

Coronary Artery Disease

Coronary artery disease happens when fatty plaques build up inside the heart's own fuel lines — the coronary arteries — narrowing them so less oxygen-rich blood reaches the heart muscle, causing chest pain and raising the risk of heart attack.

THE ONE-LINE TRUTH

Coronary artery disease is fatty plaques blocking the arteries that feed the heart muscle, creating a mismatch between oxygen supply and oxygen demand that causes chest pain, heart attacks, and heart failure.

The chain — how it unfolds

1. Endothelial injury and dysfunction — why: Risk factors like high blood pressure (mechanical shear stress), smoking (toxic chemicals), diabetes (high blood sugar damaging proteins), and high LDL cholesterol injure the endothelium (the thin protective lining inside the coronary arteries), reducing its production of nitric oxide (the artery's natural protector and dilator) and making the lining leaky so cholesterol can sneak underneath.
2. LDL infiltration and oxidation — why: Small LDL cholesterol particles penetrate the damaged endothelium, enter the artery wall, and become oxidized (chemically altered by free radicals). Oxidized LDL is recognized as foreign and dangerous, triggering an inflammatory alarm that recruits white blood cells (monocytes) into the artery wall.
3. Foam cell formation and atherosclerotic plaque growth — why: Monocytes entering the artery wall transform into macrophages (immune cells that eat debris) and gobble up the oxidized LDL in an attempt to clean it up. But the macrophages become overwhelmed and bloat into foam cells (so named because they look foamy under the microscope). Foam cells pile up, die, and release their fatty contents, forming a lipid-rich dead (necrotic) core covered by a fibrous cap of scar tissue — this is an atherosclerotic plaque. Over years the plaque grows larger and bulges into the artery channel.

4. Stable angina (exertional chest pain) — why: Once the plaque blocks about 70% or more of the artery lumen (the open channel), blood flow is adequate at rest but cannot increase enough during exercise, emotional stress, or cold exposure when the heart needs more oxygen. This supply-demand mismatch causes myocardial starved blood flow (ischemia) (lack of oxygen to heart muscle cells), which triggers chest pain or pressure (angina) — typically felt in the center or left chest, sometimes spreading to the jaw, shoulder, or arm, and relieved by rest within a few minutes.
5. Plaque rupture or erosion — why: The fibrous cap covering the plaque can suddenly crack (rupture) or wear away (erosion), especially if the cap is thin and the plaque is inflamed. Triggers include sudden blood pressure surges (heavy lifting, intense emotion, waking up in the morning), ongoing inflammation (enzymes from immune cells weaken the collagen in the cap), or mechanical stress. Rupture exposes the highly thrombogenic (clot-triggering) lipid core and tissue factor to flowing blood.
6. Acute clot formation (thrombosis) (blood clot formation) — why: When the plaque core is exposed, tissue factor instantly activates the coagulation cascade (the blood clotting system), and platelets (tiny clotting cells) stick to the ruptured surface. Within minutes a blood clot (thrombus) (blood clot) forms on top of the plaque, suddenly blocking most or all blood flow through the coronary artery. This causes an acute coronary syndrome — either unstable angina (severe chest pain at rest, but the clot is partial or breaking up so muscle is not yet dead) or heart attack (myocardial infarction) (heart attack, where the clot is large and stable enough to cause actual muscle death).
7. Heart attack (Myocardial infarction) (heart attack) — why: If a coronary artery is completely blocked for more than 20–30 minutes, heart muscle cells downstream run out of oxygen and ATP (energy molecules), cannot maintain their ion pumps, swell, and die (infarct). Dead cells release troponin (a structural protein from inside the cell) and CK-MB (an enzyme) into the bloodstream — lab tests detect these and confirm the heart attack. The infarcted muscle is replaced by scar tissue, which cannot contract, permanently weakening the heart's pumping power.
8. Left ventricular dysfunction and heart failure — why: Dead or chronically blood-starved (ischemic) (oxygen-starved) heart muscle cannot contract properly, reducing the heart's ejection fraction (the percentage of blood pumped out with each beat; normal is 55–70%). The remaining healthy muscle compensates by stretching and working harder, but over time the left ventricle dilates (enlarges) and remodels into a weaker, less efficient shape. As pumping power falls, blood backs up into the lungs (causing shortness of breath, especially when lying flat) and fluid leaks into body tissues (causing ankle swelling) — this is congestive heart failure.

Spot it — what you see, and why

- Chest pain or pressure with exertion: The coronary arteries (the blood vessels that feed the heart muscle itself) are narrowed by plaque buildup, like a pipe clogged with gunk. When you move or climb stairs, your heart beats faster and needs more oxygen. But the narrowed pipes can't deliver enough oxygen-rich blood, so the heart muscle goes hungry — that's called starved blood flow (ischemia) (not enough blood reaching the tissue to feed it). The starving muscle sends pain signals that you feel as tightness or squeezing in your chest.
- Chest pain at rest lasting more than 20 minutes: A coronary artery may be almost completely blocked or shut off by a blood clot forming on top of a plaque. Now the heart muscle is being starved of oxygen even when you're sitting still. If this lasts long enough, the muscle cells start to die — that's a heart attack (myocardial infarction) (heart attack). The pain doesn't ease because the blockage isn't opening back up on its own.
- Shortness of breath with mild activity or at rest: When the heart muscle isn't getting enough blood, it can't pump as strongly. Blood backs up into the lungs, causing fluid to leak into the tiny air sacs — called pulmonary congestion (like the lungs getting waterlogged). This makes it harder for oxygen to pass from air into your blood, so you feel short of breath even with light effort or just sitting.
- Pain in the jaw, neck, arm, or back during exertion: The pain signals from your heart travel up through nerves in your spinal cord. Because the heart and these other areas share some of the same nerve wiring, your brain sometimes gets confused about where the pain is really coming from. This is called referred pain — the ache you feel in your arm or jaw is actually the heart's distress signal taking a detour.
- Sudden severe weakness or loss of consciousness: Severe lack of oxygen to the heart muscle can trigger dangerous abnormal heart rhythms — like ventricular a fast heart rate (tachycardia) (the lower pumping chambers beating wildly and uselessly) or complete heart block (the electrical signal can't travel through the heart at all). Either way, the heart stops pumping blood effectively, so your brain doesn't get enough oxygen and you pass out or feel suddenly very weak.
- Nausea, vomiting, or indigestion-like discomfort: When the back or bottom wall of the heart isn't getting enough blood, it can irritate the vagus nerve (a long nerve that connects the brain to the stomach and heart and controls automatic body functions like digestion). This nerve sends signals that upset your stomach, making you feel sick, queasy, or like you have bad heartburn.

