

# BC CHILDREN'S HOSPITAL DIABETIC KETOACIDOSIS PROTOCOL<sup>A</sup>

FOR CHILDREN UP TO AGE 19 YEARS

THIS PROTOCOL IS ALSO AVAILABLE IN FILLABLE PDF FORMAT



ON ADMISSION

0. Confirm DKA: plasma glucose (PG)  $\geq 11$  mmol/L, ketones, capillary pH  $\leq 7.3$ ,  $\text{HCO}_3^- \leq 15$  mmol/L.<sup>B</sup>
1. Measure body weight (BW) in kilograms .....(1) \_\_\_\_\_ kg
2. Establish extent of dehydration ( $\downarrow$  BP, tears, skin turgor, capillary refill;  $\uparrow$  hematocrit) in mL/kg:<sup>C</sup>

	infants:	children:	
• mild:	5% = 50 mL/kg	3% = 30 mL/kg	
• moderate:	10% = 100 mL/kg	6% = 60 mL/kg	
• severe:	15% = 150 mL/kg	9% = 90 mL/kg	.....(2) _____ mL/kg
3. Calculate total fluid deficit: multiply (1)  $\times$  (2) .....(3) \_\_\_\_\_ mL
4. Give normal saline (NS) resuscitation bolus **only if patient is orthostatic or shocky**:<sup>D</sup>
  - recommended amount: 5–10 mL/kg BW over 1–2 hours, max  $< 30$  mL/kg .(4) \_\_\_\_\_ mL

TIME = 0–120 MIN

5. Calculate remainder of fluid deficit after fluid bolus: subtract (4) from (3) .....(5) \_\_\_\_\_ mL
6. Calculate maintenance fluid requirements for the next **48 hours**:<sup>E</sup>
  - $\rightarrow$  200 mL/kg for the first 10 kg BW
  - + 100 mL/kg for the next 10 kg BW
  - + 40 mL/kg for the rest of BW .....(6) \_\_\_\_\_ mL/48 h
7. Calculate total amount of fluid still to be given over 48 hours: add (5) and (6) .....(7) \_\_\_\_\_ mL/48 h
8. Calculate hourly rate of fluid replacement: divide (7) by 48 .....(8) \_\_\_\_\_ mL/h
9. Use **normal saline** (NS) as initial replacement fluid, at rate determined in (8). Add KCl 20–40 mEq/L only if hypokalemic and patient has adequate urine output. Continue this for 1–2 hours.

TIME = 60–120 MIN

10. After 1–2 hours, make up and start a piggyback insulin drip at 0.05–0.1 units/kg BW/h:<sup>F</sup>
  - 50 units insulin regular (Humulin® R or Novolin® Toronto) in 500 mL NS or D10/NS
  - run at 0.5–1.0 mL/kg BW/h .....(10) \_\_\_\_\_ mL/h
11. Begin “2-bag method” to replace NS<sup>G</sup>. Y together (a) NS with 40 mEq/L KCl and (b) D10–D12.5/NS with 40 mEq/L KCl. Decrease replacement fluid rate to adjust for insulin drip rate: subtract (10) from (8) .....(11) \_\_\_\_\_ mL/h
12. Aim to keep PG  $\sim 10$ –15 mmol/L by titrating the rates of these two solutions, keeping the combined rate at (11)<sup>G</sup>. Continue this for the next 6–12 hours, monitoring as below in (15) and (16).

## Rationale & Notes:

<sup>A</sup>Please note that this protocol is designed as an algorithm for treating the majority of cases of DKA in infants, children and adolescents. **It cannot replace careful clinical observation and judgment in treating this potentially very serious condition.** If you have questions or problems related to the management of DKA or diabetes, please feel free to contact the BCCH Pediatric Endocrinologist on call.

<sup>B</sup>Hyperglycemic hyperosmolar syndrome should be suspected when there is significant hyperglycemia ( $> 33$  mmol/L) and hyperosmolality ( $> 330$  mOsm/L) without ketosis or acidosis (bicarbonate  $> 15$  mmol/L). A mixed picture of DKA and HHS is possible. Mild hyperglycemia, even with ketones and mild acidosis, can often be managed without IV fluids or IV insulin.

<sup>C</sup>Rapid, deep mouth-breathing (Kussmaul respiration) often dries out the oral mucosa, making the child appear more dehydrated than s/he really is. The hematocrit and other clinical signs noted are more accurate.

<sup>D</sup>Large fluid boluses are potentially dangerous<sup>L</sup> and should be administered with caution, unless the patient is truly shocky. Only very rarely will a larger ( $> 20$  mL/kg BW) fluid bolus will be required to maintain perfusion.

<sup>E</sup>Since most patients develop DKA over days, slow metabolic repair is safest. Overhydration may contribute to cerebral edema.<sup>L</sup> Nonetheless, DKA in children often resolves in less than 48 h.

<sup>F</sup>IV insulin boluses are always contraindicated. Insulin given in the first 1–2 h of DKA repair is thought to increase mortality. This insulin rate fully inhibits ketogenesis and gluconeogenesis and should be maintained if possible. If unable to keep PG  $> 10$  mmol/L<sup>G</sup>, drop the insulin rate by 25–50%.

