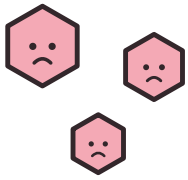
DKA, explained step by step.

The handover sheet for student nurses and new grads: what it is, why it happens, how to spot it, and what you'll be doing on shift.

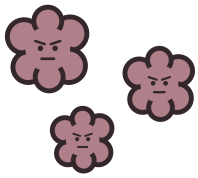
What does DKA mean?

D Diabetic



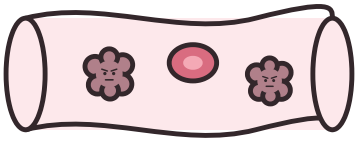
too much sugar

K Keto



ketones pile up

A Acidosis



the blood turns acidic

In one sentence

With no insulin, the cells can't use glucose, so the body burns fat for fuel. That makes **ketones**, which are acids. They build up until the **blood becomes acidic**.

Who gets it?

Mostly people with **type 1 diabetes**, and sometimes it's how type 1 is first diagnosed. It can also happen in type 2, especially on an **SGLT2 inhibitor** (the "-gliflozins").

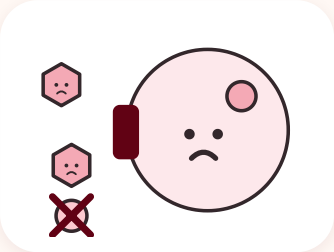
Why it matters

DKA is a **medical emergency**. The danger isn't only the sugar: it's the **acid**, the **dehydration** and the **potassium** shifts.

Don't get fooled: euglycaemic DKA

On an SGLT2 inhibitor (dapagliflozin, empagliflozin)? DKA can happen with a **near normal BGL**. If they look unwell, **check ketones anyway**.

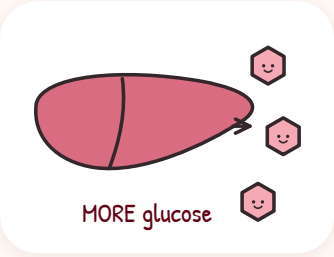
What happens inside the body



STEP 1

No insulin, no entry

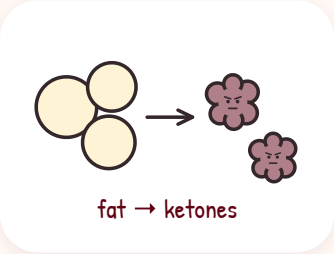
Insulin is the key that lets glucose into the cells. Without it, glucose stays in the blood and the cells are **starving next to a full plate**.



STEP 2

The body panics

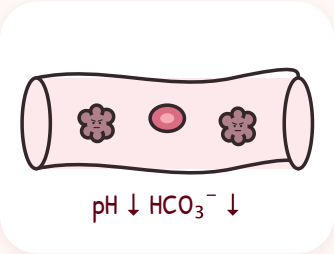
Stress hormones (glucagon, cortisol, adrenaline) tell the liver to release even **more** glucose. The BGL keeps climbing.



STEP 3

Plan B: burn fat

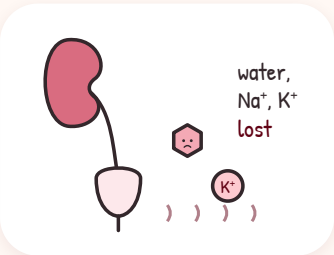
The body breaks down fat for energy. The liver turns it into **ketones**. Ketones are acids.



STEP 4

The blood turns acidic

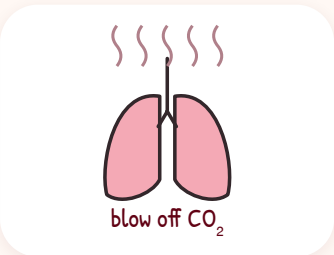
Ketones use up the bicarbonate (the blood's buffer). **pH falls, HCO_3^- falls, anion gap rises.** This is metabolic acidosis.



STEP 5

The kidneys can't keep up

Above roughly 10 mmol/L, glucose spills into the urine and drags **water, sodium and potassium** with it. Result: polyuria, thirst, severe dehydration.



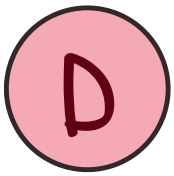
STEP 6

The lungs try to help

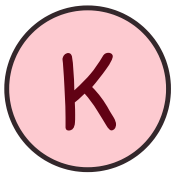
Deep, fast breathing (**Kussmaul**) blows off CO_2 to bring the pH back up. Fruity breath = acetone, a ketone you can smell.

How to spot it

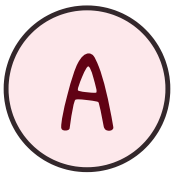
Memory trick: the letters ARE the diagnosis



Diabetes
BGL > 11 mmol/L
(or known diabetes)



Ketones
blood ketones ≥ 3 mmol/L



Acidosis
pH < 7.3 and/or
HCO_3^- < 15 mmol/L

Commonly used thresholds (e.g. JBDS). Exact cut-offs vary between Australian hospitals, so check your local protocol.

