Guideline and Procedure

HNEkidshealth Children, Young People & Families

NSW GOVERNMENT Health Hunter New England Local Health District

Paediatric Diabetic Ketoacidosis (DKA)

Sites where Guideline and Procedure applies	All HNELHD sites
This Guideline and Procedure applies to:	
1. Adults	No
2. Children up to 18 years	Yes
3. Neonates – less than 29 days	No
Target audience	Clinical staff caring for paediatric patients
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.**

Go to Procedure

Keywords	DKA, diabetic ketoacidosis, diabetes, paediatric, acidosis, ED, PICU
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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:

- 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, 2018
- See Reference Section on page 20

Guideline and Procedure 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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PURPOSE AND RISKS

Diabetic ketoacidosis (DKA) is the metabolic consequence of absolute or relative 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.

Risks can be reduced by:

1. Following the guideline and procedure to minimise the risk of electrolyte disturbances, cerebral oedema and other DKA related complications.
2. Escalate care according to this guideline and procedure.

Risk Category: Clinical Care and Patient Safety

GLOSSARY

Acronym or Term	Definition
ATP	Adenosine triphosphate
BGL	Blood glucose level
BP	Blood pressure
CNS	Central nervous system
CRP	C-reactive protein
DKA	Diabetic ketoacidosis
ED	Emergency Department
ENT	Ear, Nose and Throat
ESR	Erythrocyte sedimentation rate
EUC	Electrolytes, urea, creatinine
FBC	Full blood count
GCS	Glasgow Coma Scale
HbA1C	Glycosylated haemoglobin
HR	Heart rate
IV	Intravenous
K+	Serum Potassium
LFT	Liver function tests
NETS	Newborn and paediatric Emergency Transport Service

NG	Nasogastric
PEDOC	Paediatric Emergency Department Observation Chart
PICU	Paediatric Intensive Care Unit
RR	Respiratory rate
SC	Subcutaneous
SPOC	Standardised Paediatric Observation Chart
TSH	Thyroid stimulating hormone

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PAEDIATRIC DKA ALGORITHM

History - Polyuria, polydipsia, weight loss, abdominal pain, tiredness, vomiting, confusion, symptoms of infection.
Clinical Assessment - Hydration, perfusion, HR, BP, GCS, respiratory rate (Kussmaul), ketones, lethargy/drowsiness, infections.
Investigations - Venous blood gas, glucose, FBC, electrolytes, calcium, phosphate, magnesium, urea and creatinine, ESR, CRP, triglycerides, cholesterol and HbA1c.
If the patient has a new diabetes diagnosis then add C-peptide, insulin level, total IgA, coeliac serology, TSH, anti-thyroid Abs, anti-Insulin Ab, anti-GAD Ab, anti-islet cell Ab and anti IA2 Ab.
If the patient is febrile perform blood/urine culture and consider CXR and antibiotics.
Biochemical signs of DKA include: blood glucose level >11 mmol/L, pH <7.3, Bicarb <15 mmol/L, urinary ketones (>10 mmol/L), ketonaemia. (>3 mmol/L).

Shock and Resuscitation
Tachycardia, poor central perfusion +/- hypotension +/- reduced conscious level. Discuss with senior ED/VMO and Local Paediatrician at Regional Facility/Endocrine/PICU staff. Give sodium chloride 0.9% 10 mL/kg as a bolus and then reassess. Give further 10 mL/kg sodium chloride 0.9% as a bolus if still remains shocked continue to reassess.

