The Journal of Emergency Medicine, Vol. 49, No. 4, pp. 471–474, 2015

0736-4679/$ - see front matter

ELSEVIER

http://dx.doi.org/10.1016/j.jemermed.2015.03.022

Selected Topics: Neurological Emergencies

A THERAPEUTIC FAST FOR LYMPHOMA RESULTING IN WERNICKE ENCEPHALOPATHY

Jared Brazg, MD, Amber Ruest, MD, Susan Law, DO, and Dimitry Bosoy, MD

Maimonides Medical Center, Brooklyn, New York
Reprint Address: Jared Brazg, MD, Maimonides Medical Center, 4802 10th Avenue, Brooklyn, NY 11219

☐ **Abstract—Background: Wernicke encephalopathy is an acute neurological emergency caused by thiamine (vitamin B1) deficiency. The syndrome is associated with a significant morbidity and mortality, and prompt recognition and treatment of the syndrome in the emergency department (ED) is essential to improving patient outcomes. Numerous factors and clinical settings have been identified that predispose a patient to thiamine deficiency and subsequent Wernicke encephalopathy. Case Report: We present the rare case of a 42-year-old man with a recent diagnosis of non-Hodgkin lymphoma who opted against chemotherapy in favor of a 60-day therapeutic water-only fast. On day 53 of his fast, the patient arrived to our ED in a coma and respiratory failure. Moments after the administration of thiamine, the patient's mental status and respiratory status improved significantly. Prior to admission and transport to the medical intensive care unit, the patient was awake, alert, and following basic commands. He was ultimately diagnosed with Wernicke encephalopathy. Why Should An Emergency Physician Be Aware of This?: With the increasing incidence of patients choosing alternative medical therapies to treat a variety of diseases, numerous electrolyte, metabolic, and nutritional disorders are becoming increasingly more common in the ED setting. In some cases, patients may choose a therapeutic fast in an effort to combat a malignancy; the danger being that patients with cancers such as lymphoma are already at risk for thiamine deficiency as a result of the increased thiamine consumption associated with rapid cellular turnover. Wernicke encephalopathy is a life-threatening neurological emergency, and the emergency physician must be aware of the numerous predisposing factors to the condition, as early identification and treatment improves patient outcomes.** © **2015 Elsevier Inc.**

☐ **Keywords—Wernicke encephalopathy; neurological emergencies; thiamine deficiency; non-Hodgkin lymphoma; therapeutic fast**

INTRODUCTION

Wernicke encephalopathy is classically characterized by ophthalmoplegia, mental-status changes, and ataxia, although this triad is seen in only 16% of patients (1,2). Studies show that 82% of patients with Wernicke encephalopathy have mental status changes that may include confusion or agitation, hallucinations, or behavioral disturbances, oftentimes mimicking an acute psychotic disorder. Uncommon signs and symptoms on presentation include stupor, tachycardia, hypotension, hypothermia, visual disturbances, and seizures. Late findings include hyperthermia, increased muscle tone, choreic dyskinesia, and coma. The relatively nonspecific clinical presentation of the disease leads to a high rate of incorrect or delayed diagnosis of the disorder. A patient with Wernicke encephalopathy may present similarly to an intoxicated patient, and therefore pose an additional challenge for an emergency physician to make the diagnosis (3). Measurements of erythrocyte thiamine transketolase both prior to and after the addition of thiamine pyrophosphate have been used to diagnose thiamine deficiency, however, this test is not

Received: 14 October 2014; Final submission received: 11 March 2015;
Accepted: 24 March 2015

readily available in the emergency department (ED) (4). A serum thiamine or thiamine pyrophosphate level in serum or whole blood can also be measured by high-performance liquid chromatography (5). The sensitivity and specificity of these blood tests in symptomatic patients are unclear, as blood level may not accurately reflect brain thiamine level. Thus, a normal blood level does not always exclude the possibility of Wernicke encephalopathy.

