Clinical Case Report

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A case report of a man on peritoneal dialysis having intractable hyponatremia

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Abstract

Rationale: Hyponatremia is one of the most common electrolyte disorders in patients on peritoneal dialysis (PD). It can be associated with severe consequences, higher morbidity and mortality. Therefore, hyponatremia should be assessed and monitored more carefully in these patients.

Patient concerns: A 55-year-old male PD patient progressively developed intractable hyponatremia was admitted to our hospital.

Diagnoses: The observation that no significant salt was lost in kidney and PD drainage prompted us to seek the underlying reasons for malnutrition and chronic inflammation. And cancer and tuberculosis were further suspected, although the exact nature at last was not clearly determined due to the unfavorable prognosis.

Interventions: The hyponatremia can hardly be improved by adjusting ultrafiltration close to zero, increasing sodium intake from 2.5 g to 7g, and nutrition counselling to maintain protein intake 0.9–1.2 g/kg/day and calorie intake 27–35 kcal/kg/day. Due to poor general situation, he received tentative anti-tuberculosis treatment instead of surgery for intracranial space-occupying lesion.

Outcomes: He died at home with conservative therapy.

Lessons: It highlighted the challenge for differential diagnosis and treatment in the hyponatremia on PD patient.

Abbreviations: CT = computed tomography, ECV = extracellular volume, ICV = intracellular cell volume, PD = peritoneal dialysis.

Keywords: hyponatremia, malnutrition, peritoneal dialysis

1. Introduction

Hyponatremia (serum sodium <135 mmol/L) is reported to be one of the most common electrolyte disorders seen in hospitalized patients. It occurs in 10% to 60% of patients on peritoneal dialysis (PD) for end-stage renal disease. It can be associated with poor clinical outcomes, including malnutrition, cognitive impairment, peritonitis, and death.[1,2] In patients on PD, fluid and electrolyte balance is dependent on nonrenal routes. Thus, the underlying pathophysiology and therapeutic strategy of hyponatremia in uremic patients associated with PD is suggested to be different from that of non-PD related hyponatremia. Common mechanisms leading to hyponatremia in PD can be multiple. These include water intake in excess of sodium and/or low water excretion (insufficient ultrafiltration); deficiency in extracellular fluid sodium (low sodium diet) and/or excess sodium loss (excessive ultrafiltration); phosphate/potassium deficit leading to water redistribution; free water excess due to an inadequate vasopressin suppression secondary to drugs (opioids, psychotropic medications), endocrinopathies (hypothyroidism, hypoaldosteronism), and other diseases (neurologic, pulmonary, or paraneoplastic ones); and change in the set point for serum sodium tonicity.[3,4] Despite these, the diagnosis and management of hyponatremia often remain problematic. Here, we report a male case with hyponatremia on PD patients likely due to malnutrition, highlighting the significant diagnostic and therapeutic challenges. The case showed chronic hyponatremia resistant to routine therapy and its underlying cause was hard to be determined. We suggested an algorithmic approach for the assessment of hyponatremia in patients on PD.

2. Case report

A 55-year-old male started automated PD 1 year ago because of chronic kidney failure (estimated glomerular filtration rate 6–8 mL/min*1.73 m^2) due to IgA nephropathy combined with diabetes nephropathy. He initially had residual diuresis of 1000 to 1500 mL/day. A total of 4000 mL of 1.5% lactate dialysate on cycler in 10-hour dwell time was prescribed. The average ultrafiltration was 200 to 300 mL/day, with weekly Kt/V of 1.8 to 2.0, including weekly peritoneal Kt/V of 0.6 to 0.8 and weekly residual renal Kt/V of 1.0 to 1.3. In the follow-up, he progressively developed hyponatremia. His serum sodium decreased from 134 to 121.3 mmol/L in 3 months, with total sodium removal ranging from 2.09 to 0.7 g/day. Hypoalbuminemia was exacerbated (28.6–20.4 g/L) with 5.88 to 12.34 g/day of total protein loss via dialysate and urine. His body weight significantly lost in the past year (70–58.5 kg). By multifrequency

