Diabetic Ketoacidosis: Emergency Management in Children

Purpose

This document provides clinical practice guidelines for clinicians involved in the emergency management of children with diabetic ketoacidosis (DKA).

Scope

This guideline applies to all CHQ HHS staff involved in the care and management of children with diabetic ketoacidosis.

Related documents

Policy and standard(s)

For the management of DKA, CHQ also endorses the use of:

- National evidenced-based clinical care guidelines: Type 1 diabetes in children, adolescents and adults – prepared by Australasian Paediatric Endocrine Group for National Health & Medical Research Council 1

Procedures, Guidelines, Protocols

- Emergency management of children with diabetic ketoacidosis flow chart
- Medications and IV fluids for children with diabetic ketoacidosis
- Admission criteria for children with diabetic ketoacidosis

Forms and templates

- Diabetic Ketoacidosis insulin infusion order and blood glucose level record
Guideline

Introduction
Diabetic ketoacidosis is a life-threatening metabolic disorder and is the leading cause of morbidity and mortality in children and adolescents with type 1 diabetes. Mortality is predominantly due to cerebral oedema, which occurs in 0.3% to 1% of all episodes of DKA in children.¹

Diabetic ketoacidosis is caused by a decrease in effective circulating insulin, insulin resistance and increased production of counter-regulatory hormones.² The resulting increased hepatic and renal glucose production, and impaired peripheral glucose utilisation, causes hyperglycaemia and hyperosmolality. In addition, increased lipolysis with the overproduction of ketones leads to ketonaemia and metabolic acidosis. Hyperglycaemia and acidosis causes osmotic diuresis, dehydration and obligate loss of electrolytes.

Diagnosis

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperglycaemia (BGL &gt;11mmol/L) AND</td>
<td>Mild: venous pH &lt;7.3 or HCO3 &lt;15 mmol/L</td>
</tr>
<tr>
<td>Venous pH &lt;7.3 and/or HCO3 &lt;15 mmol/L</td>
<td>Moderate: pH &lt;7.2 or bicarbonate &lt;10 mmol/L</td>
</tr>
<tr>
<td>Presence of ketonaemia/ketonuria</td>
<td>Severe: pH &lt;7.1 or bicarbonate &lt;5 mmol/L</td>
</tr>
</tbody>
</table>

Children may present with DKA at any age, with or without a previous diagnosis of type 1 diabetes. DKA can also occur in newly diagnosed type 2 diabetes. Note also that in rare cases known patients with diabetes may have symptomatic ketoacidosis without a raised blood sugar level.

ALERT
Children can die from DKA
For early advice regarding the management of the child with DKA and to discuss the potential need for retrieval, contact Retrieval Services Queensland (RSQ) on 1300 799 127.

Assessment & Resuscitative Management

ABCD Approach
Emergency assessment and management should always involve a rapid primary survey with evaluation and management of airway, breathing, circulation and disability (ABCD). Pre-hospital treatment should be taken into consideration.

Shock at presentation – severely ill with poor perfusion and thready rapid pulse
- Only if shocked give 10ml/kg 0.9% (normal) saline as a bolus and repeat as necessary to a maximum of 20ml/kg.
- There is no evidence to support use of colloids/volume expanders over crystalloids.
- If considering additional fluid bolus’ or the use of inotropes discuss with an intensivist.
ALERT
Do NOT give a fluid bolus unless the child is in shock
Most children with DKA who present in shock will not require more than two fluid boluses of 10 ml/kg 0.9% (normal) saline. If two or more fluid boluses are administered, sepsis should be considered in the differential diagnosis.

Coma at presentation
• Consider transfer to PICU/HDU if available.
• Coma is directly related to degree of acidosis, but signs of raised ICP suggest cerebral oedema.
• Consider instituting cerebral oedema management (see below).

ALERT
Never give bolus doses of intravenous insulin.

Clinical Assessment
Clinical assessment of the child with suspected DKA should include the following:
• History:
  – Polydipsia and polyuria (may be absent in the young child).
  – Enuresis and/or wetting ‘accidents’ in a toilet trained child.
  – Weight loss and/or increased appetite.
  – Vomiting.
  – Abdominal pain.
  – Non-specific symptoms and signs of general malaise.
• Full physical examination:
  – Dehydration assessment: BP, pulse rate and volume, perfusion assessment (capillary refill time, skin colour, mentation), mucous membranes, tissue turgor.
  – Features of acidotic respiration: hyperventilation.
  – Assessment for cerebral oedema: headache, irritability, slowing pulse, rising BP and reducing conscious level. Papilloedema is a late sign.
  – Assessment for infection including appendicitis, ileus and pancreatitis.
• Biochemical confirmation:
  – BGL via finger-prick (>11mmol/L) – may be inaccurate in circulatory compromise & acidosis.
  – Blood pH <7.3 and/or HCO3 <15mmol/L.
  – Finger prick blood ketones >3.0mmol/L.
  – Glycosuria and ketonuria.
Dehydration Assessment

