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Case Report

Brain MRI: A safe and a specific tool in the diagnosis of Gayet Wernicke's encephalopathy in pregnant women: Case report ☆

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ABSTRACT

Gayet-Wernicke encephalopathy (GWE) is a neuropsychiatric syndrome due to Vitamin B1 (thiamine) deficiency, fatal in 30% of cases and preventable if treatment is initiated early, characterized by the classic triad of encephalopathy, ocular involvement: ophthalmoplegia and/or nystagmus and ataxia. GWE is mainly observed in alcoholics, but can also appear in any state of malnutrition. In obstetrics, hyperemesis gravidarum can be complicated by GWE due to low thiamine stores and increased thiamine requirements. We report a case of Gayet-Wernicke encephalopathy complicating incoercible vomiting in a pregnant woman.

Introduction

Gayet-Wernicke encephalopathy (GWE) is a neuropsychiatric syndrome due to Vitamin B1 (thiamine) deficiency, fatal in 30% of cases [1] and preventable if treatment is initiated early, characterized by the classic triad of encephalopathy, ocular involvement: ophthalmoplegia and/or nystagmus and ataxia. GWE is mainly observed in alcoholics, but can also appear in any state of malnutrition. In obstetrics, hyperemesis gravidarum can be complicated by GWE due to low thiamine stores and increased thiamine requirements. We report a case of Gayet-Wernicke encephalopathy complicating incoercible vomiting in a pregnant woman.

Case report

It is about a 32-year-old parturient, gravida 3 para 2, with no notable pathological history or complications during the first 2 pregnancies, the current 1 being unattended.

Clinical Findings: the patient was admitted in a state of confusion with a Glasgow score of 11/12 at the expense of verbal and motor response, with hyperglycemia at 2.06 g/L, tachycardia at 140 beats/min, blood pressure at 110/68 mmHg,

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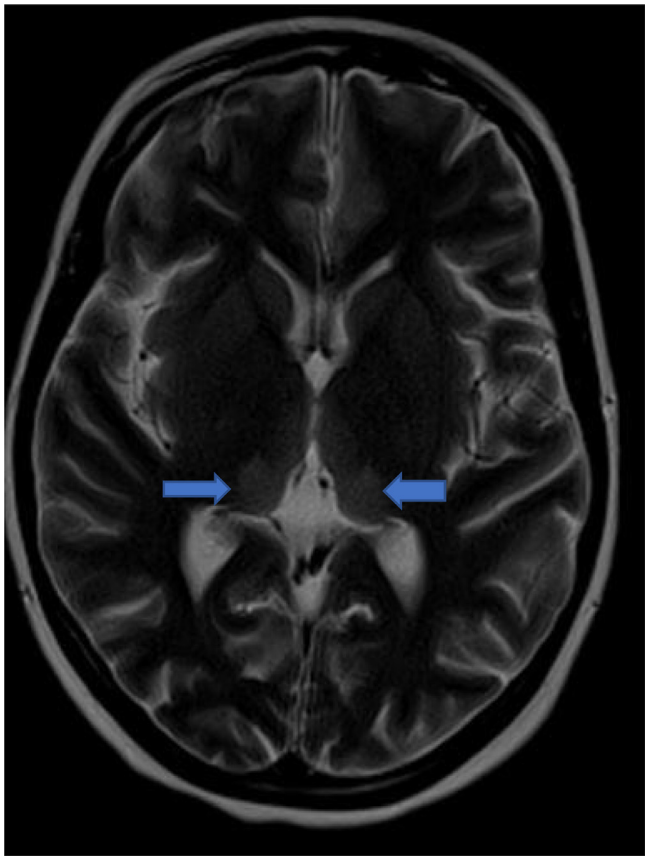

Fig. 1 – Brain MRI in axial section in T2-weighted sequence showing a bilateral hyper signal of the dorsomedial thalamus (arrows).