MILD DKA

pH >7.20
Clinically well, tolerating fluids orally

Start SC insulin
Rapid acting insulin for Breakfast, Lunch, Afternoon tea & Dinner

Long acting insulin
0.3 units/kg at 18:00 (dinner time)

Admit to hospital
Diabetic diet
Fluids as tolerated
Routine observations + BGLs

Diabetes education
Dietician
Social work

Known diabetes
Continue normal insulin regime
If BGL >8 mmol/L give subcut rapid acting insulin, according to a prescribed correction dose, second hourly

Oral fluids as tolerated

May be discharged if BGL settles and the patient is well

MODERATE DKA

pH 7.20 – 7.1
Tachycardia
Dehydration and vomiting

Keep nil by mouth

IV fluid bolus
Give sodium chloride 0.9% at 10 mL/kg over 1 hour
Repeat if required

IV fluid therapy
Rate = sodium chloride 0.9% maintenance + 10% deficit over 48 hours

IV insulin infusion
0.05 u/kg/hr
1 hour after completion of the fluid bolus

Check serum potassium
IF K+ > 5.5 reassess hourly
IF K+ < 5.5 give potassium chloride at 40 mmol/L (add to fluid)
IF K+ < 3.5 call consultant

Admit to ward

If patient and / or acidosis not improving then check:

IV fluid rate
Insulin delivery systems
Insulin dose
Consider sepsis
Cerebral oedema (headache, confusion, hypertension, bradycardia, slow respirations)

SEVERE DKA

pH <7.1

Resuscitation
Airway ± insert NG tube
Breathing (100% O2)
Circulation

Keep nil by mouth

IV fluid bolus
If shocked see above.
If not shocked give sodium chloride 0.9% sodium chloride 20 mL/kg over 1 hour
Total of all boluses should not exceed 40 mL/kg.

IV fluid therapy
Rate: sodium chloride 0.9% maintenance + 10% deficit over 48 hours.

IV insulin infusion
0.05 - 0.1 units/kg/h
1 hour after completion of the fluid bolus

Check serum potassium
IF K+ > 5.5 reassess hourly
IF K+ < 5.5 give potassium chloride at 40 mmol/L (add to fluid)
IF K+ < 3.5 call consultant

Admit to PICU or call NETS (1300 36 2500)

Paediatric Diabetic ketoacidosis JHCH 2020

MILD AND MODERATE DKA PAEDIATRIC ALGORITHM

INSULIN INFUSION 0.05 units/kg/hour

Add 1 unit/kg to 50mL sodium chloride 0.9% and run at 2.5mL/h insulin and deliver by a sideline

CRITICAL OBSERVATIONS - Hourly BGL, RR, HR, BP, fluid balance (input and output)

2 – 6 hourly neurological observations age appropriate GCS,

When blood glucose < 14 mmol/L or blood glucose falls at > 5 mmol/L/hour (not including 2 hours after initial bolus)

Change to sodium chloride 0.45% + glucose 5% + 40 mmol/L potassium chloride

When patient is well (with metabolic resolution) – transition to subcutaneous (SC) insulin

Start subcut insulin **then stop** IV insulin 60 min later

Cerebral oedema signs: headache, slowing of heart rate / respiratory rate, hypertension, irritability, age appropriate GCS < 12 or decrease of > 2 points, focal neurological signs.

Treatment of cerebral oedema: this is an emergency. Exclude hypoglycaemia, give mannitol 0.5 g/kg over 20 min, halve the IV fluid rate, monitor RR, HR, BP and SpO_2 (may need O_2 or ventilation), call senior staff and admit to PICU.

SEVERE DKA PAEDIATRIC ALGORITHM

INSULIN INFUSION 0.05 – 0.1 units/kg/hour

Add 1 unit/kg insulin to 50mL sodium chloride 0.9% and deliver by a sideline.

0.05 units/kg/h = 2.5mL/h and 0.1 units/kg/h = 5mL/h

Change to standard insulin concentrations on arrival at PICU

CRITICAL OBSERVATIONS - Hourly BGL, RR, HR, BP, fluid balance (input and output)

Neurological observations age appropriate GCS, blood gas and ketones

Potassium replacement

- Add 40 mmol/L potassium chloride to fluids when serum potassium < 5.5 mmol/L and passing urine

- **PICU only potassium management**
- Can increase up to 60 mmol/L potassium chloride in fluids via peripheral line if serum potassium remains < 3.0 mmol/L
- If serum potassium remains < 3.0 mmol/L then discuss replacement with intensivist
- If persistent hypokalaemia also check magnesium and replace if necessary
- Phosphate: If serum phosphate < 0.3 mmol/L then discuss with intensivist to change potassium chloride to potassium dihydrogen phosphate

