

**Comer Emergency Department (ED) Clinical Guidelines:  
Diabetic Ketoacidosis (DKA) – Moderately Severe to Severe**

Population: Patients with DKA

- Patients with either new-onset or known diabetes
- Classic triad:
  - Hyperglycemia (Fingerstick glucose > 250 mg/dL)
  - Ketosis (urinary ketones  $\geq 2^+$ )
  - Acidemia (Serum  $\text{HCO}_3^- < 15$  mEq/L and/or blood pH < 7.30)

History and Physical Examination:

- Obtain height and weight at triage
- Rapid cardiopulmonary assessment
- Evaluation of work of breathing and pulse oximetry on room air
- Evaluation of perfusion
- Neurological examination for evidence of cerebral edema (slowing heart rate, hypertension, increasing somnolence or irritability)
- General physical examination for evidence of focal infection, hydration status
- Check for yeast infection

Diagnostic Evaluation:

- START DIABETIC FLOWSHEET (see attachment)
- Fingerstick blood glucose
- Urinalysis (dip for ketones in ER, send U/A to lab)
- BMP (sodium, potassium, chloride,  $\text{HCO}_3^-$ , BUN, creatinine, glucose)
- Calcium, magnesium, phosphate
- Serum ketones (qualitative)
- Blood pH analysis (ABG, VBG, CBG)
- CBC with differential
- Hemoglobin A<sub>1C</sub>
- C-peptide if newly diagnosed; plus insulin level and autoantibody levels (islet-cell antibodies (ICA), insulin autoantibodies (IAA), antibodies to glutamic acid decarboxylase (GAD), and antibodies to tyrosine phosphatase-like proteins such as insulinoma associated protein (IA-2, ICA512) and antibody to cation efflux zinc transporter (ZnT8)
- Urine culture if indicated (symptomatic, history of UTI; males under 6mo, or girls under 2yrs)
- CXR if indicated (respiratory distress, cough, fever)
- CT scan of brain if there is evidence of altered mental status, focal neurologic deficit, or concern for cerebral edema
- Attempts should be made to access the medical records. The electronic medical record often contains detailed clinic notes and discharge summaries with a wealth of information (i.e., lab summaries, discharge medications, dosages, prior complications).
- Notify Peds Endo – Pgr 188-9296; to discuss management plan

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Severity Stratification:

- Hyperglycemia and Ketosis without Acidemia (pH > 7.3, HCO<sub>3</sub> > 18, minimal vomiting)
  - Often managed as an outpatient, unless newly diagnosed
  - Oral fluids and supplemental Humalog insulin sq: Please call Peds Endo for specifics on dosing insulin.
  - In known diabetics:
    - Sm ketones – give sq Humalog; please contact peds endo for specific dosing instructions
    - Mod-Lg ketones – give sq Humalog; please contact peds endo for specific dosing instructions
- Moderate DKA (pH = 7.2-7.3, HCO<sub>3</sub> = 10-20, persistent vomiting)
  - Often managed as an outpatient, unless newly diagnosed
  - IVF's (see beyond for quantity) and supplemental Humalog insulin sq (0.1-0.2 u/kg q 2-3hrs);
    - In known diabetics:
      - Sm ketones – give 10% TDD as sq Humalog
      - Mod-Lg ketones – give 15-20% TDD as sq Humalog
      - If not correcting, consider IV insulin, then admit to PICU
- Severe DKA (pH < 7.2, HCO<sub>3</sub> < 10)
  - IVF hydration
  - IV insulin
  - Careful monitoring and admission to the PICU most often recommended

Therapeutic Management:

- IV Fluids
  - All patients with DKA require supplemental fluids
  - Patients with hyperglycemia and ketosis without acidemia may be hydrated with PO fluids.
  - Patients with moderate to severe DKA and/or persistent vomiting require IV fluids.
  - For patients who are not in shock, and in whom there is evidence of adequate perfusion, a fluid bolus is usually not necessary. Instead, IV rehydration may be initiated with **NS at 1.5-2.0 x maintenance (1800-2400cc/m<sup>2</sup>/d)**, pending lab results and calculation of actual fluid deficits.
  - Another reasonable alternative is to use **NS at 4000cc/ m<sup>2</sup>/d (less any fluid boluses given) evenly distributed over 36 - 48 hrs** (method preferred by peds endo).

$$BSA = \sqrt{(ht)(wt)/3600}$$

- For patients in shock, or those with evidence of poor perfusion\*, a fluid bolus of 10-20cc/kg NS over 1/2-1 hr is recommended.
  - \*Indicators of poor perfusion may include marked tachycardia, capillary refill  $\geq$  2 seconds, signs of orthostasis, or altered mental status.

