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Case report

Correlation of magnetic resonance images with neuropathology in acute Wernicke's encephalopathy☆

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Abstract

We correlated serial brain MRIs with neuropathological findings in a 16-year-old female whose autopsy was consistent with Wernicke's encephalopathy (WE). Diffusion-weighted imaging, diffusion coefficients mapping and neuropathology findings were suggested vasogenic edema in the periaqueductal and peri-the-fourth ventricular areas. This is the first documented case report to make this direct comparison. The characteristic WE changes in the mammillary body was also correlated with the findings of MRI with contrast enhancement. Bilateral cortical lesions revealed by MRI were atypical and rare in WE and were not evidenced by pathological changes.

Keywords: Acute Wernicke's encephalopathy; Diffusion-weighted; Magnetic; Resonance imaging; Vasogenic edema

1. Introduction

Wernicke's encephalopathy (WE) is an acute neurologic disorder resulting from a deficiency of thiamine and characterized by the triad of abnormal eye movement, gait ataxia, and a global state of confusion. Being primarily a clinical diagnosis, WE can be easily missed if it is not suspected. Without appropriate treatment, the disease can be fatal. An autopsy series of over 130 patients reported that only one-fifth of acute WE was actually diagnosed prior to death [1]. Recently, magnetic resonance imaging (MRI) has proven to be helpful in the early diagnosis of WE. However, the information about MRI-histopathological correlation is limited [2].

Herein we report on a patient with autopsy findings consistent with acute WE in whom brain MRI was done 4 h before her death. The report focuses on the correlations between the histopathological findings and both specific and unusual MRI findings of WE.

2. Case report

A 16-year-old girl was brought to our emergency room by her parents with the chief complaints of progressive lightheadedness and unsteady gait of 2 weeks duration. She did not have vertigo, weakness, stiffness, or any abnormal sensations. Her nutritional intake history was very poor. She had lost 2 kg in the past month because of frequent vomiting—reason unknown. She was neither a vegetarian nor an alcohol abuser, nor did she have any contributing family medical history. On examination, the patient was alert and afebrile with stable vital signs. Her neck was supple. She had slurred speech, rotatory nystagmus, and quadriparesis (Medical Research Council [MRC] scores of 3/5 in the lower limbs and 4/5 in the upper limbs). She did not have ophthalmoplegia but complained of diplopia during bilateral horizontal gazing. Though gen-

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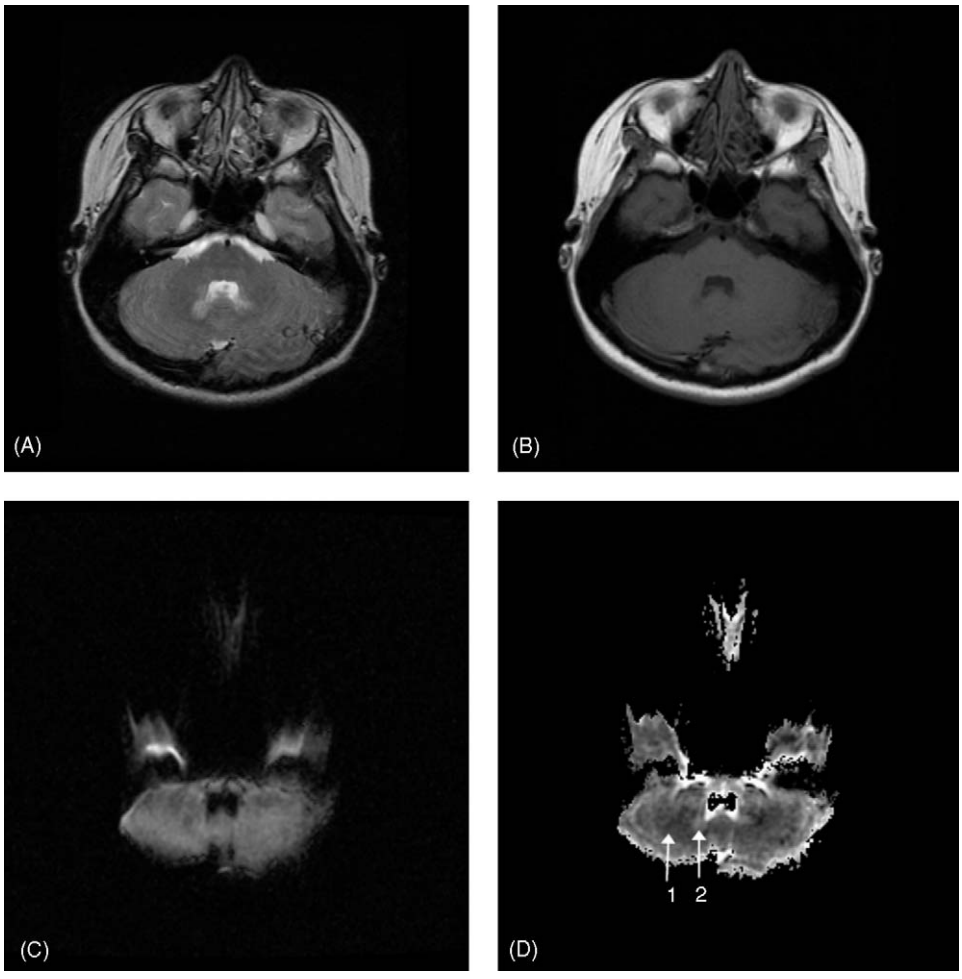