13. Re-evaluate appropriateness of replacement fluid type frequently, anticipating the need to add or increase  $\text{Na}^+$ ,  $\text{K}^+$ , dextrose, etc.
- dextrose<sup>G</sup>: aim to keep the PG ~10–15 mmol/L range
  - sodium<sup>H</sup>: corrected  $\text{Na}^+$  <140 mmol/L or falling: continue NS  
corrected  $\text{Na}^+$  140–150, stable: switch to ½NS after 4–6 h  
corrected  $\text{Na}^+$  >150, stable: switch to ½NS after 10–12 h
  - potassium<sup>I,J</sup>: patient urinating: continue KCl 20–40 mmol/L  
may give 50% of  $\text{K}^+$  as acetate or phosphate
  - bicarbonate<sup>K</sup>:  $\text{NaHCO}_3$  is **not** generally recommended
14. Close neurological observation and frequent rousing of the child with finger-pokes to detect any changes consistent with cerebral edema. Follow Glasgow Coma Scale. Severe headache, change in sensorium or BP, dilated pupils, bradycardia, irregular breathing, posturing and incontinence are signs of impending deterioration. Rapid intervention is imperative:
- airway / breathing / circulation
  - elevate head of bed
  - decrease fluid rate by one-third
  - mannitol (0.5–1 g/kg IV over 20 min) or 3% NaCl (5–10 mL/kg IV over 30 min)<sup>L</sup>
  - consider intubation and mild hyperventilation (keep  $\text{pCO}_2$  >22 mg Hg) for impending respiratory failure
  - arrange CT when stable
15. Follow laboratory parameters (use of a flowsheet is highly recommended):
- follow PG by meter every 30–60 min<sup>G</sup>: does child respond to the poke?
  - follow  $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Cl}^-$ ,  $\text{HCO}_3^-$ , anion gap, urea, creatinine, capillary pH every 2–4 hours<sup>H,I,K</sup>;  $\text{Ca}^{2+}$ ,  $\text{Mg}^{2+}$  and  $\text{P}_i$  every 2–4 hours if giving phosphate<sup>J</sup>
  - follow (preferably) plasma  $\beta$ -hydroxybutyrate every 2–4 hours or urine ketones with each void
16. Re-evaluate appropriateness of replacement fluid type frequently, anticipating the need to increase or decrease  $\text{Na}^+$ ,  $\text{K}^+$ , dextrose, etc.

Accompanying documents on our [website](#):

- [DKA Flowsheet](#)
- [DKA Sample Physician Order Sheet](#)
- [DKA Nursing Protocol](#) (including the “two-bag” method)
- [Pharmacy Recipes for Making DKA Solutions](#)

<sup>G</sup>Keeping the PG in the ~10–15 mmol/L range allows for a buffer against hypoglycemia and a too-rapid fall in plasma osmolality<sup>H</sup>. The “two-bag method” (see our [DKA Nursing Protocol](#)) is a handy way to adjust the glucose without altering the  $\text{Na}^+$  or  $\text{K}^+$  delivery. It also allows for a faster response to PG changes, and it decreases nursing and pharmacy costs and time.

<sup>H</sup>The introduction of hypotonic fluids must be considered carefully. The corrected  $\text{Na}^+$  should be calculated and followed closely: corrected  $\text{Na}^+$  = [measured  $\text{Na}^+$  + 0.36×(PG–5.6)]. If corrected  $\text{Na}^+$  falls or fails to rise as the PG falls, this could indicate excess free-water administration. It is also helpful to monitor the active osmolality [PG + 2×( $\text{Na}^+$  +  $\text{K}^+$ )], which should not fall >0.5 mOsm/kg/h. If the corrected sodium is 140–150 mmol/L and stable and the active osmolality has been dropping slowly, switching to ½NS can be considered after 4–6 h. An elevated measured  $\text{Na}^+$  in the face of hyperglycemia indicates severe dehydration and an element of the hyperglycemic hyperosmolar state. Such patients should be rehydrated with extreme caution, using fluids with higher osmolar content (e.g. NS) for longer time periods (10–12 h).

<sup>I</sup>Serum  $\text{K}^+$  levels are usually normal at diagnosis and fall precipitously with treatment. An IV fluid containing 20–40 mmol/L  $\text{K}^+$  is usually required to keep the serum  $\text{K}^+$  >3.0 mmol/L. Begin  $\text{K}^+$  and insulin together. Oral/nasogastric KCl boluses (0.5–1 mmol/kg BW) may also be administered.

<sup>J</sup>While there is no proven benefit to using potassium phosphate or acetate, it does have the theoretical advantage of repleting the severe phosphate deficit of DKA and/or ameliorating the hyperchloremia which inevitably occurs during DKA treatment. If phosphate is given, serum calcium, magnesium and phosphate levels should be monitored closely.

<sup>K</sup>The acidosis of DKA is due to both ketoacids and lactic acid, and these resolve with fluid and insulin replacement. There is no evidence that  $\text{NaHCO}_3$  is either necessary or safe in DKA, but it does have a number of deleterious effects: paradoxical CNS acidosis, hypokalemia, hyperosmolality, delayed clearance of ketones, and cerebral edema.  $\text{NaHCO}_3$  in DKA should only be considered if pH <6.9 or cardiac failure.

<sup>L</sup>Subclinical brain swelling is common in children with DKA. Cerebral edema (CE) accounts for more than half of the ~1–5% mortality rate of DKA in children. At highest risk are newly diagnosed patients, those aged <5 years, and those with initial pH <7.1 or  $\text{pCO}_2$  <18. The etiology of CE remains unclear, but aggressive hydration has been implicated in several studies. Resuscitation is successful in only 50% of cases. Most experts suggest limiting fluids to <4 L/m<sup>2</sup> body surface area, or to <2.5× maintenance fluid rate in the first 24 h, and to <50 mL/kg in the first 4 h.

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