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# Enhanced MR in the Acute Phase of Wernicke Encephalopathy

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**Summary: MR in a patient with Wernicke encephalopathy showed enhancement in the mamillary bodies and inferior quadrigeminal plate. These findings pointed to the correct diagnosis, which can be difficult to make in patients who are not alcoholics.**

**Index terms: Wernicke encephalopathy; Brain, magnetic resonance; Nutritional disorders**

Wernicke encephalopathy is caused by a nutritional deficiency of thiamine. Magnetic resonance (MR) studies show typical mesencephalic/diencephalic lesions responsible for neurologic symptoms. Early diagnosis and treatment halts progression of the disease.

## Case Report

A 35-year-old woman was hospitalized because of an acute onset of paralysis of conjugate eye movements, ataxia, nystagmus, and mental confusion. Fundus examination showed edema of the papilla. Five months before hospitalization she had undergone surgery for obstructive jaundice and pancreatitis from lithiasis and had been put on parenteral therapy. The neurologic symptoms appeared 1 month after surgery.

The MR study was performed on a 0.5-T magnet 48 hours after the onset of neurologic symptoms. T2-weighted spin-echo (repetition time 2000–100) images showed hyperintense areas surrounding aqueduct and the third ventricle. After injection of gadopentetate dimeglumine, enhancement was observed on T1-weighted images in the inferior quadrigeminal plate and mamillary bodies (Fig 1). Given the selective involvement of these typical sites, Wernicke encephalopathy was suspected, and parenteral therapy with thiamine was instituted.

The patient improved rapidly, and a follow-up MR scan 2 weeks after therapy was instituted showed reduction in the size of the mamillary bodies and persistent contrast enhancement at the level of the inferior quadrigeminal plate and mamillary bodies (Fig 2).

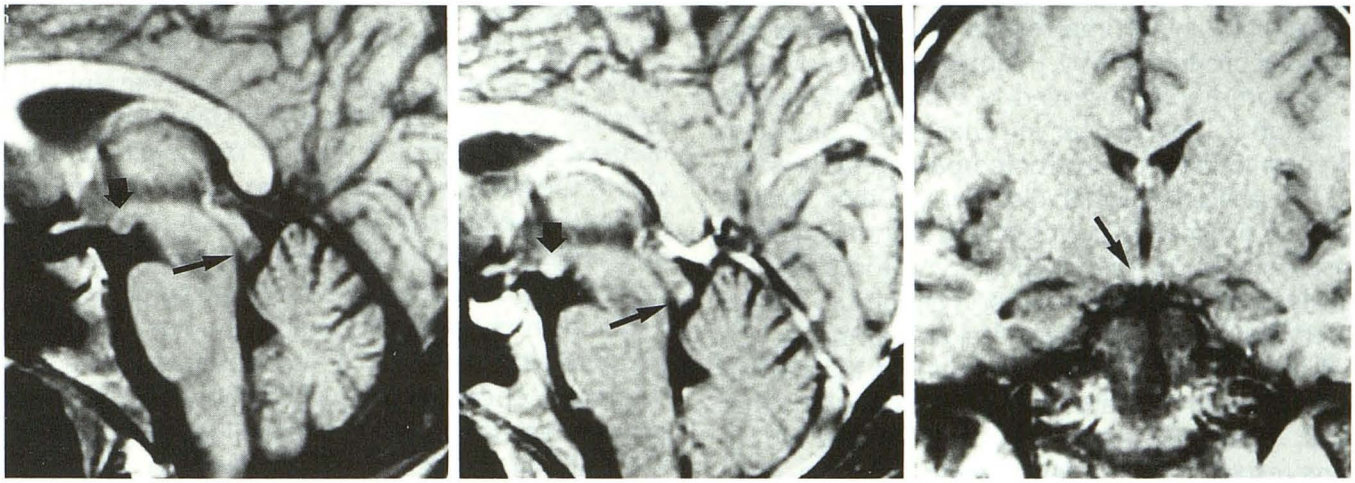


Fig. 1. T1-weighted gradient-echo (320/15 [repetition time/echo time], 90° flip angle) images in the midsagittal plane before (*A*) and after (*B*) administration of gadopentetate dimeglumine. Note the hypodense area at the level of the mamillary bodies (*wide arrow*) and inferior to the quadrigeminal plate (*thin arrow*) with enhancement after contrast. The coronal plane after contrast (*C*) shows the enhancement of the mamillary bodies (*arrow*).

