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LEARNING OBJECTIVE

TOPIC 17: Diabetic Ketoacidosis - Recognize and manage

Recognize and plan the management of a child with diabetic ketoacidosis

JUMP TO SECTION

Background

Diabetic ketoacidosis (DKA) is the **leading cause of morbidity and mortality** in children with diabetes mellitus. DKA occurs predominantly in **type 1 diabetes** but can also occur in **type 2 diabetes** during intercurrent illness or physiologic stress. In children with new-onset type 1 diabetes in the United States, DKA is present at diagnosis in **at least 30 percent**. **Cerebral injury accounts for 60 to 90 percent of DKA-related deaths**, making prompt recognition and appropriate management critical for general pediatricians.

Differential Diagnosis

When evaluating a child with suspected DKA, **distinguish DKA from hyperglycemic hyperosmolar state (HHS)** and mixed presentations.

HHS is distinguished by marked hyperglycemia (**>600 mg/dL**), **minimal acidosis (pH >7.25)**, and **absent to mild ketosis**, with effective osmolality >320 mOsm/kg. It occurs most often in African American adolescents with type 2 diabetes, typically requires greater fluid resuscitation, and has a different insulin approach than DKA.

Consider **lactic acidosis** if there is a **large anion gap but ketosis is not significant**; evaluate for shock/sepsis or HHS.

Clinical Features / Presentation

DKA most commonly presents in children with type 1 diabetes, either at **new diagnosis** or during **lapses in insulin administration**, but can also occur in type 2 diabetes during acute illness.

- **Early symptoms:** polyuria, polydipsia, weight loss, nocturia, enuresis
- **Progression:** anorexia, nausea, vomiting, abdominal pain (**can mimic acute abdomen**)
- **Respiratory: Kussmaul respirations** (deep, rapid breathing), fruity breath odor
- **Dehydration:** tachycardia, poor perfusion, decreased skin turgor (**may underestimate volume depletion**)
- **Neurologic:** drowsiness, lethargy, obtundation, coma; **severity parallels acidosis**
- **Infants:** irritability, decreased activity, **severe Candida diaper rash** rather than obvious polyuria/polydipsia

Evaluation

History

- Duration/severity of polyuria, polydipsia, weight loss
- Nausea, vomiting, abdominal pain
- Neurologic symptoms (headache, confusion, drowsiness)
- **Precipitating factors:** missed insulin, intercurrent illness, pump malfunction
- Family history of diabetes

Physical Examination

- Vital signs including respiratory pattern and blood pressure
- Perfusion and dehydration signs
- Neurologic status (GCS); **hypertension may indicate more severe DKA**

Diagnostic Tests

- **Point-of-care:** blood glucose, blood BOHB (or urine ketones)
- **Laboratory:** glucose, electrolytes with bicarbonate, BUN, creatinine, venous pH/pCO2, calcium, phosphorus, magnesium

Diagnosis

DKA is diagnosed when **all three criteria** are met:

- **Hyperglycemia:** blood glucose **>200 mg/dL** (11 mmol/L)
- **Metabolic acidosis:** venous **pH <7.3** or serum **bicarbonate <18 mEq/L**
- **Ketosis:** blood **BOHB ≥3 mmol/L** (31 mg/dL) or moderate/large urine ketones

DKA Severity

SEVERITY	PH	BICARBONATE
Mild	7.2 to <7.3	10 to <18 mEq/L
Moderate	7.1 to <7.2	5 to 9 mEq/L
Severe	**<7.1**	**<5 mEq/L**

Key Calculations

- **Anion gap** = sodium - (chloride + bicarbonate); normal is **12±2 mEq/L**
- **Corrected sodium** = measured sodium + 1.6 x [(glucose - 100)/100] mg/dL

Management and Treatment

Disposition

- **Pediatric ICU: altered consciousness**, **age <5 years**, **severe acidosis (pH <7.1)**, severe hypocapnia, or high BUN
- **Inpatient unit:** mild to moderate uncomplicated DKA if close monitoring and IV insulin available

DKA Management Pathway (ED to Resolution)

