Enhanced MR in the Acute Phase of Wernicke Encephalopathy

P. D'Aprile, M. A. Gentile, and A. Carella

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 hypointense 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).
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 symp­
toms 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 mam­
illary bodies, and perithird ventricle regions near
the thalami and hypothalamus (2–4).

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

MR plays an important role by showing the
concomitant and symmetrical involvement of
specific areas: T2-weighted images show in­
creased signal near the floor of the fourth ventri­
cle, 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 sus­
ppected 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.

References
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