Diabetic ketoacidosis in children and adolescents: An update and revised treatment protocol

Standardized pediatric-specific treatment is required to ensure safe correction of metabolic derangements associated with DKA.
Diabetic ketoacidosis in children and adolescents: An update and revised treatment protocol

- Acidosis: venous pH < 7.3 and/or bicarbonate < 15 mmol/L.
- Ketosis: presence of ketones in the blood, urine, or both.

DKA cases can be roughly divided further into mild (pH 7.20–7.29, bicarbonate 10–14), moderate (pH 7.10–7.29, bicarbonate 5–9), and severe (pH < 7.10, bicarbonate < 5).

Endocrinologists may feel comfortable treating mild DKA in reliable patients with known T1D at home or as outpatients. The majority of children with moderate DKA and all children with severe DKA should be treated in a medical facility, optimally by a pediatric endocrinologist, pediatrician, or other practitioner familiar with the unique issues that arise in DKA in the young.

Clinically, children in DKA present with the following: dehydration of up to 10% of their body weight; Kussmaul respirations; nausea, vomiting, and abdominal pain; and a progressive decrease in consciousness.

A printer friendly patient information sheet is available at www.bcmj.org.

Cerebral edema: Pathophysiology and risk factors

The major mortality factor associated with DKA in infants, children, and adolescents is cerebral edema (DKA-CE). This phenomenon has been documented on cranial CT scans prior to treatment, and it has been observed in adults as well. Approximately 0.5% to 1.5% of children presenting with DKA will develop clinically evident DKA-CE. In a recent Canadian surveillance study, 23% of children developing DKA-CE died, and another 15% survived with neurological complications.

DKA-CE accounts for 70% to 80% of diabetes-related deaths in children under 12 years. Other causes of death include electrolyte disturbances, shock, cerebral venous thrombosis, and pulmonary edema.

The exact pathophysiology underlying DKA-CE remains unclear. The accumulation of water in the brain may be due to the presence of so-called idiogenic osmole, small organic compounds that are formed in the extracellular space as a defensive response to increasing osmolality in the extracellular compartment. When fluid resuscitation is initiated, the extracellular space becomes relatively hypotonic compared with the intracellular space, resulting in an influx of water into neurons. Activation of the sodium-hydrogen exchanger mechanism, perhaps by insulin, vasopressin, or both may also lead to an influx of sodium ions (followed by water) into brain cells. Finally, it is hypothesized that DKA-CE may be due to a vasogenic mechanism representing reperfusion of hypoperfused tissue. Taken together, these factors would suggest that a thoughtful approach is necessary in the administration of fluids, electrolytes, and insulin.

A number of pretreatment (Table 1) and treatment-associated (Table 2) risk factors for the development of DKA-CE have been described. In general, younger children presenting with new-onset diabetes who have had symptoms of long duration and increased severity, as well as more pronounced dehydration and acidosis, are at highest risk of developing DKA-CE. Alternatively, sicker-appearing children may be more likely to elicit anxiety in the medical team, leading to hasty treatment decisions. Treatment regimens associated with over-zealous administration of fluids have likewise been implicated in the development of DKA-CE in a number of studies.

Most recently, a case-control study in children from the UK demonstrated for the first time that administration of insulin within the first hour of treatment was independently associated with up to a 12.7-fold risk of developing DKA-CE.

Most episodes of DKA-CE occur 4 to 12 hours after treatment has started. Timely recognition of the signs and symptoms leading to prompt initiation of treatment of DKA-CE is imperative. Most children will complain of headache and begin vomiting prior to developing an altered state of consciousness, decreased response to pain, decorticate or decerebrate posturing, cranial nerve palsies, and Cushing’s triad (hypertension, bradycardia, and abnormal respiratory pattern). Emergency treatment includes elevation of the head of the bed, a decrease in fluid rate by one-third, and administration of mannitol (0.5–1 g/kg over 20 minutes) or 3% sodium bicarbonate.

Table 1. Pretreatment factors associated with the development of DKA-related cerebral edema.

- Younger age.
- Sick appearance.
- New-onset diabetes (3.3% vs. 0.23% in known patients).
- Longer duration of symptoms.
- More severe evidence of dehydration (increased hematocrit, urea, potassium).
- Greater acidosis (lower \( pCO_2 \), pH).

