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A case of hyperglycemia induced osmotic demyelination syndrome

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ABSTRACT

Central pontine myelinolysis is an acquired demyelinating condition which is now termed as osmotic demyelination syndrome (ODS) due to associated extra pontine involvement. Although various causes of ODShave been established, rapid correction of hyponatremia remains the commonest. ODS usually presents a decrease in the level of sensorium associated with acute flaccid quadriparesis. The present reported case of 62 year old diabetic male who presented with ataxia, dysarthria and bulbar weakness. His blood sugars and serum osmolality was high. Imaging studies showed hyperintensities in pontine parenchyma suggestive of myelinolysis. The patient recovered symptomatically after correction of hyperglycemia.

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INTRODUCTION

Central pontine myelinolysis (CPM) is described as a demyelinating condition that predominantly affects the centre of the basilar portion of the pons. It was found that lesions can occur outside the pons, so-called extrapontine myelinolysis (EPM). Since this original description demyelination of CNS associated with osmotic stress has been described outside the pons (F. G. Kengne *et al.,* 2011). The more general term "osmotic demyelination syndrome"(ODS) is now preferred to the original more restrictive term "CPM" (Lampl C and Yazdi K 2002).

Most common cause of ODS is rapid correction of hyponatremia, rapidly increasing serum osmolality shifts water out of the cells as a response to correct solute imbalance, causing shrinkage of glial cells that can consequently lead to disruption of the blood-brain barrier allowing inflammatory mediators to enter the CNS damaging oligodendrocytes and myelin (F. G. Kengne *et al.,* 2011). However, it can also occur in various other conditions like alcoholism, malnutrition, hypokalemia, psychogenic polydipsia, post liver transplant, hematopoietic stem cell transplant (Yoon B *et al.,* 2008). However, association of hyperosmolar hyperglycemia with ODS is less reported.

CASE REPORT

62 year old male presented to the medical out-patient department with unsteadiness while walking for 10 days which was insidious in onset, and gradually progressive, also had dysarthria and difficulty in swallowing for 7 days. He was a known diabetic since 15 years on oral hypoglycemic drugs; there was no history suggestive of micro or macrovascular complications of diabetes. He denied the history of limb weakness, sensory disturbances, there was no bladder or bowel disturbances. No history of fever, headache and vomiting. He was a non-smoker, non-alcoholic.

On general examination conditions and vital elements of the patient were found to be normal. Examination of the central nervous system indicated that the patient was suffering from dysarthric speech. Cranial nerves examination revealed that nystagmus towards the direction of gaze, bilateral

eye closure weakness suggestive of lower motor neuron type of facial palsy, gag and palatal reflex reduced on both sides with the movement of tongue reduced on both sides suggestive of bilateral IX, X XII cranial nerve involvement. Examination of Spin motor system revealed that no motor weakness but B/L mild incoordination of bilateral upper and lower limb, stance – ataxia was present.

Laboratory studies revealed blood glucose was 542 mg/dL, HbA1c 10.6%, sodium 135 mEq/L, potassium 3.6 mEq/L, urine ketones negative, arterial blood gas revealed no evidence of acidosis and serum osmolality was 316 mOsm/kg, blood urea 38 mg/dL, creatinine 1.2 mg/dL, serum calcium and phosphorus was standard on the day of admission. MRI brain done on the day of admission revealed ill-defined multifocal early confluent DWI hyper-intensities in pontine parenchyma corresponding to low ADC values - Myelinolysis. (Figure 1). The patient was treated with intravenous infusion of regular insulin along with intravenous fluids infusion with strict monitoring of blood glucose, serum electrolytes and serum osmolality which is shown in table 1. After 5 days of treatment, blood sugars were stabilized and serum osmolality was 288 mosm/kg and patient noticed a gradual improvement in the neurological deficit and was able to walk independently and his dysarthria, swallowing difficulties improved at the time of discharge (Al-Thahab *et al.,* 2018). The patient was discharged with subcutaneous basal-bolus insulin. The patient is on regular follow up.

