Wernicke’s encephalopathy: a case report

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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.

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 hypoproteinemina 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


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