Table 2. Treatment variables associated with the development of DKA-related cerebral edema.

- Too-rapid fall (>2 mmol/L/h) in corrected sodium.
- Failure of corrected or uncorrected sodium to rise.
- Too-rapid fall (>4 mOsm/kg/h) in active osmolality.
- Use of bicarbonate to treat acidosis.
- Early insulin treatment or large insulin boluses.
- Use of fluids: administration of ≥4 L/m²/24 h or ≥50 mL/kg in the first 4 hours.
Diabetic ketoacidosis in children and adolescents: An update and revised treatment protocol

**British Columbia's Children's Hospital Diabetic Ketoacidosis Protocol**

1. Confirm DKA: plasma glucose (PG) ≥11 mmol/L, ketones, capillary pH ≤7.3, HCO₃⁻ ≤15 mmol/L.

2. Establish extent of dehydration (BMI, skin turgor, capillary refill; hematocrit) in cc/kg.
   - Children:
     - mild: 5% to 10 cc/kg
     - moderate: 10% to 15 cc/kg
     - severe: >15 cc/kg

3. Calculate total fluid deficit: multiply (1) by (2) to (3) cc.

4. Give normal saline (NS) resuscitation bolus only if patient is orthostatic or shocky.
   - recommended amount: 5-10 cc/kg BW over 1-2 hours, max <30 cc/kg.

5. Calculate remainder of fluid deficit after fluid bolus: subtract (4) from (3) cc.

6. Calculate maintenance fluid requirements for the next 48 hours.
   - add KCl 20-40 mL only if hypokalemia and patient has adequate urine output.

7. Calculate hourly rate of fluid replacement: divide (6) by 48 cc/h.

8. Use normal saline (NS) as initial replacement fluid, at rate determined in (6). Add KCl 20-40 mL only if hypokalemia and patient has adequate urine output.

9. After 1-2 hours, make up and start a piggyback insulin drip at 0.1 unit/kg BW/h.
   - regular unit insulin in a piggyback.
   - run at 1 cc/kg BW/h.

10. Close neurological observation and frequent rousing of the child with fingerpokes to detect any changes consistent with cerebral edema. Follow Glasgow Coma Scale (GCS). Severe headache, change in sensorium, or seizure activity should be reported immediately. 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)
    - consider intubation and mild hyperventilation (PEEP ≤22 cmH₂O) for impending respiratory failure

11. Follow laboratory parameters (use of a flowsheet is highly recommended):
    - follow PG by meter every 30-60 min
    - follow Na⁺, K⁺, Cl⁻, HCO₃⁻, amion gap, capillary pH every 2-4 hours
    - follow Ca²⁺, Mg²⁺, and P, every 2-4 hours if giving phosphate
    - follow urine ketones with each void or whole blood β-hydroxybutyrate (ketones) every 2-4 hours

12. Re-evaluate appropriateness of replacement fluid type frequently, anticipating the need to add or increase NaCl, K⁺, dextrose, etc.

13. Keeping the PG in the 10-15 mmol/L range allows for a better anti-glucagon response and a faster drop in the β-hydroxybutyrate level.

Rationale & Notes:

- 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 there are questions or problems related to the management of DKA or diabetes, please feel free to contact the BCH Pediatric Endocrinologist on call.

- Mild hyperglycemia, even with ketones and mild acidosis, can often be managed without IV fluids or N insulin; particularly in the older child or known diabetic who is not vomiting or seriously dehydrated.

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

- Large fluid boluses are potentially dangerous and should be administered slowly and with caution, unless the patient is truly shocky. Only very rarely will a larger (≥20 cc/kg BW) fluid bolus be required to maintain perfusion.

Since most patients develop DKA over days, slow metabolic repair is superior. Hyperhydration may contribute to cerebral edema.

- N IV insulin boluses are always contra-indicated. Insulin given in the first 1-2 h of DKA repair is thought to increase mortality. A more gentle insulin regimen may inhibit ketogenesis and glucoregulation and should be maintained if possible. If unable to keep PG <10 mmol/L, drop the insulin rate by 25-50%.

SEPTEMBER 21, 2008
HTTP://ENDO/DB.CHILDCARE.CA/FORPROFESSIONALS/DKAPROTOL.HTM

Figure 1. The 2008 BCH diabetic ketoacidosis protocol.
chloride (5–10 mL/kg IV over 30 minutes). Intubation with mild hyperventilation to keep the pCO₂ above 22 mm Hg may also be required.

