

Dyslipidemia

A plain-English guide for you and your family

WHAT IS HAPPENING

Your liver makes cholesterol (a waxy fat) and packages it into tiny balls called particles. LDL particles carry cholesterol out to your body, and HDL particles bring leftover cholesterol back to the liver to be removed. Normally your liver pulls LDL out of your blood using special hooks (receptors) on its surface, keeping the amount in your blood low and safe. But when genes are passed down that make fewer hooks, or when you eat too much saturated fat (butter, red meat, fried food), smoke, do not exercise, or have diabetes, your liver cannot clear LDL fast enough — so LDL cholesterol builds up in your blood. High LDL particles slip through the lining of your artery walls and get stuck there, forming fatty streaks that harden into plaque over years. You feel completely fine during this — no pain, no warning — because plaque grows silently. Then one day a plaque cracks open, a blood clot forms on top in seconds, and blood flow to your heart or brain stops, causing a heart attack or stroke. The good news: we can measure your cholesterol with a simple blood test, and medicines called statins can lower LDL by 30 to 50 percent, shrinking plaques and preventing most heart attacks and strokes before they happen. Dyslipidemia (high LDL or low HDL) is common, treatable, and one of the biggest things you can control to protect your heart and brain for the long run.

What you might feel

- Usually no symptoms at all
- Xanthomas (small yellow bumps on skin, especially over tendons)
- Xanthelasma (yellowish patches on eyelids)
- Corneal arcus (white or gray ring around the colored part of the eye)
- Pancreatitis (sudden, severe belly pain that may reach through to the back)
- Chest pain or pressure (angina)
- Early heart attack or stroke in family members

Call the doctor right away if...

- Sudden severe belly pain with nausea and vomiting (may be pancreatitis from very high triglycerides)
- Chest pain, pressure, or tightness, especially with activity or stress (sign that plaque is blocking heart arteries)
- Sudden weakness, numbness, slurred speech, or vision loss (possible stroke from a clot breaking off plaque)
- Multiple close family members with heart attack or stroke before age 55 in men or 65 in women (suggests inherited severe high cholesterol)
- Triglycerides above 1,000 mg/dL (high risk of pancreatitis)

What the care team will do

- Assess for signs of acute coronary syndrome: ask about chest pain quality, duration, radiation to jaw or arm, associated shortness of breath or sweating, and obtain 12-lead ECG if any cardiac symptoms present
- Check blood pressure, heart rate, and peripheral pulses in both arms and both legs; listen to carotid arteries for bruits (whooshing sounds)
- Review most recent lipid panel results: total cholesterol, LDL, HDL, and triglycerides; calculate 10-year ASCVD risk score if patient has no prior cardiovascular events
- Teach Mediterranean diet specifics: replace butter with olive oil, eat fish twice weekly, choose whole grains over white bread and pasta, fill half the plate with vegetables, eat nuts and beans for protein, limit red meat to once weekly
- Assess current physical activity level and create specific exercise prescription: 30 minutes of moderate activity like brisk walking at least 5 days per week, aiming for 150 minutes total weekly
- Educate about statin timing and side effects: take atorvastatin or rosuvastatin at the same time daily (evening preferred), report new muscle pain or weakness immediately, continue medication even when feeling well because cholesterol causes silent damage
- Schedule follow-up lipid panel 4 to 12 weeks after starting or adjusting statin therapy, and check liver enzymes (ALT and AST) and creatine kinase (CK) at baseline and if symptoms develop
- Screen for and address barriers to medication adherence: cost concerns, fear of side effects from internet stories, forgetfulness, or belief that supplements can replace statins

Medicines you may be given — and why

Atorvastatin (Lipitor), Rosuvastatin (Crestor), Simvastatin (Zocor), Pravastatin (Pravachol)

Your liver makes cholesterol using a factory enzyme called HMG-CoA reductase. A statin blocks that enzyme, so the liver makes less cholesterol inside the cell. When the cell runs low, it puts more LDL receptors (doorways) on its surface to grab LDL cholesterol from the blood. This pulls LDL out of your blood and into the liver, lowering the amount of bad cholesterol floating around and available to clog arteries.

