A case of posterior reversible encephalopathy syndrome in a patient undergoing automated peritoneal dialysis

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Abstract

A 44-year-old man undergoing automated peritoneal dialysis (PD) developed headache and dizziness with truncal ataxia and ataxic gait. Severe hypertension (systolic blood pressure/diastolic pressure: 193/83 mm Hg) and lower extremity edema were present, and his PD efficiency (weekly KT/V: 1.49) was inadequate. Magnetic resonance imaging revealed diffuse hyperintensities in the brain stem and bilateral cerebellar hemispheres on fluid-attenuated inversion recovery and apparent diffusion coefficient mapping imaging. Based on these findings, the patient was diagnosed with posterior reversible encephalopathy syndrome due to hypertension and uremia. He was treated with antihypertensive agents, and we changed the PD prescription to improve PD efficiency. Thereafter, his symptoms gradually improved, and abnormal findings on brain magnetic resonance imaging disappeared in accordance with lowering blood pressure.

Keywords Posterior reversible encephalopathy syndrome · Peritoneal dialysis · Hypertension · Renal impairment

Introduction

Posterior reversible encephalopathy syndrome (PRES) is defined as vasogenic edema of the white matter in the posterior brain regions [1]. PRES is characterized by various neurological symptoms such as headache, seizure, confusion, dizziness, and visual loss, with reversible radiological findings [1]. Several factors, such as severe hypertension, uremia, immunosuppressive drugs, infection, autoimmune diseases, and erythropoiesis-stimulating agent (ESA) in chronic kidney disease contribute to development of PRES [1–4]. Several case reports describe PRES in patients undergoing peritoneal dialysis (PD) [5–8]; however, the clinical course and treatment of PRES in patients undergoing PD have not fully clarified. We report a case of a 44-year-old man undergoing automated PD who developed PRES due to severe hypertension and uremia.

Case report

Our patient was a 44-year-old man who had been receiving PD for 6 months due to diabetic nephropathy as a complication of type II diabetes mellitus. He was undergoing automated PD, with a protocol of three cycles of 2000 ml of lactate-buffered, 1.35% glucose PD fluid over 7 h overnight. He also had hypertension that was being treated with telmisartan 20 mg/day, hydrochlorothiazide 25 mg/day, and furosemide 120 mg/day. Additionally, he had been administered 50 µg of epoetin β pegol once monthly for the treatment of renal anemia, and his hemoglobin level had been maintained at approximately 12 g/dL. One month before admission, his systolic blood pressure gradually increased above 170 mm Hg. Twenty days before admission, he developed bacterial peritonitis due to *Staphylococcus lugdunensis* infection, and antibiotic therapy with cefazolin 1 mg/day and ceftazidime 1 mg/day was started, which improved the peritonitis.

He developed headache and dizziness and visited our hospital. His blood pressure showed severe hypertension (systolic blood pressure/diastolic pressure: 193/83 mm Hg), and he had gained over 3 kg above his normal body weight. Pitting edema was also observed in both lower extremities.