Protocols for treating DKA
Because of the high rates of mortality and morbidity associated with DKA in children, young patients should be managed using standardized pediatric-specific treatment protocols to ensure safe correction of metabolic derangements while minimizing the risk of development of DKA-CE.³

The underlying principle of modern protocols is to provide even rehydration and restoration of body fluid and electrolyte deficits over a 48-hour period. This requires a meticulous approach to calculating intravenous fluid composition and rates of administration.

In 1996, the Endocrinology and Diabetes Unit at BC Children’s Hospital (BCCH) developed a DKA protocol based on the international guidelines and evidence-based knowledge that were available at that time. A companion article published in the *BCMJ* detailed the 1996 protocol and its rationale.¹⁰ Since then, new information has been published about the pathophysiology of DKA and the complications of its treatment, including DKA-CE. This has necessitated the updating of DKA protocols by a number of pediatric endocrine societies and academic institutions. In 2007, the International Society for Pediatric and Adolescent Diabetes (ISPAD) published its Clinical Practice Consensus Guidelines for DKA (updated again in 2009).⁸ The ISPAD guidelines are considered the current gold standard internationally, and the recommendations published therein have been endorsed by most major subspecialist societies.

In late 2008, the staff of the BCCH Endocrinology and Diabetes Unit published a revision of their DKA protocol to bring it into conformity with the ISPAD guidelines. The protocol is now available online¹¹ as part of the BCCH DKA Toolkit, which includes the following:

- A medical protocol for DKA (see Table 3).
- A nursing protocol for DKA.
- A flowsheet for managing DKA (see Figure 2).
- A sample physician order sheet for DKA.
- Recipes for making DKA solutions.

The five major modifications of the 1996 protocol are summarized in Table 3. As in the original protocol, the revised protocol divides the management of DKA into three periods, based on the need for monitoring and the composition of fluids to be administered:

### Figure 2: The 2008 BCCH diabetic ketoacidosis flowsheet.

<table>
<thead>
<tr>
<th>Date:</th>
<th>Time:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate</td>
<td></td>
</tr>
<tr>
<td>Respiratory Rate</td>
<td></td>
</tr>
<tr>
<td>Blood Pressure</td>
<td></td>
</tr>
<tr>
<td>Glasgow Coma Scale</td>
<td></td>
</tr>
<tr>
<td>Numb y done?</td>
<td></td>
</tr>
<tr>
<td>Blood Glucose</td>
<td></td>
</tr>
<tr>
<td>Lab</td>
<td></td>
</tr>
<tr>
<td>Urine Ketones</td>
<td></td>
</tr>
<tr>
<td>Nurse’s initials</td>
<td></td>
</tr>
<tr>
<td>Capillary H⁺</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate:</td>
<td></td>
</tr>
<tr>
<td>Capillary</td>
<td></td>
</tr>
<tr>
<td>Venous</td>
<td></td>
</tr>
<tr>
<td>Base Deficit</td>
<td></td>
</tr>
<tr>
<td>Sodium: Na⁺</td>
<td></td>
</tr>
<tr>
<td>Potassium: K⁺</td>
<td></td>
</tr>
<tr>
<td>Chloride: Cl⁻</td>
<td></td>
</tr>
<tr>
<td>Anion Gap:</td>
<td>Na⁺ + K⁺ – (Cl⁻ + HCO₃⁻)</td>
</tr>
<tr>
<td>β-Hydroxybutyrate</td>
<td></td>
</tr>
<tr>
<td>“Corrected” Sodium:</td>
<td>Na⁺ + 0.36 × (Glucose - 5.6)</td>
</tr>
<tr>
<td>“Active” Osmolality:</td>
<td>Glucose × 2 + 2 × (Na⁺ + K⁺)</td>
</tr>
<tr>
<td>Urea</td>
<td></td>
</tr>
<tr>
<td>Creatinine</td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td></td>
</tr>
<tr>
<td>Phosphate</td>
<td></td>
</tr>
<tr>
<td>Physician’s initials</td>
<td></td>
</tr>
</tbody>
</table>

November 20, 2009 endodiab.bchchildrens.ca/pdf/dkaflow.htm Page 1 of 1
On admission: attend to ABCs (airway, breathing, and circulation); complete clinical assessment; provide fluid bolus if necessary.

