

Wernicke’s encephalopathy secondary to hyperemesis gravidarum

Afshan Soomro, MD; Yelena Spitzer, MD; Shamantha Reddy, MD , FASA
Montefiore Medical Center, Department of OB Anesthesiology

Case Description

29-year-old female G2P1 was transferred to our hospital at 11 weeks gestation for further evaluation of her deteriorating mental status and management of her pregnancy in the setting of hyperemesis gravidarum and she stayed in the hospital for 270 days and spent significant amount of time in the intensive care unit (ICU).

Past Medical History and clinical course

The Patient had a history of gallstone pancreatitis s/p ERCP with biliary and pancreatic duct stents, sphincterotomy at the time of her presentation. As per history from a close family member, patient carried out her normal daily activities without problems before her pregnancy but due to severe emesis early in her pregnancy PO intake was decreased, and she reported feeling weak and having trouble walking. As her mental status changed abruptly a concerned family member brought her to the hospital. Along with hydration, addressing her nutritional status, repleting vitamins, and electrolytes, she underwent a series of tests to determine the cause of the change in her mental status.

Imaging

MRI showed hyperintensity in periaqueductal gray matter, hypothalamus, bilateral thalami and bilateral mammillary bodies consistent with Wernicke's encephalopathy (WE) .

The patient’s overall prenatal course remained complex and challenging to the entire multidisciplinary team, including prolonged care in ICU closer to the labor and delivery floor.

The patient was planned for induction at 35 week gestational age. An epidural catheter was placed for labor analgesia, patient delivered a baby (small for gestational age) with Apgar scores of 9 and 9. The patient was monitored during postpartum period and later was transferred to the floor. She is still currently admitted due to her mental status and deconditioning secondary to malnutrition and nosocomial infections during the prolonged hospital course. Her hospital course complications included acute kidney injury, COVID and pneumonia.

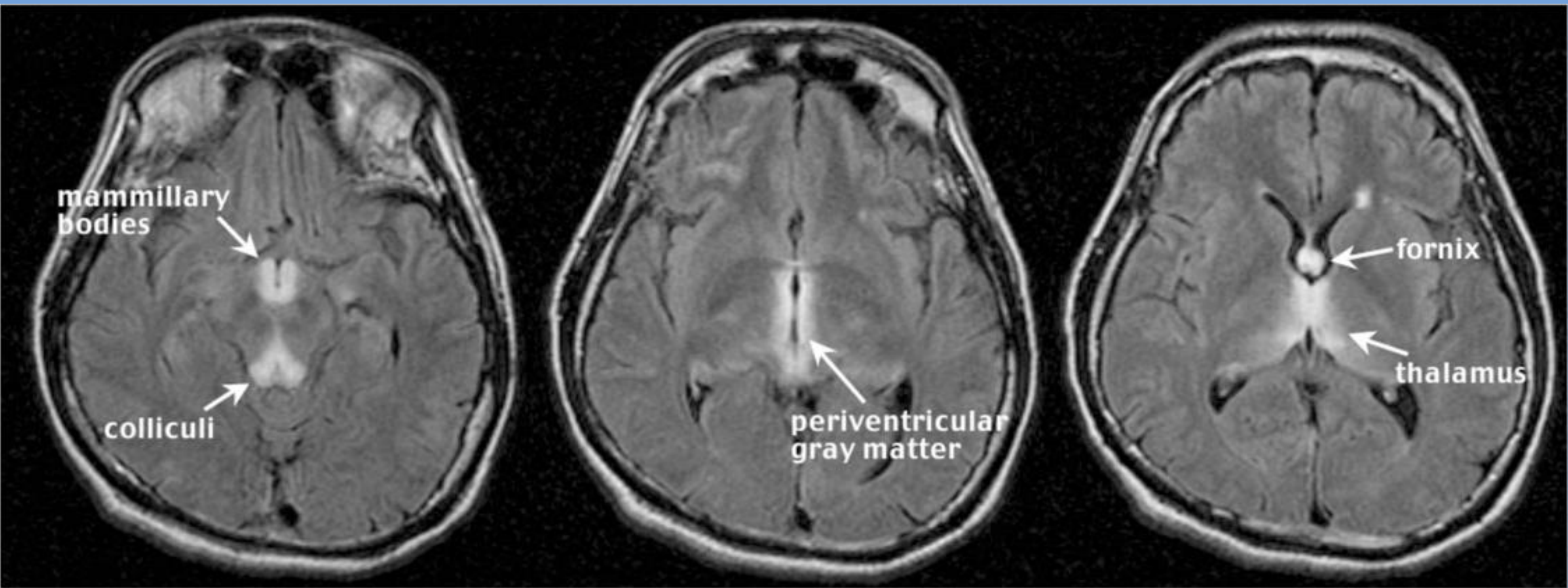
Pathology of Disease

- Wernicke’s encephalopathy is an irreversible central neurological disorder caused by severe thiamine (Vitamin B1) deficiency associated with significant morbidity and mortality if untreated. It is mostly observed in alcoholics and severely malnourished patients.
- The body stores about 25 mg to 30 mg of water-soluble vitamin thiamine (B1) which is good for about 18 days. The daily requirement is 0.4 mg/1000 kcal per day which increases in pregnancy to 1.5 mg/day. Deficiency of thiamine pyrophosphate, being an important co-factor for enzymes in the pentose phosphate pathway, affects multiple tissues particularly those with high thiamine turnover which includes neural parenchyma resulting in cell necrosis or apoptosis. WE can be precipitated in susceptible patients by administration of intravenous (IV) glucose before thiamine supplementation.
- The disease is characterized by Classic signs:
 - Encephalopathy
 - Oculomotor dysfunction
 - Gait ataxia

Common causes of delirium and confusional states

Muscle relaxers, polypharmacy
Nonprescription medications (Antihistamines) Drugs of abuse (ethanol, heroin, hallucinogens etc), Withdrawal states (eg, Ethanol, Benzodiazepines) Medication side effects (hyperammonemia from valproic acid, Serotonin syndrome)
Infections
Sepsis, Systemic infections fever related delirium
Metabolic derangements
Electrolytes disturbances (elevated or depressed) Na+, Ca+, Mg+, PO Endocrine disturbances (elevated or depressed) Thyroid, Parathyroid, Pituitary, adrenal Hypercarbia, hypoxia, hyperosmolar states or hypo-osmolar states, Inborn errors of metabolism: porphyria, Wilsons disease etc
Nutritional: Wernicke encephalopathy, Vitamin b12 deficiency, possibly folate and niacin deficiencies
Brain disorders
CNS infections: encephalitis, meningitis, brain or epidural abscess, Epilepsy Head injury, hypertensive encephalopathy

MRI findings



Pregnant female with severe emesis and presumed Wernicke’s encephalopathy. Flair images demonstrate typical findings of hyperintensity in the Mammillary bodies, periventricular gray matter.

Conclusions

WE is an uncommon and life-threatening neurological disease complicating pregnancy in the setting of hyperemesis. MRI is the investigation of choice. Timely and serious management of vomiting in pregnancy and replacing thiamine stores help treat acute neurological symptoms and can prevent both maternal and fetal morbidity.

References:

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