Academic Emergency Medicine

ORIGINAL ARTICLE

Prevalence of Wernicke's Encephalopathy When Receiving Dextrose Before Thiamine: A National Study of Veterans

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Received: 17 March 2025 | **Revised:** 6 July 2025 | **Accepted:** 6 August 2025

Supervising Editor: Latha Ganti

Funding: The authors received no specific funding for this work.

ABSTRACT

Background/Objectives: A commonly taught principle when treating emergency department (ED) patients with acute alcohol intoxication is to give thiamine before dextrose to avoid precipitating Wernicke's encephalopathy (WE). We sought to evaluate the prevalence of WE among a national sample of veterans who presented to the ED with alcohol intoxication and who then received dextrose before thiamine.

Methods: This is a retrospective, cross-sectional study of patients presenting to all Veterans Affairs (VA) Emergency Departments (ED) over a 10-year period (2010–2019). Data was obtained through a Microsoft SQL (Redmond, WA) query of the VA Corporate Data Warehouse. Inclusion criteria were any ED visit with an alcohol intoxication ICD9/10 code and/or serum ethanol result > 50 mg/dL, as well as administration of any intravenous fluids containing dextrose at any concentration. Exclusion criteria was administration of thiamine before dextrose infusion. The primary outcome was diagnosis of WE by ICD9/10 code or manual chart review in the ED, hospitalization, or follow-up visit within 90 days. Data was analyzed with descriptive statistics.

Results: 120 encounters by 114 individual patients met the inclusion/exclusion criteria, with a median age of 59 (IQR 49–64). There were 104 (91%) male patients, 77 (68%) were white, 27 (24%) were Black, and 6 (5%) were Hispanic. Most patients with a recorded AUDIT-C screened positive for AUD (94%) or had a documented history of alcohol abuse (90%). No cases of Wernicke's encephalopathy were identified by ICD code or manual chart review.

Conclusion: Our data suggest that alcohol-intoxicated patients are unlikely to develop Wernicke's encephalopathy with acute dextrose administration. While administering thiamine in alcohol-intoxicated patients is low risk and potentially beneficial, we submit that hypoglycemia treatment should not be delayed for this intervention.

1 | Introduction

Alcohol is the most used intoxicant in the United States (US). Each year over 140,000 people die from excessive alcohol use in the US, which is almost twice the number of people who died from opioid overdose in 2021 [1]. The number of alcohol-related emergency department (ED) visits continues to grow significantly—one study showed a 61% increase from 2006 to 2014 [2]. For many people with alcohol use disorder (AUD), EDs routinely serve as the first point of entry into the health care system when they present with acute alcohol intoxication. In a national study of approximately 3.7 million ED

Poster presentations: 31st Annual Wisconsin Emergency Medicine Research Forum, Madison, WI, March 2, 2023 and Society of Academic Emergency Medicine Annual Meeting 2023, Austin, TX, May 19, 2023.

visits by individuals with AUD between 2014 and 2018, the most common presenting concern was acute alcohol intoxication [3].

Patients with AUD are prone to severe nutritional deficiencies such as thiamine (vitamin B1), making them at risk for developing Wernicke's Encephalopathy (WE) [4]. WE is a neurologic illness characterized by the symptom triad of confusion, ocular disturbances, and cerebellar ataxia [5]. Since thiamine is an important coenzyme in glucose metabolism, giving dextrose to a thiamine-deficient patient may rapidly deplete already insufficient stores of thiamine and precipitate WE [5]. AUD is the most common risk factor for WE over other thiamine-deficient conditions such as bariatric surgery, malignancy, and hyperemesis gravidarum [6]. Consequently, a commonly taught principle is to avoid giving dextrose before thiamine to ED patients who present with acute alcohol intoxication [4]. However, evidence for this practice is limited to case reports or series [7, 8]. Patients presenting with acute alcohol intoxication are at high risk of hypoglycemia which, if not quickly treated, can lead to coma and death [9]. It is important, therefore, to increase our understanding of the risk of precipitating WE in this population to justify delaying treatment of hypoglycemia with dextrose until administration of thiamine.

