**Wernicke Encephalopathy Presenting with Severe Pancytopenia: A Rare Clinical Association**

**Abstract**

Wernicke's encephalopathy (WE) is a severe neurological disorder caused by thiamine deficiency, commonly associated with chronic alcoholism. Early recognition and treatment are critical to prevent irreversible neurological damage. We present a case of a 38-year-old male with chronic alcoholism and fatty liver disease who presented with confusion, ataxia, and hematological abnormalities. Initial investigations revealed anemia, thrombocytopenia, and elevated inflammatory markers. MRI findings were consistent with Wernicke's encephalopathy. The patient received intravenous thiamine, antibiotics, and supportive care. Despite complications including severe pancytopenia and pneumonia, he showed clinical improvement with multidisciplinary management. This case underscores the importance of prompt diagnosis, aggressive thiamine supplementation and vigilant monitoring in the management of WE, particularly in atypical presentations.

**Keywords:** Wernicke's encephalopathy (WE), neurological disorder, thiamine deficiency, thrombocytopenia, hematological abnormalities

**Introduction :**

Wernicke encephalopathy (WE) is an acute neurological disorder caused by thiamine deficiency [1]. Clinically, alcoholism is the most common cause of WE. The typical symptoms of WE include mental status change, ocular abnormalities, and motor problems, such as gait incoordination and ataxia. WE can be fatal if it is not managed timely, so early diagnosis and treatment are crucial [2].

**Case presentation:**

The patient is a 38-year-old male with a history of chronic alcoholism and fatty liver disease for four years, who presented to the ER in a confused and disoriented state. He complained of loss of appetite, nausea, vomiting, severe dizziness, and headache for four days, along with generalized weakness, slowed speech, forgetfulness, and a burning sensation in both palms and soles for two months, which had worsened. He was found at home in a confused and distressed state by his friends, with alcohol bottles around him, after failing to respond to their calls. On examination, he was pale, confused (GCS 14/15), had an ataxic gait, diminished reflexes, power 3/5 in all limbs, petechial lesions on the lower limbs, and ecchymosis on the right elbow. Initial vitals showed BP 95/66, HR 121, and GRBS 197. The patient was admitted to the ICU for monitoring. Initial investigations showed anemia (Hb 8.9), thrombocytopenia (Plt 57,000), leukopenia (WBC 1.1), elevated CRP (201.7), and abnormal liver function tests (TB 2.90, AST 69, ALP 156). MRI brain showed a Subtle hyperintense FLAIR signal involving the periventricular region of the third ventricle and bilateral mammillary bodies, suggestive of Wernicke's encephalopathy. X-RAY CHEST - PA VIEW showed prominent bronchovascular markings in bilateral lung fields. Bilateral costophrenic angles are clear. The trachea and mediastinum are central. The Cardiac shadow is unremarkable, while a CT chest showed right upper lobe pneumonia. USG abdomen revealed moderate hepatomegaly with diffuse fatty liver, Grade 2. No free fluid or peritoneal collection. He was started on IV Ceftriaxone, vitamin B12, thiamine injections, and electrolyte correction.

**Day 2:** The patient remained afebrile but continued to be disoriented. His CRP decreased to 182, ammonia was 33, and renal function was stable, but his hematological parameters deteriorated further (Hb 7.3, Plt 34,000, WBC 0.9). PA peripheral blood smear showed normocytic normochromic red cells with few macrocytes, oocytes, and occasional teardrop cells, and marked absolute neutropenia.

**Day 3:** The patient’s CRP continued to decrease (147), but his AST increased (92). Hemoglobin further dropped to 6.8, platelets to 29,000, and WBC increased to 1.3. Initial blood cultures showed no growth. IV Meropenem was started due to worsening infection markers.

**Day 4:** Hematological parameters worsened, necessitating transfusion of 2 units of whole blood and 6 units of platelets. Electrolytes showed hyponatremia (Na 136.4) and hypokalemia (K 2.4), so IV potassium correction was given. CRP dropped further to 111.6, and the patient remained vitally stable but continued to be disoriented.

**Day 5:** The patient showed significant improvement, with Hb rising to 9.6, platelets to 36,000, and WBC to 3.3. His potassium remained low (2.8), so additional IV potassium was administered. Repeat electrolytes showed Na 132.7 and K 3.4. As the patient wished to continue treatment in his town, he was discharged with prescribed medications, including thiamine, vitamin B12, and alcohol withdrawal management with Chlordiazepoxide.

