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EUGLYCEMIC DIABETIC KETOACIDOSIS WITH REFRACTORY METABOLIC ACIDOSIS IN A PATIENT WITH T2 DM ON EMPAGLIFLOZIN AND INTERMITTENT FASTING

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Maria Roselle Vicencio and Abigail Uy-Canto
Asian Hospital and Medical Center, Paranaque, Philippines

BACKGROUND
Euglycemic Diabetic Ketoacidosis (EuDKA) is a clinical triad of normal blood glucose levels, ketonemia, and high anion gap metabolic acidosis. It is less commonly associated with the use of Sodium-Glucose Co-Transporter 2 inhibitors (SGLT2i) such as empagliflozin. Due to the introduction of SGLT2i in the treatment of T2DM and HF, there is an increasing incidence of EuDKA.

CASE
We report a case of a 28-year-old female recently diagnosed with diabetes mellitus type 2 (T2 DM) maintained on semaglutide 0.25 mcg subcutaneously once a week and empagliflozin 10 mg once daily presenting with a three-week history of easy fatigability, generalized body weakness, nausea, vomiting and loss of appetite. At the ER, the patient experienced abdominal pain, nausea, and vomiting. The patient was managed as a case of severe refractory metabolic acidosis secondary to EuDKA. The patient was intubated for 5 days. The management consisted of insulin drip, hydration, renal replacement therapy (RRT), and bicarbonate drip. Resolution of acidosis with a pH of 7.42 and anion gap of 10.1 was achieved on the 8th day of admission.

CONCLUSION
This case reports a rare complication of SGLT2i in a patient with T2DM with retractable severe metabolic acidosis that can be effectively managed with RRT and cessation of SGLT2i. It also highlights the importance of giving D5-containing IV fluid for continued insulin administration which is important to reverse ketoacidosis while preventing hypoglycemia. Monitoring of serum ketones and serum electrolytes levels are crucial in the management of a patient with intractable acidosis needing mechanical ventilation and renal replacement therapy.

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DOES TIME TO DEVELOP POST-TRANSPLANTATION DIABETES PREDICT TIME TO GRAFT LOSS? AN ANALYSIS OF PATIENTS 20 YEARS POST-KIDNEY TRANSPLANT

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Inês Vieira, Sofia Lopes, Carla Batista, Margarida Bastos, Dírcea Rodrigues, Luísa Ruas, Isabel Paiva
Centro Hospitalar E Universitário De Coimbra, Coimbra, Portugal

INTRODUCTION
Post-transplantation diabetes (PTDM) is common after solid organ transplantation. There are data to suggest that this complication may influence the transplant outcomes, namely the risk of graft loss.

To analyze whether there is a relationship between age at diagnosis and time to graft loss.

METHODOLOGY
Retrospective study with patients transplanted between 1989-2001 who developed PTDM.

RESULTS
We included 41 patients who had transplantation 24.7 (± 2.4) years ago. Majority (68.3%) were males with a mean age at transplantation of 46.5 (± 11.3) years. The average (years) of diagnosis of DMPT was 4.3 (± 5.3). C-peptide was detectable in all patients. Diabetes autoimmunity was negative in 96.4% of patients, with 1 patient having low anti-GAD65 titers. All patients were treated with therapeutic lifestyle measures, 78.0% were started on insulin therapy (on average 6.5±7.3 years post-transplantation) and 14.6% were started on oral antidiabetics (22.7±6.1 years post-transplantation). The median HbA1c in the 1st 5 years of DMPT was 6.9 ± 1.5%, while the median Hba1c in the 2nd, 3rd, and 4th year was 6.9 ± 1.1% (n = 28), 7,2 ± 1.2% (n = 21) and 6.9 ± 1.1% (n = 12), respectively. To date, 73.2% had graft loss (mean 11.3 ± 6.1 years post-transplant) and 55.3% died (12.2 ± 6.2 years post-transplant). There was a weak but significant correlation between latency to develop DMPT and time to graft loss (r = 0.419, p=0.021).

CONCLUSION
Our study suggests a positive correlation between time to develop PTDM and graft loss. However, it is not possible to establish causality, as earlier appearance of DMPT may be influenced by the doses of immunosuppressants in patients at a greater risk of graft loss.