When blood glucose is < 14 mmol/L or blood glucose falls at > 5 mmol/L/hour once insulin started

- If corrected serum sodium < 150 mmol/L use sodium chloride 0.9% + glucose 5% + potassium chloride as the rehydration fluid
- If corrected serum sodium increases by > 5 mmol/L/hour or IS > 150 mmol/L change to sodium chloride 0.45% + glucose 5% + potassium chloride
- Discuss with intensivist /NETs before using any other fluids

Cerebral oedema signs: headache, slowing of HR /RR, hypertension, irritability, GCS < 12 or decrease by > 2 points, focal neurological signs

Treatment of cerebral oedema: This is an emergency. Exclude hypoglycaemia, give mannitol 0.5 g/kg over 20 min or hypertonic saline 3% 3 mL/kg over 20 min, halve the IV fluid rate, monitor RR, HR, BP & $satO_2$, call senior staff/admit PICU

Criteria for discharge from PICU

- Venous pH greater than 7.25 and bicarb > 15 mmol/L
- Serum potassium > 3.1 mmol/L and replacement less than 0.25 mmol/kg/hour
- GCS = 15
- Observation in white/blue zone of SPOC Chart
- Oxygen requirements as per normal ward requirements
- IV insulin 1unit/kg in 50 mL sodium chloride running at 2.5 mL/h unless otherwise stated.

GUIDELINE

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

INTRODUCTION

Diabetic ketoacidosis (DKA) 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.

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 most common 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 days with low oral intake. This is due to the depletion of liver glycogen. All people with diabetes should check for 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 just enough perfusion to avoid acute renal failure and maintain mild dehydration 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.

PROCEDURE

This procedure requires mandatory compliance.

MANAGEMENT PLAN

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

1. Assessment:
    a. History and examination
    b. Full set of observations according to the standardised observation chart (PEDOC/SPOC)
    c. Weight
    d. BGL.
2. Resuscitation (see below for more information).
3. Baseline investigations:
    a. Venous blood gas
    b. Blood sample: for formal glucose, EUC, calcium, phosphate, LFT, osmolality, FBC, ESR, CRP and, if newly diagnosed diabetes: C-peptide, insulin, autoantibodies against insulin/GAD/IA-2, islet cell antibodies, thyroid antibodies, TSH, triglyceride, cholesterol, total IgA, Vitamin D and coeliac serology
    c. HbA1c
    d. Blood cultures (if sepsis considered)
    e. Urine (for infection [mid-stream urinalysis] and ketones).
4. Notify ED Consultant, Paediatric Endocrine Consultant,VMO and Local Paediatrician at Regional Facility as soon as possible.
5. If severe DKA then notify PICU or call NETS
6. 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 ketones.
7. Treat any precipitating factor/s – assess for infection.

RESUSCITATION

ALERT

Notify PICU/NETS if any of the following is present:

a. pH less than 7.1

b. Shock (red zone PEDOC/SPOC)

c. Calculated serum osmolarity > 330 mOsm/L

d. Severe electrolyte disturbance (corrected serum sodium > 150 mmol/L or < 130 mmol/L, or serum potassium > 5.5mmol/L or < 3.0 mmol/L)

e. Altered conscious state or suspected cerebral oedema

Note: these indications for PICU admission/ NETS transfer relate to when the person first presents. These factors are indicators that the person is at high risk of developing electrolyte disturbances or cerebral oedema. Waiting for the pH to rise or the BGL to decrease does not alter the risk or the need for PICU admission/NETS transfer.

AIRWAY AND BREATHING

In shocked patients, give oxygen by facemask. If oxygen saturation is less than 95% with oxygen, then contact the ED/VMO and Local Paediatrician 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

- **pCO_2 inappropriately normal or high**

Breathing in DKA should be 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, hyperventilation or other cause of breathlessness.

