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“WERNICKE ENCEPHALOPATHY- A SEQUEL TO MISMANAGED HYPEREMESIS GRAVIDARUM - A RARE CASE REPORT.”

KEY WORDS: Wernicke encephalopathy, Hyperemesis gravidarum, Parenteral thiamine.

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ABSTRACT

Wernicke encephalopathy is an acute neurological condition characterized by a clinical triad of ophthalmoplegia, ataxia, and confusion. This disease is caused by thiamine deficiency, which primarily affects the peripheral and central nervous systems. It is a life-threatening condition best managed with parenteral Thiamine administration which results in dramatic, rapid recovery. But always prevention is better than cure and can be achieved by appropriate management of extreme starvation due to hyperemesis gravidarum. Here we present a case of Wernicke's encephalopathy resulting from hyperemesis gravidarum which was diagnosed based on clinical presentation, managed successfully with parenteral Thiamine.

INTRODUCTION

Although Wernicke encephalopathy mostly affects people who have a thiamine deficiency due to chronic alcoholism, various other causes include severe malnutrition, hyperemesis gravidarum, prolonged parenteral nutrition, malignancies, immunodeficiency syndromes, liver disease, hyperthyroidism, and severe anorexia nervosa. The female to male ratio for Wernicke encephalopathy is 1:1.7. Thiamine, one of the first B vitamins to be discovered also known as Vitamin B1, is a coenzyme that is essential for intricate organic pathways and plays a central role in cerebral metabolism. This vitamin acts as a cofactor for several enzymes in the Krebs cycle and the pentose phosphate pathway, including alpha-keto-glutamic acid oxidation and pyruvate decarboxylation. Therefore, deficiency of thiamine will lead to decreased levels of alpha-keto-glutarate, acetate, citrate, acetylcholine and accumulation of lactate and pyruvate. This deficiency can cause metabolic imbalances leading to neurologic complications including neuronal cell death in the mammillary bodies and thalamus. Studies involving computed tomography (CT) and magnetic resonance imaging (MRI) of patients with Wernicke encephalopathy revealed lesions in the thalamus with dilated ventricles and volume loss in the mammillary bodies. The classic triad of Wernicke encephalopathy is altered mental status, ataxic gait, and ophthalmoplegia. The diagnosis is made based on clinical presentation, and a definitive diagnosis is complicated as the clinical triad may not be present in up to 90% of patients.

Case Presentation

25 year old Primigravida at 18 weeks gestation was referred to delivery room of Government General Hospital with a diagnosis of intrauterine fetal demise. She was a case of primary infertility of 5 years duration and conceived spontaneously. She gave history suggestive of hyperemesis gravidarum associated with hematemesis which required hospitalization on and off since 3rd month of pregnancy. She also gave history of significant weight loss. The complaint of vomiting subsided one day ago. She was diagnosed as hypothyroid and was taking thyroid supplement at a dose of 12.5mcg per day. Her past history and family history were not significant. Upon admission, she complained of throat pain, blurring of vision, difficulty in walking, shortness of breath and fever for the past 2 to 3 days. There was no head injury, headache and seizures. On examination, her BMI was 16kg/sqm; dehydrated and afebrile. She appeared pale, not jaundiced but had bilateral vertical nystagmus. There was evidence of multivitamin deficiency in the form of glossitis, beefy red tongue, chelitis, angular stomatitis. Her vital data revealed tachycardia and tachypnoea, but otherwise normal

cardiovascular and respiratory systems. Neurological evaluation revealed cerebellar signs in the form of ataxia, tremors, abnormal gait. Motor power and tone were normal in both the extremities. Based on the history suggestive of chronic malnutrition, nystagmus, ataxia, a provisional diagnosis of Wernicke's encephalopathy was made and injection vitamin B1 was started at a dose of 100mg in 100ml normal saline, to be run over 30 minutes; 8th hourly. After this, routine induction of abortion was carried out uneventfully by medical method. She was discharged on 6th postabortal day in healthy condition with a prescription of oral vit B1 supplements at a dose of 100mg once daily for 10 days.

DISCUSSION

Wernicke encephalopathy is an acute neurological condition. The hallmark sign of Wernicke encephalopathy is ocular abnormalities especially nystagmus. Other symptoms include Pupillary sluggishness, ptosis, and anisocoria due to cranial nerve involvement of oculomotor, abducens, and vestibular nuclei. Gait ataxia is also a significant finding in Wernicke encephalopathy where patients will present with a broad-based gait. Also, gait can worsen, and in many cases, patients are unable to walk. Physical examination may include a complete neurological examination with cerebellar testing. Disorientation and altered sensorium characterize encephalopathy. Evaluation should include a thorough patient history with a focused physical examination and laboratory workup with appropriate imaging. There are no specific laboratory tests for diagnosing Wernicke encephalopathy as it is a clinical diagnosis. However, a complete blood count and the comprehensive metabolic panel can be completed to exclude other causes of central nervous system abnormalities. Moreover, normal brain imaging cannot rule out Wernicke encephalopathy and therefore not very beneficial either. Parenteral administration of thiamine is most effective and provides for rapid administration, however, in some cases, there are persistent neurological deficits, and the acute condition can progress to chronic Korsakoff syndrome. The preferred dose of thiamine treatment for Wernicke encephalopathy may be as high as 500 mg given one to three times daily parenterally. All malnourished patient may need higher doses of thiamine. Oral dosing is not reliable and not recommended. Our case was diagnosed based on the clinical history, nystagmus, gait ataxia; though the imaging studies of brain were normal. She responded promptly to parenteral Thiamine.

CONCLUSION

Wernicke encephalopathy is a clinical diagnosis in a patient of poorly managed hyperemesis gravidarum after excluding hyperthyroidism. It is a life-threatening condition best

managed with parenteral Thiamine administration which results in dramatic, rapid recovery. But always prevention is better than cure and can be achieved by appropriate management of extreme starvation due to hyperemesis gravidarum.

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