Wernicke Encephalopathy in a Patient with Pulmonary and Abdominal Tuberculosis

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Wernicke encephalopathy (WE) is an acute or subacute neurological syndrome caused by thiamine (Vitamin B1) deficiency and is usually underestimated in clinical practice. WE is suspected in merely about 6% of non-alcoholic patients and one-third of alcoholic patients.[1] Non-alcoholic causes include gastrointestinal surgery and disease, malnutrition, cancer and chemotherapeutic treatments, and long-term parenteral nutrition.[2] However, few cases were reported about WE in patients with tuberculosis. Early diagnosis and medication are vital for the prognosis of this disease.

Written informed consent was obtained from the legally authorized representative of the patient for the publication of this case report.

A 46-year-old man was admitted to hospital for cough, abdominal pain, and blurred vision. Five months before this visit, he had the onset of cough and fever. One month before the admission, he presented abdominal pain, nausea, vomit, fever, and melena. Mesenteric biopsy confirmed the diagnosis of abdominal tuberculosis. Four days before this visit, he presented blurred vision. The reason for blurred vision remained unclear. Medical history showed gastrointestinal bleeding, and others were unremarkable. The patient had a history of chronic alcoholism consumption for 20 years but stopped drinking 8 years ago. At admission, physical examination showed that he had slight mental confusion. However, other neurological examinations were unremarkable. Blood tests showed erythrocyte sedimentation rate (30.0 mm/h), C-reactive protein (23.3 mg/L). The results of routine hematological tests and arterial blood gas analysis were unremarkable. Tuberculosis infected T cells gamma interferon release test result was positive. The chest and abdominal computed tomography scan results were suggestive of pulmonary and abdominal tuberculosis. Vision acuity test showed hand movement in the right eye and CF/15 mm in the left eye. The test of anterior segment of eyeball was unremarkable. Ophthalmic fundus examination showed the disc was hyperemia and edema, and the margin was blurring with flame-shaped hemorrhages. Optic neuritis or encephalitis caused by intracranial lesions was suspected.

After admission, he was given anti-tuberculosis medication and parenteral nutrition. At the 4th day after admission, the patient suddenly developed headache and hematemesis. Brain computed tomography scan result was unremarkable. Brain magnetic resonance imaging (MRI) scan was not performed due to his noncooperation. The pressure of cerebrospinal fluid was 58 mmH_2O (1 mmH_2O = 0.0098 kPa). The routine test and biochemical indicators of cerebrospinal fluid were unremarkable. The result of occult blood test of vomit was positive. Fecal occult blood test result was negative. Gastrointestinal bleeding was considered. The reason for blurred vision and headache still remained unclear. Symptomatic and supportive treatments were administered to him, all of which were given through parenteral route. At the 7th day after admission, his symptoms significantly got worse. At the same time, he developed diplopia and dysphoria. Physical examination showed nystagmus, unsteady gait, weakness of extremities, and negative pathological reflex. Arterial blood gas analysis result was suggestive of Type I respiratory failure. Other laboratory data were as follows: sodium ions (130.5 mmol/L), chloride ion (95.4 mmol/L), blood ammonia (27.0 μmol/L, reference range, 9.0–33.0 μmol/L), 25-OH-VD (20.5 nmol/L, reference range, 47.7–144.0 nmol/L), creatine kinase (52 IU/L), lactate dehydrogenase (118 IU/L),

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and hydroxybutyrate dehydrogenase (97 IU/L). The results of brain MRI scan showed symmetric increased T1 and T2 signals in the medial dorsal thalamus, hypothalamus, and periaqueductal region [Figure 1a]. WE or encephalitis was suspected. WE was finally diagnosed on the basis of his clinical manifestations and brain MRI scan results after a consultation to a neurologist. Therefore, he was immediately given intramuscular Vitamin B1 with 100 mg twice a day for 12 days. At the following day, his mental confusion, blurred vision, and nystagmus significantly improved. However, he developed anterograde amnesia. At the 7th day after administering Vitamin B1, symptoms including diplopia, unsteady gait, and memory deterioration were resolved. A follow-up brain MRI scan results showed significantly improved [Figure 1b]. Finally, he was given oral Vitamin B1 for a year.

