Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycaemic State (HHS) - Emergency management in children

Purpose

This document provides clinical guidance for all staff involved in the care and management of a child or adolescent with diabetic ketoacidosis (DKA) and hyperosmolar hyperglycaemic state (HHS) presenting to an Emergency Department (ED) in Queensland.

This guideline has been developed by senior ED clinicians and Paediatricians across Queensland, with input from Endocrinology, Critical Care and Pharmacy, Queensland Children’s Hospital, Brisbane. It has been endorsed for statewide use by the Queensland Emergency Care of Children Working Group in partnership with the Queensland Emergency Department Strategic Advisory Panel and the Healthcare Improvement Unit, Clinical Excellence Queensland.

Key points

DKA

- potentially fatal metabolic disorder.
- diagnosis requires hyperglycaemia (blood glucose level (BGL) greater than 11mmol/L), venous pH less than 7.3 and/or HCO3 less than 15 mmol/L and moderate/large ketonaemia/ketonuria.
- cerebral oedema is a rare but serious complication requiring urgent treatment and critical care.

HHS

- state of extreme hyperglycaemia (and hence hyperosmolality) without ketosis which is usually, but not exclusively, seen in type 2 diabetes.
- diagnosis requires hyperglycaemia (BGL greater than 33.3mmol/L), venous pH greater than 7.25 and/or HCO3 greater than 15 mmol/L, small ketonuria, absent to mild ketonemia less than 1.1mmol/L and effective serum osmolality greater than 320mOsm/kg.

Management of DKA and HHS

- involves fluid and electrolyte replacement therapy (more aggressive in HHS).
- seek urgent paediatric endocrine/critical care advice (onsite or via Retrieval Services Queensland (RSQ)) for patients with severe DKA or HHS.
Introduction

Diabetes ketoacidosis (DKA)

DKA is a metabolic disorder and the leading cause of morbidity and mortality in children and adolescents with type 1 diabetes. It is caused by a decrease in effective circulating insulin, insulin resistance and increased production of counter-regulatory hormones.\(^1\)\(^,\)\(^2\) 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.

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. Occasionally, patients diagnosed with diabetes may have symptomatic ketoacidosis without a raised blood glucose level.

Management of an episode of DKA is not complete until an attempt has been made to identify and treat the cause. DKA without a preceding febrile illness or gastroenteritis in a patient with known diabetes is almost always the result of psychosocial problems and failure to appropriately administer insulin.

Cerebral oedema is a rare but devastating complication of diabetes, occurring in approximately 1% of children with DKA. It is typically described as a sudden onset of rapidly progressing neurological deterioration including altered/fluctuating level of consciousness, headache, vomiting, bradycardia, hypertension, cranial nerve palsy and abnormal posturing.

Clinical cerebral oedema can occur at any time but most commonly occurs 4-12 hours after commencement of treatment.

Risk factors for cerebral oedema

- new onset Type 1 diabetes
- elevated serum urea
- severe dehydration
- severe DKA (pH less than or equal to 7.1)
- lower bicarbonate levels
- age less than five years
- reduced level of consciousness
Hyperglycaemic Hyperosmolar State (HHS)

HHS is a state of extreme hyperglycaemia (and hence hyperosmolality) without ketosis which is usually, but not exclusively, seen in type 2 diabetes. It can also occur in neonatal diabetes and in type 1 diabetes in children and adolescents with an intellectual impairment who are unable to indicate thirst. In young persons, it is much rarer than DKA, but it is rising as the incidence of type 2 diabetes increases.

Polyuria and polydipsia may not be recognised and especially in hot temperatures, there can be extreme dehydration, fluid loss and electrolyte disturbance. Dehydration can be difficult to assess clinically and the hyperosmolality preserves intravascular volume initially. Initial treatment can cause movement of fluid out of the intravascular compartment and shock. More aggressive fluid replacement than in DKA is required to expand the intra and extravascular volume, restore normal renal perfusion and promote a gradual decline in corrected serum sodium concentration and osmolality.

Complications of HHS

- venous thromboembolism associated with central venous catheters
- rhabdomyolysis secondary to hypophosphataemia (leading to kidney injury)
- malignant hyperthermia (rare)

Assessment

**DKA Diagnosis**

Requires ALL of the following:

- hyperglycaemia (BGL greater than 11mmol/L)
- venous pH less than 7.3 and/or HCO3 less than 15 mmol/L
- moderate/large ketonaemia/ketonuria

<table>
<thead>
<tr>
<th>DKA severity</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH &lt; 7.3, HCO3 &lt; 15 mmol/L</td>
<td>pH &lt; 7.3, HCO3 &lt; 10 mmol/L</td>
<td>pH &lt; 7.1, HCO3 &lt; 5 mmol/L</td>
<td></td>
</tr>
</tbody>
</table>