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild ~ 3%</td>
<td>Is only just clinically detectable</td>
</tr>
<tr>
<td>Moderate ~ 5%</td>
<td>Dry mucous membranes, reduced skin turgor</td>
</tr>
<tr>
<td>Severe ~ 8%</td>
<td>Above with sunken eyes, poor capillary return</td>
</tr>
<tr>
<td></td>
<td>Do not use more than 8% dehydration in volume deficit calculations</td>
</tr>
<tr>
<td>Shock</td>
<td>May be severely ill with poor perfusion, thready rapid pulse (reduced BP is a very late sign)</td>
</tr>
</tbody>
</table>

**ALERT**

**Over-estimation of volume deficit in DKA is dangerous**

Volume deficit is difficult to assess accurately in DKA, particularly in the young child, and may be overestimated because of the subjective criteria used.\(^5\)

Dryness of oral mucosa in a patient in DKA may be exacerbated by the tachypnoea of Kussmaul respirations.\(^6\)

Vasoconstriction from acidosis may contribute to the appearance of cool extremities in addition to poor perfusion from dehydration.\(^6\)

Reliance on weight loss to calculate the percentage dehydration may also overestimate fluid loss because of weight loss associated with catabolism due to insulin deficiency.

Shocks with haemodynamic compromise is uncommon in childhood DKA.

**Investigations**

The following urgent baseline investigations should be requested:

- Blood glucose level.
- Finger-prick blood ketones (superior to urinary ketones).
- Only use urine ketones if blood ketones not available - please see [Appendix 1](#) for interpretation.
- Urea and electrolytes (serum urea >9.0 mmol/L may indicate severe dehydration).
- Venous pH and acid-base status.
- Hb A1C (for later analysis).

**Fever is not a feature of DKA:**

- Perform sepsis investigations as clinically appropriate - FBC, Urine MCS, CXR, CSF, throat swab, blood culture
- Also consider such investigations if the child is hypothermic, hypotensive or has a refractory acidosis or lactic acidosis.
- Note that an elevated WCC is common in DKA and doesn’t necessarily indicate sepsis.
If new diagnosis of diabetes:
- TSH and thyroid antibodies
- Coeliac screen, Total IgA and anti-tissue transglutaminase Ab (A-TgA)
- Consider collecting blood for tissue auto-antibodies (insulin antibodies, islet cell antibodies, GAD, IA2) in consultation with local specialists and in accordance to their desired practice.

Management of Moderate to Severe DKA

Background – Management of Water, Electrolyte and Acid Base Abnormalities
Diabetic ketoacidosis is characterised by loss of water and electrolytes. Administration of IV fluid, prior to giving insulin, results in substantial falls in blood glucose because the resultant increase in glomerular filtration rate (GFR) leads to increased urinary glucose excretion. The aims of fluid and electrolyte replacement therapy in DKA are:
- Restoration of circulating volume.
- Replacement of sodium and water deficit over 48 hours.
- Management of the predictable fall in the serum potassium concentration after insulin therapy commences and the ketoacidosis starts to reverse.
- Restoration of GFR with enhanced clearance of glucose and ketones from the blood.
- Administration of insulin therapy to normalise the BGL and to suppress lipolysis and ketogenesis.
- Avoidance of cerebral oedema, which may be caused by rapid fluid shifts from the extracellular fluid to the intracellular fluid compartment.

Oral Fluids and Vomiting
- Vomiting on presentation should initially be treated with IV fluids and insulin (not antiemetics). If necessary use ondansetron (zofran). Neurotropic antiemetics may interfere with the patient’s neurological status.
- All patients with moderate to severe DKA should initially remain ‘nil by mouth’ except for ice to suck.
- A nasogastric tube may be necessary in the case of gastric paresis.
- Oral fluids should only be offered after substantial clinical improvement (i.e. blood sugar < 15mmol/l and level of consciousness has improved if it was initially reduced) and no vomiting.
- When good clinical improvement occurs prior to the 48 hour rehydration period being completed, oral intake may proceed and the need for IV infusions reduced.
Rehydration Fluids

ALERT

Initial Rehydration Fluid

Specialist Paediatric ICU/ED/Endocrine input is required for fluid choices if any of the following are present:

- Neonatal DKA
- Hypernatraemia (calculate corrected sodium)
- Hyperosmolality
- Anuria
- Hyperkalaemia

Use 1 litre 0.9% NaCl + 40mmol KCl (pre-mixed bag) as the initial default fluid provided none of the above exclusions are present.

Fluid should be commenced one hour before starting insulin therapy.

Once BGL falls to ≤15mmol/L, it is necessary to add extra glucose to the IV rehydration fluid.

There are currently no suitable pre-mixed bags of 0.9% Normal Saline + 5% dextrose + 40 mmol KCl, thus this solution will need to be mixed on site.

The current CHQ-GDL-01025 Intravenous Fluid Guideline – Paediatric and Neonatal has guidance around available fluids and recipes for mixing non-standard solutions.

If additional dextrose above 5% is required to prevent hypoglycaemia, then call an intensivist for advice as there is a risk of cerebral oedema in DKA in using significantly hyponatraemic solutions.

