

Hyperlipidemia

Too much fat in the blood rusts the pipes that feed your heart and brain.

THE ONE-LINE TRUTH

In hyperlipidemia, the bedside team that spots silent vascular risk early — checking lipids, blood pressure, and family history — outperforms the team that waits for a stroke or heart attack.

The chain — how it unfolds

1. Excess LDL particles seep through the endothelium (the thin inner lining of the artery) and lodge in the wall beneath. — why: High LDL concentration in the blood creates a steep gradient that drives cholesterol into the artery wall, especially at bends and branch points where flow is turbulent and the lining is already slightly leaky.
2. Trapped LDL oxidizes and triggers an inflammatory alarm that recruits macrophages (white blood cells that eat invaders). — why: Oxidized LDL looks like a foreign invader to the immune system, so the endothelium sends out adhesion molecules (like sticky flags) that grab passing macrophages and pull them into the artery wall.
3. Macrophages engulf oxidized LDL until they are so full they become foam cells, then die and spill their contents, forming a soft, fatty core. — why: The foam cells cannot break down oxidized cholesterol fast enough, so they burst, leaving a puddle of lipid and debris that grows larger as more cells arrive and die in the same spot.
4. Smooth muscle cells migrate from the artery's middle layer and build a fibrous cap over the fatty core, creating a hardened plaque that narrows the lumen. — why: The artery tries to contain the damage by walling it off, but the cap is fragile—chronic inflammation, high blood pressure, and enzymes from macrophages steadily weaken it.
5. As plaques grow, blood flow to downstream tissues drops, causing stable angina (chest pain with exertion) when the heart muscle does not get enough oxygen during effort. — why: A 70 percent blockage can supply enough blood at rest but cannot meet the demand when the heart pumps harder during exercise, so the muscle cries out with pain (a warning that it is starving for oxygen).

6. The fibrous cap ruptures, exposing the fatty core to blood; platelets instantly form a clot to seal the tear, but the clot blocks the artery completely. — why: Your blood sees the ruptured plaque as an injury and deploys the same clotting cascade that stops a cut on your skin—except inside an artery, that clot stops all flow, causing a heart attack (if it is in a coronary artery) or stroke (if it is in a brain artery).
7. Tissue downstream of the blocked artery dies within minutes to hours, leaving permanent damage—dead heart muscle (myocardial tissue death from blocked blood flow (infarction)) or dead brain tissue (ischemic stroke). — why: Without oxygen and glucose from blood, cells run out of energy (ATP), ion pumps fail, cells swell and burst, and the damage cannot be reversed once the tissue is dead.
8. Over years, repeated plaque ruptures and clots, chronic high blood pressure from stiff arteries, and the heart's struggle to pump against resistance lead to heart failure, kidney disease, peripheral artery disease, and cognitive decline. — why: Every organ suffers when its arteries are narrowed—kidneys cannot filter waste, legs do not get enough blood to walk without pain, and the brain slowly loses neurons and connections, raising dementia risk.

Spot it — what you see, and why

- Yellow-orange bumps on the skin (xanthomas): When cholesterol in the blood climbs very high, it spills into the skin and clumps into soft yellow nodules, often over tendons (elbows, knees, hands) or around the eyes (xanthelasmas)—a visible sign the body cannot clear the extra fat.
- Cloudy white ring around the edge of the iris (corneal arcus): Cholesterol deposits settle in the outer rim of the cornea (the clear window at the front of the eye), forming a pale arc or full ring; in people under 45 this often signals familial hyperlipidemia.
- Chest pain or pressure with activity (angina): High LDL cholesterol builds waxy plaque inside the coronary arteries (the heart's fuel lines), narrowing the channel so less oxygen-rich blood reaches the heart muscle during exertion, causing a squeezing or burning pain.
- Sudden weakness, slurred speech, or vision loss (stroke symptoms): Cholesterol plaque in the carotid or brain arteries can rupture or clot off, blocking blood flow to part of the brain and killing nerve cells in minutes.
- Cramping leg pain when walking (claudication): Plaque narrows the arteries that feed the leg muscles; during walking the muscles need more oxygen but cannot get it, so they cramp and ache until you rest and demand drops.
- Severe belly pain and nausea when triglycerides are extremely high: Triglyceride levels above 500 mg/dL thicken the blood so much that tiny fat globules can clog pancreatic blood vessels, triggering pancreatitis (inflammation of the pancreas that digests itself).

