CASE REPORT

Diabetic ketoacidosis in a postoperative gastric bypass patient

Chelsea Hoenes¹,*, Qammar Rashid², and Juan Pimentel³

¹St. George’s University School of Medicine, 3500 Sunrise Highway, Building 300, Great River, NY, Grenada, West Indies, ²Atlanta Surgical Associates, 1045 Sycamore Drive, Decatur, GA, Georgia, and ³Northwest Nephrology Clinic, 5255 Snapfinger Park Drive #110, Decatur, GA, Georgia

*Correspondence address. St. George’s University School of Medicine, 3500 Sunrise Highway, Building 300, Great River, NY 11739, Grenada, West Indies. Tel: +716-374-3133. E-mail: choenes@sgu.edu

Abstract

We report a case of postoperative life threatening metabolic acidosis in a young Type-2 diabetic woman who had no history of diabetic ketoacidosis. The patient underwent a non-complicated laparoscopic Roux-en-Y gastric bypass 6 days earlier. We discuss her clinical features, pathogenesis, management and outcome. We would like to make the surgical field aware of this potentially fatal diabetic complication since bariatric procedures are now indicated for the management of diabetic morbid obesity and Type-2 diabetes prevalence is on the rise, particularly in young individuals.

INTRODUCTION

We describe a case where on January 5, a 23-year-old African American female presented on her operative day for a roux-en-Y gastric bypass for treatment of morbid obesity (BMI = 37.9). She was a known hyperlipidemic and Type-2 diabetic, for which she took daily metformin 500 mg, Empagliflozin 10 mg (an SGLT2 inhibitor), and glimepiride 4 mg. An uncomplicated surgery was followed by life threatening diabetic ketoacidosis 6 days postoperatively.

CASE REPORT

On admission for her gastric bypass, our patient was stable with normal vital signs. She had been on a liquid-only diet for 2 days pre-operatively. Her blood chemistries were within normal limits except for her blood glucose level, which was 226 mg/dl (Table 1). She had not taken her regularly scheduled medications for 3 days prior to surgery.

The surgery progressed under general anesthesia without complications. Total blood loss was minimal and the immediate postoperative status was uncomplicated, and her blood glucose was recorded at 268 mg/dl.

On postoperative Day 1, upper GI imaging was performed indicating no leak was present from the gastric bypass. Her vitals were normal, though her blood glucose level was recorded at 224 mg/dl. eGFR and anion gap were within normal limits. Creatinine and BUN were 0.8 and 10 mg/dl, respectively. For fluids, 1200 ml normal saline was administered; 240 ml of fluid was taken orally, with 450 ml of urine output. She tolerated a clear liquid diet well, with no complications. She was experiencing no pain or discomfort at this time, and she was discharged 2 days postoperatively, with instructions to take glimepiride 4 mg orally twice a day and Jardiance 10 mg orally once a day.

Six days postoperative, the patient presented to the emergency department with dyspnea, confusion and vomiting. Her BP was 177/67, HR was 150 bpm, and temperature was 99.4°F. Her abdomen was soft, non-distended, and non-tender. Her ABG revealed a pH of 6.79 and a pCO2 of 18, with bicarbonate of 2.7 mEq/l, indicating a life threatening metabolic acidosis. Lactic
Acid was significantly elevated at 5.1 mmol/l. Creatinine was measured at 1.5 mg/dl, with a BUN of 22 mg/dl. eGFR was decreased and glucose was 356 mg/dl. She was immediately brought back to the operating room for a diagnostic laparoscopy to exclude a surgical cause of her severe acidosis, the findings of which were negative.

A drug screen and a Beta-HCG level were negative. The serum osmolality was calculated at 320 mOsm/Kg. An EKG revealed sinus tachycardia and nonspecific T wave abnormalities in the inferior and lateral leads. A chest CT demonstrated no pulmonary embolism present.

She was diagnosed with diabetic ketoacidosis (DKA) and began treatment with intravenous fluids, insulin, and bicarbonate. She was intubated and mechanically ventilated. On the second hospital day, hemodialysis was initiated and continued daily for 4 days. Urine and blood cultures showed no bacterial growth. The patient gradually improved over the course of her admission, with her pH normalizing to 7.39 on the fourth hospital day. On the 5th day of hospitalization, dialysis was discontinued and she was extubated. At the time of discharge on the eighth hospital day, her serum chemistries demonstrated glucose at 145 mg/dl, BUN of 6 mg/dl, creatinine of 0.4 mg/dl and a normal anion gap of 10. At 1-month follow-up, our patient had no noted complications and was doing well.

**DISCUSSION**

The pathophysiology of DKA involves a deficiency of insulin, which results in a paucity of glucose available to tissues, despite high blood concentration. The subsequent breakdown of fats stimulates fatty acid oxidation and the production of ketoacids. DKA, therefore, presents with hyperglycemia, glycosuria and hyperketonemia [1].

