

# Dyslipidemia

Too much LDL cholesterol in the blood lets fat stick to artery walls and block blood flow to the heart and brain.

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## THE STORY IN ONE SENTENCE

Too much LDL cholesterol in the blood lets fat stick to artery walls and block blood flow to the heart and brain.

“High cholesterol builds fat plaques in arteries that can suddenly block blood flow and cause heart attacks or strokes. Statins lower cholesterol and stop this before it happens.”

## Normal anatomy — the body's job

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Your liver makes cholesterol every day and wraps it into tiny particles so it can travel through your blood. LDL particles deliver cholesterol to cells that need it, and HDL particles pick up extra cholesterol and bring it back to the liver to be thrown out. Your liver has special hooks (LDL receptors) on its surface that grab LDL particles and pull them out of the blood, keeping cholesterol levels low and safe.

## BODY'S JOB

Cholesterol is not the enemy — your body needs it to build cell walls, make hormones (like testosterone and estrogen), produce vitamin D, and help digestion. The liver balances how much it makes and how much it removes so your blood stays clean and your arteries stay smooth.

1. Your liver makes cholesterol and packs it into LDL particles (think of them as delivery trucks).
2. LDL particles float through your bloodstream, dropping off cholesterol to cells that need it.
3. HDL particles (the garbage trucks) pick up leftover cholesterol from tissues and carry it back to the liver.
4. The liver pulls LDL out of the blood using LDL receptors (like hooks) and breaks the particles down.
5. This keeps LDL cholesterol in your blood low — usually under 100 mg/dL — so nothing sticks to your artery walls.

## What breaks

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When your liver does not have enough hooks to pull LDL out, or when it makes too much cholesterol, or when the LDL particles themselves change shape and become stickier, LDL builds up in your blood and slips into artery walls.

### CLINICAL TERM

Primary dyslipidemia is caused by gene mutations (like familial hypercholesterolemia, where LDL receptors are missing or broken). Secondary dyslipidemia is caused by diet high in saturated fat and trans fat, obesity, sedentary lifestyle, smoking, type 2 diabetes (insulin resistance raises triglycerides and lowers HDL), hypothyroidism (slows LDL clearance), chronic kidney disease, or nephrotic syndrome (liver overproduces lipoproteins). The result is elevated LDL-C ( $\geq 100$  mg/dL), low HDL-C ( $< 40$  mg/dL in men,  $< 50$  mg/dL in women), or high triglycerides ( $\geq 150$  mg/dL).

## How the body compensates

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Your body does not compensate for high cholesterol in a helpful way — there is no alarm system, no pain, and no symptoms for years. Plaque grows silently, and by the time you feel chest pain or have a stroke, the damage is already severe. The only way to catch it early is to measure cholesterol with a blood test.

1. Your blood pressure and heart rate stay completely normal while plaque is building, so you feel fine and have no reason to suspect anything is wrong.
2. Your arteries try to stretch outward (remodeling) to keep the inside opening wide even as plaque grows in the wall — this hides the blockage on early imaging.
3. Your heart may grow new tiny blood vessels (collaterals) around a slowly narrowing artery to keep oxygen flowing — this delays symptoms but does not stop the plaque.
4. The danger is that a stable plaque can crack open suddenly (rupture), triggering a blood clot in seconds that blocks the artery completely — causing a heart attack or stroke with no warning.
5. If you have familial hypercholesterolemia (inherited high cholesterol), yellow bumps (xanthomas) may appear on your knuckles, elbows, or Achilles tendons, and a white ring (corneal arcus) may form around your iris — these are visible clues that LDL is dangerously high.
6. A lipid panel (blood test) is the only reliable way to see the problem early, before the first heart attack or stroke happens.

7. Once you start treatment, your body does not give you feedback that the medicine is working — you still feel normal — so sticking with daily statin therapy requires trust in the numbers, not symptoms.

