SOUTHWEST JOURNAL of PULMONARY & CRITICAL CARE

Journal of the Arizona, New Mexico, Colorado and California Thoracic Societies <u>www.swjpcc.com</u>

April 2021 Critical Care Case of the Month: Abnormal Acid-Base Balance in a Post-Partum Woman

Mohammad Abdelaziz Mahmoud, MD, DO Andrea N. Pruett, BS

Emanuel Medical Center Turlock, CA 95382

History of Present Illness

A 29-year-old healthy woman, who is 8 weeks postpartum, presented to the emergency department with severe shortness of breath, fast shallow breathing, nausea, several episodes of nonbloody nonbilious emesis, abdominal pain and malaise for 1 week. The patient delivered a healthy boy at full-term by spontaneous vaginal delivery. Her pregnancy was uneventful. She denied smoking or use of alcohol.

Physical Exam

On presentation to the emergency department her blood pressure was found to be 121/71, temperature 36.8°C, pulse 110 beats per minute, respiratory rate 20 breaths per minute and SpO2 saturation of 99% while breathing ambient air. Physical exam was remarkable except for dry mucous membranes, sinus tachycardia, and tachypnea with mild epigastric tenderness with light palpation. Which of the following *should be done*?

- 1. Complete blood count (CBC)
- 2. Metabolic panel
- 3. Chest x-ray
- 4. Arterial blood gases (ABGs)
- 5. All of the above

Key Words: acid base balance, acidosis, anion gap, breast feeding, ketones, metabolic acidosis, mudpiles, post-partum, shortness of breath, starvation ketosis

Correct! 5. All of the above

It is not clear why this woman presented with shortness of breath. Her physical examination reveals tachypnea and tachycardia but is otherwise fairly unremarkable. There are no obvious signs or symptoms pointing to a pulmonary or cardiac origin. Therefore, laboratory and radiology evaluations are indicated to narrow a huge differential diagnosis.

Her chest-ray was unremarkable as was her CBC. Results of the ABGs and metabolic panel are below.

Table 1. Initial ABGs and metabolic panel.

- pH 7.08
- pCO₂ 18mmol/L
- Sodium (Na⁺)136 Meq/L
- Potassium (K⁺) 5.2 Meq/L
- Chloride (Cl) 103 Meq/L

What *acid-base disturbance* is present?

- 1. Combined respiratory/metabolic acidosis
- 2. Metabolic acidosis
- 3. Metabolic alkalosis
- 4. Respiratory acidosis
- 5. Respiratory alkalosis

Correct! 2. Metabolic acidosis

The patient has a low pH of 7.08 which is below the normal pH of 7.40, so the patient has an acidosis. The pCO₂ is low at 18 compared to the normal 40. Therefore, the patient has a metabolic acidosis rather than a respiratory acidosis. In fact, the patient is hyperventilating resulting in a low pCO₂ partially compensating for her acidosis and explaining her tachypnea.

Metabolic acidosis results from accumulation of an acid and is divided into two groupsnormal anion gap and high anion gap. The anion gap is calculated by the following formula: Anion Gap = Sodium - (Chloride + Bicarbonate). There are a small of anions not accounted for by the formula so a normal anion gap is 8-16. The current patient's anion gap is high:

Na⁺136-(Cl⁺103+Bicarbonate 4) =Anion gap 19

Which of the following are *causes of high anion gap metabolic acidosis*?

- 1. Diabetic ketoacidosis
- 2. Ethylene glycol
- 3. Methanol
- 4. Uremia
- 5. All of the above

Correct! 5. All of the above

A mnemonic commonly used to remember the causes of a high anion gap metabolic acidosis is "goldmark" where-

G - glycols (ethylene glycol & propylene glycol) O - oxoproline, a metabolite of paracetamol / acetaminophen<math>L - L-lactate, the chemical responsible for lactic acidosis D - D-lactate M - methanol A - aspirin R - renal failure K - ketoacidosis, ketones generated from starvation, alcohol, and diabetic ketoacidosis.

"Mudpiles" is another commonly used mnemonic for high anion gap metabolic acidosis where-

M – Methanol

U – Uremia (chronic kidney failure)

D – Diabetic ketoacidosis

P – Paracetamol, Propylene glycol (used as an inactive stabilizer in many medications; historically, the "P" also stood for Paraldehyde, though this substance is not commonly used today)

I – Infection, Iron, Isoniazid (which can cause lactic acidosis in overdose), Inborn errors of metabolism (an especially important consideration in pediatric patients)

L – Lactic acidosis

E – Ethylene glycol

S – Salicylates

Working through "MUDPILES" a number of metabolic parameters were measured including-

> Methanol-methanol level normal Uremia-BUN normal

Diabetic ketoacidosis- beta hydroxybutyrate 9.47 mmol/L (normal < 0.5), urine ketones 2+, blood glucose 123 mg/dL (normal < 125). Lactate 0.4 mmol/L (normal <2.2) Salicylates-undetectable

Which of the following <u>most likely</u> accounts for the patient's acidosis?

- 1. Diabetic ketoacidosis
- 2. Ethanol intoxication
- 3. Ethylene glycol poisoning
- 4. Lactic acidosis from sepsis syndrome
- 5. Starvation ketosis

Correct! 5. Starvation ketosis

The patient reported that she was adherent to a restricted keto diet while she was breastfeeding her 8 weeks baby. A diagnosis of high anion gap metabolic acidosis due to keto diet on lactation was made. She was admitted to the ICU and she was restarted on an oral carbohydrate diet, a bicarbonate drip and IV fluids. Her anion gap and beta hydroxybutyrate normalized and she was discharged on after 3 days of hospitalization. Prior to discharge, the patient was educated that a keto diet is contraindicated during lactation.

References

- Gupta L, Khandelwal D, Kalra S, Gupta P, Dutta D, Aggarwal S. Ketogenic diet in endocrine disorders: Current perspectives. J Postgrad Med. 2017 Oct-Dec;63(4):242-251. [CrossRef] [PubMed]
- Szulewski A, Howes D, Morton AR. A severe case of iatrogenic lactation ketoacidosis. BMJ Case Rep. 2012 Mar 8;2012:bcr1220115409. [CrossRef] [PubMed]
- Paoli A, Rubini A, Volek JS, Grimaldi KA. Beyond weight loss: a review of the therapeutic uses of very-low-carbohydrate (ketogenic) diets. Eur J Clin Nutr. 2013 Aug;67(8):789-96. [CrossRef] [PubMed]
- Mohammad MA, Sunehag AL, Chacko SK, Pontius AS, Maningat PD, Haymond MW. Mechanisms to conserve glucose in lactating women during a 42-h fast. Am J Physiol Endocrinol Metab. 2009 Oct;297(4):E879-88. [CrossRef] [PubMed]