

BC CHILDREN'S HOSPITAL DIABETIC KETOACIDOSIS PROTOCOL^A

FOR CHILDREN AGES 1 MONTH TO 19 YEARS

THIS PROTOCOL IS ALSO AVAILABLE IN [NON-FILLABLE PDF FORMAT](#)

FIRST 30 MIN

0. ABCs, vital signs (with BP), neurovital signs. Place large-bore IV. Draw labs. Confirm DKA: blood glucose ≥ 11.1 mmol/L, moderate–large ketonuria ($\geq 2+$) or plasma β -hydroxybutyrate ≥ 3.0 mmol/L, and venous pH < 7.3 or plasma $\text{HCO}_3^- < 18$ mmol/L.^C Consider possibility of an element of hyperglycemic hyperosmolar state.^B

1. Measure body weight in kilograms(1) _____ kg

2. Give 0.9% saline (normal saline, NS) resuscitation bolus^D
• recommended amount: 20 mL/kg over 30 min, max 1000 mL.....(2) _____ mL

3. Give rapid repeated boluses of NS if persistent tachycardia, prolonged cap refill (> 2 sec), cool extremities:
• 20 mL/kg boluses, repeated until stable; total **extra** given:(3) _____ mL

4. Begin rehydration, calculated for even correction over 36 hours, based on admission body weight:^E
• 5–10 kg: 6.5 mL/kg/h
• 10–20 kg: 6 mL/kg/h
• 20–40 kg: 5 mL/kg/h
• > 40 kg: 4 mL/kg/h, maximum 500 mL/h(4) _____ mL/kg/h

5. Calculate **total** hourly fluid rate to be given for 36 hours: multiply (1) and (4).....(5) _____ mL/h

6. Use NS with KCl 40 mEq/L (Bag A) as initial rehydration fluid, at rate determined in (5), ensuring that patient has voided and has a plasma $\text{K}^+ \leq 5.5$ mmol/L before adding potassium to the IV fluids.

7. At 60–120 minutes after starting the first fluid bolus, make up and start a piggyback insulin drip at 0.05–0.1 units/kg/h (Bag C). Ensure plasma $\text{K}^+ \geq 3.5$ mmol/L first. Consider 0.5 mL/kg/h if pH > 7.15 .^F
• 50 units insulin regular (Humulin® R or Novolin® Toronto) in 500 mL NS or D10/NS
• run at 0.5–1 mL/kg BW/h(7) _____ mL/h

8. Begin “2-bag method”^G. Y together (Bag A) NS with 40 mEq/L KCl and (Bag B) D10–D12.5/NS with 40 mEq/L KCl. Decrease replacement fluid rate to adjust for insulin drip rate:
subtract (7) from (5).....(8) _____ mL/h

9. Aim to keep PG ~ 8 –12 mmol/L by titrating the rates of these two solutions, keeping the combined rate at (8)^G. Continue this for the next 6–12 hours, monitoring as below.

10. At 4–6 hours after initial fluids and if corrected plasma Na^+ is ≥ 145 mmol/L, stable or increasing, switch Bag A to 0.45% saline w/ 40 mEq/L KCl and Bag B to D10–D12.5/0.45% saline w/ 40 mEq/L KCl at the rate as in (8)^H.

TIME = 30 MIN–36 HOURS

Rationale & Notes:

^APlease 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, please feel free to contact the management of DKA, please feel free to contact the BCCH Pediatric Endocrinologist on call.

^BHyperglycemic hyperosmolar state (HHS) should be suspected when there is significant hyperglycemia (> 33 mmol/L) and hyper-osmolality (> 330 mOsm/L) without ketosis or acidosis (bicarbonate > 15 mmol/L, venous pH > 7.3). 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.

^CRapid, deep mouth-breathing (Kussmaul respiration) often dries out the oral mucosa, making the child appear more dehydrated than they really are. The hematocrit and other clinical signs (decreased capillary refill) are more accurate measures of dehydration.

^DRecent research shows that most children with moderate–severe DKA will require a 20 mL/kg resuscitation fluid bolus to restore perfusion, prior to the rehydration phase.

^ERecent research shows that DKA can be safely corrected over a 24- to 48-h period. This protocol is designed to correct a 10% fluid deficit (100 mL/kg) evenly over 36 h.

^FIV 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 > 8 mmol/L^G, drop the insulin rate by 25–50%. Consider 0.05 U/kg/h for pH > 7.15 . Ensure plasma $\text{K}^+ \geq 3.5$ mmol/L before starting insulin.

