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Review Article

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GAYET WERNICKE'S ENCEPHALOPATHY AND PREGNANCY: ABOUT 3 CASES AND REVIEW OF THE LITERATURE.

***Imane Laghrich, Soukaina Laaraj, Khalid Guelzim, Abdellah Babahabib and Jaouad Kouach**

Gynecology, Obstetrics Department, Mohamed V. Military Training Hospital, Rabat.

***Corresponding Author: Imane Laghrich**

Gynecology, Obstetrics Department, Mohamed V. Military Training Hospital, Rabat.

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ABSTRACT

Gayet Wernicke's encephalopathy (GWE) is a neuropsychiatric syndrome of metabolic origin deficiency secondary to a profound deficiency of thiamine (vitamin B1). GWE is mostly seen in alcoholics, but can also appear in case of malnutrition. In obstetrics, GWE is the result of hyperemesis gravidarum because of low thiamine, as well as increased thiamine requirements. It is difficult to diagnose and whose course in the absence of treatment leads to severe cognitive sequelae. Magnetic resonance imaging is the gold standard for confirming the diagnosis by the presence of hyper T2 signals in the periaqueductal, thalami, and mammary bodies. This work aims to analyze the diagnostic management and therapeutic management in pregnant women with Gayet-Wernicke encephalopathy, who have been hospitalized in the Gynecology-Obstetrics department of the Mohamed V Military Hospital of Instruction -Rabat.

KEYWORDS: Gayet Wernicke's encephalopathy, hyperemesis gravidarum, thiamin.

INTRODUCTION

Gayet-Wernicke encephalopathy (EGW) is a rare disease often of acute installation, it is a potentially fatal but preventable neuropsychiatric syndrome. It is characterized by the classic triad of encephalopathy, ataxia, ophthalmoplegia and/or nystagmus. However, it is imperative to diagnose the disease and start treatment as early as possible because it remains a clinical diagnosis that can be made even with normal blood thiamine levels and/or magnetic resonance imaging (MRI) normal.

This article reviews the context of the occurrence of EGW in pregnant women, the diagnostic process, and the therapeutic management based on three observations. In a second part, we will carry out a review of the literature and its main publications.

Methods: This is a retrospective study including all pregnant women with Gayet-Wernicke encephalopathy who were hospitalized in the Gynecology-Obstetrics department of the Mohammed V Military Hospital of Rabat during the period spread from 01/01/2020 to 01/01/2021, i.e. a duration of 12 months.

Patients and observations

Observation 1

Patient Information

This is a 26-year-old patient, nulliparous primigravida (pregnancy estimated at 12 weeks of amenorrhea), with a history of cholecystectomy 2 years ago, followed in gastroenterology for bile duct calculus with episode of pancreatitis 2 months before.

The symptomatology goes back to 6 days before his admission by the sudden installation in a context of uncontrollable vomiting (for 4 weeks) of a confusional syndrome and oculomotor disorders.

Clinical results

The clinical examination found a dehydrated and malnourished patient with, on the neurological examination, a 4/5 paraparesis, lively osteotendinous reflexes, right dysmetria with bilateral external rectus paralysis and multidirectional nystagmus.

Chronology

Incoercible vomiting installed for a month complicated 6 days ago with ataxia and oculomotor disorders.

Diagnostic approach

The cerebral MRI objectified

Signal anomaly on either side of the 3rd ventricle extending from the thalamus to the mammary region, around the aqueduct of Sylvius in Flair hypersignal and diffusion. Either an aspect compatible with Gayet-Wernicke encephalitis.

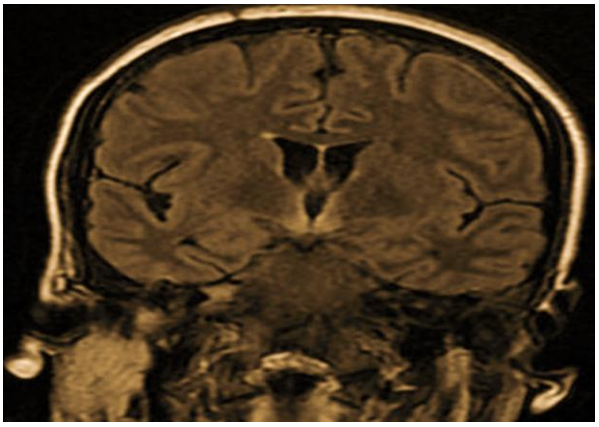

Figure 1: Brain MRI sequence in FLAIR showing symmetric hyper signal at the level of the mammillary bodies.

