Queensland Paediatric Guideline

Emergency

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. Rarely, 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

DKA severity		
Mild	**Moderate**	**Severe**
pH between 7.2 - 7.3 or HCO3 less than 15 mmol/L	pH between 7.1 - 7.2 or HCO3 less than 10 mmol/L	pH less than 7.1 or HCO3 less than 5 mmol/L

Ketone readings and probability of DKA				
Sample	**Method of testing**	**Low/Small**	**Moderate**	**High/Large**
Blood	Bedside meter Abott	Less than 0.6 mmol/L	0.6 to less than 1.5 mmol/L	Greater than or equal to 1.5 mmol/L
Urine	Bayer brand Keto-Diastix	0 mmol/L	0.5 to less than 1.5 mmol/L	Greater than or equal to 1.5 mmol/L
	Accu-chek brand Keto-Diabur-Test 5000	Negative	Less than 1.0 mmol/L	Greater than or equal to 1.0 mmol/L

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

Specific considerations in DKA

- tachypnoea secondary to acidosis can exacerbate dryness of oral mucosa[2]
- vasoconstriction from acidosis may contribute to the appearance of cool extremities[2]
- 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.

Fluid resuscitation (IV) for the management of shocked children with DKA	
Dose	Sodium Chloride 0.9% administered in 10 mL/kg bolus. Repeat as necessary to a maximum of 20 mL/kg.

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).

Requirement = Maintenance + ([Deficit – fluid bolus already given] 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	Ideal continuous insulin IV infusion dose is 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[5] however it may be considered in infants and very severe DKA. <u>If using a syringe pump:</u> 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. <u>If no syringe pump available:</u> 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		
BGL	**Fluids**	**Insulin**
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.[6] • 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,[5] 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.

Monitoring	Frequency	Comments
Vital Signs (HR, RR, BP)	Hourly	
Temperature	2-4th hourly or hourly if febrile	
Capillary (fingerprick) • BGL • Blood ketones	Hourly	• Blood ketones aid in determining resolution of DKA. • The bedside meter (Abbott brand) ketone readings greater than 4 mmol/L are less accurate. • DO NOT use either blood or urinary ketones alone as the indicator for changes to fluid or insulin regimes. Assess the whole child.
Laboratory bloods: • Venous glucose • Blood gases • U&Es • Haematocrit	2-4th hourly Consider hourly electrolyte monitoring in severe DKA.	• Capillary glucose methods may be inaccurate if poor peripheral circulation or acidosis. • Consider an IV cannula for repetitive blood sampling. An IA line may be necessary in some critically ill patients managed in PICU/ICU
Strict fluid balance	Hourly	• Watch carefully for polyuria • Consider a urinary catheter if impaired level of consciousness. • In non-toilet trained children, nappies should be weighed to ensure accurate fluid balance
Neurological observations	Hourly or more if high risk	See below for groups at high risk of cerebral oedema.
ECG monitoring	Continuous	Assess T waves for hyperkalaemia or hypokalaemia
Urinalysis for ketones	Until negative	Only required if blood ketones not available
Weight	On admission and daily	

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 20^0
- 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 failure[6]
- consider intubation and ventilation. Aim for CO2 35-40mmHg. Aggressive hyperventilation has been associated with poor outcome in retrospective studies of DKA related cerebral oedema[7]

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	
Sodium Chloride 3% (Hypertonic Saline 3%) (IV)	3 mL/kg/dose (1–5 mL/kg/dose) over 10-15 minutes 3mL/kg is expected to increase plasma sodium by approximately 2-3 mmol/L
Risks	Rebound ICP Central pontine myelinosis Subarachnoid haemorrhage Renal failure

Mannitol (IV) dosing for the treatment of raised ICP	
Mannitol (IV)	0.25-0.5 g/kg over 10-15 minutes Higher doses i.e. 1g/kg may be administered on senior advice.
Risks	Hypotension Hyperosmolality Rebound elevations in ICP Renal failure Extravasation

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 Paediatrician/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.

