

Acute Coronary Syndrome

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart is a muscle that never stops beating, so it needs oxygen-rich blood delivered every second. Three main arteries called coronary arteries wrap around your heart like a crown and carry that blood. In Acute Coronary Syndrome, a patch of cholesterol buildup inside one of those arteries suddenly cracks open. Your blood sees the crack as an injury and sends tiny sticky cells called platelets to seal it. The platelets clump together and form a clot. If the clot blocks the artery completely, the heart muscle downstream loses its oxygen supply and starts to die within twenty to forty minutes—that is a heart attack, also called a heart attack (myocardial infarction). If the clot only partly blocks the artery, the muscle is starving but not dead yet—that is called unstable angina. Either way, the heart muscle in trouble cannot squeeze properly, electrical signals get scrambled, and you feel chest pressure or pain. The faster blood flow is opened back up, the more heart muscle can be saved. That is why chest pain or pressure, especially with sweating, shortness of breath, nausea, or pain spreading to the jaw or left arm, is always a medical emergency—every minute counts.

What you might feel

- Chest discomfort—pressure, squeezing, heaviness, or tightness
- Pain spreading to the left arm, jaw, neck, back, or upper belly
- Sweating (diaphoresis)
- Nausea or vomiting
- Shortness of breath (dyspnea)
- Lightheadedness or fainting (syncope)
- Feeling the heart race or beat irregularly (palpitations)
- Atypical presentation—just feeling tired, weak, or short of breath without any chest pain (especially in women, older adults, and people with diabetes)

Call the doctor right away if...

- ST-segment elevation on the EKG—this means a coronary artery is completely blocked (STEMI, ST-elevation heart attack (myocardial infarction)) and the patient needs the artery opened within 90 minutes with a balloon and stent (percutaneous coronary intervention, or PCI) or clot-busting medicine (fibrinolytic therapy)
- Cardiogenic shock—cool clammy skin, blood pressure very low, confusion, almost no urine output—means the heart is failing and cannot pump enough blood to keep the body alive
- New heart murmur heard on exam—suggests a papillary muscle tore (causing the mitral valve to leak, called acute mitral regurgitation) or a hole opened between the pumping chambers (ventricular septal defect, or VSD); both are life-threatening mechanical problems
- Ventricular arrhythmias on the monitor—ventricular a fast heart rate (tachycardia) (V-tach, fast dangerous rhythm from the lower chambers) or ventricular fibrillation (V-fib, heart just quivers and stops pumping)—can stop the heart and cause sudden cardiac arrest
- Right ventricular tissue death from blocked blood flow (infarction) signs—low blood pressure, neck veins bulging (elevated jugular venous pressure, or JVP), lungs clear on exam—needs IV fluids, NOT water pills (diuretics) or nitroglycerin, which will drop pressure even more

What the care team will do

- Get a 12-lead ECG within 10 minutes of arrival.
- Put the patient on a heart monitor and get IV access right away.
- Give chewed aspirin as ordered — once dissection or bleeding is not suspected.
- Draw the first troponin now and repeat it on schedule.
- Treat pain and give nitroglycerin as ordered — hold it if the blood pressure is low, a right-sided MI is suspected, or erection pills were taken.
- Give oxygen only if the oxygen level is below about 90%.
- Keep the patient on bed rest and NPO (nothing to eat or drink) until the plan is clear.
- Watch closely for change: new or worse pain, rhythm changes, falling blood pressure, or a new murmur.

Medicines you may be given — and why

Aspirin

Aspirin stops tiny blood cells called platelets from sticking together. Platelets usually help seal cuts, but when plaque (a fatty bump inside a heart artery) cracks open, platelets rush over and

glue themselves into a clot that can completely block blood flow to the heart. Aspirin permanently shuts off an enzyme called cyclooxygenase-1 (COX-1) inside each platelet. Without that enzyme, platelets cannot make thromboxane A2 (the sticky chemical signal that makes them clump). This keeps the dangerous clot from growing bigger and choking off the artery.

Ticagrelor, Prasugrel, or Clopidogrel (P2Y12 inhibitors)

These drugs block a second sticky pathway on platelets. Even with aspirin on board, platelets can still clump tightly if they receive a signal through a receptor called P2Y12 (a lock on the platelet's surface). When a chemical called ADP (adenosine diphosphate) fits into that lock, the platelet gets much stickier and recruits more platelets to the clot. P2Y12 inhibitors plug the lock so ADP cannot activate it. Using aspirin plus a P2Y12 inhibitor together (called dual antiplatelet therapy) shuts down platelet clumping through two separate doors, cutting the chance that the clot will grow back or a new one will form.

Heparin, Enoxaparin (low-molecular-weight heparin), or Bivalirudin

Anticoagulants stop clots from growing by blocking the proteins in the blood that weave a tight mesh around the platelet clump. Think of a clot as a net: platelets are the knots, and clotting proteins (especially thrombin and Factor Xa) are the threads that tie everything together into a solid plug. Heparin and enoxaparin boost a natural brake in the blood called antithrombin III, which shuts down thrombin and Factor Xa. Bivalirudin grabs thrombin directly and blocks its active site. Either way, the protein mesh cannot form, so the clot stays soft and small instead of turning into a hard plug that completely blocks the artery.