Other considerations in fluid management include:

- Perform a dehydration assessment.
- Urinary losses should not be added to the initial calculation of replacement fluids.
- Calculation of effective osmolality may be useful to guide ongoing fluid and electrolyte therapy.

Bicarbonate replacement

Severe acidosis is usually reversible by fluid and insulin administration. Bicarbonate should only be considered in children who are profoundly acidotic (pH<6.9) and shocked with circulatory failure. Its only purpose is to improve cardiac contractility in severe shock.

Cardiac monitoring is required due to risk of inducing hypokalaemia.

ALERT

Administration of Sodium Bicarbonate

The decision to administer sodium bicarbonate to a child with DKA must be made in consultation with a paediatric intensivist and a paediatric endocrinologist at a Level 6 facility.

Sodium bicarbonate should not be used routinely in the management of DKA due to the association between bicarbonate treatment and increased risk of cerebral oedema.
Sodium replacement
The measured serum sodium may be falsely reduced because of the dilutional effect of the hyperglycaemia.

**ALERT**

Calculate Corrected Sodium and Osmolality

Corrected Sodium Calculation: \( Na^+ = (\text{measured } Na^+ + [2 \times (\text{glucose} - 5.5)] / 5.5) \)

Osmolality Calculation: \( \text{Osm} = 2 \times [Na^+ + K^+] + \text{glucose} \)

Hypernatraemia = corrected \( Na^+ > 150 \text{ mmol/L} \)

Hyperosmolality = \( > 310 \text{mosm/L} \)

Hypernatraemia +/- hyperosmolality in DKA:
- Requires discussion with a senior specialist in ICU/ED/Endocrine as it is usually associated with severe DKA.
- Correction of dehydration and electrolyte abnormalities should occur over 72hrs.
- 0.9% NaCl 1L + 40 mmol KCl is an appropriate initial rehydration fluid as hypotonic fluids may be associated with raised ICP.

Potassium replacement
Serum potassium levels in DKA at presentation are not a reliable indicator of total body potassium stores. Serum potassium may be reduced, normal or elevated at the time of presentation. The administration of insulin and the correction of acidosis will drive potassium back into the cells, decreasing serum levels.

**ALERT**

Potassium Replacement
Premade solutions containing potassium are preferred. Maximum potassium concentration should be 40mmol/L.

Plan for the predictable fall in the serum potassium concentration after insulin therapy commences and the ketoacidosis starts to reverse.

Potassium should be added to all rehydration fluids immediately unless the patient is anuric (after insertion of a urinary catheter) or is hyperkalaemic (>5.5 mmol/L).

Check potassium measurements every 2 hours and always cardiac monitor a patient with DKA (observing for T wave changes).

Hypokalaemia causes T wave flattening. If hypokalemia occurs temporarily reduce insulin infusion rate by 50% and discuss with PICU regarding central access and increased potassium replacement.

T wave peaking may be a sign of hyperkalaemia in a patient with pre-renal failure, check the venous potassium and, if necessary, reduce the potassium replacement until a good urine output occurs and the potassium level falls to the top of the normal range.

The following issues need to be considered in potassium replacement:
- Commence replacement with premixed bag of 1L 0.9 % sodium chloride + 40mmol KCl.
- Do not exceed a maximal potassium infusion rate of 0.3 mmol/kg/hour without consultation.
- Potassium replacement should continue throughout IV fluid therapy.
Insulin

Rehydration alone will decrease the BGL to some extent, however insulin therapy is required to normalise the BGL and to suppress lipolysis and ketogenesis.

In moderate and severe DKA, intravenous insulin is required. If IV access cannot be obtained, discuss with an endocrinologist.

Intravenous insulin infusion preparation:
- Only short-acting insulins (eg Actrapid or Humulin R) should be used for IV insulin administration.1
- The insulin infusion set should be changed every 24 hours due to the potential for the insulin to denature.1

### ALERT

**Calculating the dose of insulin**

Very serious errors have occurred when calculating the dose of insulin.
The dose of insulin should always be checked by a colleague.

<table>
<thead>
<tr>
<th>If using a syringe pump:</th>
<th>If no syringe pump available</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Add 50 units (0.5mL) short acting insulin (Actrapid or Humulin R) to 49.5mL of 0.9% NaCl in a syringe. [Insulin concentration = 1U/mL]</td>
<td>• Add 50 units (0.5mL) short acting insulin (Actrapid or Humulin R) to a 500mL bag of 0.9% NaCl. [Insulin concentration = 0.1U/mL]</td>
</tr>
<tr>
<td>• Infusion to be delivered by a syringe pump into the side arm of the IV line.</td>
<td>• The infusion should be delivered using a volumetric pump into the side arm of the IV line. If a volumetric pump is not available a separate IV site may be required for low infusion rates.</td>
</tr>
</tbody>
</table>

**Initial Intravenous insulin dosage and administration**1a:

### ALERT

**Never give bolus doses of intravenous insulin**

Insulin pump therapy should be stopped in DKA
Start insulin therapy one hour after starting fluid therapy
Continuous IV insulin infusion dose should be 0.1 units/kg/hr

- There is no firm evidence to support an initial infusion dose of 0.05 units/kg/hr1a
- In obese patients it may be prudent to start insulin infusion based on ideal body weight.