## Domino effect

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1. LDL cholesterol builds up in the blood because the liver cannot clear it fast enough. — Genes (like familial hypercholesterolemia), saturated fat in the diet, smoking, lack of exercise, diabetes, or hypothyroidism slow down or overwhelm the liver's ability to pull LDL particles out of the bloodstream.
2. LDL particles slip through the inner lining (endothelium) of artery walls and get trapped underneath. — High LDL levels and inflammation (from smoking, high blood sugar, or high blood pressure) damage the endothelium, making it leaky so LDL particles can squeeze through and get stuck in the artery wall.
3. White blood cells (macrophages) try to clean up the trapped LDL, but they turn into foam cells and die, forming a fatty streak. — Macrophages eat the LDL particles to protect the artery, but when there is too much LDL, the macrophages fill up with fat (becoming foam cells), burst, and leave behind a pool of cholesterol and dead cells — the start of plaque.
4. The fatty streak hardens into plaque as smooth muscle cells and scar tissue grow over it, narrowing the artery. — Your body tries to wall off the fatty mess by growing a fibrous cap (scar tissue) over it, but this makes the artery stiff and narrow, reducing blood flow — especially during exercise when your heart or brain needs more oxygen.
5. The plaque can crack open suddenly (rupture), and a blood clot forms on top in seconds, blocking the artery completely. — The fibrous cap can crack if blood pressure spikes, inflammation weakens it, or stress hormones surge — when it ruptures, blood touching the fatty core triggers clotting, and the clot blocks blood flow instantly, killing heart or brain tissue (heart attack or stroke).
6. Without enough oxygen, heart muscle or brain cells die within minutes, causing permanent damage or death. — Heart and brain cells need a constant supply of oxygen from blood — when flow stops, cells run out of energy (ATP), membranes break down, and the tissue dies (infarction), leading to chest pain, irregular heartbeat, paralysis, speech loss, or sudden death.
7. Statins lower LDL cholesterol by blocking the liver's cholesterol-making enzyme, shrinking plaques and preventing most heart attacks and strokes. — Statins (like atorvastatin, rosuvastatin) stop the enzyme HMG-CoA reductase, so the liver makes less cholesterol and pulls more LDL out of the blood using its receptors — this lowers LDL by 30–50%, stabilizes plaques, reduces inflammation, and cuts the risk of heart attack and stroke by about 25–35% over five years.

8. If statins are not enough, or if familial hypercholesterolemia is severe, adding ezetimibe or PCSK9 inhibitors can lower LDL even further and save lives. — Ezetimibe blocks cholesterol absorption in the gut (lowers LDL another 15–20%), and PCSK9 inhibitors (like evolocumab, alirocumab) are injections that help the liver pull LDL out of the blood faster (lowers LDL another 50–60%) — these are critical for people with genetic high cholesterol or those who cannot take statins.

## Symptoms · what the patient feels, and why

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- Usually no symptoms at all: High cholesterol and triglycerides do not hurt or feel different on their own; they quietly build up inside artery walls without warning, so most people feel completely normal until a serious problem (heart attack or stroke) happens.
- Xanthomas (small yellow bumps on skin, especially over tendons): When cholesterol in the blood stays extremely high for a long time, some of it leaks out and collects under the skin or in tendons, forming soft yellow lumps (like wax deposits); this is most common in familial (inherited) forms of high cholesterol.
- Xanthelasma (yellowish patches on eyelids): Extra cholesterol in the blood can also settle in the thin skin of the eyelids, making flat or slightly raised yellow patches; these are harmless by themselves but signal that cholesterol levels may be very high.
- Corneal arcus (white or gray ring around the colored part of the eye): Cholesterol can deposit in the edge of the cornea (the clear window at the front of the eye), creating a faint ring; in younger adults this often means inherited high cholesterol, though in older adults it can be normal aging.
- Pancreatitis (sudden, severe belly pain that may reach through to the back): When triglycerides climb extremely high (usually above 1,000 mg/dL), they can inflame the pancreas (the organ that makes digestive juices and insulin), causing intense pain, nausea, and vomiting; this is a medical emergency.
- Chest pain or pressure (angina): Over many years, high cholesterol causes waxy plaque to narrow the arteries that feed the heart muscle; when the heart does not get enough blood during exercise or stress, it sends pain signals in the chest (like a cramp from not enough oxygen).
- Early heart attack or stroke in family members: Some forms of dyslipidemia run in families (inherited gene changes make the liver remove cholesterol too slowly or make too much); when many relatives have heart attacks or strokes before age 55 (men) or 65 (women), it is a strong clue that cholesterol is dangerously high from birth.

