Original Research Paper

Volume - 12 | Issue - 10 | October - 2022 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Medicine

A CASE REPORT OF WERNICKE'S ENCEPHALOPATHY IN A PATIENT WITH SEPSIS

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ABSTRACT Thiamine (vitamin B1) is an essential water soluble vitamin that plays an important role in energy metabolism. Thiamine deficiency can cause various clinical manifestations ranging from mild neurological and psychiatric symptoms (confusion, reduced memory, and sleep disturbances) to fatal consequences like Wernicke's encephalopathy, ataxia, congestive heart failure, muscle atrophy, and even death primarily in alcoholics. Concurrent illnesses and overlapping signs and symptoms with other disorders can further complicate this.

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 Wernicke's encephalopathy in sepsis with acute kidney injury in a 26 year old male who is chronic alcoholic without other comorbidities who presented with high grade fever, vomiting for 3 days, difficulty in walking and altered sensorium for 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 :

INTRODUCTION

Wernicke's encephalopathy is an acute neurological disorder characterized by clinical triad of opthalmoplegia, ataxia and altered mental state which is associated with significant morbidity and mortality(1). Only one third patients present with the classical clinical triad(2). 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, dialysis, unbalanced nutrition, fasting, starvation, prolonged total parentalnutrition without thiamine supplementation, increased nutritional requirements as in pregnancy and lactation, thyroid disease, cancer, trauma, sepsis(2)(3)(4). 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(2)(5)

Early recognition of Wernicke's encephalopathy is vital, as prompt treatment can restore cognitive or ocular function and can prevent permanent disability(1)

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 (6)(7)

CASE REPORT

A 26 year old Male, labourer by occupation attended to the casuality department of SVS hospital with the complaints of fever high grade continuous, vomiting for 3 days and difficulty in walking and altered sensorium for 1 day. There is no history of seizures, neck stiffness, no comorbidities. He is a chronic alcoholic for 10 years consumes 180-360 ml whisky per day. On Examination his temperature was 103.5 F, Pulse162 beats per minute regular, BP 90/60 mm of Hg, GRBS 225 mg/dl. Patient was conscious, not oriented to time, place, person. GCS was E4V3M4, Pupils were 3mm B/L sluggishly reacting to light. Ocular examination revealed bilateral restriction of abduction. Motor system examination revealed Unsteady Gait with normal power and reflexes . His Hb was 11.8 gm/dl, WBC 12900 cells/cumm with a neutrophil count 80%.

Liver function tests showed Total bilirubin 1.5mg/dl and direct bilirubin 0.5mg/dl, ALP 117 U/L, ALT 25 U/L, AST 46U/L and total protein 6.4g/dl. Blood urea was 154 mg/dl and serum creatinine 5.1mg/dl. serum electrolytes were in normal range. patient tested negative for HIV, HBV, HCV, Dengue, Malaria, Covid 19. Coagulation studies like PT INR, APTT were normal. Serum lactate was 3.2 mmol/L and procalcitonin 3ng/ml. USG Abdomen showed grade 2 fatty liver. Complete urine examination showed 8-10 pus cells/hpf. Blood culture sensitivity showed 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 suggestive of Wernicke's encephalopathy. CSF analysis revealed no abnormality. The patient was treated with parenteral thiamine apart from supportive treatment and specific treatment for septicemia after which the patient improved significantly and is on follow up.

DISCUSSION

Prevalence of Wernicke's encephalopathy in the general population has been estimated from 0.4 to2.8% (3). Among patients with sepsis, estimates of thiamine deficiency range from 10% to 70%(11).

Thiamine pyrophosphate is a co-factor for enzymes alpha ketoglutarate dehydrogenase complex, pyruvate dehydrogenase complex and transketolase involved in glycolysis and oxidative decarboxylation of carbohydrates for energy production(8)(2). 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(2)(4). The contribution of thiamine deficiency to morbidity and mortality in critical illness may be substantially underestimated(11). Studies have shown that absolute or relative thiamine deficiency is found in critically ill patients and is associated with about 50% increase in mortality(9)

In the present situation our patient did not have any manifestations of thiamine deficiency previously, but developed thiamine deficiency symptoms with the onset of sepsis due to upregulation of metabolic processes resulting in more rapid depletion of thiamine stores.

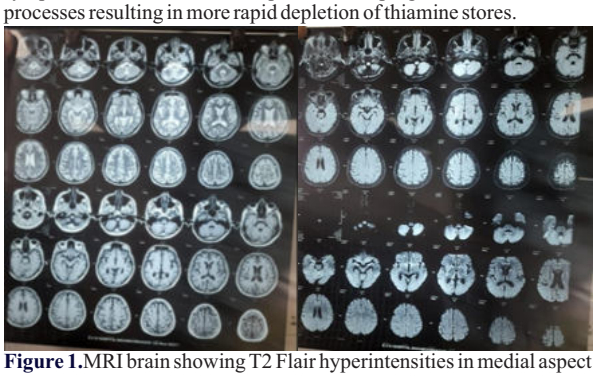

Figure 1.MRI brain showing T2 Flair hyperintensities in medial aspect of bilateral thalami and peri aqueductal grey matter showing mild restriction on DWI and no reversal on ADC, Prominent ventricular system and sulcal spaces with mild cerebral atrophy

CONCLUSION

Thiamine deficiency need to 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. Thiamine supplementation is safe in sepsis to avoid a curable disease.

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