

Hyperlipidemia

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THE STORY IN ONE SENTENCE

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

Normal anatomy — the body's job

Your liver makes cholesterol every day because your body needs it to build cell walls, make vitamin D, and create hormones. The liver packages cholesterol into tiny protein-coated balls called lipoproteins so it can travel through your watery blood. Low-density lipoprotein (LDL) delivers cholesterol out to tissues; high-density lipoprotein (HDL) picks up extra cholesterol and hauls it back to the liver to be recycled or flushed out in bile. Triglycerides come mainly from food and are packaged separately to fuel muscles. In a healthy system, LDL and HDL stay balanced, artery walls stay smooth, and blood flows freely.

BODY'S JOB

The liver regulates how much cholesterol and fat circulate in your blood. It reads signals from food, hormones, and your genes, then adjusts production and packaging to keep levels steady. HDL acts like a garbage truck, constantly sweeping excess cholesterol off artery walls before it can stick. This balance keeps your arteries clean and flexible, so oxygen-rich blood reaches every organ without resistance.

1. Your liver makes cholesterol and wraps it in LDL particles (the delivery truck).
2. LDL floats through your bloodstream, dropping cholesterol where cells need it.
3. HDL particles (the cleanup crew) collect leftover cholesterol from artery walls and tissues.
4. HDL carries the cholesterol back to the liver.

5. 5. The liver recycles it or dumps it into bile, which leaves your body in stool.
6. 6. Triglycerides from food are packaged into chylomicrons and VLDL, travel to muscles and fat for storage or fuel, then shrink as the fat is used.
7. 7. Throughout, your artery walls stay smooth and open because there is no excess fat to deposit.

What breaks

In hyperlipidemia, your liver makes too much LDL, your cells cannot pull enough of it out of the blood, or your body does not make enough HDL to haul the extra away. The result is a traffic jam of fat particles in your bloodstream. Excess LDL seeps into the artery wall, where it oxidizes (goes rancid like old oil), triggers inflammation, and hardens into plaque.

CLINICAL TERM

Primary hyperlipidemia arises from genetic defects in LDL receptor number or function (familial hypercholesterolemia), apolipoprotein structure (familial dysbetalipoproteinemia), or triglyceride clearance (familial hypertriglyceridemia). Secondary hyperlipidemia results from diabetes (insulin resistance reduces lipoprotein lipase activity), hypothyroidism (low thyroid hormone slows LDL receptor recycling), nephrotic syndrome (the kidney spills albumin and the liver overproduces lipoproteins to compensate), obesity, or a diet high in saturated fat and trans fat.

How the body compensates

At first, your artery walls try to bury the cholesterol deposits under a smooth cap of muscle cells and collagen, keeping blood flow open. Your heart pumps a little harder to push through narrowed vessels, and collateral (detour) arteries may sprout to route blood around blockages. But these fixes eventually backfire: the caps can crack, hard pumping raises blood pressure and strains the heart, and collaterals cannot match the original artery's capacity.

1. Immune cells (macrophages) in the artery wall gobble up oxidized LDL, trying to clear the damage, but they swell into foam cells and die, forming the fatty core of a plaque.
2. Smooth muscle cells migrate over the core and lay down a fibrous cap to wall it off, temporarily stabilizing the artery and keeping the lumen partly open.
3. The heart increases stroke volume and rate to maintain blood flow through narrowed arteries, which keeps organs fed but raises blood pressure and thickens the heart muscle over time (left ventricular hypertrophy).

4. Collateral vessels branch and enlarge to bypass a slowly growing blockage, but they are smaller and less efficient, so any sudden complete blockage still causes a heart attack or stroke.
5. Once the plaque cap thins or cracks—triggered by inflammation, high blood pressure, or shear stress—platelets rush in to seal the break, forming a clot that can suddenly plug the artery and cut off all blood flow.

Domino effect

1. Excess LDL particles seep through the endothelium (the thin inner lining of the artery) and lodge in the wall beneath. — 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). — 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. — 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. — 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. — 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. — 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). — 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. — 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.

Symptoms · what the patient feels, 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).
- No symptoms at all (most common): Cholesterol builds silently inside artery walls for years without hurting or showing outside signs; most people feel completely normal until a heart attack, stroke, or screening blood test reveals the problem.

Focused exam

- Xanthomas (yellow fatty bumps on elbows, knees, tendons, or eyelids): These bumps are cholesterol deposits sitting under the skin. When you see them, it tells you the blood cholesterol has been very high for a long time—often a sign of familial (inherited) hyperlipidemia.
- Arcus corneae (a white or gray ring around the colored part of the eye): This ring is cholesterol deposited in the edge of the cornea. In someone under 50, it raises suspicion for inherited high cholesterol that has been quietly damaging arteries.
- Carotid bruits (a whooshing sound heard with a stethoscope over the neck arteries): The whoosh means blood is rushing through a narrowed carotid artery—plaque has built up enough to partly block the pipe that feeds the brain. This tells you the hyperlipidemia has already caused real artery damage and stroke risk is climbing.
- Peripheral pulses (feel the pulses in the feet and compare side to side): Weak or absent foot pulses mean the arteries in the legs are clogged—a sign that cholesterol buildup has reached the point where blood flow is choked off downstream. This predicts similar blockages in the heart and brain arteries.
- Blood pressure in both arms: High cholesterol often travels with high blood pressure. Together they double the stress on artery walls and speed up plaque formation. Measuring BP tells you if you need to treat two problems at once.