**Ketone readings and probability of DKA**

<table>
<thead>
<tr>
<th>Sample</th>
<th>Method of testing</th>
<th>Low/Small</th>
<th>Moderate</th>
<th>High/Large</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>Bedside meter Abott</td>
<td>Less than 0.6 mmol/L</td>
<td>0.6 to less than 1.5 mmol/L</td>
<td>Greater than or equal to 1.5 mmol/L</td>
</tr>
<tr>
<td>Urine</td>
<td>Bayer brand Keto-Diastix</td>
<td>0 mmol/L</td>
<td>0.5 to less than 1.5 mmol/L</td>
<td>Greater than or equal to 1.5 mmol/L</td>
</tr>
<tr>
<td></td>
<td>Accu-chek brand Keto-Diabur-Test 5000</td>
<td>Negative</td>
<td>Less than 1.0 mmol/L</td>
<td>Greater than or equal to 1.0 mmol/L</td>
</tr>
</tbody>
</table>
HHS Diagnosis

Requires ALL of the following:

- hyperglycaemia (BGL greater than 33.3 mmol/L)
- venous pH greater than 7.25 and/or HCO₃ greater than 15 mmol/L (lactic acid can cause a mild acidosis)
- small ketonuria
- absent to mild ketonemia less than 1.1 mmol/L
- effective serum osmolality greater than 320 mOsm/kg

Altered level of consciousness is usual but cerebral oedema is rare.

Risk factors include:

- obesity
- signs of insulin resistance (acanthosis nigricans)
- family history of type 2 diabetes

History

History should include specific information on:

- 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

Examination

Physical examination should include an assessment of:

- weight
- hydration
- respiration (hyperventilation is a feature of acidotic respiration)
- potential cerebral oedema (signs and symptoms include headache, irritability, slowing pulse, rising BP and reducing level of consciousness. Papilloedema is a late sign)
- potential infection including appendicitis, ileus and pancreatitis

Consider sepsis in child with DKA and fever or shock (see Sepsis Guideline)
Dehydration assessment
Includes assessment of BP, pulse rate and volume, perfusion (capillary refill time, skin colour, mentation), mucous membranes and tissue turgor. Volume deficit is difficult to assess accurately in DKA, particularly in the young child.

ALERT – Volume deficit in DKA is often overestimated which may lead to over resuscitation with IV fluids. In contrast, significant fluid deficits are present in HHS and fluid resuscitation should be aggressive.

Severity of dehydration and estimation of volume deficit

<table>
<thead>
<tr>
<th>Mild ~ 3%</th>
<th>Moderate ~ 5%</th>
<th>Severe ~ 8%</th>
<th>Life -threatening - shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only just clinically detectable</td>
<td>Dry mucous membranes, reduced skin turgor</td>
<td>Dry mucous membranes, reduced skin turgor, sunken eyes, poor capillary return</td>
<td>Severely ill with poor perfusion, thready rapid pulse (reduced BP is a very late sign)</td>
</tr>
</tbody>
</table>

Specific considerations in DKA
- tachypnoea secondary to acidosis can exacerbate dryness of oral mucosa
- vasoconstriction from acidosis may contribute to the appearance of cool extremities
- catabolism due to insulin deficiency can result in weight loss

Investigations

Urgent baseline investigations for the management of DKA and HHS
- BGL (may be inaccurate via finger prick in circulatory compromise and acidosis)
- Finger-prick blood ketones (superior to urinary ketones)
- Urine ketones only if blood ketones not available
- Urea and electrolytes (serum urea greater than 9.0 mmol/L may indicate severe dehydration)
- Venous pH and acid-base status
- HbA1C (for later analysis)

Additional tests required in a child with newly diagnosed diabetes include:
- TFT (thyroid screen)
- Total IgA and TTG (coeliac screen)

Other investigations including FBC, urine M/C/S, CXR, CSF M/C/S, throat swab, and blood culture may be required on senior emergency/pediatric advice for a child who is hypothermic, hypotensive or has a refractory acidosis or lactic acidosis.

Note that an elevated WCC is common in DKA and does not necessarily indicate sepsis.
Management of DKA

Refer to Appendix 1 for a summary of the emergency management of a child with DKA.

Emergency care should always involve a rapid primary survey with evaluation of (and immediate management of concerns with) airway, breathing, circulation and disability (ABCD).

Aims of treatment

DKA is characterised by a 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.\(^3\),\(^4\)

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

Management of moderate to severe DKA

Refer to Appendix 2 for a summary of the ongoing management of a child with moderate to severe DKA.

Initial management

Shock at presentation

Child is severely ill with poor perfusion and thready rapid pulse.

<table>
<thead>
<tr>
<th>Fluid resuscitation (IV) for the management of shocked children with DKA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dose</strong></td>
</tr>
</tbody>
</table>

There is no evidence to support the use of colloids/volume expanders over crystalloids.

