

Diabetes 101

Normal Physiology

When your blood glucose levels go up for whatever reason (food intake, stress, medication), the pancreas excretes insulin in response. Insulin acts like a “key” to unlock the cell and let the glucose go into the cell where it can provide energy.

Pathophysiology

Type I: In “insulin-dependent” diabetes, the pancreas does not produce insulin as it should. It can be idiopathic or related to autoimmunity (where the body launches an attack on the beta cells)

Type II: In this type of diabetes, there is either a insufficient release of insulin, or the body’s cells are resistant to insulin, making it more difficult for insulin to “unlock” the cell and let glucose in.

In both types, glucose does not enter the cell and instead builds up in the blood causing a host of problems in the body including organ damage, blindness and neuropathic pain. In severe cases it can lead to DKA (diabetic ketoacidosis) or HHS (Hyperglycemic Hyperosmolar State), both of which can be fatal.

Normal BS = 80-110

DM if symptoms present and BS \geq 200

DM if fasting BS \geq 126 on two occasions

Goal BS < 120
Goal HbA1c < 7%

Treating Diabetes

Insulin: For all Type I DM and many Type II DM

Oral Agents: *Sulfonylureas & Meglitinides* = stimulate pancreas to secrete insulin (Glipizide, Glyburide, Repaglinide)

Starch Blockers = taken at start of high-carb meal to inhibit the enzymes that break down CHO (Acarbose)

Biguanides = Decreases glucose production in the liver while increasing glucose utilization by the cells; used a lot in early stages of Type II (Metformin)

Insulin Sensitizers = Makes the insulin receptors on cells more sensitive (Avandia, Actos-TM)

DDP-4 Inhibitors and GLP-1 Analogs = Inhibit glucagon release, stimulate insulin release, may help with weight loss (Vildagliptin, Liraglutide)

Other *Amyloid Polypeptide* = Injectable drug that slows gastric emptying so sugar doesn’t absorb as fast

DKA

This is a life-threatening condition that usually affects Type I diabetics. It involves a blood glucose > 250, a pH < 7.3, a serum bicarb of < 15 **and** moderate to severe ketones in the blood or urine. Typical blood glucose ranges are 300-800 mg/dL.

DKA diagnostic criteria

BS > 250
pH < 7.3
serum bicarb < 15
ketones present

The pathophysiology of DKA breaks down pretty much like this:

Inadequate insulin → Cells don't get the energy they need and glucose builds up in the blood → the liver thinks the body needs more energy → liver converts glycogen to glucose while fats and proteins are converted into glucose → the use of fatty acids for energy leads to the buildup of ketones while serum glucose levels increase even more → serum osmolarity goes up and fluids are pulled from cells as the body tries to normalize it → the intracellular dehydration causes a catecholamine response which further stimulates glycogenolysis, lipolysis and gluconeogenesis → more glucose is released into the bloodstream → kidneys can't handle all the glucose and start spilling it into the urine along with some ketones → diuresis ensues → fluid losses are enormous → electrolyte imbalances and dehydration → hyperosmolarity increases while acidic ketones continue to build up in the blood → dehydration and acidosis worsens → GFR decreases → kidneys aren't able to excrete glucose as effectively → serum glucose levels increase → acidosis worsens → shock, coma, death.

Early Symptoms DKA

Polyphagia
Polydipsia
Polyurea
Headache
Fatigue

Later Symptoms DKA

Nausea/Vomiting (very common!)
Extreme fatigue
Kussmaul respirations
Weight loss
Dehydration
CNS depression
Decreased LOC

Treating DKA

- Fluid resuscitation can be substantial...often up to 6 Liters!
- Insulin gtt to decrease BS by 50-70 mg/dL each hour
- Once BS is below 200 to 250, change your IV fluids to something containing a little bit of glucose, to prevent hypoglycemia. Usually this is D5W with 0.45% NaCl.
- Monitor anion gap, serum osmolality, BUN, creatinine, sodium and potassium. As insuling "unlocks" the cell, the sugar travels into the cell taking the potassium along with it which can lead to hypokalemia
- When BS is within goal, anion gap is closed, and pH is normal, the pt will transition off the insulin gtt and get a dose of lantus and something to eat. Wait two hours, then d/c the gtt and start with sub-Q sliding scale insulin.

**Note that normal values vary by institution*

Anion Gap

$Na - (Cl + HCO_3)$

Normal value*:
8-12

HHS

Hperglycemic Hyperosmolar State (sometimes referred to as Hyperglycemic Hyperosmolar Nonketotic Syndrome) occurs in type 2 diabetes and can also be fatal if not treated. The key difference between HHS and DKA is that the fluid losses are often higher in HHS, the blood glucose is often higher, and ketosis is absent or mild. In addition, there is no utilization of fats/proteins for energy in HHS.

HHS diagnostic criteria

BS > 600
pH > 7.3
serum bicarb > 15
serum osmol > 320
mild or no ketones

The pathophysiology of HHS usually follows this path:

Inadequate insulin → Cells don't get the energy they need and glucose builds up in the blood → the liver thinks the body needs more energy → liver converts glycogen to glucose → serum glucose levels increase even more → serum osmolarity goes up → fluid is pulled from inside the cells into the vasculature leading to marked intracellular dehydration → kidneys can't handle all the glucose and start spilling it into the urine → diuresis ensues → fluid losses are enormous → electrolyte imbalances and dehydration → hyperosmolarity increases → ADH is released but it is too late → dehydration worsens → hypovolemia reduces renal perfusion, GFR decreases and oliguria results → kidneys aren't able to excrete glucose as effectively → SNS releases epinephrine in response to stress → serum glucose levels increase even more → cycle continues → hemoconcentration of blood causes clot formation and infarcts in brain, heart and lung while CNS dysfunction leads to shock, coma, death.

Treating HHS

- Rapid fluid resuscitation, can typically be 7 to 10 liters!
- Monitor serum sodium as you rehydrate with 0.9% NaCl, the fluids may need to be changed to a hypotonic solution if serum sodium rises. Generally, when the blood glucose reaches 200 to 250, the fluids are changed to D5W with 0.45% NaCl anyway.
- Insulin gtt with goal of dropping blood glucose by 50-70 every hour
- Hourly monitoring of blood glucose levels
- Electrolyte monitoring and replacement (especially K, Phos, Na)
- Monitor urine output, serum osmol, BUN, creatinine
- CVP monitoring if patient in hypovolemic shock
- Watch for signs of fluid overload

Educating the Patient

Many times the patient who presents with DKA or HHS will do so because this is a new diagnosis. Most of the time, your diabetic patient will be in the hospital for some other reason, but they usually need diabetic teaching every time. Topics to cover typically include:

- how to take a blood sugar
- how to calculate correctional dosage
- when to take blood sugar, when to take insulin or oral meds
- how to calculate carbohydrates and make good dietary choices
- why they shouldn't eat candy brought in by their family members
- importance of exercise
- foot care
- signs and symptoms of hypoglycemia and hyperglycemia
- sick day protocol

Sick Day Protocol

check BS every 2-4 hours
goal BS < 200 mg/dL
check for ketones (if DM Type I)
eat small meals often
drink a lot of sugar-free fluids

When to Call Doctor

BS > 240 for more than 24 hours
vomiting/diarrhea
severe pain
moderate to large ketones
neurological symptoms