KETOSIS DISORDERS by Nick Mark MD

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ETIOLOGY OF KETOSIS

Ketone bodies are a normal metabolic energy source. Excessive unregulated production of ketones, often accompanied by an **anion gap acidosis**, is seen in several disease states:

· **Starvation ketosis** – normal consequence of fatty acid (FA) metabolism. **Tx**: provide carbohydrates (IVF or PO): The pt will produce endogenous insulin normally. Treat other deficiencies (thiamine). Monitor for development of re-feeding syndrome.

· **Alcoholic ketoacidosis** (**AKA**) – seen in chronic alcoholics, often can be treated with dextrose containing IVF & thiamine. Treat concomitant alcohol withdrawal.

· **Diabetic ketoacidosis** (**DKA**) – caused by complete insulin deficiency, leading to marked anion gap acidosis (pH < 7.35) w/ elevated blood glucose. **Euglycemic DKA** is a variant seen with SGLT inhibitors & pregnancy, where blood glucose (BG) is normal.

· **Hypeglycemic hyposmolar state** (**HHS**) –partial insulin deficiency, causing minimal ketosis but marked increase in BG & osmolality. Glucosuria causes massive **volume loss**.

· **DKA/HHS Overlap** – features of both DKA & HHS and treated the same.

	Starvation Ketosis	AKA	Euglycemic DKA	DKA	DKA/HHS Overlap	HHS
Etiology	Physiologic switch to FA metabolism.	Seen in chronic alcoholics	Seen w/ SGLT1 inhibitors, pregnancy	**DM1** (or ketosis prone DM2) Young > elderly	**DM1/2** Highest mortality	**DM2** Elderly > young
BG (mg/dL)	<250	<250	<250	**400-800**	**800-1000**	**>800-1200**
Ketones	++++	++	+++	+++	+	-
Acidosis	-	++	+++	+++	++	+
Fluid deficit (L)	-	-	**2-4 L**	**6-8 L**	**6-10 L**	**8-10 L**

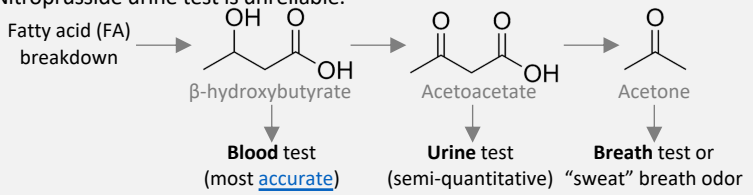
CAUSES/WORKUP OF DKA/HHS

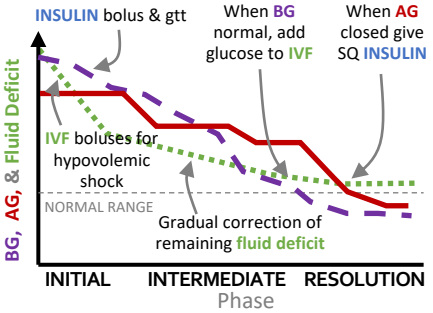
Identifying the cause of DKA/HHS is essential, because missing the underlying etiology is responsible for much of the morbidity/mortality. Consider the **5 I's**:

Etiology	Workup
Infection	BCx, Ucx, Procalcitonin, CXR, exam, POCUS
Ischemia	Lactate, Troponin, EKG
Inflammation	Lipase, CRP
Intoxication	Serum Osmolarity (Osm), Blood EtOH, Utox
Insulin (deficiency)	History (e.g. ran out of insulin, oral meds) Consider new meds (e.g. steroids, diuretics, etc)

WHICH KETONE TO CHECK?

Checking BHB is more accurate (fewer false positives & false negatives)
Nitroprusside urine test is unreliable.





TREATMENT OF KETOSIS:

The three pillars of treating DKA/HHS are **INSULIN** (to stop ketogenesis), **IVF** (to restore fluid deficit & correct hyperosmolarity), & **ELECTROLYTES** (to correct numerous derangements & prevent arrythmia)

HHS requires more **FLUID** and less **INSULIN** than DKA (because of greater fluid deficit and less acidosis)

Mild DKA can be treated with SQ INSULIN in the ED or medical wards (does not require ICU admission)

Expected AG is 2.5x albumin (usually <12 mOsm/L); with treatment AG should normalize to expected AG. If not improving consider concomitant lactic acidosis or other metabolic derangement.

Corrected Sodium accounts for spurious low Na+ measurements when blood glucose is high. For every 100 mg/dL increase in BG, the corrected Na is increased by 2.4 mEq/L.

INSULIN

GOAL: *correct* **BG**, **AG**, & acidosis
Monitor BG, Chem10

ELECTROLYTES

GOAL: *normalize* K^+, Mg^{2+}, Ca^{2+}, PO_4, and HCO_3 to avoid arrythmias
Monitor Chem10, ± VBG, EKG

IV FLUID

GOAL: restore circulating volume, *correct* **fluid deficit** & hyperosmolarity, avoid cerebral edema by correcting the corrected sodium gradually.
calculate **corrected Na** & **Fluid Deficit**
Monitor Osm, serum Na, urine output

INITIAL PHASE
Resuscitation & stabilization

Bolus **INSULIN** 10 units IV then Start **INSULIN** gtt 0.1 units/kg/hr

(Don't add K to IVF as it slows rate of infusion)

Replete K^+ to >4 mEq/L (ideally *both* PO and IV)

If HCO_3 < 6.9 give $NaHCO_3$ bolus ± gtt

(Acidosis greatly increases the risk of cardiac arrest during intubation; consider NIPPV)

If in hypovolemic shock, correct **rapidly** w/ IVF boluses (e.g. 1 L of LR or 0.9% NS)

(Use volume responsiveness to guide resuscitation; Consider balanced crystalloids. E.g Plasmalyte 148)

INTERMEDIATE PHASE
Gradual correction

Adjust **INSULIN** gtt to achieve 50 mg/dL/hr reduction in **BG**

Use caution if K^+ < 3.3 mmol/L; consider decreasing insulin gtt

Correct **Mg** deficits to reduce further K wasting

Replace **fluid deficits** with isotonic IVF ~200 ml/hr

Once **corrected Na** is >140 mEq/L switch to hypotonic IVF to avoid overcorrection (e.g. 0.45% NS, LR)

RESOLUTION PHASE
Normalization & de-escalation

Once **AG** closed, give SQ **INSULIN.** 0.1 units/kg. Stop **INSULIN** gtt 2 hrs later

Resume diet; Start basal, prandial, & correction **INSULIN**. Stop **IVF**. Begin oral **ELECTROLYTE** replacement.

Once tolerating PO

When BG < 200 mg/dL switch to glucose containing IVF (e.g. D5LR or D5 ½NS)