Seek urgent Paediatric Critical Care advice (onsite or via Retrieval Services Queensland (RSQ)) for a child in shock requiring two or more fluid boluses. Inotropes may be required.

Altered level of consciousness at presentation

Altered level of consciousness is directly related to degree of acidosis. However, consider instituting cerebral oedema management (outlined below) if signs of raised ICP.

IV rehydration fluids and insulin therapy

IV fluids and insulin are the recommended initial management.
If necessary, use Ondansetron. Other antiemetics are not recommended due to sedation/neurological side effects which may make assessments for onset of cerebral oedema difficult.

All patients with moderate to severe DKA should initially remain ‘nil by mouth’ except for ice to suck.

Consider a nasogastric tube if gastric paresis is present (vomiting caused by non-mechanical delayed gastric emptying associated with the DKA illness).

Oral fluids should only be offered after substantial clinical improvement (i.e. blood sugar less than 15mmol/l and level of consciousness has improved if initially reduced) and no vomiting. If this occurs prior to the completion of the 48-hour rehydration period, proceed with oral intake and reduce IV infusions.

Seek Paediatric Endocrine/Critical care advice (onsite or via RSQ) in the following cases:
- age less than 5 years
- hypernatraemia
- hyperosmolality
- anuria
- hyperkalaemia

Calculate fluid replacement based on dehydration assessment (See Appendix 2).

\[
\text{Requirement} = \text{Maintenance} + (\text{Deficit} – \text{fluid bolus already given}) \text{ over 48hrs}
\]

Refer to Fluid Therapy Calculation Worksheet (Appx 3).

Urinary losses should not be added to the initial calculation of replacement fluids.

Use 1 litre Sodium Chloride 0.9% + Potassium Chloride 40mmol (pre-mixed bag) as the initial default fluid unless anuria (after catheterisation) or hyperkalaemia (greater than 5.5 mmol/L) is present. If either of these are present, use Sodium Chloride 0.9% as per specialist advice.

**ALERT** – Miscalculations of added potassium have resulted in deaths. Fluids that require potassium to be added e.g. Plasmalyte or Compound Sodium Lactate solution (Hartmann’s or Ringer’s lactate) should only be used in the critical care setting.

**Insulin**

**ALERT**
- Calculate insulin doses carefully as very serious errors can occur.
- Never give bolus doses of insulin IV or insulin IM.
- Start insulin therapy **one hour after** commencing fluid therapy.

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, insulin IV is required.

Only short-acting insulins (examples include but are not limited to Actrapid or Humulin R) should be used for insulin IV administration. The insulin infusion set should be changed every 24 hours due to the potential for the insulin to denature.

If a patient on an insulin pump presents in DKA, the pump should be stopped, and an assumption made that there is a pump problem. The pump should only be restarted on advice from a Paediatric Endocrinologist or local equivalent with a new site and a new set recommended.
### Initial IV insulin infusion for the treatment of moderate to severe DKA in children

| Short-acting insulin dose | Continuous insulin IV infusion dose is ideally 0.1 units/kg/hr. Seek specialist advice for dosing in obese patients. It may be prudent to base insulin infusion on ideal body weight. There is no evidence to support an initial infusion dose of 0.05 units/kg/hr however it may be considered in infants and very severe DKA. If using a syringe pump: Add 50 units (0.5 mL) to 49.5 mL of Sodium Chloride 0.9% in a syringe. [Insulin concentration = 1 U/mL]. Infusion to be delivered by syringe pump into the side arm of the IV line. If no syringe pump available: Add 50 units (0.5 mL) to a 500 mL bag of Sodium Chloride 0.9%. [Insulin concentration = 0.1U/ml] The infusion should be delivered using a volumetric pump into the side arm of the IV line. If this is not available a separate IV site may be required for low infusion rates. |

| Monitoring | All children on insulin IV must have hourly BGLs. |

### Ongoing management

Refer to Appendix 3 for a summary of the ongoing management of a child with moderate to severe DKA.

If acidosis fails to improve or BGL rises, consider insulin error, inadequate resuscitation or alternative diagnosis including sepsis, drug overdose (such as salicylate, other prescription drugs or recreational drugs) or hyperchloraemic acidosis.

