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Osmotic demyelination syndrome in an uncontrolled type 2 diabetic – a case report

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Introduction

Osmotic demyelination syndrome is a demyelinating disorder commonly involving the pontine region, midbrain, cerebellum, thalamus and basal nuclei. The pathophysiology behind it is a sudden shift in the serum osmolality and is often seen in patients undergoing rapid correction of hyponatraemia. Diabetes mellitus is a metabolic disorder with hyperglycemia. Long standing poorly controlled diabetics develop neuropathy. It manifests as polyneuropathy, mononeuropathy, and autonomic neuropathy. Ischemic strokes are also common in diabetics. There are few articles of diabetes mellitus associated with osmotic demyelination syndrome.

Keywords: Osmotic demyelination syndrome, seizure, diabetes.

Case Report

A 55 year old female patient presented with complaints of one episode of seizure involving the left upper and lower limb with hemifacial spasm lasting for about 10 minutes with post ictal confusion for 20 minutes. No history of similar complaints in the past. No history of weakness of limbs or sensory disturbances. No history of headache or vomiting. Patient is a known case of type 2 Diabetes mellitus for 5 years on oral medications and not on medications for more than 6 months. Patient is also a knowm case of coronary artery disease – status post Coronary angiogram – single vessel disease and not on medications for 6 months.

On examination patient conscious and oriented; no pallor; pulse – 110/min, regular; Blood pressure – 130/90 mm Hg. Cardiac and respiratory system examination was normal. Abdomen was soft, no organomegaly. Examination of the central nervous system was normal with no evidence of neurological deficit. Optic fundus examination showed evidence of Proliferative diabetic retinopathy. Patient was admitted with the diagnosis of type 2 diabetes with left focal motor seizures and CAD and evaluated.

Her complete haemogram showed haemoglobin – 11.2g/dl with leukocytosis – 13000/cu mm with polymorphs 90%. Her blood sugar at admission was > 500 mg/dl and her liver and renal function tests were normal. She had

hypokalemia with a potassium of 2.6 Meq/L. Her serum sodium, chloride, bicarbonate, calcium and magnesium were normal. Serum osmolality was normal 295 mosm/L and glycated haemoglobin was elevated 11%. Urine examination showed sugar 3+ and urinary ketones were negative. Electrocardiogram showed 'T' wave inversion in lead I, aVL, V1 to V6 and troponin I was negative. Her chest X ray was normal and echocardiogram was consistent with CAD.Patient was started on iv fluids, potassium supplementation, antiplatelets, statins, antibiotic (inj. Ceftriaxone 1g iv twice daily) and insulin for blood glucose correction.

As patient developed an episode of Left focal motor seizure a urgent CT of the Brain was done which showed bilateral prominent lateral, 3rd and 4th ventricles. Later a MRI of the Brain was done with contrast which showed T2 and Flair hyperintense areas in the right inferior cerebellar peduncle, tectum of pons, midbrain, left fronto-parietal &frontal regions and right frontal regions. These areas appear hypointense in T1W1 and donot show contrast enhancement. Mild prominent ventricles noted. Above features suggest to a diagnosis of Osmotic demyelination syndrome

The patient serum sodium levels and serum osmolality was in the normal range and only metabolic derangement was uncontrolled type 2 Diabetes Mellitus and hypokalemia. The osmotic demyelination syndrome in this case can be attributed to the long standing uncontrolled serum glucose levels.

Discussion

Central pontine myelinolysis (CPM) is one of the rare neurological disorder first described in 1959 by Adams and colleagues as a disease affecting the alcoholics. The concept was extended in 1962 with extrapontine myelinolysis (EPM) with the lesions that occur outside the pons in the midbrain, cerebellum, thalamus and basal nuclei. These conditions described earlier are now called as Osmotic demyelination syndrome (ODS). The causes of myelinolysis are hyponatraemia and its rapid correction. There are also reports of ODS associated with hypernatraemia. The other causes include alcoholism, post liver transplant, malnutrition, anorexia, severe burns, AIDS, electrolyte disturbances and hyperemesis gravidarum.

The exact pathogenesis is not clearly understood. Though the shift in the serum osmolality is responsible many patients with rapid correction of hyponatraemia donot develop ODS. There are additional risk factors probably to exist. The pathogenesis explained is hyponatraemia leads to decrease levels of (glutamate, inositol and betaine) intacellular osmolytes. With rapid correction of sodium the brain cells cannot rapidly correct the osmolality and this leads to loss of water from the cell leading to demyelination.

After recovering from hyponatraemia patient may develop dysarthria and dysphagia (due to involvement of corticobulbar tract), a flaccid quadriparesis (corticospinal tract) which later becomes spastic and papillary, oculomotor abnormalities may occur if the lesion extends into the tegmentum of the pons. Patients may have varying degree of encephalopathy or in coma. The patient presents with ''the locked-in syndrome'' where cognitive function is intact but muscles are paralysed except eye blinking. Sensory disturbances are not seen and respiratory disturbances are common. In ODS involving extra pontine areas, the clinical picture may be confusing with a variety of apparently psychiatric and behavioural changes and movement disorders. There are also case reports of ODS presenting with seizures as in our case report the patient presented with left focal motor seizure. In an article by Lin CM and colleague they reported a case

of ODS involving extra pontine area presenting with generalized tonic seizures.

