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(CASE REPORT)

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Wernicke's encephalopathy complicated by perforated gastric ulcer: A case report

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Abstract

Wernicke's encephalopathy (WE) is a neurological disorder that arises from thiamine (vitamin B1) deficiency, often seen in patients with chronic alcoholism, malnutrition, or other conditions affecting thiamine absorption. This case report discusses a 50-year-old woman with a history of hypertension, chronic smoking, alcohol consumption, and a previous hemorrhagic gastric ulcer who developed WE complicated by a perforated gastric ulcer. The patient's condition highlights the importance of considering WE in the differential diagnosis of patients presenting with gastrointestinal pathology and neurological symptoms, especially when there are risk factors for thiamine deficiency.

Keywords: Wernicke's encephalopathy; Chronic alcohol consumption; Gastric ulcers; Thiamine; PPIs

1. Introduction

Wernicke's encephalopathy is an acute, life-threatening disorder resulting from thiamine deficiency, commonly seen in individuals with chronic alcoholism, malnutrition, or conditions impairing thiamine absorption. Thiamine is a cofactor for enzymes involved in carbohydrate and lipid metabolism. When thiamine is depleted, enzymatic activity decreases, leading to neurological impairment due to osmotic imbalances, edema, and neuronal cell death. Although the classic triad of ataxia, confusion, and nystagmus is typical, the symptoms may vary, and early diagnosis is crucial to prevent irreversible damage. In this case, the patient was hospitalized with gastrointestinal symptoms but later developed neurological symptoms characteristic of WE, complicating the clinical picture.

2. Case Presentation

A 50-year-old woman with a significant medical history, including hypertension (HTA), chronic smoking, alcoholism, and a hemorrhagic gastric ulcer treated non-surgically two years earlier, presented with epigastric pain of 3 days' duration. The patient also reported abdominal tenderness and muscular guarding on clinical examination. She had been prescribed omeprazole for gastric protection after her previous ulcer.

The patient's vital signs were stable, but she exhibited epigastric defense on palpation, suggesting a significant abdominal pathology. A contrast-enhanced abdominal CT revealed the following:

Esophagitis involving the lower third of the esophagus, Hypertrophic gastritis, and perforated gastric ulcer located on the posterior aspect of the Antro pyloric region, along with splenic infarction affecting the upper third of the spleen, without evidence of intra-abdominal effusion (figure1)

The gastric perforation was managed surgically, with a non-invasive approach utilizing the Taylor method, which involved nasogastric suction using a Salem tube, gentle aspiration, and the initiation of broad-spectrum antibiotics.

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Despite improvement in her abdominal condition after three days, with no abdominal pain and a soft abdomen, the patient developed new neurological symptoms.

3. Neurological Symptoms and Diagnosis

On day three of hospitalization, the patient exhibited mental slowing, disorientation, anterograde amnesia, ophthalmoplegia, nystagmus, and ataxia, suggesting a possible central nervous system (CNS) disorder. Laboratory results revealed low thiamine levels (10.7 ng/mL), confirming a thiamine deficiency (normal range: 21.3–81.9 ng/mL). Other laboratory tests for vitamins B2, B6, B12, folic acid, nicotinic acid, electrolyte balance, and kidney and thyroid functions were normal. A brain MRI showed a hypersignal in the splenium of the corpus callosum, characteristic of Wernicke's encephalopathy (Figures 2). Additional findings included periventricular white matter lesions, confirming the diagnosis of WE. The thoraco-abdominal CT confirmed the previously identified esophagitis and gastric pathology.

