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# SUCCESSFUL MANAGEMENT OF GAYET-WERNICKE ENCEPHALOPATHY IN PREGNANCY: CASE SERIES

**Ibtissam Bellajdel*, Oussama Lamzouri, Imane Benhamou, Hafsa Taheri, Hanane Saadi, Ahmed Mimouni**

Department of Gynecology and Obstetrics, Mohammed VI-Oujda University Hospital, Faculty of Medicine and Pharmacy, Mohammed Premier University, Oujda, Morocco.

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***Corresponding Author**
**Dr. Ibtissam Bellajdel**
Department of Gynecology and Obstetrics, Mohammed VI-Oujda University Hospital, Faculty of Medicine and Pharmacy, Mohammed Premier University, Oujda, Morocco.

## ABSTRACT

This article presents a retrospective observational analysis of four cases of Gayet-Wernicke encephalopathy (GWE) treated successfully in the department of gynecology at Mohamed IV university hospital. GWE is a neuropsychiatric syndrome due to thiamine (vitamin B1) deficiency, characterized by the classic triad of encephalopathy, ophthalmoplegia/nystagmus, and ataxia. The authors collected data on presenting complaints, chronicity, adequacy of treatment, neurological symptoms, pregnancy outcomes, and long-term morbidity. All cases met Caine's operational criteria for WE, and patients were followed up with via telephone to track long-term outcomes. Diagnosis of GWE is typically based on the triad of ophthalmoplegia, mental confusion, and ataxia, and MRI is the reference examination. Thiamine deficiency can cause varying degrees of neurological damage, some of which can be irreversible, and is typically observed in chronic alcoholics but is also seen in individuals suffering from severe malnutrition. Factors that can favor the development of GWE include chronic digestive or neoplastic pathologies, psychiatric pathologies, chronic end-stage renal disease, and vomiting during pregnancy. The article concludes by highlighting the importance of early recognition and treatment of GWE to prevent long-term neurological damage.

## INTRODUCTION

Gayet-Wernicke encephalopathy (GWE) is a neuropsychiatric syndrome due to thiamine (vitamin B1) deficiency, characterized by the classic triad of encephalopathy,

ophthalmoplegia/nystagmus, and ataxia. MRI is the imaging modality of choice for diagnosis.[1]

We report four cases of GWE treated successfully in the department of gynecology at Mohamed IV university hospital.

## METHODOLOGY

We conducted a retrospective observational analysis on patients diagnosed with Wernicke's encephalopathy (WE) between 2014 and 2023. Data collection relied on electronic medical records (EMRs) and Excel spreadsheets. The data included information such as presenting complaints, chronicity, adequacy of treatment, neurological symptoms, pregnancy outcomes, and long-term morbidity. We only included cases that met Caine's operational criteria for WE, which includes the classic triad of symptoms, or autopsy evidence, or a clinical response to thiamine. We also followed up with patients via telephone to track long-term outcomes.

## RESULTS

### Case 1

An 18-year-old patient with no medical history presented with uncontrollable vomiting and abdominal pain at 13 weeks of pregnancy. Biochemical analysis showed elevated pancreatic enzymes and liver transaminases, and hypokalemia. Abdominal MRI revealed Balthazar grade C pancreatitis. On the eighth day of hospitalization, the patient developed intense headaches with diplopia and tetraparesis. Angio-MRI of the brain revealed hyperintensity in both thalami on FLAIR sequences and bilateral hyperintensity of the mamillary bodies on T2 sequences, suggestive of GWE **FIGURE 1**. The patient was supplemented with 1g/day of vitamin B1, potassium, and hydration. Neurological and ophthalmic symptoms disappeared after one month.

