

Coronary Artery Disease

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart is a powerful muscle that pumps blood to your entire body, but it also needs its own steady supply of oxygen to keep working. Three main arteries called coronary arteries wrap around the outside of your heart like a crown (that's where the name coronary comes from) and deliver fresh oxygen-rich blood to the heart muscle itself. In coronary artery disease, fatty deposits called plaques slowly collect on the inner walls of these arteries, like grease building up inside a kitchen pipe. Over months and years the plaques grow larger, making the arteries narrower and stiffer so less blood can flow through. At rest your heart may get enough oxygen, but when you climb stairs, exercise, or feel stressed, your heart works harder and needs extra fuel — and the narrowed arteries cannot deliver it. That causes chest pain or pressure called angina, usually felt in the center or left side of the chest and sometimes spreading to the jaw, shoulder, or arm. If a plaque suddenly cracks open, a blood clot can form instantly and completely block the artery, cutting off oxygen to part of the heart muscle. That is a heart attack, and the muscle starts to die within minutes. Quick treatment can reopen the artery and save the muscle, which is why anyone with new or worsening chest pain, especially with sweating, nausea, or shortness of breath, should call 911 right away. The main risk factors for coronary artery disease are high cholesterol, high blood pressure, smoking, diabetes, family history, obesity, and lack of exercise — and many of these can be improved with medication, healthy eating, regular physical activity, and quitting smoking.

What you might feel

- Chest pain or pressure with exertion
- Chest pain at rest lasting more than 20 minutes
- Shortness of breath with mild activity or at rest
- Pain in the jaw, neck, arm, or back during exertion
- Sudden severe weakness or loss of consciousness
- Nausea, vomiting, or indigestion-like discomfort
- New or worsening fatigue that limits daily activities

- Heavy cold sweating with chest discomfort

Call the doctor right away if...

- 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.

What the care team will do

- Monitor for chest pain and obtain 12-lead ECG within 10 minutes
- Administer aspirin 162 to 325 mg chewable immediately if chest pain occurs
- Assess for heart failure signs including trouble breathing (dyspnea), crackles, and jugular venous distension
- Monitor blood pressure and titrate beta-blockers to target heart rate 55 to 60 beats per minute
- Ensure statin therapy is prescribed with LDL cholesterol goal below 70 mg/dL
- Educate on nitroglycerin use: one tablet under tongue every 5 minutes for chest pain, call 911 if no relief after first dose
- Assess for antiplatelet therapy adherence and check for bleeding complications
- Screen for modifiable risk factors including smoking, diabetes control with HbA1c below 7 percent, and blood pressure under 130/80 mmHg

Medicines you may be given — and why

Atorvastatin

Atorvastatin blocks an enzyme in liver cells called HMG-CoA reductase. This enzyme is the main control point for making cholesterol. When the liver cannot make enough cholesterol on its own, the liver cells pull LDL cholesterol (the 'bad' cholesterol) out of the blood by putting

more LDL catcher molecules on their surface. This lowers the amount of LDL floating in the blood. The drug also quiets down inflammation inside the fatty plaques that clog arteries, making those plaques more stable and less likely to crack open and cause a heart attack.

Aspirin

Aspirin permanently blocks an enzyme called cyclooxygenase-1 (COX-1) inside platelets by sticking an acetyl chemical tag onto a specific spot on the enzyme. This stops platelets from making thromboxane A2, a powerful signal that tells platelets to clump together and form clots. Because platelets have no control center (nucleus) to build new enzyme, one dose of aspirin shuts down that platelet for its entire 7 to 10 day life. This makes the blood less likely to clot where a plaque cracks open in a coronary artery.

Metoprolol

Metoprolol blocks beta-1 receptors on heart muscle cells. These receptors are docking spots for adrenaline and noradrenaline (the body's stress hormones). When the drug blocks these spots, the stress hormones cannot signal the heart to speed up or squeeze harder. The heart beats slower, pumps less forcefully, and sends electrical signals through the AV node (the relay station between the top and bottom chambers) more slowly. This cuts how much oxygen the heart muscle needs and helps prevent dangerous fast rhythms that can happen after a heart attack.

Nitroglycerin

Nitroglycerin is turned into nitric oxide gas inside the smooth muscle cells that wrap around blood vessels. Nitric oxide flips on an enzyme called guanylate cyclase, which makes cyclic GMP. Cyclic GMP tells the smooth muscle to relax. When veins relax, less blood comes back to the heart (lowering preload, like easing the demand on a pump). When arteries relax, the heart pumps against less resistance (lowering afterload, like opening a kink in a hose). Both changes mean the heart needs less oxygen, and chest pain goes away within a few minutes.

Clopidogrel

Clopidogrel is an inactive drug until liver enzymes turn it into the active form. The active form sticks permanently to the P2Y12 receptor on the surface of platelets. This receptor is a docking spot for ADP (a chemical released when platelets activate). When the receptor is blocked, ADP cannot tell other platelets to turn on and clump together. This breaks the snowball effect that normally builds clots at sites where plaques rupture or on the metal mesh of coronary stents.

Lisinopril

Lisinopril blocks the ACE enzyme that normally turns angiotensin I into angiotensin II. Angiotensin II is a powerful hormone that squeezes blood vessels tight and tells the adrenal glands to make aldosterone, which holds onto salt and water. When ACE is blocked, blood

vessels relax (lowering blood pressure and the resistance the heart pumps against), the kidneys let go of more salt and water (shrinking blood volume and the load returning to the heart), and harmful thickening and scarring of heart muscle after a heart attack are reduced. Blocking ACE also raises levels of bradykinin, which helps blood vessel linings work better.

Where this information comes from

- AHA/ACC 2021 Chest Pain Guideline
- ACC/AHA 2022 Guideline for Management of Patients With Chronic Coronary Disease
- AHA/ACC 2020 Guideline on Management of Patients With Non-ST-Elevation Acute Coronary Syndromes
- ACC/AHA 2023 Acute Coronary Syndrome Guideline

Last reviewed: 2026-08-29