

## Case Report

## Acute Amnesia due to Isolated Mammillary Body Infarct

Krishna Amuluru, MD,\*† Christopher G. Filippi, MD,†§ and Angela Lignelli, MD†

**Background:** There are limited reports describing acute amnesia after mammillothalamic tract infarction. Furthermore, acute infarction isolated to the mammillary body has never been reported. We present the first case of anterograde amnesia after isolated acute infarction of the mammillary body in a patient without concurrent or prior thalamic or mammillothalamic tract injury. **Methods:** A retrospective review of the patient's electronic medical record including inpatient notes and all radiological examinations was performed. **Results:** A 50-year-old woman presented with acute onset of confusion and constant repetition of the same questions. Magnetic resonance (MR) imaging of the brain showed isolated acute infarct of the left mammillary body without concurrent abnormality of the thalamus or mammillothalamic tract. MR angiography showed severe stenosis of the proximal posterior cerebral artery at the origin of the perforating mammillary artery. **Conclusions:** Isolated injury to the mammillary body is rare. In addition to recognized memory-related structures such as the thalamus and mammillothalamic tract, mammillary body injury may also play a role in memory dysfunction. Knowledge of the vascular supply of memory-related structures is important in diagnosing and understanding memory dysfunction. **Key Words:** Mammillothalamic tract—mammillary body—amnesia—cerebral infarction.

© 2015 by National Stroke Association

From the \*Department of Endovascular Surgical Neuroradiology, Rutgers, New Jersey Medical School, Newark, NJ; †Department of Neuroradiology, Columbia University Medical Center, New York, NY; ‡Department of Radiology, North Shore University Hospital-LIJ, Manhasset, NY; and §Department of Neurology, University of Vermont School of Medicine, Burlington, VT.

Received March 26, 2015; revision received May 25, 2015; accepted June 27, 2015.

Presented as Electronic Excerpta at 2014 American Society of Neuroradiology (ASNR) 52nd Annual Meeting and Symposium, May 2014, Montreal, Canada.

The work was performed at the Department of Neuroradiology, Columbia University Medical Center, 622 West 168th Street, PB 1-301, New York, NY 10032.

The authors declare that they did not receive any grant support, have nothing to disclose, have no author contributions, and have no conflict of interest.

Address correspondence to Krishna Amuluru, MD, Department of Endovascular Surgical Neuroradiology, Rutgers, New Jersey Medical School, 90 Bergen Street, Newark, NJ 07101-1709. E-mail: [kamuluru@gmail.com](mailto:kamuluru@gmail.com).

1052-3057/\$ - see front matter

© 2015 by National Stroke Association

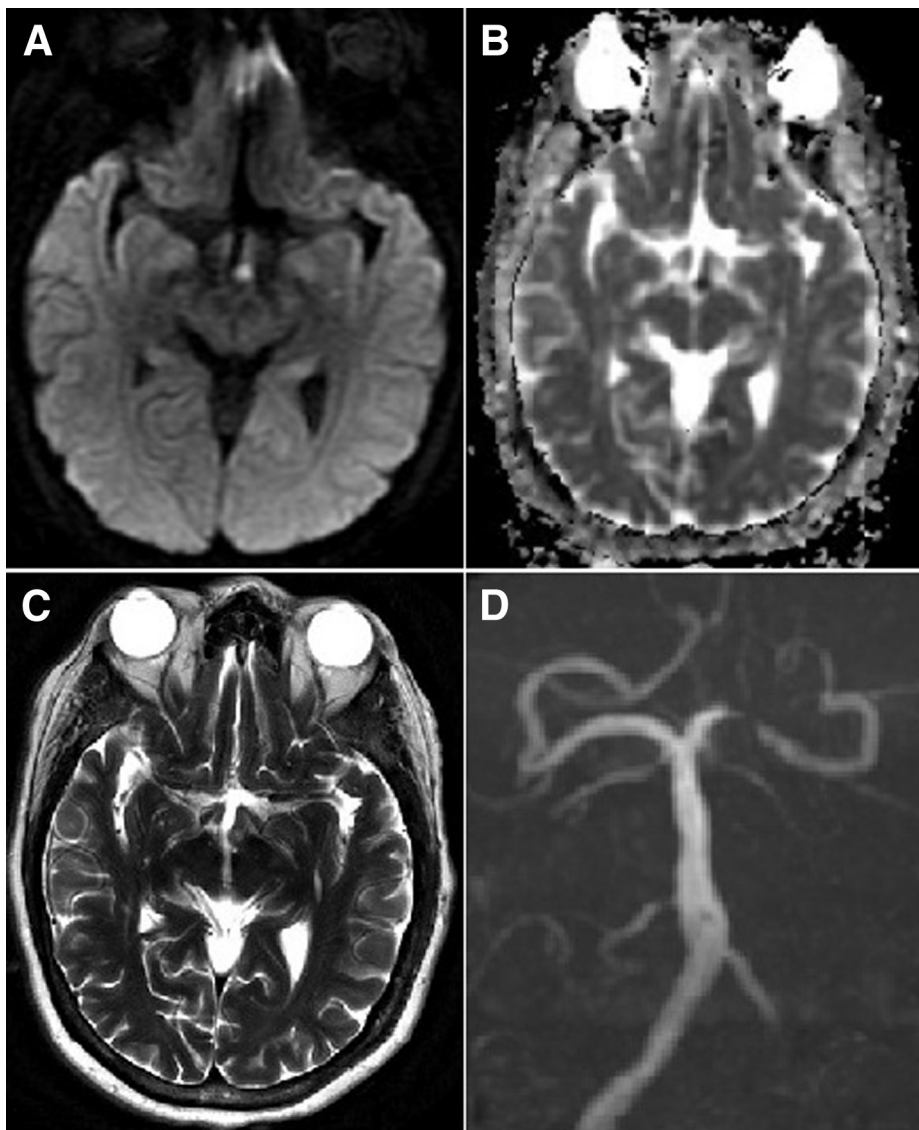
<http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2015.06.038>

