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Hyperemesis Gravidarum Induced Wernicke's Encephalopathy

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Case Report

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ABSTRACT

Introduction: Wernicke's encephalopathy (WE) is a condition caused by the depletion of Vitamin B1 levels. It causes neuropsychiatric symptoms. The triad includes Nystagmus and Ophthalmoplegia, mental status changes and Ataxia.

Case description: 29 year old female, Gravida2 Para1 Live1, with 5 months of amenorrhea brought to the hospital with reduced responsiveness for 2 days. Patient had 8-10 episodes of vomiting per day. The vomitus was non-bilious and non-projectile for last 2 months. Patient had opthalmoplegia with restricted extraocular movements. All four limbs had reduced tone. MR imaging showed bilateral thalamus hyperintensities. Serum thiamine level was 2 nmol/L. Patient was treated with 350 mg of intravenous thiamine three times a day. Patient improved clinically.

Conclusion: Hyperemesis Gravidarum is one of the rare causes of Wernicke's encephalopathy. This diagnosis should be brought to consideration when the neurological triad exist in women with hyperemesis gravidarum. Pregnant women who develop hyperemesis should be given thiamine supplementation, before intravenous or parenteral nutrition.

INTRODUCTION

Wernicke's encephalopathy (WE) is a condition caused by the depletion of vitamin B1 levels. It causes neuropsychiatric symptoms. This leads to cell death and local rise in glutamate. Most of the cases are due to chronic alcohol misuse [1]. Clinical trial of Wernicke encephalopathy includes Nystagmus and Ophthalmoplegia, Mental status changes and Ataxia [1].

In adults, autopsy studies have revealed a higher prevalence of Wernicke's encephalopathy lesions (0.8-2.8%) than is predicted by clinical studies (0.04-0.13%). Similar data have been reported in children. In particular, in adult patients who misused alcohol and those with AIDS, Wernicke's encephalopathy confirmed at autopsy had been missed by routine clinical examination in 75-80% of cases. In children, about 58% of cases have been missed at routine clinical examination [1].

CASE REPORT

He has noticed being increasingly short of breath on exertion but put this down to changing from an active job as a chef to a more sedentary job as a site manager. On 29 year old female, Gravida2 Para1 Live1, with 5 months of amenorrhea was brought to the hospital with complaints of reduced responsiveness for 2 days. Patient had swaying to both sides while walking for 3 days. On elicitation of history, patient's attenders revealed that patient used to have daily 8-10 episodes of vomiting that is non-bilious and non-projectile for last 2 months. But no treatment was sought for the same. Patient didn't had fever or loose stools. On examination patient was dehydrated, pulse rate was 110/min, and blood pressure was 100/60 mm Hg. Patient was drowsy and irritable. All four limbs were moving to painful stimuli. Bilateral pupils were reacting to light. But patient had ophthalmoplegia with restricted extraocular movements. All four limbs had hypotonia. MR imaging showed bilateral hyperintensities in thalamus, mammillary body, floor of fourth ventricle and periaqueductal signal changes. Serum thiamine level was 2nmol/L. Patient was treated with 350 mg of intravenous thiamine three times a day. From Day 2, patient became conscious and oriented. But ophthalmoplegia and ataxia were persisting. From Day 3 patient's extraocular movements improved. But lateral rectus palsy was present. From Day 5 lateral rectus palsy resolved and patient was able to sit by herself and was able to walk with support. But horizontal nystagmus was present. From Day 8, patient was able to walk by herself and was normal neurologically. Nystagmus also resolved. Injection

thiamine was given in the same dosage for a total of 14 days. Patient was able to walk by herself and was able to do day today activities at the end of 14 days. Repeat MRI scan showed normal and the hyperintensities in basal ganglia got resolved. Patient was discharged with tablet Benfotiamine 100 mg tid dose.

DISCUSSION

1 and 2 mg per day is the daily thiamine requirement for a healthy adult. But it varies based on the alcohol consumption and carbohydrate content in the diet. Alcohol can cause damage to intestinal mucosa which in turn can cause reduced absorption of thiamine [2,3]. Reidling et al. found out the intestinal transport mechanism for thiamine. It is based on THTR-1 (SLC19A2), driven by the normal pH gradient across the membrane Reidling et al., It is a sodium independent transport system. SLC19A1 is responsible for the transport of monophosphate and pyrophosphate derivatives of thiamine. Among the transporters, SLC19A2 is a high affinity transporter gene, while the SLC19A3 (THTR-2) is a lower affinity transporter. Reidling and Said have found that both intestinal and renal thiamine uptake processes are increased during thiamine deficiency. This increase may not happen when chronic thiamine deficiency is present or when chronic alcoholism is present [4,5].

Hyperemesis gravidarum is a severe form of nausea and vomiting which affects one in 200 pregnant mothers. It is also one of the most common indications for hospital admission during early pregnancy. This disorder has a higher prevalence among the low educational level, lower income group and those in part-time employment [6].

Currently MRI is considered as the best modality to diagnose Wernicke's encephalopathy. MRI has a sensitivity of only 53% but a high specificity of 93%. Increased T2 signal, bilaterally symmetrical, in the paraventricular regions of the thalamus, the hypothalamus, mammillary bodies, the periaqueductal region, the floor of the fourth ventricle and midline cerebellum - these are the common findings in MRI in case of Wernicke's encephalopathy [7]. In alcoholic and non-alcoholic patients with Wernicke's encephalopathy, MRI findings are mostly the same, except the atrophy of the Cerebellar Vermis and Mammillary bodies.

Reasons for this includes: 1. Cerebellar vermis and mammillary bodies are susceptible to thiamine deficiency in alcoholic patients; 2. MRI findings in the acute phase are contaminated by previous attacks in alcoholics. MRI brain is also helpful for knowing the prognosis of the disorder. If only the periaqueductal area, thalamus and caudate nucleus are involved, it has a good prognosis. Cortical damage is indicative of irreversible damage and poor prognosis [8].

CONCLUSION

Hyperemesis gravidarum is one of the rare causes of Wernicke's encephalopathy. This diagnosis should be brought to consideration when the neurological triad exist in women with hyperemesis gravidarum. Any pregnant women who develop hyperemesis should receive thiamine supplementation, especially before intravenous or parenteral nutrition.

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