Case Report
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A Case Report: Non-Alcoholic Wernicke Encephalopathy

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Abstract

Wernicke encephalopathy (WE) is a life-threatening neurological illness caused by thiamine deficiency. Chronic alcoholism is one of the most important cause of WE, however, WE may develop in other conditions, such as severe malnutrition, hyperemesis gravidarum, prolonged parenteral nutrition, malignancies, bariatric surgery. WE is presenting with classical findings of cerebellar, ocular and confusion. Diagnosis of WE is usually made by history and physical examination. The aim of the treatment is the rapid correction of thiamine deficiency. In this report, we present a patient who with WE had gastrointestinal tract diseases and had undergone gastrointestinal surgeries and discuss predisposing factors as well as diagnostic and therapeutic issues related to this entity.

Keywords: Wernicke encephalopathy, thiamine, delirium, gait ataxia, gastrointestinal tract diseases, emergency room.

Introduction

Wernicke encephalopathy (WE) is a life-threatening neurological illness caused by vitamin B1-thiamine deficiency. WE is an important cause of acute or subacute delirium. Thiamine is a cofactor for several enzymes in the Krebs cycle and the pentose phosphate pathway that plays a central role in cerebral metabolism. Thiamine deficiency can cause metabolic imbalances leading to neuronal cell death. Chronic alcoholism is one of the most important cause of WE, however, WE may develop in other conditions, such as severe malnutrition, hyperemesis gravidarum, prolonged parenteral nutrition, malignancies, bariatric surgery, immunodeficiency syndromes, liver disease, hyperthyroidism, and severe anorexia nervosa, hemodialysis- in patients with end-stage renal disease.[1] In this report, we present a patient who with WE had gastrointestinal tract diseases and had undergone gastrointestinal surgeries and discuss predisposing factors as well as diagnostic and therapeutic issues related to this entity.

Case

A 55-year-old-woman was admitted to the emergency room (ER) with forgetfulness, inability to walk followed by altere mental status. These complaints were started three days ago. Her relatives reported that she admited to general surgery clinic because of abdominal pain, vomiting, poor nutrition and was operated for alkaline reflux, hospitalized in the general surgery intensive care unit for 15 days. It was learned that she received parenteral nutrition while in the intensive care unit. She discharged three days ago. Then the patient was admitted to our emergency room with the above mentioned complaints. When the anamnesis was deepened, it was learned that, he had had two previous gastric surgeries, vomited for a long time and increased for a month, so that he could not eat. There was no other feature in her medical and family history. Her vital signs were stable. General examination was unremarkable except for the operation scar in the abdomen. On neurological examination, her GCS was 14. She was noncooperative and nonorientated. The patient was making nonsensical sentences composed of religious words. There was no significant abnormality in cranial nerve examinations, but horizontal nystagmus was seen on the left eye. Her fundoscopic examination was normal and she had no neck stiffness, obvious motor deficits, side signs, and pathological reflex. Cerebellar examination could not be performed because the patient was noncooperated. The results of her complete blood count, biochemistry panel, thyroid-stimulating hormone arterial blood gas were all within normal limits with the exception of non-hypoxic respiratory alcoholosis. Head computed tomography was negative for acute pathology. Lumbar puncture was performed to exclude central nervous system infection, however, cerebro-

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spinal fluid examination revealed no pathology. The patient was diagnosed as wernicke encephalopathy with anamnesis, physical examination findings. 200 mg thiamine replacement was started immediately. After thiamin replacement, the patient was sent to brain magnetic resonance imaging (MRI) and diffusion MRI. It showed symmetric mild hyperintensities on T2-weighted sequences in bilateral medial thalamus which supported the diagnosis of WE (Figure 1). Thyamine level measurement could not be performed in our hospital. The patient was consulted with the neurology clinic and tansferred to the intensive care unit. 3x200 mg IV thiamine was ordered to the patient. On the 1st day after the treatment, her speech and disorientation improved. She was transferred to the service. The patient who was mobilized on the 3rd day was observed to walk ataxia. Thiamine replacement therapy was continued. The patient, who was discharged on the 10th day, was able to walk with assistance and did not evaluate with mentally detailed neuropsychiatric tests, but there was a great improvement.

Discussion

WE is a clinical diagnosis. Recognition of risk groups, clinical symptoms and findings is of great importance in the prognosis of WE. The incidence of WE, mostly from autopsy studies, is 1-3%, however, this rate is considered to be higher in developing countries due to malnutrition.[2] The causes of WE are alcohol, malignancy, gastrointestinal illness-surgery, hyperemesis gravidarum, starvation, malnutrition, parenteral nutrition, vomiting, dialysis in renal diseases, psychiatric diseases, infections, intoxications, thyroid disease, and many other rare conditions.[3] Possible reasons of WE, in our case was gastrointestinal illness-surgery, vomiting, parenteral nutrition, so they were due to decreased oral intake and thiamine consumption. WE is presenting with classical findings of cerebellar, ocular and confusion as well as findings such as delirium, gait ataxia, delirium tremens, memory disturbance, hypothermia with hypotension.[3] Physical examination including neurological exam with cerebellar testing should be completed. Ataxia is a substantial finding in WE. Ataxia is due to combination of polyneuropathy, cerebellar damage, and vestibular paresis. Altered mental status, sensorium, disorientation, disinterest, inattentiveness, or agitation define encephalopathy. WE rarely can present with coma and death. Ocular abnormalities particularly nystagmus is the typical sign of WE.[4] Conjugate gaze palsies of oculomotor, abducens, and vestibular cranial nerve were also seen in WE. Peripheral neuropathy especially includes the lower extremity may be part of the clinical picture. Hypothermia develops due to thiamine deficiency of the temperature-regulating center in the brain stem and is accompanied by hypotension and coma.[5] Confusion, nystagmus, delirium, memory disturbance were present in our case. Initially, the non-cooperative patient was unable to perform cerebellar tests, but subsequently ataxia was detected.

