

TIA • Transient Ischemic Attack

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THE STORY IN ONE SENTENCE

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“A TIA is brain trouble (weakness, slurred speech, vision loss) that goes away in under 24 hours—usually under 1 hour—because blood flow comes back before cells die. Find where the clot came from (heart, neck artery, or small vessel disease) with brain imaging (MRI with DWI), vessel imaging (CT or MR angiogram or carotid ultrasound), and heart check (ECG, echo if a heart clot is likely). Give dual antiplatelet therapy (aspirin plus clopidogrel) for 21 days if imaging is clean, then one blood thinner forever. Start a high-dose statin (atorvastatin 80 mg). Control blood pressure, diabetes, and cholesterol. Refer to stroke neurology within 24 hours—half of all strokes after TIA happen in the first 2 days.”

Normal anatomy — the body's job

Your brain runs on oxygen delivered every second by blood flowing through a branching tree of arteries—the carotid and vertebral arteries in your neck split into smaller cerebral arteries inside your skull, feeding every brain region with a steady stream of oxygen and sugar so nerve cells can fire signals and keep you thinking, moving, seeing, and speaking.

BODY'S JOB

Brain cells are the hungriest cells in your body—they burn oxygen and sugar nonstop to make the electrical sparks that let you move your hand, understand words, and see the world. Blood vessels (arteries) are the delivery trucks: the carotid arteries in the front of your neck and the vertebral arteries in the back climb into your skull and branch like a tree, so every tiny neighborhood of brain tissue gets its own blood supply. The middle cerebral artery (the biggest branch) feeds the strip of brain that controls your face, arm, and speech on one side. The anterior cerebral artery feeds the leg area. The posterior cerebral artery feeds vision. If blood flow is steady, brain cells hum along perfectly, and you never notice. The whole system depends on smooth artery walls, steady pressure, and blood that flows without clotting too easily.

1. Heart pumps oxygen-rich blood up through the carotid arteries (front of the neck) and vertebral arteries (back of the neck).
2. Arteries climb into the skull and branch into the cerebral arteries: anterior (leg control), middle (face, arm, speech), and posterior (vision).
3. Tiny branches deliver oxygen and sugar to every brain cell, fueling the electrical signals that let you think, move, speak, and see.
4. Blood picks up waste (carbon dioxide) and flows back to the heart through veins, then to the lungs to reload oxygen.
5. The cycle repeats every few seconds—brain cells never rest, so blood flow never stops.

What breaks

A blood clot or a chunk of fatty plaque suddenly plugs one of the brain arteries, cutting off oxygen to a patch of brain cells—those cells stop working instantly (causing weakness, slurred speech, or vision loss), but the clot breaks apart or squeezes through before the cells actually die, so symptoms vanish and the brain recovers completely.

CLINICAL TERM

Transient focal cerebral starved blood flow (ischemia) due to thromboembolism, with complete symptom resolution and no tissue death from blocked blood flow (infarction) on imaging.

How the body compensates

The body opens backup arteries (collaterals in the Circle of Willis) and tries to dissolve the clot with natural enzymes—if flow returns fast enough, brain cells recover completely and

symptoms disappear. But the source of the clot is still there, and vital signs look normal even though the next clot could be minutes away.

1. The Circle of Willis (a ring of arteries at the base of the brain connecting the carotid and vertebral systems) reroutes blood around the blockage through collateral branches.
2. Natural clot-dissolving enzymes (tissue plasminogen activator, tPA, made by the vessel lining) break the clot into fragments that wash away.
3. Stunned brain cells (in the blood-starved (ischemic) penumbra—the border zone around the blockage) regain oxygen and start firing again, so weakness, numbness, or speech trouble vanishes within minutes to an hour.
4. Blood pressure may rise slightly (the brain's autoregulation reflex) to push more flow past a narrowed spot, helping collaterals deliver oxygen.
5. Vital signs and neuro exam look completely normal after symptoms resolve—this is the dangerous window, because the patient and team may think the danger has passed when the clot source (heart flutter, carotid plaque, small-vessel disease) is still active.
6. The highest stroke risk is in the first 48 hours after TIA—up to 5% will have a completed stroke in 2 days, and half of all post-TIA strokes happen in the first week—so normal appearance now does not mean safe tomorrow.