The first 1 to 2 hours: start normal saline to begin rehydration.

Thereafter: begin insulin infusion; add potassium and eventually dextrose to IV fluids; continue clinical and biochemical monitoring and adjustments.

The nursing protocol has been designed to provide nursing personnel with a more detailed explanation of how to perform initial and follow-up assessments of the DKA patient, how to prepare and manage the IV fluids and insulin infusion, and how to coordinate the necessary laboratory and bedside blood testing. Should nursing or pharmacy staff require assistance in making nonstandard IV solutions, the BCCH DKA Toolkit has a recipes page that explains how to prepare these using commercially available products.

In the emergency room and after
As with all sick patients, attending to the ABCs is the first priority. It is important to document the initial Glasgow Coma Scale score for use as a baseline. Since all fluid calculations in the protocol are based on the patient’s current weight, it is imperative that an accurate weight be obtained on all patients. An estimation of dehydration should be made, based on clinical criteria (tears, skin turgor, or capillary refill) and laboratory results (hematocrit, urea, potassium). Note that urine output cannot be relied upon as an accurate measure of dehydration because of the obligate diuresis of hyperglycemia. Similarly, with the mouth-breathing of Kussmaul respirations, oral mucous membranes will often appear drier than the patient’s actual level of fluid deficit. A normal blood pressure is reassuring, but nearly all children in the ER will be anxious and tachycardic.

It has become apparent that the biochemical picture of children presenting in DKA depends to a substantial degree on the type of fluid (milk, water, pop, fruit juice) and amount (if any) the child has been receiving in the time before presentation, so it is important to ask about this. Careful history-taking is also essential to help identify any events that might have led to recurrent DKA in a patient with known T1D. Finally, the physician must remember that underling infections or illness can trigger or accelerate the development of DKA.

Once a large-bore intravenous cannula has been placed and laboratory samples have been obtained to provide the baseline values needed (glucose, electrolytes, capillary blood gas, urea, creatinine, CBC, A1c, blood or urine ketones), fluid replacement can begin. If the patient shows evidence of cardiovascular instability, such as severe tachycardia, hypotension, or decreased capillary refill, a fluid bolus of normal saline (5–10 cc/kg) can be given over 30 to 60 minutes; very rarely, the shocked patient may require a second or third fluid push.

The BCCH DKA protocol has a formula for calculating fluid rates, depending on the patient’s weight and level of dehydration. In the initial 1 to 2 hours, normal saline is given. Thereafter, assuming the patient has been documented to have urine output, potassium (as chloride) should be added to the rehydration fluid. Insulin is started after the initial 1 to 2 hours at a dosage of 0.1 U/kg/h; instructions for making this are in the medical and nursing protocols. Once started, it is important that the insulin not be interrupted. IV insulin has a very short circulating half-life, and the metabolic derangements of DKA, which are normally suppressed by the insulin infusion, can restart within minutes of its discontinuation. If the insulin causes the patient to become hypoglycemic, it is preferable to add dextrose to the IV solutions using the two-bag system. Boluses of insulin should be avoided.

The two-bag system
Once the patient is receiving fluids and then insulin, the blood glucose will fall, often quite rapidly. The goal is to maintain the blood glucose in the 10 to 15 mmol/L range over the first day or so, to provide a buffer against the development of hypoglycemia. For this reason, dextrose should be added to the replacement fluids at this point.

To minimize turnaround time for fluid adjustments, our institution introduced the two-bag system a number of years ago, based on results published by a large pediatric centre. In short, two bags of intravenous fluids, identical in their electrolyte compos-
tion and differing only in their dextrose concentration, are run in parallel through the same cannula (see Figure 3). The total fluid rate from these two bags—determined by the protocol—will be constant, and the final concentration of dextrose can be altered simply by juggling the rates of the two bags. The insulin infusion from a third bag can be connected into the same cannula. The two-bag system is easy to institute, uses commercially available solutions, and has been shown to decrease the time needed to make a change in IV rates, to decrease the number of IV bags used during an admission, and to decrease the cost of IV solutions used.12

**Monitoring the DKA patient**

The patient in DKA will require frequent biochemical monitoring to ensure a smooth, safe correction of the metabolic derangements. The use of a flowsheet is indispensable in tracking the progress of a patient. Following the anion gap and capillary pH allows for monitoring of the correction of the acidosis. Special attention should be paid to the rise or fall of the sodium level as corrected for glucose. This is calculated as follows: corrected Na⁺ = [measured Na⁺ + 0.36 (glucose–5.6)]. A low corrected sodium that is not rising, or a corrected sodium that is dropping rapidly (> 2 mmol/L/h), suggests excess administration of free water.

