

Non-Alcoholic Wernicke-Korsakoff Syndrome Following Restrictive Diet and Prolonged Vomiting in a Young Woman: A Case Report

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ABSTRACT

Wernicke-Korsakoff syndrome (WKS) is a neurological disorder resulting from severe thiamine deficiency and is classically associated with chronic alcohol use. Non-alcoholic causes, though less well recognised, include prolonged vomiting, restrictive dietary intake, and other malnourished states. We report a 25-year-old woman with no history of alcohol consumption who presented with a two-day history of confusion, unsteadiness, and horizontal diplopia, together with an inability to recognise close family members, following three weeks of poor oral intake and recurrent vomiting associated with a restrictive diet. Examination revealed disorientation, confabulation, profound anterograde amnesia, horizontal nystagmus, and a broad-based gait. Magnetic resonance imaging demonstrated changes consistent with Wernicke encephalopathy, and serum thiamine was low. She was treated with high-dose intravenous thiamine prior to glucose administration, along with supportive care. Nystagmus and gait ataxia improved, but the amnesic deficit and confabulation persisted at discharge. This case underscores that thiamine depletion from restrictive eating and prolonged vomiting alone, independent of alcohol use, can precipitate the full Wernicke-Korsakoff triad in young women, and highlights the need for a high index of suspicion and prompt thiamine replacement to prevent irreversible neurological sequelae.

Keywords: Confabulation; Nystagmus; Wernicke; Diplopia

INTRODUCTION

Wernicke-Korsakoff syndrome is a neurological disorder resulting from severe thiamine deficiency and is traditionally associated with chronic alcohol consumption. However, thiamine deficiency may also occur in non-alcoholic individuals because of prolonged vomiting, inadequate nutritional intake, restrictive diets, starvation, gastrointestinal disorders, and rapid weight loss [1,2]. The overall incidence of the syndrome is estimated at 2.4 per 100,000 people, and in non-alcoholic populations this falls to approximately 0.39 per 100,000, with the condition being roughly three times more common in men than in women, a discrepancy that makes presentations in young women all the more noteworthy [4,5]. The core pathophysiology stems from thiamine deficiency, with alcoholism as the leading cause; however, in non-alcoholic patients, hyperemetic states, dialysis, acquired immunodeficiency syndrome, gastrointestinal surgery, refeeding syndrome, malignancy, and prolonged parenteral feeding have all been implicated [3,8,9]. Early recognition is crucial because the acute neurological manifestations may be reversible with timely thiamine replacement, whereas persistent memory impairment may become prolonged or irreversible.

The classic triad of confusion, ataxia, and nystagmus occurs together in fewer than half of affected patients, and patients frequently develop additional neurological sequelae such as pupillary reflex defects, confabulation, and anterograde amnesia. The novelty of the present case lies in the development of Korsakoff syndrome in a young, non-alcohol-dependent woman after only three weeks of poor oral intake and recurrent vomiting associated with a restrictive diet [6,7,13]. Here, we report this case, highlighting an uncommon but clinically important presentation of thiamine deficiency and emphasising the need for early suspicion and prompt treatment.

CASE DESCRIPTION

A 25-year-old female with no history of alcohol intake presented with a two-day history of confusion, unsteadiness and horizontal diplopia, accompanied by an inability to recognise close family members including her sibling. She gave a history of poor oral intake and recurrent vomiting over the preceding three weeks following a restrictive diet, with no prior neurological, psychiatric or cardiac illness and no regular medications. On examination she was afebrile, with blood pressure of 100/64 mmHg and a pulse of 88 beats per minute. She was disoriented to time and place, with confabulation and profound anterograde amnesia disproportionate to her level of alertness, GCS 14/15. Horizontal nystagmus and a broad-based gait were noted, while cardiovascular, respiratory and abdominal examinations were unremarkable.

MRI brain showed changes consistent with Wernicke encephalopathy, and serum thiamine was low. Liver function tests showed mild transaminase elevation, while renal parameters, electrolytes and complete blood count were within normal limits. This combination of amnesia with confabulation, oculomotor findings and ataxia fulfilled the classic Wernicke-Korsakoff triad, though non-alcoholic Korsakoff syndrome in a young woman is an atypical presentation rarely encountered outside chronic alcohol use. Similar cases have been described in the setting of hyperemesis gravidarum, prolonged fasting and restrictive eating disorders, where thiamine depletion from poor intake alone can produce an identical neurological picture despite the absence of any alcohol history, a pattern that continues to be under-recognised because clinicians often anchor on alcohol as a prerequisite for the diagnosis.

