Pulmonary Embolism

Pulmonary Embolism (a clot lodging in a lung artery) — a clot is blocking blood flow through the lungs.

THE ONE-LINE TRUTH

A blood clot has traveled from somewhere else in the body and is now stuck in the pulmonary arteries (the blood vessels that feed the lungs).

The chain — how it unfolds

1. A clot blocks one or more pulmonary arteries. — why: The clot (usually from a leg or pelvic vein) travels to the lungs and wedges into a branch that is too narrow to pass, cutting off blood flow to that lung tissue.
2. Pulmonary vascular resistance spikes. — why: The physical clot plus reflex squeezing of downstream lung vessels (from serotonin and thromboxane released by the clot) creates a sudden high-pressure wall the right ventricle must push against.
3. The right ventricle swells and pumps weakly. — why: The thin-walled right ventricle has little muscle reserve, so when afterload skyrockets, the chamber overstretches, loses squeeze power, and begins to fail.
4. The swollen right ventricle bulges into the left ventricle's space. — why: The heart sits in a stiff sac (pericardium) with no room to expand outward, so the bloated right side pushes through the interventricular septum and crushes the left ventricle.
5. Cardiac output (blood pumped to the body) drops and blood pressure falls. — why: The squashed left ventricle cannot fill properly, so even though it tries to squeeze, very little blood gets out to the rest of the body each beat.
6. Heart rate shoots up (tachycardia). — why: Baroreceptors (pressure sensors) in the aorta and carotid arteries detect the drop in blood pressure and signal the brainstem to flood the body with epinephrine, making the sinoatrial node (heart's pacemaker) fire faster to try to keep total flow up.
7. Low oxygen in the blood (Hypoxemia) (low oxygen in the blood) develops. — why: Blocked lung vessels cannot carry blood past the air sacs to pick up oxygen, and the failing right heart worsens ventilation-blood flow (perfusion) mismatch (some lung gets air but no blood, some gets blood but collapses), so oxygen levels plummet.

8. Dead space (lung areas with air but no blood flow) shoots up. — why: Air reaches alveoli (air sacs) downstream from the clot, but because no blood flows there, that ventilation is completely wasted — oxygen cannot jump into blood that is not there.

Spot it — what you see, and why

- Sudden shortness of breath, often with sharp chest pain when breathing in (pleuritic chest pain): A blood clot gets stuck in a lung artery and blocks blood flow to part of the lung. That lung tissue still tries to work but can't get the blood it needs, so breathing feels hard and painful — like trying to fill a tire with a kinked hose.
- Fast heart rate (tachycardia): The heart beats faster to try to push blood past the clot and keep oxygen moving to the body. It's working overtime, like a pump speeding up when a pipe is blocked downstream.
- Fast breathing (tachypnea): The lungs sense low oxygen and try to catch up by breathing faster. Your brain's breathing control center (the medulla) tells you to take more breaths per minute, trying to make up for the blocked lung section.
- Low oxygen in the blood (hypoxemia): The clot blocks blood from picking up oxygen in that part of the lung. Even though you're breathing, not enough oxygen gets into the bloodstream — like air going into a balloon that has a sealed-off section.
- Coughing up blood (hemoptysis): The blocked lung tissue can bleed because it isn't getting enough oxygen (a small 'lung attack' called pulmonary tissue death from blocked blood flow (infarction)). Blood leaks into the air sacs and gets coughed up.
- Swelling or pain in one leg (often the calf or thigh): The clot usually starts in a deep vein in the leg (deep vein clot formation (thrombosis)). Before it breaks off and travels to the lung, it can cause swelling, warmth, and aching in that leg.

Red flags — act now

- Sudden collapse, very low blood pressure, or loss of consciousness (signs of massive PE that can stop the heart)
- Coughing up blood (may mean part of the lung is dying from lack of oxygen)
- Chest pain with severe shortness of breath that gets worse quickly (clot may be large or growing)
- Heart strain markers on tests (high troponin or BNP, right ventricle dysfunction on echo) even without low blood pressure (submassive PE needs close monitoring and may need clot-busting treatment)

Treat it — in this order

1. Anticoagulation with unfractionated heparin IV bolus and infusion, or low-molecular-weight heparin (enoxaparin) subcutaneous, or fondaparinux subcutaneous — start immediately upon clinical suspicion before imaging if delay is expected and bleeding risk is acceptable → Stops the clot from getting bigger and prevents new clots from forming. Buys time for the body's own fibrinolytic system (natural clot dissolvers) to slowly break down the a traveling clot (embolus) over days to weeks.
2. Systemic thrombolysis (alteplase 100 mg IV over 2 hours, or alternative regimen per protocol) for massive pulmonary embolism (a clot lodging in a lung artery) with sustained low blood pressure (hypotension) (systolic BP < 90 mmHg for ≥ 15 minutes) or cardiac arrest → Actively dissolves the clot lodged in the pulmonary arteries within hours, dropping pulmonary vascular resistance so the right ventricle is no longer crushed by back-pressure. Restores cardiac output and can be life-saving.
3. Supplemental oxygen by nasal cannula or non-rebreather mask to target SpO2 ≥ 90% → Increases the oxygen content of blood flowing through the working parts of the lung, so tissues get more oxygen even though some lung areas are blocked and cannot participate in gas exchange.
4. Catheter-directed thrombolysis or mechanical thrombectomy (suction embolectomy, rheolytic fragmentation) for submassive pulmonary embolism (a clot lodging in a lung artery) with right ventricular dysfunction and elevated troponin or BNP but no shock → Delivers clot-busting drug or physically removes clot directly at the site inside the pulmonary artery, lowering right-heart strain faster than anticoagulation alone while using lower thrombolytic doses than systemic therapy — this lowers systemic bleeding risk compared to full-dose IV alteplase.
5. Transition to oral anticoagulation (apixaban, rivaroxaban, edoxaban, dabigatran, or warfarin) and continue for at least 3 months after a provoked pulmonary embolism (a clot lodging in a lung artery) (with a clear temporary trigger like surgery or immobilization), or indefinitely for unprovoked pulmonary embolism (no clear trigger) or recurrent events, weighing bleeding risk → Prevents recurrent venous thromboembolism and protects against another clot reaching the lungs. Lowers the 1-year recurrence rate from about 10% down to 1–2%.

Your top priorities at the bedside

1. Apply oxygen by nasal cannula or non-rebreather mask to keep SpO2 above 90%. — why: The clot blocks blood flow to lung tissue, so oxygen cannot transfer into the bloodstream. Supplemental oxygen increases the amount available for gas exchange in the unblocked lung areas and prevents tissue low oxygen (hypoxia) (low oxygen in organs).

2. Establish large-bore IV access (18-gauge or larger) and prepare for immediate anticoagulation with IV unfractionated heparin or subcutaneous LMWH (low-molecular-weight heparin). — why: Anticoagulation stops the clot from growing larger and prevents new clots from forming. Starting treatment within the first hour reduces mortality (death rate) by preventing clot extension and recurrent traveling clots (emboli) (new clots breaking off).
3. Place patient on continuous cardiac monitoring and measure vital signs every 15 minutes if unstable or every 1 hour if stable. — why: PE can cause sudden right ventricle (RV) failure because the clot increases pressure in the pulmonary arteries. A fast heart rate (Tachycardia) (heart rate over 100), low blood pressure (hypotension) (systolic BP under 90), or new arrhythmias (irregular heartbeats) signal decompensation (worsening) requiring thrombolysis or ICU transfer.