Metoprolol, Carvedilol, Atenolol, or other Beta-Blocker

Beta-blockers slow the heart rate and lower blood pressure, which reduces how hard the heart has to work. During a heart attack, part of the heart muscle is not getting enough oxygen because a coronary artery is blocked or narrowed. If the heart keeps racing and squeezing hard (driven by adrenaline flooding the body during the stress of a heart attack), the starving muscle runs out of oxygen even faster and more cells die. Beta-blockers sit on receptors in the heart where adrenaline normally docks and block adrenaline from attaching. This calms the heart down—it beats slower and squeezes less forcefully—so the oxygen-starved muscle can survive on the reduced blood supply while doctors work to open the artery.

Atorvastatin, Rosuvastatin, or other High-Intensity Statin

Statins lower cholesterol in the blood AND stabilize the cracked plaque inside arteries so it is much less likely to rupture again. The liver makes most of the body's cholesterol using an enzyme called HMG-CoA reductase (the rate-limiting enzyme in cholesterol production). Statins block that enzyme, so the liver makes far less cholesterol. When blood cholesterol drops, the liver pulls LDL cholesterol (the 'bad' kind) out of the bloodstream and out of artery plaques to meet its needs. Over weeks to months, the fatty core of the plaque shrinks. Statins

also reduce inflammation inside the plaque (lowering C-reactive protein and other inflammatory markers), which makes the fibrous cap over the plaque thicker and tougher. A stable plaque with a thick cap is much less likely to crack open and trigger another heart attack.

Nitroglycerin

Nitroglycerin relaxes and widens blood vessels, especially veins, which reduces how hard the heart has to work. When veins relax, they hold more blood (like widening a hose), so less blood returns to the heart (this is called lowering preload). With less blood filling the heart chambers, the heart does not have to stretch and squeeze as hard, which lowers oxygen demand. Nitroglycerin also relaxes arteries slightly, lowering the pressure the heart has to pump against (called afterload). Both effects together reduce the heart's workload.

Nitroglycerin may also widen coronary arteries (especially if spasm is part of the problem) and improve blood flow to blood-starved (ischemic) areas. It works by releasing nitric oxide inside vessel walls—nitric oxide is a powerful signal that tells smooth muscle in the vessel wall to relax.

Alteplase, Tenecteplase, or Reteplase (fibrinolytics / thrombolytics)

Fibrinolytics break up the clot that is blocking the coronary artery by activating the body's natural clot-dissolving system. When a clot forms, fibrin threads weave through it like a net, holding it together. The body has an enzyme called plasmin that cuts fibrin threads and dissolves clots, but plasmin is normally stored in an inactive form called plasminogen. Fibrinolytics convert plasminogen into active plasmin right at the clot, so plasmin chews through the fibrin net and the clot falls apart. Blood flow through the artery resumes, and oxygen reaches the starving heart muscle before too many cells die. This only works if given very early—ideally within 12 hours of symptom onset, but the earlier the better (greatest benefit in the first 2–3 hours).

Enalapril, Lisinopril, Ramipril, or other ACE Inhibitor (or ARB if ACE inhibitor not tolerated)

ACE inhibitors block an enzyme that makes a powerful hormone called angiotensin II, which squeezes blood vessels tight and raises blood pressure. After a heart attack, angiotensin II also triggers harmful remodeling—the heart muscle stretches, thins out, and weakens over weeks to months, raising the risk of heart failure. ACE inhibitors block the enzyme (ACE) that converts angiotensin I into angiotensin II, so angiotensin II levels drop. Blood vessels relax (lowering blood pressure and reducing how hard the heart has to pump), and the harmful remodeling is slowed or prevented. ACE inhibitors also raise levels of bradykinin (a helpful molecule that widens vessels and protects the heart), though this can cause a dry cough in some people. Started early after a heart attack (within 24 hours if the patient is stable), ACE inhibitors reduce the risk of heart failure and death.

Morphine

Morphine relieves severe chest pain and reduces anxiety during a heart attack. Pain and anxiety both trigger the release of adrenaline and noradrenaline, which make the heart beat faster and harder, raising oxygen demand in muscle that is already starving. Morphine binds to opioid receptors (mostly mu receptors) in the brain and spinal cord, blocking pain signals and producing a calming effect. It also causes veins to relax and dilate (venodilation), which reduces preload (less blood returning to the heart) and lowers the heart's workload slightly. By easing pain and anxiety, morphine helps bring heart rate and blood pressure down, which can reduce oxygen demand and limit further muscle damage.

Where this information comes from

- AHA/ACC Guideline for the Management of Patients With Non-ST-Elevation Acute Coronary Syndromes (2023)
- AHA/ACC STEMI Guideline (2023)
- ESC Guidelines for the Management of Acute Coronary Syndromes (2023)
- American College of Cardiology Clinical Consensus on High-Sensitivity Cardiac Troponin (2021)

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