Case Report

Wernicke's encephalopathy: a case report

Varsha S. Dawani[1], Manju Khanchandani[2]

[1]Consultant Psychiatrist, Shewi's Health Care Centre, Ulhasnagar.
[2]Consultant Physician, Shewi's Health Care Centre, Ulhasnagar.
Corresponding author: Varsha Dawani
Email: varshadawani@yahoo.com

ABSTRACT

Wernicke's encephalopathy (WE) is an acute neuropsychiatric condition that occurs due to reversible biochemical lesions caused by depletion of intracellular Thiamine (Vitamin B1). WE is characterized by the triad of ophthalmoplegia, ataxia, and confusion. However only 10 % of patients present with all three features and other symptoms may also be present. It is commonly regarded as a condition occurring due to malnourishment due to alcohol use but a variety of other diseases can also lead to it. It is present in the general population with a prevalence of around 2%. We present herewith a case of a 72 years old lady that presented with WE.

Key words: Wernicke's encephalopathy, thiamine, malnourishment.

(Paper received – 23rd March 2019, Peer review completed – 6th April 2019)
(Accepted – 10th April 2019)

INTRODUCTION

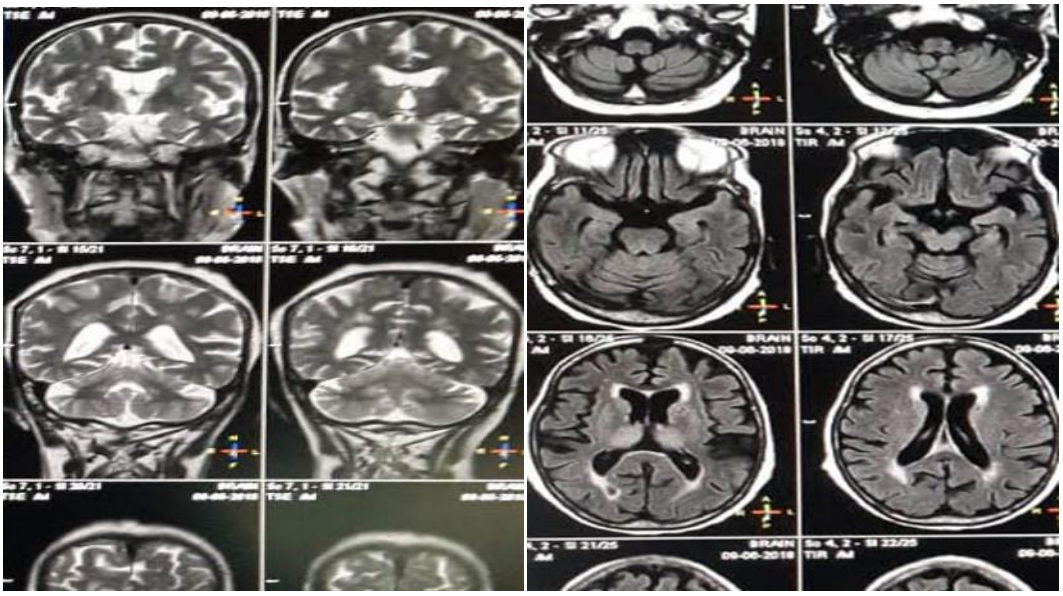
WE is caused by a thiamine deficit leading to disturbances in cerebral glucose metabolism. Typical early symptoms include oculomotor deficits (frequently bilateral abduction deficits due to 6th nerve palsies, and vertical/horizontal nystagmus), ataxia and behavioral/mental disturbances [1]. The sudden onset of symptoms raises the question of other underlying etiologies, especially of brain ischemia, revealing the absence of highly sensitive diagnostic tools in WE and the presence of the diagnostic gap between transient ischemic attack and abnormalities in diffusion-weighted imaging (DWI) in MRI [2]. It is important to note that WE may present due to a variety of causes that may be non-alcoholic in nature and is seen in gastroenterology and post gastric surgery as well [3-4]. The common presentations of WE have been noted in the elderly [5] and alcohol dependent patients [6] and it is noteworthy that the classical triad of ophthalmoplegia, ataxia and confusion is seen in just 10% cases while many unusual presentations have been reported with malnutrition being a predominant factor [7-8].

