

Case Studies

Isolated Mammillary Body Infarct Causing Global Amnesia: A Case Report

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Objectives: Mammillary bodies play an important role in human memory and emotions. Vascular lesions causing an isolated mammillary body lesion without affecting the surrounding structures are very rare. *Methods:* A 53-year-old male was brought to the emergency department with acute-onset memory problems suggestive of partial anterograde and retrograde amnesia. *Results:* Magnetic resonance imaging revealed an isolated left mammillary body infarct sparing adjacent structures. *Conclusion:* Mammillary bodies play an intrinsic role in memory formation and retrieval rather than acting as relay-only station for hippocampal projections. Non-hippocampal input from the limbic midbrain via the ventral tegmental nucleus of Gudden could be contributing to its function. **Key Words:** Mammillary body—mammillothalamic tract—amnesia—infarct—magnetic resonance imaging. © 2016 National Stroke Association. Published by Elsevier Inc. All rights reserved.

Background

Human memory is a complex mechanism by which we perceive, store, and retrieve information received from previous knowledge or experiences. It is a function of the intricate neuronal pathway with significant overlap with limbic system that deals with emotions. Mammillary bodies (MBs) are paired diencephalic structures located posterior to the hypothalamus and are a part of Papez circuit. It is traditionally considered a relay-only station for hippocampal projections to the anterior thalamic nuclei via mammillothalamic tract (MTT) with no function of its own. However, recent studies have explored the

possibility of MBs' contribution in memory independent of hippocampal formation via afferents from the ventral tegmental nucleus of Gudden.¹ Hereby, we present the first case report of isolated left MB infarct leading to partial global amnesia affecting both anterograde and retrograde memory.

Case Presentation

A 53-year-old, right-handed, African-American man with past medical history of hypertension was initially brought to the emergency department for acute-onset of confusion and memory deficit. At baseline, the patient had no neuropsychiatric deficits and was enjoying a normal life. On the morning of his presentation, he dropped his daughter off at school, and later was asking his wife where his daughter was. He also inquired about a family member who had passed away recently, and he had attended his funeral. In the emergency department, the patient denied having any neurologic deficit while exhibiting symptoms of amnesia. He was a chronic smoker and occasional drinker and was non-compliant with his hypertension medication (Tribenzor: olmesartan medoxomil, amlodipine, hydrochlorothiazide 20 mg/5 mg/12.5 mg). The rest of the patient's medical history was unremarkable.

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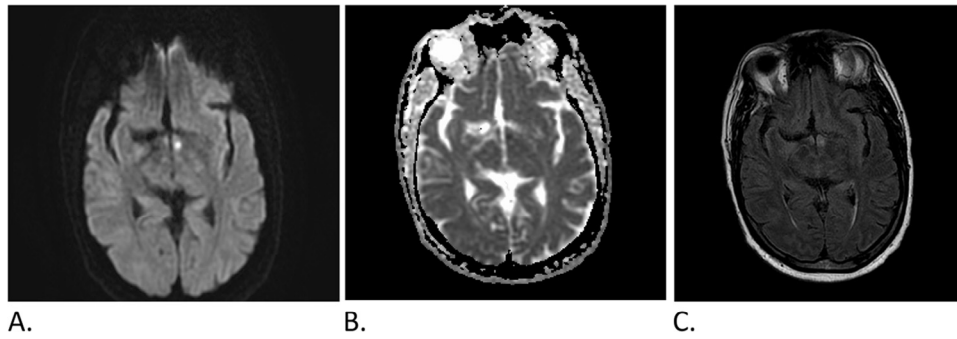


Figure 1. Magnetic resonance images of the brain: Diffusion-weighted image reveals a punctate hyperintensity in the left mammillary body (A). Corresponding drop in apparent diffusion coefficient sequence confirms the presence of cytotoxic edema secondary to an acute infarct (B). A fluid-attenuated inversion recovery sequence also reveals corresponding hyperintensity in the left mammillary body (C).

On arrival, the patient's blood pressure was 164/98 mm Hg. The rest of his vital signs were within normal limits. His examination revealed disruption of short-term memory with severe anterograde and mild-moderate retrograde components in addition to anosognosia. The rest of his neurologic examination was within normal limits.

The patient's initial laboratory findings were significant for slightly elevated serum body urea nitrogen of 22 mg/dL, serum creatinine of 1.6 mg/dL, and plasma glucose of 147 mg/dL. His remaining laboratory studies, including a complete blood count, basic metabolic panel, and liver function tests, were within normal range. Magnetic resonance imaging of the brain revealed a small punctate acute infarct in the left MB (Fig 1). Magnetic resonance angiogram was reported unremarkable; however, on close review, it was observed to have left proximal posterior cerebral artery (PCA) focal stenosis (Fig 2). The patient's transthoracic echocardiogram revealed ejection fraction of 65%, normal atrial sizes, and no evidence of thrombus. He was started on aspirin and statin. At 3-month follow-up, the patient continued to have amnesia, which was predominantly anterograde. Continuation of his symptoms beyond 24 hours differentiated it from commonly seen entity transient global amnesia.²

