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CASE REPORT

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Successful treatment of an 86-year-old patient with severe hyponatremia leading to central and extrapontine myelinolysis: A case report

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1 | INTRODUCTION

Osmotic demyelination syndrome (ODS) is a rare acute non-inflammatory central demyelinating disease, which can be divided into central pontine myelinolysis (CPM) and extrapontine myelinolysis (EPM) according to its location.[1] Very rarely, both of these conditions can be present simultaneously; and this is more likely in geriatric patients.[2] A case of CPM with EPM caused by severe hyponatremia (Na^+ = 99.2 mmol/L) in an 86-year-old female was recently diagnosed and treated successfully in our department.

2 | CASE REPORT

An 86-year-old female patient was admitted to the department of orthopedics in Beijing Hospital (March 21, 2018). She complained of having had back pain since 2 weeks before admission. The patient had a 40-year history of hypertension, with long-term use of indapamide as the antihypertensive treatment, and had been diagnosed as having lumbar spinal canal stenosis 6 months previously.

Her blood pressure was 140/80 mm Hg on admission. Physical examination demonstrated drowsiness, dysarthria, mildly sluggish pupillary response with normal pupil size, limited vertical eye movements, and facial and tongue muscle weakness. Other cranial nerve examinations revealed no abnormality. She had weakness in bilateral arms and legs (Medical Research Council [MRC] Grade 3-4), diminished bilateral deep tendon reflexes, and extensor plantar response. She could not cooperate for sensation, coordination, or gait examinations. She also had bladder and bowel incontinence. Blood tests on admission (Day 1) are shown in Table 1.

The patient was diagnosed as having severe hyponatremia (Na^+ = 99.2 mmol/L), hypokalemia (K^+ = 1.8 mmol/L), spinal canal

TABLE 1 Electrolyte results after admission

	Day 1 (0 h)	Day 1 (6 h)	Day 1 (12 h)	Day 2 (24 h)	Day 3 (48 h)	Day 4 (72 h)
Na+ (135-155 mmol/L)	100.9	99.2	111.4	121.5	124.3	133.3
K+ (3.5-5.5 mmol/L)	1.8	2.2	4.3	3.4	3.0	3.5
Fasting blood glucose (3.9-6.1 mmol/L)	13.62	10.81	9.44	7.48	3.13	4.75
Blood urea nitrogen (2.9-8.2 mmol/L)	5.7	10.3	8.3	10.6	6.2	10.2
Cl^- (96-108 mmol/L)	64.4	70.5	87.0	92.6	93.4	104.6
Calculated plasma osmotic pressure (270-310 mOsm/kg H_2O)	224.72	223.91	249.14	267.88	263.93	288.55

