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Post operative acute diabetic ketoacidosis in oncosurgery patients unmasking latent diabetes mellitus: Case series with narrative review

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Abstract

Diabetic Ketoacidosis (DKA) is a serious and potentially life-threatening complication of diabetes that arises when insulin levels are insufficient, resulting in the accumulation of ketones in the bloodstream. Postoperative hyperglycaemia triggered by surgical and anaesthetic stress is a well-recognized phenomenon and can lead to adverse clinical outcomes. This article highlights the importance of recognizing stress-induced DKA in postoperative oncosurgery patients and explores its diagnosis and management. We present a case series involving three patients who developed DKA during the postoperative period.

Keywords: Diabetic ketoacidosis; Post-operative shock; Metabolic acidosis; Ketone bodies; Anion gap; Modified radicle mastectomy; Pectoralis major myocutaneous.

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Introduction

Disrupted glucose regulation is a known risk factor associated with various complications in surgical patients, including endothelial dysfunction, postoperative sepsis, delayed wound healing, and cerebral ischemia. The physiological stress induced by surgery and anaesthesia can further exacerbate underlying glucose dysregulation, potentially triggering severe hyperglycaemia with adverse prognostic implications. While Diabetic Ketoacidosis (DKA) as an initial presentation of diabetes has been previously documented, its emergence in the immediate postoperative period as a response to perioperative stress remains uncommon. In this case series, we report instances where DKA occurred shortly after elective oncosurgery, serving as the first clinical manifestation of previously undetected glucose intolerance. Notably, none of the patients had a known history or prior symptoms suggestive of Diabetes Mellitus (DM) before this event.

Case reports

Case 1

A 39-year-old female patient, weighing 55 kg and measuring 149 cm in height, was scheduled for a right-sided Modified Radical Mastectomy (MRM). During the pre-anaesthetic evaluation, a thorough medical history was obtained, which revealed no known comorbidities or prior history of surgery or anaesthesia. Preoperative investigations were within normal limits, including a Random Blood Sugar (RBS) of 128 mg/dL.

The patient underwent the planned right MRM under general anaesthesia. The surgery lasted approximately three hours. No steroids were administered during the intraoperative period. Analgesia was managed using intravenous fentanyl and paracetamol, the latter administered 30 minutes prior to extubation. After successful reversal of neuromuscular blockade, the patient was extubated without complications and transferred to the ICU.

Two hours postoperatively, the patient developed sudden tachycardia and hypotension, along with complaints of nausea and abdominal discomfort. Arterial Blood Gas (ABG) analysis and blood sugar testing were performed. The RBS was found to be significantly elevated at 571 mg/dL, compared to a preoperative fasting value of 91 mg/dL. ABG revealed severe metabolic acidosis (pH 7.19, HCO_3^- 8 mEq/L) with a high anion gap. Urine dipstick testing showed the presence of glucose and ketone bodies, leading to a diagnosis of Diabetic Ketoacidosis (DKA).

Management began promptly with fluid resuscitation using 0.9% normal saline to restore euvolemia. A bolus of insulin was given intravenously, followed by a continuous insulin infusion. Blood glucose levels were monitored hourly, and electrolyte imbalances were corrected as necessary. The patient showed clinical improvement as the metabolic acidosis resolved and the anion gap normalized (Table 2). Subcutaneous insulin therapy was initiated before discontinuing the intravenous insulin infusion.

The patient was transferred to the ward on postoperative day 4 and subsequently discharged on postoperative day 7. She was prescribed subcutaneous insulin therapy and referred for close follow-up with an endocrinologist.

Case 2

A 34-year-old patient, weighing 72 kg and standing 168 cm tall, was scheduled for a Wide Local Excision (WLE) of the tongue. A comprehensive medical history revealed no existing comorbidities, and preoperative investigations were within normal limits, with a Random Blood Sugar (RBS) of 141 mg/dL.

