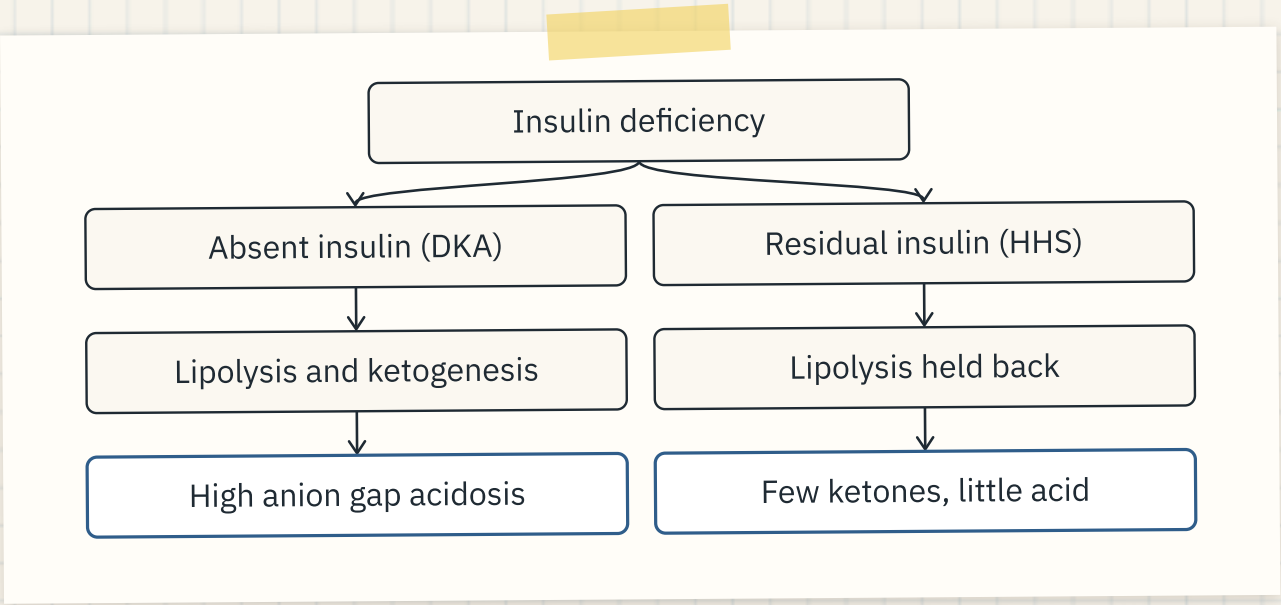
DKA vs HHS

Why only one makes acid



Step by step

1. Insulin low, counter-regulatory hormones surge
2. Gluconeogenesis and glycogenolysis: hyperglycaemia
3. Lipolysis, then ketone bodies, then bicarbonate falls
4. Glucose over about 180–200 mg/dL: osmotic diuresis

	DKA	HHS
Insulin	Absent or nearly absent	Residual
Lipolysis, ketones	Unrestrained	Held back
Acid-base	High anion gap acidosis	Little acid
Onset	Hours to a few days	Days to weeks
Mortality	Lower (treated)	5–10 times higher

DKA vs HHS

EFFECTIVE OSMOLALITY

$$2 \times Na^{+} + glucose / 18$$

HHS classic thresholds: glucose above 600 mg/dL, effective osmolality above 320 mOsm/kg

Rule of thumb

Anion gap = $Na^{+} - (Cl^{-} + HCO_3^{-})$; above 12 is high

Na^{+} 138, Cl^{-} 98, HCO_3^{-} 6: gap 34

Na^{+} 148, glucose 810: 296 + 45 ≈ 341

Exam traps

- Both crises are dehydrated and hyperglycaemic: the difference is the amount of insulin left.
- Residual insulin restrains lipolysis, so HHS has few ketones and little acid.
- Both cause an osmotic diuresis and deep volume loss.

DKA vs HHS pathophysiology: why only one makes acid

Video, written lesson, practice question and sources: medservatory.com/usmle-step-1/endocrinology/dka-vs-hhs-pathophysiology

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