

Local Guideline & Procedure



John Hunter
Children's Hospital
CHILDREN, YOUNG PEOPLE AND FAMILIES



Health
Hunter New England
Local Health District

PAEDIATRIC DIABETIC KETOACIDOSIS (DKA)

Sites where Local Guideline applies	JHH campus, excluding NICU
This Local Guideline applies to:	
1. Adults	No
2. Children up to 16 years	Yes
3. Neonates – less than 29 days	No
Target audience	All Clinical staff of John Hunter Children's Hospital; JHH ED and ICU
Description	This document provides the protocol for the management of all paediatric patients less than 18 years with DKA. DO NOT USE THE ADULT PROTOCOL TO MANAGE PAEDIATRIC DKA

[Hyperlink to Guideline](#)

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Related Legislation, Australian Standard, NSW Ministry of Health Policy Directive or Guideline, National Safety and Quality Health Service Standard (NSQHSS) and/or other, HNE Health Document, Professional Guideline, Code of Practice or Ethics:	<ul style="list-style-type: none"> • NSW Health Policy Directive 2014_036 Clinical Procedure Safety • National Evidence-Based Clinical Care Guidelines for Type 1 Diabetes for Children, Adolescents and Adults, APEG & ADS, 2011 • International Society for Pediatric and Adolescent Diabetes (ISPAD) Clinical Practice Consensus Guidelines, 2014
Local Guideline note	This document reflects what is currently regarded as safe and appropriate practice. The guideline section does not replace the need for the application of clinical judgment in respect to each individual patient but the procedure/s require mandatory compliance . If staff believe that the procedure/s should not apply in a particular clinical situation they must seek advice from their unit manager/delegate and document the variance in the patient's health record.
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RISK STATEMENT

Diabetic ketoacidosis (DKA) is the metabolic consequence of absolute insulin deficiency due to Type 1 diabetes mellitus. It is a potentially fatal condition and the most common cause of diabetes-related deaths in childhood. This guideline provides an evidenced-based protocol for Paediatric Patients with DKA as adult protocols are unsuitable.

Risk Category: *Clinical Care & Patient Safety*

GLOSSARY

Acronym or Term	Definition
BGL	Blood Glucose Level
DKA	Diabetic ketoacidosis is a potentially life threatening disorder, defined as hyperglycaemia due to insulin deficiency with pH less than 7.30.
ED	Emergency Department
GCS	Glasgow Coma Scale
HR	Heart Rate
PEDOC/SPOC	Paediatric Emergency Department Observation Chart/Standardised Paediatric Observation Chart
RR	Respiratory Rate

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This Guideline does not replace the need for the application of clinical judgment in respect to each individual patient.

INTRODUCTION

Diabetic ketoacidosis is a potentially life threatening disorder, defined as hyperglycaemia due to insulin deficiency with pH less than 7.30.

It may be the initial presentation of Type 1 diabetes or develop in a patient with established Type 1 diabetes due to failure of insulin delivery or inadequate insulin in the context of intercurrent illness.

NB: For one-page summary and checklist see Appendix 1

Pathophysiology

Insulin deficiency causes hyperglycaemia and ketogenesis.

The presence of ketones (beta-hydroxybutyrate and acetoacetate) causes acidosis. Finger-prick blood ketones by bedside meter will usually be greater than 3.0 mmol/L in DKA.

Osmotic diuresis causes dehydration and a total body deficit of all electrolytes.

Accumulation of lactate due to poor tissue perfusion may contribute to the acidosis.

Key Concerns

1. Hyperglycaemia, hyperlipidaemia and glycosuria
2. Dehydration due to osmotic diuresis
3. Severe acidosis and ketonuria
4. Hyperosmolar state

ALERT

The commonest cause of recurrent diabetic ketoacidosis is insulin omission.

Common Misconceptions

Blood glucose does not need to be markedly elevated in DKA. Concentrations as low as 7 mmol/L may occur in patients who have been sick for a number of days with low oral intake. This is due to the depletion of liver glycogen. All diabetics should check for urinary or blood ketones if they become ill.

Aims of Treatment

The aims of treatment of DKA are different between children and adults. Children are at high risk of cerebral oedema but they tolerate hypovolaemia well. Older adults tolerate hypovolaemia poorly and are at lower risk of cerebral oedema.

