

Acute Coronary Syndrome

Acute Coronary Syndrome means heart muscle is starving for oxygen right now because a blood clot has partly or completely blocked one of the arteries that feeds it.

THE ONE-LINE TRUTH

A clot blocks a heart artery, and heart muscle starts dying because it cannot get oxygen.

The chain — how it unfolds

1. Atherosclerotic plaque inside a coronary artery ruptures, and the fibrous cap covering it cracks open. — why: Atherosclerotic plaque is a buildup of cholesterol, fat, calcium, and inflammatory cells such as macrophages (immune cells that eat debris and release signals) in the artery wall. The plaque has a soft, fatty core covered by a thin fibrous cap made of collagen and smooth muscle cells. Chronic inflammation weakens the cap. Enzymes called matrix metalloproteinases (proteins that break down collagen), released by macrophages, digest the collagen. Mechanical stress from blood pressure pulsing against the plaque, especially at branch points where flow is turbulent (rough and swirling), causes the cap to crack or tear. The soft, fatty core inside is suddenly exposed to the bloodstream.
2. Platelets stick to the exposed plaque and form a blood clot (thrombus) (a blood clot). — why: Platelets are tiny cell fragments that float in the bloodstream and seal injuries. When the fibrous cap ruptures, collagen, tissue factor, and von Willebrand factor (proteins that signal damage) from the plaque core are exposed. Platelets have receptors, including GP Ib and GP IIb/IIIa (docking sites on the platelet surface), that recognize these proteins as danger signals. Platelets immediately stick to the exposed material, activate, change shape, and release chemicals such as ADP and thromboxane A₂ (signals that recruit more platelets). A clot builds on top of the ruptured plaque. The clotting cascade (a series of enzyme reactions) is also triggered, and fibrin strands weave through the platelet plug to strengthen it.

3. The a blood clot (thrombus) partly or completely blocks the coronary artery, and blood flow downstream drops or stops. — why: The clot grows rapidly and narrows or closes the artery. If the blockage is complete, oxygen-rich blood cannot reach the heart muscle supplied by that artery. If the blockage is partial, some blood still trickles through or arrives through tiny collateral vessels (backup routes that connect neighboring coronary arteries). These collaterals develop slowly over time in people with chronic narrowing, so someone with old blockages may tolerate a new clot better than someone with previously clean arteries.
4. Heart muscle downstream becomes blood-starved (ischemic) (starved of oxygen). — why: Heart muscle cells need oxygen to make ATP (the energy molecule that powers every cell function). Without oxygen, mitochondria (the cell's power plants) cannot run the electron transport chain (the main ATP-producing pathway). Cells switch to anaerobic glycolysis (making energy without oxygen), but this process is far less efficient, producing only two ATP per glucose molecule instead of thirty-six, and it generates lactic acid as a byproduct. Lactic acid builds up and makes the environment acidic. Within seconds, ATP levels drop, and the muscle starts to fail.
5. Blood-starved (Ischemic) muscle cannot contract normally, and that wall segment stops squeezing. — why: Muscle contraction requires ATP. During contraction, myosin heads (tiny motors inside muscle fibers) pull on actin filaments (protein threads that slide past each other), and each pull burns one ATP molecule. When ATP runs out, myosin heads cannot detach and reset, so the muscle fibers cannot shorten and squeeze. That wall segment moves weakly, called hypokinesis, or not at all, called akinesis. On an echocardiogram (an ultrasound of the heart), you can see the injured wall segment sit still while the rest of the heart squeezes. This reduces stroke volume (the amount of blood pumped with each beat), and cardiac output drops.
6. Electrical signals in the blood-starved (ischemic) zone become abnormal. — why: Normal heart rhythm depends on sodium, potassium, and calcium ions flowing in and out of cells in a precise pattern. Ion pumps, especially the sodium-potassium ATPase pump (a pump that uses ATP to move ions), require ATP to maintain concentration gradients (the difference in ion levels inside versus outside the cell). When ATP runs out, the pumps fail. Potassium leaks out of cells, and sodium and calcium flood in. The resting membrane potential (the electrical charge across the cell membrane) changes from its normal negative eighty-five millivolts to a less negative value. Cells depolarize (fire their electrical signal) and repolarize (reset for the next beat) in abnormal ways. The injured zone becomes electrically unstable, and dangerous arrhythmias such as ventricular a fast heart rate (tachycardia) and ventricular fibrillation can start.

