

## DIABETIC KETOACIDOSIS (DKA)

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### Background

Diabetic ketoacidosis (DKA) is a life-threatening metabolic disorder resulting from decreased effective circulating insulin, insulin resistance and increased production of counter-regulatory hormones. The frequency of DKA ranges from 16%-80% of children newly diagnosed with diabetes, depending on geographic location. It is the leading cause of morbidity and is the most common cause of diabetes-related deaths in children and adolescents with type 1 diabetes. Mortality is predominantly due to cerebral oedema which occurs in 0.3% to 1% of all episodes of diabetic ketoacidosis in children.

**Diagnosis** is based on clinical suspicion followed by biochemical confirmation:

- Hyperglycaemia (blood glucose >11 mmol/L),
- Metabolic acidosis (venous pH <7.3, Bicarbonate <15 mmol/L)
- Ketonuria / ketonaemia

### Key Points

- Always use 0.9% NaCl as initial fluid
- Never bolus insulin
- Insulin infusion to start at 1 hour after initial fluids (depending on clinical severity)
- >90% do not need fluid boluses
- Call for advice earlier rather than later

Individuals may present with DKA at any age and with or without a previous diagnosis of diabetes.

Risk factors for DKA in children with newly diagnosed type 1 diabetes include:

- young age (<5 years)
- first degree relative with type 1 diabetes
- lower socioeconomic status
- medications (high dose glucocorticoids, antipsychotics, diazoxide and immunosuppressives)

Risk factors for DKA in children with established type 1 diabetes include:

- poor metabolic control
- previous episodes of DKA
- female adolescents
- psychiatric disorders (especially eating disorders)
- lower socio-economic status.

**Infection is a common precipitant, often as a result of inadequate or omitted insulin.**

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## DIABETIC KETOACIDOSIS (DKA)

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### Pathophysiology:

- Increased hepatic and renal glucose production and impaired peripheral glucose utilisation, leading to hyperglycaemia and hyperosmolality,
- Increased lipolysis and unrestrained production of ketoacids (betahydroxybutyrate and acetoacetate), resulting in ketonaemia and metabolic acidosis.
- Osmotic diuresis (due to hyperglycaemia), loss of electrolytes and dehydration, which can exacerbate the metabolic acidosis.

### Principles of Management

Diabetic ketoacidosis (DKA) is a medical emergency. These are guidelines and cannot replace careful clinical observation and judgment in treating this potentially fatal condition (from cerebral oedema, acute hypokalaemia or hypoglycaemia).

**Contact the ENDOCRINE CONSULTANT on call in all cases (via ADHB operator or 021 974804).**

Those at **high risk for complications** (art pH<7.0, severe hyperglycaemia (BG > 50 mmol/L), cardiac decompensation, impaired consciousness) should be **discussed early**.

