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Safe and Quick Control of Hyperglycemia with Luseogliflozin in Case of High-Dose Intensive Insulin User in an Acute Ischemic Stroke Stage: A Case Report

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Abstract

Introduction: Diabetes mellitus is one of major risk factors of stroke and it hasn't been established how to manage hyperglycemia safely and quickly in an acute stroke stage, particularly in cases of high-dose insulin users.

Case Presentation: A 62-year-old female undergoing high-dose intensive insulin therapy presented with numbness of the right hand. On arrival, serum glucose level was 282 mg/dl and HbA1c 12.8%, although she had daily used total dose of 66 units of insulin. Luseogliflozin, one of sodium-glucose cotransporter-2 (SGLT2) inhibitors, coupled with mild low-carbo diet started under insulin degludec of 10 units. Her fast blood glucose became 73 mg/dl in one week and discharged home.

Conclusion: Luseogliflozin coupled with mild low-carbo diet controlled hyperglycemia safely in an acute stroke stage.

Keywords: Hyperglycemia; Luseogliflozin; Acute ischemic stroke

Abbreviations

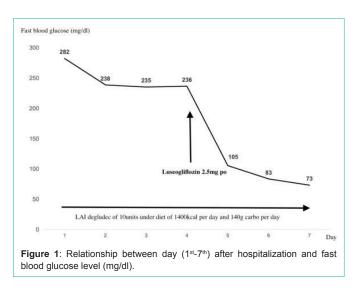
SGLT2: Sodium-Glucose Cotransporter-2; DM: Diabetes Mellitus; BMI: Body Mass Index; BW: Body Weight; SC: Subcutaneous Injection; UFAI: Ultra-Fast-Acting Insulin; LAI: Long-Acting Insulin; carbo: Carbohydrate

Introduction

Type 2 diabetes mellitus (DM) is one of major risk factors of stroke and hyperglycemia at the time of acute ischemic stroke increases the risk of hemorrhagic transformation with iv-TPA treatment and it is associated with poor clinical outcomes, longer inhospital stay, increased cost, and mortality [1-6]. However, it hasn't been established how to manage hyperglycemia safely in an acute stroke stage, particularly in cases of high-dose insulin users. Safe and effective therapy of controlling glucose level in an acute stage is required.

Case Presentation

A 62-year-old female undergoing high-dose intensive insulin therapy presented with numbness of the right hand. On arrival, her height was 158 cm, her body weight (BW) 69 kg, and her body mass index (BMI) 27.6 kg/m2. Her serum glucose level was 282 mg/dl, HbA1c 12.8%, urine glucose was positive and urine ketones positive on arrival, although she had daily used total dose of 66 units of insulin; subcutaneous injection (SC) of 16 units of ultrafast-acting insulin (UFAI) glulisine within 15 minutes before a meal three times a day and SC injection of 18 units of long-acting insulin (LAI) degludec at bedtime. However, she had suffered from diabetic retinopathy, diabetic neuropathy, diabetic renal dysfunction and bilateral arteriosclerosis obliterans, and had a mild ischemic stroke after all. She was obese and abdominal ultrasound showed fatty liver



on arrival, this was because she had eaten carbohydrates too much every day. Therapy she had undergone was unsuccessful in managing blood glucose level and in preventing micro- and macro-angiopathy. Immediately after hospitalization, mild low-carbo diet (1,400 kcal per day and 140 g of carbohydrates per day) started under LAI degludec of 10 units to control blood glucose level and luseogliflozin 2.5 mg, one of sodium-glucose cotransporter-2 (SGLT2) inhibitors was added on the 4th day (Figure 1). Intake of carbohydrates was 50 g at breakfast, 50 g at lunch and 40 g at dinner and she finally had 2 units of UFAI glulisine before breakfast and lunch as carbohydrate counting method. Her fast blood glucose level began to decrease remarkably after luseogliflozin and finally became 73 mg/dl in one week (Figure 1) and post-meal blood glucose level became less than 200 mg/dl under 2 units of UFAI glulisine before breakfast

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Table 1:

	On admission	on the 7 th day
serum glucose level (mg/di)	282	73
Urine pH	7	5
Urine glucose	4+	4+
Urine Ketone	2+	2+
Arterial pH	7.48	7.35
B.E.	-0.3	-0.37
PCO2 (mmHg)	30.1	40.3
Total dose of insulin (units)	66	14
Serum ketone (µmol/L)		1041
Body weight (kg)	69	66.6

and lunch and LAI degludec of 10 units at bedtime only. Her body weight (BW) decreased from 69 kg to 66.6 kg in one week. Her numbness disappeared and she discharged home in one week under luseogliflozin 2.5 mg po qD and total insulin dose of 14 units (Table 1). Neither acidosis nor ketoacidosis symptoms occurred, although serum ketone level increased (Table 1). Hypoglycemia of less than 70 mg/dl didn't occur, neither.

Discussion

Patients with type 2 DM are at increased risk of cardiovascular events and cardiovascular mortality [7]. The risk of stroke in patients with type 2 DM is increased 2-fold compared with individuals without diabetes mellitus; the risk of recurrent stroke is also increased [8]. Trials of intensive glucose-lowering or of specific glucose-lowering agents have not been shown to significantly reduce the risk of stroke in patients with type 2 DM even after prolonged follow-up [9,10]. A previous study reported that patients with type 2 DM at high risk for cardiovascular events who received empagliflozin, the SGLT2 inhibitor, as compared with placebo, had a lower rate of the primary composite cardiovascular outcome and of death from any cause when the SGLT2 inhibitor drug was added to standard care [11]. Indeed, stroke occurs in patients with type 2 DM, indicating unsuccessful primary prevention, but effective secondary prevention hasn't been established. In addition, it hasn't been established how to control hyperglycemia in an acute stroke stage, although hyperglycemia must be controlled soon to improve clinical outcome following stroke. Therefore, our novel strategy of SGLT2 inhibitors coupled with mild calorie-and carbo-limited diet may be promising to control hyperglycemia safely and quickly in an acute stroke stage and to prevent recurrence of strokes. Her BW decreased within one week and serum ketones increased, however, acidosis didn't occur.

Conclusion

Luseogliflozin coupled with mild low-carbo diet successfully controlled blood glucose level safely in an acute stroke stage, and dramatically reduced total dose of insulin.

Author's Contribution

TM and YT treated the patient, analyzed and all authors interpreted the patient data. TM was a major contributor in writing the manuscript.

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