

# Chapter 38

## Blood Gas III

Pramod Chetty

A patient with closed fracture of the lower extremity is scheduled for an ORIF. The patient is an unaccompanied, slender, 26-year-old male who cannot give a good history due to confusion and has deep, rapid breathing with a distinctive odor. His vital signs show mild hypotension, tachycardia, and low-grade fever. Investigations demonstrate  $\text{Na}^+$  132,  $\text{K}^+$  4.8,  $\text{Cl}^-$  92,  $\text{HCO}_3^-$  12, BUN 24 mg, creatinine 1.6 mg,  $\text{Ca}^{++}$  7.8 mg, and blood sugar of 318 mg/dl. Arterial blood gas shows a pH of 7.24,  $\text{PCO}_2$  28,  $\text{PO}_2$  76,  $\text{HCO}_3$  12, BE of 14, and  $\text{O}_2$  sat of 93%. His CBC is normal with mild leukocytosis and evidence of hemoconcentration. The chest X-ray is unremarkable and EKG shows sinus tachycardia.

1. What is the likely initial diagnosis of this patient and how can you confirm the diagnosis?
2. What are abnormal laboratory values in the BMP and ABGs that are seen in this condition?
3. What is the major differential diagnosis in this clinical condition?
4. What are the principles in the treatment of this condition?
5. How do the results of the BMP and ABG trend during the treatment of this condition?
6. How will you continue management of this patient with the planned surgery?

---

P. Chetty, MD  
Department of Anesthesiology, OU Health Sciences Center,  
750 NE 13th Street, Suite 200, PO Box 53168, Oklahoma City, OK 73104, USA  
e-mail: [Pramod-chetty@ouhsc.edu](mailto:Pramod-chetty@ouhsc.edu)

## Answers

1. The presentation of this young patient with altered sensorium, “Kussmaul” breathing, hyperglycemia, and metabolic acidosis strongly suggests diabetic ketoacidosis (DKA). The diagnosis can be *confirmed by the presence of ketone bodies* in the urine and serum [1]. Concomitant lactic acidosis must also be investigated [2, 3]. As with any patient with a traumatic injury and altered sensorium, radiological testing for cervical spine and cranial pathology must be done.
2. The laboratory values in DKA will show evidence of metabolic acidosis, electrolyte derangements, and evidence of severe dehydration [4].

### (a) BMP

- $\text{Na}^+$ —there is a total body loss of  $\text{Na}^+$ ; the levels can be low normal. Correction must be made for undermeasurement of  $\text{Na}^+$  due to hyperglycemia (add 1.6 meq/L to the measured  $\text{Na}^+$  for every 100 mg of glucose above 100 mg/dl level).
- $\text{K}^+$ —there can be a significant total body loss of 3–10 meq/kg of  $\text{K}^+$ . The initial serum  $\text{K}^+$  level may be paradoxically high due to both volume contraction and decreased movement into the intracellular compartment [1].
- $\text{Cl}^-$ —will be decreased.
- $\text{HCO}_3^-$ —will be decreased.
- Anion gap—will be increased above normal 10–14 meq/L [5]. This gap is calculated by the formula:

$$\text{AG} = \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$$

- BUN—will be increased.
- Creatinine—may be mildly increased.
- $\text{Ca}^{++}$ —may be decreased. Additionally magnesium and phosphate depletion can also occur.
- Glucose—increases to levels greater than 250–600 mg/dl [4] but *rarely* may be normal, when called euglycemic DKA [6].