11. Re-evaluate appropriateness of replacement fluid type frequently, anticipating the need to add or increase Na⁺, K⁺, dextrose, etc.
 - dextrose^G: aim to keep the PG ~8–12 mmol/L range
 - sodium^H: corrected Na⁺ <145 mmol/L, or falling regardless of level: continue NS
corrected Na⁺ ≥145, stable or increasing, switch to ½NS after 4–6 h
 - potassium^{I,J}: patient urinating and K⁺ remains ≤5.5: continue KCl 40 mmol/L may give 50% of K⁺ as phosphate
 - bicarbonate^K: NaHCO₃ is **not** generally recommended
12. Children with DKA have high risk for acute kidney injury (AKI). Use Schwartz formula to calculate expected baseline creatinine (EBC).^L
13. 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 all fluid bags to 5 mL/h pending physician reassessment
 - mannitol 20% (0.5–1 g/kg, 2.5–5 mL/kg IV over 15 min) or NaCl 3% (2.5–5 mL/kg IV over 15 min)^M
 - consider intubation and mild hyperventilation (avoid hypocapnia) for impending respiratory failure
 - arrange CT when stable
14. Follow laboratory parameters (use of a flowsheet is highly recommended):
 - follow PG by meter every 30–60 min^G: does child respond to the poke?
 - follow Na⁺, K⁺, Cl⁻, HCO₃⁻, anion gap, urea, creatinine, venous pH every 2–4 hours^{H, I, K}; Ca²⁺, Mg²⁺ and P_i every 2–4 hours if giving phosphate^J
 - follow (preferably) plasma β-hydroxybutyrate every 2–4 hours or urine ketones with each void
15. Re-evaluate appropriateness of replacement fluid type frequently, anticipating the need to increase or decrease Na⁺, K⁺, dextrose, etc.

^GKeeping the PG in the ~8–12 mmol/L range allows for a buffer against hypoglycemia and a too-rapid fall in plasma osmolality^H. The “two-bag method” (see our [DKA Nursing Protocol](#)) is a handy way to adjust the glucose without altering the Na⁺ or K⁺ delivery. It also allows for a faster response to PG changes, and it decreases nursing and pharmacy workload and costs.

^HAt some point, the patient will require a switch to the use of hypotonic fluids. The corrected Na⁺ should be calculated and followed closely: corrected Na⁺ = [measured Na⁺ + 0.36×(PG–5.6)]. It is also helpful to monitor the active osmolality [PG + 2×(Na⁺ + K⁺)], which should not fall >0.5 mOsm/kg/h. If the corrected sodium is ≥140 mmol/L and stable or rising and the active osmolality has been dropping slowly, switching to ½NS should be considered after 4–6 h of fluids. An elevated measured Na⁺ in the face of hyperglycemia indicates severe dehydration and an element of the hyperglycemic hyperosmolar state. Such patients should be rehydrated using fluids with higher osmolar content (e.g. NS) for longer time periods (10–12 h).

^ISerum K⁺ levels are usually normal at diagnosis and fall precipitously with treatment. An IV fluid containing 20–40 mmol/L K⁺ is usually required to keep the serum K⁺ ≥3.5 mmol/L. Begin K⁺ and insulin together. Oral/nasogastric KCl boluses (0.5–1 mmol/kg BW) may also be administered.

^JWhile there is no proven benefit to using potassium phosphate, it does have the theoretical advantage of repleting the severe phosphate deficit of DKA and/or ameliorating hyperchloremia which inevitably occurs during DKA treatment. A phosphate <0.32 mmol/L should be treated. If phosphate is given, serum calcium, magnesium and phosphate levels should be monitored closely.

^KThe acidosis of DKA is due to both ketoacids and lactic acid, and these resolve with fluid and insulin replacement. There is no evidence that NaHCO₃ is either necessary or safe in DKA, but its use has a number of deleterious effects: paradoxical CNS acidosis, hypokalemia, hyperosmolality, delayed clearance of ketones, and cerebral edema. NaHCO₃ in DKA should only be considered if pH <6.9 or cardiac failure.

^LEBC (μmol/L) = 36.5 × height (cm)/120. Measured creatinine 1.5–1.99× EBC = Stage 1, 2–2.99× EBC = Stage 2, ≥3× EBC = Stage 3 AKI.

^MSubclinical 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 pCO₂ <18. The exact etiology of CE remains unclear. Resuscitation is successful in only 50% of cases.

Accompanying documents on our [website](#):

- [DKA Flowsheet](#) and [DKA Sample Physician Order Sheet](#)
- [DKA Glucose, Fluid and Insulin Management](#)
- [DKA Nursing Protocol](#) (including the “two-bag” method)
- [DKA Recipes for Making Solutions](#)