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Wernicke Encephalopathy Induced by Ileus after Gastrectomy in Gastric Cancer: A Case Report

Chun Cao[1], Xing Li[2], Yunfeng Yao[1*]

[1]Department of Oncology, The 960th Hospital of the Chinese PLA Joint Logistic Support Force, Jinan, China
[2]Department of Cardiology, The 960th Hospital of the Chinese PLA Joint Logistic Support Force, Jinan, China
Email: *yaoyunfeng.1976@163.com

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Abstract

Wernicke encephalopathy (WE) is a clinically significant condition where delayed treatment can lead to severe consequences. We report a case of a 52-year-old woman with recurrent gastric cancer one year after surgery, who developed a postoperative bowel obstruction. Following supportive care with clinical improvement of her primary condition, she rapidly developed dizziness, lethargy, and confusion. Brain MRI confirmed the diagnosis of WE. Prompt intravenous thiamine (vitamin B1) supplementation resulted in rapid neurological symptom resolution. Follow-up MRI demonstrated remarkable regression of the positive brain lesions.

Keywords

Wernicke Encephalopathy, Thiamine Deficiency, Gastric Cancer, Bowel Obstruction

1. Introduction

Wernicke encephalopathy (WE) represents an acute neurological emergency caused by thiamine (vitamin B1) deficiency, disrupting cerebral energy metabolism due to impaired glucose utilization. First described by Carl Wernicke in 1881 in alcoholic patients, WE classically present a triad of ocular abnormalities (nystagmus, ophthalmoplegia), ataxia, and confusion or memory impairment. Historically linked to chronic alcoholism, contemporary evidence underscores its rising incidence in non-alcoholic conditions, including hyperemesis gravidarum, bariatric surgery, malnutrition, gastrointestinal malignancies, and extreme caloric re-

striction—particularly in pediatric obesity management. This epidemiological shift necessitates heightened clinical vigilance beyond traditional risk profiles [1]. Diagnosis remains notoriously challenging. Few patients exhibit the complete triad, most present with partial or atypical symptoms like isolated vertigo, hypotonia, or confabulation, leading to underrecognizing. Although MRI is beneficial for diagnosing, reliance on classic signs or imaging alone delays life-saving intervention. The European Federation of Neurological Societies (EFNS) guidelines thus emphasize presumptive treatment based on clinical suspicion in high-risk scenarios [2]. Treatment demands urgent high-dose parenteral thiamine. The cornerstone of treatment is prompt parenteral administration of high-dose thiamine. Intravenous (IV) delivery is paramount due to its rapid bioavailability, with initial doses of 500 mg IV thiamine administered 2 - 3 times daily for 3 - 5 days, followed by 250 - 500 mg daily for up to a week. High-dose IV thiamine (500 mg) is a safe, effective first-line intervention for WE. Adherence to evidence-based dosing and integrated rehabilitation optimizes neurological recovery and reduces mortality [2].

2. Case Presentation

A 54-year-old female patient was diagnosed with gastric cancer two years ago and underwent radical subtotal gastrectomy, followed by adjuvant chemotherapy. Six months later, follow-up examinations revealed tumor recurrence with metastasis, prompting a change in the chemotherapy regimen. During treatment, the patient developed a surgical wound-related intestinal obstruction, which was managed with symptomatic and nutritional support, enema, and medications to promote gas passage and defecation, leading to symptomatic relief. However, she soon developed neuropsychiatric symptoms including dizziness, lethargy, and confusion. Upon the onset of the condition, the patient's vital signs were stable, with a heart rate of 80 bpm, respiratory rate of 20 breaths/min, blood pressure of 110/65 mmHg, and body temperature of 36.5˚C. Following the emergence of psychiatric symptoms, laboratory tests revealed the following: Complete blood count: WBC 5.38×10^9/L, neutrophils 77%, RBC 4.3×10^{12}/L, Hb 116 g/L, platelet count 151×10^9/L; Liver function: ALT 28 U/L, AST 26 U/L, total protein 74 g/L, albumin 43 g/L, total bilirubin 21.1 μmol/L, direct bilirubin 7 μmol/L; Biochemistry: potassium 3.35 mmol/L, sodium 136.7 mmol/L, calcium 2.35 mmol/L, magnesium 0.83 mmol/L, creatinine 44 μmol/L, urea 5.9 mmol/L, glucose 5.13 mmol/L; Coagulation profile: D-dimer 1.08 mg/L. No significant abnormalities were detected in urinalysis, stool routine, or electrocardiogram. Chest CT showed new small pulmonary nodules without enhancement, minimal pleural effusion, and no signs of inflammation. Abdominal CT findings were consistent with status post-gastrectomy, improved bowel wall edema, bilateral hydronephrosis, right-sided ureteral dilation, and a small amount of ascites. Neurological examination indicated drowsiness, slowed responsiveness, coherent speech, clear articulation, and spatial disorientation. The tongue protruded midline, with limited abduction and upward gaze bilaterally. Muscle strength was largely normal in the upper extremities and