**Ongoing intravenous insulin dosage and administration**1a:

- **When the BGL falls to ≤15 mmol/L:** Glucose should be added to the IV fluid to prevent hypoglycaemia.
  A solution of 0.9% NaCl + 5% glucose + 40mmol KCl per litre is used (see below re glucose solutions).
  **DO NOT** reduce the insulin infusion rate. The insulin dose needs to be maintained at 0.1units/kg/hr to switch off ketogenesis.
- **When the BGL falls >5mmol/L/hr:** Some suggest adding glucose if the initial rate of fall of blood glucose is greater than 5mmol/L/hr to protect against cerebral oedema1a. There is no good evidence for this practice. BGL will often fall quickly because of rehydraton.
• **Resolution of ketoacidosis**: The dose of insulin should remain at 0.1 units/kg/hr at least until resolution of ketoacidosis (pH >7.3, HCO3 >15mmol/L and or closure of anion gap) – then consider transition to subcutaneous insulin. To transition subcutaneous insulin should be commenced 1 hour before stopping intravenous treatment.

• **If BGL rises out of control or pH not improving after 4-6hrs**: Consult senior staff and re-evaluate for sepsis, insulin error, inadequate resuscitation, hyperchloraemic acidosis, salicylate or other prescription or recreational drugs. Consider restarting protocol over again.

• **If the patient uses an insulin pump and presents in DKA**: The pump should be removed as the assumption would be there is a pump problem. The pump should therefore only be restarted on the advice of an endocrine team or local equivalent and would be recommenced using a new site with a new set.

**ALERT**

Glucose warnings
50% glucose is extremely hypertonic and should NOT be administered without dilution.
0.9% sodium chloride with 10% glucose is very hypertonic and the site should be monitored for local reactions.

**Management of Hypoglycaemia**

• **If the BGL falls below 4mmol/L**, give a bolus of 2 mL/kg of 10% glucose over 3 minutes. Ensure fluid running has 5% dextrose and consider if there is a requirement for 10% dextrose. Insulin can temporarily be reduced for 1 hour. **DO NOT** stop the insulin infusion while glucose is being infused1a, as insulin is required to switch off ketone production.

• **If issues with maintaining BGL between 5-10mmol/L remain despite running a solution containing 5% dextrose**: the glucose concentration in the IV fluid may be further increased to 0.9% NaCl + 10% glucose + 40mmol KCl/L (this requires intensivist input as some mixtures of this solution are significantly hyponatraemic and may contribute to cerebral oedema). The insulin infusion rate should only be decreased if the BGL remains below the target range despite this glucose supplementation.

**ALERT**

There are currently no suitable pre-mixed bags of 0.9% Normal Saline + 5% dextrose + 40 mmol KCl, thus this solution will need to be mixed on site.

The current CHQ-GDL-01025 Intravenous Fluid Guideline – Paediatric and Neonatal has guidance around available fluids and recipes for mixing non-standard solutions.

If additional dextrose above 5% is required to prevent hypoglycaemia, then call an intensivist for advice as there is a risk of cerebral oedema in DKA in using significantly hyponatraemic solutions.

**Monitoring – Careful clinical observation and reassessment**

Use the Statewide Diabetic Ketoacidosis (DKA) Insulin Intravenous Infusion or Subcutaneous Insulin Order and Blood Glucose Level Record Form
Clinical and laboratory monitoring should include:

<table>
<thead>
<tr>
<th>Monitoring</th>
<th>Frequency</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital Signs (HR, RR, BP)</td>
<td>Hourly</td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>2-4&lt;sup&gt;th&lt;/sup&gt; hourly, hourly if febrile</td>
<td>Blood ketones are monitored to assist in determining resolution of DKA. The bedside meter (Abbott brand) ketone readings &gt;4mmol/L are less accurate. The whole child needs to be assessed – DO NOT use either blood or urinary ketones alone as the indicator for changes to fluid or insulin regimes.</td>
</tr>
<tr>
<td>Capillary (fingerpick) BGL and blood ketones</td>
<td>Hourly</td>
<td></td>
</tr>
<tr>
<td>Laboratory bloods:</td>
<td>2-4&lt;sup&gt;th&lt;/sup&gt; hourly</td>
<td>Capillary glucose methods may be inaccurate in the presence of poor peripheral circulation and acidosis. In severe DKA it may be necessary to monitor electrolytes hourly. An IV cannula may be placed for repetitive blood sampling. An IA line may be necessary in some critically ill patients managed in PICU.</td>
</tr>
<tr>
<td>• Venous glucose</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Blood gases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• U&amp;Es</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Haematocrit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strict fluid balance</td>
<td>Hourly</td>
<td>Watch carefully for polyuria If level of consciousness is impaired a urinary catheter may be necessary.</td>
</tr>
<tr>
<td>Neurological Observations</td>
<td>Hourly or more frequently in high risk groups such as &lt;2yr olds and those with pH below 7.1</td>
<td>Warning signs and symptoms of cerebral oedema • Headache • Inappropriate slowing of heart rate • Recurrence of vomiting • Change in neurological states (restlessness, irritability, increased drowsiness, incontinence) or specific neurological signs (such as cranial nerve palsies, pupillary response) • Rising BP, decreased oxygen saturation If cerebral oedema is suspected notify senior staff immediately.</td>
</tr>
<tr>
<td>ECG monitoring</td>
<td>Continuous</td>
<td>Assess T waves for hyperkalaemia or hypokalaemia</td>
</tr>
<tr>
<td>Urinalysis for ketones</td>
<td>Until negative</td>
<td>Only required if blood ketones not available</td>
</tr>
<tr>
<td>Weight</td>
<td>On admission &amp; daily</td>
<td></td>
</tr>
</tbody>
</table>

