

DVT · Deep Vein Thrombosis

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THE STORY IN ONE SENTENCE

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“A clot in a deep leg vein (Deep vein thrombosis) is a clot stuck in a deep vein. The urgent question is: did a piece break off and travel to the lungs?”

Normal anatomy — the body's job

Deep veins carry blood from your legs back up to your heart. Your calf muscles squeeze the veins like a pump with every step. One-way valves inside the veins catch the blood and stop it from falling backward. The smooth inner lining of the vein wall keeps blood flowing without clotting. This system runs quietly all day, moving blood uphill against gravity.

BODY'S JOB

The deep veins are the main highway for blood returning from your legs to your heart and lungs. When you walk, your calf muscles contract and squeeze the veins, pushing blood upward. Tiny one-way valves open to let blood through, then snap shut so it cannot slide back down. The endothelium—the smooth cell layer lining the inside of the vein—releases chemicals that keep blood liquid and prevent clots from forming. Blood also keeps moving steadily, which stops clotting factors from piling up in one spot. Together, these three things—steady flow, a healthy vein wall, and balanced clotting chemistry—keep your blood moving safely home.

1. Your heart pumps oxygen-rich blood out through arteries to your legs and feet.
2. Tiny capillaries deliver oxygen and nutrients to every muscle and skin cell.
3. Used blood, now low in oxygen and carrying waste, collects in small veins.
4. These small veins merge into larger deep veins that run alongside your leg bones, protected by muscle.

5. When you walk or flex your foot, your calf muscles squeeze these deep veins like a tube of toothpaste, pushing blood upward.
6. One-way valves inside the veins open as blood flows up, then close immediately so blood cannot fall back down.
7. The smooth endothelial lining keeps blood from sticking or clotting along the vein wall.
8. Blood travels up through the femoral vein in your thigh, through the iliac vein in your pelvis, and into the inferior vena cava—the body's main vein—returning to the right side of your heart.
9. Your heart then pumps that blood to your lungs to pick up fresh oxygen, and the cycle starts again.

What breaks

A clot forms in a deep vein when one or more parts of the normal system break down. Blood may sit still too long, the vein wall may get damaged, or the blood itself may clot too easily. Any of these—or a combination—tips the balance toward clotting instead of flowing.

CLINICAL TERM

Virchow's triad describes the three factors that cause DVT: stasis (slow or stopped blood flow), endothelial injury (damage to the vein lining), and hypercoagulability (blood that clots too easily). Stasis happens during long plane or car trips, bedrest after surgery, paralysis, or immobility from illness. Endothelial injury occurs from central venous catheters, trauma, surgery (especially orthopedic), or direct vein damage.

Hypercoagulability is caused by cancer (especially pancreatic, lung, or blood cancers), estrogen-containing contraceptives or hormone therapy, pregnancy and the postpartum period, inherited clotting disorders (factor V Leiden, prothrombin G20210A mutation, protein C or S deficiency, antithrombin deficiency), antiphospholipid syndrome, nephrotic syndrome, inflammatory diseases, smoking, and obesity. When one or more of these factors is present, clotting factors accumulate faster than natural anticoagulants can control them, and a blood clot (thrombus) (clot) forms and anchors to the vein wall—most commonly in the calf, popliteal, femoral, or iliac veins. The clot grows in both directions, blocking flow and triggering inflammation.

How the body compensates

Your body tries to route blood around the clot through smaller backup veins, but this causes swelling, pain, and pressure. Vital signs may stay normal at first because the body is working

hard to cope—but that hides how close the patient is to a dangerous tipping point, especially pulmonary embolism (a clot lodging in a lung artery).