WE is a clinical emergency resulting from hypovitaminosis. It is usually considered as a metabolic disease caused by chronic alcoholism. In recent years, more and more cases about WE were diagnosed in non-drinkers. In our case, tuberculosis and subsequent incomplete ileus decreased the absorption of nutrients. Finally, the patient appeared blurred vision due to long-term lack of absorption of Vitamin B1. We reviewed many English literatures related to this disease and found that there were a few cases about WE associated with tuberculosis.

The typical triad of symptoms in WE includes mental disorder, cerebellar ataxia, and ophthalmoplegia. However, which were reported in only about 8% of patients in clinical practice.[1] It is difficult for clinical practitioners to distinguish it from other neuropsychiatric disorders, especially in pediatric patients for their atypical clinical symptoms[3] and in patients with liver failure due to the difficulty of distinguishing WE from hepatic encephalopathy.[4] The patient in our case gradually developed blurred vision, mental confusion and unsteady gait, which made it difficult for a non-neurologist to diagnose early. At the beginning, tuberculous meningitis (TBM) was considered. However, due to lack of the typical cerebrospinal fluid profile (a predominance of lymphocytes, low glucose, and elevated protein) and MRI signs (tuberculomas, hydrocephalus, infarction, pachymeningitis, etc.), TBM was excluded subsequently.

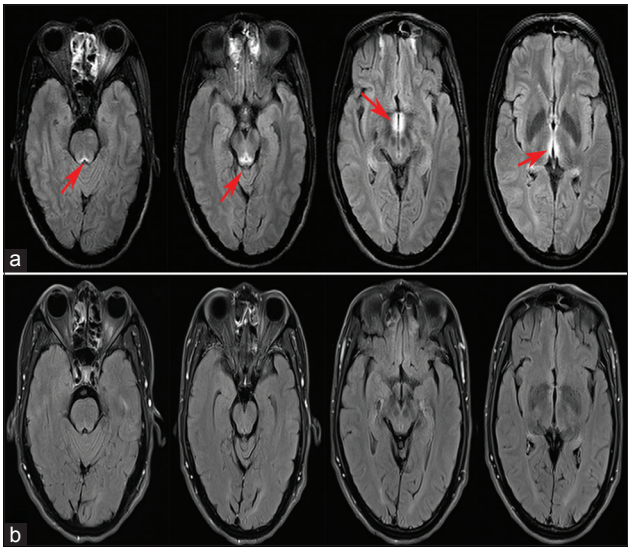

Figure 1: High signal lesions in brain magnetic resonance imaging results of the patient before receiving thiamine therapy, as indicated by the red arrows (a). Magnetic resonance imaging results of the patient after receiving thiamine therapy for 3 months (b).

Considering WE is a neuropsychiatric disorder, clinical practitioners may routinely examine brain spinal fluid and prescribe brain computed tomography scan. However, these results may be unremarkable in the early stage. Brain MRI is more powerful to support the diagnosis of acute WE. Typically, MRI scan results are symmetrical lesions in the mammillary bodies, thalami, tectal plate, and periaqueductal area which showed increased T2 signal in these sites.[5] In our case, brain computed tomography scan result was unremarkable. Brain MRI scan was finally performed and showed typical imaging results.

Delayed diagnosis and treatment can lead to progression of this disease. Ultimately, the patient may develop infantile beriberi, shock, Wernicke–Korsakoff syndrome, or even death. The EFNS guidelines recommend that thiamine should be given 200 mg three times a day and the best route is intravenous instead of intramuscular.[1] The patient in our case received intramuscular Vitamin B1 with 100 mg twice daily for 12 days. His mental confusion, blurred vision, and nystagmus significantly improved although we just prescribed a small dose of Vitamin B1.

WE was diagnosed in a tuberculous patient who had no history of alcohol consumption in recent years. It is difficult for us to early diagnose WE. Carefully collection of medical history and cooperation with the department of neurology is necessary. Although our patient got rapid relief and had a good prognosis after a small dose of thiamine, we still need more researches to get specific and better recommendation for dosage, route, and duration of Vitamin B1.

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Conflicts of interest

There are no conflicts of interest.

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