

Diabetic Ketoacidosis (DKA) in Pregnancy:

A Management Guideline for the Department of Obstetrics and Gynaecology



Tygerberg Hospital

Context:

Diabetic Ketoacidosis represents a *hyperglycaemic and obstetric emergency* posing significant risk for maternal and fetal morbidity and mortality.

Although typically encountered in Type 1 Diabetes mellitus (T1DM), pregnant women with any form of hyperglycaemia (including Type 2 Diabetes mellitus (T2DM), Gestational diabetes (GDM) and newly diagnosed diabetes) may be affected. DKA may develop at any time during pregnancy but is most common in the second and third trimesters.

For the following reasons, a high index of suspicion of DKA in any pregnant woman who is unwell, is judicious:

- Symptoms can be misleading, non-specific and resemble normal pregnancy.
- DKA can happen with mild hyperglycaemia (euglycaemic DKA).
- DKA can occur without prior diagnosis of diabetes.

Common presenting symptoms:

- Nausea, vomiting, abdominal pain,
- generalised fatigue, blurred vision,
- polyuria, polydipsia,
- evidence of volume depletion (dry mucous membranes, decreased skin turgor),
- tachycardia, hypotension
- Severe symptoms: Rapid, shallow breathing (Kussmaul's respiration), ketotic breath, altered mental state.

Diagnostic Criteria for DKA in Pregnancy:

1. <u>Hyperglycaemia:</u> BG > 11mmol/l

(Some guidelines omit hyperglycaemia in the definition. DKA in Pregnancy can frequently occur at lower or normal BG levels - euglycemic DKA. Therefore, if other criteria are met, DKA can be diagnosed in the presence of normal/near-normoglycaemia and is still an emergency).

- 2. <u>Metabolic acidosis</u>: $pH \le 7.3 / HCO_3 \le 18 mmol/l$ (High anion gap >12)
- 3. Ketonaemia: Blood ketones >3mmol/l / Urine ketones 2+
 - Note: Blood beta-hydroxybutyrate is the preferred test for the diagnosis and monitoring of diabetic ketoacidosis (this is currently best done on a validated point of care (Freestyle[®]) glucometer using ketone strips).
 - $\circ~$ Anion gap >12mmol/L indicates increased anion gap metabolic acidosis.

Aspects of Treatment:

- 1. Confirm diagnosis (clinical suspicion and blood gas confirmation)
- 2. Secure airway and oxygenation
- 3. Admit to highest level of care available (if Obstetric Critical Care Unit is full, initial management will need to be in labour ward until OCCU bed available).
- 4. Obtain IV access and insert indwelling urinary catheter.
- 5. Volume Repletion: Fluid rehydration
- 6. Electrolyte Repletion: Potassium replacement (hypokalaemia can be life-threatening mom and baby)
- 7. Address Hypoinsulinaemia and ketogenesis with IV Insulin: *weight-based fixed-rate insulin infusion to reverse ketosis* (avoid hypoglycaemia by adjusting IV fluid type)
- 8. Identify and address precipitating cause.
- 9. Ongoing close surveillance with monitoring of blood glucose, electrolytes and fluid status and adjusting treatment as indicated.
- 10. Delay fetal monitoring (even in viable babies) until the acute phase is over and then only start fetal monitoring once the mother is stable enough for potential surgery.
- 11. Patient education and prevention of recurrence of DKA.

FLUIDS		
Type of Fluid	Examples	Indications
Isotonic	0.9% NaCl (also Ringer's lactate, Balsol, Plasmalyte B)	For rehydration (in absence of hypernatremia)
Isotonic with 5% Dextrose	0.9% NaCl with 5% Dextrose	BG <14 and No hypernatremia
Isotonic with 10% Dextrose	0.9% NaCl with 10% Dextrose (see 'Calculation' section for method of mixing to 10% concentration)	BG <10 and No hypernatremia
Hypotonic	0.45% NaCl	Na >150 (add 5% dextrose) or Hyperchloremic (normal anion gap) acidosis