1. **Rapid assessment** including weight, vital signs, GCS, point-of-care glucose/ketones. Estimate dehydration as **8%** if pH <7.1, BUN >20 mg/dL, or new-onset diabetes; otherwise assume **6%**.
2. **Initial volume expansion** with **10 to 20 mL/kg isotonic saline (0.9% NaCl)** over 20 to 30 minutes (20 mL/kg for moderate-severe, 10 mL/kg for mild). Repeat boluses as needed for hemodynamic instability.
3. Approximately one hour after starting IV fluids, begin **continuous IV insulin at 0.1 units/kg/hour** (no bolus). Address potassium based on initial level:
   - **Hypokalemic:** start replacement immediately; **delay insulin** until potassium approaches normal
   - **Normokalemic:** add **40 mEq/L potassium** to IV fluids (not in initial bolus) when starting insulin because total body potassium is depleted due to significant losses of total body K from osmotic diuresis and extracellular shifts
   - **Hyperkalemic:** defer adding potassium until level normalizes and urine output documented
4. **Replace remaining deficit** over 24 to 48 hours using 0.45 to 0.9% NaCl. **Add dextrose when glucose falls to 250 to 300 mg/dL**. If glucose falls **below 150 mg/dL** before ketoacidosis resolves, increase dextrose to 10 or 12.5%.
5. **Discontinue insulin infusion** when: anion gap normalizes (12±2 mEq/L) or **BOHB ≤1 mmol/L**, venous **pH >7.3** or bicarbonate >18 mEq/L, glucose <200 mg/dL, and patient tolerates oral intake. **Transition to subcutaneous insulin 15 to 30 minutes before stopping IV insulin.** Selected mild cases in older children with established diabetes may use subcutaneous insulin with close monitoring.

Bicarbonate is generally **not recommended**; consider only for **pH ≤6.9** with impaired cardiac contractility or life-threatening hyperkalemia.

Monitoring During DKA Treatment

PARAMETER	FREQUENCY
Vital signs	Hourly
Fluid intake/output	Hourly
Neurologic status (GCS)	At least hourly
Blood glucose	Hourly
Blood BOHB	Every 2-4 hours
Electrolytes, BUN, Cr, VBG	Every 2-4 hours
Ca, Mg, phosphorus	Every 4-6 hours

Complications

Cerebral injury occurs in **0.3 to 0.9 percent** of children with DKA and is the **leading cause of DKA-related mortality**. **Treat based on clinical signs without waiting for neuroimaging.**

Cerebral Injury: Recognition and Immediate Response

RISK FACTORS	SUSPICIOUS FINDINGS	WHEN TO TREAT	IMMEDIATE ACTIONS
Severe acidosis at presentation; elevated BUN; severe hypocapnia; **age <5 years** and/or new-onset diabetes	**Minor:** headache (new/worsening); vomiting during treatment; irritability/lethargy; diastolic BP >90 mmHg **Major:** abnormal/deteriorating mental status; age-inappropriate incontinence; HR slowing >20 bpm **Diagnostic:** abnormal motor/verbal response to pain; posturing; abnormal pupils/CN palsy; abnormal respiratory pattern	**1 diagnostic criterion**, OR **2 major criteria**, OR 1 major + 2 minor, OR **1 major + 1 minor if <5 years**	**Mannitol 0.5-1 g/kg IV** over 15 min (may repeat in 30 min), OR **3% saline 2.5-5 mL/kg** over 10-15 min Elevate HOB 30°; **avoid hypotension**; consult neurosurgery; **do not delay for imaging**

Other complications include venous thrombosis (**avoid central venous catheters** when possible), acute kidney injury (usually resolves with treatment), and mild pancreatic enzyme elevations (usually not clinically significant).

Prevention / Counseling / Guidance

In children with established diabetes, **insulin omission and mismanagement account for most DKA episodes**. Prevention strategies include reinforcing parental involvement, sick-day management education, insulin pump troubleshooting, and frequent contact with the diabetes care team. During intercurrent illnesses, families should monitor glucose and ketones, maintain hydration, and adjust insulin as needed. **Never stop insulin during illness.** Consider psychosocial concerns in patients with poorly controlled diabetes/recurrent DKA (e.g., **adolescents with eating disorders may reduce/stop insulin to promote weight loss**).

JUST IN CASE (CURATED EXAM-DAY SUPPORT)

- Diabetic Ketoacidosis in Children: Clinical Features and Diagnosis - UpToDate
- Diabetic Ketoacidosis in Children: Treatment and Complications - UpToDate
- Diabetic Ketoacidosis - Pediatrics in Review (AAP)

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