Domino effect

1. A clot or plaque fragment plugs a brain artery (most often the middle cerebral artery, which feeds the face/arm/speech area, or a branch of it). — The blockage cuts off oxygen and glucose delivery to downstream brain cells—those cells stop making ATP (the energy molecule), so ion pumps fail and electrical signals stop within seconds.
2. Brain cells stop working immediately, causing sudden focal symptoms: arm/face weakness, slurred speech (if the left hemisphere language areas are hit), vision loss (if posterior cerebral artery), numbness, or trouble understanding words. — Without ATP, neurons cannot maintain the sodium/potassium gradients needed to fire action potentials—the brain region goes electrically silent, so the body function it controls (movement, speech, vision) disappears instantly.
3. Collateral arteries (backup branches in the Circle of Willis) open, or the clot breaks apart from natural tPA in the blood, restoring blood flow before cells die—usually within 1 hour, always within 24 hours by definition. — The Circle of Willis connects the carotid and vertebral systems, so blood can detour around a blocked artery; tPA dissolves fibrin strands holding the clot together, letting fragments wash downstream and flow resume.

4. Oxygen returns, stunned neurons recharge (ischemic penumbra recovers), and every symptom vanishes—the exam is completely normal again. — Cells in the penumbra (the at-risk zone around the blockage) were blood-starved (ischemic) but not infarcted (dead)—once they get oxygen, ATP production restarts, ion pumps work again, and normal signaling comes back, so strength, speech, and vision return to baseline.
5. The clot source remains: atrial fibrillation (an irregular, quivering heartbeat) keeps pooling blood in the heart, carotid plaque is still rough and ready to throw another clot, or small-vessel disease keeps arteries stiff and narrow. — A TIA does not fix the upstream problem—it just means the first clot was small or dissolved fast. The same broken system (irregular heartbeat, cholesterol buildup, high blood pressure damage) will produce another clot unless treated.
6. The next a traveling clot (embolus) may be larger, stick longer, or hit a spot with poor collaterals—brain cells die (infarction), and the deficit is permanent (ischemic stroke). — If collaterals cannot reroute enough blood or the clot does not dissolve in time, neurons run out of ATP for more than a few minutes and undergo irreversible injury (cell membrane breaks down, calcium floods in, cells swell and die)—that tissue is gone forever.
7. Risk is highest in the first 48 hours (5% stroke rate) and first 7 days (10% total)—so TIA is a medical emergency requiring same-day imaging, antiplatelet therapy, statin, and neurology referral. — The clot-producing condition is active right now—imaging finds the source (carotid narrowing (stenosis) >50% may need surgery; atrial fibrillation (an irregular, quivering heartbeat) needs anticoagulation; small-vessel disease needs blood pressure and diabetes control), and dual antiplatelet therapy (aspirin + clopidogrel for 21 days) cuts early stroke risk by two-thirds if started within 24 hours.

Symptoms · what the patient feels, and why

- Sudden weakness or numbness on one side of the body (face, arm, or leg): A clot or piece of plaque briefly blocks a brain artery, so part of the motor cortex (the strip of brain that sends movement signals) or the sensory cortex (the strip that receives touch signals) stops getting oxygen and can't fire properly. That side of the body goes numb or weak because the signals aren't getting through.
- Sudden trouble speaking or understanding words (slurred speech, wrong words, or can't get words out): The blockage hits Broca's area (the speech-making zone in the front-left brain) or Wernicke's area (the word-understanding zone in the side-left brain). Without oxygen those cells pause, so speech comes out garbled or the person can't make sense of what they hear.

- Sudden vision loss or double vision in one or both eyes: The clot blocks an artery feeding the occipital lobe (the back of the brain that reads what your eyes send in) or the brainstem pathways that coordinate eye movement. The visual signal stops reaching the brain or the eyes can't line up, so vision goes dark or doubles.
- Sudden severe dizziness, loss of balance, or trouble walking: The blockage cuts flow to the cerebellum (the coordination center at the back-bottom of the brain) or the vestibular pathways (the inner-ear-to-brain balance system). Those areas can't keep you steady, so you feel spinning or can't stay upright.
- Sudden severe headache with no clear cause: When blood vessels in the brain suddenly stretch or spasm because of the blockage or the brain tissue swells even a little from the lack of oxygen, pain sensors in the vessel walls and the meninges (the wrapping around the brain) fire and create a sharp headache.
- Confusion or sudden trouble thinking clearly: The blockage starves parts of the frontal or parietal lobes (thinking and attention zones) of oxygen. Those neurons slow down or pause, so the person can't focus, plan, or track what's happening around them.
- All symptoms resolve completely within 24 hours (usually within minutes to one hour): The clot or plaque fragment breaks apart on its own or moves downstream, blood flow restarts, and the starved brain cells wake back up because they didn't die—they only paused. By definition a TIA leaves no lasting damage on imaging.
- Normal neurologic exam when the patient arrives at the hospital: Because the blockage cleared and blood flow returned before permanent injury, the brain cells recovered fully. The National Institutes of Health Stroke Scale score is zero because all the functions—strength, sensation, speech, vision, coordination—are working again.

