Intractable life-threatening hyperkalaemia in a diabetic patient

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Introduction

Hyperkalaemia is a common, but potentially lethal clinical problem. We wish to draw the attention of the reader to the unique ability of hypertonicity engendered by hyperglycaemia to induce severe hyperkalaemia especially in the setting of insulin absence or reduced insulin responsiveness.

Case report

A 44-year-old woman with a 10-year history of type 2 diabetes mellitus (DM) presented to our hospital emergency room complaining of fatigue and vomiting. She had been diagnosed with diabetic nephropathy 2 years earlier with a baseline creatinine of 4.5 mg/dl. On examination she had a blood pressure of 115/70 mmHg and pulse rate of 92/min with orthostatic changes. Her laboratory data at this time was remarkable for serum sodium of 139 mEq/l, potassium was 5.6 mEq/l with increased T wave amplitude, blood urea nitrogen was 120 mg/dl and serum bicarbonate of 17 mEq/l. An impression of volume depletion was made and the patient was given 2.5 l of 0.9% saline in 5% dextrose solution. For hyperkalaemia with ECG changes she was given calcium gluconate and one ampoule of 50% dextrose (25 g). However, insulin was not given with the 50% dextrose in error.

Four hours later, repeat chemistries revealed a serum potassium concentration of 9.2 mEq/l. Sodium polystyrene sulfonate (kayexalate) (60 g) was given orally with monitoring of serial serum potassium values. Despite giving repeat oral doses of 30–60 g sodium polystyrene sulfonate and albuterol 25 mg by nebulizer over the next 6–8 h, serum potassium remained dangerously high until emergent dialysis was done 10 h later as illustrated in Figure 1.

Fig. 1. Serial serum potassium levels over time.
volume replacement. This was clearly aggravated by administration of 50% dextrose without concomitant insulin in the treatment of hyperkalaemia. Hyperkalaemia resolved with haemodialysis. The patient continues to follow-up at our chronic haemodialysis facility. The fact that an intravenous glucose infusion can precipitate severe hyperkalaemia in diabetics, is well documented [1,2]. This response is clearly paradoxical as glucose infusion in non-diabetic subjects engenders hypokalaemia due to insulin release [2]. The mechanism for the hyperkalaemic response to glucose infusion in diabetics is unclear but it is thought to involve hypertonicity [3,4], hypoaldosteronism and possible effect of glucagon [5]. The glucose infusion in the setting of absolute or relative insulin deficiency (DM) acted as effective osmole to increase extracellular fluid (ECF) osmolarity. The resulting cell shrinkage increases potassium efflux from the cell. A role for underlying hypoaldosteronism in this hyperkalaemic response is underscored by the fact that prior desoxycorticosterone (DOCA) therapy prevents this response [2]. Glucose infusion in diabetic subjects also paradoxically elevates glucagon levels, a hormone that may also increase potassium efflux [5,6]. The survival of this patient, in the face of severe hyperkalaemia, is really a testament to the relative resistance of patients with chronic renal failure to hyperkalaemia [7].

**Teaching point**

Extreme caution should be exercised in giving glucose and/or hypertonic solutions (mannitol, amino acids, etc.) to diabetic patients especially those with renal insufficiency, as the risk of iatrogenic hyperkalaemia is high. For the same reason, insulin must be given in pari-passu with dextrose in the emergency management of hyperkalaemia with ECG changes.

**References**