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bioimpedance analysis, it was observed that his intracellular cell volume (ICV)/extracellular volume (ECV) ratio remained stable at about 0.69 to 0.75 but decreased to 0.65 on hospitalization. The hyponatremia can hardly be improved by adjusting ultrafiltration close to zero, increasing sodium intake from 2.5 to 7 g, and nutrition counseling to maintain protein intake 0.9 to 1.2 g/kg/day and calorie intake 27 to 35 kcal/kg/day. The hemoglobin decreased to 76 g/L from 112 g/L in spite of sufficient serum ferritin status and increased recombinant human erythropoietin treatment. After a 3-day history of nausea, dizzy, and hypotension, he was admitted to our hospital where he was found to be fully conscious. Serum osmolality was 292 mOsm/kg; urine osmolality was 284 mosm/kg. B-type natriuretic peptide level was normal. Additional endocrine analysis did not support hypoaldosteronism or hypothyroidism. No indications of cirrhosis was observed. His erythrocyte sedimentation rate was 37 mm/1sth, and C-reaction protein was 64.10 mg/L, but procalcitonin and tumor markers were nonsignificant. However, pituitary magnetic resonance imaging indicated irregular mixed signal (about 2.3×1.7 cm) in posterior left occipital lobe, and long T2 weighted imaging signal in surrounding cerebral white matter. Head enhancement computed tomography (CT) suggested it was likely to be malignant, as it showed a 2.5×2.0 cm low-density lesion in the posterior left occipital lobe with the CT value of 35 HU, and edema in surrounding tissues. Abdominal CT showed abdominal and pelvic effusion, and multiple pelvic enlarged lymph nodes. Chest enhancement CT indicated multiple patches, nodules, or cavities in the upper lobe and dorsal segment of the lower lobe of the lungs. Multiple enlarged mediastinal lymph nodes were also observed (Fig. 1 and Table 1). Due to poor general situation, he received tentative antituberculosis treatment instead of surgery for intracranial space-occupying lesion. However, a month later, he died at home with conservative therapy. Within the whole course of 1-year follow-up, it was difficult to normalize his hyponatremia, with highest serum sodium reaching 129.6 mmol/L. The study was approved by the medical ethics committee of Peking University. The patient and his family provided informed consent.

3. Discussion

This case highlights the significant diagnostic and therapeutic challenges for hyponatremia on PD patient. To evaluate hyponatremia in PD patients, it is necessary to rule out pseudohyponatremia and hyperosmotic hyponatremia (hyperglycemia, mannitol use, and renal failure); evaluate body weight (BW) change; and evaluate ICV/ECV ratio (Fig. 2).[3,4]

At first, pseudohyponatremia and hyperosmotic hyponatremia should be taken into account by considering formula including effective osmolality (mmol/kg H_2O) = $2 \times$ (serum Na (mmol/L) + serum K (mmol/L)) + serum glycaemia (mmol/L) and corrected (Na+) = measured (Na+) + $2.4 \times$ {[glucose(mmol/L) − 5.5 (mmol/L)]/5.5 (mmol/L)}. Thus, it was likely that the patient had hypo-osmotic hypo-natremia in fact.

BW loss during the development of hyponatremia reflects the total body water loss. Although ultrafiltration failure should be evaluated on the concept of sodium sieving and could lead to total body water gain. The patient had edema and polyserositis, but it seemed that he had no clinical signs of volume overload but possible signs of volume contraction (hypotension, BW loss, and no increase in B-type natriuretic peptide). Thus, he had hypo- or at least normal effective circulating blood volume. At this stage, syndrome of inappropriate antidiuretic hormone secretion or pituitary adenoma should be carefully differentiated. As routine laboratory tests endorsed in diagnostic criteria were not suitable for PD patient, fluid restriction showing therapeutic effect in syndrome of inappropriate antidiuretic hormone secretion, a volume-expanded state, could be an evidence. Instead, the patient