Interpretation of Blood Ketones (use Freestyle [®] glucometer with KETOstrips – available in OCCU)			
Value	Interpretation	Follow-up	
0.0 – 0.5 mmol/L	Normal	Nil unless clinical picture changes	
0.6 – 1.5 mmol/l	Ketones present	Check 4-hourly	
1.6 – 2.9 mmol/L	High risk for DKA	Treat if clinical suspicion of DKA. Otherwise recheck 2-hrly	
≥3.0mmol/l	DKA confirmed	Initiate treatment	

Treatment Pillars

o meatment mars				
1. FLUID	2. POTA	SSIUM	3. INSULIN	
Principle:_total volume loss (approximates 6-10L) should be replaced within the first 24-36 hours)	Principle: Hyp acidosis result potassium def serum potassia Rapid DKA cor	poinsulinaemia, and in total body icit <i>despite normal</i> <i>um concentration</i> . rection increases risk	Principle: IV Insulin therapy is needed to correct the underlying metabolic derangement – addresses both hyperglycaemia and ongoing ketogenesis.	
<u>Fluid:</u> Isotonic fluid (e.g. 0.9% NaCl) <u>Rate:</u>	ot hypokalaemia and life- threatening cardiac arrythmias or respiratory muscle weakness.		Delay insulin initiation until K+ is ≥3.5mmol/l.	
1 litre over an hour, then	AFTER the first	t litre of stat fluid. KCl	In a state in factions. Due no entries	
1 litre over 2 hrs (500ml/h), then 1 litre over 4 hrs (250ml/h)	supplementation should be added to the Rehydration fluid (1L) and run at appropriate rehydration rate. NB: MARK the fluid clearly as		Insulin infusion Preparation: (2 healthcare professionals shou be present / check concentration Need concentration of 1 unit/n	
Rehydration (rest of 24 – 48 hrs)	containing (x)	mmol/L KCl.	of short-acting insulin	
To avoid HYPOglycaemia, check	neck <u>Rehydration Phase:</u> <u>If K⁺ < 3.5 mmol/I:</u> Continue fluid rehydration but withhold IV insulin		E.g., Mix 200 units Actrapid® with 200 ml 0.9% NaCl	
prescribing fluid:			Prime IV-line with insulin solution before connecting to avoid delay in patient receiving insulin-containing	
<u>BG ≥ 14mml/l:</u>	Add 40 mmol	KCI/I in the rapid	fluid.	
0.9% NaCl at 150ml/hr	rehydration fluid vaculiter. Check K+ after an hour.		Fixed rate, weight-based Insulin infusion: 0.1 units/kg/h	
BG 10.0 -13.9 mmol/L:	<u>If K+ ≥3.5mm</u> c	ol/I: start insulin	(e.g., 6 ml/hr if 60 kg)	
5% dextrose @ 150ml/Hr	infusion 1hr after rehydration fluid started.		Titrate insulin hourly according to targets. Increase by 1 U/h if targets	
<u>BG <10mmol/L</u>	Maintenance Phase:		below are not reached.	
Assess acidosis:	K⁺ level	K ⁺ dose	Hourly targets:	
If still acidotic, aim to maintain insulin infusion but increase	(mmol/l)		 ✓ 10% to 20% drop in BG ✓ 0.5 mmol/l drop in blood 	
dextrose concentration to 10%.	<3.5	maintenance fluid	ketones ✓ 3 mmol/l increase in HCO3.	
infusion rate (e.g. 0.1u/kg to	3.5 – 4.9	20mmol KCl/l in 1L		
0.05u/kg) and continue 5% dextrose		maintenance fluid	Addressing acidaemia requires	
infusion.	≥5.0	No added KCl	insulin but takes longer to correct	
		Repeat K ⁺ in an	than hyperglycaemia does.	
Maintenance Fluid		noui	DO NOT STOP INSULIN while in	
hypernatremia)	Monitor K ⁺ 4hrlv once stable.		DKA (unless BG < 4 mmol/l)	
<i>Rate:</i> 125 - 150 ml/h				
(min 3L/24hr)			Keep BG between 8 - 12mmol/l until DKA resolved.	
			until DKA resolved.	

Cautionary notes: Insulin Therapy

Insulin type: Only short (and ultra-short)- acting insulin is used in the management of DKA.

Dangers of Hypoglycaemia:

While insulin therapy is essential to address acidaemia in DKA, hypoglycaemia, is life-threatening too.

