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**Gadolinium-enhanced MR findings in a pediatric case of Wernicke encephalopathy.**

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# Gadolinium-Enhanced MR Findings in a Pediatric Case of Wernicke Encephalopathy

Scott B. Harter and Steven R. Nokes

**Summary:** This report describes the postcontrast MR findings of Wernicke encephalopathy seen in a malnourished 11-year-old boy. The examination showed increased signal on T2-weighted images in the periaqueductal gray matter and medial thalami. On T1-weighted acquisition, these areas showed decreased signal intensity, but on postcontrast T1-weighted examination, they showed moderately intense enhancement. Also noted on the postcontrast examination was mamillary body enhancement.

**Index terms:** Wernicke encephalopathy; Nutritional disorders; Pediatric neuroradiology

Magnetic resonance (MR) and computed tomography findings of Wernicke encephalopathy have been previously described (1–6). Herein we describe a case of Wernicke encephalopathy in a malnourished 11-year-old boy who underwent gadolinium-enhanced MR soon after presentation.

## Case Report

An 11-year-old boy self-imposed a near-starvation diet for the 3 months before admission, apparently after he was teased about his obesity. Subsequently he had a documented 48-lb weight loss. He was admitted for treatment of weakness, lethargy, and dehydration.

Physical findings on admission were nonspecific, and included listlessness and mild confusion. Laboratory findings were also nonspecific. Cranial computed tomography findings without contrast were normal. Supportive therapy yielded no improvement in his condition. He had a fall during admission. Physical exam after the fall revealed bilateral abducens nerve palsies (intranuclear ophthalmoplegia) and, therefore, an MR study with gadopentetate dimeglumine was performed.

Cranial MR demonstrated abnormal increased signal on T2-weighted images in the periaqueductal gray matter (Fig 1A) and the medial thalami (Fig 1B). Signal intensity in the mamillary bodies on T2-weighted images was normal (Fig 1C). There was corresponding abnormal decreased signal on T1-weighted images in the periaqueductal gray matter (Fig 2A) and in the medial thalami (Fig

2B). Signal intensity in the mamillary bodies on T1-weighted images was normal (no figure). Following administration of gadopentetate dimeglumine, there was intense enhancement in the periaqueductal gray matter (Fig 3A), the medial thalami (Fig 3B), and the mamillary bodies (Fig 3C).

These findings were reported to the attending physician as consistent with Wernicke encephalopathy. Parenteral thiamine was administered immediately. Within hours, dramatic improvement occurred. The patient's neurologic status returned to normal with continued thiamine administration. Seven days after admission, he was discharged, neurologically intact. After addressing nutritional problems, there has been no recurrence of symptoms. Further supportive evidence for the diagnosis was a baseline thiamine level obtained just after the MR. The serum thiamine level was 0.1 mg/dL, with normal being 0.2 to 2.0 mg/dL. The result of this test was not available for several days after treatment was initiated.

## Discussion

Wernicke encephalopathy is caused by nutritional deficiency of vitamin B<sub>1</sub>, or thiamine. As described by Wernicke in 1881 (7), the disease is characterized by the onset of ocular muscle paresis, nystagmus, ataxia, and mental disturbances. In the early phase, there is apathy and confusion. If untreated, the usual (90%) course is development of Korsakoff psychosis, the dominant features of which are memory disturbance and confabulation (8).

Neuropathologic changes (edema, increased cellularity, occasionally hemorrhage) occur in the medial thalamus, the hypothalamus, the periaqueductal tissues of the midbrain, the gray matter of the floor of the fourth ventricle, and in the mamillary bodies (1, 3, 8, 9). The disease is found most commonly in alcoholics, but may be associated with any form of severe thiamine deficiency, including starvation, parenteral

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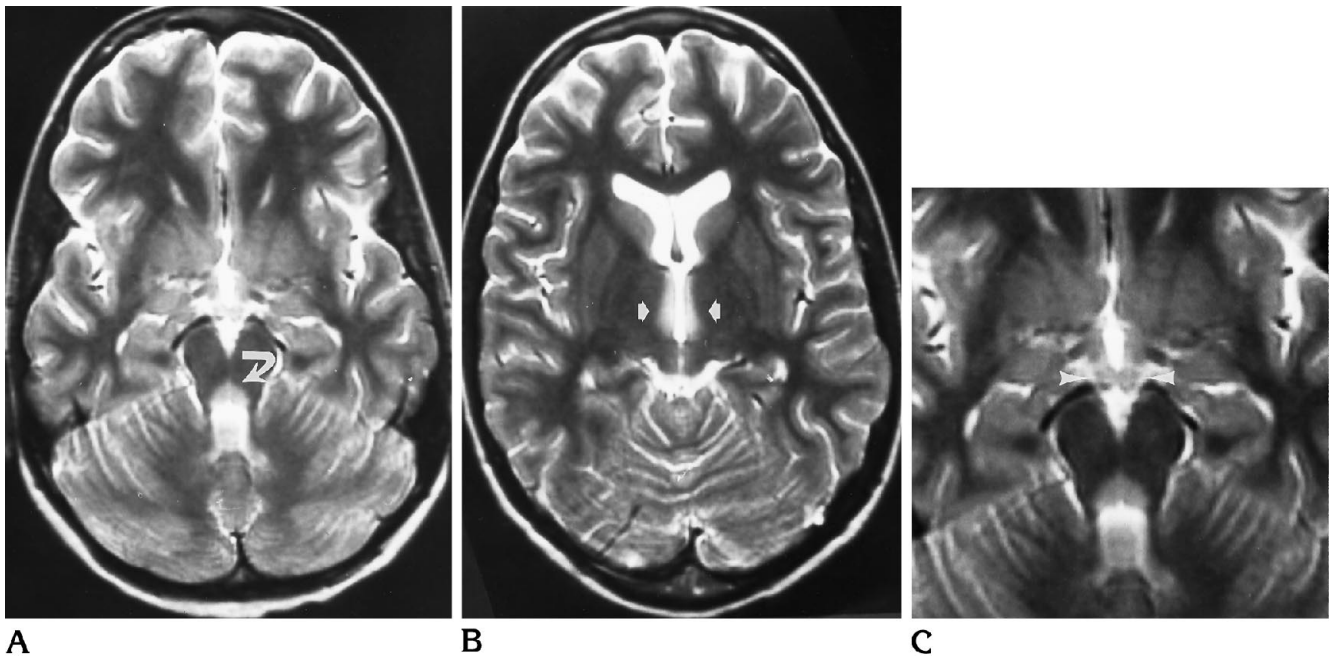


Fig 1. A through C, Fast spin-echo T2-weighted axial images (6000/60/1 [repetition time/echo time/excitations]) at the levels of the midbrain, the third ventricle, and the mammillary bodies, respectively. There is increased signal in the periaqueductal gray matter (A), and the medial thalami (B). Signal in the mammillary bodies is normal (C).

feeding, bowel obstruction, hyperemesis gravidarum, and hematologic malignancy (6, 7).

In this case, the MR was ordered because of abnormal physical findings (intranuclear ophthalmoplegia) noticed after a fall. However, the physical findings and subsequent MR findings were not felt to be posttraumatic.

The exact mechanisms underlying the pathogenesis of the lesions seen in Wernicke enceph-

alopathy are incompletely understood. Intracellular edema in astrocytes, oligodendrocytes, myelin sheaths, and neuronal dendrites is the earliest finding (10). Thiamine is important in maintaining osmotic gradients across cell membranes; thus, thiamine deficiency is associated with intracellular and extracellular edema (11). Progressive changes include demyelination, petechial hemorrhage, and cellular prolif-

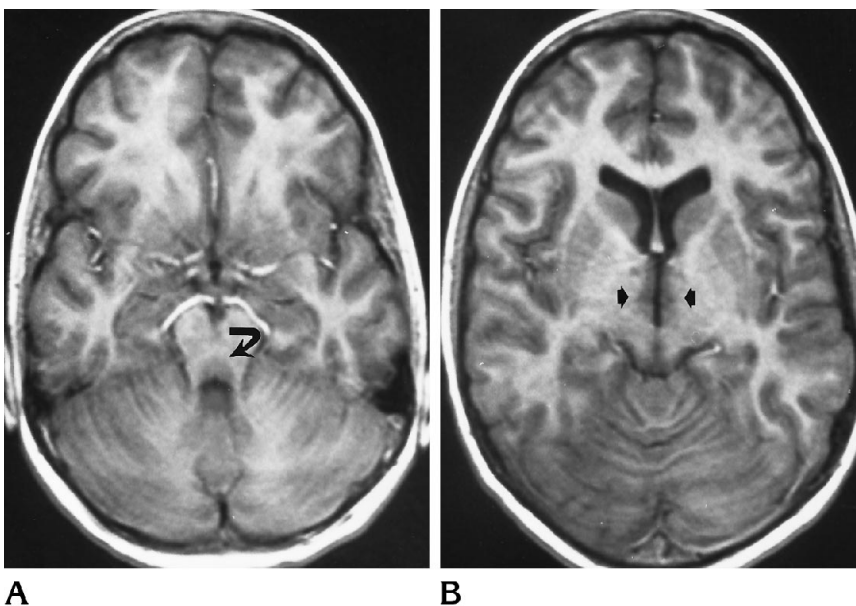


Fig 2. A and B, T1-weighted axial images (spoiled gradient-echo, 24/5/1, 35° flip angle) at the levels of the midbrain and the third ventricle, respectively. There is decreased signal in the periaqueductal gray matter (A) and the medial thalami (B).

