Case Report

Wernicke's encephalopathy following hyperemesis gravidarum

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Abstract

Wernicke's encephalopathy (WE) is a potentially reversible yet serious neurological manifestation caused by vitamin B_1 (thiamine) deficiency. It is commonly associated with heavy alcohol consumption. Other clinical associations are with hyperemesis gravidarum (HG), starvation, and prolonged intravenous feeding. Most patients present with the triad of ocular signs, ataxia, and confusion. It can be associated with life-threatening complication like central pontine myelinolysis (CPM). We report two cases of WE following HG, with two different outcomes.

Keywords: Central pontine myelinolysis, hyperemesis gravidarum, Wernicke's encephalopathy

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Introduction

Wernicke's encephalopathy (WE) is a rare neurological disorder, due to thiamine deficiency and is precipitated by administration of glucose-containing fluids before thiamine supplementation. 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.[1] It is typically diagnosed among alcoholics (12.5%), but in nonalcoholics prevalence varies from 0.04-0.13%.[2] Almost 80% cases remain undiagnosed, as the majority are diagnosed on autopsy.[3] Many cases of WE in pregnancy with hyperemesis gravidarum (HG), were first reported in 1914.[2] There is no specific laboratory test available to support the diagnosis. Hyperemesis can further be complicated by life-threatening condition like central pontine myelinolysis (CPM) due to electrolyte fluctuations.[4] We report two cases of WE following HG, where clinical findings were supported by magnetic resonance imaging (MRI) of brain.

Case Report

Case 1

A 25-year-old woman, at 22 weeks gestation, presented with excessive vomiting for several weeks followed by progressive weakness of lower limbs, altered sensorium, and blurred vision. Her previous pregnancy had resulted in fetal loss, due to hyperemesis. On admission, she had stable vitals, with 7/15 Glasgow Coma Scale (GCS). Physical examination revealed sluggishly reacting pupils with papilledema, left sided facial twitching with ptosis of the eye, bilateral conjugate palsies with nystagmus, and flaccid paralysis of all four limbs with bilateral flexor plantar reflexes. Fetal sonography was normal. Her serum biochemistry during her stay is shown in Table 1. MRI finding was suggestive of WE [Figures 1 and 2]. She was electively intubated and ventilated and was administered intravenous thiamine 100 mg thrice daily along with other vitamins, electrolytes, and trace elements. Gradually, her ocular signs disappeared and she showed neurological recovery with improvement in muscle power.

However, within next few days, her general condition deteriorated and she became hyperpyrexic (>40°C). A repeat MRI (brain) was highly suggestive of CPM [Figure 3]. She underwent lower segment cesarean

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section (LSCS) at 30 weeks and a male baby was delivered (weight 800 g, APGAR 6 and 8). Unfortunately, patient died of multiorgan failure on 4th postoperative day.

Case 2

A 23-year-old parturient was admitted with altered sensorium and acute renal failure after spontaneous abortion at 19 weeks gestation. She presented with excessive vomiting of several weeks duration. On admission she was confused and physical examination revealed restricted ocular movements, bilateral convergent, and horizontal nystagmus with hypotonia of all four limbs. Though her vitals were stable, she was in acute renal failure. Her serum biochemistry during her stay is shown in Table 2. MRI brain was highly suggestive of WE [Figure 4].

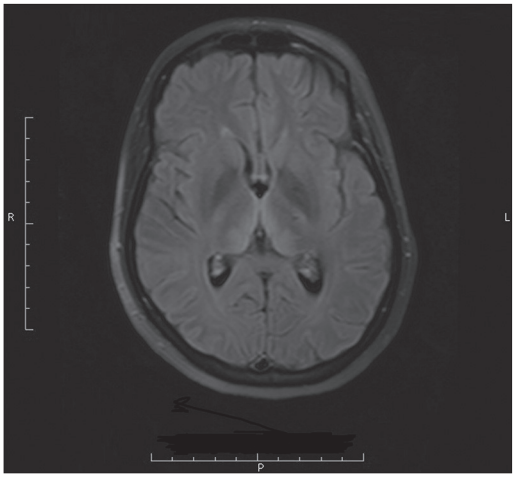

Figure 1: Bilateral symmetrical increased signal intensity of the posteromedial aspect of both thalami on fluid attenuated inversion recovery images suggesting Wernicke's encephalopathy

