

**MSH GUIDELINES FOR PEDIATRIC MANAGEMENT OF DIABETIC KETOACIDOSIS**

This is not intended as a rigid protocol, but is offered as a guideline. Discuss each case promptly with the Pediatric Endocrine Fellow and Attending on call. Each case needs individual assessment and frequent reassessment with the Diabetes Team during the course of treatment.

\*\*\* 24-hour Pediatric Endocrine Contact via 212-241-6936 or pager listed on Amion \*\*\*

**Diabetic Ketoacidosis is defined as:**

1. **Glucose >200mg/dL and**
2. **Moderate to large ketonuria/ketonemia and**
3. **Venous pH <7.3 or HCO<sub>3</sub> <15mEq/L<sup>1</sup>**

**A. INITIAL ASSESSMENT and MONITORING**

**History:**

Duration of illness, symptoms (polydipsia, polyphagia)

- Assessment of fluid loss (polyuria, nocturia, vomiting)
- Weight loss, abdominal pain, nausea and fatigue
- Medications (i.e. steroids)
- If known diabetes – review patient's home diabetes management, including insulin regimen, adherence and time of last insulin injection.

**Physical Examination (symptoms consistent with DKA)**

- Constitutional: weight loss, tachycardia
- HEENT/Skin: level of dehydration (dry mucous membranes, skin turgor, capillary perfusion, peripheral pulses), fundoscopic exam
- Respiratory: Kussmaul respirations, fruity odor breath
- Neurologic: level of consciousness, mental status, GCS

**Labs:**

- Obtain Point of Care Testing (POCT) blood glucose, VBG and urine dipstick
- Start 2 large bore IV lines
- Obtain chem10, CBC, HbA1C
- Calculate anion gap:  $[Na - (H_2CO_3 + Cl)]$

If patient is **not** known to have Diabetes Mellitus, before starting insulin treatment also obtain: Total insulin, C-peptide, DM related autoantibodies (Insulin Ab, GAD Ab, IA2 Ab, Zinc Transporter 8 Ab)

If clinically indicated – EKG, Urine pregnancy, Urine toxicology screen, Cultures, CXR.

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**B. TREATMENT OF DKA**

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- A. Patient should be NPO
- B. Normal Saline bolus (10cc/kg) over 1 hr
- C. Reassess hydration status to determine need for 2<sup>nd</sup> bolus (10cc/kg)
- D. Start Insulin IV infusion at 0.1Units/kg/hr
- E. Initiate 2 bag system at 1.5-2X maintenance rate<sup>2</sup>
  - Bag 1: Normal Saline
  - Bag 2: Dextrose 10% ½ Normal Saline
    - Add dextrose to fluids when blood sugar <300mg/dL
    - Adjust fluid rates based on glucose and electrolytes
- F. Add Potassium to fluids when serum K <5.0 mEq/L. Start with 20 mEq/L KCl and 20 mEq/L Kphos

Do not stop insulin infusion. If there is a rapid decline in blood glucose or hypoglycemia maximize IV dextrose concentration before considering a decrease in insulin rate. Goal is to decrease blood sugar by 50-100mg/dL/hr and to then maintain BG at 150-200 mg/dL<sup>1</sup>. Insulin infusion should be continued until pH > 7.3 and/or serum bicarb > 18 and/or anion gap normalizes.

**C. MONITORING AND OBSERVATION**

- i. Q1hr POCT glucose
- ii. Q2hr VBG
- iii. Q4hr chem10
- iv. Q1 hour vital signs and neuro checks
- v. Urinalysis every void until ketones have cleared
- vi. Strict Is/Os

Over-vigorous management can produce rapid changes in glucose, osmolality and pH and may contribute to development of complications including hypoglycemia, hypokalemia, hypocalcemia, hypernatremia, fluid overload and hyperchloremic acidosis.

**Cerebral edema** occurs in up to 1% of children with DKA and accounts for >50% of the mortality rate<sup>3,4</sup>. Patients at highest risk are those who are newly diagnosed and age < 5 years<sup>1</sup>. Peak incidence is around 8-12 hours after initiation of therapy. Diagnostic criteria for cerebral edema include abnormal response to pain, decorticate/decerebrate posture, cranial nerve palsy and/or abnormal respiratory pattern. If patient develops any of these symptoms or severe headache, mental status changes, bradycardia, or hypertension perform a complete neurological examination and consider empiric treatment with elevation of HOB, decreasing IVF rate, and mannitol IV. This is a medical emergency.

**D. TRANSITION FROM IV TO SUBCUTANEOUS INSULIN**

When the child is stable, alert, ready to eat and parameters improved (pH > 7.3, serum bicarb > 18, anion gap closed) they may be transitioned from IV to subcutaneous insulin. Insulin doses are individualized for each patient and must be discussed with the Pediatric Endocrinology Team.

- Once patient's food tray has arrived determine the total dose of short acting insulin (Lispro) using 2 calculations:
  - Insulin sensitivity factor (ISF)- used to correct for high BG levels
  - Insulin carbohydrate factor (ICF)- used to cover carbohydrates consumed  
(The Pediatric Endocrine Team will provide both calculations)
- Administer subcutaneous injections of both long acting insulin (Lantus) and short acting (Lispro) insulin
- Wait 20 minutes before discontinuing IV insulin
- Allow patient to eat. Should be ordered for a regular diet with NO concentrated sweets
- Change IV fluid to non-dextrose containing fluid and decrease IV fluid rate to adjust for PO fluid intake
- Once transitioned to subcutaneous insulin check POCT glucose premeal, bedtime, midnight, and 3am. Patient should not go >6 hours without a glucose check.

