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CIRRHOSIS A RARE CAUSE OF CPM

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Abstract:

A 74-year-old gentleman with cirrhosis, Type 2 Diabetes Mellitus and peripheral neuropathy developed central pontine Myelinolysis. The man's history of most recent admissions shows that he was having multiple metabolic disturbances on different occasions like erratic Blood glucose readings, Hyponatremia, hypoalbuminemia, hypokalaemia, and once had hypophosphatemia. He presented to Princess Alexandra Hospital A/E during this admission (November, 2018) with complaint of weakness of legs, slurred speech and progressive dysphagia for 1 week. His Neurological examination mandated the MRI head with focus on posterior fossa lesions. The MRI was reported as having central pontine myelinolysis with rest of brain studies being normal. His further tests on viral serology, infection screening, TSH level, Vitamin B12 level, Folic acid level and autoimmune screening were all normal. Lumbar Puncture reported to be having elevated proteins while xanthochromia screening shown high bilirubin and Oxyhaemoglobin. There were no further evidences to suggest subarachnoid haemorrhage as well. Patient's CPM was correlated clinically to metabolic disturbances and corrective measures taken for those derangements during recent admissions to hospital and all of these derangements were either directly or indirectly caused by Liver Cirrhosis.

Background: *A commonly understood cause of central pontine myelinolysis is sudden correction of hyponatremia with fluids which would increase serum osmolality rapidly. In patients with decompensated liver disease and Diabetes few more rare causes need to be considered which put them at risk of CPM. It is well known that Cirrhosis patients are mostly hyponatremic due to multiple pathophysiological phenomena and due to diuretics they consume for ascites management. If these patient undergo osmolar corrections it is vital to consider that osmolality is not only controlled by sodium but also by Glucose, potassium and salbumin which, if are deranged too, they would increase the risk of CPM.*

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INTRODUCTION:

CASE PRESENTATION:

A 74-year-old man of British origin was admitted to Princess Alexandra Hospital A/E department in November 2018 with history of subacute onset of weakness of legs, ataxia, dysphagia and slurring of speech for past 2 weeks. His wife was concerned about his recent multiple falls and he had started using wheel chair at home due to fear of falls. He was unable to use knife and fork for eating, and lost a tone of weight due to swallowing issues. Past Medical History was significant for Decompensated Alcoholic Liver disease, type 2 Diabetes Mellitus and peripheral Neuropathy.

Patient was abstinent from Alcoholism since last 8 years and on list for liver transplant at Royal Free Hospital, London. Patient was treated on various occasions for therapeutic paracentesis, esophageal varices, and deranged blood glucose levels and once for subacute bacterial peritonitis. He was on furosemide, spironolactone for ascites management. On CNS examination he demonstrated positive signs for ataxic gait, dysdiadochokinesia, past pointing, abnormal heel shin test. His MRI head was requested and formal referral to neurologist was sent

INVESTIGATIONS:

His blood tests had shown Na,[129] K,[2.8] PO4,[0.65] albumin,[28] and capillary Blood Glucose level of,[25] which would fall as low as, [2.2] next morning. HBA1C was,[131] (normal range 20-42). Previous data shown he had persistently hyponatremia low albumin level and erratic blood glucose reading where level would range between,[1.7-30].His MRI was reported as having central pontine myelinolysis which could explain his symptoms
of ataxia and dysphagia etc.

Hospital Neurologist recommended to test his for HIV, Treponema, Vitamin B12, Folate, CMV, Borellia, syphilis, autoimmune screening and get CT scan of chest abdo-pelvis to rule out paraneoplastic causes of myelinolysis, all test turned out negative and CT CAP was normal for any malignancy. Lumbar puncture was performed to rule out any brain infection, which was reported negative for infections but did demonstrate high proteins and bilirubin level which would go in the favour of myelin degradation again. After ruling out above mentioned causes of CPM,
patients previous care record was studied in detail and it was found that he had persistent hyponatremia, hypokalaemia and erratic blood glucose readings in last few months, he had gone through ascetic tap thrice for relief of shortness of breathing in last 6 months which would be replaced by albumin each time. His last ascetic tap was done 2 weeks before this presentation and had drained 10 litres of fluid after which he was investigated for SBP and
was given 5% albumin and 0.9% normal saline. His PO4 level was low too during previous Hospitalization.
In the light of his previous readings of low sodium, low potassium, low Phosphate, low glucose and correction of low Na with fluids during previous admission it was presumed that likely cause of CPM in this gentleman was derangement of electrolytes and low glucose levels because all other differential causes of myelinolysis were ruled out by blood testing and imaging.