Diagnostics · every test answers a question

- Lipid panel (total cholesterol, LDL, HDL, triglycerides) after 9–12 hour fast → How high is the cholesterol, and which type is elevated?
- 10-year ASCVD risk calculator (using age, sex, race, cholesterol, BP, diabetes, smoking) → What is this person's chance of having a heart attack or stroke in the next 10 years?
- Hemoglobin A1c → Does this person also have diabetes, which would raise cardiovascular risk even higher?
- Basic metabolic panel (BMP) including creatinine and eGFR → Are the kidneys working normally, and can they handle the medicines we might prescribe?
- Liver function tests (ALT, AST) before starting statin → Is the liver healthy enough to tolerate a statin, which the liver processes?
- Thyroid-stimulating hormone (TSH) → Is an underactive thyroid causing or worsening the high cholesterol?

Treatment logic

- 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.
- 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.
- 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.
- 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.
- 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 ≥ 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.
- Icosapent ethyl 2 g twice daily (if triglycerides 150–499 mg/dL despite statin) → A purified omega-3 fatty acid that lowers triglycerides and reduces inflammation in artery walls. In people already on a statin with high triglycerides, it cuts the risk of heart attack and stroke by about 25%.
- Fibrate (fenofibrate 145 mg daily) if triglycerides ≥ 500 mg/dL → Activates PPAR-alpha receptors in the liver, which ramp up enzymes that break down triglycerides and boost HDL. This prevents triglyceride levels from climbing so high that the pancreas becomes inflamed (pancreatitis).

Medications

Atorvastatin (Lipitor) · Statin (HMG-CoA reductase inhibitor)

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.

1. Atorvastatin enters liver cells and binds tightly to the HMG-CoA reductase enzyme, turning it off.
2. With less cholesterol being made inside the liver, the liver cells need more cholesterol to keep working.
3. The liver responds by putting more LDL receptors on its surface — these are like docking stations that grab LDL cholesterol from the blood.
4. More LDL gets pulled out of the blood and into the liver, so blood LDL levels drop.
5. Over weeks, this also lowers total cholesterol and can raise HDL (the 'good' cholesterol) a little.

SAFETY WATCH

Muscle pain or weakness (myopathy) — rare but serious. If muscles hurt badly or feel very weak, tell your doctor right away. · Liver enzyme rise — the doctor will check blood tests before starting and a few months later to make sure the liver is handling the drug well. · Avoid grapefruit juice in large amounts — it raises atorvastatin levels in the blood and increases the risk of side effects. · Use reliable birth control if you can become pregnant — statins can harm a developing baby. · Diabetes risk — statins may nudge blood sugar up slightly in some people, so blood sugar is monitored.

Rosuvastatin (Crestor) · Statin (HMG-CoA reductase inhibitor)

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.

1. Rosuvastatin binds to and shuts off the HMG-CoA reductase enzyme in liver cells.
2. The liver makes less cholesterol internally.
3. The liver compensates by increasing LDL receptors on its surface.
4. More LDL cholesterol is cleared from the bloodstream into the liver.
5. Blood LDL levels fall significantly, often by 50% or more at higher doses.

SAFETY WATCH

Muscle pain or damage (myopathy, rhabdomyolysis) — very rare but serious. Report severe muscle pain, dark urine, or unusual weakness immediately. · Liver enzyme elevation — blood tests monitor liver function before and during treatment. · Avoid grapefruit juice in large amounts. · Not safe during pregnancy or breastfeeding — effective contraception is required for those who can become pregnant. · May slightly raise blood sugar — diabetes screening is recommended for at-risk patients. · Kidney function — lower doses are used if kidney function is reduced.

Ezetimibe (Zetia) · Cholesterol absorption inhibitor

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.

1. Ezetimibe binds to the NPC1L1 transporter on the surface of intestinal cells.
2. Cholesterol from food and bile cannot be absorbed as efficiently.
3. Less cholesterol enters the blood from the gut.
4. The liver senses the drop and increases LDL receptors to pull more LDL from the blood.
5. Blood LDL levels fall by about 15-20% when ezetimibe is used alone, and even more when combined with a statin.

SAFETY WATCH

Generally very well tolerated with few side effects. · Mild diarrhea or stomach upset can happen but is uncommon. · Liver enzymes — when combined with a statin, liver tests are monitored as with statin therapy alone. · Muscle problems are rare but can occur, especially if combined with a statin; report any severe muscle pain.

Fenofibrate (Tricor, Fenoglide) · Fibrate (PPAR-alpha agonist)

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.

1. Fenofibrate enters cells and binds to the PPAR-alpha receptor.
2. PPAR-alpha switches on genes that make enzymes to burn fatty acids (the building blocks of triglycerides).

