DIABETIC KETOACIDOSIS (DKA)

PRACTICE GUIDELINE ©

DOCUMENT SUMMARY/KEY POINTS

Variations from this guideline may be required for individual patients but this should only occur under consultant supervision or in the intensive care setting.

- Refer to Algorithm for the management of DKA [next page].
- This guideline is for use within SCHN, including ward areas, emergency departments, intensive care units and other clinical areas. **Recommendations are based on the International Society for Paediatric and Adolescent Diabetes DKA Guideline (2018)**.
- Use standard concentrations for insulin preparation.

CHANGE SUMMARY

- Due for mandatory review; no major changes to practice.
- Minor changes made throughout to clarify/simplify instructions.
- Appendix 2 (how to increase glucose concentrations of IV fluid bags) now links to the Paediatric Injectable Medicines Handbook (PIMH): via CIAP or via the SCHN intranet

READ ACKNOWLEDGEMENT

- Clinical staff, nurses and medical officers, in Emergency Departments, Intensive Care Units and other Ward areas where diabetic patients are managed should read and acknowledge they understand the contents of this document.

This document reflects what is currently regarded as safe practice. However, as in any clinical situation, there may be factors which cannot be covered by a single set of guidelines. This document does not replace the need for the application of clinical judgement to each individual presentation.

Approved by:	SCHN Policy, Procedure and Guideline Committee	
Date Effective:	1st December 2021	**Review Period:** 3 years
Team Leader:	Endocrinologist	**Area/Dept**: Endocrine SCH and CHW

Algorithm for the Management of DKA

IMMEDIATE ASSESSMENT

Clinical History
Polyuria, Polydipsia
Nocturia, Nocturnal enuresis
Weight loss
Abdominal pain
Nausea and Vomiting
Lethargy, Confusion

Clinical Signs
Dehydration
Weight loss (weigh patient)
Deep sighing respiration (Kussmaul)
Smell of ketones
Lethargy/drowsiness/↓ LOC

Biochemical features & Investigations
Ketones ≥ 3 mmol/L on blood ketone meter (or in urine)
Elevated blood glucose
Acidaemia (pH venous < 7.3 or HCO_3 < 15 mmol/L)
Pathology tests: UEC, BGL, VBG, CMP, Osmolality, Antibodies (Insulin, GAD & IA-2), Coeliac screen, Serum IgA, TSH, fT4 and thyroid antibodies,
Other investigations as indicated

Diagnosis Confirmed – DIABETIC KETOACIDOSIS

Mild (3%) Dehydration and mild acidosis [pH 7.2 - 7.3]
Clinically well
Tolerating fluids orally

Therapy
Start with subcut insulin
Continue oral hydration

No improvement

Moderate (4-7%) Dehydration but not in shock and/or pH <7.2
Clinically acidotic (hyperventilation)
Vomiting

IV Therapy
Sodium chloride 0.9% 10 mL/kg over 30-60 min
Calculate fluid requirements
Replace deficit over 48 hours
Commence sodium chloride 0.9%
Add potassium chloride 40 mmol/L unless hyperkalaemic (consider more if hypokalaemic)

Commence IV insulin infusion 1 hour after fluids
(2 hours if hypokalaemic [potassium < 3.5 mmol/L])
Check glucose prior to starting insulin
Commence at 0.05-0.1 unit/kg/hour
lower rate indicated if hypokalaemic, at risk cerebral oedema, HHS and in all at SCH

Severe (≥ 8%) Dehydration and/or pH <7.1
Shock (reduced peripheral pulses, tachycardia, raised urea)
Reduced conscious level

Resuscitation
Airway ± NG tube
Breathing (100% Oxygen)
Circulation
Disability
Exposure
Fluids:
- Sodium chloride 0.9% 10 mL/kg bolus
- Reassess and repeat if signs of shock are not reversed
- Generally do not exceed 20 mL/kg
- Reassess hydration status and continue fluids for the current hydration status (post bolus)

Critical Observations
Hourly blood glucose level
Hourly fluid input & output
Neurological status at least hourly
UEC, VBG, glucose, osmolality, CMP (2nd hourly)
Monitor ECG for T wave abnormalities

Acidosis NOT improving

Re-evaluate
IV fluid calculations
Insulin delivery systems & dose
Consider sepsis

Neurological **WARNING SIGNS**: severe or progressive headache, slowing heart rate, irritability, decreased conscious level, incontinence, specific neurological signs

Exclude Hypoglycaemia

Is it Cerebral Oedema?

