CASE REPORT

Wernicke's Encephalopathy: A Rare Complication of Hyperemesis Gravidarum

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Abstract

Aim: A case of Wernicke's encephalopathy (WE) due to hyperemesis gravidarum is presented for its rarity and favorable outcome of pregnancy due to timely diagnosis and management.

Background: Wernicke's encephalopathy is a rare reversible neurological complication of hyperemesis gravidarum due to vitamin B1 (thiamine) deficiency. Hyperemesis gravidarum (HG) is associated with prolonged starvation and electrolyte imbalance leading to Wernicke's encephalopathy, which is a triad of ocular signs, ataxia, and confused mental state. It also leads to a serious complications like central pontine myelinolysis (CPM). During pregnancy, Wernicke's encephalopathy has poor outcomes in mother and baby.

A fifth gravida presented at 14 weeks of gestation with Wernicke's encephalopathy was treated with thiamine, recovered after 34 days, and delivered by cesarean section at 36.5 weeks, a live healthy baby.

Conclusion: A favorable outcome of pregnancy can be there with a high index of suspicion, timely diagnosis, and management with thiamine.

Clinical significance: Immediate supplementation of thiamine in antenatal women with prolonged vomiting in pregnancy, especially before starting intravenous glucose can prevent this reversible neurological condition.

Keywords: Ataxia, Case report, Hyperemesis gravidarum, Mental confusion, Thiamine, Wernicke's encephalopathy.

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Introduction

Wernicke's encephalopathy (WE) is a rare, potentially fatal neurological disease resulting from thiamine deficiency, particularly due to prolonged fasting, intravenous fluids, and hyperemesis gravidarum (HG). It was first noted by Carl Wernicke in 1881, in women with the triad of ocular signs, ataxia, and confused mental state.[1]

Wernicke's encephalopathy is usually seen in alcoholics (12.5%), its prevalence varies from 0.04 to 0.13% in women with HG.[2] 80% of cases are diagnosed only on autopsy as it has serious maternal sequelae, so one needs to be vigilant of this entity as it is often not diagnosed. So high index of suspicion is necessary.[3]

Wernicke's encephalopathy during pregnancy with HG was first seen in 1914.[2] The diagnosis is difficult as there are no laboratory tests, and a woman can present with nonspecific symptoms and neurological signs. Hyperemesis can also be associated with central pontine myelinolysis (CPM) due to electrolyte imbalance and this can be dangerous to life.[4]

We report a rare case of WE following HG diagnosed clinically in a fifth gravida with minimal features on magnetic resonance imaging (MRI) of the brain and with a favorable pregnancy outcome due to timely management.

Case Description

A 24-year-old fifth gravida with 14 weeks of pregnancy attended antenatal clinic with excessive vomiting since 6th week of gestation associated with hazy vision, less power in lower limbs which was increasing, and change in sensorium since 3 days.

A provisional diagnosis of WE was made clinically due to altered sensorium with HG. On examination, she had cachexia, her vitals were stable, she had altered sensorium, blurred vision, and ptosis of an eye, and her lower limb power was grade II with bilateral flexor plantar reflexes. On fundus examination, there was papilledema, and obstetric ultrasonography showed single live intrauterine pregnancy of 13.4 weeks. Her blood investigations are as shown in Table 1. Urine ketones were in large amounts. Magnetic resonance imaging was suggestive of edema in mammillary body and medial thalami (Fig. 1). From history, clinical examination, MRI report diagnosis of WE was made, and treatment was initiated.

Her sodium levels were corrected by intravenous 3% sodium chloride. Ketosis was corrected by intravenous Ringer lactate and normal saline, and she was ventilated with elective intubation due to altered sensorium and was supplemented with intravenous thiamine 100 mg thrice a day, vitamins, electrolytes, and trace elements. Clinical improvement following supplementation of thiamine confirms the diagnosis of WE.

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Table 1: Shows comparison of laboratory investigations on day 1, day 10, and day 24 with normal levels

Investigation	*Day 1*	*Day 10*	*Day 24*	*Normal range*
Serum sodium	118 mmoL/L	140 mmoL/L	140 mmoL/L	135–147 mmoL/L
Serum potassium	4 mmoL/L	3.2 mmoL/L	4.2 mmoL/L	3.5–5 mmoL/L
Serum chloride	106 mmoL/L	100 mmoL/L	110 mmoL/L	100–110 mmoL/L
Blood urea	16 mg/dL	20 mg/dL	18 mg/dL	5–20 mg/dL
Serum creatinine	0.8 mg/dL	0.8 mg/dL	1.2 mg/dL	0.8–1.2 mg/dL
Serum bilirubin	0.6 mg/dL	0.8 mg/dL	1.2 mg/dL	0.2–1 mg/dL