This case highlights the importance of early recognition and management of Wernicke’s encephalopathy, alcohol withdrawal, and nutritional deficiencies. It also emphasizes the need to continuously monitor hematological parameters and electrolyte imbalances in critically ill patients. Long-term follow-up is crucial to prevent recurrence and manage the complications of chronic alcoholism.



**Fig 1: MRI of the brain**: Red circle- hypertensive lesion in the third ventricle, and blue circle- involved bilateral mammillary bodies.

**Examination before discharge:**

General Appearance: Alert; Skin: Bruise, Pallor ++ Petechial lesions found on bilateral lower limbs, ecchymosis seen on right elbow, Intact; Head & Scalp: Normocephalic; Face: Symmetrical; Eyes: Normal red reflex present; Ears: Not deformed; Nose: Not deformed; Throat: Not inflamed; Mouth: Moist mucosa; Neck: Normal range of Movement; Respiratory: Normal Breathing; Cardiovascular System: Regular Pulse; Gastrointestinal Tract & Abdomen: No Distension; Rectal: Normal; Genitourinary: Normal Genitalia for the age & Sex; Hematology & Lymphatic: No pallor; Musculoskeletal: hands tremor ataxic gait, no nystagmus, power 3/5 in all limbs. Diminished reflexes, plantar down going. ; Neurology : Alert; Psychology : Comfortable; Development : Normal development of age

**Discussion:**

Wernicke's encephalopathy (WE) is a neuropsychiatric condition caused by a thiamine deficit. It was originally described by Carl Wernicke in 1881. WE is an acute condition that can cause irreversible brain damage if treatment is not received for an extended period. Working memory is impacted by Korsakoff syndrome (KS). Patients with lesions in the diencephalon-hippocampal circuit are unable to integrate short-term memories into long-term memories. Information must be gathered, and previously stored information must be integrated to adjust to new situations. Because of this, KS patients are unable to accomplish anything but their usual routines. Even though the primary symptom of KS, according to the previous classifications, is irreversible memory loss, there are also behavioral and cognitive abnormalities that occur at the same time [3]. Males are more likely to experience WE from alcohol abuse (1.69 males for every female patient), but females are more likely to experience WE from non-alcohol-related causes (1.84 females for every male patient). The cause of the condition also affects the age at which WE first appears. In individuals with alcohol dependence, the disorder often manifests at the age of over 40, although non-alcohol-related reasons are more prevalent in younger age groups [4,5].

The enzymes transketolase, pyruvate dehydrogenase, and alpha-ketoglutarate dehydrogenase all require thiamine, a crucial cofactor for the metabolism of carbohydrates. All of the body's cells use thiamine, but the brain and heart are particularly susceptible to deficiencies. Additionally, thiamine is required for the preservation of the myelin sheath. Thiamine deficiency is caused by alcohol primarily through reduced dietary intake in alcoholics, alcohol's interference with thiamine uptake from the gastrointestinal tract by blocking transport carrier molecules, and concurrent magnesium deficiency in chronic alcoholics, which impairs the function of thiamine-utilizing enzymes [6]. A study conducted by He, Xiaohua, et al. revealed significantly white matter lesions in the alcohol/pyrithiaimine group than in the water/pyrithiaimine group, which is consistent with the theory that long-term intentional alcohol use aggravates thiamine deficiency and has negative effects on white matter in the brain. This combined impact suggests that alcohol-related brain damage in human alcoholism may be caused by a combination of alcohol and thiamine deficiency [7]. The superior cerebellar vermis, medial thalamus, mamillary bodies, and periaqueductal grey matter are the main brain regions affected. This leads to the classic symptoms of WE [8].

