EM CASE OF THE WEEK.

BROWARD HEALTH MEDICAL CENTER
DEPARTMENT OF EMERGENCY MEDICINE

BROWARD HEALTH
Care Warriors

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Diabetic Ketoacidosis

A 55-year-old obese male with no past medical history presents to the ED with abdominal pain, nausea, vomiting, polyuria, and polydipsia persisting for the past 24 hours. He has never experienced these symptoms prior to this episode. He denies fever, chills, or any sick contacts. The patient is afebrile, slightly tachycardic and remaining vitals are within normal limits. On physical exam, he has dry oral mucosa and a fruity breath odor. The patient's POC blood glucose was found to be 556 mg/dL. Which of the following is the most appropriate initial treatment for this patient's condition after the patient is initially evaluated?

A. Order regular insulin 0.1 U/kg as IV bolus

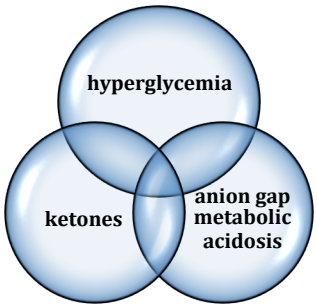
B. Start regular IV insulin 0.1 U/kg/hour as continuous IV infusion

C. Order 4 mg IV Zofran and wait for lab results before proceeding

D. Start IV fluids 1.0 L of 0.9% NaCl/hour

E. Order 20 mEq potassium/hour

The correct answer is D. Start IV fluids 1.0 L of 0.9% NaCl/hour. This patient's presentation is consistent with diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic nonketotic state (HHNS). Both have similar signs and symptoms as hyperglycemia. DKA is the triad of hyperglycemia, anion gap metabolic acidosis and ketonemia, whereas patients with HHNS have little or no ketoacid accumulation and serum glucose frequently exceeds 1000 mg/dL. In both DKA and HHNS, IV fluid hydration is essential to treatment and is the initial treatment. Answers A, B, and E and incorrect because a serum glucose, serum potassium, serum bicarbonate, serum sodium and arterial blood gas are needed before initiating any further treatment. Potassium may need to be replaced prior to initiation of insulin and IV fluids may be changed depending on corrected sodium level. Answer C is incorrect because IV hydration may be initiated immediately prior to awaiting lab results.



Diabetic Ketoacidosis is defined as a triad of

- **hyperglycemia**
- **anion gap metabolic acidosis**
- **ketonemia**

Precipitating Factors:

- Insulin deficiency
  - New onset diabetes
  - Insulin noncompliance
- Physiologic stress
  - Trauma
  - Pancreatitis
  - Infection (sepsis, pneumonia, UTI)
  - Myocardial Infarction
  - Cerebrovascular accident
- Drugs
  - Second generation atypical antipsychotic agents: clozapine (Clozaril), olanzapine (Zyprexa)
  - Corticosteroids
  - Cocaine

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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Clinical Presentation:

Diabetic ketoacidosis usually evolves rapidly over a 24-hour period. Patients present with symptoms of hyperglycemia such as polyuria, polydipsia, and weight loss. As the degree or duration of hyperglycemia progresses, neurological symptoms, including lethargy, focal signs, obtundation, and coma, can develop. Neurological deterioration primarily occurs in patients with plasma osmolality above 320 to 330 mosmol/kg. Patients may also present with abdominal pain, nausea, and vomiting. Abdominal pain is associated with the severity of metabolic acidosis and occurs more frequently in patients with a serum bicarbonate ≤5 mEq/L. Signs of volume depletion such as decreased skin turgor, dry axillae and oral mucosa, low jugular venous pressure, tachycardia, and hypotension may be apparent. Patients may have a fruity odor due to exhaled acetone, which is similar to the scent of nail polish remover, and deep respirations reflecting the hyperventilation called Kussmaul respirations to compensate for the metabolic acidosis.

DKA usually evolves rapidly over 24 hours and is more common in type 1 diabetes, whereas HHNS is usually insidious over several days and more common in patients older than 65.

Diagnostic Evaluation: Diabetic ketoacidosis is a medical emergency and requires prompt recognition and management.