### (b) ABG

- pH—usually less than 7.3
  - $\text{PaCO}_2$ —usually lower due to respiratory compensation for metabolic acidosis
  - $\text{PaO}_2$ —usually low normal unless a pneumonic process causes it to be low
  - $\text{HCO}_3^-$ —will be lower due to metabolic acidosis
  - BE—will be lower to indicate significant metabolic acidosis
  - $\text{O}_2$  saturation—will be in the low 90 s with  $\text{O}_2$  supplementation unless a pneumonic process causes it to be lower
3. The major differential diagnosis in this scenario would be non-ketotic hyperosmolar hyperglycemia (NHH) [1]. In this condition the patient is generally a type 2 diabetic and as such would likely be an older and often overweight patient. The patient can

present with altered mentation or in a coma. The blood sugar levels are frequently higher ( $>600$  mg/dl) and there is no ketone body formation [4]. Therefore metabolic acidosis if present would likely be due to the precipitant cause such as infection with lactic acidosis. The reason for the absence of ketone bodies is due to the presence of some circulating insulin. This insulin can prevent the alteration in fatty acid metabolism leading to ketosis but due to peripheral insulin resistance still leads to very high serum glucose levels [6]. The presence of increased insulin counter regulatory hormones (esp. glucagon) exacerbates the hyperglycemia due to increased hepatic gluconeogenesis [7, 8]. The resultant osmotic diuresis leads to the severe dehydration ( $\sim 12$  L loss), azotemia, and hyperosmolarity ( $>330$  mOsm/L) [4].

Serum osmolarity is calculated by the formula  $2(\text{Na}^+ + \text{K}^+) + \text{Glucose}/18 + \text{BUN}/2.8$ .

The precipitating causes can be infection, stoppage of medication, newly diagnosed diabetes, stroke, MI, subdural hematoma, and GI diseases. The treatment of this condition is hydration, correction of electrolyte aberrations, and treatment of the causative process. Insulin use will be needed to gradually bring down the blood sugar.

#### 4. The principles for treatment of DKA are

- (a) Insulin therapy to decrease hyperglycemia and stop production of ketone bodies.
- (b) Hydration with isotonic solutions. Deficit may be up to 9 L in the average adult.

Start with saline and convert to isotonic fluids with  $\text{K}^+$  when  $\text{K}^+$  levels start to decrease, and urine output is maintained [9]. Change to hypotonic solution if  $\text{Na}^+$  level  $>150$  meq/L [6].

Bicarb therapy is only reserved for severe acidosis ( $\text{pH} < 7.1$ ).

- (c) Replacement of other specific electrolytes  $\text{Ca}^{++}$ ,  $\text{Mg}^{++}$ ,  $\text{PO}_4$ .
  - (d) Treatment of precipitating cause—infections, interruption of insulin, MI, trauma, stress.
  - (e) Mental status changes—may need to have airway protected and ventilator assistance.
  - (f) Ileus and other GI presentations, e.g., acute cholecystitis, either due to systemic ketosis or incidental, must be clinically managed.
- #### 5. The trending changes for electrolytes and the ABG with treatment will be:

##### (a) BMP

- $\text{Na}^+$ —should be in the upper normal range.
- $\text{K}^+$ —after initial fluid resuscitation with use of NS (first 4 h), the  $\text{K}^+$  levels will drop associated with the intracellular migration due now to the presence of insulin.  $\text{K}^+$  can be added to IV fluids once the level goes below 4 meq/L, and a steady urine output is maintained.
- $\text{Cl}^-$ —will increase with use of normal saline (NS). Excessive use of NS can lead to hyperchloremic acidosis.
- $\text{HCO}_3^-$ —use of replacement  $\text{NaHCO}_3$  is not required unless acidosis is severe ( $\text{pH} < 7.1$ ).

- Anion gap—will move toward normal gap of <11 meq/L.
- BUN—azotemia, if present, will normalize with hydration and increased urine production.
- Creatinine—as volume status and GFR improves, it should normalize unless kidneys are affected.
- $\text{Ca}^{++}$ —can be low due to loss from osmotic diuresis—careful augmentation along with associated  $\text{Mg}^{++}$  and phosphate supplementation for their measured deficiencies.
- Glucose—the target is to gradually bring the blood sugar (BS) level down ~75–100 mg/h using regular insulin as an IV bolus (0.1 u/kg) followed by continuous infusion IV (0.1 u/kg/h) [4]. Rates of insulin infusion can be progressively ramped up with use of any standard protocol. Once BS levels reach the lower 200 s/dl, then 5% glucose should be added to the IV fluids to prevent hypoglycemia [10]. Target blood sugar is in the range 120–150 mg/dl.