Focused exam

- Weakness or numbness on one side of the body that came and went: When a clot or a piece of plaque briefly blocks a blood vessel feeding the brain, one stripe of brain tissue loses oxygen for a few minutes. That stripe controls movement and feeling on the opposite side of the body. The weakness or numbness clears completely when the clot breaks apart or moves on and blood flows again. The key sign of a TIA is that everything goes back to normal — the brain did not die, it just got starved briefly.
- Slurred speech or trouble finding words that cleared up: The left side of the brain (in most people) holds the speech centers — Broca's area (which makes words come out) and Wernicke's area (which understands words). When a clot blocks the artery feeding those spots, speech gets jumbled or stops. When the clot clears and blood flows again, speech comes back. This tells you exactly where the blood got cut off.

- Vision loss in one eye or double vision that came and went: The eye and the muscles that move it need steady blood flow from branches of the carotid and vertebral arteries. A TIA can block the ophthalmic artery (the eye's main supply line) or the part of the brain that controls eye movement (the brainstem or occipital cortex). When the block clears, vision returns. This is the brain and eye telling you they almost ran out of fuel.
- Completely normal neurologic exam when the patient arrives at the hospital: By definition, a TIA lasts less than 24 hours — most clear in under an hour. By the time you see the patient, the clot has dissolved or moved and the brain is working again. A normal exam now does not mean nothing happened; it means the damage was temporary. This is the hallmark of TIA and the reason imaging often looks normal.
- Carotid a whooshing sound over a vessel (bruit) (a whooshing sound heard with a stethoscope over the neck): A a whooshing sound over a vessel (bruit) means blood is rushing through a narrowed spot in the carotid artery (the main vessel feeding the front two-thirds of the brain). That narrowing is often caused by a cholesterol plaque. A piece of that plaque can break off and block a smaller artery in the brain, causing a TIA or stroke. Hearing a a whooshing sound over a vessel tells you the patient needs urgent imaging of the neck vessels.
- Blood pressure reading: High blood pressure (over 140/90 mmHg) damages the lining of arteries and makes plaques grow faster. It also makes clots more likely to form. Checking blood pressure tells you one of the main risk factors for another TIA or a full stroke. Lowering it steadily over time helps protect the brain.

Diagnostics · every test answers a question

- MRI brain with diffusion-weighted imaging (DWI) → Is there any dead brain tissue, or did the blood flow come back before the cells died?
- CT head (non-contrast) → Is there bleeding in the brain, or a large stroke that we need to see right now?
- Carotid Doppler ultrasound or CT/MR angiography of the neck → Is there a dangerous narrowing in the carotid or vertebral arteries that could throw another clot?
- 12-lead ECG and continuous telemetry monitoring → Is the heart beating irregularly and forming clots that travel to the brain?
- Transthoracic echocardiogram (TTE) or transesophageal echocardiogram (TEE) → Is there a clot sitting in the heart, or a hole in the heart letting clots sneak through?
- Fasting lipid panel (total cholesterol, LDL, HDL, triglycerides) → Is cholesterol building plaques in the arteries?
- Hemoglobin A1c → Does the patient have diabetes, which damages blood vessels and raises stroke risk?

- Complete blood count (CBC) with platelet count → Are there too many platelets (making clots more likely) or too few (raising bleeding risk if we start blood thinners)?