Seek urgent Paediatric Critical Care advice (onsite or via RSQ) if:
- acidosis fails to improve after two hours
- BGL rises
## Ongoing management of child with moderate to severe DKA

<table>
<thead>
<tr>
<th>BGL</th>
<th>Fluids</th>
<th>Insulin</th>
</tr>
</thead>
</table>
| Falls at rate of greater than 5 mmol/L/hr | BGL will often fall quickly because of rehydration.  
No evidence supports the practice of adding glucose to protect against cerebral oedema.  
Only add glucose if BGL is less than or equal to 15 mmol/L (see below). | DO NOT reduce rate. |
| Falls to less than or equal to 15 mmol/L | Add Glucose 5% to IV fluid bag of Sodium Chloride 0.9% + Potassium Chloride 40 mmol (will need to be mixed onsite) to prevent hypoglycaemia. | DO NOT reduce rate.  
The insulin dose needs to be ideally maintained at 0.1 units/kg/hr to switch off ketogenesis. |
| Issues maintaining 5-10 mmol/L despite running a solution containing Glucose 5% | Increase glucose concentration to Sodium Chloride 0.9% + Glucose 10% + Potassium Chloride 40 mmol.  
Seek specialist advice when mixing solution as some mixtures are significantly hyponatraemic and may contribute to cerebral oedema.  
Monitor site for local reactions as solution is hypertonic. | Only reduce the rate if BGL remains below the target range despite this glucose supplementation.  
Note problems with hypoglycaemia can occur if there has been a miscalculation of the insulin dose. Consider preparing the insulin infusion again and recommencing. |
| Falls below 4 mmol/L | Administer a bolus of 2 mL/kg of Glucose 10% over three minutes.  
Ensure fluid running has Glucose 5% and consider Glucose 10%. | Temporarily reduce by 50% and seek urgent paediatric endocrine/critical care advice.  
DO NOT stop the infusion.  
It takes ~20 minutes for insulin infusion cessation to take clinical effect so will not assist in acute hypoglycaemia.  
Ongoing insulin administration is necessary while Glucose is being infused, as insulin is required to switch off ketone production. |
Seek Paediatric Endocrine/Critical Care advice (onsite or via RSQ)

Seek senior Paediatric/Endocrine advice as per local practice

Consider seeking Paediatric/Endocrine advice as per local practice

**ALERT** – Glucose 50% is extremely hypertonic and should **NOT** be administered without dilution

**Electrolyte considerations in IV fluid management**

**Sodium replacement and osmolality**

Seek Paediatric Endocrine/Critical Care advice (onsite or via RSQ) if hypernatraemia (Na+ greater than 150 mmol/L) and/or hyperosmolarity (greater than 310 mosm/L).

Correction of dehydration and electrolyte abnormalities should occur over 72 hours.

Hypotonic solutions may be associated with raised intracranial pressure (ICP).

**Potassium replacement**

Plan for the predictable fall in the serum potassium concentration after insulin therapy commences and the ketoacidosis starts to reverse. 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 potassium levels. The maximum potassium concentration should be 40 mmol/L.

Do not exceed a maximal potassium infusion rate of 0.3 mmol/kg/hr without consultation.

Potassium replacement should continue throughout IV fluid therapy.

**ALERT** – Miscalculations of added potassium have resulted in deaths. Outside of the ICU/PICU setting, use pre-mixed bags with Potassium Chloride 40 mmol/L.

Check potassium measurements every two hours (iStat, blood gas or formal U&Es). All patients with DKA must be on a cardiac monitor while in ED to alert clinicians to arrhythmias and ECG/T wave changes.

- Hypokalaemia causes T wave flattening. If hypokalemia occurs, temporarily reduce insulin infusion rate by 50% and discuss with Paediatric Critical Care 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 (greater than or equal to 1 ml/kg/hour) occurs and the potassium level falls to the top of the normal range. Reducing the potassium replacement is done by changing the fluid to Sodium Chloride 0.9% with Potassium Chloride 20 mmol/L which is available as a pre-mix bag.

**Bicarbonate replacement**

Severe acidosis is usually reversible by fluid and insulin administration. Bicarbonates only purpose is to improve cardiac contractility in severe shock.

**ALERT** – Sodium bicarbonate is **not** routinely recommended due to increased risk of cerebral oedema. The decision to administer sodium bicarbonate to a child with DKA **must** be made in consultation with a paediatric intensivist/endocrinologist at a Level 6 facility.
Resolution of acidosis
The dose of insulin should remain at 0.1 units/kg/hr at least until the resolution of acidosis (pH greater than 7.3, HCO3 greater than 15 mmol/L) and/or closure of anion gap. As the resolution of ketosis takes much longer, ketosis alone will not delay the transition to SC insulin.

Seek specialist advice on transitioning to SC insulin providing all of following have occurred:
- resolution of acidosis
- clinical improvement and no vomiting
- tolerating oral fluids

Insulin IV infusion must continue for one hour after administration of SC insulin.

Clinical and laboratory monitoring
Refer to the Queensland DKA Subcutaneous Insulin Order and Blood Glucose Level Record Form.

The monitoring outlined below should continue until the child is well.