## Case Report

A 50-year-old woman with hypertension, diabetes, and previously normal behavior was brought to the emergency room by her daughter with acute onset of confusion. She was constantly repeating the same questions and had difficulty forming new memories. She could recall that a plumber came to her house the morning of admission but she did not recall him leaving.

On arrival, her temperature was 37°, blood pressure was 142/82 mm Hg, and her heart rate was 77 and regular. On mental status testing, she was unable to recall 3 words 5 minutes after successful registration. Her neurologic examination was otherwise normal. Laboratory tests including complete blood count, basic metabolic panel, hepatic metabolic panel, urinalysis, and urine toxicity were normal.

Magnetic resonance imaging of the brain demonstrated left mammillary body restricted diffusion (Fig 1, A,B). T2-weighted sequences demonstrated asymmetric enlargement and abnormal hyperintensity of the left mammillary



**Figure 1.** MRI brain. Axial diffusion-weighted image (A) and associated ADC map (B) demonstrate focal restricted diffusion involving the left mammillary body. T2-weighted sequence (C) shows asymmetric enlargement and abnormal hyperintensity of the left mammillary body. MRA with 3D time of flight reconstruction (D) shows focal area of narrowing involving the P1 segment of the left posterior cerebral artery. Abbreviations: ADC, apparent diffusion coefficient; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.

body (Fig 1, C). She was admitted with a differential that included stroke, seizure, atypical Wernicke encephalopathy, or an inflammatory process. A search for alternative etiology of her confusion, including infectious and metabolic workup, was negative. Continuous electroencephalogram monitoring showed no epileptiform activity. Magnetic resonance angiography showed signal loss within the proximal left posterior cerebral artery (PCA; Fig 1, D). Her diagnosis was established as an acute mammillary body infarction due to high-grade stenosis of the left PCA.

During her hospitalization, her examination remained unchanged. She had stable persistent difficulty with three-item recall and repeatedly failed to recognize the physician who examined her the prior day. Her language function was normal with normal fluency and prosody of speech, and intact naming and repetition without phonemic or semantic paraphasic errors. Her executive

function was normal and she was able to attend to her activities of daily living. Her vision was normal. Upon discharge on hospital day number 10, her anterograde amnesia was unchanged.

## Discussion

The mammillary bodies lie at the posterior margin of the hypothalamus and are a part of the circuit of Papez, which also includes the hippocampal formation, anterior thalamus, cingulate gyrus, and their interconnections. Although Papez originally suggested that this pathway was the basis for emotion, current understanding suggests that it has a unique role in memory and cognition.<sup>1</sup>

Within this network of structures involved in memory, the anterior thalamic nuclei and the direct hippocampal–anterior thalamic connections via the fornix are critical components. This is evidenced by several reports

showing that damage or disconnection of the anterior thalamic nuclei is consistently associated with profound memory and learning impairments in rodents and anterograde amnesia in humans.<sup>2,3</sup>