She was started on high-dose intravenous thiamine replacement prior to any glucose administration, along with supportive fluids and correction of her nutritional deficits. Nystagmus and gait ataxia improved over the following days, though the amnesic deficit and confabulation persisted at discharge, consistent with reports that oculomotor and cerebellar signs tend to recover well while the memory disorder often remains fixed. She was discharged on oral thiamine supplementation with neurology, psychiatry and dietetic follow-up arranged, and counselled regarding adequate nutritional intake going forward.

DISCUSSION

Non-alcohol-related Wernicke-Korsakoff syndrome has been documented predominantly in younger patients, particularly women, in the setting of nutritional deficiency, persistent vomiting, fasting, restrictive diets, and other causes of malnutrition [6,7,13]. A systematic review of 623 reported cases found that persistent memory impairment occurred in a substantial proportion of patients, demonstrating that cognitive deficits may continue despite improvement of the acute neurological manifestations [1]. Another review of 586 non-alcohol-related cases identified persistent vomiting and marked weight loss as important precipitating factors [2]. These observations support the clinical relevance of recognising thiamine deficiency even in patients without alcohol exposure, and are consistent with the presentation described here.

In the present case, magnetic resonance imaging showed changes consistent with Wernicke encephalopathy, namely hyperintense signals in the peri-third-ventricular grey matter, medial thalami, and vermis, alongside a low serum thiamine level, findings that are classically described in this condition [12]. The patient additionally fulfilled the complete Wernicke-Korsakoff triad of confusion, ataxia, and nystagmus, a combination present together in fewer than half of affected patients, together with confabulation and profound anterograde amnesia disproportionate to her level of alertness.

Management of Wernicke-Korsakoff syndrome should not await confirmatory investigations; high-dose intravenous thiamine, typically 500 mg in the acute stage administered prior to any glucose supplementation, followed by 100 mg three times daily during the first week and continued supplementation for up to three months, remains the cornerstone of therapy, alongside correction of fluid and nutritional deficits [14,15]. While the general treatment approach is well established, the optimal thiamine dosage continues to be debated in the literature. Magnesium, a cofactor for transketolase, may also require supplementation, as deficiency is more common in alcohol-dependent patients but can occur in any debilitated individual [16]. In this case, intravenous thiamine was continued for 37 days; although cognitive function improved clinically and radiologically, persistent Korsakoff deficits remained at discharge, as is classically described, and structured neurorehabilitation was recommended to optimise long-term functional outcomes [11].

The prognosis of Wernicke-Korsakoff syndrome remains guarded despite treatment; acute mortality is low, but long-term median survival ranges from six to eleven years, with death most commonly attributable to infection, malignancy, cardiovascular disease, malnutrition, and other medical comorbidities. Consistent with reports that oculomotor and cerebellar signs tend to recover well while the memory disorder often remains fixed, this patient's nystagmus and gait ataxia improved substantially, whereas her amnesic deficit and confabulation persisted at discharge. This case

reinforces that nutritional thiamine depletion alone, in the absence of alcohol use, can produce the full Wernicke-Korsakoff spectrum, and that persistent vomiting with severe nutritional depletion should be regarded as an important warning sign in young patients [10].

CONCLUSION

This case highlights non-alcoholic Wernicke-Korsakoff syndrome as an uncommon yet clinically significant cause of acute confusion, oculomotor dysfunction, and amnesia in young women, even in the complete absence of alcohol exposure. A three-week history of restrictive dietary intake and recurrent vomiting was sufficient to precipitate the full Wernicke-Korsakoff triad in this previously healthy 25-year-old woman. Clinicians should maintain a high index of suspicion for thiamine deficiency in any patient with prolonged poor oral intake or vomiting, regardless of alcohol history, and should institute empirical high-dose intravenous thiamine without delay, as prompt treatment can reverse oculomotor and cerebellar signs even though the amnestic component frequently persists, underscoring the importance of early recognition and sustained multidisciplinary follow-up.

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