<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>2-4th hourly or hourly if febrile</td>
<td></td>
</tr>
<tr>
<td>Capillary (fingerprick)</td>
<td>Hourly</td>
<td>Blood ketones aid in determining resolution of DKA.</td>
</tr>
<tr>
<td>BGL</td>
<td></td>
<td>The bedside meter (Abbott brand) ketone readings greater than 4 mmol/L are less accurate.</td>
</tr>
<tr>
<td>Blood ketones</td>
<td></td>
<td>DO NOT use either blood or urinary ketones alone as the indicator for changes to fluid or insulin regimes. Assess the whole child.</td>
</tr>
<tr>
<td>Laboratory bloods:</td>
<td>2-4th hourly Consider hourly electrolyte monitoring in severe DKA.</td>
<td>Capillary glucose methods may be inaccurate if poor peripheral circulation or acidosis.</td>
</tr>
<tr>
<td>Venous glucose</td>
<td></td>
<td>Consider an IV cannula for repetitive blood sampling. An IA line may be necessary in some critically ill patients managed in PICU/ICU</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</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Consider a urinary catheter if impaired level of consciousness.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>In non-toilet trained children, nappies should be weighed to ensure accurate fluid balance</td>
</tr>
<tr>
<td>Neurological observations</td>
<td>Hourly or more if high risk</td>
<td>See below for groups at high risk of cerebral oedema.</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 and daily</td>
<td></td>
</tr>
</tbody>
</table>
Management of cerebral oedema

If cerebral oedema is suspected initiate immediate treatment and urgently seek Paediatric Critical Care advice (onsite or via RSQ). Do not delay for Neurology consultation or imaging.

When to suspect cerebral oedema

Warning signs and symptoms:
- headache
- inappropriate slowing of heart rate
- recurrence of vomiting
- change in neurological status (restlessness, irritability, increased drowsiness, incontinence)
- specific neurological signs (such as cranial nerve palsies, pupillary response)
- rising BP
- decreased oxygen saturation

Biochemical red flags:
- rapid fall in the calculated osmolarity with treatment (usually serum sodium rises as the glucose falls resulting in a relatively stable calculated osmolarity)
- development of hyponatraemia during therapy or rapidly falling sodium
- initial sodium in the hypernatraemic range

Immediate management of cerebral oedema
- raise the head of the bed to 20°
- administer high-flow oxygen via a non-rebreathing mask with a reservoir bag
- reduce the rate of fluid administration as per specialist advice
- administer Mannitol or Hypertonic Saline 3% IV in patients with signs of cerebral oedema before impending respiratory failure6
- consider intubation and ventilation. Aim for CO2 35-40mmHg. Aggressive hyperventilation has been associated with poor outcome in retrospective studies of DKA related cerebral oedema7

**ALERT** – Intubation and ventilation of a patient with severe DKA is an extremely high-risk procedure. This must be discussed with a critical care specialist.

Sodium Chloride 3% (IV) dosing for the treatment of raised ICP

<table>
<thead>
<tr>
<th>Sodium Chloride 3% (Hypertonic Saline 3%) (IV)</th>
<th>3 mL/kg/dose (1–5 mL/kg/dose) over 10-15 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risks</td>
<td>Rebound ICP</td>
</tr>
<tr>
<td></td>
<td>Central pontine myelinosis</td>
</tr>
<tr>
<td></td>
<td>Subarachnoid haemorrhage</td>
</tr>
<tr>
<td></td>
<td>Renal failure</td>
</tr>
</tbody>
</table>
Management of mild DKA

The following management is recommended in a child who meets ALL of the following criteria:

- clinically well (stable vital signs, normal GCS)
- tolerating oral fluids and normal perfusion
- less than 5% dehydrated
- pH between 7.2 and 7.3

![ALERT – Insulin pump therapy should be discontinued in mild DKA.]

Insulin

Seek Paediatric/Endocrine advice as per local practice prior to administering insulin.

**Subcutaneous insulin for the management of children with mild DKA**

| Short-acting insulin (Actrapid or Humulin R) OR Ultra-short acting insulin analog (Humalog [lispro] or NovoRapid [aspart]) | 0.1 – 0.2 units/kg every four to six hours depending on the response. For children less than 5 years of age, 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. |

Clinical monitoring

Refer to the Queensland DKA Subcutaneous Insulin Order and Blood Glucose Level Record Form.

Clinical reassessment of the child at frequent intervals is mandatory.
Management of HHS

Urgent specialist Paediatric Critical Care/Endocrine advice must always be sought for HHS. The following provides an outline to guide initial management while awaiting specialist advice.

Emergency assessment and management should always involve a rapid primary survey with evaluation and management of airway, breathing, circulation and disability (ABCD).

**Shock at presentation**

Patient is severely ill with poor perfusion and thready rapid pulse. Occurs more commonly than in DKA.

### Fluid resuscitation (IV) for the management of shocked children with HHS

| Bolus dose | Sodium Chloride 0.9% administered in 20 mL/kg bolus. Repeat boluses as needed to reverse shock. |

**IV rehydration fluids**

**ALERT** – The management of fluids and electrolytes in HHS is extremely complex and early specialised PICU Consultation is imperative.

