Aim
This protocol outlines the assessment and management of Diabetic Ketoacidosis (DKA) in children and adolescents.

Introduction
DKA is characterised by the triad of hyperglycaemia, metabolic acidosis and increased total body ketone concentration. DKA results from absolute or relative deficiency of circulating insulin and the effects of increased levels of counter-regulatory hormones.

The biochemical criteria for the diagnosis of DKA
1. Hyperglycaemia – blood glucose > 11 mmol/L
2. Ketonaemia
3. Acidosis – venous pH <7.3 or bicarbonate < 15mmol/L

Where to manage patients with DKA
- Patients with a pH <7.2 at presentation require admission to Paediatric Intensive Care Unit (PICU) at PMH. Infants and young children and patients with complications may also require admission to PICU with a pH >7.2.
- Other patients with a pH >7.2 may be managed on the ward with subcutaneous insulin.

Risk
DKA is life threatening and requires immediate treatment

DKA is the most common cause of diabetes-related deaths in children and adolescents. Most deaths in DKA occur as a result of cerebral oedema.¹

Causes and precipitants
- Insufficient insulin: newly diagnosed
- Missed insulin dose(s)
- Infections/illness
Clinical features
- Dehydration
- Deep, rapid breathing (Kussmaul) respiration
- Nausea and vomiting, abdominal pain
- Lethargy, drowsiness
- Confusion, loss of consciousness

Assessment
1. Confirm the diagnosis and determine its cause - e.g.
   - Infection/illness
   - New onset, insulin omission
2. Weigh the patient
3. Level of consciousness (refer to Neurological Observations)
   - Watch for cerebral oedema (headache, deteriorating conscious state, falling heart rate, rising blood pressure)
4. Investigations
   Venous blood sample (place an IV line if possible as this will be needed if DKA is confirmed) for the following:
   - FBC
   - Blood glucose, urea, creatinine, electrolytes (sodium, potassium, chloride, bicarbonate)
   - Blood ketones
   - Calcium, phosphate, magnesium
   - HbA1c
   - pH, pCO₂
   - Where indicated, investigations for precipitating cause (ie if clinical signs of infection consider septic workup including blood culture)

Management of DKA
The goals of therapy when treating DKA are to:
- Correct dehydration
- Correct acidosis and reverse ketosis
- Restore normal blood glucose levels
- Avoid complications of therapy

Consider need for consultation with PICU Consultant/Registrar or on call Paediatric Endocrinologist.
1. **Airway/breathing**
   - In the comatose patient, secure the airway and insert nasogastric tube on straight drainage to prevent pulmonary aspiration.
   - Oxygen therapy if oxygen saturation < 95% or patient with severe circulatory impairment or shock
   - Do not ventilate without discussion with PMH ICU Consultant

2. **Supportive measures to consider if appropriate**
   - Establish arterial or venous access for convenient and painless repetitive blood sampling. This should be kept patent with a non-glucose containing fluid.
   - Apply cardiac monitor to detect potassium related abnormalities (hyperkalaemia – peaked T-waves, widened QRS or hypokalaemia – flattened or inverted T waves, ST depression, wide PR interval)
   - Consider antibiotics for febrile patients after obtaining appropriate cultures

3. **Fluid requirements**

   Intravenous or oral fluids that may have been given at another hospital should be factored into the assessment and calculation of fluid deficit and replacement needs.

   Corrected (i.e. actual) Na = add 2 mmol/L of Na for every 5.5 mmol/L of glucose above 5.5 mmol/L

   Effective osmolality = 2 x (Na + K) + glucose

   - If there is haemodynamic compromise, **immediately commence resuscitation with 0.9% saline 10ml/kg** and repeat until blood pressure is restored.
   - Do not exceed 30 ml/kg 0.9% saline
   - All fluids given must be documented

   **Calculate fluid requirement**

   Requirement = maintenance + deficit

   This tends to be overestimated. Do not use greater than 5% dehydration in calculations

   Deficit (ml) = % dehydration x body weight (kg) x 10

   **Maintenance fluid requirements**

<table>
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<tr>
<th>Body weight</th>
<th>Fluid requirement</th>
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<tr>
<td>3-10kg</td>
<td>100 ml/kg/day</td>
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<tr>
<td>10-20kg</td>
<td>1000 ml + (50 ml/kg per day for each kg &gt; 10kg)</td>
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<tr>
<td>&gt;20kg</td>
<td>1500 ml + (20 ml/kg per day for each kg &gt; 20kg)</td>
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   Add calculated maintenance for 48 hours and estimated deficit then subtract amount already given as resuscitation fluid to give total evenly over 48 hours.
Diabetic Ketoacidosis – Assessment and Management

