. He continuously repeated the same questions within one to two minutes of receiving an answer. The remainder of his neurological examination was within normal limits, including cranial nerves II-XII, motor examination, sensory testing, reflexes, coordination, balance, and gait testing. He denied any prior psychiatric history or use of psychiatric medications.

Noncontrast computed tomogram (CT) of the head showed a geographic hypodense area in the expected location of the left fornix without mass effect or edema suggestive of a subacute infarct (Fig. 1). In addition, an old infarct was demonstrated in the left occipital lobe, as well as small old lacunar infarcts in the right basal ganglia and left cerebellum.

Magnetic resonance imaging/angiography (MRI) of the head and neck demonstrated high signal on diffusion-weighted image (DWI) with corresponding low signal on apparent diffusion coefficient (ADC) in the posterior left fornix, involving the body, crus, and the fimbria compatible with acute infarction (Fig. 2). Magnetic resonance angiography (MRA) of the head and neck showed normal appearance of the intracranial vessels (Fig. 3).

The patient was treated medically with aspirin, lisinopril, atorvastatin, amlodipine, and levetiracetam. In addition, he was counseled to avoid cocaine abuse. The patient was from out-of-town and was unfortunately lost to follow-up after his discharge.

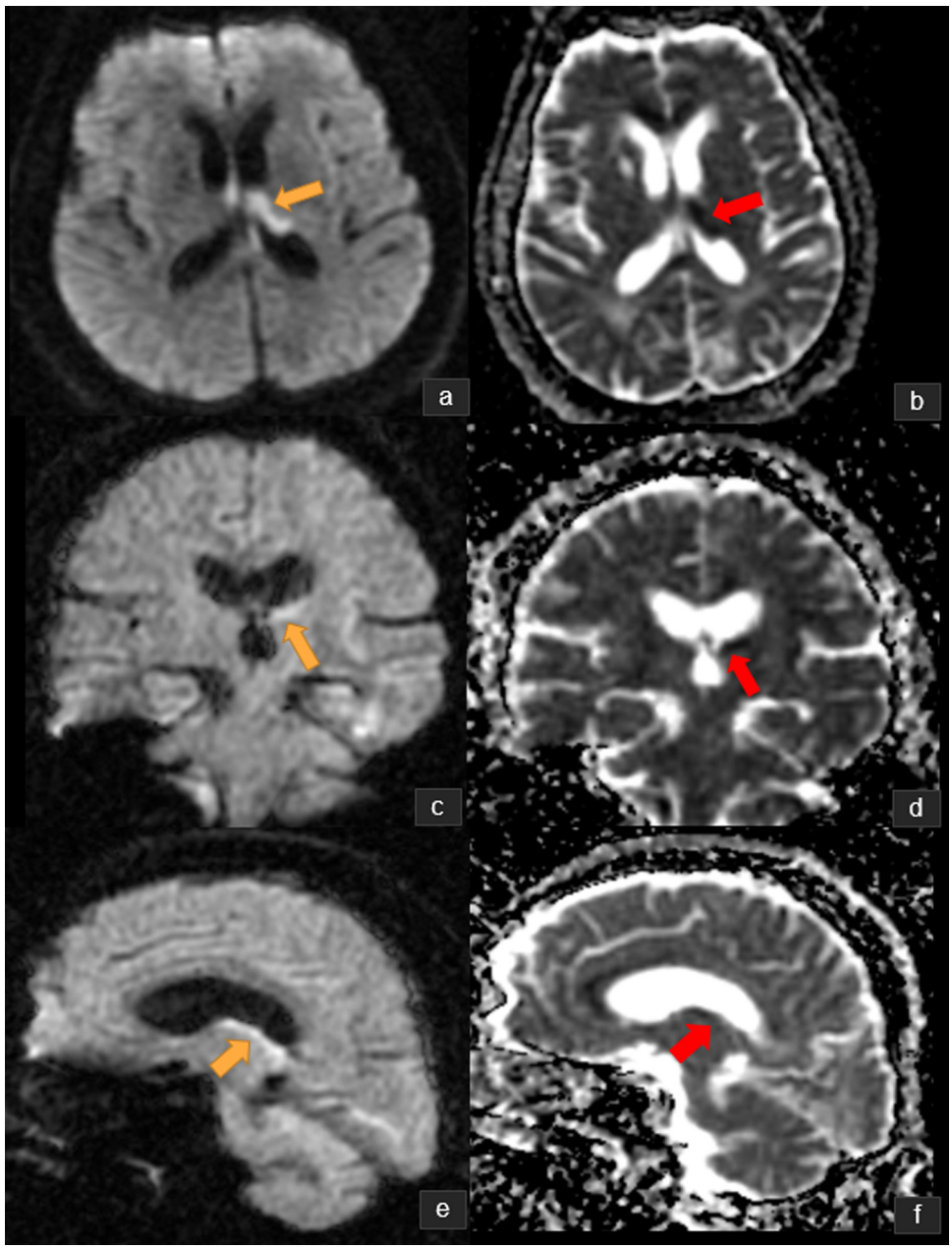

Fig. 2 – Sixty-year-old male with a history of substance abuse presenting with altered mental status and acute onset of amnesia. Findings: Noncontrast MRI brain with diffusion-weighted imaging (DWI) in the axial (a), coronal (c), and sagittal (e) planes with corresponding apparent diffusion coefficient (ADC) maps in the axial (b), coronal (d), and sagittal (f) planes demonstrate an area of hyperintensity (orange arrows) and hypointensity (red arrows), respectively, suggestive of an acute ischemic injury in the left fornix.

