Case Report

Altered sensorium unmasking Wernicke–Korsakoff psychosis: A case report

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ABSTRACT

Wernicke–Korsakoff syndrome (WKS) is an acute neuropsychiatric illness produced by chronic deficiency of thiamine (vitamin B1), usually in association with chronic alcoholism. It is characterized by the sudden onset of Wernicke's encephalopathy (WE) with subsequent chronic Korsakoff's psychosis if untreated. Here, we describe a 68-year-old man who presented with slurred speech, ataxic gait, memory loss, bewilderment, and disorientation. A past history of alcohol dependence, clinical assessment, elevated liver enzymes, and magnetic resonance imaging findings of mammillary bodies and thalamus involvement led to a Wernicke–Korsakoff psychosis diagnosis. The patient responded well to thiamine replacement therapy and supportive management. The case again emphasizes early identification and treatment of WKS, especially in elderly patients with a history of chronic alcohol use.

Key words: Chronic alcoholism, Encephalopathy, Memory loss, Thiamine deficiency, Wernicke–Korsakoff syndrome

Wernicke–Korsakoff syndrome (WKS) is a thiamine deficiency-associated neurological disorder. Wernicke's encephalopathy (WE) is the acute presentation, and it consists of a triad of features: Ophthalmoplegia (or nystagmus), ataxia, and confusion [1,2]. It may advance to Korsakoff's psychosis, which is anterograde and retrograde amnesia, confabulation, and severe cognitive dysfunction, unless diagnosed and treated in time. The disorder is most commonly encountered in chronic alcoholics, who are unable to absorb and utilize thiamine [3,4]. Alcohol not only leads to nutritional deficiencies but also has a direct neurotoxic effect, which worsens the clinical picture [5]. WKS is frequently underdiagnosed, particularly in older patients, where confusion or impairment of memory may be inappropriately blamed on other causes such as dementia or delirium [6]. Magnetic resonance imaging (MRI) plays an important role in the diagnosis of characteristic brain lesions [7] of the mammillary bodies and periventricular areas. Initial parenteral thiamine treatment is the heart of therapy [8] and may result in dramatic recovery. WE, although classically linked to chronic alcoholism, has been described in other non-alcoholic illnesses like hyperemesis gravidarum, making it a matter of urgent recognition and intervention to avoid the development of Wernicke–Korsakoff psychosis [9]. If untreated, thiamine deficiency can result in WE and eventually permanent Korsakoff's psychosis [10]. The clinical syndrome of early mental loss is studied in the present paper based on new findings that suggest the neuroprotective effect of thiamine [11]. Timely recognition and prompt thiamine supplementation are therefore critical, because delayed treatment markedly increases the risk of progression from the acute reversible phase of Wernicke encephalopathy (WE) into the irreversible residual phase of WKS [12,13].

The rationale for reporting this case is to emphasize the importance of early identification and intervention in elderly patients with chronic alcohol use and to prevent permanent cognitive sequelae associated with delayed treatment.

CASE PRESENTATION

A 68-year-old man arrived at the Tiruppur Government Hospital complaining of memory problems, forgetfulness, and disorientation that had persisted for 7 days. He also had poor balance, which caused him to walk unsteadily. In addition, the patient's speech has been slurred for the past 2 days. Medical history revealed that 5 years ago, he had been diagnosed with type 2 diabetes mellitus and 6 years ago he was diagnosed with hypertension, which both were controlled very well on normal drugs, 500 mg of metformin twice daily, and amlodipine 5 mg twice

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a day, respectively. While reviewing social history, it was revealed that the patient had a significant history of alcohol use for more than 30 years.

Upon general examination, the patient was afebrile and aware despite being confused. His vital signs were normal. Systemic examination was also uneventful.

Complete blood count, renal function tests, and serum electrolytes were normal. Liver function tests, however, showed elevated total bilirubin (2.3 mg/dL), direct bilirubin (1.1 mg/dL), serum glutamic-oxaloacetic transaminase (150 IU/L), and serum glutamate pyruvate transaminase (86 IU/L). These results were suggestive of mild hepatic impairment, presumptively secondary to chronic alcohol use. Brain MRI revealed hyperintensities of the mammillary bodies, thalamus, and periventricular white matter, which are characteristic of Wernicke encephalopathy. Based on the clinical presentation, a history of chronic alcoholism, deranged liver function tests, and MRI reports, a diagnosis of altered sensorium secondary to Wernicke–Korsakoff psychosis was made.

The patient was admitted on an inpatient basis and was managed with intravenous thiamine 100 mg 3 times daily and chlordiazepoxide 10 mg twice daily, atorvastatin 10 mg twice daily, aspirin 75 mg once daily, metformin 500 mg twice daily, and enalapril 2.5 mg twice daily. The patient kept improving steadily for the next 6 days, with progressive improvement in orientation, balance, and amelioration of slurring of speech. At the time of discharge, injectable thiamine was substituted with oral thiamine 100 mg once daily, and the other medications remained as ordered. Follow-up was suggested to have a screening done for any intellectual impairment or symptom recurrence.

DISCUSSION

WKS is a reversible, but often unrecognized, form of cognitive impairment, particularly found in cases of chronic alcohol use, where the early administration of thiamine, preferably through intravenous (parenteral), leads to substantial resolution of symptoms in many patients [1,3,6]. If the condition is permitted to progress without diagnosis, it will result in a Korsakoff state with incomplete recovery of cognitive function [2,9]. The elevation of liver enzymes and bilirubin was also indicative of underlying alcohol-induced liver disease and likely played a role [4] in the neuropsychiatric symptoms observed.