Hence the aim of treatment of DKA in **children is to achieve sufficient perfusion to avoid acute tubular necrosis but to keep the patient relatively dehydrated while the metabolic defect is corrected.**

Fluid therapy in children aims to correct the fluid deficit over 48 hours whilst in adults the fluid deficit is replaced in 24 hours or less.

ALERT

Look for an infective cause or other precipitating causes. Treat appropriately.

In severe DKA the white cell count may be elevated due to the acidosis itself.

MANAGEMENT PLAN

Key elements of the management of a paediatric patient in DKA who has presented to the Emergency Department include:

1. Assessment including:
 - a. History & examination
 - b. Full set of observations according to the standardised observation chart (PEDOC/SPOC)
 - c. Weight
 - d. Blood glucose (BGL)
2. Resuscitation (see page 6. below for more information)
3. Baseline investigations including:
 - a. Venous blood gas
 - b. Blood sample including: Glucose, EUC, calcium, magnesium, phosphate, osmolality, FBC and, if newly diagnosed diabetes, C-peptide (and if sufficient blood available), add autoantibodies against insulin/GAD/IA-2, islet (ICA) antibodies, thyroid antibodies, lipids, TSH and coeliac screen
 - c. HbA1c
 - d. Blood cultures (if sepsis considered)
 - e. Test urine (for infection [mid-stream urinalysis] and ketones)
4. Notify ED Consultant and Paediatric Endocrine Consultant as soon as possible
5. Initiate definitive treatment and continue close monitoring
 - a. IV fluids and potassium replacement
 - b. Insulin infusion
 - c. Observations (BGL, complete PEDOC/SPOC observations)
 - d. Accurate measurement of urine output
 - e. Monitor electrolytes, venous blood gas and urinary ketones
6. Treat any precipitating factor(s) – assess for infection

RESUSCITATION

ALERT

Notify ICU if any of the following is present:

- a. pH less than 7.1
- b. Child is less than 2 years of age
- c. Shock (red zone PEDOC/SPOC)
- d. Calculated serum osmolarity greater than 330 mOsm/L
- e. Severe electrolyte disturbance (corrected Na⁺ greater than 150 or less than 130 mmol/L, or K⁺ greater than 5.5 or less than 3.0 mmol/L)
- f. Altered conscious state or suspected cerebral oedema
- g. Blood glucose greater than 50 mmol/L

Airway and Breathing

In shocked patients, give oxygen by face-mask. If oxygen saturation is less than 95% with oxygen then contact the ED consultant immediately.

If the patient has impaired consciousness then maintain the airway and breathing.

Respiratory arrest is imminent if the respiration rate is:

- Inappropriately normal or slow for age

and/or

- pCO₂ inappropriately normal or high

Breathing in DKA is rapid and deep due to the acidosis (Kussmaul breathing).

Avoid the use of opioids or other pain relief that can suppress respiration. Inhibiting the respiratory drive in DKA can lead to rapid worsening of the acidosis.

ALERT

Kussmaul breathing may be misdiagnosed as asthma, hysterical hyperventilation or any other cause of breathlessness.

Not everyone can smell ketones on the breath as not everyone has the required receptor in the neuro-epithelium of the nose.

Abdominal pain and vomiting may be due simply to the ketonaemia itself, but can be misdiagnosed as gastroenteritis, appendicitis or other acute intra-abdominal problems. If pain relief is given in the form of opioids, the Kussmaul breathing can be suppressed leading to rapid worsening of the acidosis.

Circulation

If the patient is in shock (hypotensive, peripheral circulatory shutdown, oliguria) resuscitate with a bolus of intravenous sodium chloride 0.9%.

Paediatric Fluid Bolus

If the heart rate is in the yellow zone (PEDOC/SPOC) give 10 mL/kg sodium chloride 0.9% over 30 minutes

For patients in shock i.e. red zone (PEDOC/SPOC), give 20 mL/kg sodium chloride 0.9%

Reassess after the bolus.

Repeat bolus if

- Central capillary refill time greater than 3 seconds
- Heart rate remains in red zone

Notify the consultant immediately if a second bolus is required.

Repeat the 10 mL/kg bolus up to 2 times (maximum of 30 mL/kg over 3 hours).

