



Foundations Frameworks

Approach to Hyperglycemia and Insulin

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Diabetes is the most common cause of hyperglycemia. There are four major types:

1. **Type 1 Diabetes Mellitus:** This results from absent or diminished insulin production, usually due to autoimmune destruction of pancreatic beta cells. These patients require insulin supplementation
2. **Type 2 Diabetes Mellitus:** These patients have insulin resistance resulting from decreased receptor production or receptor defects. There are a wide variety of therapies for this, including insulin, oral hypoglycemics, and metformin
3. **Gestational Diabetes:** This is pathophysiologically like DM2 but resolves in the post-partum period
4. **Secondary Diabetes:** This may be like Type 1 or Type 2 DM but is due to other causes (e.g. pancreatitis-induced beta cell destruction, medication-induced insulin resistance, etc.). Exogenous steroids may also cause hyperglycemia and mimic diabetes

Hyperglycemia by itself is not usually dangerous and can be treated with IV fluids or supplemental insulin. Acute complications of hyperglycemia, however, need to be ruled out

- **New-onset DM2:** In the asymptomatic but hyperglycemic patient without a formal diagnosis of DM, workup and initiation of oral medications is appropriate
 - Labs: CBC, chem, UA, HgbA1C, LFTs, lipase
 - Diagnosis: HgbA1c > 6.5%
 - Meds: Start metformin 500 mg q12 hr or 850 mg qD
 - Disposition: Usually home, with PCP follow-up within 1 week
- **Hyperglycemia in DM:** In the hyperglycemic patient with a known diagnosis of DM, adverse consequences such as DKA and HHS (below) must be ruled out. This will often have a precipitant as well, most frequently med non-compliance or infection, but consider cardiac causes or other systemic stressors
 - Labs: VBG, CBC, chem, LA, workup for infectious precipitant (e.g. UA, CXR), consider troponin, BNP, etc.
 - Diagnosis: Hyperglycemia without other metabolic abnormalities
 - Meds: IV normal saline, insulin per pt's own sliding scale regimen
 - Disposition: Usually home, with PCP follow-up
- **Diabetic Ketoacidosis:** Mainly a complication of DM1, this does exist to a lesser degree in DM2 as well
 - Labs: VBG, CBC, chem, Mg, UA, LA, acetone (if available), workup for infectious precipitant (e.g. CXR)
 - Diagnosis: Hyperglycemia with **metabolic anion gap acidosis and ketosis** – make sure to apply Winter's formula to detect concomitant respiratory/metabolic alkaloses. Rarely, DKA may occur with euglycemia, but acidemia and ketosis must be present to make this diagnosis

- Meds: IV normal saline/LR bolus (corrects dehydration from hyperglycemia-driven osmotic diuresis), replete K as needed, then start insulin gtt (DKA results from insulin deficiency, with the ketosis being driven by unopposed glucagon)
 - Once the patient's glucose hits 250, change IVF to D5NS and titrate insulin for goal glucose 150-200 until acidosis/anion gap resolves
 - Check the K every hour and replete when it gets to 4.0
- Disposition: Generally the ICU
- **Hyperglycemic Hyperosmolar State**: Mainly a complication of DM2 (sometimes concurrently with DKA) and *usually involves mental status changes* due to fluid shifts
 - Labs: VBG, serum/urine osms, CBC, chem, LA, UA, workup for precipitant
 - Note that electrolytes will need to be monitored here too, but less aggressively than for DKA, as you don't need to give insulin (see below)
 - Diagnosis: Marked hyperglycemia **without ketosis** (but acidemia may be present if an acidemic state, e.g. sepsis, is the precipitant), longer prodrome than DKA
 - Meds: Copious IV fluids (fixes profound dehydration from hyperglycemia-driven osmotic diuresis), can consider insulin but this is not essential as insulin is still secreted in HHS
 - Disposition: ICU

References:

- Maloney G and Glauser J. *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 9e. Chapter 118: "Diabetes Mellitus and Disorders of Glucose Homeostasis." Elsevier 2018, pp. 1533-1547.
- Evans M, Schumm-Draeger PM, Vora J, King AB. A review of modern insulin analogue pharmacokinetic and pharmacodynamic profiles in type 2 diabetes: improvements and limitations. *Diabetes Obes Metab* 2011; 13:677.>