HYPEREMESIS GRAVIDARUM-INDUCED WERNICKE’S ENCEPHALOPATHY

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INTRODUCTION: Wernicke’s Encephalopathy (WE) is a rare neurological disorder [1], caused by Vitamin B1 (thiamine) deficiency, that potentially reversible when diagnosed and treated early [1]. Majority of cases reported are among alcoholics, less common cases seen in starvation, prolonged parenteral nutrition and hyperemesis gravidarum. [2] Diagnosis is clinical [1], with high incidence of encephalopathy, ataxia and oculomotor abnormalities. There are no specific diagnostic laboratory tests available but there are high sensitivity MRI findings for diagnosis. Due to the clinical severity and mortality associated with WE, and the potential reversibility with timely treatment.

CASE PRESENTATION: A 40-year-old woman, 15 weeks pregnant, came to the emergency department complaining of generalized weakness and excessive vomiting since the beginning of her pregnancy. On admission was hypotensive, tachycardic, dry oral mucosae, GCS of 15. Fetal ultrasound revealed fetal demise and was admitted to the ICU where she was treated with intravenous hydration, correction of metabolic acidosis, acute kidney injury and electrolyte derangements. Her mental status progressively declined, eventually became comatose. An extensive work up for infectious and autoimmune meningoencephalitis and stroke was unrevealing for a cause. Was given empiric antibiotics and steroids without any discernible improvement. Intravenous thiamine 500 mg every 8h was instituted empirically for suspected WE and by the third dose showed improvement in clinical status. GCS improved, followed simple commands, motor function improved and resolved nystagmus. MRI brain revealed increased signal intensity in the medial thalamic and the periaqueductal gray matter on FLAIR and T2-weighted pulse sequences, consistent with WE. Clinical condition continued to gradually improve.

DISCUSSION: WE is a rare neuropsychiatric condition caused by deficiency of thiamine, either by decreased intake or absorption. Vitamin B1 is an essential cofactor for important enzymes in the Kreb’s and Pentose Phosphate Cycle. With thiamine deficiency, there is reduced energy production, lactic acid accumulation and neuronal depolarization and necrosis due to excitotoxicity. [3] Majority of diagnosed cases are observed in alcoholics and also with malnutrition and hyperemesis gravidarum. The prevalence of non-alcoholic cases diagnosed is rare. Treatment with high dose intravenous thiamine [3] can reverse the syndrome in some cases. Clinical improvement has been observed within a few hours, with ocular signs improving first followed by confusion.

CONCLUSIONS: WE is a medical emergency that requires high level of clinical suspicion and when promptly treated with high dose intravenous thiamine, can potentially reverse, within hours, the neurological signs and prevent irreversible neurologic damage and death.


DISCLOSURES: No relevant relationships by Sandra Donahue, source=Web Response

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