Assume 12-15% fluid deficit as starting point for patients presenting with HHS.

**Fluid Requirement = Maintenance + (Deficit – to be given over 48hrs) + Urinary Losses**

Use Sodium Chloride 0.9% + Potassium Chloride 40 mmol (pre-mixed bag) as initial default fluid unless:

- potassium is greater than 5.5 mmol/L
- renal function is compromised
- the patient is anuric

If any of these three are present, use Sodium Chloride 0.9% with no added Potassium Chloride as initial fluid, and seek urgent advice.

Replace urinary losses with Sodium Chloride 0.45%.

Fluid replacement alone will reduce the Glucose by 4-5 mmol/hour (measure hourly). If level falls faster the intensivist will consider adding glucose to the bag, however this should only be done on critical care advice.

**Start insulin** 0.025-0.05 u/kg/hour ONLY WHEN the BGL is not falling with rehydration alone. Titrate to achieve reduction of blood glucose by no more than 3-4 mmol/hour.

Accurate fluid balance is critical to guide initial and ongoing management. Urinary catheter insertion or strict fluid balance with weighing of nappies or measuring all output is recommended.
Complications of HHS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venous thromboembolism</td>
<td>Consider prophylaxis in patient with central venous catheter</td>
</tr>
</tbody>
</table>
| Rhabdomyolysis secondary to hypophosphataemia  | Measure phosphate, calcium, magnesium and creatinine kinase levels every two hours.  
                                                                                                       • In hypophosphataemic states, replace phosphate as IV potassium phosphate/potassium chloride mixture 0.2-0.5 mmol/kg in 24 hours if less than 0.5 mmol/L  
                                                                                                       • In hypomagnesaemia, replace magnesium as magnesium chloride 25-50 mg/kg/dose for four doses (administered every six hours) |
| Malignant hyperthermia                        | Occurs rarely. Discuss with Critical Care. Dantrolene may be required.      |

Escalation outside of ED

Clinicians can contact the services below if escalation of care outside of senior clinicians within the ED is needed, as per local practices. Transfer is recommended if the child requires a higher level of care.

Critically unwell or rapidly deteriorating child

<table>
<thead>
<tr>
<th>Service</th>
<th>Reason for contact by clinician</th>
<th>Contact</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paediatric Critical Care</td>
<td>For urgent advice/assistance for children with severe DKA and any of: • shock requiring two or more fluid boluses • suspected cerebral oedema • age less than 5 years For urgent advice/assistance in all children with HHS.</td>
<td>Onsite or via Retrieval Services Queensland (RSQ). For facilities with no onsite service contact RSQ on 1300 799 127: • for access to specialist telephone advice • to coordinate the retrieval of a critically unwell child Notify early of children potentially requiring transfer. In the event of retrieval, inform your local Paediatric service.</td>
</tr>
<tr>
<td>Paediatric Endocrinology</td>
<td>For advice on the management of severe DKA including the following high-risk groups: • first presentation • suspected cerebral oedema • hypernatraemia • hyperosmolality • hyperkalaemia • HHS</td>
<td>Onsite or via RSQ (see above).</td>
</tr>
<tr>
<td>Service</td>
<td>Reason for contact by clinician</td>
<td>Contact</td>
</tr>
<tr>
<td>-------------------------------</td>
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<td>-------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| Immediate assistance with airway | For onsite help with the management of airway, including intubation and ventilation.          | The most senior resources available onsite at the time as per local practices. Options may include:  
  • paediatric critical care  
  • critical care  
  • anaesthetics  
  • paediatrics |

**Non-critical child**

<table>
<thead>
<tr>
<th>Reason for contact by clinician</th>
<th>Contact</th>
</tr>
</thead>
<tbody>
<tr>
<td>For specialist advice prior to administering insulin in child with mild DKA.</td>
<td>Onsite/local endocrinologist or onsite/local paediatric service as per local practice</td>
</tr>
<tr>
<td>For specialist advice on the management, disposition and follow-up of other children with DKA as required.</td>
<td></td>
</tr>
<tr>
<td>To inform the treating service of the ED presentation for all children previously diagnosed with diabetes (regardless of the presenting condition).</td>
<td></td>
</tr>
</tbody>
</table>

For assistance with local inter-hospital transfers of non-critical patients. Onsite/local paediatric service as per local practice.

For assistance with inter-hospital transfer of non-critical patients into and out of Queensland Children’s Hospital. View QH Inter-hospital transfer request form (QH only) Children’s Advice and Transport Coordination Hub (CATCH) (07) 3068 4510 (24-hour service).

For assistance with decision making regarding safe and appropriate inter-hospital transfer of children in Queensland. View the Queensland Paediatric Transport Triage tool – Medical or call CATCH on (07) 3068 4510 (24-hour service).

