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Case Report

Wernicke's encephalopathy: A case report

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ABSTRACT:

We described a case of an alcoholic patient who presented with a history of Jaundice for 25 days, generalised weakness and loss of appetite for 20 days and altered level of consciousness for 2 days. On examination, patient was grossly icteric and there were no palpable lymphnode. Central nervous system examination revealed low GCS (E2V2M3), Pupil bilaterally pin point, Planter extensor bilaterally and abdominal examination showed mild hepatomegaly. MRI of Brain showed symmetrical hyperintense areas on T2W and FLAIR images seen in the Mamillary bodies, dorsomedial thalami, tectal plate, periaqueductal area and around the third ventricle. The lesions are hypointense on T1W images and showed diffusion restriction on DW images. These findings are compatible with Wernickes Encephalopathy. He was started on thiamine supplementation with which neurological signs improved. Patient had a history of RTA about 1 month prior to ICU admission. Then he underwent Spleenectomy operation. Again a laparotomy operation was done due to intra-abdominal collection and repair of the omental vessels done with thorough peritoneal collection. It is important to consider Wernickes encephalopathy in alcoholic patients with intra-abdominal surgery.

Introduction:

Wernickes Encephalopathy (WE) is an often unrecognized disease of nutritional deficiency and can lead to death if not treated. Although WE usually results from chronic alcoholism, non alcoholic causes such as gastrointestinal tract surgery, AIDS, chronic malnutrition, prolonged parenteral nutrition, hyperemesis gravidarum and rarely in malignancy, are reported in 20-50% of cases. It is often underdiagnosed because clinicians may be less likely to recognize this can also mislead us about diagnosis, hence we present this case.

Case presentation:

A-42- years old man presented with a history of jaundice for 25 days, generalised weakness and loss of appetite for 20 days and altered level of consciousness for 2 days. Patient had a history of RTA (Road Traffic Accident) on 07/05/22. Then he underwent Spleenectomy operation on 09/05/22. Again a laparotomy operation done on 18.05.22 due to intra-abdominal collection and repair of the omental vessels done with thorough peritoneal collection. He had history of raised temperature for 3-4 episodes. On examination, patient was grossly jaundiced and there were no palpable lymphnode. Central nervous system examination revealed low GCS (E2V2M3), pupils bilaterally pin point, plantar extensor bilaterally and abdominal examination showed mild hepatomegaly. His blood pressure was 100/60mmHg and pulse rate is 110 beats/minute. Other system examination reveals no abnormality. Our initial diagnosis was hepatic encephalopathy, stroke or ADEM (acute disseminated encephalomyelitis).

Complete blood count revealed normal findings. RBS was within normal limit. AST, ALT was normal. Alkaline Phosphatase was 289 u/l (Normal-38-126 u/ L)on 16.06.22. S. bilirubin was 53.7 micromol/l (Normal-<22 m.mol/L), S. Ammonia 19micromol/L (Normal: 9-33mmol/L). Renal function test and thyroid study were normal. HIV serology by ELISA was negative. Blood and Urine culture were sterile. Test for malarial parasite was negative. X ray chest was normal. A MRI of Brain was done (due to low GCS, 1day after ICU admission) showed symmetrical hyperintense areas on T2W and FLAIR images seen in the Mamillary bodies, dorsomedial thalami, tectal plate, periaqueductal area and around the third ventricle. The lesions are hypointense on T1W images and showed diffusion restriction on DW images. These findings are suggestive of Wernickes Encephalopathy.

An ultrasound of the abdomen showed mild chronic perenchymal liver disease with mild hepatomegaly, bile sludge in GB, suggestive of acute cholelithiasis/ Hypoalbuminaemia, mild pancreatitis, scanty ascites, mild bilateral pleural effusion. MRCP was suggestive of Cholangitis, Pancreatitis, Cholelithiasis, mild hepatomegaly, minimal peritoneal collections.