Our patient is of interest because she had relatively low blood glucose at presentation (euglycemic DKA), which certainly confounded her diagnosis of DKA. Known risk factors for DKA were promptly excluded in our patient (Table 2). She was not taking steroids, had no evidence of an acute abdomen, and she resumed her Empagliflozin (Jardiance) therapy postoperatively.

**Table 1** Laboratory studies

|                | Preoperative | Postoperative (days) |
|----------------|--------------|----------------------|
|                | 1 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
| pH             | 6.79 | 7.12 | 7.23 | 7.39 | 7.44 |
| pCO2           | 18 | 26 | 32 | 33.7 | 35.4 |
| pO2            | 226 | 176 | 123 | 93 | 77 |
| HCO3           | 2.7 | 8.4 | 13.4 | 20.6 | 24 |
| Glucose        | 226 | 189 | 356 | 156 | 249 | 218 | 168 | 124 | 145 |
| BUN            | 8 | 10 | 22 | 7 | 9 | 14 | 16 | 7 | 6 |
| Creatinine     | 0.6 | 0.8 | 1.5 | 1.1 | 1.2 | 0.9 | 0.7 | 0.4 | 0.4 |
| Sodium         | 137 | 141 | 135 | 148 | 148 | 150 | 147 | 144 | 142 |
| Potassium      | 3.8 | 4.5 | 4.8 | 3.2 | 3 | 3 | 2.8 | 3 | 3.1 |
| Carbon dioxide | 22 | 21 | 5 | 11 | 10 | 22 | 27 | 27 | 27 |
| Chloride       | 104 | 110 | 103 | 116 | 112 | 112 | 108 | 107 | 105 |
| Anion Gap      | 11 | 10 | 27 | 22 | 26 | 16 | 12 | 10 | 10 |
| Albumin        | 3.8 | 3.1 | 4.4 | 2.8 | 2.5 | 2.2 | 2.1 | 2.1 |
| Lactic Acid    | 5.1 | 0.7 | Neg. |
| D-Lactate      | 149 | 107 | 52 | 74 | 67 | 93 | 125 |
| Hgb-A1C        | 15.4 | 13.2 | 17.8 | 13.2 | 10 | 10.4 | 10.3 |
| eGFR           | 49 | 42.8 | 55.5 | 39.8 | 28.8 | 30.8 | 30.9 |

**Table 2** Factors that can precipitate DKA

- Non-adherence to diabetic therapy
- Infections
- Alcohol abuse
- Psychological stress
- Pregnancy
- Cardiovascular events
- Trauma
- Mediations
- Cushing disease
- Acute gastrointestinal disease

Empagliflozin is a selective inhibitor of sodium glucose cotransporter 2 (SGLT2), a new class of antidiabetic agents that acts on the kidney.

Recent reports linked SGLT2 inhibitors to the development of ‘euglycemic’ diabetic ketoacidosis (DKA). At the time of our patient’s hospitalization, there were no reports in the literature of Empagliflozin associated DKA. Interestingly, most of the cases of DKA reported in patients with T2DM treated with SGLT2 inhibitors were in the postoperative setting.

In 2015, the FDA warned about the safety of SGLT2 inhibition treatment in T2DM because of increased risk of DKA [2, 3]. Several mechanisms have been posited for how SGLT2 inhibitors precipitate DKA in patients with either T1DM or T2DM, including the increased transport of glucose into pancreatic alpha cells, decreasing the insulin: glucagon ratio and volume depletion, which leads to decreased glucose oxidation, increased fat oxidation, and thus, stimulation of ketone body formation on top of increased renal ketone body reabsorption. A reduced caloric and carbohydrate intake in our postoperative patient may also have reduced insulin secretion.

The signs of DKA may be missed in young, healthy patients and it is often mistakenly assumed that DKA is only a remote complication in patients with T2D. Even in surgical patients with seemingly uncomplicated diabetes, further insight and study of the safety of this class of drugs is warranted to prevent a potentially fatal postop
complication of DKA. This particular case is, therefore, cautionary to general or gastrointestinal surgeons who plan invasive procedures on patients using SGLT2 inhibitors.

REFERENCES
1. Rosenstock J, Ferrannini E. Euglycemic diabetic ketoacidosis: a predictable, detectable, and preventable safety concern with SGLT2 inhibitors. Diabetes Care 2015;38:638–42.
2. U.S. Food and Drug Administration. FDA Drug Safety Communication: FDA warns that SGLT2 inhibitors for diabetes may result in a serious condition of too much acid in the blood. FDA 2015. http://www.fda.gov/Drugs/DrugSafety/ucm446845.htm.
3. Storgaard H, Bagger J, Knop F, Vilsbøll T, Rungby J. Diabetic ketoacidosis in a patient with type 2 diabetes after initiation of sodium-glucose cotransporter 2 inhibitor treatment. Basic Clin Pharmacol Toxicol 2016;118:168–70.