Wernicke encephalopathy in a patient with liver failure
Clinical case report
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Abstract
Early recognition and diagnosis of Wernicke encephalopathy is pivotal for the prognosis of this medical emergency, especially in patients with liver failure which predisposes individuals to develop hepatic encephalopathy. For these patients, distinguishing between hepatic encephalopathy and Wernicke encephalopathy is a challenge in real-world clinical practice.

A male patient with 21-year medical history of liver cirrhosis presented diarrhea and ascites. One month before this visit, he was noted to have poor appetite and progressive fatigue. After admission, although several major symptoms, including diarrhea, ascites, hyponatremia, and hypoproteinemia, were greatly improved through appropriate treatments, his laboratory indicators were not changed much. His appetite was not reversed at discharge. On the 5th day after discharge, the patient suddenly became reluctant to speak and did not remember the recent happenings. Simultaneously, unsteady gait and strabismus occurred. On the basis of clinical manifestations and brain magnetic resonance imaging scan results, the patient was diagnosed as Wernicke encephalopathy and these relative symptoms were resolved after intravenous vitamin B1.

To our knowledge, this is the second case report of Wernicke encephalopathy developing in a critically ill cirrhotic patient without hepatocellular carcinoma or operative intervention. Wernicke encephalopathy may be underdiagnosed in these patients and this case raises physicians’ awareness of its possible onset.

Abbreviations: HBV = hepatitis B virus, MRI = magnetic resonance imaging.

Keywords: differential diagnosis, liver failure, Wernicke encephalopathy

1. Introduction
Wernicke encephalopathy is an acute neurological disorder resulting from thiamine (vitamin B1) deficiency and occurs frequently in patients with compromised absorption, increased metabolism, or increased carbohydrate intake.[1] Delay in its recognition and treatment may lead to significant morbidity, irreversible neurological damage, or even death.[2] Several case reports describe Wernicke encephalopathy as a rare clinical condition; however, it is suggested, by guidelines from the European Federation of Neurological Societies, that Wernicke encephalopathy is not a rare disorder, but rather a rare diagnosis.[3] Here, we present a critically ill patient with decompensated liver cirrhosis who developed Wernicke encephalopathy.

2. Consent
Written informed consent was obtained from the patient for the publication of this case report. A copy of the written consent is available for review by the editor of this journal.

3. Case report
A 61-year-old man was admitted to the hospital for diarrhea and newly onset ascites. The patient had a 21-year medical history of liver cirrhosis associated with hepatitis B virus (HBV) infection and did not have a habit of alcohol consumption. His serum HBV DNA was not detected 15 years ago. After that, he did not receive any relative examinations or treatments due to economic reasons. One month before the current admission, he was noted to have poor appetite and progressive fatigue. Admission laboratory evaluation yielded the following: white blood cell count, 1.71 × 109; neutrophil percentage, 83.0%; red blood cell count, 3.60 × 1012; platelet count, 35 × 109; hemoglobin, 99 g/L; international normalized ratio, 1.51; blood ammonia, 24.7 μmol/L (reference range, 0–40 μmol/L); serum HBV DNA, <40 IU/L; serum alanine aminotransferase, 29 IU/L; serum aspartate aminotransferase, 40 IU/L; serum alkaline phosphatase, 122 IU/L; serum gamma glutamyl transferase, 42 IU/L; serum total bilirubin, 27.2 μmol/L (reference range, 0.3–1.9 μmol/L); serum albumin, 26 g/L (reference range, 35–55 g/L); serum creatinine, 92 μmol/L (reference range, 62–115 μmol/L); serum sodium, 130 mmol/L; and serum potassium, 4.0 mmol/L (reference range, 3.5–5.0 mmol/L).
A diagnosis of Wernicke encephalopathy should be considered in any patient presenting two of these symptoms.

Figure 1. Caine criteria for diagnosis of Wernicke encephalopathy.

Diagnostic difficulties in the differential and confirmed diagnosis. It is reported that the diagnosis is confirmed in 0.4% to 2.8% of autopsies, yet may be overlooked in 68% of patients with alcoholism and 94% of patients without alcoholism. The Caine criteria for diagnosis of Wernicke encephalopathy have been demonstrated to be optimal and are summarized in Fig. 1. Till now, only 4 Wernicke encephalopathy cases associated with liver diseases (Table 1) have been reported in the English literature.