With clinical suspicion the condition is diagnosed by imaging. Often a MRI (Magnetic resonance imaging) is the investigation of choice and T2 images shows hyperintense areas where demyelination has occurred. CSF analysis shows elevated proteins with mononuclear pleocytosis. Electroencephalography may show diffuse bihemispheric slowing and Brainstem evoked potentials may reveal abnormalities when neuroimaging is not conclusive. Treatment is mostly supportive care with correction of malnutrition and neurorehabilitation. Some patients with ODS show very good recovery while some may need ventilator support, respiratory infections and become bedridden.

Our patient presented with new onset of left focal seizures. She was a known type 2 diabetic not on treatment for 6 months and when she presented her sugars were more than 500 mg/dl. In addition she had hypokalemia (serum potassium – 2.6 MEQ/L) and other electrolytes were normal. There was a similar case report by Shintani M and colleagues presented a case of ODS in a case of diabetic with hypokalemia who presented with normoglycemia and in addition had nephrotic syndrome. There was another case report of ODS in a uncontrolled type 2 diabetic and this patient entire electrolyte panel was normal. In another article by David Hopkins and colleagues reported two cases of diabetes with vomiting presenting with ODS.

Long standing diabetes leads to diabetic neuropathy and vascular insult to the central nervous system. There are very few reports of myelinolysis associated with diabetics. As marked shifts in osmolality occurs in patients with diabetics it may be responsible for myelinolysis occurring in diabetics. Also electrolyte disturbances occurring in ketoacidosis and hyperglycemic hyperosmolar state could explain, but myelinolysis is not so commonly reported when compared to the burden of diabetes globally. Is there adaptive process in the brain to osmotic stress in diabetics is also questioned. Also with current understanding that ODS presents with varied clinical manifestations most of the cases are not diagnosed.

To conclude diabetes is a major cause of morbidity and mortality globally and patients with diabetes with neurological manifestations showed be looked for myelinolysis. Patient with diabetes should have a strict glycemic control. Future researches are required in myelinolysis occurring in diabetics.

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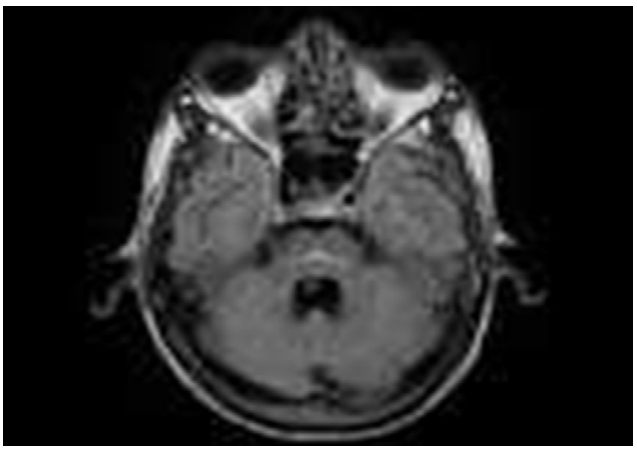
List of Figure

Figure : 1



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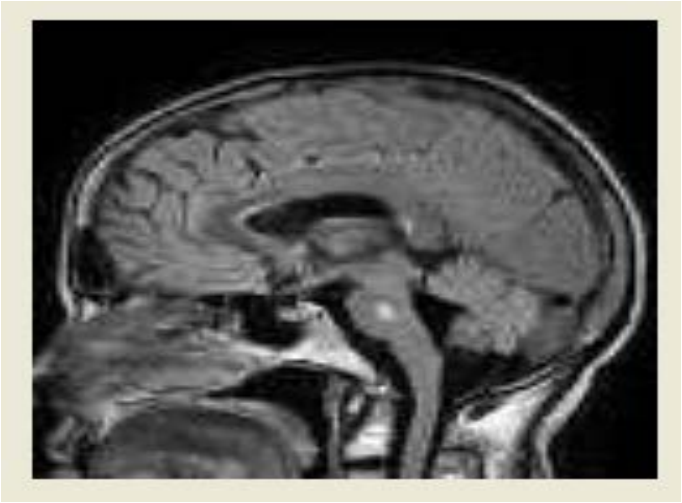
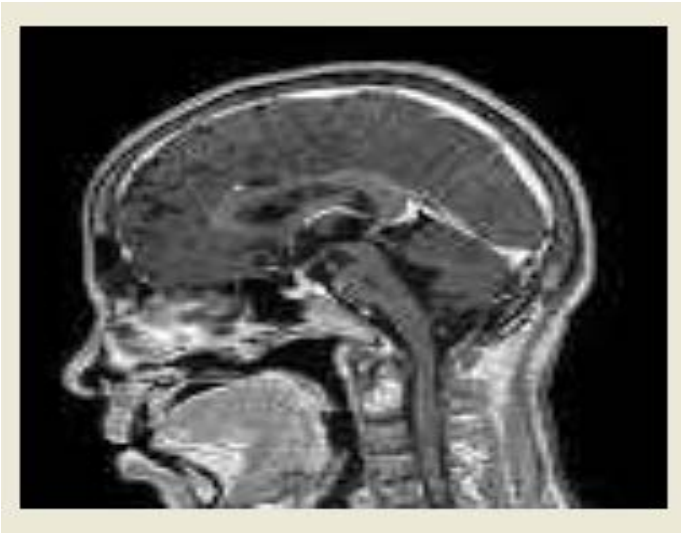


Figure : 3



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