Diabetic Ketoacidosis Following Bariatric Surgery in Patients With Type 2 Diabetes

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Diabetic ketoacidosis (DKA) is a life-threatening complication mainly linked to type 1 diabetes. Clinical features of DKA after bariatric surgery are mostly unknown and likely underreported especially in those with type 2 diabetes. The objective of this study is to emphasize occurrence and clinical presentation of DKA in patients with type 2 diabetes after bariatric surgery. We also aim to describe diagnostic challenges related to new medications such as sodium–glucose cotransporter 2 inhibitors (SGLT-2i) that can cause euglycemic DKA (1,2).

We report four cases of DKA in three patients with type 2 diabetes after bariatric surgery at a single institution from January 2010 to December 2015. All cases presented within 30 days following surgery, were classified as moderate to severe based on criteria from the American Diabetes Association (3,4), and required admission to the intensive care unit. Table 1 provides detailed description of the cases.

Baseline glycated hemoglobin A_{1c} (HbA_{1c}) was 9.4% (79 mmol/mol) for patient 1, 9.5% (80 mmol/mol) for patient 2, and missing for patient 3. All patients had type 2 diabetes treated with insulin for a median duration of 15 years (range 2–17), which represents global trends as more patients undergo surgery with worse insulin resistance and less β-cell reserves.

Median time to DKA was 13 days (range 3–27). Main presenting symptoms were nausea, vomiting, and abdominal pain, which are common soon after bariatric surgery. All cases were likely precipitated in part by inadequate insulin therapy or noncompliance compounded by decreased oral intake and dehydration in the early postoperative period. This observation is in keeping with a recent study by Aminian et al. (5) describing four cases of DKA in patients with type 2 diabetes after bariatric surgery. Unlike their report where septic complications were main culprits in three patients, we observed no DKA as a result of postoperative infectious complications confirmed by abdominal imaging. Median time to treatment with insulin infusion was 1.3 h (range 0.6–2.1), which highlights the challenge in timely diagnosis in these patients who present with common symptoms after bariatric surgery. Moreover, the findings emphasize the need for better preoperative patient education and closer glucose follow-up during the hospital stay. A visit to an endocrinologist within the first 2 weeks after discharge is especially important as all cases occurred within the first month.

Interestingly, patient 3 presented on day 9 with euglycemic DKA while on SGLT-2i (canagliflozin). In a recent case series, Peters et al. (2) reported euglycemic DKA linked to SGLT-2 inhibition. However, our study is the first to report this condition in patients with type 2 diabetes after bariatric surgery. Here, DKA could have also been precipitated by lower oral intake after surgery and omission of insulin. Delay between presentation to emergency department and initiation of insulin infusion was 2.1 h, which underlines the difficulty of making the proper diagnosis given the "euglycemic" presentation.

Finally, our findings emphasize the need for a high index of suspicion for DKA in patients with type 2 diabetes who present early after bariatric surgery, especially those on SGLT-2i. Mild hyperglycemia should not preclude prompt diagnosis and timely delivery of treatment. Diagnosis can be hastened with urinalysis demonstrating ketones. Our observation further suggests stopping SGLT-2i before planned surgery, possibly prior to starting the 2-week course of low-calorie diet.

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Table 1—Characteristics of patients with type 2 diabetes with DKA after bariatric surgery

	Patient 1		Patient 2	Patient 3
Case	1	2	3	4
Age (years)	62		54	58
Sex	Female		Female	Male
BMI (kg/m^2)	41.0		37.8	49.0
Baseline HbA_{1c} (%)	9.4		9.5	N/A
Duration of T2DM (years)	2		15	17
Preoperative medications	Levemir, glyburide (10 mg b.i.d.), and Komboglyze (2.5/1,000 mg b.i.d.)		Levemir, Humalog, and metformin (800 mg b.i.d.)	Levemir, NovoRapid, metformin (1,000 mg b.i.d.), and canagliflozin (300 mg q.d.)
Insulin doses (IU)				
Long-acting	0–0–0–36		30–0–0–40	0–0–0–65
Short-acting			25–25–25–0	35–35–35–0
Surgery	LSG		LRYGB	LSG*
Time to presentation (days)	17	27	3	9
Symptoms of presentation	Nausea, vomiting, abdominal pain, and poor oral intake	Nausea, vomiting, diarrhea, and altered mental status	Nausea and shortness of breath	Nausea, vomiting, and abdominal pain
Precipitating factors	Omission of insulin and dehydration	Omission of insulin† and dehydration	Omission of insulin	Canagliflozin and omission of insulin
Arterial pH‡	N/A	7.1	6.8	7.2
Anion gap (mmol/L)	30	25	22	26
Bicarbonate (mmol/L)	4.5	4.5	3.7	9.4
Serum glucose (mmol/L)	34.3	29.4	25.0	17.0§
Serum ketones‖ (mmol/L)	9.7	9.4	10.3	10.5
Urine ketones	Positive	Positive	Positive	Positive
White blood cells (10^9/L)	16.8	29.1	23.7	17.5
Serum creatinine (mmol/L)	220	177	125	210
Severity	Moderate–severe	Severe	Severe	Moderate–severe
Organ dysfunction	AKI	AKI and encephalopathy¶	AKI	AKI
Time to treatment (h)	0.6	0.6	2.0	2.1
Where treated	ICU	ICU	ICU	ICU

AKI, acute kidney injury; ICU, intensive care unit; LRYGB, laparoscopic Roux-en-Y gastric bypass; LSG, laparoscopic sleeve gastrectomy; N/A, not available; T2DM, type 2 diabetes mellitus. *The operation was converted to laparotomy for lysis of dense adhesions. †The omission of insulin in this presentation was because of noncompliance with recommended regimen by the patient. ‡None of the patients had a sample of arterial blood gas upon presentation to the emergency department. The reported values are from the first 2 h after presentation, hence the initial arterial pH may have been lower. §Patient presented with euglycemic DKA, which is likely caused by SGLT-2i (canagliflozin). ‖Serum ketones were measured as a direct assay of plasma β-hydroxybutyrate (normal range is $<$0.6 mmol/L). ¶Patient required ventilatory support because of her metabolic encephalopathy upon presentation.

version. A.An. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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