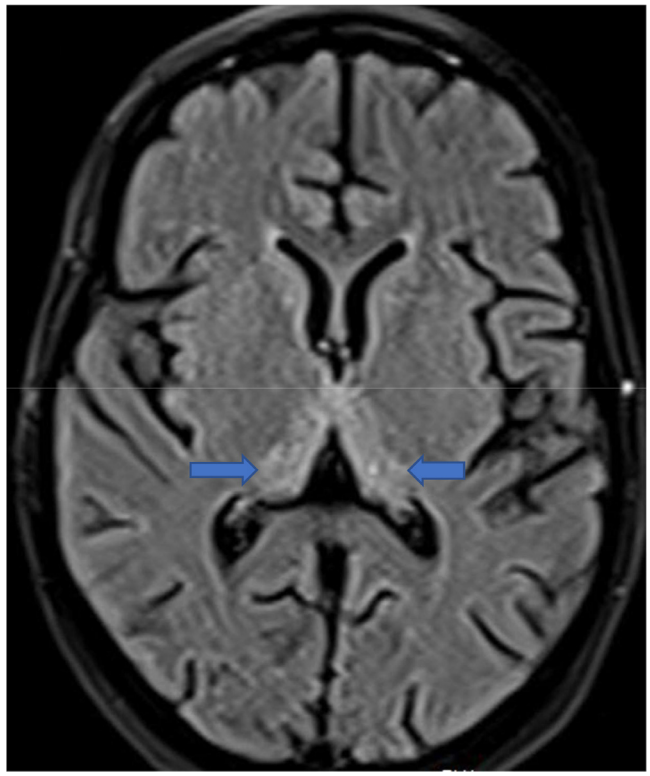

Fig. 2 – Brain MRI in axial section in Flair showing a bilateral hyper signal of the dorsomedial thalamus (arrows).

polypnea at 24 cycles/minute, apyretic, normally colored conjunctiva, body mass index at 17 kg/m^2. Neurological examination revealed bilateral horizontal nystagmus, hypotonia of the 4 limbs without sensory level, and the abolition of osteotendinous reflexes (patellar).

The history of illness began at 14 weeks of gestation when the patient presented vomiting of medium abundance becoming more frequent throughout the day, reaching 6 episodes per day. The vomiting was followed by epigastric abdominal pain and significant weight loss according to the patient's family. The evolution was marked by asthenia and a fluctuating disorder of the conscience, hence the consultation in our formation.

Diagnostic assessment: biological explorations revealed creatinine level at 7 mg/L, urea level at 0.18 g/L, serum potassium level at 3.8 mmol/L and natremia at 139 mmol/L. Liver function was normal, total bilirubin at 12 mg/dL with conjugated bilirubin at 7 mg/dL. The TSH ultra-sensitive test was normal at 2.2 mIU/L. Prothrombin level and activated partial thromboplastin time were normal. The urine dipstick showed no abnormality.

The patient underwent an abdominal ultrasound that showed no abnormalities. The evolution was marked by a progressive worsening of the sensory-motor deficit in all four limbs, complicated by paraplegia with the abolition of the deep tendon reflexes. A brain CT scan requested came back as normal.

A cranial magnetic resonance imaging (MRI) was requested which showed signs in favor of a Gayet-Wernicke encephalopathy: T2 (Fig. 1) and FLAIR (Figs. 2 and 3) hyper signal involving the bilateral thalamus with diffusion restriction at this level.

Diagnosis: The clinical and biological picture corresponded to a Gayet-Wernicke encephalopathy associated with peripheral neuropathy in the context of hyperemesis gravidarum.

Therapeutic interventions: the patient was admitted and treated in the intensive care unit receiving rehydration with crystalloids, treatment of Hyperemesis Gravidarum with doxylamine 10 mg/day, and gastric protection with protein pump inhibitors at a rate of 40 mg/day, a vitamin therapy with thiamine 600 mg/day was started.

Follow-up and outcome of interventions: after 1 week under vitamin B1 therapy, the patient regained her initial state of consciousness with a clear improvement of spatio-temporal orientation. The vomiting stopped and oral feeding was started.

Patient Perspective: the patient, who at first was admitted in a state of obnubilation, testifies after having gradually regained her state of consciousness, which was initially manifested by a smile testifying to her satisfaction and later by her verbal response, providing her testimony with an emotional component and a lot of gratefulness that was dedicated to the medical team and being appreciative of the development of medicine, which, according to her, did miraculously save her life and that of her baby.