CASE REPORT

A 72 years old lady referred to the psychiatrist with history of sudden onset change in behavior since 3- 4 days with complaints of confusion, staring spells, not talking, decreased appetite, unable to walk or maintain balance. On enquiry patient and relatives gave history of and investigated for low hemoglobin and hypoproteinemia since 3 to 4 months for which patient received blood transfusion, intravenous iron preparation and multivitamins, intravenous albumin. One week prior to presentation, the patient developed axillary lymph nodes and Fine needle aspiration Cytology (FNAC) was done. FNAC results were suggestive of TB and the patient was started Anti TB medication. Hence when patient was referred for a psychiatry evaluation, INH induced psychosis was suspected and she was admitted for detailed work up. On admission, her vital parameters were stable but was not cooperative for detailed mental status

examination (MSE). On MSE the patient had poverty of speech, staring spells, was grossly oriented in time, place and person. The above symptoms were of acute onset since the past 2 days. In the ward, on day one patient developed tremors and twitching of whole body especially arms. Blood investigations done were within normal limits. A magnetic resonance imaging study of the brain was done and revealed symmetric areas of diffusion restriction and altered signal in both thalami, around the 3rd ventricle and aqueduct and both mammillary bodies were noted. The above radiological findings were suggestive of WE and the patient was started on high doses of vitamin B1 and B12 both intravenous as well as orally. The patient improved in 3 days of treatment and was discharged on 6th day on Anti TB medication, thiamine and iron supplements. She has been following up and has been doing well.

Figure – MRI images of the patient



DISCUSSION

WE is clinically diagnosed, since there are no highly sensitive tools to prove this pathological entity. A low serum level of thiamine and a low erythrocyte transketolase activity can support the diagnosis but lack sensitivity and may not be available immediately. Suggestive MRI findings (bilateral lesions around the third/fourth ventricle, in the medial parts of the thalamus, corpora mamillaria, mesencephalon, tegmentum and periaqueductal) can strengthen the diagnosis, but sensitivity only amounts to about 50% [9]. Thiamine pyrophosphate is an essential coenzyme for cerebral glucose metabolism and involved in the regulation of the osmotic gradient across the cell membranes. Functional loss of cortical cells during lack of thiamine is attributed to disturbances in metabolism of glucose, local accumulation of lactate, pyruvate and alanin leading to local acidosis [10] and extra-/intracellular edema that may lead to demyelinisation, glial proliferation, hemorrhagic lesions and necrosis at the WE-typical lesion site. WE is an important condition and clinicians must be aware of the same when patients who are malnourished present with neurological problems.

REFERENCES

1. Sechi G, Serra A. Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. Lancet Neurol 2007;6(5):442-55.
2. Galvin R, Bråthen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. Eur J Neurol 2010;17(12):1408-18.

3. Zuccoli G, Pipitone N. Neuroimaging findings in acute Wernicke's encephalopathy: review of the literature. Am J Roentgenol 2009;192(2):501-8.
4. Thomson AD, Marshall EJ. The natural history and pathophysiology of Wernicke's encephalopathy and Korsakoff's psychosis. Alcohol Alcohol 2005;41(2):151-8.
5. Fei GQ, Zhong C, Jin L, Wang J, Zhang Y, Zheng X, Zhang Y, Hong Z. Clinical characteristics and MR imaging features of nonalcoholic Wernicke encephalopathy. Am J Neuroradiol 2008;29(1):164-9.
6. Weidauer S, Nichtweiss M, Lanfermann H, Zanella FE. Wernicke encephalopathy: MR findings and clinical presentation. Eur Radiol 2003;13(5):1001-9.
7. Jung YC, Chanraud S, Sullivan EV. Neuroimaging of Wernicke's encephalopathy and Korsakoff's syndrome. Neuropsychol Rev 2012;22(2):170-80.
8. Lindberg MC, Oyler RA. Wernicke's encephalopathy. Amer Fam Physician 1990;41(4):1205-9.
9. Naidoo DP, Bramdev A, Cooper K. Wernicke's encephalopathy and alcohol-related disease. Postgrad Med J 1991;67(793):978-81.
10. Wilson RK, Kuncl RW, Corse AM. Wernicke's encephalopathy: beyond alcoholism. Nat Rev Neurol 2006;2(1):54-8.

****

Acknowledgements – Nil
Source of Funding – Nil
Conflict of Interest – Nil