Mamillary Body Enhancement on MR as the Only Sign of Acute Wernicke Encephalopathy

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Summary: We report a case of Wernicke encephalopathy in which the only sign of acute disease was enhancement of the mamillary bodies. This case demonstrates the utility of gadolinium enhancement at MR imaging as a means of diagnosing or confirming the syndrome of Wernicke encephalopathy even in the absence of atrophy or T2 abnormalities within the diencephalon and mesencephalon.

Index terms: Wernicke encephalopathy; Hypothalamus; Brain, magnetic resonance; Degenerative brain disease

Diencephalic and mesencephalic abnormalities in patients with Wernicke encephalopathy (WE) have been described at computed tomography (CT) to include atrophy or hemorrhage (1–4) and described at magnetic resonance (MR) to include atrophy and hyperintensity on T2-weighted images (5–11). We report a case of clinically acute WE in which the only imaging abnormality was diffuse symmetric enhancement of the mamillary bodies at MR imaging.

Case Report

A 59-year-old woman presented with complaints of dizziness and confusion after a 3-week period of severe nausea and vomiting. She had undergone radical pancreaticoduodenectomy (Whipple procedure) 5 months previously for treatment of ampullary adenocarcinoma. There was no history of alcohol intake. Physical examination disclosed lateral gaze nystagmus, unsteadiness of gait and stance, labile mood, and disorientation to time. A clinical diagnosis was not initially made, and MR was requested. Scanning was performed on a 1.5-T superconducting magnet (General Electric Medical System, Milwaukee, Wis; 4.7.9 Advantage). Sequences included sagittal spin-echo (500/11/1 [repetition time/echo time/excitation]) with 5.0-mm thickness and 1.0-mm gap, axial spin-echo (2500/30/0.5) (90° flip angle) with 5.0-mm thickness and 2.5-mm gap, and axial 3-D spoiled gradient-echo sequence (24/5/1) (35° flip angle) with 64 partitions 2.5 mm in thickness performed before and after intravenous administration of gadopentetate dimeglumine (Berlex, 0.2 mmol/kg). Flow compensation was used on the T2-weighted spin-echo sequence only. Reformatting of the volume data was performed on an independent console workstation. The only detectable abnormality was diffuse symmetric enhancement of the mamillary bodies (Fig 1). The mamillary body volume, calculated using the method of Charness and De LaPaz (7), was 52.3 mm (normal 51.7 ± 2.5 mm). A diagnosis of acute WE was proposed. The patient was treated with intramuscular thiamine and her nystagmus and ataxia improved, although she remained somewhat confused 1 month later. The history, physical findings, and response to thiamine administration all clinically confirm the diagnosis of WE. Relevant laboratory evaluation consisted only of radioassay of urine thiamine, which was negative, consistent with the diagnosis. Follow-up MR was not clinically indicated.

Discussion

WE is a common clinical condition caused by a deficiency of dietary thiamine that is most often seen in alcoholics, and presenting symptoms may include the classic triad of oculomotor dysfunction, ataxia, and global confusion (12). In addition to alcoholism, WE can be seen in any clinical setting in which thiamine is not ingested in adequate amounts or is lost because of protracted vomiting. WE is a reported complication of protracted vomiting caused by partial gastric resection (13), which this patient underwent as part of the Whipple procedure. The adult autopsy incidence of up to 2.2% (14, 15) testifies to the underdiagnosis of this syndrome, which is recorded clinically in only 0.04% to 0.13% of all hospital admissions (15, 16).

Prior imaging studies have demonstrated bilaterally symmetric lesions of the paraventricular regions of the thalamus, the hypothalamus, mamillary bodies, periaqueductal region, floor of the fourth ventricle, and midline cerebellum. Char-

