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Managing Wernicke's encephalopathy in hospitalized patients: a brief review of diagnostic considerations and thiamine treatment

Abstract

Wernicke's encephalopathy is heterogeneously defined, treatable, and reported in a number of disorders that cause thiamine deficiency. Despite well-documented approaches to diagnosis and treatment of this neurological condition, thiamine dosing guidelines remain inconsistent and understudied. Furthermore, identification of symptoms is often delayed or missed entirely. This review explores the historical evolution of Wernicke's encephalopathy, its pathophysiology, clinical presentation in hospitalized patients, and treatment considerations. We discuss a structured paradigm for inpatient evaluation and special considerations for high-risk populations, including pregnant patients, older adults and individuals with co-occurring substance use beyond alcohol consumption. Wernicke's encephalopathy remains a clinical diagnosis, though evolving understanding of its etiology, presentation, and epidemiology presents opportunity for earlier detection and treatment.

Keywords: Wernicke's encephalopathy, alcohol use disorder, thiamine deficiency

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Abbreviations: WE, Wernicke's encephalopathy; KS, Korsakoff's syndrome; TPP, thiamine pyrophosphate; GI, gastrointestinal; HE, hepatic encephalopathy; IV, intravenous; IM, intramuscular; CHS, cannabinoid hyperemesis syndrome; HG, hyperemesis gravidarum; AMS, altered mental status; ATP, adenosine triphosphate

Introduction

Wernicke's encephalopathy (WE) derives its name from Dr. Carl Wernicke, who identified and defined the condition as a form of hemorrhagic encephalitis.[1] The classic Wernicke's triad includes ocular dysfunction, gait ataxia, and altered mental status (AMS), however, all three are present in fewer than 40% of cases.[2,3] While WE is commonly associated with severe alcohol use, it is more broadly caused by thiamine (vitamin B1) deficiency, which can occur in diverse medical and psychiatric conditions, including sepsis, hyperemesis gravidarum, anorexia nervosa, or after bariatric surgery.[1]

Hospitalized patients often face nutritional challenges either prior to admission or during a prolonged hospital course, and close monitoring of their neurological condition, as well as a thorough medical history, are paramount in diagnosing WE. Early recognition and treatment with thiamine can reverse debilitating symptoms and prevent progression to chronic neurologic impairment. This review highlights the historical background, pathophysiology, clinical presentation, and treatment considerations for hospitalized patients and introduces a structured paradigm for inpatient evaluation.

A brief history of the Wernicke-Korsakoff clinical spectrum

Both WE and its progressed form, Korsakoff's psychosis or syndrome, were separately identified as severe neurological disorders in the late 19th century.[4] In 1881, Dr. Carl Wernicke, a neuropsychiatrist and anatomist, reported three fatal cases marked by ocular dysfunction, ataxia, and delirium, with postmortem punctate hemorrhages in a similar bilateral distribution along the third and fourth ventricles.[5,6] These cases had seemingly little else in common, though one patient suffered from sulfuric acid poisoning with persistent emesis over several weeks, and the other two were known to have a history of significant alcohol use. In retrospect, these patients likely developed a thiamine deficiency from nutrient depletion via vomiting, and malabsorption.

While Wernicke did not identify the underlying biochemical etiology of this presentation, his report marked the first widely disseminated and cohesive description of what later became known as the clinical triad of symptoms of this condition.[4] Six years later, in 1887, neuropsychiatrist Dr. Sergei Korsakoff described memory loss and psychosis that he correlated with alcohol-associated neuropathy. Korsakoff hypothesized that the underlying etiology of neuropathy in these patients also impaired brain functioning, resulting in psychiatric symptoms he described as psychosis polyneuritica.[4,7] Although later known as Korsakoff's psychosis, Korsakoff originally identified a range of psychiatric symptoms, including persistent depression, anxiety, and irritability, which prompted a clinical reframing of this condition as a syndrome. Korsakoff also identified medical conditions beyond alcohol use as potential causes of this presentation, including typhoid fever, postpartum infection, and intestinal obstruction.[4]

Korsakoff's syndrome (KS) is characterized by residual and persistent neurologic effects, including memory impairment, in patients who had WE.[7] WE and KS were conceptualized as a clinical continuum by Dr. Karl Bonheoffer who observed KS develop only in surviving cases of WE.[7,8] Thiamine itself was identified as a critical nutritional element in animals and structurally characterized in the 20th century.[9,10] Together, these discoveries laid the foundation for understanding WE as a thiamine deficiency disorder.

