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Case Report

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Wernicke Encephalopathy in a Non-Alcoholic Obese Adolescent: A Case Report

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Abstract

We present an uncommon case report of Wernicke encephalopathy in a non-alcoholic obese adolescent. The patient was a 17-year-old obese male with a 4-month history of rapid weight loss (34 kg) and restrictive dieting pattern; he was admitted to general pediatric inpatient unit due to intractable vomiting and abdominal pain for one week. On day 8 of hospitalization, he developed ophthalmoplegia, weakness, and abnormal gait with mild halluciantion and memory impairment. He was eventually diagnosed with Wernicke encephalopathy clinically and by brain magnetic resonance imaging (MRI). Supplementation with thiamine and other nutrition deficient micronutrients began. He showed steady improvement, however his muscle strength did not return to baseline. As such patient was transferred to an inpatient rehabilitation facility. Understanding the risk factors, clinical presentations and brain MRI findings ensure higher chances of accurate diagnosis and appropriate treatment of Wernicke encephalopathy in children.

Introduction

Wernicke encephalopathy (WE) is a life-threatening condition caused by a deficiency in thiamine, also known as Vitamin B1. WE is known to occur mainly in adult patients with heavy alcohol use. However, it is also seen in all age groups with prolonged severe malnutrition, high-calorie malnutrition, and extended fasting [1-4]. WE tends to be more common in low-income countries and is often under diagnosed in high-income countries like the U.S, especially in previously healthy adolescents [5].

WE presents with the classical triad of altered mental status, ataxia, and oculomotor abnormality [2]. Thiamine deficiency can be divided into two separate categories: Wet (cardiovascular) beriberi, dry beriberi and Wernicke-Korsakoff syndrome (nervous) depending on the manifestations [3]. If left untreated, WE can progress to an irreversible neuropsychiatric state of anterograde and retrograde amnesia with confabulation known as Korsakoff syndrome (KS). In KS, patients develop lesions in the diencephalon-hippocampal system contributing to the inability to store new memories [6]. It has been found that thiamine plays a major role in both the Kreb's cycle and the pentose phosphate pathway, explaining why a deficiency in this vitamin has serious systemic consequences [3].

Case Presentation

A 17-year-old non-alcoholic obese male with a 4-month history of rapid weight loss (34 kg) and restrictive dieting pattern who was admitted due to intractable vomiting and abdominal pain for one week. Recent diet included water-fasting for three weeks with transition to small portioned meals for the latter three weeks.

On admission, his lab results were significant for AST 95 u/L, ALT 169 u/L, but low alkaline phosphatase 40 u/L, he had normal complete blood count, coagulation study, and thyroid function. CT abdomen revealed diffusely fatty infiltrated liver. Antiemetics and supportive treatment were started. On day 2 of hospitalization, he still had persistent nausea and multiple episodes of nonbilious nonbloody emesis with flat affect. His abdominal ultrasound ruled out superior mesenteric artery (SMA) syndrome. He also underwent esophagogastroduodenoscopy which showed minimal chronic gastritis with lactase enzyme deficiency from duodenal biopsy. On day 3 of hospitalization, he was fatigued and was unable to walk without support. He continued to

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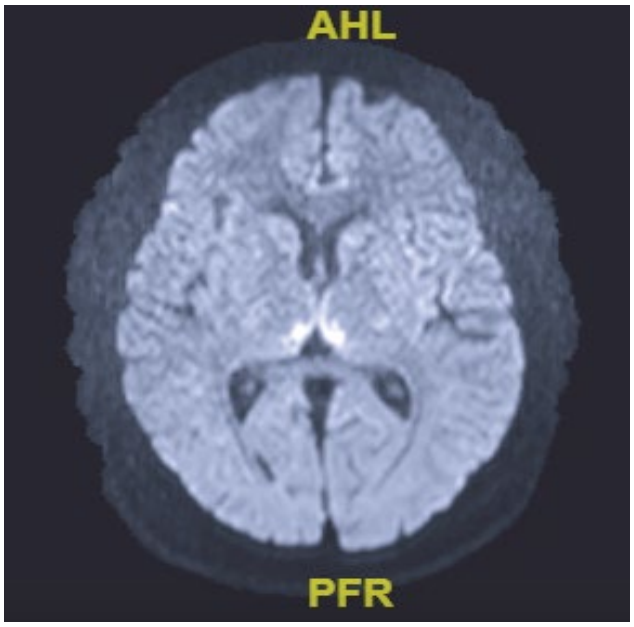

Figure 1: MRI brain on day 8 of hospitalization. There is abnormal increased T2 signal in the periaqueductal gray matter and medial thalami which is consistent with Wernicke encephalopathy.

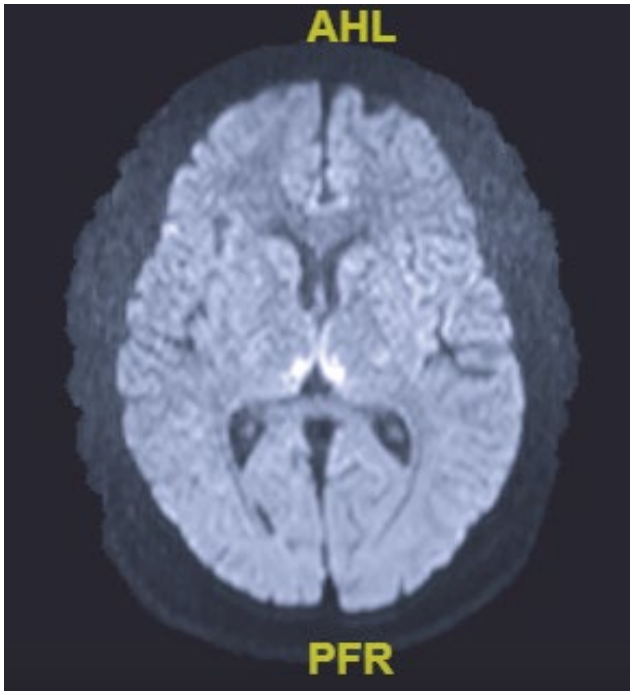

Figure 2: Repeated MRI brain on day 11 of hospitalization. No change in the appearance of the brain when compared with the previous study. The findings are suggestive of Wernicke encephalopathy.

have emesis after feeds, which improved with Phenergan. Day 6 of hospitalization, patient didn't have emesis and he tolerated clear liquid diet. Extensive lab workup of Epstein-Barr virus (EBV) and cytomegalovirus (CMV) were investigated including EBV viral capsid antigen (VCA) IgG positive, EBV nuclear antigen positive, and CMV IgG Ab reactive, EBV VCA IgM and EBV early antigen (EA) were negative.

On day 8 of hospitalization, patient developed hallucinations, dysconjugate gaze, and weakness on legs. Concerning for occupying lesion or pseudotumor cerebri, patient underwent brain MRI and it resulted. Wernicke encephalopathy (Figure 1). He received IV thiamine 500 mg three times a day. Vitamin levels were collected before thiamine administration and it resulted normal vitamins A, B1, B6, and B12 level. Vitamin C, D 25-hydroxy, and E alpha tocopherol were low. On day 11 of hospitalization, he was alert with improved cognition and tolerated oral intake. Patient had repeated brain MRI and it still demonstrated Wernicke encephalopathy (Figure 2), and spine MRI showed degenerative disc desiccation at L4-5 and L5-S1. Patient had slow and steady progress with walking; eventually he was discharged to inpatient rehabilitation center.

Discussion

Suspicion for Wernicke encephalopathy needs to be higher in patients who are exposed to an unbalanced nutrition, recurrent vomiting, and diarrhea. With the condition being generally vague, administration of thiamine is necessary as delay can lead to neurological impairment and even death. The mechanism of neuronal damage in the thalamus, pons, cerebral cortex, basal ganglia and cerebellum is unknown, but have been attributed to mechanisms of energy depletion, increased lactic acid build-up, NMDA mediated excitotoxicity, and oxidative stress [7].

About 58% of cases of Wernicke encephalopathy are missed during routine encounters [8]. Recognizing the risk factors ensure the early diagnosis. This pediatric patient has had a significant history of restrictive dieting pattern based on a three-week water fast with an extended three-week low-calorie diet. In addition, he experienced intractable emesis that accelerated his clinical deterioration.

Including vitamin deficiency like thiamine deficiency in the differential diagnosis is essential when a child presents with severe malnutrition. Four days of thiamine deficiency leads to a decrease in alpha ketoglutarate-dehydrogenase mainly in astrocytes. Astrocytes play a critical role in blood-brain barrier permeability, ionic gradients, and glutamate concentrations [8]. Because of possible irreversible changes that can occur, early detection is important. A revised criteria for Wernicke encephalopathy now includes presence of dietary deficiencies and two signs of the three from the clinical triad (mental status abnormalities, ophthalmoplegia, and in coordination of gait and trunk ataxia) [8]. Normal serum vitamin B1 level cannot exclude WE.

Early detection and treatment is beneficial as thiamine has a high safety profile. Many studies have taken place where a total of more than 300,000 patients were treated without significant adverse reactions [9]. The importance of early detection is due to the low-risk factors of thiamine administration, the reversibility of the disease when treated early and high complication rates when left untreated.

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