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**Case Report**

# Intractable Metabolic Acidosis Requiring Hemodialysis in a Patient of Lada Treated with Sglt – 2 Inhibitor

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## ABSTRACT

Sodium Glucose co-transporter – 2 (SGLT-2) inhibitors are class of oral antidiabetic drugs used in the treatment of Type II diabetes Mellitus. They act on the SGLT- 2 protein expressed in the renal proximal convoluted tubules to reduce the absorption of filtered glucose, decrease the renal threshold of glucose and promote glucose excretion. They have gained popularity due to their beneficial effects on heart and kidneys. However their use is associated with increased risk of Euglycemic ketoacidosis. Here we present a case where SGLT – 2 inhibitor used in combination with Insulin in a patient of LADA was associated with D.K.A where severe Metabolic acidosis persisted even after correction of hyperglycemia and dehydration which did not respond to continuous infusion of Dextrose, insulin and Sodium bicarbonate, ultimately requiring Hemodialysis.

**Key Words**: *SGLT -2, inhibitors, Diabetic ketoacidosis, Euglycemic ketoacidosis, metabolic acidosis*

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## INTRODUCTION

SGLT – 2 Inhibitors are known to cause Euglycemic Diabetic Ketoacidosis, hence proper selection of patients before initiating treatment with these agents are imperative specially so if the patient is suspected to be a case of LADA[1,2,3,4]. We report a case of 42 years old male diagnosed with LADA and initiated on Premix Insulin, Metformin and SGLT – 2 inhibitor, who presented with Diabetic Ketoacidosis, developed severe intractable metabolicacidosis even after correction of hyperglycemia, requiring Hemodialysis.

## Case Report

42 years old male, serving personnel, posted in Campbell Bay Island of Nicobar District was evacuated to Naval Hospital at Port Blair in the afternoon of 14/2/22 with complaint of sudden onset of breathlessness since the morning of same day. There was history of polyurea and polydypsia since 5 days prior to admission. There was no history of fever, pain abdomen, vomiting, diarrhea. Patient was diagnosed as a case of LADA in November 2021 and was onInj. Ryzodeg, Tab. Metformin 500mg BD and Dapagliflozin 10 mg OD since January 2022. His blood sugar was under control when last checked a month back.

On examination the patient was found to be conscious, oriented, afebrile, dehydrated. His Pulse rate was 110 min., BP was 140/90, Respiratory rate was 36/min., had acidotic breathing with SPo2 of 100% in room air. There were no crepitations in the chest, C.V.S examination showed tachycardia with normal CNS and per abdomen examination. His investigations showed that his blood sugar was 425 mg% and urine ketones were ++++. His ABG showed that pH was 7.0, $PCO_2$= 24, $P0_2$ = 103 and HCO3 = 8.3. Except for elevated total count in CBC all other investigations were normal. (Given in the table below). A diagnosis of Diabetic Ketoacidosis was made and treatment was initiated with I.V fluids, insulin infusion, bicarbonate infusion and IV antibiotics. Metformin and Dapagliflozine were stopped. His Dehydration was corrected within 12 hours and blood sugar was brought under 200mg% within 24 hours after which he was put on continuous infusion of glucose with neutralizing dose of insulin but he continued to have severe ketoacidosis requiring continuous infusion of sodium bicarbonate. On 16/02/22 at 06:30 pm he developed severe breathlessness with a respiratory rate of 52/min, PR = 120/min, BP = 130/80, hydration-normal, RS=clear. His ABG showed that HCO3 level was 3.5, PH =6.9, PCo2 = 14 mmHg and Po2 = 148 and blood sugar was 130 mg%. In view of persistent severe metabolic acidosis not responding to IV sodium bicarbonate he was taken up for Hemodialysis without ultra filtration at G.B. Pant Hospital, Port Blair which is a tertiary level hospital attached to ANIIMS. The patient underwent three sessions of Hemodialysis of four hour each daily for next three days, Inj. sodabicarb was stopped from next day and was put on Tab. sodabicarb 1 gram three times a day. His acidosis got corrected on 18/02/22 and the patient was shifted to command

Hospital, Bangalore on 23/02/22. At the time of shifting he was asymptomatic, his sugars were under control with Insulin, Urine ketones were negative and his bicarbonate level was normal.