We hypothesized that in patients who presented to the ED with acute alcohol intoxication and received dextrose before thiamine, the frequency of WE associated at any point during hospitalization or follow-up visit would be low. The purpose of this study was to evaluate the prevalence of Wernicke's encephalopathy among a large, national sample of veterans with acute alcohol intoxication who received dextrose before thiamine in the ED.

2 | Methods

2.1 | Study Design and Setting

This was a retrospective, cross-sectional study of patients presenting to all Veterans Health Administration (VHA) EDs in the United States over a 10-year period between 2010 and 2019. The VHA is the largest integrated healthcare system in the United States and includes 172 hospitals that provide care to over nine million veterans [10]. This study was approved by the Milwaukee VA Institutional Review Board and was performed in compliance with the Strengthening the Reporting of Observational studies in Epidemiology (STROBE) statement for cohort studies.

2.2 | Selection of Participants

Inclusion criteria were (1) adult veteran (age ≥ 18 years old), (2) VHA ED visit in the study period with an alcohol intoxication ICD9/10 code (ICD9–303.0, 305.0, 291.4; ICD10–F10.12*x*, F10.22*x*, F10.92*x* [*x* = wildcard]), and/or serum ethanol result > 50 mg/dL, and (3) administration of any intravenous (IV) fluids containing dextrose at any concentration. Exclusion criteria were administration of thiamine (either oral or IV) before dextrose infusion. Patients were identified using the VA Informatics and Computing Infrastructure by Microsoft SQL query performed on the VA Corporate Data Warehouse.

2.3 | Data Measurements, Outcome, and Analysis

The primary outcome was a diagnosis of WE either defined by ICD9/10 code (ICD9-265.2; ICD10- E51.2) or documented in the electronic medical record (EMR). We used patient identifiers to access the EMR in the Veterans Health Information Systems and Technology Architecture (US Department of Veteran's Affairs). Physician abstractors (J.J., A.F.) performed manual chart review on every patient that met inclusion criteria. The abstractors were not blinded to the study hypothesis. All charts were reviewed by both physician abstractors, with discrepancies resolved by review, discussion, and consensus. The records reviewed included nursing and triage notes, ED physician notes, outpatient medication lists, admission History and Physical (H&P) notes, and discharge summaries. To determine if thiamine was given before dextrose, we compared the documented time of thiamine administration to dextrose administration in the EMR medication log. All cases with simultaneous dextrose and thiamine administration (i.e., identical timestamps) were excluded. When both dextrose and thiamine were given, the time to thiamine infusion was defined as the time between dextrose infusion and the time thiamine was given. The following criteria were used to review the EMR for a clinical impression for WE if not already diagnosed by ICD9/10 code: (1) Notation of visual disturbances, ataxia, and/or confusion in the physical exam, (2) Administration of high-dose thiamine for treatment of WE, and (3) Clinical impression of Wernicke's Encephalopathy in the ED note, admission H&P, discharge summary, or outpatient follow-up visit within 90 days. Additional data included demographics, history of alcohol abuse as documented via ICD 9/10 code in the problem list, whether thiamine was listed as an outpatient medication, type of IV dextrose (D50, crystalloid, electrolyte supplementation, antibiotic), volume of IV dextrose, ED disposition, length of stay, and outpatient follow-up within 90 days. We also reported Alcohol Use Disorders Identification Test-Concise (AUDIT-C) scores, a three-question screening tool to identify individuals who may be engaging in risky drinking behavior [11]. We defined this as the highest score recorded at any point within 90 days of the ED visit in question. Descriptive statistics were performed using Microsoft Excel.

