

# EVALUATION AND MANAGEMENT OF THE PATIENT WITH DIABETIC KETOACIDOSIS (DKA)

Pediatric Endocrinology  
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## I. Pertinent history in DKA patients

- A. Prodrome usually longer in new onset insulin dependent diabetes mellitus (IDDM)
- B. Polyuria ( $\pm$  enuresis), polydipsia
- C. Weight loss
- D. Fatigue, malaise
- E. Anorexia, abdominal pain, nausea, vomiting
- F. Possible history of antecedent illness
- G. Family history of Type 1 DM (new patients)
- H. Social history
  - 1. Who are the caregivers at various times of the day?
  - 2. Psychosocial stressors
- I. Compliance (known patients)
  - 1. Insulin
  - 2. Capillary blood glucose testing
  - 3. Diet
  - 4. Possibility of EtOH or substance abuse (adolescents)
- J. For known patients, past hospitalizations and ER visits, "Frequent flyers" account for a disproportionate number.

## II. Physical findings in DKA

- A. Dehydration
  - 1. Dry mucosa
  - 2. Sunken eyes
  - 3. Prolonged capillary refill
  - 4. Tachycardia
  - 5. Orthostatic changes
  - 6. Shock: hypotension, pallor, mottling, cool extremities
- B. Ketoacidosis
  - 1. Kussmaul respirations
  - 2. "Fruity" breath odor
- C. Nonfocal abdominal tenderness
- D. Lethargy or Significant alterations in mental status
- E. Look carefully for signs of infection

## III. Initial laboratory evaluation

- A. STAT- Bedside
  - 1. Capillary blood glucose ("Dextrostix")
  - 2. Urine dipstick for ketones and glucose
- B. STAT- Monitor Closely
  - 1. Serum Glucose
  - 2. Venous Blood Gas (VBG)
  - 3. Electrolytes
  - 4. Phosphorus, Calcium, & Magnesium
- C. STAT- Monitor Less Frequently
  - 1. Serum Ketones (Beta-hydroxybutyrate (BOH))
  - 2. BUN/Creatinine, Urinalysis (U/A)
  - 3. CBC with differential

- D. NOT URGENT- But Necessary
1. Amylase/Lipase
  2. Hemoglobin A<sub>1</sub>C
  3. Cultures (If necessary)
  4. Antibodies (Islet Cell Ab (ICA), anti-GAD65 Ab, insulin auto-Ab (IAA) – **NEW Dx**
- E. GENERAL GUIDELINE for LAB FREQUENCY

<u>LAB</u>	<u>FREQUENCY</u>
Capillary blood Glucose (CBG)	q Hour
Serum Glucose	q 2 hrs, d/c when CBG < 300 mg/dL
Venous Blood Gas (VBG)	q 2 hrs until pH > 7.19, then q 4 hrs
Electrolytes, Ca, Mg, Phos	q 4 hrs
Urine dip for Ketones	At initial Presentation*
Serum Ketones (beta-hydroxy-butyrate)	At initial Presentation*
BUN, Creatinine	At initial Presentation*
CBC with differential	At initial Presentation*
Urinalysis	At initial Presentation*
Cultures (if indicated)	At initial Presentation*
Amylase, Lipase	At initial Presentation*
Hgb A <sub>1</sub> C	At initial Presentation
Antibodies (ICA,IAA,GAD65)	At initial Presentation <sup>Δ</sup>

\* May need to repeat depending on clinical situation

<sup>Δ</sup> Collect only if new onset

- F. Comments concerning labs
1. Capillary blood glucose measurements are unreliable when >500 or <50. They should be confirmed with a serum glucose determination if necessary.
  2. Continue hourly capillary blood glucose measurements while the patient is acidotic and on an IV insulin infusion.
  3. Correct serum sodium for hyperglycemia:  $\text{Corrected } [\text{Na}^+] = [\text{Na}^+] + 1.6([\text{glucose}] - 100)/100$
  4. Calculate the anion gap ( $\text{Nml} = 8 - 16 \text{ meq/L}$ ) =  $\{(\text{Na} + \text{K}) - (\text{Cl} + \text{HCO}_3)\}$ . An elevated anion gap is typical in DKA and usually reflects the accumulation of beta-hydroxybutyrate (BOH), acetoacetate, and lactate.
  5. If patient presents obtunded or comatose, check plasma osmolality and follow closely.
  6. An elevated BUN may reflect a decrease in extracellular volume and a decrease in GFR or, it may be increased secondary to protein breakdown.
  7. Leukocytosis with a left shift is common but does not usually imply infection - it is often secondary to a stress response.

