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Case Report

Metabolic Acidosis in a Diabetic Patient Treated by Jardiance

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Abstract

Patients affected by Type II Diabetes are often subjected to multi-drug therapy, thus they are at risk of developing several side effects. Metabolic acidosis is one of these. It has often been identified as a side effect during treatment with Metformin, in which case it is usually associated with hyperlactactemia. Empagliflozin is an anti-diabetic drug known to reduce cardiovascular complications as well as renal function declinein patients with Type II diabetes. Recent studies have shown that this class of antidiabetic drugs harbour a risk of inducing euglycemic diabetic ketoacidosis, especially in case of an acute condition (infection, acute phase of a chronic illness) but also in the case of poor compliance to treatment. We present a case of a 75-year-old patient who presented with idiopathic metabolic acidosis without hyperlactatemia, with a preserved renal function and with ketonuria, following treatment with Empagliflozin.

Keywords: Diabetes; Hyperlactactemia; Jardiance; Metabolic acidosis

Learning Points

This is a clinical case of special interest, especially for recently graduated doctors. 75 year old patient presented idiopathic metabolic acidosis without hyperlactatemia, with a Preserved renal function and ketonuria after treatment with empagliflozin.

Introduction

Diabetes is a widespread disease in the whole world. In 2014, it was estimated that 422 million adults were affected by Diabetes. The worldly prevalence for Diabetes (normalized according to age) has nearly doubled since 1980, going from 4.7% to 8.5%. Moreover, the WHO has pointed out that 9/9% to 21% of the world's inhabitants present a state of pre-diabetes. Since 39% of the world's adults are overweight with 13% being obese, screening for pre-diabetes should be carried out systematically in all admitted patients. In diabetic patients, medically induced metabolic acidosis is often identified as being hyperglycemic with hyperlactactemia. In case of acidosis (arterial pH <7.3) during treatment with Jardiance, it is euglycemic (glucosemia <250 mg/dL); lactate is not increased but instead there is a reduction of bicarbonates (<18mEq/L) [1]. As for the physiopathology, this condition finds its explanation partly in the increase of regulatory hormones such as Glucagon, Cortisol and Epinephrine and on the other hand in the reduction of insulinemia. This triggers lipolysis, leading to ketogenesis. The incidence of euglycemic metabolic acidosis in diabetic patients has increased since the introduction of SLGT2s, especially Empagliflozin [2,3].

Case Description

In February 2021, a 75-year-old Caucasian female patient presented to our Hospital for faintness, without loss of consciousness. She had recently been bereaved. It was reported that in the last days, she had been eating and drinking too little. She also complained of nausea without vomit and pollakiuria since 72 hours. Her medical history included the implantation of a pacemaker and type II Diabetes, which was treated by Empagliflozin and Linagliptin. Neurologic examination and cardiovascular evaluation were both reassuring. As of the cerebral CT Scan, it only showed fronto-temporal cerebral atrophy. Arterial blood gas analysis was carried out in the emergency room and showed euglycemic metabolic acidosis without hyperlactatemia. Renal function was preserved. Ketonuria was present. HbA1c was measured at 9.4%. Empagliflozin treatment was interrupted and the patient was treated by hydration IV and Sodium Bicarbonate 8.4%. Clinical and biological evolution was satisfactory. The patient's therapy was modified following the diabetologist's consent; treatment was undertaken with Metformin + Gliclazide. Testing of HbA1c was scheduled three.

Discussion

Recent studies have demonstrated the induction of euglycemic metabolic acidosis in diabetic patients treated with Empagliflozin. A recent study publication in the Cureus Journal of Medical Science presented another case of euglycemic metabolic acidosis in a 23-year-old patient affected by Covid19 [4]. The Current Diabetes Review published a review in 2017 which elaborated the various etiologies of metabolic acidosis, among which Empagliflozin was included. The European Journal of Pharmacology published a review that also demonstrated an increase of the incidence of medical-induced metabolic acidosis in diabetic patients. It also showed that there was a higher incidence in patients treated with bitherapy (Metformin+Empagliflozin) when compared to patients treated with Metformin alone [5]. Another study that was published in the JDI (Journal of Diabetes Investigation) in April 2021 addressed a case of a 59-year-old patient who suffered from Diabetes since 12 years. She had been treated by insulin and Empagliflozin. He had developed empyema which had led

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to debridement by thoracoscopy and intrathoracic washing. During surgery, the patient had presented euglycemic acidosis which had resolved in the 24 hours following surgery, thanks to early treatment [6].

According to the FDA, between May 2013 and June 2015, 73 cases of ketoacidosis requiring admission into hospital were reported in patients treated with SGLT2 inhibitors. The mean period before the presentation of the first symptoms is of 43 days (between 1 and 36 days). The triggering factors had been identified in just half of the cases [7].

Conclusion

There are multiple causes that lead to metabolic acidosis. Nonetheless, an increase in the incidence of acidosis has been noted in relation to therapy with SGLT-2 inhibitors. These drugs had already proven to be useful, especially for patients who harbor renal and cardiac diseases, but nowadays it is necessary to assess the risk of side effects, especially in the case of an acute phase (infection, surgery, acute phase of a chronic illness,…). Tools such as the "STOP DKA Protocol" have recently been created for this purpose. Therefore, it is crucial to evaluate the presence of ketones in the serum and in the urine in all the patients under treatment with SGLT-2 inhibitors who present metabolic acidosis.

Disclosure

Conflict of interest

The authors declare no conflict of interest.

Patient consent

Yes.

Acknowledgment

There is no other contribution.

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