

# Pediatric Diabetic Ketoacidosis Clinical Pathway

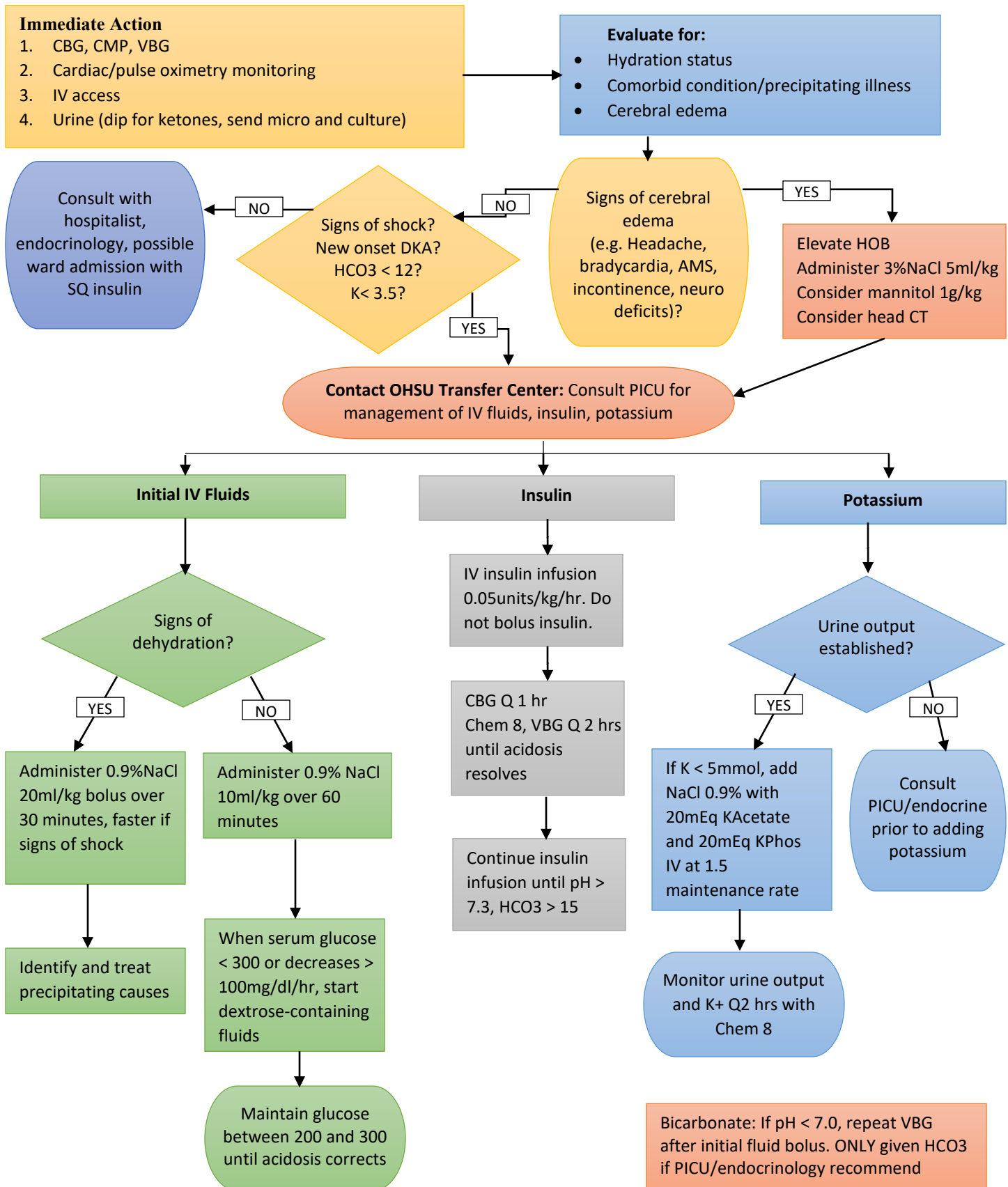
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<b>Outcomes/Goals</b>	<ol style="list-style-type: none"> <li>1. Initiate prompt, care of the pediatric patient presenting with DKA</li> <li>2. Correction of dehydration and hyperglycemia, resolution of electrolyte abnormalities, identification of precipitating and co-morbid illnesses</li> </ol>
<b>Inclusion Criteria</b>	<p>Patients 1 to 18 years who present in diabetic ketoacidosis, defined as presence of:</p> <ul style="list-style-type: none"> <li>• Blood glucose &gt;250mg/dl</li> <li>• Venous pH &lt;7.3 OR bicarbonate &lt;15mmol/L</li> <li>• Ketonuria</li> </ul>
<b>Exclusion Criteria</b>	<ul style="list-style-type: none"> <li>• Children &lt; 1 year</li> <li>• Patients with hyperglycemia without acidosis</li> </ul>
<b>NURSE</b> documentation	<p>ESI level II</p> <p>Full vital signs, weight, onset of symptoms, strict I/O, signs/symptoms of infection, assess cardiopulmonary and neurological stability including signs/symptoms of shock, respiratory distress, dehydration, mental status, focal neurologic deficits</p>
<b>INTERVENTIONS</b> Initiate on arrival	<p>IV access (2 peripheral sites in separate extremities once identified as PICU admission)</p> <ul style="list-style-type: none"> <li>• If unable to get PIV, consider central line or IO. Avoid arterial access.</li> </ul> <p>Venous blood glucose and urinalysis for ketones</p> <p>Cardiac/pulse oximetry monitoring with: Q1 hour vitals and Q1 hour neuro checks</p> <p>NPO, strict I/O</p>
<b>DIAGNOSTICS</b>	<p>A. All patients:</p> <ol style="list-style-type: none"> <li>1. Hourly capillary or venous glucose monitoring</li> <li>2. Serum: glucose, CMP, phosphorus, POC VBG w/lactate, and chem8, then chem8 + POC VBG/lactate Q2-4 hours until acidosis resolved and transitioned to SQ insulin</li> <li>3. Urine microscopy and urinalysis – culture if indicated</li> <li>4. Urine ketones Q void until ketosis resolves</li> </ol> <p>B. All <u>new</u> diabetic patients consider: A1C, TTG, IgA</p>
<b>PHYSICIAN (LIP)</b>	
