Wernicke's encephalopathy in a terminally ill patient with primary cervical cancer: A case report and literature review

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Abstract. Wernicke's encephalopathy (WE) is an acute or subacute neuropsychiatric condition associated with thiamine deficiency that is more often seen in cases of alcohol abuse. The current study presents a rare case of primary cervical cancer complicated by WE. A 44-year-old woman who underwent a laparoscopic radical hysterectomy with endoscopic pelvic lymphadenectomy for primary cervical adenocarcinoma in 2014 developed multiple metastases in the pelvic and abdominal cavities, right iliopsoas muscle and iliac wing 2 years post-surgery. The patient was hospitalized due to the rupture of a mass in the right lower abdomen in August 2019. A computed tomography scan demonstrated the spreading of primary cervical cancer to the right lower abdomen, which broke through the skin. In this terminal stage of cervical cancer accompanied with malnutrition, the patient suddenly presented with cognitive impairment, particularly in recent and immediate memory, as well as bilateral sustained nystagmus during hospitalization. Brain magnetic resonance imaging showed hyperintensity in the periaqueductal midbrain on T2 fluid-attenuated inversion recovery imaging. Based on these findings, a diagnosis of WE was made, and thiamine (100 mg) was immediately administered intramuscularly three times a day. After a week, the patient's eye movement disorder and recent memory improved gradually. The present case report with literature review aims to demonstrate the significant comorbidity between cancer and WE, raising awareness about the importance of recognizing the risk of thiamine deficiency in advanced cancer to prevent the development of WE.

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Abbreviations: MMSE, mini-mental state exam; MoCA, montreal cognitive assessment; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; TPN, total parenteral nutrition; WE, Wernicke's encephalopathy

Key words: primary cervical cancer, WE, thiamine deficiency, malnutrition, case report

Introduction

Wernicke's encephalopathy (WE) is an acute or subacute neuropsychiatric syndrome that is caused by thiamine (vitamin B1) deficiency. This vitamin plays indispensable roles in various physiological processes, including functioning as a coenzyme in the metabolism of carbohydrates, fats and proteins, making it a crucial enzyme regulating the proper functioning of the nervous system, muscles and cells (1). In humans, intestinal bacteria can synthesize only small amounts of thiamine, and thus, dietary intake is necessary (2). Generally, the occurrence of WE is associated with alcohol abuse. Evidence from numerous clinical case reports has indicated that WE is also common in patients with cancer, particularly those with advanced cancer. Reduced thiamine availability and storage capacity in patients with cancer may arise from starvation, malabsorption, malnutrition, chemotherapeutic agents use (e.g., 5-fluorouracil) and disease progression (3-5). Moreover, in patients with cancer undergoing gastrointestinal surgeries or experiencing complications such as vomiting, diarrhea or intestinal obstruction, thiamine absorption may be compromised, leading to thiamine deficiency (4). There are also rare factors contributing to thiamine deficiency, such as prolonged hospitalization and administration of total parenteral nutrition (TPN) without thiamine support supplementation.

The classic clinical triad of WE comprises alterations in consciousness, eye movement disorders and ataxia. In terms of diagnosis, WE is primarily based on the presence of one or two core manifestations. Magnetic resonance imaging (MRI) is currently considered the most reliable tool for diagnosing WE, providing high specificity and accurate positive predictive value, but low sensitivity (6). The prevalence of WE is in the range of 0.4 to 2.8%, as determined by several autopsy studies (7,8). However, in patients with cancer, WE may be underdiagnosed due to factors such as the under-recognition of early symptoms and the lack of specific diagnostic criteria for critically ill patients. The present study describes the case of a

woman with advanced primary cervical cancer who developed WE, highlighting the need for early recognition and monitoring of thiamine levels in patients with cancer, particularly those with malnutrition or undergoing prolonged hospitalization.

