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Case Series

Wernicke's encephalopathy in pregnancy

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ABSTRACT

Wernicke's encephalopathy (WE) is a reversible neurological emergency which is a rare but known complication of hyperemesis gravidarum due to thiamine deficiency. Prolonged vomiting in pregnancy results in thiamine depletion. Most frequently Wernicke's encephalopathy is found among persons suffering from excessive drinking. Unusually it can also be seen in women presenting with hyperemesis gravidarum with pre-existing malnutrition, as avitaminosis can result from the acute malnutrition associated with prolonged pregnancy-related hyperemesis. The early recognition of its clinical signs and symptoms is essential to establish the suspected diagnosis and can be confirmed by MRI. Most patients present with the triad of ocular signs, ataxia, and confusion. It can be associated with life-threatening complication like central pontine myelinolysis. Here we stress upon the importance of early diagnosis and prompt treatment of WE. The aim of this report is to present cases of Wernicke's encephalopathy induced by hyperemesis gravidarum except one case which was acute in onset. The course of the disease, clinical signs, diagnostic tools, treatment and its results are presented.

Keywords: Wernicke's encephalopathy, Hyperemesis gravidarum, Thiamine deficiency

INTRODUCTION

Wernicke's encephalopathy is a rare neurological disorder, due to thiamine deficiency in hyperemesis gravidarum and is also precipitated by administration of glucose-containing fluids before thiamine supplementation.[1] It is an acute neuropsychiatric thiamine deficiency disorder associated with alcoholism and malnutrition. It was described by Carl Wernicke in 1881 in patients presenting with the triad of ocular signs, ataxia, and confusion that is seen in 60% of cases. It is typically diagnosed among alcoholics (12.5%), but in non-alcoholic prevalence varies from 0.04-0.13%. Many cases in pregnancy with hyperemesis gravidarum (HG), were first reported in 1914.[1] Hyperemesis can further be complicated by life-threatening condition like central pontine myelinolysis due to electrolyte fluctuations. Here we present 3 cases of Wernicke's.

CASE SERIES

Case 1

23 year old primigravida with 16 week 5 days of amenorrhea with chronic hypertension came to casualty with vomiting for 3 months aggravated since 2 days with 10 episodes per day associated with giddiness. On examination, patient was conscious, oriented and vitals were normal. Hydration was poor, urine acetone was found to be positive, all other blood investigations were normal.

She was treated with intravenous (IV) fluids, antiemetics and thiamine. As giddiness didn't subside, magnetic resonance imaging (MRI) brain was taken. MRI report was showing lesion in periaqueductal area diagnosed to be Wernicke's encephalopathy. The patient was initially

treated with injection thiamine 100 mg IV thrice daily for 5 days with significant symptomatic improvement and changed to oral thiamine till delivery.

Case 2

21 year old G2P1L1 with 18 weeks 5 days of pregnancy presented with vomiting since one month, 5 to 6 episodes per day, blurring of vision and giddiness. On examination, she was conscious, oriented, severely dehydrated and drowsy. Her blood reports were showing creatinine 1.24 mg/dl, potassium was 3.9 mEq/l, sodium was 136 mEq/l and urine acetone was positive. She was started on antiemetics and IV fluids. Blurring of vision and drowsiness didn't subside. Neurophysician opinion was obtained and was advised to take MRI brain. MRI brain was showing periaqueductal hyper intensified areas and diagnosed to be Wernicke's.

She was treated with IV fluids and injection thiamine 150 mg TDS for 5 days followed by oral supplements. Caesarean section was done for obstetric indications. Both baby and mother are fine.

Case 3

25 year old primigravida with 35 weeks of pregnancy presented with acute onset of giddiness and double vision. She was not a known case of diabetes, hypertension, hyperthyroidism and alcoholism. On examination she was having ataxia, nystagmus and her general condition was fair. MRI brain showing altered signal in the cerebellar vermis (Figure 1).

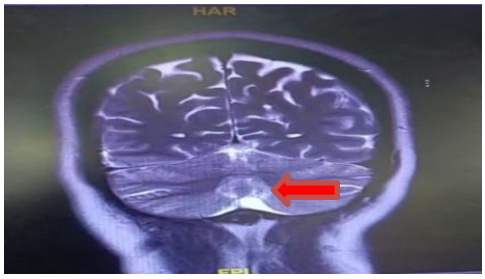

Figure 1: MRI brain showing altered signal in the cerebellar vermis.

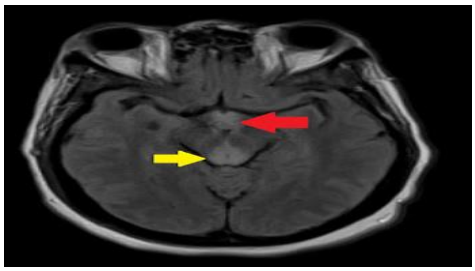

Figure 2: MRI in we shows hyperintense T2/flair signal involving the mammilary bodies, dorsomedial thalami, tectal plate, periaqueductucal area and around third ventricle.