3 | Results

3.1 | Characteristics

Of the 251,310 patient encounters for alcohol intoxication at VHA EDs from 2010 to 2019, 296 encounters met inclusion criteria and received dextrose in the ED. Of these, 176 encounters were excluded due to receiving thiamine before dextrose (Figure 1). This resulted in 120 encounters by 114 individual patients. Characteristics of these 114 patients are shown in Table 1. The median age was 59, and the majority were male (91%) and of white race/ethnicity (68%). Most patients with a recorded AUDIT-C screened positive for risky drinking

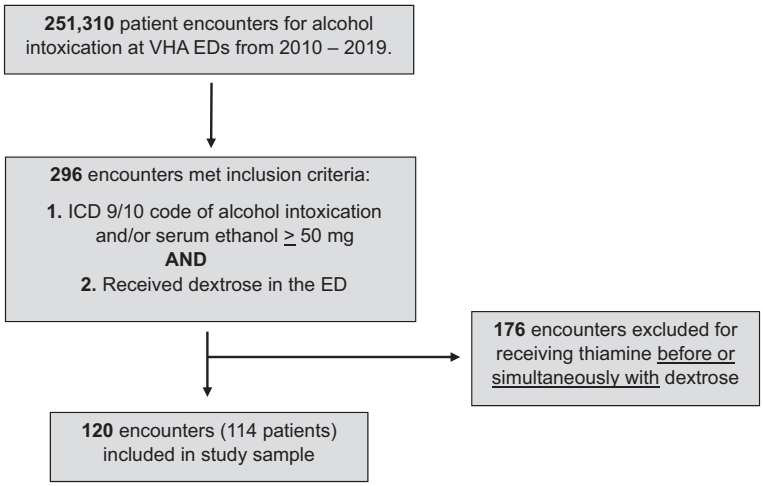

FIGURE 1 | Study sample diagram.

TABLE 1 | Patient demographics.

	***N*=114**
Age, median year (IQR)	59 (49–64)
Male, *n* (%)	104 (91%)
Race/ethnicity, *n* (%)	
White	77 (68%)
Black	27 (24%)
Hispanic	6 (5%)
Unknown	7 (6%)
AUDIT-C (3 item) recorded	98
Positive (> 3 men, > 2 women)	92 (94%)
Negative	6 (6%)
Mean (STD)	9.4 (3.7)
History of alcohol abuse	103 (90%)

behavior (94%; 92/98) or had documented history of AUD (90%; 103/114).

3.2 | Treatment and Outcomes

As shown in Table 2, a third of patients (33%) had thiamine listed in their outpatient medication list. Less than a third of patients (28%) received thiamine in the ED, while most patients (68%) received thiamine at some point during hospitalization (ED and inpatient). For those that received thiamine, the median time between thiamine and dextrose administration was 260 min.

Only 5% of patients received dextrose through D50 formulation. The other 95% received dextrose through other dextrose-containing fluids such as D5 normal saline or lactate ringer's (60%), antibiotics (18%), and electrolyte supplementation (13%).

Over half of patients (59%) received 0.5 L to 2 L of dextrose-containing fluids, while 35% received < 0.5 L and 6% received > 2 L.

Less than half of patients (46%) were discharged from the ED while the remainder were admitted to a hospital ward (38%), mental health facility (10%), or Intensive Care Unit (6%). Median length of stay was 4 days (IQR 3–7.5).

Most patients (86%) had an outpatient follow-up within 90 days, with median time to follow-up 7 days (IQR 2–15).

As shown in Table 2, we found no cases of WE either through ICD 9/10 code or manual chart review.

4 | Discussion

This study provides new insights into the prevalence of WE in acute alcohol intoxicated patients who receive dextrose prior to thiamine in an emergency department setting. We examined a large, national sample of veterans presenting to the ED with alcohol intoxication and found no cases of WE among 120 patient encounters that received dextrose before thiamine or without thiamine. To put this in perspective, there were 2254 cases of WE among the remainder of the 251,310 VA patient encounters for alcohol intoxication from 2010 to 2019. While our results from this retrospective study are not conclusive, they do suggest that acute administration of dextrose to alcohol-intoxicated patients is unlikely to precipitate WE.