Excessive use of fluid boluses is associated with cerebral oedema and death.

Giving more than two boluses is rarely required. Remember that a contribution to decreased peripheral perfusion comes from acidosis, which will only correct gradually as the acidosis is reversed.

If the fluid boluses do not restore adequate perfusion then notify the consultant and consider inotropic support.

ALERT

Ketoacidosis is often accompanied by ileus and a nasogastric tube may be needed if the level of consciousness is depressed. Nil by mouth, except for ice to suck, until metabolically stable (pH greater than 7.3) and bowel sounds are present, at which point low calorie fluids can be offered.

DEFINITIVE TREATMENT

i. Fluid therapy

Initially:

Use sodium chloride 0.9% until potassium is indicated (see Electrolytes section below).

IF serum potassium greater than 5.5 mmol/L reassess each hour

IF serum potassium 3.5–5.5 start potassium chloride at 40 mmol/L

IF serum potassium less than 3.5 mmol/L the patient may require rapid potassium replacement

NB: This last step (rapid potassium replacement) needs to be done in ICU with monitoring

If potassium is indicated, use sodium chloride 0.9% with potassium chloride 40 mmol/L intravenous solution.

When the BGL is 14 mmol/L or less

Change to sodium chloride 0.45% with glucose 5% and with potassium chloride 40 mmol/L premixed solution.

Rates and considerations

Do not replace ongoing fluid losses. Polyuria is usually short-lived and rarely interferes with fluid replacement.

If the patient has DKA then assume they are at least 10% dehydrated. The patient must have their weight measured prior to starting IV therapy and an accurate fluid balance should be kept.

Aim to correct the deficit over 48 hours. Therefore Fluid Rate = Maintenance + Deficit Replacement given over 48 hours

Correct over 72 hours if the patient is very ill, very young (less than 2 years), has been sick for a long time or the corrected serum sodium is in the hypernatraemic range.

See Appendix 2 for recommended fluid infusion rates.

ALERT

Rapid rehydration is associated with the risk of cerebral oedema & death.

Patients requiring intravenous infusions for DKA must be NIL BY MOUTH to prevent excess/rapid rehydration.

If the patient has already received excessive rehydration at another hospital, the fluid given should be subtracted from the total requirement before calculating the replacement needed over 48 hours.

Note: Premixed bags of fluid should be used if possible and potassium should not be added to premixed bags already containing potassium. *Please see JHCH Clinical Guideline 13.31 for a list of premixed fluids available in JHCH*

http://intranet.hne.health.nsw.gov.au/data/assets/pdf_file/0006/133926/13_31_potassium_f inal.pdf

ii. Insulin infusion

ALERT

The insulin infusion should start one hour after fluid boluses are completed.

Insulin should be given by continuous intravenous infusion. Intramuscular or subcutaneous routes are unreliable in this setting. If the patient has a subcutaneous insulin pump, this should be ceased and the pump's subcutaneous cannula removed when the insulin infusion is started.

Blood glucose should be done every hour. Over the first two hours, rehydration alone will cause a rapid fall in blood glucose. However, after this, the aim is to decrease glucose by 4–5 mmol/L per hour. When the BGL is 14 mmol/L or less, glucose is added to the fluid regimen.

Paediatric Insulin Infusion Rate

Start infusion at **0.05 to 0.1 Unit/kg/hour** of short acting (soluble) insulin via a volumetric pump.

Use Actrapid or Humulin R diluted in sodium chloride 0.9%.

The insulin infusion is diluted by adding insulin 1 Unit/kg body weight into
50 mL of sodium chloride 0.9%: therefore:

$$5 \text{ mL/h} = 0.1 \text{ Unit/kg/h}$$

$$2.5 \text{ mL/h} = 0.05 \text{ Unit/kg/h}$$

Use lower insulin doses of 0.05 Unit/kg/hour (2.5 mL/h) especially if:

- The child is young (less than 5 years)
- There is hyperosmolarity (greater than 330 mOsm/L)
- There is hypokalaemia (less than 3.5 mmol/L, when there is potassium chloride 40 mmol/L in the IV fluids)
- if the BGL is less than 5 mmol/L, when glucose 5% solutions are running

If the BGL continues to drop below 5 mmol/L and fluids containing glucose 5% are running, then higher concentrations of glucose can be used (glucose 7.5%).