## Focused exam

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- Lipid panel: LDL cholesterol high, HDL cholesterol low, triglycerides high or normal: Dyslipidemia is defined by these numbers. LDL is the bad cholesterol that sticks in blood vessels. HDL is the good cholesterol that cleans it up. You cannot diagnose dyslipidemia without checking.
- Xanthomas (bumpy yellow patches on skin) or xanthelasma (yellow patches near eyes): These are cholesterol deposits in the skin. They mean very high cholesterol, especially in familial hypercholesterolemia. They tell you the patient has had high cholesterol for a long time.
- Corneal arcus (white ring around the colored part of the eye): Cholesterol deposits in the eye. Like xanthomas, this means the body has been storing cholesterol where it should not be. It suggests genetic dyslipidemia.
- No chest pain or leg pain, but patient is 50 years old with high cholesterol: Dyslipidemia is silent — plaque builds up without symptoms. By the time someone feels chest pain, the vessel may already be almost blocked. This is why we screen and treat before symptoms happen.
- Blood pressure high, weight high, or smoker: These are other breaks that make dyslipidemia worse. High cholesterol alone is dangerous. High cholesterol plus high blood pressure plus smoking is much more dangerous. The risk adds up.
- ASCVD risk calculator score above 7.5% over 10 years: This calculator adds up all the breaks in the patient — age, cholesterol, blood pressure, smoking, diabetes. It tells you how likely a heart attack or stroke is. High score means treat harder.
- History of early heart attack or stroke in family (before age 55 in men, 65 in women): Familial hypercholesterolemia is a gene break that makes cholesterol very high from birth. If the family had early heart disease, this patient's genes may be broken too. Treat very hard.

## Diagnostics · every test answers a question

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- Lipid panel can be fasting or non → Numbers are clues, not final answers. They point back to how the body works.
- Use CAC for borderline → Numbers are clues, not final answers. They point back to how the body works.
- The question is always → Numbers are clues, not final answers. They point back to how the body works.
- Lipid panel can be fasting or non → Imaging only helps when it answers one specific question that changes how you treat the patient.

- Use CAC for borderline → Imaging only helps when it answers one specific question that changes how you treat the patient.

## Treatment logic

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- High-intensity statin (atorvastatin 40-80mg or rosuvastatin 20-40mg daily) → Blocks the enzyme that makes cholesterol in the liver, so less LDL particles get made and fewer stick to blood vessel walls. This slows plaque buildup before it causes a heart attack.
- Lifestyle changes: Mediterranean diet, 150 minutes exercise per week, weight loss, smoking cessation → Eating better and moving your body tell your liver to make less cholesterol and burn fat instead of storing it. This fixes the broken upstream steps — the actual causes, not just the number.
- Ezetimibe (10mg daily) if LDL stays above goal after statin → Blocks cholesterol from entering the bloodstream through the intestines, so fewer fat particles are available to build plaque in vessels.
- PCSK9 inhibitor (evolocumab or alirocumab) for familial hypercholesterolemia or ASCVD with LDL >70 despite statin + ezetimibe → Stops the liver from destroying LDL receptors, so more LDL gets pulled out of the blood and does not accumulate in vessel walls.
- Icosapent ethyl (2g twice daily) or fibrate (fenofibrate) if triglycerides >200 despite statin → Lowers triglyceride particles that also trigger inflammation and plaque growth in blood vessels when LDL is already high.
- Calculate 10-year ASCVD risk and check lipid panel every 4-12 weeks after starting or changing therapy → Tells you if the medicine is actually stopping plaque buildup by showing the numbers moving toward safe targets (LDL <70 for ASCVD, <100 for primary prevention).

## Medications

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### **Atorvastatin (Lipitor), Rosuvastatin (Crestor), Simvastatin (Zocor), Pravastatin (Pravachol) · Statin (HMG-CoA reductase inhibitor)**

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.

1. Block HMG-CoA reductase, the enzyme that builds cholesterol inside the liver cell
2. Liver cholesterol drops, so the cell senses it needs more
3. Cell builds more LDL receptors and puts them on its surface

4. These receptors grab LDL particles from the blood and bring them inside
5. Blood LDL level falls; less cholesterol is available to stick in artery walls

**SAFETY WATCH**

Muscle aches or weakness (myalgia) — common and usually mild; very rarely, severe muscle breakdown (rhabdomyolysis) can happen if muscle pain is ignored · Liver enzyme elevation — check blood tests before starting and as needed; serious liver injury is rare · New-onset diabetes — statins can nudge blood sugar up slightly in people already at risk; the heart benefit still outweighs this risk · Memory or confusion — reported but not proven in large trials; tell your doctor if thinking feels off · Avoid grapefruit juice with atorvastatin, simvastatin, or lovastatin — it blocks the enzyme that clears the drug, raising blood levels and muscle-injury risk

**Ezetimibe (Zetia) · Cholesterol absorption inhibitor**

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.