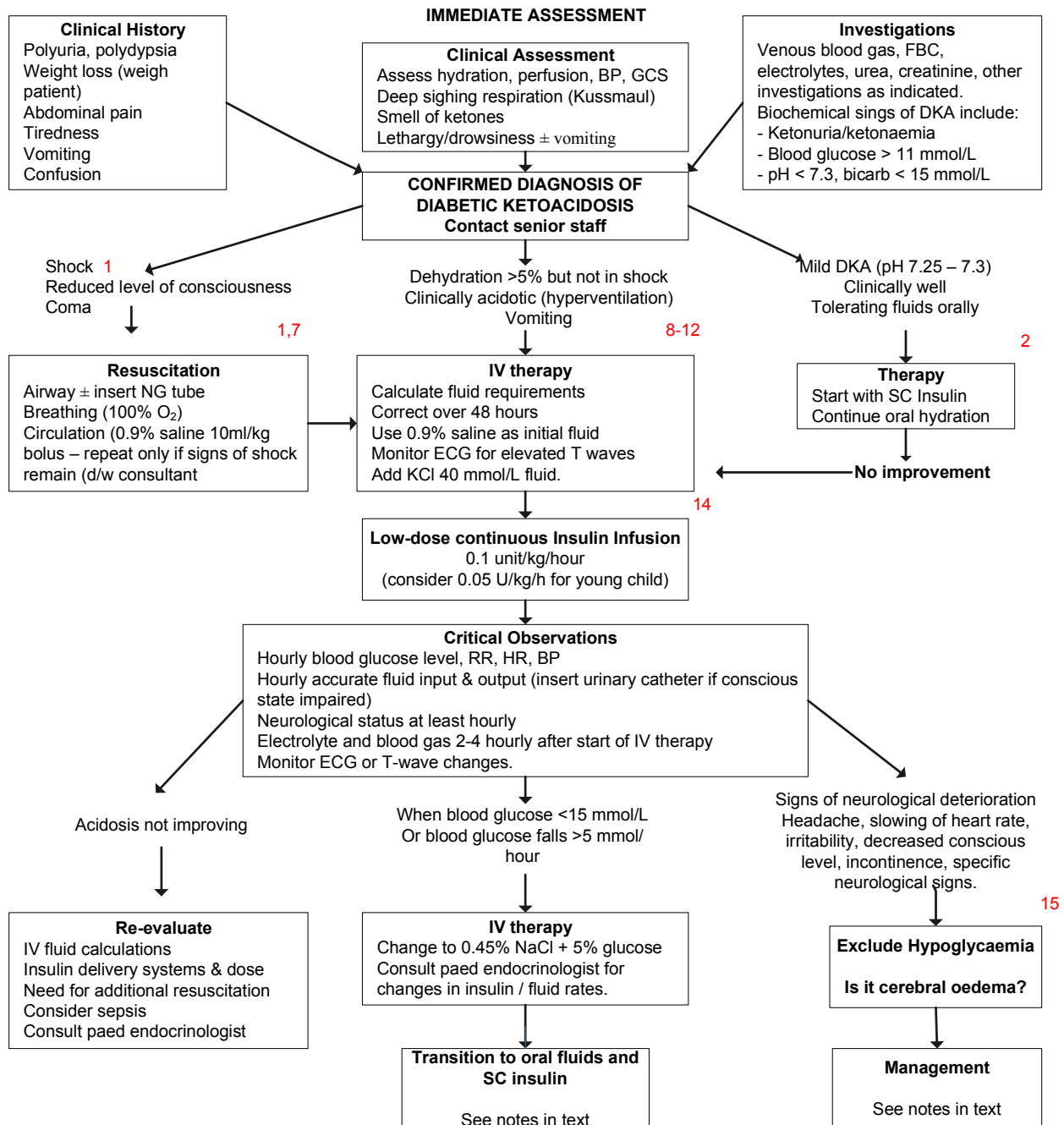
The aim of treatment is the slow, smooth restoration of clinical and biochemical normality while avoiding anticipatable complications. Usually the goal is a return to normal metabolic parameters within 48-72 hours. Safe treatment is dependent on careful observation of progress, biochemical monitoring and meticulous record keeping. While initial management will often be in the emergency department, careful consideration should be given to where the child should be managed for ongoing care (see below).

These guidelines are based on the national guidelines for DKA management developed by a working group of the Paediatric Society of New Zealand (see references) and reformatted for this publication. Guidelines are divided into background, an algorithm providing an overview, management notes and an appendix. Management notes are divided into instructions (left column) and rationale (right column).

## DIABETIC KETOACIDOSIS (DKA)

### Overview of Management

DKA is a serious life-threatening disorder and guidelines cannot replace careful observation and judgement. **Contact the on call PAEDIATRIC ENDOCRINOLOGIST in all cases (via ADHB operator or 021 974804).**



Modified from Clinical Practice Guidelines of Australasian Paediatric Endocrine Group, March 2005. Reference numbers refer to right margin notes in this guideline's text

## DIABETIC KETOACIDOSIS (DKA)

### Fluid Calculator

#### A. Assessment:

- A, B, C – look for shock<sup>1</sup>
- Neurological exam (GCS / AVPU)
- Diagnosis (glucose >11mmol/L, ketonuria, pH <7.30)<sup>2,3</sup>
- Assess hydration.<sup>8</sup>
- Blood tests & investigations including 5ml plain tube.<sup>4,5,6</sup>
- Measure / estimate body weight in kilograms: ❶ \_\_\_\_\_ kg

#### B. Resuscitation – if signs of shock are present<sup>1</sup>

- Consider airway +/- NG tube
- Oxygen (100% via mask)
- Gain secure IV access
- Fluid bolus (10ml/kg) of 0.9% saline (if shocked)
- Repeat fluid bolus only if signs of shock remain<sup>1,7</sup>
- Total fluid bolus given ..... ❷ \_\_\_\_\_ ml