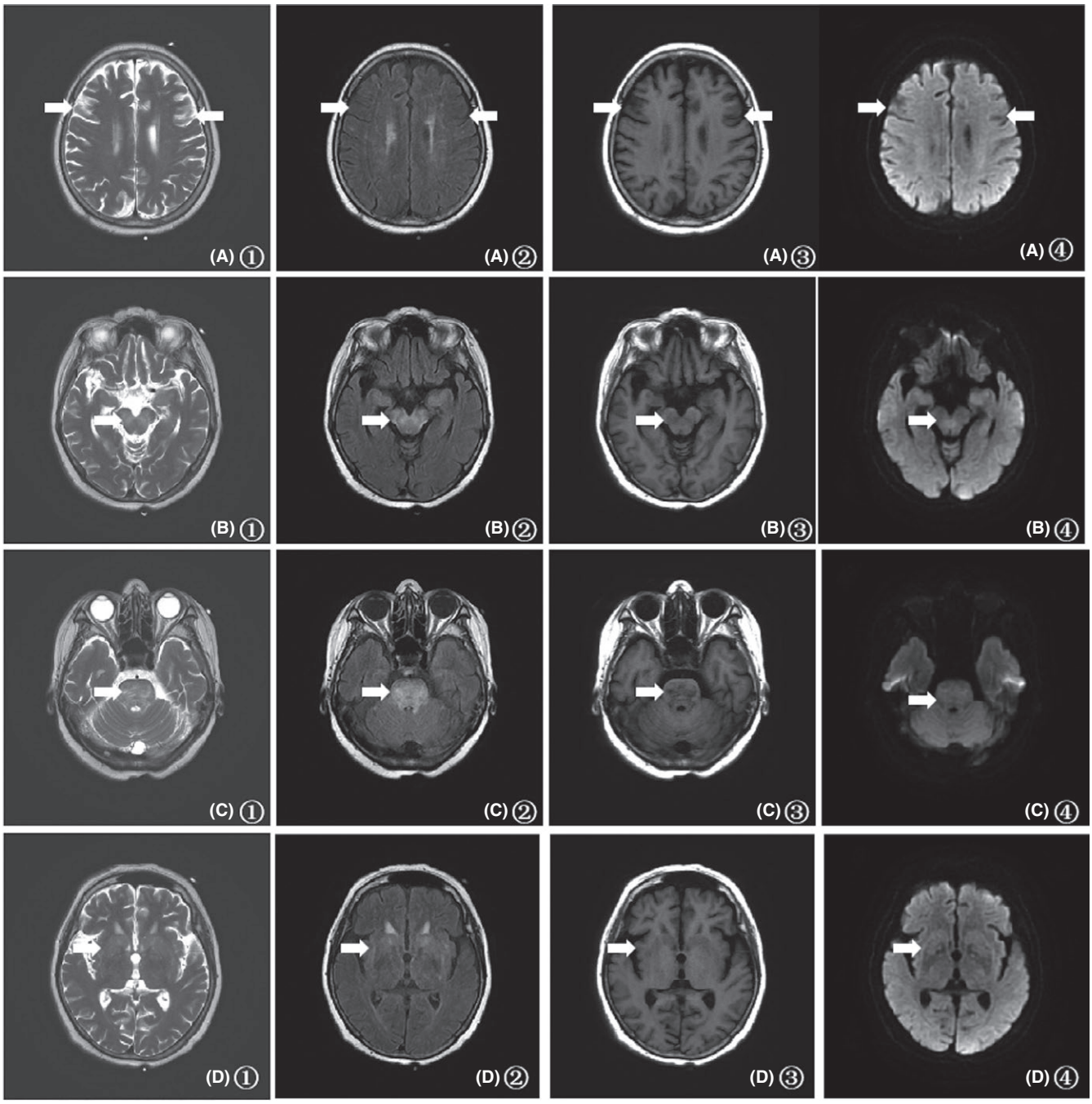

FIGURE 1 At Day 20 of hospitalization (April 10, 2018), magnetic resonance imaging (MRI) showed speckle and patchy hyperintensities in the basal ganglia and thalamus (D①-④), the pons (C①-④), midbrain (B①-④), and the white matter area of the frontal and parietal cortices (A①-④), which showed a slight hypointensity on T1 weighted imaging (WI; ③A-C), a hyperintensity on T2WI (①A-C) and fluid-attenuated inversion recovery (FLAIR; ②A-C), and isointensity on diffusion-weighted imaging (DWI; ④A-C). ①, A-D: T2WI; ②, A-D: FLAIR; ③, A-D: T1WI; ④, A-D: DWI

stenosis, and hypertension. She was given sodium (45.6 g in total during the first 24 h) and potassium supplementation on admission during the first day. The patient's neurological symptoms were relieved significantly. However, blood sodium level rose to 121.5 mmol/L during the first 24 h. The patient became comatose again on the second day, with heart rate rising to 120 bpm and blood pressure dropping to 62/52 mm Hg. She was transferred to the surgical intensive care unit (SICU).

