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A Case Report of SGLT2 Inhibitor-associated Diabetic Ketoacidosis- Hyperglycemic Hyperosmolar State (DKA/HHS) Overlap

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Abstract

Sodium glucose co-transporter-2 inhibitors are associated with a range of adverse effects including Diabetic ketoacidosis both euglycemic and Hyperglycemic. However, this case report highlights a rare case of DKA/HHS overlap in a patient who was treated with SGLT2i. Most patients recover with prompt recognition and treatment, patient education about identifying early signs remains a cornerstone of early identification and treatment.

Keywords: DKA • HHS • Sodium-glucose cotransporter-2 inhibitors • SGLT2i

Introduction

Sodium-Glucose cotransporter-2 inhibitors (SGLT2i) are a relatively newer class of oral anti-diabetic drugs in the management of Diabetes mellitus with significant benefits such as improved cardio-metabolic profile and reno-protective effects. Although rare, serious adverse effects remain a cause of Concern including Diabetic Ketoacidosis (DKA) both Euglycemic and hyperglycemic DKA. However, we report a rare occurrence of Diabetic ketoacidosis- Hyperglycemic Hyperosmolar state (HHS) overlap in a patient treated for one month with SGLT2i.

Case Presentation

A 30-year-old female presented to a primary care clinic with complaints of dysuria. The patient denied any history of fever, flank pain, pyuria, or hematuria. Her past medical history was not significant. She was evaluated by her primary care physician, initial lab investigations (Table 1) revealed 7-8 pus cells in urine and random blood glucose of 320 mg/dl, HbA1c was sent which showed a value of 9.6%. She was prescribed Oral Anti-diabetic drugs (SGLT2i and metformin) and oral antibiotics for Urinary tract infection.

The patient was compliant and doing well on medications for around one month following which she presented to the emergency room with a history of multiple episodes of vomiting, pain Abdomen, Altered sensorium and breathlessness. The family of the patient denied any new history of fever, dysuria, cough or lower urinary tract symptoms. The family denied any history of alternative medication use, paracetamol ingestion or non-compliance to home medications. On Admission, the patient was drowsy with a GCS of 7 (E2V2M3). She was dehydrated and tachypneic (Respiratory Rate 26), BP of 110/70 mmHg, Pulse rate of 120/minute, Temperature was normal. On clinical examination, there were no signs of meningeal irritation, no signs of infection. The patient was overweight with a BMI of 26.7. Signs of insulin resistance in the form of Grade I Acanthosis nigricans and few skin tags were present. Lab investigations revealed random blood glucose of 500 mg/dl, serum ketones were elevated, hypernatremia (serum Na+ 161 mEq), hypokalemia (k+ 2.64 mEq), Neutrophilic leukocytosis and elevated creatinine. NCCT head, NCCT Abdomen And chest x-ray were normal. USG Abdomen and pelvis revealed Grade-II fatty liver. Anti-Gad 65 was positive and C-peptide levels were low so the patient was diagnosed as having Type-1 Diabetes mellitus.

Table 1. Laboratory Investigations.

Laboratory Investigation	Result
Hemoglobin	12.5 gm/dl
Total leucocyte count	30600/uL (H)
Neutrophils	82% (H)
Lymphocytes	11%
Platelets	361000/uL
Random blood glucose	>500 mg/dl (H)
Ketones	6.1 mmol/l (H)
Blood pH	7.018 (L)
Sodium	161 mEq/L (H)
Potassium	2.64 mEq/L (L)
Bicarbonate	5.4 mEq/L (L)
Lactate	1.15
Total bilirubin	0.45 mg/dL
ALT	38 U/L
Albumin	4.19 g/dL
Urea	43 mg/dL
Creatinine	1.17
Amylase	86
Calcium	9.2 mg/dL
Phosphorus	1.78 mg/dL
HbA1C	13.2% (H)
GAD-65	Positive
C-peptide	Low

H: High, L: Low

Based on history, clinical examination and laboratory investigations the patient was diagnosed to have DKA/ HHS overlap (presence of high blood sugars and hypernatremia). Her intravascular volume loss was also corrected by giving IV fluid bolus and started on continuous insulin infusion as per adult DKA management protocol with supportive therapy and treatment was adjusted according to Blood glucose and electrolyte monitoring. The patient was switched to Multiple Subcutaneous Insulin Injections (MSII) after the resolution of ketoacidosis and the insulin dose was titrated to the blood glucose log. The patient improved remarkably and was discharged on MSII with fairly

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acceptable glycemic control. Oral anti-diabetics were discontinued and the patient is doing well on follow-ups.

Results and Discussion

Sodium-glucose cotransporter-2 inhibitors are a newer class of oral drugs for type-2 diabetes mellitus, they predominantly act by preventing glucose reabsorption from the proximal renal tubule by blocking sodium glucose cotransporter-2 [1,2]. There is a decent increase in the use of these drugs as many clinical trials have shown significant cardio and renoprotective effects, especially in patients with type-2 diabetes mellitus [3]. DKA is defined as ketoacidosis, high anion gap (>12 meq) with elevated blood sugar >250 mg/dl, in 2015 FDA issued a warning regarding the risk of DKA with the use of SGLT2i use [4]. SGLT2i usually leads to Euglycemic DKA i.e. DKA with preserved blood glucose levels below 250 mg/dl, however not all patients on these drugs develop euglycemic DKA [5]. These drugs can lead to volume depletion and acute kidney injury.

Since there are no guidelines to diagnose DKA/HHS overlap, the presence of high blood sugars with associated hypernatremia points towards the co-existence of DKA and HHS.

Conclusion

DKA is a rare but serious adverse effect associated with SGLT2 inhibitors, with recent interest and approval of these drugs for type-1 diabetes mellitus in some countries, the treating physician should exercise caution while treating young patients with diabetes with SGLT2i. Clinicians should have a high index of suspicion for DKA in the setting of SGLT2i use. Most patients recover with early recognition and treatment and patient education about identifying early signs of DKA remains the cornerstone of prompt management.

Acknowledgement

None.

Conflict of Interest

None.

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