CASE REPORT

Identifying and Addressing Wernicke-Korsakoff Syndrome in Patients Presenting with Alcohol Withdrawal - A Case Report

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ABSTRACT

Wernicke's Encephalopathy (WE), a severe neurological disorder characterized by confusion, ataxia, and ophthalmoplegia, results primarily from thiamine (vitamin B1) deficiency. Strongly associated with alcohol dependence syndrome, WE reflects the detrimental impact of alcohol on nutritional status and thiamine metabolism. This case report explores the relationship between alcohol use disorder (as per DSM-5 criteria) and WE, presenting a detailed examination of a patient exhibiting classical WE symptoms during alcohol withdrawal, diagnosed according to DSM-5 criteria for Alcohol Withdrawal Syndrome. A retrospective case study methodology was applied, focusing on a 44-year-old male patient from Saveetha Medical College Hospital, presenting with symptoms of alcohol withdrawal and diagnosed with WE. Treatment involved high-dose parenteral thiamine, anxiolytics, anti-craving medications, and cognitive rehabilitation strategies. This case underscores the critical need for early recognition and prompt treatment of WE in patients with a history of alcohol use disorder.

Malaysian Journal of Medicine and Health Sciences (2024) 20(SUPP13): 81-83.doi:10.47836/mjmhs.20.s13.18

Keywords: Wernicke's Encephalopathy, Alcohol Use Disorder, Thiamine Deficiency, Alcohol Withdrawal, Nutritional Deficiencies.

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INTRODUCTION

Wernicke's Encephalopathy (WE), a neurological disorder characterized by a triad of symptoms including confusion, ataxia, and ophthalmoplegia, is a condition of grave concern in the field of neurology and psychiatry. This syndrome, first described by Carl Wernicke in the late 19th century, is primarily attributed to a severe deficiency of thiamine (vitamin B1) within the body. While Wernicke's Encephalopathy (WE) can emerge from various causes, there is a significant and well-documented association with alcohol use disorder, as defined by DSM-5 criteria, also known as alcoholism or alcohol use disorder. The connection between WE and alcohol use disorder is complex, warranting thorough investigation due to its profound clinical implications. Chronic alcohol consumption is a major contributor to the onset of WE, largely because of its adverse effects on thiamine metabolism and the resultant nutritional deficiencies. Alcohol use disorder typically results in suboptimal dietary practices, compromised absorption in the gastrointestinal tract, and liver damage, all of which play pivotal roles in precipitating thiamine scarcity, a crucial factor in the development of WE.

Chronic alcohol consumption detrimentally influences thiamine absorption, utilization, and storage in the body. The ensuing thiamine deficiency, coupled with alcohol use disorder as per DSM-5 criteria, constitutes a considerable risk factor for the onset of Wernicke's Encephalopathy (WE). The nexus between these two conditions is noteworthy. Individuals grappling with alcohol use disorder are at an increased risk of suboptimal nutrition due to diminished appetite, compromised absorption of essential nutrients, and impaired liver

function. These conditions can further intensify thiamine deficiency, amplifying the risk of developing Wernicke's Encephalopathy.

The clinical manifestations of Wernicke's Encephalopathy, including confusion, memory deficits, and ataxia, may be masked by symptoms associated with alcohol intoxication and withdrawal, as outlined in DSM-5, thus complicating timely diagnosis and intervention. This difficulty in diagnosis highlights the critical need for heightened awareness and vigilance within clinical settings. Untreated Wernicke's Encephalopathy can evolve into Korsakoff syndrome, a severe and enduring neurological condition marked by profound memory loss and confabulation.

Successful treatment and prevention of Wernicke's Encephalopathy in patients with alcohol use disorder hinge on tackling the underlying issue via cessation of alcohol use, nutritional reinforcement, and thiamine supplementation. This case report aims to provide an in-depth examination of the complex interplay between Wernicke's Encephalopathy and alcohol use disorder. Given the scarcity of case reports on this subject, the current case is notably fascinating, and the unique insights offered in this report make a significant contribution to the field.

Ethical Considerations

The research was conducted in alignment with the highest ethical standards, ensuring patient confidentiality and informed consent throughout the study. Approval from the institutional review board (IRB) was obtained (IEC-Reference number 016/11/2023/IEC/SMCH) prior to the commencement of the study, ensuring adherence to ethical research practices.

CASE REPORT

A 44-year-old male with a history of alcohol use disorder, meeting DSM-5 criteria, presented with symptoms of alcohol withdrawal and Wernicke's Encephalopathy. The patient had a long-term history of alcohol use, consuming 2-3 units of 180ml of liquor per session, leading to significant socio-occupational dysfunction. His prolonged alcohol use was associated with diminished appetite, compromised absorption of essential nutrients, and impaired liver function. The patient also reported a history of tobacco use, quantified at 2-3 packets daily over the last 15 years. The initiation of alcohol use was influenced by peer pressure and curiosity, evolving into a dependence as defined by DSM-5 criteria, characterized by both psychological and somatic withdrawal symptoms. The patient's condition led to multiple de-addiction service engagements over a five-year span, with an increasing cognitive decline, irritability, and hostility towards family members.

