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Case Report

Gayet-Wernicke Encephalopathy Complicating a Pregnancy of 17 Weeks of Amenorrhea: A Case Report

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Abstract

Gayet-Wernicke encephalopathy is a neuropsychiatric emergency due to thiamine (vitamin B1) deficiency, secondary to several factors, it is a disorder characterized by confusion of acute onset, nystagmus, partial ophthalmoplegia and ataxia. The diagnosis is mainly clinical. The disorder may resolve with treatment, persist, or degenerate into Korsakoff's psychosis. We report a case of Gayet-Wernicke encephalopathy in a 39 year old non-ethylic patient, pregnant at 17 weeks of age who presented with consciousness disorders with paresthesia of the 4 limbs and dyspnea. Clinical and magnetic resonance imaging (MRI) have an important place, especially in the diagnosis of non-alcoholic Wernicke's encephalopathy.

Keywords: nystagmus, magnetic resonance imaging (MRI), Gayet-Wernicke encephalopathy.

INTRODUCTION

Gayet-Wernicke encephalopathy (WE) is a severe neurological complication caused by thiamine deficiency in malnourished individuals, especially alcoholics, patients operated for digestive surgery or suffering from gastrointestinal tract's diseases or incoercible vomiting. WE can lead to serious complications such as Korsakoff's syndrome, coma, or even death. To avoid these devastating outcomes, the diagnosis must be made early. Classically it manifests by confusion with oculomotor disorders and ataxia [1]. However, other neurological manifestations are possible. MRI is the examination of choice to confirm diagnosis and assess the lesion [2].

We report in this work a case of gayet wernicke encephalopathy discovered in the context of gravidic vomiting and we will discuss through this case the clinical symptoms, the diagnostic means, the complications, the therapeutic modalities as well as the prevention of this syndrome.

CASE REPORT

39 year old patient, with no history of alcohol consumption, hypertensive, diabetic, G2P2 current pregnancy estimated at 17 SA, was admitted to the emergency room for persistent vomiting evolving over two-months. The clinical status of the patient worsened gradually; she was confused, glascow score at 12, TA=16/9 pulse at 110, she presented ataxia, horizontal nystagmus, paresthesia of the 4 members.

A gynecological examination finds minimal bleeding with a dilated cervix at 2cm, the ultrasound showed an arrested pregnancy with a biometry corresponding to 18 SA, and the patient had expelled 45 minutes after her admission.

During her hospitalization in intensive care, the patient presented a deterioration of her neurological and respiratory condition, and intubation was indicated.

A cephalic MRI performed showed signal abnormalities of the periaqueductal region, the walls of the V3, the tectal lamina, and the posterior thalami in favor of a Gayet-Wernicke encephalopathy (Fig 1).

The therapeutic protocol in the intensive care unit was as follows: vit B1 B6 supplementation, LMWH, Loxen, rehydration with motor and respiratory physiotherapy sessions.

The evolution was marked by the improvement of her respiratory function currently the patient is tracheotomized with krishaber cannula.

