

VTE in pregnancy

VTE in pregnancy means give low-molecular-weight heparin (LMWH, a daily shot that safely thins the blood). Do NOT give warfarin (pills that cross the placenta and harm the baby) or DOACs (newer pills that lack pregnancy safety data). Keep giving LMWH for at least 6 weeks after delivery — the clot risk stays high until the body fully resets.

THE ONE-LINE TRUTH

Pregnancy makes blood clot more easily, so clots can form in leg veins and break off into the lungs.

The chain — how it unfolds

1. Pregnancy hormones and slowed blood flow make the blood stickier, so clotting proteins and platelets clump together inside a deep vein — usually in the left leg (because the left iliac vein is squeezed harder by the uterus and the right iliac artery crossing over it). — why: Estrogen and progesterone tell the liver to crank out more clotting factors (fibrinogen, factors VII, VIII, X) and make less protein S (a natural clot-stopper). At the same time, the heavy uterus presses on the iliac veins and slows blood flow in the legs by half. Slow, sticky blood gives clotting proteins time to stick together and form a clot — even though there's no cut or injury.
2. The clot grows and jams the vein, blocking blood from draining out of the leg, so fluid and pressure back up into the tissues — the leg swells, feels heavy and tight, and hurts (especially the calf or thigh). — why: Blood keeps flowing down into the leg through arteries, but the clot plugs the vein so blood can't flow back up to the heart. Pressure builds behind the clot and pushes fluid out of the vein into the surrounding tissue (edema). Inflammatory signals (bradykinin, histamine) leak from the trapped blood and irritate nerves, causing pain. The leg looks puffy, red or purple, and feels warm because blood and fluid are stuck there.

3. Part of the clot breaks off (embolizes) and floats up through the inferior vena cava, through the right side of the heart, and into the pulmonary arteries — the blood vessels that carry oxygen-poor blood into the lungs to pick up oxygen. — why: The clot is fragile — muscle movement, changes in blood flow, or just the force of blood pushing against it can snap off a piece. Once free, the clot travels with the blood flow: up the leg veins, into the big vein in the belly (inferior vena cava), into the right atrium of the heart, through the right ventricle, and out into the pulmonary arteries. It keeps floating until it reaches a vessel too narrow to pass through, and it gets stuck there — blocking blood flow to part of the lung.
4. The clot jams a pulmonary artery, blocking blood from reaching the air sacs (alveoli) where oxygen is supposed to move into the bloodstream — that part of the lung keeps breathing in air, but no blood flows past to pick up the oxygen (called dead space ventilation), so oxygen levels in the blood drop. — why: Oxygen can only get into your blood when air and blood meet at the thin walls of the alveoli. If a clot blocks the blood vessel feeding a section of lung, air still fills those alveoli but the blood never arrives — so the oxygen just sits there, useless. The rest of the lungs try to compensate, but if the clot is big enough (blocking 30–50% or more of lung circulation), the working lung tissue can't keep up and oxygen saturation falls (hypoxemia). You feel suddenly short of breath and may gasp for air.
5. The body panics — the brainstem (medulla) senses low oxygen and high carbon dioxide, so it makes you breathe faster and deeper (tachypnea, hyperventilation), and the sympathetic nervous system floods the blood with adrenaline, making the heart race (tachycardia) to try to pump more blood through the fewer open lung vessels and deliver what little oxygen is available. — why: The medulla in the brainstem constantly monitors oxygen (via chemoreceptors in the carotid arteries and aorta) and carbon dioxide levels. When oxygen drops or CO₂ rises, it sends urgent signals to the diaphragm and chest muscles: breathe faster, breathe deeper. At the same time, the sympathetic nervous system releases adrenaline and noradrenaline, which make the heart beat harder and faster (trying to push more blood per minute) and tighten blood vessels to keep blood pressure up. This is the body's emergency mode — you feel your heart pounding, you're breathing fast, you may feel dizzy or anxious because your brain isn't getting enough oxygen.

6. If the clot is large or keeps growing, the right side of the heart struggles — it has to pump against the blocked, high-pressure pulmonary arteries, so it strains and can fail (right heart strain or acute cor pulmonale), causing chest pain, a drop in blood pressure, and — in the worst cases — cardiac arrest and death. — why: The right ventricle is a thin-walled pump designed to push blood gently into the low-pressure lungs. When a big clot blocks the pulmonary arteries, pressure shoots up behind the clot — the right ventricle has to push much harder than it was built for. It dilates (stretches) and weakens, and blood backs up into the body (causing neck veins to bulge, liver to swell, legs to puff up). The overstretched heart muscle doesn't get enough oxygen itself (ischemia), causing chest pain. If the right heart fails completely, it can't pump blood forward to the lungs and left heart, so blood pressure crashes, organs shut down, and the heart can stop. This is why massive PE is a leading cause of maternal death.
7. Every sign — puffy painful leg, sudden shortness of breath, chest pain, fast heart, low oxygen, bulging neck veins — is the body's alarm screaming that a clot is blocking blood flow somewhere. The only fix is to find the clot (using ultrasound for DVT, CT scan or V/Q scan for PE) and treat it by thinning the blood (anticoagulation with LMWH) to stop the clot from growing and let the body slowly dissolve it, or — in life-threatening cases — break it up fast with clot-busting drugs (thrombolytics) or pull it out (thrombectomy). — why: None of the body's emergency responses — faster breathing, racing heart, tighter blood vessels — can fix the root problem: a solid clot physically blocking a blood vessel. All they do is buy time. The only real solution is to stop new clots from forming (anticoagulation) so the body's natural clot-dissolving system (plasmin) can slowly break down the existing clot over days to weeks, or — if the patient is crashing — use powerful clot-busting enzymes (tPA, tissue plasminogen activator) to dissolve the clot in minutes to hours, or thread a catheter up to the clot and suck it out or break it apart. In pregnancy, LMWH (enoxaparin, dalteparin) is the safe choice — it doesn't cross the placenta, it's reversible, and it works fast. Warfarin crosses the placenta and causes birth defects; DOACs lack safety data. You keep the patient on LMWH through delivery and for at least 6 weeks postpartum (12 weeks total minimum) because clot risk stays high until hormones normalize and blood flow returns to normal.