The encephalomyelogram shows impairment of the femorocutaneous nerve of deficiency origin.
The biological assessment objectified hypokalemia, hyponatremia with hepatic cytolysis and hypothyroidism. Kidney function was fine.
Therapeutic interventions:
The treatment consisted of rehydration with isotonic saline, antiemetics, proton pump inhibitors and potassium load. Parenteral supplementation with vitamin B1 and vitamin B6 was instituted.
Follow-up and results of therapeutic interventions:
The evolution was marked by the improvement of the symptoms of EGW which ended up disappearing after 10 days. Vitamin supplementation was continued at a preventive dose of 100mg/day for the rest of the pregnancy.

Observation 2
Patient Information
25-year-old female patient, 3rd act, primiparous (Pregnancy 1: early spontaneous abortion, Pregnancy 2: vaginal delivery), Pregnancy 3: the current pregnancy is estimated at 17 weeks+6 days, with no particular history.

Clinical Results
Neurological examination finds
- horizontal diplopia
-horizontal nystagmus beating to the right
- balance disorder with incoordination of walking and ataxia.

Chronology
Admitted for management of uncontrollable vomiting dating back to 2 months complicated a week ago with headache, dizziness, blurriness and double vision.

Diagnostic approach
Brain MRI had objectified
On the FLAIR sequence, symmetrical and bilateral lesions in hypersignal at the level of the thalamus, quadrigeminal tubercles and at the periaqueductal level. The venous MRI angiography was normal. (Figure 2)

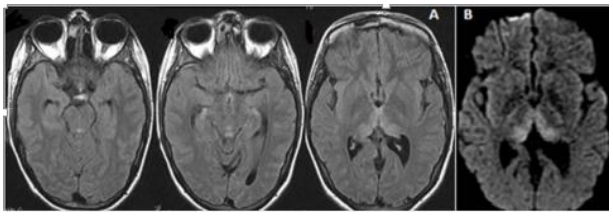

Figure 2: Brain MRI in FLAIR sequence (A), diffusion (B) showing symmetric and bilateral hyper signal lesions in the paramedian region of the thalamus, mammillary bodies, and periaqueductal region.

The biological assessment had objectified hypokalaemia, hyponatremia, a lipasemia twice the normal, hepatic assessment and renal function correct, thiamine dosage was low.

Therapeutic Intervention
The patient benefited from rehydration with correction of ionic disorders with cessation of oral feeding, antiemetics and gastric protection intravenously, anticoagulation at a preventive dose and vitamin B1 B6 supplementation.

Follow-up and results of therapeutic interventions
The evolution was marked by the improvement of the symptoms of the EGW with correction of the biological disorders. Oral vitamin supplementation was maintained at a preventive dose of 250 mg/day during the rest of the pregnancy, which resulted in premature rupture of the membranes with premature delivery at 34 weeks of amenorrhea with simple sequels in the mother and the newborn. born.

Observation No. 3
Patient Information
This is a 27-year-old patient, 2nd primiparous procedure (1st pregnancy: vaginal delivery), with no notable pathological history.

Clinical Results
On neurological examination
Patient disoriented in time and space, confused
multidirectional nystagmus,
VII nerve paresis,
Taxi Walk,
Static and kinetic cerebellar syndrome,
Osteotendinous reflexes present and symmetrical.

Chronology
Patient admitted in a picture of confusion with memory disorders, oculomotor disorders and ataxia of progressive installation for a week. In addition, the family reports the notion of incoercible vomiting for a month and a half in a context of weight loss of more than 10% of the initial weight, with the notion of hospitalization in another hospital structure.

Diagnostic approach
The obstetric ultrasound objectified a monofetal and evolving pregnancy with a biometry corresponding to 18 SA.

The biological assessment showed hypokalemia, hyponatremia, hypochloremia, hepatic cytolysis 10 times normal, lipasemia 2 times normal, hyperthyroidism, serologies for viral hepatitis (A, B and C) were negative. Liver ultrasound was unremarkable.

The clinical and biological picture was in favor of Gayet-Wernicke encephalopathy secondary to hyperemesis gravidarum with severe hydro-electrolyte disorders.

Therapeutic interventions
The patient received emergency rehydration with correction of hydro-electrolyte disorders, vitamin therapy.

Follow-up and results of therapeutic interventions
A marked improvement in clinical and biological signs after one week. The MRI done 10 days later was normal.

DISCUSSION
Gayet Wernicke's encephalopathy (EGW) (called Wernicke-Korsakoff in the Anglo-Saxon literature) described for the first time by Wernicke in 1881.[1]

It is a rare but fatal complication secondary to a thiamine deficiency. It is frequently encountered in alcoholics, but several cases have been reported in the context of uncontrollable vomiting during pregnancy. Its frequency is underestimated because several autopsy cases have been described.[1] Its association with hyperhemesis gravidarum was first described by Sheehan in 1939.