#### **!V. Clinical Monitoring**

- A. Vital signs q hr
1. Respiration Pattern (Kussmaul?), pulse, BP
  2. Neuro checks
    - a. Pupils, Mental status
    - b. Glasgow Coma Score (Modified for young children) in obtunded patients
- B. Cardiac monitor
- C. Strict I & O
- D. If patient still in shock after initial bolus infusion(s), **consider** central line and CVP monitoring.

V. **Initial Resuscitation Fluids**

- A. Shock - i.e. hypotension, pallor, mottling, cool extremities, obtunded mental status
  - 1. 20 ml/kg NS (normal saline, 0.9% NaCl) IV push (over 30 min)
    - a. Reassess after bolus
- B. Not in Shock:
  - 1. 10-20 ml/kg NS IV over 30-60 min

VI. **Decisions for further therapy in DKA after initial fluid bolus infusions**

- A. Threshold values for DKA treatment:
  - 1. Does the patient appear ill ?
  - 2. Can the patient tolerate oral fluids/eat ?
  - 3. Does the patient have significant acidosis (pH <7.30, HCO<sub>3</sub> < 18) ?
- B. If, after initial hydration, the patient does not have significant acidosis and can eat, (s)he may be managed with subcutaneous insulin. NS at maintenance may be continued to help clear ketones.
- C. If the patient fails one or more of these criteria, (s)he is treated as described below.

VII. **Replacement Fluids: After initial fluid bolus infusions**

- A. General Guidelines:
  - 1. Total rate of all fluids should be **1.75 (≤ 20 kg) - 2.0 (> 20 kg) times maintenance**. This corresponds to < 4 L/M<sup>2</sup>/24 hrs. Exceeding this **MAY** increase risk of cerebral edema.
  - 2. If this rate is used, it is **NOT** necessary to calculate and replace deficits.
  - 3. Include the volume of the insulin infusion in calculating the rate.
  - 4. For patients in shock, additional fluids may be required to maintain CVP or urine output.
  - 5. The initial fluid rate of **1.75 - 2.0 x maintenance** is used until acidosis is resolved and the patient can tolerate oral fluids. Remaining rehydration can be completed orally, but **1-1.5 x maintenance** IV fluids may be continued until serum ketones are clear, especially if acidosis is resolving during the night and the patient is sleeping. It is not usually necessary to maintain a fluid restriction after acidosis has resolved.
- B. Maintain a fluid restriction for 48 hrs if:
  - 1. Signs/symptoms of increased ICP (see end of protocol for discussion of cerebral edema)
  - 2. Initial serum glucose > 1,000 mg/dl
  - 3. Initial corrected serum sodium > 150 meq/L.
- C. **Beware of uncorrected serum sodium failing to increase as serum glucose decreases**
  - 1. A decrease in serum sodium implies free water overload
  - 2. Indicates an increased risk for cerebral edema
  - 3. Fluids may need to be restricted
- D. Sodium content of fluids
  - 1. Use NS for the first 1-2 hours and 1/2 NS thereafter.
  - 2. Consider 1/4 NS if corrected Na increasing on therapy and is ≥ 145 meq/l.
- E. Potassium content of fluids:
  - 1. Patients usually present with high normal or elevated serum potassium levels. Despite this, the patient is usually **total body potassium depleted** upon initial presentation.
  - 2. Guidelines for potassium replacement -- SEE BELOW

<u>Initial Potassium (mEq/L)</u>	<u>Amount KCl (mEq/L)</u>	<u>Amount K<sub>2</sub>PO<sub>4</sub> (mEq/L)</u>
< 4.0	30	30
4.0-5.5	20	20
> 5.5	0*	0*