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- Remember, however, that altered mental status itself may be a sign of cerebral edema or a hyperosmolar state, and a rapid fluid bolus may worsen the condition.
- Many patients with Severe DKA will have some evidence of poor perfusion and therefore require an initial fluid bolus of NS.

<u>Sodium Correction:</u>	$\text{Corrected Na}^+ = (\text{Measured Na}^+) + \frac{(1.6)(\text{Glucose} - 140)}{100}$
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<u>Fluid Deficit:</u>	$\text{Water deficit (in liters)} = (0.6)(\text{body wt in kg})\{[(\text{Measured Na})/(140)] - 1\}$
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- **Avoid overaggressive fluid administration** and instead opt for frequent re-evaluations, and repeat boluses as necessary. You can always give more, you can never give less.
  - Following initial fluid resuscitation, the remaining calculated deficits are replaced evenly over 24--48hrs using an isotonic fluid (NS with 30-40 mEq K<sup>+</sup>/L- see potassium and phosphate sections below). Please see Peds ER DKA order set for fluid management if patient remains in ED for an extended time period.
  - In cases of severe hyperosmolality, or marked hyperglycemia with relative hyponatremia (Na<sup>+</sup> above 145 mEq/L after correction for hyperglycemia using 1.6 mEq/L for every 100 mg/dl blood glucose above 100 mg/dl), calculate fluid rate to replace deficit evenly over 48-72 hours. Target to correct Na no faster than 8-10 mEq/day (max 12 mEq/day)
- **Insulin**
  - Patients with Mild to Moderate DKA may receive sq Humalog insulin as described above.
  - Patients with Severe DKA require **continuous IV Regular Insulin at 0.1 U/kg/hr**. A bolus of insulin is usually not necessary unless there is going to be a long delay in receiving the insulin drip from the pharmacy. In such cases, a bolus of regular insulin 0.1 U/kg (10 U max) may be given to approximate the first hour of continuous infusion.
  - After the insulin drip starts, do not drop the glucose by more than 100/hr or the corrected Na by more than 1-2 mEq/4hrs.
  - The purpose of the insulin is to correct the ketoacidosis. Do not reduce or discontinue the insulin drip based solely upon the blood glucose.
  - The insulin drip should be continued until the pH > 7.30 and/or the HCO<sub>3</sub> > 18, and the serum ketones have cleared.
  - The lowest effective rate of insulin is 0.05 U/kg/hr. Additional glucose may be necessary in the maintenance IV fluids in order to maintain a blood glucose of at least 150 mg/dL.
  - When the patient's status is appropriate for discontinuation of IV insulin, sq Lantus and short-acting insulin may be given 30 minutes prior to stopping the IV insulin drip. Please call peds endo to discuss specifics about dosing.
  - Take the dextrose out of the IV when you stop the insulin drip.
- **Dextrose**
  - When blood glucose ≤ 250 mg/dl, change fluid to D5NS

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- Maintain blood glucose between 150-250 mg/dl. If necessary, higher concentrations of glucose may be obtained with supplemental PO intake or 7.5-12% dextrose containing IV infusions.
  - Take the dextrose out of the IV when you stop the insulin drip.
  - Please refer Peds ER DKA order set for fluid and dextrose management if patient remains in ED for an extended time period.
- **Bicarbonate**
    - The use of bicarbonate is controversial and is rarely necessary unless shock, hypoxia, renal failure, or sepsis are present.
    - Consider use of HCO<sub>3</sub> only if patient is suffering from shock, renal failure, cardiac insufficiency, or if pH < 7.0 (only after discussion with the attending).
    - Do not administer HCO<sub>3</sub> as a bolus.
    - Instead, add 44mEq (1 amp)/liter of IV fluid. It should be added to a rehydration fluid with less Na<sup>+</sup> (i.e., 1/2 NS) so that the sodium concentration does not exceed 75 mEq/L.
    - Correct to 1/2 of normal.
  - **Potassium**
    - DKA is associated with a total body depletion of potassium, regardless of the initial serum K<sup>+</sup>.
    - Correction of acidosis and supplemental insulin both cause intracellular shift of K<sup>+</sup> into cells resulting in a fall in serum potassium concentrations.
    - If initial serum K<sup>+</sup> < 5.5 and the patient is making urine (no renal failure) add 30-40 mEq of K<sup>+</sup>/L IV fluid. Give 1/2 as K-phos and the other 1/2 as KCl.
    - Potassium salts can be added as phosphate, chloride, and/or acetate. It is recommended that neither the K-phos nor the K-acetate should constitute more than 15-20 mEq/L each.
  - **Phosphate**
    - Phosphate depletion occurs because of acidosis and urinary losses, and may lower ATP and 2,3-DPG levels.
    - Giving too much phosphate (i.e., more than 20 mEq/L) may cause hypocalcemia and tetany.
    - Check repeat phosphate levels if giving supplemental phosphate (K-phos)
    - 1 ml of potassium phosphate has 3 mMol phosphate/ml and 4.4 mEq potassium/ml