Pathophysiology and clinical presentation of thiamine deficiency

Humans and animals do not synthesize thiamine, a vitamin essential to carbohydrate metabolism and neurologic function. Thiamine is stored primarily in the liver as thiamine pyrophosphate

(TPP), a cofactor for multiple enzymes involved in energy production. Through its role in generating acetyl-CoA, a key substrate in the Krebs cycle, TPP supports adenosine triphosphate (ATP) synthesis. Thiamine deficiency disrupts this pathway, reduces ATP production, increases oxidative stress and leads to neuronal loss and microhemorrhage.[11,12]

Human dietary thiamine needs range between 1 and 2 mg daily, and total body stores can hold up to 50 mg. If not replenished adequately through diet, thiamine can be depleted within six weeks.[13] WE is initially reversible, and caused by the inability of thiamine-depleted cells to respond to metabolic needs. Inadequate treatment, such as delayed or subtherapeutic thiamine dosing, can lead to irreversible structural neurologic changes and persistent cognitive deficits.[14] Given this, it is essential to assess patients' nutritional intake in the weeks prior to hospitalization, regardless of alcohol use history, as even short periods of inadequate intake can lead to thiamine deficiency.

Preclinical studies have demonstrated that alcohol interferes with the absorption of thiamine in the gastrointestinal (GI) tract.[15] Similarly, conditions that shorten or impair the absorptive surface of the GI tract, such as bariatric and gastric bypass surgeries, Crohn's disease, and pyloric stenosis, can reduce thiamine absorption in patients and lead to deficiency.

Although the diagnosis of WE remains clinical, brain imaging can provide supporting evidence. In a retrospective observational study by Arendt and colleagues (2024), MRI identified microbleeds in the mamillary bodies of patients both with and without a history of severe alcohol use.[1] Notably, atrophied mamillary bodies have been identified in autopsy studies of patients with known WE, and these findings are considered specific to WE, though their identification tends to occur mainly on autopsy.[16,17] Other common loci for lesions include the periaqueductal grey and medial thalamic nuclei, though significant correlations between imaging and clinical findings were only identified in the mamillary bodies and short-term memory.[1]

AMS in WE may present as confusion, disorientation, and mild memory impairment, though atypical presentations, such as indifference, inattention, or even coma, are possible.[18–20] Oculomotor abnormalities include nystagmus - the most common ocular finding - as well as ptosis, conjugate gaze palsy, and dysfunctional pupillary reaction.[17] Gait ataxia and weakness may precede other signs of WE by days to weeks, though a fourth criterion, dietary deficiency, develops first and has been proposed by Caine and colleagues.[21]

Because WE and KS are commonly associated with alcohol use disorder, distinguishing between these and symptoms of hepatic encephalopathy (HE), can be a challenging undertaking. An overview of WS, KS and HE is described in Table 1. A summary of common clinical symptoms associated with WE is included in Table 2.

Table 1 Risk factors, clinical features, and lab abnormalities for Wernicke's encephalopathy, Korsakoff syndrome and hepatic encephalopathy

	Wernicke's encephalopathy	**Korsakoff syndrome**	**Hepatic encephalopathy**
Risk factors	Thiamine deficiency Alcoholism Hyperemesis gravidarum Bariatric surgery Hemodialysis	Untreated WE Thiamine deficiency Alcoholism	Cirrhosis and other liver diseases Portosystemic shunts
Clinical features	Mental status changes Ophthalmoplegia and other ocular abnormalities Gait ataxia Hypothermia Hypotension Coma	Severe anterograde amnesia Retrograde amnesia Executive function deficits Confabulations Anosognosia	Diurnal sleep pattern disturbances Personality changes Bradykinesia Asterixis Physical stigmata of liver dysfunction Coma
Lab abnormalities	↓ Blood thiamine[3] Lesions of the mamillary bodies	↓ Blood thiamine[7] Lesions of the mamillary bodies, medial thalami, and corpus callosum	↑ Blood ammonia[74] Liver tests (e.g., ↑ bilirubin, ↓ albumin levels)