Spot it — what you see, and why

- Leg swelling on one side (usually the left leg): The growing uterus presses on the left iliac vein (the big vein that drains blood from the left leg), slowing the flow and letting blood pool and clot. The left side is affected more often because of the way the vessels sit under the uterus.
- Leg pain or tenderness, especially deep in the calf or thigh: A clot inside the deep veins stretches the vein wall and triggers inflammation, which the body reads as pain. The clot blocks normal blood drainage, so pressure builds up.

- Warmth and redness over the swollen area: Inflammation from the clot sends more blood to the skin surface to try to heal the injury, making the area feel warm and look red or purple.
- Sudden shortness of breath and chest pain (sharp, worse with a breath in): A piece of the leg clot breaks off, travels through the heart, and gets stuck in the lung arteries (pulmonary a traveling clot lodging in a vessel (embolism), or PE). This blocks oxygen exchange and irritates the lung lining, causing sharp pain and trouble breathing.
- Fast heart rate (over 100 beats per minute at rest): When a clot blocks part of the lung circulation (PE), less oxygen gets into the blood. The heart speeds up to try to deliver enough oxygen to the brain and body despite the shortfall.
- Coughing up blood or pink, frothy spit: The blocked lung artery raises pressure behind it, forcing blood to leak into the tiny air sacs. That blood then comes up when you cough.

Red flags — act now

- Sudden shortness of breath or chest pain (sign of a pulmonary embolism (a clot lodging in a lung artery), which can be life-threatening)
- Coughing up blood
- Feeling faint, dizzy, or passing out (sign that blood pressure is dropping)
- Fast heart rate at rest (over 100) with trouble breathing
- Severe, sudden leg swelling with very tight, shiny skin (may mean a large clot)

Treat it — in this order

1. Start low-molecular-weight heparin (LMWH) immediately: enoxaparin 1 mg/kg subcutaneous twice daily OR dalteparin 200 units/kg subcutaneous once daily (max 18,000 units per day) → LMWH blocks factor Xa, a key protein in the clotting chain, so the blood cannot make new clots and the existing clot stops growing. This gives the body time to slowly dissolve the clot with its own enzymes (plasmin). LMWH does not cross the placenta, so it is safe for the baby.
2. Elevate the swollen leg above the level of the heart and apply graduated compression stockings (20–30 mmHg) → Gravity helps blood and fluid drain out of the leg back toward the heart, and the stocking squeezes the leg veins to push blood upward. This reduces swelling and pain quickly and may lower the chance of long-term vein damage (post-thrombotic syndrome).

3. Hold LMWH at least 24 hours before planned delivery or epidural anesthesia (12 hours before if using preventive (prophylactic) low dose) → Stopping the blood thinner lets clotting factors recover so the patient does not bleed too much during birth and does not get a spinal a pooled collection of blood (hematoma) (bleeding into the spine) if an epidural needle is placed. The epidural space has delicate veins that can bleed dangerously if blood is too thin.
4. Resume LMWH or start warfarin 4–12 hours after vaginal delivery (12–24 hours after cesarean section) and continue anticoagulation for at least 6 weeks postpartum, minimum total 3 months from diagnosis → Postpartum is the highest-risk time for new clots because delivery triggers a huge release of clotting factors. Continuing blood thinners lets the old clot scar over and stick to the vein wall so it cannot break loose and travel to the lungs. Warfarin and LMWH are both safe during breastfeeding (warfarin does not enter breast milk in meaningful amounts).
5. Place an inferior vena cava (IVC) filter if anticoagulation is absolutely contraindicated (e.g. active uncontrolled bleeding) or if clots keep breaking off despite full-dose LMWH → The filter sits in the large vein that carries blood from the legs to the heart and catches clot pieces before they reach the lungs, blocking pulmonary embolism (a clot lodging in a lung artery). It acts like a net or strainer.

Your top priorities at the bedside

1. Check oxygen saturation with pulse oximeter and listen to lung sounds every 2 to 4 hours — why: Pulmonary embolism (a clot lodging in a lung artery) blocks blood flow through the lungs so oxygen levels drop and the patient cannot breathe well; catching this early saves lives
2. Measure the circumference of both calves and thighs at the same spot each shift and compare the numbers — why: A difference of 2 centimeters or more between legs confirms DVT and tracking the measurements shows if the clot is getting worse or the treatment is working
3. Give the LMWH shot into the fatty tissue of the abdomen at the exact same times each day and rotate injection sites — why: Consistent timing keeps anticoagulation (blood thinning) levels steady to prevent new clots; rotating sites prevents skin bruising and hard lumps that block drug absorption