

A Good Prognosis of Central Pontine Myelinolysis in a Type II Diabetic Patient

Dear Editor,

Central pontine myelinolysis (CPM) is an osmotic demyelination syndrome that is typically associated with hyponatremia or the rapid correction of hypernatremia. The specific mechanism by which the osmotic demyelination occurs is not completely understood. Here we describe a patient presenting with CPM confirmed by magnetic resonance imaging (MRI) who had persistent hyperglycaemia as a result of untreated type 2 diabetes in the presence of normal serum sodium levels, and had a good prognosis.

Case Report

A 51-year-old male was admitted to our hospital with progressive dysarthria, dysphagia and mild tetraparesis for a period of 37 days. He experienced dizziness at day 1, which progressed to an inability to walk unassisted. At day 25, he developed anarthria and dysphagia when admitted to our hospital. He had a history of untreated type 2 diabetes for more than 20 years, which resulted in impaired vision in the left eye due to diabetic retinopathy. His fasting blood glucose after admission was 434.52 mg/dL. Although he had not had an alcoholic drink in over a year, he had a 20-year history of alcohol consumption, consuming 250 mL per day. In addition, he had poorly controlled hypertension. On admission, his vital signs were normal. The first neurological examination determined that the patient was oriented to person, place and time with deficits including reeling gait, dysarthria, bulbar palsy, mild tetraparesis and absent of ankle- and knee-jerk. The results of the laboratory tests are shown in Table 1. In addition, the electromyogram showed slowed nerve conduction velocity in limbs, specifically the right sural nerve. His brain MRI is shown in Figures 1A to 1C.

He was treated with insulin and intravenous fluids to control blood pressure. Two days after insulin administration, his bulbar palsy and paresis gradually improved with a normalisation of blood glucose. Insulin was discontinued after 1 week. A follow-up MRI after week 4 (T2), showed no obvious change of the pontine lesion (Fig. 1D). However, the patient showed significant improvement in speech, gait and dystaxia.

CPM is a demyelinating disorder first identified by Adams et al.¹ Although the exact aetiology and pathogenesis remains unknown, it is frequently associated with chronic

Table 1. Results of Laboratory Test on Patient

Lab Tests	Patient Data	Normal Range
Blood chemistry and serological tests		
Glucose (mg/dL)	434.52	70.02-114.84
Albumin (g/dL)	0.03	0.04-0.05
LDL-C (mg/dL)	196.93	0.00-140.83
CH (mg/dL)	272.76	0.00-221.31
ALP (IU/L)	132	40-129
BUN (mg/dL)	23.36	7.89-20.00
Na (mEq/L)	136	136-145
K (mEq/L)	3.96	3.50-5.20
Urine tests		
Proteinuria (g/24h)	1.5	-
Glycosuria	4+	-
Cerebrospinal fluid		
Initial pressure (mmH ₂ O)	125	70-200
Cell (/mm ³)	1	0-10
Protein (mg/dL)	0.65	0.12-0.60
Glucose (mg/dL)	1.5	0.4-0.7
Cl (mEq/L)	122	120-132

hyponatremia and rapid correction or an increase in serum osmolality secondary to the development of hypernatremia.² MRI is the gold standard to identify and diagnose CPM. Typical findings include a symmetrical decrease in signal intensity in T1-weighted image and increased signal intensity in T2-weighted image in the central portion of the pons. The prognosis for CPM is poor and the mortality is very high.

Our patient had poorly controlled diabetes with developed neuropathies. Patients with poorly controlled diabetes and persistent hyperglycaemia often develop renal dysfunction, which are contributing factors to serum osmolality. In such cases, greater osmotic shifts and over-hydration may occur resulting in an increased risk of cerebral oedema.³ Besides, diabetes can cause disruption of cerebral autoregulation, endothelial cells and the blood-brain barrier, leading to plasma leakage and cerebral oedema,⁴ eventually causing brain oedema. Although our patient exhibited such symptoms, his prognosis was good. The initial neurological deficits were significantly improved following a reduction

