

Rapid resolution of life-threatening hyperkalaemia in diabetic ketoacidosis with intensive insulin therapy

Soniya Abraham ¹, Jay Parekh,¹ Lakshmi Polisetty,¹ Kulothungan Gunasekaran²

¹Internal Medicine, Bridgeport Hospital, Bridgeport, Connecticut, USA

²Division of Pulmonary Diseases and Critical Care Medicine, Bridgeport Hospital, Bridgeport, Connecticut, USA

Correspondence to

Dr Soniya Abraham;
soniya7ab@gmail.com

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DESCRIPTION

A 28-year-old man with a history of type 1 diabetes mellitus on a continuous subcutaneous insulin infusion pump presented to the emergency department with generalised malaise and elevated blood sugar. His insulin pump was found to be turned off, which the patient was not aware of. The initial physical findings included a blood pressure of 124/70 mm Hg and a pulse rate of 89 beats/min. His plasma glucose was more than 1500 mg/dL (normal range: 70–100 mg/dL), serum sodium was 104 mmol/L (normal range: 136–144 mmol/L) and potassium was 9.2 mmol/L (normal range: 3.4–4.8 mmol/L). Arterial blood gas analysis showed a pH of 7.15, bicarbonate of 9.9 mmol/L and a base excess of –18.0 mmol/L. The initial ECG revealed typical features associated with hyperkalaemia with peaked T waves and a prolonged QRS interval (Figure 1A). The patient was found to be in diabetic ketoacidosis (DKA) and was admitted to the medical intensive care unit. He received 3 L of normal saline, 1 g of calcium gluconate and 100 mEq of sodium bicarbonate during the course. He was started on an intravenous insulin infusion, and labs showed marked improvement in his plasma glucose to 649 mg/decilitre and serum potassium to 4.2 mEq/L after 6 hours of therapy. Repeat ECG showed

Learning points

- ▶ Extreme hyperkalaemia with marked ECG changes can occur in patients with diabetic ketoacidosis (DKA) with no pre-existing renal disease.
- ▶ In such a situation, severe hyperkalaemia can be treated with intensive insulin therapy without urgent haemodialysis.
- ▶ Physicians should be aware of deleterious potassium changes in DKA, and prompt interventions can avoid fatal arrhythmias.

normalisation of the T waves and QRS interval (figure 1B).

DKA is an endocrinology emergency characterised by hyperglycaemic, metabolic acidosis and ketosis.¹ Insulin deficiency combined with an excess of counterregulatory hormones such as glucagon, catecholamines, and cortisol results in hyperglycaemic and ketoacidosis, leading to DKA. DKA usually presents with normal or mildly elevated potassium levels despite depleting total body potassium stores secondary to kaliuresis, vomiting and decreased intake.² Severe hyperkalaemia is a rare and life-threatening emergency that requires urgent intervention; otherwise, it could result in fatal cardiac arrhythmias.¹ Severe hyperkalaemia can be managed acutely by antagonising cardiac toxicity by stabilising the myocyte membrane using calcium salts, shifting potassium from extracellular compartment to intracellular compartment, and removing potassium directly plasma using haemodialysis. Insulin and beta-adrenergic agonists stimulate the Na/K ATPase pump, which shifts potassium to the intracellular compartment in exchange for sodium. Haemodialysis remains definite therapy for extreme hyperkalaemia (K>8 meq).^{3,4} Our case shows the resolution of severe hyperkalaemia associated with DKA by intensive insulin therapy alone without the need for haemodialysis.

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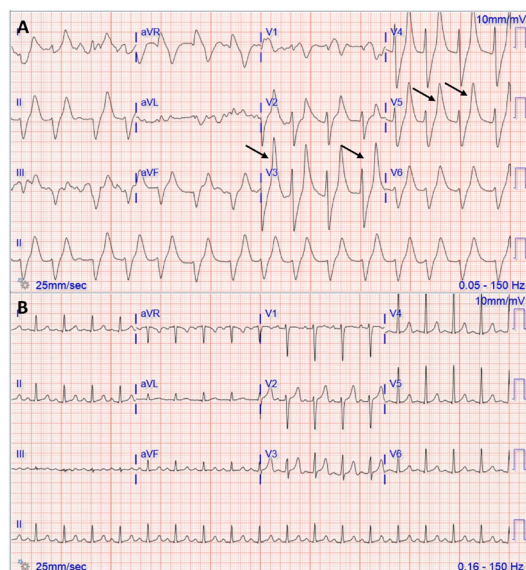


Figure 1 (A) ECG at the time of presentation showing peaked T waves with prolonged QRS interval consistent with hyperkalaemia. (B) ECG after 6 hours of intensive insulin therapy showing normalisation of T waves and QRS interval.



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ORCID iD

Soniya Abraham <http://orcid.org/0000-0003-1596-258X>

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