\* Order 20 mEq/L of KCl and 20 mEq/L of K<sub>2</sub>PO<sub>4</sub> (K-PHOS) but do not infuse until K < 5.6

- 3. If initial serum K<sup>+</sup> > 5.5 mEq/L, then follow serum K<sup>+</sup> every hour until it is 5.5 mEq/L. This usually occurs within the first 1-2 hr of IV hydration. NS can be used until this point. It is best to order fluids from the pharmacy with K<sup>+</sup> as soon as the initial evaluation is complete, b/c serum K<sup>+</sup> usually <5.5 by the time those fluids are available.

4. **DO NOT ADD potassium to fluids if the patient has:**
  - a. Oliguria/Anuria
  - b. Acute renal failure
  - c. Cardiac arrest or serum potassium > 5.5 mEq/L.
5. Await the initial serum K<sup>+</sup> level before running fluids containing potassium. If there is a laboratory delay (> 1 hr), then add potassium (20 mEq/L KCl + 20 mmol/L K phosphate) as long as none of the conditions mentioned in **item 4** (above) are present.
6. **Watch for hypokalemia as treatment is initiated** -- potassium *decreases* with:
  - a. Administration of both glucose and insulin.
  - b. Correction of acidosis.
  - c. Rehydration--promotes renal function, increasing renal elimination of K<sup>+</sup>.
7. On the cardiac monitor, follow lead II for evidence of hypo- or hyperkalemia. If K<sup>+</sup> abnormalities are suspected, lead II should also be checked on an ECG machine because T-wave morphology on the cardiac monitor may be difficult to interpret.
8. **ECG Effects dependent on serum K -- SEE BELOW**

<b><u>Serum Potassium (mEq/L)</u></b>	<b><u>Effect on ECG</u></b>
< 2.5	Depressed ST, Biphasic T wave, U wave
> 6.0	Peaked T wave
> 7.5	Long PR, Wide QRS, Peaked T wave, V-Fib/Asystole
> 9.0	Absent P wave, Sinusoidal wave, V-Fib/Asystole

F. **Sodium Bicarbonate:**

With provision of fluid, insulin, and electrolytes, metabolic acidosis usually corrects spontaneously (owing to the interruption of ketoacid production, metabolism & excretion of ketoacids, and the generation of bicarbonate in the kidney). Bicarbonate should **ONLY** be given if there is cardiac irregularity or arrest.

1. **WHEN USING BICARBONATE:**  
Give one mEq/kg IV (max. dose = 50 mEq) over 30 minutes if serum pH < 7.00 **AND patient is in poor clinical condition** -- i.e. shock with hypotension, acute renal failure, symptomatic hyperkalemia (i.e. muscle weakness or paralysis, or changes in cardiac conduction), sepsis, cardiovascular instability (i.e. hypotension, impaired myocardial function), or obtunded/comatose. It is rarely necessary to give additional doses.
2. **DO NOT GIVE BICARBONATE:**
  - a. As an IV push
  - b. If patient is hypokalemic (HCO<sub>3</sub> drives K into cell, worsening hypokalemia).
  - c. If unable to adequately ventilate patient (i.e., if there is a component of respiratory acidosis) as it may be difficult to eliminate the CO<sub>2</sub> produced. PCO<sub>2</sub> will increase and serum pH will acutely decrease and you may have to intubate and ventilate.
3. **ARGUMENT FOR BICARBONATE**
  - a. Severe acidosis impairs myocardial contractility and is a CNS depressant.
  - b. A rapid, slight improvement in acidosis may stabilize the patient until definitive treatment takes effect.
4. **ARGUMENTS AGAINST BICARBONATE**
  - a. No difference in rate of recovery of patients treated with & without bicarbonate.
    - (1) Bicarbonate is associated with increased risk of cerebral edema in children.
  - b. **Paradoxical CNS acidosis caused by the CO<sub>2</sub> produced from HCO<sub>3</sub>**
    - (1) CO<sub>2</sub> crosses the blood-brain barrier more readily than bicarbonate and results in a lower CSF pH. This can cause a rebound alkalosis later in therapy, but is rarely a problem with the recommended dose.
  - c. **Bicarbonate shifts the oxy-hemoglobin dissociation curve to the left**
    - (1) Leads to impaired oxygen release to tissues and increased lactate.
    - (2) Rarely a problem with oxygenation, therefore not a major concern.

