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Abstract

Emergency physicians frequently treat hyperemesis gravidarum and should be aware of possible complications. Wernicke encephalopathy secondary to thiamine deficiency should be considered in the differential diagnosis of acute encephalopathy in pregnant women. A seventeen-week pregnant 27-year-old woman presented to the Emergency Department with nausea, emesis, and right upper quadrant abdominal pain. Ultrasound diagnosed gallbladder sludge. Surgical consultant offered cholecystectomy versus expectant management. She improved with IV hydration, ondansetron, and was discharged on hospital day 3 with a diagnosis of hyperemesis gravidarum and gallbladder sludge. Three days later she presented with continued emesis and altered mental status. She and family members denied alcohol or illicit drug use. Vital signs were pulse 99/minute, blood pressure 115/70, temperature 36.4°C, respiratory rate 18, and oxygen saturation 99%. Neurological examination was delirium, variable mentation, and inability to follow commands. She had internuclear ophthalmoplegia with bilateral nystagmus. CT scan of brain was negative. MRI found abnormal T2-weighted signal in the central pons and medial thalami. Radiographic differential included central pontine myelinolysis, dysmyelinating conditions from malnutrition, toxic encephalopathy, and Wernicke encephalopathy. Thiamine level was below the limits of detection. Alcohol and urine drug screen were negative. Diagnosis was thiamine deficiency secondary to hyperemesis gravidarum with Wernicke encephalopathy. Emergency physicians frequently treat hyperemesis gravidarum. Nutritional status should be evaluated in patients who are unable to take neonatal vitamins. Awareness should exist of possible complications, including Wernicke encephalopathy secondary to thiamine deficiency.

Introduction

Hyperemesis gravidarum, defined as nausea and vomiting of pregnancy (1,2,3), is commonly seen in the Emergency Department, particularly during the first trimester of pregnancy. The approach to management is antiemetics, rehydration with glucose containing fluids, and reversal of urine ketones. Criteria for admission is a failure of a trial of oral feeding and inability to reverse urine ketones in a timely fashion. Nutritional status is not always addressed in the Emergency Department, though a controlled study of hyperemesis gravidarum patients found that the mean dietary intake of most nutrients fell below 50% of the recommended dietary allowances and differed significantly from pregnant patients without hyperemesis gravidarum (3). More than 60% of the patients had suboptimal biochemical status of thiamine, riboflavin, vitamin Bs, vitamin A, retinol-binding protein, vitamin C, calcium, and albumin (3). Wernicke encephalopathy, with variable degrees of confusion, ocular abnormalities, and ataxia due to thiamine deficiency, is an unusual but preventable complication of hyperemesis gravidarum (4), as is beri beri (5). A case is presented of a pregnant woman with severe hyperemesis gravidarum who presented with mental confusion and a thiamine level below the limits of detection.

Case Report

A 27-year-old woman with no prior pregnancies presented to the hospital 17 weeks pregnant with nausea, emesis, and right upper quadrant abdominal pain. Ultrasound diagnosed gallbladder sludge. Lipase and hepatic panel were negative. Surgical consultant offered acute cholecystectomy versus expectant management. She improved with IV hydration, ondansetron, and was discharged on hospital day 3 with a diagnosis of hyperemesis gravidarum and gallbladder sludge. Three days later she presented with continued emesis and altered mental status. She and family members denied alcohol or illicit drug use. Vital signs were pulse 99/minute, blood pressure 115/70, temperature 36.4°C, respiratory rate 18, and oxygen saturation 99%. Neurological examination was delirium with waxing and waning of mentation. She was intermittently unable to follow commands. She was intermittently able to participate in examination. Cranial nerve examination was with obvious internuclear ophthalmoplegia with nystagmus in the abducting eyes bilaterally. CT scan of brain was negative. MRI found abnormal T2-weighted signal in the central pons and medial thalami (Figure 1). On the Flair T2 coronal view, the medial thalamus and pons had abnormal signals (Figure 2). Radiographic differential included central pontine myelinolysis, dysmyelinating conditions from malnutrition, Wernicke encephalopathy, toxic encephalopathy from alcohol use or other drugs. Thiamine level was below the limits of detection. Alcohol and urine drug screen were negative. Diagnosis was thiamine deficiency secondary to hyperemesis gravidarum with Wernicke encephalopathy. She was treated with intravenous thiamine. Ultrasound demonstrated fetal demise so dilatation and curettage was performed. Hospital course was complicated by the development of hypercalcemia secondary to hyperparathyroidism for which she underwent resection of a 2 gram left parathyroid adenoma with normalization of parathyroid hormone levels. An upper extremity venous thrombosis was treated with fractionated heparin with transition to

warfarin. A repeat MRI two weeks after the initial MRI revealed abnormal signals in the pons and thalami that were unchanged from the initial MRI. Discharge to a skilled nursing facility was recommended, but her family elected to treat her at home with a home health aid.

Discussion:

Hyperemesis gravidarum is a common complication of pregnancy that is often severe enough to require Emergency Department treatment. Wernicke encephalopathy is a rare complication of hyperemesis gravidarum. A 2006 review of the world's literature identified 56 reported cases (5). Since that review, four more cases have been reported (6,7,8). Emergency physicians should be aware that nutritional deficiencies including thiamine deficiency can occur in woman with hyperemesis gravidarum. Historical information might be able to identify at risk individuals. Women who have been unable to keep down prenatal vitamins for days to weeks may be at greatest risk.

