

DVT and PE

DVT and PE are two stops on the same dangerous road: a clot forms in a deep vein, then breaks off and blocks the lung—anticoagulate immediately and risk-stratify to prevent recurrence and death.

THE ONE-LINE TRUTH

A blood clot forms in a deep vein (usually the leg), and if it breaks loose and travels to the lung, it becomes a PE (pulmonary a traveling clot lodging in a vessel (embolism))—both are part of the same disease process called VTE (venous thromboembolism).

The chain — how it unfolds

1. Virchow's triad is triggered: blood flow slows, the vein wall is injured, or blood gets stickier. — why: Long immobility (flight, surgery, bed rest), trauma, surgery, pregnancy, birth control pills, cancer, smoking, or inherited clotting disorders tip the balance so platelets and clotting proteins (fibrin) stick together instead of staying dissolved.
2. A solid clot (thrombus) forms inside a deep vein, usually in the calf or thigh. — why: Platelets clump on the damaged or slow-flow area, fibrin threads weave through them like a net, and red blood cells get trapped—forming a rubbery, solid plug that sticks to the vein wall and grows bigger over hours to days.
3. The clot blocks blood from draining out of the leg, causing unilateral swelling, pain, warmth, and redness. — why: Blood coming up from the foot hits the blockage and backs up, so pressure rises in the small veins and capillaries below the clot—fluid leaks out into the tissue (edema), the leg swells, feels heavy and tight, and may turn red or feel warm because blood is pooling and inflammatory chemicals are released.
4. A piece of the clot breaks loose (embolizes) and floats through the bloodstream. — why: Proximal clots (thigh or pelvis) are more likely to break off because blood flow is faster there; the fragment travels up the inferior vena cava, through the right atrium and right ventricle, and is pumped into the pulmonary arteries.
5. The clot gets stuck in a pulmonary artery branch—this is a pulmonary embolism (a clot lodging in a lung artery) (PE). — why: Pulmonary arteries branch and get narrower and narrower as they spread through the lungs; the clot wedges into a branch it cannot fit through, blocking blood flow to that section of lung tissue.

6. The blocked lung tissue cannot pick up oxygen (ventilation-blood flow (perfusion) mismatch), causing sudden shortness of breath, low oxygen (hypoxemia), fast breathing (tachypnea), and sometimes sharp chest pain (pleuritic pain). — why: Air still reaches the alveoli (tiny air sacs), but no blood flows past them to pick up the oxygen—so that part of the lung is wasted. The rest of the lung tries to compensate, and the person breathes faster and harder, but oxygen levels drop because too much lung is out of service. If the blocked lung tissue dies (pulmonary tissue death from blocked blood flow (infarction)), it releases inflammatory chemicals that irritate the pleura (lung lining), causing sharp chest pain that gets worse with deep breathing.
7. The right ventricle has to pump harder against the blocked pulmonary arteries, causing right heart strain, fast heart rate (tachycardia), elevated jugular venous pressure (JVP), and sometimes a new heart murmur or gallop. — why: The blockage raises pressure in the pulmonary arteries (pulmonary high blood pressure (hypertension)); the right ventricle, which is thin-walled and used to pumping against low pressure, suddenly faces high resistance—like trying to blow through a pinched straw. It dilates, struggles, and may fail. Troponin (a heart muscle injury marker) can rise, ECG may show right heart strain (S1Q3T3 pattern, right bundle branch block, or T-wave inversions in V1–V4), and the person's heart races to try to keep oxygen delivery going.
8. In massive PE, the right ventricle fails completely, causing obstructive shock: blood pressure drops (hypotension), the patient feels dizzy or faints (syncope), and may go into cardiac arrest. — why: When more than 50% of the pulmonary circulation is blocked, the right heart cannot pump enough blood forward into the lungs, so the left heart has nothing to pump out to the body—cardiac output crashes, blood pressure falls, organs (especially the brain) do not get enough oxygen, and the patient can die within minutes without emergency treatment (systemic thrombolysis, catheter embolectomy, or surgical embolectomy).

