Wernicke’s encephalopathy in a patient with hyperemesis gravidarum, hepatitis A and pancreatitis

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DESCRIPTION
A pregnant woman in her early 20s, G3P1011, with no medical history, came to the hospital because of altered mental status, nausea, vomiting and weakness. At her 6 weeks’ gestation, she developed severe nausea and vomiting and was treated for hyperemesis gravidarum (HG). The symptoms did not resolve, and she was admitted in another institution. The patient was diagnosed with pancreatitis and hepatitis A, and 2 days later, she left the hospital against medical advice. The patient continued with the nausea and vomiting secondary to possible pancreatitis and hepatitis A. At 15 weeks’ gestation, she visited the emergency room of our hospital with severe nausea, vomiting and altered mental status.

The patient had more than 8 weeks of persistent nausea and vomiting, decreased oral intake, and a new onset of somnolence, confusion, and unstable ambulation. In the emergency department, on the physical examination, cardiopulmonary and abdomen were normal. The abdomen was not distended, and the uterus was palpable at the midpoint between the umbilicus and the pubic symphysis. The patient was confused, able to nod to commands, but dysarthric, and unable to ambulate due to imbalance. Laboratory studies revealed total bilirubin of 3.4 mg/dL, aspartate transaminase of 291 U/L, alanine transaminase of 256 U/L and lipase of 984 U/L. Abdominal ultrasound and CT scan of the brain were normal. MRI of the brain revealed increased intensity of the bilateral mammillary bodies, dorsomedial thalami and periaqueductal grey matter (figure 1).

The patient was diagnosed with Wernicke’s encephalopathy (WE) secondary to HG, pancreatitis and hepatitis A. She was started on thiamine 500 mg intravenously three times a day for 2 days, followed by 250 mg intravenously daily for 5 days in combination with other prenatal vitamins such as folic acid and vitamin B complex. She was discharged to acute inpatient rehabilitation on oral thiamine and multivitamins. At the time of discharge, she was oriented to person, time, place and situation, speech clear and able to perform all her activities of daily living with assistance.

WE is an acute neuropsychiatric emergency resulting from a deficiency of thiamine. In industrialised countries, 90% of the cases of thiamine deficiency are associated with alcohol misuse. Non-alcoholic causes of WE such as gastrointestinal surgery, starvation and HG are less frequent.

Rane et al described nausea affecting up to 80% of pregnant women; however, HG affects up to 3% of pregnant women. It may lead to severe nutritional deficiency in the mother and the fetus. In our patient, the combination of increased demand during pregnancy, deficient oral intake secondary to nausea and vomiting, malabsorption during hepatitis A and HG, and the lack of thiamine deficiency prophylaxis were the causes of WE. Green top guideline recommends prophylaxis for WE with 100 mg of parenteral thiamine each day the patient received intravenous dextrose. During and after previous hospital admission, our patient, with more than 8 weeks of vomiting, did not receive thiamine supplements.

In previous admission, WE was not diagnosed in our patient because altered mental status was not present. Young age is a protective factor against all forms of altered mental status. Oudman et al reported that in a young woman with HG, mental alterations were not the most common presenting sign within the triad of WE.

Wijnia et al reported infections in 35 of 68 patients during the initial phase of Wernicke-Korsakoff encephalopathy, one of them with pancreatitis. We believe that comorbid infections such as pancreatitis and hepatitis A in our patient may be symptoms of thiamine deficiency and a marker of more severe malnutrition.

Liver function abnormalities have been reported in up to 67% of patients with HG. In our patient, liver function abnormalities were elevated secondary to hepatitis A and HG.

In a review by Oudman et al, pregnant patients became thiamine depleted between 10 and 15 weeks of gestation and have been vomiting for a median of 7 weeks before developing WE. Severe complications such as pregnancy loss and maternal death
Images in... were observed in 50% and 5% of cases, respectively. The history of nausea and vomiting related to HG, hepatitis A and pancreatitis sets our patient apart from other patients with concurrent HG and WE. Our patient started with symptoms earlier during pregnancy which lasted for more than 8 weeks. Our patient did not have any pregnancy complications and followed up in the obstetrics clinic at 20 weeks of gestation. At that time, she was alert, awake, oriented to time, person, place and situation. The physician considered that her overall medical condition improved.

Contributors HAB, BCB and DC conceptualised and designed the case. HAB, BCB and ZA performed data collection and analysis, and wrote the initial manuscript. HAB, BCB and ZA performed subsequent article revision, data integration, image collection and editing. DC was the attending physician in the case, obtained patient consent and performed final manuscript editing. HAB, BCB, ZA and DC reviewed the final manuscript and approved the final version to be submitted.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

REFERENCES

Learning points
► Hyperemesis gravidarum can be a cause of Wernicke’s encephalopathy (WE).
► Nausea and vomiting associated with hepatitis and pancreatitis can be associated with vitamin deficiency during pregnancy.
► Prompt administration of thiamine leads to rapid improvement of some of the symptoms in patients with WE.

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