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A Rare Complication of Hyperemesis Gravidarum-Wernickes Encephalopathy

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Abstract: ***Wernicke's encephalopathy is a rare neurological syndrome that presents in the setting of thiamine deficiency. It is mainly associated with excessive alcohol intake. other causes include malnutrition, malignancy, gastric bypass surgery hemodialysis and hyperemesis gravidarum. It is a very rare complication of pregnancy. Wernicke's encephalopathy is characterized by clinical triad which includes ataxia, acute delirium and oculomotor disturbances. MRI confirms the diagnosis by the presence of hyper signals most frequently in the periaqueductal level of thalami and mamillary bodies in these cases, we report three cases of wernicke's encephalopathy in the context of hyperemesis gravidarum that developed the classical clinical trial of wernicke's encephalopathy along with spontaneous nystagmus and typical imaging findings on the brain imaging. subsequent treatment with thiamine led to rapid improvement in the clinical status and favourable course of pregnancy.***

Keywords: Hyperemesis Gravidarum, Wernickes Encephalopathy, neurological syndrome

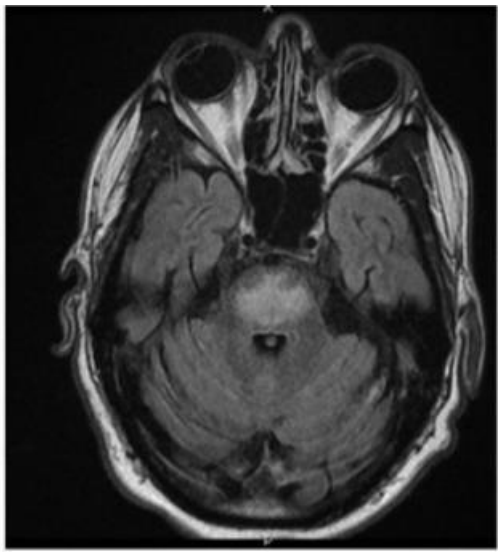
1. Introduction

Wernicke's encephalopathy is rare neurological disorder, due to thiamine deficiency and it's precipitated by administration of glucose containing fluids before thiamine supplementation. It was described by Carl wernicke in 1881, in patients presenting with triad of ocular signs, ataxia and confusion that is seen in 60% of cases. It typically diagnosed among alcoholics (12.5%), but in non alcholics prevalence varies from 0.04-0.13%. Due to electrolytes abnormalities, it may lead to life threatening complications like central Pontine myelinolysis. The aim of this report is to present the case of wenicke's encephalopathy induced by hyperemesis gravidarum. The course of disease, clinical signs, diagnostic tools, treatment and it's results are presented.

2. Case Report

Case series 1
37 yr old G2P1L1/prev LSCS/at 16 wks of gestation, presented in an unconscious state with complaints of nausea, vomiting since conception, fever with breathlessness for past 1 day. Vitals-Her BP was not recordable, pulse feeble, RR-38/min, Temp-100 degree. Patient was diagnosed to have starvation ketoacidosis (RBS_442 mg/dl). Due to poor GCS, patient was intubated and maintained on mechanical ventilation. Neurophysician opinion obtained, as the patient had features of ataxia, ophthalmoplegia and global confusion a diagnosis of wernicke's encephalopathy was made and the same treated with injection thiamine. Pregnancy was terminated in view of maternal hemodynamic instability.

MRI brain revealed features of central pontine myelinolysis



She was treated with Intravenous fluids, thiamine and antiemetics. Neurophyscian review obtained and advised to continue T. prednisolone for 2 weeks. patient condition improved and hence discharged and advised for oral contraceptives.

Case series 2:
A 23 yr old primi, at 23 weeks of gestation, presented with history of general weakness, inability to walk for past 1week. she gives history of hyperemesis was on IV fluids most of time.

On examination: patient conscious, well oriented, vitals normal. Nystagmus+ with ataxia gait. All investigations were sent and found to be normal except hypokalemia. Fundus examination revealed blurred disc margins and peripappillary edema. MRI brain showed hyperintensity of Mamillary bodies.