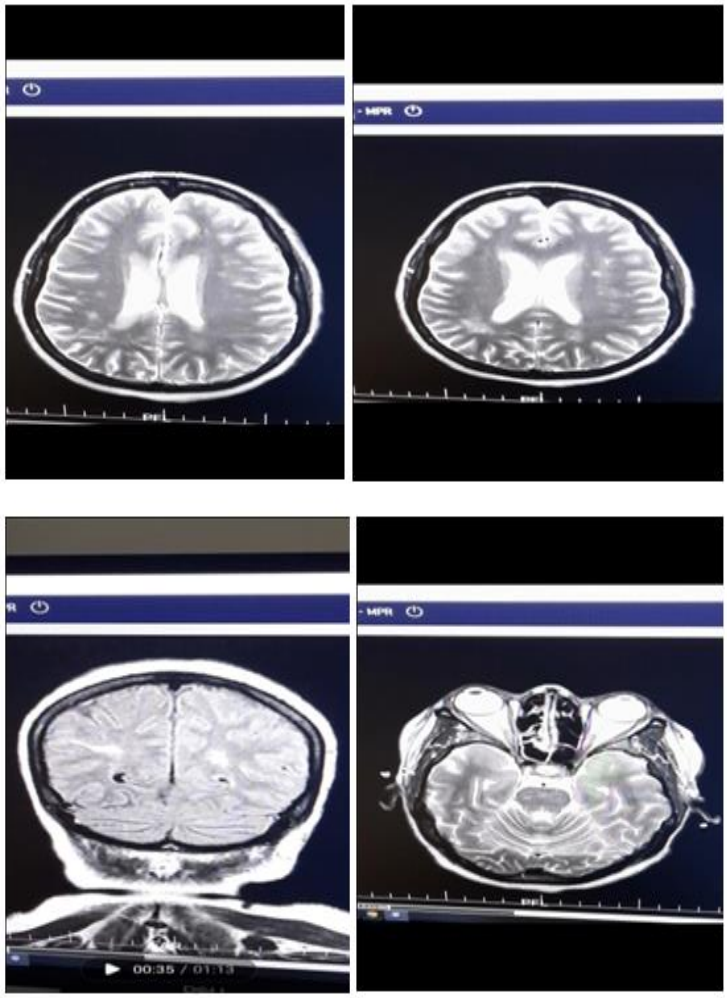

Fig 1: Cerebral MRI

DISCUSSION

WE is mostly seen in alcoholics but can also occur in any malnourished state. The prevalence of WE in a nonalcoholic patient varies from 0.04% to 0.13% [3]. It should be considered in patients with anorexia nervosa, prolonged vomiting associated with chemotherapy, gastrointestinal disease, haemodialysis, and HG [4]. In a large literature review, the most frequent causes of WE in nonalcoholic patients were neoplastic disease (18.1%) and gastrointestinal surgery (16.8%) [5].

Thiamine is an important co enzyme for 3 enzymes: α-ketoglutarate dehydrogenase complex, pyruvate dehydrogenase complex, and transketolase (enzyme metabolism and degradation of glucose). It is also essential in maintaining osmotic gradients across cell membranes. The mechanism through which its deficit causes brain lesions is unknown, although it is believed that neuronal damage begins once the metabolism in brain regions with high metabolic requirements and high thiamine turnover is inhibited [1]. Time to deplete the body's store of thiamine is about 3 weeks. The daily requirement of thiamine is around 1.1 mg/day for females, and it increases to 1.5 mg/day, particularly during pregnancy and lactation [6], and even more by the impaired absorption due to HG.

The clinical diagnosis of WE is missed in 75–80% of cases [6]. The classical triad of gait ataxia, eye signs (nystagmus, ophthalmoplegia) and global confusion, as described by Karl Wernicke in 1881, is seen in only 16–20% of patients. More rarely, a decrease in deep tendon reflexes, peripheral polyneuropathy and decreased tone or dysarthria were observed [7].

Caine *et al*., developed what are known as operational criteria for the diagnosis of WE. Based on these criteria, WE should be diagnosed when 2 of the following 4 signs are present: dietary deficiency, oculomotor abnormalities, unsteadiness of stance and gait, and either altered mental state or mild memory

impairment [8]. Our patient presented with the classical clinical triad following intractable vomiting, MRI also detected bilateral and symmetric specific lesions. The clinical and radiological presentation was in favor of high suspicion of an acute thiamine deficiency, indicating immediate thiamine supplementation.

WE is considered a medical emergency. Early identification and immediate intervention, as outlined are crucial to producing positive patient outcomes. Even in the absence of a confirmed diagnosis, it is advised that clinicians initiate interventions in suspected cases.

The prognosis of WE depend on the stage of the disease and prompt institution of thiamine. Evolution can include the full reversibility of disorders, motor sequelae, Korsakoff's syndrome, coma and even death 1. The mortality rate ranges from 20 to 30% [9].

CONCLUSION

WE is a medical emergency that requires intravenous thiamine administration as soon as the diagnosis is made to prevent any neurological sequelae, including Korsakoff syndrome. This rare complication in non-alcoholic patients deserves to be better known by clinicians. Preventive treatment of patients at risk of thiamine deficiency may prevent fatal consequences.

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