Management of Diabetic Ketoacidosis (DKA) in Pediatrics

Physician Direct Connect Line
(614) 722-2052
1(866) 722-2052
I. Assessment in the Emergency Department

A. Initial Nurse Assessment
   1. Establish baseline mental status. If altered mental status, alert MD immediately
   2. Initial lab assessment
      a. blood glucose by glucometer
      b. arterial or venous blood gas
      c. serum for glucose, electrolytes, CBC with diff, magnesium, calcium, phosphorous, hemoglobin A1C, and beta hydroxybutyrate.
      d. urine for urinalysis (glucose and ketones)
      e. for new onset: “Islet antibody screen”(GAD, ICA, IAA antibodies), and if not acutely ill, TSH
   3. Start a DKA flowsheet

B. Initial MD Assessment
   1. History and physical with documentation of baseline mental status
   2. Ensure above lab tests sent

C. ICU admission guidelines:
   1. Altered mental status
   2. pH<7.10
   3. Age less than 2 years with pH<7.24
   4. Blood glucose>1000mg/dl
   5. Corrected sodium>155 mmol/L
      Corrected serum sodium= (Serum sodium + [{serum glucose - 100} x 1.6]/100

Table of Contents

I. Assessment in the Emergency Department 3
II. Initial Fluid Therapy: Normal Saline Bolus 4
III. Maintenance Fluids 4
IV. Insulin Therapy 6
V. Labs 7
VI. Always be alert for Cerebral Edema 8
VII. Stopping IV therapy 8
Pathophysiology 9
II. Initial Fluid Therapy: Normal Saline Bolus
1. If less than 7% dehydrated, NS bolus is optional
2. If 7-10% dehydrated, give 10 mL/kg NS bolus over 30 minutes
3. If >10% dehydrated or with cardiovascular compromise, repeat 10 mL/kg NS boluses until cardiovascularly stable (normal peripheral pulses, cap refill time (<3 sec), urine output restored, normal BP)

III. Maintenance Fluids
A. Rate: Run IVF at 3000 mL/m2/day, or 1.5-2.0 times maintenance, (or calculate maintenance plus ongoing losses plus deficit replacement over 48 hours) unless hypernatremic dehydration (see below)
   \[ m2 = \sqrt{\text{height}(\text{cm}) \times \text{weight}(\text{kg})}/3600 \]
B. Sodium content: First determine corrected Na with the following equation:
   \[ \text{Corrected serum sodium} = \text{Serum sodium} + \left\{ \text{serum glucose (mg/dl)} - 100 \ (\text{mg/dl}) \right\} x 1.6/100 \]
   1. If hypernatremic dehydration (defined as corrected Na over 150 mmol/L)
      a. Always manage with help of endocrinology or ICU staff
      b. The initial goal is to lower the serum glucose to 200-300 mg/dl over 24 hours with little or no decrease in the corrected serum sodium
      c. Consider 3/4 NS or NS infusion to accomplish controlled rate of sodium correction
      d. Fluid rate in this case should be 2000 mL/m2 to control the drop in osmolarity
   2. If corrected Na ≤ 150 mmol/L, use 1/2 NS for maintenance fluid.
C. Potassium content: Remember K+ will fall as acidosis corrects with insulin therapy
   1. If K>6.0 mmol/L, do not add potassium to fluids
   2. If K+ between 4 and 6 mmol/L, give a total of 40 meq/L of K+ (suggested 20 meq KCl/L + 20 meq KPhos/L)
   3. If K+ less or equal to 4 mmol/L, give a total of 60 meq/L of K+ (suggested 30 meq KCl/L + 30 meq KPhos/L)
D. Phosphate content: Remember Phos will also fall with correction of acidosis with insulin therapy. IV phosphate administration is recommended. This is usually given with potassium as above (see II.C).
E. Glucose content:
   1. Add glucose to the IV fluids when:
      a. the serum glucose is decreasing at greater than 100mg/dl/hr.
      b. When the blood glucose falls below 400 mg/dl use a “two bag system” of IV fluid Y’ed together. The bags have the identical composition except that one contains no dextrose and the other contains D10. By changing the relative rates for each bag, D0, D5, and D10 can be rapidly selected.
   2. Start with D5 and increase to D10 or D12.5, as needed to maintain glucose 150-200 mg/dl. Decrease insulin infusion rate only if necessary to avoid hypoglycemia once on D12.5.
Plan Ahead: The serum glucose usually falls 100-200 mg/dl after initial NS bolus because of dilution and increased GFR. If initial glucose in the ED is under 500 mg/dl BEFORE the NS bolus, consider ordering D5 in the maintenance fluids to have ready once the NS bolus has completed, if not using the “two bag system.”

IV. Insulin Therapy:
A. If ketone-positive and bicarb ≤ 15, start an insulin drip (Regular Insulin) at 0.1 units/kg/hour
   a. Remember, need insulin to correct acidosis. If blood glucose is dropping, add glucose to fluids. Do not decrease insulin drip unless absolutely necessary (i.e. new fluids aren’t available yet) and then turn drip back up ASAP.
   b. Disconnect the insulin pump for patients on a pump.
   c. Optional to continue to administer Lantus q day for patients on Lantus. Continuing Lantus makes it easier to transition back to sub cu insulin.