1. Block the NPC1L1 transporter on gut cells that normally shuttle cholesterol across the intestine wall
2. Less cholesterol enters the bloodstream from the gut
3. Liver senses lower cholesterol delivery
4. Liver increases LDL receptor activity to pull more LDL from blood
5. Blood LDL drops by an additional 15 to 20 percent when added to a statin

**SAFETY WATCH**

Generally very well tolerated with few side effects · Diarrhea or joint pain — uncommon · Liver enzyme elevation when combined with a statin — check blood tests as you would for statin alone · Allergic reactions — rare rash or swelling; stop the drug and call your doctor

**Evolocumab (Repatha), Alirocumab (Praluent) · PCSK9 inhibitor (monoclonal antibody)**

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.

1. Inject the antibody under the skin every two or four weeks
2. Antibody binds to PCSK9 protein in the blood
3. PCSK9 can no longer tag LDL receptors for breakdown
4. LDL receptors stay on liver cells longer
5. More receptors pull more LDL from the blood into the liver
6. Blood LDL drops dramatically

#### **SAFETY WATCH**

Injection-site reactions — redness, swelling, or itch where the needle goes in · Flu-like symptoms — muscle aches, tiredness — usually mild · Allergic reactions — very rare but serious; watch for rash, trouble breathing, or swelling · Cost and insurance coverage — these are expensive biologic drugs; prior authorization is usually required

### **Inclisiran (Leqvio) · Small interfering RNA (siRNA) targeting PCSK9**

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.

1. Inject inclisiran under the skin; it travels to the liver
2. RNA enters liver cells and binds to PCSK9 messenger RNA
3. PCSK9 mRNA is cut and destroyed before it makes protein
4. Liver produces less PCSK9
5. More LDL receptors survive on the cell surface
6. Blood LDL drops by 50 percent; effect lasts six months

#### **SAFETY WATCH**

Injection-site reactions — redness, pain, or swelling · Generally well tolerated; long-term safety data are still growing · Allergic reactions — very rare · Cost and access — newer drug, often requires prior authorization

### **Bempedoic acid (Nexletol) · ATP citrate lyase inhibitor**

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.

1. Block ATP citrate lyase in the liver
2. Liver produces less acetyl-CoA, the starting material for cholesterol
3. Cholesterol synthesis drops inside liver cells
4. Liver increases LDL receptor expression to pull LDL from blood
5. Blood LDL falls by 15 to 25 percent when used alone, more when combined with ezetimibe or a statin

#### SAFETY WATCH

Tendon rupture — rare but reported, especially Achilles tendon; avoid if you have tendon problems · Gout flares — can raise uric acid; use cautiously if you have gout · Elevated liver enzymes and creatine kinase — monitor blood tests · Generally well tolerated with fewer muscle symptoms than statins

### **Fenofibrate (Tricor, Lofibra), Gemfibrozil (Lopid) · Fibrate (PPAR-alpha agonist)**

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).

1. Activate PPAR-alpha receptors in liver and fat cells
2. Cells increase fatty-acid oxidation (burning fat for fuel)
3. Liver makes less VLDL (the triglyceride-rich particle)
4. Lipoprotein lipase activity goes up, breaking down circulating triglycerides faster
5. Blood triglycerides drop; HDL rises modestly

#### SAFETY WATCH

Muscle pain and rhabdomyolysis — risk is higher when combined with a statin, especially gemfibrozil; fenofibrate is safer to combine · Gallstones — fibrates increase cholesterol in bile · Kidney function — can raise creatinine; monitor blood tests and adjust dose in kidney disease · Liver enzyme elevation — check blood tests before and during treatment · Do NOT use gemfibrozil with a statin — serious muscle-injury risk; fenofibrate is preferred if both are needed

**Icosapent ethyl (Vascepa) · Purified omega-3 fatty acid (EPA only)**

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.

1. Take 2 grams twice daily with food
2. Liver makes less VLDL (the particle that carries triglycerides)
3. Blood triglycerides drop by 20 to 30 percent
4. EPA incorporates into cell membranes and reduces inflammatory signals
5. Artery plaques become less likely to rupture
6. Risk of heart attack and stroke falls in high-risk patients

**SAFETY WATCH**

Atrial fibrillation (an irregular, quivering heartbeat) — small increase in irregular heartbeat risk in some trials; tell your doctor if you feel palpitations · Bleeding — may increase bleeding risk slightly, especially if you take aspirin or a blood thinner; report unusual bruising or bleeding · Fishy aftertaste or burping — take with food to reduce this · Allergic reaction to fish — do not use if you are allergic to fish or shellfish

**Niacin (nicotinic acid; Niaspan) · B vitamin (nicotinic acid) at prescription dose**

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.

