

John Hunter Hospital ICU

DIABETIC KETOACIDOSIS MANAGEMENT PLAN:

1. Assessment (weight, blood glucose level (BGL), blood pressure (BP), heart rate (HR), respiratory rate (RR), temperature, history & examination)
2. Resuscitation
3. Baseline Investigations (BGL, blood gas, full blood count (FBC), electrolytes, blood cultures, ward test urine)
4. Notify Paediatric or Adult Diabetes team as soon as possible.
5. Initiate definitive treatment
 - a. IV fluid and potassium replacement
 - b. Insulin infusion (start 1 hour after fluids are commenced)
 - c. Observations (BGL, BP, HR, RR, temp, neurological obs & fluid balance)
 - d. Monitor electrolytes, blood gas and urinary ketones
6. Treat any precipitating factor(s) - look for infection
7. Notify ICU if indicated

Criteria for Considering ICU Admission

1. pH < 7.1 at the time of ICU consultation or 7.0 or less at any time during the presentation.
2. Shock
3. Any child less than 3 years of age with DKA
4. Calculated serum osmolality is greater than 330 mOsm/L
5. Monitoring required for K⁺ replacement (> 0.3 mmol/kg/hour KCl)
6. Impaired conscious state (GCS<12) or suspected cerebral oedema

AIMS OF TREATMENT **PAEDIATRIC** vs **ADULT**

The aims of treatment of DKA are different for paediatrics and adults.

Children are at high risk of cerebral oedema but they tolerate hypovolaemia well.

Adults tolerate hypovolaemia poorly and are at low risk of cerebral oedema.

Hence the aims of treatment of DKA for:

Paediatrics is to achieve perfusion to avoid acute tubular necrosis but keep them relatively dehydrated while the metabolic defect is corrected.

Adults is to correct hypovolaemia and the metabolic defect.

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

A. RESUSCITATION

[Link to paediatric fluid calculator](#)

I. Fluid Therapy

If patient is in shock (hypotensive, peripheral circulatory shutdown, oliguria) resuscitate with a bolus of intravenous normal saline.

Paediatric – heart rate >100 bpm 10 -20 mL/kg over 60 minutes

Reassess after the bolus. Repeat up to 2 times (a total of 60 mL/kg) if no improvement then notify the consultant immediately. Excessive use of fluid boluses is associated with cerebral oedema and death.

Adult – Not in shock: Normal saline 3L at 500mL/hr
In shock: Normal saline 3L at 1000mL/hr

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

II. Airways and Breathing

In severely shocked patients, give oxygen by face-mask. If oxygen saturation is less than 95% with oxygen then contact the department consultant immediately. Early notification of ICU and endocrine teams will assist in organisation of ICU admission.

If the patient has impaired consciousness then maintain the airway and breathing. If the respiration rate is normal or slow then respiratory arrest is imminent. Breathing in DKA is rapid and deep (Kussmaul respiration) due to the acidosis.

B. DEFINITIVE TREATMENT

Fluid therapy

Paediatric fluid therapy – See Appendix 1

Use normal Saline until the BGL is 14 mmol/L then change to N/2 with 5% Dextrose pre-pack.

*If pre-packaged is not available: to make up N/2 with 5% Dextrose - take 1 Lt 5% Dextrose and add 20 mL High Potency 23.4% sodium chloride (4.6 grams) **OR** 500mL 5% Dextrose + 10 mL High Potency 23.4% sodium chloride (2.3 grams)*

Each bag of fluid should also contain 40 mmol/L of Potassium Chloride unless otherwise indicated (see potassium section). Potassium should not be added to premixed bags already containing potassium. See [13.31 Potassium Prescribing Guideline](#)

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

Assess degree of dehydration. If the patient has signs of dehydration then assume they are at least 10% dehydrated. The patient must have a weight 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 + 5% deficit** per 24 hours

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

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

Adult fluids

After the initial boluses have finished, **Fluid Rate = 250 mL/hour**

Use normal saline until the BGL is 14 mmol/L then change to N/5 with 4% Dextrose.

