

Hyperlipidemia

A plain-English guide for you and your family

WHAT IS HAPPENING

Your blood carries many things—oxygen, sugar, and also fats called lipids. The two most important are cholesterol (a waxy builder used to make cell walls and hormones) and triglycerides (stored energy). When there is too much of either, the extra fat starts to stick to the inside of your arteries like grease on a pipe. Over years, this builds a hard, rough patch called plaque. Plaque narrows the pipe, making it harder for blood to push through. Worse, the plaque can crack open, and your blood forms a clot to seal the crack—but that clot can plug the artery completely, cutting off blood to your heart (a heart attack) or brain (a stroke). The dangerous part is that you feel nothing for years while the damage is building. Most people only find out they have high cholesterol from a simple blood test. The good news is that catching it early and lowering the fat in your blood—with medicine (usually a statin), exercise, and eating less saturated fat—can stop the plaques from growing, keep clots from forming, and protect your heart and brain for decades.

What you might feel

- Yellow-orange bumps on the skin (xanthomas)
- Cloudy white ring around the edge of the iris (corneal arcus)
- Chest pain or pressure with activity (angina)
- Sudden weakness, slurred speech, or vision loss (stroke symptoms)
- Cramping leg pain when walking (claudication)
- Severe belly pain and nausea when triglycerides are extremely high
- No symptoms at all (most common)

Call the doctor right away if...

- 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.

What the care team will do

- 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.
- Check blood pressure in both arms and document accurately, because many hyperlipidemia patients need ACE inhibitors or ARBs that require baseline BP before starting.
- 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.
- Teach the patient to take statins in the evening because cholesterol synthesis peaks at night, and explain the need for liver enzyme monitoring at baseline and 8 to 12 weeks.
- Assess for xanthomas (yellowish cholesterol deposits under skin around eyes or on tendons) and arcus corneae (white ring around the iris), which signal severely elevated lipids needing urgent specialist referral.
- Document current statin use and ask about muscle symptoms every visit, using a pain scale for any reported muscle aches (myalgias) (muscle aches) to catch early rhabdomyolysis before CK rises.
- Calculate the 10-year ASCVD risk score using age, sex, race, cholesterol numbers, blood pressure, diabetes status, and smoking history to determine statin intensity needed.
- Provide written diet education focusing on replacing saturated fats with unsaturated fats, adding soluble fiber from oats and beans, and limiting trans fats from processed foods.

Medicines you may be given — and why

Atorvastatin (Lipitor)

Atorvastatin blocks an enzyme in the liver called HMG-CoA reductase (the enzyme that makes cholesterol). When this enzyme is blocked, the liver makes less cholesterol. The liver then pulls more LDL cholesterol (the 'bad' cholesterol) out of the blood to use for other jobs, which lowers the total amount of LDL in your bloodstream.

Rosuvastatin (Crestor)

Rosuvastatin blocks the same cholesterol-making enzyme (HMG-CoA reductase) in the liver as atorvastatin, but it is more potent milligram-for-milligram. It forces the liver to pull even more LDL cholesterol out of the blood, making it a strong choice for people with very high LDL or high cardiovascular risk.

Ezetimibe (Zetia)

Ezetimibe works in the small intestine, not the liver. It blocks a transporter protein called NPC1L1 that normally pulls cholesterol from food (and bile) into the intestinal cells. When this transporter is blocked, less cholesterol gets absorbed, so less enters the bloodstream. The liver then has to pull more LDL out of the blood to make up the difference.

Fenofibrate (Tricor, Fenoglide)

Fenofibrate turns on a receptor in the liver and muscles called PPAR-alpha (peroxisome proliferator-activated receptor alpha). This receptor controls genes that break down fats. When PPAR-alpha is activated, the liver speeds up the breakdown of triglycerides (a type of fat in the blood) and makes more HDL (the 'good' cholesterol). Fenofibrate is especially helpful for people with very high triglycerides.

Icosapent ethyl (Vascepa)

Icosapent ethyl is a purified form of EPA (eicosapentaenoic acid), an omega-3 fatty acid from fish oil. It lowers triglycerides by reducing the liver's production of VLDL (very-low-density lipoprotein), the particle that carries triglycerides in the blood. It also has anti-inflammatory effects on blood vessel walls, which may lower the risk of heart attack and stroke beyond just lowering triglycerides.

Evolocumab (Repatha) or Alirocumab (Praluent)

These drugs are injected antibodies that block a protein called PCSK9. Normally, PCSK9 tags LDL receptors on liver cells for destruction. When PCSK9 is blocked, more LDL receptors survive on the liver's surface, so the liver can pull much more LDL cholesterol out of the blood. This results in very large drops in LDL — often 50-60% lower.

Bempedoic acid (Nexletol)

Bempedoic acid blocks an enzyme in the liver called ATP citrate lyase, which sits earlier in the cholesterol-making pathway than the enzyme statins block. By shutting down this enzyme, the liver makes less cholesterol. Because bempedoic acid only works in the liver (not in muscles), it causes much less muscle pain than statins, making it a good option for people who cannot tolerate statins.

Where this information comes from

- AHA/ACC 2018 Guideline on the Management of Blood Cholesterol
- ATP IV (Adult Treatment Panel IV) National Lipid Association
- USPSTF 2022 Lipid Screening Recommendation
- Cochrane systematic reviews on statin therapy

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