Table 2 Clinical manifestations of thiamine deficiency

Cardiorespiratory
High output cardiac failure
Lower extremity edema
Pulmonary hypertension
Tachypnea
Neurological
Cognitive impairment
Hyperirritability
Ophthalmoplegia
Nystagmus
Amnesia
Confusion
Ataxia
Peripheral neuropathy
Weakness
Gastrointestinal
Loss of appetite
Abdominal pain
Lactic acidosis
Nausea
Vomiting

Thiamine dosing

Prompt thiamine replacement is the cornerstone of WE management, and a thorough patient history and serial assessments can expedite diagnosis. Thiamine stores must be replenished to treat WE, but the recommended dose and route is variable in the literature. There are also no significant side effects reported with thiamine

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administration, though anaphylaxis has been reported as a rare occurrence.[14,22]

One review of thiamine replacement in 138 patients across six different studies reported intravenous (IV) dosing ranging from 100-1500 mg daily and intramuscular (IM) dosing up to 300 mg daily.[23] Serum levels rise more rapidly with IV administration, but IM dosing has been shown to provide more stable serum concentrations over time.[24,25] Oral thiamine has lower bioavailability than IV or IM, although some clinical improvement has still been reported with oral supplementation alone.[12] Beyond differences in route and dosing, various formulations have also been described in the literature. In the early 2000's, the Royal College of Physicians recommended Pabrinex, a compound preparation containing 250 mg of IV thiamine along with riboflavin and pyridoxine, likely because this was the only commercially available IV thiamine product in the United Kingdom at the time.[14] Data on this topic remains limited: a 2013 Cochrane review identified only two randomized double-blind, placebo-controlled trials on thiamine dosing, of which only one study was published and had enough data for sufficient quantitative analysis.[26,27] Prucker and colleagues (2019) conducted a comprehensive literature review of recent thiamine guidelines and noted ongoing heterogeneity between professional medical association guidelines, though use of parenteral thiamine remained a consistent clinical recommendation across organizations.[28] Notably, recommendations vary by country, with US guidance from the American Society of Addiction Medicine (ASAM) and American Psychiatric Association (APA) recommending between 50 to 100 mg of parenteral thiamine in the setting of moderate to severe alcohol withdrawal.[28,29] Medical associations from other countries, such as the Italian Society on Alcohol and the British Association for Psychopharmacology, the Australian Government Department of Health, have recommended higher doses over 200 mg daily.[28,30,31]

In contemporary inpatient practice, most experts recommend initiating thiamine 200-500 mg IV three times a day for at least 3-5 days, followed by oral supplementation if risk factors persist.[30,32,33] As the side effect profile of thiamine is generally benign, the therapeutic benefit of higher dosing favors its use.

Thiamine supports enzymes involved in glucose metabolism, and thiamine deficiency impairs glycolysis and citric acid cycle processes, leading to the accumulation of toxic intermediaries, such as lactic acid.[34] Historically, clinical guidance has recommended administering thiamine prior to glucose in hypoglycemic patients, to prevent worsening of lactic acidosis and precipitation of WE.[35–37] However, subsequent studies have not demonstrated significant differences in neurological function resulting from glucose administered either pre- or post-thiamine. Importantly, prolonged delay in either glucose or thiamine administration should be avoided in patients who require repletion of both.[17,34,38,39]

Important considerations for the hospitalized patient

Hospitalized patients are at a heightened risk of delirium and nutritional deficiencies that may exacerbate their neurological function.[40,41] These risks are especially pronounced in older, pregnant, or post-surgical patients, who often face compounded nutritional challenges.[42,43] Acute illness, surgeries and interruptions in nutritional support in the inpatient setting can lead to more rapidly evolving nutritional deficiencies.[44,45] In addition, hospitalized patients often present atypically and are more likely to have comorbidities or sedation that can confound the diagnostic picture and contribute to under-recognition of WE, particularly in patients without a history of alcohol use.[3,45]

Thiamine deficiency should be considered in any patient presenting with malnutrition, failure to thrive or prolonged nausea and vomiting. Thiamine levels can be measured, though hospital availability varies, and results may not be available on an emergent basis. Furthermore, there are no established cut-offs for WE diagnosis and serum thiamine levels do not necessarily reflect stores in the brain, limiting the utility of thiamine level testing in the acute inpatient setting.[46,47] A history of GI disease or surgery, particularly bariatric surgery, hyperemesis gravidarum, parenteral nutrition or cancer with rapid tumor growth or active chemotherapy also increases the risk of nutritional deficiencies.[44,45] Beyond medical illness, patients with psychiatric conditions such as anorexia nervosa, and those with substance use are also at risk.

