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AJNR Am J Neuroradiol 1994, 15 (3) 594-596
http://www.ajnr.org/content/15/3/594

Wernicke-Korsakoff Syndrome Caused by Psychogenic Food Refusal: MR Findings

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Summary: A 37-year-old woman developed Wernicke encephalopathy after prolonged psychogenic food refusal. MR revealed characteristic signal abnormalities in the midbrain and dorsal thalamus. Follow-up scans showed atrophy and third ventricular enlargement. Wernicke encephalopathy can occur in nonalcoholics, and MR imaging is useful in both the diagnosis and follow-up.

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

Wernicke encephalopathy is a clinical syndrome of delirium, external ophthalmoplegia, and truncal ataxia (1, 2). Korsakoff psychosis is an amnestic syndrome that often follows untreated Wernicke encephalopathy (1, 2). Although this syndrome occurs most frequently in association with chronic alcoholism, recent case reports have emphasized that it can occur in nonalcoholics as well. We report the brain magnetic resonance (MR) findings in a nonalcoholic patient who developed Wernicke-Korsakoff syndrome from psychogenic food refusal.

Case Report

A 37-year-old woman went to an emergency room in April 1991 for evaluation of a 20-year history of progressive fatigue, emesis and weight loss, reclusive behavior, delusions that certain foods were dangerous to her health, odd food preferences (eg, eating only pears for weeks at a time), a 70-pound weight loss, and thinking difficulties. She denied a history of alcohol abuse; this was confirmed by her family. On evaluation, severe cachexia, ophthalmoplegia, confusion, short-term memory deficits, and disorientation were noted. Brain MR was obtained on a 1.5-T unit (Figs 1 and 2). Lumbar puncture was noncontributory, and electroencephalogram showed diffuse slowing. She was treated with high-dose parenteral thiamine. Her short-term memory deficits and confusion persisted. She then developed severe cardiopulmonary complications requiring ventilator support and central alimentation. A 10-week follow-up MR scan (0.3-T) in June 1991 revealed a partial resolution of the periaqueductal T2 signal abnormalities and cortical atrophy. In November 1991 she was transferred to our hospital for further management. Physical examination revealed an extreme cachexia, flexion contractures of both lower extremities, confusion, disorientation, apraxia, marked short-term memory deficits, some deficits in remote memory, dysarthria, vertical nystagmus, lateral gaze palsy, positive palmomental, glabellar tap and snout reflexes, ataxia, and bilateral extensor plantar reflexes. Serum and cerebrospinal fluid studies were normal. Electroencephalogram showed mild background (7 Hz) and bitemporal (4 to 5 Hz) slowing. A follow-up MR scan was performed on a 1.5-T scanner (Fig 3). She was treated with nutritional supplementation, surgical correction of her flexion contractures, and extensive physical rehabilitation. Now at home, she has achieved 75% of her premorbid function and 100% of her premorbid weight.

Discussion

The clinical and imaging findings confirmed the diagnosis of Wernicke encephalopathy in this patient. In addition, the patient had a primary psychiatric diagnosis with delusions regarding food and chronic fatigue. Psychogenic food refusal was the likely cause of this woman's thiamine deficiency and Wernicke encephalopathy. The prevalence of Wernicke encephalopathy has been estimated at 2.2% in consecutive autopsy studies in adults, although the clinical diagnosis is probably less frequent (3). Chronic alcohol abuse remains the most common reported cause for thiamine deficiency and Wernicke encephalopathy. However, recent case studies have documented a variety of other causes that also may precipitate this syndrome (2). Patients receiving therapy for human immunodeficiency virus,

Received September 10, 1992; accepted pending revision November 6; revision received May 25, 1993.

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AJNR 15:594–596, Mar 1994 0195-6108/94/1503–0594