The surgery was performed under general anaesthesia and lasted approximately 4.5 hours. No intraoperative steroids were administered. The patient was sedated intraoperatively using a fentanyl infusion and subsequently transferred to the ICU while intubated with a nasal Endotracheal Tube (ETT) size 8.0 cuffed, placed on mechanical ventilation.

Approximately four hours postoperatively, the patient developed tachycardia (heart rate of 152 bpm) and hypotension (blood pressure of 86/46 mmHg). Fluid resuscitation was initiated, and an additional dose of sedation was administered. An Arterial Blood Gas (ABG) analysis revealed severe metabolic acidosis (pH 7.21, anion gap 21 - Table 3), and RBS had risen sharply to 556 mg/dL. Urine dipstick testing showed the presence of both glucose and ketone bodies, leading to a diagnosis of Diabetic Ketoacidosis (DKA).

Management began with aggressive fluid replacement and intravenous insulin therapy, aiming to reduce serum glucose by 75–100 mg/dL per hour. Blood sugar was monitored hourly. The patient's hemodynamic instability improved, with decreasing vasopressor requirements as blood glucose and acidosis were corrected.

The patient remained on mechanical ventilation for 48 hours, with intravenous insulin continued until normalization of the anion gap (Table 4). By Postoperative Day (POD) 3, ABG values had improved and the anion gap had resolved. The patient was successfully extubated and maintained on supplemental oxygen. Intravenous insulin was discontinued, and subcutaneous insulin therapy was initiated.

The patient was transferred to the ward and, on POD-7, discharged on subcutaneous insulin with instructions for close endocrinological follow-up to manage newly diagnosed diabetes.

Case 3

A 45-year-old male patient, weighing 68 kg and measuring 178 cm in height, was scheduled to undergo Wide Local Excision (WLE) with Pectoralis Major Myocutaneous (PMMC) flap reconstruction. Preoperative evaluation was unremarkable, with no prior history of surgical procedures or anaesthesia exposure. His Random Blood Sugar (RBS) was 138 mg/dL.

The procedure was performed under general anaesthesia and lasted approximately 7 hours. Intraoperatively, the patient was maintained under sedation using a fentanyl infusion, along with intermittent bolus doses of atracurium for neuromuscular blockade. The surgery proceeded without complications, and the patient was transferred to the ICU while intubated with a 7.5 mm cuffed nasal endotracheal tube and placed on mechanical ventilation.

Upon ICU admission, the patient exhibited tachycardia (heart rate of 138 bpm) and hypotension (blood pressure of 98/52 mmHg). Despite receiving additional sedation and fluid boluses, the hypotension persisted and worsened, necessitating initiation of low-dose vasopressor support. Arterial Blood Gas (ABG) analysis showed metabolic acidosis with a pH of 7.18 and an anion gap of 26 (Table 5). Concurrently, blood glucose was significantly elevated at 492 mg/dL.

Management was initiated with resuscitation using 0.9% normal saline, and intravenous insulin therapy was started with bolus doses followed by continuous infusion. Once euvolemia was achieved, insulin was tapered based on targeted blood glucose monitoring. As the anion gap narrowed and acidosis resolved (Table 6), the patient's condition stabilized.

The patient was successfully extubated on postoperative day 2 with stable vital signs and controlled RBS. Intravenous insulin infusion was transitioned to subcutaneous insulin before discontinuation. The patient was shifted to the ward on POD-4 with continued RBS monitoring and discharged on POD-7 with subcutaneous insulin therapy and a scheduled follow-up with an endocrinologist.

Table 1

Ph	7.19
Po_2	94
Pco_2	32
Hco_3	8
BE	-16
AG	24
Na/ k	134 / 4.1

Table 2

Ph	7.38
Po_2	108
Pco_2	41
Hco_3	23
BE	-1.5
AG	9
Na/ k	146 / 4.8

Table 3

Ph	7.23
Po_2	89
PCo_2	38
Hco_3	10
BE	-13
AG	21
Na/ k	140/ 4.5
Ph	7.42
Po_2	125
Pco_2	39
Hco_3	25
BE	+1
AG	8
Na/ k	139/ 4.2

Table 4

Ph	7.18
Po_2	154
Pco_2	28
Hco_3	6
BE	-18
AG	26
Na/ k	144 / 3.9

Table 5

Ph	7.389
Po_2	89
Pco_2	37
Hco_3	22
BE	-2
AG	11
Na/ k	136 / 4.1

Discussion

Diabetic Ketoacidosis (DKA) is identified through a combination of elevated blood glucose levels (generally over 250 mg/dL), decreased blood pH indicating acidosis, reduced bicarbonate levels, and the presence of ketones in either the blood or urine.