CASE REPORT

A 42-year-old man with a recent diagnosis of non-Hodgkin lymphoma (stage 2) was brought in by ambulance to our ED with coma and respiratory failure. His wife provided the patient's history. Two months prior, he underwent a biopsy of an abdominal mass and was diagnosed with non-Hodgkin lymphoma. After meeting with an oncologist, he opted against chemotherapy and decided instead on a therapeutic fast consisting only of distilled water. The patient arrived to our ED on day 53 of his 60-day fast. Over the past few days, he had become increasingly confused, fatigued, and unable to get out of bed. An episode of apnea and cyanosis an hour prior to his arrival to the ED had ultimately prompted his wife to call an ambulance. She denied witnessing any seizure-like activity, and there was no history of any fevers, chills, headaches, abdominal pain, nausea, vomiting, or diarrhea. There was no known illicit drug use or alcohol abuse. He was not taking supplements, and she reported a 34-kg weight loss since the beginning of the fast.

The patient's heart rate was 158 beats/min, respiratory rate 24 breaths/min, and temperature was 38°C (100.4°F). Blood pressure and oxygen saturation on 100% nonrebreather mask were within normal limits. Point-of-care blood glucose level was 166 mg/dL. He appeared cachectic, and was unresponsive and unarousable. The head was normocephalic and atraumatic. Pupils were constricted to 2 mm with a fixed stare. Corneal reflex was intact, but there was no gag reflex. There was no oculocephalic reflex upon dolling. The patient did not withdraw to pain or respond to sternal rub. His breathing was labored, and breath sounds were clear bilaterally. On abdominal examination, a small palpable mass was felt in the right lower quadrant. Skin was warm and well perfused.

Electrocardiogram showed sinus tachycardia at a rate of 118 beats/min. Basic laboratory studies were unremarkable, with a normal white blood cell count of 8.8 K/UL (reference range 4.8–10.8), neutrophils 74.8% (reference range 37.9–70.5%), and sodium level of 143 mmol/L (reference range 133–146). Magnesium was 2.8 (reference range 1.93–2.45). Phosphorus was elevated to 7.3 (reference range 2.7–4.9). Urine toxicology panel, blood alcohol, salicylate, and acetaminophen levels were all negative. Blood gas revealed a pH of 7.27, with a PCO_2 of 77 mm Hg and HCO_3 of 36 mmol/L, consistent with a primary respiratory acidosis with metabolic compensation. Vitamins B1, B6, B12, and folate levels were also sent.

Prior to imaging, the patient was intubated for airway protection and respiratory failure. After intubation, the patient went into a narrow complex supraventricular tachycardia and was converted to normal sinus rhythm with 6 mg of adenosine.

Head computed tomography (CT) scan without contrast demonstrated symmetric hypodense lesions in the medial and anterior portion of the thalami, consistent with acute nonhemorrhagic infarcts, Wernicke encephalopathy, or demyelination such as acute disseminated encephalomyelitis. Magnetic resonance imaging (MRI) of the brain with and without intravenous (i.v.) contrast was also ordered.

Prior to MRI, and with a high level of suspicion for Wernicke encephalopathy, the patient received 200 mg i.v. thiamine, moments after which the patient was noted to be opening his eyes and following commands. Repeat neurological examination demonstrated absence of gaze in the horizontal plane. He had intact corneal reflexes and upward gaze (with upbeat nystagmus). Bilateral arms were antigravity, but severely dysmetric when performing finger to nose (Figure 1).

The patient was admitted to the medical intensive care unit. Initial thiamine level was 32 nmol/L (reference range 75–200 nmol/L). He was given 500 mg thiamine i.v. three times daily for 2 days, followed by 500 mg daily for the next 5 days. The patient was extubated on day 4, developed septic shock likely secondary to aspiration pneumonia, requiring vasopressors meropenem and vancomycin. After resolution of septic shock, he was transferred to the medical floor, where he had a portacath placed, and chose to begin chemotherapy. Over the course of his hospital stay, his mental status returned to baseline. The patient's only neurologic deficit was the inability to adduct his right eye to midline. Repeat head CT 19 days after the initial head CT scan showed interval resolution of the medial thalamic and midbrain hypodensity. He was discharged to home on day 25 of his hospital stay (Figure 2).

DISCUSSION

Wernicke encephalopathy can be challenging to diagnose, especially in the ED setting, where we frequently deal with alternative explanations for a patient's symptoms, such as alcohol intoxication, acute psychotic disorders, or other more common encephalopathic diseases. Knowledge of the varied presentations and predisposing clinical scenarios by emergency medicine