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A Rare Case of Hypokalemia Associated Central Pontine Myelinolysis: A Case Report

Authors

Dr Shruthi Swamy¹, Dr Vijay M.B², Dr Krishna M.V³

¹Junior Resident, Department of General Medicine, RRMCH, Bangalore ²Professor, Department of General Medicine, RRMCH, Bangalore ³Professor & Head, Department of General Medicine, RRMCH, Bangalore

Abstract

Central pontine myelinolysis (CPM) is a rare disorder characterized by non-inflammatory demyelination in the central pons. It was first reported in necropsy cases of alcoholism and malnutrition. It is a condition most frequently related to rapid correction of hyponatremia. CPM is characterized by disturbance of consciousness, quadriparesis and mutism. It has been considered to have a poor prognosis. 100 cases of CPM associated with electrolyte disturbance have been reported worldwide since 1959. Hypokalemia associated CPM has also been reported. We hereby present a 60-year-old male, who had come with complaints of fever, vomiting and headache for five days. Vitals were normal. Patient was found to be drowsy, having slurred speech with deteriorating sensorium and flaccid quadriparesis. Serum electrolytes revealed hyponatremia, hypokalemia and hypochloridemia. Other blood parameters were normal. Sodium and potassium corrections were done as per recommended guidelines within normal limits. There was improvement in sensorium but the quadriparesis persisted. MRI imaging of brain was done which showed signal intensity of the pons on axial T2 weighted images. A final diagnosis of Central Pontine Myelinolysis was made. Central pontine myelinolysis is a rare complication of long standing hypokalemia. Electrolyte abnormality other than sodium should be investigated and rectified.

Introduction

Central pontine myelinolysis (CPM) is a rare disorder characterized by non-inflammatory demyelination in the central pons. CPM is characterized by disturbance of consciousness, quadriparesis and mutism. It was first reported in necropsy cases of alcoholism and malnutrition. It is a condition most frequently related to rapid correction of hyponatremia. 100 cases of CPM associated with electrolyte disturbance have been reported worldwide since 1959. Cases of hypokalaemia associated CPM have also been reported.

Case Report

A 60 yr old male, without remarkable family history or past history presented with fever, vomiting and headache for five days. Vitals were normal. Patient was found to be drowsy, having slurred speech with deteriorating sensorium and flaccid quadriparesis. Serum electrolytes revealed hyponatremia, hypokalemia and hypochloridemia. Other blood parameters were normal. Sodium and potassium corrections were done as per recommended guidelines within normal limits. There was improvement in sensorium but the quadriparesis persisted. MRI imaging of brain was

JMSCR Vol||05||Issue||09||Page 27744-27746||September

done which showed DWI hyperintensities and ADC hypointensities in the pons on axial T2 weighted images. A final diagnosis of Central Pontine Myelinolysis was made.

Trend of Electrolytes

<u>Day</u>	<u>Sodium</u>	Potassium	Chloride
	<u>(mEq)</u>	(mEq)	<u>(mEq)</u>
1	112	2.1	83
2	116	2.0	86
3	123	2.4	97
4	131	2.9	99
5	133	3.0	102
6	141	3.2	108

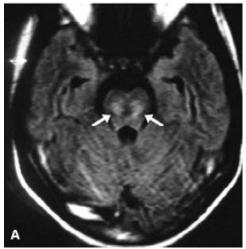


Figure A. MRI imaging using DWI showing hyperintensities in the pons

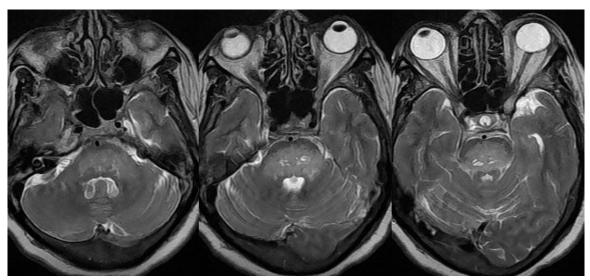


Figure B. Serial axial T2 weighted MR images (TR=3200 ms, TE=82.9ms) showing hyperintensities in pons

Discussion

In CPM, spastic quadriplegia, pseudobulbar palsy (characterized by head and neck weakness, dysphagia, and dysarthria), or encephalopathy in association with non-inflammatory demyelination centered within the pons is seen. The mechanism of myelinolysis is linked to intramyelinitic splitting, vacuolization, and rupture of myelin sheaths, which is presumably caused by osmotic effects in the setting of correction of sodium levels⁽⁵⁾. Studies have suggested that reintroducing the hyponatremia may be beneficial, little research has been done in humans ^(3,15).

A recent study suggested that CPM tends to occur in hyponatremia complicated by hypokalemia because a decreased concentration of Na,K-ATPase in endothelial cell membrane during hypokalemia may predispose the cell susceptible to injury by osmotic stress associated with the rapid rise in the serum sodium concentration. Here CPM was likely to be induced by a slight increase in osmotic pressure attributable to fluid infusion such as of electrolytes and glucose in the presence of severe hypokalaemia.

Conclusion

Central pontine myelinolysis is a rare complication of long standing hypokalemia. Electrolyte abnormality other than sodium should be investigated and rectified. This case report

suggests that further caution is necessary when serum electrolytes are corrected in such patients.

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