7. ST-segment changes appear on the EKG. — why: The ST segment on an EKG represents the moment between ventricular depolarization (when the ventricles contract) and repolarization (when they reset). Starved blood flow (Ischemia) creates an abnormal electrical current, called an injury current, between the injured zone and the healthy zone during this moment. If the injury goes through the full thickness of the heart wall, called transmural starved blood flow, the ST segment lifts up, called ST elevation, in the EKG leads facing that wall. If the injury affects mainly the subendocardium (the inner layer), the ST segment drops down, called ST depression, or the T wave flips upside down, called T-wave inversion. The pattern and location of ST changes tell you which coronary artery is blocked: ST elevation in leads II, III, and aVF means the right coronary artery and the inferior wall; ST elevation in V1 through V4 means the left anterior descending artery and the front wall; ST elevation in I, aVL, V5, and V6 means the circumflex artery and the side or back wall.
8. Cardiac troponin leaks into the bloodstream. — why: Troponin is a protein inside heart muscle cells that regulates contraction by controlling calcium binding to actin and myosin. The troponin complex has three parts: troponin C binds calcium, troponin I inhibits contraction, and troponin T binds the complex to tropomyosin (another regulatory protein). When muscle cells die or their membranes break down from severe starved blood flow (ischemia), troponin spills out into the bloodstream, like grain spilling from a torn sack. Troponin I and troponin T are specific to heart muscle, so a rise in blood troponin means heart muscle is dying. Troponin is more specific and sensitive than older markers such as CK-MB. Troponin levels rise within two to six hours after symptom onset, peak at twelve to twenty-four hours, and stay elevated for one to two weeks. The first troponin test may be normal if drawn too early, within the first hour or two. That is why current guidelines recommend repeating troponin at three to six hours if the first is normal but suspicion is high. High-sensitivity troponin assays (newer tests approved in the United States and widely used in Europe) detect much smaller amounts and rise faster, sometimes within one to two hours, and can rule out heart attack (myocardial infarction) more quickly using zero-hour and one-hour or two-hour protocols.

Spot it — what you see, and why

- Chest discomfort—pressure, squeezing, heaviness, or tightness: When heart muscle does not get enough blood (ischemia, meaning the tissue is starving), the dying cells release chemicals like adenosine and bradykinin. These chemicals wake up pain nerves in the heart wall that send signals up the spinal cord to the brain. The brain reads those signals as deep pressure or squeezing, not a sharp stab.

- Pain spreading to the left arm, jaw, neck, back, or upper belly: Pain nerves from the heart and nerves from your arm, jaw, and belly all plug into the same spots in the spinal cord (they share the same nerve root level). So when the heart nerve fires, the brain sometimes guesses the pain is coming from your arm or jaw instead. This mix-up is called referred pain.
- Sweating (diaphoresis): When the heart is in trouble, your body's alarm system (the sympathetic nervous system, the branch that runs fight-or-flight) switches on. That system tells sweat glands all over your body to turn on, just like when you are scared or running hard.
- Nausea or vomiting: Pain nerves from the heart wake up the vagus nerve (a big nerve that runs from the brainstem down to the stomach and heart). The vagus nerve sends a signal to the vomit center in the brainstem, making you feel sick or throw up. This happens especially when the bottom wall of the heart (inferior wall) is hurt.
- Shortness of breath (dyspnea): When heart muscle is starving, it cannot squeeze well. The left ventricle (the main pumping chamber on the left side) cannot push all the blood out, so blood backs up into the lungs. Fluid leaks from the blood vessels into the air sacs (pulmonary congestion), making it hard to breathe.
- Lightheadedness or fainting (syncope): If the heart cannot pump enough blood forward (low cardiac output, meaning not enough blood is leaving the heart) or if the heart starts beating in a dangerous way (arrhythmia), blood pressure drops. The brain does not get enough oxygen and you feel dizzy or pass out.