Guard against allowing the blood glucose to drop to hypoglycaemic levels (<4.0mmol/L). Counter-regulatory hormones will be activated resulting in rebound ketosis, necessitating a longer duration of treatment.

Rapid correction of hyperglycemia and hyperosmolarity may also shift water rapidly to the hyperosmolar intracellular space and so induce cerebral oedema.

Management of Hypoglycaemia during a DKA:

- Confirm BG<4mmol/L with a second point-of-care glucose test.
- Stop insulin infusion.
- Give 50ml 50% dextrose IV stat.
- Repeat blood glucose 15min later.
- If still <4mmol/L repeat 50ml 50% dextrose IV stat.
- If >4mmol/L restart insulin infusion at half the rate. Use 10% Dextrose fluid.
- If there is a second hypoglycaemic episode, discard the insulin solution and mix a new bag (1unit Actrapid /ml 0.9% NaCl) and ensure that two health professionals are present to check the concentration is accurate.
- If hypoglycaemia is an ongoing concern, please escalate to the Department of Internal Medicine / Endocrinology for review.

Thromboprophylaxis:

The cumulative risk of diabetes, ketosis and pregnancy places the patient at high risk for thromboembolic events. Routine thromboprophylaxis is indicated. Use Enoxaparin 40mg SC daily or unfractionated heparin 7500iU SC twice daily as thromboprophylactic agents.

Nil per Os vs Early Oral Feeding:

At initial treatment of the DKA, it is safest and simplest to keep the patient nil by mouth.

If, however, the patient is stabilising and requests food, early oral feeding should not be discouraged even if acidosis is not fully resolved. <u>Prerequisites:</u> Patient awake, orientated and maintaining airway. Test tolerance of clear fluids prior to allowing solids. IV insulin infusion should be adequate to cover the requirements of small meals. No subcutaneous insulin boluses should be used at this time.

Monitoring				
MEASURE	ACUTE PHASE		STABLE PHASE	
	Frequency	Treatment Aim	Frequency	Treatment Aim
Blood Glucoso	Hourby	10-20% drop in	Hourly	BG range
Biobu Glucose	Hourry	BG per hour	(until 4hrs on SCI)	8-12mmol/L
Potassium (K ⁺)				
(On calibrated blood gas	Hourly	3.5-5.5	4-hourly	3.5-5.5
machine)				
		Increase of		Normalising of all
Blood gas	2-4hourly	3mmol/l/hr in	4-6hourly	narameters
		bicarbonate		purumeters
Input / Output	Hourly	0.5ml/kg/hr	4-hourly	0.5ml/kg/hr
Blood Ketones		0.5mmol drop in		
(point-of-care Freestyle	2-hourly	ketones / hr	12-hourly	Negative ketones
machine; ketone strips)		,		
		Aim for normal		Aim for normal
Plasma	12-hourly	range, adjusting	12-hourly	range, adjusting
Na⁺, K⁺, Cl⁻	12 1100119	fluids to	(daily once normal)	fluids to
		compensate.		compensate.

DKA Precipitant – Identify, Record, and Treat:

- Actively <u>look for infection/inflammation</u> by history and clinical examination (e.g., pyelonephritis, respiratory, chorioamnionitis, ear infection, sinusitis, cellulitis, tooth abscess, cholecystitis, deep vein thrombosis)
- Do <u>empiric screening</u> (e.g., urine-, sputum (for MCS / GXP) & blood cultures)
- Check for <u>other causes of DKA</u> (e.g., lipohypertrophy on injection sites, insulin dosing errors, skipping of
 insulin due to food insecurity or ignorance, using expired insulin, beta-sympathomimetic drugs or
 administered glucocorticoids (e.g., for fetal lung maturity).

Fetal Monitoring during DKA

- Electronic fetal monitoring prior to the mother being metabolically stable enough for an emergency anaesthetic, is NOT recommended.
- Typically, DKA is not an indication for delivery and CTG abnormalities seen in the acute phase of DKA, are usually reversible.
- Confirm (and document) the fetal heart once the mother is stable, and daily until maternal metabolic abnormalities are corrected.