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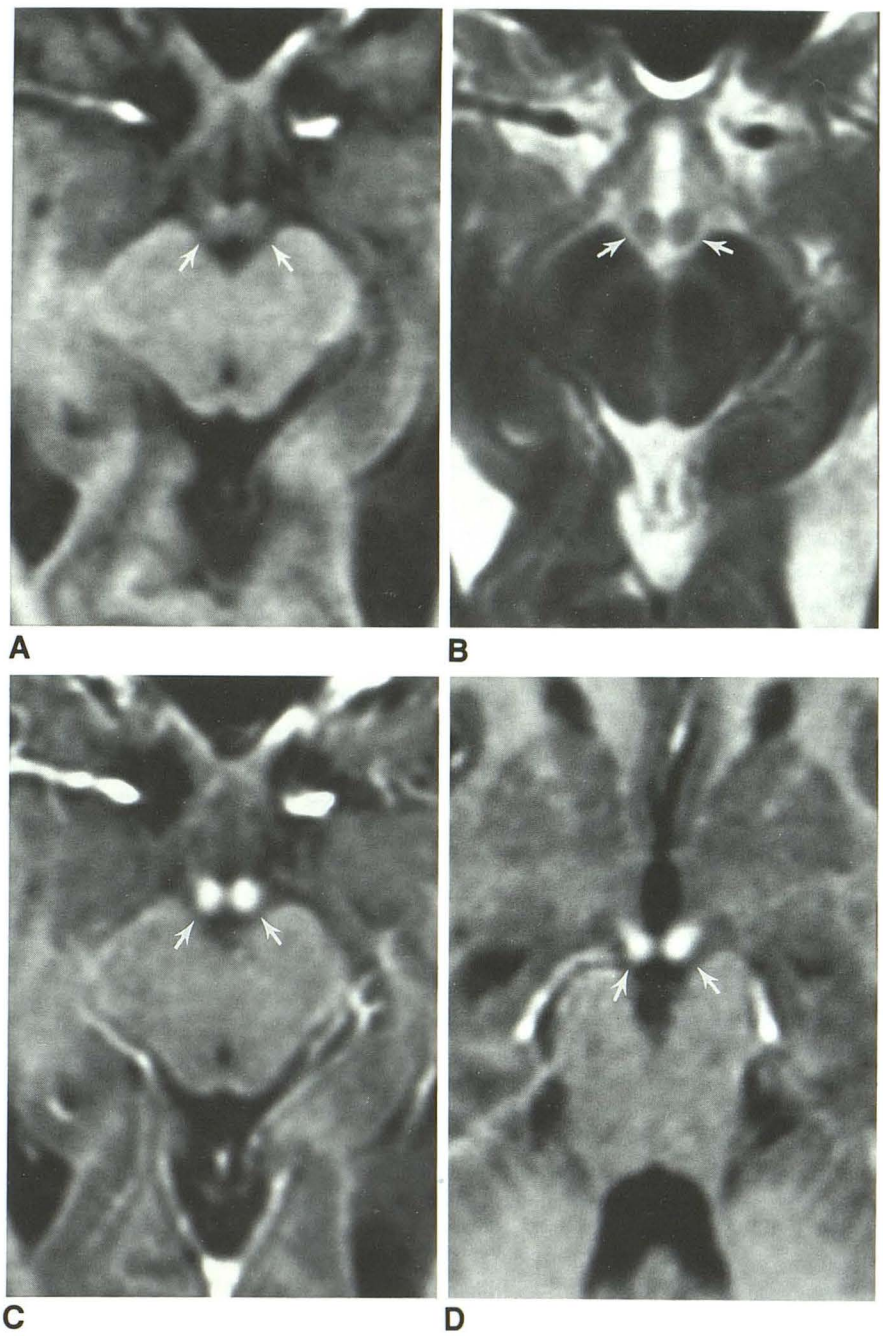

Fig. 1. Precontrast axial 3-D spoiled gradient-echo (24/5) (*A*) and axial spin-echo (2500/90) (*B*) images show normal-appearing mamillary bodies (*arrows*). Postcontrast axial 3-D spoiled gradient-echo (*C*) and postcontrast reconstructed paracoronal 3-D spoiled gradient-echo (*D*) images show bilateral mamillary body enhancement (*arrows*).

ness and De LaPaz (7) described mamillary body atrophy using MR, with sagittal scans consistently demonstrating atrophy in chronic WE patients compared with controls. Drayer (11) described hyperintense thalami on T2-weighted images, along with atrophic mamillary bodies and cerebellar changes. Donnal et al (5), along with Gallucci et al (6), reported several cases showing that these hyperintense areas surrounding the third ventricle and aqueduct diminished on follow-up examinations although concomitant ex vacuo enlargement of the aqueduct and third ventricle was noted.

This case shows that the mamillary bodies may demonstrate striking gadolinium enhancement in acute WE before or without the development of T2 abnormalities, allowing another means of imaging diagnosis. One might also expect enhancement of hyperintense periaqueductal areas, if present. The exact mechanism underlying the pathogenesis of WE and, more specifically, resulting in enhancement of the mamillary bodies,

is not known. One postulate is that the deficiency in thiamine-related phosphoric esters in cell membranes results in failure to maintain the normal osmotic gradient between the extracellular and intracellular spaces (6), thereby resulting in blood-brain barrier breakdown. The presence of mamillary body enhancement in the absence of detectable T2 abnormality initially seems paradoxical. However, the conspicuity of the contrast between the paramagnetic effect of gadolinium in the mamillary bodies and the surrounding cerebrospinal fluid is understandably more noticeable than any subtle change in T2 that may occur in the gray matter mamillary bodies. The effect may be likened to the increased detection of small cortical metastases in postcontrast T1-weighted images when compared to ordinary T2-weighted images (17). It is also possible that T2 changes could be either emerging or regressing, similar to the "fogging effect" in cerebral infarction, such that T2 differences from normal gray matter are minimal even though striking gadolinium enhancement may be present (18).

In conclusion, gadolinium enhancement of mamillary bodies in WE may in some cases lead to earlier diagnosis and treatment, which holds the promise of reducing or preventing permanent tissue damage and clinical sequelae thereof (12).

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