Management
Manage ABCDEF
Urgent consultation with ICU regarding management & transfer
Consider hypertonic sodium chloride 3% [3-5 mL/kg] or mannitol 0.5 – 1 g/kg
Adjust IV fluids to maintain normal BP, but avoid over hydration
Consider CT brain - **only** after patient stabilised

Blood glucose ≤ 15 mmol/L or falling > 5 mmol/L/hour after the initial fall associated with the first 1-2 hours of fluid therapy

IV Therapy
Change to IV fluids containing glucose 5% but can be titrated up to glucose 12.5% to maintain BGL > 5 mmol/L
Either sodium chloride 0.9% or Plasma-Lyte 148
Sodium chloride 0.45% should only be used after discussion with an intensivist or endocrinologist

Improvement
Clinically well, tolerating oral fluids

Transition to subcut Insulin
Start subcut insulin **then** stop IV insulin after an appropriate interval

Algorithm for the Management of diabetic ketoacidosis. Adapted from ISPAD Clinical Practice Consensus Guidelines 2018 Compendium[1]

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1 Background

1.1 Diagnosis of DKA

Diagnosis
Hyperglycaemia: blood glucose greater than 11 mmol/L
Venous pH less than 7.3 or bicarbonate less than 15 mmol/L
Presence of ketonaemia (greater than or equal to 3 mmol/L) or ketonuria

- It may be the initial presentation of type 1 or type 2 diabetes or develop in a patient with established diabetes, due to failure of insulin delivery or inadequate insulin in the context of intercurrent illness
- Recurrent DKA in adolescence is almost always due to insulin omission
- In patients on insulin pumps, DKA is often a result of an undetected infusion set failure

1.2 Clinical Features of DKA

- Dehydration
- Polydipsia and polyuria
- Nocturia, nocturnal enuresis
- Weight loss
- Kussmaul respiration (high respiratory rate and large tidal volume giving a sighing quality)
- Acetone smell on the breath and flushed cheeks due to ketosis
- Shock (rapid pulse rate, low blood pressure, poor peripheral circulation, mottling and peripheral cyanosis, lactic acidosis)
- Nausea, vomiting (may be mistaken for gastroenteritis)
- Abdominal pain (may mimic an acute abdominal condition)
- Weakness, fatigue
- Disordered sensorium (disoriented, drowsy, or comatose)

1.3 Pathophysiology

- Insulin deficiency causes hyperglycaemia and ketogenesis
- The presence of ketones (beta-hydroxybutyrate and acetoacetate) causes acidosis
- 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