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Truncal ataxia and ataxic gait without limb ataxia were present without disturbance of consciousness or vision. He was admitted to our department for further examination and treatment. Laboratory tests on admission showed severe renal dysfunction (creatinine: 11.6 mg/dL, blood urea nitrogen: 52 mg/dL), and mildly elevated inflammatory indicators (white blood cell count: 9100/ μ L, C-reactive protein: 0.23 mg/dl). His PD fluid was clear without increased cells count (cell count: 28/mm³). His PD efficiency was inadequate (weekly KT/V: 1.49). A detailed description of the patient's laboratory data on admission is shown in Table 1. Chest x-ray showed enlarged cardiothoracic rate (53.5%), but no abnormal findings in either lung field. Head computed tomography showed no obvious intracranial hemorrhage or mass lesion. Brain magnetic resonance imaging (MRI) revealed diffuse hyperintensities in the brain stem and bilateral posterior cerebellar hemispheres on fluid-attenuated inversion recovery (FLAIR) imaging and apparent diffusion coefficient mapping (ADC-MAP) (Fig. 1a, b, arrows) but not on diffusion-weighted imaging. Based on these findings, he was diagnosed with PRES due to severe hypertension related at least in part to volume overload and uremia. Antihypertensive therapy with continuous intravenous nicardipine infusion was started. Oral antihypertensive drugs and diuretic drugs were changed to olmesartan 40 mg/day, amlodipine 10 mg/day, and doxazosin 0.5 mg/day, hydrochlorothiazide 25 mg/day, and furosemide 120 mg/day to decrease blood pressure and fluid retention, respectively. Also, 2 L of lactate-buffered, 1.35% glucose PD fluid was added to the PD prescription for ultrafiltration and to improve PD efficiency. Thereafter, his blood pressure decreased gradually and symptoms including headache, dizziness, truncal ataxia, and ataxic gait gradually improved. The patient's clinical course after admission is shown in Fig. 2. On the tenth admission day, his systolic blood pressure level decreased to approximately 130 mm Hg, his body weight decreased by 3 kg, and his PD efficiency also improved (weekly KT/V: 1.59). On the 25th admission day, repeated brain MRI revealed that the abnormal findings detected on admission had completely disappeared (Fig. 1c, d). He was discharged on the 28th admission day with no sequelae.

Discussion

We reported PRES in a patient receiving automated PD. PRES is defined as reversible vasogenic edema that frequently occurs in the posterior brain regions [1, 9–11]. Typical clinical symptoms of PRES are headache, seizures, visual disturbances, and focal neurological symptoms [1]; our patient experienced headache, dizziness, and ataxia. The diagnosis of PRES is based on clinical symptoms and imaging findings [12]. Brain MRI in PRES reveals

Table 1 Laboratory results on admission

Examination	Value	Reference range
Blood test		
White blood cells (/ μ L)	9100	3900–9800
Neutrophil (%)	84.9	40–74
Lymphocytes (%)	8.5	19–48
Monocyte (%)	5.0	3.4–9.0
Eosinophil (%)	0.8	0–7
Basophil (%)	0.2	0–2
Red blood cells (/ μ L)	472×10^4	$427\text{--}570 \times 10^4$
Hemoglobin (g/dL)	13.5	12.0–17.6
Hematocrit (%)	41.6	39.8–51.8
Mean corpuscular volume (fL)	88.1	83–101
Platelets ($\times 10^3$ / μ L)	234	130–369
Total protein (g/dL)	4.8	6.4–8.2
Albumin (g/dL)	2.0	3.9–5.1
Total bilirubin (mg/dL)	0.33	0.2–1.0
Aspartate aminotransferase (mU/mL)	20	11–30
Alanine aminotransferase (mU/mL)	2	4–30
Lactate dehydrogenase (mU/mL)	679	110–220
Sodium (mEq/L)	140	138–145
Potassium (mEq/L)	3.6	3.6–4.8
Chloride (mEq/L)	101	100–110
Calcium (mg/dL)	5.6	8.6–10.1
Phosphate (mg/dL)	8.7	2.7–4.6
Blood urea nitrogen (mg/dL)	52	8–20
Creatinine (mg/dL)	11.6	0.65–1.07
C-reactive protein (mg/dL)	0.23	<0.20
Blood glucose (mg/dL)	125	70–100
HbA1c (%)	5.7	4.6–6.2
Thyroid stimulating hormone (μ U/mL)	1.823	0.35–4.94
Free thyroxine (ng/dL)	0.73	0.70–1.48
Free triiodothyronine (pg/mL)	2.92	1.71–3.71
Urine test		
pH	7.5	5–7.5
Specific gravity	1.028	1.005–1.025
Protein	3+	–
Glucose	3+	–
Red blood cell (/HPF)	1–4	0–4
White blood cell (/HPF)	10–19	0–4
24-h urine collection test		
Urinary volume (mL/day)	160	
Creatinine clearance (mL/min)	0.7	70–130
Protein (mg/day)	2045	20–60
Peritoneal dialysis related tests		
Weekly total Kt/V (/week)	1.49	
Renal Kt/V (/week)	0.42	
Dialysate Kt/V (/week)	1.07	
Dialysate cells count (/ μ L)	28	

