

## Two Cases of Wernicke's Encephalopathy: A Rare Complication of Hyperemesis Gravidarum

Amina Beddi\*, Guezri Hamza, Fatiha. Aghrib, Dalal.Laoudiyi, Kamilia. Chbani, Siham. Salam, Lahcen. Ouzidane

Department of Pediatric Radiology, Hospital El Harrouchi CHU Ibn Roch

DOI: [10.36347/sasjm.2021.v07i11.005](https://doi.org/10.36347/sasjm.2021.v07i11.005)

| Received: 19.09.2021 | Accepted: 28.10.2021 | Published: 23.11.2021

\*Corresponding author: Amina Beddi

### Abstract

### Case Report

Wernicke's encephalopathy (WE) is a serious neurologic condition resulting from thiamine deficiency (vitamin B1). The majority of cases involve alcoholism; however, nonalcohol-associated WE do occur and is under-recognized. Magnetic resonance imaging is the gold standard imaging modality to confirm the diagnosis. We report two cases of young women with abundant vomiting during the first trimester of pregnancy (hyperemesis gravidarum), causing symptomatic Wernicke's encephalopathy. The diagnostic was made by MRI showing a T2-weighted hyperintensity in the periaqueducal, thalami and mammillary bodies. However, it is imperative that WE is diagnosed and treatment is started as quickly as possible.

**Abstract:** Wernicke's Encephalopathy, Gravidarum.

Copyright © 2021 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

## INTRODUCTION

Wernicke's encephalopathy (WE) is often thought of in the context of alcoholism and neurologic abnormalities. The relationship with other diseases that lead to thiamine deficiency is less often recognized in a patient who presents with a variety of neurologic complaints.

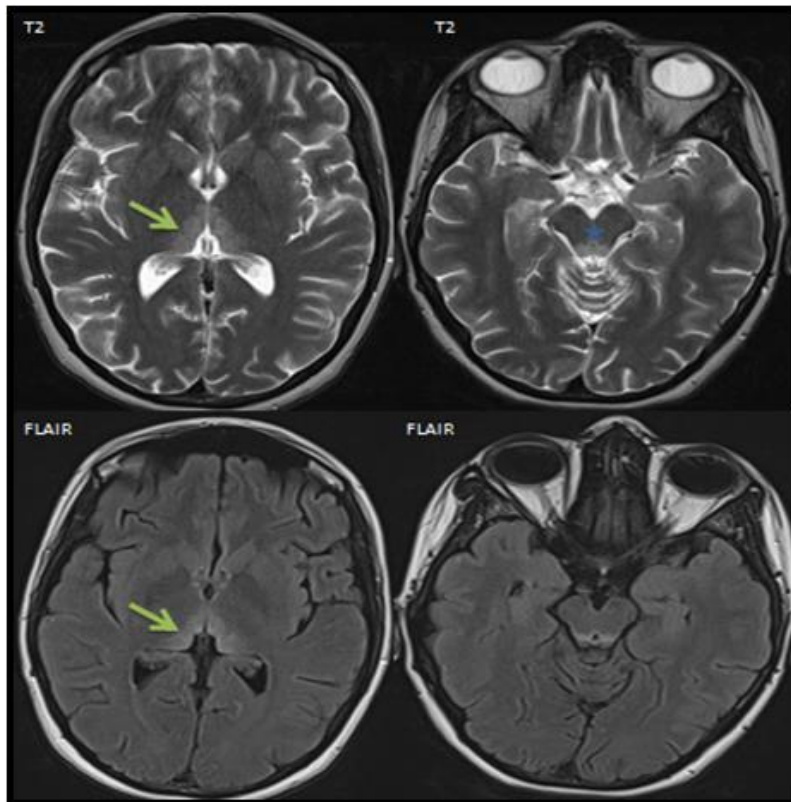
The classical clinical triad and magnetic resonance imaging (MRI) play an important role, especially in the diagnosis of non-alcoholic Wernicke's encephalopathy causing a damage of the hippocampomammillothalamic network. It also affects the grey matter in contact with the aqueduct of Sylvius and the fourth ventricle.

It is a neurologic emergency that requires immediate attention to prevent permanent neurological morbidity and mortality. We report two cases of Wernicke encephalopathy complicating incoercible vomiting in pregnancy.

## PRESENTATIONS OF THE CASES

### Case 1

The patient was 22 years old with no notable pathological history, 14 weeks pregnant and reported incoercible vomiting in pregnancy, resistant to antiemetics. She was referred to the emergency department for a neurological symptomatology made of diplopia, intracranial hypertension syndrome (HTIC), headache and then a confusional disorder, an abolition of osteotendinous reflexes and hypoesthesia essentially in the lower limbs. The biological workup revealed moderate hepatic cytolysis, moderate elevation of pancreatic enzymes, and metabolic alkalosis with hypokalemia. A magnetic resonance imaging (MRI) of the brain was performed urgently showed the appearance of hyper signals at the periaqueductal level, of the mammillary bodies (Figure 1), of the two thalami and around the 3rd ventricle (Figure 1) very suggestive of a WGE. Parenteral vitamin B1 supplementation (1g/24h) associated with vitamin B6 was instituted and then relayed orally (vitamin B1 500 mg\*2/d) until the end of the pregnancy. Two months later, the evolution was marked by a clear regression of the symptoms.



