



Gayet-Wernicke Encephalopathy Major Complication of Hyperemesis Gravidarum

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Abstract

Wernicke's encephalopathy is a deficiency pathology caused by a profound deficiency in thiamine (vitamin B1). It most often occurs on an alcoholic ground, but it can occur in other pathological situation, sometimes it is difficult to diagnose but the evolution in the absence of treatment leads to severe cognitive sequelae. Magnetic resonance imaging is the reference examination to confirm the diagnosis, but in 40% of cases it is strictly normal. The diagnosis then remains based on the clinical signs and above all, on the significant improvement after treatment with thiamine. We report the case of a 32-year-old woman with hyperemesis gravidarum, causing symptomatic Wernicke's encephalopathy with normal radiology.

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Introduction

Gayet Wernicke's Encephalopathy (EGW) is a neurological emergency secondary to a deficiency in thiamine (vitamin B1) causing damage to the hippocampo-mamillothalamic network (Patez 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 with a mortality rate reaching 30% [1]. If EGW 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 uncontrollable vomiting in a pregnant woman

Observation

This is a 32-year-old patient pregnant with 20 SA without ATCD admitted to the maternity ward in a picture of consciousness disorder and bilateral ophthalmoplegia.

History of her illness dates back to the first month of her pregnancy when the patient presented uncontrollable vomiting treated several times with symptomatic treatment.

The evolution was marked by a progressive worsening of his general condition, the installation of a confusional state, diplopia and a walking disorder.



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The neurological examination revealed a confusional state, temporal and spatial disorientation, significant memory disorders, static and kinetic cerebellar syndrome, abolition of osteotendinous reflexes and hypoaesthesia mainly in the lower limbs.

Ophthalmological examination showed bilateral ophthalmoplegia at the expense of the external oculomotor nerve (VI).

Biological assessment revealed moderate hepatic cytolysis, moderate elevation of pancreatic enzymes, metabolic acidosis with hypokalaemia.

A lobar puncture was made normal return, a CT completed by following an angio MRI without particularity.

Despite an unremarkable radiological assessment but in front of a clinical symptomatology very evocative of Wernicke's encephalopathy, oral vitamin B1 supplementation was instituted, the evolution was marked by a progressive regression of the symptomatology.

Discussion

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

One in five patients does not present any of the clinical signs of the classic EGW triad, and these signs are sometimes difficult to differentiate from those of acute or chronic alcohol intoxication, the main risk area for this pathology in the Western world [7,8]. This vitamin B1 deficiency may be related to several pathological situations such as malnutrition, anorexia nervosa, prolonged parenteral nutrition without thiamine supplementation, or even gastrointestinal tumors, and chemotherapy. In our patient, hypovitaminosis B1 was secondary to uncontrollable vomiting in a context of hyperemesis gravidarum.

Hyperemesis gravidarum complicates 0.5 to 2% of pregnancies [9]. This syndrome is defined by profuse vomiting in the first trimester of pregnancy leading to weight loss, extracellular dehydration and metabolic alkalosis with hypokalaemia. Transient hyperthyroidism can be observed and contributes to the severity of hypokalaemia [10].

The diagnosis of EGW 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 impairment of III and VI) and balance disorders, related to a central vestibular syndrome and a cerebellar syndrome, this triad is however only complete in 30% of cases and the deficiency can then manifest itself by hypothermia, hypotension, tachycardia, hallucinations, headache, fatigue, abdominal discomfort. Dysarthria, dysphagia, hypotonia of the lower limbs, hypoacusis, myoclonia, dyskinesia, dystonia, epilepsy, psychic disorders such as psychosis with auditory hallucinations and delusions of persecution or bulimia have also been described. Peripheral neuropathy is often associated, but rarely sought [11]. Korsakoff syndrome is described in 80% of cases following an EGW, due to lesions of the hippocampo-mamillo-thalamic circuit, with predominance of mammillary abnormalities [11].

In imaging, MRI shows abnormalities in 60% of cases, which implies that normal imaging does not exclude the diagnosis [12,13], this is the case of our patient, but we can observe in the days following the onset 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 above all at the level of the mammillary tubercles. Diffusion sequences show zones in hypersignal predictive of long-term neurological sequelae [14].

The diagnosis remains based on the clinical signs and above all, on the significant improvement after treatment with thiamine. The scanner has not proved its diagnostic usefulness. The main differential diagnoses should be kept in mind (stroke, deep vein thrombosis, Creutzfeldt-Jakob disease, Miller-Fisher syndrome, cytomegalovirus encephalitis, lymphoma). Finally, blood tests for vitamin B1 deficiency require access to specialized laboratories and the results are only obtained late, which makes their usefulness limited in clinical practice. For care and treatment, different protocols have been proposed. Above all, vitamin B1 therapy should be introduced quickly, parenterally, for some until the vomiting stops and a normal diet is resumed for others until the end of pregnancy. The reversibility of the disorders and the prognosis essentially depend on the duration of the neurological signs before the introduction of the treatment.

Conclusion

Our clinical case reminds us that Gayet and Wernicke encephalopathy should always be suspected in the face of any confusion or oculomotor sign in a pregnant woman with vomiting during pregnancy, despite unremarkable imaging, and also to emphasize the importance of prevention through prescription. systematic vitamin B1 in all pregnant women with hyperemesis gravidarum.

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