Some experts have proposed following the active osmolality, a measure of the osmotically active molecules (which does not include urea) in DKA as follows: active osmolality = [2 (Na⁺ + K⁺) + glucose]. A rapid fall in the active osmolality (> 4 mOsm/kg/h) has also been associated with the development of DKA-CE.

Traditionally, ketones have been assessed by using semiquantitative urine dipsticks (measuring acetoacetate, which is actually present to a lesser degree in DKA). In recent years, quantitative blood beta-hydroxybutyrate levels have been used increasingly to follow the development and resolution of ketosis. Blood beta-hydroxybutyrate rises more rapidly and corrects more quickly than urine ketones and is thus a more sensitive indicator of impending DKA. Many patients using insulin pumps are trained to check themselves using a home monitor to detect early ketones associated with infusion-site problems. A blood beta-hydroxybutyrate greater than or equal to 0.4 mmol/L is abnormal in children with diabetes.13

**Table 4. Comparison of urine and blood ketones.** Adapted from Brink S, Laffel L, Likitmaskul S, et al.13

<table>
<thead>
<tr>
<th>Urine ketones</th>
<th>Blood ketones (β-hydroxybutyrate)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td>&lt; 0.5 mmol/L</td>
</tr>
<tr>
<td>Trace</td>
<td>0.5 mmol/L</td>
</tr>
<tr>
<td>Small</td>
<td>1.5 mmol/L</td>
</tr>
<tr>
<td>Moderate</td>
<td>4.0 mmol/L</td>
</tr>
<tr>
<td>Large</td>
<td>8.0 mmol/L</td>
</tr>
<tr>
<td>Very large</td>
<td>16 mmol/L</td>
</tr>
</tbody>
</table>

**Caveats**

While it is generally straightforward to diagnose and treat DKA, caution must be taken when treating a child in...
DKA to ensure that comorbidities are not missed. Many children presenting in DKA have abdominal pain, tachypnea, and polyuria; it is important to ascertain with time whether these are part of the DKA picture or there is an underlying appendicitis, pneumonia, or urinary tract infection. Fever is not seen in uncomplicated DKA and should always be investigated. However, white blood cell counts are often quite elevated in DKA with a profound left shift, due to elevated plasma catecholamines; this normally resolves within the first 4 to 6 hours of treatment.

It has been recognized increasingly that many children presenting with DKA-like symptoms may have a coexistent—and sometimes predominant—element of hypernatremic dehydration, the hyperglycemic hyperosmolar state, or both. This is observed especially in children with T2D, as well as those with T1D who have consumed large amounts of salt- or carbohydrate-containing fluids prior to presentation. In these children, hypernatremia (corrected Na⁺ > 150 mmol/L), hyperglycemia (> 33 mmol/L), and/or hyperosmolarity (> 320 mOsm/kg) may be present, but the acidosis is mild (pH > 7.30, bicarbonate > 15 mmol/L), and ketones are absent or only mildly elevated (blood beta-hydroxybutyrate < 1 mmol/L). In such cases, treatment must be modified to address the unique biochemical disturbance of the patient.

The ketoacidosis of DKA resolves with fluid and insulin treatment. Bicarbonate should not be used in children with DKA, except in the very rare situation where profound acidosis is causing decreased cardiac output. The use of bicarbonate has been associated with an increased risk of DKA-CE, paradoxical CNS acidosis, hyperosmolality, delayed correction of acidosis, and longer hospital stays. As well, the use of phosphate-containing solutions in DKA remains controversial. Prospective studies have not identified a benefit with the use of phosphate, but most experts would consider substituting some of the potassium chloride in the replacement fluids with potassium phosphate in the face of weakness or severe hypophosphatemia. In this case, the patient must be monitored for the development of hypocalcemia.

Occasionally, patients treated according to the DKA protocol do not exhibit an increase in pH after the first 4 to 6 hours of treatment. Almost invariably, this is due to an error in the preparation of the insulin infusion. In this case, a new insulin bag should be made.

