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Case Report

Hyperemesis Gravidarum Induced Wernicke's Encephalopathy

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Abstract

Wernicke' encephalopathy is a rare but potentially reversible neurological disorder characterized by ophthalmoplegia, ataxia and confusion. Wernicke's encephalopathy occurs as result of severe vitamin B1 (i.e., thiamine) deficiency. The most common etiology of Wernicke's encephalopathy is malnutrition among alcoholics. Here, we report an unusual case of hyperemesis gravidarum induced Wernicke's encephalopathy. A 25-year-old female, G2P1, with recent miscarriage at 20 weeks gestation and hyperemesis gravidarum presented with 1-month history of confusion, blurred vision, and progressive bilateral lower extremity weakness. The magnetic resonance imaging of brain revealed bilateral symmetrical hyperintensities in the thalami, mammillary bodies, and surrounding aqueduct of the midbrain suggesting Wernicke's encephalopathy. Patient was subsequently managed with aggressive thiamine replacement therapy. Her symptoms gradually improved, and she was eventually discharged to an inpatient rehabilitation facility for long term physical therapy. Wernicke's encephalopathy is a rare complication of hyperemesis gravidarum. This case highlights the importance of recognizing neurological changes due to thiamine deficiency during pregnancy. We would like to emphasize the importance of thiamine supplementation and monitoring in pregnant women with severe and prolonged vomiting history.

Keywords: Hyperemesis Gravidarum; Wernicke's Encephalopathy; Thiamine Deficiency; Ataxia; Confusion; Ophthalmoplegia

Abbreviations

WE: Wernicke's Encephalopathy; WBC: White Blood Cells; Hgb: Hemoglobin; ANA: Antinuclear Antibody; ss: Sjogren Syndrome; ss: Sjogren Syndrome; ACE: Angiotensin-Converting Enzyme; CRP: C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate; MRI: Magnetic Resonance Imaging; IV: Intravenous

Introduction

Wernicke' encephalopathy (WE) is a rare but potentially reversible neurological disorder characterized by ophthalmoplegia, ataxia and confusion. WE occurs as result of severe vitamin B1 (i.e. thiamine) deficiency and can manifest in necrosis of the thalamus, hypothalamus and mammillary bodies. WE is most frequently reported among alcoholics. Less common causes of WE have been reported in severe starvation, malignancy, anorexia nervosa, thyrotoxicosis and hyperemesis gravidarum [1]. However, the prevalence of nonalcoholic causes remains low, approximately 0.04% to 0.13% [2]. Here we report a case of Wernicke's encephalopathy as a result of hyperemesis gravidarum.

Case Presentation

A 25-year-old female, G2P1, with a recent miscarriage at 20 weeks gestation and hyperemesis gravidarum presented with 1-month history of confusion, blurred vision, and progressive bilateral lower extremity weakness which resulted in her inability to ambulate. She endorsed associated numbness and occasional

10/10 shooting pain in her lower extremities. Patient denied alcohol use and family history of neurological disorders. On admission, patient was afebrile, mildly tachycardic with heart rate of 107, blood pressure and respiratory rate were within normal limits. On physical exam, she was alert and oriented to self, place and time. However, patient was confused about the reason for being in the hospital. Her pupils were equal and reactive to light bilaterally. Ocular movements were intact without obvious signs of nystagmus. Patient has bilateral lower extremity weakness 3/5. Sensation to light touch was symmetrical and intact bilaterally. However, patient reported numbness sensation in the lower extremities. Straight leg raise test was negative for pain. Finger to nose test were negative bilaterally. Deep tendon reflexes were +2 throughout. Gait assessment was deferred due to patient's inability to ambulate. Lab findings include WBC at 7.0 X $(10)^3$/uL, Hgb at 11.3 g/dL, elevated homocysteine at 31.9 µmol/L, B12 and folate were within normal limits (705 pg/mL and 5.7 ng/mL, respectively). Neurology was consulted for initial concern of Guillain-Barre syndrome. However, autoimmune work up including ANA, Sjogren's ssA/ssB, ACE serum, CRP, ESR, para-neoplastic screen and infectious workup were all negative. MRI brain with and without contrast revealed bilateral symmetrical hyperintensities in the thalami, mammillary bodies, and surrounding aqueduct of the midbrain (Figure 1). These findings are typically seen in Wernicke's encephalopathy. MRI lumbar/thoracic/cervical spine was negative for any abnormalities.

Figure 1: MRI brain with and without contrast revealed bilateral symmetrical hyperintensities in the thalami, mammillary bodies, and surrounding aqueduct of the midbrain. Findings were consistent with Wernicke's encephalopathy.

Patient's confusion, blurred vision, and progressive bilateral lower extremity weakness were attributed to Wernicke's encephalopathy due to hyperemesis gravidarum induced thiamine depletion. As a serum thiamine level was not available immediately, patient was treated with IV thiamine 500 mg three times daily for three days and then transitioned to oral thiamine 250 mg daily for five days. Patient worked with physical therapy and occupational therapy during her hospital stay. Gradual improvement of mentation, vision, bilateral lower extremity weakness, numbness and pain were reported. Upon discharge, patient was accepted at an inpatient rehabilitation facility to continue physical therapy.