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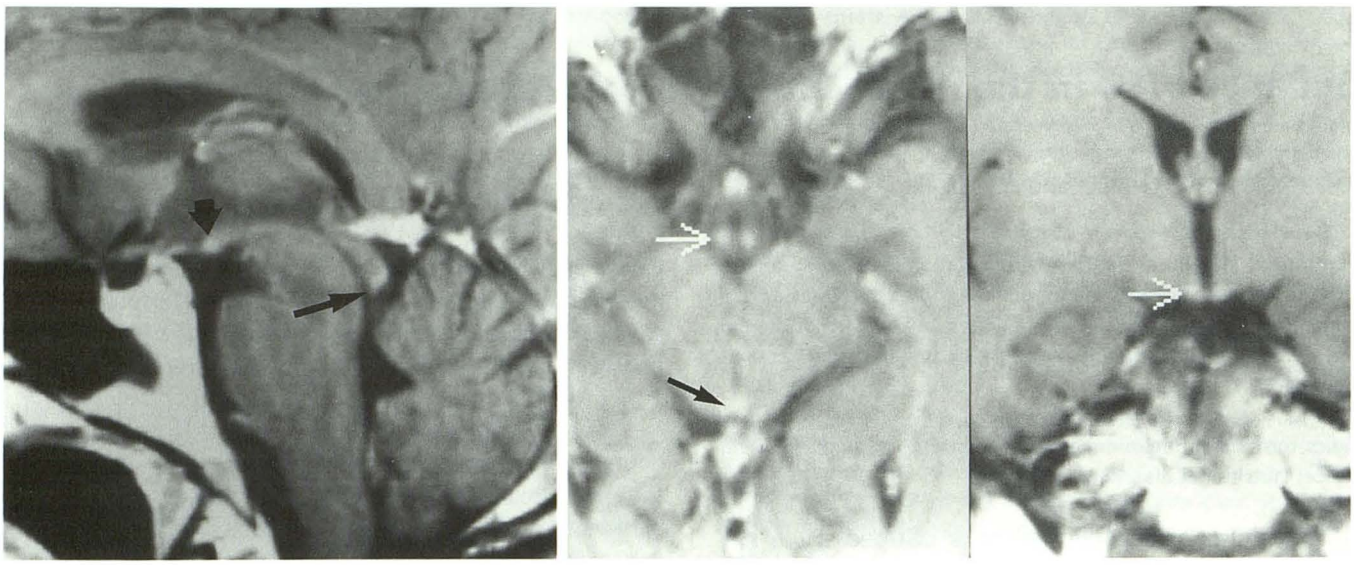

Fig. 2. Follow-up MR T1-weighted images in the midsagittal (*A*) and axial and coronal plane (*B*) (spin-echo, 500/15) with gadopentetate dimeglumine 2 weeks after institution of therapy show reduction in the side of mamillary bodies and persistent enhancement at the level of the inferior quadrigeminal plate (*thin black arrow*) and mamillary bodies (*wide arrow* and *white arrow*).

A 6-month clinical follow-up showed that her short-term memory was still impaired.

## Discussion

Wernicke syndrome results from a nutritional disorder brought on by thiamine deficiency. It is most commonly observed in chronic alcoholics, although it may appear in other conditions such as chronic uremia, protracted parenteral therapy (1), or gastrointestinal disorders. Other factors such as pregnancy or infections may trigger the onset of the disease. Typical symptoms include paralysis of eye movements, nystagmus, ataxia, deterioration of consciousness, and amnesia.

If thiamine therapy is not initiated the patient may become comatose and die. On the other hand, when proper therapy is instituted symptoms resolve within a few weeks. However, once the memory disorder stabilizes—in particular short-term memory disorders—improvement in memory is observed in only a small number of patients.

Patients with Wernicke disease present with symmetrical lesions of the periaqueductal gray matter, the floor of the fourth ventricle, the mamillary bodies, and perithird ventricle regions near the thalami and hypothalamus (2–4).

It may be difficult to formulate a clinical diagnosis in patients with an atypical history (nonalcoholics) and incomplete symptomatology. Imaging modalities may contribute greatly by providing an early diagnosis for this life-threatening disease.

MR plays an important role by showing the concomitant and symmetrical involvement of specific areas: T2-weighted images show increased signal near the floor of the fourth ventricle, in the periventricular region of the thalamus, and in the periaqueductal gray matter (3, 4). After appropriate vitamin treatment the signal becomes normal (1), and these areas may atrophy (4).

Involvement of the mamillary bodies has been documented in 98% of the autopsy cases of Wernicke encephalopathy (5); however, it has been detected by imaging modalities only during the chronic phases with MR findings showing atrophy of the mamillary bodies (4, 6, 7). It is difficult to detect signal changes in the mamillary bodies in the axial plane during the acute phase because they are usually lost because of partial volume averaging with the suprasellar cistern (3). In this case, Wernicke encephalopathy was suspected on the basis of the MR findings showing contrast enhancement of inferior quadrigeminal plated and mamillary bodies. Our diagnosis was

confirmed when most of the patient's symptoms rapidly resolved after thiamine treatment.

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