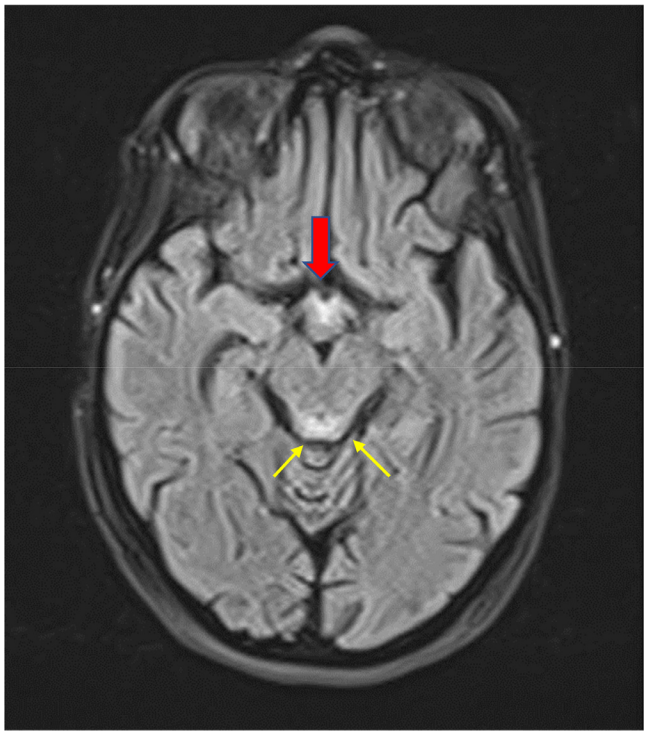

Fig. 3 – Brain MRI in axial section FLAIR showing a bilateral hyper signal of the mamillary bodies (red arrow) and of the periaqueductal region (yellow arrows).

Discussion

Gayet-Wernicke encephalopathy was first described by Carl Wernicke, a German neurologist, in 1881 as an acute upper hemorrhagic encephalitis in 2 alcoholic patients and a patient with pyloric stenosis following ingestion of sulfuric acid with repeated vomiting [2–6].

In pregnant women, GWE is related to acquired thiamine (vitamin B1) deficiency [7]. Vitamin B1 deficiency can be manifested by numerous nonspecific signs (asthenia, hearing loss, stupor, vegetative signs: hypotension, hypothermia, bradycardia), particularly in the elderly [8].

Thiamine deficiency leads to brain damage mainly in the mesodiencephalic regions rich in thiamine-dependent energy processes [9].

Thiamine is an important coenzyme for 3 critical enzymes of the Krebs and pentose phosphate cycle: transketolase, ketoglutarate dehydrogenase, and pyruvate dehydrogenase. Deficiency of thiamine and these enzymes causes focal lactic acidosis, alteration of brain energy, and depolarization of neurons due to excitotoxicity induced by the n-methylD-aspartate receptor. Ultimately, this results in the alteration of the blood-brain barrier, and the generation of free radicals, leading to cell death by necrosis and apoptosis [10].

Nausea and vomiting are common in pregnancy, and these manifestations affect up to 85% of pregnant women, but incoercible vomiting, hyperemesis gravidarum, complicates 0.5%-2% of pregnancies [11,12], The largest series in the literature including cases of GWE in pregnant women is that of Di Gangi. et al in 2012 which included 63 cases [13,14],

When Gayet-Wernicke encephalopathy is suspected, MRI is the reference examination. It shows hyper signals in T2, FLAIR, and diffusion, typical by their localization and their symmetrical character around the aqueduct of Sylvius, the third ventricle, and especially at the level of the mammary tubercles [15]. In our patient, this examination allowed us to retain the diagnosis of Gayet-Wernicke encephalopathy. The diagnosis of Wernicke's encephalopathy can be confirmed by measuring the blood concentration of thiamine or its derivatives before any supplementation [16]. But in our patient, no assay was performed because of the therapeutic urgency and the unavailability of this examination at our hospital. Other abnormalities are often observed in this context of vomiting in pregnancy and Gayet-Wernicke's encephalopathy, mainly hyperthyroidism, and hypokalemia, and do not require any specific treatment except symptomatic treatment [17,18].

Gayet-Wernicke encephalopathy is a medical emergency. Treatment should be early, as soon as the diagnosis is suspected, and should not be delayed by vitamin tests. There is no consensus on the dosing of thiamine or the duration of treatment [19]. Our patient benefited from treatment with a diluted intravenous vitamin complex containing vitamins B1, B6, and B12 for 1 week with a favorable outcome.

The prognosis of GWE remains grave if treatment is not started early or the diagnosis is not suspected or confirmed.

Conclusion

Gayet-Wernicke encephalopathy (GWE) is a potentially fatal neurological syndrome due to thiamine deficiency.

The clinical picture is varied, generally, the clinical triad is present in most cases (confusion, ataxia, ophthalmoplegia).

On MRI, hypersignals of the affected areas are frequently found in T2, FLAIR sequence. Therapeutic management consists of administering thiamine as soon as possible.

Any pregnant woman with hyperemesis gravidarum or malnutrition should receive daily thiamine supplementation.

What is already known about this topic:

- Gayet Wernicke's encephalopathy is a rarely diagnosed pathology, especially in third world countries.
- Gayet Wernicke's encephalopathy, if being underdiagnosed engages the vital prognosis if the treatment is not quickly instituted.

What this study adds:

- The interest in presenting this case relies on the good evolution seen in our patient.
- By presenting this case, we hope to ensure the survival of pregnant women affected by this pathology, especially in the African continent.

Patient consent

Informed consent was approved by the patient being fully aware of the stakes of this case report and wanting willingly to be a part of it.

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