

Case report

Central pontine and extrapontine myelinolysis associated with type 2 diabetic patient with hypokalemia

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Abstract

Central pontine myelinolysis (CPM) is a demyelinating disease of the pons often associated with the demyelination of extrapontine areas of the central nervous system. Although the etiology and pathogenesis are unclear, CPM is usually associated with hyponatremia or its rapid correction, and chronic alcoholism is also a common underlying condition. We observed a 43-year-old man with diabetes mellitus who developed central pontine and extrapontine myelinolysis with no apparent evidence of hyponatremia, serum hyperosmolality or associated rapid correction, or history of alcohol abuse. On admission, the patient was lethargic with dysarthria, dysphagia, and mild tetraparesis and his face and lower extremities were severely edematous. Laboratory examination showed normoglycemia and normonatremia, although hypokalemia, elevated HbA_{1c}, and nephrotic syndrome were also present. Magnetic resonance imaging (MRI) revealed abnormal signal intensity in the pons, the deep layers of the cerebral cortex, and the adjacent white matter consistent with central pontine and extrapontine myelinolysis. Generalized edema was reduced by the use of diuretics and extracorporeal ultrafiltration without significant changes of serum sodium or osmolality. His consciousness level and paresis gradually improved within a few weeks. Our patient is a rare case of CPM associated with diabetes without apparent evidence of sodium or glucose imbalances.

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1. Introduction

Central pontine and extrapontine myelinolysis are neurologic complications that have been associated

with the rapid correction of hyponatremia [1,2]. Pontine and extrapontine myelinolysis have also been described in association with other underlying conditions, such as alcoholism and malnutrition [3,4]. These conditions are characterized by loss of myelin with the sparing of neuron in the central pons as well as certain extrapontine sites, such as the internal capsule, basal ganglia, cerebellum, and

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cerebrum. Chronically, CPM is characterized by rapid evolving paraparesis or quadriparesis with pseudo-bulbar symptoms, such as dysarthria and dysphagia.

We describe a case of diabetic patient accompanied by central pontine and extrapontine myelinolysis without hyponatremia, hyperosmolality or associated rapid corrections.

2. Case report

A 43-year-old man with a history of type 2 diabetes for more than 10 years was admitted to our hospital on April 9, 2003, due to lethargy, dysarthria, dysphasia, mild tetraparesis, and severe generalized edema.

He was previously admitted to our hospital due to nephrotic syndrome in June 2002, at which time, diabetic nephropathy was diagnosed. Vibration sense was decreased and neurogenic bladder dysfunction was also diagnosed. Proliferative diabetic retinopathy was observed and photocoagulation was recommended. His condition was complicated with diabetic microangiopathy, but he did not have cerebrovascular disease or ischemic heart disease. After his discharge, he discontinued his doctor visit and eventually lost his sight due to diabetic retinopathy.

Laboratory data on the present admission (Table 1) showed hypokalemia (2.6 mEq/l, normal range: 3.5–4.8), hypoalbuminemia (1.8 mg/dl, normal range: 3.8–5.3), proteinuria, and hyperlipidemia, but serum sodium (sodium; 146 mEq/l, normal range: 135–147), osmolality (297 mOsm/kg, normal range: 275–295) and glucose (101 mg/dl, normal range: 60–109 mg/dl) were almost normal. HbA_{1c} was, however, at a high value of 9.1%. Magnetic resonance imaging (MRI) of the brain (Fig. 1(a)–(d)) revealed increased signal intensity in the central portion of pons, the deep layers of the cerebral cortex and the adjacent white matter consistent with central pontine and extrapontine myelinolysis in T2 weighted images. The T1 weighted images decreased in intensity. CSF examination did not reveal any remarkable changes. These clinical images seemed to be consistent with central pontine and extrapontine myelinolysis.

The patient's generalized edema was reduced after 2 weeks by the use of the diuretic furosemide and by the extracorporeal ultrafiltration method (ECUM)

Table 1

Laboratory data on the present admission

CBC	
WBC	7300 ml ⁻¹
RBC	429 × 10 ⁴ ml ⁻¹
Hb	11.5 g/dl
Ht	35.7%
Plt	52.9 × 10 ⁴ m ⁻¹
Serological	
CRP	2.49 mg/dl
Urinalysis	
Protein	300↑
Glucose	—
Ketone body	+
Occult blood	2+
pH	7.0
WBC	3+
Arterial blood gas analysis	
pH	7.460
pO ₂	73.5 mmHg
pCO ₂	39.0 mmHg
HCO ₃	27.1 mEq/l pl
BE	3.2
Blood chemistry	
Na	146 mEq/l
K	2.6 mEq/l
Cl	108 mEq/l
Ca	7.6 mg/dl
P	4.4 mg/dl
BUN	18.6 mg/dl
Cr	1.8 mg/dl
UA	6.7 mg/dl
TP	4.7 g/dl
Alb	1.8 g/dl
Ch-E	291 IU/l
GOT	12 IU/l
GPT	4 IU/l
LDH	175 IU/l
γ-GTP	9 U/l
T-cho	351 mg/dl
TG	192 mg/dl
HDL	53 mg/dl
LDL	282 mg/dl
Glucose	101 mg/dl
HbA _{1c}	9.1%

without significant changes of serum sodium or osmolality (Fig. 2). His consciousness and paresis gradually improved relative to the improvement of serum potassium level and nutrition. T2 weighed MRI images after 6 months showed slight reduction of the high intensity in the pons (Fig. 1(e)).

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