

Chest Pain and ACS Triage

Chest pain triage: sort by who will die first if you wait, then go after the upstream break before the body runs out of time.

THE ONE-LINE TRUTH

Chest pain triage means finding the life-threatening causes first: STEMI (complete blockage heart attack), NSTEMI (partial blockage heart attack), unstable angina (heart pain at rest or getting worse), aortic dissection (tear in the body's biggest artery), pulmonary embolism (a clot lodging in a lung artery) (blood clot in the lung), cardiac tamponade (the heart squeezed by fluid around it) (blood or fluid squeezing the heart so it cannot fill), tension pneumothorax (air trapped in the chest pushing the heart sideways), and esophageal rupture (hole torn in the swallowing tube).

The chain — how it unfolds

1. A coronary artery gets blocked by a clot, or the heart is forced to work harder than its blood supply can support. — why: Whether the problem is a ruptured plaque with clot formation (thrombosis), a spasm, or a supply-demand mismatch, the result is the same: part of the heart muscle is starving for oxygen.
2. Blood-starved (Ischemic) heart muscle screams in pain that feels like pressure, tightness, squeezing, or a heavy weight on the chest. — why: When cells run out of oxygen they switch to anaerobic metabolism, which makes lactic acid and other waste products that irritate nerve endings; your brain reads that signal as deep, crushing discomfort—not sharp or stabbing, but dull and relentless.
3. The pain radiates to the jaw, neck, shoulders, or left arm. — why: The heart and those areas share overlapping nerve pathways in the spinal cord (cervical and thoracic segments), so your brain cannot always tell exactly where the pain is coming from—a phenomenon called referred pain.
4. The sympathetic nervous system floods your body with adrenaline and stress hormones. — why: The brainstem senses that the heart—the body's most critical pump—is in danger, so it triggers a full-blown fight-or-flight response to try to boost blood flow and oxygen delivery.

5. You start sweating (diaphoresis), feel nauseated, and have an overwhelming sense of dread or doom. — why: Adrenaline opens sweat glands, slows digestion (causing nausea), and activates the fear centers in your brain; these are not random symptoms—they are the body's all-hands alarm.
6. An ECG shows ST-segment elevation (STEMI) or depression (NSTEMI/starved blood flow (ischemia)), T-wave inversion, or new Q waves. — why: Blood-starved (Ischemic) or dying heart cells cannot conduct electricity normally; the ECG maps the electrical signature of injury, and the pattern tells you which coronary artery is blocked and which wall of the heart is affected.
7. Troponin level rises in the blood within 3 to 6 hours and peaks at 12 to 24 hours. — why: When heart muscle cells die, their membranes break open and spill troponin—a protein normally locked inside—into the bloodstream; the higher the troponin, the more muscle has been damaged.
8. If the blockage is not opened quickly, the affected heart muscle dies (infarcts) and turns into scar tissue that cannot pump. — why: Heart muscle has almost no ability to regenerate; once it is dead, it is replaced by fibrous scar, so the heart becomes weaker, raising the risk of heart failure, arrhythmias, and sudden death—which is why every minute counts in getting the artery open again.

Spot it — what you see, and why

- Pressure, squeezing, or heaviness in the center of the chest (often brought on by activity, relieved by rest): The heart muscle is not getting enough oxygen-rich blood because a coronary artery (one of the heart's own supply pipes) is narrowed or blocked, so the muscle 'cries out' with this tight, heavy feeling.
- Pain or discomfort spreading to the left arm, jaw, neck, back, or stomach: The heart and these areas share the same nerve pathways in the spinal cord, so the brain sometimes 'reads' heart pain as coming from the arm, jaw, or belly—a trick called referred pain.
- Sweating (cold, clammy skin) without obvious reason: The body's stress system (sympathetic nervous system) kicks in when the heart is struggling, triggering sweat glands all over—like an alarm bell that something serious is happening inside.
- Shortness of breath (with or without chest discomfort): When part of the heart muscle is starved for oxygen, it pumps less effectively; blood and fluid back up into the lungs, making each breath feel harder and leaving the person gasping for air.
- Nausea or vomiting: The heart and stomach share some of the same nerve signals, and the body's stress response can upset the stomach, especially during a heart attack; women often notice this symptom more than men do.
- Light-headedness or feeling faint: The weakened heart cannot pump enough blood to the brain, so blood pressure drops and the person feels woozy or close to passing out.

