Wernicke's syndrome associated with hyperemesis gravidarum

Mohamed Amine Bouslama, Afraa Brahim, Ahmed Nedhir Sfeyhi, Khalil Tarmiz, Khaled Ben Jazia

ABSTRACT

Introduction: Hyperemesis gravidarum during pregnancy can be complicated by Wernicke's encephalopathy (WE). It is a neuropsychiatric syndrome due to thiamine deficiency generated by increased requirement and loss. It is a fatal but preventable complication. The management and prevention of this disorder is still unclear for lack of guidelines.

Case Series: These are two cases of Wernicke's encephalopathy complicating hyperemesis gravidarum occurred in two women during the first trimester of pregnancy. Diagnosis was confirmed by MRI scan, and the treatment consisted of intravenous thiamine and parenteral nutrition with a good maternal and fetal outcome.

Conclusion: Wernicke's encephalopathy must be suspected in women showing its characteristic clinical signs to avoid worse outcome, such as maternal or fetal death or permanent neuromuscular sequelae. Medical management by thiamine is simple and efficient.

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CASE SERIES

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Introduction: Hyperemesis gravidarum during pregnancy can be complicated by Wernicke's encephalopathy (WE). It is a neuropsychiatric syndrome due to thiamine deficiency generated by increased requirement and loss. It is a fatal but preventable complication. The management and prevention of this disorder is still unclear for lack of guidelines. **Case Series:** These are two cases of Wernicke's encephalopathy complicating hyperemesis gravidarum occurred in two women during the first trimester of pregnancy. **Diagnosis** was confirmed by MRI scan, and the treatment consisted of intravenous thiamine and parenteral nutrition with a good maternal and fetal outcome. **Conclusion:** Wernicke's encephalopathy must be suspected in women showing its characteristic clinical signs to avoid worse outcome, such as maternal or fetal death or permanent neuromuscular sequelae. **Medical management** by thiamine is simple and efficient.

Keywords: Esophageal rupture, Hyperemesis gravidarum, Thiamine deficiency, Wernicke's encephalopathy

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INTRODUCTION

Hyperemesis gravidarum is uncontrollable vomiting during pregnancy that can lead to serious complications like liver damage, esophageal rupture or Wernicke's encephalopathy (WE), especially if it remains untreated.

Wernicke's encephalopathy is an acute neurological disorder related to poor thiamine absorption. It is a medical metabolic emergency which can lead to death if not managed aggressively. It was described by Carl Wernicke in 1881, in patients presenting with the triad of ocular signs, ataxia, and confusion [1].

There are currently no suitable, evidence-based guidelines for managing or preventing this disorder.

We report two clinical cases of WE in pregnancy presenting in the department of intensive care of Farhat Hached Teaching Hospital.

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CASE SERIES

Case 1

A 28-year-old primigravida with no notable medical history was hospitalized in ICU at 14 weeks gestation for uncontrollable vomiting occurs six times per day during eight weeks before hospitalization. Vomiting was clear and not associated with abdominal pain.

Despite initial therapy with antiemetics and intravenous glucose, the evolution was by the persistence of vomiting and the appearance of neurological disorders involving rotatory vertigo and horizontal nystagmus. Laboratory investigations had shown severe hypokalemia 2.1 mmol/L and mild liver cytolysis: aminotransferase 185 U/L. Cerebral MRI scan had been demanded because of neurological signs. It showed periventricular and periaqueductal hyper intensities in T2 sequence affirming WE syndrome. Therapy consisted on metoclopramide 10 mg intravenously 8 hours, rehydration with 2000 ml saline solution with added potassium 20 mmol given over four hours, and thiamine supplementation 500 mg/day. Neurological signs disappeared after 48 hours of hospitalization. Total recovery was noted after 12 days. The patient delivered eutrophic baby at 40th gestation week with good outcome.