3. The liver produces more lipoprotein lipase (an enzyme that breaks down triglycerides in the blood).
4. Triglyceride levels drop, often by 30–50%.
5. HDL cholesterol rises modestly (by about 10–15%).
6. LDL may drop slightly, or LDL particles may shift to a less harmful type.

SAFETY WATCH

Muscle pain or damage (especially when combined with a statin) — report severe muscle aches or weakness. · Liver enzyme elevation — blood tests monitor liver function. · Gallstones — fibrates can increase the risk of gallstones forming. · Kidney function — dose is adjusted if kidney function is reduced; not used if kidney function is very poor. · Blood thinner interaction — fenofibrate can boost the effect of warfarin, so INR (blood clotting time) must be watched closely if both are used.

Icosapent ethyl (Vascepa) · Prescription omega-3 fatty acid (EPA)

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.

1. Icosapent ethyl is absorbed and travels to the liver.
2. It slows down enzymes that assemble triglycerides into VLDL particles.
3. The liver releases fewer VLDL particles into the blood.
4. Blood triglyceride levels drop.
5. EPA also reduces inflammation in the blood vessel lining, which may stabilize plaques and lower cardiovascular events.

SAFETY WATCH

Atrial fibrillation (an irregular, quivering heartbeat) (irregular heartbeat) — slightly higher risk seen in trials; tell your doctor if you feel a fluttering or racing heart. · Bleeding — omega-3s can thin the blood slightly, so watch for easy bruising, nosebleeds, or unusual bleeding, especially if you take aspirin or a blood thinner. · Allergic reaction — rare; avoid if you have a serious fish or shellfish allergy. · Generally well tolerated; side effects are mild (burping, fishy aftertaste).

Evolocumab (Repatha) or Alirocumab (Praluent) · PCSK9 inhibitor (monoclonal antibody)

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.

1. The antibody (evolocumab or alirocumab) is injected under the skin.
2. It enters the bloodstream and binds tightly to PCSK9 protein.
3. PCSK9 can no longer attach to LDL receptors on liver cells.
4. LDL receptors are not destroyed and stay on the liver surface longer.
5. The liver clears much more LDL cholesterol from the blood.
6. Blood LDL levels drop dramatically within days to weeks.

SAFETY WATCH

Injection site reactions — redness, swelling, or soreness where the shot is given (usually mild). · Allergic reactions — rare; watch for rash, itching, trouble breathing. · Flu-like symptoms — mild muscle aches or fatigue can occur. · Very low LDL levels — generally safe, but long-term effects of extremely low LDL are still being studied. · Cost — these drugs are expensive and usually reserved for high-risk patients or those who cannot tolerate statins.

Bempedoic acid (Nexletol) · ATP citrate lyase inhibitor

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.

1. Bempedoic acid is taken as a pill and travels to the liver.
2. Inside liver cells, it is converted to its active form.
3. The active drug binds to and blocks ATP citrate lyase.
4. The cholesterol-making pathway is interrupted early.
5. The liver makes less cholesterol and upregulates LDL receptors to pull more LDL from the blood.
6. Blood LDL levels drop by about 15–25% (more when combined with a statin or ezetimibe).

SAFETY WATCH

Tendon rupture — rare but reported; avoid if you have a history of tendon problems, and report sudden severe pain in a joint or tendon. · Elevated uric acid — can trigger gout in people prone to it; uric acid levels are monitored. · Elevated liver enzymes — liver tests are checked before and during treatment. · Generally well tolerated with low risk of muscle side effects compared to statins.

Red flags

- 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.

Nursing priorities

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. — 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. — 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. — 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.
4. 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. — The liver enzyme HMG-CoA reductase makes most cholesterol during sleep hours, so evening dosing blocks production when it is highest. Statins can cause hepatotoxicity (liver damage) shown by elevated ALT and AST enzymes, requiring dose reduction or discontinuation before permanent damage occurs.
5. 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. — Visible cholesterol deposits indicate LDL levels often above 300 mg/dL or familial hyperlipidemia, conditions requiring aggressive therapy beyond primary care scope. These physical findings also confirm chronic elevation and very high cardiovascular event risk within 5 years without treatment.
6. 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. — Statins cause myopathy (muscle disease) in up to 10 percent of patients, with severe rhabdomyolysis releasing myoglobin that clogs kidney tubules causing acute renal failure. Creatine kinase levels above 10 times normal with symptoms require immediate statin cessation and IV hydration to protect kidneys.
7. 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. — The ASCVD calculator predicts heart attack and stroke probability and guides evidence-based treatment: risk above 7.5 percent needs moderate to high-intensity statin, while risk below 5 percent may allow lifestyle modification alone. This prevents both overtreatment of low-risk patients and undertreatment of high-risk ones.
8. 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. — Saturated fat raises LDL by increasing liver cholesterol production, while soluble fiber binds bile acids in the intestine forcing the liver to use cholesterol to make new bile. Dietary changes can lower LDL by 10 to 15 percent, sometimes avoiding medication in borderline cases or allowing lower statin doses.

Sources

- 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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