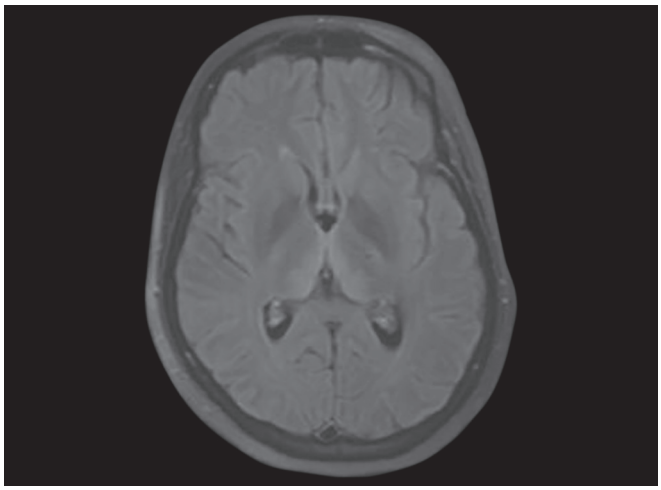

Fig. 1: Shows MRI

Over a period of 28 days, her vision improved, her lower limb muscle power improved, and she regained her normal sensorium. She was discharged from the hospital after 34 days. On antenatal review, she was normal, reached 36.5 weeks, and underwent lower segment cesarean section for fetal distress in an emergency with a 2.6 kg baby. Her postoperative period was normal.

Table 1 shows the comparison of laboratory investigations on day 1, day 10, and day 24 with normal levels.

Discussion

Wernicke's encephalopathy, a reversible neurological disorder, is seen in HG because of thiamine (B1) deficiency, a cofactor required in various phases of carbohydrate metabolism. During pregnancy, the basal metabolism is very high, and when is associated with HG, the cells lack thiamine, it affects energy production resulting in neuronal damage.[1] Another postulation is that thiamine deficient cells cannot maintain an osmotic gradient leading to edema of intra and extracellular spaces.[5] The thiamine levels fall during pregnancy with starvation in hyperemesis, but if before thiamine administration, glucose is supplemented, the fall of thiamine levels is drastic, resulting in neuronal damage. In addition, the fetus and placenta also take up the vitamin thiamine resulting in poor pregnancy sequelae as spontaneous abortion and fetal loss.[6]

Presentation of women with nonspecific symptoms, neurologic signs, and non-reliable blood investigations make the diagnosis difficult. So Caine's criteria are suggested for diagnosis, which includes that the woman should have at least two signs out of four like dietary deficiency, oculomotor abnormality, cerebellar dysfunction, and memory impairment.[7]

Typical bilateral and symmetrical hyper-intense lesions in the thalamus, around the third ventricle on T2 weighted and FLAIR are characteristic of WE. Magnetic resonance imaging has specificity of 93% and sensitivity of 53% in diagnosing WE. But a normal MRI doesn't rule out WE.

Our patient had a typical history of severe vomiting (HG), with starvation which led to WE. She came with ptosis, blurred vision, altered sensorium, and weakness in the legs. Ketones were in large amounts. MRI of the brain though showed minimal changes, treatment was started immediately. ACOG 2018 mentions that ketosis should be corrected promptly.

The MRI of the woman showed complete resolution of changes at 4 weeks. Some case studies of hyperemesis associated with hypernatremia, hypokalemia, or hypophosphatemia results in CPM.[6]

Thiamine deficiency due to starvation from hyperemesis is associated with poor maternal and neonatal outcomes with a mortality of 40% in neonates due to small for gestational age babies. As per the European Federation of Neurological Societies (EFNS) recommendation, thiamine is to be supplemented in dose of 200 mg thrice daily via intravenous route prior to glucose infusion and is continued till improvement in signs and symptoms.

In 1994, the beneficial effect of steroid therapy in WE was postulated to reduce the edema around the thalami and maxillary body, and thus stabilization of blood–brain barrier.

Conclusion

Wernicke's encephalopathy is potentially reversible neurological complication of hyperemesis gravidarum, which if diagnosed early and treated promptly can result in successful outcome of pregnancy. Thiamine supplementation for 4 weeks, electrolytes, and glucose infusion homeostasis are very essential treatment modalities in women with HG to prevent CPM.

It is very essential to keep the diagnosis of WE in mind with the triad of ocular symptoms, altered sensorium, weakness of extremities, and immediate supplementation of thiamine in antenatal women with prolonged vomiting in pregnancy, especially before starting intravenous glucose.

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