

Acute Ischemic Stroke

A plain-English guide for you and your family

WHAT IS HAPPENING

Your brain runs on a constant river of blood. It has no way to store oxygen or sugar, so if even one artery gets blocked by a clot, the brain tissue downstream starts to die almost immediately. That is a stroke. The most common kind happens when a clot either forms right on top of hard cholesterol buildup inside a brain artery, or breaks loose from the heart—often because the upper chambers are quivering instead of squeezing (atrial fibrillation)—and shoots up into the brain. The warning signs hit fast: one side of the face droops, one arm goes weak, speech comes out slurred or makes no sense, vision cuts out on one side, or the person suddenly cannot walk straight. There is a clot-busting medicine (tPA) and a procedure where a thin tube snakes up to grab and pull the clot out (thrombectomy). Both can save huge amounts of brain, but only inside a narrow time window—ideally within four and a half hours for the medicine and six to twenty-four hours for the tube, depending on which artery is blocked. Every minute counts. Call 911 the moment you notice even one of these signs. Do not wait to see if it gets better on its own.

What you might feel

- Sudden face droop on one side
- Sudden arm or leg weakness on one side
- Slurred speech or trouble finding words
- Loss of vision on one side in both eyes
- Sudden dizziness, loss of balance, or trouble walking
- Sudden severe headache unlike any before
- Confusion or decreased alertness
- Double vision or trouble swallowing
- Sudden numbness on one side of the face, arm, or leg
- Sudden loss of coordination in one arm or leg

Call the doctor right away if...

- Sudden severe headache with the deficit—may be bleeding in the brain instead of a clot; get an emergency CT scan immediately before giving any blood thinner
- Neurologic symptoms getting rapidly worse after starting tPA—suggests bleeding inside the skull from the clot-buster; stop the infusion, repeat CT right away, and reverse the medicine with clotting factors and platelets
- Coma with tiny pinpoint pupils—basilar artery stroke cutting off blood to the brainstem; very high risk of death; secure the airway and consider emergency thrombectomy to pull the clot out
- Blood pressure above 185/110 before giving tPA—too high increases bleeding risk; must lower it carefully with IV medicine like labetalol or nicardipide before starting the clot-buster
- Seizure at the moment stroke symptoms start—raises the chance the clot will turn into bleeding later; confirm with imaging before giving reperfusion therapy

What the care team will do

- Confirm last-known-well time with a reliable witness
- Activate the stroke alert - CT within 25 min of arrival
- Hold oral intake until swallow screen is passed
- Q15 neuro checks x2 h post-tPA, then q30 x6 h
- Head of bed flat unless BP or oxygenation demands 30 degrees
- Fall precautions and skin protection

Medicines you may be given — and why

Alteplase (tPA)

Turns on plasminogen, a natural protein in your blood that breaks down clots. Once activated, plasminogen becomes plasmin—an enzyme that cuts the clot into pieces. The clot dissolves and blood flow returns to brain tissue that was starving for oxygen.

Aspirin

Blocks the enzyme COX-1 inside platelets forever. COX-1 makes thromboxane A2, a chemical that tells platelets to stick together. When COX-1 is off, platelets cannot clump and form new clots on damaged artery walls.

Clopidogrel

Blocks the P2Y₁₂ receptor on platelets. This receptor is a docking site for ADP, a chemical that activates platelets and makes them sticky. When the receptor is blocked, platelets cannot respond to ADP and stay calm instead of clumping.

Dual Antiplatelet Therapy (DAPT): Aspirin + Clopidogrel

Uses two drugs that block platelets in different ways. Aspirin stops thromboxane A₂ production, and clopidogrel blocks the ADP receptor. Together they shut down two main pathways that make platelets sticky, lowering the chance of a second stroke in the next few weeks.

Apixaban, Rivaroxaban, Dabigatran, or Warfarin

Blocks clotting factors in the blood (proteins that build clots). Apixaban and rivaroxaban block Factor Xa, the enzyme that turns prothrombin into thrombin. Dabigatran blocks thrombin directly. Warfarin blocks vitamin K, which the liver needs to make several clotting factors. All four reduce clot formation from the heart in patients with atrial fibrillation (an irregular, quivering heartbeat) (irregular heartbeat that lets blood pool and clot in the atrium).

Atorvastatin or Rosuvastatin

Blocks HMG-CoA reductase, the enzyme the liver uses to make cholesterol. When cholesterol production drops, the liver pulls LDL cholesterol out of the blood to use instead. Blood LDL levels fall. Lower LDL means less cholesterol builds up in artery walls, slowing plaque growth and stabilizing plaques so they do not rupture and cause another stroke.

Antihypertensives (ACE inhibitors, ARBs, thiazide diuretics, calcium channel blockers)

Lower blood pressure through different pathways. ACE inhibitors (lisinopril, enalapril) block angiotensin II formation, relaxing arteries. ARBs (losartan, valsartan) block angiotensin II receptors. Thiazide diuretics (hydrochlorothiazide) remove sodium and water through urine. Calcium channel blockers (amlodipine) relax artery walls. All reduce the force blood pushes on artery walls, lowering stroke risk over time.

Where this information comes from

- AHA/ASA 2019 Guidelines for the Early Management of Acute Ischemic Stroke
- AHA/ASA 2018 Guidelines for Endovascular Treatment
- NINDS tPA Stroke Study
- DAWN and DEFUSE-3 trials

Last reviewed: 2026-07-07