It is also imperative to address the severe pancytopenia in the lab work-up of our case. In a case study that involved a case of pancytopenia in an adult patient with thiamine-responsive megaloblastic anaemia (TRMA), the authors initially hypothesised the causes of pancytopenia to be the sudden discontinuation of thiamine supplementation, an acute viral infection, a side effect of antipsychotic medications and a myelodysplastic syndrome. Due to multiple ruling-out of causes, the ultimate etiology appeared to be the sudden discontinuation of thiamine supplementation [9]. Thiamine is primarily stored in the liver. Conditions like end-stage chronic liver failure are closely connected with low thiamine levels, which can lead to WE. This condition may be exacerbated by regular alcohol consumption, as long-term alcohol treatment to experimental animals has been shown to deplete thiamine reserves in both the brain and liver [5]. In a case setting of hepatic encephalopathy (HE) with cirrhosis, hyperammonemia state is caused by hepatic failure, which is exacerbated by reduced ammonia metabolism due to muscular atrophy. In the presence of hyperammonemia, brain astrocytes oversynthesize glutamine, causing cell swelling and the accumulation of reactive oxygen species, ultimately contributing to the neuropsychiatric manifestation of HE. However, not everyone responds to ammonia-reducing medicines alone. This incomplete remission of neuropsychiatric symptoms after lactulose and rifaximin occurred because chronic liver failure causes both hyperammonemia and thiamine deficiency. Depletion of liver thiamine reserves reduces the action of alpha-ketoglutarate dehydrogenase, increasing glutamate concentration in peripheral tissues, which eventually enters the central nervous system and causes the HE symptoms [10].

Several studies have demonstrated that only one-third of patients presented with the typical triad, and in the majority of cases, just one or two of the triad were present. Ataxia (23%), ocular symptoms (29%), and polyneuropathy (11%) were less frequent presenting symptoms than altered mental status (82%), which was the most prevalent [11]. When two or more of Caine's Criteria are met, a patient is diagnosed with WE. The suggested operational criteria demonstrate that Wernicke's encephalopathy can be identified antemortem with a high level of specificity [12].

Diagnosis is mostly clinical. Whole blood thiamine levels are normally used to assess deficiency of thiamine, but a study by Davies et al. concluded that even normal blood thiamine levels do not safely rule out WE [13]. Imaging can be used to confirm the situation. It is possible to use both CT and MRI imaging. MRI might be regarded as the preferred imaging method due to its significantly higher sensitivity in aiding in diagnosis confirmation. CT is not an adequate tool for diagnosis. In the acute stage of WE, MRI displays a high-intensity signal on T2-weighted images in the periaqueductal and medial thalamic areas. Third ventricle dilatation and mammillary body and cerebellar atrophy are more noticeable in the latter stages [14].

A key contributor to readily preventable irreversible neurological morbidity and mortality is undiagnosed/untreated WE [15]. There are no established thiamine dosage recommendations for the treatment of this condition. There is only agreement that high dosages of thiamine should be administered early. Due to its accessible availability, favorable safety profile, and affordability, clinicians should have a low barrier to initiating a thiamine treatment. Neuronal cell death or the development of irreversible Korsakoff syndrome requires immediate medical attention [6]. It is worthwhile to mention that there is no set dosage of thiamine currently [16]. A case series depicted that thiamine usage resulted in a quick improvement in mental status over the course of 3-5 days in all three cases. 500 mg of intravenous thiamine HCl was given t.i.d. in each of the three instances [17]. Numerous case reports claim that non-alcoholic diseases have been healed by intravenous thiamine treatment at doses of 100 or 200 mg. However, among alcoholics, this hasn't always been the case. Higher daily dosages may be required for alcoholic patients with WE; 500 mg three times a day has been suggested [8,18]. Patients with suspected WE were given ≥ 500 mg of thiamine over a duration of three days, and around 73% showed improvement in symptoms after treatment. IV thiamine was also reported to have been very effective and safe for the treatment of acute WE [19]. It is also crucial to begin thiamine supplementation before carbohydrates [6]. The primary preventive strategy involves the fortification of daily food items with thiamine. Additional preventive strategies include oral prophylactic medication for alcohol dependence and the use of oral thiamine to treat patients experiencing alcohol withdrawal while they are in the hospital.

**Conclusion:**   
In conclusion, this case highlights a rare presentation of Wernicke’s encephalopathy with concurrent pneumonic consolidation and pancytopenia, emphasizing the importance of early recognition and intervention. The patient showed improvement with appropriate management, including neurological, hematological, and respiratory support. Publishing this case is crucial to increasing awareness among healthcare professionals about the atypical presentations of Wernicke’s encephalopathy, particularly when associated with pancytopenia. By sharing this case, we aim to enhance the clinical vigilance of the healthcare team, ensuring early diagnosis and timely management to improve patient outcomes.

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