ALERT

The minimum insulin infusion rate allowed is 0.05 Unit/kg/h (i.e. 2.5 mL/h) unless specifically discussed with the Paediatric Endocrinologist on-call

iii. Correcting pH

If pH is not correcting then consider the following:

1. Patient not receiving insulin (check syringe, line and cannula and ensure the line was primed with insulin)
2. Inadequate perfusion (check fluid balance and cardiac status)

3. Sepsis (ENT, chest, abdomen, urine, skin)
4. Insulin resistance (these patients will require higher insulin infusion rates)
5. Electrolyte disturbance (hyperchloraemic acidosis, hypophosphataemia, hypomagnesaemia)
6. Cerebral oedema
7. Lactic acidosis (reperfusion of tissues following fluid resuscitation in the first 2 hours mobilises tissue lactate and may cause a minor drop in pH. However, the pH should be improving by 4 hours and if not, contact consultant).

ELECTROLYTES

Electrolytes should be monitored every two to six hours depending on the clinical situation.

I. Sodium replacement

Serum sodium needs to be corrected for the dilutional effect of hyperglycaemia and hyperlipidaemia.

Corrected Sodium Calculation

$$\text{Corrected Sodium} = \text{Sodium} + \text{glucose}/3$$

If corrected sodium begins to rise rapidly with sodium chloride 0.9%, fluids may need to be changed to sodium chloride 0.45% or sodium chloride 0.45% with glucose 5% and potassium chloride 40 mmol/L.

ALERT

If corrected sodium exceeds 160 mmol/L extreme caution is needed with the rate of rehydration, particularly in infants. Fluid replacement needs to be over 72 hours and patients must be nil by mouth until the electrolyte disturbance has resolved.

Hyponatraemia during treatment usually reflects over-zealous volume correction with insufficient electrolyte replacement.

II. Potassium replacement

Always check the serum potassium before commencing potassium replacement

IF serum potassium greater than 5.5 mmol/L reassess each hour

IF serum potassium 3.5–5.5 mmol/L start potassium chloride at 40 mmol/L

IF serum potassium less than 3.5 mmol/L the patient may require rapid potassium replacement

ALERT

IF serum potassium less than 3.0 mmol/L, call the ED consultant immediately and notify ICU/Paediatric Endocrinology and monitor ECG.

If the potassium is less than 3.5 mmol/L and there is potassium chloride 40 mmol/L in the IV fluid, then decrease the insulin infusion rate to 0.05 Unit/kg/h. Repeat the serum potassium in 2 hours. If the potassium has not started to increase, then more rapid potassium replacement is required.

If the required potassium replacement rate is greater than 0.3 mmol/kg/h, ICU admission is necessary, therefore call the ED consultant and notify ICU/paediatric endocrinology.

A "normal" appearing serum potassium in the face of severe acidosis indicates marked depletion of total body potassium stores. After fluid boluses have been finished, potassium chloride (40 mmol/L) should be added to the IV fluid unless serum potassium is greater than 5.5 mmol/L and/or the patient is anuric (pre-renal renal failure).

An ECG should be performed if there is hypo- or hyperkalaemia. If greater than 0.3 mmol/kg/h potassium is needed, then cardiac monitoring is necessary.

III. Osmolarity

Serum osmolarity can be calculated directly using the following approximation:

Serum Osmolarity Calculation

Serum Osmolarity = 2 x (sodium + potassium) + glucose + urea

NB: The sodium is the laboratory value, not the calculated, corrected sodium

A hyperosmolar state (greater than 330 mOsm/L) exists with severe hyperglycaemia and/or hypernatraemia. In the face of marked hyperglycaemia, a serum sodium in the "normal range" should ring warning bells.

In this situation, paediatric patients will require the therapy to be tailored to minimise the risk of cerebral oedema (correction of dehydration and electrolyte imbalance over 72 hours).

NB: A solution of sodium chloride 0.9% with of potassium chloride 40 mmol/L has an osmolality of 370 mOsm/kg, and may exacerbate a hyperosmolar state.

IV. Bicarbonate

The use of bicarbonate in DKA is no longer recommended. There may be a place in very rare cases of extremely severe acidosis (e.g. arterial pH less than 6.8) or the severely shocked patient. Urgent consultant advice should be obtained. Consider bicarbonate therapy only in patients with cardiogenic shock due to acidosis or with symptomatic hyperkalaemia.