graded 2+ in the lower extremities. Both plantar reflexes were negative. Due to suspected cerebral involvement, we need consideration of cerebral infarction, hematencephalon, or metastatic encephaloma. The brain Magnetic resonance imaging (MRI) was performed consequently. MRI showed T2 hyperintensities involving the medial sides of the two thalami, the dorsal side of the brainstem, periphery of the third ventricle and the cerebral aqueduct of the midbrain (**Figure 1**). There were no contrast enhancement lesions. These imaging findings raised the suspicion of WE and ruled out cerebral infarction, hematencephalon, or metastatic encephaloma basically. As the patient with recurrent gastric cancer complicated by intestinal obstruction, she experienced inadequate nutritional intake and malabsorption during treatment, which lead to vitamin B1 deficiency. Moreover, to meet the daily glucose metabolic demands, daily intravenous fluids often contained dextrose, accelerating vitamin B1 consumption. Based on the clinical presentation, a diagnosis of Wernicke's encephalopathy due to vitamin B1 deficiency was considered. Intravenous vitamin B1 supplementation was promptly initiated at a dose of 500 mg dissolved in normal saline, administered three times daily for three consecutive days. This was followed by intramuscular injection at 200 mg once daily for one week, and then switched to oral administration at 40 mg Qd for a month after discharge. Patient's symptoms of gaze palsy and drowsiness were improved obviously after reception of vitamin B1 intravenous infusion. Within one week, the drowsiness, confusion, and memory impairment resolved completely, with significant overall psychological improvement. The reconfirmation of the brain MRI showed T2 hyperintensities in original cerebral zone is decreasing (**Figure 2**). After discharge, the patient continued oral vitamin B1 at 40 mg once daily. During three months of follow-up, there was no recurrence of the aforementioned symptoms.

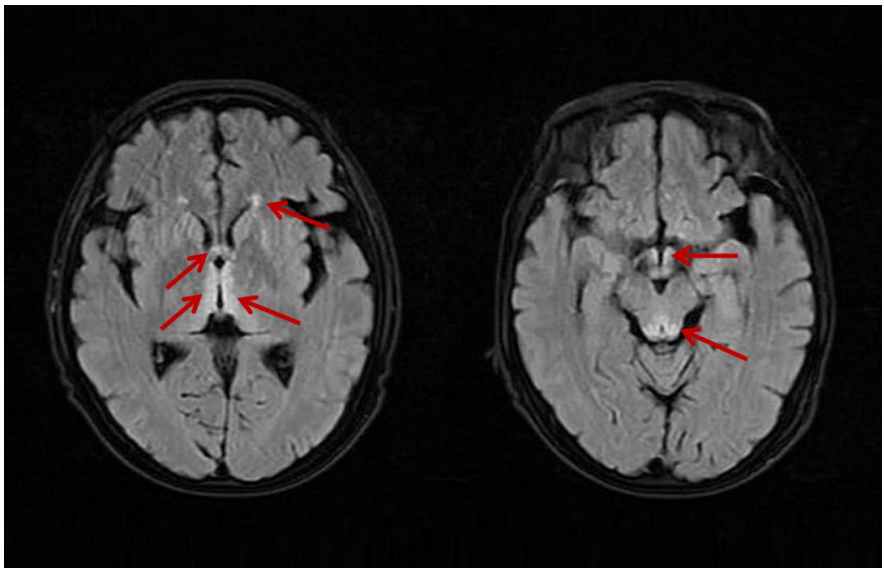

Figure 1. Results of cranial magnetic resonance scan: T2 hyperintensities in the area (the red arrow points).

