A pregnant patient with DKA, septic shock and a lactate mystery

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Abstract

Diabetic ketoacidosis (DKA) is a life-threatening complication in pregnant diabetic mothers. Infection, especially involving the urinary tract in females is the most prominent precipitating factor for DKA. When a patient in DKA presents with uncontrolled hyperglycemia, ketonemia and hypovolemia, intravenous fluids and insulin infusions according to the blood glucose levels become the cornerstone of treatment.

Keywords

Diabetic ketoacidosis; surrogate pregnancy; sepsis; acute kidney injury; renal replacement therapy

Background

Poor control over the blood sugar levels in a diabetic patient makes them susceptible to infections. When the host mounts a dysregulated response to this infection, it leads to serious organ dysfunction described as sepsis, which in turn can precipitate ketoacidosis. In a pregnant diabetic mother, the normal physiological changes and altered metabolic demands can trigger DKA. This is a case report about a pregnant woman with infection - precipitated DKA presenting to the hospital in septic shock. We report this case to highlight the challenging circumstances in which the patient was treated and to analyze the clinical outcomes.

Case Presentation

A 33-year-old G3P2L2 was a surrogate mother at 8 weeks of gestation. She presented to the ER in a drowsy and unresponsive state. Earlier, she had complained of breathlessness, which had worsened over the day, and constipation with abdominal distension for the past 2 days. The patient had also complained of a swelling in the groin region associated with purulent discharge per vaginum for the past 3 days.

She was on steroid and hormone replacement therapy as a part of Assisted Reproductive Technology (ART). There was no known past medical illness.

Her vitals on arrival were Heart Rate (HR) of 135 beats/min, Blood pressure of 90/50 mm Hg, SPO2 of 98% with 15-liter O2/min via NRBM and a Respiratory Rate of 28 breaths/min. Peripheries were cold and clammy. Initial arterial blood gas analysis showed severe metabolic acidosis and blood glucose level of 494 mg/dl (Table 1) Urine ketones were positive.

Table (1) ABG on arrival and after 2 h.

Parameter	Arrival	After 2 h
Mode of oxygen support	15 l/min NRBM.	80% FiO2 VC mode
pH	6.89	7.08
PO2	141	304
PCO2	12	17
HCO3 std	<3.0	7.0
Lactate	1.1	0.4
Na+	122	128
K+	4.6	3.9
Base excess	-28.3	-23.1

In view of poor GCS E2 V2 M5, tachypnea, and hemodynamic instability, the airway was secured after obtaining consent and the patient was mechanically ventilated.

An Arterial line and Central Venous Catheter were secured. Patient was catheterized and urine output was monitored.

Based on the provisional diagnosis of diabetic ketoacidosis, fluid resuscitation and insulin infusion were initiated. An initial fluid bolus of 2 L of normal saline was followed with noradrenaline infusion. Further fluid resuscitation was continued according to

dynamic parameters of fluid responsiveness.

Insulin infusion was initially started at 4 units per hour and further adjustments were made according to hourly capillary blood glucose monitoring. Serum beta hydroxybutyrate was 4.6 mol/L.

Other significant labs on admission included leucocytosis (20,250 cells/cumm), elevated serum procalcitonin (1.74 ng/ml) and a high HbA1c (12.5). Blood and urine samples were drawn for culture sensitivity tests and empirical antibiotics therapy with meropenem was started within 1 h. Serial arterial blood gas analyses were done at regular intervals to monitor pH and serum electrolyte levels. Screening echocardiogram was normal. Abdominal ultrasound showed bilateral increased renal cortical echoes, thickened edematous gallbladder wall and thickened endometrium with minimal fluid collection in it.

A Transvaginal Ultrasound showed a single, intrauterine, gestational sac with no fetal cardiac activity. Per vaginal examination showed a sequel area of a small ruptured vaginal abscess.
The patient had a fever spike of 101°F and was persistently tachycardic. Her level of consciousness improved and she opened her eyes to call in around 12 h of ICU admission. In spite of adequate fluid resuscitation and vasopressor support, the patient continued to be anuric and had persistent metabolic acidosis.

On the second day in ICU Continuous Renal Replacement Therapy was initiated (creatinine 3.15 mg/dl) and patient required dual vasopressor support (INJ. Noradrenaline and INJ. Vasopressin) to maintain blood pressure. Blood sugar levels and serum electrolyte levels were monitored at regular intervals. During this period, Insulin infusion was continued and she also received intravenous potassium supplements (Table 2). Overnight, the metabolic acidosis improved and her vasopressor requirement decreased.

Table (2) CBG levels and daily insulin requirements

Date	Highest CBG in mg/dl	Mean CBG in mg/dl	Lowest CBG in mg/dl	TotalInsulin given in units
Day 1	464	356	233	35U IV infusion
Day 2	318	199	133	49U IV infusion
Day 3	245	183	121	11.5U IV infusion
Day 4	331	200	86	16U S/C

On day 3, Patient was fully awake and responding to commands but failed the spontaneous breathing trial. She was weaned off the ventilator and extubated on day 4. The same day she was off vasopressors and CRRT was discontinued. However, her urine output remained minimal (creatinine 1.64 mg/dl) and she continued to be on SLED. During her ICU stay, she received nutritional support, DVT prophylaxis and physiotherapy as per ICU protocol. Swabs taken from the vaginal abscess grew Candida albicans. All other cultures (blood and urine) turned out to be negative for microorganism growth. Surprisingly in spite of severe shock requiring fluid resuscitation and vasopressors, she never manifested lactic acidosis at any point in time (Table 3). We were unable to explain this.

Table (3) ABG before, during and after CRRT

Parameter	ICU day 1	ICU day 1 2 h after initiating CVVHD	ICU day 2
Mode of oxygen support	40% FiO2 in VC mode	40% FiO2 in VC mode	40% FiO2 in VC mode
pH	7.12	7.36	7.37
PO2 (mmHg)	175	190	201
PCO2 (mmHg)	27	26	32
HCO3 std (mmol/L)	10.2	17.4	20.2
Lactate (mmol/L)	0.7	1.0	1.0
Na+ (mmol/L)	140	143	142
K+ (mmol/L)	3.0	2.8	2.9
Base excess (mmol/L)	-19	-9.7	-6.1

Outcome and follow up

Oral diabetic diet, physiotherapy and mobilization were continued on a regular basis during the patient's stay in the hospital. She was discharged home after adequate counselling on the need for regular blood glucose

monitoring with insulin administration. She was also advised follow up for blood sugar management and dialysis.

Discussion

This patient had undiagnosed diabetes mellitus. Sepsis, pregnancy and steroid supplementation used as a part of ART were the precipitating factors for diabetic ketoacidosis in this patient. In addition, the hormonal supplementation she was receiving would have further confounded the situation. We were baffled by the absence of lactic acidosis in spite of her presentation with septic shock and AKI requiring dual vasopressor support and RRT respectively. However, all her initial cultures (both blood and urine) turned negative except for the vaginal swab as mentioned earlier. We were unable to identify any other foci of infection. Infections especially involving the urinary tract, with a female predominance have been an established cause of DKA [1,2]. The hormones secreted during pregnancy increase insulin resistance and cause hyperglycaemia. This and the decreased overall buffering capacity leads to worsening acidosis, severe fluid depletion and marked metabolic disturbances [3]. The presence of AKI in this patient would certainly have worsened her metabolic acidosis. The clinical presentation can vary, ranging from non-specific nausea, vomiting or dehydration to a full-blown shock as with this patient.

The diagnosis of DKA in pregnancy is focused on laboratory abnormalities that include hyperglycemia (>200–300 mg/dL), anion gap acidosis (AG >12mEq/L), pH <7.30, HCO3 <15 mEq/L, and ketonemia or ketonuria [3]. Aggressive and early fluid resuscitation and intravenous insulin help in correcting acidosis and hyperglycemia [2]. Underlying infection or other causes of DKA should be concurrently pursued and treated at the earliest. The acute kidney injury due to organ dysfunction is a risk factor for mortality and significantly prolongs the hospital stay in critically Ill patients [4].

Conclusion

Our patient didn't fully recover her renal functions at the time of discharge and required renal replacement therapy which stresses the importance of how quickly sepsis can devolve into a life-threatening complication with significant morbidity.

References

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