Red flags — act now

- 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

Treat it — in this order

1. Aspirin 162–325 mg chewed immediately (non-enteric-coated tablet) → Irreversibly blocks cyclooxygenase-1 (COX-1, the enzyme inside platelets that makes clotting signals) so platelets cannot produce thromboxane A₂ (the chemical messenger that tells platelets to stick together). Chewing gets the drug absorbed through the lining of the mouth into the bloodstream within 5 minutes instead of waiting 30–60 minutes for a swallowed pill to dissolve in the stomach. Cuts 30-day death rate by about 23% even before any other treatment starts.
2. Second antiplatelet drug—ticagrelor 180 mg loading dose, prasugrel 60 mg loading dose, or clopidogrel 600 mg loading dose (dual antiplatelet therapy, called DAPT) → Blocks the P2Y₁₂ receptor on the platelet surface, a completely different pathway from aspirin—this stops ADP from activating platelets. Using two drugs hits platelet clumping from two angles at once, like locking two separate doors. Keeps the artery open after it has been unblocked by balloon and stent or clot-busting drugs, and cuts the chance of another heart attack or stent clot formation (thrombosis) (new clot forming on the stent) by 20–30%.
3. Anticoagulation—unfractionated heparin IV bolus (60–70 units per kg body weight, max 5000 units) plus continuous infusion, or enoxaparin 1 mg per kg subcutaneous injection every 12 hours, or bivalirudin continuous infusion → Heparin amplifies antithrombin III (a natural brake on clotting) to shut down the coagulation cascade—the chain reaction that weaves fibrin strands into a stable mesh holding the clot together. Bivalirudin directly inhibits thrombin itself. Both prevent new clot from forming on top of the existing a blood clot (thrombus), inside stents, or on catheters threaded through arteries during PCI.
4. Urgent percutaneous coronary intervention (PCI)—door-to-balloon time under 90 minutes for STEMI, within 24 hours for high-risk NSTEMI (GRACE score >140, refractory starved blood flow (ischemia), hemodynamic instability, or life-threatening arrhythmia) → A guide catheter threaded from the radial artery at the wrist (or femoral artery in the groin) delivers a deflated balloon to the blockage under live X-ray guidance. Inflating the balloon crushes the a blood clot (thrombus) and plaque against the artery wall and cracks open the fibrous cap, then a drug-eluting stent (a tiny mesh tube coated with medication that prevents scar tissue from growing back) is deployed to scaffold the artery open. Immediately restores forward blood flow and salvages blood-starved (ischemic) myocardium before irreversible tissue death (necrosis) (cell death) happens.

5. Fibrinolytic therapy (alteplase, tenecteplase, or reteplase IV) if PCI cannot be performed within 120 minutes of first medical contact and symptom onset is within 12 hours → Converts plasminogen (an inactive enzyme floating in the blood) into plasmin (the active enzyme that chews up fibrin), chemically dissolving the clot mesh. Restores TIMI 3 flow (normal brisk flow with dye filling the whole artery) in about 50–60% of patients. Most effective when given within 3 hours of symptom onset—every 30-minute delay cuts the benefit and more muscle dies.

Your top priorities at the bedside

1. Get a 12-lead ECG within 10 minutes of arrival. — why: A STEMI is diagnosed on the ECG, and every minute of delay is heart muscle dying.
2. Put the patient on a heart monitor and get IV access right away. — why: Deadly rhythms like ventricular fibrillation are most likely in the first hours of a heart attack.
3. Give chewed aspirin as ordered — once dissection or bleeding is not suspected. — why: Chewing gets it working fast. Aspirin stops platelets from making the clot bigger.