Management of Cerebral Oedema

**ALERT**

Children can die from DKA

For early advice regarding the management of the child with DKA and to discuss the potential need for retrieval, contact Retrieval Services Queensland (RSQ) on 1300 799 127. Emergency management of suspected cerebral oedema must be initiated as a matter of extreme urgency, and must not be delayed by requests for neurology consultation or performance of a CT or MRI. Discussion with PICU consultant is essential after initial treatment.
Cerebral oedema is a rare but devastating complication of diabetes, occurring in approximately 1% of children being managed for DKA. It is typically described as having a sudden onset and manifesting as rapidly progressive neurological deterioration (altered/fluctuating conscious level, headache, vomiting, bradycardia, hypertension, cranial nerve palsy, abnormal posturing).

Clinical cerebral oedema can occur suddenly and at any time. The most common period for this to occur is 4-12 hours after commencement of treatment.

Risk factors include:
- New onset Type 1 diabetes
- Elevated serum urea nitrogen
- Severe dehydration
- Severe DKA (pH ≤ 7.1)
- Lower bicarbonate levels
- Age ≤ 5 years
- Reduced level of consciousness

**ALERT**
Excessive administration of IV fluids may contribute to the development of cerebral oedema.

Biochemical red flags associated with the development of cerebral oedema include:
- A rapid fall in the calculated osmolarity with treatment. Usually the serum sodium rises as the glucose falls resulting in a relatively stable calculated osmolarity
- Development of hyponatraemia during therapy
- An initial corrected sodium in the hypernatraemic range

**If a biochemical red flag is detected contact paediatric ICU/ED/endocrinologist**

**Treatment – initiate immediately**
- Raise the head of the bed to 20°
- Give high-flow oxygen via a non-rebreathing mask with a reservoir bag
- Reduce the rate of fluid administration
- Give IV mannitol (0.5 -1.0 g/kg over 20 minutes) or hypertonic saline 3% (3-5ml/kg over 15 minutes) in patients with signs of cerebral oedema before impending respiratory failure.
- Intubation and ventilation may be necessary, but aggressive hyperventilation has been associated with poor outcome in retrospective studies of DKA related cerebral oedema. Aim for CO2 35-40mmHg.
- The patient must be discussed with an intensivist & should be transferred to an intensive care facility and a neurological assessment and MRI or CT scan arranged.
Management of Mild DKA

The following management should only be considered in a child who:

- Is clinically well
- Is tolerating oral fluids
- Is less than 5% dehydrated
- Has a pH between 7.25 and 7.3
- Has normal perfusion

**ALERT**
Insulin pump therapy should be discontinued even for mild DKA.

Insulin therapy in mild DKA

*Subcutaneous insulin:*
Short acting insulin (Actrapid or Humulin R) or ultra-short acting insulin analog (Humalog [lispro] or NovoRapid [aspart]) should be used.
Give 0.1 – 0.2 units/kg every 4 - 6 hours subcutaneously depending on the response. For young children <5 years, a smaller dose of 0.05 units/kg may be used. If the BGL remains elevated, a further dose of 0.05 units/kg can be given after 2 - 3 hours.

Monitoring in mild DKA

Use the [Statewide Diabetic Ketoacidosis (DKA) Subcutaneous Insulin Order and Blood Glucose Level Record Form](#).

Clinical reassessment of the child at frequent intervals is mandatory.

- Vital Signs – hourly
- Temperature – 4 hourly
- Blood Glucose – finger prick or venous BGL at a minimum one (1) hourly
- Capillary blood Ketones – hourly
- Strict Fluid Balance – in mild DKA it is still crucial to monitor fluid balance (in particular fluid intake) as even mild DKA can develop cerebral oedema at any time
- Neurological observation - hourly (unless endocrinologist requests that observation not required)

Disposition

Mild and moderate cases of DKA may be managed in a general paediatric ward depending on local practice. A paediatrician with training and expertise in the management of DKA should direct inpatient management. The child with mild to moderate DKA should receive care in a unit that has:

- Experienced nursing staff trained in the monitoring and management of DKA
- Written guidelines for DKA management in children
• Access to laboratories that can provide frequent and timely measurements of biochemical variables
• Access to appropriate education and psychosocial assessment services

ALERT
Signs of severe DKA that should prompt consideration for treatment in a Level 6 High Dependency Unit or Paediatric Intensive Care Unit include:

• All cases of severe DKA
• Lack of appropriate staff / facilities to care for child with mild/moderate DKA
• Long duration of symptoms
• Cardiovascular compromise or shock not responding to treatment
• Requirement for respiratory support (intubation/ventilation)
• Depressed level of consciousness/neurological deterioration/cerebral oedema
• Increased risk for cerebral oedema (including < 5 years of age, new onset)

Contact RSQ for these patients to discuss management with a paediatric intensivist (1300 799 127).