Elevated Blood glucose

A blood glucose level exceeding 250 mg/dL (13.9 mmol/L) is a significant marker.

Metabolic acidosis

- **Decreased blood pH:** An arterial pH below 7.3 is a defining characteristic of diabetic ketoacidosis (DKA).
- **Reduced bicarbonate levels:** Serum bicarbonate levels under 18 mEq/L—typically between 15 to 18 mEq/L in mild cases—also support the diagnosis.

Presence of ketones:

- **Ketonemia:** Detection of ketones in the bloodstream.
- **Ketonuria:** Detection of ketones in the urine.

The physiological stress induced by surgery and anaesthesia likely triggered underlying, previously undiagnosed diabetes mellitus, leading to its first presentation as overt Diabetic Ketoacidosis (DKA). Intraoperative blood glucose monitoring was not considered necessary at the time. The seemingly stable intraoperative period may be attributed to the masking of DKA symptoms by the effects of general anaesthesia, with clear clinical signs only emerging in the early postoperative phase once anaesthetic effects diminished.

DKA is associated with a mortality rate ranging from 2% to 5%. Various factors—including infections, missed or insufficient insulin doses, alcohol intake, surgical interventions, prolonged fasting, or dietary indiscretions—can precipitate or accelerate the onset of diabetes mellitus, sometimes previously unrecognized, leading to an acute presentation as DKA. Common symptoms at presentation include nausea, vomiting, and abdominal pain.

Anaesthesia and surgery trigger a typical metabolic stress response, leading to the release of catabolic hormones such as epinephrine, norepinephrine, cortisol, glucagon, and growth hormone. This stress response suppresses both insulin secretion and its physiological action. Catecholamines promote gluconeogenesis and glycogenolysis while simultaneously inhibiting glucose uptake and insulin release. Additionally, these hormones drive lipolysis and ketone production. Beyond the insulin resistance caused by circulating stress hormones, surgical stress can also impair pancreatic β-cell function. This results in decreased plasma insulin levels and a diminished insulin response to glucose. Although the exact mechanism remains unclear, this dysfunction shows a poor correlation with intraoperative catecholamine levels but a strong inverse relationship with plasma epinephrine postoperatively.

Perioperative hyperglycaemia may also be influenced by other factors, including the administration of dextrose-containing fluids, hypothermia, elevated lactate levels, steroid use, and heparin therapy. In this case, only 0.9% normal saline was used intraoperatively, and no exogenous steroids were given. Furthermore, the 7-hour preoperative fasting period, combined with severe hyperglycaemia and a high anion gap metabolic acidosis, was not indicative of starvation ketoacidosis.

Conclusion

Detecting patients with undiagnosed glucose intolerance during the preoperative period—especially in the absence of clear symptoms or medical history—and ensuring appropriate management can help lower the risk of complications like Diabetic Ketoacidosis (DKA) or hyperosmolar Hyperglycaemic State (HHS). Given the high prevalence of unrecognized diabetes in the general population, early identification and intervention are crucial for enhancing patient outcomes.

Clinical significance

- Diabetic ketoacidosis can occur in the perioperative setting not only under general anesthesia but also in the postoperative setting. Other factors may include intraoperative steroid administration, inadequate depth of anesthesia, and pre-existing conditions contributing to diabetic ketoacidosis.

- Supportive therapy with fluid resuscitation and insulin therapty is key in the management of DKA.

Declarations

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Patient consent statement: We hereby confirm that we have obtained written informed consent from the patient in this case report for publication of their clinical information

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