Monitoring	Frequency
Vital Signs (HR, RR)	Hourly
Temperature and BP	4th hourly (or hourly temperature if febrile)
Capillary (fingerprick) BGL	Standard BGL (before meals and 2am)
Strict fluid balance	Hourly
Neurological observations	Hourly – unless advised otherwise by endocrinologist/treating paediatrician.

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

Condition	Management
Venous thromboembolism	Consider prophylaxis in patient with central venous catheter
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 and advice 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.

Child is critically unwell or rapidly deteriorating child

Includes the following children:

- DKA and any of:
  - shock requiring two or more fluid boluses
  - suspected cerebral oedema (severe headache or neurological deterioration)
  - age less than 5 years
  - pH < 7.15 continuing within first two hours of presentation despite treatment
  - physiological triggers based on age (see below)
- any child with HHS

Less than 1 year	1-4 years	5-11 years	Over 12 years
• RR >50 • HR <90 or >170 • sBP <65 • SpO2 <93% in oxygen or <85% in air • GCS ≤12	• RR >40 • HR <80 or >160 • sBP <70 • SpO2 <93% in oxygen or <85% in air • GCS ≤12	• RR >40 • HR <70 or >150 • sBP <75 • SpO2 <93% in oxygen or <85% in air • GCS ≤12	• RR >30 • HR <50 or >130 • sBP <85 • SpO2 <93% in oxygen or <85% in air • GCS ≤12

Reason for contact	Who to contact
For immediate onsite assistance including airway management	The most senior resources available onsite at the time as per local practices. Options may include: • paediatric critical care • critical care • anaesthetics • paediatrics • Senior Medical Officer (or similar)
Paediatric critical care and endocrine advice and assistance	Onsite or via Retrieval Services Queensland (RSQ). If no onsite paediatric critical care service contact RSQ on **1300 799 127**: • for access to paediatric critical care and endocrine telephone advice • to coordinate the retrieval of a critically unwell child RSQ (access via QH intranet) **Notify early of child potentially requiring transfer.** **Consider early involvement of local paediatric/critical care service.** In the event of retrieval, inform your local paediatric service.

Non-critical child

Includes the following children (as a guide):
• first presentation of DKA (non-critical) • mild DKA prior to administering insulin • as needed for any other child with DKA **Inform the treating service of the ED presentation for ALL children previously diagnosed with diabetes (regardless of the presenting condition).**

Reason for contact	Who to contact
Advice (including management, disposition or follow-up)	Follow local practice. Options: • onsite/local paediatric service • Queensland Children's Hospital experts via Children's Advice and Transport Coordination Hub (CATCH) on 13 CATCH (13 22 82) (24-hour service) • local and regional paediatric videoconference support via Telehealth Emergency Management Support Unit TEMSU (access via QH intranet) on 1800 11 44 14 (24-hour service)
Referral	First point of call is the onsite/local paediatric service

Inter-hospital transfers

Do I need a critical transfer?	• discuss with onsite/local paediatric service • view Queensland Paediatric Transport Triage Tool
Request a non-critical inter-hospital transfer	• contact onsite/local paediatric service • contact RSQ on 1300 799 127 for aeromedical transfers • contact Children's Advice and Transport Coordination Hub (CATCH) on 13 CATCH (13 22 82) for transfers to Queensland Children's Hospital

Non-critical transfer forms	• QH Inter-hospital transfer request form (access via QH intranet)
	• aeromedical stepdown (access via QH intranet)
	• commercial aeromedical transfers:
	  ○ Qantas
	  ○ Virgin
	  ○ Jetstar

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

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Guideline approval

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Disclaimer

This guideline is intended as a guide and provided for information purposes only. The information has been prepared using a multidisciplinary approach with reference to the best information and evidence available at the time of preparation. No assurance is given that the information is entirely complete, current, or accurate in every respect. We recommend hospitals follow their usual practice for endorsement locally including presenting it to their local Medicines Advisory Committee (or equivalent) prior to use.

