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A Case Report of Hypomagnesemia in Wernicke's Encephalopathy

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Abstract
Magnesium is the second most abundant intracellular divalent cation in the body. Hypomagnesemia is a possible source of Wernicke's encephalopathy, as it plays a role of cofactor in the phosphorylation of thiamine.[1,2] Alcoholics with thiamine deficiency cause severe neurological damage due to hypomagnesemia.[3-6] Here we report a case of 58yrs old male of Wernicke's encephalopathy.
Keywords: *chronic alcoholism, hypomagnesemia, Wernicke's encephalopathy,*

Introduction

Wernicke's encephalopathy is anacute reversible neuropsychiatric disease caused by thiamine deficiency. Chronic alcoholism and malnutrition are the most common risk factors of developing Wernicke encephalopathy. Alcoholism is defined as repeated alcohol-related difficulties in at least 2 of 11 life areas that cluster together in the same 12 month period. About 8.9 – 12.5% of chronic alcoholic individuals develop Wernicke's encephalopathy.[7,8] It is characterized by clinical trial of 1) disorder of mental status or confusion 2) Stance and gait ataxia and 3) Oculomotor abnormalities. These symptoms can be completely resolved by timely recognition of WE and by appropriate treatment. If unrecognized or insufficiently treated, it will lead to korsakoff's syndrome (anterograde and retrograde amnesia) or death.[2]

Case Report

58 year old chronic alcoholic was brought to casualty on 22/10/17 at 6pm in altered sensorium with H/o irritability with irrelevant speech since 22/10/17 afternoon 1pm. H/o binge alcohol intake for past 2 days. H/o persistent vomiting since morning for which patient was taken to private clinic where treated with antiemetic and IV fluids. Followed by patient developed confusion and then brought to our institution for further management. O/E- patient was restless and irritable. GCS - 13/15.BP 110/80mmhg, PR-80/min, SPO_2-98% in RA, RR-30/min, Temp- 98.6°F. No Pallor/ Icterus/ Clubbing/Cyanosis/Lymphadenopathy and Pedal edema. CVS –S_1S_2 heard. No murmur. RS – Normal vesicular breath sounds heard. no added sounds. P/A- Soft, not tender. CNS- Higher mental function: Patient disoriented to time, place and self. Irritable and agitated. Bilateral pupil

2mm RTL. All cranial nerves intact. No sensory deficit. No motor deficit. No meningeal signs. Patient had ataxic gait. The laboratory data was as following: Hb-12.6g/dl WBC-16.7*10^3/µL PLT-199*10^3/µL. ESR ½ hr-12mm 1hr -30mm. RBS -95mg/dl. LFT–T.BILIRUBIN – 0.9, D.BILIRUBIN-0.2, SGOT-127, SGPT – 32, ALP–110. TOTAL PROTEIN – 6.2, ALBUMIN – 4.0 GLOBULIN -2.2. UREA -32, CREATININE – 0.9. SERUM ELECTROLYTES-Na^+-137, K^+-3.6, CL^-–99. SERUM AMYLASE- 30U/L, SERUM CALCIUM – 8.8g/dl. Patient was treated with high dose thiamine and supportive management.MRI BRAIN T2 flair shows hyper intensity of bilateral mammillary bodies and periaqueductal grey matter. Hence diagnosis of WERNICKE'S ENCEPHALOPATHY was made. Symptoms were not resolved even after supplementation of thiamine. Then Serum magnesium was done. Reports revealed hypomagnesemia (0.8 mg/dl). Patient was treated with inj. Magnesium Sulphate. Serial serum Magnesium monitoring was done.

After 12hrs, Patient consciousness improved. Serum magnesium reverted to 1.9 mg/dl. Supplementation of thiamine and magnesium resolved the symptoms completely. This case report proves that hypomagnesemia can be an additional factor for Wernicke encephalopathy.

Discussion

Magnesium is the fourth most abundant element in the body.Majority of this mineral is in the bones and teeth. Only 1% is extracellular.[9] Dietary magnesium content range from 140 to 360mg/day, of which 30 - 40% is absorbed by ileum and distal part of jejunum and excreted by renal. Renal magnesium reabsorption maintains the serum magnesium concentration. 70% of filtered magnesium is reabsorbed by thick ascending loop of henle. Intracellular magnesium act as important cofactor for thiamine phosphorylation which in turn responsible for glucose and amino acid metabolism acting as cofactor for transketolase, α ketoglutarate dehydrogenase and pyruvate dehydrogenase enzymes. Magnesium also plays a role in excitability of neuromuscular membranes, antagonist of NMDA receptor and also has direct vasodilatory effect.[1,2,9]