Not everyone can smell ketones on the breath.

Abdominal pain and vomiting may be due to the ketonaemia itself, but can be misdiagnosed as gastroenteritis, appendicitis or other acute intra-abdominal problems. If abdominal pain is severe, seek senior advice before routinely giving opioids. If opioid analgesia is given the Kussmaul breathing may be suppressed leading to 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 HR is in the yellow zone on PEDOC/SPOC give 10 mL/kg sodium chloride 0.9% over 1 hour.

For patients in shock (red zone on PEDOC/SPOC) give 10 mL/kg sodium chloride 0.9% over 15 min; reassess after the bolus.

Give a further 10 mL/kg bolus if:

– Central capillary refill time greater than 3 seconds

– HR remains in red zone

Notify the consultant immediately if a second bolus is required.

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

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

If perfusion and shock is not improving despite fluid boluses notify PICU/ED consultant immediately. Consider other causes of shock +/- need for inotropes.

ALERT

Ketoacidosis is often accompanied by ileus. A NG tube may be needed if the level of consciousness is depressed. Patients should remain **nil by mouth** until metabolically stable (pH greater than 7.3), at which point low calorie fluids can be offered.

DEFINITIVE TREATMENT

Fluid therapy

Initially:

Use sodium chloride 0.9% until potassium is indicated.

IF serum potassium greater than 5.5 mmol/L, reassess each hour
IF serum potassium 3.5–5.5 mmol/L, add potassium chloride 40 mmol/L to IV fluids
IF serum potassium less than 3.5 mmol/L, the patient may require rapid potassium replacement

NB: Rapid potassium replacement needs to be done in PICU or in consultation with NETS with appropriate 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

For moderate DKA, change to sodium chloride 0.45% + glucose 5% + potassium chloride 40 mmol/L premixed solution.

For severe DKA/PICU, change to sodium chloride 0.9% + glucose 5% + 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. Polyuria resolves once the BGL is less than 10 mmol/L.

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

See Appendix 1 for recommended fluid infusion rates.

ALERT

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

Patients requiring intravenous infusions for DKA must be **nil by mouth** to prevent excess/rapid rehydration.

If the patient has already received 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.*** Refer to HNE Intravenous Potassium Chloride-Paediatrics PD2019_058: PCP 5

INSULIN INFUSION

ALERT

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

Insulin should be given by continuous IV 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.

MILD AND MODERATE DKA PAEDIATRIC INSULIN INFUSION RATE

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

Use Actrapid or Humulin R 1 unit/kg body weight diluted in 50 mL sodium chloride 0.9%.

The insulin infusion rate will be:

0.1 unit/kg/h = 5 mL/hour

0.05 units/kg/h = 2.5 mL/hour

0.025 units/kg/h = 1.25 mL/hour

MANAGEMENT OF INSULIN INFUSION AND BLOOD GLUCOSE LEVELS

The target BGL during insulin infusion is 5 – 8 mmol/L.

BGL (mmol/L)	Effect/Management
>8	Osmotic diuresis occurs which contributes to electrolyte disturbances
5–8	Target range
3.5–5	Ensure the IV fluid contains glucose 5% and decrease the insulin rate (see below)
<3.5	Hypoglycaemia – treat with 2 mL/kg glucose 10% IV (repeat in 5 minutes if needed) Notify the consultant.

The insulin infusion rate should be decreased if:

- The BGL continues to drop below 5 mmol/L and fluids containing glucose 5% are running
- The BGL drops at a rate > 5 mmol/L/h (excluding the first 2 hours of resuscitation where the BGL decreases due to rehydration following the fluid boluses)

Try 0.025 units/kg/h for one hour. Further insulin titration can be performed depending on BGL response. Insulin infusion rates below 0.025 units/kg/h may be required in the very young or very insulin sensitive patient. In this situation discussion with the consultant is required and serum ketones or pH should be monitored. Infusion rates <0.0125 units/kg/h may not be adequate to clear ketones and so may lead to worsening acidosis.

