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Case report

Neuromyelitis optica and Wernicke encephalopathy share the similar imagings, any correlations?

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Abstract

Two cases with similar brain imaging findings were reported. Both brain MRIs were characterized by abnormal signal intensities in thalamus, surrounding third ventricle, periaqueductal areas and posteromedial thalami. Through clinical observation and a series of auxiliary examinations, one patient was diagnosed as Wernicke encephalopathy (WE) and the other as neuromyelitis optica (NMO). Although WE and NMO are totally different diseases entity, but similar lesions area aroused hypothesis that potential correlations may exist between these two diseases. Astrocytes may be the target cells connecting these two diseases. Potential benefit of thiamine supplementary therapy is suggested for NMO treatment.

Keywords: Wernicke encephalopathy (WE); Neuromyelitis optica(NMO); Imagings; Astrocytes

1. Introduction

Neuromyelitis optica (NMO) and Wernicke encephalopathy (WE) are totally different diseases entity. NMO is an autoimmune disease which NMO-IgG immune response leads to demyelination. The antibodies target aquaporin-4 in the cell membranes of astrocytes which acts as a channel for the transport of water across the cell membrane. While WE results from deficiency of vitamin B1 (thiamine hydrochloride) which helps to breakdown glucose. Specifically, it acts as an essential coenzyme in the TCA cycle and the pentose phosphate shunt. Human body only has 2–3 weeks of thiamine reserves, which are readily exhausted without intake, or if depletion occurs rapidly, such as in chronic inflammatory states or in diabetes. From a hyperemesis gravidarum patient, unexpectedly we found that the cranial MRIs of these two diseases are quite similar. The common lesions would involve thalamus, hypothalamus, peri-third ventricle and fourth ventricle, periaqueductal, brain stem and cerebellum. Their initial presentations are easy misdiagnosis, including visual changes, intractable vomiting. Are there any correlations between them two?

2. Case report

2.1. Case 1

A 24-year-old woman was admitted for blurred vision for 9 h and convulsion for 1 h. Nine hours before admission, blurred vision occurred abruptly and aggravated gradually and lost vision after a dispute with others. Sudden convulsions occurred during the patient's consultation. Two times of outbreaks occurred in 2–3 min. The past medical history implied the patient now is 3 months pregnant with nausea and vomiting for more than a month. Physical examination upon

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admission indicated no light perception was found in both eyes. Laboratory examinations showed blood glucose 10.7 mmol/L, K^+3.5 mmol/L, Na^+132 mmol/L, Cl^- 9.5 mmol/L; ENA and ANCA spectrum, Widal–Pei test, syphilis and HIV blood test were all negative. Serum thiamine was 51 nM/L (normal > 70). Head CT scan revealed suspicious flake-like hypoattenuated lesions in the left occipital lobe, and MRI showed slightly longer T1 and T2 signal intensities in bilateral occipital cortex, and slightly longer T1 and T2-weighted sheeted signal intensities symmetrically distributed surrounding aqueduct and third ventricle (Fig. 1). Lumbar puncture showed cerebral spinal fluid pressure of 100mmH_2O, protein 1+, cells 1+, sugar 3.4 mmol/L, total protein 0.3 g/L, Cl-127 mmol/L, and LDH 64U/L. Acid-resistent bacilli, fungi, and neocryptococcosis in cerebral spinal fluid were all negative. AQP-4 antibody was also negative.

Wernicke Encephalopathy was diagnosed. After vitamin B1 supplementing for 5 days, her neurologic examination showed a slight recovery of conscious, vision acuity was 1 m/FC (finger counting). 7 days later, visual acuity and visual field of both eyes were normal. The patient's condition was relatively stable, but her emotional expressions were indifferent and unresponsive. Ten days later, the patient's condition improved gradually, but with memory impaired. After 20 days continuous treatment, her consciousness and cognitive function were resolved completely. A follow-up cranial MRI scanning 2 weeks later FLAIR showed high punctiform and banding-shaped signal in left parietal lobe and slightly higher signal intensity in surrounding cerebral aqueduct, the lesion reduced significantly compared to prior examinations. Laboratory results were normal. Vaginal delivery was induced after admission into obstetric department. One year after discharge, a telephone call follow-up confirmed that her blood pressure and visual acuity were normal and no seizures had reoccurred.