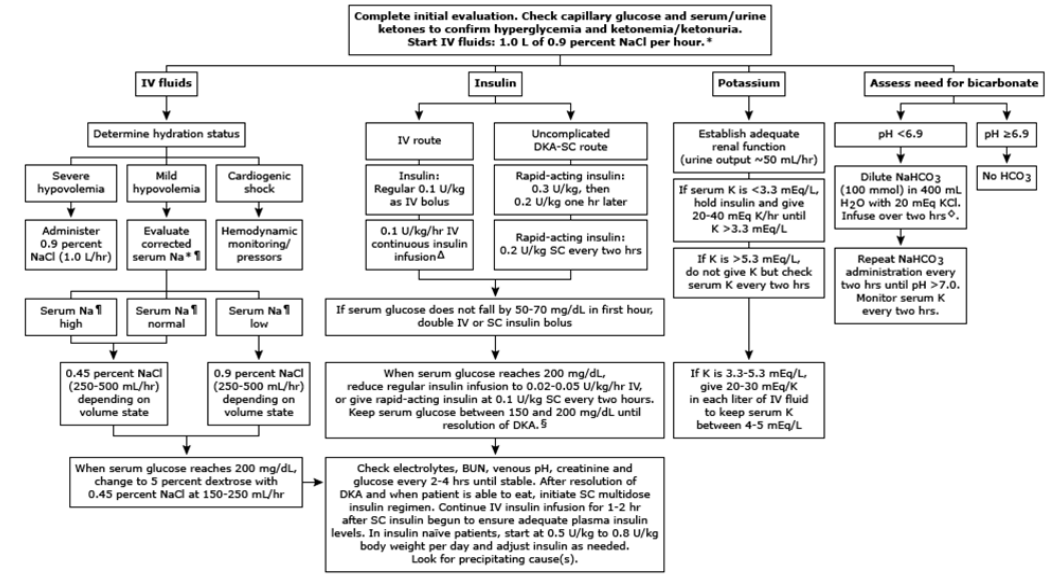
Initial Evaluation:

- airway, breathing, circulation
- mental status
- possible precipitating events (myocardial infarction, source of infection)
- volume status

Initial Labs:

- serum glucose
- serum electrolytes with anion gap
- BUN
- plasma creatinine
- CBC with differential
- urinalysis and urine ketones by dipstick
- plasma osmolality
- serum ketones
- arterial blood gas
- electrocardiogram

Protocol for the management of adult patients with DKA



DKA diagnostic criteria: Serum glucose >250 mg/dL, arterial pH <7.3, serum bicarbonate <18 mEq/L, and at least moderate ketonuria or ketonemia. Normal laboratory values vary; check local lab normal ranges for all electrolytes.

BUN: blood urea nitrogen; DKA: diabetic ketoacidosis; HCO_3: bicarbonate; IV: intravenous; K: potassium; Na: sodium; NaCl: sodium chloride; $NaHCO_3$: sodium bicarbonate; SC: subcutaneous.

* After history and physical exam, obtain capillary glucose and serum or urine ketones. Begin one liter of 0.9 percent NaCl over one hour and draw arterial blood gases, complete blood count with differential, urinalysis, serum glucose, BUN, electrolytes, chemistry profile, and creatinine levels STAT. Obtain electrocardiogram, chest radiograph, and specimens for bacterial cultures, as needed.

¶ Serum Na^+ should be corrected for hyperglycemia (for each 100 mg/dL glucose >100 mg/dL, add 2.0 mEq to sodium value for corrected serum sodium value).

Δ An alternative IV insulin regimen is to give a continuous intravenous infusion of regular insulin at 0.14 units/kg/hour; at this dose, an initial intravenous bolus is not necessary.

◇ 100 mmol sodium bicarbonate = 100 mEq sodium bicarbonate.

§ Please refer to the topic on DKA for the definition of DKA resolution.

(via UpToDate "Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Treatment)

For a list of educational lectures, grand rounds, workshops, and didactics please visit ***BrowardER.com*** and **click** on the ***"Conference"*** **link**.

All are welcome to attend!

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Management: Management is aimed at correction of fluid and electrolyte abnormalities, specifically the hyperosmolality, hypovolemia and metabolic acidosis. Insulin administration is also key.