Spot it — what you see, and why

- Leg swelling (usually one leg): A blood clot in a deep vein blocks blood from flowing back to the heart, so fluid backs up and the leg gets puffy and tight.
- Leg pain or tenderness (often feels like a cramp in the calf): The clot irritates the vein wall and stretches it, and the backed-up blood presses on tissues, which triggers pain signals.
- Warmth and redness over the swollen area: The body sends extra blood and immune cells to the clot site to try to heal it, which makes the skin feel hot and look red.
- Sudden shortness of breath: If a piece of the clot breaks off and travels to the lungs (a PE), it blocks blood vessels in the lung so oxygen cannot get into the blood as easily.

- Sharp chest pain that gets worse when you breathe in deeply or cough: The blocked lung blood vessels cause the lung tissue to swell and the membrane around the lung (pleura) to get irritated, which hurts when the lung expands.
- Fast heart rate (tachycardia): The heart pumps faster to try to make up for the fact that less blood is getting oxygen in the blocked lung.

Red flags — act now

- Sudden trouble breathing or very fast breathing
- Chest pain that feels sharp or stabbing, especially with a deep breath
- Coughing up blood
- Feeling faint, dizzy, or passing out
- Fast or irregular heartbeat with sweating or confusion

Treat it — in this order

1. Start anticoagulation immediately: DOAC (apixaban 10 mg twice daily × 7 days then 5 mg twice daily, or rivaroxaban 15 mg twice daily × 21 days then 20 mg once daily) OR low-molecular-weight heparin (enoxaparin 1 mg/kg twice daily) bridged to warfarin (INR goal 2-3) → Anticoagulation blocks clotting proteins (factor Xa or thrombin) so the clot stops growing. Your body's own enzymes slowly break down the clot over weeks to months. This fixes the upstream problem—stops the blood from clotting more while the vein or lung heals.
2. Use low-molecular-weight heparin (LMWH, enoxaparin) instead of DOAC if the patient is pregnant, has active cancer, or has severe kidney disease (CrCl under 30) → LMWH is safe in pregnancy (DOACs cross the placenta and harm the baby). It also works better in cancer patients because cancer makes blood clot more aggressively. LMWH does not need dose adjustment by kidney function as much as some DOACs. Still blocks factor Xa to stop clot growth.
3. Give thrombolysis (tissue plasminogen activator, tPA 100 mg IV over 2 hours, or catheter-directed tPA) if massive PE with shock (systolic BP under 90) or submassive PE with right heart strain on echo → tPA activates plasminogen, which dissolves the clot actively right now instead of waiting weeks. This unclogs the lung vessels fast so blood can flow and oxygen can get through. Buys time when the patient is about to die from right heart failure.
4. Place an inferior vena cava (IVC) filter if the patient has a new clot but CANNOT take anticoagulation (active bleeding, recent brain surgery, severe bleeding disorder) → A metal basket sits in the big vein below the heart and catches clots before they reach the lung. Prevents PE when anticoagulation is too dangerous.

5. Elevate the swollen leg above heart level, start walking as soon as it's safe (early mobilization), and fit the patient for compression stockings (30–40 mmHg) once swelling is controlled → Elevation and muscle movement (walking) help veins pump blood back to the heart against gravity, so fluid doesn't pool. Compression stockings squeeze the leg so veins can't stretch and leak fluid. Together they reduce pain and swelling.

Your top priorities at the bedside

1. Check oxygen saturation with pulse oximeter and apply oxygen to keep SpO₂ above 90 percent if the patient has any shortness of breath or chest pain — why: PE blocks blood flow to part of the lung so that area cannot pick up oxygen; low oxygen levels mean the clot is large or the patient is decompensating (getting worse)
2. Start continuous pulse oximetry and cardiac monitoring to watch for arrhythmias (irregular heartbeat) and a fast heart rate (tachycardia) (fast heart rate) — why: A large PE strains the right side of the heart and can cause dangerous rhythms or right heart failure; a fast heart rate (tachycardia) over 100 is an early warning sign
3. Keep the patient on bed rest with the affected leg elevated above the level of the heart — why: Elevation uses gravity to reduce swelling and pain; bed rest prevents the clot from breaking loose and traveling to the lungs, though this is controversial and short-term only