2.2. Case 2

A-21-year-old woman was admitted for complaint of vertigo and vomiting for 3 days and drowsiness for 1 day. Three days before admission, vertigo occurred abruptly after morning rises, accompanied with nausea and obviously vomiting. The patient did not have headache, fever, physical activity disturbance and limb paresthesia. She was not given a diagnosis and treatment. One day before admission, she was

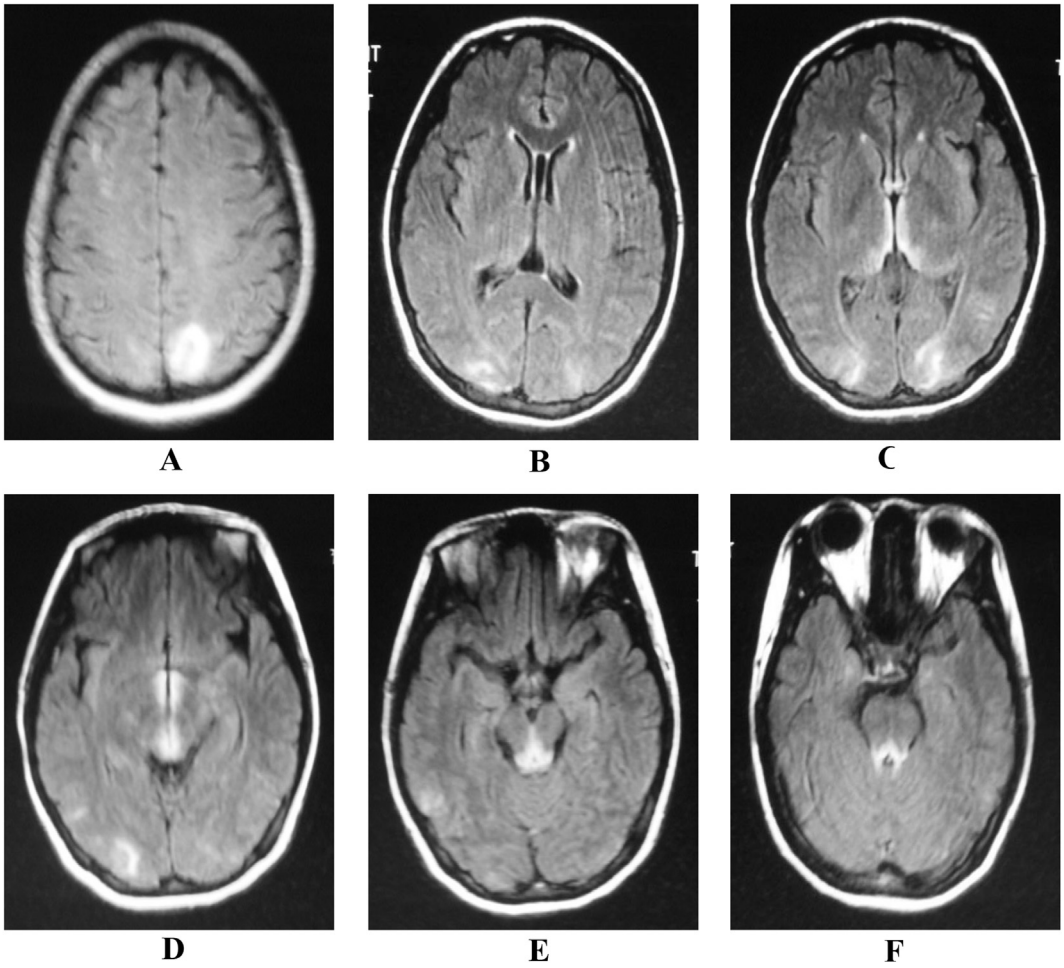

Fig. 1. Axial FLAIR images showing asymmetric hyperintensities in bilateral occipital lobe (A, B), surrounding third ventricle and posteromedial thalami (C, D), periaqueductal areas (E, F).