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Non-Alcoholic Gayet-Wernicke Encephalopathy in an Elderly Malnourished Patient: A Case Report and Review of Diagnostic and Therapeutic Approaches

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Abstract **Case Report**

Wernicke's encephalopathy results from a thiamine deficiency. It is an underdiagnosed pathology, particularly in the geriatric population, with irreversible consequences. We report a case of a Wernicke's encephalopathy in an elderly patient who was hospitalized for anorexia, in a context of inappropriate re-feeding. Neurological deterioration was observed following the instatement of parenteral nutrition. Brain MRI evidenced anomalies suggestive of Wernicke's encephalopathy. Intravenous treatment with thiamine provided an improvement in mental alertness, nevertheless contrasting with an aggravation of her cognitive disorders. This case illustrates the gravity of Wernicke's encephalopathy, which entails a risk of severe complications among elderly, malnourished patients. It is a diagnostic and therapeutic emergency.

Keywords: Non-alcoholic Wernicke encephalopathy, Gayet-Wernicke encephalopathy, Thiamine deficiency, Elderly malnutrition, Acute neuropsychiatric syndrome.

INTRODUCTION/BACKGROUND

Gayet-Wernicke encephalopathy (GWE) is a severe neurological disorder caused by thiamine (vitamin B1) deficiency, with multiple etiologies, the most common being alcohol dependence. Recently, an increasing number of non-alcoholic GWE cases have been reported [1], particularly among malnourished populations, in oncological pathologies [2], gastrointestinal disorders following bariatric surgeries [3], and in hyperemesis gravidarum [4]. Its symptomatology is subtle and nonspecific, with the typically described triad (confusion, oculomotor disturbances, ataxia) present in only 16 to 20% of cases [5]. This condition is often underdiagnosed and undertreated, particularly in the geriatric population, leading to serious and potentially irreversible consequences. We report here the case of an 80-year-old malnourished female patient who developed Gayet-Wernicke encephalopathy.

CASE REPORT

A 80-year-old man was admitted to the hospital for a one-month history of worsening nausea, vomiting, anorexia, weakness, vertigo and a general deterioration in health, accompanied by a 10-kilogram weight loss within a month. Her medical history includes severe major cognitive impairment due to Alzheimer's disease and chronic gastritis he does not consume alcohol.

Upon admission, the patient's overall condition was precarious, with significant clinical malnutrition: he weighed 57 kg at a height of 1.75 meters, resulting in a BMI of 13.9 kg/m², and had an albumin level of 15 g/L. he exhibited advanced cognitive impairment, with signs of delirium, agitation, psychomotor slowing, fluctuating alertness, and disorientation in time and space. Additionally, he had been experiencing vertigo and postprandial vomiting for 15 days, with a distended abdomen but no signs of bowel obstruction, An abdominal-pelvic CT scan showed no significant abnormalities.

A neurologic examination showed horizontal nystagmus and ataxia. Cerebral magnetic resonance imaging (MRI) revealed FLAIR hyperintensities in the periaqueductal region, mammillary bodies and the walls of the third ventricle, (Figure 1) consistent with Gayet-Wernicke encephalopathy.

Laboratory examination showed low thiamine levels of 35 nmol/L (ref 76-183 nmol/L).

The patient was immediately treated with intravenous thiamine to prevent irreversible complications and subsequently improved within 12 hours. The patient continued to receive 500 mg of intravenous thiamine every eight hours for three days with full resolution of nystagmus, ataxia, nausea, vomiting, and fatigue.

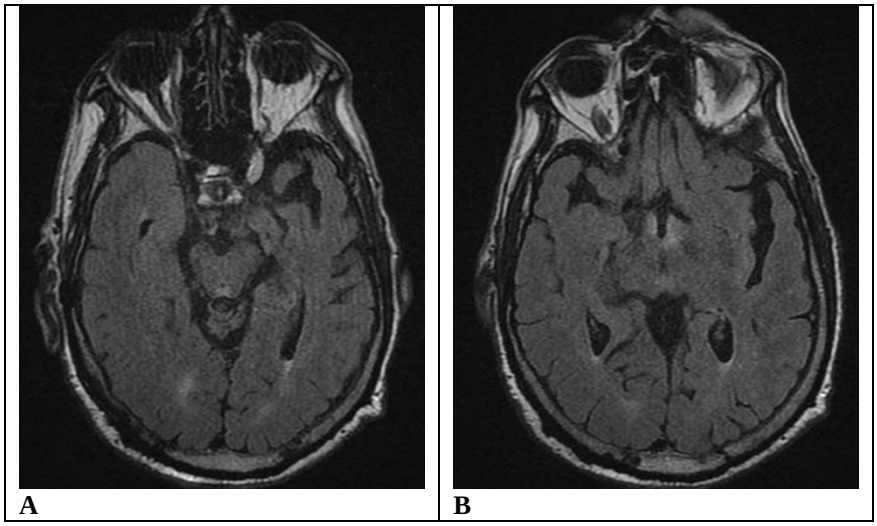

Figure 1: Magnetic resonance imaging (MRI) of the brain was performed without contrast; (A) Fluid-attenuated inversion recovery (FLAIR) MRI demonstrated hyperintensity of the periaqueductal midbrain, (B) the mammillary bodies and the walls of the third ventricle