The pathophysiology of WKS suggests that thiamine deficiency is associated with impaired energy metabolism in neurons in parts of the brain such as the mammillary bodies [5,7] and medial thalamus as well as periaqueductal grey matter. These structures are exquisitely sensitive to thiamine deficiency and the presence of neurological symptoms. Our patient presented with altered sensorium, memory issues, and gait ataxia that were consistent with WE. The mammillary body and periventricular hyperintensities on MRI further supported the diagnosis [7].

Several recent case reports have emphasized the diverse and frequently overlooked presentation of WKS. In one case, Bagash *et al*. (2021) presented a 64-year-old female with chronic alcohol use who experienced development of progressive gait ataxia and cognitive decline over a 9-month timespan before diagnosis, and in their consideration of the literature, emphasized that WE can be mistaken for a chronic, insidious process rather than the classical presentation of acute ophthalmoplegia, ataxia, and confusion [13]. Early treatment and diagnosis for the patient likely prevented this from occurring in this case. Chronic alcohol use is the primary cause of WE, but it also impacts how much thiamine is absorbed, stored, and utilized in the body [14]. Likewise, Koca *et al*. (2022) reported a non-alcoholic 65-year-old male with cholangiocellular carcinoma who developed encephalopathy whilst on total parenteral nutrition, and MRI confirmed WE, followed by complete recovery after parenteral thiamine, demonstrating that WKS has also been seen in non-alcoholic individuals, particularly malnourished individuals [15]. Barata *et al*. (2020) highlighted in a case series from liaison psychiatry that many inpatients with cognitive changes on the general medical ward were diagnosed retrospectively with WKS, reinforcing that WKS is still vastly under-recognized in the general medical inpatient settings [16]. In addition, Butnariu *et al*. (2024) presented a unique case which mimicked a left-hemispheric stroke, as the patient presented with hemiparesis and aphasia, but was ultimately diagnosed with WKS based on MRI findings and rapid clinical improvement after thiamine [17]. Taken together, the above literature illustrates the varied clinical presentation of WKS and the need for a high index of suspicion for WKS among alcoholic patients and non-alcoholic patients, to mitigate irreversible neurological outcomes.

When evaluating individuals with confusion, gait disturbance, and altered mental status, there are a variety of differential diagnoses that need to be considered and ruled out before diagnosing WKS. For example, hepatic encephalopathy (HE) is an important consideration in chronic alcohol abusers who present with confusion and disorientation; however, HE has an increase in serum ammonia, is characterized by asterixis, and features of liver failure, whereas ocular findings and MRI lesions in the mammillary bodies and thalamus are findings seen in WE [18]. Cerebrovascular accident (stroke) may appear characteristic of WE but typically has focal neurological deficits and limited diffusion patterns that correlate with vascular territories on MRI, in contrast to the symmetrical thalamic and periventricular lesions of WE [19]. Alcohol withdrawal delirium and delirium tremens can exhibit agitation, tremors, and confusion like WE. However, they do not have the ophthalmoplegia, gait ataxia, or rapid response to thiamine infusion, as we do in WE [20]. Other causes like metabolic or infectious encephalopathies (hypoglycemia, hypoxia, meningitis, and encephalitis) must also be ruled out based, in part, on the laboratory and neuroimaging [21]. Clinical suspicion based on

history, supporting MRI evidence, and close observation of the patient's response to thiamine treatment, at least historically, has been a major role in distinguishing WKS from other causes of the aforementioned conditions.

Recent studies continue to further one's understanding of WKS beyond chronic alcohol abuse. Doğan *et al*. (2018) published a case of WE from non-alcoholic gastrointestinal tract disease, noting that prolonged vomiting, intestinal obstruction, or malabsorption can rapidly deplete thiamine stores and mimic neurovascular or infectious causes of encephalopathy [22]. This reinforces the need for clinicians to address gastrointestinal disease as a precipitating cause, especially in frail or nutritionally at-risk patients. Arendt *et al*. (2024) provided a comparison of imaging in alcoholic and non-alcoholic WE and noted that atypical MRI lesions may include cerebellar, cranial nerve nucleus, or corpus callosum lesions in non-alcoholic presentations, suggesting diagnosis should not be focused solely on the typical thalamic or mammillary body lesion [23]. MRI's diagnostic capability is made clear by radiological heterogeneity where the clinical presentation is partial or confusing. In an individual report, Mantero *et al*. (2021) reported a case of a young female patient with a history of a longstanding eating disorder who presented with non-alcoholic beriberi and WE, demonstrating further that chronic restriction of diet, and not merely the intake of alcohol itself, can lead to thiamine deficiency and severe neurological consequences [24]. Collectively, these findings support the notion that WKS needs to be considered in any differential diagnosis of a patient with protracted vomiting, malnutrition, or unknown etiology encephalopathy with or without an alcoholic history.

CONCLUSION

Wernicke–Korsakoff syndrome should always be in the differential diagnosis of altered mental status in an older patient, especially among those who are chronic alcoholics and/or malnourished. This case illustrates the undeniable value of early diagnosis and immediate parenteral thiamine administration, which can produce substantial clinical improvement. Clinician recognition of the characteristic triad, supplemented with MRI findings, and the necessity for immediate thiamine supplementation (even before laboratory tests confirm thiamine deficiency) will include a significant number in optimizing outcomes for the patient. In addition, patient and caregiver education regarding the neurotoxicity of alcohol and the need for continued follow-up over lengthy time periods is also paramount in preventing repetition and permanent neurological consequences.

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