Title: Rare Association of Central Pontine Myelinolysis with Diabetic Hyperglycemia: CPM Associated with Hyperglycemia

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A 56-year-old woman with a poorly controlled type-2 diabetes mellitus presented with progressive dysarthria, dysphagia, and general weakness for 5 days. On admission, she had 36.8°C temperature, 111/53 mmHg blood pressure, 59 beats/min heart rate, and 20 breaths/min respiratory rate. On neurological examination, she was lethargic with pseudobulbar palsy and quadriplegia. Laboratory studies revealed blood glucose was 545 mg/dL, HbA1c 17.3%, sodium 137 mEq/L, blood urea nitrogen 19 mg/dL, and calculated serum osmolality was 311 mOsm/kg. Emergent computed tomography of brain showed a central pontine hypodensity (Figure 1). A follow-up Magnetic Resonance Imaging (MRI) of brain showed a central pontine hyperintensity with peripheral sparing on T2-Weighted (T2W) and Diffusion-Weighted (DW) images (Figure 2), consistent with acute Central Pontine Myelinolysis (CPM). After insulin infusion, her hyperglycemia was corrected and her neurological deficit improved gradually after 1 month.

CPM is an acute noninflammatory demyelinating condition involving the central pons. Hyperosmotic stress result from rapid
correction of chronic hyponatremia is the most common cause [1]. Chronic alcoholics, malnourished, and debilitated patients are at risk. MRI characteristically shows central pontine T2 hyperintensity with peripheral sparing1. Diffusion-weighted MRI may help for diagnosing suspected acute cases early [1]. The differential diagnosis includes infarct, metastasis, glioma, multiple sclerosis, acute disseminated encephalomyelitis, and secondary radiation changes [1].

CPM associated with diabetic hyperglycemia is rare [2]. The disruption of cerebral autoregulation, endothelial cells and the blood-brain barrier by hyperglycemia leading to cerebral edema may cause CPM [2]. Early diagnosis may lead to good prognosis.

There are no conflicts of interest to declare.

References