**VIII. Guidelines for Administration of Glucose and Insulin:**

- A. Start continuous intravenous infusion of regular insulin at 0.1 units/kg/hr:
1. If child's weight  $\geq 20$  kg, Mix 100 units Regular insulin in 100 ml NS (1:1 ratio).
  2. If child's weight  $< 20$  kg, Mix 25 units Regular insulin in 250 ml NS (1:10 ratio).
  3. Flush tubing with this solution prior to administration.
  4. Start continuous insulin infusion ASAP. (**Order insulin as soon as dx confirmed.**)
- B. Add glucose to fluids when :
1. Serum glucose  $\leq 300$  mg/dl
  2. If after initial hydration, blood glucose is decreasing at a rate  $> 100$  mg/dl/hr.
  3. If blood glucose is not decreasing rapidly, reevaluate insulin and fluid rates.
- C. Prepare two bags of IV fluids (Order with insulin infusion)
1. 2 bags of 1/2 NS with appropriate mixture of KCl and K phosphate (see K criteria above)
  2. 1 with 10% dextrose and the other without (the 2 fluids identical except for the dextrose)
  3. Start fluids to give 1.75 - 2.0 x maintenance rate (based on body weight).
  4. Glucose infusion rates can be titrated by raising and lowering the rate of D10 delivered and adjusting the infusion rate of the glucose-free solution to keep the total hourly fluid volume constant.
- D. Subsequent management of glucose levels in DKA
1. Increase the ratio (i.e., the glucose infusion rate) if serum glucose continues to fall too rapidly -- i.e. greater than 50-100 mg/dl/hr.
  2. The rate of decrease of serum glucose is often greater than this limit during the first hour of rehydration as the intravascular volume expands (this increases GFR which increases glucosuria).
  3. The serum glucose usually corrects before the metabolic acidosis, and the degree of acidosis bears little relationship to the degree of hyperglycemia.
  4. **Avoid decreasing the rate of the insulin infusion** while the patient is **ACIDOTIC** since this will delay the clearance of ketones and the correction of the acidosis. Increase the glucose infusion rate instead.
  5. The insulin infusion rate may be increased to 0.15-0.2 units/kg/hr if, with no glucose in the IV fluids, hyperglycemia and acidosis have not improved after 4-6 hrs. This is rarely necessary. Be sure to recheck all fluids and IV lines for leaks or poor connections.
  6. Aim for a glucose level of 100-200 while the patient is on IV insulin. Levels  $>200$  lead to glycosuria and osmotic diuresis. Levels  $<100$  place patient at risk for hypoglycemia.
  7. If acidosis almost resolved ( $\text{pH} \geq 7.30$ ), it is permissible to decrease the insulin infusion rate rather than use very large volumes or high glucose-containing solutions. It is also possible to give the patient ~4-6 oz of glucose-containing juice or soda at this time.
  8. **Hypoglycemia should rarely occur in patients on IV insulin.**
    - a. Signs and symptoms of hypoglycemia include: somnolence, headache, confusion, fatigue, seizures, loss of consciousness, palpitations, anxiety, tremors, and diaphoresis.
    - b. Treatment of hypoglycemia:
      - (1) Mild hypoglycemia (60-80) - increase rate of glucose infusion.
      - (2) In severe ( $<40$ ) or very symptomatic (seizures, loss of consciousness) hypoglycemia, push 2 ml/kg D<sub>25</sub> IV.
      - (3) Follow capillary blood glucose (cbg) every 10-20 min until stable.
      - (4) Once hypoglycemia is corrected, restart insulin drip at a lower rate and/or increase the rate of IV glucose.

