Diabetic Ketoacidosis (DKA): Initial Management of the Pediatric Patient

EVIDENCE INFORMED PRACTICE TOOLS

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PURPOSE AND INTENT
To prevent death and neurological morbidity from DKA by assisting the emergency medicine practitioner with optimal management strategies.
Note: This guideline is appropriate for the majority of cases of DKA but does not replace clinical judgment. It is intended for use along with the Standard Order Forms (see PHOR 387; PHOR408). Consultation with a Pediatric Endocrinologist is required.

1. Practice Outcomes

The goals of treatment are to:

- restore perfusion to increase glucose utilization in the periphery and reverse the progressive acidosis.
- stop ketogenesis by administering insulin, to reverse proteolysis and lipolysis, stimulate glucose uptake and processing; normalizing blood glucose concentration.
- correct electrolyte imbalances.
- avoid the complications of treatment insofar as possible, including cerebral edema, hypoglycemia and hypokalemia and hypophosphatemia.

To prevent cerebral edema, these goals must be met in a manner that prevents a rapid decrease in the serum osmolarity. Treatment requires:

- meticulous attention to the patient’s response to fluid and insulin therapy
- careful observation of glycemia to prevent hypoglycemia which may occur abruptly during insulin therapy
- admission to an inpatient setting

2. Background

Diabetic ketoacidosis (DKA) is a complex metabolic state of hyperglycemia, ketosis, and acidosis, which results from untreated absolute or relative deficiency of insulin, respectively, in Type 1 or Type 2 Diabetes Mellitus. DKA is the most common cause of diabetes-related deaths in children. The most important cause of mortality and severe morbidity in children with DKA is cerebral edema.

Common Causes of DKA:

2.1 The initial presentation of Type 1 or Type 2 diabetes mellitus
2.2 Insulin omission through missed injections or pump failure
2.3 Insufficient insulin during an intercurrent illness, such as:
   2.3.1 Balanitis/Vaginitis
   2.3.2 Viral URTI, OM, myocarditis
3. Guidelines

Community and rural practitioners:
3.1 Rural physicians/practitioners should continue to follow the protocol until transfer is accomplished. If a child has minimal dehydration and is not vomiting, urgent transfer to a centre with specialized pediatric intensive care may not be necessary, but this should be discussed with the Pediatric Endocrinologist on call.
3.2 Otherwise, initiate arrangements for transfer as soon as the diagnosis of DKA is established and if required initial stabilization of Airway, Breathing and Circulation (initial ABC’s) have been addressed. The ongoing treatment with insulin, ongoing biochemical monitoring, and especially treatment of cerebral edema is inappropriate in centers without specialized pediatric intensive care.
3.3 Contact the Pediatric Endocrinologist on call regarding the assessment and management of any child with DKA.

Confirmation of the diagnosis of DKA:
3.4 For a diagnosis of DKA, all of the following criteria must be present:
   - Plasma Glucose ≥ 11 mmol/L
   - Capillary pH ≤ 7.30*
   - Capillary HC03 ≤ 15 mmol/L*
   - Ketones present in blood or urine (serum betahydroxybuterate > 2mmol/L or urine Ketones Large > 8 mmol/L)
3.5 If severe metabolic acidosis is present in the absence of hyperglycemia, rule out other causes such as inborn errors of metabolism, alcoholic ketoacidosis, or salicylate overdose
   *NOTE RE BLOOD GASES: use capillary blood gases to document acidosis. If capillary gases and betahydroxybutyrate levels are not available, use blood ketone strips to determine level of betahydroxybutyrate. If >3, DKA is probable. If blood gases are not available in your center use a urine dipstick (Ketostiks) to determine the level of ketosis. Arterial blood gases are not recommended in young children.