Case report

In June 2014, a 44-year-old woman underwent laparoscopic radical hysterectomy with endoscopic pelvic lymphadenectomy for the treatment of primary cervical adenocarcinoma [pathological T1b2N0M0 stage 1B, according to the 7th edition of the Union for International Cancer Control-American Joint Committee on Cancer staging system (9)] at the Cancer Hospital, Chinese Academy of Medical Sciences (Beijing, China). In June 2016, multiple metastases were detected in the pelvic and abdominal cavity, right iliopsoas muscle and iliac wing during a follow-up positron emission tomography-computed tomography (PET-CT) examination at the Cancer Hospital, Chinese Academy of Medical Sciences. Subsequently, multiple cycles of various chemotherapy regimens were administered as follows: Paclitaxel liposomes (240 mg) combined with nedaplatin (120 mg) was administered over 6 cycles via intravenous infusion; oral altretamine was administered for 4 months (specific dosage unavailable); liposome-encapsulated doxorubicin (60 mg) combined with lobaplatin (50 mg) was administered over 3 cycles via intravenous infusion; and albumin-bound paclitaxel (300 mg) combined with oxaliplatin (200 mg) was administered over 2 cycles by intravenous infusion. In June 2019, the patient developed a fever of 38.5˚C, and in July, TPN was provided at an external hospital due to intestinal obstruction. At the Department of Oncology, Guang'anmen Hospital, China Academy of Chinese Medical Sciences (Beijing, China), symptoms of a fever, abdominal pain and vomiting were observed, which were ascribed to the unresolved intestinal obstruction. The patient was treated with an enema and anti-infective drugs, including levornidazole and sodium chloride injection (0.5 g, every 12 h) combined with piperacillin sodium and tazobactam sodium injection (4.5 g, every 8 h) via intravenous infusion, which restored the temperature to normal level and alleviated the abdominal pain. Subsequently, the patient was discharged with a tolerable small liquid diet.

By August, the patient had lost ~4.6 kg, which represented a loss of 5-7.5% of total body weight occurring in <2 months, and they now returned to the hospital due to the spontaneous rupture of a mass in the right lower abdomen. Based on the Malnutrition Universal Screening Tool assessment (Fig. 1), the patient was considered to be at high risk of malnutrition (score 3) due to substantial weight loss and the current acute disease status, despite having a body mass index of 23.03 (10). A computed tomography scan confirmed that the primary cervical cancer had spread to the right lower abdomen and broken through the skin (Fig. 2). The wound, with significant exudate, was cleaned twice daily, and the patient continued to receive TPN due to poor oral intake. At 10 days after admission, the patient exhibited sudden cognitive impairment, especially in recent and immediate memory. An examination performed by a neurologist showed that she was conscious but disoriented with regard to time, place and person. The Mini-Mental State Exam (MMSE) and Montreal Cognitive Assessment (MoCA) scores were 19/30 and 17/30, respectively (11,12). In addition, ophthalmoplegia with bilateral sustained nystagmus was observed. Eye movement was normal to the left side, but abnormal to the right side. The patient could not walk due to cancer metastases within the right iliopsoas muscle and iliac wing. Consequently, a gait assessment was not performed. Mild upper-limb ataxia was observed and tendon reflexes exhibited a slight response. All other neurological examinations, including assessments of muscle tone and strength, were normal. Brain MRI and magnetic resonance angiography (MRA) were performed immediately. The MRA demonstrated normal blood vessels without restricted diffusion, which excluded the possibility of ischemic or hemorrhagic stroke. No significant abnormalities were detected on T1-weighted imaging and diffusion-weighted imaging, which further ruled out the possibility of brain metastases. However, brain MRI demonstrated hyperintensity in the periaqueductal midbrain on T2 fluid-attenuated inversion recovery imaging (Fig. 3). The patient's laboratory results were as follows: White blood cell count, $6.34x10^9$/l (normal range, $3.5\text{-}9.5x10^9$/l); red blood cell count, $3.69x10^{12}$/l (normal range, $4.3\text{-}5.8x10^{12}$/l); hemoglobin, 86 g/l (normal range, 120-160 g/l); platelets, $277x10^9$/l (normal range, $125\text{-}350x10^9$/l); alanine aminotransferase, 14.4 U/l (normal range, 7-40 U/l); aspartate aminotransferase, 15.9 U/l (normal range, 13-35 U/l); serum albumin, 32.62 g/dl (normal range, 40-55 g/l); potassium, 4.88 mmol/l (normal range, 3.5-5.3 mmol/l); magnesium, 0.90 mmol/l (normal range, 0.77-1.03 mmol/l); folate, 5.3 ng/ml (normal range, 3.38-5.38 ng/ml); and vitamin b12, 605 pg/ml (normal range, 211-911 pg/ml). Serum thiamine levels were not tested due to laboratory constraints. Several other conditions, including brain metastases, acute cerebrovascular disease and electrolyte imbalance, were considered and excluded based on clinical symptoms, imaging examination and laboratory results. Furthermore, the TPN regimen did not include routine thiamine supplementation, which may have promoted the development of WE. Thiamine (100 mg) was immediately administered intramuscularly three times a day. After 1 week of treatment, the patient's eye movement disorder and recent memory improved, with the MMSE and MoCA scores changing to 23/30 and 20/30, respectively. The patient was subsequently discharged and returned to her hometown. After 3 months, a telephone follow-up was conducted. It was noted that the patient showed rapid progression of the primary tumor, along with ongoing partial recent memory impairment and spatial disorientation. Ultimately, the patient passed away due to complications related to the tumor. The timeline of the disease course is presented in Fig. 4.