#### C. Calculate Fluid Therapy:

- Determine **deficit** (ml/kg) based on estimated dehydration<sup>8</sup>
- Mild: thirsty, dry mucous membranes 4%=40ml/kg
- Mod: reduced turgor, abnormal respiration 6%=60ml/kg
- Severe: shock<sup>1</sup> 10%=100ml/kg

Enter estimate (ml/kg) here..... ❸ \_\_\_\_\_ ml/kg

Calculate total deficit: multiply ❶ by ❸..... ❹ \_\_\_\_\_ ml

Only if >20ml/kg fluid bolus given then subtract ❷ from ❹)..... ❹b \_\_\_\_\_ ml

Divide **deficit** over 48hr (divide ❹ or ❹b by 48) .. ❺ \_\_\_\_\_ ml/hr

Calculate **maintenance** fluids for next 48hr.<sup>9</sup>

Weight: First ten kg 4ml/kg/hr \_\_\_\_\_

Second ten kg 2ml/kg/hr \_\_\_\_\_

Every kg after 20kg 1ml/kg/hr \_\_\_\_\_

Total **maintenance** fluids ..... ❻ \_\_\_\_\_ ml/hr

Calculate **total hourly fluid rate**: add ❺ and ❻ ... ❼ \_\_\_\_\_ ml/hr

#### D. Type of fluid:

- Initial fluids:**<sup>10</sup> Start with 0.9% saline.
- Potassium:**<sup>11</sup> add 40mmol per litre (20mmol per 500ml) once insulin infusion has started. Consult if K<sup>+</sup>>6mmol/L or in acute renal failure.
- Dextrose:**<sup>12</sup> Change to 0.45% saline with 5% dextrose when BG <15mmol/L Higher dextrose may be required to maintain BG 10-15mmol/L without reducing insulin infusion.

- <sup>1</sup> Signs of shock: tachycardia, peripheral shutdown, and hypotension (late sign).
- <sup>2</sup> Mild hyperglycemia (BG < 30-35), even with ketonuria and mild acidosis (pH >7.25), can often be managed without IV fluids or IV insulin, particularly in the older child or known diabetic who is not vomiting or seriously dehydrated.

<sup>3</sup> Identify possible precipitants (infection, insulin omission)

<sup>4</sup> Blood: glucose, urea, electrolytes, calcium, blood gas, osmolality, ketones, FBC, 5ml plain tube (newly diagnosed, for diabetes associated antibodies), +/- culture

<sup>5</sup> Urinalysis: MC+s, ketones

<sup>6</sup> Consider CXR, throat swab

<sup>7</sup> Large fluid boluses are potentially dangerous and should be administered only if the patient is truly shocked. Only very rarely will a large (>20 ml/kg) bolus be required to maintain perfusion. **This decision should be made by paediatric endocrinologist.**

<sup>8</sup> Assessment of dehydration in DKA is difficult and often overestimated: rapid, deep mouth-breathing often dries out the oral mucosa; urine output is unreliable due to osmotic diuresis; acidosis reduces peripheral perfusion.

<sup>9</sup> Since most patients develop DKA over days, slow metabolic repair is generally safest. Overhydration may contribute to cerebral edema. In **most cases aim** to give deficit over **48hr**. If there is **hyponatraemia** (corrected Na >150mmols/L, see formula below) or **hyperosmolality** (osmolality >310mosmol/L, see formula below) give deficit plus maintenance **over 72 hours**.

<sup>10</sup> As the hyperglycemia resolves, the serum Na<sup>+</sup> should normally rise, with the corrected Na<sup>+</sup> remaining stable. A decrease in the corrected Na<sup>+</sup> is a sign of relative fluid overload, and may increase the risk of cerebral oedema. Decrease the fluid rate.

$$Na^{+}_{corrected} = Na^{+}_{measured} + (0.3 \times (\text{glucose} - 5.5))$$

$$\text{Osmolality} = 2(\text{Na}^{+} + \text{K}^{+}) + \text{glucose} + \text{urea}$$

<sup>11</sup> The total body potassium is invariably depleted, although the initial serum K<sup>+</sup> is often normal or high because of metabolic acidosis. Depletion is rapidly unmasked by therapy. If serum K<sup>+</sup> falls below 3.0mmol/L, the first warning for which could be flattening of T waves on ECG monitor, **give 0.5mmols/kg/hr for 4 hours** and reassess.

