Diabetic Ketoacidosis (DKA)

DKA is a life-threatening complication of diabetes characterized by hyperglycemia, ketosis, and acidosis, requiring urgent management.

Definition and Epidemiology

- **Definition:** DKA is an acute metabolic complication of diabetes with hyperglycemia (glucose >250 mg/dL), ketosis (↑ ketones), and acidosis (pH <7.3, HCO3- <18 mEq/L).
- **Prevalence:** ~30% of type 1 diabetes patients experience DKA; 5-10% in type 2; 50-100 cases/10,000 diabetic patients annually.
- **Risk Factors:** Type 1 diabetes, infection, insulin omission, new-onset diabetes, substance use (alcohol, cocaine).

Pathophysiology

- **Mechanisms:** Insulin deficiency + counterregulatory hormone excess (glucagon, cortisol) → ↑ Gluconeogenesis, ↓ glucose uptake → Hyperglycemia.
- **Ketogenesis:** ↑ Lipolysis → Free fatty acids → Ketone production (beta-hydroxybutyrate, acetoacetate) → Acidosis.
- **Potassium Shift:** Acidosis (↓ pH) causes K+ to shift out of cells (H+/K+ exchange to buffer H+) → Normal or ↑ serum K+ despite total body depletion (urinary losses).
- **Effects:** Dehydration (osmotic diuresis), electrolyte imbalances (↓ K+, ↓ Na+), organ dysfunction.
- **Key Pathway:** Insulin deficiency → ↑ Glucose → Osmotic diuresis → Dehydration + ketosis → Acidosis.

Causes and Precipitants

Category	Causes/Precipitants	Notes
Common	- Infection: UTI, pneumonia (30-50% of cases). - Insulin omission: Non-adherence, pump failure. - New-onset diabetes: Undiagnosed type 1. - Stress: MI, trauma, surgery.	Infection: Most common trigger; check UA, CXR.

Category	Causes/Precipitants	Notes
Other	- Substance use: Alcohol (poor intake), cocaine (↑ stress hormones). - Medications: Steroids, antipsychotics.	Cocaine: Sympathetic surge → ↑ Counterregulatory hormones.
Rare	- SGLT2 inhibitors (euglycemic DKA). - Pregnancy (↑ insulin resistance). - Thyrotoxicosis (↑ metabolic demand).	SGLT2i: Glucose <200 mg/dL but ketosis/ acidosis.

DKA Diagnostic Criteria (ADA)

Parameter	Threshold	Notes
Glucose	>250 mg/dL	May be lower in euglycemic DKA (SGLT2i).
pH	<7.3	Severe if <7.0; Kussmaul respirations.
HCO3-	<18 mEq/L	Reflects metabolic acidosis.
Ketones	Positive (serum/urine)	Beta-hydroxybutyrate >3 mmol/L (most sensitive).
Anion Gap	>12 mEq/L	AG = Na+ - (Cl- + HCO3-).

Clinical Presentation

- **Symptoms:**
  - Polyuria, polydipsia, dehydration (early).
  - Nausea, vomiting, abdominal pain (ketosis).
  - oConfusion, lethargy (acidosis, dehydration).
  - Kussmaul respirations (rapid, deep; pH <7.2).
- **Exam:**
  - **Dehydration:** Dry mucous membranes, tachycardia, hypotension.
  - **Fruity breath (ketones:** acetoacetate).
  - **AMS:** Confusion (pH <7.1), coma (pH <7.0).
  - **Substance Use:** Alcohol (poor intake → ↓ insulin), cocaine (↑ stress → DKA).
- **Red Flags:** pH <7.0, K+ <3.0 or >5.5 mEq/L, altered mental status.

Diagnostic Workup

- **Labs:**
  - **Glucose:** >250 mg/dL (or euglycemic DKA).
  - **ABG/VBG:** pH <7.3, HCO3- <18 mEq/L.
  - **Ketones:** Serum beta-hydroxybutyrate (>3 mmol/L), urine ketones.
  - **Electrolytes:**
    - Anion gap >12 (Na+ - (Cl- + HCO3-)).

        - **K+:** Normal or ↑ on presentation (acidosis shift); risk of ↓ K+ with treatment.
        - **Na+:** Corrected Na+ for hyperglycemia (↑ 1.6 mEq/L per 100 mg/dL glucose >100).
    - **Infection:** CBC (WBC ↑), UA, CXR, blood cultures.
    - **Other:** BUN/Cr (dehydration), lactate (sepsis), troponin (MI).
- **Imaging:** CXR (pneumonia), CT head (if AMS + focal deficits).
- **Key Tip:** Beta-hydroxybutyrate is most sensitive for ketosis; urine ketones lag behind resolution.