TREATMENT:

Patient was assessed inpatient by Physiotherapy and Occupational Therapy department; he was deemed a candidate for neurorehabilitation and hence referred for neurorehabilitation. Patient and family were explained about new diagnosis of central pontine myelinolysis and they were explained that basic goal in this case would be to help the patient to gain mobility as much as possible. Undoubtedly, it was a bad news for patient who was abstinent from alcohol for last 8 years, hoping to have a new liver to improve quality of life.

OUTCOME AND FOLLOW-UP:

Community physiotherapy and occupational team will take care of his follow up for his mobility and post-rehabilitation management plan. Patient was also seen by gastroenterology team and advised to continue follow up as outpatient, Diabetic team will continue to visit in community for his erratic blood glucose levels monitoring and control.

DISCUSSION:

CPM also named sometimes osmotic demyelination syndrome has been attributed to hyponatremia correction and alcoholism in past. One widely accepted theory for CPM is that rapid shifting of fluids into neuronal cells of pontine causes the pons to adapt to this low osmolality of plasma by accepting water inside. However, if plasma osmolality rises too rapidly this osmotic gradient puts pons at risk of demyelination. Now it needs to be established further that which other factors can increase the plasma osmolality in addition to rapid IV saline infusion. It has been written much about those factors in past and more research and need to be done in this particular area. This prompted the need to write about co-risk factors responsible for CPM. Few postulations are that

in liver failure erratic blood glucose levels may cause sudden fluctuations in plasma osmolality during hyperglycaemic episodes. It may increase the risk of demyelination in those patients who are already hyponatremic and hypokalemic (ascites and diuretic drugs cause it in cirrhosis patients). Further to this, low phosphate level caused by cirrhosis adds to risks for CPM in cirrhotic patients. Albumin is another factor responsible for causing plasma osmolality fluctuations in patients with
cirrhosis. It is important to consider that cirrhotic patients undergo the ascitic fluid drainage procedure where almost 8-10 liters of fluid is drained and replaced with hyperosmolar albumin solutions (20%).If these patients simultaneously are facing other metabolic derangements like hyperglycemia, hypophosphatemia and hypokalemia the risks for cellular damage are increased in pontine myelin. Therefore multiple derangements in electrolytes
and blood glucose level should be carefully addressed by multidisciplinary approach where we can involve Diabetic team, internal medicine physicians, dieticians and hepatologists to prevent such complications (CPM) in future.

LEARNING POINTS:

1. CPM (Central Pontine Myelinolysis) should be considered as important differential in cirrhotic patient presenting with symptoms and signs of brainstem/cerebellar lesions especially in those who had correction of deranged electrolytes in past or those who were treated for metabolic disturbances on multiple occasions.

2. This case highlights rare causes of CPM e.g. erratic blood glucose levels, hypophosphatemia, hypokalemia and low albumin. A commonly established cause of CPM is hyponatremia and its sudden corrections but in Liver Cirrhosis multiple derangements happen in the form of fluctuating blood glucose levels in diabetics, low phosphate level, hypokalemia and hypoalbuminemia which all sum up the risks to cause CPM.

3. These postulations have mandated the need for further studies in cirrhotic patient and it may help to prevent the CPM in Cirrhotic patients especially those who are waiting for new hopes in the form of liver transplant to have good quality of life. In those cases it is extremely crucial to manage these metabolic derangements by involving multispecialty teams to prevent the Central Pontine Myelinolysis.

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