Fluids (if indicated)	<p>A. If signs of dehydration, give 20 ml/kg NS to rehydrate. If no signs of dehydration, give 10ml/kg NS bolus over 30-60 minutes. Avoid excessive fluid administration.</p> <p>B. For patients on an insulin gtt, add dextrose (up to D10) when blood glucose &lt;300 mg/dl <b>OR</b> if glucose decreases more than 100mg/dl/hour – <b>see pg 3 for volume and rate</b>. Keep glucose between 200-300 mg/dl until acidosis corrects.</p>
Medication	<p>A. Insulin intravenous: start after consult with endocrine</p> <p>0.05 unit/kg/hour Regular insulin. (Requires RN double check)</p> <ul style="list-style-type: none"> <li>• Remember to flush 0.55 mL Microbore syringe tubing with 10-20 mL of Insulin prior to starting drip <b>or</b> flush 1-2 mL and allow to dwell for 20 minutes. Alaris 25 mL tubing should be flushed with 50 mL Insulin prior to starting drip.</li> <li>• After adding dextrose to fluids, titrate to prevent glucose from decreasing &gt;100mg/dl/hour.</li> <li>• <u>Do not</u> bolus insulin.</li> <li>• <u>Do not</u> use patient's personal pump if they meet criteria for DKA.</li> </ul> <p>B. Potassium: after initial hydration if K&lt;5mmol and urine output established use <b>NaCl 0.9% with 20 mEq KAcetate and 20 mEq KPhos IV at 1 ½ maintenance rate</b>. If no urine output established consult endocrine/PICU prior to adding replacement.</p>
Neurologic Deterioration	<p>If any signs of neurologic deterioration, consider hypoglycemia or cerebral edema. Warning signs of cerebral edema: headache, slow heart rate, irritability, AMS, incontinence, neurologic deficits.</p> <p>Interventions:</p> <ol style="list-style-type: none"> <li>1. Elevate head of bed</li> <li>2. Administer 3% saline 5 mL/kg IV over 30 min</li> <li>3. Consider decreasing IV fluids by 1/3</li> <li>4. Consider Mannitol 0.5-1 grams/kg over 10-15 min</li> <li>5. Consider intracranial imaging</li> </ol>
Identify Hyperosmolar Hyperglycemic State (HHS); if present	<p>A. Glucose &gt;600 mg/dL; serum osm &gt;320mOsm/L; may have bicarb &gt;15 mmol/L; mild acidosis</p> <p>B. HHS may be isolated and may develop hypotension if serum osm falls rapidly. (BP supported by hyperosmolality in the context of severe dehydration).</p> <p>C. Contact Endocrinology team.</p>
<b>Admission Criteria</b>	PICU admission criteria (any one): hemodynamically unstable, AMS, insulin drip, hypokalemia

