A 76 years old woman was referred to our emergency room (ER) due to acute onset of conscious disturbance. She had been well until a week prior to her visit to our ER, when she fell down and injured her right knee. She had received some unknown intravenous drug treatment for knee pain and insomnia. At 10 PM the day before her visit, she could still talk with her family. By 2 AM the next day, she was found sitting unresponsively on the chair with stool incontinence. She didn’t have dyspnea and cyanotic face then, and there was no evidence of carbon monoxide intoxication. The patient was then brought to a local hospital, where she received endotracheal intubation for conscious disturbance. Non-contrast brain computed tomography (CT) scan was normal. After transferring to our ER at 5AM, she remained unconscious and her pupils were equally small but reactive. Her respiratory rate was 20/min on arrival and the pulse oxymeter showed that her oxygen saturation was 100%. Her serum sodium was 114 mmol/L (135-148 mmol/L), aspartate transaminase was 61 U/L (0-39 U/L), alanine transaminase 28 U/L (0-54 U/L), and ammonia was 3 µmol/L (9-33 µmol/L). After hyponatremia was corrected to 128 mmol/L by 1 PM, she regained full consciousness and was extubated smoothly. She was then admitted for further care.

The serum sodium was 130 mmol/L the next day and the follow-up brain CT showed hypodense lesion over bilateral globus pallidus. Extrapontine myelinolysis was suspected. Non-contrast brain magnetic resonance image (MRI) revealed bilateral globus pallidus lesion,

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Figure. Non-contrast brain CT before correction of hyponatremia was normal (A); compared to the one performed two days later, showing hypodense lesions over bilateral globus pallidus (white arrow) after correction of hyponatremia (B); follow-up non-contrast MRI 1 week later showed increased signal intensity in bilateral globus pallidus on DWI (C: white arrow) and T2WI (D: white arrowhead).

Extrapontine Myelinolysis in a Patient Following Correction of Hyponatremia

Cheng-Yang Hsieh and Chin-Wei Huang
which was hyperintense on diffusion-weighted image (DWI) and T2 weighted image (T2WI). Her mental state gradually improved and she was discharged 2 weeks later with minimal disoriented speech.

Extrapontine myelinolysis (EPM), like central pontine myelinolysis, belongs to the osmotic demyelination syndrome. It often occurs after rapid correction of prolonged and severe hyponatremia. The usual locations of EPM in order of frequency are cerebellum, lateral geniculate body, external capsule, hippocampus, putamen, cerebral cortex / subcortex, thalamus, and caudate nucleus\(^{1}\). In addition, the clinical symptoms of EPM correlate with the structure affected. In this patient, the unique clinical presentation and MRI imaging of EPM narrowed down the differential diagnosis, such as hypoxia, carbon monoxide intoxication, Leigh disease, and Wilson disease\(^{2}\). However, these possibilities could be ruled out by clinical history and laboratory data. The patient had EPM involving bilateral globus pallidus from a relatively rapid correction of marked hyponatremia and subsequent changes seen in neuroimaging studies. Lim et al had reported a case with myelinolysis involving bilateral globus pallidus in addition to caudate nucleus, putamen, and central pontine region after a rapid correction of hyponatremia\(^{3}\). Nevertheless, it is rare to involve globus pallidus only in EPM.

References: