Arrhythmias in an obese patient with diabetic ketoacidosis

Edouard Mills, Mushtag Rahman

Department of Diabetes and Endocrinology, Northwick Park Hospital (London Northwest Healthcare NHS Trust)

Introduction

- Diabetic ketoacidosis is one of the most common medical emergencies in diabetic patients.
- Changes in patient demographics, with increasing prevalence of obesity and other insulin-resistant states, have led to changes in the management of DKA to fixed rate intravenous insulin infusions calculated on weight.

Discussion

- The Joint British Diabetes Societies guidelines suggest that giving more than 15 units of insulin per hour should be only with specialist advice.
- Insulin administration causes shift hypokalaemia due to \bullet redistribution of potassium from the extracellular to the intracellular fluid compartment. Failure to acknowledge and properly manage hypokalaemia in DKA can result in severe, symptomatic hypokalaemia with detrimental effects on the neuromuscular and cardiopulmonary systems.
- We present the case of an obese patient who developed hypokalaemia and hypophosphataemia, which later led to new atrial flutter with a rapid ventricular response. This persisted even after correction of these biochemical abnormalities.

Case description

- 38 year old, obese man (BMI 60 kg/m2), who presented to the Emergency Department with severe osmotic symptoms.
- Admission investigations confirmed ketoacidosis (pH 7.06, bicarbonate 7.7 mmol/L, base excess -18.7 mmol/L, potassium 3.8 mmol/L, blood ketones 5.7 mmol/L and glucose 18 mmol/L) and a new diagnosis of diabetes mellitus made.
- Admission ECG was sinus rhythm.
- Managed in a level 2 facility with fixed-rate intravenous insulin, 0.1 U/kg, as per national guidelines: 18 U/h, weight 182 kg.

- Hypokalaemia causes cardiac myocyte hyperpolarisation leading to increased excitability and delayed repolarisation. Notable ECG findings in hypokalaemic patients include early T-wave flattening with subsequent ST-T depression, U waves, and prolongation of the QU, QT and PR intervals. All of these electrophysiological changes can result in atrial and ventricular arrhythmias.
- Mild to moderately severe hypophosphataemia is usually asymptomatic. Major clinical sequelae usually occur only in severe hypophosphataemia; 0.32 mmol/L. If severe hypophosphataemia is present for longer than 2-3 days, serious complications can be seen, including rhabdomyolysis, respiratory failure, acute haemolytic anemia, and fatal arrhythmias.
- Our patient's tachyarrhythmia will have been driven by electrolyte deficiencies, hypokalaemia more so than hypophosphataemia; the high dose of insulin will have precipitated both.

Clinical course

- His progress was complicated by hypokalaemia and hypophosphataemia (Figure 1 and Figure 2)
- Subsequently developed new atrial flutter with a rapid ventricular rate
- Intravenous potassium and phosphate were given in an attempt to manage his tachyarrhythmia, but the insulin rate was unchanged
- Later transferred to the Cardiac Care Unit and required DC cardioversion as the atrial flutter continued despite correction of electrolyte imbalance.

• The hypophosphataemia in DKA tends to be due to inter-compartment shifts rather than absolute deficiency secondary to insulin; its association with correction of atrial tachyarrhythmias is less clear than hypokalaemia.

Learning point

• We recommend that greater care should be exercised with the dosing of insulin in obese patients with DKA in order to avoid dysrhythmias.

References

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Figure 1: Phosphate result from admission

Figure 2: Potassium results from admission