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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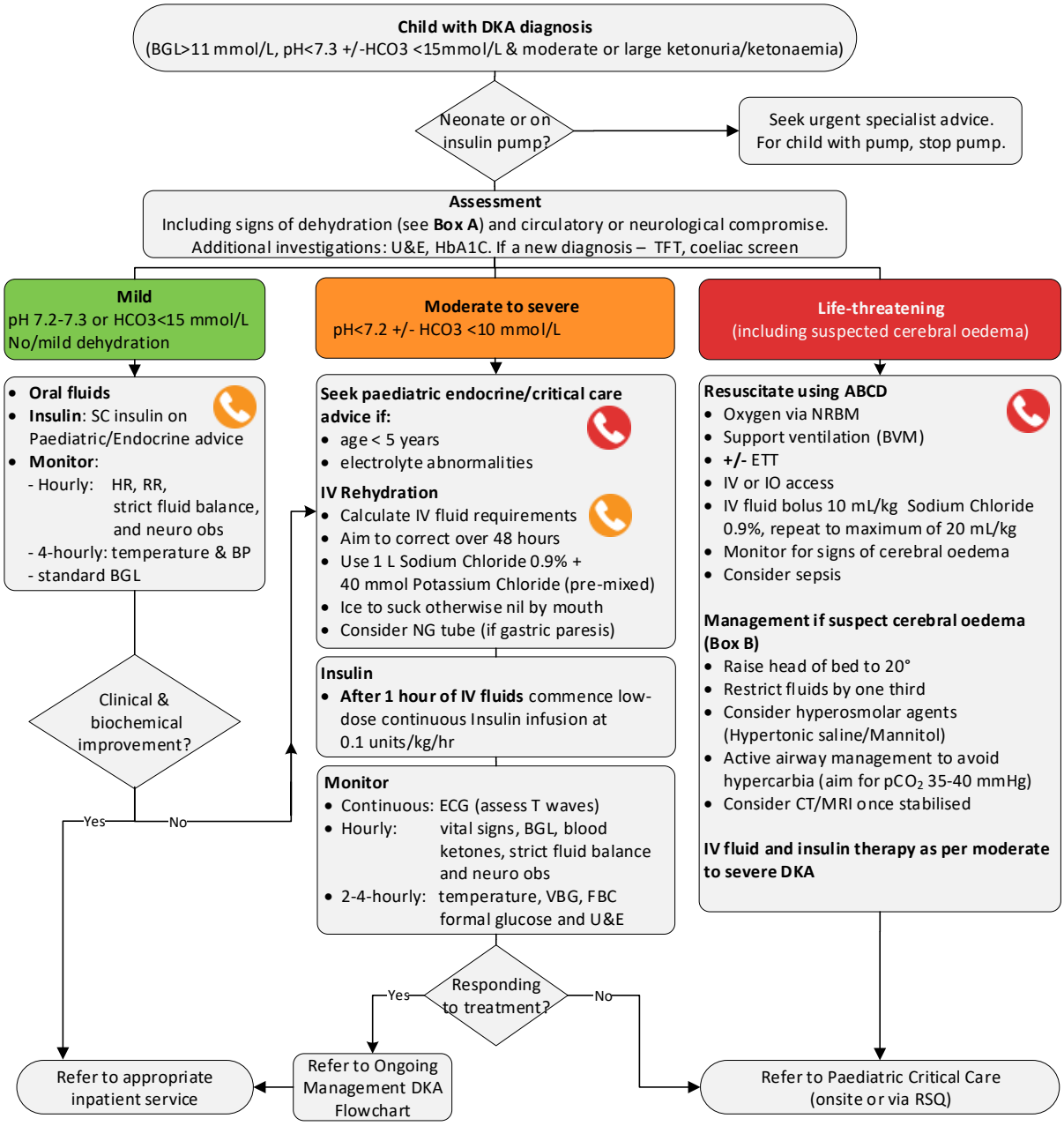

- Calculate insulin doses carefully as very serious errors can occur. Never give bolus IV or IM insulin.
- Miscalculations of added potassium to fluids can be fatal. Outside of critical care, pre-mixed fluid bags are recommended.

Box A: Hydration assessment in DKA

Volume deficit is often overestimated in DKA which can result in over resuscitation with IV fluids.

Specific considerations in DKA include:

- 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 may result in weight loss

Box B: Signs and symptoms of cerebral oedema

- 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

Seek senior Paediatric/Endocrine advice as per local practice.