On examination, the patient was moderately built with clinical signs of icterus, pallor, and digital clubbing. Neurological assessment showed vertical nystagmus, appendicular dysmetria, ambulatory ataxia, and bilateral resting tremors in the upper limbs. Mental Status Examination revealed fluctuating levels of consciousness, disorientation to time, and ataxic gait. His emotional state was anxious, and thought processes were preoccupied with substance cravings. Cognitive assessment showed deficits in immediate and recent memory recall, abstract reasoning, and judgment, with insight classified as Grade 2. A Mini-Mental State Examination (MMSE) score was 20/30, indicating moderate cognitive impairment.

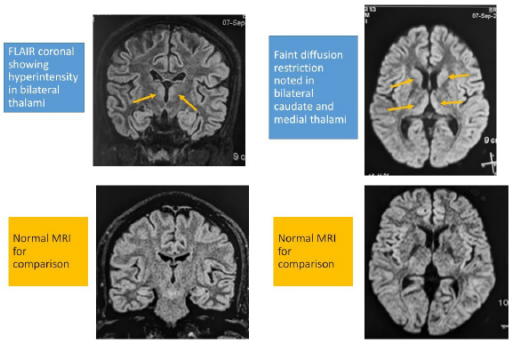

Fig.1 : MRI Findings

DISCUSSION

The patient received a comprehensive treatment regimen, including parenteral administration of thiamine at 500 mg three times daily intravenously for three days, followed by a transition to 250 mg intramuscularly for five days, and then oral thiamine at 100 mg per day as maintenance therapy. This aggressive thiamine replenishment protocol was essential to rapidly restore thiamine levels, prevent further neurological damage, and address acute symptoms of Wernicke's Encephalopathy. In addition to thiamine, the patient was administered anxiolytic therapy with Lorazepam at 6 mg per day in divided doses to manage withdrawal symptoms and anxiety. Anti-craving medication, specifically Acamprosate at 1998 mg per day in a divided regimen, was included to support long-term alcohol abstinence and reduce cravings.

Cognitive rehabilitation strategies were implemented, involving memory training exercises, orientation strategies, and structured cognitive tasks to help recover cognitive functions impaired by the encephalopathy. Under continuous medical oversight, the introduction of Donepezil combined with Memantine further enhanced the patient's memory functions, showing substantial improvement in cognitive symptoms over the course of treatment.

Significance of Treatment Process

The comprehensive treatment process is significant not only for symptom management but also for preventing irreversible neurological damage associated with Wernicke's Encephalopathy. Early and aggressive thiamine supplementation plays a crucial role in halting the progression of cognitive and neurological impairments. Anxiolytics and anti-craving medications support the patient's mental health and commitment to abstinence, which is essential to long-term recovery and relapse prevention. Cognitive rehabilitation aids in restoring impaired functions, allowing for improved quality of life and reintegration into daily activities.

Educational Initiatives and Awareness

To effectively manage and prevent Wernicke's Encephalopathy in populations with high alcohol consumption, healthcare providers should be educated on the early signs and symptoms of WE, the importance of prompt thiamine administration, and the comprehensive management of alcohol withdrawal. Suggested educational initiatives include continuing medical education (CME) workshops, clinical guidelines dissemination, and integration of WE management protocols in medical curricula. Public health campaigns could also target communities with high alcohol consumption rates to raise awareness about the risks of alcohol-related thiamine deficiency and the critical need for early medical intervention.

Potential Side Effects of Treatment vs. Addiction Outcomes

The high-dose thiamine treatment is generally well-tolerated, with side effects such as irritation at the injection site, mild allergic reactions, or gastrointestinal disturbances being relatively rare and manageable compared to the severe outcomes of untreated alcohol addiction and Wernicke's Encephalopathy. These include irreversible brain damage, cognitive decline, and progression to Korsakoff Syndrome, which significantly impacts the patient's quality of life and survival. Thus, the benefits of thiamine treatment far outweigh its minimal risks, highlighting the importance of its inclusion in standard care protocols for at-risk individuals.

CONCLUSION

This case report underscores the critical need to recognize and treat Wernicke's Encephalopathy in patients with alcohol withdrawal symptoms. The complex relationship between alcohol use disorder and WE, as defined by DSM-5 criteria, calls for increased vigilance among healthcare professionals. Timely intervention with high-dose parenteral thiamine, alongside comprehensive management strategies including anxiolytics, anti-craving medications, and cognitive rehabilitation, is vital in preventing the progression to Korsakoff Syndrome. These findings emphasize the importance of awareness and education initiatives targeted at healthcare providers to enhance the diagnosis and management of Wernicke's Encephalopathy in individuals with Alcohol Use Disorder.

ACKNOWLEDGEMENT

We extend our sincere gratitude to the Department of Psychiatry at Saveetha Medical College and Hospital for their invaluable support and resources that facilitated this study. We thank our colleagues for their constructive feedback and assistance during the preparation of this manuscript. Our deepest appreciation also goes to the patient and their family for their cooperation and consent to share this case for academic and educational purposes.

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