Treatment logic

- Dual antiplatelet therapy: aspirin 325 mg loading dose, then 81 mg daily, PLUS clopidogrel 300–600 mg loading dose, then 75 mg daily for 21 days (CHANCE/POINT protocol), then aspirin alone → Two antiplatelet drugs together stop platelets from clumping and forming new clots in the first three weeks after a TIA, when stroke risk is highest (up to 10–20% in the first 90 days, mostly in the first week). After 21 days, you drop clopidogrel and continue aspirin alone to avoid bleeding risk.
- High-intensity statin: atorvastatin 80 mg or rosuvastatin 20–40 mg daily, regardless of baseline LDL → Statins lower LDL cholesterol and stabilize the cholesterol plaques inside arteries so they are less likely to crack open and form a clot. They also cool down inflammation in the vessel wall. The benefit starts within weeks and grows over months.
- Blood pressure control: target < 140/90 mmHg for most patients, < 130/80 mmHg if diabetes or chronic kidney disease (individualize based on age and comorbidities) → Lowering blood pressure reduces the force hammering on artery walls, which slows plaque growth and lowers the chance that a plaque will rupture. ACE inhibitors, ARBs, and thiazide diuretics are common first choices.
- Carotid imaging (Doppler ultrasound, CT angiography, or MR angiography) followed by carotid endarterectomy or stenting within 2 weeks if symptomatic narrowing (stenosis) $\geq$ 70% (or 50–69% in select cases) → Removing the plaque (endarterectomy) or propping the artery open with a mesh tube (stenting) restores normal blood flow and removes the rough surface where clots form. Early surgery (within 2 weeks of the TIA) cuts stroke risk dramatically in patients with severe narrowing.
- Anticoagulation (warfarin with INR 2–3, or a DOAC such as apixaban 5 mg twice daily, rivaroxaban 20 mg daily, edoxaban 60 mg daily, or dabigatran 150 mg twice daily) if atrial fibrillation (an irregular, quivering heartbeat) or left ventricular a blood clot (thrombus) is found → Anticoagulants block clotting factors in the blood (warfarin blocks vitamin K-dependent factors; DOACs block factor Xa or thrombin directly) and prevent new clots from forming in the heart. This stops clots from shooting up to the brain. CHA₂DS₂-VASc score $\geq$ 2 in men or $\geq$ 3 in women means anticoagulation is needed.
- Diabetes control: target A1c < 7% with metformin, SGLT2 inhibitors, or GLP-1 agonists (which also lower cardiovascular risk) → Keeping blood sugar steady prevents the sugar from damaging the lining of blood vessels and making plaques grow. SGLT2 inhibitors and GLP-1 agonists also protect the heart and kidneys directly.

- Lifestyle modification: smoking cessation, weight loss if BMI $\geq$ 25, regular aerobic exercise (150 minutes per week), and a Mediterranean or DASH diet → Stopping smoking lets the artery lining heal and lowers clot risk within weeks. Weight loss and exercise lower blood pressure, improve cholesterol, and make the body more sensitive to insulin. The Mediterranean diet (rich in vegetables, fish, olive oil, nuts) cools inflammation and stabilizes plaques.

Medications

Aspirin + Clopidogrel (dual antiplatelet therapy) · Antiplatelet agents

Both drugs stop blood cells called platelets from clumping together. Aspirin blocks an enzyme (COX-1) that tells platelets to stick. Clopidogrel blocks a receptor (P2Y12) that also signals platelets to clump. Together, they make it much harder for a new clot to form in a narrowed brain artery, lowering the chance of a full stroke right after a TIA.

1. Aspirin blocks the COX-1 enzyme inside platelets, stopping them from making thromboxane A₂, a chemical that tells platelets to stick together.
2. Clopidogrel blocks the P2Y12 receptor on the platelet surface, stopping a different 'stick together' signal triggered by ADP (a molecule released when vessels are damaged).
3. With both pathways blocked, platelets lose their ability to form dangerous clots in narrowed arteries.
4. Blood flows more smoothly through tight spots, and the brain gets steady oxygen and fuel.

SAFETY WATCH

Watch for any unusual bleeding—nosebleeds, bleeding gums, dark or bloody stools, easy bruising, or blood in urine. · Aspirin can irritate the stomach lining; take with food if it bothers your stomach. · Tell your doctor before any surgery or dental work—you may need to pause one or both drugs to avoid excess bleeding. · Dual therapy is usually given for 21 days (based on POINT and CHANCE trials), then only aspirin continues long-term. · Call 911 immediately if you have signs of major bleeding or stroke (sudden weakness, slurred speech, severe headache).