Consultation
Key stakeholders who reviewed this version:
• Dr Jason Acworth, Director of Emergency, Lady Cilento Children’s Hospital, Brisbane.
• Dr Natalie Deuble, Paediatric Emergency Specialist, Lady Cilento Children’s Hospital, Brisbane.
• Prof Jerry Wales, Director of Endocrinology and Diabetes, Lady Cilento Children’s Hospital, Brisbane.
• Kate Trenoweth, Clinical Practice Facilitator, Lady Cilento Children’s Hospital, Brisbane
• Dr Geoff Pearce, Paediatric Emergency Specialist, Lady Cilento Children's Hospital, Brisbane
• Dr Dana Newcomb, GP Liaison, Office of Executive Director Medical Services, CHQ
• LCCH ED and Endocrine SMOs

Acknowledgements
Children’s Health Queensland would like to acknowledge the contribution made by members of the Greater Brisbane metropolitan area clinical procedures working group to the creation of the original version of this guideline.
## Definition of terms

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
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<tbody>
<tr>
<td>AVPU</td>
<td>Alert, Voice, Pain, Unresponsive</td>
</tr>
<tr>
<td>BGL</td>
<td>Blood glucose level</td>
</tr>
<tr>
<td>BP</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>Children</td>
<td>0-16 years of age</td>
</tr>
<tr>
<td>CHQ</td>
<td>Children’s Health Queensland</td>
</tr>
<tr>
<td>CNS</td>
<td>Central nervous system</td>
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<tr>
<td>CRP</td>
<td>C-reactive protein</td>
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<tr>
<td>CSCF</td>
<td>Clinical Services Capability Framework</td>
</tr>
<tr>
<td>CXR</td>
<td>Chest x-ray</td>
</tr>
<tr>
<td>DKA</td>
<td>Diabetic ketoacidosis</td>
</tr>
<tr>
<td>ECF</td>
<td>Extracellular fluid</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
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<tr>
<td>EDIS</td>
<td>Emergency Department Information System</td>
</tr>
<tr>
<td>FBC</td>
<td>Full blood count</td>
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<tr>
<td>GCS</td>
<td>Glasgow Coma Scale</td>
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<tr>
<td>GFR</td>
<td>Glomerular filtration rate</td>
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<tr>
<td>HR</td>
<td>Heart rate</td>
</tr>
<tr>
<td>ICF</td>
<td>Intracellular fluid</td>
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<tr>
<td>IM</td>
<td>Intramuscular</td>
</tr>
<tr>
<td>IV</td>
<td>Intravenous</td>
</tr>
<tr>
<td>K⁺</td>
<td>Potassium</td>
</tr>
<tr>
<td>Kussmaul breathing</td>
<td>Deep and laboured breathing associated with metabolic acidosis</td>
</tr>
<tr>
<td>Na⁺</td>
<td>Sodium</td>
</tr>
<tr>
<td>NaCl</td>
<td>Sodium chloride</td>
</tr>
<tr>
<td>NBM</td>
<td>Nil by mouth</td>
</tr>
<tr>
<td>PICU</td>
<td>Paediatric Intensive Care Unit</td>
</tr>
<tr>
<td>RR</td>
<td>Respiratory rate</td>
</tr>
<tr>
<td>RSQ</td>
<td>Retrieval Services Queensland</td>
</tr>
<tr>
<td>SC</td>
<td>Subcutaneous</td>
</tr>
<tr>
<td>U&amp;E’s</td>
<td>Urea and electrolytes (serum electrolyte analysis)</td>
</tr>
<tr>
<td>VBG</td>
<td>Venous blood gas</td>
</tr>
</tbody>
</table>
References and suggested reading


Guideline revision and approval history

<table>
<thead>
<tr>
<th>Version No.</th>
<th>Modified by</th>
<th>Amendments authorised by</th>
<th>Approved by</th>
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<tr>
<td>1.0</td>
<td>Greater Brisbane metropolitan area clinical procedures working group</td>
<td>Greater Brisbane metropolitan area clinical procedures editorial group</td>
<td>General Manager Operations, Children’s Health Services</td>
</tr>
<tr>
<td>2.0</td>
<td>DKA Working Group LCCH</td>
<td>Divisional Director, Critical Care</td>
<td>Executive Director, Medical Services</td>
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</table>

Keywords

Children, diabetic ketoacidosis, DKA, emergency management, 00706

Accreditation references

NSQHS Standards (1-10): 1.3.1, 1.4.1, 1.5.1

Appendix 1 – Ketone readings and probability of DKA (use with BGL and pH)