Each bag of fluid should contain 40 mmol/L of Potassium Chloride unless otherwise indicated (see potassium section). Premixed bags of N/5 with 4% Dextrose and 30 mmol/L Potassium Chloride may be used if available.

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

III. Insulin Infusion

In paediatrics, the insulin infusion should start after fluid boluses are completed.

In adults, the insulin infusion should start 2 hours after IV fluids started.

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.

A blood glucose measurement should be attended 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 levels by 4-5 mmol/L per hour. .

When blood glucose levels fall to 14 mmol/L, dextrose is added to the fluid regime (Paediatrics: N/2 with 5% dextrose; Adults: N/5 with 4% dextrose).

Paediatric Insulin Infusion Rate

Start infusion at **0.1 Units/kg/hour** of short acting insulin via a volumetric pump.

Actrapid HM TM or Humulin RTM diluted in normal saline

The insulin infusion is made by putting insulin 1 unit/Kg body weight into 50 mL of normal saline:

5 mL/ hr = 0.1 units/Kg/hr

2.5 mL/hr = 0.05 units/Kg/hr

Lower doses (e.g. 0.05 to 0.02 U/kg/hour) may be advisable in the very young child (<4 years), especially if there is hyperosmolality.

Adult Insulin Infusion Rate

Insulin infusion at **5 Units/ hour** of short acting insulin via a volumetric pump.

Actrapid HM TM or Humulin RTM diluted in normal Saline

e.g. 50 units insulin in 50 ml normal saline = 1 unit/mL

Run at 5 mL/hr

If the BGL drops below 5mmol/L and dextrose containing fluids are running then the insulin rate can be reduced.

The minimum insulin infusion rates allowed are:

Paediatrics - 0.05 units/Kg/hr (0.025 units/Kg/hr for under 4 year olds).

Adults – 2.5 units/hr.

If the patients BGL continues to drop with the lower insulin rates then higher concentrations of dextrose can be used (7.5% dextrose or 10% dextrose).

DO NOT DISCONTINUE - Both glucose and insulin are needed to correct the acidosis, hence the insulin infusion must never be ceased (until the urine is cleared of ketones and subcutaneous insulin has been administered).

If pH is not correcting then consider the following:

1. Patient not receiving insulin (check syringe, line and cannula).
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 (can cause a drop in pH in the first 2 hours).

IV. ELECTROLYTES

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

(1) Sodium Replacement

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

$$\text{Corrected Sodium} = \text{Sodium} + 2X(\text{glucose} - 5.6 \text{ mmol/L}) / 5.6$$

An easy alternative equation is

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

If corrected sodium begins to rise rapidly with normal saline, fluids may need to be changed to 0.45% saline or even 0.225% saline with 3.75% dextrose. Frankly lipaemic plasma will also falsely lower serum sodium measurements. Look in the retina for lipaemia retinalis, when blood vessels appear creamy in colour from blood fats.

If corrected sodium exceeds 160 mmol/L extreme caution is needed with the rate of rehydration, particularly in infants.

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

(2) Potassium Replacement

Always check the serum potassium before commencing potassium replacement

IF $K^+ > 5.5$ reassess each hour

IF $K^+ < 5.5$ start potassium chloride at 40 mmol/L

IF $K^+ < 3.5$ the patient may require rapid potassium replacement rates. If the potassium replacement rate is over 0.3 mmol/kg/hr they will require ICU admission, therefore call the department consultant and notify ICU / endocrinology.

A "normal" appearing K^+ level in the face of severe acidosis indicates marked depletion of total body potassium stores. After fluid boluses have been finished, potassium chloride (40 mmol/litre) should be added to the I.V. fluids unless $K^+ > 5.5$ mmol/L and/or the patient is anuric (pre-renal renal failure).

An ECG should be performed if there is hypo- or hyperkalaemia. If > 0.3 mmol/kg/hour KCl is needed then cardiac monitoring is necessary.

Premixed IV fluids containing potassium chloride should be used when ever possible.

In adult patients, premixed bags containing 30 mmol/L potassium chloride can be used if premixed bags containing 40 mmol/L are not available.