Note: using glucose concentrations greater than 5% causes potassium to enter the cells resulting in hypokalaemia. Fluids containing glucose concentrations greater than 5% should not be used without discussion with the consultant.

Correcting pH / acidosis

If the pH is not correcting, then consider the following:

1. Patient is not receiving insulin: Check syringe, line and cannula and ensure the line was primed with insulin
2. Inadequate tissue perfusion: Check fluid balance and cardiac status
3. Sepsis: ENT, chest, abdomen, urine, skin
4. Electrolyte disturbance: Hyperchloraemic acidosis, hypophosphataemia, hypomagnesaemia
5. Cerebral oedema
6. 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 if not, contact consultant
7. Insulin resistance: These patients will require higher insulin infusion rates (Note: this is very rare in paediatrics and should only be considered once the other possible causes have been excluded)

ELECTROLYTES

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

Sodium replacement

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

Corrected Sodium Calculation

$$\text{Corrected Sodium} = \text{Serum sodium (mmol/L)} + \frac{\text{blood glucose (mmol/L)}}{3}$$

Rapid increases in serum sodium have been associated with excess sodium administration during rehydration, especially in infants. This is because the fluid deficit is intracellular, and water and potassium enter the cell leaving the sodium in the extra cellular fluid.

The measured serum sodium concentration should rise as the glucose falls (corrected serum sodium should stay the same). Failure of the measured serum sodium to rise, associated with falling corrected serum sodium, indicates excess free water administration and is associated with an increased risk of cerebral oedema.

- If the corrected serum sodium falls to less than 136 mmol/L, continue with sodium chloride 0.9% (rather than sodium chloride 0.45%) as the rehydration fluid.
- If the corrected serum sodium increases by greater than 5 mmol/L/h or to greater than 150mmol/L, then hypernatraemia may aggravate the hyperosmolar state produced by hyperglycaemia. If corrected serum sodium is greater than 150 mmol/L, consider changing IV fluid to sodium chloride 0.45% and slowing the rate of rehydration after discussion with an Intensivist or Endocrinologist.

ALERT

If corrected serum sodium exceeds 160 mmol/L extreme caution is needed with the rate of correction, particularly in infants. Rapid drops in serum sodium can lead to cerebral oedema. Discuss fluid replacement with PICU intensivist. Patients must be nil by mouth until the electrolyte disturbance has resolved.

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 add potassium chloride at 40 mmol/L to IV fluids
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/NETS and notify PICU/Paediatric Endocrinology/VMO and Local Paediatrician and monitor ECG.

If the serum 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 0.025 unit/kg/h. Check magnesium and replace if necessary. Repeat the serum potassium in 2 hours. If the serum potassium has not started to increase, then more rapid potassium replacement is required.

If the required potassium replacement rate is greater than 0.25 mmol/kg/hr, cardiac monitoring and PICU admission is necessary. Call the paediatric endocrinology consultant and notify PICU and

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 0.25 mmol/kg/hr potassium is needed, then cardiac monitoring is necessary.

Osmolarity

Serum osmolarity can be calculated directly using the following approximation:

Serum Osmolarity Calculation

Serum osmolarity = 2 x (serum sodium + serum potassium) + blood glucose + serum urea

NB: The serum 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 indicates hypernatremia and 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 monitoring of neurological status is required.

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 with impaired cardiac contractility). 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 Paediatric Endocrinologist, PICU or NETS advice ONLY:

Bicarbonate dose (mmol) for total repair of base deficit

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

Only give one quarter of this dose at one time and note response before repeating

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.

Calcium

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

Phosphate

Hypophosphataemia can occur during treatment for DKA because insulin increases phosphate consumption (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, paraesthesia, 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 dihydrogen 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 dihydrogen phosphate concentrations greater than 10 mmol/L can cause hypocalcaemia. IV phosphate should be ceased once the patient starts eating, as there are large amounts of phosphate in food. Oral phosphate replacement can be given if required.