**Hourly rate = (48hr maintenance + deficit – resuscitation fluid already given)**

A calculator tool using these formulae is available through Kids Health WA Paediatric Acute Care Guidelines website. [PMH ED DKA Fluid Calculator](#)

**Example:**

20 kg boy who is > 5% dehydrated and has already had 20mL/kg saline will require:

5% X 20 kg X 10 = 1000mL deficit

plus maintenance = 1500mL each 24 hours, therefore 3000mL each 48 hours = 4000mL

MINUS 20 kg X 20 mL = 400 mL resuscitation fluid

3000ml (48hr maintenance) + 1000ml (deficit) – 400ml (resuscitation fluid given) / 48

Therefore, fluid requirement is 2600mL over 48 hrs = 75 mL/hr

- Do not include urinary losses in calculations
- Continue normal saline until the plasma glucose is less than 15 mmol/L
- When the plasma glucose falls below 15 mmol/L, change fluids to 5% glucose in 0.9% saline to prevent rapid decrease in plasma glucose and hypoglycaemia
- The use of large amounts of 0.9% saline is associated with the development of hyperchloaemic metabolic acidosis; this needs to be factored in to the assessment of acid base status, particularly in assessing the timing of conversion to subcutaneous insulin (see below)
- Glucose concentration may be increased to 7.5% if the plasma glucose falls below 5 mmol/L – increase glucose concentration rather than the fluid rate
- Oral fluids only offered after substantial clinical improvement and no vomiting
- Include oral intake in calculations
- Rapid changes in fluid, electrolytes and serum osmolality can cause cerebral oedema. Monitor changes in mental state (using GCS, Neurological Observations). Fluid management may need to be altered accordingly. If in doubt, discuss with senior clinician.

4. **Potassium**

Once a child has been resuscitated, potassium should be commenced immediately with rehydration fluid unless anuria present. Potassium replacement therapy is required for treatment of DKA. This is because a total body deficit of potassium occurs and correction of the acidosis in the absence of potassium therapy will usually rapidly result in hypokalaemia. Patients may have hyperkalaemia, hypokalaemia or normokalaemia at presentation however there is always massive depletion of total body potassium. Levels in the blood will fall once insulin is commenced.

- Initially add 20 mmol potassium chloride to every 500mL bag of fluid (40 mmol KCl per litre)
Diabetic Ketoacidosis – Assessment and Management

- **It may be necessary to give potassium through a sideline in PICU.** Sideline potassium – start at 2 mmol/kg/day and increase if hypokalaemia develops.
- In general no more than 6 mmol/kg/day is required
- Give no more than 0.4 mmol/kg/hr without discussion with PICU consultant
- If serum potassium is >4.5 mmol/L, cease the potassium infusion
- When metabolic acidosis is corrected, potassium supplements may be reduced
- Refer to the [Potassium Monograph](#) for further information on administration and monitoring.

5. **Insulin administration** *(begins after the initial fluid resuscitation)*

- Insulin is essential to switch off ketogenesis and reverse the ketoacidosis
- Continuous low dose insulin is the preferred method
  - Venous pH <7.2 = intravenous insulin recommended with admission to PICU
  - Venous pH >7.2 = subcutaneous insulin and ward management
- Refer to the [Insulin Monograph](#) for further information on administration and monitoring.

**Intravenous infusion**

- Patients having IV insulin infusion MUST be managed in PICU
- Add 50 units (0.5 mL) of either Actrapid or Humulin R (*not* Humulin NPH) to 50 mL of 0.9% saline
- When saline is used, flush the line (20 mL) to prevent loss of insulin through binding to the tubing
- Give insulin at an insulin infusion rate of 0.1 units/kg/hour (maximum 5 units/hr).
  **Do not give IV insulin boluses**
- **Do not reduce the insulin infusion rate while ketoacidosis persists**
- When the ketoacidosis is corrected the insulin infusion may be reduced to 0.05 units/kg/hr (or less if BGL less than 5 mmol/L and glucose is being infused).
- If the blood glucose level does not decrease after the first 4 hours of the infusion, increase the infusion rate to 0.15 units/kg/hr
- Once the child is rehydrated, the keto acidosis corrected (ie pH>7.30 and HCO₃ >15) and the blood glucose level stable, give subcutaneous insulin 0.1 units/kg and **2 hours later cease the insulin infusion.** Subcutaneous insulin at the same dose should then be given every 4 hours.

