E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/15.01.32

Case Report

Wernicke's encephalopathy In a Rural Tertiary Teaching Hospital: A Case Report

Dr.Shaik Fathimunni[1*], Dr.Uma MA[2], Dr.Karthik[3], Dr. Oersala Thasneem ara[4]
[1*]Post graduate, Department of General Medicine, PES Institute of Medical Sciences and Research (PESIMER), Kuppam, A.P.
[2]Professor and HOD, Department of General Medicine, PES Institute of Medical Sciences and Research (PESIMER), Kuppam, A.P.
[3]Assistant Professor, Department of General Medicine, PES Institute of Medical Sciences and Research (PESIMER), Kuppam, A.P.
[4]Senior Resident, Department of General Medicine, PES Institute of Medical Sciences and Research (PESIMER), Kuppam, A.P.
Corresponding Author: Dr.Shaik Fathimunni
Email: [1*]Shaikmunni086@gmail.com
Received: 18.02.25, Revised: 21.03.25, Accepted: 11.04.25

ABSTRACT
Background: Wernicke's encephalopathy is a neurologic disorder caused by thiamine (vitamin B1) deficiency, often associated with chronic alcoholism, malnutrition, or other conditions that impair thiamine absorption or utilization. It presents with a classic triad of confusion, ataxia, and ophthalmoplegia, but the full triad is rarely present in all cases.
Case Presentation: We report a case of Wernicke's encephalopathy in a 50-year-old male who presented with acute onset of confusion, unsteady gait, and blurred vision. His medical history was significant for chronic alcohol use and poor dietary intake. He was admitted to our rural tertiary hospital for evaluation and management.
Conclusion: Early recognition and prompt thiamine administration are critical for improving outcomes in patients with Wernicke's encephalopathy, especially in resource-limited settings.

INTRODUCTION

Wernicke's encephalopathy is a life-threatening condition, predominantly seen in patients with alcohol dependence, malnutrition, or those with gastrointestinal diseases that impair nutrient absorption. Despite being treatable with thiamine supplementation, early diagnosis is often missed, particularly in settings where there may be a lack of awareness or diagnostic facilities.[1,2]

A case report on Wernicke's encephalopathy (WE) in a rural tertiary teaching hospital would typically focus on a detailed clinical presentation, diagnosis, and management of a patient with the condition in a setting with limited resources. Here is an outline of how such a report might be structured

Case Presentation

A 50-year-old male was admitted to the emergency department with complaints of confusion, unsteadiness while walking, and blurred vision. His family described him as having a history of chronic alcoholism (approximately 10 years of heavy drinking), poor nutrition, and a lack of regular meals. He also had a history of repeated episodes of vomiting and diarrhoea over the past month due to self-medication with antacids.

On examination, the patient was disoriented to time and place, had nystagmus, and displayed an ataxic gait. The neurological examination revealed ophthalmoplegia with bilateral lateral rectus palsy. No signs of fever or meningeal irritation were noted. The rest of the physical examination was unremarkable except for mild tachycardia.

Investigations

Routine laboratory tests showed:

- Hemoglobin: 10.2 g/dL (suggesting mild anemia)
- Serum electrolytes: normal
- Liver function tests: mildly elevated liver enzymes (likely related to alcohol use)
- Serum thiamine levels: significantly low
- MRI of the brain: showed characteristic changes consistent with Wernicke's encephalopathy (e.g., bilateral thalamic involvement). (Figure 1 and 2)

E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/15.01.32

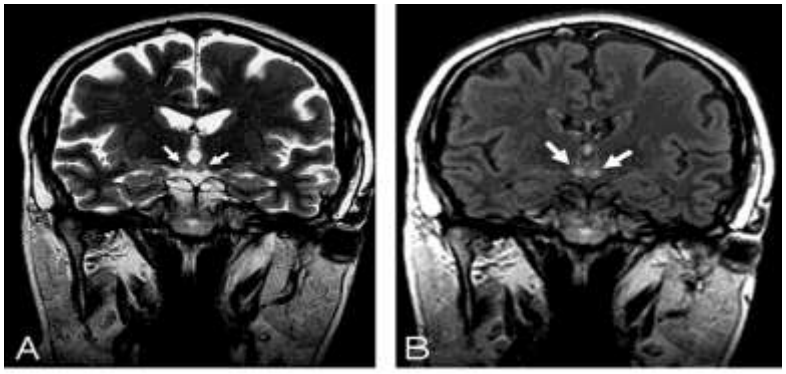

Figure 1: Coronal T2-Weighted (A) and Coronal FLAIR Images (B) Show High Signal Intensity Circumscribed to the Mamillary Bodies (White Arrows).