### Case 2

A 37-year-old woman, with a history of uterine scarring, developed uncontrollable vomiting at 5 weeks of gestation, which became intractable at 12 weeks, associated with epigastric pain, hypokalemia, and was hospitalized in the intensive care unit. She was re-hospitalized at 17 weeks of gestation with anterograde amnesia, diplopia, and flaccid paralysis of the lower limbs. Liver enzymes were elevated, and brain MRI **(FIGURE 2)** showed hyperintensities in the periaqueductal region, mamillary bodies, third ventricle, and both thalami on T1, T2, and T2 FLAIR sequences. Vitamin B1 therapy was initiated at 500mg X 3/day for 10 days,

followed by 250mg X 3/day for another 10 days, then switched to oral therapy, along with antiemetics. At 26 weeks of gestation, the patient remained paraplegic with persistent anterograde amnesia and bilateral blindness, and a fetal death was detected. She underwent fetal extraction, and after one month, her clinical condition improved, although global motricity of the lower limbs remained reduced. Follow-up MRI revealed regression of the hyperintensities and hypotrophy of the mamillary bodies.

**Case 3**

A 32-year-old woman, G4P2, with no notable medical history, was admitted for intractable vomiting and electrolyte disturbance at 12 weeks of gestation. Fetal ultrasound showed missed abortion with fetal biometry corresponding to 12 weeks and 4 days. On the fifth day of hospitalization, the patient experienced dizziness, diplopia, and decreased visual acuity. Biochemical analysis was unremarkable. Brain MRI **(FIGURE3)** revealed bilateral and symmetrical hyperintensity of the thalamic pulvinars suggestive of GWE. The patient received vitamin B1 supplementation and showed complete recovery, with disappearance of all GWE signs.

**Case 4**

A 34-year-old female patient, without notable medical history, who was 13 weeks pregnant, presented to the emergency department with uncontrollable vomiting, mental confusion, and incoherent speech. The clinical examination revealed a confused patient with a Glasgow score of 14, stable on the hemodynamic and respiratory levels, afebrile, and with a normal obstetric examination.

The biological assessment revealed hypokalemia at 2.8 mmol/L, normal TSH, normal lipasemia, and a normal liver and kidney function test. Glycemia was 1 g/L.

A brain MRI was performed, **FIGURE 4** revealing an aspect of Gayet and Wernicke encephalopathy with bilateral and symmetrical hypersignal of the thalamic pulvinars. The vitamin B1 level revealed a deficiency with a low level of 45 mg/L. The patient was supplemented with vitamin B1 and had a good evolution.

