Case report

DOI: https://doi.org/10.4038/sljpsyc.v15i1.8426

Wernicke encephalopathy due to hyperemesis gravidarum in pregnancy

N E Hendawitharana, G D Punchihewa

Abstract

Wernicke's encephalopathy is a life-threatening acute or sub-acute neurological emergency characterized by a triad of ataxia, confusion, ophthalmoplegia caused by thiamine deficiency, mostly observed in chronic alcohol users. Wernicke's encephalopathy due to causes other than alcohol abuse is uncommon and often misdiagnosed. In pregnancy, it is an unknown pathology, with poor prognosis, which occurs secondarily to hyperemesis gravidarum. We present a rare case of a 30-year-old female with mild learning disability, at 15 weeks of gestational age, with a history of previous hospital admission for severe vomiting and referred to Maternal Mental Health Clinic for abnormal behaviour. She had classic symptoms of Wernicke's Encephalopathy on presentation, and was immediately treated with thiamine. Her symptoms improved in a few days.

Keywords: Wernicke's encephalopathy, hyperemesis gravidarum

SL J Psychiatry 2024; 15(1): 55-57

Introduction

Wernicke's encephalopathy (WE) is a potentially fatal, but avoidable neuropsychiatric syndrome due to thiamine deficiency, first described in 1881 by Carl Wernicke. The clinical presentation is extremely varied with the presence of the typical clinical triad, for most cases (confusion and ataxia, ophthalmoplegia). However, it may be incomplete or associated with other clinical signs such as convulsion, dysarthria. Alcohol dependence is the most common cause but it can also result from an unbalanced diet, vomiting, anorexia nervosa, untreated inflammatory bowel disease, and bariatric surgery. The relation between Wernicke encephalopathy and hyperemesis gravidarum was described in 1939 by Sheehan. Wernicke encephalopathy in pregnancy generally occurs in women who are malnourished, due to the increased demands of pregnancy. Hyperemesis further depletes thiamine stores (1). Though, Wernicke's encephalopathy is uncommon in pregnancy, hyperemesis gravidarum is quite common. Wernicke encephalopathy generally occurs at 14 to 18 weeks gestation after two to three weeks of vomiting (2). Therapeutic management consists of the administration of thiamine as soon as possible to avoid sequel of irreversible neurological damage. We report an unusual etiology of Wernicke's encephalopathy: hyperemesis gravidarum.

Case report

A 30 year old, pregnant patient with a period of gestation of 15 weeks was referred to us with confusion and abnormal behavior, suspecting a psychiatric illness. The patient had a history of being successfully treated for a urinary tract infection, followed by being treated with anti-emetics and intravenous fluids for hyperemesis gravidarum in another hospital and discharged with much improvement two weeks prior to presentation. During the following 10 days, she was noticed to be withdrawn, preoccupied, with poor sleep and refusing meals. Three days prior to admission she had excessive fear about falling, refusing to walk, visual disturbances and suspiciousness. She had been easily irritable with intermittent crying spells and had not been able to recognize familiar faces. On examination patient was awake, restless, distracted and fearful, responding to non-apparent stimuli. She followed one step commands on repeated verbal stimuli. Her affect ranged from anxious to dysphoria and there was lability of mood. She had persecutory ideas, visual and auditory hallucinations. She was not oriented to time and place and had poor insight. She was hemodynamically stable. The neurological examination revealed loss of equilibrium with incoordination of gait. The power, tone, reflexes of bilateral upper and lower limbs were normal. The pupils

were bilaterally equal and reactive. Ocular movements showed restricted bilateral convergence, ocular clonus, with multidirectional nystagmus.

Laboratory tests showed the following changes: drop in albumin (27 g/L, reference: 35-53 g/dL), calcium (2.03mmol/L, reference: 2.1-2.57mmol/L), magnesium (0.64 mmol/L, reference: 0.72-1.06mmol/L), and potassium (3.3 mmol/l, reference: 3.5-5.5 mmol/l) with normal full blood count indices and liver enzymes and renal profile. Blood picture revealed features suggestive of mixed deficiency. Transient hyperthyroidism was also observed, with TSH (0.065mU/L, reference 0.3-4.0 mU/L) free T4 (0.74mU/L, reference: 0.9-1.7mU/L) below normal limits. Ultrasound showed single live intrauterine fetus. Magnetic resonance imaging (MRI), was not done due to unavailability, and as patient was confirmed to have Wernicke's Encephalopathy clinically by a consultant physician and neurologist. She responded well with thiamine replacement

Due to the main diagnostic suspicion of WE, thiamine replacement was initiated with a 500 mg intravenous loading dose every eight hours for five days, and then maintained with 100 mg orally for another 14 days. The patient also received potassium replacement orally, after which she showed an improvement in her neurological condition, being discharged after seven days of hospitalization for prenatal follow-up.

Discussion

Wernicke's encephalopathy is a neuropsychiatric syndrome due to a potentially fatal but preventable thiamine deficiency, characterized by the classic triad of encephalopathy, ophthalmoplegia, ataxia, and mental confusion (3). Thiamine, a water soluble vitamin provided by diet, is an essential cofactor of three enzymes involved in energy metabolism, in glucose degradation. The body stores 25 to 30 mg of thiamine (B1), which is good for about 18 days. The daily requirement is 0.4 mg/1,000 kcal per day, which increases in pregnancy to 1.5 mg/day (4). Deficiency of thiamine affects multiple tissues particularly those with high thiamine turnover which includes neural parenchyma resulting in cell necrosis or apoptosis (2).

The diagnosis of WE is primarily clinical. In the operational criterion for the identification of Wenicke's encpahlopathy, Caine et al (5) proposed that Wernicke's encephalopathy is diagnosed if any two of the following four signs exist: ophthalmoplegia, ataxia, altered mental status, and malnourishment (5). Nystagmus is the most common ocular sign and confusion is the most common presenting symptom (5) which was also evident in our patient.

Although Wernicke's encephalopathy is reversible, major complications can arise in the pregnant woman and her unborn child without active management. They are permanent neurologic lesions and Korsakoff syndrome, which is fatal in 10-20% of cases, and may also lead to miscarriage, preterm birth, and intrauterine growth retardation. Therefore, any pregnant woman with frequent vomiting should receive thiamine supplements, and if she develops any neurological manifestation, Wernicke's encephalopathy should be considered. Wernicke's encephalopathy can progress to Wernicke-Korsakoff syndrome, resulting in more chronic symptoms such as anterograde amnesia, which takes more time to resolve after treatment. Therefore, diagnosis and treating as fast as possible will result in less severe disease. The gold standard treatment is to replace thiamine which results in the resolution of symptoms in a few hours to a few days depending on the severity of the disease.

Conclusion

Wernicke's encephalopathy is an uncommon and life-threatening neurological disease complicating pregnancy in the setting of hyperemesis. The diagnosis is primarily clinical. Timely and proper management of vomiting in pregnancy and early replacement of thiamine will prevent development of serious neurological consequences and maternal and fetal morbidity.

Acknowledgements

The authors acknowledge the patient and her relatives for giving written informed consent to publish the case history.

Statement of contribution

All the authors were involved in the conception, literature survey and writing of the manuscript. All authors approved the final draft.

Declaration of interests

There are no conflicts of interest.

N E Hendawitharana, University Psychiatry Unit, Teaching Hospital, Karapitiya, Sri Lanka

G D Punchihewa, Department of Psychiatry, Faculty of Medicine, University of Ruhuna, Sri Lanka

Corresponding author: N E Hendawitharana

E-mail: nisalie@hotmail.com

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