Discussion

Memory is one of the most fascinating and vital functions of the brain. A significant amount of knowledge regarding the complex pathways involved in memory has been derived from the lesions in human as well as animals. Memory regions are primarily composed of medial temporal areas (hippocampal and parahippocampal gyri) and medial diencephalic areas (MBs, anterior thalamic nuclei, and mediodorsal thalamic nuclei).³ MBs are a paired structure located just posterior to the hypothalamus. They receive hippocampal inputs and convey it to the anterior and mediodorsal nucleus of the thalamus via MTT to play a significant role in emotions and memory.⁴ Recent studies have revealed that non-hippocampal projection from the limbic midbrain via the ventral tegmental nucleus of Gudden is vital to an MB's contribution to memory, as shown in Figure 3.¹ Often, the lesions involving MBs involve adjacent MTT or thalamic nuclei; hence, the exact role of the MB in memory remains elusive. A recent study of 12 patients with thalamic amnesia revealed that damage to MTT produces severe memory impairment as compared with damage to the mediodorsal nucleus of the thalamus.⁴ Much implicated anterior nucleus of the thalamus was found to be damaged in only 1 out of the 12

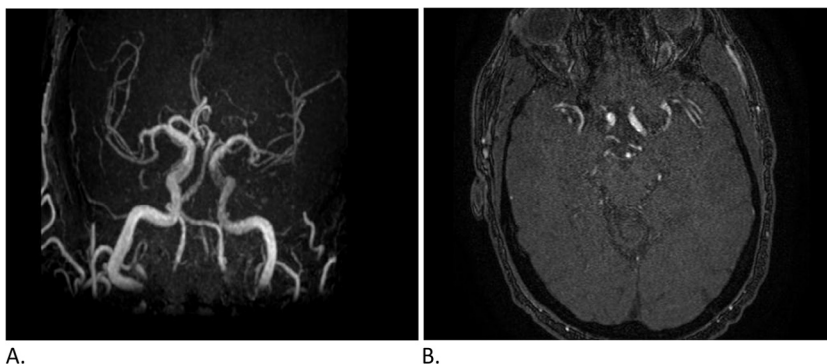


Figure 2. Magnetic resonance angiogram time of flight reconstruction image reveals the presence of the left fetal posterior cerebral artery with diminution versus occlusion of the left P1 segment (A). The presence of minimal flow on source images in the left P1 segment was confirmed on source images (B).

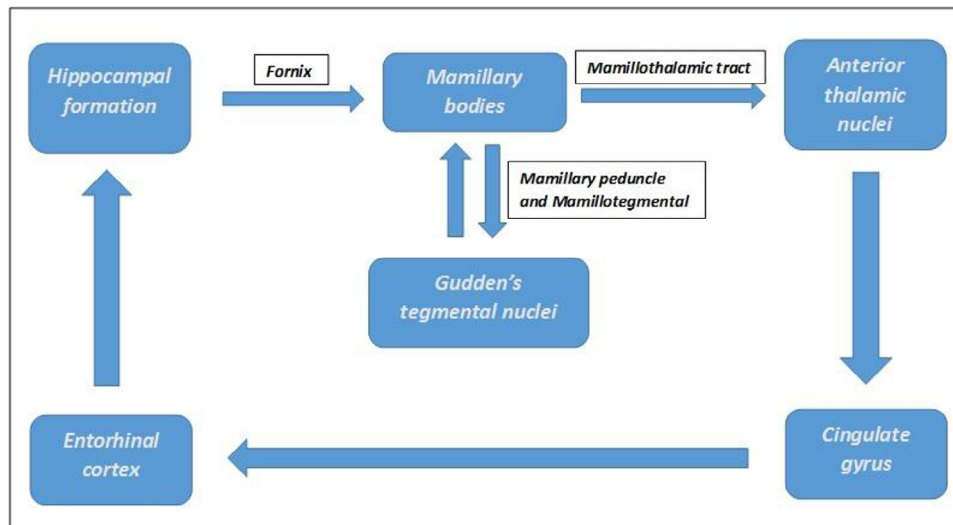


Figure 3. A simplified flowchart representation of updated “Papez circuit” involved in memory consolidation. Hippocampal pathways project to anterior thalamic nuclei. Later, thalamic projection provides input to cingulate gyrus and reaches back to hippocampal formation via entorhinal cortex. The figure shows that the ventral tegmental nucleus of Gudden makes reciprocal connections with mammillary body, providing non-hippocampal projections and possibly playing a vital role in memory independent of hippocampal pathways. Conduction tracts “Fornix,” “Mammillothalamic tract,” and “Mamillary peduncle and mamillotegmental tract” are represented above the respective arrows.

patients. However, none of those patients had damage to the MB. Our patient, in fact, had one of the rare isolated lesions of the left MB with sparing of surrounding MTT and thalamic nuclei. This lesion left the patient with partial but global amnesia, strongly suggesting the vital role of the MB in memory encoding and retrieval. There was no apparent deficit in executive function, but the presence of anosognosia indicates interruption of thalamic projection of MBs.⁴ Our patient’s magnetic resonance images (Fig 1) were a replica of a case reported by Amuluru et al; however, the clinical deficit was restricted to anterograde amnesia.⁵ MB receives its blood supply from perforators originating from proximal PCA (P1 segment) or posterior communicating artery.^{6,7} In our patient, he had a diminutive left P1 segment with left fetal PCA (Fig 2). Although animal experiments and functional neuroimaging continue to enhance our knowledge regarding the MB anatomy, its function, and its connections, lesion studies are equally important in further characterization.

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