

Peripheral Arterial Disease and Acute Limb Ischemia

A plain-English guide for you and your family

WHAT IS HAPPENING

Your leg arteries are like pipes carrying oxygen-rich blood from your heart down to your feet. Normally these pipes stay wide open so muscles and skin get all the oxygen they need, even when you walk or run. In chronic PAD (peripheral arterial disease), sticky plaque builds up inside the artery walls year after year — like rust narrowing a pipe — so blood flow slows down. When you walk, your leg muscles need more oxygen, but the narrowed artery cannot deliver it fast enough. That mismatch causes cramping pain (claudication), usually in the calf, that forces you to stop and rest. The pain goes away in a few minutes because resting muscles need less oxygen. If the blockage gets worse over time, even resting does not bring enough blood, so you feel pain at night in bed (rest pain), sores appear on your toes or heel, and tissue starts to die (gangrene). Acute limb starved blood flow (ischemia) (ALI) is totally different: a clot suddenly plugs the artery — often a clot that broke off from the heart during atrial fibrillation (an irregular, quivering heartbeat) or from the aorta — and blood stops in minutes instead of years. The leg goes pale, then mottled purple, feels ice-cold, hurts terribly, goes numb, and you cannot move your toes (the 6 P's: pain, pale skin (pallor), pulselessness, poikilothermia, tingling or numbness (paresthesia), paralysis). This is a true emergency; the leg will die in hours without surgery or a clot-busting catheter. None of this is random. Every strange finding — cramping with walking, cold foot, absent pulse, purple spots, rest pain, sores — is your body's alarm that the upstream pipe is clogged. Fixing the break means opening the pipe: for chronic PAD, stop smoking (nicotine shrinks arteries), walk 30–45 minutes three times a week with a trainer (exercise grows tiny backup vessels around the blockage), take aspirin or clopidogrel every day (keeps platelets from clumping), take a statin (shrinks plaque and stops new plaque), and if rest pain or dead tissue appears, get a balloon-and-stent or bypass to restore flow. For ALI, heparin stops the clot from growing, and surgery or a catheter pulls the clot out within hours. Time is muscle — literally.

What you might feel

- Leg pain or cramping when you walk that goes away when you rest (claudication)

- Cool or cold feet or lower legs compared to the rest of your body
- Weak or absent pulse in your feet or behind your knee
- Shiny, smooth, or hairless skin on your legs and feet
- Sores or wounds on your toes, feet, or legs that heal very slowly or not at all
- Sudden, severe pain in your leg or foot that doesn't go away (acute limb starved blood flow (ischemia))
- Pale, mottled (blotchy), or blue-tinged skin on your leg or foot
- Numbness, tingling, or weakness in your leg or foot
- Ankle-brachial index (ABI) below 0.9
- Ankle-brachial index (ABI) below 0.4

Call the doctor right away if...

- Sudden, severe leg or foot pain that won't go away—this may be acute limb starved blood flow (ischemia), a blood-flow emergency that can lead to amputation if not treated within hours.
- Leg or foot that turns pale, mottled, or blue, feels ice-cold, or has no pulse—tissue may be dying and needs urgent specialist care.
- Numbness, tingling, or paralysis in the leg or foot—nerves and muscles are starving for oxygen and may be permanently damaged if flow is not restored quickly.
- Non-healing ulcer or black, dead tissue (gangrene) on the foot or toes—this is critical limb starved blood flow (ischemia) and requires urgent vascular surgery or interventional radiology.
- Mistaking acute limb starved blood flow (ischemia) for a pinched nerve, simple cramp, or a clot in a deep leg vein (deep vein thrombosis)—acute limb starved blood flow is a vascular emergency and every hour of delay raises the risk of losing the limb.

What the care team will do

- Assess the 6 Ps every 1 to 2 hours: Pain, Pale skin (Pallor) (pale color), Pulselessness (no pulse), Tingling or numbness (Paresthesia) (numbness or tingling), Paralysis (cannot move), Poikilothermia (cold to touch)
- Notify the vascular surgeon immediately and start IV heparin bolus 80 units per kilogram then infusion 18 units per kilogram per hour if acute limb starved blood flow (ischemia) is suspected
- Keep the affected leg at heart level or slightly below, never elevate it above the heart
- Protect the leg from pressure by using a heel protector or pillow under the calf, and avoid tight sheets or blankets

- Check bilateral pedal pulses (dorsalis pedis and posterior tibial) with Doppler ultrasound and compare to the unaffected side, documenting pulse quality every 2 to 4 hours
- Monitor for reperfusion injury after revascularization: check potassium level, urine output, and watch for compartment syndrome (severe pain, swelling, tense leg)
- Assess capillary refill in toes by pressing the nail bed and counting seconds until pink color returns; normal is less than 3 seconds
- Check ankle-brachial index (ABI) by measuring ankle systolic pressure divided by arm systolic pressure using Doppler; normal is 0.91 to 1.3