1.4 Complications of DKA

Cerebral oedema	*Hypokalaemia*	*Thrombosis*	*Hyperglycaemic Hyperosmolar State [HHS] and mixed HHS DKA*
Usually in the first 24 hours after therapy is started. Usually presents with decreased consciousness, headache and signs of raised intracranial pressure, but there may be minimal symptoms until sudden collapse. May become life-threatening due to brain herniation. The cause of cerebral oedema is controversial; more recent data suggest that dehydration and cerebral hypoperfusion are associated with DKA-related cerebral oedema, since the degree of cerebral oedema that develops during DKA correlates with the degree of dehydration and hyperventilation at presentation, but not with initial osmolality or osmotic changes during treatment. **Risk factors** for cerebral oedema: - severe acidosis and dehydration - new onset diabetes - younger age - longer duration of symptoms - extended history of suboptimal control (in those with existing diabetes - hypernatraemia, hyponatraemia, or falling serum sodium during therapy - excessive fluid replacement Cerebral oedema may occur unpredictably	Total body deficit of potassium (of ~ 3 to 6 mmol/kg) is present before initiation of therapy, irrespective of plasma concentration Potassium moves into cells as the acidosis is corrected with insulin administration Bicarbonate therapy increases the risk of hypokalaemia	There is an increased thrombotic tendency; this may manifest as dural sinus thrombosis, basilar artery thrombosis, venous thrombosis and pulmonary embolism. Prophylactic low dose heparin is not routinely recommended, but should be considered and discussed with intensivists for patients who have additional risk factors for thrombosis e.g. central venous catheter, young age, severe dehydration or coexisting HHS.	**Criteria for the diagnosis of HHS are**: - Plasma glucose greater than 33 mmol/L - pH greater than 7.3 - Serum bicarbonate greater than 15 mmol/L - Absent or mild ketonaemia - Calculated serum osmolality greater than 320 mOsm/kg: [(2 x corrected plasma sodium) + plasma glucose] - Altered consciousness or seizures “Mixed HHS DKA” is when the patient meets criteria for DKA with acidosis and ketosis, and the calculated serum osmolality is greater than 320mOsm/kg DKA treatment should be modified after discussion with an intensivist and endocrinologist on-call. (see Step 4: ongoing management)

2 Procedure

Step 1: Initial Assessment and Investigations

- **Resuscitation**: **A**irway, **B**reathing, **C**irculation, **D**isability (neurological assessment), **E**xposure, **F**luids
- Level of consciousness (Glasgow Coma Scale)
- Degree of dehydration. Clinical estimates of volume deficit in DKA are subjective and often inaccurate (both over and under). A reasonable assumption is 5-7% dehydrated in moderate DKA and 8-10% dehydrated in severe DKA. The severity of DKA is categorised by the degree of acidosis[1]:
  - Mild: venous pH <7.3 or serum bicarbonate <15 mmol/L
  - Moderate: venous pH <7.2 or serum bicarbonate <10 mmol/L
  - Severe: venous pH <7.1 or serum bicarbonate < 5 mmol/L
- Measure blood glucose and blood ketones with bedside meter, (or urinalysis for ketones if blood ketone meter not available)
- Send baseline blood sample for:
  - Blood glucose level (BGL), urea, electrolytes and creatinine (UEC), calcium, magnesium phosphate (CMP), osmolality, venous blood gas (VBG) and full blood count (FBC) and other investigations as indicated.
  - Calculate the corrected sodium = measured sodium + 0.3 (BGL - 5.5) mmol/L. If corrected sodium is greater than 150 mmol/L, discuss with the on-call intensivist.
  - Calculate the anion gap ([sodium + potassium] – [bicarbonate+ chloride]).
  - If newly diagnosed diabetes add insulin autoantibodies (IAA), glutamic acid decarboxylase (GAD), tyrosine phosphatase antibodies (IA-2), thyroid stimulating hormone (TSH), free T4, thyroid autoantibodies (thyroglobulin antibody, TRAB, TPO antibody), total serum IgA and coeliac screen.
- Consider a source of infection, which may have precipitated the onset of DKA
- Obtain patient's weight, and most recent prior weight if available and height (when feasible) so that BMI can be determined

Step 2: Start IV fluids

- If moderate dehydration (5-7%) give 10 mL/kg sodium chloride 0.9% over 30-60 minutes to restore the peripheral circulation
- If severe dehydration (8-10%) give 10 mL/kg bolus (5-10 minutes) of sodium chloride 0.9%. Reassess and repeat if signs of shock are not reversed. More than 2 boluses are rarely required

Ongoing rehydration fluids:

- **Reassess hydration status PRIOR to starting ongoing rehydration**
- **Start rehydration** with sodium chloride 0.9% at a rate to give maintenance plus correction of fluid deficit over 48 hours (see table of IV fluid rates in Appendix 1 as a quick guide)
  - Consider reducing rehydration rates if large volume fluid resuscitation has already been given (greater than 20 mL/kg)
  - Plasma-Lyte 148 is a suitable alternative if pre-packaged glucose 5% preparations are available but should not be used outside of the tertiary hospitals
  - The maximum weight is 70kg when calculating deficit and maintenance fluid rates, unless otherwise clinically indicated; to avoid excessive amount of fluid in obese and older patients

