ORIGINAL RESEARCH PAPER

General Medicine

DECODING THE CONUNDRUM: WERNICKE'S ENCEPHALOPATHY PRECIPITATED BY HYPEREMESIS GRAVIDARUM - A SINGULAR CASE ANALYSIS

KEY WORDS: Wernicke's Encephalopathy, Hyperemesis Gravidarum, Thiamine

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ABSTRACT

Wernicke's encephalopathy (WE), a life-threatening complication of hyperemesis gravidarum, is primarily caused by a deficiency of thiamine (vitamin B1). This report outlines a case of Wernicke's encephalopathy triggered by hyperemesis gravidarum in a 28-year-old female, highlighting the significance of thiamine deficiency and appropriate supplementation of thiamine in patients with hyperemesis gravidarum

INTRODUCTION

Nausea and vomiting are very common during pregnancy, affecting up to 80% of pregnancies, with symptoms often being most severe during the first trimester (1-3). Wernicke encephalopathy is a serious neurological complication of thiamine deficiency that usually affects alcoholics (4,5). It is an acute neuropsychiatric syndrome characterized by the classic triad of ataxia, oculomotor abnormalities, and mental status change, along with cerebellar dysfunction and an altered mental state (6,7). Although hyperemesis gravidarum has been identified as a predisposing factor, WE in obstetric patients with persistent vomiting and feeding difficulties remains an under-recognized disorder (4). The objective of this paper is to review the clinical characteristics of WE in hyperemesis gravidarum and to raise the clinician's index of suspicion regarding this neuropsychiatric diagnosis and its preventability.

Case Study

A 28-year-old woman, who was 16 weeks pregnant, experienced excessive vomiting for eight weeks, followed by progressive weakness of her limbs, altered mental state, and blurred vision for the past three days. She did not have a history of significant alcohol consumption. On admission, her vital signs were stable, with a Glasgow Coma Scale (GCS) score of 12/15. Her physical examination revealed slow reaction to light with left eye conjugate palsy, nystagmus, and power of 4/5 in all four limbs with bilateral flexor plantar reflexes. Rest of the systemic examination was normal. Her serum biochemistry and blood tests during her stay are shown in Table 1. The MRI findings were indicative of Wernicke's encephalopathy [Figures 1-3]. She was admitted to the intensive care unit and was given intravenous thiamine, 500 mg three times a day for three days, followed by 250 mg twice a day for five days, along with other vitamins, electrolytes, and trace elements. Her hypernatremia was corrected by administering 0.45% normal saline with potassium chloride supplementation and free water of 200 ml every four hours, without developing signs of Central Pontine Myelinosis (CPM). Over time, her ocular symptoms improved, and she showed neurological recovery with improved muscle power.

Table 1: shows serial blood investigations of the patient

Investigation	Normal Range	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Hemoglobin	13-18gm/dl	14.1	11.1	10.1	11	11.8	11.4
White blood cell counts	4000-11000mg/dl	10,800	7,070	8,800	9,000	8,600	7,500
Platelets	150000-450000 mg/dl	224000	150000	160000	175000	200000	180000
Serum sodium	135-145 mEq/L	160	164	159	153	144	140
Serum potassium	3.5-5.3 mEq/L	3.0	3.3	2.9	3.4	3.2	3.8
Serum chloride	98-108 mEq/L	125	133	131	125	120	118

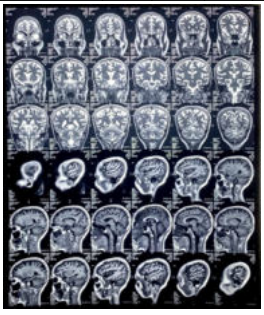

Figure 1

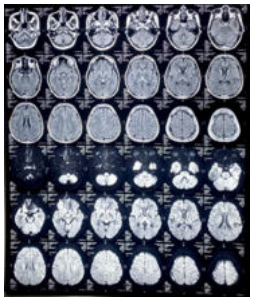

Figure 2

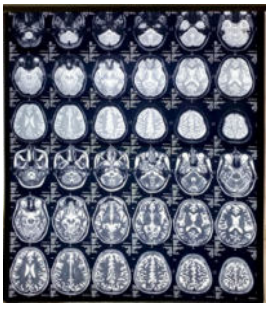

Figure 3

(Figures 1-3 showing ill-defined T2 and FLAIR hyperintense signals involving periventricular white matter around the third and fourth ventricle and periaqueductal white matter which were features suggestive of Wernicke's Encephalopathy)

DISCUSSION:

WE occurs as a result of thiamine (B1) deficiency, which is an essential cofactor in various stages of carbohydrate

metabolism. If cells with high metabolic requirements have inadequate stores of thiamine, energy production drops, and neuronal damage occurs (8). Body stores of B1 fall rapidly during fasting (9). Intravenous dextrose administered before correcting thiamine will worsen the situation. In pregnancy, it occurs due to excessive vomiting, poor intake, and increased metabolic demand. Additionally, the sequestration of the vitamin by the fetus and placenta (10) can lead to devastating complications such as spontaneous abortion and fetal loss (11). The assessment of blood transketolase activity and thiamine pyrophosphate (TPP) is not very reliable. Magnetic resonance imaging is the imaging modality of choice as it is highly specific (93%) and relatively safer than computed tomography scan. Prompt supplementation of thiamine leads to improvement in ocular signs within hours to days (12). If ocular palsies fail to respond, other diagnoses should be considered. In one report, recovery of vestibular functions began during the second week after thiamine treatment, and improvement in gait ataxia coincided with recovery of vestibular function (13,14)

CONCLUSION:

WE is a complication that can arise in cases of hyperemesis gravidarum, which is caused by a combination of poor nutrition, frequent vomiting, and the increased metabolic demands of pregnancy. Wernicke encephalopathy can also be triggered by the administration of glucose-containing solutions without prior thiamine supplementation (15). To prevent this complication, pregnant women who experience vomiting and feeding difficulties for more than three weeks should be prescribed oral or intramuscular thiamine supplements (100 mg/day). Intravenous thiamine supplements should be given to patients with longstanding hyperemesis gravidarum before giving parenteral carbohydrate solutions or nutrition. It is crucial to note that WE is a potentially reversible condition if treated early, and thiamine supplementation is essential for women with hyperemesis gravidarum. Additionally, maintaining electrolyte and glucose homeostasis is important to prevent complications such as cerebral edema. We strongly emphasize the importance of prompt thiamine supplementation in pregnant women with prolonged vomiting, especially before starting intravenous or parenteral nutrition

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