Central Pontine Myelinolysis Associated with Diabetic Hyperglycemia

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**CLINICAL IMAGE**

A 56-year-old woman presented at the emergency department with progressive dysarthria, dysphagia, and general weakness for 5 days. She had a 20-year history of alcohol abuse and a new-onset type-2 diabetes mellitus. On neurological examination, she had pseudobulbar palsy and quadriplegia. Laboratory studies revealed blood glucose was 545 mg/dL, HbA1c 17.3%, sodium 136 mEq/L, potassium 3.7 mEq/L, blood urea nitrogen 19 mg/dL, and calculated serum osmolality was 316 mOsm/kg. Noncontrast brain computed tomography showed a central pontine hypodensity. T2-weighted (T2W) and diffusion-weighted (DW) magnetic resonance imaging (MRI) of brain showed central pontine hyperintensity with peripheral sparing (Figure 1) consistent with acute central pontine myelinolysis (CPM). Her hyperglycemia was corrected after insulin infusion, 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 sparing [1]. However, initially negative MRI does not exclude CPM. Diffusion-weighted MRI may help for diagnosing suspected acute cases early [1].

CPM developed in the setting of diabetic hyperglycemia is uncommon [2]. Hyperglycemia can cause endothelial cells injury and disruption of blood-brain barrier leading to vasogenic edema and in turn causes demyelination [2]. Early recognition and treatment may lead to favorable outcomes.

**REFERENCES**


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Submitted: 29 August 2014
Accepted: 30 September 2014
Published: 02 October 2014

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