To our knowledge, there is one prior study beyond the level of a case report or series that has evaluated this. Merlin et al. [12] found no significant difference in Glasgow Coma Scale (GCS) between hypoglycemic patients that received thiamine and did not receive thiamine by Emergency Medical Services (EMS). Important limitations of this study include the use of GCS as a surrogate marker for WE, the inability to identify whether included patients were at increased risk for thiamine deficiency, patient selection from a single center and geographical location, and the evaluation of treatment and outcomes restricted to the prehospital setting [12].

TABLE 2 | Treatment and outcomes.

	***N*=120**
Thiamine	
Patient had thiamine listed in outpatient medication list	40 (33%)
Received thiamine in the ED	33 (28%)
Received thiamine at any point during hospitalization (ED and inpatient)	81 (68%)
Median time to thiamine, minutes (IQR)	260 (113–391)
Dextrose	
Type[a]	
Crystalloid (D5 normal saline or lactated Ringer's)	72 (60%)
Electrolyte supplementation (i.e., magnesium)	16 (13%)
Antibiotic (i.e., ceftriaxone)	21 (18%)
Other (i.e., diltiazem)	9 (8%)
D50	6 (5%)
Volume	
≤500 mL	42 (35%)
500 mL to 2 L	71 (59%)
≥2 L	7 (6%)
Disposition	
ED discharge	55 (46%)
Mental health admission	12 (10%)
Floor admission	46 (38%)
ICU admission	7 (6%)
Median length of stay, days (IQR)	4 (3–7.5)
Outpatient follow-up within 90 days	103 (86%)
Median time to follow-up, days (IQR)	7 (2–15)
Number of patients who were discharged with no follow up within 90 days	8 (7%)
Primary outcome: Wernicke's Encephalopathy	
ICD 9/10 code	0 (0%)
ED or inpatient documentation suggesting Wernicke's Encephalopathy	0 (0%)

[a]Do not add up to 120 because some patients received more than 1 type.

Since the most frequently cited case series by Watson et al. in 1981, multiple emergency medicine (EM) researchers have appropriately questioned the practice of prioritizing thiamine before dextrose [13, 14]. The current edition of Tintinalli's Emergency Medicine: A Comprehensive Study Guide states thiamine supplementation prior to dextrose for patients with acute alcohol intoxication is not required [15]. However, numerous clinical references such as UpToDate and other EM, neurology, and internal medicine textbooks still advise that dextrose should not be given to thiamine-deficient patients prior to thiamine [16–19]. We believe a potential serious consequence of this practice is a delay in treatment of hypoglycemia. It is well-established that patients with AUD are at increased risk for developing hypoglycemia [9, 20]. Hypoglycemia causes many harmful physiologic effects, most importantly in the brain. Neurons are critically dependent on an immediate and continuous supply of glucose from the bloodstream since they have a limited ability to store or synthesize glucose [21]. The brain accounts for approximately 25% of the body's glucose utilization, consuming almost 5.5 mg of glucose per 100 g of brain tissue per minute [22]. Because brain metabolism quickly fails with hypoglycemia, the central nervous system has multiple physiologic mechanisms to quickly detect low blood glucose and maintain glucose homeostasis [23]. Seven patients in our study were hypoglycemic and appropriately treated with D50. Although this represents a small proportion of our sample, the data in our study overall indicates no immediate poor consequences of acute dextrose administration in alcohol-intoxicated patients without thiamine supplementation. Based on our results, we agree with prior EM researchers and advise immediate treatment of hypoglycemia without waiting for thiamine administration.