Risks of therapy include:

- Hypokalaemia and cardiac arrhythmias from sudden correction of pH
- Exacerbation of hypernatraemia
- Paradoxical worsening of CNS acidosis
- Precipitation of cerebral oedema

Bicarbonate paediatric dose:

Under consultant advice ONLY:

Bicarbonate dose (mmol) for total repair of base deficit

= 1/3 (base deficit x body weight in kg)

Only give 1/4 of this dose at one time and note response before repeating

V. Chloride

Hyperchloraemia may develop in the course of therapy (due to the use of sodium chloride in solutions) and fluids may need to be changed from 0.9% to 0.45% sodium chloride.

There is no evidence that hyperchloraemic acidosis adversely affects outcome and hence treatment should be conservative and response monitored.

VI. Calcium

Hypocalcaemia may occur if some of the potassium replacement is given as potassium phosphate. No more than 10 mmol/L of potassium phosphate should be added to any solution.

VII. Phosphate

Hypophosphataemia can occur during treatment for DKA because insulin increases phosphate utilisation (formation of ATP, protein phosphorylation etc.).

The significance of hypophosphataemia in this setting is unclear.

Severe hypophosphataemia can occur but is usually asymptomatic unless phosphate is less than 0.32 mmol/L. Clinical manifestations can include: Metabolic encephalopathy (irritability, paraesthesiae, confusion, seizures, coma); impaired myocardial contractility, cardiac arrhythmias; respiratory failure due to weakness of the diaphragm; muscle dysfunction with proximal myopathy, dysphagia and ileus. Rhabdomyolysis can occur rarely.

No treatment should be given unless the serum phosphate is less than 0.5 mmol/L. If this occurs, then 10 mmol/L of potassium phosphate (and 30 mmol/L of potassium chloride [so the total potassium concentration is 40 mmol/L]) should be added to the IV solution.

Using potassium phosphate concentrations greater than 10 mmol/L can cause hypocalcaemia. IV phosphate should be ceased once the patient starts eating (because there are large amounts of phosphate in food). Oral phosphate replacement can be given if required.

CEREBRAL OEDEMA

If conscious state is altered, then hourly neurological observations should be done.

Diagnostic criteria

- Abnormal motor or verbal response to pain
- Decorticate or decerebrate posture
- Cranial nerve palsy (especially III, IV and VI)
- Abnormal neurogenic respiratory pattern (e.g. grunting, Cheyne-Stokes respiration, apnoea)

Major criteria

- Altered mentation/fluctuating level of consciousness (GCS less than 12)
- Agitation or irrational behaviour
- Sustained heart rate deceleration (decrease more than 20 beats per minute) not attributable to improved intravascular volume or sleep state
- Age-inappropriate incontinence

Minor criteria

- Vomiting
- Headache
- Lethargy or not easily rousable
- Diastolic blood pressure greater than 90 mmHg
- Age less than 5 years

Diagnosis of cerebral oedema should be suspected if there is:

- One diagnostic criterion OR
- Two major criteria OR
- One major and two minor criteria

Such criteria combinations have a sensitivity of 92% and a false positive rate of only 4%.

Risk factors for cerebral oedema:

- Severe acidosis and dehydration
- Extended history of poor control (presumed increase in osmoprotective adaptation)
- Young age
- Hypernatraemia, hyponatraemia or falling serum sodium during therapy
- Excessive fluid replacement has been implicated

ALERT

Early detection of subtle symptoms and signs is extremely important.

