International Journal of Research and Review
Vol.7; Issue: 5; May 2020
Website: www.ijrrjournal.com
Case Report *E-ISSN: 2349-9788; P-ISSN: 2454-2237*

Triad of Wernicke's Encephalopathy in an Alcohol Withdrawal Patient - A Case Report

Preethi Rebello[1], Sriraksha R Nayak[2], Pavithra P Rao[1], Galina Lisa D'Souza[2], Uzair Jawed Memon[2], Asmita Mukund Toro[2], Avinash Joe[3], Aruna Yadiyal[4]

[1]Assistant Professor, [2]Post Graduate Resident, [3]Senior Resident, [4]Professor,
Department of Psychiatry, Fr Muller Medical College, Kankanady, Mangalore - 575002, Karnataka.

Corresponding Author: Pavithra P Rao

ABSTRACT

Wernicke's encephalopathy (WE) is an acute neurological condition with psychiatric manifestations characterized by the classic triad of confusion, ophthalmoplegia and ataxia. The presence of all features of the classic triad in one patient is uncommon and not present in up to 90% of patients with WE. Early diagnosis and management of WE is important for better outcome and improved quality of life in these patients. This case reports a rare presentation of all the symptoms of the triad of WE in one patient with Alcohol Dependence Syndrome.

Key words: Wernicke's Encephalopathy, Ataxia, Ophthalmoplegia, Global Confusion

INTRODUCTION

Wernicke's Encephalopathy (WE) is a neurological condition which is acute in onset. It is usually characterized by a clinical triad of ophthalmoparesis, ataxia, and confusion. It is a disorder which is caused by deficiency of Vitamin B1 which is thiamine and it affects central as well as the peripheral nervous system. As it can be a life threatening illness it requires adequate and early diagnosis and management. [(1-2)] Patients with Alcohol dependence syndrome, malnutrition, cancer should be suspected with WE if they present with one or more than one of the characteristic symptoms of ophthalomoplegia, ataxia and altered mental status. Apart from the above mentioned causes of WE, hyperemesis gravidarum, prolonged parenteral nutrition, immunodeficiency syndromes, liver disease, hyperthyroidism, and severe anorexia nervosa are some of the other disorders which can present with symptoms of WE. The pathogenesis of WE in all being the same, that is thiamine deficiency due to impaired absorption of thiamine from the intestine, a possible genetic predisposition, inadequate diet, reduced storage of thiamine in the liver, and other nutritional deficiencies. [(3-5)]

The major part of the process of reaching a diagnosis of WE is based on the history and examination of the patient, which includes both physical and mental status examination. Though the classic triad of WE is altered mental status, ataxic gait, and ophthalmoplegia, a definitive diagnosis is complicated as the clinical triad may not be present in up to 90% of patients. This is a case report which presents a patient with all the symptoms of the triad of WE and hence is unique as it is a very uncommon presentation.

CASE DESCRIPTION

47 year old, married male from a lower socioeconomic status was brought to the outpatient department with history of Alcohol consumption since past 10 years and meeting criteria for Alcohol dependence syndrome. Patient's last drink was reported as being 20 days before presenting to the hospital. He had stopped alcohol after multiple episodes of vomiting and was treated for gastritis and given intravenous

fluids by a local physician. After 3 days of stopping alcohol he had gone into withdrawal delirium with history suggestive of Visual and auditory hallucinations, fluctuation of consciousness, decreased sleep, fearfulness and increased activity. Patient's wife also noticed that he was having difficulty in walking and was unstable requiring assistance. This was noticed 10 days after stopping alcohol. As he did not improve within 1 week like his past withdrawal episodes and as this episode had an added symptom of instability of gait family members brought him for admission and further management. A detailed History was taken and there was no history of other medical co-morbidities.

On general physical examination, his vitals were stable and BMI (Body Mass Index) was 15.24 kg/m^2. He was noticed to have dry skin, angular stomatitis. Central nervous system examination revealed a horizontal nystagmus and staggering gait requiring help from family members. On mental status examination he was having fluctuation of consciousness, disorientation to time and place. There was presence of Visual, auditory hallucinations and delusion of persecution. His remote memory was intact but there was confabulation with telescoping on recent memory assessment. Other systems were within normal limits.

On blood investigations his Complete Blood counts, Renal function test, Thyroid function test were normal. He had elevated liver enzymes and was non reactive for immunocompromised states. His ultrasound abdomen showed grade II fatty liver. Electroencephalogram was normal.

MRI (Magnetic Resonance Imaging) brain was done and it showed classical features of WE i.e. T2/ FLAIR hyperintensities with restricted diffusion involving periaqueductal grey matter, dorsomedial thalami (left > right), around III ventricle, mamillary bodies, colliculi.

Based on clinical examination with the presence of the rare triad along with corroboration with the neuroimaging finding of classical characteristics of WE, a diagnosis of Wernicke's encephalopathy and Alcohol dependence syndrome withdrawal delirium (resolving) was made.

