Dear Editor,

Dialysis patients and patients with end-stage renal disease are at risk for hyperkalaemia because renal potassium excretion is reduced or completely absent. Elevated potassium concentration leads to reduced myocardial conduction, and can cause acute death because of arrhythmia.¹

A 67-year-old haemodialysis patient presented at the emergency room after a sudden collapse and paralysis of his legs. Electrocardiograms showed typical changes compatible with severe hyperkalaemia: peaking of the T wave, prolongation of the PR interval, loss of the P-wave amplitude and widening of the QRS complex (figure 1).² Laboratory analysis revealed severe hyperkalaemia. (K = 9.3 mmol/l; reference levels are 3.5-4.7 mmol/l). The patient was treated with calcium, insulin, glucose and sodium polystyrene sulphonate. Acute dialysis was arranged. Soon after treatment was initiated, the symptoms resolved and the electrocardiogram normalised.

A thorough analysis was performed to reveal the cause of this life-threatening hyperkalaemia. The patient was aware of potassium-rich products and adhered to his potassium-restricted diet. The only recent change in his diet was the addition of Becel pro.activ blood pressure spread®. His neighbour advised him to use the product to improve blood pressure regulation. Becel pro.activ blood pressure spread® is a low-sodium and potassium-enriched spread which helps maintain a healthy blood pressure.³ Without the consumption of the spread the patient had a potassium intake of 50 mmol per day. By measuring the weight of a slice of bread before and after addition of the spread we calculated an added potassium intake of 40 mmol a day. It seemed that this was the cause of the hyperkalaemia. No other cause of the sudden increase in potassium levels could be identified. After discontinuation of the Becel pro.activ spread, no excessive potassium levels were measured.

Some case reports in the past have been published in which hyperkalaemia was caused by salt substitutes.⁴⁻⁶ Nowadays not only potassium-enriched salt substitutes are produced but low-sodium products also use potassium instead of salt. Severe hyperkalaemia requires emergency treatment. Management of this condition is based on small studies and expert opinions. Reports focus on the level of potassium and do not describe mortality or cardiac arrhythmia after the treatment. Despite this, treatment with calcium, insulin and a beta agonist are considered effective.⁷⁻⁹

Causes of hyperkalaemia in dialysis patients are pseudohyperkalaemia, extreme potassium intake, inadequate dialysis, drugs (in patients with considerable residual renal function), acidosis, cell lysis, fasting and constipation.¹⁰ Excessive potassium intake is a very important preventable cause. Special attention should be given to low-salt and potassium-enriched products. These products are often recommended because they are assumed to lower cardiovascular risks. Indeed, effective dietary protocols for patients with hypertension include a low-salt and

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**Figure 1. Electrocardiogram at presentation showing changing characteristics for hyperkalaemia**
high-potassium diet. This can provoke excessive potassium intake and finally lead to life-threatening hyperkalaemia as in our patient. This case report adds that even potassium-enriched spread can cause a life-threatening hyperkalaemia. It should create an awareness of this preventable cause of hyperkalaemia among doctors and dieticians. Furthermore this is the first case report to describe Becel pro.activ as a source of hyperkalaemia.

REFERENCES