She was also treated with thiamine injection 150 mg TDS for 2 weeks followed by oral supplements till her postnatal period of 6 weeks. She delivered by spontaneous vaginal delivery with a good outcome baby.

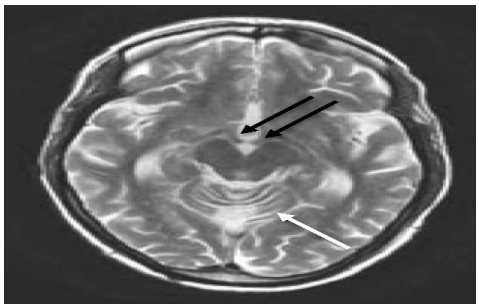

Figure 3: Axial T2 weighted MRI of the head at the level of midbrain shows prominent vermian folia and abnormal increased signal in the superior vermis (white arrow) as well as normal size and signal intensity of the mamilary bodies (black arrow).

DISCUSSION

WE is due to deficiency of thiamine (B_1), which is an essential cofactor for several key enzymes like transketolase, alpha-ketoglutarate dehydrogenase and pyruvate dehydrogenase important in carbohydrate metabolism. The body stores 25 mg to 30 mg of vitamin thiamine which is sufficient for 18 days.[2] In pregnancy due to excessive vomiting, poor intake, increased metabolic demand and sequestration of the vitamin by the foetus and placenta there will be deficiency of thiamine. If the cells with high metabolic requirements have inadequate stores of thiamine, energy production drops, and neuronal damage occurs. Thiamine dependant enzymes play an essential role in cerebral energy utilization that's why thiamine deficiency can cause brain tissue injury.[3] Intravenous dextrose administered before correction of thiamine will aggravate the condition further, as there is consumption of left out thiamine and lead to the WE.[1]

The classical triad of WE include encephalopathy, oculomotor dysfunction, and gait ataxia.[3] But in most of the patients only one or two features will be there. Confusion will be the most common presenting symptom, followed by staggering gait and ocular problems.

In our report, first case presented with giddiness followed by chronic vomiting. Second case presented with giddiness, and blurring of vision followed by chronic vomiting whereas third case presented with acute onset of Giddiness and blurring of vision without any prior history of vomiting. So symptoms common to our cases are giddiness and blurring of vision.

The encephalopathy is characterized by profound disorientation, indifference, and inattentiveness. Nystagmus will be the most common ocular finding and is typically evoked by horizontal gaze to both sides. Ataxia

primarily involves stance and gait and is likely due to a combination of polyneuropathy, cerebellar involvement and vestibular dysfunction. WE is mostly a clinical diagnosis as lab tests are not so convincing.

WE can progress to Korsakoff s syndrome in some instances which is irreversible.[4]

MRI is the imaging modality of choice because it is highly specific (93%) and comparatively safer than computed tomography scan in pregnancy. Reversible cytotoxic oedema seems to be the most distinctive lesion in WE, and the most useful sequences to detect it are t2, flair, and DWI.[5] Periventricular regions of diencephalon, mesencephalon, brainstem, and superior vermis of the cerebellum are sensitive to thiamine deficiency due to cellular dependence on oxidative metabolism that causes T2W hyperintensities in the region. Typical findings include areas of increased T2 and flair signals, decreased T1 signal, and diffusion abnormality surrounding the aqueduct and third ventricle and within the medial thalamus, dorsal medulla, tectal plate, and mamillary bodies (Figures 2 and 3). All three cases in report were having hyper intensified area around periaqueductal area.

Guidelines by the European federation of neurological societies (EFNS) recommend that thiamine should be given 200 mg thrice daily via intravenous route, started before any carbohydrate, and continued until there is no further improvement in signs and symptoms.[6] In non-alcoholic patients, an intravenous dose of thiamine 100-200 mg once daily could be enough; whereas in alcoholic patients, higher doses may be required.

All reported cases also recovered completely after treating with parenteral thiamine followed by oral thiamine supplementation. This shows the timely diagnosis and treatment with the thiamine reverses the condition.

CONCLUSION

As obstetricians we should think of medical complication of hyperemesis gravidarum, if we are not treating it properly may lead to complications like Wernicke's. In our cases early recognition of the symptoms and signs of Wernicke's lead to early intervention and prevention of irreversible sequelae. Thorough knowledge of the IV fluids is very important to treat the condition as dextrose may precipitate the condition. Early prediction of the Wernicke's is important to treat the condition and to prevent the irreversible sequelae. Appropriate treatment with thiamine is helpful.

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