Patient was started on Parenteral Thiamine supplementation for management of WE and thereafter continued on oral thiamine along with maintenance of adequate hydration. Perceptual abnormalities were managed with low dose antipsychotics with Benzodiazepines for managing sleep disturbance. Once patient's delirium features resolved, gait and balance physiotherapy was given. Patient and family were Psychoeducated to improve the insight into illness and expressed emotions were addressed. Sleep and appetite improved initially and perceptual abnormalities subsequently. Minimal ataxia, nystagmus and confabulation persisted at the time of discharge.

CASE DISCUSSION

WE has enormous morbidity and mortality risk. Overlap of WE symptoms of ataxia and confusion with alcohol withdrawal features lead to difficulty in differentiating between the two. But the presence of nystagmus and MRI features pointing towards WE helped in diagnosis and early management of the WE, leading to better improvement and lesser morbidity. The severity of Alcohol dependence, Vomiting and anorexia and delay in seeking help led to the worsening of WE symptoms in this patient. The presence of the triad which is uncommon helped in faster diagnosis of the disorder and the result of the MRI brain done, further confirmed the diagnosis and helped in initiating the treatment of the disorder at the earliest. Delay in recognition and treatment of WE can worsens prognosis leading to permanent brain damage, long-term institutionalization and even death. [(6)] Early recognition and appropriate management with parenteral thiamine helps improve prognosis and ensures better quality of life. After thiamine is administered parenterally, the first to improve is global confusion while ataxia and nystagmus may remain for quite a

while. A number of patients with WE will continue to have ataxia, confabulation and ophthalmoplegia for a long time and this causes problems in their socio-occupational functioning and decreases their quality of life along with causing care giver burden on family members attending to them. There are some reports which indicate that there is premature mortality in patients with this disorder but there is a lack of long term studies regarding the same. [(7-9)]

The presence of all the symptoms of triad is reported to be in the range of only 10 to 16 % of patients of WE. [(1,10)] This case report is unique in reporting an uncommon presentation of all the symptoms of the triad of WE in one patient.

CONCLUSION

WE is a neuropsychiatric manifestation with the classic triad which is acute in onset caused by severe thiamine deficiency. It is a life threatening disorder with poor outcome in number of patients. Early diagnosis and management is one of the measures to decrease the high morbidity and mortality risk associated with the disorder. Maintaining a high degree of suspicion, and a thorough neurological examination, in patients presenting with disorders which cause WE will go a long way in early recognition and appropriate management of the WE and hence increasing the quality of life in such patients.

REFERENCES

1. Vasan S, Kumar A. Wernicke Encephalopathy. [Updated 2019 Nov 16]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470344/
2. Karakonstantis S, Galani D, Korela D, Maragou S, Arna D, Basta M. Missing the early signs of thiamine deficiency. A case associated with a liquid-only diet. Nutr Neurosci. 2018 Aug 09;1-3
3. Oudman E, Wijnia JW, Oey MJ, van Dam MJ, Postma A. Preventing Wernicke's encephalopathy in anorexia nervosa: A systematic review. Psychiatry Clin. Neurosci. 2018 Oct;72(10):774-779
4. Shabbir S, Tong O, Gluck L, Robbins M. Convergence Spasm in Wernicke Encephalopathy. Neurohospitalist. 2018 Jan;8(1):NP1-NP2
5. Tang L, Alsulaim HA, Canner JK, Prokopowicz GP, Steele KE. Prevalence and predictors of postoperative thiamine deficiency after vertical sleeve gastrectomy. Surg Obes Relat Dis. 2018 Jul;14(7):943-950
6. Flynn A, Macaluso M, D'Empaire I, Troutman MM. Wernicke's Encephalopathy: Increasing Clinician Awareness of This Serious, Enigmatic, Yet Treatable Disease. Prim care companion CNS Disord. 2015;17(3). doi: 10.4088/PCC.14r01738. eCollection 2015.
7. Nakamura ZM, Tatreau JR, Rosenstein DL, Park EM. Clinical Characteristics and Outcomes Associated With High-Dose Intravenous Thiamine Administration in Patients With Encephalopathy. Psychosomatics. 2018 Jul - Aug;59(4):379-387
8. Sanvisens A, Zuluaga P, Fuster D, Rivas I, Tor J, Marcos M, Chamorro AJ, Muga R. Long-Term Mortality of Patients with an Alcohol-Related Wernicke-Korsakoff Syndrome. Alcohol Alcohol. 2017 Jul 01;52(4):466-471
9. Zahr NM, Kaufman KL, Harper CG. Clinical and pathological features of alcohol-related brain damage. Nat Rev Neurol. 2011 May;7(5):284-94
10. Harper C.G., 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 and Psychiatry. 1986; 49 :341-5

How to cite this article: Rebello P, Nayak SR, Rao PP et.al. Triad of Wernicke's encephalopathy in an alcohol withdrawal patient - a case report. International Journal of Research and Review. 2020; 7(5): 243-245.

******