- **Add potassium chloride to the rehydration fluid**
  - If hyperkalaemic (>5.5 mmol/L), oliguric or known to have renal failure, withhold potassium until urine output is documented and potassium less than 5.5 mmol/L. Ask child to void.
  - If normokalaemic (3.5 – 5.5 mmol/L) add 40mmol/L potassium to rehydration fluids AFTER the initial volume expansion and concurrent with starting insulin infusion
  - If hypokalaemic (<3.5 mmol/L) **discuss with intensivist or endocrinologist**. Add 60mmol/L potassium chloride (or potassium dihydrogen phosphate) to the intravenous fluids and before starting insulin therapy. Consider delaying commencement of insulin or reducing insulin infusion rate to 0.03 – 0.05 units/kg/hour.
  - Pre-mixed solutions containing potassium chloride should be used wherever possible
  - DO NOT use potassium containing fluids to give a fluid bolus
  - Reassess with electrolyte results initially every 1-2 hours, then every 2-4 hours
  - Consider potassium dihydrogen phosphate if patient is hyperchloraemic or serum phosphate falling. Consult with the endocrinologist or intensivist on-call. Monitor calcium levels if phosphate is administered
  - Note: Plasma-Lyte-148 contains 5 mmol/L of potassium chloride – this must be taken into consideration when adding extra potassium

  (*Refer to Potassium Management Practice Guideline for more information*)

Step 3: Insulin infusion

- Delay starting insulin until 1 hour of fluid administration has been given (or delay longer if hypokalaemic while further fluids containing potassium are given- discuss with endocrinology team)

- Commence insulin infusion at 0.05 – 0.1 units/kg/hour with a 50 mL syringe pump (or volumetric infusion pump), **as a sideline** to the rehydration fluid (to avoid unopposed insulin infusion if a cannula fails)
- Consider using the lower insulin dose if hypokalaemic, young age, risk factors for cerebral oedema exist or HSS are present
- Insulin infusion rate of 0.05 units/kg/hour (or lower) is always commenced **at SCH Randwick**
- Prime the IV line with the prepared infusion and discard a small amount
- A new insulin infusion must be prepared every 24 hours as detailed below:

<table>
<tr><th>If using a “smart” syringe pump with dose error reduction software*</th><th>If no smart pump is available</th></tr>
<tr><td>Patient >10kg:
Add 50 units of insulin (Actrapid or Humulin R) to a 50 mL syringe containing 49.5 mL sodium chloride 0.9% (so that concentration is 1 unit per mL)</td><td rowspan="2">All patients:
Add 100 units of insulin (Actrapid or Humulin R) to 1000 mL bag of sodium chloride 0.9% (so that concentration is 0.1 unit per mL)</td></tr>
<tr><td>Patient ≤10kg
Add 25 units of insulin (Actrapid or Humulin R) to a 50 mL syringe containing 49.75 mL sodium chloride 0.9% (so that concentration is 0.5 unit per mL)</td></tr>
</table>

* A smart syringe pump has dose error reduction software which enables the user to select the drug that they are administering, enter the concentration of the solution, weight of the patient and the intended dose per hour to be delivered. Smart pumps in use at SCH and PICU at CHW.

- Insulin infusion must be clearly labelled in accordance with MoH PD2016_058 Labelling of Injectable Medicines, Fluids and Lines

Step 4: Ongoing management

- Site second IV in the other arm for venous sampling (22 gauge cannula minimum) for subsequent blood sample for BGL, UEC, CMP, VBG and osmolality
- Any of the following criteria usually require admission to ICU, however these are not absolute criteria and any child causing concern should be discussed with the intensivist:
  - severe acidosis with initial pH less than 7.1
  - severe electrolyte disturbance (corrected sodium greater than 150 or less than 130 mmol/L, or potassium greater than 5.5 or less than 3.0 mmol/L)
  - blood glucose greater than 50 mmol/L

