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Euglycemic Diabetic Ketoacidosis – A Therapeutic Challenge

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Introduction

Euglycemic Diabetic ketoacidosis is a clinical syndrome occurring in both Type 1 & Type 2 DM. Euglycemic DKA is characterized by euglycemia (blood glucose less than 250 mg/dl) with severe metabolic acidosis and ketonemia. The incidence of Euglycemic DKA has increased with the introduction of sodium-glucose transporter 2 inhibitors. It also presents a challenge for physicians due to its various etiologies and the presence of normal glucose levels which often results in a delayed diagnosis. Euglycemic DKA is an uncommon diabetic complication associated with several risk factors such as fasting, surgery, pregnancy and recently the use of SGLT2 inhibitors. Euglycemic DKA can lead to serious complications if not recognized early and treated appropriately with fluids, dextrose and insulin.

Case Summary

A 37-year-old male patient, a case of Type 2 DM came with complaints of abdominal pain and vomiting for 2 days. He was on SGLT2 inhibitors (empaglifozin) for the last one month. The patient with the above-mentioned complaints initially went to a local hospital where he was diagnosed with Euglycemic DKA, He was treated conservatively initially but as it failed to correct the metabolic acidosis, he also underwent 2 sessions of hemodialysis. However, there was no improvement in his general condition, hence he was referred to us for further management. At admission to our hospital patient was conscious and oriented. His pulse was 120/ min and blood pressure was 100/60 mm Hg. He was tachypnoeic at rest with a respiratory rate of 38/ min. His oxygen saturation in room air was 92%. ABG showed uncompensated metabolic acidosis with pH of 7.10 and a bicarbonate value of 5.9mmol/l. His blood glucose was 166 mg/dl, serum ketones were high -127mmol/l. He also had hyponatremia and hypokalemia. The patient was intubated in casualty due to respiratory distress and then shifted to the medical ICU. He was treated with a 5% dextrose infusion, insulin infusion and IV bicarbonate infusion. He developed hypotension with a BP of 90/60 mmHg and hence he was started on inotropes. Subsequent arterial blood gases showed no improvement and even deterioration in pH to 7.05 and bicarbonate of 5.4 mmol/ l. He developed generalized tonic-clonic seizures and his sensorium deteriorated, hence he had to be intubated. He was started on mechanical ventilation and anti-epileptics were also administered. A nephrologist's opinion was taken for refractory acidosis and advised Continuous renal replacement therapy if conservative management fails. We reviewed the literature on the management of Euglycemic DKA changed to high-calorie glucose infusion (10% dextrose) with insulin and continued the aggressive fluid resuscitation. Within six hours of starting 10% dextrose with concomitant insulin infusion, the recalcitrant acidosis improved. The pH corrected to 7.470 and bicarbonate to 27mmol/l. His vitals improved, and he was weaned off inotropes and ventilatory support. Once his general condition improved, he was shifted to the ward.

Serial ABG VALUES BEFORE 10% DEXTROSE

pH	*7.033*	*7.058*	*7.059*
HCO3(mmol/l)	*6.5*	*8.5*	*8.9*
PaCO2 (mmHg)	*25*	*31*	*21.9*
Blood Glucose (mg/dl)	*205*	*200*	*196*

Serial ABG VALUES AFTER 10% DEXTROSE

pH	*7.472*	*7.474*	*7.515*
HCO3(mmol/l)	*27.1*	*35.5*	*31.8*
PaCO2 (mmHg)	*37.6*	*49*	*39.7*
Blood Glucose (mg/dl)	*147*	*148*	*141*

We continued the high-caloric infusion along with insulin infusion for 24 hours. Once the patient resumed oral intake, insulin was continued as a basal-bolus regimen. The patient was discharged in a stable condition.

Discussion

Euglycemic DKA due to SGLT2 inhibitors has 2 important pathophysiological features. Firstly, a significant calorie loss from the increased glycosuria brought by the SGL2 inhibitors leads to lipolysis and the generation of ketone bodies. The second contributory factor is the imbalance between insulin and glucagon levels. There is an increase in glucagon levels in response to the decrease in blood glucose levels due to glycosuria brought about by SGLT 2 inhibitors. The increase in glucagon levels provides a strong drive to promote the production of ketone bodies. There is also a concomitant decrease in insulin levels following a reduction in blood glucose levels. High caloric glucose infusion and tight glycemic control are key factors in the management of the SGLT2 inhibitor-induced euglycemic DKA. Timely intervention prevents the use of dialysis in such patients and protects the patient from its psychosocial impact and financial burden.

References

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