General Overall Approach to DKA: (specifics in American Diabetes Association algorithm)

1) Infusion of isotonic saline (0.9% sodium chloride) 1.0 L/h
- May need to adjust to 0.45% NS dependant on corrected Na
- When serum glucose reaches 200 mg/dl, change to 5% dextrose with 0.45% NS at 150-250 ml/hr

2) Correct any potassium deficit prior to administration of insulin (may influence choice of fluid replacement)

Typical laboratory characteristics of DKA and HHS*

	DKA			HHS
	Mild	Moderate	Severe	
Plasma glucose (mg/dL)	>250	>250	>250	>600
Plasma glucose (mmol/L)	>13.9	>13.9	>13.9	>33.3
Arterial pH	7.25 to 7.30	7.00 to 7.24	<7.00	>7.30
Serum bicarbonate (mEq/L)	15 to 18	10 to <15	<10	>18
Urine ketones¶	Positive	Positive	Positive	Small
Serum ketones - Nitroprusside reaction	Positive	Positive	Positive	≤ Small
Serum ketones - Enzymatic assay of beta hydroxybutyrate (normal range <0.6 mmol/L)Δ	3 to 4 mmol/L	4 to 8 mmol/L	>8 mmol/L	<0.6 mmol/L
Effective serum osmolality (mOsm/kg)◦	Variable	Variable	Variable	>320
Anion gap§	>10	>12	>12	Variable
Alteration in sensoria or mental obtundation	Alert	Alert/drowsy	Stupor/coma	Stupor/coma

DKA: diabetic ketoacidosis; HHS: hyperosmolar hyperglycemic state.
* There may be considerable diagnostic overlap between DKA and HHS.
¶ Nitroprusside reaction method.
Δ NOTE: Many assays for beta hydroxybutyrate can only report markedly elevated values as >6.0 mmol/L.
◦ Calculation: 2[measured Na (mEq/L)] + glucose (mg/dL)/18.
§ Calculation: (Na+) – (Cl– + HCO3–) (mEq/L). See text for details.

3) Low-dose IV insulin 0.1 U/kg/hr continuous infusion to patients with moderate-severe DKA with serum potassium ≥ 3.3 mEq/L
- Delay until potassium replacement has begun and concentration has increased because insulin will drive potassium into the cells and hypokalemia can potentiate cardiac arrhythmias
- Decrease to 0.02-0.05 U/kg/hr when serum glucose reaches 200 mg/dl
- Switch to subcutaneous insulin regimen after closure of anion gap and when patient is able to eat

Take Home Points

- There are many precipitating factors of diabetic ketoacidosis, including insulin deficiency from new onset diabetes or insulin noncompliance, physiologic stress such as trauma, pancreatitis, infection, MI, and CVA, and drugs such as second generation atypical antipsychotics, corticosteroids and cocaine.
- Diabetic ketoacidosis (DKA) is defined as the triad of hyperglycemia, anion gap metabolic acidosis and ketonemia.
- Patients may present with polyuria, polydipsia, weight loss, abdominal pain, nausea, vomiting, signs of volume depletion, fruity breath odor, deep respirations (Kussmaul respirations) and in severe cases neurological symptoms.
- Hyperosmolar hyperglemic nonketotic state (HHNS) has similar signs and symptoms as hyperglycemia, but HHNS has little or no ketoacid accumulation and serum glucose frequently exceeds 1000 mg/dL
- Management is aimed at correction of fluid and electrolyte abnormalities, specifically the hyperosmolality, hypovolemia, and metabolic acidosis (in DKA).
- In both DKA and HHNS, IV fluid hydration is essential to treatment and is the initial treatment.
- Serum glucose, serum potassium, serum bicarbonate, serum sodium and arterial blood gas are needed before initiating any further treatment. Potassium may need to be replaced prior to initiation of insulin and IV fluids may need to be changed depending on corrected sodium level.

ABOUT THE AUTHOR

This month's case was written by Taylor Schaubschlager. Taylor is a 4th year medical student from NSU-COM. She did her emergency medicine rotation at BHMC in August 2016. Taylor plans on pursuing a career in Internal Medicine after graduation.

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