The Intercontinental Journal of Emergency Medicine

Case Report

DOI: 10.51271/ICJEM-0061

Wernicke-Korsakoff syndrome in the emergency department: a case report

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Cite this article: Akben S, Gedik MS, Mangır L. Wernicke-Korsakoff syndrome in the emergency department: a case report. *Intercont J Emerg Med.* 2025;3(3):62-64.

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Received: 16/06/2025 ◆ **Accepted**: 07/09/2025 ◆ **Published**: 23/09/2025

ABSTRACT

Wernicke-Korsakoff syndrome (WKS) is a severe, potentially irreversible neuropsychiatric illness frequently linked with alcohol use disorder and thiamine (vitamin B1) deficiency. The acute phase, Wernicke encephalopathy (WE), presents with confusion, ataxia, and oculomotor dysfunction. The triad of symptoms may not always be present, however, and diagnosis is therefore troublesome. In this case, a chronically alcoholic and malnourished patient came with a change in mental status, which led to the diagnosis of WE and was initiated on high-dose thiamine treatment. Prompt treatment led to quick clinical recovery. Diagnosis of WE in the emergency department and prompt treatment as a component of multidisciplinary care is paramount to avoid long-term neurological impairment.

Keywords: Confusion, disorientation, emergency department, thiamine, Wernicke encephalopathy

INTRODUCTION

Wernicke–Korsakoff syndrome (WKS) is a severe neuropsychiatric disorder resulting from a lack of thiamine (vitamin B1) and frequently occurring alongside alcohol use disorder. The first phase of the syndrome, Wernicke encephalopathy (WE), is presumably reversible and presents with the classic triad of confusion, ataxia, and oculomotor dysfunction. It must, however, be remembered that the triad of symptoms may not occur concurrently in all individuals, thereby rendering the diagnosis difficult.[1-3]

Although WE is most commonly associated with alcohol-related brain damage, it may also result from many other causes of thiamine deficiency, including those due to malnutrition, gastrointestinal malabsorption, post-bariatric surgery syndrome, and prolonged fasting.[1,3,4] Diagnosis is usually based on a clinical evaluation, which requires a high degree of clinical suspicion.[1,2,5]

The initiation of parenteral thiamine therapy at high doses prior to confirmation of the diagnosis can potentially result in the rapid resolution of neurological symptoms. Nevertheless, with delayed treatment and diagnosis, there is a much greater risk of developing long-term cognitive dysfunction and increased mortality.[6] It is thus extremely critical to always include Wernicke's encephalopathy in the differential diagnosis of patients presenting in emergency departments, particularly those with preceding histories of alcohol use, malnutrition, or susceptibility to malabsorption. Treatment should be initiated immediately if two or more of the following features are present: nutritional deficiencies, ocular findings, cerebellar abnormalities, and changes in mental status.[7]

CASE

A 40-year-old male patient was brought to the emergency department by family members, who reported that he had experienced abnormal eye movements, severe numbness, lethargy, behavioural changes and disorientation over the previous few days. He was a chronic alcoholic who had attempted to abstain from alcohol for the previous week. During this period, he had also reduced his oral intake and stopped taking antianxiety medications. He had also stopped taking vitamin B supplements, and his diet had deteriorated. His family reported that he had not consumed food for the past two days, could not recognise his environment, and was unable to communicate his thoughts coherently.

On physical examination, the patient was somnolent and was only able to communicate minimally with those around him. His spontaneous speech was mostly incomprehensible, and he could not answer questions appropriately. He was unable to identify his relatives, describe his environment, or give the date, suggesting impairment of orientation to place,

time, and person. There were no motor deficits or noticeable articulatory impairment. He had a stable gait and normal extremity movement. Pupils were isocoric, and light reflexes were normal. Vital signs were stable (temperature: 36.7°C; blood pressure: 124/78 mmHg; pulse: 88 bpm; SpO_2: 97%).