With resuscitation, ventilation support, electrolyte supplementation, and nutritional support in the SICU, the patient's vital signs stabilized eventually. However, her neuropsychiatric symptoms persisted; she remained sleepy, with weakness of all limbs (MRC Grade 3). On the seventh day of admission, we checked her brain computed tomography examination, but did not find any obvious abnormalities. On the 20th day, brain magnetic resonance imaging (MRI) showed speckled and patchy abnormal signals in the pons, bilateral

TABLE 2 GCS assessment and ICDSC assessment

	Day 1	Day 2 (24 h)	Day 2 (36 h)	Day 3 (48 h)	Day 22 (April 10, 2018)	Day 69 (May 28, 2018)
GCS	E2V4M4	E3V4M6	E1V1M3	E2V2M4	E4V4M5	E4V5M6
ICDSC	6	3	None	None	4	2

E, eye response; GCS, Glasgow Coma Scale; ICDSC, Intensive Care Delirium Screening Checklist; M, motor response; V, verbal response.

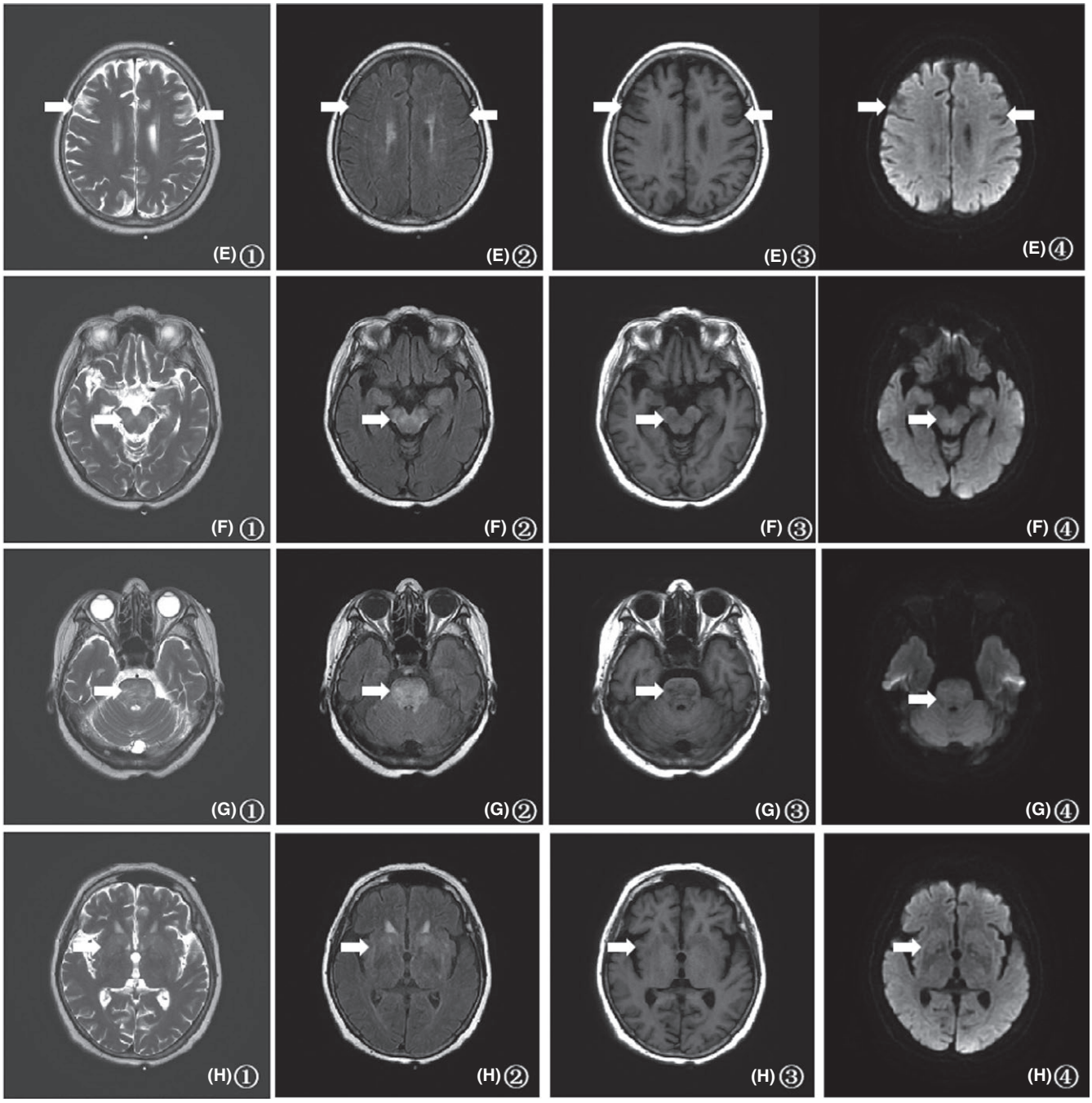

FIGURE 2 After 2 months (on May 28, 2018), brain magnetic resonance imaging (MRI) showed that the lesions in the basal ganglia and thalamus (H①-④), the pons (G①-④), midbrain (F①-④), and the white matter area of the frontal and parietal cortices (E①-④) had all reduced in size (compared with Figure 1). ①, E-H: T2 weighted imaging (WI); ②, E-H: fluid-attenuated inversion recovery; ③, E-H: T1WI; ④, E-H: diffusion-weighted imaging

cerebrum pedicles, bilateral lateral ventricle, midbrain, and the white matter area of the frontal and parietal cortices, which showed slight hypointensity on T1 weighted imaging, hyperintensity on T2 weighted imaging and fluid-attenuated inversion recovery, and isointensity on diffusion-weighted imaging (Figure 1). Incorporating the patient's medical history, clinical signs, and MRI finding, the diagnosis of CPM+EPM was considered. Through hemodynamic support, nutrition therapy, and active functional rehabilitation exercise, the patient's consciousness and orientation were significantly improved (Table 2). After 2 months, her brain MRI showed that the lesions in the pons and bilateral cerebrum pedicles had reduced in size (Figure 2). The patient was then transferred to a secondary hospital for rehabilitation.