WE is a clinical diagnosis with the signs and symptoms. Diagnosis of WE is usually made by history and physical examination. A complete blood count, serum glucose levels, the comprehensive metabolic panel, arterial blood gas, toxic drug screening, lumbar puncture, electroencephalogram and imaging study can be completed to exclude other causes of central nervous system abnormalities such as hepatic encephalopathy, stroke, alcohol withdrawal syndrome, delirium tremens, chronic hypoxia, meningitis- encephalitis normal pressure hydrocephalus, psychosis, closed-head injury, nonconvulsive status epilepticus and postictal state. Brain imaging studies such as computed tomography and magnetic resonance imaging (MRI) of patients with WE can

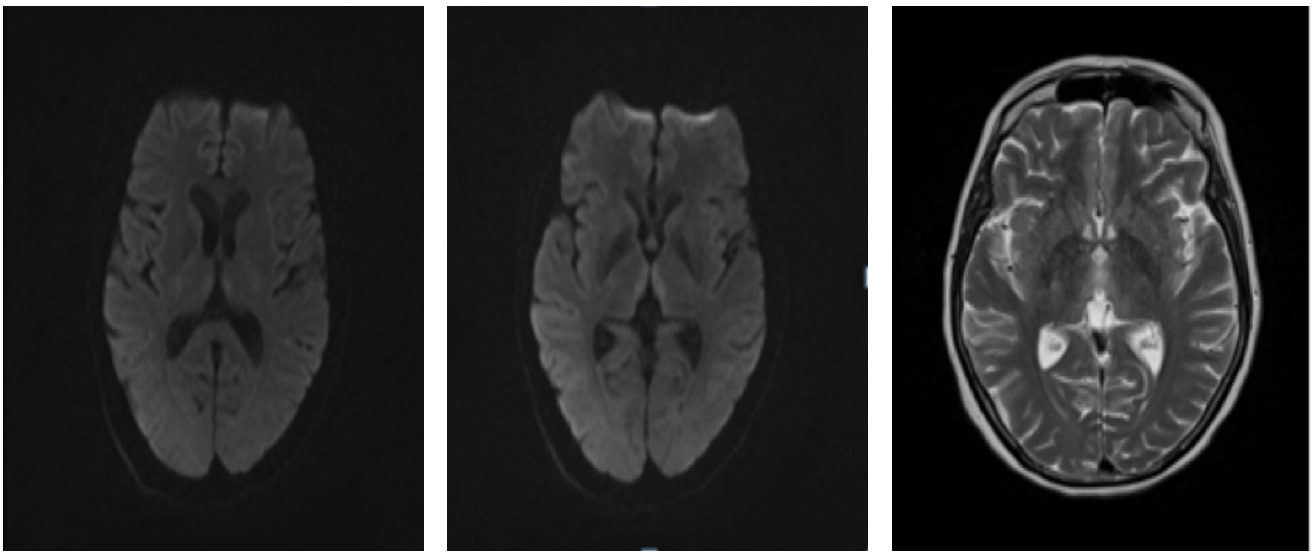

Figura 1. Brain MRI and diffusion MRI showed symmetric mild hyperintensities on T2-weighted sequences in bilateral medial thalamus.

exhibited generally symmetrical lesions in the thalamus with dilated ventricles and volume loss in the mammillary bodies.[6] Normal imaging cannot rule out WE.[5] Biomarkers, including an assay for thiamine, are not used diagnostic purposes, besides, no study has obviously described the sensitivity, specificity, and accuracy of thiamine levels in relation to active disease.[7] Erythrocyte transketolase levels reliably detect thiamine deficiency but are not necessary for the diagnosis of WE.[8] Blood pyruvate and lactate measurements are sensitive and helpful but not specific for thiamine deficiency illnesses. High-Performance Liquid Chromatography for Thiamine Detection is available in many countries. In our case, there was no significant pathology in our laboratory findings except MRI findings that support WE.

WE is one of the medical emergencies that must be noticed immediately. Studies suggest that up to 80% of patients with WE may not be diagnosed, therefore not be treated.[9] The aim of the treatment is the rapid correction of thiamine deficiency. Parenteral replacement of thiamine is most effective the patient's condition to some degree in almost all cases, although, in some cases, neurological deficits may persist despite replacement.[4] Thiamine is indicated for the treatment of suspected or manifest WE. It should be given, before any carbohydrate, 200 mg thrice daily, preferably intravenously (IV).[3] Ataxia, ophthalmoplegia and confusion usually resolve rapidly, within hours, after replacement of thiamine if diagnosed early in the disease course, on the other hand, memory and learning impairment tend to improve slowly as in our patient.[10] Management of WE after emergency diagnosis and thiamine replacement; requires a team approach including neurologist, intensivist, internist, endocrinologist, and psychiatrist. A dietary consult should be done to assess the calorie needs and determine how to provide the food as well as thiamine. Neurological injury, ataxia, Korsakoff syndrome, ophthalmoplegia are complications of WE.

Conclusion

WE is a notably morbid and mortal condition that can be prevented or reversed if diagnosed and treated early. WE should be suspected in any patient with form of malnutrition, feeding with prolonged parenteral nutrition, undergoing gastrointestinal surgery and also presenting with any symptoms and findings such as acute altered mental status, ophthalmoplegia, ataxic gait, delirium.

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