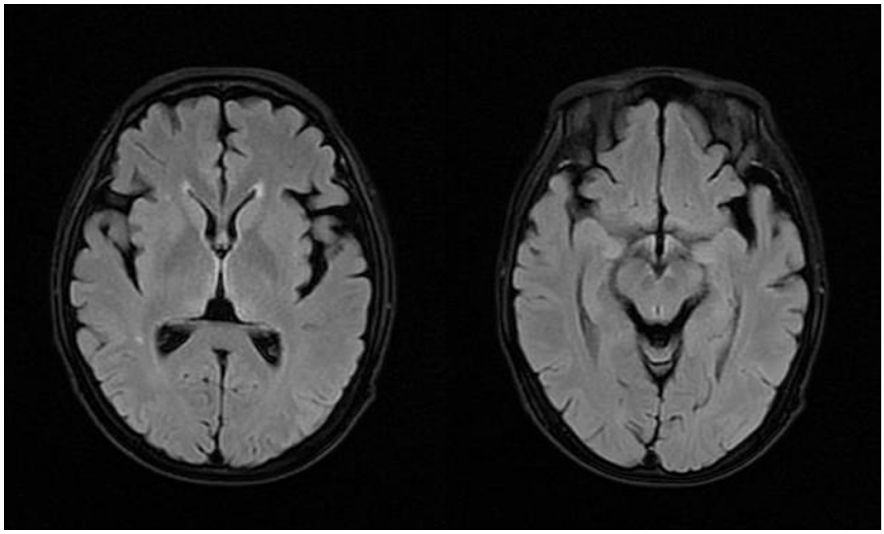

Figure 2. Reexamination of cranial magnetic resonance scan after therapy: The areas of T2 hyperintensities are decreased.

3. Discussion

Wernicke encephalopathy (WE), a disease caused by vitamin B1 deficiency, is a usual disorder in clinical. Vitamin B1 deficiency is categorized into alcohol-related and non-alcohol-related causes. Chronic excessive alcohol consumption is a well-established cause of vitamin B1 deficiency, and majority of Wernicke's encephalopathy cases in clinical practice are associated with long-term chronic alcoholism. However, beyond alcoholism, Wernicke's encephalopathy is not uncommon in various malnutrition-related conditions [3]. These include malabsorption, inadequate dietary intake, increased metabolic demands, or enhanced loss of water-soluble thiamine, such as in cases of anorexia nervosa, prolonged parenteral nutrition, long-term renal dialysis, hyperemesis gravidarum, gastrointestinal disorders, and post-gastrointestinal surgery [3].

Wernicke encephalopathy is an acute neurological disorder resulting from severe deficiency of thiamine (vitamin B1), which serves as an essential cofactor for several key enzymes involved in cerebral energy metabolism. Thiamine deficiency impairs the function of α-ketoglutarate dehydrogenase, transketolase, and pyruvate dehydrogenase complexes, leading to decreased ATP production, mitochondrial dysfunction, and failure of oxidative metabolism [4]. This results in widespread neuronal cell injury, particularly in regions with high metabolic demand such as the thalamus, mammillary bodies, and brainstem. Additionally, compromised blood-brain barrier integrity, glutamate-mediated excitotoxicity, and increased oxidative stress further contribute to the characteristic neuropathological changes [5] [6]. Without rapid thiamine replenishment, irreversible structural damage and neurological deficits may ensue.

Wernicke's encephalopathy (WE) typically presents with acute or subacute onset, where vomiting and nystagmus are the earliest manifestations. Ophthalmoplegia represents one of the characteristic features of the disorder, and ataxia

often develops following the occurrence of ocular symptoms [3]. The most patients, initial symptoms are severe and progress rapidly, within days, to an inability to stand or walk [6]. Milder cases may exhibit cerebellar ataxia, characterized by a broad-based gait and a tendency to fall. Some patients may also demonstrate slurred speech and incoherent articulation [7]. More than 80% of patients present with psychiatric symptoms, though these may sometimes be subtle and require careful clinical evaluation. The classic clinical triad of WE consists of ocular motor abnormalities, ataxia, and encephalopathy. However, only a minority of patients (approximately one-third) exhibit all three components of the triad. Most present with only one or two features. Additional symptoms may include peripheral sensory and motor disturbances, as well as thermoregulatory dysfunction such as hyperthermia or hypothermia. Common complications encompass peripheral neuropathy and cardiac arrhythmias [3].

For the treatment of WE, prompt thiamine supplementation is the foremost method. Parenteral administration, either intravenous or intramuscular, is most effective, while oral administration is not recommended. The initial dose of thiamine is 500 mg every 8 h for 2 - 3 days, followed by 250 mg for 3 - 5 days or until complete clinical improvement [2]. Although the therapy is not complex, delayed intervention can lead to irreversible neurological damage. Therefore, in clinical practice, routine thiamine (vitamin B1) supplementation should be provided to patients with high-risk factors for thiamine deficiency to prevent the occurrence of Wernicke's encephalopathy.

4. Conclusion

Based on this case, we think prevention is superior to treatment. In clinical practice, lacking of routine thiamine is usually recognized first by dietitian, not the physician. Moreover, the factors that lead to deficiency of vitamin B1 are often ignored except for alcohol abuse. Hence, for the patient who suffered gastrointestinal disorders and vomiting, unable to receive enteral nutrition, luxus consumption and unable to absorb, supplementation of vitamin B1 is a necessary treatment.

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Author Contributions

All the authors contributed to the article. Among them, the corresponding author Yao Yunfeng was responsible for proposing the writing ideas for this case, Cao Chun was responsible for organizing and summarizing the case materials as well as writing the article, and Li Xing was responsible for revising and improving the draft.

Conflicts of Interest

The authors declare that they have no known competing financial interests or per-

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