Discussion

Thiamine is a water-soluble vitamin absorbed in the small intestine, with a daily requirement of 1-2 mg in healthy adults (13). The body's thiamine reserves can be nearly exhausted within ~18 days. The deficiency primarily arises from insufficient dietary intake, reduced gastrointestinal absorption or impaired utilization. Natural sources of thiamine include whole grains, legumes, leafy greens, nuts and seeds, fish and pork, which are essential for maintaining adequate thiamine levels in the body. Thiamine has essential roles in

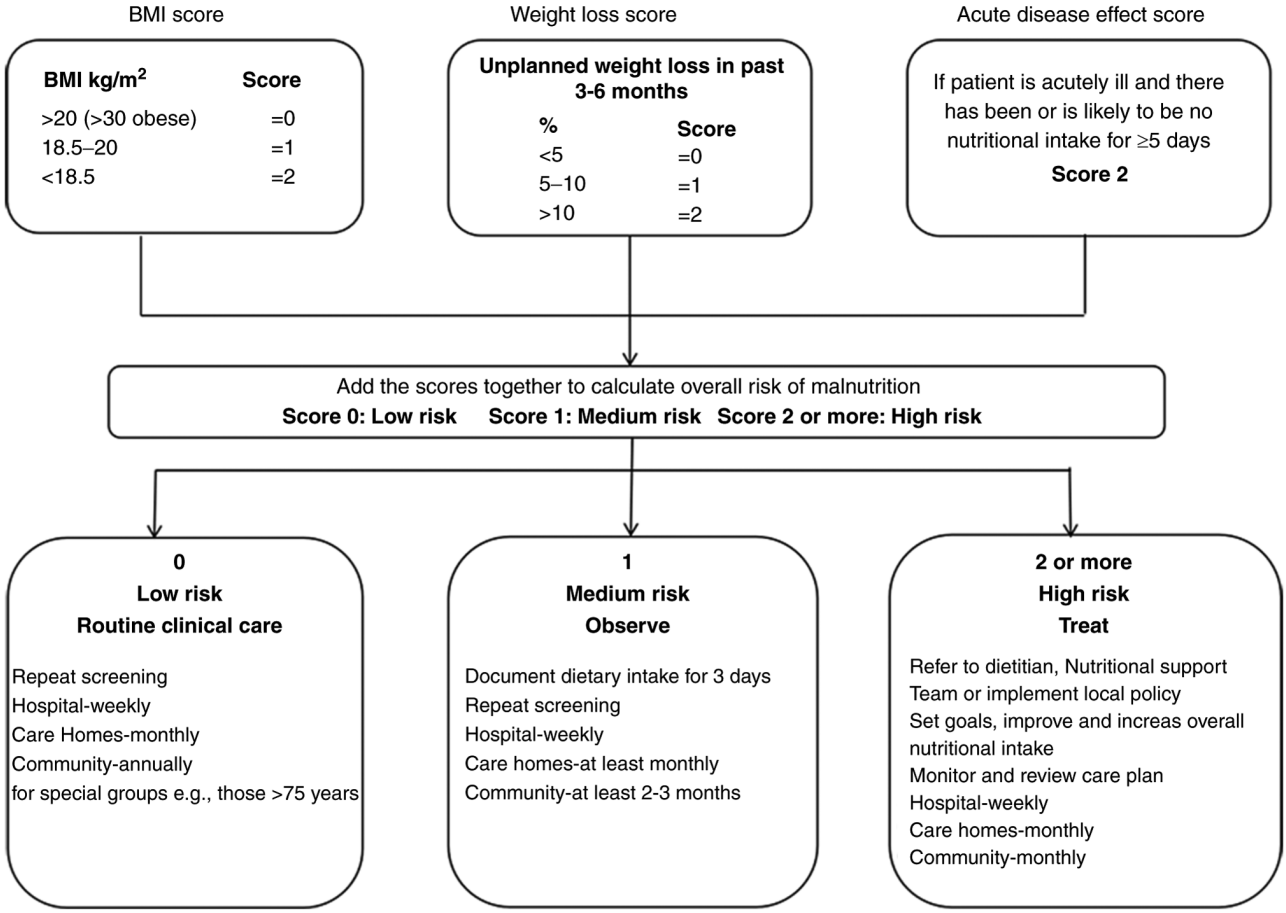

Figure 1. Malnutrition Universal Screening Tool assessment. BMI, body mass index.

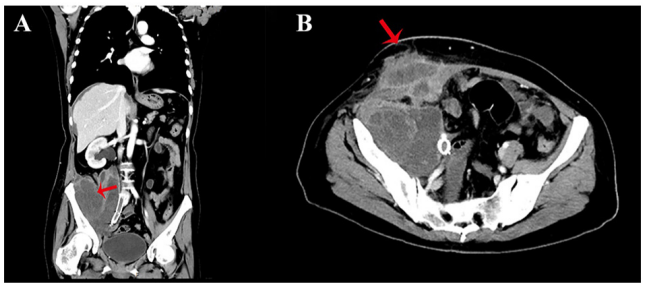

Figure 2. Representative CT scan of the abdomen and pelvis. (A) Coronal CT scan for multiple metastases within the pelvic cavity and abdominal cavity (arrow). (B) Horizontal CT scan for rupturing of metastases (arrow). CT, computed tomography.

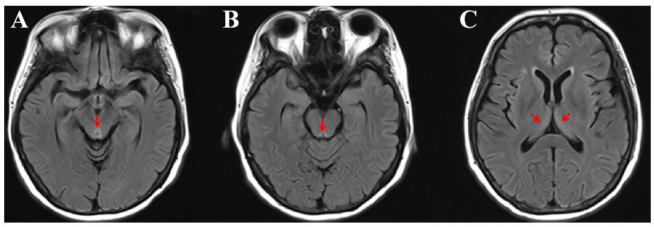

Figure 3. Brain magnetic resonance imaging. (A) T2 FLAIR imaging showing hyperintensity in the periaqueductal midbrain (arrow). (B) T2 FLAIR imaging showing hyperintensity in the periaqueductal gray matter (arrow). (C) T2 FLAIR imaging showing hyperintensity in the thalami (arrow). FLAIR, fluid-attenuated inversion recovery.

the regulation of cerebral metabolism, synapse formation and neurotransmitter synthesis (6,14,15). In neuronal and glial cells, thiamine pyrophosphate, the active form of thiamine, acts as a cofactor that modulates the complete oxidation of nutrients via the Krebs cycle to influence energy production. Thiamine is also involved in the synthesis of various neurotransmitters, such as glutamate and γ-aminobutyric acid, and its deficiency leads to cellular damage, impairing cerebellar activity (14,15). Thiamine deficiency induces neuropathological damage, including neuronal loss, micro-hemorrhages, endothelial swelling and gliosis in selective brain regions, primarily affecting the mammillary bodies, thalamus, cerebellum, cerebral aqueduct, and the third and fourth ventricles (16). Several mechanisms, such as cellular energy failure, lactic acidosis, oxidative and nitrosative stress, and N-methyl-D-aspartate receptor-mediated excitotoxicity, have been proposed to explain the association of thiamine deficiency with neuronal cell damage and loss (17).