1. Blood backs up behind the clot, raising pressure in the vein and forcing fluid out through the vein walls into the surrounding tissue—this is why the leg swells.
2. Smaller collateral veins try to carry the extra blood around the blockage, but they are too narrow to handle the full load—so pressure stays high and swelling continues.
3. The body releases inflammatory chemicals at the clot site, causing warmth, redness, and pain—this is not infection, it is your immune system reacting to the clot as if it were an injury.
4. The clot may grow longer and wider if anticoagulation is not started promptly, extending upstream and downstream and increasing the risk that a piece will break off.
5. Vital signs—blood pressure, heart rate, oxygen level—often stay normal while the clot is confined to the leg, because the heart and lungs are not yet affected. This stability is deceptive and can lead clinicians to underestimate risk.
6. The tipping point to watch for is pulmonary embolism (a clot lodging in a lung artery): a clot fragment breaks loose, travels through the right heart, and lodges in the lung arteries—causing sudden shortness of breath, chest pain, rapid heart rate, low oxygen, and potential cardiovascular collapse.
7. In rare cases (phlegmasia cerulea dolens), the clot is so massive it blocks nearly all venous outflow from the leg. Pressure inside the leg rises so high it compresses the arteries, cutting off oxygen delivery—the leg turns blue-purple, becomes exquisitely painful, and tissue begins to die. This is a surgical emergency requiring thrombectomy or fasciotomy.
8. Watch every DVT patient for signs of PE: new shortness of breath, chest pain (especially sharp pain with breathing), rapid breathing, rapid heart rate, lightheadedness, fainting, coughing up blood, or sudden anxiety. These signs mean the clot has traveled and the window to prevent disaster is narrowing fast.
9. Trend over time, not just a single snapshot. A patient whose leg swelling is increasing, whose pain is worsening despite analgesia, or who develops any respiratory or cardiovascular symptom is moving toward the cliff edge—not stable.

Domino effect

1. A clot forms and anchors inside a deep vein, usually in the calf or thigh. — One or more of Virchow's triad—stasis, endothelial injury, or hypercoagulability—tips the balance toward clotting. Clotting proteins and platelets stick together and attach to the vein wall, forming a solid plug that blocks blood flow.

2. Blood backs up behind the clot, unable to return to the heart. — The vein is like a one-way road with a traffic jam. Blood keeps arriving from the leg tissues but cannot leave, so pressure rises inside the vein and fluid leaks out into surrounding tissue—this is swelling (edema) (swelling).
3. The leg swells, feels heavy and tight, and hurts—especially the calf or thigh. — Fluid trapped in the tissue stretches the skin and presses on nerves. The swelling is usually in one leg only, because the clot blocks one side. Walking or standing makes it worse because gravity adds more pressure.
4. The skin over the clot feels warm and may look red or pale. — Inflammation at the clot site releases heat and chemicals that dilate nearby blood vessels, causing warmth and sometimes redness. In severe cases, if blood flow is very poor, the leg may look pale or dusky instead.
5. The clot grows longer, extending upstream and downstream inside the vein. — If anticoagulation is not started, clotting continues. The clot propagates like a slow-growing icicle, layer by layer, because stasis and inflammation persist around it. Larger clots are more likely to break off.
6. A piece of the clot breaks loose and travels through the bloodstream as an a traveling clot (embolus). — Blood flow tugs on the clot. If a fragment detaches, it floats freely through the vein, passes through the right atrium and right ventricle of the heart, and enters the pulmonary arteries feeding the lungs.
7. The a traveling clot (embolus) lodges in a lung artery, causing pulmonary embolism (a clot lodging in a lung artery) (PE). — The pulmonary arteries branch into smaller and smaller vessels. The clot travels until it reaches a branch too narrow to pass, then gets stuck—blocking blood flow to part of the lung. That lung tissue cannot exchange oxygen, and the right heart strains to push blood past the blockage.
8. The patient develops sudden shortness of breath, chest pain, rapid heart rate, low oxygen, and may collapse. — Blocked lung arteries mean less oxygen reaches the blood, so the body gasps for air and the heart races trying to compensate. A large PE can cause the right ventricle to fail, dropping blood pressure and leading to shock or cardiac arrest. This is the life-threatening endpoint of untreated DVT.
9. In rare cases, phlegmasia cerulea dolens develops: the entire leg turns blue-purple, swells massively, and loses pulses. — When a very large clot blocks nearly all venous drainage from the leg, pressure inside the leg skyrockets. This pressure compresses the arteries, shutting off oxygen delivery—the leg becomes blood-starved (ischemic) (starved of blood). Without emergency treatment (thrombectomy, thrombolysis, or fasciotomy), the leg tissue dies and the patient may lose the limb or develop gangrene and sepsis.