Thus, clinicians must be proactive in evaluating for WE and have a low threshold to initiate empiric parenteral thiamine. Empiric treatment should be started as soon as the diagnosis is suspected, as delays can result in irreversible deficits.[17]

We have developed a paradigm for evaluating WE in the hospital setting, as illustrated in Figure 1. This approach considers Caine's criterion of dietary deficiency and underscores the diagnostic value of both clinical assessment and history gathering.[21] We introduce the 4N paradigm for WE assessment: nutritional and neurological evaluation, as well as consideration of how the patient presents now, and how they present "normally," at their baseline. These four aspects should also be considered from two dimensions: clinically and through the lens of available collateral and history.

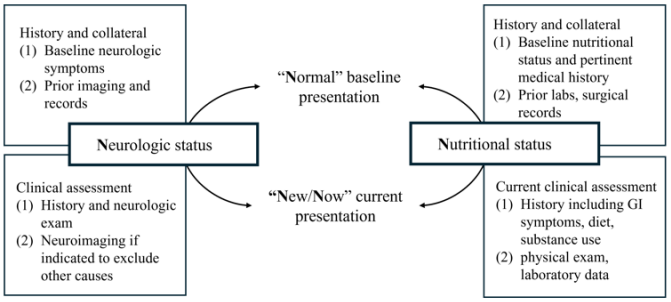

Figure 1 "4 N" Paradigm for early and iterative assessment for Wernicke's encephalopathy.

Iterative re-assessment daily, more often if critically ill or symptoms change rapidly

Nutritional evaluation: Assess dietary intake, weight, persistent vomiting or diarrhea. Laboratory studies such as hemoglobin, prealbumin, albumin, electrolytes, and micronutrient levels such as B12 and iron, can be helpful in evaluating a patient's underlying risk of nutritional deficiencies.[48]

Neurological evaluation: A thorough history and exam should be completed that includes assessment for the WE triad – confusion or memory impairment, gait disturbance and oculomotor dysfunction (e.g., nystagmus, ophthalmoplegia, gaze palsies). Clinicians should note that the full triad is uncommon, so the presence of one or two of these findings should raise suspicion. Patients should also be evaluated for atypical features such as blurred vision, urinary retention, hypotension, hypothermia, infection or rapid weight loss, as hospitalized patients are more common to present with these.

The patient's current nutritional and neurological evaluation should be compared with their baseline presentation by reviewing prior data, history and collateral, and this should be taken into context in the evaluation.

This paradigm should be applied iteratively throughout the hospital course, recognizing that symptoms may be subtle or evolving. Serial neurologic examinations should be performed at least daily, with more frequent assessments in critically ill patients or when symptoms change. Clinicians should also reassess frequently after thiamine initiation to monitor for improvement. To illustrate the practical implementation of the 4N framework, two representative cases are presented in Table 3.

Table 3 Case examples applying the "4N" paradigm

Case	Neurological findings		Nutritional findings		Next steps
1. A 58-year-old man with alcohol use disorder is admitted with acute pancreatitis. At baseline, he lives independently and is cognitively intact, although he reports a long history of a limited diet consisting mostly of bread and soup. In the week before admission, he developed severe nausea and vomiting, leading to markedly reduced oral intake. On exam, he is confused, disoriented and demonstrates new nystagmus.	Normal	New/now	Normal	New/now	
	Collateral and chart confirm intact cognition and neurologic function.	Confusion and new nystagmus represent a clear deviation from baseline.	Long-standing alcohol use and limited diet place him at baseline risk for Wernicke's encephalopathy.	Acute decline in intake with pancreatitis further elevates risk.	Together, these findings strongly support WE, and immediate parenteral thiamine is indicated, with daily reassessment.
2. A 28-year-old pregnant woman with hyperemesis gravidarum presents after several weeks of persistent vomiting and poor oral intake. Prior to pregnancy, she was well nourished and neurologically intact. At the time of evaluation, she exhibits mild memory deficits and unsteady gait.	No prior deficits.	New mild memory deficits and unsteady gait.	Previously well-nourished.	Prolonged vomiting and poor intake with electrolyte disturbances.	New nutritional and neurologic compromise compared with a healthy baseline prompts empiric parenteral thiamine.

