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A Case of Late Recognition of Euglycemic Diabetic Ketoacidosis Associated with Sodium–Glucose Cotransporter-2 Inhibitor after One Anastomosis Gastric Bypass Surgery

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ABSTRACT

Euglycemic Diabetic Ketoacidosis (EuDKA) is a serious and recognized complication associated with the use of sodium-glucose cotransporter-2 inhibitors (SGLT2i), a new group of oral hypoglycemic agents, and it can be easily overlooked by medical care providers because of the absence of marked hyperglycemia in contrast to DKA. Very-low-calorie diet regimens and poor postoperative intake can trigger EuDKA, which can present a diagnostic challenge. The caregiving team needs to be aware of this complication in the postoperative period.We report this case to highlight the delayed recognition of a potentially life-threatening condition.

Keywords*: Euglycemic diabetic ketoacidosis; SGLT2 inhibitors; Postoperative complications; Sodium–glucose cotransporter-2 inhibitors; Very-low-calorie diet.*

INTRODUCTION:

Euglycemic diabetic ketoacidosis (EuDKA) is an acute rare complication of diabetes that can occur in both type 1 and type 2 diabetes, and it is more common in type 1 diabetes. The diagnostic triad is the same as for Diabetic Ketoacidosis (DKA) with the exception of hyperglycemia (>250 mg/dl) [(1)].The other two criteria are ketonuria and metabolic acidosis. The blood glucose level is typically under 200 mg/dl. SGLT2i induces normoglycemia by promoting glycosuria, leading to a reduction in blood glucose levels and a decrease in insulin secretion. This, together with decreased carbohydrate intake and increased glucagon secretion, will trigger lipolysis and ketogenesis while maintaining normoglycemia [(4)]. Several conditions can lead to carbohydrate starvation, such as anorexia, fasting, and gastroparesis. Other trigger factors may include stress, surgery, infection, and volume depletion. Patients on SGLT2i such as canagliflozin, dapagliflozin, empagliflozin, or ertugliflozin, along with the aforementioned triggering factors, can develop EuDKA[(5)].

Case Presentation:

A 60-year-old woman with class 1 obesity (BMI 34) underwent a laparoscopic one anastomosis gastric bypass along with umbilical hernia repair. Her medical history included type 2 diabetes on SYNJARDY 12.5/1000, hypertriglyceridemia (INEGY 10/20), and dyslipidemia (LIPANTHYL 145MG), along with failed back surgery syndrome. The surgery proceeded without complications, and she was discharged two days later. Seventy-two hours after surgery, she returned to the emergency room complaining of fatigue and malaise and was treated with intravenous fluids. Her vital signs were within normal limits, and her blood glucose was 116 mg/dL. On the fifth postoperative day, she was readmitted due to generalized weakness, palpitations, and headache, with a blood sugar level of 207 mg/dL. At that time, she was afebrile, her oxygen saturation was 100% on room air, her heart rate was 104 beats per minute with sinus rhythm, and her blood pressure measured 147/70 mmHg. Laboratory tests showed a bicarbonate level of 7 mEq/L, normal electrolytes, a hematocrit of 50.4%, and normal cardiac enzymes. She was admitted to the hospital for observation and symptomatic treatment. Investigations, including tests to exclude infection and an anastomotic leak, were planned, and she was placed

in the ward for monitoring. After the onset of encephalopathy, additional tests such as arterial blood gas (ABG) analysis and urine ketone testing were performed, resulting in a preliminary diagnosis of euglycemic diabetic ketoacidosis (EuDKA). The neurology and cardiology teams were consulted. Arterial Blood gas analysis revealed severe metabolic acidosis. She was promptly transferred to the Intensive Care Unit (ICU), where she received intravenous fluids, bicarbonate, glucose, and insulin therapy. Following these interventions, she demonstrated rapid clinical improvement and normalization of her blood gas values (Table: 1)