Fetal Outcomes:

• Fetal mortality is quoted up to 36%. Surviving fetuses have a risk of long-term neurodevelopmental abnormalities due to a direct toxicity of ketone bodies on the fetal brain. For this reason, <u>please book all</u> women admitted with DKA for a fetal neuro ultrasound at TBH 3-4 weeks after DKA.

Transition to Subcutaneous Insulin

Prerequisites:

- Patient stable, awake & able to tolerate oral intake
- DKA is biochemically resolved strict criteria in pregnancy not established, but these can be used:
 - pH > 7.3
 - HCO3 ≥18mmol/
 - blood ketones < 0.6 mmol/l</p>
 - anion gap ≤12.0mmol/L

NB! INSULIN OVERLAP

To prevent reoccurrence of DKA, <u>IVI and SC insulin need to overlap by at least 4hours</u>. Do not stop IVI insulin earlier than 4hrs after first basal SC insulin (Protaphane[®] or Glargine) is given.

Glucose monitoring during transition:

- Continue hourly BG for 6 hours after IVI is stopped.
- Thereafter BG is checked before and after meals (pre & post prandial BG-monitoring)

Preventing Recurrent DKA:

- If new diagnosis of diabetes or not previously done:
 - Do Anti-GAD / Anti-Islet Antibodies.
 - Prescribe glucometer and testing strips.
- ALL patients with DKA
 - Early referral to Diabetes Educator for education on survival skills, insulin technique.
 - Confirm patient is provided with written information about the correct use of insulin.

Multi-disciplinary Approach is Essential:

Acute management should be the responsibility of the on-call team in the area where the patient is admitted (e.g.,

OCCU or labour ward). However, please involve an MDT early.

After-hours:

- Involve internal medicine registrar on call if there are concerns.
- Involve on-call Obstetrics consultant.

Office-hours:

- Refer patient to Special Care Team early to help streamline take-over of management when patient is stable.
- Notify Dr on call for Endocrinology. Request consult if needed.
- Refer patient early to Diabetes Educator (currently Mrs Lourentia van Wyk).

Useful Calculations:

- 1. How to mix 5% or 10% Dextrose solution: For 5% solution:
 - Start with 1L 0.9% NaCl
 - o remove 100ml volume
 - $\circ~$ add 100ml 50% dextrose to 1L 0.9% NaCl bag = 5% solution

For 10% solution:

- Start with 1L 0.9% NaCl
- o remove 200ml volume
- $\circ~$ add 200ml 50% dextrose to 1L 0.9% NaCl bag = 10% solution

2. Calculating the Anion Gap

[Na] – ([Cl] – [HCO3]) = anion gap (or use MDCalc[®] or equivalent application)

A difference of ≥12mEq/L along with a bicarbonate level (<18mEq/L) demonstrates an anion gap metabolic acidosis a defining feature of DKA.

Pitfalls & Pearls:

- <u>Avoid using only urinary ketones to diagnose DKA</u> as these may be present during fasting, exercise or pregnancy even without any metabolic abnormality. In DKA, the two main ketones produced are acetoacetate and betahydroxybutyrate. Urinary dipsticks measure only acetoacetate and is associated with both false positive and false negative results.
- <u>Practicalities Serum ketone testing:</u> Use dedicated ketone testing strips on Freestyle® point-of-care glucometers.
 Freestyle® glucometers and ketone sticks should be available in both C2A west and east (OCCU).
 The strips can be ordered from the pharmacy.

• Venous blood gas vs Arterial Blood gas:

*VENOUS blood samples are adequate to assess and monitor acidosis in the context of DKA. <u>Practical tips:</u>

- Still use HEPARIN PRIMED SYRINGES whether ABG or VBG.
- Do not take VBG from the IV cannula where IV Fluid was attached. Use different vein distal to cannula site.

VBG vs ABG - Interpreting aspects of blood gas:

<u>pH:</u> VBG pH is on average 0.03-0.04 less than the ABG pH values (even in DKA). <u>Bicarbonate:</u> HCO3 correlates well between VBG and ABG. (it's a calculated, not measured value. If absolute value needed – send serum to laboratory).

Lactate: good correlation. Difference <1. VBG useful to trend patient.

