

Acute Limb Ischemia

A plain-English guide for you and your family

WHAT IS HAPPENING

Arteries are like highways that carry oxygen-rich blood to the muscles and nerves of your arms and legs. Normally, small side-road blood vessels—called collateral circulation—can take over if a main artery slowly narrows over months or years. But when a clot suddenly blocks a major artery, those side roads cannot open fast enough to help. The block can come from five main causes: a traveling clot thrown off by the heart (embolism, often from atrial fibrillation (an irregular, quivering heartbeat) or a damaged heart valve), a clot forming right at the blockage site in an artery already narrowed by cholesterol plaque (thrombosis), direct injury from trauma, a tear in the artery wall (dissection), or a clot in a previous bypass graft. Pain hits first—often severe and sudden—because nerve cells run out of oxygen within seconds and fire distress signals. Then the limb turns pale (pallor) because the body squeezes tiny blood vessels shut to save oxygen for the brain and heart. Next, you lose feeling (numbness or tingling, called tingling or numbness (paresthesia)) because nerves die within 30 to 60 minutes without oxygen. After that, you lose the ability to move (paralysis) because muscle cells starve and stop working. The limb turns cold because no warm blood is flowing. The clock is ticking: tissue dies completely in 4 to 6 hours. Even when doctors restore blood flow, the washout of dead-cell contents—potassium, acid, and myoglobin (a muscle protein)—back into the bloodstream can harm the kidneys, trigger dangerous heart rhythms, and cause shock. Every symptom is the body's desperate attempt to cope with oxygen starvation, and every minute counts.

What you might feel

- Sudden severe pain in the limb
- Pale skin (Pallor) (pale or white limb)
- Pulselessness (absent or weak pulses below the blockage)
- Tingling or numbness (Paresthesia) (tingling, numbness, or abnormal sensation)
- Paralysis (inability to move the limb)
- Poikilothermia (cold limb)
- Mottled or turning blue (cyanotic) (blue-purple) skin

- Muscle rigidity or stiffness

Call the doctor right away if...

- Paralysis or inability to move the limb (indicates severe starved blood flow (ischemia) and high risk of irreversible damage)
- Loss of sensation or complete numbness (nerve death is occurring)
- Muscle rigidity or woody, hard feeling to the calf or forearm (muscle tissue death from blocked blood flow (infarction), often unsalvageable)
- Mottled or turning blue (cyanotic) (blue-purple) skin (severe oxygen starvation, tissue dying)
- Absent Doppler arterial signal (no blood flow detectable, limb-threatening emergency)

What the care team will do

- Start intravenous unfractionated heparin immediately with an 80 units per kilogram bolus, then continuous infusion at 18 units per kilogram per hour, and notify the provider stat for order confirmation.
- Call the vascular surgery team immediately and communicate the six Ps: Pain, Pale skin (Pallor) (pale color), Pulselessness, Tingling or numbness (Paresthesia) (tingling or numbness), Paralysis (cannot move), and Poikilothermia (cold limb).
- Keep the affected limb at heart level or slightly dependent (hanging down); do NOT elevate the leg or arm above the heart.
- Perform and document neurovascular checks every 15 minutes: pulses by Doppler ultrasound (a handheld device using sound waves), capillary refill (pressing the nail bed and timing color return), skin temperature, color, sensation to light touch, and motor function (wiggle toes or fingers).
- Protect the limb from any trauma: use a bed cradle to keep sheets off the leg, pad bony areas, and avoid tight blood pressure cuffs or tourniquets on the affected side.
- Monitor for signs of reperfusion injury after revascularization: high potassium (hyperkalemia) (high potassium, normal is 3.5 to 5.0 milliequivalents per liter), myoglobinuria (tea-colored urine from muscle breakdown), metabolic acidosis (acid building up in the blood) (blood pH below 7.35), and compartment syndrome (severe pain, tense swollen limb, pain with passive stretch of muscles).
- Assess and document pain level every 15 minutes using a 0-to-10 numeric scale, and give prescribed opioid analgesics (strong pain medicines) intravenously for severe pain.

- Maintain nothing-by-mouth (NPO) status in case emergency surgery or catheter-based intervention is needed, and start a second large-bore IV line (18-gauge or larger) for rapid fluid or blood product administration.

Medicines you may be given — and why

Unfractionated heparin (IV bolus then continuous drip)

Heparin stops clots from getting bigger. It does NOT dissolve the clot that's already there—it only keeps new clot from piling on. Think of it like turning off the glue factory so no more sticky threads form. The existing blockage stays, but heparin buys time until surgery can remove it or a clot-buster drug can dissolve it.

Tissue plasminogen activator—tPA (alteplase) by catheter-directed thrombolysis

tPA is a clot-buster. It wakes up plasminogen (a sleeping enzyme in the blood) and turns it into plasmin (the active enzyme that chews up fibrin threads holding the clot together). The clot dissolves from the inside, and blood flow can restart. The doctor threads a thin tube (catheter) right up to the clot and drips the tPA slowly over 12 to 24 hours so it works gently and locally—this lowers the bleeding risk compared to giving tPA into the whole body at once.

Warfarin (started after heparin, once the acute clot is treated)

Warfarin slows down the liver's production of clotting factors. It blocks vitamin K, which the liver needs to finish building factors II, VII, IX, and X (think of vitamin K as the final assembly tool). Without those factors, the blood clots much more slowly. Warfarin is used long-term to prevent NEW clots after the acute blockage is fixed—especially if the cause was atrial fibrillation (an irregular, quivering heartbeat) (the heart's chaotic rhythm that lets blood pool and clot in the atrium) or if the patient has a mechanical heart valve.

Rivaroxaban, apixaban, edoxaban, or dabigatran (direct oral anticoagulants, DOACs)

These drugs block one specific clotting factor directly in the blood—no need to go through the liver or wait days like warfarin. Rivaroxaban, apixaban, and edoxaban lock onto factor Xa (the enzyme that turns prothrombin into thrombin). Dabigatran locks onto thrombin itself (the enzyme that makes fibrin threads). Either way, the clotting cascade stops, and new clots can't form. They work within hours, and you don't need routine blood tests to monitor them.

Aspirin (after revascularization, often combined with a statin and blood pressure control)

Aspirin stops platelets from sticking together. Platelets are tiny cell fragments that rush to any tear or rough spot in a blood vessel and pile up to form a plug. Aspirin permanently blocks an enzyme inside platelets called cyclooxygenase-1 (COX-1), so they can't make thromboxane A2 (a chemical signal that tells more platelets to clump). Without that signal, platelets are much less sticky. This prevents clots from forming on atherosclerotic plaques (the fatty, rough deposits in artery walls). After the acute limb starved blood flow (ischemia) is fixed, aspirin helps keep the artery open long-term—especially if the blockage was caused by in-situ clot formation (thrombosis) (a clot forming right on a ruptured plaque) rather than an a traveling clot lodging in a vessel (embolism) from the heart.

Where this information comes from

- Society for Vascular Surgery (SVS) 2020 Clinical Practice Guidelines on Acute Limb Ischemia
- American College of Cardiology (ACC) / American Heart Association (AHA) 2016 Guidelines on Lower Extremity Peripheral Artery Disease
- Cochrane Review on Thrombolysis for Acute Limb Ischemia
- European Society for Vascular Surgery (ESVS) 2020 Guidelines on Acute Limb Ischemia

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