DISCUSSION

Wernicke encephalopathy is an acute neuropsychiatric syndrome caused by thiamine deficiency with a classic triad of nystagmus and ophthalmoplegia, mental status changes, and ataxia [6]. It occurs more commonly in patients who abuse alcohol Furthermore, cases of non-alcoholic Gayet-Wernicke encephalopathy are increasingly being reported in individuals suffering from dementia, those who are neglected or abused, and in patients receiving inadequate parenteral nutrition [7]. This condition is currently underdiagnosed, yet its incidence in the general population is relatively common, ranging from 0.4% to 2.8%.

The diagnosis of Gayet-Wernicke encephalopathy (GWE) is clinical and relies on the often incomplete triad described by Wernicke: confusion, oculomotor disturbances, and cerebellar ataxia [8]. The most consistent sign is altered consciousness or a confusional state [9]. Repeated vomiting and significant weight loss are major predictive factors for non-alcoholic GWE [10]. There is no specific diagnostic test for GWE. Direct blood measurement of thiamine or its phosphorylated forms is recommended but should never delay treatment. To date, there is no consensus or clinically relevant threshold to define a vitamin B1 deficiency, and a normal thiamine level does not always exclude the diagnosis of deficiency [7].

Cerebral computed tomography (CT) has a diagnostic sensitivity of only 13% in GWE and is not recommended, as it is usually normal in the initial stages. It may show hypodensities in the cerebellum and mammillary bodies, progressing to mammillary body atrophy in the chronic phase. The sensitivity of cerebral magnetic resonance imaging (MRI) is 53-58%, with a specificity of 93%. In non-alcoholic GWE patients, FLAIR sequence lesions are present in 100% of cases, making MRI the preferred diagnostic tool. However, its primary limitation is its accessibility and availability. Brain lesions typically appear 2 to 3 weeks after the onset of the first symptoms, corresponding to the time it takes to deplete vitamin B1 stores. These lesions are initially glial before becoming neuronal, and the damage is irreversible once neuronal involvement occurs. The most vulnerable areas are seen as hyperintensities on T2 and FLAIR sequences (typically bilateral), restricted diffusion, including the thalami, mammillary bodies, quadrigeminal plate, cerebellum, and periaqueductal region. Contrast enhancement can also be seen in the same regions, most commonly of the mammillary bodies [11, 12].

In the differential diagnosis of imaging findings suggestive of Gayet-Wernicke encephalopathy, several conditions should be considered. Leigh disease, for instance, typically spares the mammillary bodies, which distinguishes it from Gayet-Wernicke encephalopathy. Metronidazole-induced encephalopathy should also be included in the differential, as it involves the dentate nuclei, cranial nerve nuclei, and the splenium. Additionally, bilateral medial thalamic abnormalities may be indicative of an artery of Percheron infarct or central venous thrombosis. These conditions present with overlapping imaging features, necessitating careful evaluation of the specific regions involved to ensure accurate diagnosis [13].

The treatment of acute Wernicke-Korsakoff syndrome primarily involves the administration of intravenous thiamine hydrochloride, often in conjunction with other vitamins and minerals, alongside addressing the underlying cause, such as alcohol cessation. Without treatment, the condition carries a high mortality rate of up to 20%. The European Federation of Neurological Societies and the Royal College of Physicians recommend administering 500 mg of parenteral thiamine three times daily until the symptoms of acute Wernicke encephalopathy resolve. This treatment is lifesaving and can potentially reverse the acute neuropsychiatric manifestations of the syndrome. Suboptimal treatment is defined as administering less than 500 mg of parenteral thiamine as the initial dose, which may result in poorer outcomes [14, 15].

CONCLUSION

Gayet-Wernicke encephalopathy (GWE) is an underrecognized condition, particularly in the geriatric population. Its prevention is crucial for elderly, multimorbid, and malnourished patients, due to the potential for severe or even fatal complications. MRI plays a fundamental role in the diagnosis of GWE, providing essential imaging evidence to support clinical findings.

Declaration of Interest: All authors declare no conflicts of interest.

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