Appendix 2 – Fluid therapy calculation for children with DKA

Appendix 3 – DKA Flowchart
Appendix 1 – Ketone readings and probability of DKA (use with BGL and pH)

<table>
<thead>
<tr>
<th></th>
<th>Low</th>
<th>Moderate #</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Urine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bayer brand</td>
<td>0 mmol/L</td>
<td>0.5 mmol/L to &lt; 1.5 mmol/L</td>
<td>≥ 1.5 mmol/L</td>
</tr>
<tr>
<td>Keto-Diastix</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Accu-chek brand</td>
<td>Negative</td>
<td>&lt; 1.0 mmol/L</td>
<td>≥ 1.0 mmol/L</td>
</tr>
<tr>
<td>Keto-Diabur-Test 5000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Blood – capillary</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bedside meter Abbott</td>
<td>&lt; 0.6 mmol/L</td>
<td>0.6 mmol/L to &lt; 1.5 mmol/L</td>
<td>≥ 1.5 mmol/L</td>
</tr>
</tbody>
</table>
## Appendix 2 – Fluid therapy calculation for children with DKA

<table>
<thead>
<tr>
<th>Body weight in kg:</th>
<th>1</th>
<th>kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fluid bolus given</td>
<td>2</td>
<td>mL</td>
</tr>
</tbody>
</table>

**Deficit – fluid bolus already given (given over 48hrs)**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Fluid Bolus</th>
</tr>
</thead>
<tbody>
<tr>
<td>No signs of dehydration (tolerating fluids orally)</td>
<td>Continue with oral rehydration</td>
</tr>
<tr>
<td>Moderate 5% Dry mucous membranes, reduced skin turgor</td>
<td>50 mL/kg</td>
</tr>
<tr>
<td>Severe 8% Above with sunken eyes &amp; poor capillary return</td>
<td>80 mL/kg</td>
</tr>
<tr>
<td>Shock severely ill, thready pulse, poor perfusion</td>
<td>10 mL/kg stat</td>
</tr>
</tbody>
</table>

Enter deficit estimate (mL/kg) | 3 | mL/kg |
Calculate total deficit: Multiply 1 by 3 | 4 | mL |
If fluid bolus was given: then subtract 2 from 4 | 5 | mL |
Divide deficit over 48hr (divide 5 by 48) | 6 | mL/hr |

**Note:** Deficit given over 72 hours if Na+ corrected > 150 mmol/L or hyperosmolality > 310 mosm/L

### Maintenance Fluids

<table>
<thead>
<tr>
<th>Weight</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 10kg</td>
<td>4 mL/kg/hr</td>
</tr>
<tr>
<td>Second 10kg</td>
<td>2 mL/kg/hr</td>
</tr>
<tr>
<td>Every kg after 20kg</td>
<td>1 mL/kg/hr</td>
</tr>
</tbody>
</table>

Total maintenance fluids | 7 | mL/hr |
Calculate total hourly fluid rate: add 6 and 7 |  | mL/hr |
WORKED EXAMPLE: 34kg child, 10ml/kg fluid bolus (shock), 8% dehydrated

<table>
<thead>
<tr>
<th>Body weight in kg</th>
<th>34 kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fluid bolus given</td>
<td>340 mL</td>
</tr>
</tbody>
</table>

Deficit – fluid bolus already given (given over 48hrs)

<table>
<thead>
<tr>
<th>No signs of dehydration (tolerating fluids orally)</th>
<th>Moderate 5% Dry mucous membranes, reduced skin turgor</th>
<th>Severe 8% Above with sunken eyes &amp; poor capillary return</th>
<th>Shock severely ill, thready pulse, poor perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continue with oral rehydration</td>
<td>50 mL/kg</td>
<td>80 mL/kg</td>
<td>10 mL/kg stat</td>
</tr>
</tbody>
</table>

Enter deficit estimate (mL/kg) | 80 mL/kg |

Calculate total deficit: Multiply 34 kg by 80 mL/kg | 2720 mL |

If fluid bolus was given:

then subtract 340 mL from 2720 mL | 2380 mL |

Divide deficit over 48hr (divide 2380 mL by 48) | 50 mL/hr |

Note: Deficit given over 72 hours if Na⁺ corrected > 150 mmol/L or hyperosmolality > 310mosm/L

Maintenance Fluids

<table>
<thead>
<tr>
<th>Weight</th>
<th>First 10kg</th>
<th>4 mL/kg/hr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Second 10kg</td>
<td>2 mL/kg/hr</td>
</tr>
<tr>
<td></td>
<td>Every kg after 20kg</td>
<td>1 mL/kg/hr</td>
</tr>
</tbody>
</table>

Total maintenance fluids | 64 mL/hr |

Calculate total hourly fluid rate: add 50 mL/hr and 64 mL/hr | 114 mL/hr |
Appendix 3 – DKA Flowchart

Child presents to emergency service with clinical features suggesting DKA
Check finger prick BSL & ketones:
- Consider pre-hospital management given
- Assess for signs of dehydration and circulatory or neurological compromise
- Investigations – VBG, FBC, U&E’s, ketones (urine & blood)
- On confirmation of diagnosis, contact senior medical staff

Assessment

- No clinical signs of dehydration
  - NRV DKA (pH 7.25 to 7.3)
  - Clinically well
  - Tolerating oral fluid

- Clinical signs of dehydration
  - No signs of shock present
    - Clinically acidic (hyperventilation)
    - Vomiting

Initiate Therapy

- Start with SC insulin (see below)
- Continue oral hydration

Clinical & biochemical improvement?

