Central Pontine Myelinolysis

Masanori Shimodaira, M.D. [1,2], Erisa Sorimati, M.D. [1], Kazufumi Honda, M.D. [1]

[1]Department of Hematology, Endocrinology and Metabolism, Tokyo Metropolitan Hiroo Hospital, Tokyo, Japan
[2]Division of Laboratory Medicine, Nihon University School of Medicine, Tokyo, Japan

BRIEF HISTORY

A 68-year-old man was referred to Tokyo Department Hiroo Hospital because of non-projectile vomiting and watery diarrhea which continued for three days. On admission, his initial blood tests were normal except for severe hyponatremia with a sodium level of 99 mmol/L (normal, 135–145 mmol/L). Normal saline was administered intravenously, and the sodium level began to normalize at 99 mmol/L on day 2, 105 mmol/L on day 3, 120 mmol/L on day 5, and 129 mmol/L on day 7. One week later, he started complaining of difficulty in swallowing and speaking accompanied by unsteadiness of gait and eventual inability to walk. Initial magnetic resonance imaging (MRI) and computed tomography (CT) of the brain on day 7 showed no abnormality. However, his condition continued to deteriorate, and a repeat MRI on day 14 revealed a well-defined lesion in the pons of low T1 signal intensity (Figure 1 A) as well as a trident-shaped lesion of high T2 signal pontine intensity (Figure 1 B). A diagnosis of central myelinolysis (CPM) was made.

CPM is an acute demyelinating condition of the brain stem and recognized complication of the treatment of patients with chronic hyponatremia (>48 h). The risk of CPM is believed to be associated with a rapid (>8 mmol/L/day) correction or overcorrection of the serum sodium concentration. However, there is no accepted safe rate of correction [1]. Other risk factors identified include alcoholism, malnutrition, liver disease, and interestingly, liver transplantation. Symptoms of CPM include tetraplegia, pseudobulbar palsy, and acute changes in mental status leading to coma or death without intervention. Conventional imaging findings (MRI and CT) typically lag behind clinical manifestations, thus limiting the utility of imaging in early diagnosis, and imaging is advocated later to confirm CPM diagnosis [2]. There are no effective therapeutic methods for CPM treatment and recovery varies ranging from none to substantial improvement [3].

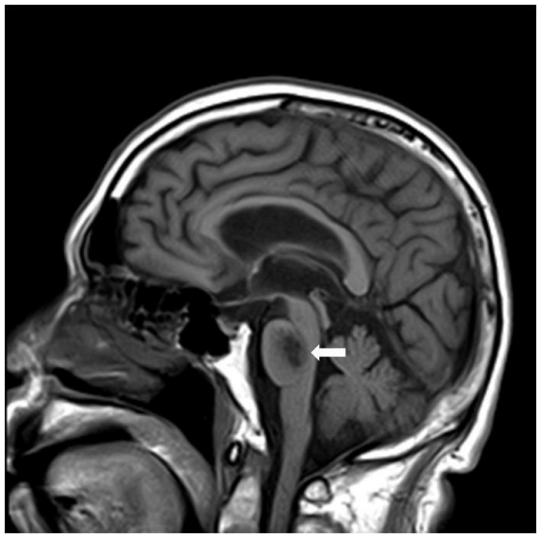
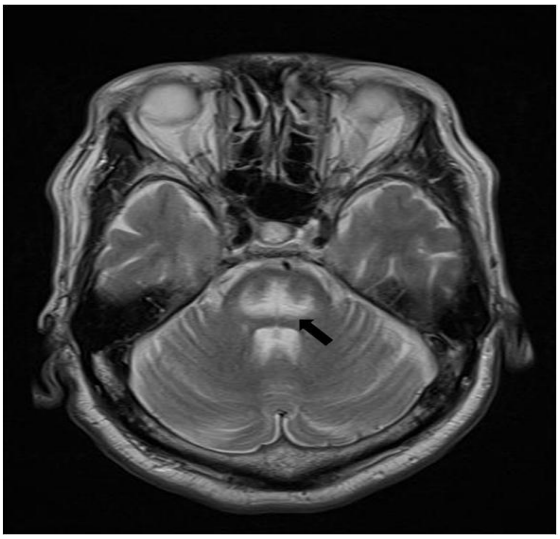


Figure 1 (A) Sagittal T1-weighted image of a 68-year-old man. The pontine lesion did not show any extension into the midbrain and medulla (white arrow).



(B) Axial T2-weighted image of the same patient demonstrating a high-signal trident-shaped lesion in the central pons (black arrow).

Conflicting Interest: None declared

This article has been peer reviewed.

Article Submitted on: 12th August 2011

Article Accepted on: 10th September 2011

Funding sources: None Declared.

Correspondence to Masanori Shimodaira, M.D. Department of Endocrinology and Metabolism, Tokyo Metropolitan Hiroo Hospital, Tokyo, Japan

Address: 2-34-10 Ebisu, Shibuya-ku, Tokyo 150-0013, Japan

Email: masanori19810813@yahoo.co.jp

REFERENCES

1. Adrogué HJ, Madias NE. Hyponatremia. *N Engl J Med.* 2000;342:1581-9.
2. Laureno R, Illo wsky Karp B. Myelinolysis after correction of hyponatremia. *Ann Intern Med.* 1997;126:57–62
3. Menger H, Jörg J. Outcome of central pontine and extrapontine myelinolysis (n = 44). *J Neurol.* 1999;246:700-5.