**Subcutaneous administration**

- Initially 0.1 units/kg of Actrapid or Humulin R, then 0.1 units/kg every 2 hours
- When the acidosis is corrected change to 0.1 units/kg every 4-6 hours
Diabetic Ketoacidosis – Assessment and Management

- For repeated subcutaneous administration, the use of an Insufion (indwelling subcutaneous soft cannula) will be considered. Refer to Subcutaneous Indwelling Catheter - Insufion

6. **Bicarbonate**

Bicarbonate administration is not routinely recommended as it may cause paradoxical CNS acidosis. Continuing acidosis indicates insufficient fluid and insulin replacement. In rare circumstances some extremely sick children may benefit from cautious administration. **Bicarbonate is not to be given unless discussed with PICU Consultant.**

7. **Monitoring and management**

- Patient initially should be “Nil by mouth” except for ice to suck
- Strict fluid balance hourly, use a urinary catheter in the comatose child
- Hourly observations (more frequent if clinically indicated)
  - Pulse, BP, Respiratory rate,
  - level of consciousness (GCS)
  - neurological status (pupillary responses, assess for change e.g. restlessness, irritability, headache)
- Blood glucose level every 2 hours until stable, then 4 hourly
- Blood gases every 2 hours until stable, then 4 hourly
- Electrolytes at 2 hours and then 4 hourly until the acidosis is corrected. Patient should have 2 IV cannulas sited – one for infusions and one for sampling. If a second IV is unobtainable, capillary samples will be taken for ABG and formal bloods.
- Some patients will have an arterial line inserted if their condition warrants it.

**Hazards**

1. **Hyper/hyponatraemia**

   - Measured serum sodium is depressed by the dilutional effect of the hyperglycaemia. To ‘correct” sodium concentration, use the following formula:

   \[
   \text{Corrected (ie actual) Na} = \text{add 2 mmol/L of Na for every 5.5 mmol/L of glucose above 5.5 mmol/L.}
   \]

   Effective osmolality = 2 x (Na + K) + glucose

   - If Na is >160 mmol/L discuss with senior doctor
   - If sodium does not rise as the glucose falls during treatment or if hyponatraemia develops, it usually indicates overzealous volume correction and insufficient electrolyte replacement. This may place the patient at risk of cerebral oedema.
2. Hyperglycaemia

- If blood glucose falls below 4 mmol/L and the patient is still ketogenic, give IV 10% dextrose 2-5ml/kg as a bolus and use a 10% dextrose concentration for ongoing IV fluids (with 0.9% NaCl and K+ supplements). Do not discontinue the insulin infusion.

- If hypoglycaemia occurred despite use of 10% dextrose in the preceding 2 or more hours, the rate of the insulin infusion may be decreased to 0.05 units/kg/hr as long as the ketosis and acidosis are clearing.

- Continue with a 10% dextrose concentration in IV fluids until BGL stable.

- If blood glucose falls below 4 mmol/L and most recent pH is >7.3, oral treatment for hypoglycaemia (jelly beans + 1 serve of complex carbohydrate) can be used instead of an IV bolus of dextrose 10%.

3. Cerebral Oedema

Clinical cerebral oedema occurs suddenly, usually between 6-12 hours after starting therapy (range 2-24 hours). Mortality or severe morbidity is very high without early treatment.

**Prevention**

- Slow correction of fluid and biochemical abnormalities. Optimally, the rate of fall of blood glucose and serum osmolality should not exceed 5 mmol/hr but in children there is often a quicker initial fall in glucose.

- Patients should be nursed head up (ie elevate the head of the bed)

**Risk factors**

- First presentation
- Long history of poor control
- Young age (<5 yrs)
- Low pH and elevated serum urea at presentation

**Warning signs**

- Headache, irritability, lethargy, depressed consciousness, incontinence, thermal instability
- Very late signs – bradycardia, increased BP and respiratory impairment
- No sodium rise as glucose falls, hyponatraemia during therapy, initial adjusted hypernatraemia
- Always exclude hypoglycaemia

**Treatment**

- Inform senior staff immediately and manage in PICU
- Mannitol 20% 0.5g/kg IV over 10-15 min and repeat if there is no initial response in 30 min to 2 hours or 3% saline 5ml/kg over 5 mins and repeat if no initial response.
Give immediately when clinical diagnosis is suspected – do not delay for confirmatory brain scan.

- Reduce fluid input by 1/3
- Nurse head up
- Sedation, intubation and ventilation may be necessary (must hyperventilate to the same level or lower than that achieved by the patient with spontaneous breathing).

### Related internal policies, procedures and guidelines

- **Neurological Observations**, PMH Clinical Practice Manual

### References


2. Dehydration, Royal Children Hospital Clinical Guideline (2015)

3. ISPAD Clinical Consensus Guidelines 2014 Compendium


5. National evidence-based Clinical Care Guidelines for Type 1 Diabetes in Children, Adolescents and Adults 2011, Ch 17, Diabetic ketoacidosis, p128-135

### Useful resources (including related forms)

- **PMH DKA Emergency Management Pathway** (www.kidshealthwa.com)
- **PMH ED DKA Fluid Calculator**
Diabetic Ketoacidosis – Assessment and Management

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