If Cerebral Oedema is suspected:

- Exclude hypoglycaemia (which can mimic cerebral oedema)
- Mannitol 0.5 gram per kg IV infusion over 20 minutes or hypertonic sodium chloride 3%
- Repeat if there is no response in 30 minutes
- Reduce IV infusion rate by 1/3
- Consider intubation if GCS is less than 8

**Cerebral oedema is a medical emergency – activate a rapid response.
Call the ED consultant immediately and notify ICU and paediatric endocrinologist.**

CHILDREN WHO MEET THE FOLLOWING CRITERIA USUALLY REQUIRE ADMISSION TO ICU

- pH is less than 7.1
- Child is less than 4 years of age
- Shock (red zone PEDOC/SPOC)
- Calculated serum osmolarity is greater than 330 mOsm/L
- Severe electrolyte disturbance (corrected Na⁺ greater than 150 or less than 130 mmol/L, or K⁺ greater than 5.5 or less than 3.0 mmol/L)
- Altered conscious state or suspected cerebral oedema
- Blood glucose greater than 50 mmol/L

CRITERIA FOR DISCHARGE FROM ICU

The ICU discharge process requires careful medical and nursing handover to the paediatric endocrinology team. The patient must meet the following criteria:

- Venous pH greater than 7.25 and base deficit less than -6 mEq/L
- Potassium greater than 3.1 mmol/L and replacement less than 0.3 mmol/kg/hour
- GCS = 15
- Heart rate in white zone of PEDOC/SPOC
- Oxygen requirement less than 1 L/min or FiO₂ less than 0.5

IMPLEMENTATION AND MONITORING COMPLIANCE

The implementation of this guideline will be communicated to all staff within the JHCH/JHH, through managers, education boards, educator network and clinical network streams.

Guideline and procedures will all be available through HNE LHD PPG directory and HNEKidshealth website.

Compliance will be monitored with audits yearly, conducted by the Endocrine team. Results and associated action plan where required, will be sent to the JHCH Clinical Quality and Patient Care Committee.

APPENDICES

Appendix 1: Paediatric DKA fluid replacement – John Hunter Children’s Hospital

Appendix 2: Paediatric DKA Algorithm – John Hunter Children’s Hospital

REFERENCES

1. [National Evidence-Based Clinical Care Guidelines for Type 1 Diabetes for Children, Adolescents and Adults, APEG & ADS, 2011](#)
2. [International Society for Pediatric and Adolescent Diabetes \(ISPAD\) Clinical Practice Consensus Guidelines, 2014](#)
3. Kamel, KS and Halperin, ML. Acid-Base Problems in Diabetic Ketoacidosis. N Engl J Med 2015;372: 546–554.
4. [NSW Health GL2015_008 Standards for Paediatric Intravenous Fluids: NSW Health](#) (second edition)
5. [JHCH Guideline 13.31 Use of Potassium Chloride in JHCH \(excluding NICU\)](#)
6. [NSW Health PD2013_043 Medication Handling in NSW Public Health Facilities](#)

FEEDBACK

Any feedback on this document should be sent to the Contact Officer listed on the front page.

CONSULTATION

REVISED: A/Professor Bruce King, June 2012

REVISED: A/Professor Patricia Crock, February 2015

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APPROVAL

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JHH ICU: 6th September 2016

JHH ED: 7th September 2016

Appendix 1: Diabetic Ketoacidosis (DKA) Guideline

One-page Summary and Checklist

Step 1: Initial Assessment

- Airway, Breathing, Circulation
- Level of consciousness (Glasgow Coma Scale, see Emergency SPOC charts)
- Hydration
- Measure blood glucose (and blood ketones, if available) with bedside meter
- Test urine for ketones and glucose
- Obtain patient's weight

Send baseline blood sample (at same time as siting IV cannula):

- Glucose, EUC, calcium, magnesium, phosphate, osmolality, venous pH, FBC
- if newly diagnosed diabetes; C-peptide (and if sufficient blood available), add autoantibodies against insulin/GAD/IA-2, islet antibodies (ICA), thyroid antibodies, lipids TSH and coeliac screen

Step 2: If patient looks well and pH greater than 7.3 then IV fluids may not be required and subcutaneous insulin can be given (consult paediatric endocrine team for insulin type/dose).

If patient looks unwell, site IV cannula without waiting for pH result and start sodium chloride 0.9% infusion (or Plasma-Lyte 148, if available):

- Only if shocked, give 20 mL/kg bolus of sodium chloride 0.9% and oxygen by face mask (repeat fluid boluses of 10 mL/kg are rarely required in DKA – total maximum 30 mL/kg)
- Set ongoing fluid rate to give maintenance plus replacement of deficit over 48 hours (see Table in Appendix 2). Consider reducing rehydration rates if excessive fluid resuscitation has already been given (greater than 30 mL/kg)
- Add potassium chloride (initially 4–5 mmol/kg/day) if K⁺ less than 5.0 mmol/L, unless patient is oliguric or known to have renal failure

The over-riding principle is to correct the metabolic derangements (acidosis, dehydration and hyperglycaemia) **slowly**. Rapid correction has been associated with cerebral oedema.