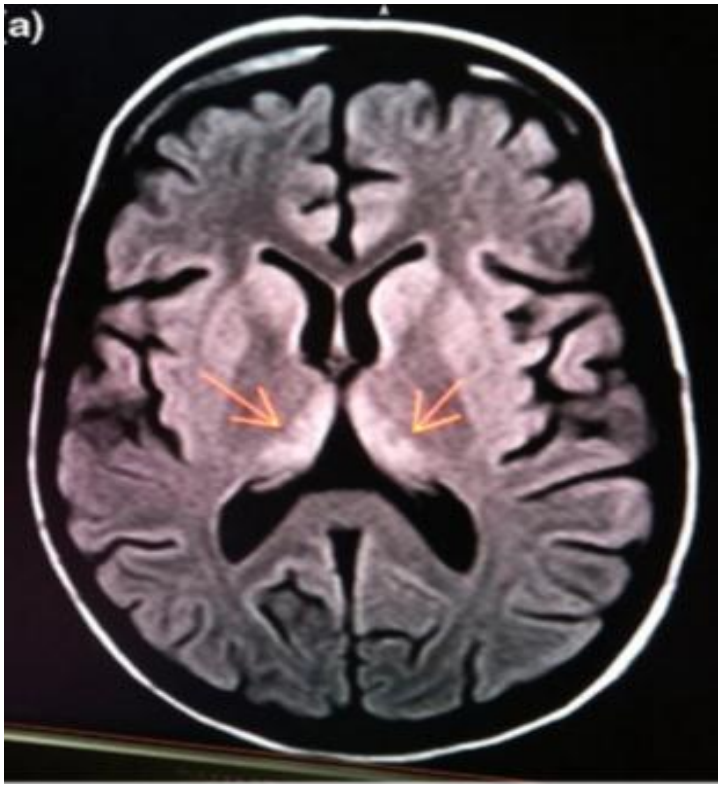


**Figure 1: FLAIR sequence showing an hypersignal in both thalamus.**

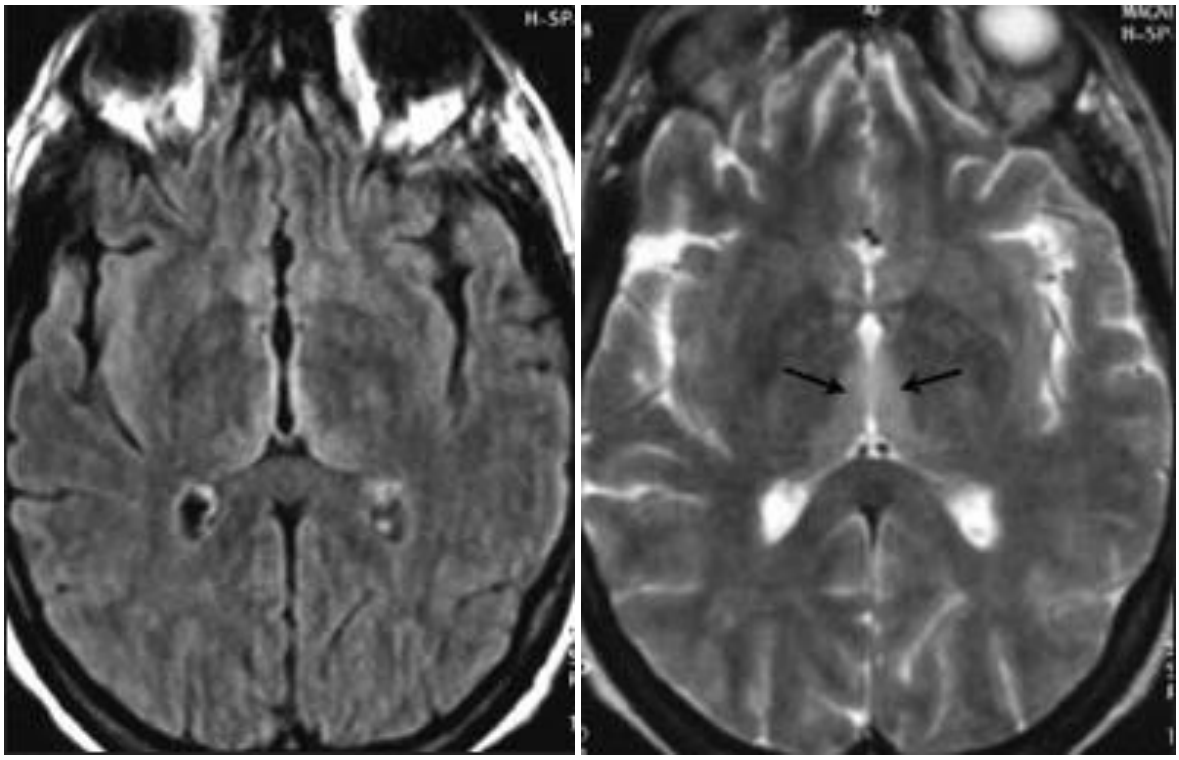

**Figure 2: Axial encephalic MRI in T2 and FLAIR showing hypersignals on either side of V3 and the thalamus.**

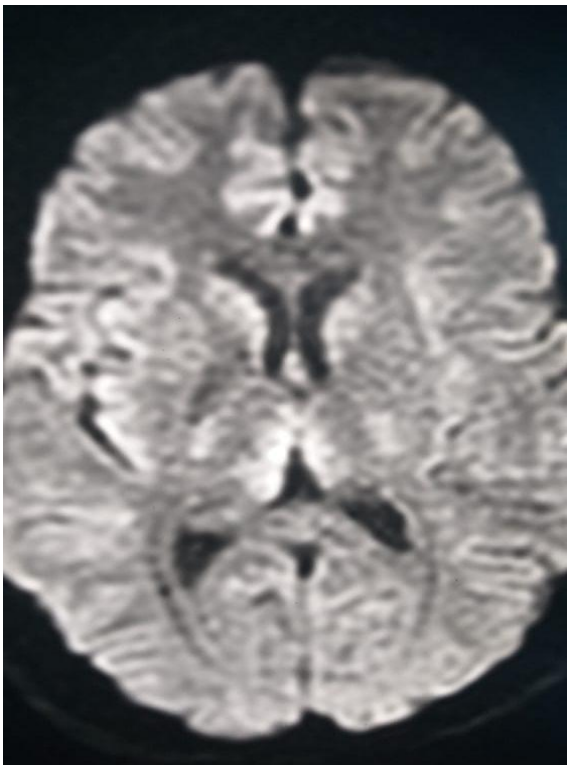