Seek urgent Paediatric Endocrine/Critical Care advice (onsite or via Retrieval Services Queensland (RSQ) on 1300 799 127)

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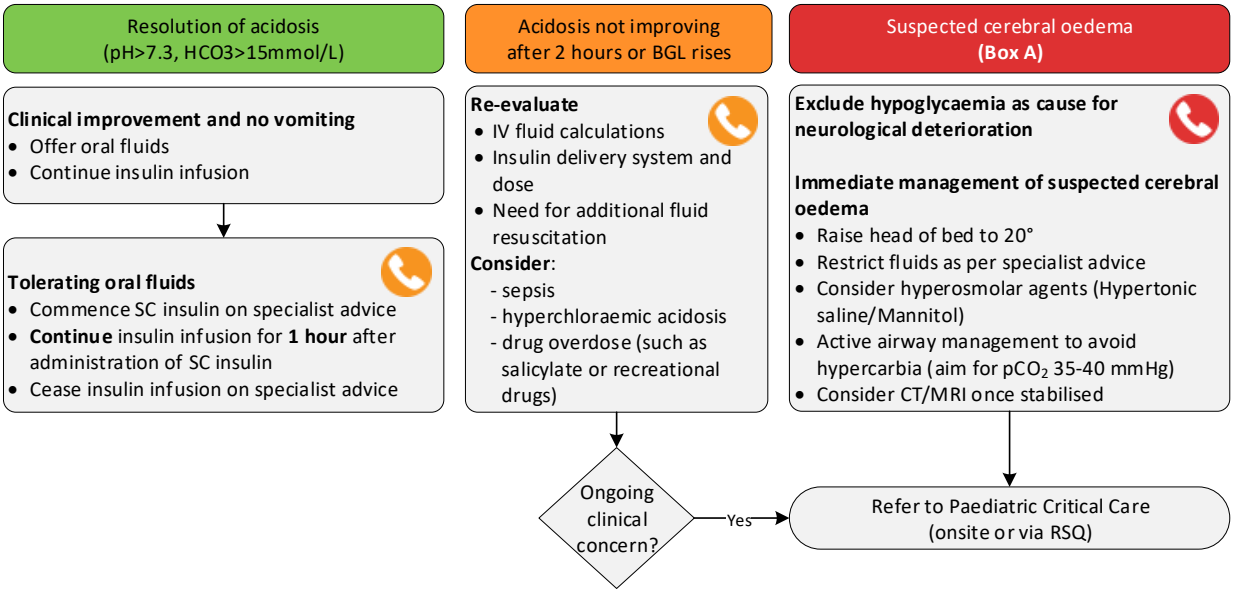
Seek Paediatric Endocrine/Critical Care advice (onsite or via Retrieval Services Queensland (RSQ)) if electrolyte abnormalities are identified

Ongoing management of child with moderate to severe DKA		
BGL	**Fluids IV**	**Insulin**
Falls at rate of greater than 5 mmol/L/hr	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 Sodium Chloride 0.9% + 40 mmol Potassium Chloride	**DO NOT** reduce rate
Issues maintaining 5-10 mmol/L despite running a solution containing Glucose 5%	Increase the Glucose concentration to Sodium Chloride 0.9% + Glucose 10% + 40 mmol Potassium Chloride/L	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).
Falls below 4 mmol/L	Administer a bolus of 2 mL/kg of Glucose 10% over 3 minutes. Ensure fluid running has Glucose 5% and consider Glucose 10%	Temporarily reduce by 50% and seek urgent specialist advice. **DO NOT** stop infusion.

Management of possible clinical scenarios



BOX A: When to suspect cerebral oedma	
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)

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Body weight in kg: ..... ❶ 34 kg

Total **fluid bolus** given ..... ❷ 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) ..... ❸ 80 mL/kg

Calculate total deficit: Multiply ❶ by ❸ ..... ❹ 2720 mL

If **fluid bolus** was given:

then subtract ❷ from ❹ ..... ❺ 2380 mL

Divide **deficit** over 48hr (divide ❺ by 48) ... ❻ 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..... ❼ 74 mL/hr

Calculate total hourly fluid rate: add ❻ and ❼ 124 mL/hr