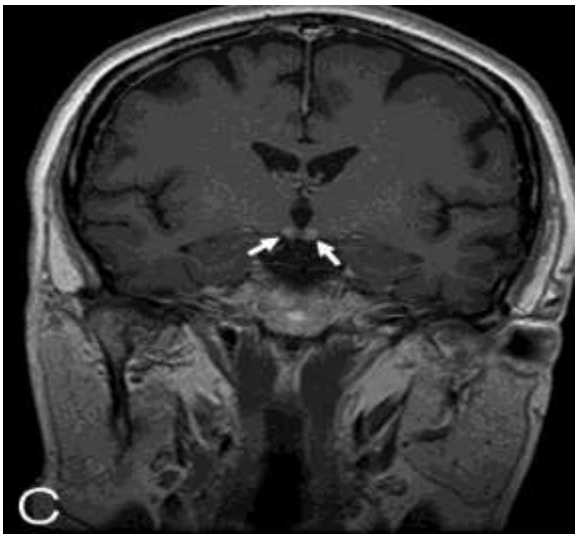

Figure 2: After Administration of Contrast Media, Central Enhancement (White Arrows) of Both Mamillary Bodies is seen on Coronal T1-Weighted Image (C).

A clinical diagnosis of Wernicke's encephalopathy was made, based on the patient's presentation and laboratory findings.

Management

The patient was immediately started on intravenous thiamine (500 mg TID for 3 days), followed by oral thiamine supplementation. Supportive care, including hydration, nutrition, and electrolyte correction, were also provided. Nutritional rehabilitation and counseling for alcohol cessation were also provided.

Outcome

Within 48 hours, the patient's confusion began to improve, and he was able to recall basic information. After a week of treatment, the ophthalmoplegia improved significantly, and he regained near-normal gait stability. He was discharged with oral thiamine supplementation and counseling on the importance of a balanced diet and alcohol cessation.

DISCUSSION

Wernicke's encephalopathy can be underdiagnosed, especially in resource-poor settings, where advanced neuroimaging and specialized laboratory tests may not be readily available. The classic triad of mental confusion, ataxia, and ophthalmoplegia is not always present, further delaying diagnosis. In rural hospitals, a high index of suspicion based on clinical signs such as confusion, ataxia, and ophthalmoplegia, coupled with a history of alcoholism or malnutrition, is crucial for timely diagnosis.[2,3] Thiamine deficiency impairs energy metabolism in the brain, leading to selective vulnerability of brain regions such as the mammillary bodies, thalamus, and periaqueductal gray matter. Early thiamine supplementation can reverse symptoms and prevent progression to Korsakoff's psychosis or death.[4] Thiamine supplementation remains the cornerstone of treatment. Management involves high-dose intravenous thiamine, usually 500 mg three times daily for 2-3 days, followed by oral thiamine.[4] Delayed or missed

treatment can lead to poor outcomes and permanent neurological sequelae.[5] It is important to initiate therapy early to prevent irreversible brain damage and other sequelae like Korsakoff syndrome. While the full triad of symptoms (confusion, ataxia, ophthalmoplegia) is seen in only about 20% of cases, prompt intervention significantly improves outcomes.[4,5]

CONCLUSION

Wernicke's encephalopathy should be considered in any patient presenting with neurological symptoms, particularly in individuals with a history of chronic alcoholism or malnutrition. Rural tertiary hospitals, although often limited in resources, can still play a critical role in the timely diagnosis and treatment of this condition, significantly improving patient outcomes.

REFERENCES

1. Sechi G, Serena C. Wernicke's encephalopathy: new clinical settings and recent advances in therapy. Lancet Neurology. 2007; 6(5):442-448.
2. Thomson AD. Clinical presentations of Wernicke's encephalopathy: A 21st century update. J Neurol Neurosurg Psychiatry. 2018; 89(4):315-323.
3. Litten RZ, Moss HB. Alcohol use disorders and the global burden of disease: An overview. Alcohol Research: Current Reviews. 2013; 35(1):57-61.
4. Galvin R, Brathen G, Ivashynka A, et al. EFNS guidelines for diagnosis, therapy, and prevention of Wernicke's encephalopathy. European Journal of Neurology. 2010; 17(12):1408-1418.
5. Oudman E, Wijnia JW, Oey MJ, et al. Preventing Wernicke encephalopathy in alcoholism: A randomized controlled trial. Alcohol and Alcoholism. 2018; 53(2):112-120.