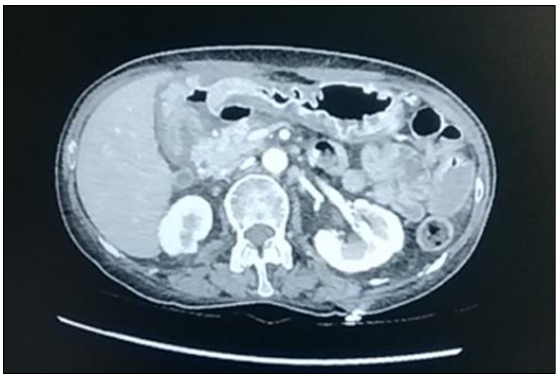

Figure 1 Abdominal CT scan Perforated peptic ulcer

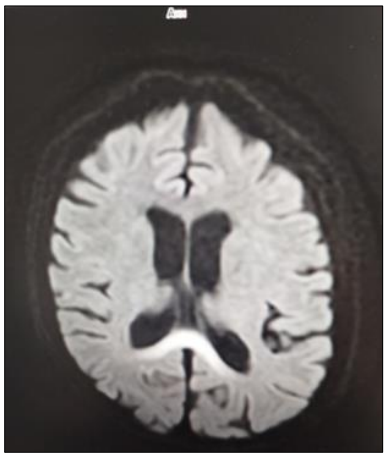

Figure 2 A brain MRI showed a in the splenium of the *corpus callosum*

3.1. Management

Given the clinical and radiological findings, the patient was diagnosed with Wernicke's encephalopathy due to thiamine deficiency. Immediate treatment was initiated with intravenous thiamine (1500 mg/day), based on current clinical guidelines. Her condition began to improve significantly within the following weeks. By day 21 of hospitalization, the patient regained the ability to walk independently, with significant resolution of her neurological symptoms. A follow-up brain MRI performed a month later showed partial resolution of the initial hypersignal and white matter changes, consistent with a favorable response to therapy.

4. Discussion

Wernicke's encephalopathy occurs due to thiamine deficiency, which leads to impairment in key metabolic processes, particularly those involving the pyruvate dehydrogenase complex and transketolase enzymes [1]. Thiamine deficiency

impairs carbohydrate metabolism, causing neuronal dysfunction, cytotoxic edema, and eventual neuronal cell death. Symptoms of WE can vary, but the classic triad of ataxia, confusion, and nystagmus is seen in 16–38% of cases. In this patient, the neurological presentation included disorientation, nystagmus, and ataxia [2].

This case highlights several important aspects of WE, particularly in patients with gastrointestinal disease and alcoholism. The patient's gastric ulcer and hypertrophic gastritis likely contributed to impaired thiamine absorption. The use of proton pump inhibitors (PPIs) like omeprazole, which are commonly prescribed to manage gastric ulcers, can further exacerbate thiamine malabsorption by reducing gastric acid secretion. While hypomagnesemia is often associated with thiamine deficiency, it was not observed in this case, suggesting that gastric mucosal lesions played a more significant role in the impaired thiamine absorption [3].

Although thiamine is absorbed primarily in the duodenum, high-affinity thiamine transporters are also present in the stomach, suggesting that gastric absorption may contribute to thiamine intake. Genetic predispositions may also play a role in some cases, with polymorphisms in thiamine transporters or transketolase potentially influencing the severity of deficiency and susceptibility to WE, despite the absence of other classical risk factors.

The treatment of WE is based on thiamine supplementation, with intravenous administration being the preferred route due to the poor bioavailability of oral thiamine (3.7–5.3%). The recommended regimen is 500 mg of intravenous thiamine three times daily for the first three days, followed by 250 mg daily until symptoms resolve or plateau. Continuous supplementation is necessary for patients at ongoing risk, such as those with alcohol use disorder or gastrointestinal pathologies.

5. Conclusion

This case underscores the importance of considering Wernicke's encephalopathy in patients with neurological symptoms and gastrointestinal disease, particularly in those with a history of chronic alcoholism and gastric ulcers. Early recognition and prompt thiamine replacement are essential to prevent irreversible neurological damage. Additionally, gastrointestinal conditions, such as gastric ulcers, hypertrophic gastritis, and the use of proton pump inhibitors, can impair thiamine absorption and contribute to the development of WE. Clinicians should be vigilant in recognizing the signs of WE in at-risk patients and initiate treatment early to optimize patient outcomes.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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