In patients with advanced cancer, the risk of developing of WE is increased due to multiple factors, including poor nutritional intake, vomiting, severe malnutrition and the use of chemotherapy agents. A literature review of all WE cases in patients with cancer from studies that were published in the last 3 years and retrieved from PubMed (https://pubmed.ncbi.nlm.nih.gov/) is presented in Table I (18-28). Key terms used in the search included ‘Wernicke's Encephalopathy’, ‘cancer’, ‘chemotherapy’, ‘neurological complications’ and ‘case report’.

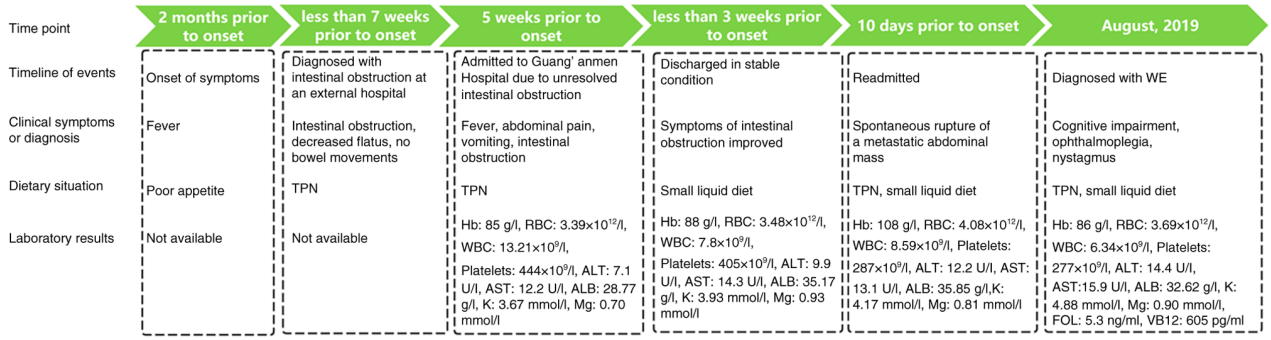

Figure 4. Case summary timeline. TPN, total parenteral nutrition; Hb, hemoglobin; RBC, red blood cell; WBC, white blood cell; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALB, albumin; K, potassium; Mg, magnesium; WE, Wernicke's encephalopathy.

Inclusion criteria were studies that reported on patients with cancer diagnosed with WE and were available through PubMed. Exclusion criteria included studies focusing on non-cancer-related causes of WE or those with insufficient data on patient treatment or outcomes. Table I summarizes a series of cases of WE in patients with cancer, emphasizing several key findings. Most patients had advanced cancer, particularly gastrointestinal, lung and hematological malignancies, and were undergoing chemotherapy or had significant nutritional deficiencies. Common symptoms of WE included confusion, ataxia and ophthalmoplegia, although atypical symptoms, such as isolated cognitive changes or motor disturbances, were also observed. Diagnosis was primarily clinical, with MRI providing typical imaging findings in some cases. Treatment involved thiamine supplementation, though the timing and dosage of administration varied. Patient outcomes ranged from full recovery to long-term neurological deficits, with faster diagnosis and prompt thiamine treatment generally associated with better outcomes (18-28). Clinically, WE is often underdiagnosed or diagnosed late due to the unclear clinical guidelines and criteria, especially in patients with advanced cancer. This challenge is particularly complicated by the significant overlap between advanced cancer symptoms and those of WE. The classic triad of symptoms has been reported in 16.5% of patients (29). This suggests that regular monitoring of thiamine levels in patients with cancer, particularly those with persistent nausea, vomiting or weight loss, is imperative to improve early detection and prevention of WE. In a number of cases, thiamine supplementation is administered after the development of neurological damage. However, early intervention may reduce the degree of neurological damage. This highlights the significance of incorporating regular nutritional assessments, including thiamine levels, in the routine care protocols, especially for patients at high risk for severe malnutrition.