- hyperosmolar state (corrected serum osmolality greater than 320mOsm/kg) or if the patient meets the criteria for HHS
- abnormal or falling GCS
- other neurologic or haemodynamic compromise
- DKA (pH < 7.3) in a child aged less than 2 years

- **Monitor with**:
  - **i.** hourly pulse, respiratory rate, blood pressure, neurology observations and 2-4 hourly temperature;
  - **ii.** hourly blood glucose with bedside glucose meter or venous blood gas, while on IV insulin infusion;
  - **iii.** hourly accurate fluid balance chart;
  - **iv.** 2 hourly blood ketones with bedside meter. If blood ketone strips are not available, test all urine for ketones (until negative);
  - **v.** 2-4 hourly (initially 2 hourly) VBG, UEC, CMP, serum osmolality, BGL;
  - **vi.** Calculate the corrected sodium, (see below), anion gap and osmolality, (if serum osmolality is unavailable);
  - **vii.** reassess state of hydration every few hours
- Urinary catheter and nasogastric tube are not usually required but should be considered if reduced level of consciousness, severe dehydration and NGT if possible ileus

Monitor BGL and rate of fall of BGL

- Change fluids to those containing glucose 5% when BGL less than or equal to 15 mmol/L (or rapidly approaching 15 mmol/L), or if the rate of fall in BGL exceeds 5 mmol/L/hour after the first 2 hours
- Consider reducing the insulin infusion rate from 0.1 units/kg/hour to 0.05 units/kg/hour if rate of fall in BGL remains greater than 5 mmol/hour

Do not reduce the insulin infusion rate below 0.05 units/kg/hour until ketones are cleared and the acidosis and/or anion gap is corrected.

- Modification of the glucose content of the IV fluids may be required to maintain the BGL between 5 and 10 mmol/L or to reduce the rate of fall in glucose

  This can be achieved by either increasing the glucose concentration of the IV fluids (as detailed in Appendix 2) OR running two IV fluid lines, with the same sodium and potassium content, but with different glucose concentrations to allow titration. This method may be particularly useful during transportation when it is difficult to make up a new glucose concentration.
- When bedside ketones less than 1.0 mmol/L consider reducing the insulin infusion rate to 0.05 units/kg/hour if not already
- **At CHW** further adjustments should be made as directed in Appendix 3: IV Insulin Infusion for Diabetic Ketoacidosis – Adjustment Algorithm if required

Management of hypoglycaemia

- BGL 3.1 – 4 mmol/L:
  - Temporarily cease insulin infusion and check for problems with the administration of the glucose-containing fluids
  - Recheck blood glucose after 15 minutes
  - Increase glucose content of infused fluids and/or consider reducing the insulin infusion to 0.05 units/kg/hour once the BGL is greater than 5 mmol/L. Only reduce insulin infusion rate below 0.05 units/kg/hour if ketones less than 1.0 mmol/L, acidosis cleared and/or after discussion with intensivist or endocrinologist
- BGL less than 3.1 mmol/L or symptomatic hypoglycaemia:

  As above **PLUS** administer 2 mL/kg IV bolus of glucose 10% and recheck blood glucose in 15 minutes.

Monitor the corrected Sodium

Corrected Sodium = measured sodium + 0.3 (glucose - 5.5 mmol/L)

- The measured sodium concentration should rise as the glucose falls (corrected sodium should stay the same). Failure of this to occur indicates excess free water administration and is associated with an increased risk of cerebral oedema
- If corrected sodium changes by more than 5 mmol/L/hr and is greater than 150 mmol/L or less than 140 mmol/L, discuss with an intensivist.
- **Sodium chloride 0.9% (with addition of glucose and potassium as indicated) is usually appropriate for the duration of IV fluid therapy**
- Plasma-Lyte 148 is a suitable alternative and minimises the development of hyperchloraemic acidosis. Plasma-Lyte 148 should only be used if staff are experienced with its use, as it already contains 5mmol/L potassium chloride and 1% glucose, and pre-packaged solutions with different glucose concentrations are usually not available.
- **"Mixed HHS DKA"** is when the patient meets criteria for DKA with acidosis and ketosis, and the calculated serum osmolality is greater than 320mOsm/kg