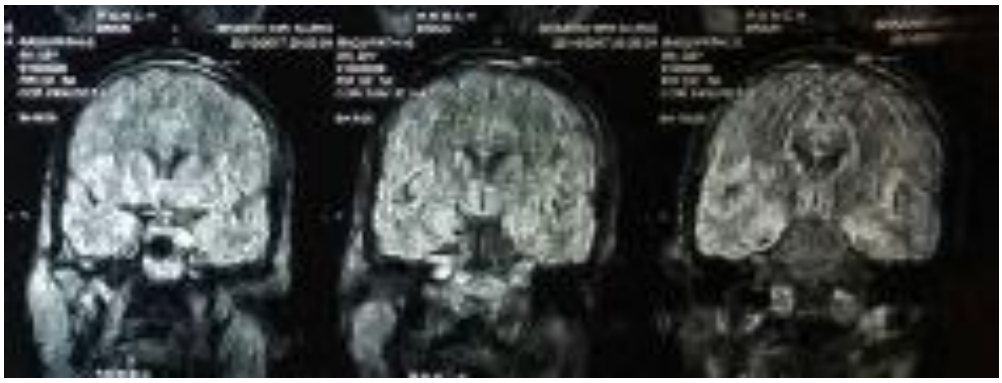
Wernicke encephalopathy is due to deficient supply of thiamine to brain which was described by Carl Wernicke in 1881. Wernicke encephalopathy occurs in one third of alcoholic patient, but also occurs in patients with hyperemesis gravidarum, long starvation, hemodialysis, malignancy, abdominal surgery. Vomiting or diarrhoea is common in chronic alcoholism, if inadequate thiamine intake is associated with nutritional deficiency. Thiamine absorption is reduced to about 90% in alcoholics due to damage to intestinal mucosa and also in hepatic disease which reduces the storage of thiamine. Thomson et al and cook et al reported that the circulating level of thiamine is low in 30–80% of alcoholics. Infections, delirium tremens and alcohol withdrawal increase the metabolic rate where requirement is more in these cases. Binge drinking cause glutamate induced excitability and damage to neurons.[10] Glucose infusion precipitates Wernicke encephalopathy in thiamine deficiency.[11] Blood transketolase activity is markedly attenuated in Wernicke encephalopathy.[12,13]

Wernicke encephalopathy is characterized by classic triad of mental status disorders, oculomotor abnormalities and gait ataxia in 1881. The mental status disorders ranging from global confusional state to delirium tremens and coma due to involvement of mammillary bodies and thalamus.[14] Oculomotor abnormalities like horizontal haze nystagmus which is more common,[14,15] ophthalmoplegia and gaze palsies. Stance and gait ataxia affects the vestibular nuclei causing mild abnormal tandem walking to unable to stand or walk. Other less common signs are polyneuropathy, orthostatic hypotension, dysphagia or hoarseness, hypothermia, bilateral facial paresis, bulbar paresis etc. Nearly 10% of cases have all three main features.[16] Caine et al's operational criteria has sensitivity of 85% and

specificity of 100% In differentiating WE from KS and hepatic encephalopathy. Ophthalmoplegia resolve rapidly, whereas global confusion resolves within hours or days. More than 80% of Wernicke encephalopathy patients develop korsakoff syndrome and 17% lead to death.[14,17]

Diagnosis of Wernicke's encephalopathy is based on history, neurological evaluation, laboratory measurement of thiamine and magnesium and MRI brain. Laboratory assessment of thiamine and its phosphate esters is performed by high performance liquid chromatography (HPLC).[18,19] MRI has sensitivity 53-54% and specificity of 93% in detecting Wernicke's encephalopathy.[20] Reversible cytotoxic oedema is peculiar to WE. There is endothelial proliferation, demyelination and neuronal loss. Typical lesions involve medial thalami, mammillary bodies, tectal plate and periaqueductal area which are symmetric. Atypical lesions are observed in cerebellum, cerebellar vermis, cranial nerve nuclei, red, dentate, caudate nuclei, splenium and cerebellar cortex. In alcoholics, Gadolinium contrast uptake is more in thalamus and mammillary bodies. Atrophy of mammillary bodies and enlargement of third ventricle is unique to chronic WE and korsakoff syndrome. Amnesic defect in korsakoff syndrome is due to lesion in dorsal medial thalami.

Treatment of Wernicke's encephalopathy include administration of thiamine 500mg three times daily intravenously in 100ml normal saline over 30mins for 2 – 3 days followed by 250mg iv daily for next 3 – 5 days. Aggressive magnesium correction 40 - 60 meq/day as infusion over 4 – 12hrs depending on severity.



Hyperintensity noted in bilateral mammillary bodies

Conclusion

All admitted alcoholic patients should be treated with prophylactic thiamine to reduce the risk of Wernicke's encephalopathy. In clinical environment triad is not typical. Early diagnosis and adequate treatment is the treatment of WE. Oral thiamine and magnesium is ineffective in acute Wernicke encephalopathy. Intravenous therapy of magnesium takes several days to correct intracellular magnesium deficiency. Magnesium correction is of importance in thiamine refractoriness to attenuate permanent neurological damage. MRI is of major importance in diagnosing WERNICKE'S ENCEPHALOPATHY.

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