Abstract

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SGLT2 Inhibitors and ketogenic diet: a dangerous association in T2 Diabetes Mellitus patients

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Background: Sodium glucose co-transporter 2 inhibitors (SGLT 2- I) are a novel class of oral antidiabetic drug used for Type 2 Diabetes Mellitus (T2DM) treatment. Their action is carried out mainly enhancing glycosuria and switching from glucose to lipid catabolism. Currently SGLT2 inhibitor is highly used for their protective rule on renal and cardiac output in patient suffering from DM2. Genital mycotic infection is described as the most common adverse drug reaction. In 2015, FDA issued a warning regarding risk for this class of inducing euglycemic diabetic ketoacidosis (EDKA) but no attention has been paid on diet modification in patient being treated with SGLT2 inhibitors.

Case Report: A 52 year-old man presented to emergency department with abdominal pain, fatigue and vomiting Formerly obese, he developed T2DM2 ten years before and was in treatment with SGLT 2 inhibitor/biguanide association (empaglifozin/ metformina 12,5/1000 mg 2/day) since one year. Of note, he referred to have started a ketogenic diet three days before the beginning of symptoms. Laboratory evaluation revealed a severe metabolic acidosis with elevated anion gap (27.7): ph 6.89, pCo2 9.2 mmHg, HCO3 1.8 mmHg, lactate 3.2 mmol/L and chetonuria (80 mg/dl). Slightly altered glucose level was observed, 220 mg/ dl defining a case of EDKA. Additionally leucocytosis neutrofila was observed and atrial flutter evidenced at ECG with frequency of 150 bpm. High insulin level (87) and low c peptide value (0.9) were found. Three days after patient discharge, c peptide value was raised to 3.6 confirming diagnosis of type 2 DM. Anti-diabetic treatments was stopped. Treatment was started as per protocol with 0.9% saline solution for fluid resuscitation, insulin and glucose infusion, early potassium replacement and alkali administration for severe deprivation. Symptoms resolved in few days and hemogasanalysis performed three days after hospitalization showed normalization of all parameters with ph.7.4, Pco2 36 mmHg, lactate levels 0.6 mmol/L, hco3 23mm Hg, glucose 190 mg/dl and absence of chetonuria.

Conclusions: In the case presented the risk of EDKA due to SGLT2-I treatment has been significantly increased by ketogenic diet. The association between a very low carbohydrate diet with drug-induced glycosuria and increased ketones production because of the increased glucagon levels induced keto-acidosis: in the absence Of any carbohydrate reserve fat is only source of energy It is important for care provider to adapt dietary modification to diabetes medication in-patient with T2DM. According to last reports, patient on SGLT2 inhibitors treatment should be advised to avoid this kind of diet. Further recommendation is needed to allow a safe management of this treatment.