Discussion

WE due to causes other than chronic alcohol abuse is an uncommon and often overlooked diagnosis. A systematic review in 2019 reported only 177 cases [3]. The most frequent causes of nonalcoholic patients were neoplastic disease (18.1%) and gastrointestinal surgery (16.8%) [4]. In the setting of hyperemesis gravidarum, an acute deficiency of thiamine results from body storage being unable to meet the increased metabolic demands. The biological active form of vitamin B1 is thiamine pyrophosphate, and it is an essential cofactor for enzymes involved in the pentose pathway [2,5]. There is about 25-30 mg of thiamine storage in the body which can last for about 18 days [6]. The recommended daily allowance is 0.4 mg/1000 kcal which can be easily met by an average adult diet [6]. However, in pregnancy the requirement of thiamine may increase up to 1.5 mg/day [7]. Thiamine deficiency affects energy metabolism, more profoundly evident in tissues with high thiamine turnover such as neural parenchyma and can result in cell death from necrosis or apoptosis [2,8].

Here we present a patient with history of hyperemesis gravidarum who developed Wernicke's encephalopathy as a result of severe thiamine deficiency from intractable emesis and decreased oral intake. Our patient presented with confusion, blurred vision, and inability to ambulate due to weakness. Patients with WE usually present with encephalopathy, ataxia, and oculomotor symptoms (nystagmus and gaze palsies being the most common). However, the classic triad is not always present. In pathological proven cases of WE (n = 131), 37.1% had one and 27.8% had two signs; 18.6% had none of the signs of the classic triad [9]. Encephalopathy usually presents as global confusion, lethargy and sometimes agitation. Most adults also have peripheral neuropathy such as absent deep tendon reflexes, hypotonia and weakness of the lower limbs [10].

Upper motor neuron and pontine involvement in WE suggest central pontine myelinolysis likely due to susceptibility of myeline to thiamine deficiency. Symptoms such as hyper-reflexia, gaze palsies, spastic quadriparesis, spastic dysarthria or bifacial weakness can be witnessed [9]. A large number of patients also report associated antegrade and retrograde amnesia called Korsakoff's psychosis. Our patient did not have any upper motor neuron, pontine involvement or memory deficiency on admission.

MR imaging along with clinical presentations are most useful in diagnosing WE. MRI is the imaging modality of choice because it is highly specific (i.e., 93%) and comparatively safer than computed tomography scan [11]. T2- hyperintensity may be seen in the medial thalami, in the peri-aqueductal gray matter and adjacent to the third and fourth ventricles [12]. These areas are sensitive to thiamine deficiency as they depend on oxidative phosphorylation. Our patient's MRI brain with and without contrast revealed bilateral symmetrical hyperintensities in the thalami, mammillary bodies, and surrounding aqueduct of the midbrain (Figure 1). The MRI brain in alcoholic WE often show additional vermian atrophy and contrast enhancement of the mammillary bodies [2].

Our patient's clinical presentation and radiological findings were in favor of an acute thiamine deficiency requiring immediate thiamine supplementation. Guidelines by the European Federation of Neurological Societies recommend that thiamine should be given 200 mg thrice daily via intravenous route, started before any carbohydrate, and continued until there is no further improvement in signs and symptoms [4]. In nonalcoholic patients, an intravenous dose of thiamine 100-200 mg once daily could be enough; whereas in alcoholic patients, higher doses may be required [13]. Due to clinical severity of our case, we chose to proceed with aggressive thiamine supplementation of IV thiamine 500 mg three times daily for three days and then transitioned to oral thiamine 250 mg daily for five days. Intravenous dextrose was not administered as it will aggravate matters further before correcting thiamine deficiency. Ultimately, our patient's symptoms gradually improved.

In general, the prognosis of patients with hyperemesis gravidarum induced WE is good if detected and treated early. Clinical improvement can be seen with adequately thiamine repletion [10,14]. On the other hand, the outcome of pregnancy in patients with WE are poor. Sequestration of thiamine by the fetus and placenta can have devastating complications like spontaneous abortion and fetal loss [15]. Prior study has shown ten out of 29 patients had healthy babies while the rest had either suffered from severe neurological deficits or resulted in fetal loss [10].

Conclusion

In conclusion, clinicians should be wary of the risk of Wernicke's encephalopathy associated with hyperemesis gravidarum in pregnant patients. Although not common, WE are a serious complication that can be potentially reversed when addressed promptly. Our case highlights the importance of recognizing neurological changes due to thiamine deficiency during pregnancy. We would like to emphasize the importance of thiamine monitoring and empiric supplementation in pregnant women with severe and prolonged vomiting history.

Conflict of Interest

The authors have no financial interest or any conflict of interest to declare.

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