Wernicke's Encephalopathy with Normal Neuroimaging - Suspect and Treat - A Case Report

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INTRODUCTION

Wernicke's encephalopathy (WE) is an unrecognized nutritional deficiency which often goes unnoticed. WE is clinically often composed of a triad including nystagmus, ophthalmoplegia and altered mental status. Although this triad practically is present only in a handful of cases[1] it is also described as an acute neuropsychiatric presentation of thiamine deficiency. Early diagnosis and prompt treatment are of utmost importance here as it can prevent chronic brain damage which is often the end effect of thiamine deficiency.

Wernicke's encephalopathy is most commonly found in patients with chronic alcoholism, less frequent in non-alcoholic patients. In non-alcoholic patients, Wernicke's encephalopathy might develop due to erosion of upper portion of gastrointestinal tract or secondary to intractable vomiting, inadequate dietary intake or malabsorption. Other causes include malignancies (gastric cancer, leukaemia, lymphoma), hyperemesis, anorexia, thyroid conditions.[1,2]

Wernicke's encephalopathy is caused due to thiamine (B1) deficiency. B1 is a water-soluble vitamin which acts as a co-factor for carbohydrate metabolism. It is also important for neuronal cell function.[2] This vitamin can't be synthesised in the human body and thus dietary intake play a very important role.

Symptoms of thiamine deficiency Include - Nystagmus, ataxia, encephalopathy, mental confusion. Early onset includes symptoms like: - headache, irritability, fatigue and abdominal discomfort. Prophylactic thiamine supplementation forms a major treatment for patients at risk for developing refeeding syndrome (RFS).
RFS is an underdiagnosed condition which is characterised by potential shift in the fluid and electrolytes.[3]

PRESENTATION OF CASE

A 26-year-old male patient came with complain of vomiting. Patient was a chronic alcoholic since 15 years drinking around 200 ml alcohol daily. There was history of slurred speech and generalized weakness and swaying on either side while walking since 10 days and relatives also gave history of altered sensorium since 2 days.

There was no history of any psychiatric illness or medication history from the past. On examination, patient was emaciated, patient was conscious and confused, not oriented to time, place and person. His blood pressure was 100/60 mmHg, pulse 92/min. When checked for ocular movements, ophthalmoplegia was present as shown in image 1 and 2.

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Magnetic resonance imaging (MRI)of brain was done for this patient which showed no any abnormality. Even though MRI findings of Wernicke's encephalopathy were not present on MRI, classical triad of confusion, ataxia and ophthalmoplegia and clinical improvement in patient after thiamine administration was observed. Thus, patient diagnosis of Wernicke's encephalopathy was made, and treatment was started accordingly. Patient was treated with IV antibiotics and injection thiamine and showed dramatic improvement in 5 days course in hospital. He was later discharged.

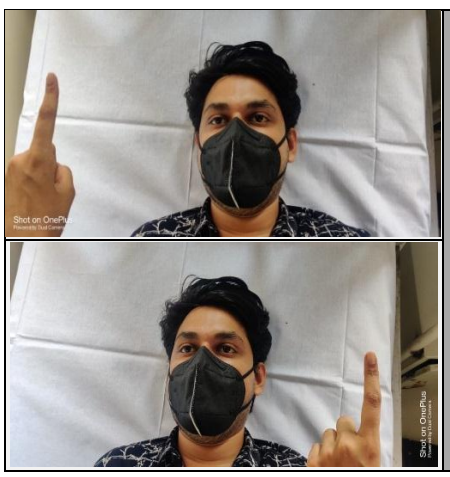

Figure 1 and 2 Showing Bilateral Lateral Rectus Palsy Suggestive of Ophthalmoplegia

DISCUSSION

Nutritional disorders are very common in a country like India where malnutrition is so rampant. Amongst the nutritional disorders, vitamin deficiencies are very common. Wernicke's encephalopathy is an uncommon nutritional disorder caused due to deficiency of vitamin b1 called thiamine. Although diagnosis of such a condition is often missed, patient can show dramatic improvement when prompt treatment is initiated.[4]

Wernicke's encephalopathy can be a life-threatening condition following acute deficiency of thiamine. Thiamine acts as a cofactor for various enzymes in Krebs cycle as well as the pentose phosphate pathway. It also plays a vital role in carbohydrate metabolism. Any deficiency or malfunctioning of this vitamin can lead to accumulation of various intermediate products of the various pathways.[5] These toxic intermediates can have serious effects on the body including the brain.

The neuropsychiatric features of thiamine deficiency ranges from nystagmus, ophthalmoplegia, confusion, gait disturbances and various mental changes. These mental changes are owing to the accumulation of toxic intermediate metabolites. If untreated can often lead to deterioration and then to coma and even death at times. These changes in the brain are characteristic and usually discovered post autopsy as diagnosis of Wernicke's encephalopathy is often missed. The characteristic changes are often seen in the mamillary or thalamic nuclei which leads to episodes of acute confusion, mental sluggishness, apathy sometimes coma. The ophthalmological changes include nystagmus, bilateral recti palsy and conjugate gaze palsy.[6] As early diagnosis is of utmost importance, there are no specific investigations other than estimating blood thiamine levels or measuring.

Erythrocyte transketolase activity. The most accurate diagnosis is obtained by MRI. MRI shows symmetric involvement of the mammillary bodies, tectal plate, the periaqueductal grey matter, periventricular region and others. Signal hyper intensities on T2 sequences along with fluid attenuated inversion recovery (FLAIR) and diffusion weighted images within posteromedial thalami and third ventricle are the most common abnormalities detected on MRI.[7] According the literature available, MRI diagnostic sensitivity is poor, hence many patients with Wernicke's encephalopathy may not present with diagnostic findings on MRI, our case also did not have any diagnostic findings on MRI and diagnosis was purely made clinically. As complex as this disorder may sound, the treatment option is very simple, that is administration of intravenous thiamine. A recommended regimen allows administration of around 500 mg of thiamine IV over 30 minutes, 8 hourly for 2 consecutive days followed by 500 mg once daily for 5 days along with administration of other essential vitamin B supplement.[8,9]

CONCLUSIONS

As MRI has poor sensitivity to diagnose Wernicke's encephalopathy, diagnosis can be made clinically, and MRI should just be used to confirm the diagnosis if needed. Although Wernicke's encephalopathy may appear diagnostically challenging, it's mere simple treatment can be lifesaving and prevent fatal complications.

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