CEREBRAL OEDEMA

If conscious state is altered, 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, apneusis)

Major criteria

- Altered mentation/fluctuating level of consciousness (GCS less than 12)
- Agitation or irrational behaviour
- Sustained HR 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 of cerebral oedema is extremely important.

If cerebral oedema is suspected:

Exclude hypoglycaemia (which can mimic cerebral oedema)

Give mannitol 0.5 g/kg IV infusion over 20 minutes

Repeat if there is no response in 30 minutes

If there is still no response, then give hypertonic sodium chloride 3% 2.5 mL/kg

Reduce IV infusion rate by one third

Transfer to PICU, contact NETS

Consider intubation if impending respiratory failure

Cerebral oedema is a medical emergency – activate a rapid response/CERS Protocol.

In ED call the ED consultant immediately/CERS protocol and notify PICU/NETS and paediatric endocrinologist/VMO and Local Paediatrician.

Cerebral imaging should be performed after the patient is medically stable.

CHILDREN WHO MEET THE FOLLOWING CRITERIA AT PRESENTATION REQUIRE ADMISSION TO PICU/ESCALATION OF CARE VIA NETS

- pH less than 7.1

- Shock (red zone on PEDOC/SPOC)
- Calculated serum osmolarity greater than 330 mOsm/L
- Severe electrolyte disturbance (corrected serum sodium greater than 150 or less than 130 mmol/L, or serum potassium greater than 5.5 or less than 3.0 mmol/L)
- Altered conscious state or suspected cerebral oedema

CRITERIA FOR DISCHARGE FROM PICU

The PICU 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 bicarbonate >15 mmol/L
- Serum potassium greater than 3.1 mmol/L and replacement less than 0.25 mmol/kg/hour
- GCS = 15
- HR in white/blue zone of PEDOC/SPOC
- Oxygen requirement as per normal ward requirements

IMPLEMENTATION AND MONITORING COMPLIANCE

The implementation of this guideline will be communicated to all staff using the CE News and within the clinical network.

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

Compliance will be monitored with yearly audits and results and an associated action plan where required. This information will be sent to the CYPFS Clinical Quality and Patient Care Committee.

Appendices

Appendix 1: Paediatric DKA Fluid Replacement

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, 2018

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. NSW Health PD2013_043 Medication Handling in NSW Public Health Facilities

6. HNELHD PD2019_058:PCP 5 Intravenous Potassium Chloride – Paediatrics

FEEDBACK

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

CONSULTATION

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REVISED: A/Professor Patricia Crock, February 2015

REVISED: Professor Bruce King, May - July 2020

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CYPFS CPGAG approval date: May 2020

HNE QUM approval date: June 2020

CYPFS CQ&PCC approval date: July 2020

APPENDIX 1: PAEDIATRIC DKA FLUID REPLACEMENT

Assessing the degree of dehydration in DKA is difficult. In all cases assume the patient is 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, has been sick for a long time or the corrected serum sodium is in the hypernatraemic range, then electrolyte monitoring should occur at least every 4 hours.

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**
3 kg	13 mL/h	19 mL/h
4 kg	17 mL/h	25 mL/h
5 kg	21 mL/h	31 mL/h
6 kg	25 mL/h	38 mL/h
7 kg	29 mL/h	44 mL/h
8 kg	33 mL/h	50 mL/h
9 kg	38 mL/h	56 mL/h
10 kg	42 mL/h	63 mL/h
11–13 kg	45 mL/h	70 mL/h
14–16 kg	50 mL/h	85 mL/h
17–20 kg	60 mL/h	100 mL/h
21–25 kg	65 mL/h	115 mL/h
26–30 kg	70 mL/h	130 mL/h
31–35 kg	80 mL/h	150 mL/h
36–40 kg	80 mL/h	160 mL/h
41–45 kg	80 mL/h	170 mL/h
46–50 kg	80 mL/h	180 mL/h
51–55 kg	80 mL/h	190 mL/h
56–60 kg	80 mL/h	200 mL/h
greater than 60 kg	80 mL/h	210 mL/h