Case Report

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A Typical MRI Features of Wernicke's Encephalopathy: A Case Report

Telmen Khuvituguldur[1,3], Munkhbaatar Dagvasumberel[1,2], Delgerdalai Khashbat[1,2]

[1]Department of Radiology, School of Medicine, Mongolian National University of Medical Sciences, Ulaanbaatar, Mongolia;
[2]Department of Radiology, Mongolia-Japan Teaching Hospital, Mongolian National University of Medical Sciences, Ulaanbaatar, Mongolia;
[3]Diagnostic Imaging Center, The First Central Hospital of Mongolia, Ulaanbaatar, Mongolia.

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Corresponding Author:
Delgerdalai Khashbat (M.D., Ph.D)
Department of Radiology, School of Medicine, Mongolian National University of Medical Sciences, Ulaanbaatar, Mongolia
E-mail: delgerdalai@mnums.edu.mn
ORCID: https://orcid.org/0000-0003-3083-2003

Objective: This case report highlights the rare occurrence of Wernicke encephalopathy (WE) in a non-alcoholic patient with atypical imaging findings. **Methods:** Diagnosis is made based on clinical and imaging findings. **Results:** A 44-year-old man with a history of inability to walk, ataxia, and did not have a habit of alcoholism. Magnetic resonance imaging (MRI) showed symmetrical bilateral basal ganglia changes. **Conclusion:** We should always be included in the differential diagnosis for bilateral basal ganglia lesions. MRI has proven useful in diagnosing WE in patients with occasional neurological symptoms and atypical imaging findings.

Introduction

Wernicke's encephalopathy (WE) is a potentially critical neurological disorder precipitated by the lack of thiamine.[1] The clinical diagnosis of WE is based on the classical triad which includes ocular dysfunction, altered consciousness, and ataxia, however this triad occurs in only 16 to 25 percent of all patients, which explains in part why WE is often clinically underdiagnosed.[2,3] Among the diagnostic methods, MRI is considered the most effective method to confirm a diagnosis of WE. The specificity of MRI to detect diagnostic features of acute WE was 93%, however, the sensitivity of MRI imaging was relatively low, indicating that it can be ruled out.[4,6] MRI is should be used to support the diagnosis and recovery of acute WE both in alcoholics and non-alcoholics is recommended in many countries.[5,10] The main pathogenesis of WE is reversible cytotoxic edema, and it is easily shown on MRI. In following areas, the maintenance of cellular osmotic gradients is considered to be strictly related to thiamine levels.[7,8] In the case of WE, typical findings are represented by symmetric high signal intensity in the thalami, mamillary bodies, midbrain, tectal plate, and periaqueductal gray on MRI. Atypical MRI findings are represented by symmetric high signal intensity of the cerebellum, cranial nerve nuclei, basal ganglia and cerebral cortex.[9,11-15]

Case Presentation

A 44-year-old man with a history of inability to walk and ataxia was admitted to the teaching hospital of the Mongolian National University of Medical Sciences. The patient had no other significant past medical or surgical history and denied any history of alcohol consumption. According to his sister, his wife had passed away 2 months ago, which caused him to go into a depressed state of loss of appetite and sleeplessness. A physical examination revealed stable vital signs and normal temperature, but no particular findings were found. He exhibited spatial and temporal disorientation and demonstrated gait ataxia on a neurologic examination. His pupils were isochoric with prompt light reflex. A cranial nerve examination showed horizontal nystagmus and diplopia on both sides. No focal weakness or sensory changes were noted. The laboratory test results on that day revealed normal blood counts and serum electrolytes.

An abdominal ultrasound scan and chest X-ray showed no apparent abnormalities. MRI was performed on a 1.5T Scanner (Magnetom Symphony, Siemens, Germany) using an 8-channel head array coil. Conventional sequences, including T1WI, T2WI, and FLAIR, diffusion-weighted imaging (DWI), apparent diffusion coefficient (ADC) maps, susceptibility-weighted imaging (SWI), and time-of-flight (TOF) magnetic resonance angiography (MRA) were acquired. The brain MRI scan revealed symmetrical T2WI and FLAIR high signal intensity, T1WI slightly decreased signal intensity involving the bilateral basal ganglia, including the putamen, thalamus, midbrain, periaqueductal gray, posterior putamen, globus pallidus, external capsule, posterior part of pons (Figures 1, 2). DWI showed high signal intensities in the bilateral basal ganglia (Figure 1B). A cervical and thoracic spine MRI without contrast was also performed, and no significant abnormalities were found. These findings and clinical presentation are consistent with WE, likely precipitated by malnutrition related to the patient's recent severe emotional stress and depression. We suspected a WE and administered thiamine 1500 mg/day promptly intravenously. On day 5, gaze nystagmus, diplopia, and extrapyramidal symptoms gradually improved. A follow-up MRI on day 11 revealed high signal intensity in basal ganglia on FLAIR and T2WI slightly reduced.