# Clinical Pathway Decision Making Process

## Pediatric DKA

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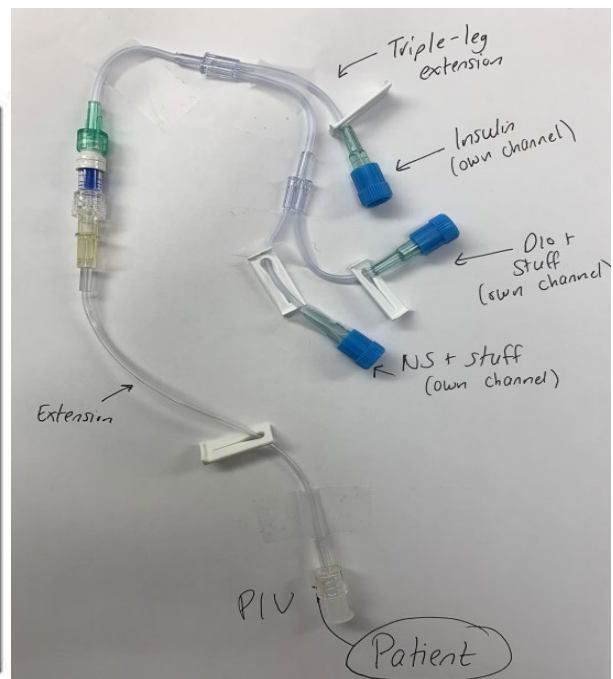
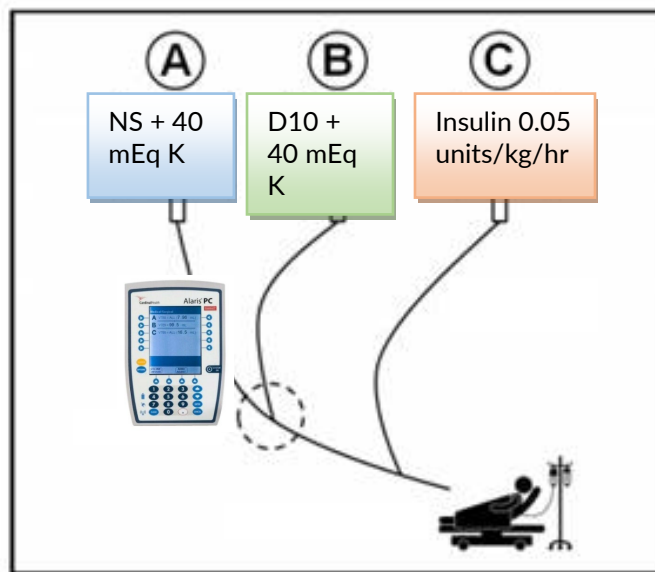
## IV Fluid Administration in Diabetic Ketoacidosis