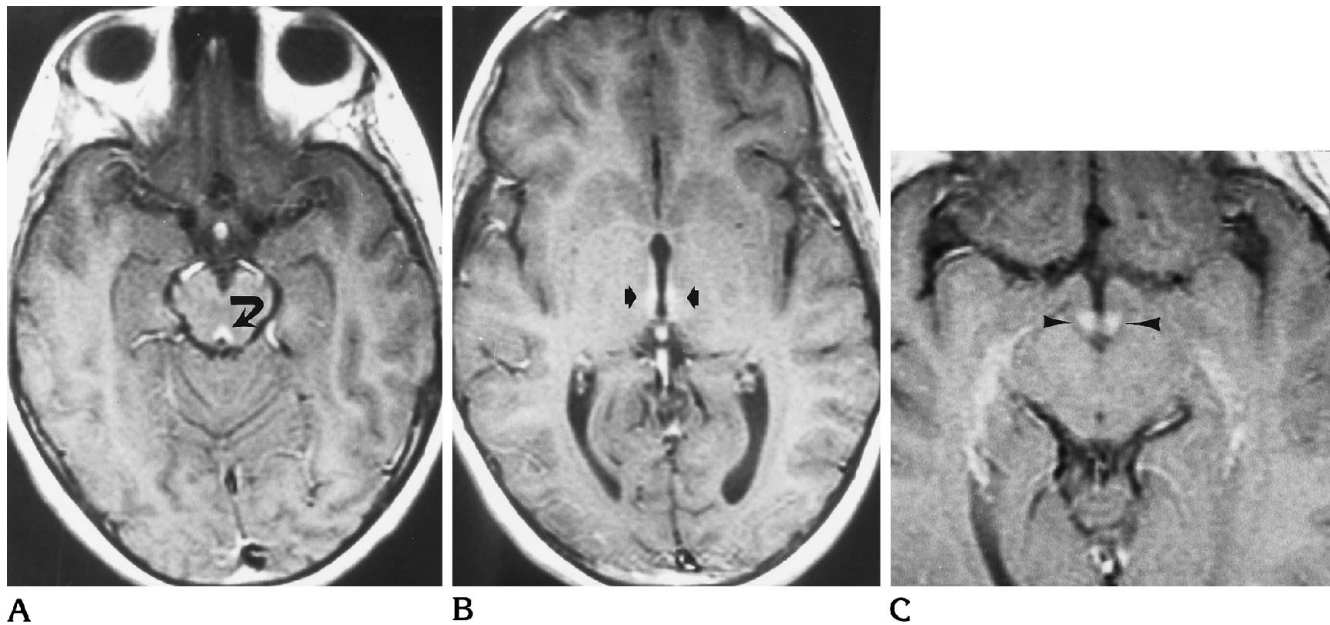


Fig 3. A through C, T1-weighted postgadolinium axial images (600/14/2) at the levels of the midbrain, the third ventricle, and the mamillary bodies, respectively. There is intense enhancement in the periaqueductal gray matter (A), the medial thalami (B), and the mamillary bodies (C).

eration. Severe involvement is characterized by tissue necrosis (12). Sites of disease are symmetric and include the mamillary bodies, medial thalami, and periaqueductal regions. Indeed, microscopic involvement of the mamillary bodies was described in 100% of 47 cases (12). Precontrast signal abnormality in this case was confined to the medial thalami and periaqueductal regions. However, postcontrast enhancement was evident in the mamillary bodies as well as these areas. The reason for the lack of precontrast signal abnormality in the mamillary bodies is uncertain. They did not appear to be atrophic.

In this acute/subacute presentation of Wernicke encephalopathy, the explanation of contrast enhancement can probably be related to demyelination, one of the relatively early histopathologic changes. Certainly demyelination is associated with enhancement, the classic example of which is seen in multiple sclerosis. Considering the complete clinical recovery, significant cellular necrosis is an unlikely cause of the enhancement.

MR provided crucial information in this case. The diagnosis of Wernicke encephalopathy was not suspected before the study. This particular case demonstrates the interesting findings of marked enhancement of several lesions. It also serves as another illustration of the usefulness

of MR in the diagnosis of Wernicke encephalopathy, even in an unusual pediatric presentation.

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