Symptoms · what the patient feels, and why

- Swelling in one calf or thigh: A clot (a solid plug made of blood) blocks the deep vein so blood cannot flow back up toward the heart. The blood gets stuck below the clot and pushes fluid out into the tissue, making the leg puff up.
- Pain or tenderness in the calf or thigh: The trapped blood stretches the vein wall and irritates the tissue around it. When you press or walk, you squeeze the swollen area and feel the soreness.
- Warmth over the swollen area: The clot triggers inflammation (your body's alarm response). Extra blood flow rushes to the area to start repair, and that extra blood makes the skin feel warmer than the other leg.
- Redness or darker color of the skin on the leg: Blood pools and cannot drain properly. The extra blood under the skin changes its color—lighter skin may look red or purple, and darker skin may look deeper brown or ashen.
- Swollen veins you can see or feel just under the skin: The deep vein is blocked, so blood tries to find another route home. It backs up into the smaller surface veins, making them bulge out like overfilled garden hoses.
- The leg feels heavy or tight: Fluid collects in the tissue because the vein cannot carry it away. The extra weight and pressure make the leg feel full and sluggish.
- Shortness of breath or chest pain: A piece of the clot can break off and travel through the bloodstream to the lungs, blocking a lung artery (this is called a pulmonary embolism (a clot lodging in a lung artery)). The blocked artery starves part of the lung of oxygen, so breathing becomes hard and the chest hurts.
- Fast heart rate or feeling dizzy: If a large piece of clot reaches the lungs, the heart has to pump harder to push blood past the blockage. Not enough oxygen reaches the brain, so you feel light-headed or your heart races to try to catch up.

Focused exam

- One leg is swollen compared to the other (unilateral leg swelling): A clot in the deep vein blocks blood from draining out of the leg, so fluid backs up and the leg swells. When you see one leg bigger than the other, you know something is blocking the return path on that side.
- The leg feels warm and hurts when you press on the calf or thigh (tenderness and warmth): The clot irritates the vein wall, causing inflammation. Inflammation brings extra blood and immune cells, which heat the tissue and make it sore. This tells you the clot is active and the body is reacting to it.

- When you press your thumb into the swollen leg, the dent stays for a few seconds (pitting swelling (edema)): Fluid trapped in tissue does not spring back right away. This shows the swelling is from backed-up fluid, not fat or muscle. It confirms that blood is not draining properly.
- The calf or thigh is more than 3 cm bigger around than the other leg (measured at the same spot on both sides): Real swelling from a clot is big enough to measure. Measuring both legs at the same level (for example, 10 cm below the knee) proves the difference is real and not just how you are looking at it.
- Skin on the swollen leg looks red, or in severe cases blue or purple: Poor drainage makes blood sit still in the tissue, which turns the skin red. A blue or purple color means blood is barely moving at all—this is a sign of severe blockage and the leg is in danger.
- You can feel a firm, rope-like cord under the skin along the path of a vein: Sometimes the clotted vein is close enough to the surface that you can feel it as a hard string. This is a direct sign that the vein is full of clot.

Diagnostics · every test answers a question

- D-dimer blood test → Is a blood clot unlikely enough that we can safely skip imaging?
- Compression duplex ultrasound of the leg veins → Is there a clot in the deep veins, and if so, where is it?
- Wells score for DVT (clinical prediction rule) → How likely is DVT before we do any tests?
- Complete blood count (CBC) with platelet count → Is the platelet count safe before starting blood thinners, and is there anemia from hidden bleeding?
- Creatinine and estimated glomerular filtration rate (eGFR) → Are the kidneys working well enough to clear the blood thinner we plan to use?
- PT/INR and aPTT (if warfarin or heparin is planned) → Is the blood clotting time in a safe range for the anticoagulant we are using?

Treatment logic

- Direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban, started immediately → Blocks factor Xa in the clotting cascade, so the clot cannot grow larger and new clots cannot form. Your body's natural cleanup system (fibrinolysis) slowly breaks down the clot over weeks to months. Apixaban is 10 mg twice daily for 7 days, then 5 mg twice daily; rivaroxaban is 15 mg twice daily for 21 days, then 20 mg once daily.
- Compression stockings (20–30 mmHg) worn during the day and leg elevation above heart level when resting → Squeezes the leg veins to push blood upward against gravity, which reduces swelling and prevents blood from pooling. This lowers the chance of post-thrombotic syndrome (long-term pain and swelling after DVT).

