CASE 7.3
Diabetic Ketoacidosis | Level 3

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1. What subjective and objective evidence supports the diagnosis of DKA in this patient?

SUBJECTIVE FINDINGS: Reported high blood glucose and ketones in urine at home, emesis, headaches, and abdominal pain

OBJECTIVE FINDINGS: Respiratory rate 40 breaths per minute with Kussmaul breathing; serum blood glucose of 647 mg/dL; glycosuria of 500 mg/dL; ketonuria greater than 80 mg/dL; metabolic acidosis demonstrated via a venous blood gas (pH= 7.14, pCO_2 = 25 mm Hg, HCO_3 = 9.8 mmol/L, BE = –18.8 mmol/L, SpO_2 = 86.1%); anion gap = 37 mmol/L; Cl^- = 96 mmol/L; and GCS = 15

Based on the patient's presenting laboratory values, she is considered to have moderate DKA with a venous pH less than 7.2 and the HCO_3 less than 10 mmol/L. The likely precipitating event for DKA in this patient was the acute illness and nonadherence to insulin therapy.

2. Describe the necessary laboratory parameters to obtain before developing an appropriate pharmacologic regimen for this patient.

When a patient arrives to the ED with DKA, cardiac monitoring (e.g., ECG) and pulse oximetry are imperative to assess for arrhythmias secondary to hyperkalemia and tissue oxygenation, respectively. Throughout the acute phase, monitoring vital signs—including BP, HR, RR, SpO_2, temperature, intake/output, and GCS—are essential because patients are at greater risk for cerebral edema during this time. A venous blood gas, urinalysis, HbA_{1c}, and urine pregnancy test in females should be done on admission to the ED. In addition, bedside blood glucose monitoring must be done every hour, and a comprehensive metabolic panel (CMP)—including phosphorous and magnesium—are monitored approximately every 4 hours during the acute phase. Depending on the patient's status, this length of time could range from 24 to 48 hours.

Cerebral edema is a complication of DKA with the potential to cause irreversible damage. If a patient has symptoms of cerebral edema, namely altered mental status, a prompt reduction in intracranial pressure is required with the administration of mannitol (dosed at 0.25 to 1 g/kg/dose) or 3% hypertonic saline (dosed as a continuous IV infusion at 0.1 to 1 mL/kg/hr). The onset and progression of cerebral edema usually develops within the first 4 to 12 hours after treatment

initiation; however, it may occur prior to treatment or develop as late as 24 to 48 hours after treatment. Therefore, monitoring should occur hourly in the first 4 to 12 hours and every 4 hours for the next 1 to 2 days after treatment initiation.

3. What are the overall goals of therapy related to DKA? Given these goals, develop a pharmacologic regimen for the treatment of DKA.

There are three overall goals related to the treatment of DKA. Patients presenting with DKA are often dehydrated due to the osmotic diuresis induced by glycosuria. Therefore, the first goal is to provide fluid replacement and volume expansion to improve oxygenation and tissue perfusion. This is necessary to prevent multisystem organ failure. On presentation of a patient with DKA, hydration status must be assessed. The patient was dehydrated as indicated by BUN = 39 mg/dL and SCr = 2.3 mg/dL. Her physical exam had positive findings of hypoxemia (SpO_2 of 86%), hypotension, tachycardia, and tachypnea, which collectively indicate dehydration and volume depletion. IVFs are necessary to expand the intravascular space and improve tissue perfusion and oxygenation. The patient received an IV isotonic crystalloid 0.9% saline at 20 mL/kg over 1 to 2 hours. For this patient, the dose of 700 mL IV over 1 hour was appropriate. The repeat bolus dose was also appropriate if the patient remained hemodynamically unstable indicated by the continued presence of hypotension, tachypnea, and tachycardia. Resuscitation volumes should never exceed 40 to 50 mL/kg (1,750 mL for this patient) in the first 4 hours to avoid the risk of cerebral edema and herniation.

The second goal of therapy is to suppress ketone production by stopping lipolysis (breakdown of triglycerides into free fatty acids and glycerol) and ketogenesis (production of ketone bodies by the liver as an energy source for the brain and heart in periods of starvation). This is accomplished with the administration of insulin, which will also decrease the elevated glucose. In this patient, 1 to 2 hours following the initial fluid bolus, an insulin continuous infusion was started to halt the production of ketones and reduce serum glucose concentrations. The insulin dose of 0.1 units/kg/hr (3.5 mL/hr) in normal saline was appropriate and should be continued until the ketosis resolves, which is indicated by a pH greater than 7.3, bicarbonate level greater than 15 mmol/L, or a resolved anion gap. Reducing the serum glucose level must not exceed a rate of 100 mg/dL/hr; if this rate is exceeded, the insulin dose must be decreased to 0.05 units/kg/hr. The principal outcome and primary goal of insulin therapy is to suppress lipolysis and to reverse ketosis more so than to decrease the serum glucose concentration. Once the ketone production has stopped and oral intake is tolerable, the IV insulin infusions may be transitioned to the subcutaneous (SC) route. The IV infusion should be discontinued 2 hours after the patient eats and SC insulin is administered. At that point, the patient can resume her home insulin pump settings.

Lastly, the third goal of therapy is to restore electrolyte balance and avoid complications such as hypokalemia, hypophosphatemia, hypoglycemia, or cerebral edema. The devastating complication of DKA is cerebral edema; therefore, appropriate management of DKA is crucial. After the initial IVF saline bolus, the remaining fluid deficit should be administered over the next 48 hours with either 0.45% or 0.9% saline infusion depending on the patient's sodium level. Because of the low initial sodium level of 130 mmol/L, the most appropriate option for this patient is 1 liter of 0.9% saline, which contains a higher amount of sodium (154 mEq/L) than the 0.45% saline. The rate of infusion for the IVF should include the maintenance rate plus additional necessary fluid replacement depending on the degree of dehydration on presentation but should not exceed twice the maintenance rate (in this case, 75 mL/hr). The maintenance rate is calculated utilizing the Holliday-Segar method. The patient weighs approximately 35 kg, which would be 1,500 mL for the first 20 kg and 20 mL/kg for each kilogram greater than 20 kg (20 mL/kg × 15 = 300 mL) or 1,800 mL/day. Because this patient initially presented dehydrated, the replacement fluid in normal saline should be infused at a rate of 112.5 mL/hr (approximately 1.5 times the maintenance rate).