

T2dm

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally, when you eat, your blood sugar rises. The pancreas (a small organ behind your stomach) releases insulin into your blood. Insulin is like a key that unlocks the door on muscle and fat cells so sugar can go inside and be used for energy. Insulin also tells the liver to stop making new sugar. In type 2 diabetes, the locks on those cell doors get rusty—they don't respond to insulin anymore (this is called insulin resistance). At first, your pancreas tries to help by making more and more insulin, like turning the key harder. But after months or years, the beta cells in the pancreas (the tiny factories that make insulin) get exhausted and start to fail. Now you have high insulin resistance AND low insulin, so sugar stays in the blood. High blood sugar acts like glue—it sticks to proteins in the walls of your smallest blood vessels (capillaries in the eyes, kidneys, and nerves) and slowly breaks them down. Bigger blood vessels (arteries to the heart and brain) also get stiff and clogged faster. Your body tries to flush out the extra sugar by making you pee a lot, which makes you very thirsty. But the real damage is silent for years: vision fades, kidneys leak protein and lose function, nerves burn or go numb, and the heart and brain are at high risk for heart attack and stroke. Every odd symptom—frequent infections, slow-healing cuts, dark velvety skin patches—points back to this one upstream problem: cells stopped listening to insulin, sugar rose, and time did the damage.

What you might feel

- Peeing a lot (polyuria)
- Being very thirsty (polydipsia)
- Losing weight without trying
- Feeling tired and weak all the time
- Blurry vision
- Dark, velvety patches of skin (acanthosis nigricans)
- Cuts and sores that heal slowly
- Yeast infections and skin infections that keep coming back
- Tingling or numbness in hands or feet

- No symptoms at all (found on routine lab work)

Call the doctor right away if...

- Confusion, extreme thirst, or very dry mouth with blood sugar over 600 (sign of hyperosmolar hyperglycemic state, a life-threatening emergency)
- Chest pain, trouble breathing, or pain in the jaw or arm (possible heart attack — diabetes raises heart disease risk)
- Sudden vision loss or floaters and flashes (could be bleeding in the eye from damaged blood vessels)
- A wound or sore that won't heal, is red and hot, or smells bad (sign of a serious infection that can spread)
- Severe belly pain, nausea, and vomiting with high blood sugar (could be diabetic ketoacidosis (the acid crisis of missing insulin), rare in type 2 but dangerous)

What the care team will do

- Check blood sugar with fingerstick glucometer and review the last hemoglobin A1c (3-month average sugar) result
- Examine both feet completely including between toes, checking for cuts, blisters, redness, warmth, or foul smell
- Measure blood pressure and check if patient is on a statin and ACE inhibitor or ARB
- Ask about the last eye exam and schedule one if more than 12 months ago
- Weigh the patient and calculate BMI (body mass index) to assess obesity
- Review when patient last ate and hold metformin for 48 hours if getting IV contrast dye for CT scan or procedure
- Teach patient to check feet daily at home and report any cuts or color changes immediately
- Confirm patient has glucose tablets or gel at home and knows signs of low blood sugar

Medicines you may be given — and why

Metformin

Metformin tells the liver to make less sugar and helps muscle cells listen better to insulin's signal so they can pull sugar out of the blood. It does NOT make the pancreas pump out more insulin — instead, it fixes the insulin-resistance problem at the liver and muscles.

GLP-1 receptor agonists (examples: semaglutide, dulaglutide, liraglutide)

GLP-1 drugs copy a natural gut hormone called GLP-1 (glucagon-like peptide-1) that tells the pancreas to release insulin only when sugar is high, tells the stomach to slow down emptying, and tells the brain's appetite center to feel full. This lowers blood sugar, protects the heart, and often leads to weight loss.

SGLT2 inhibitors (examples: empagliflozin, dapagliflozin, canagliflozin)

SGLT2 inhibitors block a transporter protein in the kidney that normally grabs sugar back from the urine and returns it to the blood. When this transporter is blocked, extra sugar spills into the urine and leaves the body. This lowers blood sugar, protects the heart, slows kidney damage, and helps you lose a little weight because you are peeing out calories.

DPP-4 inhibitors (examples: sitagliptin, linagliptin, saxagliptin)

DPP-4 inhibitors block the enzyme DPP-4, which normally chews up GLP-1 (your body's natural insulin-boosting gut hormone) within minutes. By blocking DPP-4, your own GLP-1 lasts longer in the blood and gently tells the pancreas to make more insulin when sugar is high and tells the liver to make less sugar.

Sulfonylureas (examples: glipizide, glyburide, glimepiride)

Sulfonylureas force the pancreas beta cells to release insulin all the time, whether blood sugar is high or not. They close potassium channels on the beta cell surface, which flips the cell into release mode and insulin pours out. This lowers blood sugar but can push it too low if you skip a meal.

Thiazolidinediones / TZDs (pioglitazone)

TZDs turn on a master switch called PPAR-gamma (peroxisome proliferator-activated receptor gamma) inside fat and muscle cells that rewires how the cells handle fat and sugar. This makes the cells much more sensitive to insulin, so they pull more glucose out of the blood. TZDs also shift fat storage away from dangerous belly fat and liver fat to safer under-skin fat.

Insulin (examples: basal insulins like glargine, degludec, detemir; rapid-acting insulins like lispro, aspart, glulisine; premixed insulins)

Insulin is the same hormone your pancreas normally makes. When injected, it tells cells throughout the body to open their glucose gates and pull sugar out of the blood. Basal (long-acting) insulin covers background needs all day and night; rapid-acting (mealtime) insulin covers the sugar spike from food.

Statins (examples: atorvastatin, rosuvastatin, simvastatin, pravastatin)

Statins block the enzyme HMG-CoA reductase (3-hydroxy-3-methylglutaryl coenzyme A reductase) that the liver uses to make cholesterol. With less cholesterol being made, the liver pulls LDL (bad cholesterol) out of the blood to use as raw material. This lowers LDL, stabilizes artery plaques, and cuts the risk of heart attack and stroke — the leading cause of death in people with diabetes.

ACE inhibitors (examples: lisinopril, enalapril, ramipril) or ARBs (examples: losartan, irbesartan, telmisartan)

ACE inhibitors and ARBs both block angiotensin II (a hormone that squeezes blood vessels tight and pushes the kidneys to hold onto salt and water). ACE inhibitors stop angiotensin II from being made; ARBs stop it from working. Either way, blood vessels relax, blood pressure drops gently, and the kidneys are protected from diabetes damage — especially if you already have protein leaking into your urine.

Where this information comes from

- American Diabetes Association Standards of Care 2024
- CDC Diabetes Guidelines
- USPSTF Screening Recommendations
- ADA/EASD Consensus Report on Type 2 Diabetes Management

Last reviewed: 2026-07-07