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Case Report

Wernicke's encephalopathy: obstetric diagnostic challenge! (a rare complication of hyperemesis gravidarum): a case report and review of literature

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ABSTRACT

Wernicke's encephalopathy (WE) is an acute neurological disorder caused by a deficiency in thiamine. It is characterized by triad of altered mental status, ataxia and ophthalmoplegia. Most of the cases reported were secondary to long-term alcohol use. We reported a rare case of WE due to hyperemesis gravidarum in a 29-year-old P1L1A1 women at 22 weeks of gestation who had spontaneous abortion. Patient manifested with features of mental confusion, nystagmus, and gait ataxia. Diagnosis was established after MRI findings suggestive of WE in thalamus. Patient clinically improved after treatment with thiamine. We emphasize the importance of thiamine supplementation to women with hyperemesis to prevent life threatening complications.

Keywords: Hyperemesis gravidarum, WE, Thiamine deficiency

INTRODUCTION

WE is a neurological disorder due to thiamine deficiency which can be potentially fatal but preventable.[1] It is typically diagnosed among alcholics and other underlying causes of thiamine deficiency may be due to multiple aetiology. The clinical presentation is extremely varied typical triad of confusion, ataxia and ophthalmoplegia in most of cases which was described by Carl Wernicke in 1881.

Nausea and vomiting are very common during pregnancy seen among 80% of pregnancies.[2] Hyperemesis gravidarum (HG) affects up to 3% of pregnant women in the first trimester of pregnancy. Maternal complications include dehydration, malnutrition, vitamin deficiencies, peripheral neuropathy and more serious neurological complications, including central pontine myelinolysis (CPM) and WE.[4-8] Therapeutic management consists of the administration of thiamine to avoid irreversible neurological damage. The relation between WE and HG was described in 1939 by Sheehan.[9] Here, we described the case of a 29-year-old pregnant female with WE due to HG. This highlighted that clinical suspicion was necessary to recognize signs and symptoms to begin appropriate management.

CASE REPORT

We presented the case of 29-year-old P1L1A1 who presented with spontaneous abortion at 22 weeks of gestation was referred from peripheral hospital in view of altered mental status. During her admission patient was disoriented and had was found to have nystagmus and gait disturbance and others features suggestive of malnourishment. With the help of patient attenders detailed history was elicited which revealed that her illness had begun around 18 weeks of amenorrhea, when she developed multiple episodes of vomiting at first trimester on daily basis, which was associated with weight loss and

epigastric pain. She had haemoglobin of 8.6% with microcytic hypochromic peripheral blood picture. Patient had hyponatremia and hypokalemia with elevated serum LDH and liver functions.