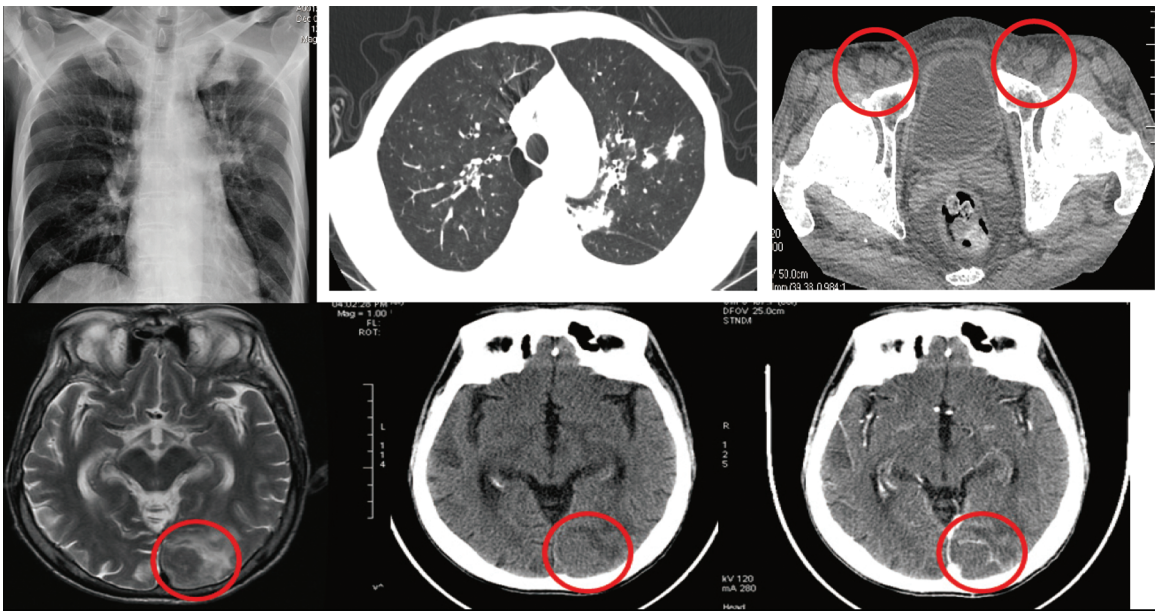

Figure 1. The medical images of the patient. Chest enhancement CT indicated multiple patches, nodules, or cavities in the upper lobe, and dorsal segment of the lower lobe of the lungs along with mediastinal multiple lymph node enlargement. Abdominal CT showed abdominal and pelvic effusion, and multiple pelvic enlarged lymph nodes. Pituitary MRI indicated irregular mixed signal (about 2.3×1.7 cm) in posterior left occipital lobe, and long T2WI signal in surrounding cerebral white matter. Head enhancement CT suggested the space occupying lesion likely to be malignancy, as it showed a 2.5×2.0 cm low-density lesion in the posterior left occipital lobe, with the CT value of 35 HU as well as edema of the surrounding tissues. CT = computed tomography, MRI = magnetic resonance imaging, T2WI = T2 weighted imaging.

Table 1

The clinical follow-up data of the patient.

Date	Weight, kg	Blood pressure, mm Hg	Serum sodium, mmol/L	Urine volume, mL	PD drainage volume, mL	Sodium removal in urine, g/d	Sodium removal in PD drainage, g/d	ICV/ECV	Peritoneal dialysis scheme	Hemoglobin, g/L	Fasting glucose, mmol/L	Albumin, g/L	Prealbumin, mg/L	Urine protein loss, g/d	PD protein loss, g/d	Sodium intake, g/d
2014/08/12			139							98	5.58	28.6				
2014/10/10	70		134							96	11.29	28.3				
2014/12/29	65	132/76	135	1107	240			0.72	1.5%*2000 mL*2 (10 h), rest empty	100	19.5	25.9	169.2			
2015/01/26	65.5	135/81	133	1600	321	1.56	0.75	0.75	↓	112	2.88	28.5	189.1	2.14	6.87	
2015/02/25	65.6	131/78	132.5	1885	151	3.24	0.57	0.75	↓	107	10.26	28.2	153.1	3.4	9.04	
2015/03/24	64.5	120/60	129.3					0.69	↓	84	13.21	24.2	144.4			2.5
2015/04/21	63.2		129			2.12		072	↓	84	12.05	25.6	155.8	1.29	7.78	3.2
2015/05/19	59		127.3	1450	260			1.04	1.5%*2000 mL*2 (10 h), long-dwell 2000 mL (14 h)	84	11.31	24.1	116			
2015/06/16	58.5	122/65	127.5	971	281	1.70	0.23		↓	91	6.63	22.2	129.1	1.19	8.52	3.6
2015/07/14	56.1		130.5	828	312	0.80	1.10		↓	88	13.67	24.1	150.3	0.76	7.86	5
2015/08/11	57	124/76	134	1033	437				↓	82	5.1	22.8	127.5			↓
2015/09/08	59		132	1186	322	1.26	0.83		↓	87	14.95	22.2	164.1	0.62	8.71	↓
2015/10/07	59.2		132	775	377				↓	99	5.31	22.9	159			↓
2015/11/09	60	140/85	127.1	620	4				Targeting 0 ultrafiltration	81	9.95	22.2	102.3			↓
2015/11/25	58.5	130/70	121.3	550	440	0.40	0.30	0.65	↓	76	8.4	20.4	79.4	0.65	5.23	6–7
2015/12/14	62	130/90	129.6	750	344				↓	85	5.72	23.1	70.9			↓