Fig. 1. Initial MRI obtained on day 1. (A) Axial fast spin-echo T2-weighted image shows hyperintense lesions around the fourth ventricle and the cerebellar hemispheres with the dentate nuclei and the deep white matter involved. (B) Axial T1-weighted image does not demonstrate gadolinium enhancement on the corresponding section. (C) DWI shows no obvious diffusion restriction on the corresponding section. (D) The corresponding ADC value of the left cerebellar peduncle is increased (arrow 2—average: 9.01×10^{-3} mm^2/s; S.D.: 1.91×10^{-3} mm^2/s) as compared with the normal cerebellar tissue (arrow 1—average: 7.30×10^{-3} mm^2/s; S.D.: 1.03×10^{-3} mm^2/s).

eralized hyperreflexia was found, Babinski response could not be elicited and her muscle tone was generally normal. There was no involuntary movement nor palatal myoclonus. MRI on admission (day 1) showed hyperintensities in the T2-weighted images and increased values of apparent diffusion coefficient (ADC) around the fourth ventricle and bilaterally in the nearby deep white matter of the cerebellar hemispheres (Fig. 1A and D). T1-weighted images after gadolinium administration and diffusion-weighted imaging (DWI) scans did not reveal any change in signal intensity (Fig. 1B and C). Cerebrospinal fluid examination showed cell counts, protein and glucose values to be normal. Except for a low potassium level (2.9 mequiv/dl), which might have been the consequence of the patient's excessive vomiting, all other biochemistry studies including liver and renal functions were normal. The patient's hemogram, C-reactive protein (CRP) level, chest X-rays (CXR), and urinalysis results were also normal.

Acute disseminated encephalomyelitis was our initial tentative impression because the brain MRI revealed multiple non-enhanced white matter lesions and no other possible etiologies were determined at that time although the imaging findings were not very typical; therefore, the patient was given pulse therapy with methylprednisolone 1000 mg/day for 3 days.

During the course of the hospitalization the patient vomited so frequently and intensely that we had to introduce tube feedings and IV normal saline and glucose water to sustain her nutrition and hydration. However, no vitamin supplement was given in the first 3 days. All of these we later considered might have worsened her condition. On the fourth day of the admission (day 4), she suddenly developed hyperventilation (respiratory rate up to 40 min^{-1}) and became lethargic. Ventilator support was needed because of respiratory failure. She had leukocytosis (white blood cell count, 16,600/mm^3) and an elevated CRP level (8.3 mg/dl), but CXRs, routine urinalysis, and abdominal sonography did not show active infection. Investigations were performed to detect infections due to bacteria, viruses, fungi, tuberculosis,