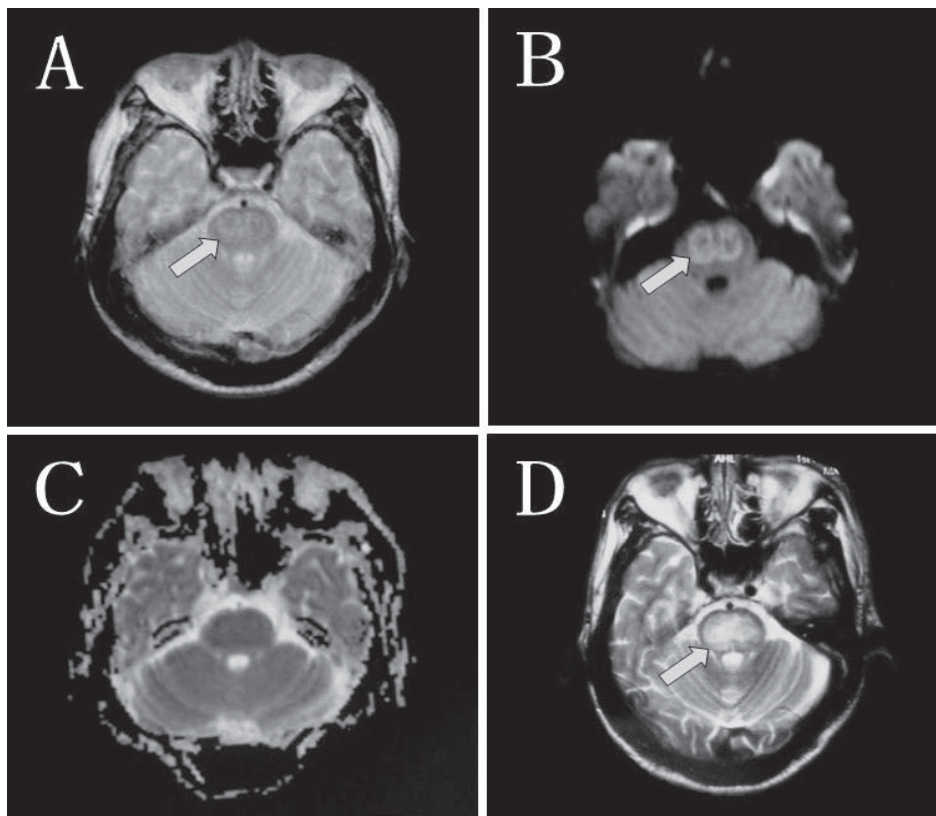


Fig. 1A. Brain MRI reveals an increased symmetrical ring-shaped signal intensity, the centre of which is inhomogeneous, in the central portion of the pons on a T2-weighted image.

Fig. 1B. The diffusion-weighted images (DWI) shows marked hyperintense lesions, the centre of which was also inhomogeneous.

Fig. 1C. The apparent diffusion coefficient (ADC) map shows an isointensity signal in the pontine lesion.

Fig. 1D. A followed-up brain MRI reveals an increased symmetrical ring-shaped signal intensity, the center of which is also inhomogeneous, in the central portion of the pontine lesion on a T2-weighted image.

in his blood glucose after the administration of insulin. However, his follow-up MRI did not show obvious change to the pontine lesion.

Conclusion

The prognosis of CPM varies widely. Our findings indicate the prognosis of CPM is not dependent on the severity of clinical presentation or laboratory results during the acute phase of the condition or on concomitant risk factors, including alcohol use and hypertension. Moreover, the prognosis of CPM may not be related to the severity of pontine deficits or the persistence of the pontine lesions associated with poor clinical improvement. Our patient may return to normal functioning, but the condition may also lead to death. A follow-up MRI showed that the extent of the disease was not obviously decreased, but the patient showed significant improvement. Recent case studies have reported that the apparent diffusion coefficient (ADC) measurements may eventually be of prognostic predictive

value. Normal ADC map predicts a good prognosis because of the vasogenic oedema instead of cellulos damage.⁵ Therefore we think that vasogenic oedema, not cellular oedema contributed to the patient's recovery. Besides, chronic alcoholism and hypertension for this case cannot be excluded. To improve prognosis and quantity of life it is important to prevent misdiagnosis of the disease and initiate early treatment.

REFERENCES

1. Adams RD, Victor M, Mancall EL. Central pontine myelinolysis: a hitherto undescribed disease occurring in alcoholic and malnourished patients. *AMA Arch Neurol Psychiatry* 1959;81:154-72.
2. Sterns RH, Riggs JE, Schochet SS Jr. Osmotic demyelination syndrome following correction of hyponatremia. *N Engl J Med* 1986;314:1535-42.
3. Andreoli TE. Disorders of fluid volume, electrolyte, and acid-base balance. In: Wyngaarden JB, Smith LH, Bennett JC, editors. *Cecil Textbook of Medicine*. 19th ed. Philadelphia: W. B. Saunders Company, 1992.
4. Jacob RJ, Fan X, Evans ML, Dziura J, Sherwin RS. Brain glucose levels are elevated in chronically hyperglycemic diabetic rats: no evidence for protective adaptation by the blood brain barrier. *Metabolism* 2002;51:1522-4.
5. Dervisoglu E, Yegenaga I, Anik Y, Sengul E, Turgut T. Diffusion magnetic resonance imaging may provide prognostic information in osmotic demyelination syndrome: report of a case. *Acta Radiol* 2006;47:208-12.

Fan Lou,^{1MM}, Xiaoguang Luo,^{1MD}, Dan Cao,^{1MDS},
Yan Ren,^{1MD}

¹Department of Neurology, First Affiliated Hospital of China Medical University

Address for Correspondence: Dr Yan Ren, Department of Neurology, First Affiliated Hospital of China Medical University, Liao Ning 110001, China. Email: loufan1018@yahoo.com.cn