ISSN: 2754-4931

Journal of Gastroenterology & Hepatology Reports

SCIENTIFIC Research and Community

Case Report

Open Access

Wernicke's Encephalopathy in the Setting of Severe Malnutrition and Alcohol Abuse

Lyndie Wilkins Parker DO[1]*, Nathan Duffin DO[1] and Yamine Saddouk MD DO[2]

[1]Department of Internal Medicine, Mountain Vista Medical Center/Midwestern University, Mesa, AZ, USA

[2]Department of Internal Medicine, Program Director, Mountain Vista Medial Center/Midwestern University, Mesa, AZ, USA

ABSTRACT

Wernicke's encephalopathy is an acute, potentially fatal syndrome attributable to thiamine deficiency. Prompt identification and treatment is essential to prevent the development of Korsakoff syndrome, an irreversible memory disorder associated with both retrograde and anterograde amnesia. Here, we present a case of a 39-year-old woman admitted to the ICU with a diagnosis of alcohol withdrawal and severe malnutrition. On admission, the patient was noted to have horizontal nystagmus and underwent aggressive thiamine repletion resulting in resolution of her symptoms.

***Corresponding author**

Lyndie Wilkins Parker, Department of Internal Medicine, Mountain Vista Medical Center/Midwestern University, Mesa, AZ, USA, DO 1301 S. Crismon Road, Mesa, Arizona 85209, USA. Phone: 970-556-1603; E-mail: lyndieparker9@gmail.com

Received: March 22, 2022; **Accepted:** March 28, 2022; **Published:** April 04, 2022

Introduction

Thiamine, or B1, is water-soluble vitamin and an essential co-enzyme of multiple metabolic pathways in the brain [1]. A full-grown adult requires 1-2mg of thiamine a day, however in states of high metabolic demand, such as chronic alcohol use or anorexia, individuals will have higher requirements. Absorbed in the duodenum and stored in the liver, thiamine depletion can occur within 18 days if it is not actively repleted [2].

Originally described in 1881 by Carl Wernicke, Campbell and Russell identified thiamine as the probable underlying nutritional deficiency and stressed the importance of repletion in the 1940s [2]. Since that time, the use of high dose thiamine has revolutionized treatment of Wernicke's encephalopathy.

Early symptoms include headache, fatigue, mental status changes, ocular abnormalities, gait incoordination and ataxia. These symptoms are largely thought to result from involvement of the mamillary bodies. Symptoms can progress to agitation, hallucinations, and other behavioral disturbances.

Case Report

39-year-old female with history of alcohol abuse (last drink 2 months prior to admission) and degenerative joint disease presented for evaluation of a near-syncopal event. The patient stated she had poor oral intake over the past several months. She occasionally ate a microwave meal, but was unable to tolerate a full meal secondary to early satiety and nausea. The patient was found to have multiple electrolyte derangements including a potassium of 2.8, phosphorus of 0.40, magnesium of 1.7, albumin of 2.7, Beta-hydroxybutyric acid of 101, a thiamine of 133.2 (66.5-200.0), and a hemoglobin of 6.6. Serum alcohol and urine alcohol were negative. On exam, the patient had horizontal nystagmus, abnormal finger-to-nose test, and BMI of 14.1kg/m2. The patient was started on high dose thiamine, 500mg IV three times day for 3 days, and then 200mg IV daily. During her hospital course, she was evaluated by nutrition and started on ensure shakes three times daily and magic cup nutritional support twice daily. She was fluid resuscitated and her dietary habits gradually improved. Her horizontal nystagmus resolved on day 4 of her hospital course and ultimately the patient was discharge with outpatient support several days later.

Discussion

Chronic alcohol misuse only results in Wernicke's encephalopathy when the dietary intake is not sufficient. Concurrent liver impairment, poor dietary intake, and decreased intestinal absorption increase the risk of developing thiamine deficiency. The majority of thiamine in serum is protein bound to albumin with over 90% contained within erythrocytes. In the case described above, the patient had adequate stores of thiamine, but given her low hemoglobin and albumin, ineffective transportation of such stores. While the patient in this case had refrained from alcohol intake for over 2 months prior to presentation, she had a long-standing history of alcohol ingestion for many years. Alcohol metabolism causes increased demand of thiamine, but concurrently decreases the amount of thiamine transported across the intestinal mucosa and impairs conversion of thiamine to thiamine pyrophosphate [3]. Moreover, the patient in this case had hypomagnesemia, magnesium is a necessary co-factor in the thiamine pyrophosphokinase pathway and magnesium deficiency can lead to refractory response to thiamine until magnesium is repleted [2]. Catabolic states are known to cause the down regulation of enzymes associated with thiamine, and, consequently, optimization of diet plays an essential role in resolution [4].

Diagnosis of Wernicke's encephalopathy can be confirmed by thiamine blood concentration or via measurement of red blood cell transketolase. MRI can also confirm Wernicke's with increased symmetric T2 signal in the paraventricular regions of the thalamus, hypothalamus, mamillary bodies, and midline cerebellum [2]. In this case, no MRI was performed.

Given its high morbidity and mortality, rapid diagnosis and treatment of Wernicke's is essential. High dose thiamine should be started immediately. While there is no consensus on optimal dosing, current guidelines indicate high doses are required in alcoholic patients, with a suggested dose of 500mg IV. As the half-life of thiamine is 96 minutes, three-times a day dosing is recommended so a sufficient concentration reaches the brain [4]. In patients with severe alcohol withdrawal or poor nourishment, prophylactic treatment with 250mg IM thiamine should be done for 3-5 days [2]. Thiamine should always be given prior or concurrently with glucose as glucose can precipitate Wernicke's encephalopathy in thiamine-deficient patients. 29% of patients will have ocular abnormalities including nystagmus and conjugate-gaze palsies which result from lesions of the pontine tegmentum [2]. Recovery from ophthalmoplegia is rapid, although many patients continue to have a persistent, discrete horizontal nystagmus. Ataxia and mental status gradually improve with optimization of nutrition and thiamine stores.

Conclusion

Wernicke's encephalopathy often presents with multiple non-specific neurological signs. Rapid identification, early treatment, and aggressive management are essential to prevent long term consequences. Addressing nutritional status and optimization of all micronutrients is essential to ensure optimization of administered thiamine.

References

1. Praharaj Samir Kumar, Ravindra N Munoli, Sonia Shenoy, Suma T Udupa, Linda Susan Thomas, et al. (2020) High-Dose Thiamine Strategy in Wernicke-Korsakoff Syndrome and Related Thiamine Deficiency Conditions Associated with Alcohol Use Disorder. Indian Journal of Psychiatry 63: 121-126.
2. Sechi, GianPietro, Alessandro Serra (2007) Wernicke's Encephalopathy: New Clinical Settings and Recent Advances in Diagnosis and Management. The Lancet Neurology, 6: 442-455.
3. Saad Laura, Luiz FAL Silva, Claudio EM Banzato, Clarissa R Dantas, Celso Garcia, et al. (2010) Anorexia Nervosa and Wernicke-Korsakoff Syndrome: A Case Report. Journal of Medical Case Reports 4: 217.
4. Jayaprakash B, Rao KN, Patil N, Bhattacharjee D, Maden M, Rau NR, et al. (2016) Wernicke's Encephalopathy-'Pushing the Envelope' of Patient's Profile: A Case Report. Annals of Neurosciences 23: 188-193.

Copyright: ©2022 Lyndie Wilkins Parker, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.