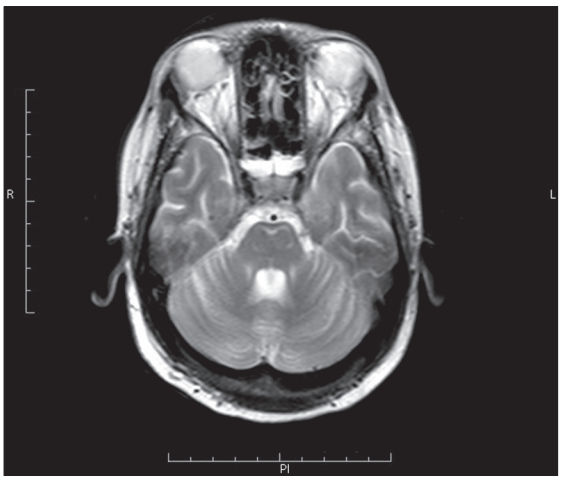

Figure 3: Ill-defined areas of T2 increased signal intensity within the pons sparing the corticospinal tract with no evidence of diffusion restriction and hypointense signal on T1 weighted images and the picture suggestive of central pontine myelinolysis

She received 300 mg/day of intravenous thiamine along with other vitamins, electrolytes, and trace elements. Her renal functions improved with renal support. She was discharged from hospital with significant neurological recovery. She was lost to follow-up, so repeat MRI could not be done.

Discussion

WE occurs due to deficiency of thiamine (B_1), which is an essential cofactor in various stages of carbohydrate metabolism. If the cells with high metabolic requirements have inadequate stores of thiamine, energy production drops, and neuronal damage ensues.[1] Body store of B1 falls rapidly during fasting is correct instead of Body stores B_1 of falls rapidly during fasting.[5] Intravenous dextrose administered before correction of thiamine will aggravate

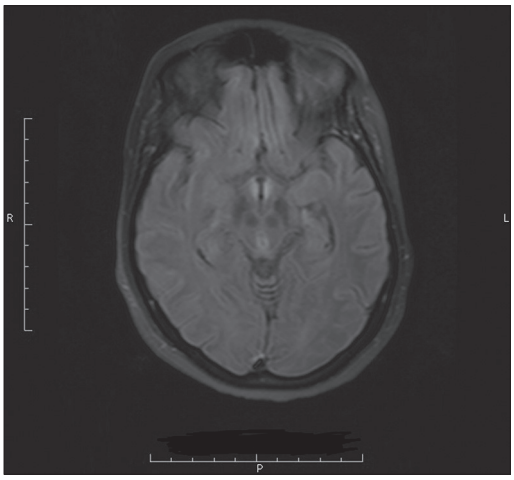

Figure 2: Increased signal intensity of the periaqueductal gray matter in the midbrain and mammillary bodies, with no change in the signal intensity seen in the T1 weighted images suggestive of Wernicke's encephalopathy

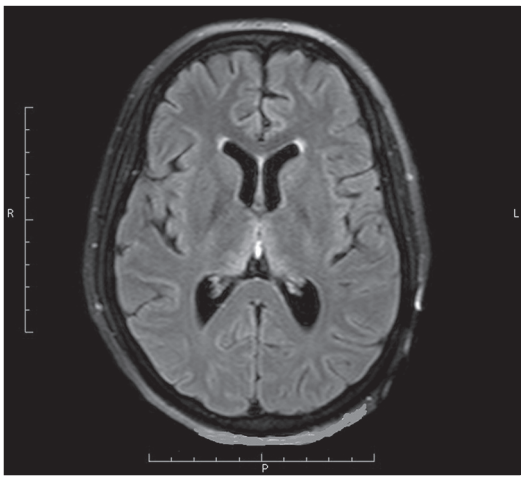

Figure 4: Generalized widening of extra-axial cerebrospinal fluid spaces in both supra- and infratentrorial region with periaqueductal hyperintensity signal in long reptition times images and similar changes were also seen involving mammillary bodies, T2 hyperintense signal at medial aspect of both thalami with diffusion restriction suggestive of Wernicke's encephalopathy