Existing DKA protocols, including the BCCH protocol, provide an algorithm for treating the majority of cases of DKA in infants, children, and adolescents based on our best current understanding of research and the medical literature. However, no protocol has been designed that completely eliminates the occurrence of DKA-CE, and no protocol can replace careful clinical observation and judgment when treating this potentially very serious condition.

**Recurrent DKA**

Most pediatric endocrinologists will maintain that the vast majority of recurrent DKA episodes are preventable. The physician should bear in mind that recurrent DKA is the result of insulin omission, either deliberate or accidental, until proven otherwise. An A1c taken at admission, and perhaps an insulin level, will help identify the patient with recurrent bouts of “gastroenteritis” who is in reality in borderline metabolic control or who has been missing insulin injections (or both). Insulin omission is frequently associated with eating disorders in teenage girls, depression in children or youth, insufficient parental supervision, or a poor psychosocial situation. In these instances, it is often beneficial to solicit the help of a social worker, counselor, or psychiatrist before discharge.

In some instances, parents or patients will mistakenly discontinue all insulin on sick days because of a fear of hypoglycemia with poor oral intake. This situation may also lead to hyperglycemia and DKA. Family education around proper sick-day management, with reinforcement at subsequent visits, should help to prevent this problem.

With the rise in use of insulin pumps in the pediatric population (approximately 30% to 40% of children who have diabetes in BC are on an insulin pump), a new cause of DKA is being observed: insulin pump infusion-site problems (and, much more uncommonly, insulin pump malfunction). Since pumps carry only rapid-acting insulin analogs, any disruption in insulin delivery can lead to rapid development (within 2 to 4 hours) of hyperglycemia and ketosis. Home monitoring of ketones, rapid replacement of dislodged infusion sites, and the administration of a correction dose of rapid-acting insulin (preferably by pen or syringe if ketones are present) are essential. Family members should be educated about managing infusion-site problems at the time of pump initiation, with reinforcement of pump training at subsequent visits.

**Coming off protocol**

While the protocol is designed to correct DKA over a 48-hour period, many children are metabolically corrected more quickly. Once the acidosis has resolved (pH > 7.3 and anion gap normal) and the patient is feeling well and is ready to begin eating and drinking, the use of subcutaneous insulin can be started or re-established. This is most
Diabetic ketoacidosis in children and adolescents: An update and revised treatment protocol

DKA is associated with significant fluid and biochemical derangements, necessitating a thoughtful, structured approach to its management.

Easily done at breakfast or dinner. The patient will require a dose of intermediate-acting (e.g., NPH) or basal (e.g., glargine or detemir) insulin, as well as a dose of short-acting (e.g., regular) or rapid-acting (e.g., lispro, aspart, or glulisine) preprandial insulin. The insulin drip should be discontinued 15 to 30 minutes after the first injection of rapid-acting insulin or 60 to 120 minutes after regular insulin. The physician may choose to continue the IV fluids (without glucose) in the patient who still has a mild fluid deficit.

Summary

The management of pediatric DKA in our province often begins in local emergency rooms with primary care providers who see this condition only rarely. Yet it is the medical care that children receive in the first hours that can have the greatest impact on their outcome and survival. It is essential for all emergency rooms and associated medical personnel to have a plan in place for dealing with this relatively uncommon condition, including access to the necessary medical supplies and diagnostic equipment needed to make a rapid and accurate diagnosis. A thoughtful plan of action must be formulated to maximize patient safety. It must be recognized that DKA is treated very differently in children and adults, particularly with respect to fluid administration. Pediatric tertiary care centres such as BCCH stand ready to assist local and regional hospitals and medical staff in dealing with pediatric DKA at all times. Ultimately, the goal is to decrease the incidence of DKA in children by educating the public about the signs and symptoms of diabetes, reminding T1D families about avoidance of recurrent DKA, and training professionals to make an earlier diagnosis of diabetes in children presenting with suspicious symptoms.

Acknowledgments

The BCCH DKA Protocol Toolkit was revised by the pediatric endocrinologists from the Endocrinology and Diabetes Unit at BC Children’s Hospital, who are grateful for the input received from colleagues in Nursing, Intensive Care, General Pediatrics, Emergency Medicine, Laboratory Medicine, and Pharmacy.

Competing interests

None declared.

References