## **IX. Phosphate, calcium, and magnesium content of fluids**

- A. Phosphate
1. Despite the actual serum phosphate value, the patient is usually total body depleted of phosphorus upon presentation
  2. Serum PHOS decreases during treatment of DKA (Insulin drives Phos intracellularly).
  3. Serum phosphate > 1.5 mg/dL is OK.
  4. If serum phosphate < 1.5 mg/dL, then give all potassium replacement as K-phosphate.
  5. Severe hypophosphatemia (< 1 mg/dL) is rare in DKA.
  6. Hyperphosphatemia may lead to hypocalcemia or metastatic calcification.
  7. Do not give phosphate if there is clinical or biochemical evidence of hypocalcemia.
- B. Calcium
1. Symptomatic hypocalcemia is rare in DKA.
  2. Treat symptomatic hypocalcemia with 100 mg/kg IV calcium gluconate (max. 2 g/dose).
    - a. Administer slowly over 30 minutes.
    - b. Monitor HR & rhythm during infusion.
    - c. Avoid extravasation.
- C. Magnesium
1. Symptomatic hypomagnesemia is rare in DKA
  2. Correct hypomagnesemia (serum Mg < 1.0 mEq/L) before correcting hypocalcemia.
    - a. MgSO<sub>4</sub> 25-50 mg/kg IV over 1-2 hours or IM

## **X. Stopping IV therapy**

- A. Endpoint of IV therapy for DKA is **NOT** normoglycemia - it is **CORRECTION OF ACIDOSIS**
- B. The criteria for stopping IV insulin are essentially identical to the criteria for starting it:
1. Whether the patient can eat
  2. Resolution of acidosis: pH >7.30, HCO<sub>3</sub> > 18
  3. Convenient Meal Time (i.e., breakfast, lunch, or supper)
- C. It is best to switch to s.c. insulin at the next regular meal.
1. Order an ADA diet with 3 meals and 2 (< 8 y/o) or 3 snacks. Estimate calories as 1000 + 100 \*age (yrs) unless the child is pubertal. Sedentary adolescents need 2500 kcal/day whereas active adolescents may need 3000-4000/day. So for pubertal children, try and get an idea about the calories from the child and family, but also get a nutrition consult as a guide. Make sure the tray is **AVAILABLE (in sight) BEFORE** proceeding.
  2. Administer s.c. insulin (New patient)
    - a. The estimated insulin dose for a new patient is ~ 0.5 - 1.0 units/kg/day.
    - b. A simple to remember, **RULE OF THUMB** is:

(1) Severe DKA	1.0-1.5 units/kg/day
(2) Moderate DKA	0.8 units/kg/day
(3) Mild DKA	0.6 units/kg/day
(4) Non-Acidotic	0.4-0.5 units/kg/day
    - c. If the patient is started on an NPH & Humalog or Novolog regimen, give 0.4 units/kg (~ 2/3 NPH and 1/3 Humalog or Novolog) at breakfast, 0.3 units/kg of Humalog or Novolog before supper, and 0.3 units/kg of NPH at bedtime. If the odd chance occurs that the child is initially started on insulin at lunch, give 0.2 units/kg of Humalog or Novolog and 0.2 units/kg NPH.
    - d. If the patient is started on a Lantus & Humalog or Novolog regimen, give 0.4 units/kg as Lantus and 0.15 units/kg of Humalog or Novolog. The patient should continue to receive 0.15 units/kg of Humalog or Novolog with each scheduled meal, but the lantus should continue to be given ~ every 24 hrs.
    - e. Patients **SHOULD NOT** be switched from IV to SC insulin in the middle of the night or, at an inconvenient time of day.
    - f. Patient is to begin eating **IMMEDIATELY AFTER** s.c. insulin injection
    - g. If using Regular insulin, stop IV insulin & glucose 20 mins after injection
    - h. If using Huma/Novolog, stop IV insulin & glucose immediately after injection
    - i. Continue IV solution **without** glucose at maintenance if ketones present.

