

An Insight to Wernicke Encephalopathy

Dr. Blessy Rachal Boban, Dr. Sherin Alexander

Department of Clinical Pharmacy, Believers Church Medical College Hospital, Thiruvalla, Kerala, India

ABSTRACT

Wernicke encephalopathy (WE) is an acute neurological condition with several manifestations. It is characterized by a clinical triad with confusion, ataxia and nystagmus. There are different causes of WE and the prime cause is the deficiency of thiamine due to chronic alcoholism. Here we discuss a case of Wernicke encephalopathy and in our case, thiamine deficiency is the etiology.

KEYWORDS: *Wernicke encephalopathy, clinical triad, thiamine deficiency, ataxia, confusion, nystagmus*

How to cite this paper: Dr. Blessy Rachal Boban | Dr. Sherin Alexander "An Insight to Wernicke Encephalopathy" Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN: 2456-6470, Volume-5 | Issue-3, April 2021, pp.996-997, URL: www.ijtsrd.com/papers/ijtsrd41100.pdf



Copyright © 2021 by author (s) and International Journal of Trend in Scientific Research and Development Journal. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY 4.0) (<http://creativecommons.org/licenses/by/4.0>)



INTRODUCTION

Wernicke encephalopathy (WE) is a life-threatening, neurological condition characterized by a clinical triad of ophthalmoparesis (paralysis of one or more extra ocular muscles) with confusion, nystagmus (involuntary eye movements) and ataxia (lack of muscle control or coordination of voluntary movements), caused by thiamine (Vitamin B1) deficiency, which primarily affects peripheral and central nervous system. Various causes of WE include thiamine deficiency due to chronic alcoholism, liver disease, severe malnutrition, malignancies, prolonged parenteral nutrition, immunodeficiency syndromes and severe anorexia nervosa.¹ Other symptoms and signs that have been described in Wernicke encephalopathy include irritability, tachycardia, hypotension, hypo or hyperthermia, hearing loss, seizures, spastic paraparesis, delirium, coma, acute psychosis, miosis, anisocoria (unequal pupil size), papilloedema and retinal haemorrhages.²

The prevalence of the disease is slightly higher in male population than females with ranges from 30 to 70 years.^{3,4} WE in non-alcoholics usually presents as a dramatic acute syndrome, whereas WE in alcoholics may more frequently present as a subclinical syndrome.⁵

CT scanning is not a reliable test for WE. MRI is a powerful tool which should be used to support the diagnosis of acute WE both in alcoholics and non-alcoholics. It could also be used to follow the recovery of patients.⁶

Thiamine administration can improve symptoms to a certain extent, especially if administered promptly. It is also important to give thiamine before any carbohydrate, because it is well known that glucose infusion precipitates WE in thiamine deficiency.⁷ The preferred dose of thiamine is 500 mg one to three times daily parenterally. Malnourished

patients need higher doses of thiamine. Many WE patients should encourage abstinence from alcohol.⁸

CASE REPORT

This was a case in Believers Church Medical College Hospital, Thiruvalla. A 72 year old male patient presented with complaints of giddiness, altered sensorium and heavy breathing. On examination, he was found to have pallor, palmar erythema, icterus and ascites.

The patient was apparently alright till 7th June 2020, then he suddenly developed giddiness and loss of vision. He also had altered sensorium, labored breathing and two episodes of urinary incontinence. He was binge drinking for the past four days of about 750 ml. He was taken to a local hospital and CT brain was done. CT showed cystic hygroma and brought to Believers Church Medical College Hospital for further management. He had a past medical history of CHILD C, Chronic Liver Disease. He was a chronic alcoholic and a reformed smoker. He had no history of trauma or snake bite. On initial examination, his vitals were Blood Pressure - 180/90mmHg, pulse – 92beats per minute, Respiratory Rate - 15bpm, SPO₂ – 99%. He was found to have deranged blood reports (Total count:14800 cells/mcL, C Reactive Protein :13.5 mg/L, Platelet: 0.52 lakh/mcl, AFP:9.65 IU/ml, Total bilirubin:6.33 mg/dl, Indirect bilirubin:4.36 mg/dl, SGOT/SGPT: 136/89 U/L, Albumin : 2.77 g/dl, ALP: 133 U/L, INR:1.49, ammonia: 199 mmol /l and Creatinine:0.55 mg/dl).

MRI brain was done which showed the possibility of Wernicke's encephalopathy and the presence of chronic small vessel ischemic changes. Frequent epileptiform abnormalities arising from both hemispheres was mentioned in EEG. USG and CECT showed chronic liver

disease with portal hypertension, portosystemic collaterals, prostatomegaly, bilateral inguinal hernia and cardiomegaly. In view of poor Glasgow Coma Score (7/15), he was intubated and kept him on mechanical ventilation. General medicine & neurology consultation was sought to rule out other causes of altered sensorium. Medicine consultant opined as hepatic/Wernicke's encephalopathy and Neurology consultant advised MRI brain which showed possibility of Wernicke's encephalopathy to be considered. High dose of Injection. Thiamine 200mg BD was started as advised by a Neurologist. On the 3rd day of admission, altered sensorium improved gradually and he was extubated. Psychiatry consultation was sought in view of restlessness and advised Injection. Lorazepam 1 mg if needed. Surveillance Endoscopy was done on 18th June 2020 to rule out varices which showed only the antral gastritis. He was treated with Tab. Ursodeoxycholic acid 300mg two times a day, Injection. Pantoprazole 40mg two times a day, Injection. Piperacillin-Tazobactam 4.5g every sixth hourly, Tab. Rifaximin 550mg two times a day, Tab. Zinc acetate once a day, Tab. Ademetionine 400mg three times a day, Tab. Calcium carbonate+vitamin D3 500mg once a day, Tab. Melatonin 3mg at night, Tab. Levetiracetam 750mg two times a day, Syrup. Lactulose 30ml two times a day, Injection. Thiamine 500mg three times a day. He became stable and hence being discharged.