Treatment goals:
3.6 The goals of treatment are to:
   - restore perfusion, which will increase glucose utilization in the periphery and reverse the progressive acidosis.
   - stop ketogenesis by administering insulin, which will reverse proteolysis and lipolysis, stimulate glucose uptake and processing; normalizing blood glucose concentration.
   - correct electrolyte imbalances.
   - avoid the complications of treatment insofar as possible, including cerebral edema, hypoglycemia and hypokalemia and hypophosphatemia.
3.7 These goals must be met in a manner that prevents a rapid decrease in the serum osmolarity. Treatment usually requires:
   • meticulous attention to the patient’s response to therapy
   • careful observation of glycemia to prevent hypoglycemia which may occur abruptly as fluid and insulin therapy are initiated and insulin resistance resolves.
   • admission to an inpatient setting

**Insulin**

3.8 Insulin bolus is **NOT** recommended in DKA in children

3.9 Start an insulin infusion 1 hour after IV fluids are initiated

3.10 Initial Insulin dose: **0.1 units/kg/hr** per continuous infusion

3.11 If rate of BG drop is greater than 5 mmol/L/hr **AND** dextrose in IV has been optimized, decrease insulin infusion to 0.05 units/kg/hr

**IV Fluids and Hydration:**

3.12 **Background:**
With insulin deficiency, hyperglycemia causes an obligate diuresis and urinary losses of water and electrolytes (sodium, potassium, chloride), resulting in extracellular fluid volume (ECFV) depletion. Although DKA is a medical emergency, few children in DKA require a rapid infusion of fluids or “bolus” to resuscitate from a hypotensive or shock state. If required, bolus fluids should be given for resuscitation and cardiovascular stabilization. Once the patient is no longer in shock/hypotensive state, fluids are given cautiously to avoid excess fluid and cerebral edema.

3.13 **Initial Fluid Resuscitation**

3.13.1 Purpose: Administer as needed for SHOCK and/or cardiovascular instability.  
* (e.g. poor pulses and perfusion, postural hypotension)

3.13.2 Volume and rate:  
Administer **20 mL/kg over 20-30 minutes**. Repeat once if vascular status still compromised.

3.13.3 Fluid type  
Administer **0.9% sodium chloride** (normal saline) for initial fluid management as children are depleted in both Na and free water.  
**NOTE:** Please see the Fluid worksheet on reverse of Standard Order Sheet:  
**NOTE:** subtract any initial fluids administered to treat cardiovascular instability from the fluids to be administered to correct the total calculated fluid depletion and provide maintenance.
3.14 **Hourly Fluid Rate:**

3.14.1 The intravenous fluid rate **SHOULD NOT** exceed 2X maintenance rate

3.14.2 To calculate the rate: **Please see reverse on Standard Order Sheet**

**Calculation:**

Fluids to correct Volume Depletion plus ongoing losses minus fluid given as bolus for resuscitation

3.14.3 **To calculate volume depletion:**

3% volume depletion= 30 mL/Kg:
Most children in mild DKA will be 3%-6% volume depleted

6% volume depletion= 60 mL /Kg: Children with dry mucous membranes, tachycardia.

9% volume depletion= 90 mL /Kg: Children who have had significant weight loss, Kussmaul breathing, altered LOC, tachycardia, sunken eyes.

**Administer remaining fluid deficit (minus fluid bolus) over 48 hours**

**NOTE:** dehydration in the range of 5 to 10% is commonly observed. Rapid, deep mouth breathing (Kussmaul respiration) often dries out the oral mucous membranes, which makes the child appear more dehydrated than he/she really is. Other clinical signs are more accurate, including documented weight loss, tachycardia, poor peripheral perfusion (cool extremities, thready pulses, delayed capillary refill), sunken eyes, and reduced skin turgor.

3.14.4 **Calculation of ongoing losses**

4 mL /Kg/hr: First 10 kg of body weight
2 mL /Kg/hr: Next 10 kg of body weight
1 mL /Kg/hr: Remainder of kg of body weight

**NOTE:** This applies to children with underlying normal renal, cardiac and neurologic status. If known abnormalities exist, the "maintenance" calculation may not be appropriate
3.15 **Dextrose in IV fluids**
Dextrose is added to IV fluids to prevent a rapid drop in serum osmolarity with hydration and insulin therapy. Rapid shifts in osmolarity are associated with rapid intracellular fluid shifts, potentially increasing the risk of neurologic complications.

