Encephalopathy: Proton MR Spectroscopy in Wernicke Encephalopathy

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Proton MR Spectroscopy in Wernicke Encephalopathy

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Summary: Wernicke encephalopathy is caused by thiamine deficiency. Although the clinical picture has been well established for some time, clinical diagnosis is attained in only 20% of the cases. MR imaging techniques contribute to early diagnosis of Wernicke encephalopathy. We herein report MR imaging and proton MR spectroscopic findings for a patient with clinical and biochemical features consistent with Wernicke encephalopathy. Increased lactate and typical MR imaging findings are discussed in the context of the known pathophysiology of Wernicke encephalopathy.

Wernicke encephalopathy is a metabolic disorder caused by thiamine (B1 vitamin) deficiency and is classically characterized by the clinical triad of ocular movement abnormalities, ataxia, and confusional state. Its prevalence in various neuropathologic studies is variable (nearly 2.8%) and its incidence is reported to be 1.7% (2). Despite this notably high frequency, clinical diagnosis of Wernicke encephalopathy is made in only 20% of the cases (3). It is important to understand that Wernicke encephalopathy has been described as being associated with many different non-alcohol-related pathologic conditions that share the common denominator of causing malnutrition and include hematologic malignancies (4).

During recent years, MR imaging has proved to be very useful in confirming the diagnosis of Wernicke encephalopathy and in contributing to earlier detection (5). Recently, reports of patients with Wernicke encephalopathy have shown restricted diffusion on diffusion-weighted images of brain regions characteristically compromised in this entity (6, 7). Only one description of proton MR spectroscopic changes in a patient with Wernicke encephalopathy has been reported (8). Even though the changes shown by diffusion-weighted imaging and especially proton MR spectroscopy have not yet been fully understood, they seem to constitute a new tool for understanding and diagnosing Wernicke encephalopathy. We herein report the case of a patient with reversible Wernicke encephalopathy who developed cerebral lesions and had changes shown by diffusion-weighted imaging and proton MR spectroscopy.

Case Report
A 34-year-old woman with acute myeloid leukemia was admitted to our center in July 2000 because of persisting nausea and vomiting associated with significant weight loss. Two months earlier, she had undergone bone marrow transplantation, and a few days later, she developed appetite loss, nausea, and vomiting that were initially attributed to chemotherapy. The symptoms did not resolve and were proved to be refractory to any medical treatment, so the patient was hospitalized. At admission, she was alert and well oriented without any neurologic abnormality. Because of oral intolerance, she was treated with intravenous administered dextrose saline solutions without vitamin supplementation. Blood examinations showed only moderate elevation of pancreatic enzyme concentration. The results of sonography and contrast-enhanced CT of the abdomen were normal, and an upper gastrointestinal tract videodenscopy revealed a mild superficial gastritis. The findings of contrast-enhanced MR imaging of the brain performed 2 days after admission were normal. During the ensuing 5 days, the patient’s condition evolved with progressive frontal cephalgia and dizziness. A neurologic examination showed mild inattention, central nystagmus, and axial ataxia. New laboratory tests did not reveal any significant data. The results of CT of the brain were normal. CSF examination revealed 2 cells/mm³ normal protein and low glucose concentration (28 mg/dL). Blood and CSF cultures were negative for bacterial or fungal infection, and the results of HIV and Venereal Disease Research Laboratories tests were also negative. A bone marrow biopsy ruled out acute myeloid leukemia relapse. The patient’s condition rapidly worsened, adding diplopia, lethargy, confusion, appendicular ataxia, and mild generalized weakness associated with decreased deep tendon reflexes. A second CSF examination revealed 2 cells/mm³ normal glucose (71 mg/dL), and elevated protein (57 mg/dL) concentration with an elevation of lactate to 50 mg/dL (normal range, 5.7–22 mg/dL). Cytomegalovirus serology and CSF protein chain reaction for tuberculosis and herpes virus were negative. Repeat MR imaging of the brain performed 10 days after admission and 6 days after the beginning of neurologic symptoms disclosed hyperintensities on T2-weighted, fluid-attenuated inversion recovery, and diffusion-weighted images in both dorsal and medial nuclei of the thalamus, periaqueductal gray matter, and superior and anterior cerebellar vermis without contrast enhancement or mass effect. The diffusion-weighted imaging abnormalities correlated with decreased signal intensity in the same areas on apparent diffusion coefficient maps, indicating restrictive diffusion. Two single voxel proton spectroscopic sequences with point-resolved spectroscopic volume selection (Philips) were measured with 2000/136, 272 (TR/TE, second TE). A total of 128 measurements were averaged, yielding an imaging time of 4 min 24 s, with 512 data points. Water suppression was achieved with an inversion pulse. A cubic 8-mL voxel was localized in the region of the T2 hyperintensity in the dien...
FIG 1. Proton MR spectroscopic image (TE, 272) shows a positive doublet centered at 1.3 ppm without reduction of N-acetylaspartate.

FIG 2. Proton MR spectroscopic image (TE, 136) confirms lactate as a negative doublet at this TE.
increased apparent diffusion coefficient was visually completely, and no sequelae remained. In this case, the neurologic symptoms in our patient reversed completely, which is a finding that has recently been correlated with edema by some authors, especially when it is seen on diffusion-weighted imaging (6, 7).

The apparent diffusion coefficient for our patient was decreased, which is a finding that has been associated with cytotoxic edema (6, 7). The apparent diffusion coefficient value.

In short, Wernicke encephalopathy is a potentially reversible condition with high morbidity and mortality rates that usually depend on the delay in starting thiamine treatment. Because the classic clinical triad is present in only 16% of cases (3), other paraclinical methods have become extremely important. The combination of conventional MR imaging with some of its new developments, such as diffusion-weighted imaging and proton MR spectroscopy, seems to constitute a powerful diagnostic tool with potentially prognostic value.
References