Commentary

Wernicke's encephalopathy is typically diagnosed among alcoholic and malnourished patients or when a digestive disease or intervention (bariatric surgery) responsible for malabsorption leads to thiamine deficiency (vitamin B1).[1] Its occurrence during hyperemesis gravidarum is known but remains exceptional. When it occurs it can lead to devastating complications for the fetus and pregnancy (spontaneous abortion, fetal loss) and for the mother (possible progression to an irreversible Korsakoff's syndrome).[2,3] Some points deserve to be learned from these patients. First, the diagnosis should be systematically and early considered when we are confronted to mental changes, central nystagmus, and/or ophthalmoplegia or gait ataxia in a woman with hyperemesis gravidarum. Nevertheless and because each of these major symptoms can appear alone, the diagnosis could become more difficult. Whenever it is suspected, Wernicke's encephalopathy should be treated immediately with intravenous thiamine administration. Duration and dosage of thiamine supplementation remain debated, but initial parenteral administration of at least 200 mg daily to 500 mg three times daily is generally accepted.[4,5] In the case of Sutamnartpong *et al*.[6] we may observe that their patient was treated with lower doses of thiamine (100 mg intravenously daily for five days) with dramatic improvement. Another point to keep in mind is that, unfortunately, in several cases, Wernicke's encephalopathy is the direct consequence of a too much delayed or inadequate management of hyperemesis gravidarum with, for example, the prescription of glucose infusions in patients not previously substituted with thiamine (sometimes in patients with whom, sweets, better tolerated, can already constitute the basal diet!). Indeed, glucose intakes significantly increase thiamine consumption and can become the trigger to the onset of symptoms or to their aggravation. That is why these at-risk patients should systematically benefit from prophylactic thiamine administration.[7]

Central pontine myelinolysis (osmotic demyelination) is more frequently observed after the rapid correction of hyponatremia, generally with people with accompanying debilitating conditions such as alcoholism, liver diseases, or systemic illnesses. In rare cases, it has been reported in association with hyperemesis gravidarum and/or with Wernicke's encephalopathy with or without demonstration of natremia fluctuations,[8] as in the case proposed in this issue by Sutamnartpong *et al*.[6] The pathophysiological mechanisms involved remain unclear. Both for central pontine myelinolysis and Wernicke's encephalopathy, MRI plays a major role by showing in most cases a suggestive aspect, even if normal imaging does not rule out Wernicke's encephalopathy. However, treatments should not be delayed and must be started as soon as the diagnosis is suspected. The typical appearance of Wernicke's encephalopathy is hyperintensities in T2/T2-Flair weighted images of periacqueductal gray matter, around the third ventricle, mammillary bodies, and medial thalamus. Regarding central pontine myelinolysis, heterogeneous hyperintensities in T2/T2-Flair images of the central part of the pons, respecting its outer rims and cortico-spinal tracts, are classic (trident-shaped sign in axial images). Extrapontine myelinolysis is often associated with T2 hyperintensities of the basal ganglia and central white matter. Diffusion weighted imaging (DWI) and apparent diffusion coefficient (ADC) may provide additional information.

Finally, this case report highlights the importance of the prevention of neurological complications in hyperemesis gravidarum, and if these complications are suspected, they must be given an early and appropriate treatment.

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