For access to generalist and specialist acute support and advice via videoconferencing, as per locally agreed pathways, in regional, rural and remote areas in Queensland. Telehealth Emergency Management Support Unit (TEMSU) 1800 11 44 14 (24-hour service) TEMSU (access via QH intranet).

To request aeromedical inter-hospital transfer in Queensland. Retrieval Services Queensland (RSQ) 1300 799 127 (24-hour service) RSQ (access via QH intranet).
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 facility with all of the following:

- 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

Transfer to a Paediatric Critical Care service may be considered for the following:

- lack of appropriate staff/facilities to care of a 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 less than five years of age and new onset)

All patients with severe DKA and HHS will require admission to a Paediatric Critical Care service.

Related documents

Guidelines

- ISPAD (International Society for Paediatric and Adolescent Diabetes) Clinical Practice Consensus Guidelines 2018

References

# Guideline approval

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<td></td>
<td>Effective date</td>
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<td>Review date</td>
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## Keywords
- Diabetes Ketoacidosis, DKA, Hyperosmolar Hyperglycaemic State, HHS, hyperglycaemia, ketonaemia, ketonuria, paediatric, emergency, guideline, children, 60016

## Accreditation references
- NSQHS Standards (1-8): 1, 4, 8

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The guideline is not a substitute for clinical judgement, knowledge and expertise, or medical advice. Variation from the guideline, taking into account individual circumstances may be appropriate.

This guideline does not address all elements of standard practice and accepts that individual clinicians are responsible for:
- Providing care within the context of locally available resources, expertise, and scope of practice
- Supporting consumer rights and informed decision making in partnership with healthcare practitioners including the right to decline intervention or ongoing management
- Advising consumers of their choices in an environment that is culturally appropriate and which enables comfortable and confidential discussion. This includes the use of interpreter services where necessary
- Ensuring informed consent is obtained prior to delivering care
- Meeting all legislative requirements and professional standards
- Applying standard precautions, and additional precautions as necessary, when delivering care
- Documenting all care in accordance with mandatory and local requirements

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CHQ-GDL-60016 – Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycaemic State (HHS) - Emergency management in children
Appendix 1

CHQ-GDL-60016 – Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycaemic State (HHS) - Emergency management in children

Child with DKA diagnosis
(BGL>11 mmol/L, pH<7.3 +/- HCO3 <15mmol/L & moderate or large ketonuria/ketonaemia)

Assessment
Including signs of dehydration (see Box A) and circulatory or neurological compromise.
Additional investigations: U&E, HbA1C. If a new diagnosis — TFT, coeliac screen

Mild
pH 7.2-7.3 or HCO3<15 mmol/L
No/mild dehydration

- Oral fluids
- Insulin: SC insulin on Paediatric/Endocrine advice
- Monitor:
  - Hourly: HR, RR, strict fluid balance, and neuro obs
  - 4-hourly: temperature & BP
  - standard BGL

Moderate to severe
pH<7.2 +/- HCO3 <10 mmol/L

Seek paediatric endocrine/critical care advice if:
- age < 5 years
- electrolyte abnormalities

IV Rehydration
- Calculate IV fluid requirements
- Aim to correct over 48 hours
- Use 1 L Sodium Chloride 0.9% + 40 mmol Potassium Chloride (pre-mixed)
- Ice to suck otherwise nil by mouth
- Consider NG tube (if gastric paresis)

Insulin
- After 1 hour of IV fluids commence low-dose continuous Insulin infusion at 0.1 units/kg/hr

Resuscitate using ABCD
- Oxygen via NRBM
- Support ventilation (BVM)
- +/- ETT
- IV or IO access
- IV fluid bolus 10 mL/kg Sodium Chloride 0.9%, repeat to maximum of 20 mL/kg
- Monitor for signs of cerebral oedema
- Consider sepsis

Management if suspect cerebral oedema (Box B)
- Raise head of bed to 20°
- Restrict fluids by one third
- Consider hyperosmolar agents (Hypertonic saline/Mannitol)
- Active airway management to avoid hypercarbia (aim for pCO2 35-40 mmHg)
- Consider CT/MRI once stabilised

IV fluid and insulin therapy as per moderate to severe DKA
Clinical & biochemical improvement?

- Yes
  - Refer to appropriate inpatient service
- No
  - Refer to Ongoing Management DKA Flowchart

Responding to treatment?

- Yes
  - Refer to Paediatric Critical Care (onsite or via RSQ)
- No

Clinical & biochemical improvement?

- Yes
  - Refer to Paediatric Critical Care (onsite or via RSQ)
- No

Resuscitate using ABCD
- Oxygen via NRBM
- Support ventilation (BVM)
- +/- ETT
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Clinical & biochemical improvement?