Women with hyperemesis gravidarum with ketonuria are rehydrated with glucose containing intravenous fluids. Emergency physicians give parenteral thiamine to alcoholics before administering glucose due to fears of precipitating Wernicke's encephalopathy from glucose infusion (9). A literature review concluded that the level of evidence is case reports, case series, animal studies, and expert opinion, but without "true clinical research" (10). Consideration of administering intravenous thiamine before rehydration with glucose containing intravenous fluids may be prudent in cases of severe hyperemesis gravidarum with the possibility of thiamine deficiency, particularly if there are other digestive issues such as the gallbladder sludge in this case.

Conclusion: Emergency physicians frequently treat hyperemesis gravidarum. Nutritional status should be evaluated in patients who are unable to take food or neonatal vitamins. Awareness should exist of possible complications, including Wernicke encephalopathy secondary to thiamine deficiency. Consideration of administering thiamine before rehydration with glucose containing fluids may be prudent in severe cases of hyperemesis gravidarum.

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Figure Captions

Figure 1. MRI AX FRFSE T2 Transverse image showing abnormal sign in pons.

Figure 2. Flaire T2MRI Coronal image showing abnormal sign in Medial Thalamus & pons.

Figure 1.

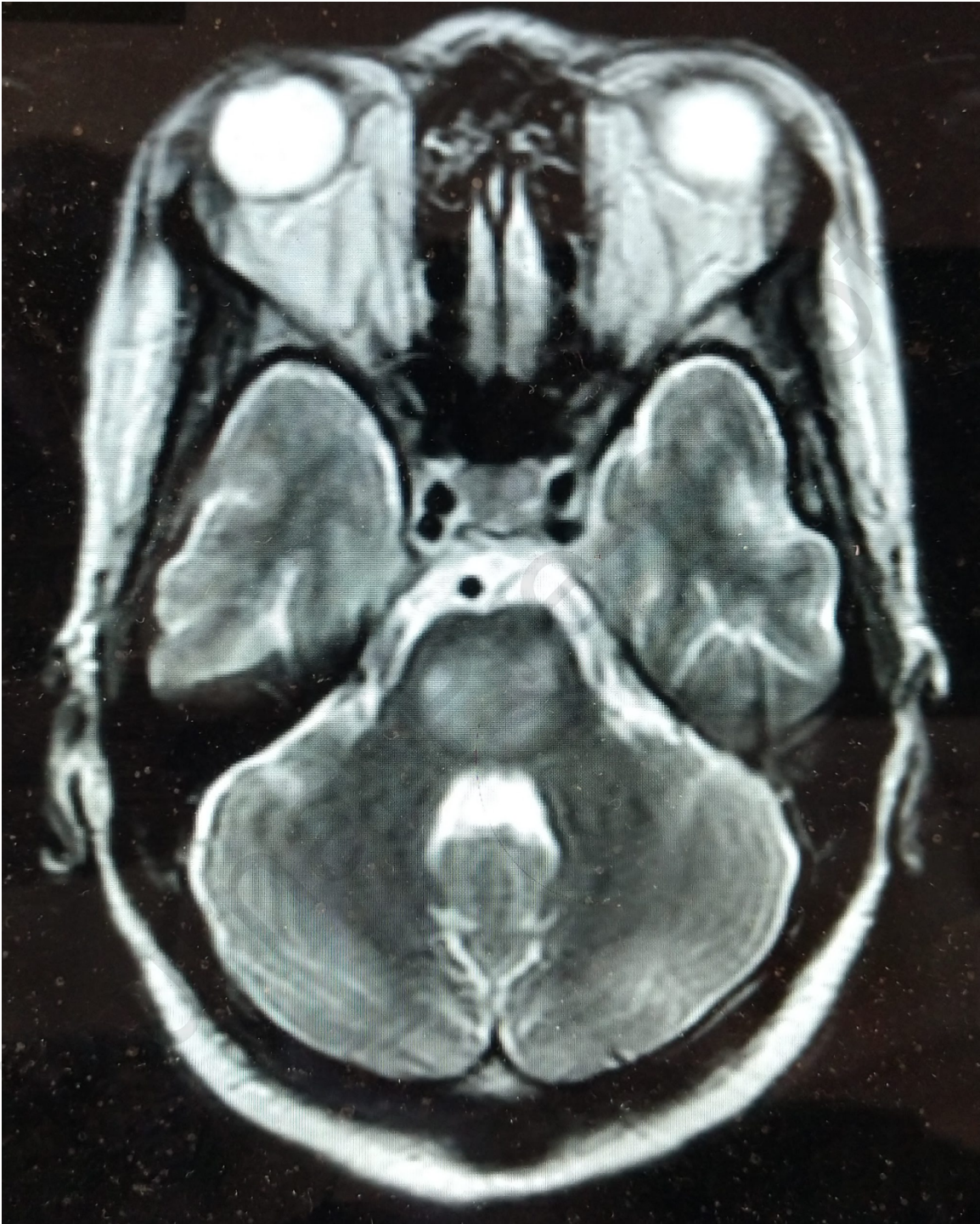


Figure 2.

