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Research Paper

Gayet Wernicke's encephalopathy: a rare complication of hyperemesis gravidarum

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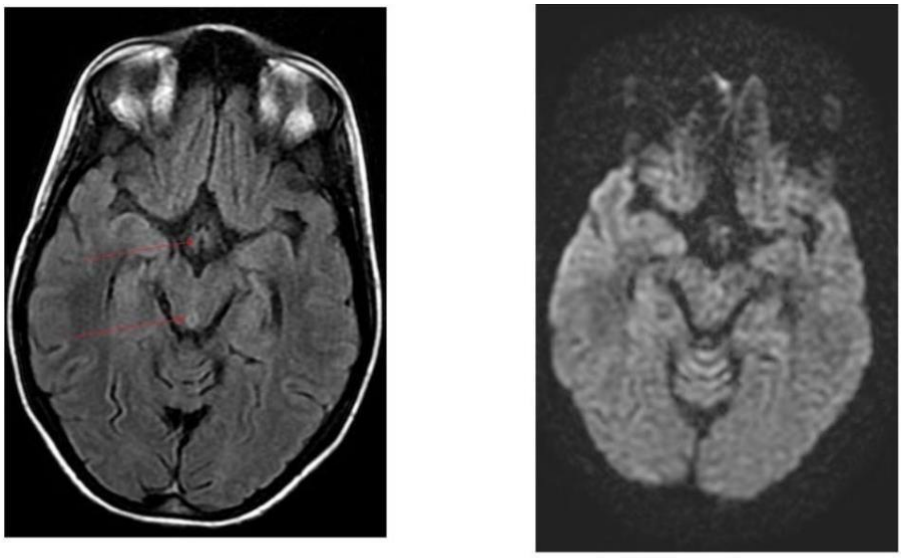
I. Introduction

Gayet Wernicke's encephalopathy (GWE) is a neurological emergency secondary to a deficit thiamine (vitamin B1) causing damage to the hippocampo-mamillothalamic network (Papez circuit). It also affects the gray matter in contact with the aqueduct of Sylvius and the fourth ventricle. It is a serious central neurological condition which mortality rate is estimated at 30% [1]. If GWE is a known complication of alcoholism, it should be known that it can occur outside of this addiction [2,3]. We report a case of Gayet-Wernicke encephalopathy complicating incoercible vomiting in a pregnant woman.

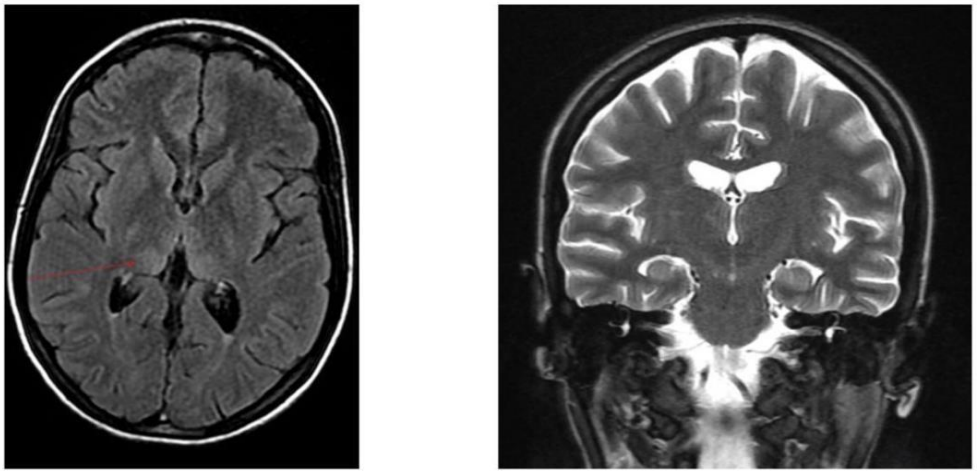
II. Clinical observation

This is a 23-year-old patient with no specific pathological records,
Addressed to the Gynecological Emergency
Department II of the Hassan II University Hospital, for the mamangment of incoercible gestational vomiting on pregnancy of 15SA + 3 days evolving for 02 months with alteration of the general state and confusional syndrom with a significant temporospatial disorientation.
Neurological examination revealed static cerebellar syndrome, abolition of osteotendinous reflexes and hypoesthesia mainly in the lower limbs,

The initial biological assessment showed moderate hyponatremia at 127, with hypokaliemia at 3, TSH slightly decreased to 0.21, lipasemia to 2 * N, Vit B12 correct, 1 the remains of the was normal.
An encephalic magnetic resonance imaging (MRI) was performed showed T2 hypersignal and periaqueductal flair, mamillary bodies and pulvinar, non-restrictive in diffusion (Figure 1 and 2) very suggestive of an GWE.
Neurologist examination: found an asthenic patient not able to stand and walk, sensory and motor deficit rated 4/5 on the extensors of the right arm, and flexors of the two lower legs.
Psychiatric opinion found the diagnosis of a Depressive Syndrome with suicid idea
Psychiatric examination finds a patient stable on the Psycho-Motor level, slows down, sad fasciated, reports mood sadness, depressive syndrom, anorexia, anhedonia.
Therapeutically: Injectable Vitamin B1 (500mg diluted in SSI/d), RHD, + PARENTERAL DIET oliclinomel type N4, becidouze 1cp*3/d, omeprazole 20mg/d, Setraline 50mg/d, LOVENOX 0.4 CC/24H.
20 days later, the evolution was marked by a clear regression of static Cerebellar syndrome and hypoesthesia of the lower limbs, memory disorders otherwise persisted.