  Calculated serum osmolality =
  (2 x corrected plasma sodium) + plasma glucose

  Children with mixed HHS DKA are at significantly higher risk of complications. The principles of management to be considered are:
  - Fluid deficits are greater than in DKA alone, and may be as high as 12-15%
  - An initial fluid bolus of 10 mL/kg sodium chloride 0.9% should be given, and will likely need to be repeated. No further boluses should be given unless overtly shocked.
  - Intravenous fluids after this **should be guided by an intensivist or endocrinologist**

- The insulin infusion should only be commenced after any fluid boluses and only at a lower rate (0.03 – 0.05 units/kg/hour) with consideration given to deferring starting the insulin infusion for 1-2 hours, depending on the rate of fall in the blood glucose
- Adjust fluid administration rate as needed to maintain normal blood pressure and account for excess urinary losses, whilst avoiding excessive fluid administration; avoid hypotension that might compromise cerebral perfusion pressure
- Aim for a gradual reduction of sodium (less than 0.5 mmol/L per hour) and glucose (less than 5 mmol/L per hour)
- Frequent circulatory and fluid re-assessment is essential
- In HHS, monitoring of mental state, continuous cardiac monitoring, CK levels and temperature are required. Rhabdomyolysis, malignant hyperthermia, arrhythmias and venous thrombosis are known complications of HHS
- Heparin may be considered if prolonged immobility or central venous catheter required

Cerebral oedema

- Severe or progressive headache, irritability, confusion, depressed consciousness, unstable body temperature, incontinence, specific neurological signs (e.g. cranial nerve palsy, especially III, IV, and VI) bradycardia and hypertension (late signs) may indicate increased intracranial pressure. Signs may be subtle and a high index of suspicion is needed
- Raised intracranial pressure due to cerebral oedema is an emergency requiring urgent treatment by:
  - Elevating the head of the bed to 30 degrees
  - Immediately reducing the rate of IV fluids by at least one third and adjust insulin infusion rate by the same amount. Adjust fluid administration rate as needed to maintain normal blood pressure while avoiding excessive fluid administration; avoid hypotension that might compromise cerebral perfusion pressure
  - Discuss with ICU consultant and consider further fluid restriction by temporarily stopping all IV fluids that reduce plasma tonicity, including insulin infusion
  - Give hypertonic saline (sodium chloride 3%), 3-5 mL/kg over 10 -15 minutes and repeat after discussion with an intensivist if there is no initial response. Monitor the change in corrected sodium
  - Mannitol 0.5 – 1 g/kg by IV infusion over 10 – 15 minutes may be used as an alternative to hypertonic saline. If no response or deterioration after hypertonic saline the addition of mannitol may be indicated after expert consultation with an intensivist (Hypertonic saline (3%) 2.5 mL/kg is equimolar to mannitol 0.5 g/kg)
  - Transfer to ICU, urgent intubation and ventilatory support required in patients with signs of herniation (pupil dilatation) or who are too comatosed to maintain compensatory hyperventilation
  - *After* treatment for cerebral oedema has been started, and following consultation with an intensivist, consider cranial imaging and neurosurgical consult

Monitor the anion gap (normal anion gap = 8 – 16 mmol/L)

- In parallel with pH improvement, the blood ketone level should fall and the anion gap should return to normal

Anion gap calculated as **(sodium + potassium) – (bicarbonate + chloride)**

- **If the anion gap is not falling**, check for problems with administration of the insulin infusion, and consider increasing the insulin infusion rate
- **If anion gap is falling**, but pH remains low due to hyperchloraemia, consider changing the rehydration fluid from sodium chloride to Plasma-Lyte 148, which has lower chloride concentration than sodium chloride 0.9% (98 mmol/L versus 154 mmol/L)