3.15.1 Add dextrose to normal saline if:
- Blood Glucose drops below 17 mmol/L and/or
- Blood Glucose drops ≥ 5mmol/L/hr

3.15.2 Dextrose may be added to normal saline in the forms of D5W, D10W or D12.5W as required to keep BG 10 to 15 mmol/L.

3.15.3 * IV Dextrose should be maximized PRIOR to decreasing the insulin infusion.

3.15.4 Effective Osmolarity: (2x Na + BG)

3.16 **Sodium concentration in IV fluid**

3.16.1 If corrected plasma Na is ≤ 140 mmol/L or falling, 0.9% sodium chloride should be continued.

3.16.2 If corrected plasma Na is 140-150 mmol/L, stable (4 to 6 hours) switch to 0.45% sodium chloride.

3.16.3 If corrected plasma Na is ≥ 150 mmol/L, continue 0.9% sodium chloride for 10 to 12 hours until Na is stable or decreasing.

3.16.4 True Serum Na: Na + [0.3 (BG - 5)]

**Bicarbonate:**

3.17 Administration of bicarbonate is not recommended

3.18 Acidosis in DKA is due to both ketone bodies and lactic acid. Acidosis related to DKA resolves with fluid administration and insulin replacement.

3.19 Bicarbonate has a number of deleterious effects, including hypokalemia, metabolic alkalosis, and delayed clearance of ketones

**Potassium:**

3.20 Serum Potassium (K) should be monitored as per standard order sheet (see PHOR387)

3.21 Levels are usually normal at diagnosis but drop rapidly after insulin therapy is initiated due to intracellular shifting of K. A child with a serum K < 4.0 mmol/L who has had urine output and has normal renal function should be started on K replacement by adding K to the IV fluids as below (3.22). * A K bolus is not routinely required in children with DKA.

3.22 Potassium Chloride (KCl) at a rate of 5 mmol/kg over 24 hours, should be added to the IV fluids only once urine output has been established. The approximate concentration of potassium chloride in the IV fluids will be 20 to-40 mmol/L.

3.23 IV additives should be carefully titrated to achieve/maintain normokalemia.
**Monitoring: Laboratory Parameters**
As per physicians standard order sheet (PHOR387)
Call to notify the clinical chemistry laboratory that you are admitting a patient in DKA and will require STAT bloodwork

**Monitoring: Clinical Parameters**
As per physicians order sheet (PHOR387)

Other investigations as clinically indicated:

3.24 Imaging studies
Obtain studies appropriate for suspected cerebral edema

3.25 ECG monitoring
in severe DKA to assess T-waves;
Causes of cardiac dysrhythmia may include hyperkalemia, hypokalemia and hypocalcemia

3.26 Investigate for infection if indicated:
blood cultures
chest x-ray
urinalysis/urine C & S
lumbar puncture if not contraindicated
other cultures as history and physical exam suggest

**Potential Complications of initial Treatment and Prevention**

3.27 Cerebral edema:
Highest Risk Associated With:
New Diagnosis of diabetes
Age ≤ 5 years
Initial pH ≤ 7.1 or pCO2 ≤ 18

3.28 Rapid Fluid Shifts with Treatment Initiation:
Associated with:
Use of insulin in the first 1 to 2 hours of therapy
Over hydration/rapid correction of hyperglycemia
use of bicarbonate

3.29 Hyperchloremic acidosis:
Associated with the use of normal saline fluid
Monitor/Treat by:
Follow serum Cl measurements
Anticipate need to decrease NaCl in intravenous fluid
3.30 Hypoglycemia:
Causes include increasing sensitivity to exogenous insulin with correction of acidosis and insufficient serum glucose for insulin to metabolize
Anticipate a decrease in blood sugar with insulin initiation
Anticipate need for Dextrose in IV

3.31 Hypokalemia:
Causes include total body potassium depletion from renal wasting (polyuria) and intracellular shifting of potassium with insulin therapy.
Anticipate decrease in serum K with insulin therapy.
Serum potassium begins to reflect actual total body potassium depletion as volume depletion and acidosis resolve
Administer KCL 20-40 mmol/L in IV once urine output established.

5. References

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