Treated with Inj. thiamine, Iv fluids. Patient condition improved from second day onwards and on day 5 nystagmus

very much improved and gait becomes better. She delivered at term, alive boy baby by normal vaginal delivery

Case Series 3

A 30-year-old woman, 10 weeks pregnant and presented with intractable vomiting in pregnancy was admitted to the hospital she complained of dizziness and headache but those symptoms quickly subsided. But she developed vertigo and nystagmus, but her mental status was alert, 3 days later, she was presented with diplopia and complained of difficulty in closing her eyes. MRI showed hypersignals of periaqueductal region, Mamillary bodies. She was treated with thiamine. Her symptoms gradually improved after 2 weeks. She developed gestational diabetes mellitus and was treated with OHA. she delivered an alive Male baby by LSCS.

3. Discussion

Wernicke‘s encephalopathy occurs due to deficiency of thiamine, which is as an essential cofactor in various stages of Carbohydrate metabolism. If the cells with high metabolic requirements, have an inadequate store of thiamine, energy production reduced and neuronal damage occurs. Intravenous dextrose administered before correction of thiamine will aggravate further risks. In Pregnancy, it happens due to excessive vomiting, poor intake and increased metabolic demand. In addition, sequestration of the vitamin by fetus and placenta, can have devastating complications like spontaneous abortion, fetal loss. MRI is the imaging modality of choice, because it is highly specific and comparatively safer than CT. Our patients had a significant history of severe vomiting during pregnancy, with poor intake which led to wernicke’s encephalopathy. They presented with confusion, encephalopathy, ophthalmoplegia. MRI brain was diagnostic and treatment was started without delay. Thiamine is a water-soluble vitamin and is absorbed throughout the small intestine and more efficiently in the upper jejunum. Thiamine acts as a cofactor for enzymes such as transketolase, alpha-ketoglutarate dehydrogenase, and pyruvate dehydrogenase which play a major role in carbohydrate metabolism. Thiamine also has a protective effect on retinal neurons against glutamate toxicity and aids in the survival of hippocampal neurons in vivo studies. It also facilitates neurotransmission and the release of neurotransmitters such as acetylcholine, dopamine, and norepinephrine. During HG, thiamine storage is depleted and the absorption rate is decreased significantly due to excessive vomiting, poor oral intake, and high metabolic demand. In our patient, we believe that the rapid correction of hypoglycemia without any replacement of thiamine caused the depletion of residual storage.

The classic triad of WE is often seen in patients with HG as compared to those with WE associated with alcoholism. Confusion is the most common findings followed by nystagmus and gait instability. In our patient, the etiology of the gait instability is multifactorial and due to a combination of ocular dysfunction, poor nutritional status, orthostatic intolerance, and other dysfunctions of other, higher structures. Our patient had a classic clinical picture in the appropriate setting, and the diagnosis was confirmed by the low levels of Thiamine. Patients with WE show a selective pattern of damage in the subcortical regions of the brain that can involve the thalamus, mammillary bodies, midbrain, and brainstem structures, including the vestibular system. The exact pathophysiology of the selective pattern of neurodegeneration remains unclear. Reversible cytotoxic edema is noted in the subcortical region and predominately seen in the T2/FLAIR and diffusion-weighted imaging (DWI) sequences. Our patient had bilateral thalamic hyperintensities on FLAIR and DWI changes significantly on the left side as compared to the right. The repeat MRI scan during her follow-up showed the resolution of previously seen imaging findings. Thiamine replacement should always precede the replacement of sugars during the treatment of WE. A low threshold of suspicion and quick intervention are keys to the prevention of significant neurological injury. The dosage and duration of thiamine replacement are not well defined in these patients.

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

4. Conclusion

Wernicke’s encephalopathy is a potentially reversible conditions if treated early. Thiamine supplementation is crucial for women with hyperemesis gravidarum. Moreover, replacement of electrolytes and glucose homeostasis is also important to prevent CPM. It occurs probably due to hypokalemia, hypernatremia and hyperosmolality in hyperemesis. MRI facilitated the diagnosis by finding FLAIR hypersignals in a region of interest (periaqueductal, thalamus, Mamillary bodies). we would like to emphasize the importance of prompt thiamine supplementation in pregnant women with prolonged vomiting, especially before starting intravenous or parental nutrition.

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