

Hyperemesis Gravidarum's Most Serious Sequelae

In the ICC, multiple patients present with hyperemesis gravidarum (HG). Patients range from vomiting for a couple of days to several weeks and some even have dramatic weight loss and nutritional decline. One main value that is used to determine the degree of dehydration is the amount of ketones in the urine. Patients are traditionally hydrated with normal saline or D5 and PO challenged until they can clear their ketones. If their ketones still persist after multiple boluses and urine catches, they are admitted to the hospital until their condition improves. In situations with concern for the patient's nutritional deficiency, thiamine was often given along with the treatment regimen. The article [Wernicke's Encephalopathy Following Hyperemesis Gravidarum](#) describes the extreme consequences of thiamine deficiency in hyperemesis gravidarum.

Background: Wernicke's encephalopathy (WE) is a rare but very serious neurological disorder that can result from **vitamin B1 (thiamine) deficiency**. It is **typically associated with alcoholism, but is also a result of hyperemesis gravidarum, starvation, and prolonged IV feeding**. The common symptoms include a triad of **ocular signs, ataxia and confusion, and can progress to the life-threatening condition Central Pontine Myelinolysis if electrolyte fluctuations are not controlled**. We'll discuss two cases of WE following HG.

Case 1: 25yo F, 22w gestation, with excessive vomiting for several weeks and progressive weakness of lower limbs, AMS, and blurred vision. GCS 7, with physical exam showing sluggishly reactive pupils, papilledema, L-sided facial twitching and ptosis, nystagmus and flaccid paralysis of all four limbs. Fetal sonography was normal. Her labs showed a K of 2.3 and Na of 139. MRI showed increased signaling of the bilateral posteromedial aspect of both thalami and of the periaqueductal gray matter in the midbrain and mammillary bodies, both suggestive of WE. She was intubated and ventilated and IV thiamine was administered TID along with other vitamins and electrolytes. Her symptoms gradually disappeared and she began to show signs of neurological recovery. After a few days, she deteriorated again and became febrile. A repeat MRI showed increased signaling in the pons sparing the corticospinal tract, highly suggestive of CPM. She underwent a C Section but died of multiorgan failure on POD 4.

Case 2: 23yo F presented with excessive vomiting for several weeks and was noted to have AMS and acute renal failure after spontaneous abortion at 19w gestation. Her physical exam revealed restricted ocular movements, nystagmus and hypotonia of all four limbs. She was in acute renal failure but with stable vitals. Her K was 2.9 and Na was 139. Her MRI showed signs of WE, including hyperintense signaling at the medial aspect of both thalami. She received IV thiamine with additional vitamins and electrolytes. Her renal function improved and she was discharged from the hospital after several days with significant neurological recovery, but was unfortunately lost to follow up.

Discussion: Thiamine is **essential in carbohydrate metabolism** and the lack of thiamine can cause energy production to drop, leading to the neurologic damage of WE. Administering IV dextrose before the thiamine is corrected will worsen the situation (controversial). In pregnancy, thiamine stores are depleted due to excessive vomiting, poor intake, and increased metabolic

demand. The TTP (thiamine pyrophosphate) lab value is not very reliable, so **MRI** is the diagnostic modality of choice due to its high specificity and safety compared to the CT scan. The MRIs along with the clinical correlations confirmed WE in both of our case patients. This allowed treatment to be started early and efficiently. In the first patient, the follow-up MRI revealed resolution of the WE but features suggestive of CPM likely due to the electrolyte disturbances. This is related to the fact that thiamine deficiency may change the myelin sheath and make it more sensitive to changes in the Na, P, and K levels. The second patient responded well to treatment, likely because her spontaneous abortion indirectly improved her nutritional status and thiamine absorption.

Take-home Point: WE is a potentially reversible condition if it is diagnosed and treated early. It is crucial to treat women with HG and a compromised nutritional status. According to the Guidelines by the European Federation of Neurological Societies (EFNS), IV thiamine should be given 200 mg TID and before any carbohydrate substance, and given for as long as there continues to be neurologic improvement.

References/Further Reading

- Kantor S, Prakash S, Chandwani J, Gokhale A, Sarma K, Albahrani MJ. Wernicke's encephalopathy following hyperemesis gravidarum. Indian J Crit Care Med 2014;18:164-6
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