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Case Report

A Case Report of Wernicke's Encephalopathy in a Patient with Sepsis

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Abstract

Thiamine (vitamin B1) deficiency is rare but if it occurs can cause vague symptoms like loss of appetite, fatigue, sleep disturbances to severe neurological disorders like polyneuropathy, Wernicke's encephalopathy and korsakoffs syndrome and Cardiovascular manifestations like high output cardiac failure primarily in alcoholics. Many patients with sepsis, critical illness develop altered mental states, variously described as disorientation, confusion, delirium and encephalopathy without obvious explanation.

We report a case of wernickes encephalopathy in sepsis with acute kidney injury in a 36 year old male who is chronic alcoholic without other Comorbidities who presented with high grade fever, vomiting's since 3 days, altered sensorium and difficulty in walking since 1 day. Leucocyte count was elevated on hemogram, renal function tests were abnormal suggesting acute kidney injury, ESR and CRP were raised. MRI findings were suggestive of Wernicke's encephalopathy and there was significant improvement in symptoms after thiamine supplementation.

Keywords: Thiamine; Leucocyte; Wernicke's Encephalopathy

Introduction

Wernicke's encephalopathy is an acute neurological disorder characterized by clinical triad of opthalmoplegia, ataxia and confusion which is associated with significant morbidity and mortality. This disease is caused by thiamine deficiency which is seen primarily in alcoholics. Other causes include diet rich in polished rice/processed grains, increased loss as in diarrhoea and vomiting's, renal replacement therapy, chronic malnutrition, prolonged total parental nutrition without thiamine supplementation, increased nutritional requirements as in pregnancy and lactation, hyperthyroidism, trauma, sepsis. Pathological changes detected by MRI are periventricular lesions around the third ventricle, aqueduct and fourth ventricle, lesions in the dorsal medial nuclei of the thalamus. petechial hemorrhages are found in few acute cases and mammillary bodies atropy is seen in most chronic cases. Endothelial proliferation, demyelination and some neuronal loss is frequently noticed.

Symptoms of thiamine deficiency are often underdiagnosed and under treated as the clinical features overlap with other conditions mainly in patients with severe illness in the ICU.

Case Report

A 26 year old male, labourer by occupation attended to the casualty department of SVS hospital with the complaints of fever-high grade continuous, vomitings since 3 days and altered sensorium and difficulty in walking since 1 day. There is no history of seizures, neck stiffness, no comorbidities. He is a chronic alcoholic since 10years consumes 180-360 ml whisky per day. On Examination his BP was 90/60 mm of Hg, PR 162 beats per minute regular, GRBS 225 mg/dl, temperature 103.5 F. Patient was conscious irritable GCS E4V4M4, Pupils were 3 mm B/L sluggishly reacting to light. ocular abnormalities include restricted abduction bilaterally and horizontal nystagmus. Motor system examination revealed unsteady Gait with normal muscle power and reflexes. His hb was 11.8 gm/dl, wbc 12900 with a neutrophil count 80%. Liver

function tests : ALP 117 U/L,ALT 25 U/L, AST 46U/L, Total bilirubin 1.5 mg/dl and direct bilirubin 0.5 mg/dl, total protein 6.4g/dl.b. urea 154 mg/dl and serum creatinine 5.1 mg/dl. serum electrolytes were in normal range, viral screening was negative, coagulation studies were normal. Serum lactate and procalcitonin levels were raised. USG Abdomen showed grade 2 fatty liver. Complete urine examination showed 8-10 pus cells/hpf. Blood culture sensitivity-coagulase negative staphylococcus. MRI brain revealed T2 Flair hyperintensities in medial aspect of bilateral thalami and peri aqueductal grey matter showing mild restriction on DWI and no reversal on ADC, Prominent ventricular system and sulcal spaces noted with mild cerebral atrophy S/o wernicke's encephalopathy. CSF analysis was done to rule out other causes of encephalopathy which showed mildly elevated protein with cell count being normal. The patient was treated with parenteral thiamine and supportive treatment after which the patient improved significantly and is on follow up.

Discussion

Prevalence of Wernicke's encephalopathy world wide has been estimated from 0.4 to 2.8% Thiamine pyrophosphate is a co-factor for enzymes alpha ketoglutarate dehydrogenase, pyruvate dehydrogenase and transketolase involved in glycolysis and oxidative decarboxylation of carbohydrates for energy production. decreased utilization of cerebral glucose and mitochondrial damage occurs in thiamine deficiency which is precipitated in the presence of risk factors like alcoholism and sepsis. Studies have shown that absolute or relative thiamine deficiency is found in critically ill patients and is associated with about 50% increase in mortality [1-12].

Figure 1: MRI brain showing T2 Flair hyperintensities in medial aspect of bilateral thalami and peri aqueductal grey matter showing mild restriction on DWI and no reversal on ADC, Prominent ventricular system and sulcal spaces with mild cerebral atrophy.

Conclusion

Thiamine deficiency need be suspected in clinical conditions such as neurological disorder in alcoholics, in sepsis where it may be misdiagnosed as sepsis associated encephalopathy and appropriate treatment should be initiated early to avoid potentially dangerous consequences.

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