What you might see

- Polyuria and extreme thirst
- Nausea, vomiting, **abdominal pain** (often mistaken for gastro)
- Deep, fast breathing (Kussmaul)
- Fruity breath
- Dehydration: dry mouth, tachycardia, low BP
- Drowsiness or confusion

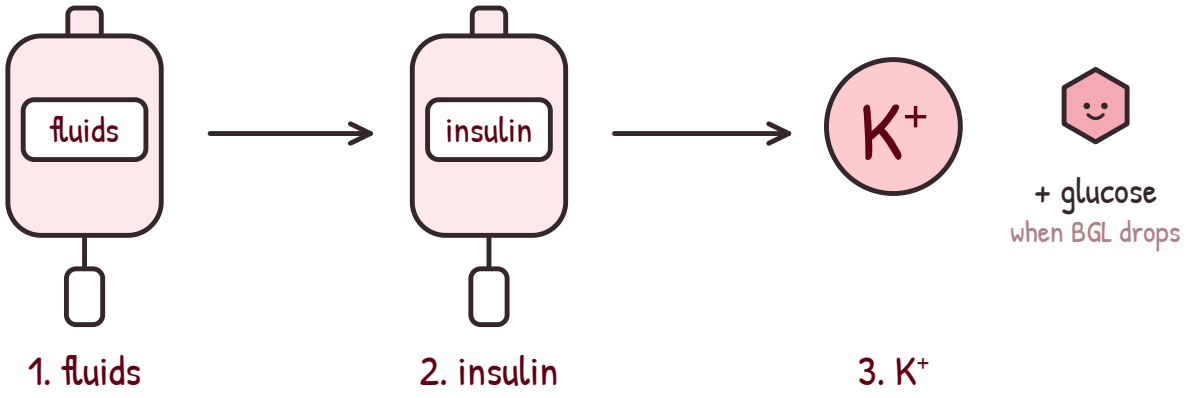
Memory trick: the 6 I's (triggers)

- **I**nfection (the most common)
- **I**nsulin missed, or a pump problem
- **I**nitial presentation (new type 1)
- **I**nfarction (MI, stroke)
- **I**ntoxication (alcohol, drugs)
- **I**atrogenic (steroids, SGLT2 inhibitors)

What the bloods usually look like

BGL	High (often > 11, but see euglycaemic DKA on page 1)
Ketones	High (≥ 3 mmol/L)
pH / HCO_3^-	Low: metabolic acidosis
Anion gap	High: $Na^+ - (Cl^- + HCO_3^-)$, the ketones are the "unmeasured" acids
K^+	Normal or high at first, even though total body K^+ is low
Na^+	Can look low because the high glucose pulls water into the blood
Urea / creatinine	Often up from dehydration

Treatment, and your job on shift



Fluids fix the dehydration. **Insulin** (usually a fixed-rate IV infusion) switches off ketone production. **Potassium** is replaced because insulin pushes K^+ into the cells. When the BGL comes down, **glucose is added** so the insulin can keep running safely.

What you'll monitor

CHECK	USUALLY
BGL	Hourly
Blood ketones	Hourly at first
VBG: pH, HCO_3^-, K^+	Regularly, often 2-hourly at first, then less often as they improve
Obs and neuro obs (GCS)	Hourly, or as the patient's condition needs
Strict fluid balance	Hourly urine output (may need an IDC)
Cardiac monitoring	If K^+ is abnormal

Frequencies differ between hospitals. Follow your local DKA protocol and chart.

Escalate early if you see

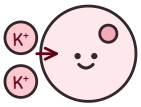
Headache or a drop in GCS (think cerebral oedema, especially in younger patients) · K^+ out of range · a hypo · ketones not falling or HCO_3^- not rising · poor urine output · any Q-ADDS or local MET criteria.

Memory tricks for your next shift



It's an acid problem, not a sugar problem.

That's why the insulin keeps running after the BGL is normal: it's still clearing ketones.



Insulin opens the door, and K^+ walks in too.

Serum K^+ can drop fast once insulin starts. Know the K^+ before and during the infusion.



Fill the tank first.

Fluids are usually the first thing started. Most patients are several litres down.



Sugar dropping? Add sugar, keep the insulin.

Glucose goes into the fluids so the insulin can keep working without a hypo.



Never drop the basal.

Long-acting insulin is usually continued during DKA treatment. A person with type 1 should never stop it, even when sick and not eating.



-gliflozin? Check ketones.

SGLT2 inhibitors can cause DKA with a near normal BGL.

When is it resolved?

Many protocols use **blood ketones < 0.6 mmol/L** and **pH > 7.3**, but criteria vary between hospitals. When switching to subcutaneous insulin, the IV infusion is usually **kept running for a while after** the first subcut dose, so there's never a gap in insulin.

Want more handover sheets like this?

Join the waitlist for The Handover Club: pathophys, bloods and meds explained the way uni didn't. **Link in the bio of @thehandoverclub.**