(3) Osmolality

Serum osmolality can be calculated directly using the following approximation:

$$\text{Serum Osmolality} = 2x (\text{sodium} + \text{potassium}) + \text{glucose}$$

Note: the sodium is the lab value not the corrected sodium.

A hyperosmolar state (>330 mOsm/kg) 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 minimize the risk of cerebral oedema (correction of dehydration and electrolyte imbalance over 48 - 72 hours).

NB: A solution of Normal saline with 30 mmol/L of KCl has an osmolality of 370 mOsm/kg, and may exacerbate a hyperosmolar state.

(4) Bicarbonate

Its use in DKA has gone out of vogue except in rare cases of severe acidosis (e.g. arterial pH <6.8) or the severely shocked patient. **N.B.** Urgent consultant advise should be obtained.

Risks of therapy: (1) hypokalaemia and cardiac arrhythmias by sudden correction of pH (2) exacerbation of hypernatremia (3) paradoxical worsening of CNS acidosis & (4) precipitation of cerebral oedema.

Paediatric dose:

Bicarbonate dose (mmol) for total repair of base deficit
= 1/3 (base deficit x body weight in kg).

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

Adult dose:

50 mmol

(5) 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 normal saline to 0.45% saline.

There is no evidence that hyperchloraemic acidosis adversely affects outcome and hence treatment should be conservative. Time cures all cases!

(6) Calcium

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

(7) Phosphate

Hypophosphataemia may occur because the insulin causes utilization of phosphate (formation of ATP, protein phosphorylation etc).

The significance of hypophosphataemia in this setting is unclear. Severe hypophosphataemia is associated with cardiac arrhythmias but whether this occurs in DKA is unknown.

No treatment should be given unless the serum phosphate is less than 0.5 mmol/L. If this occurs then 10mmol/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 over 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).

V. CEREBRAL OEDEMA

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

Diagnosis of cerebral oedema should be suspected if there are one diagnostic criterion, 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%.

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 < 12)
- ☒ 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 arousable
- ☒ Diastolic blood pressure > 90 mm Hg
- ☒ Age <5 yr

If suspected then treat:

Exclude Hypoglycaemia (which can mimic cerebral oedema)

Mannitol 0.5 grams per kg IV infusion over 20 minutes

Repeat if there is no response in 30 minutes

Halve IV infusion rate.

This is a medical emergency – call the department consultant and notify ICU and endocrinologist.

VI. Treat any precipitating factor(s) for DKA

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.

Criteria for Discharge from ICU

1. Venous pH > 7.25 and base deficit < 6
2. Potassium > 3.1 mmol/L and replacement < 0.3 mmol/kg/hour
3. GCS =15
4. Heart rate in normal range
5. Oxygen requirement < 1lt/min (child) or <50% (adult)

Note: The Discharge process requires careful medical and nursing handover to the paediatric endocrinology team.

Traps!

Blood glucose levels do not need to be markedly elevated in DKA.

Levels 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 ketones if sick.

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 this receptor in their nose.

Abdominal pain and vomiting may be due simply to the ketonemia itself, but can mask gastroenteritis, appendicitis or other intra-abdominal problems.

The commonest cause of recurrent diabetic ketoacidosis is insulin omission.

REFERENCES:

1. The Australian Clinical Practice Guidelines on the Management of Type 1 Diabetes in Children and Adolescents, APEG, 2005
www.nhmrc.gov.au/publications/files/cp102.pdf
2. International Society for Pediatric and Adolescent Diabetes ISPAD Clinical Practice Consensus Guidelines, 2006-2008
www.ispad.org/FileCenter.html?CategoryID=5

AUTHOR: Dr Bruce King

CONSULTATION:

ICU Executive JHH – Dr Martin Rowley, Dr. Ken Havill
Emergency Executive JHH – Dr Mark Lee
Endocrine Team JHCH – Dr Trish Crock, Dr Don Anderson

SIGN OFF:

Kaleidoscope CPGAG approval 3rd August, 2009
KGNS Quality approval August, 2009