- Low-molecular-weight heparin (LMWH, such as enoxaparin 1 mg/kg subcutaneous twice daily) bridging to warfarin in patients with cancer or severe kidney disease (eGFR < 30) → LMWH works immediately by boosting antithrombin, which shuts down factor Xa and thrombin. Warfarin takes 3–5 days to reach full effect, so LMWH keeps the clot from growing while you wait. In cancer patients, LMWH alone (without warfarin) is often continued for months because it works better than warfarin in active cancer.
- Extended anticoagulation (at least 3 months; longer or lifelong if unprovoked or recurrent DVT) → Keeps the blood from clotting again after the first clot is gone. A provoked DVT (clear trigger like surgery or broken leg) can often stop after 3 months. An unprovoked DVT (no clear reason) or a second DVT means higher risk of another clot, so treatment continues as long as bleeding risk stays low.
- Early walking and movement (ambulation) as symptoms allow, NOT strict bed rest → Moving the leg muscles pumps blood out of the veins and prevents more clot from forming due to stasis (still blood). Old teaching said stay in bed, but we now know walking is safe once anticoagulation is started and actually lowers clot risk.
- Catheter-directed thrombolysis (clot-busting drug delivered by a thin tube threaded into the clot) in severe cases like phlegmasia cerulea dolens (blue, painful, swollen leg with no blood flow) → Injects tissue plasminogen activator (tPA) directly into the clot to dissolve it much faster than anticoagulation alone. This can save the leg when the clot is so big it stops all blood flow and threatens tissue death.
- Inferior vena cava (IVC) filter placement (a small metal trap inserted into the large vein in the abdomen) only when anticoagulation is absolutely contraindicated (for example, active brain bleeding) → Catches clot pieces that break off from the leg before they reach the lungs, preventing pulmonary embolism (a clot lodging in a lung artery). It does NOT treat the DVT itself.

Medications

Apixaban (Eliquis) or Rivaroxaban (Xarelto) · Direct oral anticoagulant (DOAC)

These drugs block Factor Xa, a protein in your blood that helps clots form. By stopping Factor Xa, the medicine keeps your blood from making new clots on top of the one already stuck in your leg vein, so your body has time to slowly break down the clot on its own.

1. Factor Xa normally turns prothrombin (an inactive protein) into thrombin (the active clot-builder).
2. The DOAC locks onto Factor Xa and stops it from working.
3. Without Factor Xa, much less thrombin gets made.
4. With less thrombin, fibrin threads (the sticky net that holds a clot together) don't form as easily.

5. Your existing clot stops growing, and over weeks your body's cleanup crew (enzymes) quietly dissolves it.

SAFETY WATCH

Bleeding is the main risk—any cut or bump can bleed longer or harder than usual. · Watch for unusual bruises, blood in urine or stool, coughing up blood, or severe headache. · Tell every doctor and dentist you take a blood thinner before any procedure. · Do NOT stop suddenly without talking to your doctor—stopping can let a new clot form fast.

Enoxaparin (Lovenox) or Dalteparin (Fragmin) · Low-molecular-weight heparin (LMWH)

These are injectable medicines that boost the power of antithrombin, a natural brake on clotting in your blood. By making antithrombin work much faster, LMWH stops Factor Xa and thrombin so new clot cannot grow.

1. You inject the medicine under your skin (usually in your belly).
2. LMWH finds antithrombin in your blood and makes it 1,000 times more active.
3. Supercharged antithrombin then grabs and disables Factor Xa and some thrombin.
4. Without these clotting proteins working, your blood stays thinner and new clot stops forming.
5. Your body slowly clears away the existing clot over time.

SAFETY WATCH

Bleeding is the top risk—watch for unusual bruising, blood in urine or stool, or very heavy periods. · You must inject it once or twice a day; missing doses lets clotting restart. · Tell your doctor if you have kidney problems—the drug is cleared by your kidneys and can build up. · Avoid aspirin or ibuprofen unless your doctor says it is safe, because they also thin blood. · Rare risk of low platelet count (HIT); report any new rash or unusual clotting.

Warfarin (Coumadin) · Vitamin K antagonist

Warfarin slows down your liver's ability to make four clotting proteins (Factors II, VII, IX, and X) by blocking vitamin K, which those proteins need to work. With fewer working clotting factors, your blood stays thinner.

1. You swallow a warfarin pill every day.
2. The drug blocks an enzyme (VKORC1) that recycles vitamin K in your liver.
3. Without enough active vitamin K, your liver makes clotting factors, but they come out incomplete and cannot do their job.

4. Over 3–5 days, the old working clotting factors break down naturally and are replaced by these weak ones.
5. Your blood becomes thinner, so new clots cannot form easily.

SAFETY WATCH

Bleeding is the biggest danger—cuts, falls, and even hard coughing can cause serious bleeding. · You need regular blood tests (INR) to make sure your dose is right—too much causes bleeding, too little lets clots form. · Many foods (especially leafy greens high in vitamin K) and other medicines change how warfarin works; keep your diet steady and tell every doctor what you take. · Avoid alcohol in large amounts and contact sports. · If you become pregnant or plan to become pregnant, tell your doctor immediately—warfarin can harm a growing baby.