The anterior thalamic nuclei also receive input from the mammillary bodies via the mammillothalamic tract (MTT). Animal model evidence has long suggested that the major participatory role of the mammillary bodies in anterior thalamic function is the indirect relay of “extended hippocampal” inputs to the anterior thalamus via the MTT.<sup>4</sup> However, recent advances are changing the understanding of the independent importance of the mammillary bodies, the mammillothalamic tract, and their contribution to anterior thalamic function.<sup>5</sup>

In humans, the association of memory dysfunction with the mammillary bodies and the MTT continues to evolve. Tumor-associated damage to the mammillary bodies causing memory deficits has been reported; however, these cases involved adjacent structures as well.<sup>6,7</sup> There are only a few reports documenting memory dysfunction following MTT infarction in humans.<sup>8,9,10</sup> Acute infarction isolated to the mammillary body has never been reported. We present the first case of anterograde amnesia after isolated acute infarction of the mammillary body in a patient without concurrent or prior thalamic or MTT injury.

Our case differs from previously reported cases in that our patient’s pathology was confined to the mammillary body. Park et al<sup>8</sup> reported global amnesia due to unilateral infarct of the left MTT in a patient with prior anterior right thalamic hemorrhage. Yoneoka et al<sup>10</sup> reported Korsakoff syndrome due to left MTT ischemia in a patient with additional infarction involving the anterior left thalamus as well as prior right MTT injury. Similarly, Schott et al<sup>9</sup> reported amnesia due to a left thalamic infarct with interruption of the MTT. Not only did our patient’s acute injury spare the thalamus and MTT, but she also had no prior injury to these regions.

The mammillary body has a blood supply discrete from the MTT and thalamus. The MTT shares a common vascular supply with other memory-related structures such as the fornix, the anterior and mediodorsal thalamic nuclei, and internal medullary lamina.<sup>8</sup> It is supplied via the tuberothalamic artery, which arises from the posterior communicating artery.<sup>11</sup> The mammillary body, however, is predominantly supplied by proximally perforating

mammillary branches off the P1 segment of the PCA.<sup>12</sup> In our patient, a thromboembolic event at the origin of the perforating mammillary artery caused an acute infarct of the mammillary body while sparing the MTT and thalamus.

Determining the contribution of an individual structure to memory is difficult because it is rare to find isolated damage in a clinical setting.<sup>10</sup> We present this case of anterograde amnesia due to isolated mammillary body infarct to elucidate this fascinating anatomical contribution to memory.

## References

1. Vertes RP, Albo Z, Viana Di Prisco G. Theta-rhythmically firing neurons in the anterior thalamus: implications for mnemonic functions of Papez’s circuit. *Neuroscience* 2001;104:619-625.
2. Aggleton JP, Sahgal A. The contribution of the anterior thalamic nuclei to anterograde amnesia. *Neuropsychologia* 1993;31:1001-1019.
3. Harding A, Halliday G, Caine D, et al. Degeneration of anterior thalamic nuclei differentiates alcoholics with amnesia. *Brain* 2000;123:141-154.
4. Aggleton JP, Brown MW. Episodic memory, amnesia, and the hippocampal–anterior thalamic axis. *Behav Brain Sci* 1999;22:425-444.
5. Dillingham CM, Frizzati A, Nelson AJ, et al. How do mammillary body inputs contribute to anterior thalamic function? *Neurosci Biobehav Rev* 2015;54:108-119.
6. Hildebrandt H, Muller S, Bussmann-Mork B, et al. Are some memory deficits unique to lesions of the mammillary bodies? *J Clin Exp Neuropsychol* 2001;23:490-501.
7. Tanaka Y, Miyazawa Y, Araoka F, et al. Amnesia following damage to the mammillary bodies. *Neurology* 1997;4:160-165.
8. Park KC, Yoon SS, Chang DI, et al. Amnesic syndrome in a mammillothalamic tract infarction. *J Korean Med Sci* 2007;22:1094-1097.
9. Schott JM, Crutch SJ, Fox NC, et al. Development of selective verbal memory impairment secondary to a left thalamic infarct: a longitudinal case study. *J Neurol Neurosurg Psychiatry* 2003;74:255-257.
10. Yoneoka Y, Takeda N, Inoue A, et al. Acute Korsakoff syndrome following mammillothalamic tract infarction. *Am J Neuroradiol* 2004;25:964-968.
11. Pedroza A, Dujovny M, Artero JC, et al. Microanatomy of the posterior communicating artery. *Neurosurgery* 1987;20:228-235.
12. Kaya AH, Dagcinar A, Ulu MO, et al. The perforating branches of the P1 segment of the posterior cerebral artery. *J Clin Neurosci* 2010;17:80-84.