Base deficit (BE) correlates well between ABG, VBG

pO2 and pCO2: least correlation between VBG and ABG. If these values are imperative, an ABG should be done.

*ABG most relevant with hypoxaemic patients.

- If, for some reason, <u>IV insulin with infusion pump is not available (e.g., at referral hospital, while awaiting transport</u>), an alternative regimen of 0.1u/kg insulin lispro (e.g., Humalog[®]) can be used subcutaneously as a temporising measure until patient is transferred to unit where insulin driver available. This dose of insulin can be repeated every 2 hours. Hourly BG monitoring needs to continue. If Insulin lispro is not available, short-acting human insulin (Actrapid[®]) can be used with due cognisance that it's effect can last 6 hours, thus sequential dosages will likely have an additive effect, increasing the risk for hypoglycaemia.
- A <u>raised white cell count</u> should not be over-interpreted as this may be due to both the pregnancy and the DKA itself.
- If <u>hyperchloremic (normal anion gap) acidosis</u> occurs in the recovery phase of DKA (possibly due to use of 0.9% NaCl), minimise hyperchloraemia by using 0.45% saline or 5% dextrose water.
- <u>IV Bicarbonate</u>: This is generally not recommended (can worsen hypoxaemia, hypokalaemia, intracellular acidosis, and cerebral oedema) unless the ABG/VBG shows pH<6.9 (and then under expert supervision)
- <u>Individualised fluid management</u> needs to be made (in conjunction with OCCU consultant) in women with *kidney disease, heart failure or pre-eclampsia*.

*Summary of this guideline for quick reference available as Addendum 1.

Abbreviations used:		
ABG -arterial blood gas	IVI (intravenous insulin)	
BG (blood glucose) in mmol/l	MDT – multi-disciplinary team	
D (dextrose)	SCI (subcutaneous insulin)	
DKA – diabetic ketoacidosis	T1DM – Type 1 Diabetes mellitus	
GDM – Gestational Diabetes	T2DM – Type 2 Diabetes mellitus	
JDBS – Joint British Diabetes Society	VBG – venous blood gas	

References:

- SEMDSA. (2017). SEMDSA 2017 Guidelines for the Management of Type 2 diabetes mellitus SEMDSA Type 2 Diabetes Guidelines Expert Committee. JEMDSA 2017; 22(1)(Supplement 1): S1-S196. *Journal of Endocrinology, Metabolism and Diabetes of South Africa*. www.jemdsa.co.za
- Dhanasekaran, M., Mohan, S., & Egan, A. (2022). Diabetic Ketoacidosis in Pregnancy: An Overview of Pathophysiology, Management, and Pregnancy Outcomes. *EMJ Diabetes, August*. https://doi.org/10.33590/emjdiabet/10194487
- Eshkoli, T., Barski, L., Faingelernt, Y., Jotkowitz, A., Finkel-Oron, A., & Schwarzfuchs, D. (2022). Diabetic ketoacidosis in pregnancy Case series, pathophysiology, and review of the literature. *European Journal of Obstetrics and Gynecology and Reproductive Biology*, 269, 41–46. https://doi.org/10.1016/j.ejogrb.2021.12.011
- Mohan, M., Baagar, K. A. M., & Lindow, S. (2017). Management of diabetic ketoacidosis in pregnancy. *The Obstetrician & Gynaecologist*, 19(1), 55–62. https://doi.org/10.1111/tog.12344
- Bonner, S., & Schofield, J. (2019). Diabetic Ketoacidosis in Pregnancy. In *Obstetric Decision-Making and Simulation* (pp. 277–285). https://doi.org/10.1017/9781108296793.035

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DIABETIC KETOACIDOSIS (DKA) IN PREGNANCY (Addendum 1)

DKA Basics:

- Is caused by an extreme lack of insulin that forces the body to produce ketones for energy. •
 - Is synonymous with at least 10% dehydration.
 - Rehydration and insulin are used for treatment.
 - Hypokalaemia (life threatening) should be pre-empted & prevented.
 - Hypoglycaemia (due to the aggressive & fixed rate insulin infusion needed to reverse ketosis) is a real risk that should be prevented in advance by adjusting IV fluid type.