DISCUSSION

Our patient had a typical clinical triad of Wernicke's Encephalopathy which was confirmed using MRI scan. Etiology of this case is thiamine deficiency due to chronic alcoholism. Thiamine replacement and supportive measures were done.

Talib et al⁹ reported a case on Wernicke Encephalopathy due to Hyperemesis Gravidarum in Pregnancy. This condition usually occurs in pregnant women who are malnourished and hyperemesis further depletes thiamine stores. In this case report, it was a 35 years old female with 16 weeks of gestation. Thiamine supplementation was done and hence her condition improved.

Zhao et al¹⁰ reported a case of Wernicke Encephalopathy in 61 year old liver cirrhosis male patient, without hepato cellular carcinoma or operative intervention. Condition diagnosed using MRI scan and thiamine was initiated. But the patient died of bleeding due to rapid deterioration of liver function.

UdyavaraKudru C, et al¹¹ also reported a case of Wernicke's encephalopathy in a patient with gastric carcinoma. Patient is a 35 year old man. CT of brain and CSF analysis were normal. MRI of brain suggestive of WE. CT of abdomen showed gastric outlet obstruction with liver metastasis. Upper gastroduodenoscopy showed ulcerative lesions involving the antrum, pylorus and duodenum and biopsy revealed adenocarcinoma.

CONCLUSION

Wernicke's Encephalopathy can develop in any individual, even in well-nourished individuals, who are benefit of a thiamine source for more than a few weeks and possess any of the underlying conditions. Furthermore, in light of the varied and often non-specific presentation, the suspicion of WE should always be at the back of our minds in the presence of any one of the classic signs and symptoms. Once the diagnosis of WE is ascertained on the basis of clinical presentation, thiamine supplementation takes priority over laboratory evaluation.

ACKNOWLEDGMENT

We would like to thank Dr. Aleena Paul, Clinical specialist, Department of Gastroenterology, Believers Church Medical College Hospital for the valuable suggestions during the progression of the case report. We would also thank our patient, his caregivers and hospital authority for the co-operations.

CONFLICT OF INTEREST

The authors declare that there are no conflicts of interests.

ABBREVIATIONS

WE: Wernicke Encephalopathy; **CT:** Computed Tomography; **CSF:** Cerebrospinal Fluid; **MRI:** Magnetic Resonance Imaging; **CRP:** C Reactive Protein; **INR:** International Normalized Ratio; **EEG:** Electroencephalogram; **CECT:** Contrast-Enhanced Computed Tomography; **ALP:** Alkaline Phosphatase; **AFP:** Alpha Fetoprotein.

REFERENCES

- [1] Rebello P, Nayak SR, Rao PP, D'Souza GL, Memon UJ, Toro AM, Joe A, Yadiyal A. Triad of Wernicke's Encephalopathy in an Alcohol Withdrawal Patient-A Case Report.
- [2] Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. *Journal of Neurology, Neurosurgery & Psychiatry*. 1986 Apr 1; 49(4):341-5.
- [3] Goodwin DW. The Wernicke-Korsakoff Syndrome: A Clinical and Pathological Study of 245 Patients, 82 with Post-Mortem Examinations. *JAMA*. 1972 Jan 17; 219(3):389-.
- [4] Adams RD. Principles of neurology. McGraw-Hill, Health Professions Division; 1997 Jul 1.
- [5] Galvin R, Bräthen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. *European Journal of Neurology*. 2010 Dec; 17(12):1408-18.
- [6] Antunez E, Estruch R, Cardenal C, Nicolas JM, Fernandez-Sola J, Urbano-Marquez A. Usefulness of CT and MR imaging in the diagnosis of acute Wernicke's encephalopathy. *AJR. American journal of roentgenology*. 1998 Oct; 171(4):1131-7.
- [7] Sechi G, Serra A. Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. *The Lancet Neurology*. 2007 May 1; 6(5):442-55.
- [8] Onishi H, Ishida M, Uchida N, Shintani D, Nishikawa T, Hasegawa K, Fujiwara K, Akechi T. Subclinical thiamine deficiency identified by preoperative evaluation in an ovarian cancer patient: Diagnosis and the need for preoperative thiamine measurement. *Palliative & supportive care*. 2019 888Oct; 17(5):609-10.
- [9] Talib V, Sultana S, Hamad A, Yaqoob U. Wernicke encephalopathy due to hyperemesis gravidarum in pregnancy: a case report. *Cureus*. 2018 Jul; 10(7).
- [10] Zhao P, Zhao Y, Wei Z, Chen J, Yan L. Wernicke encephalopathy in a patient with liver failure: Clinical case report. *Medicine*. 2016 Jul; 95(27).
- [11] Kudru CU, Nagiri SK, Rao S. Wernicke's encephalopathy in a patient with gastric carcinoma: a diagnosis not to miss. *Case Reports*. 2014 Mar 20; 2014.