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### C. Calculate Insulin infusion.

Add 50 units of regular insulin (Actrapid or Humulin R) to 49.5ml of 0.9% saline. This will make a 1 unit/ml solution. It should be carefully labelled and can last for 24 hours. Piggyback infusion into IV fluids with a syringe pump.

No bolus; start at 0.1 unit/kg/hr<sup>13</sup>. (unless otherwise directed by paediatric endocrinologist)

Rate = 0.1ml/kg/hr \* ① \_\_\_\_\_ kg = ..... ② \_\_\_\_\_ ml/hr

### D. Other therapies

Sodium bicarbonate is rarely, if ever, needed and should be given only after discussion with a paediatric diabetes specialist.<sup>14</sup>

### E. PICU Admission:

Consider PICU admission (discuss with consultant) if

- <2 years,
- pH<7.1 (check with arterial pH),
- altered consciousness,
- need for arterial line,
- severe hyperosmolar dehydration,
- or inadequate staffing on Children's Unit.

Those in HDU(26a) should have ECG monitoring, which should also be considered for those not in HDU. Consider NG tube.

### F. Observation / Monitoring

Continuous ECG monitoring (elevated T-waves, arrhythmia)

Blood glucose hourly (meter)

Na<sup>+</sup>, K<sup>+</sup>, capillary/venous pH / HCO<sub>3</sub><sup>-</sup> every 2-4 hours

Strict fluid balance (may need IDC).

Check all urine for glucose and ketones.

Neurological observations at least hourly (see below)

Re-evaluate the appropriateness of fluid type frequently, anticipating the need to add or increase K<sup>+</sup>, dextrose, etc.

### G. Neurological Monitoring and Cerebral Oedema

Close neurological monitoring is required to detect the warning signs and symptoms of cerebral oedema, which usually develops before the classical symptoms of raised intracranial pressure.<sup>15</sup>

**Cerebral Oedema is a "Paediatric Blue 100" emergency.**

Suspect if: 2 major OR 1 major and 2 minor criteria:

**Major criteria:** age inappropriate incontinence, altered mentation, sustained deceleration in heart rate (>20 bpm) not related to sleep or initial resuscitation.

**Minor criteria:** vomiting, headache, lethargy (not easily roused from sleep), age<5 years, diastolic BP >90mmHg.

<sup>12</sup> When the patient's BG begins to fall, dextrose is added to the IV fluid to keep the BG in the 10–15 mmol/L range. This buffers against hypoglycemia and a too-rapid fall in the osmolarity. When BG <15mmol/L, change fluids to **0.45% Saline plus 5% Dextrose** (see appendix). If BG continues to fall, change to 0.45% Saline plus 10% Dextrose. If BG falls further reduce the infusion to 0.05 units/kg/hr = 0.05mls/kg/hr.

<sup>13</sup> This relatively high dose of insulin is chosen to inhibit ketogenesis and gluconeogenesis and should be maintained. The aim is to provide a gradual fall in osmolality (1-2 mosmols/hr) and blood glucose (4-5mmols/hr). If improvement is occurring faster than this, and the patient is receiving saline with no dextrose, **slow down the fluid** replacement rather than the insulin infusion. (It is acceptable for a more rapid improvement to occur initially with the resuscitation fluids, prior to starting insulin.) If however the patient has dextrose added, **increase the dextrose** concentration rather than adjusting the insulin infusion.

The consultant **may consider** starting at 0.05U/kg/hr in the very young.

<sup>14</sup> The acidosis of DKA is due to both ketone bodies and lactic acid, and it resolves with fluid and insulin replacement. There is **no** proven benefit to giving NaHCO<sub>3</sub>, and it does have a number of deleterious effects, including hypokalemia, metabolic alkalosis, and delayed clearance of ketones. Continuing acidosis usually means insufficient resuscitation. Bicarbonate should **only be considered** in children who are **profoundly acidotic** (pH < 7.0) **AND** who have **circulatory failure**.

