**Case report**

**Non-alcoholic Wernicke’s encephalopathy as a cause of profound shock after abdominal surgery**

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**Learning Point for Clinicians**

Wernicke’s encephalopathy (WE) should be taken into consideration in surgical patients with prolonged parenteral therapy whom develop refractory hypotension and neurological deficit. Total parenteral nutrition with inadequate thiamine supplement may result in WE.

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**Case report**

A 31-year-old man was admitted to a local hospital 1 month ago for an operation of enterolysis. He had a history of penetrating abdominal injury with a rupture of his small intestine 8 years ago and has experienced recurrent ileus ever since the surgery. He is a non-drinker and denied any systemic disease. Due to persistent vomiting and abdominal pain after surgery, total parenteral nutrition was given, which is also useful for gastric decompression. One week prior to being admitted to our hospital, he began to have blurred vision, hearing impairment and progressive deterioration of consciousness. He was then transferred to our hospital. On examination, he was comatose (Glasgow Coma Score 2T), afebrile, with a blood pressure of 63/33 mmHg, heart rate of 94 beats/min and respiratory rate of 16 breaths/min. Pupils were 2 mm without light reflex, eyeballs were fixed, corneal reflex and Doll’s eye sign were absent and muscle power of the four limbs was grade 0. Deep tendon reflex and plantar reflex could not be elicited. There was no neck stiffness or edema of extremities. Pertinent laboratory values on admission disclosed no significant abnormalities except for total protein 4.9 g/dl (7.1–8.7 g/dl) and albumin 2.9 g/dl (4.3–5.0 g/dl). Cerebrospinal fluid analysis and other surveys of infections were unremarkable. Transthoracic echocardiography showed normal systolic function of left ventricle with an ejection fraction of 65%.

Magnetic resonance imaging (MRI) of brain showed bilateral symmetrical hyperintensity in the frontal cortices, dorsal putamen, thalami, fornices, mamillary bodies, periventricular regions of the third ventricle, periaqueductal area, tectum of the midbrain and dorsal pons and medulla (Figure 1). Corresponding changes of diffusion-weighted imaging and apparent diffusion coefficient maps were found in some areas. The clinical and imaging findings favored the diagnosis of non-alcoholic WE. Shock was unresponsive to vigorous fluid replacement and inotropic agents, but dramatically improved in 12 h after intravenous thiamine supplement. The patient’s vital signs stabilized gradually; however, poor neurological recovery was observed. He remained comatose and was only found to have mild withdrawal of limbs to painful stimuli 2 months after treatment.

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Discussion

WE is a serious, potentially fatal acute or subacute neurological disorder caused by thiamine (Vitamin B1) deficiency. Thiamine is important for cell membranes to maintain the osmotic gradients and in its biologically active form, thiamine pyrophosphate, is a co-factor in intermediate carbohydrate metabolism. The body’s reserve of thiamine is ~30 mg and is sufficient to sustain the normal level for ~18 days. A deficiency of thiamine may lead to beriberi, Korsakoff’s syndrome and WE.

WE is characterized by the classical triad of ophthalmoplegia, ataxia and mental status changes. However, the classical triad is found in only 16% affected patients in a necropsy study of 131 cases with Wernicke–Korsakoff syndrome. In addition to alcoholic patients, WE may also be associated with other conditions, such as prolonged parenteral therapy, without adequate thiamine supplementation, gastric and intestinal surgery, protracted vomiting and in any clinical condition in which thiamine is not adequately ingested or absorbed. In fact, WE is clinically underdiagnosed, particularly when patients present with atypical clinical symptoms, such as shock, hypothermia or hearing loss, or have no history of alcohol consumption.

MRI is currently considered the most valuable tool in the diagnosis of WE. Typical MRI findings show symmetrical lesions of the medial thalami, periventricular regions of the third ventricle, mammillary bodies and the periaqueductal gray matter. Atypical locations of lesions, such as splenium, vermis of cerebellum, cranial nerve nuclei, caudate nuclei, cerebral cortex, dentate nuclei and red nuclei have been reported. Chu et al. reported two patients with WE, showing disappearance of signal abnormalities of MRI after thiamine therapy and supposed the mechanism of reversible cytotoxic edema caused by thiamine deficiency. In our case, the combinations of typical and atypical imaging findings enclose the full blown pictures of WE.

Hypothalamic lesions and/or coexistent cardiovascular berberi can lead to dysfunction of autonomic system with resultant hypotension, tachycardia and hypothermia—which are uncommon presenting symptoms of WE. The pathogenesis might be due to decrease of efferent sympathetic outflow. In addition, cortical and thalamic involvement may be indicative of a poor neurological

Figure 1. Axial T2 fluid attenuated inversion recovery MRI at the levels of high convexity (a), basal ganglia (b), midbrain (c) and pons (d) show bilateral symmetrical hyperintensity in the frontal cortices, thalami, mammalian bodies, dorsal putamen, periventricular regions of the third ventricle, periaqueductal area and dorsal pons. The abnormal areas show restricted water diffusion on corresponding diffusion-weighted imaging (e-h) and apparent diffusion coefficient maps (i-l).
outcome.\textsuperscript{7} Cognat \textit{et al}.\textsuperscript{8} described a 42-year-old HIV-positive woman who developed severe dysautonomia and classical manifestations of WE after 4 days of recurrent episodes of vomiting. Treatment with parenteral thiamine induced dramatic improvement within a few days. Wang \textit{et al}.\textsuperscript{9} reported a 57-year-old man receiving total parenteral nutrition after radical distal gastrectomy, developed refractory hypotension before the appearance of characteristic signs of WE. They suggested that severe lesions of the bilateral thalami may account for the hypotension. In our patient, the etiology of WE was due to prolonged parenteral therapy, in which the demand for increased basal needs of thiamine was unmet. Although shock was rapidly corrected by thiamine supplement, the neurological deficit sustained. Early identification and timely treatment of WE is crucial because it is reversible and curable. We highlight that WE should be taken into consideration in surgical patients with prolonged parenteral therapy whom develop refractory hypotension and neurological deficit.

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\textbf{References}


