64e Fluid and Electrolyte Imbalances and Acid-Base Disturbances: Case Examples

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CASE 1

A 23-year-old woman was admitted with a 3-day history of fever, cough productive of blood-tinged sputum, confusion, and orthostasis. Past medical history included type 1 diabetes mellitus. A physical examination in the emergency department indicated postural hypotension, tachycardia, and Kussmaul respiration. The breath was noted to smell of "acetone." Examination of the thorax suggested consolidation in the right lower lobe.

Laboratory Data	Value	Units
Sodium	130	meq/L
Potassium	5.0	meq/L
Chloride	96	meq/L
CO2	14	meq/L
Blood urea nitrogen (BUN)	20	mg/dL
Creatinine	1.3	mg/dL
Glucose	450	mg/dL
Arterial Blood Gases	**On Room Air**	
pH	7.39	
P_{CO_2}	24	mmHg
P_{aO_2}	89	mmHg
$[HCO_3^-]$	14	meq/L
Anion gap	20	meq/L
Urinalysis		
Urine ketones	Positive 4+	
Glucose	Positive 4+	
Serum Ketones	Strongly positive 1:8	
Chest X-Ray		
Pneumonic infiltrate, right lower lobe		

APPROACH TO DIAGNOSIS

The diagnosis of the acid-base disorder should proceed in a stepwise fashion:

1. The normal anion gap (AG) is 8–10 meq/L, but in this case, the AG is elevated (20 meq/L). Therefore, the change in AG (ΔAG) = ~10 meq/L.
2. Compare the ΔAG and the $\Delta[HCO_3^-]$. In this case, the ΔAG, as noted above, is 10, and the $\Delta[HCO_3^-]$ (25 – 14) is 11. Therefore, the *increment in the AG* is approximately equal to the *decrement in bicarbonate*.
3. Estimate the respiratory compensatory response. In this case, the predicted P_{aCO_2} for an $[HCO_3^-]$ of 14 should be approximately 29 mmHg. This value is obtained by adding 15 to the measured $[HCO_3^-]$ (15 + 14 = 29) or by calculating the predicted P_{aCO_2} from the Winter equation: $1.5 \times [HCO_3^-] + 8$. In either case, the predicted value for P_{aCO_2} of 29 is significantly higher than the measured value of 24. Therefore, the prevailing P_{aCO_2} exceeds the range for compensation alone and is too low, indicating a superimposed respiratory alkalosis.
4. Therefore, this patient has a *mixed acid-base disturbance* with two components: (a) high AG acidosis secondary to ketoacidosis and (b) respiratory alkalosis (which was secondary to community-acquired pneumonia in this case). The latter resulted in an additional component of hyperventilation that exceeded the compensatory response driven by metabolic acidosis, explaining the normal pH. The finding of respiratory alkalosis in the setting of a high AG acidosis suggests another cause of the respiratory component. Respiratory alkalosis frequently accompanies community-acquired pneumonia.

The clinical features in this case include hyperglycemia, hypovolemia, ketoacidosis, central nervous system (CNS) signs of confusion, and superimposed pneumonia. This clinical scenario is consistent with diabetic ketoacidosis (DKA) developing in a patient with known type 1 diabetes mellitus. Infections in DKA are common and may be a precipitating feature in the development of ketoacidosis.

The diagnosis of DKA is usually not challenging but should be considered in all patients with an elevated AG and metabolic acidosis. Hyperglycemia and ketonemia (positive acetoacetate at a dilution of 1:8 or greater) are sufficient criteria for diagnosis in patients with type 1 diabetes mellitus. The $\Delta[HCO_3^-]$ should approximate the increase in the plasma AG (ΔAG), but this equality can be modified by several factors. For example, the ΔAG will often decrease with IV hydration, as glomerular filtration increases and ketones are excreted into the urine. The decrement in plasma sodium is the result of hyperglycemia, which induces the movement of water into the extracellular compartment from the intracellular compartment of cells that require insulin for the transport of glucose. Additionally, a natriuresis occurs in response to an osmotic diuresis associated with hyperglycemia. Moreover, in patients with DKA, thirst is very common and water ingestion often continues. The plasma potassium concentration is usually mildly elevated, but in the face of acidosis, and as a result of the ongoing osmotic diuresis, a significant total-body deficit of potassium is almost always present. Recognition of the total-body deficit of potassium is critically important. The inclusion of potassium replacement in the therapeutic regimen at the appropriate time and with the appropriate indications (see below) is essential. Volume depletion is a very common finding in DKA and is a pivotal component in the pathogenesis of the disorder.

APPROACH TO MANAGEMENT

Patients with DKA often have a sustained and significant deficit of sodium, potassium, water, bicarbonate, and phosphate. The general approach to treatment requires attention to all of these abnormalities. Successful treatment of DKA involves a stepwise approach, as follows:

1. *Replace extracellular fluid (ECF) volume deficits.* Because most patients present with actual or relative hypotension and, at times, impending shock, the initial fluid administered should be 0.9% NaCl infused rapidly until the systolic blood pressure is >100 mmHg or until 2–3 L cumulatively have been administered. During the initial 2–3 h of infusion of saline, the decline in blood glucose can be accounted for by dilution and increased renal excretion. Glucose should be added to the infusion as D_5 normal saline (NS) or D_5 0.45% NS once the plasma glucose declines to 230 mg/dL or below.
2. *Abate the production of ketoacids.* Regular insulin is required during DKA as an initial bolus of 0.1 U/kg body weight (BW) IV, followed immediately by a continuous infusion of 0.1 U/kg BW per hour in NS. The effectiveness of IV insulin (not subcutaneous) can be tracked by observing the decline in plasma ketones. Because the increment in the AG above the normal value of 10 meq/L represents accumulated ketoacids in DKA, the disappearance of ketoacid anions is reflected by the narrowing and eventual correction of the AG. Typically, the plasma AG returns to normal within 8–12 h.
3. *Replace potassium deficits.* Although patients with DKA often have hyperkalemia due to insulin deficiency, they are usually severely K^+ depleted. KCl (20 meq/L) should be added to each liter of IV fluids when urine output is established and insulin has been administered.
4. *Correct the metabolic acidosis.* The plasma bicarbonate concentration will usually not increase for several hours because of dilution from administered IV NaCl. The plasma $[HCO^-]$ approaches 18 meq/L once ketoacidosis disappears. Sodium bicarbonate therapy is often not recommended or necessary and is contraindicated