Severe Hyperkalemia in a Peritoneal Dialysis Patient After Consumption of Salt Substitute

KEY WORDS: Hyperkalemia; salt substitutes; chronic kidney disease.

In normal individuals, potassium homeostasis is maintained very delicately, governed by the mechanisms of daily potassium consumption and renal excretion. Potassium balance can be maintained even if potassium intake is slowly increased from 60 mmol to 500 mmol daily. By contrast, in end-stage renal disease patients receiving peritoneal dialysis (PD) treatment, the main driving force for the elimination of potassium is the diffusive gradient between blood and PD fluid. Peritoneal dialysis fluid contains no potassium, and a typical continuous ambulatory PD (CAPD) regimen of 8 L eliminates approximately 40 mmol potassium daily, which is about the normal potassium intake of Chinese adults in Hong Kong (1). Thus, CAPD patients rarely develop hyperkalemia with a normal dietary intake of potassium. In fact, they are susceptible to hypokalemia if they continue the low-potassium diet from their pre-dialysis period.

Here, we report a PD patient who, having adopted a low-potassium diet, developed hyperkalemia stemming from the intake of a potassium-containing salt substitute.

CASE REPORT

A 57-year-old Chinese woman developed ESRD in 2008 because of renal tuberculosis and obstructive uropathy. Treatment with CAPD was started in January 2009. Her CAPD prescription was 3 daily exchanges with 2.5% dextrose solution. Ultrafiltration was about 800 mL daily, urine output was approximately 400 mL daily, and residual renal function was 1.5 mL/min/1.73 m². This patient had been very compliant with her low-potassium diet regimen—so much so that a daily 600-mg potassium chloride tablet was required to maintain normal serum levels of potassium. Her other medications included phosphate binders, erythropoietin, and angiotensin converting-enzyme inhibitor (ACEI).

In November 2010, the patient's routine blood tests revealed hyperkalemia. Serum potassium was 6.7 mmol/L; bicarbonate was 23 mmol/L. Total and peritoneal Kt/V were 2.11 and 1.78 respectively. No deterioration in residual renal function had been observed. The patient was asymptomatic, with normal blood pressure and pulse rate. Physical examination and echocardiogram were unremarkable. The patient denied an increase in the consumption of potassium-rich foods. The oral potassium supplement and ACEI were stopped. Ion-exchange resin was prescribed to lower the patient's serum potassium. Serum potassium normalized while the woman remained on the ion-exchange resin, but it bounced back to more than 6.0 mmol/L after resin treatment stopped.

The patient's food diary was once again reviewed. She was taking neither potassium-rich food nor herbal medicines. On further direct questioning, we found that, 3 weeks earlier, she had started using a new brand of salt that she had bought at a supermarket and that turned out to contain potassium (LoSalt: Klinge Foods, East Kilbride, UK). She had started substituting LoSalt for normal salt after she had been advised to reduce her sodium intake because of mild fluid overload. Unfortunately, she was not aware that LoSalt is potassium-containing, and the warning label on the package was too small to be noticeable. LoSalt contains two thirds potassium chloride and one third sodium chloride. The patient's estimated daily intake of LoSalt was 3750 mg—that is, 95 mmol of potassium daily.

After stopping the LoSalt, our patient experienced no further episodes of hyperkalemia.

DISCUSSION

In CAPD patients, hyperkalemia is less common than hypokalemia (2–4). In a local study by Szeto *et al.* (4), the prevalence of hyperkalemia in a cohort of 266 patients was 3%, much lower than the prevalence of hypokalemia (20.3%). Our center has similar prevalences of hyperkalemia and hypokalemia.

Apart from excessive intake of potassium-containing food, other causes of hyperkalemia in PD patients include inadequate dialytic potassium removal in patients who skip PD exchanges. Drugs that impair urinary potassium excretion, including ACEIs, angiotensin receptor blockers, and nonsteroidal anti-inflammatory drugs, may cause hyperkalemia in PD patients with significant residual renal function. Conditions causing potassium shifts from the intracellular to the extracellular compartment (for example, metabolic acidosis and rhabdomyolysis) may also cause hyperkalemia; but, by far, the most common cause of hyperkalemia in PD patients is excessive intake of potassium because of dietary indiscretion, or less commonly, use of nutritional supplements, herbal remedies, and potassium-containing salt substitutes (as in this case).

Use of a salt substitute that contains potassium has the combined advantages of reduced sodium intake and increased potassium intake. However, patients with renal failure or with diabetes mellitus and hyporeninemic hypoaldosteronism and patients taking ACEIs or angiotensin receptor blockers may fail to excrete the excess potassium load, resulting in life-threatening hyperkalemia.

John *et al.* (5) reported a 65-year-old patient with diabetes and heart failure who developed hyperkalemia (8.5 mmol/L) after consuming salt substitute. Doorenbos *et al.* (6) and Pal *et al.* (7) reported another two patients, one with ESRD on hemodialysis and the other with stage 4 chronic kidney disease who developed life-threatening hyperkalemia (>9 mmol/L) after consuming LoSalt.

Prevention of hyperkalemia is important in patients with chronic kidney disease. Patients are therefore often advised to follow low-salt diet that they may find tasteless. They may then turn to the so-called salt substitute, not knowing that, to produce the salty taste, most of these products replace the sodium with potassium. To prevent life-threatening hyperkalemia from misuse of salt substitutes, chronic kidney disease and dialysis patients should be educated to avoid using salt substitutes in their low-salt diet. In fact, we educate all of our dialysis patients to avoid salt substitutes. In the present case, the patient was unaware that LoSalt is a salt substitute. Thus, our case indicates a need for the local food governing authority to enforce clear warnings on the labels of such products.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

Terence Yip*
Winnie Wan
Pui-Chi Hui
Sing-Leung Lui
Wai-Kei Lo

Dr. Lee Iu Cheung Memorial Renal Research Centre
Tung Wah Hospital
The University of Hong Kong
Hong Kong SAR, PR China

*email: yipterence@gmail.com

REFERENCES

1. Kwok TC, Chan TY, Woo J. Relationship of urinary sodium/potassium excretion and calcium intake to blood pressure and prevalence of hypertension among older Chinese vegetarians. *Eur J Clin Nutr* 2003; 57:299–304.
2. Oreopoulos DG, Khanna R, Williams P, Vas SI. Continuous ambulatory peritoneal dialysis—1981. *Nephron* 1982; 30:293–303.
3. Spital A, Sterns RH. Potassium supplementation via the dialysate in continuous ambulatory peritoneal dialysis. *Am J Kidney Dis* 1985; 6:173–6.
4. Szeto CC, Chow KM, Kwan BC, Leung CB, Chung KY, Law MC, *et al.* Hypokalemia in Chinese peritoneal dialysis patients: prevalence and prognostic implication. *Am J Kidney Dis* 2005; 46:128–35.
5. John SK, Rangan Y, Block CA, Koff MD. Life-threatening hyperkalemia from nutritional supplements: uncommon or undiagnosed? *Am J Emerg Med* 2011; 29:1237.e1–2.
6. Doorenbos CJ, Vermeij CG. Danger of salt substitutes that contain potassium in patients with renal failure. *BMJ* 2003; 326:35–6.
7. Pal B, Hutchinson A, Bhattacharya A, Ralston A. Cardiac arrest due to severe hyperkalaemia in patient taking nabumetone and low salt diet. *BMJ* 1995; 311:1486–7.
doi:10.3747/pdi.2011.00198