Case 2

A 24-year-old pregnant woman at 19th gestation week was admitted in the ICU with the history of excessive vomiting for several weeks followed by progressive weakness of lower limbs. Vomiting was blood-streaked with epigastric pain. At the admission, the physical examination found a drowsiness and bilateral nystagmus.

Serum biochemistry showed altered hepatic indices: aminotransferase 67 U/L, acute renal failure: creatinine clearance: 43 ml/min, and lactic acidosis: 3.6 mmol/l.

A metabolic encephalopathy was suspected. The cerebral MRI scan revealed a neuroradiologic picture compatible with WE: images of signal-intensity alterations with different intensity patterns are seen in the thalami, signal-intensity alterations in the mammillary bodies (Figures 1 and 2)

The patient was treated with 500 mg of thiamine per day associated with total parenteral nutrition. The fetal well-being was good, checked daily.

Vomiting and drowsiness had disappeared after 48 hours. Nystagmus had regressed at 13 hospital days, so thiamine supplementation was stopped. Maternofetal outcome was good.

DISCUSSION

Hyperemesis gravidarum is pathology of the first trimester of pregnancy which impact is about 0.6% [2]. It can be complicated by WE in case of thiamine deficiency.

In fact, thiamine plays a vital role in the metabolism of carbohydrates. Thiamine is a cofactor for several essential enzymes in the Krebs cycle and the pentose phosphate pathway [3].

In the setting of thiamine deficiency, thiamine-dependent cellular systems begin to fail, resulting eventually in cell death that feeds the localized vasogenic response. Thiamine-dependent enzymes play an essential role in cerebral energy utilization that's why thiamine deficiency can cause brain tissue injury most notably in regions with higher metabolic demands.

Diagnosis is based on the classic clinical triad made by encephalopathy, ataxic gait, and some variant of oculomotor dysfunction (nystagmus in 93% of cases). But symptoms may be vague and non-specific, i.e., headaches,

Figure 1: FLAIR coronal images signal-intensity alterations with different intensity patterns are seen in the thalami, signal-intensity alterations in the mammillary bodies.

Figure 2: T2-weighted images showing the high signal intensities in the bilateral paramedian thalami with mammillary bodies.

fatigue and irritability [4].

Magnetic resonance imaging scan is the most valuable method to confirm the diagnosis with a 93% of specificity.

The typical signs of WE are increased periventricular and periaqueductal signal Intensity (FLAIR and T2) and mammillary bodies [5].

Wernicke's syndrome is fatal but reversible medical emergency. Neurologic deficits could persist despite treatment. Mortality range is about 20% [6].

If WE is suspected, parenteral thiamine should be immediately given at the dose of 500 mg/day during the two first days then 250 mg/day until the resumption of oral feeding [7].

Some authors recommend the thiamine intake until the delivery [8]. Wernicke's syndrome threaten the fetal prognosis. It is associated with an increased risk of low birth weight, neurodevelopmental disorders, intrauterine growth restriction, preterm delivery, and fetal and neonatal death. Ischemic stroke can be seen in fetuses of women affected by US with no specific explanations [9].

Early diagnosis and vitamin therapy within the first 24 hours prevent fetal worse outcome [10]. Anti-emetics are also useful as adjoining therapy to reduce the intensity of vomiting.

A chronic WE can develop in Korsakoff syndrome which consists in impairment in memory with loss of working memory. Moreover, in severe forms of WE, muscular sequelae are possible [8].

For our two patients, no incidents had been recorded, and the remission was complete.

CONCLUSION

Wernicke's syndrome is a rare but serious neurologic complication of hyperemesis gravidarum that must be suspected in pregnant women in the first trimester showing the clinical symptoms. Magnetic resonance imaging findings confirm the diagnosis. Maternofetal outcome depends on the rapidity of the treatment by thiamine.

Author Contributions

Mohamed Amine Bouslama – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Afraa Brahim – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Ahmed Nedhir Sfeyhi – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Khalil Tarmiz – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Khaled Ben Jazia – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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