No

- Transfer to Paediatric Inpatient Service

Yes

- Commence low-dose continuous insulin infusion at 0.1 units/kg/hr
  - Consider 0.05 units/kg/hr for young child < 5
  - Commence 1 hour after start of IV therapy

Critical Observations

- Hourly BSL, RR, HR, BP
- Hourly fluid input and output
- Neurological status at least hourly
- U & Es/VBG 2-4 hrly after start of IV therapy
- Monitor ECG for T-wave changes

Acidosis not improving

Re-evaluate

- IV fluid calculations
- Insulin delivery system & dose
- Need for additional fluid resuscitation
- Consider sepsis

- Transfer to Local Paediatric Inpatient Service or Endocrinology Unit / PICU at Level 6 Service as per Criteria on next page

Blood glucose < 15 mmol/L

IV Therapy

- Change to 5% Glucose + 0.9% NaCl + 40 mmol KCl (per 1 L bag)
  - May need to adjust insulin infusion if above ineffective (not < 0.05 units/kg/hr)

- Go to IV hypertonic saline (3% NaCl) 3-5 mL/kg over 30 mins OR mannitol 0.5-1.0 g/kg (over 20 mins)

Warning signs include:
- headache or irritability
- slowing heart rate
- decreased conscious level
- incontinence
- specific neurological signs

- Exclude hypoglycaemia as cause
- Is it cerebral oedema?

Management

- Give IV Mannitol 0.5-1.0 g/kg (over 20 mins) or IV 3% NaCl 3-5 mL/kg over 30 mins
- Restrict IV fluids by one third
- Call senior staff
- Transfer to ICU
- Consider CT/MRI - only after patient stabilised

Improvement?

Clinical well
- Tolerating oral fluids
- Transition to SC insulin

Transition to IV insulin
- Start SC insulin then stop IV insulin 90 mins later

- Re-evaluate
- IV fluid calculations
- Insulin delivery system & dose
- Need for additional fluid resuscitation
- Consider sepsis

- Transfer to Local Paediatric Inpatient Service or Endocrinology Unit / PICU at Level 6 Service as per Criteria on next page

Confirmation of Diagnosis DKA
Biochemical signs of DKA include:
- Ketonuria/ketonaemia
  - BSL > 11 mmol/L
- pH < 7.25, Bicarb < 15 mmol/L

Signs of shock present

- Reduced peripheral pulses
- Reduced conscious level/soma

Emergency Management (Resuscitate using ABC)

- Call emergency &/or paediatric consultant
- Support ventilation (BVM)
- Consider ETT intubation if not responding
- Give IV fluid boluses 0.9% NaCl 10 mL/kg aliquots as required
- Neurological deterioration present – give IV/hypertonic saline (3% NaCl) 3-5 mL/kg over 30 mins OR mannitol 0.5-1.0 g/kg (over 20 mins)

- Exclude hypoglycaemia as cause
- Is it cerebral oedema?

- Give IV Mannitol 0.5-1.0 g/kg (over 20 mins) or IV 3% NaCl 3-5 mL/kg over 30 mins
- Restrict IV fluids by one third
- Call senior staff
- Transfer to ICU
- Consider CT/MRI - only after patient stabilised

Medications & IV Fluids

Ongoing treatment

Insulin (IV)

- Dose: 0.1 units/kg/hour
  - Syringe pump: Add 50 units of short-acting insulin (Actrapid or Humulin R) to 49.5 mL 0.9% NaCl (≈ 1 unit of insulin per mL)
  - No syringe pump: Add 50 units of short-acting insulin (Actrapid or Humulin R) to 500 mL 0.9% NaCl (~ 1 unit of insulin per 10 mL)

Insulin (SC)

- Dose: 0.1-0.2 units/kg, 4-6 hourly depending on response (Actrapid or Humulin R)

IV 0.9% Normal Saline with 40 mmol potassium

Use pre-mixed 1 litre bag of 0.9% NaCl + 40mmol KC1 as initial fluid unless neonatal DKA, hypernatremia (corrected Na), hypoglycaemia, anuria or hyperkalaemia in which case consultation with PICU required

IV 0.9% Normal Saline with 40mmol potassium and 5% glucose

Use pre-mixed 1 litre bag of 0.9% NaCl + 40mmol KCl, remove 100 mL and add 50 g (100 mL) of 50% dextrose as per http://www.qheps.health.qld.gov.au/childrenshealth/resources/guidelines/gdl-01025.pdf

Note: If higher concentrations of glucose required consult with PICU due to risk of hypernatremia and cerebral oedema