3 | DISCUSSION

Osmotic demyelination syndrome is a rare neurological syndrome characterized by symmetrical non-inflammatory oligodendrocyte apoptosis and myelin loss, while neuronal cells and axons are usually intact, with infiltration of a large number of phagocytes and astrocytes.[3] In 1962, Martin found that demyelinating lesions may also involve extrapontine tissues, such as basal ganglia, inner capsule of the thalamus, cerebellum, neostriatum, amygdala, and midbrain. EPM accounts for about 10% of ODS cases.[4]

The major causes of ODS include chronic alcoholism, malnutrition, and correcting hyponatremia.[5] Other causes include liver transplantation,[6] brain trauma, pituitary tumors, postpartum hemorrhage, hypertonic state, impaired liver function, renal dialysis, acute porphyria, lithium poisoning, and hyperemesis gravidarum. ODS is caused by imbalance in intracranial osmotic pressure, which leads to destruction of the blood–brain barrier, brain cell dehydration, intramedullary edema, and oligodendrocyte destruction.[7] CPM firstly involves the corticobulbar tract, which is characterized by dysarthria and dysphagia. Further involvement of the corticospinal tract and basal part may cause flaccid or spastic paralysis, nystagmus, and gaze disturbance. MRI is currently the main method of diagnosis of CPM/EPM, while early stage ODS can show normal signs on MRI. Patients with clinical diagnosis of CPM/EPM should repeat the brain MRI examination 10-14 days after disease onset. Diffusion-weighted imaging can reveal a hyperintense lesion as early as 1 week after the onset.[8]

Diuretics are one of the important causes of hyponatremia; long-term use of indapamide has the risk of severe hyponatremia,[9] and ODS might occur during its correction. In particular, patients with chronic hyponatremia must be managed with caution. Sodium supplementation should be slow and should not exceed 0.5 mmol/L/h, and should not exceed 8 mmol/L/d.[10,11]

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