Red flags — act now

- Chest pain with sweating, shortness of breath, or feeling faint—these suggest the heart is in serious trouble right now.
- New irregular heartbeat or very slow or very fast pulse—the heart's electrical system may be failing.
- Blood pressure dropping or signs of shock (cool, clammy skin; confusion; weak pulse)—the heart is not pumping enough blood to keep the body alive.
- Sudden tearing pain to the back—the aorta may be ripping open, a true surgical emergency.
- Active bleeding or very high bleeding risk in someone who needs blood thinners—treatment must be carefully balanced to save the heart without causing dangerous bleeding.

Treat it — in this order

1. 12-lead EKG within 10 minutes of arrival → Shows exactly which heart artery is blocked, how bad the blockage is, and whether the full thickness of the heart wall is dying (ST elevation). This tells you whether the patient needs the cardiac catheterization lab opened immediately or can be managed with medicine and stress testing later.
2. For ST-elevation MI (STEMI): Activate the cath lab for percutaneous coronary intervention (PCI) within 90 minutes, or give fibrinolytic therapy (e.g. tenecteplase, alteplase) within 30 minutes if PCI is not available. Give aspirin 162–325 mg chewed, a P2Y₁₂ inhibitor (clopidogrel 600 mg, ticagrelor 180 mg, or prasugrel 60 mg), and anticoagulation (unfractionated heparin bolus 60 units/kg then 12 units/kg/hr, or enoxaparin 1 mg/kg subcutaneous every 12 hours) → Aspirin stops platelets from clumping. The P2Y₁₂ inhibitor blocks a second platelet pathway so the clot cannot grow. Heparin keeps new clot from forming while the cardiologist threads a catheter up to the blockage and opens it with a balloon and stent. Fibrinolytics dissolve the clot chemically if you cannot reach a cath lab in time. Time is muscle—every minute of delay kills more heart cells.
3. High-intensity statin (atorvastatin 80 mg or rosuvastatin 40 mg) started immediately and continued daily → Stabilizes the cholesterol plaque that ruptured and started the whole cascade. Reduces inflammation in the artery wall so another plaque does not break open in the next days or weeks. Lowers LDL cholesterol over time to slow plaque growth.

4. For aortic dissection: IV beta-blocker (esmolol 500 mcg/kg bolus then 50–200 mcg/kg/min, or labetalol 20 mg IV push then 40–80 mg every 10 minutes) to slow heart rate to 60 bpm, THEN vasodilator (nicardipine 5 mg/hr IV or nitroprusside 0.3 mcg/kg/min IV) to lower systolic blood pressure to 100–120 mmHg. Emergent cardiothoracic surgery consult for Type A; medical management ± endovascular repair for Type B → Every heartbeat hammers the torn aorta and pushes the tear wider. Slowing the heart and dropping the pressure stops the tear from extending and reduces the risk of rupture while you get imaging and prepare for surgery or intervention.
5. For pulmonary embolism (a clot lodging in a lung artery): Anticoagulation with unfractionated heparin (80 units/kg bolus, then 18 units/kg/hr IV), low-molecular-weight heparin (enoxaparin 1 mg/kg subcutaneous every 12 hours), or a direct oral anticoagulant (apixaban 10 mg twice daily × 7 days, then 5 mg twice daily; rivaroxaban 15 mg twice daily × 21 days, then 20 mg daily). If massive PE with shock or severe right heart strain, give systemic fibrinolysis (alteplase 100 mg IV over 2 hours) or catheter-directed therapy → Heparin and oral anticoagulants stop the clot from growing while the body's own enzymes slowly dissolve it over days to weeks. Fibrinolytics or catheter suction dissolve or remove the clot faster when the right heart is failing under the pressure load and the patient is about to crash.

Your top priorities at the bedside

1. Treat every chest-pain arrival as an emergency: ECG in hand within 10 minutes. — why: The ECG is the one test that finds a STEMI, and the clock starts at the door.
2. Never leave a 'normal-looking' chest-pain patient in the waiting room. — why: Women, older adults, and people with diabetes have quiet heart attacks — these are the ones most often missed.
3. Take blood pressure in both arms if dissection is possible. — why: A big pressure difference between arms is a classic dissection clue.