Bicarbonate

- **Bicarbonate is very rarely used**
- Bicarbonate administration is associated with paradoxical worsening of cerebral acidosis and hypokalaemia (due to correcting acidosis too quickly) and was of no benefit in a retrospective case series. Consider bicarbonate therapy only in patients with cardiogenic shock due to acidosis or with symptomatic hyperkalaemia, under the direction of the intensivist on-call

Step 5: Meals and transition to subcutaneous insulin

- Adjustment of the insulin infusion once the patient is eating:
  - For main meals: double the infusion rate when the patient starts eating, for one hour before returning to the basal rate
  - For snacks: double the infusion rate when the patient starts eating, for 30 minutes, before returning to the basal rate
- The infusion can be stopped when the patient is alert, stable [BGL less than 12, pH greater than 7.3, bicarbonate greater than 15] and ready to eat a meal
- When ketoacidosis has resolved, oral intake is tolerated, and the change to SC insulin is planned, a dose of basal insulin should be administered in addition to rapid-acting insulin. The most convenient time to change to SC insulin is just before a mealtime
  - stop the infusion:
    - 30 minutes after *rapid acting* subcutaneous insulin has been given OR
    - 90 minutes after regular or *long acting* subcutaneous insulin has been given
- Once established on subcutaneous insulin, the frequency of BGL monitoring can be reduced to pre-prandial (including meals and snacks), plus midnight and 3 am

3 References

1. Wolfsdorf JI, Glaser N, Agus M, Fritsch M, Hanas R, Rewers A, Sperling MA, Codner E. Pediatr Diabetes. ISPAD Clinical Practice Consensus Guidelines 2018: Diabetic ketoacidosis and the hyperglycemic hyperosmolar state. 2018 Oct;19 Suppl 27:155-177. doi: 10.1111/pedi.12701
2. Priyambada L, Wolfsdorf JI, Brink SJ, Fritsch M, Codner E, Donaghue KC, Craig ME. ISPAD Clinical Practice Consensus Guideline: Diabetic ketoacidosis in the time of COVID-19 and resource-limited settings-role of subcutaneous insulin. Pediatr Diabetes. 2020 Dec;21(8):1394-1402. doi: 10.1111/pedi.13118.
3. Kuppermann N, Ghetti S, Schunk JE, Stoner MJ, Rewers A, McManemy JK, Myers SR, Nigrovic LE, Garro A, Brown KM, Quayle KS, Trainor JL, Tzimenatos L, Bennett JE, DePiero AD, Kwok MY, Perry CS 3rd, Olsen CS, Casper TC, Dean JM, Glaser NS; PECARN DKA FLUID Study Group. Clinical Trial of Fluid Infusion Rates for Pediatric Diabetic Ketoacidosis. N Engl J Med. 2018 Jun 14;378(24):2275-2287. doi: 10.1056/NEJMoa1716816.

Appendix 1: IV Fluid Rates

Table 1: IV fluid rates (mL/hour) to give maintenance fluids plus replacement of the deficit over 48 hours

Weight (kg)		Dehydration			
		3%	5%	7%	10%
5		24	26	28	31
7		34	36	39	44
8		38	42	45	50
10		48	52	56	63
12		53	58	63	71
14		59	65	70	79
16		64	71	78	88
18		70	77	85	96
20		75	83	92	104
22		78	87	96	110
24		81	91	101	116
26		84	95	105	122
28		87	98	110	128
30		90	102	115	133
32		93	106	119	139
34		95	110	124	145
36		98	113	128	151
38		101	117	133	157
40		104	121	138	163
42		107	125	142	168
44		110	128	147	174
46		113	132	151	180
48		116	136	156	186
50		119	140	160	192
52		122	143	165	198
54		125	147	170	203
56		128	151	174	209
58		130	155	179	215
60		133	158	183	221
62		136	162	188	227
64		139	166	193	233
66		142	170	197	238
68		145	173	202	244
70		148	177	206	250

Appendix 2: Increasing glucose concentration of IV fluid bags

The following information is from the Paediatric Injectable Medicines Handbook:

There are variable practices with regards to the method to change the glucose concentration of an existing IV fluid bag containing glucose, sodium chloride or a combination of glucose and sodium chloride. Some methods consider the overage and withdrawing of the fluid before addition of 50% glucose solution while other methods limits accessing of the bags and calculates an approximate glucose concentration.