In the current case, the patient presented with a nearly 2-month illness, including fever, vomiting and infection. Although TPN was administered, with regimens comprising the supplementation of 12 vitamins, thiamine was not included. The patient experienced a sudden onset of anterograde amnesia, characterized by an inability to recall the reason for the hospitalization, repeatedly asking the same questions and failing to recognize the attending physician. The anterograde amnesia was associated with the occurrence of lesions in the mammillary bodies and thalamus. The mammillary bodies, which regulate memory, are directly connected to the hippocampus, thalamus and midbrain. The mammillary bodies receive memory information from the hippocampus, and thalamic connections transmit the information from the mammillary bodies to the thalamus (30). In the present case, analysis of the MoCA and MMSE scores demonstrated cognitive deficits in the spatial and temporal orientations, immediate memory and calculation ability. These signs, and symptoms such as eye movement abnormality and confusion, along with the MRI scan findings, were consistent with WE.

Although several double-blind randomized clinical trials have explored the effective treatment strategies, there is no consensus regarding the treatment of WE. Pharmacokinetic studies have suggested that administration of thiamine twice or three times daily may induce a satisfactory clinical response compared with a single daily dose, due to the short plasma half-life of free thiamine (31). So far, few guidelines for WE treatment have been proposed, and most of them have not been updated. The Royal College of Physicians of the United Kingdom recommends intravenous administration of two pairs of high-potency B-complex vitamins (each pair containing 250 mg of thiamine) twice daily for 3 days in patients with confirmed or suspected WE (32). However, according to the European Federation of Neurological Societies, patients with suspected or confirmed WE should receive intravenous administration of 200 mg thiamine three times daily, until the clinical signs and symptoms stabilize (7). A recent systematic review reported that the most frequently used regimen in case studies is a dose of 500 mg thiamine administered intravenously three times daily (33). It has been reported that early and adequate thiamine supplementation can alleviate symptoms in 90% of patients, with mental status showing the most rapid improvement (33). However, another randomized controlled trial showed that different dosing regimens (100, 300 and 500 mg) did not significantly improve cognitive or neurological outcomes in both asymptomatic at-risk patients and symptomatic WE patients (34). For effective treatment of WE, several factors should be considered. In most cases, thiamine administered via the intravenous route has a high safety profile. However, anaphylactic responses may occur during or shortly after the intravenous injection, especially with multiple administrations (35). Therefore, intravenous injections should be administered over 30 min (36). Cases of WE induced by magnesium depletion have been reported, as magnesium functions as a co-factor promoting the phosphorylation of thiamine (37). Consequently, magnesium depletion may exacerbate

Table I. Review of cases of Wernicke's encephalopathy in patients with cancer from studies published in PubMed over the last 3 years.

First author, year	Malignancy	Age, years	Sex	Nutritional status	Medication history	Treatment	Prognosis	(Refs.)
Zhang *et al*, 2022	Esophageal cancer	64	Male	Underweight (BMI=19.88 kg/m^2)	Radiotherapy	Intravenous thiamine 500 mg/day for 3 days, then 200 mg/week, then oral thiamine 100 mg/day	Neurological symptoms improved, mild memory impairment	(18)
Nikjoo *et al*, 2022	Gastric cancer	38	Male	Malignancy	Gastric stent placement	Intravenous thiamine 250 mg/8 h for 2 days, then 250 mg/day	Neurological improvement	(19)
Lin *et al*, 2023	Gastric cancer	67	Female	Malignancy, weight loss	Post-gastrectomy, TPN	Intravenous thiamine 100 mg/day, 200 mg/8 h, 500 mg/8 h	Died from secondary respiratory failure	(20)
Gross *et al*, 2023	Duodenal adenocarcinoma	50	Female	Obese, Poor nutritional intake	Laparoscopic gastric band placement 11 years ago, TPN	Intravenous thiamine 500 mg/8 h for 2 days, then 250 mg/day for 5 days, then 100 mg orally daily	Dizziness, diplopia and nausea largely resolved	(21)
Koca *et al*, 2022	Cholangiocellular carcinoma	65	Male	Not mentioned	Gastrojejunostomy, radiotherapy, chemotherapy (gemcitabine), TPN	Intravenous thiamine 200 mg/8 h, then 100 mg/day	Orientation and cognitive function recovered, nystagmus persisted	(22)
Ibnawadh *et al*, 2023	Pancreatic cancer with colonic fistula	39	Male	Obese, weight loss (BMI=38 kg/m^2)	Surgical intervention for fistula and abscess, gastrojejunostomy, TPN	Intravenous thiamine 500 mg/day for 5 days, then 250 mg/day for 7 days, then 100 mg/day	Transitioned to palliative care and died	(23)
Slim *et al*, 2022	Sigmoid colon tumor	66	Male	Underweight (BMI=17 kg/m^2)	Post-surgical, TPN	Intravenous thiamine 1,000 mg/day for 3 days	Died on the 24th day after surgery	(24)
Brown and Hutt-Williams, 2022	Metastatic breast cancer	54	Female	Malnutrition, prolonged vomiting	Chemotherapy (docetaxel, trastuzumab, pertuzumab) targeted therapy, (trastuzumab, emtansine), radiotherapy	Intravenous Pabrinex for 48 h, then oral thiamine	Speech returned, memory and mobility improved	(25)
Azapagasi *et al*, 2024	Acute myeloid leukemia	7	Female	Not mentioned	Chemotherapy, TPN	500 mg thiamine via nasogastric enteral tube	Symptom remission	(26)