PD = peritoneal dialysis.

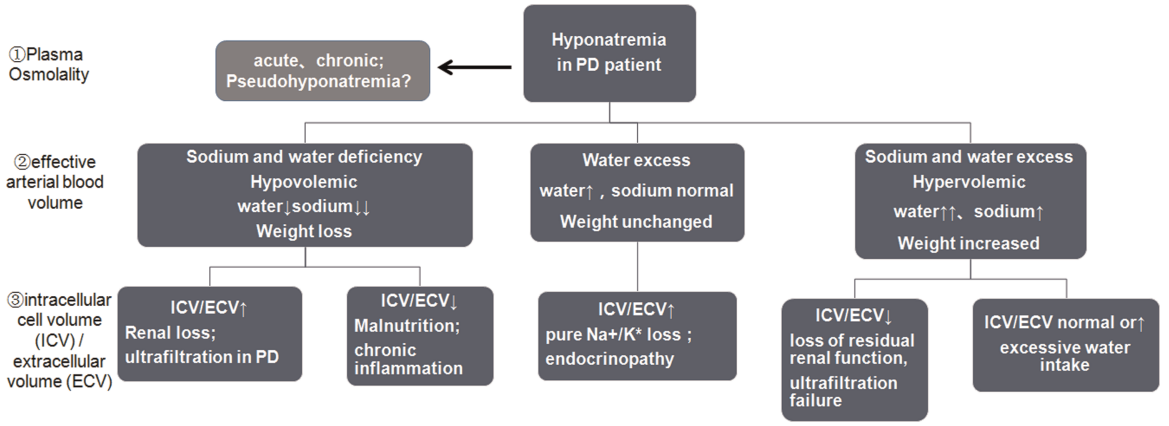

Figure 2. A diagnostic approach for hyponatremia on peritoneal dialysis patient.

showed no response but significant negative salt balance, suggesting alternative explanations to the problem.

Concomitant loss of extracellular Na^+, via the gastrointestinal tract, the kidney and PD, may be the major routes of Na^+ and water loss. This may be characterized by BW loss and increased ICV/ECV ratio.[3,4] However, in malnourished patients with low protein intake, intracellular K^+ and phosphate will be lost concomitantly to be electroneutral, producing a primary deficit of K^+ and phosphate.[4] Reduced intracellular tonicity will cause intracellular water shifting to extracellular components, subsequently diluting extracellular osmoles (mainly Na^+). PD patients with hyponatremia and an unexpected decrease of ICV or ICV/ECV ratio may point to malnutrition, which was consistent with the current case. However, it should also be noted that data from bioimpedance were advisory instead of mandatory.

The observation that no significant salt was lost in kidney and PD drainage prompted us to seek the underlying reasons for malnutrition and chronic inflammation. And cancer and tuberculosis were further suspected, although the exact nature at last was not clearly determined due to the unfavorable prognosis, reminding us to pay more attention to intractable hyponatremia in PD patients.[5–7] The case reinforces the notion that the specific reason for hyponatremia in certain cases is still vague despite extensive investigation, and malnutrition might be caused by or concomitant with multiple co-morbidities in patients on PD.

Author contributions

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Investigation: T. Su, Y. Yang.
Methodology: T. Su.
Writing – original draft: X-J. Zhou.
Writing – review & editing: J. Dong, X-J. Zhou.

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