Table 1: Arterial blood gas values

Time point(hr)	0	1	2	3	4	5	6	7	8	10	12	18	24	72
Ph	6.7	6.9	6.9	6.9	6.9	6.9	7.02	7.01	7.07	7.16	7.21	7.28	7.31	7.34
HCO3 (meq/l)	1.5	2.8	2.4	2.4	3	3.4	3.9	3.7	4.5	5.3	6	9.8	11.6	12.2
Pco2(mmhg)	10.1	11.7	12.1	12.2	13.8	14.2	15.2	14.7	15.6	14.9	15.1	21.4	23.4	23.2
AG	28.3	26.2	25.3	24.9	27.1	26.2	25.3	23.4	22.5	21.5	19.7	13.7	15.7	14.9
HCO3 :bicarbonate; pCO2 :partial pressure of carbon dioxide; AG: anion gap														

Discussion:

Surgical weight-loss methods such as the one anastomosis gastric bypass (OAGB) have become widely accepted as successful approaches for managing obesity and its associated disorders, such as type 2 diabetes mellitus (T2DM).Sodium-glucose cotransporter-2 inhibitors (SGLT2i),a new categoryof oral hypoglycemic medications for T2DM, lower blood sugar by promoting the removal of glucose through urine, which also helps with reducing body weight[4] .Sodium-glucose cotransporter-2 inhibitor(SGLT2i)-linked euglycemic diabetic ketoacidosis(EuDKA)[1,2,3] is a serious and increasingly recognized side effect of this group of oral diabetes medications, often posing a diagnostic difficulty that can lead to delayed diagnosis, improper management, and life-threatening acidosis[4]. These inhibitors carry a reduced risk of low blood sugar because they do not interfere with the body's own glucose production during hypoglycemia, and the amount of glucose lost in urine depends on kidney filtration and blood sugar levels. Their action does not rely on insulin, making them appropriate for treating type 2 diabetes at any stage, and they may also be used in type 1 diabetes[6].Empagliflozin has been shown in outcome trials to decrease the risk of major adverse cardiovascular events in patients with T2DM [7]. The mild metabolic shift from glucose to ketone body use has been postulated to be a more efficient energy source, which improves cardiac and renal efficiency, thus improving cardiac contractility and renal function. This, in combination with weight loss and reduction in blood pressure, is proposed to contribute to the cardiovascular benefits [8]. DKA is defined as the triad of hyperglycemia (>14 mmol/l), metabolic acidosis with an elevated anion gap, and elevated plasma ketones. SGLT2i-associated DKA is somewhat different in its pathogenesis, though treatment principles are the same. The term 'euglycemic' DKA has been used when describing SGLT2i-associated DKA, but it could be a misnomer as euglycemia is not universal and is actually uncommon [9,10]. Ketone buildup in diabetic ketoacidosis (DKA) associated with sodium-glucose cotransporter-2 inhibitors (SGLT2i) results from the combined effects of reduced insulin and elevated glucagon, which stimulate fat breakdown and ketone production in the liver. The glucose-lowering effect of SGLT2 inhibitors, [11] via increased urinary glucose excretion, causes a decrease in insulin release from pancreatic beta cells. Additionally, by promoting glycosuria and reducing sodium reabsorption in the kidneys, SGLT2 inhibitors may increase ketone levels indirectly by enhancing renal ketone reabsorption. Multiple factors can precipitate the development of euglycemic diabetic ketoacidosis (EDKA) in T2DM [12]. It is essential for healthcare professionals to be aware of the attributable factors, as very-low-calorie diet regimens and poor postoperative intake can trigger EuDKA[13]. In the preoperative period, stopping the SGLT2 inhibitor 24-48 hours before surgery did not have a significant effect. Optimal methods for preventing postoperative EDKA remain uncertain and deserve further review [14].The Centre for Perioperative Care (CPOC) recommends omission before surgery, whereas the ESC recommends 3 days prior to surgery [15, 16]. At this time, clinicians should consider pausing SGLT2 inhibitor 3–4days before surgery, and insulin therapy should be personalized and adjusted accordingly [17].

Conclusion:

This case report points out that treating physicians failed to recognize DKA due to relative normoglycemia, resulting in a delay in effective treatment. Also, early identification of the precipitating factors could have minimized the potential risks. EuDKA is likely to be under recognized because of its atypical presentation. Understanding this clinical entity and vigilance towards monitoring plasma/capillary ketones can facilitate early identification and assist in management.

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