<sup>15</sup> Subclinical brain swelling is common in children with DKA. Cerebral oedema accounts for more than half of the ~1–5% mortality rate of DKA in children. At highest risk are newly diagnosed diabetics, those aged <5 years, and those with pH <7.1. The aetiology is multifactorial and remains unclear, although over hydration has been implicated in several studies. Resuscitation is successful in only 50% of cases. Cerebral oedema is unpredictable but most commonly occurs in the first 24 hrs after starting rehydration when the general condition of the patient may seem to be improving. The **"too late" signs and symptoms** are: abnormal motor or verbal response to pain; decorticate or decerebrate posturing; cranial nerve palsy; neurogenic respiration and seizures.

**Vigilant observations throughout the first 24 hrs must not diminish.**

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### H. Cerebral Oedema Management:<sup>16</sup>

If suspected, treat immediately:

- Mannitol 0.5g/kg by rapid IV infusion
- Halve IV fluid rate
- PICU transfer (?intubation and hyperventilation)
- Seek neurosurgical review
- Consider radiological imaging (after stabilised).

**Cerebral Oedema is a "Paediatric Blue 100" emergency.**

<sup>16</sup> Mannitol should be immediately available during the treatment of DKA.

<sup>17</sup> Patients **newly diagnosed** with diabetes:  
Prepubertal 0.5 – 1 units/kg/day  
Pubertal 1 - 1.5 units/kg/day

2/3 pre breakfast; 1/3 pre dinner  
2/3 intermediate acting; 1/3 short acting

### I. Transitioning to SC insulin and oral fluids

When the patient's appetite has returned, HCO<sub>3</sub> >16, and blood glucose <15, **oral fluids can commence**; give water initially.

At the **next breakfast or dinner mealtime: stop the fluid** infusion, but **continue the insulin** infusion. **Give an appropriate dose of subcutaneous insulin.** After 15 minutes, **feed** the patient and finally **stop the insulin** infusion **30 minutes after the subcutaneous insulin.**<sup>17</sup>

### J. Appendices & References

1. Emergency contact details for paediatric endocrinologist: 021 974804 or 93 4307
2. Weight estimation: weight (kg) = 2(age+4)
3. Formulae for making 0.45% saline with 5% dextrose:  
Add 50ml 50% dextrose to 500ml bag of 0.45% saline  
OR add 25ml 50% dextrose to 500ml bag of 0.45% saline with 2.5% dextrose.
3. Guide / check of fluid administration (including deficit and maintenance) rate to be given over 48hr according to degree of hydration. This does not account for fluid bolus.

Weight (kg)	Mild <4%	Mod (4-7%)	Sev >7%	Weight (kg)	Mild <4%	Mod 4-7%	Sev >7%	Weight (kg)	Mild <4%	Mod 4-7%	Sev >7%	Weight (kg)	Mild <4%	Mod 4-7%	Sev >7%
5	24	27	31	20	75	87	104	<b>38</b>	101	125	156	<b>54</b>	124	158	203
7	33	38	43	22	78	91	110	<b>40</b>	104	129	162	<b>56</b>	127	162	208
8	38	43	50	24	80	95	115	<b>42</b>	107	133	168	<b>58</b>	130	167	214
10	48	54	62	26	83	100	121	<b>44</b>	110	137	174	<b>60</b>	133	171	220
12	53	60	70	28	86	104	127	<b>46</b>	113	141	180	<b>62</b>	136	175	226
14	58	67	79	30	89	108	133	<b>48</b>	116	146	186	<b>64</b>	139	179	232
16	64	74	87	32	92	112	139	<b>50</b>	119	150	191	<b>66</b>	142	183	238
18	70	80	95	34	95	116	145	<b>52</b>	122	154	197	<b>68</b>	145	187	244

Table adapted from the Royal Children's Hospital (Melbourne) Clinical Practice Guidelines for Diabetes Mellitus

### References:

1. Paediatric Society of New Zealand Working Group, National guidelines for the management of moderate to severe diabetic ketoacidosis (DKA) in children and young people, 2005.
2. Australasian Paediatric Endocrine Group, Clinical Practice Guidelines: Type I diabetes in children and adolescents, March 2005, website: [www.nhmrc.gov.au/publications](http://www.nhmrc.gov.au/publications).