Laboratory examinations revealed normal electrolyte, glucose, liver, and kidney function levels. Complete blood count results showed no signs of infection. Cranial computed tomography and magnetic resonance imaging scans did not reveal any acute pathologies. Infection, metabolic disorders, cerebrovascular accidents, and head injury were excluded as differential diagnoses based on the clinical and laboratory findings.

The evaluation revealed the patient's malnutrition, long-term alcoholism, thiamine deficiency, and progressive cognitive disorientation. A provisional diagnosis of WE was established, and the patient was started on high-dose parenteral thiamine replacement (500 mg intravenous administration, three times daily). Electrolyte replenishment and fluid resuscitation were also initiated as adjunct therapy. The patient's neurological status, level of consciousness, and response to intravenous therapy were closely monitored by the nursing team.

Six to eight hours after thiamine treatment began, there was a remarkable improvement in the patient's level of consciousness. Their orientation to place and time showed partial recovery, and they were able to communicate more effectively with their environment and recognise their relatives. On the following day, further improvement in mental status was observed, and orientation was significantly enhanced. The patient was subsequently admitted to the neurology ward for ongoing assessment and long-term supportive therapy.

DISCUSSION

We are referring to WKS, an acute clinical syndrome that, if not diagnosed and treated early, can result in irreversible neurological damage. Although it is usually characterised by the classical triad of confusion, ataxia, and oculomotor dysfunction, the absence of these features in some patients may lead to delayed diagnosis.[1,3,8] Thus, it is of paramount importance to maintain a high index of clinical suspicion, particularly in the emergency setting, to establish an early diagnosis.

Effective communication and division of labour among a multidisciplinary team are key to patient management in individuals presenting to emergency departments with suspected WE. In the present case, key nursing interventions included monitoring levels of consciousness using the Glasgow Coma Scale, repeated orientation and vital sign checks, observation for possible adverse reactions during intravenous thiamine infusion, and maintenance of fluid and electrolyte balance as dictated by laboratory findings. These interventions were essential to ensure patient safety and achieve clinical stabilisation.[9]

In the described case, acute alterations in consciousness and disturbances in orientation in patients with reduced oral intake, cessation of vitamin supplementation, and alcohol withdrawal must be included in the broad differential diagnosis. Following the exclusion of hypoglycaemia, electrolyte imbalance, sepsis, hepatic encephalopathy, stroke, and other toxic-metabolic causes, WE should be considered in the differential diagnosis of individuals with a history of malnutrition and thiamine deficiency, even in the absence of the classic triad.[8,10,11]

There is no definitive laboratory test for WE; thus, the diagnosis is primarily based on clinical presentation and associated risk factors. Magnetic resonance imaging (MRI) may support the diagnosis, most commonly revealing symmetrical signal enhancement in the thalamus, mammillary bodies, and periventricular regions. These imaging abnormalities, however, are not always present and typically become apparent only in more severe cases.[3,12] Hence, in the presence of strong clinical suspicion, it is recommended that high-dose parenteral thiamine therapy be initiated immediately, without awaiting confirmatory diagnosis.[2]

CONCLUSION

This case illustrates that WE is a significant clinical condition that is rarely diagnosed in emergency department practice, yet can lead to permanent neurological impairment if diagnosis is delayed. Diagnosis requires a systematic clinical assessment and a high index of suspicion, as delays are associated with worsening cognitive function and an increased risk of mortality.

Early treatment with high-dose parenteral thiamine can result in considerable clinical improvement in carefully selected patients within a limited time frame. Accordingly, patients with a history of alcohol use or risk factors such as malnutrition or malabsorption who present with confusion, memory loss, and disorientation should be thoroughly assessed for WE. Enhancing clinical suspicion may prevent long-term neurological damage through effective and inexpensive treatment.

ETHICAL DECLARATIONS

Informed Consent
The patient signed and free and informed consent form.

Referee Evaluation Process
Externally peer-reviewed.

Conflict of Interest Statement
The authors declare no conflict of interest.

Financial Disclosure
The authors declared that this study has received no financial support.

Author Contributions
All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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