3. Administer s.c. insulin (Established patient)
  - a. If the patient is not new, the previous home insulin regimen may be used.
  - b. Patient is to begin eating **IMMEDIATELY AFTER** s.c. insulin injection
  - c. If using Regular insulin, stop IV insulin & glucose 20 mins after injection
  - d. If using Huma/Novolog, stop IV insulin & glucose immediately after injection
  - e. Continue IV solution **without** glucose at maintenance if ketones present.

#### XI. Criteria for PICU admission

Depending on the medical center, all patients receiving IV insulin *may require* PICU admission. If that is not the case, the following are **GENERAL** criteria for PICU admission.

- A. Age < 3 years
- B. Current Serum pH < 7.10
- C. Mental Status Changes
- D. Cardiovascular Instability, i.e. failure to improve perfusion after 2 bolus infusions
- E. Respiratory Insufficiency
- F. Initial Serum Glucose > 1,000 mg/dL

#### XII. Cerebral Edema

- A. Most patients in DKA have some degree of asymptomatic cerebral edema that usually doesn't require specific treatment.
- B. Symptoms from cerebral edema usually occur within the first 16 hr after initiation of therapy for DKA. Patient is usually in the process of recovery with improvements in acidosis/hyperglycemia.
- C. Signs & symptoms of cerebral edema are those associated with increased intracranial pressure (ICP). Some of these signs (esp. 1 & 2) can also occur in hypoglycemia, which should be quickly ruled out.
  1. Severe headache
  2. Mental status changes (irritability, decreased cooperation, disorientation, decreased level of consciousness). Many patients in DKA are lethargic, but this usually improves with therapy.
  3. Bradycardia/hypertension/respiratory insufficiency (**Cushing's triad**). Be aware of the normal heart rate for the age. The heart rate in a patient with DKA should decrease with IV fluid therapy, but **NOT** to below the normal range.
  4. Recrudescence of vomiting. Most patients in DKA are vomiting on presentation, but this should improve on therapy.
  5. One or both pupils fixed and dilated.
  6. Papilledema.
  7. Focal neurologic signs.
  8. Polyuria secondary to diabetes insipidus or, conversely, oliguria secondary to SIADH.
  9. Coma.
- D. Proposed risk factors for development of cerebral edema:
  1. New-onset diabetes
  2. History of prolonged ketoacidosis (several days)
  3. Extended history of poor diabetic control leading to chronic hyperosmolality.
  4. Age <5
  5. Moderate-to-severe acidosis (serum pH < 7.20)
  6. Elevated BUN
  7. Receiving > 4 L/M<sup>2</sup>/day of fluids
  8. Excessive swings of serum glucose, plasma osmolality, and serum pH
  9. Variable rate of fluid replacement
  10. Rate of decrease of serum glucose > 100 mg/dL/hr
  11. Failure of serum sodium to increase as serum glucose decreases.
  12. Rapidly decreasing plasma osmolality or very low osmolality during 1<sup>st</sup> 24 hr of therapy.

- E. Therapy for cerebral edema (in approximately this chronologic order)
1. In obtunded patients, assess the airway and ensure that the patient is breathing.
  2. Raise head-of-bed 30 degrees.
  3. Head in midline position.
  4. Decrease IV fluid rate to less than maintenance (0.5 – 0.67 x maintenance).
  5. Mannitol
    - a. It is prudent to have mannitol at the bedside in any patient with severe DKA.
    - b. 0.5 g/Kg IV over 5 min, may repeat 0.25 g/kg q5-10 minutes x 2 until a response (pupils, vitals, consciousness) is obtained.
    - c. There is little consensus about the exact dose; speed is critical.
  6. Hyperventilation
    - a. If the patient is obtunded, this may be started immediately with bag and mask.
    - b. Intubation with paralysis and sedation are more effective.
  7. Stat ICU consult (and subsequent transfer) if patient is not already in ICU.
  8. Head CT, if the patient is sufficiently stable.
  9. Stat neurosurgical consult - ICP monitoring.