- Yes
  - Refer to appropriate inpatient service
- No

Responding to treatment?
Appendix 2

Acidosis not improving after 2 hours or BGL rises

Resolution of acidosis (pH>7.3, HCO₃>15mmol/L)

Suspected cerebral oedema (Box A)

Ongoing management of child with moderate to severe DKA

<table>
<thead>
<tr>
<th>BGL</th>
<th>Fluids IV</th>
<th>Insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Falls at rate of greater than 5 mmol/L/hr</td>
<td>Only add Glucose if BGL is less than or equal to 15 mmol/L (see below)</td>
<td>DO NOT reduce rate</td>
</tr>
<tr>
<td>Falls to less than or equal to 15 mmol/L</td>
<td>Add Glucose 5% to Sodium Chloride 0.9% + 40 mmol Potassium Chloride</td>
<td>DO NOT reduce rate</td>
</tr>
<tr>
<td>Issues maintaining 5-10 mmol/L despite running a solution containing Glucose 5%</td>
<td>Increase the Glucose concentration to Sodium Chloride 0.9% + Glucose 10% + 40 mmol Potassium Chloride/L</td>
<td>Only reduce the rate if BGL remains below the target range despite this glucose supplementation. Consider Insulin error (infusion may need to be made up again and recommenced).</td>
</tr>
<tr>
<td>Falls below 4 mmol/L</td>
<td>Administer a bolus of 2 mL/kg of Glucose 10% over 3 minutes. Ensure fluid running has Glucose 5% and consider Glucose 10%</td>
<td>Temporarily reduce by 50% and seek urgent specialist advice. DO NOT stop infusion.</td>
</tr>
</tbody>
</table>

Management of possible clinical scenarios

**Clinical improvement and no vomiting**
- Offer oral fluids
- Continue insulin infusion

**Tolerating oral fluids**
- Commence SC insulin on specialist advice
- Continue insulin infusion for 1 hour after administration of SC insulin
- Cease insulin infusion on specialist advice

**Acidosis not improving after 2 hours or BGL rises**
- IV fluid calculations
- Insulin delivery system and dose
- Need for additional fluid resuscitation Consider:
  - sepsis
  - hyperchloraemic acidosis
  - drug overdose (such as salicylate or recreational drugs)

**Suspected cerebral oedema (Box A)**
- Exclude hypoglycaemia as cause for neurological deterioration
- Immediate management of suspected cerebral oedema
  - Raise head of bed to 20°
  - Restrict fluids as per specialist advice
  - Consider hyperosmolar agents (Hypertonic saline/Mannitol)
  - Active airway management to avoid hypercarbia (aim for pCO₂ 35-40 mmHg)
  - Consider CT/MRI once stabilised

**Ongoing clinical concern?**

Refer to Paediatric Critical Care (onsite or via RSQ)

**BOX A: When to suspect cerebral oedema**

**Signs and symptoms**
- headache
- inappropriate slowing of heart rate
- recurrence of vomiting
- change in neurological status (restlessness, irritability, increased drowsiness, incontinence)
- specific neurological signs
- rising BP
- decreased oxygen saturation

**Biochemical red flags:**
- rapid fall in the calculated osmolarity with treatment (usually serum sodium rises as the glucose falls resulting in a relatively stable calculated osmolarity)
- development of hyponatraemia during therapy or rapidly falling sodium
- initial sodium in the hypernatraemic range

**Consider seeking senior paediatric/endocrine advice as per local practice.**

**Seek senior paediatric/endocrine advice as per local practice.**

**Seek urgent paediatric critical care / endocrine advice (onsite or via Retrieval Services Queensland (RSQ) on 1300 799 127)**
Appendix 3

CHQ-GDL-60016 – Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycaemic State (HHS) -
Emergency management in children

Body weight in kg: ............................................ ① 34 kg
Total fluid bolus given ............................................ ② 340 mL

Deficit – fluid bolus already given (given over 48hrs)
No signs of dehydration (tolerating fluids orally) Continue with oral rehydration
Moderate 5% Dry mucous membranes, reduced skin turgor 50 mL/kg
Severe 8% Above with sunken eyes & poor capillary return 80 mL/kg
Shock severely ill, thready pulse, poor perfusion 10 mL/kg stat

Enter deficit estimate (mL/kg) ........................................ ③ 80 mL/kg
Calculate total deficit: Multiply ① by ③ .............................. ④ 2720 mL

If fluid bolus was given:
then subtract ② from ④ ........................................... ⑤ 2380 mL

Divide deficit over 48hr (divide ⑤ by 48) ... ⑥ 50 mL/hr

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

Maintenance Fluids
Weight: First 10kg 4 mL/kg/hr
Second 10kg 2 mL/kg/hr
Every kg after 20kg 1 mL/kg/hr
Total maintenance fluids ........................................... ⑦ 74 mL/hr

Calculate total hourly fluid rate: add ⑥ and ⑦ .......................... ⑧ 124 mL/hr