**Figure 3: Encephalic MRI: hypersignal in the thalamus.**

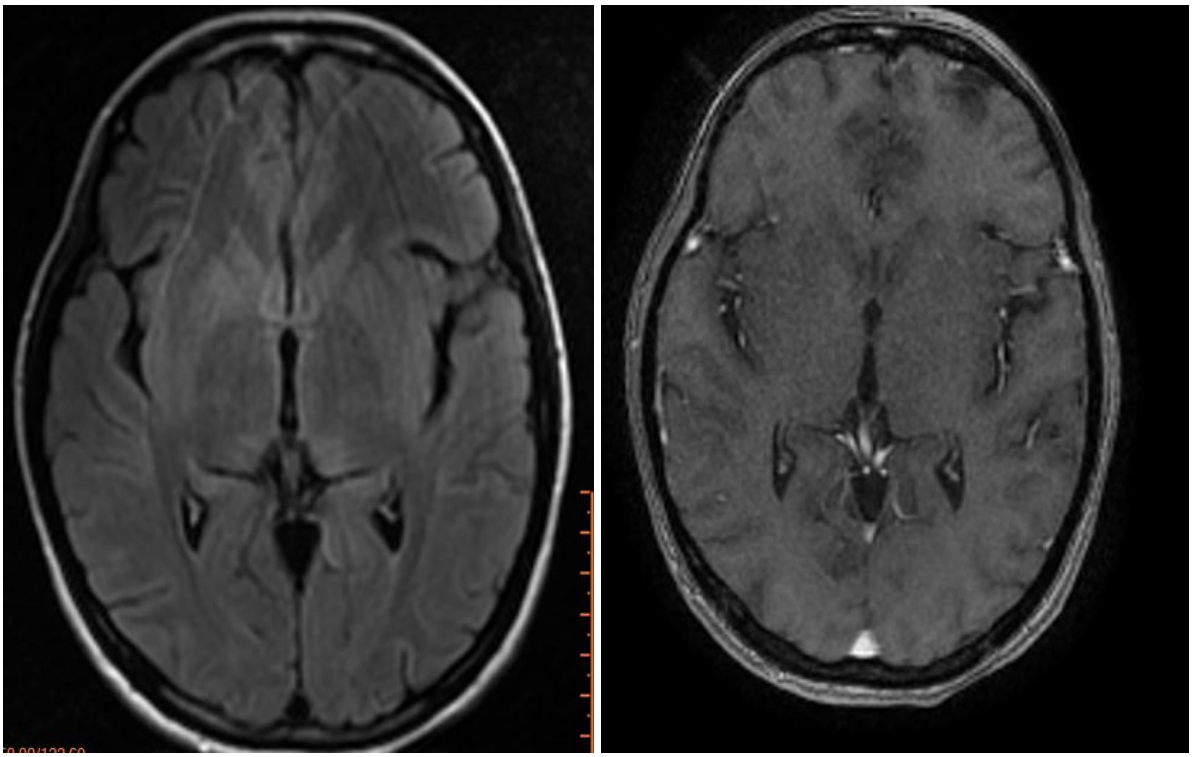

**Figure 4: An aspect of Gayet and Wernicke encephalopathy with bilateral and symmetrical hypersignal of the thalamic pulvinars.**