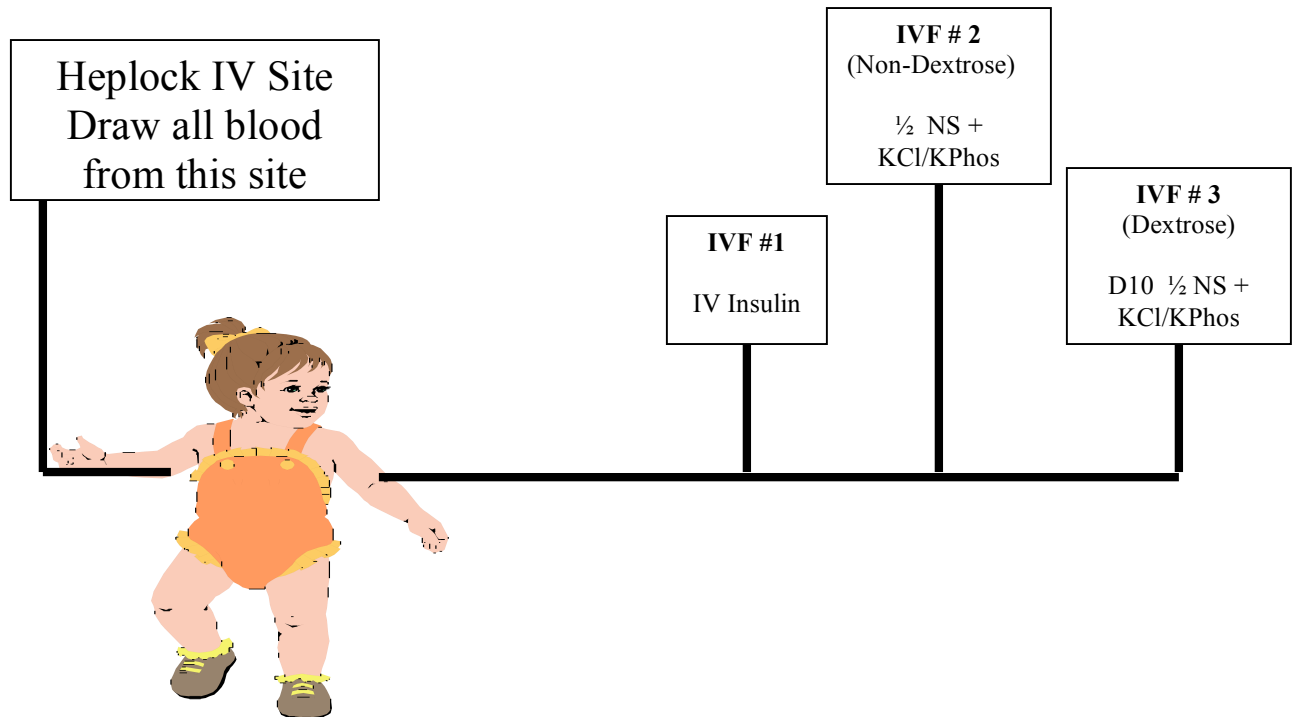
### **XIII. Evaluation of infections in patients with DKA**

- A. Infections are frequent precipitants of DKA in patients with Type 1DM
- B. Patients with Type 1 DM are prone to bacterial infections, especially if DM not well controlled.
  1. Urine
  2. Sinuses
  3. Skin and subcutaneous tissue
  4. Lungs
- C. Fever may be absent in a patient with DKA, particularly if the patient is poorly perfused.
- D. Physical exams and chest X-rays are unreliable for diagnosing pneumonia in dehydrated patients. Rales and infiltrates may become apparent with rehydration.
- E. Conversely, CBCs are initially of little diagnostic value because high levels of stress hormones (epinephrine and cortisol) lead to demargination of neutrophils, causing leukocytosis & a left shift.