Ezetimibe (Zetia)

Cholesterol enters your gut from food and from bile (liver fluid). Ezetimibe blocks a transporter protein (NPC1L1) in the gut-lining cells, so less cholesterol gets absorbed into your bloodstream. The cholesterol stays in the gut and leaves in stool. Your liver responds by pulling more LDL out of the blood to make up for what it did not absorb, lowering blood LDL even more.

Evolocumab (Repatha), Alirocumab (Praluent)

PCSK9 is a protein your liver releases that tags LDL receptors for destruction. Fewer receptors mean less LDL is pulled from the blood. This drug is an antibody (a protein shield) that binds and blocks PCSK9, so LDL receptors stay on the liver cell surface longer. More receptors pull more LDL out of the blood, dropping LDL cholesterol by 50 to 60 percent.

Inclisiran (Leqvio)

Inclisiran is a tiny piece of RNA (genetic instruction) that enters liver cells and silences the gene that makes PCSK9. With less PCSK9 produced, LDL receptors stay on the cell surface longer, pulling more LDL out of the blood. The effect lasts months because the RNA is stable, so you only need an injection twice a year after the first two doses.

Bempedoic acid (Nexletol)

Bempedoic acid blocks an enzyme (ATP citrate lyase) one step earlier in the cholesterol-making pathway than statins do — but only in the liver, not in muscles. This means the liver makes less cholesterol, LDL receptors go up, and blood LDL drops — without the muscle side effects many people get from statins. It is an oral pill taken once daily.

Fenofibrate (Tricor, Lofibra), Gemfibrozil (Lopid)

Fibrates turn on a gene switch (PPAR-alpha) in the liver that tells the cell to burn more fatty acids for energy and to make less triglyceride. They also boost production of lipoprotein lipase,

the enzyme that breaks down triglyceride-rich particles in the blood. The result is lower triglycerides (often by 30 to 50 percent) and a modest rise in HDL (good cholesterol).

Icosapent ethyl (Vascepa)

Icosapent ethyl is pure EPA (eicosapentaenoic acid), an omega-3 fat from fish oil. It lowers triglycerides by reducing the liver's production of VLDL particles and by speeding the breakdown of triglycerides in the blood. It also reduces inflammation in artery walls and stabilizes plaques, which lowers the risk of heart attack and stroke in people who already have heart disease or diabetes and high triglycerides despite statin therapy.

Niacin (nicotinic acid; Niaspan)

Niacin reduces the liver's release of VLDL (which carries triglycerides) and slows the breakdown of HDL, so HDL cholesterol rises (often by 15 to 35 percent) and triglycerides fall. It also modestly lowers LDL. However, large trials showed no additional heart benefit when added to statin therapy, so it is now used rarely — mainly when HDL is very low and other options have not worked.

Lomitapide (Juxtapid)

Lomitapide blocks the MTP enzyme inside liver and gut cells. MTP is needed to load triglycerides and cholesterol into VLDL and chylomicron particles. Without it, the liver and gut cannot release these particles into the blood, so LDL and triglycerides drop dramatically. It is used only for homozygous familial hypercholesterolemia (the most severe inherited form) when other drugs are not enough.

Evinacumab (Evkeeza)

Evinacumab blocks ANGPTL3, a protein that normally stops lipoprotein lipase (the enzyme that breaks down triglycerides) from working. By blocking ANGPTL3, the drug lets lipoprotein lipase stay active longer, so triglyceride-rich particles and LDL are cleared faster from the blood. It is approved only for homozygous familial hypercholesterolemia, often in people who do not respond well to PCSK9 inhibitors.

Where this information comes from

- AHA/ACC Cholesterol Guideline 2018
- ESC/EAS Dyslipidaemia Guidelines 2019
- National Lipid Association
- Cochrane Reviews

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