EM CASE OF THE WEEK.
BROWARD HEALTH MEDICAL CENTER
DEPARTMENT OF EMERGENCY MEDICINE

BROWARD HEALTH
Care Warriors

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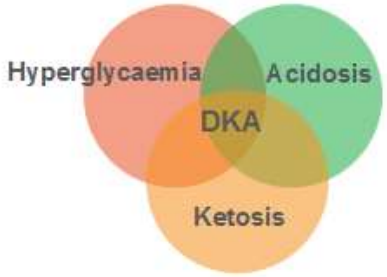
DKA

A 29-year-old man with type 1 diabetes mellitus presents to the emergency department following three bouts of vomiting this morning. He states that he was feeling weak and nauseated for the past two days after eating at a roadside diner. He complains of generalized abdominal pain but denies experiencing diarrhea or constipation. The patient states he was travelling for work this past week for a conference and accidentally left his medication bag at home. His temperature is 98.4°F (36.8°C), blood pressure is 100/75 mm Hg, pulse is 110/min, and respiratory rate is 23 /min. Physical examination shows a distressed thin man, with dry oral mucosa, and poor skin turgor. His abdomen is soft, non-distended, and diffusely tender on palpation. There are no signs of rebound tenderness or guarding. Bowel sounds are auscultated.

Which electrolyte imbalance would you expect to see in this patient? [1]

Choices	Sodium	Bicarbonate	Chloride
A	120	14	94
B	130	30	80
C	135	14	94
D	133	21	100
E	140	24	98



Diabetic Ketoacidosis (DKA) is an acute emergency characterized by hyperglycemia, ketoacidosis, and ketonuria.

It occurs when absolute or relative insulin deficiency inhibits the ability of glucose to enter cells for utilization as metabolic fuel.

Excess fat breakdown and increased ketogenesis from increased free fatty acids, which are made into ketone bodies (beta-hydroxybutyrate >acetoacetate).[2]

EM Case of the Week is a weekly "pop quiz" for ED staff.

The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.

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The correct answer is C.

This patient with type 1 diabetes presents with acute nausea, vomiting, and abdominal pain in the setting of medication non-compliance. This is a classic clinical presentation of **diabetic ketoacidosis** (DKA). On physical examination, patients with DKA usually have signs of volume depletion, including decreased skin turgor, dry oral mucosa, tachycardia, and hyperventilation, which are all seen in this patient. Severe DKA may manifest with neurologic symptoms (i.e. altered mental status).

The fact that the patient has forgotten his medications at home, it is a reasonable assumption that he has missed his insulin injections, precipitating his DKA. The absolute absence of insulin prevents cells from utilizing glucose. Fat becomes an alternative energy source, and in the process of fat breakdown, ketones are formed. The excess ketones cause an anion-gap metabolic acidosis, and patients with DKA compensate by hyperventilating.

Therefore, the diagnosis of DKA requires evidence of hyperglycemia (>300 mg/dL), ketonemia, and a high **anion gap metabolic acidosis.** Serum pH is acidotic (pH < 7.3) and bicarbonate is reduced (<18 mEq/L). Serum anion gap is calculated using the following equation: **Anion gap = Serum Sodium - (Serum Bicarbonate + Serum Chloride).**[1]

Signs/symptoms:

DKA: **D**elirium/psychosis, **K**ussmaul respirations (rapid, deep breathing), **A**bdominal pain/nausea/vomiting, Dehydration.[2]

Labs:

- Hyperglycemia, increased H+, decreased bicarb, increased AG metabolic acidosis, increased blood ketone levels, leukocytosis.
- ***Hyperkalemia depleted intracellular K+ due to transcellular shift from decreased insulin and acidosis.***
- Osmotic diuresis leads to increased K+ loss in urine causing a total body K+ depletion.[3]

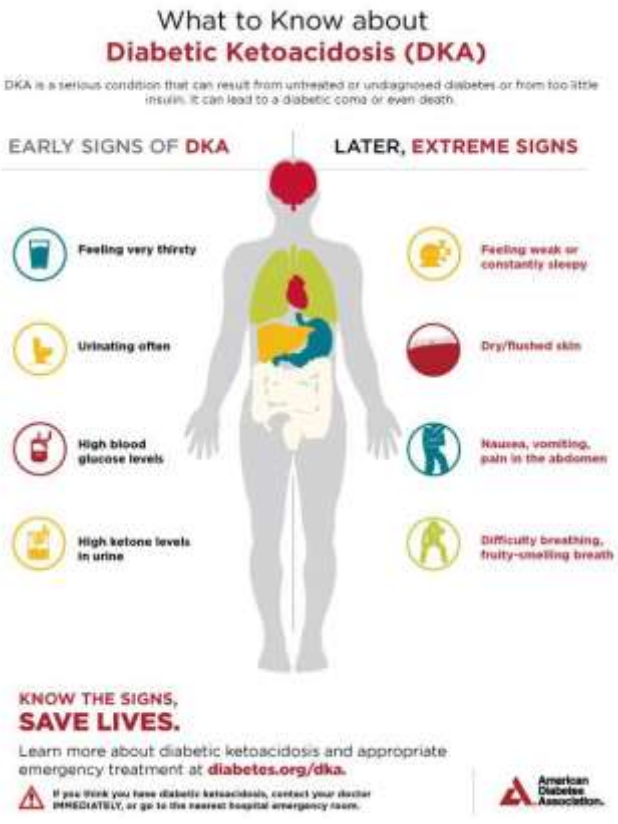
Treatment:

- Correction of fluid loss with intravenous fluids.
- Correction of hyperglycemia with insulin.
- Correction of electrolyte disturbances, particularly potassium loss.
- Correction of acid-base balance.
- Treatment of concurrent infection, if present.[4]

Table 1. Five Actions of Insulin for Diabetic Ketoacidosis (DKA) Treatment

Action	DKA Condition Treated
Drives glucose into cells	Hyperglycemia
Drives potassium into cells	Hyperkalemia
Anabolic	Catabolism
Blocks fat breakdown	Free fatty acid production
Blocks protein breakdown	Ketoacid production

Take Home Points:



ABOUT THE AUTHOR

This month's case was written by Natalie Ceballos. Natalie is a 4th year medical student from FIU HWCOM. She did her emergency medicine rotation at BHMC in October 2019. Natalie plans on pursuing a career General Surgery.

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