Alteplase (tPA) · Thrombolytic (clot-buster)

Alteplase is an enzyme that directly dissolves fibrin, the sticky net holding a blood clot together. It is delivered through a thin tube (catheter) placed right into the clot in your vein, so it breaks down the clot from the inside out.

1. A radiologist threads a catheter through a small nick in your skin into the vein where the clot sits.
2. Alteplase is dripped slowly through the catheter directly onto the clot.
3. The drug turns plasminogen (an inactive protein stuck in the clot) into plasmin (an active scissor enzyme).
4. Plasmin cuts apart the fibrin mesh that holds the clot together.
5. The clot dissolves quickly—often within hours to a day—and blood flow returns.

SAFETY WATCH

High bleeding risk—this is only used in severe cases where the leg is badly swollen, painful, or at risk of tissue death. · Cannot be used if you had recent surgery, stroke, or any active bleeding. · Requires close monitoring in a hospital, often in an ICU. · Very rarely, pieces of clot can break loose and travel to the lungs during treatment.

Red flags

- Sudden shortness of breath, chest pain, or coughing up blood — the clot may have moved to the lungs (pulmonary a traveling clot lodging in a vessel (embolism)).
- Swelling in both legs at once, or one leg that turns very pale and cold — the main vein near the belly may be blocked, or a large artery may be in trouble.

- Calling one-sided leg swelling 'a skin infection' in someone who just had surgery, was on a long trip, or cannot move much — DVT can look like an infection but needs different treatment fast.

Nursing priorities

1. Keep the patient on bed rest with the affected leg elevated above heart level until the doctor confirms anticoagulation (blood thinner) is started and working. — Movement can shake the clot loose and send it to the lungs as a pulmonary embolism (a clot lodging in a lung artery); gravity and rest help blood drain from the leg and reduce pressure on the clot until medicine makes it stable.
2. Check oxygen saturation, respiratory rate, and heart rate every 2 to 4 hours and report oxygen below 94 percent, breathing faster than 20, chest pain, or sudden shortness of breath immediately. — A pulmonary embolism (a clot lodging in a lung artery) (clot in the lung) can happen suddenly when part of the leg clot breaks off, and low oxygen or fast breathing are the earliest warning signs that need emergency treatment.
3. Measure the circumference of both calves and thighs at the same spot (mark with a pen) every shift and document the difference in centimeters. — Shrinking swelling tells you the blood thinner is working and the clot is not growing; worsening swelling means the clot may be getting bigger or treatment is failing.
4. Start the first dose of anticoagulant (direct oral anticoagulant or low-molecular-weight heparin injection) within 2 hours of the doctor's order, and teach the patient never to skip doses. — Blood thinners do not dissolve the clot, but they stop it from growing larger while the body slowly breaks it down; any delay or missed dose lets the clot expand or break off into the lungs.
5. Assess the affected leg for new numbness, tingling, pale color, loss of pulse, or severe pain not relieved by medicine, and notify the doctor stat if any appear. — These signs mean compartment syndrome (pressure inside the leg muscles cuts off blood flow) or phlegmasia cerulea dolens (the entire deep vein system is blocked), both surgical emergencies that can lead to losing the leg.
6. Apply graduated compression stockings (20 to 30 millimeters of mercury pressure) from toes to knee once daily ambulation (walking) is approved, and remove them at bedtime. — Compression squeezes blood up and out of the leg veins, preventing post-thrombotic syndrome (chronic swelling, pain, and skin damage) that happens in up to 40 percent of DVT patients within two years.

7. Check for signs of bleeding—gums, urine color, stool color, bruising, nosebleeds—every shift and teach the patient to report blood in urine or black tarry stools immediately. — Anticoagulants prevent clotting everywhere in the body, so any small injury or ulcer can bleed heavily; catching bleeding early allows dose adjustment before life-threatening bleeding (hemorrhage) happens.
8. Encourage ankle pumps (pointing toes up and down 10 times every hour) and deep breathing exercises even while on bed rest, and help the patient walk short distances as soon as the doctor allows. — Muscle contraction squeezes veins and pushes blood back toward the heart, reducing stasis (still blood) that causes new clots; early safe movement also prevents pneumonia and muscle wasting from prolonged bed rest.

Sources

- American College of Chest Physicians (CHEST) Antithrombotic Guidelines
- American Society of Hematology (ASH) VTE Guidelines 2020
- ACCP Evidence-Based Clinical Practice Guidelines (9th & 10th editions)
- CDC Venous Thromboembolism Resources

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