*Infusion rates on fluids should range from 0-1.5x hourly maintenance rate:*

**Rate Bag A + Rate Bag B = total infusion rate**

GLUCOSE	RATE BAG A: NO DEXTROSE	RATE BAG B: D10 NS W/K	FINAL DEXTROSE CONCENTRATION
$\geq 300$	1.5 x maintenance rate	0	D0
$>200 \leq 300$	0.75 x maintenance rate	0.75 x maintenance rate	D5
$\leq 200 >150$	0	1.5 x maintenance rate	D10
$<150$	<b>Contact provider if POC glucose drops below 150</b>		

**Notify provider if POC Glucose drops greater than 100 mg/dL per hour**



## Pediatric DKA: Goals of Clinical Pathway

1. Initiate prompt, care of the pediatric patient presenting with DKA
2. Minimize / decrease risk of complications through slow decrease in blood glucose of 50-100mg/dl/hour until glucose <300
3. Correction of dehydration and hyperglycemia, resolution and anticipation of electrolyte abnormalities, identification of precipitating and co-morbid illnesses through coordinated care and close patient monitoring

Diabetic ketoacidosis (DKA) is a complex metabolic state of hyperglycemia, ketosis, and acidosis. Hyperglycemia causes an osmotic diuresis that leads to excessive loss of free water and electrolytes. Resultant hypovolemia leads to tissue hypoperfusion and lactic acidosis. Successful treatment of DKA includes correction of the dehydration and hyperglycemia, anticipation and resolution of electrolyte abnormalities, identification of precipitating and comorbid illnesses, and frequent patient monitoring.

Data Considerations	Rationale
Cerebral Edema and Fluid Resuscitation	DKA-associated cerebral edema (CE) has a low incidence, occurring in about 1% of all episodes of DKA. Overly rapid correction of serum hyperglycemia and osmolarity has been thought to contribute to the risk of CE. However, there is no good quality evidence suggesting that rapid fluid resuscitation is associated with cerebral edema. Further, recent evidence suggests no association between rates and volumes of fluid resuscitation and development of cerebral edema. Given this, our approach is to use fluid judiciously if signs of dehydration are absent but to fluid resuscitate per standard practice for a patient displaying signs of dehydration.
Potassium Replacement	Despite what may be severe total body potassium depletion, apparent serum hyperkalemia is often observed in patients with diabetic ketoacidosis prior to volume resuscitation. The risk of cardiac arrhythmias may be increased, therefore, continuous cardiac monitoring is recommended.
Bicarbonate Use	While ketosis and lactic acidosis produce a metabolic acidosis, supplemental bicarbonate is not recommended. A pediatric trial of bicarbonate in severe metabolic acidosis during DKA (pH <7.15) showed no benefit when compared with placebo. <sup>2</sup> Indeed, multiple studies suggest that bicarbonate therapy may cause paradoxical intracellular acidosis, worsening tissue perfusion and hypokalemia, and cerebral edema. Acidosis usually resolves with isotonic fluid volume replenishment and insulin therapy.

### References:

1. Kuppermann N, Ghetti S, Schunk JE, et al. Clinical Trial of Fluid Infusion Rates for Pediatric Diabetic Ketoacidosis. *N Engl J Med* 2018; 378:2275
2. Nallasamy K, Jayashree M, Singhi S, Bansal A. Low-dose vs standard-dose insulin in pediatric diabetic ketoacidosis: a randomized clinical trial. *JAMA Pediatr.* 2014 Nov;168(11):999-1005
3. Chua HR1, Schneider A, Bellomo R. Bicarbonate in diabetic ketoacidosis - a systematic review. *Ann Intensive Care.* 2011 Jul 6;1(1):23.
4. Glaser N, Barnett P, McCaslin I, et al. Risk factors for cerebral edema in children with diabetic ketoacidosis. The Pediatric Emergency Medicine Collaborative Research Committee of the American Academy of Pediatrics. *N Engl J Med* 2001; 344:264.
5. Knopp, Jennifer L., et al. Capacity of Infusion Lines for Insulin Adsorption: Effect of Flow Rate on Total Adsorption. *Journal of Diabetes Science and Technology* 2021, 15(1), 109-120. DOI: 10.1177/1932296819876924