Central Pontine Myelinolysis in a Chronic Alcoholic Patient with Hyperglycemic Hyperosmotic State

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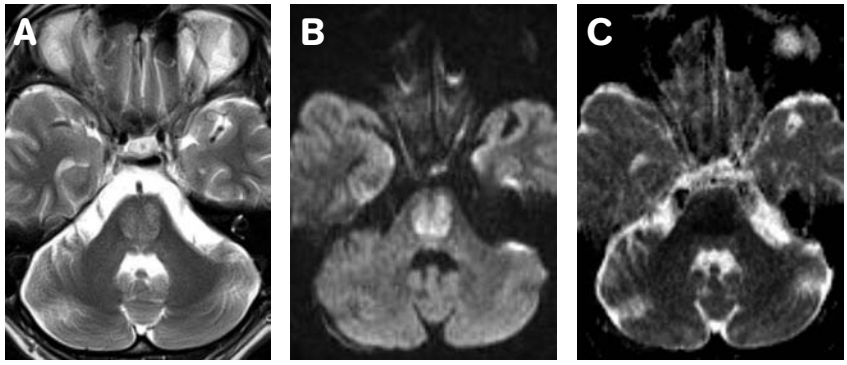

Figure 1. (A) Axial T2WI MR shows a high signal area in the central pons sparing the peripheral white matter, which is a classical finding of central pontine myelinolysis. (B, C). Axial DWI and ADC-map MR show diffusion restriction, with a pattern that precisely matches the hyperintensity on the T2WI.

A 36-year-old man who had poorly controlled type 2 diabetes mellitus and a history of chronic alcoholism for 10 years presented to our emergency department with unsteady gait, dysarthria and easy choking. He denied binge drinking before this episode. Laboratory tests revealed blood glucose was 823mg/dl, sodium 145mmol/L, and calculated serum osmolality was 336mOsm/kg. Brain CT revealed no abnormality. MRI of brain demonstrated central pons T2 hyperintensity with sparing of periphery, indicating the acute stage of disease process (Fig 1). The patient was diagnosed to have central pontine myelinolysis. Fluid resuscitation, insulin infusion and thiamine were given under close monitoring. The ataxia and dysphagia resolved almost completely after 1 month while dysarthria and hand tremor persisted in 4-month follow-up.

Central pontine myelinolysis (CPM) has been a recognized as a complication of rapid correction of hyponatremia [(1)]. A variety of medical conditions have also been reported to be related to CPM. It was postulated that CPM in hyperglycemic hyperosmotic state (HHS) was due to a hypertonic insult [(2)]. CPM could be caused by a rapid change in osmolality resulting in damage to the endothelium, which may further release myelin toxin and produce vasogenic edema in the central pons [(3)]. Notably, chronic alcoholism is more vulnerable to CPM [(4)]. Various pathophysiologic mechanisms

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were proposed including direct toxicity of alcohol on pons or the possibility of undocumented rapid change in sodium level during binge drinking [4].

The typical MR imaging features of CPM are round or triangular shape T2 hyperintensities in central pons. The peripheral pons and corticospinal tracts are usually spared. The lesions may be transitory and resolve completely after adequate treatment. In acute stage, lesions of CPM need to differentiate from acute basilar infarction which, however, usually had wedge-shaped lesion without sparing of peripheral and basilar artery is usually thrombosed.

All physicians should be familiar with typical imaging of CPM and be alert of possibility of CPM in HHS patients. Early diagnosis may improve the final prognosis.

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