Step 3: Insulin infusion (Actrapid) at 0.05–0.1 Unit/kg/h, as a side-line to the rehydration fluid. Delay starting insulin infusion until 1 hour of IV fluid administration has been given.

Step 4: Site second IV cannula (22 gauge minimum) for venous sampling and send second blood sample for glucose, EUC, venous pH.

Step 5: Consider nasogastric tube (to prevent aspiration in an obtunded patient) and urinary catheter (if needed to allow strict fluid balance).

Step 6: Ongoing management

- Keep nil by mouth (except ice to suck)
- Review indications for ICU
- Continue monitoring using PEDOC/SPOC
- Assess adjustments to fluid and electrolyte replacement
- Change IV fluids to glucose 5% when blood glucose falls below (or is rapidly approaching) 14 mmol/L (don't decrease the insulin infusion below 0.05 Unit/kg/h in DKA, because the ketosis is likely to worsen)

Step 7: Transition to subcutaneous insulin

(Courtesy of Associate Professor Charles Verge, Sydney Children's Hospital)

Appendix 2: Paediatric DKA fluid replacement

John Hunter Children's Hospital

Assessing the degree of dehydration in DKA is difficult. In all cases assume they are 10% dehydrated. The patient must have a weight measured prior to starting IV therapy and an accurate fluid balance should be kept.

Aim to correct the deficit over 48 hours.

NB: If the patient is very ill, very young (less than 2 years), has been sick for a long time or the corrected serum sodium is in the hypernatraemic range then correct over 72 hours.

Therefore **Fluid Rate = Maintenance + 10% Deficit** replaced over 48 hours

Paediatric DKA Fluid Replacement			
Weight	Maintenance fluid	Assume 10% dehydrated	
		Maintenance plus deficit replacement over 48 h	Maintenance plus deficit replacement over 72 h
3 kg	13 mL/h	19 mL/h	17 mL/h
4 kg	17 mL/h	25 mL/h	22 mL/h
5 kg	21 mL/h	31 mL/h	28 mL/h
6 kg	25 mL/h	38 mL/h	33 mL/h
7 kg	29 mL/h	44 mL/h	39 mL/h
8 kg	33 mL/h	50 mL/h	44 mL/h
9 kg	38 mL/h	56 mL/h	50 mL/h
10 kg	42 mL/h	63 mL/h	56 mL/h
11–13 kg	45 mL/h	70 mL/h	63 mL/h
14–16 kg	50 mL/h	85 mL/h	75 mL/h
17–20 kg	60 mL/h	100 mL/h	85 mL/h
21–25 kg	65 mL/h	115 mL/h	100 mL/h
26–30 kg	70 mL/h	130 mL/h	110 mL/h
31–35 kg	80 mL/h	150 mL/h	125 mL/h
36–40 kg	80 mL/h	160 mL/h	135 mL/h
41–45 kg	80 mL/h	170 mL/h	140 mL/h
46–50 kg	80 mL/h	180 mL/h	150 mL/h
51–55 kg	80 mL/h	190 mL/h	155 mL/h
56–60 kg	80 mL/h	200 mL/h	160 mL/h
greater	80 mL/h	210 mL/h	170 mL/h

Clinical Audit Tool

(National Standard 1: 1.7.2 The use of agreed clinical guidelines by the clinical workforce is monitored)

Criterion no.	Criterion	Exceptions	Definition of terms and/or general guidance	Data source	Frequency	Position Responsible
1	To minimise the risk of clinical deterioration of the patient and prevent acute complications of DKA	None	The aim is to prevent mortality and minimise morbidity from diabetic DKA – including hypokalaemia, hyper- or hyponatraemia and cerebral oedema.	IIMS data Retrospective chart audits x10	12 monthly	Paediatric Endocrinology and Diabetes Team
2						
3						
4						
5						

Reference: *Electronic audit tool - National Institute for Health and Clinical Excellence (NICE): www.nice.org.uk/nicemedia/live/10996/56372/56372.xls*