Hypersignal FLAIR périaqueducal et au niveau des tubercules mamillaires, non restrictif en diffusion



Hypersignal FLAIR, T2 du pulvinar

III. Discussion

most often underdiagnosed with a clinical prevalence of 0.04% to 0.13%, compared to 0.8% and 2.8% in Gayet Wernicke's encephalopathy (or Wernicke anatomical pathology [5,6]. This underdiagnosis is Korsakoff's in Anglo-Saxon literature) was first largely related to misleading clinical forms in patients described by Wernicke in 1881 in an alcoholic man not recognized as being at risk [7,8], and a woman with incoercible vomiting [4]. It is a metabolic pathology related to thiamine deficiency,

As well as frequent atypical presentations. One in five patients has none of the clinical signs of the classic GWE triad; moreover, these signs are sometimes difficult to differentiate from those of acute or chronic ethyl poisoning, the main risk ground for this pathology in the Western world [7,8]. This vitamin B1 deficiency can complicate other pathological situations such as malnutrition, anorexia nervosa, prolonged parenteral nutrition without thiaminic supplementation, or gastrointestinal tumors, and chemotherapy. In our patient, hypovitaminosis B1 was secondary to incoercible vomiting in the context of hyperemesis gravidarum. Hyperemesis gravidarum complicates 0.5 to 2% of pregnancies [9]. This syndrome is defined by profuse vomiting of the first trimester of pregnancy leading to weight loss, extracellular dehydration. Impermanent hyperthyroidism may be.

The first case of hyperemesis gravidarum was reported by Henderson in 1914 and rare sporadic cases have been described since [11]. The article by TogayIsikay et al. in 2001 made it possible to take stock of 30 clinical cases of GayetWernicke's encephalopathy in the context of hyperemesis gravidarum published between

1968 and 2000 [12]. In 1997, Olindo et al. Highlight the association of this syndrome with centropontine myelinolysis in the context of gestational vomiting [13]. The diagnosis of GWE is above all clinical with the classic triad [14] which combines psychic disorders (confusional syndrome, apathy, bradypsychism, hypersomnia), oculomotor disorders (horizontal or multiple nystagmus, oculomotor paralysis by involvement of III and VI), and balance disorders, in connection with a Central vestibular syndrome and cerebellar syndrome; however, this triad is only complete in 30% of cases and the deficiency can then manifest itself in hypothermia, hypotension, tachycardia, hallucinations, headache, fatigue, abdominal discomfort. Dysarthria, dysphagia, hypotonia of the lower limbs, hypoacusis, myoclonusia, dyskinesias, dystonia, epilepsy, psychosis-type psychic disorders with auditory hallucinations and delirium of persecution or bulimia have also been described. Peripheral neuropathy is often associated, but rarely sought [14]. Korsakoff syndrome is described in 80% of cases during GWE, due to damage to the hippocampo-mamillo-thalamic circuit, with predominance of mamillary abnormalities [14]. In imaging, MRI shows abnormalities in 60% of cases, implying that normal imaging does not exclude diagnosis [15,16]. We can observe in the days following the installation of clinical signs, hypersignals in T2, FLAIR and diffusion, typical by their location and their symmetrical character around the aqueduct of Sylvius, the 3rd ventricle (V3), the medial face of the thalami and especially at the level of the mamillary tubercles. Diffusion sequences objectify hypersignal areas predictive of long-term neurological sequelae [17]. In addition, these lesions inconsistently take the contrast after injection of Gadolinium chelate [18,19]. Atypical localizations have been reported, with signal abnormalities, in the form of hyperintensities in T2 and possibility of contrast taking at the level of the upper vermis, the head of the caudate nuclei and lenticular nuclei, the red nuclei, the nuclei of the facial nerve, the nuclei of the abducen nerves and the vestibular nuclei, as well as at the level of the central and precentral corte. These atypical locations can make diagnosis difficult [19]. MRI can be an interesting tool for the early diagnosis of unusual or severe forms with coma. Gold Diagnosis remains based on clinical signs and above all, on significant improvement after thiamine treatment. The scanner has not proven its diagnostic usefulness. It should be done in mind the main differential diagnoses (stroke, deep vein thrombosis, cytomegalovirus encephalitis, lymphoma). Finally, blood tests for vitamin B1 deficiency require access to specialized laboratories and results are obtained only late, making their usefulness limited in clinical practice. For management and treatment, different protocols have been proposed. Above all, vitamin B1 should be introduced quickly, parenterally, for some until vomiting has stopped and normal feeding resumed for others until the end of pregnancy. The reversibility of the disorders and the prognosis depend mainly on the duration of neurological signs before the introduction of treatment. Depending on this period, the evolution can go from the complete reversibility of clinical signs and MRI images if treatment is initiated early, to motor sequelae, Korsakoff syndrome (atrophy of the mamillary bodies), coma, or even death in 17% of cases [20] in case of diagnostic or therapeutic delay. The favorable evolution of the images is not correlated with the clinical course. If ophthalmoplegia regresses within hours of starting treatment, ataxia may take longer to recover (sequelae in 25% of cases) and more or less serious psycho-memory sequelae may be noted. In terms of fetal prognosis, according to the article by Spruill and Kuller, the fetal evolution is favorable in the various published cases, when the initiation of treatment was carried out within 24 hours after the onset of neurological disorders [20].

IV. Conclusion:

The clinical context of our patient leads us to suspect other pathologies such as cerebral venous thrombosis, a stroke, or another metabolic disorder. MRI facilitated the diagnosis by finding FLAIR hypersignals in a region of interest (periacqueductal, thalamus or mamillary body). It is appropriate to mention this deficiency in front of all neurological manifestations in a pregnant patient with hyperemesis gravidarum in order to avoid irreversible sequelae.

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