Red flags — act now

- Chest pain, jaw pain, or shortness of breath—may signal a heart attack from a ruptured cholesterol plaque.
- Sudden face droop, arm weakness, or slurred speech—may signal a stroke from a blocked brain artery.
- Severe belly pain with vomiting—triglycerides above 500 mg/dL can cause pancreatitis, a life-threatening emergency.
- Yellow skin bumps (xanthomas) or cloudy eye rings (arcus) before age 45—often means familial hyperlipidemia with very high inherited cholesterol that needs urgent treatment to prevent early heart disease.
- New leg pain that does not go away with rest—may signal a clot or severe artery blockage cutting off blood to the limb.

Treat it — in this order

1. High-intensity statin (atorvastatin 40–80 mg or rosuvastatin 20–40 mg daily) → Blocks HMG-CoA reductase, the liver enzyme that makes cholesterol. This cuts LDL by 50% or more and shrinks the amount of sticky fat floating in the blood that clogs artery walls. Over months to years, it slows new plaque growth and stabilizes old plaque so it is less likely to rupture and cause a clot.
2. Moderate-intensity statin (atorvastatin 10–20 mg, rosuvastatin 5–10 mg, simvastatin 20–40 mg, or pravastatin 40–80 mg daily) → Lowers LDL by 30–49%, which is enough for people with moderate ASCVD risk (7.5–19.9% 10-year risk) or those who cannot tolerate high-intensity dosing. It still reduces the cholesterol pool feeding artery plaque.
3. Ezetimibe 10 mg daily (added to statin if LDL remains ≥ 70 mg/dL on statin alone) → Blocks the Niemann-Pick C1-Like 1 protein in the small intestine, which normally pulls cholesterol from food and bile back into the bloodstream. This cuts another 15–20% off LDL and gives you a second way to lower cholesterol when statin alone is not enough.
4. PCSK9 inhibitor (evolocumab 140 mg SC every 2 weeks or alirocumab 75–150 mg SC every 2 weeks) → Blocks PCSK9, a protein that normally tags LDL receptors for destruction. With PCSK9 blocked, the liver keeps more receptors on its surface and pulls LDL out of the blood faster—dropping LDL by another 50–60% on top of statin. This is reserved for familial hyperlipidemia (LDL ≥ 190 despite statin) or people who have already had a heart attack and need the lowest possible LDL.

5. Lifestyle modification: diet with < 7% calories from saturated fat, 25–35 g fiber daily, 150 minutes moderate aerobic exercise per week, weight loss if BMI $\geq$ 25 → Lowers LDL by 5–10%, raises HDL slightly, and improves insulin sensitivity and blood pressure. Exercise burns triglycerides and helps the body clear fat from the blood. Fiber binds bile acids in the gut, forcing the liver to use cholesterol to make new bile—this pulls cholesterol out of circulation.

Your top priorities at the bedside

1. Assess for acute coronary syndrome by asking about chest pain character, checking vital signs for a fast heart rate (tachycardia) or low blood pressure (hypotension), and obtaining a 12-lead ECG within 10 minutes if any cardiac symptoms present. — why: Hyperlipidemia causes atherosclerosis (plaque buildup in arteries) that can rupture and cause heart attacks. Early ECG detection of ST-segment elevation or depression guides immediate reperfusion therapy, and every minute of delay increases heart muscle death.
2. Check blood pressure in both arms and document accurately, because many hyperlipidemia patients need ACE inhibitors or ARBs that require baseline BP before starting. — why: ACE inhibitors and ARBs lower cardiovascular risk independent of cholesterol lowering by reducing afterload (pressure the heart pumps against) and preventing vessel remodeling. Low blood pressure (Hypotension) below 90 systolic is a contraindication that would cause dangerous drops requiring dose adjustment or alternative therapy.
3. Review the lipid panel results—total cholesterol, LDL (low-density lipoprotein), HDL (high-density lipoprotein), and triglycerides—and identify which number is driving the diagnosis. — why: Treatment differs based on the lipid type: isolated high LDL needs statins first; high triglycerides above 500 mg/dL risk pancreatitis (inflamed pancreas) and need fibrates or omega-3 fatty acids; low HDL below 40 in men needs exercise emphasis. Knowing the pattern prevents wrong medication choices.