Both methods are described in appendix 2 of the **Paediatric Injectable Medicines Handbook (PIMH).** The methods are:

- Formulas considering the IV Fluid bag overage volume
- Formulas not considering the IV Fluid bag overage volume and withdrawal of fluid

It is important to ensure consistency in the preparation process for an individual patient to avoid variations in concentration and the amount of glucose and sodium delivered.

The formulas also apply for IV fluid bags containing sodium chloride.

The **Paediatric Injectable Medicines Handbook** can be accessed via the *SCHN Intranet* **or** *CIAP* (Clinical Information Access Portal).

The links below go to the **Appendices** section on the respective sites.

When clicked, look for appendix 2.

SCHN Intranet: http://injectables.webapps.schn.health.nsw.gov.au/pages/appendices

OR

CIAP: https://pimh.schn.health.nsw.gov.au.acs.hcn.com.au/pages/appendices *(for use outside of SCHN)*

Appendix 3: IV Insulin infusion for Diabetic Ketoacidosis – Adjustment Algorithm CHW only

Only to be used when bedside ketones <1.0 mmol/L or acidosis improved (bicarbonate 12-15 mmol/L)

The table indicates the change in insulin infusion rate from the current hourly rate according to the current BGL and rate of change of BGL in the previous hour.

	Change in BGL from last hour						
Current BGL (mmol/L)	**Falling quickly Fall of > 4 mmol/L/hour**	**Falling moderately Fall of 2-4 mmol/hour**	**Falling slowly Fall of 0.6-2 mmol//L/hour**	**No change (within 0.5 mmol/L of last hour)**	**Rising slowly Rise of 0.6-2 mmol/L/hour**	**Rising moderately Rise of 2-4 mmol/L/hour**	**Rising quickly Rise of > 4 mmol/L/hour**
> 15 mmol/L	Decrease by 20%	No change	Increase by 10%	Increase by 10%	Increase by 20%	Increase by 20%	Increase by 20%
10.1 –15 mmol/L (when BGL first falls to <15 mmol/L, first step is to add glucose to IV fluids before adjusting insulin infusion)	Decrease by 20%	No change	No change	Increase by 10%	Increase by 20%	Increase by 20%	Increase by 20%
5.1 – 10 mmol/L	Decrease by 20%	Decrease by 20%	Decrease by 10%	No change	No change	No change	Increase by 20%
4.1 – 5 mmol/L	Decrease by 50%*	Decrease by 20%	Decrease by 20%	Decrease by 10%	No change	No change	
3.1 – 4 mmol/L	Cease temporarily. Recheck BGL in 15 mins & recommence infusion when BGL >5 mmol/L at 50% lower than the previous rate Give IV glucose bolus 2 mL/kg of 10% glucose only if symptomatic						
< 3 mmol/L or symptomatic hypoglycaemia	Cease temporarily Give IV glucose bolus 2 mL/kg of 10% glucose. Recheck BGL in 15 mins & when BGL >5 mmol/L recommence infusion at 50% lower than the previous rate						

NB: Call the endocrinologist/intensivist on call if acidosis is not improving

Appendix 4: Abbreviations

BGL	Blood Glucose Level
CMP	Calcium, Magnesium, Phosphate
CK	Creatine kinase
CRT	Capillary Refill Time
DKA	Diabetic Ketoacidosis
ECG	Electrocardiogram
fT4	free thyroxine
FBC	Full Blood Count
GAD	Glutamic Acid Decarboxylase
GCS	Glasgow Coma Score
HSS	Hyperglycaemic Hyperosmolar State
IAA	Insulin autoantibodies
IA-2	Tyrosine Phosphatase Antibodies
NGT	Nasogastric tube
TSH	Thyroid Stimulating Hormone
UEC	Urea, Electrolytes, Creatinine
VBG	Venous Blood Gas