1. Block the liver's release of VLDL particles
2. VLDL carries triglycerides, so blood triglycerides drop
3. Slow the kidney's removal of HDL from the blood
4. HDL cholesterol rises
5. LDL falls modestly as a secondary effect

**SAFETY WATCH**

Flushing — hot, red, itchy skin, especially on the face and chest; take aspirin 30 minutes before the dose or use extended-release forms to reduce this · Liver toxicity — can raise liver enzymes or cause serious liver injury, especially at high doses; monitor blood tests · High blood sugar — can worsen diabetes control · Gout — raises uric acid; can trigger gout attacks · Stomach upset, nausea, or ulcers · Use has declined sharply; most guidelines no longer recommend it

**Lomitapide (Juxtapid) · Microsomal triglyceride transfer protein (MTP) inhibitor**

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.

1. Block MTP enzyme in the endoplasmic reticulum (cell's factory)
2. Liver cannot package triglycerides and cholesterol into VLDL
3. Gut cannot package dietary fat into chylomicrons
4. Less VLDL and chylomicrons enter the blood
5. LDL drops (because LDL comes from VLDL breakdown)
6. Blood LDL can fall by 40 to 50 percent

**SAFETY WATCH**

Liver fat accumulation (steatosis) and liver injury — black-box warning; liver enzymes and fat must be monitored with MRI or biopsy; only available through a restricted program (REMS) · Diarrhea and nausea — very common; take with water two hours after dinner · Fat-soluble vitamin deficiency (A, D, E, K) — must take daily supplements · Strict low-fat diet required — no more than 20 percent of calories from fat to reduce side effects · Not for use in pregnancy — can harm the fetus · Many drug interactions — check with pharmacist before adding any new medication

**Evinacumab (Evkeeza) · Monoclonal antibody against angiopoietin-like 3 (ANGPTL3)**

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.

1. Infuse the antibody intravenously once a month
2. Antibody binds and neutralizes ANGPTL3
3. Lipoprotein lipase activity increases
4. Triglyceride-rich particles and LDL are cleared faster
5. Blood LDL drops by about 50 percent in homozygous FH patients

#### **SAFETY WATCH**

Infusion reactions — nausea, dizziness, or flushing during or after the infusion · Flu-like symptoms — fatigue, body aches · Allergic reactions — rare but watch for rash, trouble breathing, or swelling · Very limited long-term safety data — this is a new drug (approved 2021) · Extremely expensive and requires prior authorization

## **Red flags**

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- 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)

## **Nursing priorities**

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1. 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 — Cholesterol plaque can rupture suddenly and form a clot that blocks a coronary artery, causing heart attack; early detection and treatment within 90 minutes restores blood flow and prevents permanent heart muscle death
2. Check blood pressure, heart rate, and peripheral pulses in both arms and both legs; listen to carotid arteries for bruits (whooshing sounds) — High cholesterol causes plaque throughout the arterial system; weak or absent pulses indicate severe blockage in peripheral arteries, blood pressure differences between arms suggest subclavian artery narrowing (stenosis), and carotid bruits warn of stroke risk from narrowed neck arteries

3. 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 — Treatment intensity depends on absolute LDL number and overall cardiovascular risk; patients with LDL above 190 mg/dL or 10-year ASCVD risk above 20 percent need high-intensity statins, while triglycerides above 500 mg/dL require immediate intervention to prevent pancreatitis
4. 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 — Dietary saturated fat in red meat and dairy directly raises LDL cholesterol by increasing cholesterol production in the liver, while soluble fiber from vegetables and whole grains binds cholesterol in the intestines and removes it in stool before it enters the bloodstream
5. 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 — Aerobic exercise raises HDL cholesterol (the protective type that carries cholesterol away from arteries back to the liver) and lowers triglycerides by increasing the enzymes that break down fat particles in the blood
6. 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 — The liver makes most cholesterol at night, so evening dosing maximizes statin effectiveness by blocking HMG-CoA reductase (the enzyme that produces cholesterol); early detection of myopathy (muscle inflammation) prevents progression to rhabdomyolysis, and medication adherence prevents plaque progression even without symptoms
7. 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 — Lipid levels guide treatment intensification; if LDL remains above goal despite maximum statin dose, adding ezetimibe or PCSK9 inhibitors is necessary; elevated liver enzymes above three times normal or CK above ten times normal require statin discontinuation
8. 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 — Statin therapy reduces cardiovascular events by 25 to 35 percent, but only if taken consistently; many patients stop statins due to misinformation about side effects or try unproven alternatives like red yeast rice, leaving them unprotected against progressive plaque buildup

## Sources

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

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