Review of prior case reports identifies a risk of precipitating WE only after prolonged and large amounts of dextrose are administered before thiamine supplementation [4]. Most of these patients received anywhere from 1 to 3 L of 5% dextrose-containing fluids (100–300 g dextrose) before developing symptoms at least 24 to 48 h later [4]. Over half (59%) of the patients in our study received 0.5 to 2 L of 5% dextrose-containing fluids (25–200 g dextrose), and most (73%) did not receive any thiamine in ED. However, all admitted patients received thiamine upon admission within 6 h. While chronic thiamine supplementation is potentially beneficial for patients with AUD, we believe it should be initiated after prioritizing immediate treatment of hypoglycemia [24–26]. In essence, we think the evidence of benefit for rapidly treating hypoglycemia in these patients outweighs the evidence of potential harm if it is not delayed for thiamine administration. As such, we submit that treatment protocols, electronic order sets, and educational resources should emphasize the immediate administration of intravenous dextrose in patients with suspected critical hypoglycemia regardless of the availability of thiamine, even as thiamine should also be administered as soon as is practical in any patient at risk for development of WE.

5 | Limitations

The results of this retrospective, observational study should be interpreted with caution due to several limitations. Because we evaluated a veteran population, our study sample was predominantly white males, which may affect generalizability. Second, we may have incompletely characterized patients at risk for thiamine deficiency since there are other thiamine-deficient conditions such as bariatric surgery and hyperemesis gravidarum [27, 28]. However, AUD is the most common risk factor for WE over these other conditions [3, 6]. Next, we used acute ethanol intoxication as a surrogate for AUD, which is a risk factor for

WE because sustained alcohol ingestion leads to severe nutrient deficiencies such as thiamine. We believe acute alcohol intoxication is a reasonable surrogate for AUD because prior studies have demonstrated it. (1) It is the most common entry point into the healthcare system among patients with AUD and (2) AUD screening in the ED is often missed [3, 29]. Furthermore, most patients in our study with a recorded AUDIT-C inpatient scored positive (94%; 92/98) or had documented history of alcohol abuse (90%; 103/114). Another limitation is a third of our patients (33%) had thiamine listed in their outpatient medications at the time of ED admission, so it is possible they were not thiamine-deficient on presentation to the ED. Fifth, the documented times of dextrose and thiamine administration in the EMR may be inaccurate, which may have biased our sample. However, the case with the shortest time between dextrose and thiamine was 16 min and there were only 5 included cases where the time between dextrose and thiamine was < 60 min. Sixth, we may have missed cases of WE because it may not present with the classical triad of symptoms and WE symptoms can overlap with alcohol intoxication or withdrawal, resulting in underdiagnosis in ED encounters. Additionally, most diagnoses of WE happen after the index visit and we may have missed cases of WE diagnosed on subsequent visits, particularly if veterans presented for follow up at EDs outside the VA system. However, most of our patients (86%) had an outpatient follow-up within 90 days with a median time to follow up of 7 days at which there were no documented diagnoses or clinical concern for WE. Next, patients in our study received lower total amounts of dextrose compared to patients in prior case reports (25–200 g compared to 100–300 g), and risk of precipitating WE is higher with larger amounts of dextrose [4]. Finally, since we found zero instances of our primary outcome of Wernicke's Encephalopathy, we did not calculate inter-rater reliability or 95% confidence intervals around the prevalence estimate.

6 | Conclusion

In summary, the data from this large, national, cross-sectional sample over a decade suggests that alcohol-intoxicated patients are unlikely to develop Wernicke's encephalopathy with acute dextrose administration. While administering thiamine in alcohol-intoxicated patients is low risk and potentially beneficial, we submit that hypoglycemia treatment should not be delayed for this intervention [27–29].

Author Contributions

A.F. and D.G. conceived of the study. A.F. and J.J. designed the study. A.F., K.S., and J.J. participated in data collection. J.J. and A.F. analyzed and interpreted the data. J.J. drafted the manuscript and all authors contributed substantially to its revision.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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