## DISCUSSION

Gayet-Wernicke's encephalopathy is an uncommon acute neuropsychiatric complication resulting from a deficiency in vitamin B1 (thiamine).[1] Thiamine deficiency can cause varying degrees of neurological damage, some of which can be irreversible.[2] This condition is typically observed in chronic alcoholics but is mainly seen in individuals suffering from severe malnutrition.[3] Favoring factors include chronic digestive or neoplastic pathologies, psychiatric pathologies, chronic end-stage renal disease, and vomiting during pregnancy.[4]

The diagnosis of Gayet-Wernicke's encephalopathy is initially clinical, based on the triad of ophthalmoplegia, mental confusion, and ataxia.[5] In the case of suspected Gayet-Wernicke's encephalopathy, MRI is the reference examination.[6] It shows hypersignals in T2, FLAIR, and diffusion, which are typical due to their location and symmetrical character around the aqueduct of Sylvius, the third ventricle, and especially at the level of the mammary tubercles.[7] In some cases, blood concentration of thiamine or its derivatives can be measured before supplementation to confirm the diagnosis of Wernicke's encephalopathy.[8] However, this was not done in our patient due to the therapeutic intervention.

Our patients presented with persistent neurological signs despite good hemodynamic and biological patterns. Therefore, we conducted a radiological exploration, which was an MRI angiography, to eliminate any underlying neurological cause (vascular, tumoral, or infectious). This examination allowed us to confirm the diagnosis of Gayet-Wernicke's encephalopathy.

Gayet-Wernicke's encephalopathy is a medical emergency and treatment should begin as soon as the diagnosis is suspected without waiting for vitamin determinations.[9] There is no consensus on the dosage of thiamine or the duration of treatment.[10] Our patients were treated with an intravenous vitamin complex containing vitamins B1, B6, and B12. The prognosis of Gayet-Wernicke's encephalopathy is highly variable and depends on early diagnosis and treatment.[11] The mortality rate for all etiologies combined is 17-20%.[12]

Hyperemesis gravidarum (HG) is a condition that occurs most often in the first trimester of pregnancy.[13] Before the onset of the classical triad of Wernicke's encephalopathy, there is a prodromal phase associated with nausea, vomiting, blurring of vision, or diplopia.[14,15] Thiamine is a water-soluble vitamin, and its demand is increased in pregnancy. This is compounded by recurrent vomiting and inadequate food intake, especially during the first

trimester.[16] Thiamine acts as a cofactor for enzymes such as transketolase, which plays a major role in carbohydrate metabolism. It aids in the survival of neurons and facilitates neurotransmission. The exact pathophysiology of neurodegeneration occurring due to thiamine deficiency is not clear.

Wernicke encephalopathy is a medical emergency that requires prompt treatment to prevent permanent brain damage and potentially fatal complications. The primary treatment for Wernicke encephalopathy is high-dose thiamine replacement therapy, which is typically administered intravenously.[13] Prompt thiamine replacement can reverse the symptoms of Wernicke encephalopathy in many cases, but the long-term prognosis can depend on the severity of the initial injury and the timing and adequacy of treatment.[17]

However, even with treatment, some patients may experience residual cognitive or neurological deficits or develop complications such as Korsakoff syndrome, a chronic memory disorder that can occur as a consequence of untreated or inadequately treated Wernicke encephalopathy. Other complications that may arise include seizures, coma, or even death in severe cases.[18]

It is essential to identify and treat Wernicke encephalopathy early, as prompt treatment can significantly improve outcomes and reduce the risk of complications.[19] Therefore, early recognition of the symptoms of Wernicke encephalopathy is crucial, especially in individuals who are at higher risk, such as those with alcohol use disorder or malnutrition.[18]

Diagnosis of HG is usually clinical, but neuroimaging should be performed to exclude other neurological conditions with similar presentations.[16] Computerized tomography is an insensitive test for Wernicke's encephalopathy.[9] The characteristic MRI findings include areas of increased T2 and fluid-attenuated inversion recovery signals, decreased T1 signal, and diffusion abnormality surrounding the aqueduct and third ventricle and within the medial thalamus.

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