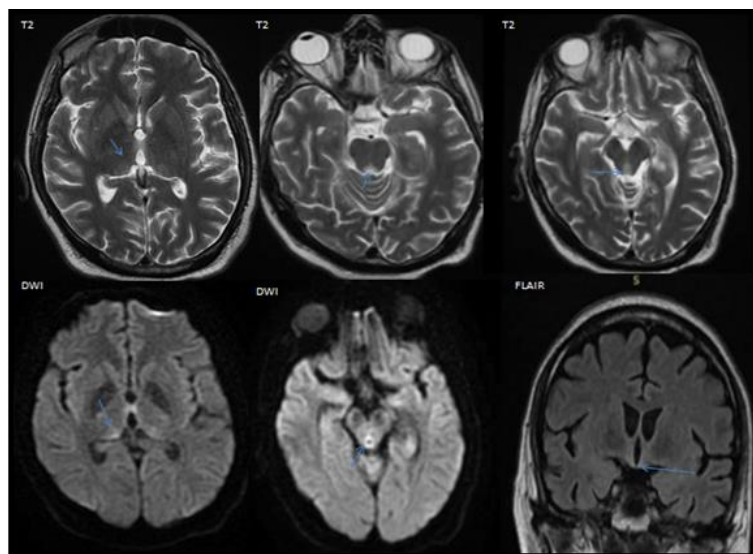
**Fig-1:** Axial fluid-attenuated inversion recovery (FLAIR) and sequence T2 weighted images from when the patient was encephalopathic show symmetrical changes of abnormal high signal in the periaqueductal area (asterix), and posterior medial thalami (arrows).

**Case 2**

A 30-year-old woman, 10 weeks pregnant and reported incoercible vomiting in pregnancy was admitted to the hospital she complained of dizziness and headache, but those symptoms quickly subsided. Vertigo and nystagmus developed, but his mental status was alert, and he did not show sensory or motor changes in the extremities. Three days later, he presented diplopia and esotropia, and complained of difficulty in closing his eyes. Brain magnetic resonance

imaging (MRI) was performed showed bilateral and symmetrical T2 and FLAIR hypersignals of the periaqueductal region, cerebral tubercles (colliculi), mammillary bodies (Figure 2), we note a restriction of diffusion.

The blood level of thiamine was low (0.5 U/L; reference range 2-7 U/L). Thiamine was subsequently administered. Over the following two weeks, his mental status gradually, but not completely, improved.



**Fig-2: (Before treatment)** MRI weighted images FLAIR and T2 axial: bilateral and symmetrical hypersignal around the periaqueductal, the mammillary bodies and the colliculi; Coronal FLAIR weighted images: hyperintensities in the periventricular region around the third ventricle; in the axial Diffusion Weighted Images highlighting WE changes in bilateral thalami and periaqueductal (blue arrows).

## DISCUSSION

Gayet-Wernicke's encephalopathy is a rare neurological pathology, due to a vitamin B1 deficiency, but known complication of severe hyperemesis gravidarum. The mortality rate ranges from 10–20%. It is essentially encountered in alcoholics, but can also occur in a context of incoercible vomiting or in states of severe malnutrition. It is a therapeutic emergency requiring the administration of vitamin B1 (Thiamine)[1, 2].

The diagnosis is clinical with the triad (found in 60% of cases): confusional syndrome, oculomotor disorders (nystagmus, oculomotor paralysis) and an ataxic gait [3].

MRI is the key examination to confirm the diagnosis by the presence of T2 and Flair hypersignals in the acute phase, most frequently in the periaqueductal gray matter, in the mediodorsal nucleus of the thalami, and in the mammillary bodies. Isolated contrast of the mammillary bodies may be the only abnormality initially observed. The evolution is marked by a disappearance of the signal abnormalities as well as atrophy of the mammillary bodies and thalami, leading to enlargement of the third ventricle.

In imaging, MRI shows abnormalities in 60% of cases, which means that normal imaging, does not exclude the diagnosis [1-4-5].

Moreover, these lesions take up contrast inconsistently after injection of Gadolinium chelate. Atypical localizations have been reported, with signal abnormalities in the form of T2 hyperintensities and possible contrast uptake in the superior vermis, head of the caudate and lenticular nuclei, red nuclei, facial nerve nuclei, abducens and vestibular nuclei, as well as

in the central and precentral cortex. These atypical locations can make the diagnosis difficult [1-6].

## CONCLUSION

Wernicke's encephalopathy is an exceptional but potentially reversible complication, the diagnosis of which is of its potentially lethal character. To be considered in the presence of neurological manifestations in a pregnant patient with hyperemesis gravidarum. MRI facilitated the diagnosis by finding FLAIR hypersignals in a region of interest (periaqueductal, thalamus or mammillary bodies).

## REFERENCES

1. Nasri, S., Aichouni, N., Ettayeb, M., Mebrouk, Y., & Kamaoui, I. (2020). Encéphalopathie de Wernicke: complication rare de l'hyperemesis gravidarum. *The Pan African Medical Journal*, 36.
2. Spruill, S. C., & Kuller, J. A. (2002). Hyperemesis gravidarum complicated by Wernicke's encephalopathy. *Obstetrics & Gynecology*, 99(5), 875-877.
3. Welsh, A., Rogers, P., & Clift, F. (2016). Nonalcoholic Wernicke's encephalopathy. *Canadian Journal of Emergency Medicine*, 18(4), 309-312.
4. Phayphet, M., Rafat, C., Andreux, F., Parraga, E., Rondeau, É., & Hertig, A. (2007). Hyperemesis gravidarum: une cause rare d'encéphalopathie de Wernicke. *La Presse Médicale*, 36(12), 1759-1761.
5. Patel, S., Topiwala, K., & Hudson, L. (2018). Wernicke's encephalopathy. *Cureus*, 10(8).
6. Sechi, G., & Serra, A. (2007). Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. *The Lancet Neurology*, 6(5), 442-455.