# Diagram of Fluid Regimen

## DKA Management



# DKA FLOWSHEET

Please attach this form to the child's bed at the initiation of DKA therapy

Patient Name: \_\_\_\_\_ MR Number: \_\_\_\_\_  
 Date of Birth/Age: \_\_\_\_\_ Today's Date: \_\_\_\_\_  
 Weight (kg): \_\_\_\_\_ Height (cm): \_\_\_\_\_  
 BSA (m<sup>2</sup>): \_\_\_\_\_ BMI (kg/m<sup>2</sup>): \_\_\_\_\_

Maintenance IVF Rate: \_\_\_\_\_ cc/hr  
 1.5 x Maintenance IVF Rate: \_\_\_\_\_ cc/hr  
 1.75 x Maintenance IVF Rate: \_\_\_\_\_ cc/hr  
 2.0 x Maintenance IVF Rate: \_\_\_\_\_ cc/hr **DO NOT EXCEED THIS RATE**

Time	T/P/R/BP	Glucose (ser/cap)	Na/K	Ca/Phos	pH/BE	NS bolus (cc)	#1 IVF (cc/hr)	#2 IVF D10 (cc/hr)	Insulin (cc/hr)	Total Fluid (cc/hr)

## Example

- A. 8 y/o, 30 kg child presenting in DKA (pH 7.11, glu 678, Na 130, K 4.2, Phos 4.6, HCO<sub>3</sub> 8, large ketones)
1. Initial Fluid Bolus
    - a. NS 300 or 600 cc (20 cc/kg) IV
- B. After bolus, peripheral perfusion improves, crt 2 sec, HR 110 bpm
1. Continue to treat with NS at 2.0 x maintenance until fluids from pharmacy arrive
    - a. NS at 140 cc/hr (maintenance IVF ~ 70 cc/hr)
  2. Order fluids from Pharmacy STAT
    - a. 1/2 NS + 20 mEq Kcl + 20 mEq K<sub>2</sub>PO<sub>4</sub>/liter
    - b. D10 1/2 NS + 20 mEq Kcl + 20 mEq K<sub>2</sub>PO<sub>4</sub>/liter
    - c. Mix 100 units of Regular Insulin in 100 cc of NS (1.0 unit/cc)
- C. Fluids arrive and new labs: glu 388, pH 7.08
1. Calculate fluid delivery rates
    - a. Insulin: 30 kg x 0.1 u/kg/hr = 3 units/hr, 3 u/hr = 3 cc/hr
    - b. Since 2.0 x maintenance = 140 cc/hr, total fluid able to deliver = 140-3 = 137 cc/hr
    - c. Since Glucose still > 300, Run {1/2 NS + 20 mEq Kcl + 20 mEq K<sub>2</sub>PO<sub>4</sub>/liter} at 137 cc/hr
    - d. The D10 fluid bag should be hung **BUT HEPCLOCKED** for now
  2. Continue to monitor CB glucose q 1 hr and other labs as described in protocol
- D. Next CB glucose collected after insulin/IV fluids started is 243
1. Run the D10 bag at 68.5 cc/hr and decrease the Non-dextrose IV bag to 68.5 cc/hr
- E. Next CB glucose is 147
1. Although it is desired to keep CB glucose 100-200, with rapid drop, increase D10 to 100 cc/hr and further decrease the Non-dextrose solution to 37 cc/hr (Remember to keep total fluids 140 cc/hr).
- F. Patient does well and by AM, CB glucose 124, pH 7.33, Na 141, K 4.4, Phos 4.0, urine ketones small:
1. Order breakfast tray – ADA 1800 kcals with 3 meals & 2 snacks (afternoon/bedtime)
  2. Subcutaneous Insulin
    - a. If NPH/Humalog or Novolog, give ~30 units/day (1.0 units/kg/d – severe acidosis)
      - 1) AM: 14 NPH/7 Humalog for breakfast
    - b. If Lantus/Humalog or Novolog
      - 1) AM: 15 Lantus/5 Humalog for breakfast
  3. When tray arrives, give s.c. insulin, and pt is **TO EAT IMMEDIATELY** after injection
  4. After injection, d/c IV D10 bag and IV insulin
  5. Since Ketones present, continue Non-dextrose bag at 1-1.5xmaintenance (70 - 100 cc/hr)
  6. Change glucose checks to qac,qhs,qMN,q0300
  7. Repeat BMP ~ 4-8 hrs after discontinuing ALL IV fluids