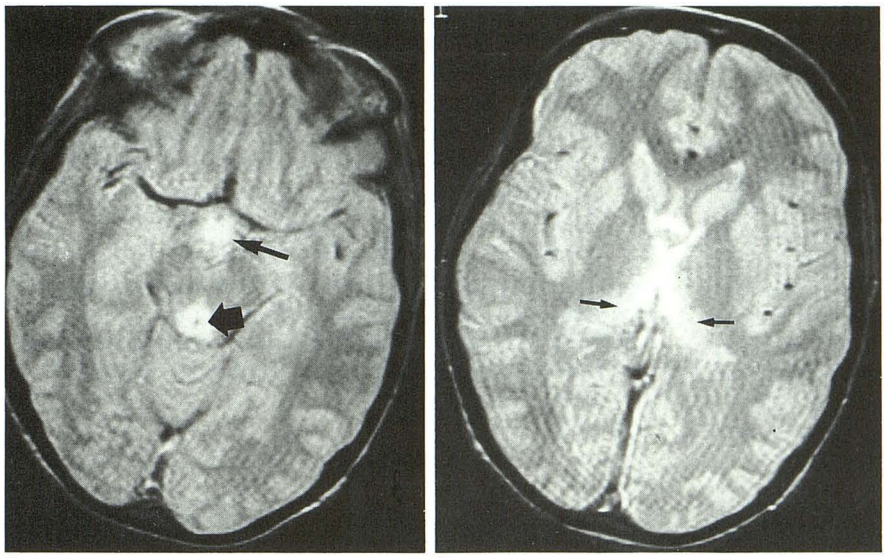

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Fig. 1. Proton-density MR image (2500/30 [repetition time/echo time]) from April 1991 shows symmetric signal abnormalities in the periaqueductal areas of the midbrain (*wide arrow*) and the hypothalamic/mamillary body region (*thin arrow*). In this figure and those that follow, motion artifacts are present.

Fig. 2. Proton-density MR image (2300/30) from April 1991 shows signal abnormalities in the periventricular areas of the third ventricle and dorsal thalamus.

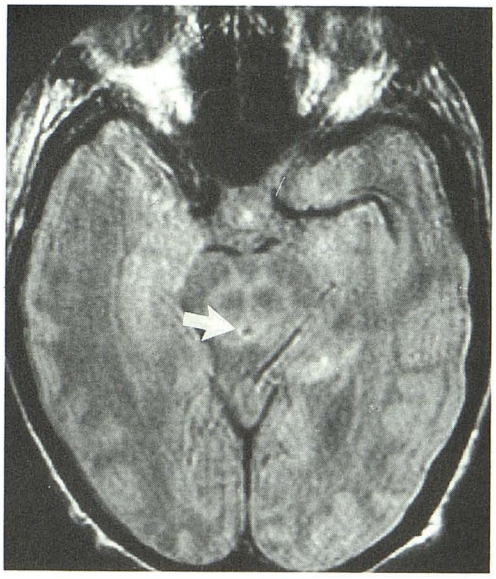

Fig. 3. Proton-density MR image (2200/30) from November 1991 shows dilatation of the aqueduct with decreased but persistent signal abnormalities in the midbrain. Motion artifacts are present.

chemotherapy for malignancies, immunosuppressants after bone marrow transplantation, or peritoneal dialysis or hemodialysis; patients on prolonged tube feedings; patients who had gastric or intestinal surgery; patients in critical care units who do not receive nourishment by mouth; and patients who remain ill with extended fever or dehydration are all at high risk. In addition, patients with eating disorders, severe malnourishment or beri beri, thyrotoxicosis, hyperemesis gravidarum, and diarrheal disorders are also at risk.

The role of thiamine in the pathophysiology of Wernicke syndrome may be related to its involvement in the function of excitable membranes and neurotransmitter production (4). Neuropathologic changes described in autopsies include marked edema; swelling of glia, myelin sheaths, and neuronal dendrites; demyelination; symmetric petechial hemorrhage; and glial proliferation (4). Some case reports describe MR findings in Wernicke encephalopathy (4–7). Symmetric signal abnormalities on T2-weighted MR images in the periaqeductal areas and bilaterally in the dorsal thalami are the typical acute or subacute findings. Follow-up MR scans in some reports have revealed atrophy of mamillary bodies, cortical atrophy, and third ventricular and aqeductal dilitation. Mamillary atrophy, best visualized on sagittal or coronal scans, has been reported to exceed the degree of cortical atrophy and is believed to be irreversible (8). Early thiamine replacement may result in complete resolution of thalamic and midbrain MR signal abnormalities, whereas delayed treatment appears to lead to a partial resolution of these changes. The areas of increased signal on T2-weighted images are believed to be pathognomonic for Wernicke syndrome. Some of the abnormalities mentioned above also may may be detected by CT (9), but with less sensitivity.

An increased awareness of the cardinal clinical signs, risk factors, and utility of MR imaging is

essential for the early recognition and reversal of this devastating disorder.

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