Table I. Continued.

First author, year	Malignancy	Age, years	Sex	Nutritional status	Medication history	Treatment	Prognosis	(Refs.)
Feng *et al*, 2022	Diffuse large B-cell lymphoma	67	Male	Malnourished	History of chronic alcohol abuse, 1 year of sobriety	Intramuscular thiamine 200 mg/12 h	Metamorphopsia, ataxia and cognitive improvement, with persistent ataxia after discharge	(27)
Malik *et al*, 2024	Malignant melanoma	79	Male	Poor nutritional intake, weight loss	Immunotherapy (nivolumab)	Intravenous thiamine 200 mg/8 h for 3 days	Mental status improved	(28)

TPN, total parenteral nutrition; BMI, body mass index.

thiamine deficiency, implying that clinicians should correct magnesium deficiencies and pay attention to other nutritional deficiencies (38). In the present case, the patient's TPN comprised 12 vitamins, and the serum magnesium and other vitamin levels were within the normal range. Therefore, supplementation with additional magnesium or other vitamins was deemed unnecessary. Data indicates that only 25% of patients with WE achieve a full recovery, while 50% show gradual improvement and the remaining 25% fail to show any improvement (39). It should be noted that the prognosis of WE in patients with advanced cancer is complex and influenced by several factors, such as the stage of malignancy, the degree of tumor progression, the timeliness of diagnosis and adequate thiamine supplementation (23-26). In the present study, it was observed that the patient's glassy eyes improved gradually after treatment for 1 week. At this point, the patient had partial recent memory recall, but the immediate memory was still impaired. The patient returned to her hometown after 1 week of thiamine treatment. After 3 months, a follow-up phone call revealed that the patient had experienced rapid tumor progression, accompanied with partial impairment of recent memory and disorientation in terms of place.

Thiamine deficiency is often not detected by oncologists, which results in delays or missed treatments. For high-risk cancer patients, regular monitoring of thiamine levels, early nutritional support and timely recognition of deficiency symptoms are essential to improve outcomes. To improve patient outcomes, future research should focus on creating comprehensive prevention and treatment guidelines for high-risk or WE patients. These guidelines should be easily accessible and readily implemented by healthcare providers to minimize the risk of underdiagnosis and undertreatment.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Guang'anmen Hospital, China Academy of Chinese Medical Sciences (Beijing, China).

Patient consent for publication

Written consent for publication was obtained from the patient's guardian.

Authors' contributions

HZ wrote the manuscript and participated in the management of the patient. YMZ designed the case report, and participated

in the diagnosis and management of the patient. SLW participated in the diagnosis of the patient and revised the manuscript for important intellectual content. All authors have read and approved the final manuscript. YMZ and SLW confirm the authenticity of all the raw data.

Competing interests

The authors declare that they have no competing interests.

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