DKA defined: •

- Ketonaemia: Blood ketones >3mmol/l / Urine ketones >2+
- **Hyperglycaemia: BG > 11 mmol/l
- Metabolic Acidosis: pH < 7.3 / HCO₃ < 18 mmol/l
- **Interpretation of Blood Ketones:**
- 0.0 0.5 mmol/l: normal
- 0.6 1.5 mmol/l: ketosis (check 4 hourly)
- 1.6 2.9 mmol/l: high risk for DKA (check 2hrly)
- **Normoglycaemia does not exclude DKA in Pregnancy. Euglycemic DKA is a life-threatening emergency.

Summary of Management Guideline

- Initiate Rehydration. Keep pt NPO. Do a Venous Blood Gas (Arterial BG if A-line in situ).
- Before starting insulin, take the necessary precautions to prevent hypokalaemia.
- An hour after fluid initiation, start with a fixed rate of 0.1 U Insulin/kg/h ALONGSIDE potassium as indicated.
- Continue to monitor & prevent hypokalaemia.
- Monitor blood glucose (BG) hourly and take precautions to prevent hypoglycaemia.

1) Fluid

Rapid Rehydration (1st 8 hrs)

using 0.9% NaCl:

1 litre stat, then

1 litre over an hour, then

1 litre over 2 hrs (500ml/h), then

1 litre over 4 hrs (250ml/h)

Rehydration (rest of 24 – 48 hrs)

using 0.9% NaCl: 150 ml/h

Maintenance Fluid (0.9% Saline): 125 - 150 ml/h (min 3L/24hr)

AVOID HYPOGLYCAEMIA:

Aim to keep BG between 8 and 12

BG < 14: 5%D @ 150 ml/h (replace 100ml IV-fluid with 100ml 50% dextrose)

BG < 10: If still acidotic, use 10%D with same rate IVI (replace 200ml IVfluid with 200ml 50% dextrose) BG < 10: If acidosis is improving, continue 5%D & halve IVI dose (e.g., 0.1 to 0.05 U/kg) BG< 4: Stop insulin infusion. Give 50ml 50% dextrose IV stat. Repeat blood glucose 15min later. If >4mmol/L: restart insulin infusion at half the rate. Use 10% Dextrose fluid.

Type of fluid & Examples

<i>.</i>	
Isotonic: 0.9% NaCl (Normal Saline),	For rehydration & maintenance in the
Balsol, Plasmalyte B, Ringer's lactate)	absence of hypernatremia
Isotonic 5%D: replace 100ml of the	Use when BG < 14 mmol/l in the
isotonic fluid with 100ml 50% dextrose	absence of hypernatremia
Hypotonic 5%D: 0.45% NaCl with 100ml	Corrected Na > 150 mmol/l / Hyper- chloremic acidosis with normal
nula replaced by 100ml 50% dextrose	anion gap

2) Potassium

During rehydration phase:

Safe potassium level is required prior to initiating IV insulin.

If K⁺< 3.5 mmol/l: give 40mmol KCl in 1L rehydration fluid at appropriate rehydration rate.

Check K⁺ hourly

Rehydration and Maintenance:

Ongoing K⁺ monitoring and supplementation as needed in each vaculiter according to K⁺ level.

Potassium replacement (mmol/l)	
< 3.5	40 mmol KCl/L in Rehydration/ Maintenance fluid
3.5 – 4.9	20 mmol KCl/L In Rehydration / Maintenance fluid
<u>≥</u> 5.0	Nil (repeat K+ in 1hour)

Indications

Insulin 3)

NB: Do not start insulin if K⁺ is unknown or < 3.5 mmol/l

Prepare IV-insulin @ 1 unit/ml:

Mix 200 units Actrapid® with 200ml 0.9% NaCl. Prime IV-line with the insulin-solution before connecting. (At least 2 HPs should be present when IVI is prepared)

Give 0.1 units/kg/h (e.g. 6 ml/h if 60 kg)

Hourly targets:

- ✓ A 10% to 20% drop in BG
- ✓ 0.5 mmol/l drop in blood ketones
- ✓ 3 mmol/l increase in HCO₃

INFORM a senior doctor if targets are not reached; increase IVI rate by (1 U/hr) until reaching targets.