Medicines you may be given — and why

Aspirin

Aspirin stops platelets (tiny sticky cells in your blood that clump together to form clots) from sticking to each other. It blocks an enzyme inside platelets called COX-1 (the factory that makes thromboxane A2, a signal that tells platelets to activate and clump). By shutting down that factory permanently, aspirin keeps blood flowing more smoothly through narrowed arteries and lowers the chance of a sudden clot that could completely block the artery and cut off blood to your leg, or cause a heart attack or stroke.

Clopidogrel (Plavix)

Clopidogrel stops platelets from clumping by blocking a receptor on their surface called P2Y12 (a docking spot for ADP, a chemical messenger that tells platelets to activate and stick together). When that receptor is locked, platelets cannot respond to ADP and stay calmer, so blood flows more smoothly through narrowed arteries and the chance of a sudden clot drops. It is often used instead of aspirin if aspirin causes side effects, or together with aspirin after a stent is placed in an artery.

Atorvastatin or Rosuvastatin (statins)

Statins lower cholesterol by blocking an enzyme in your liver called HMG-CoA reductase (the main factory worker that makes cholesterol). With less cholesterol being made, your liver pulls more LDL cholesterol (the kind that builds up in artery walls) out of your blood to use for other jobs. Lower LDL slows down plaque growth, stabilizes the plaque already there so it is less likely to crack open and cause a clot, and lowers your risk of heart attack, stroke, and worsening leg pain.

Cilostazol

Cilostazol widens blood vessels and keeps platelets from clumping by blocking an enzyme called phosphodiesterase-3 (PDE3) inside smooth muscle cells (the cells that wrap around arteries and squeeze or relax them) and platelets. Blocking PDE3 raises the level of cyclic AMP

(cAMP, a messenger inside cells), which tells smooth muscle to relax (opening the artery wider) and tells platelets to stay calm. Wider arteries and smoother blood flow mean more oxygen reaches your leg muscles, so walking hurts less and you can walk farther before pain starts (intermittent claudication improves).

Heparin (unfractionated heparin, UFH) or Enoxaparin (low-molecular-weight heparin, LMWH)

Heparin stops blood clots from growing by turbocharging a natural brake in your blood called antithrombin III (a protein that shuts down clotting enzymes). Heparin grabs antithrombin III and makes it work thousands of times faster to block thrombin and factor Xa (two key enzymes in the clotting chain). With those enzymes blocked, the clot stops growing and your body's own clot-dissolving system has time to start breaking it down. In acute limb starved blood flow (ischemia) (sudden blockage cutting off blood to your leg), heparin is given immediately to keep the clot from getting bigger while the vascular surgeon prepares to remove it or open the artery.

Alteplase (tPA, tissue plasminogen activator) or other thrombolytic agents

Alteplase dissolves blood clots by turning on plasminogen (an inactive enzyme sitting in your blood and trapped in the clot) and changing it into plasmin (the active enzyme that chews up clot fibers). Plasmin breaks down fibrin (the mesh that holds the clot together), so the clot falls apart and blood flow is restored. In acute limb starved blood flow (ischemia), a catheter is threaded into the blocked artery and alteplase is dripped directly onto the clot (catheter-directed thrombolysis) to dissolve it from the inside. This works best for clots that are fresh (less than 14 days old) and is used when the limb is not immediately dying (Rutherford class I or IIa, meaning sensory or motor loss is not yet severe).

Rivaroxaban (in combination with aspirin, for high-risk PAD)

Rivaroxaban blocks factor Xa (a key enzyme in the clotting chain) directly, without needing antithrombin III. Blocking factor Xa stops thrombin from being made, so clots form more slowly. In people with peripheral artery disease who have a very high risk of heart attack, stroke, or acute limb starved blood flow (ischemia), rivaroxaban 2.5 mg twice daily plus aspirin (the COMPASS trial regimen) lowers the chance of a major cardiovascular event (heart attack, stroke) or major amputation better than aspirin alone. It is used for chronic prevention over months to years, NOT for treating acute limb starved blood flow.

Where this information comes from

- AHA/ACC 2016 Guideline on the Management of Patients with Lower Extremity Peripheral Artery Disease

- European Society for Vascular Surgery (ESVS) 2020 Clinical Practice Guidelines on the Management of Acute Limb Ischaemia
- Society for Vascular Surgery (SVS) Lower Extremity Threatened Limb Classification System
- UpToDate clinical reviews on peripheral arterial disease and acute limb ischemia

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