Abstract:
Wernicke's Encephalopathy is a rare but known complication of hyperemesis gravidarum resulting from the combination of poor nutritional intake, frequent vomiting, and increased metabolic demands of pregnancy. Our patient presented with Wernicke's Encephalopathy resulting from prolonged hyperemesis gravidarum presented with rapidly evolving ataxia, blurring of vision and altered sensorium. This is an unusual cause of Wernicke's Encephalopathy, a potentially fatal medical emergency due to thiamine deficiency. Characteristic brain MRI findings and rapid response to thiamine suggested that she had Wernicke's Encephalopathy, possibly due to vomiting and dextrose administration without thiamine supplements. A high index of suspicion is required, since delayed or lack of treatment may lead to high morbidity and mortality. We stress upon the importance of early diagnosis and prompt treatment of Wernicke's Encephalopathy.

Keyword: Wernicke's encephalopathy, Hyperemesis Gravidarum, non alcholic wernicke's encephalopathy, thaimine deficiency,

INTRODUCTION:
Nausea and vomiting of pregnancy has been a very common age old phenomenon. Though not well understood, it occurs in almost 70 percent of pregnant women. While “morning sickness” remains common, it is usually more troublesome when it is serious\(^2\). We describe a young pregnant woman with hyperemesis gravidarum complicated by wernicke's encephalopathy. It presents with ocular and gaze palsies, ataxia and derangement of higher mental function. Wernicke's encephalopathy is a potentially reversible condition caused by thiamine deficiency. It is usually suspected in the setting of chronic alcoholism and might not be recognized when associated with other conditions\(^3\).
Wernicke's encephalopathy is an acute neuropsychiatric disorder resulting from thiamine (vitamin B1) deficiency. Traditionally, the clinical diagnosis of Wernicke’s encephalopathy rests on the classical triad consisting of ocular signs, altered consciousness, and ataxia(1). Ocular signs associated with WE includes nystagmus, bilateral lateral rectus palsies, and conjugate gaze palsies reflecting involvement of the oculomotor, abducens, and vestibular cranial nerves nuclei.

CASE REPORT:
22yr old primigravida in her 18th week of gestation with the diagnosis of hyperemesis gravidorum for past 4 months on symptomatic treatment presented with blurring of vision, altered sensorium, unable to walk with tendency to fall back for past 2 days. She also had nausea & intractable vomiting for past 4 days treated in private hospital with intravenous fluids and antiemetics. On examination, she was drowsy, had bilateral upgaze & downgaze nystagmus, bilateral gaze retraction, with ataxia, responding to commands, and with severe dehydration. Laboratory studies were remarkable only for ketonuria. Other parameters including liver function test, thyroid function test, abdominopelvic ultrasound were normal.

Routine hyperemesis therapy started. Neurologist opinion obtained. MRI taken after 2 days of admission.

DISCUSSION:
In 1881, Carl Wernicke described the syndrome that now bears his name. Wernicke originally named the disease “polioencephalitis hemorrhagica superioris”

T2 weighted and Fluid Attenuated Inversion Recovery (FLAIR) images demonstrated hyperintensity in bilateral paramedian thalamus (Fig 1)

![Fig. 1 MRI BRAIN T2 AXIAL & FLAIR CORONAL](image1)

Hyperintensity in periaqueductal grey matter of midbrain (Fig 2)

![Fig. 2 MR BRAIN T2 AXIAL & FLAIR CORONAL](image2)

Hyperintensity in mamillary bodies (Fig 3)

![Fig. 3 MRI BRAIN](image3)

Diffusion restriction noted in the above mentioned region in DWI & ADC images. Patient started with Inj. Thiamine 500mg IV TDS for 5 days. Blurring of vision improved after 2 days, patient improved slowly. Inj Vit B complex IM given every week along Oral Vit B complexes. Patients symptoms recovered except ataxia. Review MRI taken after 1 ½ month it showed improvement. Past discharged 26th week of her gestation.
Wernicke's Encephalopathy is caused by a deficiency in thiamine (vitamin B1), a water-soluble compound essential for carbohydrate metabolism. However, the disease may not manifest in all patients with thiamine deficiency because genetic susceptibility may be involved in some patients who develop the disorder. Moreover, blood serum levels of thiamine may not be reduced at the time of clinical onset, and replacement of the vitamin does not always fully reverse the clinical picture, limiting the value of a therapeutic test under certain circumstances.

Wernicke's encephalopathy is recognized if there are two of the following four signs: (i) dietary deficiencies, (ii) oculomotor abnormalities, (iii) cerebellar dysfunction, and (iv) either an altered mental state or mild memory impairment.

The prevalence of Wernicke's encephalopathy in a non-alcoholic patient varies from 0.04% to 0.13%. Wernicke's encephalopathy in a patient with hyperemesis gravidarum was first described by Sheehan in 1939. The classic clinical triad is present in only a subset of patients. Clinical presentation is usually subtle especially in non-alcoholic patients and in those with deep coma whose neurological evaluation is often limited.

Non-alcoholic Wernicke's encephalopathy manifests in many different clinical settings, such as gastrointestinal tumours, hyperemesis gravidarum, chemotherapy, acquired immunodeficiency syndrome, prolonged therapeutic fasting, prolonged parenteral nutrition and bariatric surgery, anorexia nervosa and can even be secondary to socioeconomic factor.

Atypical MRI findings appear to be more prevalent in non-alcoholic Wernicke's encephalopathy. Imaging features MR abnormalities in WE patients are classically reported in the literature as bilateral and symmetrical lesions around the third ventricle, in the dorsomedial portions of the thalami and the periaqueductal region of the mid-brain, characterised by high signal intensity on T2 weighted sequences. Our patient presented with typical findings of Wernicke's.

Thiamine is an essential cofactor for energy metabolism. Thiamine is an important co-enzyme for three critical enzymes in the Kreb's and pentose phosphate cycle: transketolase, ketoglutarate dehydrogenase, and pyruvate dehydrogenase complex. Result in focal lactic acidosis, cerebral energy impairment, depolarization of neurons due to n-methyl-D-aspartate receptor mediated excitotoxicity. Ultimately, it results in alteration of blood brain barrier, generation of free radical, prompting cell death by necrosis and apoptosis. Delayed treatment leads to irreversible neuronal necrosis and gliosis especially of the medial dorsal nuclei which manifests as incomplete recovery of memory defects. Ultimately, it results in alteration of blood brain barrier, generation of free radical, prompting cell death by necrosis and apoptosis.

CONCLUSION:
Wernicke's encephalopathy should be suspected in any nutritionally compromised patient who shows altered mental status before manifestation of the classical triad as, if left untreated, it could lead to irreversible and persistent neurological sequelae or death. Hyperemesis is a potentially life-threatening complication of pregnancy. All practitioners caring for
pregnant women should be familiar with the indications for hospital referral. A protocol for the management of Hyperemesis Gravidorum should be available in all units to ensure accurate recognition, fluid, electrolyte and vitamin replacement and pharmacological treatment.

REFERENCES:


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