HbA1c hemoglobin A1c, *HPF* high-power field

Fig. 1 Brain MRI on admission and on the 25th admission day. Diffuse hyperintense signals in the brain stem and bilateral cerebellar hemispheres are seen on FLAIR (arrows, **a**) and ADC-MAP (arrows, **b**) on admission. These abnormalities disappeared on repeat brain MRI on the 25th admission day (**c, d**). ADC-MAP apparent diffusion coefficient mapping, FLAIR fluid-attenuated inversion recovery, MRI magnetic resonance imaging

symmetrical, reversible hyperintensities seen as posterior brain regions on FLAIR and ADC-MAP images [3, 13]. Brain MRI in our patient revealed diffuse hyperintensities in the brain stem and bilateral posterior cerebellar hemispheres, which were consistent with the previous reports [3, 13]. The pathogenesis of PRES is considered failed cerebral autoregulation secondary to hypertension or endothelial dysfunction [1]. Other factors, including uremia, cytotoxic and immunosuppressive drugs, infection, autoimmune diseases, and ESA in chronic kidney disease contribute to development of PRES [1–4]. In the previous cases of PRES in patients undergoing PD, hypertension, inadequate PD efficiency, infections and administration of ESA were considered to contribute to development of PRES [4–8]. A cross-sectional survey showed that patients undergoing PD tended to be overhydrated and suffered

high blood pressure [14]. Additionally, ultrafiltration and sodium removal which contribute to develop hypertension have been reported to be lower in automated PD than continuous ambulatory PD [15]. Previous studies reported that 23.6% of PRES cases developed in the setting of infection, 80% of which was attributed to Gram-positive cocci [16]. Our patient had inadequate PD efficiency (weekly KT/V: 1.49) associated with automated PD and signs of hypervolemia including leg edema and enlarged cardiothoracic rate. He had been treated with ESA and his hemoglobin level was increased to 13.5 g/dL on admission. Also, the patient suffered from peritonitis due to Gram-positive cocci 20 days before the onset of PRES. Therefore, uncontrolled hypertension, fluid retention, uremia secondary to inadequate PD efficiency, administration of ESA, and prior peritonitis may have contributed to development of PRES.

Fig. 2 Patient's clinical course. The horizontal axis shows the number of days from admission

Regarding PRES treatment, prompt and aggressive antihypertensive therapy with intravenous antihypertensive drugs are recommended [17]. Also, specific treatments aimed at the underlying diseases are required [12]. In our patient, intravenous antihypertensive drugs were administered in addition to oral antihypertensive drugs, and we changed the PD prescription to improve ultrafiltration and remove uremic toxins. We did not perform hemodialysis for ultrafiltration to remove uremic toxins because of concern that our patient's brain injury might be exacerbated by rapid ultrafiltration and removal of uremic toxins and because of the increased risk of brain hemorrhage related to anticoagulants. These adverse effects following hemodialysis were reported in patients with other brain injuries including cerebral infarction and brain hemorrhage [18, 19]. However, our patient was successfully treated with a combination of intensive antihypertensive drugs and an improved PD prescription. Further studies are needed to investigate the ideal therapy for PRES in patients undergoing PD. In conclusion, we discuss a patient with PRES who was undergoing automated PD, and who was successfully treated with aggressive antihypertensive therapy combined with improved PD efficiency.

Compliance with ethical standards

Conflict of interest The authors have declared that no conflict of interest exists.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the insti-

tutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from the patient described in this case report.

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