Keep BG between 8 - 12mmol/l until **DKA resolved:**

- Use 5% Dextrose when BG <14.
- Use 10% Dextrose when BG <10.

NB: Only if acidosis is improving, continue 5%D & halve the rate of insulin (e.g., from 0.1 to 0.05 U/kg) when BG falls below 10 mmol/l.

DO NOT STOP INSULIN while in DKA (unless BG < 4 mmol/l)

NB: To prevent DKA relapse, See STEP 7 (Transition to SC insulin) as a 4-6hr overlapping of IVI and SCI is required.

Monitoring (part of step 1 to 3)

- Hourly **blood glucose** (BG) aiming for a 10 to 20% drop in BG per hour.
- 1 2 Hourly blood ketones aiming for a 0.5 mmol/l drop per hour (6 hrly urine dipsticks only if blood ketone strips not in stock).
- Hourly **Potassium** on calibrated blood gas machine during acute phase; 4 hourly during stable phase.
- Blood gas hourly during acute phase aiming for a 3 mmol/l/h increase in bicarbonate; 4 6 hrly during stable phase.
- 12 hourly formal plasma Na⁺, K⁺ & Cl (if previously abnormal; otherwise daily)
- In & Output aiming for a urine output of 0.5 ml/kg (acute phase hourly; stable phase 4hrly).

4) Other Investigations

- Plasma BG (grey top)
- CXR when indicated
- Calcium, magnesium & phosphate
- HbA1C (if not done in the previous 6 weeks)
- Serum ketones only if blood ketone strips not in stock
 Calculate anion gap if refractory acidosis*
- Calculate anion gap in refractory actuosis
 (hyperchloremia can cause normal anion gap (≤ 12 mmol/l)
- Serum osmolality if Na⁺ abnormal on ABG/VBG
- Amylase / lipase if severe abdominal pain
- Antibodies for T1D testing (Anti-GAD &/Anti-Islet) if not yet done (even if known with T2D)
 *Insulin does not play a role in metabolic acidosis caused by hyperchloremia.

5) Prescription checklist

• Fluids

Potassium replacement

Insulin

- Blood ketone strips (if not in stock)
- NPO in initial phase of DKA
- Thromboprophylaxis
- Order glucometer with strips soon after admission (if new diagnosis / patient does not have own meter)
- Education on Survival Skills before discharge (causes & prevention of DKA, sick-day management, DSME, etc.)
- Ensure patient is provided with written information re the correct use of insulin at home

6) Thromboprophylaxis

• Routinely prescribe thromboprophylaxis: Enoxaparin 40mg SC daily or unfractionated heparin 7500iU SC twice daily.

7) Transition to subcutaneous insulin

- Patient stable, awake & able to tolerate oral intake
- DKA is resolved (pH > 7.3, HCO₃ ≥ 18 mmol/l, blood ketones less than 0.6 mmol/l & anion gap ≤12)
- NB: Ensure an overlap of at least 4 hours <u>after</u> Basal (e.g., Protaphane or Glargine) SCI is given before stopping IVI (to prevent recurrence of DKA)
- Continue hourly blood glucose (BG) for 6 hours after IVI is stopped; thereafter BG is checked before and after meals.

RECORD CAUSE OF DKA CLEARLY IN NOTES:

- Actively look for infection (e.g., urine-, sputum (for MCS / GXP) & blood cultures) and treat accordingly.
- Check for other causes of DKA (e.g., lipohypertrophy on injection sites, skipping of insulin due to food insecurity or ignorance & using expired insulin) in those known with diabetes and educate accordingly

BEFORE TRANSFER OF THE PATIENT FROM THE UNIT TO A WARD:

- Please ensure that the necessary education is done to avoid readmissions due to another DKA.
- A senior doctor and his/her consultant should approve discharge from the high care unit to a ward.
- Make sure that patient care, after discharge from critical care unit, is taken over by either Obstetric Special Care team or Endocrinology. Follow-up, patient education, and continuation of care is imperative.

Abbreviations used BG (blood glucose) in mmol/l; D (dextrose); Na & K in mmol/l; IVI (intravenous insulin); SCI (subcutaneous insulin) SME (diabetes self-management education; TOD (Target Organ Damage), HPs (healthcare professionals)