In this case, insufficient dietary intake (poor appetite) and abnormal glucose metabolism caused by impaired liver function contribute to the development of Wernicke encephalopathy. For patients with underlying liver cirrhosis, distinguishing between hepatic encephalopathy and Wernicke encephalopathy sometimes becomes a tough problem, especially in an emergency. As is known, hepatic encephalopathy, which may be associated with the increased ammonia or endozepine and is characterized by a wide spectrum of psychiatric and behavioral disturbances and motor disorders, is a common complication of liver diseases.

Table 1

| Year  | Authors                  | Underlying liver diseases in cases reported                      |
|-------|--------------------------|-------------------------------------------------------------------|
| 2015  | Souladopoulos et al      | Acute liver failure related to hepatitis B virus infection         |
| 2010  | Zhang et al              | Spleenectomy in the setting of liver cirrhosis                    |
| 2009  | Shin et al               | Liver transplantation                                             |
| 2005  | Onishi et al             | Hepatocellular carcinoma with liver cirrhosis                     |

As seen in the cases reported, Wernicke encephalopathy often starts with symptoms such as confusion, ataxia, and ophthalmoplegia. In this case, the patient also presented with memory loss, unsteady gait, and strabismus. Although Wernicke encephalopathy is a disease of known etiology, the above triad is not always present, which leads to diagnostic difficulties in the differential and confirmed diagnosis. It is reported that the diagnosis is confirmed in 0.4% to 2.8% of autopsies, yet may be overlooked in 68% of patients with alcoholism and 94% of patients without alcoholism. The Caine criteria for diagnosis of Wernicke encephalopathy have been demonstrated to be optimal and are summarized in Fig. 1.

4. Discussion

Wernicke encephalopathy is a clinical emergency. Classically, its clinical triad consisted of confusion, ataxia, and ophthalmoplegia. Although Wernicke encephalopathy is a disease of known etiology, the above triad is not always present, which leads to diagnostic difficulties in the differential and confirmed diagnosis. It is reported that the diagnosis is confirmed in 0.4% to 2.8% of autopsies, yet may be overlooked in 68% of patients with alcoholism and 94% of patients without alcoholism. The Caine criteria for diagnosis of Wernicke encephalopathy have been demonstrated to be optimal and are summarized in Fig. 1.

Till now, only 4 Wernicke encephalopathy cases associated with liver diseases (Table 1) have been reported in the English literature. In this case, insufficient dietary intake (poor appetite) and abnormal glucose metabolism caused by impaired liver function contribute to the development of Wernicke encephalopathy. For patients with underlying liver cirrhosis, distinguishing between hepatic encephalopathy and Wernicke encephalopathy sometimes becomes a tough problem, especially in an emergency. As is known, hepatic encephalopathy, which may be associated with the increased ammonia or endozepine and is characterized by a wide spectrum of psychiatric and behavioral disturbances and motor disorders, is a common complication of liver diseases.

First, an absolute majority of hepatologists are not familiar with Wernicke encephalopathy. Second, no mental alteration is usual in cases with hepatic encephalopathy. Factually, distinguishing between hepatic encephalopathy and Wernicke encephalopathy in real-world clinical practice is very difficult. First, an absolute majority of hepatologists are not familiar with Wernicke encephalopathy. Second, no mental alteration is unique in the 2 disorders. Third, distinctions in the neuroimaging findings between the 2 disorders are not easily identified. Like MRI findings in Wernicke encephalopathy, MRI scans in several cases with hepatic encephalopathy also present with symmetrical T2 high signal intensities in the bilateral cerebellar hemispheres and brachium pontis. Regarding the present case, some physicians who participated in the discussion insisted on their diagnosis of hepatic encephalopathy and they did not give up their opinions until the condition of the patient was reversed by intravenous vitamin B1. Therefore, when difficulties exist in distinguishing between hepatic encephalopathy and Wernicke encephalopathy, intravenous vitamin B1 (200 mg) can be considered as a discriminative method or a preemptive treatment.
the risk of incident Wernicke encephalopathy in critically ill patients with liver diseases.

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