Discussion

The fornix is a white matter tract bundle that acts as the primary outgoing pathway of the hippocampus, and plays an important function in memory [3]. A review of the literature has highlighted several uncommon causes of TGA secondary to anatomical disruption or injury to the brain structures involved in memory propagation, including the fornix. Direct unilateral fornix injury with resultant memory impairment secondary to accidental trauma or iatrogenic causes from cerebral intervention like surgical resection have been described in case reports [4,5]. Pathophysiological causes such as vasculitis, cardioembolism, and even pregnancy-related pathology have been shown to cause bilateral forniceal injury leading to memory impairment [6,7].

Some authors describe the potential symptoms depending upon the laterality of the injury to the functional distribution of the fornix bundles. Injuries to the right fornical fibers result in visuo-spatial memory dysfunction, while lesions to

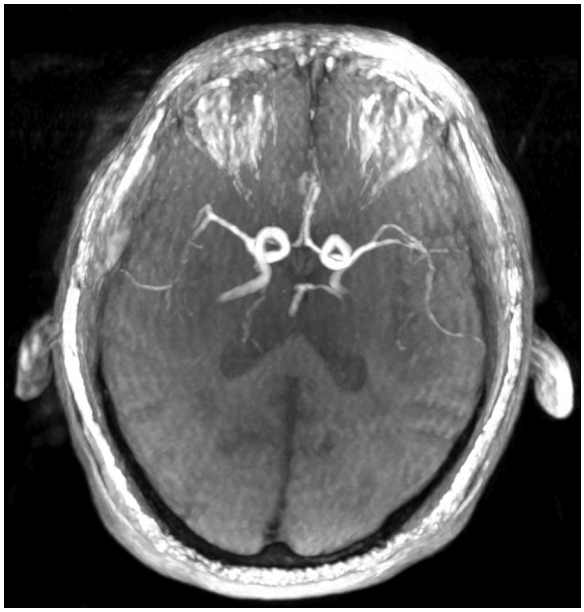

Figure 3 – 60-year-old male with a history of substance abuse presented with altered mental status and acute onset of amnesia. Magnetic resonance angiography (MRA) of the brain demonstrating normal vasculature of the brain. No filling defect was identified.

the left fornix bundles lead to verbal and memory impairment [3,8]. On the other hand, infarction of the thalamus can cause memory impairment and language deficits have been described according to the literature [9]. However, injuries to either anterior or posterior portion of the fornix appear sufficient to induce amnesic syndrome [10]. In our case, the patient was initially diagnosed with delirium secondary to cocaine use with a component of vascular dementia. Our patient demonstrated severe verbal memory dysfunction, which correlates with imaging findings of left forniceal injury. However, a complete detailed neuropsychological assessment was not performed, specifically the visuo-spatial memory was not tested due to COVID-19 pandemic related restrictions.

The anterior fornix is supplied by the subcallosal branches of the anterior cerebral artery or the perforating branches of the anterior communicating artery. The posterior fornix is supplied by the lateral posterior choroidal artery (LPChA) arising from the posterior cerebral artery [11]. Other authors have reported the anterior portion of fornical body and fornical column, specifically the pars libera, can be supplied by the medial posterior choroidal artery (MPChA), which arises from the posterior cerebral artery [11]. In addition, the MPChA forms anastomosis with the LPChA to supply the fornix has been described [12]. In our patient, there was no evidence of involvement of the anterior column, the anterior cingulate cortex, or the genu of the corpus callosum. In addition, there was no involvement of the thalamus. Therefore, in our case, the amnesia is associated with posterior fornix infarct and best explained by left posterior choroidal arteries, medial PChA and lateral PChA, as the injuries were restricted to the territory of posterior circulation, not the subcallosal artery.

Our patient had numerous risk factors for cerebrovascular disease, including his history of substance abuse, with evidence of multiple chronic infarcts involving different regions of the cerebrum. In view of the absence of a thromboembolic source, the TGA is likely precipitated by cerebral infarction of the fornix, the mechanism of steno-occlusion was, most likely, small-vessel disease. Our patient was treated with standard secondary stroke risk factor modification, a cholesterol-lowering agent, blood pressure recommendations, and lifestyle coaching.

Conclusion

This case highlights the importance for physicians to maintain an index of suspicion for fornix infarct in the event of acute amnesia. Brain imaging studies should be considered for patients presenting with acute onset amnesia. Early detection is needed to prevent unnecessary extensive workup and immediate treatment is important for good recovery.

Patient Consent

The authors declare to have obtained a written consent of the patient to publish the material in a journal/article.

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