In our patients, hypovitaminosis B1 was secondary to uncontrollable vomiting in a context of hyperemesis gravidarum, the latter complicating 0.5 to 2% of pregnancies.[2] This syndrome is defined by profuse vomiting occurring during the first trimester of pregnancy complicated by weight loss, extracellular dehydration and hypokalaemia with metabolic alkalosis.

Transient hyperthyroidism can be observed and contributes to the severity of this vomiting as well as to the severity of the hypokalaemia.[3]

In the literature, Henderson had reported the first case related to hyperemesis gravidarum in 1914 and then rare sporadic cases were observed.[4]

The diagnosis of the EGW is especially clinical posed in front of the triad[5] which associates.
- Psychic disorders: confusional syndrome, apathy, Brady psychism, hypersomnia
- Oculomotor disorders: horizontal or multiple nystagmus, oculomotor paralysis due to damage to III and VI).
- Balance disorders related to central vestibular syndrome and cerebellar syndrome.

However, this triad is only complete in 30% of cases, the deficiency can also manifest itself by hypotension, hypothermia, hallucinations, headaches, asthenia, abdominal discomfort.

In addition, dysarthria, dyskinesias, dysphagia, hypoacusis, myoclonus, hypotonia of the lower limbs, dystonia, epilepsy, psychosis with auditory hallucinations and delusions of persecution or bulimia have also been described.

Korsakoff's syndrome is described in 80% of cases following an EGW, due to lesions of the hippocampo-mamillo-thalamic circuit, with predominance of mammillary abnormalities.[5]

In imaging, MRI makes it possible to objectify abnormalities in 60% of cases, and therefore normal imaging does not exclude the diagnosis.[6,7] In the days following the onset, clinical signs, hypersignals in T2, FLAIR and diffusion, typical localized around the aqueduct of Sylvius, the 3rd ventricle (V3), the medial surface of the thalami and especially at the level of the mammillary tubercles, having a symmetrical character. Diffusion sequences highlight zones in hypersignal predictive of neurological sequelae observed in the long term.[8] Moreover, these lesions take contrast inconsistently after injection of gadolinium.[9,10]

Atypical localizations have been reported, with signal anomalies, in the form of hyperintensities on T2 with possible contrast enhancement at the level of the head of the caudate nuclei of the superior vermis, and of the lenticular nuclei, red nuclei, nuclei of the facial nerve, and vestibular nuclei, as well as in the central and precentral cortex. These atypical locations sometimes make the diagnosis difficult.[10] MRI plays a key role in the early diagnosis of unusual or severe forms with coma. However, these signal anomalies are not pathognomonic even if they are very evocative.

The diagnosis is based on the clinical signs and above all, on the spectacular improvement after treatment with thiamine. Moreover, the CT scan has not proved its usefulness for the diagnosis of this pathology.

Finally, biological assessments looking for a vitamin B1 deficiency require the use of specialized laboratories and the results are obtained only late, which makes their usefulness very limited in clinical practice.

The main differential diagnoses are: stroke, deep vein thrombosis, Miller-Fisher syndrome, cytomegalovirus encephalitis, lymphoma, Creutzfeldt-Jakob disease).

For therapeutic management, several protocols have been proposed. Above all, thiamine supplementation should

be quickly introduced, parenterally, then orally for some until vomiting stops and normal food is resumed for others until the end of pregnancy.

For the fetal impact, we note above all intrauterine growth restriction, prematurity and fetal death in utero. But we still do not know the consequences on the brain development of the child and the possible long-term neurological sequelae.

The evolution of the disease depends on the delay in treatment, which can range from complete reversibility of clinical and radiological signs if treatment is started early, to motor neurological sequelae, Korsakoff syndrome (mammillary body atrophy), coma, or even death in 17% of cases[11] if diagnostic or therapeutic delay. It should be noted that the favorable evolution of the images is not always correlated with the evolution of the clinical signs.

The ophthalmoplegia may regress rapidly in the hours following the initiation of treatment, however, the ataxia may take longer to recover (may have sequelae in 25% of cases); thus psycho-mnesic sequelae may appear.

For the fetal prognosis, in the literature, the fetal evolution is favorable in the various published cases, when the start of the treatment was carried out within 24 hours after the onset of the neurological disorders.[11]

CONCLUSION

Gayet-Wernicke encephalopathy is a rare complication of hypermesis gravidarum, serious but reversible, whose diagnosis is urgent given its potentially fatal nature.

Conflicts of interest

The authors declare no conflict of interest

Contribution of the Authors

All authors contributed to the conduct of this work. The authors also declare that they have read and approved the final version of the manuscript.

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