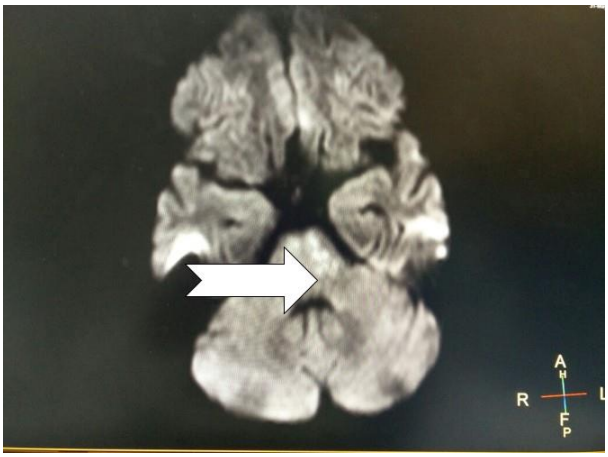

Figure 1: MRI brain showing patchy areas of hyperintensities in pons suggestive of demyelination

DISCUSSION

Osmotic demyelination syndrome is a lethal complication that usually occurs following rapid correction of hyponatremia. ODS following hyperglycemic hyperosmolar state (HHS) is one of the causes which is being less reported. These demyelinating changes usually occur in the pons and also in extra pontine are as like cerebellum, lateral geniculate body, external capsule, hippocampus, putamen and thalamus (Gocht A, Colmant HJ 1987).

Classical clinical presentation of ODS is acute flaccid quadriparesis pseudobulbar palsy, and emotional lability and altered sensorium (Martin RJ 2004; Karp BI 1993) but the patient had subacute onset of ataxia, dysarthria, nystagmus with bulbar weakness and patient did not have motor weakness suggesting that pontocerebellar, corticobulbar tracts were involved predominantly sparing the corticospinal tracts (Lateef *et al.,* 2018).

Previous case reports of hyperglycemia causing ODS was associated with electrolyte abnormality (Hegazi MO, Mashankar A 2013). In 1989 McComb *et al.,* reported the first case of an ODS related to an HHS, but this patient had associated hypernatremia with HHS. In 2008 O'Malley *et al.,* reported a female patient presented with hyperglycemia and normal sodium values later developed flaccid quadriparesis with pseudobulbar palsy following rapid correction of hyperglycemia which was associated with a rapid rise in plasma sodium from a baseline of 135 mmol/L to a peak of 164 mmol/L. Karla Victoria Rodríguez-Velver *et al.,* in 2014 had listed the cases of ODS following correction of HHS and highlighted that ODS, can be the initial manifestation of HHS rather than its correction, like our case. In 2015 Saini *et al.,* had reported a 45 year old female, diabetic presented with an unsteady gait, dizziness and right-sided weakness on evaluation diagnosed as hyperglycemia presenting as CPM (Azhar Omaran, 2017). Gradual improvement of neurological condition was noted following correction of hyperosmolar hyperglycemic state.

In 2017 Kote *et al.,* reported a case of HHS presented as ODS, but this patient had gradual worsening of neurological status and patient died on 15th day of admission. Cause of ODS has attributed long-standing diabetes rather than treatment of HHS. The cause of HHS leading to ODS (Pontine and Extra Pontine Myelinolysis) is probably due to osmolar shift and high blood glucose-induced cerebral oedema due to disruption of cerebral autoregulation, endothelial cells and blood-brain barrier leading to plasma leakage (Andreoli TE *et al.,* 1992). This case illustrates the variable presentation of ODS; our patient had a subacute presentation of ataxia, dysarthria and bulbar weakness. Our patient did not have other electrolyte abnormalities apart from HHS which would have been the major factor for good recovery. Thus, on looking at the previous studies, it is found that hyperglycemic state as the independent cause of ODS and its prognosis depends on serum osmolality, associated

Table 1: Serial Biochemical parameters during inpatient care

	Day 1	Day2	Day3	Day4	Day5
Blood sugars(F/PP) mg/dL	542 (random)	198 244	153 176	130 162	114 143
Serum sodium mEq/L	135	138	130	134	138
Serum potassium mEq/L	3.6	3.4	4.2	4.0	4.3
Blood urea mg/dL	38	34	30	30	28
Serum osmolality (Measured) mosm/kg	319	--	290	--	288

dyselectrolytemia, the rate of correction of electrolyte imbalance and serum osmolality.

CONCLUSION

ODS should be kept in mind when the diabetic patient was presenting with a different neurological presentation during HHS. Although multiple causes of ODS are present, HHS should be considered as one of important cause next to rapid correction of hyponatremia. Cautious monitoring should be done to avoid rapid fluctuations in serum osmolality which is important for good recovery of the patient.

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