B. If ketone negative hyperglycemic hyperosmolar syndrome (serum glucose >1000 mg/dL, osm >380, little to no acidosis, and MS changes), consult Endocrinology before starting insulin drip.

   These patients should get no insulin or a reduced rate (0.01 units/kg/hour, initially) in order to correct the hyperosmolar state slowly.

C. If isolated hyperglycemia or ketone-positive without significant acidosis (bicarb > 15), can usually treat with sub cu insulin. Hyperglycemia will cause dehydration, so use of IVF should be considered to speed metabolic recovery.

D. If treating ketone-positive patient with sub cu insulin boluses, they will need additional rapid acting insulin to clear ketones:

   - Small ketones: 5% of normal BASAL insulin dose added to bolus
   - Mod ketones: 20% of normal BASAL insulin dose added to bolus
   - Large ketones: 30% of normal BASAL insulin dose added to bolus

V. Labs:
   a. Bedside glucose every hour while on insulin drip
   b. Electrolytes every 2 hours until bicarb greater than 15 meq, then can space out to every 4 to 6 hours
   c. Ca, Mg, Phos every 4 hours
   d. Urine ketones or blood ketones (serum beta hydroxbutyrate) every 3-4 hours until negative X3.

   Interpretation of serum ketones
   - < 0.5  Negative
   - 0.6-0.9  Small
   - 1.0-1.5  Moderate
   - > 1.6  Large
VI. Always Be Alert for Cerebral Edema

a. Risk factors: young children, new diagnosis, large fall in osmolarity, severity of acidosis, 3-24 hours after start of fluid therapy.
b. Signs and symptoms: headache only (early), then, mental status changes, HTN, bradycardia, pupil abnormalities, posturing
c. Treatment (always in conjunction with Endo or ICU staff):
   1. Decrease rate of IVF by 1/2
   2. Mannitol 250-1000 mg/kg IV over 20-30 minutes
   3. Alternative: 3% NS bolus, 5-10 ml/kg IV over 30 minutes.

VII. Stopping IV therapy

A. Maintain IV administration of insulin until:
   1) The patient is hungry and able to take adequate intake by mouth, and
   2) The anion gap is resolved, and
   3) HCO3 rises to 18 meq/L. Except, after high volumes of IV saline, hyperchloremia may prevent HCO3 from rising above 15 mg/dl.

B. For a patient not already receiving insulin glargine (Lantus) or insulin detemir (Levemir) give the basal insulin one hour before discontinuing the IV insulin drip. Give the initial dose of Lantus or SC rapid acting insulin 10 minutes before discontinuing the IV insulin.

C. Maintain IV fluid therapy until the patient has demonstrated successful oral.

Pathophysiology

A. General Principles of Therapy of DKA
   1. Patients with significant acidosis (pH<7.2) are always dehydrated.
   2. New onset patients are at increased risk for cerebral edema probably because they have become dehydrated over a longer period of time than an individual with diabetes who becomes acutely ill.
   3. Avoid cerebral edema by correcting deficits in fluid and electrolytes over 48 hours, by keeping the total amount of fluid administered to less than 4000 ml/M2/24 hours and by avoiding hypotonic solutions early in the course of treatment.
   4. Insulin is required to inhibit fatty acid release and subsequent ketogenesis. Thus, if the blood glucose is falling rapidly or to a low concentration, it is better to increase the concentration of glucose in the IV infusion than to reduce the rate of infusion of insulin. The latter will allow ketone production to resume in DKA due to the ongoing catabolic insulin resistant state.

B. Potassium
   1. In DKA, a normal or low serum potassium in the presence of acidosis is evidence of severe total body potassium depletion. This is because acidosis drives potassium out of the intracellular compartment, where it is present in highest concentrations, to the serum from which it is subsequently lost by osmotic diuresis.
2. Insulin administration will rapidly lower serum potassium concentration.
3. Hypokalemia can result in cardiac arrhythmias.

C. Phosphate
1. Due to the osmotic diuresis induced by hyperglycemia in DKA, the initial serum phosphate concentration may be decreased. In addition, insulin administration will lower the serum phosphate level. Decreased phosphate levels will result in depletion of 2,3 DPG, shifting the oxyhemoglobin dissociation curve to the left if the acidosis is corrected too rapidly. This shift will hamper O2 delivery to the tissues. Therefore, IV phosphate administration is recommended.

D. Ketones
1. Ketones exist as acetoacetate (AA), beta hydroxybutyrate (BHB) and acetone (AC). In DKA there is a predominance (up to 10X) of BHB over AA and AC. Therefore measurement of serum BHB is preferred. The urine ketone test strip (nitroprusside reaction) measures AA, reacts poorly with AC, but does not measure BHB. This may lead to an underestimation of the concentration of ketones present. As the acidosis resolves, BHB is metabolized to AA. This process results in a worsening or prolonged ketonuria of less clinically significant. [The blood ketone meter (Precision Xtra and NovaMax Plus) measure BHB directly and does not measure AA or AC.]

E. Hypocalcemia and hypomagnesemia
1. Chronic osmotic diuresis can result in magnesium deficiency. Hypomagnesemia results in functional hypoparathyroidism (inhibition of both PTH secretion and action). Thus consider an IM injection of magnesium sulfate for persistent hypocalcemia if concomitant hypomagnesemia is present.

NOTES: