

## Practice Point

# Current recommendations for management of paediatric diabetic ketoacidosis

Karen E. Gripp MD, Evelyne D. Trottier MD, Sidd Thakore MD, Jonathan Sniderman MD, Sarah Lawrence MD

Canadian Paediatric Society, Acute Care Committee, Ottawa, Ontario, Canada

Correspondence: Canadian Paediatric Society, 100–2305 St Laurent Blvd, Ottawa, Ontario K1G 4J8, Canada.  
Tel: 613-526-9397, fax: 613-526-3332, e-mail: [info@cps.ca](mailto:info@cps.ca), website: [www.cps.ca](http://www.cps.ca)

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### ABSTRACT

Treatment of paediatric diabetic ketoacidosis (DKA) includes careful attention to fluids and electrolytes to minimize the risk of complications such as cerebral injury (CI), which is associated with high morbidity and mortality. The incidence of cerebral edema in paediatric DKA has not decreased despite the use of fluid-limiting protocols based on restricting early fluid resuscitation. New evidence suggests that early isotonic fluid therapy does not confer additional risk and may improve outcomes in some patients. Protocols and clinical practice guidelines are being adjusted, with a particular focus on recommendations for initial and ongoing fluids and electrolyte monitoring and replacement. Initial isotonic fluid resuscitation is now recommended for all patients in the first 20 to 30 minutes after presentation, followed by repletion of volume deficit over 36 hours in association with an insulin infusion, electrolyte supplementation, and careful monitoring for and management of potential CI.

**Keywords:** Cerebral edema; Cerebral injury; Diabetes mellitus; Diabetic ketoacidosis; Paediatric.

### BACKGROUND

Diabetic ketoacidosis (DKA) is defined by the presence of hyperglycemia, ketosis, and acidosis as measured by serum pH or bicarbonate (Table 1) (1,2). DKA can occur in any patient with an absolute or relative insulin deficiency. Risk factors for DKA include younger age, lower socioeconomic status, and delayed diagnosis in new patients. For children and youth with known diabetes, additional risk factors include previous DKA, poor glycemic control, unrecognized insulin pump malfunction, infection, some medications (e.g., long-acting insulin analogues, atypical antipsychotics, glucocorticoids), ethnicity, limited access to care, coexisting mental health or social and family issues, peripubertal stage, and adolescence (1,3).

Additional synoptic guidance for initial and ongoing management of DKA can be found at these websites:

- [Translating Emergency Knowledge for Kids \(https://trekk.ca/\)](https://trekk.ca/)
- [Canadian Pediatric Endocrine Group \(https://cpeg-gcep.net/\)](https://cpeg-gcep.net/)

### CLINICAL PRESENTATION AND DIAGNOSIS

In DKA, hyperglycemia leads to urinary losses of both water and electrolytes, resulting in volume depletion and metabolic disturbances. Low insulin levels reduce glucose utilization, and subsequent cellular glycopenia triggers increased glucagon release, lipolysis, and oxidation of free fatty acids, with ensuing ketoacidosis (4,5). Serum potassium may be normal or elevated due to extracellular shifts, but total body potassium is invariably low due to osmotic diuresis and active urinary excretion. DKA should be differentiated from hyperosmolar hyperglycemic state, which is characterized by more severe volume depletion and extreme electrolyte imbalances in the absence of significant ketosis and acidosis.

Presenting symptoms of DKA may include polyuria, polydipsia, polyphagia, weakness, nausea, vomiting, abdominal pain, decreased level of consciousness, Kussmaul breathing, and acetone breath.

Initial clinical assessment should include ABCDs and evaluation of tachypnea and altered breathing patterns, perfusion, fluid balance, and level of consciousness. Further evaluation

should solicit history of precipitating factors (e.g., infection, intoxication) and medication adherence for individuals with known diabetes. Assessing volume depletion can be difficult, with substantial inter-rater variability (6–8). For fluid calculations, it is suggested to use a minimum of 5% depletion for a patient with mild DKA, or 7% to 10% for more severe DKA (1).

Laboratory evaluation should include plasma glucose, electrolytes (including calcium, magnesium, and phosphate), urea, creatinine, anion gap, blood gas, osmolality, and serum/urine ketones to determine the severity of DKA (Table 2). A beta-hydroxybutyrate (BHB) level should be measured, if available. Blood BHB correlates better with changes in pH and blood bicarbonate than urine ketones, which can be falsely negative. An improved BHB level may indicate that persistent acidosis is due to hyperchloremia. The need for additional studies will be based on clinical circumstances (e.g., hemoglobin A1c, evaluation for infection, electrocardiogram if serum potassium is elevated).

### CEREBRAL INJURY IN DKA

Cerebral injury (CI) occurs more commonly in children than adults (3,4,9–12) (Table 3). The frequency of clinically significant CI may be as high as 1%, and those individuals experience a morbidity between 21% and 25% and a mortality between 21% and 24% (3,13). Clinical CI is more frequent in severe DKA (9–11) and may be present before treatment is initiated (10,14,15) (Table 4). Although patients with DKA present with volume depletion, intravenous (IV) fluids have traditionally been restricted in paediatric patients out of concern for potentiating CI (16).

As an alternative to the traditional osmotic hypothesis of CI, cerebral hypoperfusion and reperfusion likely play a significant role in DKA-related CI. Several levels of evidence, including computed tomography (CT), magnetic resonance imaging, and cerebral blood flow studies, have shown changes in biochemical markers and progression from cytotoxic to vasogenic edema (17–19).

### MANAGEMENT RECOMMENDATIONS

Treatment of DKA includes progressive volume expansion and a careful reduction in plasma glucose. The diagnosis of DKA

**Table 1.** Triad of laboratory findings in DKA

- Hyperglycemia (>11 mmol/L)
- Serum ketosis (beta-hydroxybutyrate  $\geq$ 3 mmol/L) and/or ketonuria (moderate or large)
- Acidosis (pH <7.3 or measured bicarbonate <18 mmol/L) with an anion gap >12

Data taken from references (1,2).

**Table 2.** Determination of severity of DKA

DKA severity	Acidosis	Bicarbonate
Mild	pH 7.2–7.29	10 mmol/L to <18 mmol/L
Moderate	pH 7.1–7.19	5 mmol/L to 9 mmol/L
Severe	pH <7.1	<5 mmol/L

**Table 3.** Risk factors for cerebral injury

- New-onset diabetes
- Longer duration of symptoms
- Young age (<5 years)
- Severe acidosis (pH <7.1 or HCO<sub>3</sub> <5)
- Laboratory evidence of severe dehydration (elevated urea, hematocrit)
- Hypocapnia (pCO<sub>2</sub> <21)
- Insulin therapy in first hour of management and/or insulin bolus
- Rapid administration of hypotonic fluids
- Use of sodium bicarbonate
- Failure of measured sodium to rise during treatment

Data taken from references (3,4,9–12)

**Table 4.** Warning signs of cerebral injury

- Altered level of consciousness, especially after initial improvement
- Headache, particularly if severe, worsening, or commencing after treatment onset
- Irritability in young children
- Vomiting
- Urinary incontinence
- Hypertension (may be diastolic)
- Bradycardia (unrelated to sleep or improved vascular volume)
- Respiratory depression or oxygen desaturation
- Cranial nerve palsies

Data taken from reference (1).

should be confirmed (Table 1) before initiating any interventions. It is important to remember that DKA in children is managed differently than in adults. The differences to consider in paediatric patients include (3,4):

- Extra caution when administering IV fluids
- Initiating insulin only after IV fluids
- Replacing potassium earlier and more aggressively
- Avoiding insulin boluses and sodium bicarbonate

Goals for management are to:

- Correct volume depletion
- Correct acidosis
- Stop ketogenesis
- Correct electrolyte imbalances
- Restore normal blood glucose
- Monitor for and prevent complications (CI, hypoglycemia, symptomatic electrolyte deficiencies, hyperchloremic acidosis)
- Manage coexistent illness or precipitating factors

### Fluids

All patients with DKA require careful monitoring and attention to fluid administration, particularly children and youth at higher

risk for CI. In paediatric patients without clinical symptoms of CI, no harm has been demonstrated by using more liberal initial fluid resuscitation, with the primary goal of improving tissue perfusion (13,20,21).

Both saline and balanced crystalloids are appropriate for use as IV fluids in DKA. Balanced crystalloids (e.g., Ringer’s lactate, Plasmalyte) are recognized as safe alternatives to saline for both bolus and ongoing infusions and may minimize hyperchloremic metabolic acidosis, as well as potentially reduce CI and renal injury (22–25). While ongoing therapy with 0.45% NaCl with dextrose and added potassium can be safe for most patients with DKA and a normal neurologic status, hypotonic fluids should be avoided in all patients with symptoms of CI.

Specific fluid recommendations for DKA include the following:

- Administer 10 mL/kg to 20 mL/kg (to a maximum 1,000 mL) of isotonic fluid with 0.9% NaCl or a balanced crystalloid for **all** patients over 20-30 minutes, regardless of hemodynamic status.
- In settings of hypotension or compensated shock, give a fluid bolus within 10 to 15 minutes and administer additional isotonic fluid rapidly, in 10 mL/kg increments to a maximum of 40 mL/kg, in consultation with a paediatric intensivist.
- After the initial isotonic fluid bolus(es), calculate a starting hourly fluid rate to incorporate both maintenance and deficit fluids, based on replacing an assumed 10% deficit over 36 hours (26) (Table 5). Administering twice the usual rate of maintenance fluids is generally safe until the detailed fluid calculation is completed. Changes in the hourly rate may be considered for patients with less severe volume depletion, and many can transition safely from IV to oral rehydration when ketoacidosis has resolved.
- Dextrose-free isotonic fluids should be continued with a goal of decreasing blood glucose by no more than 5 mmol/L/hour until the glucose level is between 15 mmol/L and 17 mmol/L. At this point, dextrose (usually 5%) should be added and adjusted to maintain a blood glucose of 7 mmol/L to 11 mmol/L. This range of blood glucose helps minimize glucosuria, which occurs when the glucose level exceeds 12 mmol/L.
- A two-bag protocol, with each bag containing the same amount of electrolytes but only one containing dextrose, can be used to quickly adjust administered dextrose concentration with finesse (and some cost saving) in response to blood glucose (Table 6) (27). The second bag should

**Table 5.** Calculation of total hourly IV fluid rate

Weight	mL/kg/h
5 to <10 kg	6.5
10 to <20 kg	6
20 to <40 kg	5
≥40 kg	4 (to a maximum 250 mL/h)

Data taken from reference (26).

(ideally) contain 12.5% dextrose to maximize peripheral venous delivery if needed, but 10% dextrose may also be used.

- Patients with mild DKA may be treated with reduced amounts of IV fluids or oral fluids if they are tolerating enteral intake.

### Insulin

Insulin should be started **only after the first hour** of fluid therapy (9) AND when potassium levels are >3.0 mmol/L, and never as an IV bolus. Replace potassium before starting insulin if potassium levels are less than or equal to 3.0 mmol/L.

- An infusion of rapid-acting insulin at 0.05 unit/kg/hour to 0.1 unit/kg/hour is preferred, without a weight-based maximum. However, intermittent subcutaneous insulin can be used if IV administration is not possible or in some cases of mild DKA, with the support of expert consultation.
- If blood glucose has been decreasing at a rate greater than 5 mmol/L/hour AND administration of IV dextrose has been maximized, reduce insulin infusion to 0.05 unit/kg/hour.
- A further gradual decrease to no less than 0.025 unit/kg/hour can be considered if blood glucose continues to fall rapidly, or as a bridge until the next mealtime (and associated subcutaneous insulin dose) if acidosis has corrected.

### Electrolytes

Patients with DKA present with a relative or total body deficiency of sodium, potassium, phosphate, and magnesium. Hyperglycemia results in factitious hyponatremia but measured Na can be used to calculate the initial anion gap (28). Assessment

**Table 6.** Two-bag protocol: Infusion rates to achieve desired dextrose concentration\*

Desired dextrose concentration	Infusion rate of bag #1	Infusion rate of bag #2
	(No dextrose)	(12.5% dextrose) <sup>†</sup>
No dextrose	Total hourly IV fluid rate × 1	Do not infuse
D5 (5%)	Total hourly IV fluid rate × 0.6	Total hourly IV fluid rate × 0.4
D7.5 (7.5%)	Total hourly IV fluid rate × 0.4	Total hourly IV fluid rate × 0.6
D10 (10%)	Total hourly IV fluid rate × 0.2	Total hourly IV fluid rate × 0.8
D12.5 (12.5%)	Do not infuse	Total hourly IV fluid rate × 1

\*Bag #1 and bag #2 should have the same constituents except for dextrose.

Recommended fluids include Ringer’s lactate, Plasmalyte, or 0.9% NaCl (may use 0.45% NaCl, if no concerns of cerebral injury), all with added KCl (40 mmol/L).

<sup>†</sup>If using 10% dextrose, a desired dextrose concentration of D5 can be obtained by using a factor of 0.5 for each bag. A desired concentration of D7.5 can be attained by multiplying the rate for bag #1 by 0.25 and bag #2 by 0.75.

and decision-making should be based on the corrected serum sodium (corrected sodium = measured sodium +  $[\{\text{glucose} - 5\} \times 0.3]$ ). An elevated corrected sodium indicates more severe volume depletion. Corrected sodium levels should be carefully monitored because a decrease (or increase) of more than 2 mmol/L/hour to 3 mmol/L/hour may indicate excessive (or inadequate) fluid resuscitation, with increased risk for CI or acute kidney injury (29,30).

DKA treatment lowers serum potassium levels, and supplemental potassium of at least 40 mmol/L should be added to IV fluids when measured potassium is  $<5$  mmol/L and after recent urine output is documented.

Serum phosphate may be normal or high despite a total body deficit. Insulin therapy will lower serum phosphate. Consider replacement if measured phosphate is below 0.5 mmol/L, or if there are concerns of cardiac dysfunction, respiratory failure, gastrointestinal dysmotility, or metabolic encephalopathy (31,32). Follow local protocols for phosphate administration.

Sodium bicarbonate should not be given as a treatment for metabolic acidosis because it increases the risk for CI (25). Adequate treatment with fluids and insulin is sufficient for the correction of acidosis, unless indicated as part of active cardiopulmonary resuscitation or symptomatic hyperkalemia.

### Monitoring and treatment of cerebral edema

Laboratory studies should be repeated every 2 hours at a minimum during insulin infusion, with measurement of glucose hourly. Additional monitoring includes neurologic status, volume status, and urine output, with added cardiac monitoring in the setting of severe DKA with potentially dangerous metabolic derangements of potassium or calcium.

Frequent reassessment of neurologic status is essential to identify and manage CI. If CI is suspected, do not wait for cranial imaging before initiating therapy. CI management should include:

- Minimizing patient movement and agitation
- Raising the head of bed to 30°
- Maintaining the patient's head in the midline position
- Administering isotonic fluids (change to isotonic if using 0.45% NaCl)
- Reducing rate of IV fluids to 75% of calculated hourly rate if sufficient to maintain adequate perfusion.
- Osmolar therapy, either 3% saline delivered at 5 mL/kg over 10 to 15 minutes (to a maximum 250 mL) or mannitol 0.5 g/kg to 1 g/kg (to a maximum 100 g) over 15 to 20 minutes (33,34)
- Urgent consultation with critical care

Intubation is best avoided. However, a patient's baseline hypoventilation must be maintained if there is a drop in respiratory drive and level of consciousness. Consultation with a paediatric intensivist is advised *before* intubation.

### DISPOSITION

While some mild DKA can be corrected in an emergency department setting, most paediatric patients with DKA should be admitted for ongoing monitoring and management supported by expert consultation. Any abnormal mental status in a child

or youth with DKA should prompt urgent referral to the nearest tertiary care centre.

A paediatric intensivist should be consulted for children with signs or symptoms of CI. Further considerations for consultation with critical care include more severe DKA with a pH  $<7.0$  or bicarbonate  $<5$  mmol/L and less severe cases in children under 5 years of age, especially when presenting outside of a tertiary care centre.

### SUMMARY

The management of DKA continues to evolve with new literature and understanding of both pathophysiology and optimal therapy. Treatment of DKA includes correction of volume depletion, acidosis, electrolyte imbalances, and hyperglycemia. Current evidence suggests that administering an initial 10 mL/kg to 20 mL/kg of isotonic IV fluid to paediatric DKA patients is both safe and desirable as part of established evidence-based guidelines for management.

Best practice points:

1. All patients should initially receive 10 mL/kg to 20 mL/kg isotonic fluid, either 0.9% saline or a balanced crystalloid.
2. Calculate ongoing fluids per TREKK guidelines over 36 hours, based on 10% dehydration.
3. Fluid administration via a two-bag system allows tighter glycemic control and less glucosuria.
4. Insulin should be given *only* after 1 hour of IV fluids and never as a bolus.
5. Follow corrected sodium levels to assess the adequacy of fluid replacement.
6. Replace potassium early and anticipate the need for replacing phosphate.
7. Avoid sodium bicarbonate.
8. Anticipate, monitor for, and manage cerebral injury.

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### POTENTIAL CONFLICTS OF INTEREST

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**Authors:** Karen E. Gripp MD, Evelyne D. Trottier MD, Sidd Thakore MD, Jonathan Sniderman MD, Sarah Lawrence MD