Table 1: Case I-Serum biochemistry

Test	Normal range	Day 1	Day 15	Day 30	Day 45
S. Chloride	100-110 mmol/L	106	111	99	126
S. Potassium	3.5-5 mmol/L	2.3	4.2	3	5
S. Sodium	135-147 mmol/L	139	140	145	154
S. Creatinine	40-90 μmol/L	105	45	36	126
S. Urea	2.5-6.7 mmol/L	3.6	1.1	3.1	8.6
S. Magnesium	0.65-1.1 mmol/L	1.09	0.67	0.73	1.14
S. Phosphate	0.7-1.4 mmol/L	0.13	0.9	1.32	1.5
S. Albumin	35-50 g/L	16	18	17	11
S. Bilirubin	3-17 μmol/L	15	14	4	30

S: Serum

Table 2: Case II-Serum biochemistry

Test	Normal range	Day 1	Day 7	Day 15
S. Chloride	100-110 mmol/L	81	101	109
S. Potassium	3.5-5 mmol/L	2.9	3.6	4.9
S. Sodium	135-147 mmol/L	139	139	143
S. Creatinine	40-90 μmol/L	519	406	201
S. Urea	2.5-6.7 mmol/L	44.7	22.5	11.3
S. Magnesium	0.65-1.1 mmol/L	0.96	1.19	1.11
S. Phosphate	0.7-1.4 mmol/L	0.36	1.32	1.12
S. Albumin	35-50 g/L	18	18	17
S. Bilirubin	3-17 μmol/L	96	39	4

S: Serum

matters further. In pregnancy it happens due to excessive vomiting, poor intake, and increased metabolic demand. In addition, sequestration of the vitamin by the fetus and placenta,[6] can have devastating complications like spontaneous abortion, fetal loss.[7] Lab assessment of blood transketolase activity and thiamine pyrophosphate (TPP) are not very reliable. MRI is the imaging modality of choice because it is highly specific (93%) and comparatively safer than computed tomography (CT) scan.

Our patients had a significant history of severe vomiting during pregnancy, with poor intake which led to WE. They presented with acute mental confusion, encephalopathy, and opthalmoplegia. MRI of the brain was diagnostic and treatment was started without any delay.

The follow-up MRI of first patient, at 4 weeks revealed complete resolution of WE, but features of CPM which could be attributed to severe electrolyte disturbances.[8] Adams *et al*., described CPM as a unique clinical entity in 1958.[9] Several case reports illustrate, that hyperemesis causing hypernatremia, hypokalemia, or hypophosphatemia eventually lead to CPM.[10] Moreover, thiamine deficiency may render the myelin sheaths of central pons more sensitive to changes in serum sodium, phosphate, and potassium.

Our second patient responded to treatment remarkably. Spontaneous abortion, indirectly improved her nutritional status and thiamine absorption.

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.[11] In nonalcoholic patients, an intravenous dose of thiamine 100-200 mg once daily could be enough; whereas in alcoholic patients, higher doses may be required.[12]

Conclusion

WE is a potentially reversible condition if treated early. Thiamine supplementation is crucial for women with HG. Moreover, replacement of electrolytes and glucose homeostasis is also important to prevent CPM. CPM occurs probably due to hypokalemia, hypernatremia and hyperosmolality in hyperemesis.[11] We would like to emphasize the importance of prompt thiamine supplementation in pregnant women with prolonged vomiting in pregnancy, especially before starting intravenous or parenteral nutrition.

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