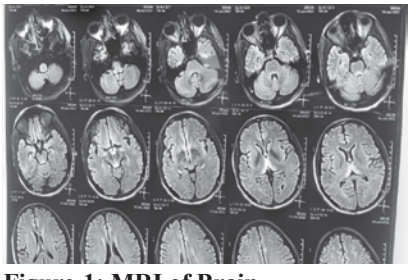

Figure-1: MRI of Brain

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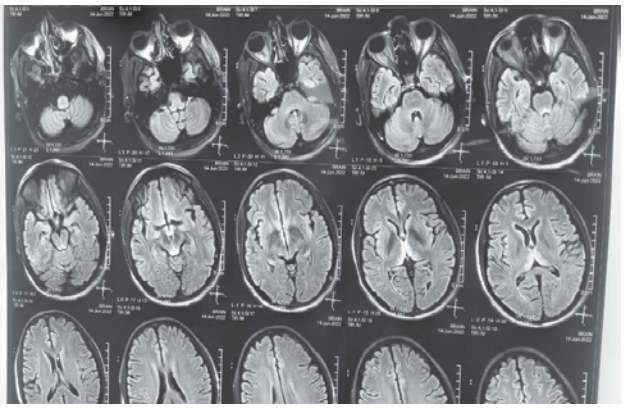

Fig 2: MRI of Brain

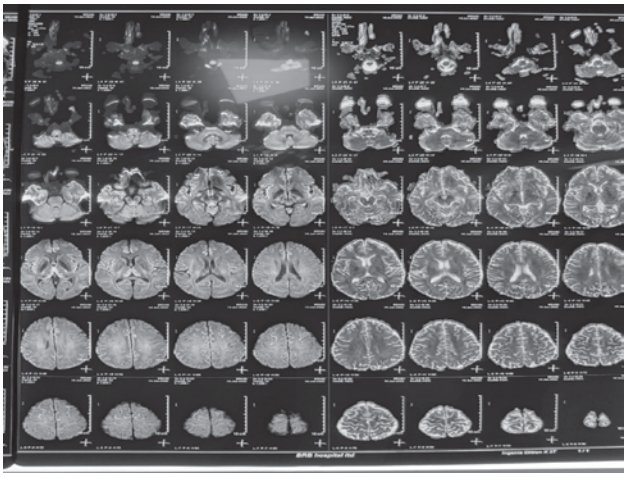

Figure-03: MRI of Brain

The patient was started on thiamine intravenously 200mg with dilution on 0.9% normal saline, twice daily for 2 consecutive days, then 200mg, once daily for an additional 5 days.

Low GCS was improved after just 1 day following thiamine supplementation. Patient was on conservative treatment then and was clinically better at discharge.

Discussion:

WE is a medical emergency caused by thiamine deficiency. Thiamine is a co factor for several enzymes in the Krebs cycle and the pantose phosphate pathway. And thus play a vital role in carbohydrate metabolism. A decrease in their activity may lead to increase buildup of toxic intermediates which are unable to metabolized.[1,2]

Patients presents with features of headache, dizziness, fatigue, irritability, abdominal distension at early stage and later on mental status change. Our patient presented with low mentation. About 82% patients have mental status changes according to autopsy based series.[3,4] These changes occurs due to involvement of thalamic or mammillary bodies and range from acute confusional state to mental sluggishness, apathy and inability to concentrate and if not treated, it leads to coma and death.

Alcoholism along with malnutrition , undernutrition and decrease or inadequate food intake is the main cause here in our patient's WE.

MRI Brain is currently considered as the most valuable method to confirm a diagnosis of WE. MRI shows symmetric involvement of Mammilary bodies, Thalami, the tectal plate, the periaquiductal grey matter and the periventricular region of the 3rd ventricle.[5-7] Here in our patient differential diagnosis was HE as USG Abdomen was in favour of CLD, Stroke as his GCS was significantly low within short period, as history of previous fever, altered level of consciousness , so, ADEM was also a differential diagnosis.

Usual dose of thiamine is though 500 mg initially[8-11], but we started 200 mg, and our patient had a dramatic response within 24 hours.

Conclusion:

To conclude, acute WE is a rare but life threatening condition often overlooked in the unknown alcoholic population, especially in post surgical , malnourished or undernourished patients, resulting in the further progression of an easily treatable condition. Treatment should be initiated at the earliest possible time to avoid persistent brain damage. The prognosis of WE is favourable if diagnosed and treated early, as shown in our patient who was treated with thiamine and made a dramatic recovery.

Learning Points:

- WE may occur in anyone who develops a thiamine deficiency state in post surgical case even with a brief history of under nutrition or nutritional deficiency.
- MRI of Brain is the single most important investigation to diagnose WE, even without true history of alcohol.
- Use of 200mg thiamine intravenously seems to have similar effect as like as 500mg thiamine intravenously in alcoholic post surgical patients.
- Early diagnosis and timely administration of thiamin will help prevent the devastating consequences of a treatable disease.

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