20 mEq K Phosphate = 13.6 mMol K Phosphate
15 mEq K Phosphate = 10.2 mMol K Phosphate
10 mEq K Phosphate = 6.8 mMol K Phosphate

mMol based on phosphorous content; mEq based on potassium content  
pediatric dose not to exceed > 100 mMol phosphorous per day

**Ongoing Monitoring of Patients with Severe DKA**

- Bedside glucose q 1hr

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- Serum electrolytes (BMP plus calcium and phosphate), pH, and serum ketones every 3-4 hrs depending on severity until acidosis has cleared.
- Dip all urine for ketones
- Vital signs and neurological checks every 1-2 hrs for at least 24hrs to watch for possible cerebral edema.
- Accurate Intake and Output records.
- Flow sheets are recommended.
- Frequent physician re-evaluations timed and documented in the medical record.

Major Complication: Cerebral Edema in DKA

- Symptomatic cerebral edema occurs in approximately 1% of patients with DKA. Asymptomatic cerebral swelling, however, is believed to occur more frequently.
- It occurs most frequently 8-24hrs after the initiation of therapy.
- The pathophysiology of cerebral edema remains unclear.
- Although historically there has been a putative association with the administration of bicarbonate and/or excessive hypotonic fluid resuscitation, it should be remembered that cerebral edema has been documented in patients prior to the initiation of any therapy.
- In addition, although most treatment guidelines in the recent years have decreased the administration of bicarbonate and overzealous fluids, there has been no significant change in the overall rate of cerebral edema.
- Risk factors that have been identified retrospectively for the development of DKA include:
  - Younger age, newer-onset diabetes
  - Higher glucose, BUN, creatinine
  - Lower pH, HCO<sub>3</sub>, pCO<sub>2</sub>
  - Smaller increases in serum sodium concentration during therapy
- Hyperosmolar therapy with mannitol should be immediately available for use in the event that signs of cerebral edema occur (lowering heart rate, elevating blood pressure, excessive irritability, confusion or somnolence, visual complaints). With progression of symptoms, consider intubation, mannitol, and controlled ventilation (aim to keep pCO<sub>2</sub> 35-40).
  - Notify the PICU of all patients receiving hyperosmolar therapy in the ED.

Disposition assignment:

- Admission recommended for:
  - Severe DKA (pH < 7.2, HCO<sub>3</sub> < 10) ⇒ Consult PICU Service
  - Ill-appearing children
  - Children under 5 yrs of age
  - Prior history of cerebral edema
  - Altered mental status or acute neurologic deficit
  - Persistent vomiting
  - Evidence of poor adherence to recommended therapy or inadequate follow-up.
- In patients who are discharged it is imperative that the following are ensured:
  - Patients and parents receive detailed instructions regarding dosing recommendations and are comfortable with home management
  - Close follow-up is available by the primary care physician and/or clinic responsible for care, even if it is only by phone.

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**Notification of Endocrine Service**

- **The Endocrine Service should be notified of all patients with DKA.**

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**DISCLAIMER:**

This clinical guideline has been developed for the purpose of unifying the general emergency care of patients with diabetic ketoacidosis (DKA). It is intended to aid, rather than substitute for, professional judgment. It is not intended to serve as a rigid protocol or a written proxy for the standard of care. Failure to comply with this guideline does not represent a breach of the standard of care.