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Case Report

Wernicke's Encephalopathy Due to Hyperemesis Graviderum : A Rare Presentation

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Abstract

Wernicke's encephalopathy is a common and preventable acute neurological symptom due to deficiency of thiamine. Alcoholism also causes Thiamine deficiency. It can occur in non-alcoholic conditions such as prolonged starvation, hyperemesis graviderum, bariatric surgery, HIV, AIDS. The characteristics clinical triad of disease is ophthalmoplagia,ataxia & global confusion. Here a patient was studied who was diagnosed on M.R.I as Wernicke's encephalopathy.

Key words: Encephalopathy; Ataxia; Vomiting; MRI.

INTRODUCTION

Wernicke's encephalopathy is also known as Wernicke's disease. It occurs due to exhaustion of vitamin reserves. Thiamine requirement is increased in pregnancy especially in hyperemesis graviderum, in high glucose intake. So its depletion due to less intake or increase administration of glucose without thiamine may result in Wernicke's Encephalopathy. Here Neurological symptoms are due to lesions of central nervous system. It is characterized by ophthalmoplagia ,ataxia & confusion. Overall incidence is about 2%. Only small percentage of patients experience all these 3 symptoms. Usually all symptoms are found in over use of alcohol. Thiamine deficiency is associated with alcoholism because it affects thiamine uptake as well as its utilization. Other symptoms are also found like amblyopia, hearing loss, dysphagia, hypothermia, cardio circulatory dysfunction. Lack of thiamine also affects major energy consumer myocardium and patient may develop cardiomegaly, heart failure. Cardiac abnormalities are one of the most important trigger of death in Wernicke's encephalopathy[1-3].

CASE REPORT

A patient of 28 years having 1 child delivered by C-Section was admitted in a private clinic with 12 weeks amenorrhea, vertigo, vomiting, and history of 10% glucose replacement at home. She had repeated ante natal checkup for vertigo & vomiting, she was also advised for admission. This was her 1st time admission in hospital so treatment was started with fluid replacement for correction of dehydration, mantainence of hyperemesis chart and with some special investigations like Serum electrolytes where Hypokalemia was diagnosed and treated accordingly. But the

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patient did not improve rather became drowsy and developed nystigmus within 2 days of admission. So medicine department was consulted. Patient was shifted to H.D.U for restlessness, slurring of speech & irrelevant behavior with nystigmus in all directions. Hence the patient was deteriorating, a medical board was arranged among gynecologist, physician, neurologist & psychiatrist. The case was concluded as pregnancy with brain stem lesion & hypokalemia with a advice for M.R.I of brain. The patient was kept in N P O. with all symptomatic treatment with special attention to mantainence of intake output chart.

On M.R.I there was oedema in mamillary body, brain stem nuclei, peri equiductal grey matter & medial thalami. Ultimately Wernicke's encephalopathy was reported in M.R.I. so the patient was treated with Injection Thiosine 2cc I/V daily for 10 days, then it was switched to Tab. Beovit, 1 tab BD for 3 months.

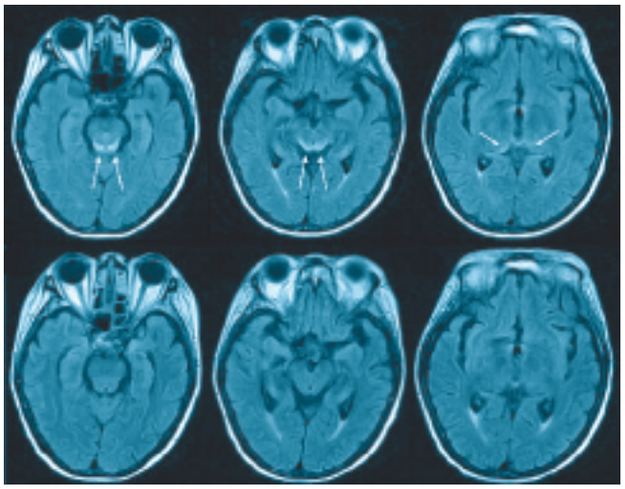

Figure 1 : M.R.I of Wernicke's encephalopathy.

USG was done to see patient's wellbeing at 14 weeks of gestation. The patient was improving day by day, but irrelevant behavior persisted and forgetfulness developed. The patient was advised for consultation of psychiatrist during antenatal period. Rest of her pregnancy period was uneventful only forgetfulness persisted. As she had previous cesarean section her delivery was planned at 38 weeks of gestation. But GOD BLESSED her here, with normal Vaginal Delivery at 37 completed weeks of gestation within half an hour of her admission in hospital with labor pain. She was discharged from hospital with healthy baby but not cured from forgetfulness. So in Antenatal Checkup every patient should be motivated for adequate treatment which is necessary for her.

DISCUSSION

Wernicke's encephalopathy is a Neurological Disorder induced by vitamin B_1 deficiency. It is the most important encephalopathy due to a single vitamin deficiency. Doctor Carl Wernicks- a German Neurologist described it in 1881 as classic triad of mental confusion, ataxia & ophthalmoplagia in three patients- 2 male and 1 female. On autopsy he detected punctuate hemorrhage affecting the grey matter around 3rd & 4th Ventricle and Aquiduct of Sylvus. Later on CAINE proposed atleast 2 signs out of four for recognition of Wernicke's encephalopathy like:-

- Dietary deficiency
- Occulomotor abnormality
- Cerebellar dysfunction
- Memory impairment

Wernicke's encephalopathy due to hyperemesis gravidarum was 1st described by SHEEHANS in the year 1939. The incidence in non alcoholic patients is near about 0.04% to 0.13%. An obstetrical and gynecological survey was done on April 2006 over case report review of literature where 49 cases were reported for Wernicke's encephalopathy due to hyperemesis graviderum[1]. Thiamine deficiency is characteristically associated with chronic alcoholism. Because alcohol affect Thiamine uptake as well as utilization. Wernicke's encephalopathy can also develop in non alcoholic conditions like hyperemesis graviderum, in total parenteral nutrition, in infant who are fed on thiamine deficient infant formula. Thiamine plays a vital role in metabolism of Carbohydrate. It is a co-factor for several essential enzymes in Krebs cycle & Pentose Phosphate pathway. So in deficiency of thiamine, thiamine dependent cellular system begins to fail resulting in cell death by necrosis and apoptosis. Here lastly the patient was improved by I/V Thiamine supplementation after diagnosis of the case by M.R.I report[4-6].

CONCLUSION

M.R.I is a costly investigation not free from its hazards, so it should be reserved for special cases where clinical solution is not possible. A clinical diagnosis of the case earlier may help to reduce the long run sufferings of the patients. We should give emphasis on earlier clinical diagnosis and prompt Thiamine supplementation in pregnant women with prolong vomiting during pregnancy especially with intravenous nutrition.

DISCLOSURE

All the authors declared no competing interest.

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