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Abstract Form

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Project Title:	Mineral Mining: A Confounding Case of Wernicke Encephalopathy

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction: Wernicke encephalopathy can be a consequence of heavy alcohol use, and it classically presents with disorientation, ocular motor dysfunction, and ataxia. However, other medical complications may muddle this clinical picture.

Case Report: A 26-year-old previously healthy female presented with several months of worsening bilateral lower extremity weakness. She endorsed having multiple shots of hard liquor daily for several years and reported a low appetite for the past month following a friend's death. Her last drink was three days before admission. On admission, vital signs were stable except for mild tachycardia, and the patient was completely disoriented and unable to ambulate due to unsteadiness. She had an abnormal finger-to-nose test, stocking-glove pattern neuropathy, and bilateral vertical nystagmus. She also had leukopenia, normocytic anemia, and low serum thiamine, folate, and copper. Per neurology recommendations, she was empirically treated for Wernicke encephalopathy using IV thiamine 500 mg TID for 2 days, followed by 200 mg daily for 5 days. MRI brain was negative for mamillary body or thalamic changes. Further encephalopathy workup was negative, including lumbar puncture, Vitamin B12, methylmalonic acid, HIV, RPR, serum ethanol, and urine drug screen. After completing her parenteral thiamine therapy, she was discharged with oral thiamine and folate supplementation for alcohol use disorder and gabapentin for neuropathy. She was instructed to purchase over-the-counter copper supplements for her copper deficiency. The patient made a full recovery other than she did not remember being hospitalized during a follow-up phone visit.

Discussion: This patient's neurological status was initially attributed to Wernicke encephalopathy given her disorientation, vertical nystagmus, and cerebellar signs such as a positive finger-to-nose test. However, she incidentally had low serum copper. Although rare, it occurs in people with malnutrition, such as this patient who endorsed significantly decreased eating for the past month prior to admission, and it can be treated with supplements or cocoa. Copper deficiency is not typically associated with alcohol use based on current literature. However, it can lead to anemia, leukopenia, and neuropathy, all of which were present in this patient. A stocking-glove pattern neuropathy and macrocytic anemia are not unexpected in patients with alcohol use, but this patient had normocytic anemia, leukopenia, and no mamillary body or thalamic changes in her brain MRI, which suggests that her neurologic symptoms were not solely from Wernicke encephalopathy. This case illustrates that although Wernicke encephalopathy is usually considered in the differential diagnosis of patients with neurologic abnormalities in the setting of heavy alcohol use, copper deficiency should also be assessed, especially in patients with poor food intake. Properly managing patients with such a complex clinical picture is essential to improve their neurologic status, avoid irreversible complications such as Korsakoff psychosis, and treat reversible vitamin and mineral deficiencies.