**INVESTIGATIONS**

| DATE | 14.02.22 | 15.02.22 | 16.02.22 | 17.02.22 | 18.02.22 | 19.02.22 | 22.02.22 |
|---|---|---|---|---|---|---|---|
| Hb. | 14.7 | 14.6 | 15 | 14,8 | 13.2 | | 11.0 |
| TLC | 17360 | 15880 | 13040 | 14400 | 13160 | | 4400 |
| S.BIL | 0.5 | 0.6 | 0.4 | | 0.4 | | 0.6 |
| SGOT | 16 | 18 | 23 | | | | 30 |
| SGPT | 10 | 14 | 20 | | 27 | | 22 |
| CREATININ | 0.8 | 0.8 | 0.9 | | 1.0 | 0.7 | 0.5 |
| sNA* | | 132 | | | 143 | 133 | 138 |
| K* | | 4.2 | | | 4.5 | 4.2 | 3.8 |
| RBS | 425mg/dl | 200mg/dl | 232 mg/dl | 200mg/dl | 90 mg/dl | 205 mg/dl | |
| URINE FOR KETONE BODIES | ++++ | ++++ | +++ | +++ | Negative | Negative | Negative |
| URINE CULTURE | | | NO GROWTH | | | | |
| BLOOD CULTURE | | | NO GROWTH | | | | |

**CXR, ECG, Ultrasound Abdomen, CT angiogram: Normal**

**ABG:**

| *DATE* | *TIME* | *PH* | *HCO3* | *PCO2* | *PO2* |
|---|---|---|---|---|---|
| 14/02/22 | 06:00 PM | 7.0 | 10.9 | 24 | 103 |
| 15/02/22 | 06:15 AM | 7.22 | 8.2 | 20 | 111 |
| 15/02/22 | 01:30 PM | 7.18 | 13.7 | 26 | 89 |
| 16/02/22 | 12:15 PM | 7.02 | 7.10 | 26 | 98 |
| 16/02/22 | 05:30 PM | 6.9 | 3.5 | 14 | 148 |
| 17/02/22 | 01:15PM | 7.1 | 2.6 | 7.1 | 152.3 |
| 18/02/22 | 09:15 AM | 7.2 | 7.9 | 15 | 158 |
| 18/02/22 | 06:30 PM | 7.35 | 18.1 | 25 | 112 |
| 19/02/22 | 02:10 PM | 7.4 | 20.5 | 31.3 | 93.4 |
| 21/02/22 | 06:30 AM | 7.4 | 20.4 | 30.6 | 96 |
| 22/02/22 | 9.15 AM | 7.4 | 24.0 | 30.5 | 99 |

## DISCUSSION

LADA (Latent Auto-immune Diabetes of Adults) also known as type 1.5 diabetes is adult onset diabetes which shares features of both type I and type 2 Diabetes Mellitus. Diagnosing LADA has treatment implications because of high risk of progression to insulin dependency. The optimal treatment of LADA is not established. Sulfonylureas are better avoided as they exhaust β – cells, Glitazones and Exenatide have favorable outcomes, where-as Metformin needs to be used with caution. SGLT- 2 Inhibitors should be avoided for fear of causing Euglycemic D.K.A[2,4]. The proposed mechanism of SGLT-2 inhibitors associated Euglycemic D.K.A implicates glycosuria leading to decreased plasma glucose level, decreased insulin, increased glucagon levels leading to upregulation of lipolysis and ketogenesis[1]. Also the pharmacological effects of SGLT-2 inhibitors may persist beyondseveral half lives of elimination.Our patient was initially diagnosed as Type II Diabetes Mellitus in November 2021 at Military Hospital, Chennai, where he presented with osmotic symptoms and weight loss and was found to have a HbA1c of 13.8%. The patient was initiated on Basal insulin, sulphonylureas and Metformin. Since his blood sugars were not coming under control and since the patient had a low body mass Index, he was investigated and was found to have elevated GAD and Islet cell antibodies, hence he was diagnosed as LADA and referred to the Endocrinologist at Command Hospital, Bangalore in January 2022 for further management. At Bangalore he was initiated on Premix insulin (Ryzodeg), Dapagliflozine 10mg once a day and metformin 500 mg twice a day. With the regime his blood sugarswere brought under control before discharge. He reported for his duty at his place of posting i.e. Campbell Bay, Nicobar District one week prior to developing the symptoms of Diabetes ketoacidosis for which he was shifted to our hospital after stabilization. On admission to our hospital on 14/02/22, the patient was in Diabetes ketoacidosis, but despite correction of dehydration and hyperglycemia, continued to be in ketoacidosis with severe metabolic acidosis which did not respond to infusion of dextrose insulin and

sodium bicarbonate necessitating hemodialysis. Although the metabolicacidosis in Diabetic Ketoacidosis gets corrected with correction of dehydration and hyperglycemia, some patients with severe acidosis with PH<7.0 may require bicarbonate infusion but rarely require dialysis[5]. Our case is a rare case where Hemodialysis was required to correct severe Metabolicacidosis in a patient of LADA treated with Insulin and SGLT – 2 inhibitor. This re-emphasis the point that caution should be exercised in initiating the patients on SGLT – 2 inhibitor in patients with LADA even in combination with Insulin.

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