Red flags — act now

- Chest pain lasting more than 20 minutes with sweating or nausea — suggests a heart attack happening right now and needs an ECG and trip to the catheterization lab (where doctors can open the blocked artery) immediately.
- Sudden severe chest pain that rips or tears into the back — may mean an aortic dissection (the main artery from the heart is tearing apart, not a coronary blockage). Needs an urgent CT scan and fast blood pressure control.
- New shortness of breath or leg swelling with chest pain — suggests a large heart attack or a torn valve muscle inside the heart. Needs an urgent ultrasound of the heart (echocardiogram) to see what's broken.
- Passing out or losing consciousness with chest pain — may mean a deadly heart rhythm like ventricular a fast heart rate (tachycardia) or complete heart block. Needs immediate heart monitor and shock treatment if needed.

Treat it — in this order

1. High-dose statin (atorvastatin 40–80 mg or rosuvastatin 20–40 mg daily) → Statins block an enzyme in the liver that makes cholesterol, dropping LDL by half or more. They also calm inflammation inside plaques and make plaques less likely to crack open and cause a clot. Over time, this cuts the risk of heart attack, stroke, and death by 25–35% in people with coronary disease. The benefit grows the longer you take it.
2. Two antiplatelet drugs: aspirin (81–162 mg daily) plus a P2Y₁₂ inhibitor (clopidogrel 75 mg, ticagrelor 90 mg twice daily, or prasugrel 10 mg daily) → Platelets are tiny blood cells that clump together to form clots. When a plaque cracks, platelets rush in and can seal off the artery completely. These medicines block two different steps in platelet clumping, so even if one pathway tries to activate, the other is jammed. Using two together (dual antiplatelet therapy) lowers the chance of clotting inside a stent by over 80% and cuts repeat heart attacks by 20–30% after an acute coronary syndrome, compared to aspirin alone.
3. Beta-blocker (metoprolol, carvedilol, or bisoprolol) → Beta-blockers attach to receptors on the heart and block adrenaline's push, slowing the heart rate and weakening each beat slightly. This makes the heart need less oxygen—helpful when arteries cannot deliver enough. They lower blood pressure, reduce chest pain episodes, and, after a heart attack, prevent dangerous rhythm problems and cut the risk of dying, especially if the heart is weak (ejection fraction under 40%).

4. ACE inhibitor (lisinopril, enalapril, ramipril) or ARB (losartan, valsartan) if ACE inhibitor causes cough → These medicines block a hormone cascade (the renin-angiotensin-aldosterone system) that tightens blood vessels and makes the heart swell and stiffen after injury. By stopping that, they lower blood pressure, ease the heart's workload, and prevent the heart from reshaping in harmful ways after a heart attack. They reduce death and future heart events by 20–25% in people with coronary disease who also have a weak heart or diabetes.
5. Percutaneous coronary intervention (PCI): threading a balloon and stent through an artery in the wrist or groin to the blockage, inflating the balloon to squash the plaque, then leaving a tiny metal mesh tube (stent) to hold the artery open → PCI mechanically opens the artery in minutes, restoring blood flow immediately. It stops a heart attack in its tracks (if done within 90 minutes of arrival for ST-elevation MI, it saves lives), relieves chest pain in most patients, and prevents further muscle death. The stent keeps the artery open long-term.

Your top priorities at the bedside

1. Monitor for chest pain and obtain 12-lead ECG within 10 minutes — why: Coronary artery disease (CAD) causes narrowed heart arteries that may lead to acute coronary syndrome (sudden blockage causing heart attack). Early ECG identifies ST-elevation heart attack (myocardial infarction) (STEMI, complete artery blockage) versus non-STEMI, guiding time-sensitive treatment like percutaneous coronary intervention (PCI, balloon and stent to open artery).
2. Administer aspirin 162 to 325 mg chewable immediately if chest pain occurs — why: Aspirin inhibits platelet aggregation (clumping of blood cells that form clots) within 5 minutes when chewed. This reduces a blood clot (thrombus) (blood clot) size in coronary arteries and lowers mortality by 23 percent in acute heart attack (myocardial infarction) (heart attack) per AHA/ACC guidelines.
3. Assess for heart failure signs including trouble breathing (dyspnea), crackles, and jugular venous distension — why: Chronic CAD weakens the myocardium (heart muscle) over time, reducing ejection fraction (percentage of blood pumped out each beat). Left ventricular dysfunction causes pulmonary edema (fluid flooding the lungs) (fluid in lungs) and peripheral swelling (edema) (swelling in legs), requiring diuretics (water pills) and ACE inhibitors (medicines that relax blood vessels).