Special populations

Wernicke's encephalopathy in patients with a history of other substance use

Although WE is commonly associated with alcohol use disorder, there is growing evidence that WE can also occur in the setting of other substance use, including in patients with cocaine use disorder.[49] Several case reports have also identified WE in patients with significant cannabis use.[50–52] Munroe and colleagues report the case of a patient who used cannabis daily for several months and, in the setting of suspected cannabinoid hyperemesis syndrome (CHS), developed AMS and ocular abnormalities.[51] CHS, which is characterized by chronic emesis, can lead to dehydration and electrolyte abnormalities, as well as unintentional weight loss due to poor nutritional intake. [53] Thus, CHS may contribute to thiamine deficiency and, if not promptly addressed, WE.

Wernicke's encephalopathy and co-morbid psychiatric disorders

WE has also been reported in patients with severe mental illness (SMI), and in cases of poor insight and malnutrition, progression to KS has been documented.[54] Patients with SMI are vulnerable to dietary deficiencies, and are more likely to have poorer nutritional status and weight fluctuation than their healthy counterparts.[55] The prevalence of poor nutritional status was recently illustrated by Risch and colleagues (2022) in a study that identified over a third of patients with SMI screened positive for malnutrition.[56] WE symptoms can also overlap with adverse reactions to psychiatric medications, warranting close monitoring of patients on these medications who are also at risk for WE. This is especially pertinent in cases of serotonin syndrome (SS) where neuromuscular hyperactivity and myoclonus impede gait. SS is likewise associated with AMS which can also be present in cases of WE.[57] WE has been reported in a case of concurrent serotonin syndrome (SS) in a patient on the serotonergic antidepressant fluoxetine, highlighting the need to monitor patients on psychiatric regimens for WE that may be masked by confounding differentials.[58]

Wernicke's encephalopathy and older adults

Older adults are at risk for malnutrition and subsequent WE for a number of well-documented reasons. Malnutrition has been reported in 14 to 60% of older adults.[59,60] Causes include polypharmacy, which can yield GI complications and lower appetite, as well as chronic diseases like cancer or diabetes, which increase metabolic demands or impair nutrient absorption.[61] Thiamine deficiency has been identified in 6 to 14% of older adults in nursing facilities.[62,63] WE symptoms have also been reported in a number of case reports and case series of older adults.[64–66] As such, older adults should be monitored closely for thiamine deficiency and WE.

Pregnancy and the risk of Wernicke's encephalopathy

Nutritional demands increase in pregnancy, and thiamine deficiency may occur and be furthermore exacerbated by associated nausea and vomiting. More severe symptoms, or hyperemesis gravidarum (HG), occur in approximately 3% of cases and can profoundly impair nutrient absorption.[44,67,68] These clinical manifestations can deplete thiamine stores and lead to WE in pregnant populations. These patients often present with severe vomiting alongside neurological symptoms of WE.[67–70] Notably, the clinical symptoms of HG, including nausea, vomiting, and loss of appetite, are non-specific and overlap with thiamine deficiency signs, potentially delaying

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appropriate WE treatment.[44] Furthermore, pregnant patients tend to be younger, and as such AMS is a less predominant clinical feature of WE, further complicating its diagnosis.[44,68] Data on the potential fetal effects of WE are limited, though fetal thalamic alterations and demise have been reported.[71–73] This highlights the critical need for timely diagnosis and treatment of WE in pregnancy.

Conclusion

Over a century and a half after Carl Wernicke's reports, the diagnostic approach to WE continues to evolve. While thiamine remains the gold-standard treatment, consistent dosing guidelines are lacking. Furthermore, though WE is highly treatable, thiamine is underprescribed at discharge, and further advocacy and education around its importance are needed.[47] Areas for future exploration include characterization of prognostic neuroimaging changes, as well as the development of rapid and sensitive imaging tools to identify them. Additionally, continued appraisal of therapeutic dosing of thiamine and longer-acting formulations may improve clinician confidence in prescribing and promote better treatment adherence.

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Conflicts of interest

The authors declare that they have no pertinent conflicts of interest to disclose.

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