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Case Report

Euglycemic Diabetic Ketoacidosis and Adrenal Insufficiency

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ABSTRACT

Euglycemic Diabetic Ketoacidosis (EGDKA) is somewhat a masked illness. It is a diagnostic challenge for the treating physician, as it can hide behind the most important factor leading to a suspicion of a ketoacidosis state – Hyperglycemia. Normal sugar levels, along with symptoms such as nausea, vomiting, and normal or decreased appetite should also raise a strong suspicion. SGLT-2 Inhibitor is already widely reported as the most common etiological factor leading to EGDKA. But we report a somewhat uncommon presentation of EGDKA in a patient with Adrenal Insufficiency. Much less reported but still prevalent, Adrenal Crisis induced EGDKA is a severe condition, but easily manageable if identified early and treated aggressively.

KEYWORDS: Adrenal Crisis, SGLT-2 Inhibitors, Polyarthritis, Steroid dependency, High anion gap acidosis

INTRODUCTION

Euglycemic Diabetic Ketoacidosis is a rather rare complication of Diabetes Mellitus, which is steadily increasing in recent years. The primary cause of Euglycemic Diabetic Ketoacidosis (EGKA) can be narrowed down to the increase in Insulin/Glucagon ratio. Patients with EGKA can present with severe metabolic acidosis and presence of abnormally high ketones in blood. Despite occurring in patients suffering from diabetes mellitus, there is euglycemic state of the patient (Blood glucose 250 mg/dl). This proposes a challenge especially in early diagnosis or identifying ketosis.[1] Recently the incidence is steadily rising because of the introduction of Sodium Glucose Transporter -2 (SGLT-2) as oral hypoglycemic agents.[2,3]

CASE REPORT

A 47 years old female presented to Pacific Medical College and Hospital, Udaipur with complaints of generalized weakness since 15 days and difficulty in breathing with nausea and vomiting since 3-4 days. Patient had history of polyarthritis for last 10 years and she was taking oral Betamethasone 0.5 mg since then. Patient had increased the dosage to 1.5 mg since last three years. About 15 days back before the day of admission patient developed vague complaints of fatigue, noticed weight loss and nausea. She went to a local physician for the complaints and got diagnosed with T2DM. She was prescribed Voglibose along with Metformin and Glimepiride combination. Patient had stopped taking her steroids since last three days abruptly, and subsequently she developed fever with chills and vomiting.

Her systemic examination was unremarkable, and on admission random blood sugar was 190 mg/dl. Her HbA1C level was 12.1%. She developed severe metabolic acidosis

(pH 7.22, pCO2: 28.0, cHCO3: 11.1).Despite Euglycemic state a suspicion of Euglycemic Diabetic Ketoacidosis was made as her ABG was suggestive. Her Urine examination was strongly positive for Ketones (+++).Blood sample was sent for serum cortisol level. In spite of severe acidosis and stress of illness her serum cortisol was low (4.30), which also contributed to the euglycemic state.

She was provisionally diagnosed as a case of T2DM with polyarthritis (Steroid dependent), euglycemic diabetic ketoacidosis and adrenal crisis (abrupt steroid withdrawal).

An insulin infusion was initiated to keep the sugar levels according to therapeutic target. Her steroids were restarted and IV fluids, electrolytes management and appropriate antibiotics were initiated. Patient responded well to the treatment. Subsequent urine examination showed absence of ketones and ABG was stabilized. Patient was shifted to ward 5 days later and discharged on the 10th day.

DISCUSSION

Euglycemic Diabetic Ketoacidosis was first reported in 1973.[4] But, the sugar level cut off has been further reduced to 250.[5]The primary mechanism inducing EGDKA is because of decrease in serum insulin levels and increase in glucagon levels along with other counter regulatory hormones. This results in the increase of Insulin/Glucagon ratio, which subsequently manifests as ketoacidosis. This high anion gap acidosis instigates a respiratory compensation which as seen in the present patient and can result into dyspnea.[6,7] The contrast here can be loss or decreased appetite mainly due to volume reduction along with blood sugar <250 mg/dl.[8] Normally the counter-regulatory hormones increases in DKA and they manifest as hyperglycemia. But in this case serum cortisol was low compared to the stress of the illness, which can be the cause of euglycemia.

SGLT-2 Inhibitors are the new class of oral hypoglycemic drugs. SGLT-2 participates in a competitive inhibition at the proximal convoluted tubule; they stop the glucose re-absorption (Filtered from urine). This transfers the metabolism which was utilizing glucose initially to lipid, mimicking a fasting/starvation like situation.[9] Old Diabetes with poor control and lower levels of BMI are more predisposed to developed EGDKA.[10]

What was observed here is that despite being diabetic her appetite was reduced and still she was in ketosis. We replaced the steroid as steroid replacement is also of paramount importance along with glycemic control. So this lights the fact that steroid withdrawal can also be a precipitating factor of Ketosis. We should suspect EGDKA in patients with adrenal insufficiency also. There is scant published knowledge available for the same.

Nicole et al. studied 5 cases admitted for diabetic ketoacidosis and summarized that resolution of ketoacidosis in adrenal insufficiency steroid replacement needs to be increased in situations of stress. Although it was a hypothesis generating research, it still can be taken as a paramount importance.[11]

A case reported by Azmi Mohammed et al. of a 26 year old female with weight loss of 32 kilograms in last 18 months. The paper similarly concluded that prompt treatment of adrenal crisis is a requirement, euglycemic or hypoglycemic ketosis can further complicate into starvation, nausea and vomiting and collateral history is important.

Even though it is rare but Type 1 diabetes can hide behind Adrenal Insufficiency and it can manifest as EGDKA. Strong Suspicion, prompt diagnosis and early and vigorous treatment is the mainstay for management.[12]

CONCLUSION

It is a diagnostic challenge for the physician to identify euglycemic ketoacidosis early and manage it timely. It is easy to miss out as serum glucose levels are not in the diagnostic range. But SGLT-2 inhibitors have proven notorious to cause such instances, and thus suspicion is always made for the patient ongoing treatment with these drugs.

But, as has been seen in this case, another strong contributing factor can be adrenal insufficiency. So, proper history and evaluation is paramount in ruling out adrenal insufficiency as it can equally cause the severe disease.

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