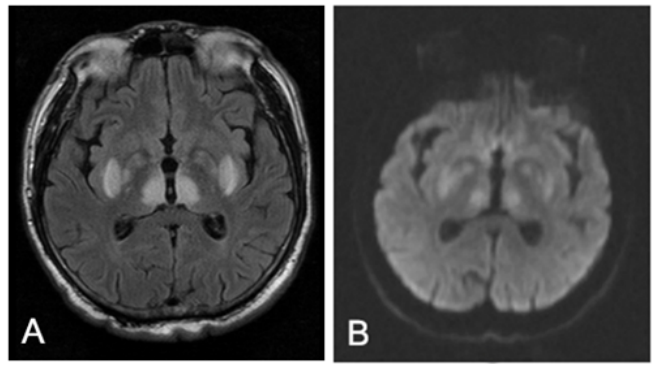

Figure 1. Axial FLAIR (A) image showed the symmetrical increased signal intensity of the posterior putamen, external capsule, and thalamus. DWI (B) showed symmetric restricted diffusion.

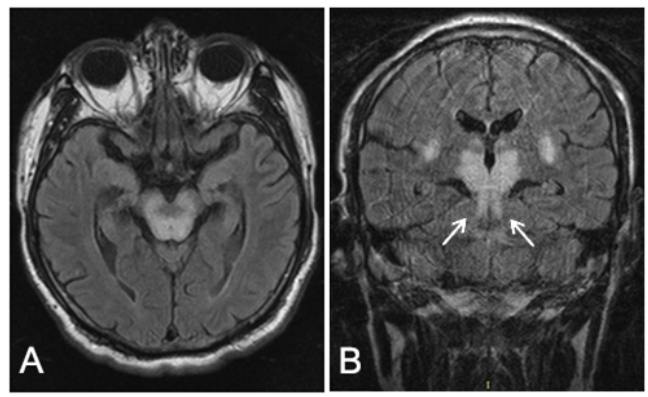

Figure 2. FLAIR axial (A) and coronal (B) images showed symmetrical increased signal intensity of the midbrain, including periaqueductal gray, tectal plate, and dorsal portion of pons (white arrows).

Discussion

The basal ganglia and thalamus are symmetrically affected by a wide variety of diseases, such as toxic poisoning (by carbon monoxide, methanol, cyanide) and systemic metabolic abnormalities, neurodegeneration with brain iron accumulation, vascular abnormalities, and some focal inflammatory and infectious conditions (neuro-Behçet disease, flavivirus encephalitides, toxoplasmosis) or neoplasms.[16-18] The treatment of suspected or manifest WE is based on the administration of thiamine.[10,11] If not treated or inappropriately treated with low doses of thiamine, WE can lead to irreversible brain damage that can cause death.[1,2,4,6,19,23] Previous studies have shown that MRI is the most effective diagnostic test for WE.[1,2,5] On the contrary, CT proved useless in diagnosing WE.[1,4,5] T2WI and FLAIR symmetric high signal intensity in the typical and atypical areas are thought to be associated with cytotoxic edema due to thiamine-related metabolic breakdown.[21,22] It was previously

believed that symmetric basal ganglia lesions occur specifically in children due to an increase in the rate of thiamine-dependent metabolism.[6] However, there have been reports that WE may affect the basal ganglia in adults and children.[20] According to the study by Ashikaga R. and his colleagues,[24,25] an MRI imaging of WE lesion visibility was found to be better with FLAIR imaging than T2WI, which is consistent with our case.

Conclusion

MRI is valuable in the diagnosis of WE, particularly in patients presenting with atypical symptoms and imaging findings or with no history of chronic alcohol use. The neuroimaging findings of WE may be helpful to diagnose the disease early to prevent morbidity and mortality. We should always be considered in the differential diagnosis for abnormalities of the basal ganglia in patients with malnutrition, chronic alcoholism, gastrointestinal surgery, and prolonged vomiting.

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