Management Flowsheet: DKA

- **Step 1:** Fluids: NS 1-1.5 L bolus (20 mL/kg), then 250-500 mL/h; switch to 0.45% NS if corrected Na+ normal.
- **Step 2:** Insulin: IV regular insulin 0.1 U/kg bolus, then 0.1 U/kg/h infusion; hold if K+ <3.3 mEq/L.
- **Step 3:** Electrolytes:
    - **K+:** If <5.3 mEq/L, add 20-40 mEq/L to fluids (target 4-5 mEq/L); monitor closely as acidosis corrects (K+ shifts into cells).
    - **Bicarb:** Only if pH <6.9 (100 mEq NaHCO3 IV over 2h).
- **Step 4:** Monitor:
    - Glucose q1h (target 150-200 mg/dL; ↓ 50-75 mg/dL/h).
    - VBG/electrolytes q2-4h (close gap, HCO3- >15, watch for ↓ K+).
- **Step 5:** Transition: Gap closed → Dextrose 5% + insulin drip; overlap SC insulin 1-2h before stopping drip.
- **Step 6:** Treat precipitant: Antibiotics (infection), education (insulin adherence).

Treatment

- **General Principles:** Correct hyperglycemia, acidosis, dehydration, and precipitant; monitor closely.
- **Fluids:**
    - NS 1-1.5 L bolus, then 250-500 mL/h (total deficit 6-10 L).
    - Switch to 0.45% NS if corrected Na+ normal; add dextrose 5% when glucose <200 mg/dL.
- **Insulin:**
    - **IV regular insulin:** 0.1 U/kg bolus, then 0.1 U/kg/h.
    - **Goal:** ↓ Glucose 50-75 mg/dL/h; continue until gap closes (AG <12).
    - Hold if K+ <3.3 mEq/L (risk of arrhythmia).

- **Electrolytes:**
  - **K+:** Add 20-40 mEq/L to fluids if K+ <5.3 mEq/L; recheck q2h. Acidosis correction causes K+ to shift back into cells, risking severe hypokalemia (arrhythmias, weakness).
  - **Bicarb:** Only if pH <6.9 (100 mEq IV over 2h).
  - **Phos:** If <1 mg/dL, replace (potassium phosphate 20-30 mmol IV).
- **Monitor:**
  - Glucose q1h, VBG/electrolytes q2-4h, neuro checks (cerebral edema risk in kids).
  - Watch K+ closely as pH rises (risk of hypokalemia).
- **Treat Precipitant:**
  - **Infection:** Antibiotics (e.g., ceftriaxone 1 g IV q24h for UTI).
  - **Substance Use:** Alcohol (education, monitor withdrawal), cocaine (stop use).
- **Key Tip:** Do not stop insulin drip until anion gap closes, even if glucose <200 mg/dL (add dextrose).

Examples

- **1. Case 1:** DKA from Infection (UTI)
- **Presentation:** 30 y/o F, type 1 DM, fever, nausea, glucose 600 mg/dL, pH 7.1, HCO3- 10 mEq/L, UA: +LE, +nitrites.
- **Interpretation:** DKA (glucose >250, pH <7.3, HCO3- <18), triggered by UTI.
- **Management:** NS 1 L bolus, insulin 0.1 U/kg/h, K+ 20 mEq/L in fluids, ceftriaxone 1 g IV, monitor q1-2h (K+ drops to 3.0 mEq/L as pH corrects → Add 40 mEq/L).

- **Case 2:** DKA from Insulin Omission (Non-Adherence)
- **Presentation:** 25 y/o M, type 1 DM, missed insulin doses, vomiting, glucose 450 mg/dL, pH 7.2, beta-hydroxybutyrate 5 mmol/L, K+ 4.0 mEq/L.
- **Interpretation:** DKA (hyperglycemia, ketosis, acidosis), due to insulin omission.
- **Management:** NS 1.5 L bolus, insulin 0.1 U/kg/h, K+ 20 mEq/L in fluids, education on adherence, monitor gap (K+ falls to 2.8 mEq/L → Increase replacement).

Complications

- **Acute:** Hypokalemia (arrhythmias), cerebral edema (rare, kids), hypoglycemia (overcorrection).

- **Long-Term:** Recurrent DKA (non-adherence).

Prognosis

- **Mortality:** <1% with treatment; 5-10% if delayed.
- **Recovery:** Gap closes in 12-24h with proper management.

Key Pearls

- DKA = Glucose >250, pH <7.3, HCO3- <18, +ketones, AG >12.
- Fluids first, then insulin; hold insulin if K+ <3.3 mEq/L.
- Acidosis causes K+ to shift out of cells; correction → Severe hypokalemia → Monitor q2h.
- **Treat precipitant:** Infection (most common), non-adherence.
- SGLT2i → Euglycemic DKA; check ketones even if glucose <200.

References

- **UpToDate:** “Diabetic Ketoacidosis” (2025).
- **ADA Guidelines:** DKA Management (2023).
- **NEJM:** “DKA and HHS” (Kitabchi, 2009).