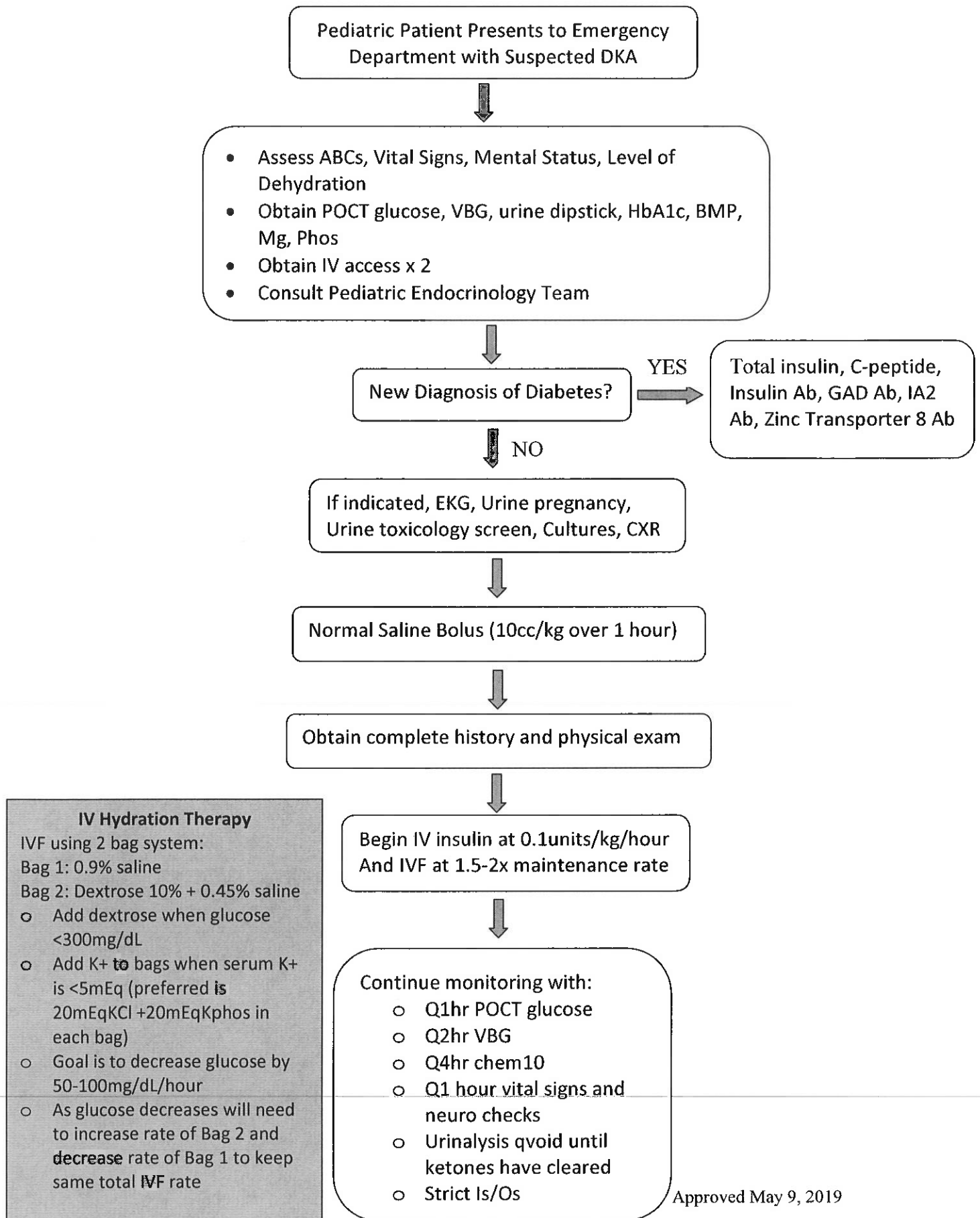
**Transition to Subcutaneous Insulin Caveat**

The majority of patients, in particular new onset diabetes patients, are transitioned to a basal/bolus subcutaneous insulin regimen. There is no particular time of day basal insulin is required to be given; patients will typically choose to administer this insulin either in the morning or in the evening at bedtime<sup>5</sup>.

The timing of transition to subcutaneous insulin can therefore pose a temporal challenge with regards to establishing a daily regimen for the patient. In general, endocrine will advocate for basal insulin in the morning or night in order to promote compliance. There is emerging evidence suggesting that basal insulin given during the treatment of DKA is both safe and may shorten the course of DKA. The decision of when to initiate basal insulin should therefore be discussed by the primary team and the Pediatric Endocrine team.

**References:**

1. Rosenbloom, A. The Management of Diabetic Ketoacidosis in Children. Diabetes Therapy. December 2010; 1:103-120
2. Schunk, J et al. Clinical Trial of Fluid Infusion Rates for Pediatric Diabetic Ketoacidosis. The New England Journal of Medicine. June 2018; 378: 2275-2287
3. Patel, A et al. Incidence, Trends, and Outcomes of Cerebral Edema Among Children with Diabetes Ketoacidosis in the United States. Clinical Pediatrics. 2016; 55(10) 943-951.
4. Wolfsdor J et al. Diabetic Ketoacidosis in Infants, Children, and Adolescents. A consensus statement from the American Diabetes Association. 2006, Diabetes Care; 29:5.
5. Barski, L et al. Basal Insulin for the Management of Diabetic Ketoacidosis. European Journal of Internal Medicine. January 2018; 47:14-16.



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