(b) ABG

- pH—with hydration alone, the acidosis should start to correct. Insulin is needed to prevent further ketone production and bring down glucose levels.
  - $\text{PaCO}_2$ —as metabolic acidosis is corrected, the respiratory alkalosis should normalize.
  - $\text{PaO}_2$ —with normalization of vascular volume, the oxygenation should improve.
  - $\text{HCO}_3$ —if there is severe metabolic acidosis, then correction with exogenous bicarb will be required. Level should normalize with decreased ketone body formation and elimination of the same by the normal buffer systems.
  - BE—abnormality will normalize with therapy.
  - $\text{O}_2$  saturation—with fluid resuscitation the maintenance of normal  $\text{O}_2$  saturation will be easier.
6. Once the patient has had definitive treatment for DKA and has shown metabolic stabilization, surgery can proceed. The principles for perioperative management would include:
- (a) Continuing the use of appropriate fluids and electrolyte and IV insulin administration by infusion.
  - (b) Precautions for a full stomach before induction if not already intubated.
  - (c) Type 1 diabetics can have a difficult airway due to stiffening of tissues of the upper airway and rigidity of the cervical spine [7].
  - (d) Arterial line and good venous access for this particular case would be appropriate. Central venous access for volume estimation in major surgery or in patients with comorbidity would be appropriate.

- (e) Glucose checks at least hourly under anesthesia with BMP and ABG at regular intervals.
- (f) At the end of the procedure, extubation would depend on preinduction status, intraoperative course, and emergence profile. The postoperative care should continue in an ICU setting with treatment for both initiating and coexisting clinical issues.
- (g) Once stable, the diet and treatment plan must be made with type, amount, and route of administration of insulin determined.

## References

1. Magee MF, Bhatt BA. Management of decompensated diabetes. Diabetic ketoacidosis and hyperglycemic hyperosmolar syndrome. *Crit Care Clin.* 2001;17(1):75–106.
2. Lu J, Zello GA, Randell E, Adeli K, Krahn J, Meng QH. Closing the anion gap: contribution of D-lactate to diabetic ketoacidosis. *Clin Chim Acta.* 2011;412(3–4):286–91.
3. Cox K, Cocchi MN, Saliccioli JD, Carney E, Howell M, Donnino MW. Prevalence and significance of lactic acidosis in diabetic ketoacidosis. *J Crit Care.* 2012;27(2):132–7.
4. Kasper DL, Jameson JL, Hauser S, Loscalzo J, Fauci AS, Longo D, editors. *Harrison's principles of internal medicine.* 19th ed. New York: McGraw-Hill Medical; 2015. Chap. 418.
5. Roizen MF, Fleisher LA. Anesthetic implications of concurrent disease. In: Miller RD, editor. *Miller's anesthesia.* 8th ed. Philadelphia: Churchill Livingstone/Elsevier; 2015.
6. Delaney MF, Zisman A, Kettyle WM. Diabetic ketoacidosis and hyperglycemic hyperosmolar nonketotic syndrome. *Endocrinol Metab Clin N Am.* 2000;29(4):683–705.
7. McAnulty GR, Robertshaw HJ, Hall GM. Anaesthetic management of patients with diabetes mellitus. *Br J Anaesth.* 2000;85(1):80–90.
8. Nattrass M. Diabetic ketoacidosis. *Medicine.* 2010;38(12):667–70.
9. Chua HR, Venkatesh B, Stachowski E, Schneider AG, Perkins K, Ladanyi S, et al. Plasma-Lyte 148 vs 0.9% saline for fluid resuscitation in diabetic ketoacidosis. *J Crit Care.* 2012;27(2):138–45.
10. Nyenwe EA, Kitabchi AE. Evidence-based management of hyperglycemic emergencies in diabetes mellitus. *Diabetes Res Clin Pract.* 2011;94(3):340–51.