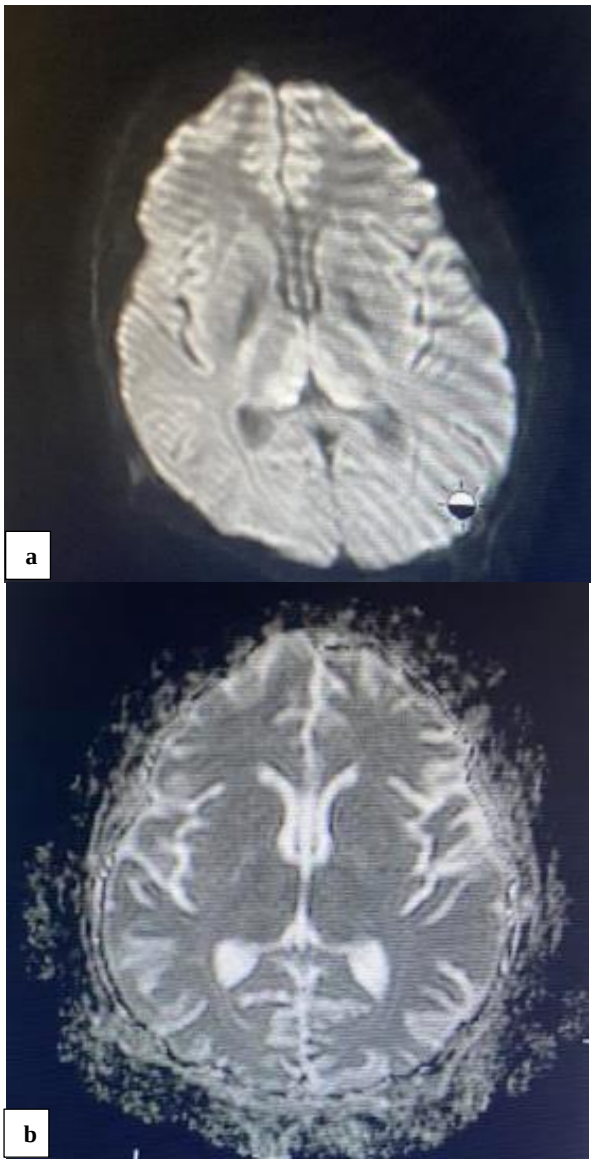

Figure 1 (a and b): Hyperintense signal on T2 and FLAIR with diffusion restrictions in bilateral thalamus.

According to Caine's criteria, the clinical and biological picture was in favor of hyperemesis gravidarum with severe hydroelectrolytic disorders.[11] Treatment began before confirmation of diagnosis, patient received thiamine with an emergency dose of 500 mg intravenously followed by loading dose every 8 hours for 3 days. There was drastic improvement in patient mental status. MRI was done for confirmation of diagnosis which showed hyperintense signal on T2 and FLAIR with diffusion restrictions in bilateral thalamus, characteristic of Wernicke´s encephalopathy (Figure 1). Patient then maintained with 100 mg orally for another 14 days. The patient also received potassium replacement intravenously and 1 pint of PRBC was transfused, she showed a gradual improvement in her neurological condition, being discharged after 14 days of hospitalization.

DISCUSSION

The diagnosis of WE is primarily clinical. In Caine et al operational criterion for the identification of WE, proposed that WE was diagnosed if any two of the following four signs exist: ophthalmoplegia, ataxia, altered mental status, and malnourishment.[10] Nystagmus is the most common sign and confusion is the most common presenting symptom. The more symptoms of WE, the more likely chronic Korsakoff syndrome will develop: a cognitive disorder characterized by severe amnesia, executive problems, and confabulation, which leads to lifelong impairment.[8,11]

WE is better diagnosed by the Caine's operational criteria, since not all patients have the classic triad. Caine's operational criteria for WE are: Wernicke's classic triad; autopsy evidence of WE; or clinical response to thiamine. The defining signs and symptoms for WE were dietary deficiencies, oculomotor abnormalities (reported as nystagmus or ophthalmoplegia), cerebellar dysfunction (reported as falling or imbalance), and an altered mental state (reported as delirium, confusion, and problems in alertness, or cognition).[10]

The body stores ~25 to 30 mg of water-soluble vitamin thiamine (B1), which is good for ~18 days. The daily requirement is 0.4 mg/1,000 kcal per day, which increases in pregnancy to 1.5 mg/day.[12] Deficiency of thiamine pyrophosphate, which is an important co-factor for enzymes in the pentose phosphate pathway, affects multiple tissues, particularly those with high thiamine turnover that include neural parenchyma resulting in cell necrosis or apoptosis resulting in brain tissue injury.[13] Thiamine is essential for carbohydrate and amino acid metabolism deficiency also results shunting of glucose to anaerobic pathway resulting in metabolic abnormalities.

WE in pregnancy generally occurs in women who are malnourished, due to the increased demands of pregnancy. Hyperemesis further depletes thiamine stores.[14] However, WE is uncommon in pregnancy but HG is pretty common. Wernicke encephalopathy generally occur at 14 to 18 weeks gestation after 2 to 3 weeks of vomiting,13 which was the case with our patient presented at 22 weeks with history of vomiting for 4weeks and was severely malnourished, emaciated with dry patchy skin. Malnourishment related WE is pregnancy is rare but which is aggravated due to hyperemesis.

WE is also associated with spontaneous fetal loss, fetal growth restriction.

The diagnosis may be reinforced by a brain MRI, which shows symmetric high T1, T2, and T2 flair signal

intensities in the mammillary body, medial thalamus, periventricular, and periaqueductal regions. The sensitivity and specificity of brain MRIs are 53%; and 93%; for the diagnosis of WE, respectively.[15,16]

CONCLUSION

WE is an uncommon and life-threatening neurological disease complicating pregnancy in the setting of hyperemesis. Timely and serious management of vomiting in pregnancy and replacement of thiamine stores help treat acute neurological symptoms and can prevent both maternal and fetal morbidity.

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