Aspirin + Ticagrelor (dual antiplatelet therapy, alternative) · Antiplatelet agents

Aspirin blocks the COX-1 enzyme in platelets to stop one sticking signal. Ticagrelor directly blocks the P2Y12 receptor (like clopidogrel does) but works faster and more completely.

Together they prevent new clots from forming in brain arteries after a TIA, cutting stroke risk in the days and weeks after the warning event.

1. Aspirin shuts down COX-1, stopping platelets from making thromboxane A₂, a powerful clumping signal.
2. Ticagrelor sits on the P2Y₁₂ receptor and blocks it directly (it does not need liver activation like clopidogrel does), stopping the ADP clumping signal.
3. Platelets cannot stick together as easily, so dangerous clots are less likely to form.
4. Blood flows more freely through narrow spots, keeping the brain fed with oxygen and glucose.

SAFETY WATCH

Watch for any unusual bleeding—bleeding gums, nosebleeds, bruising, dark stools, blood in urine. · Ticagrelor can cause shortness of breath in some people; it usually goes away but tell your doctor if it bothers you. · Dual therapy is typically given for 30 days (based on the THALES trial), then only aspirin or a single antiplatelet continues. · Tell your doctor before any surgery or dental work—you may need to stop these drugs temporarily. · Call 911 immediately if you have signs of major bleeding or a new stroke.

High-intensity statin (e.g., atorvastatin 40-80 mg or rosuvastatin 20-40 mg) · Statin (HMG-CoA reductase inhibitor)

Statins block an enzyme in the liver (HMG-CoA reductase) that makes cholesterol. With less cholesterol being made, the liver pulls LDL (bad cholesterol) out of the blood to use for making cell parts. Lower LDL means less buildup in artery walls, which stabilizes existing plaques and slows new plaque from forming. Stable plaques are less likely to crack open and trigger a clot.

1. The statin enters liver cells and blocks HMG-CoA reductase, the enzyme that makes cholesterol.
2. The liver runs low on cholesterol and turns up receptors on its surface to grab LDL cholesterol from the blood.
3. LDL levels drop, and less cholesterol is available to deposit in artery walls.
4. Existing plaques become more stable and less inflamed, lowering the chance they will rupture and cause a clot.
5. Over time, the risk of a full stroke or heart attack goes down.

SAFETY WATCH

Muscle aches are the most common side effect; tell your doctor if your muscles hurt or feel unusually weak. · Rarely, statins can cause liver enzyme elevations—your doctor will check blood tests periodically. · Very rarely, they can cause rhabdomyolysis (severe muscle breakdown)—go to the ER if you have dark urine and severe muscle pain. · Avoid grapefruit juice with some statins (especially atorvastatin and simvastatin) because it raises blood levels and side-effect risk. · Do not stop your statin without talking to your doctor—stopping suddenly raises your stroke and heart attack risk.

Antihypertensive medications (e.g., ACE inhibitor, ARB, thiazide diuretic, calcium channel blocker) · Blood pressure-lowering agents (various classes)

High blood pressure damages artery walls over time, making plaques more likely to form and rupture. Controlling blood pressure takes stress off vessel walls and slows disease progression. Different drug classes work in different ways—some relax blood vessels, some reduce fluid volume, some block stress hormones—but all bring pressure down to a safer range (target usually <130/80 mm Hg after TIA).

1. ACE inhibitors (e.g., lisinopril) block the enzyme that makes angiotensin II, a hormone that narrows blood vessels and raises pressure.
2. ARBs (e.g., losartan) block the receptor where angiotensin II acts, achieving a similar effect.
3. Thiazide diuretics (e.g., hydrochlorothiazide) help the kidneys flush out extra salt and water, shrinking blood volume and lowering pressure.
4. Calcium channel blockers (e.g., amlodipine) stop calcium from entering vessel wall muscle cells, so vessels stay relaxed and wide.
5. With lower pressure, artery walls take less pounding, plaques stay more stable, and the risk of rupture and clot formation drops.

SAFETY WATCH

Stand up slowly—some BP drugs can cause dizziness when you first stand (orthostatic low blood pressure (hypotension)). · ACE inhibitors can cause a dry cough in some people; if it bothers you, an ARB is usually an alternative. · Diuretics make you urinate more; take them in the morning so you do not wake up at night. · Check your blood pressure at home regularly and keep a log for your doctor. · Do not stop blood pressure medicine suddenly—stopping can cause a dangerous rebound spike in pressure.

Oral anticoagulant (e.g., apixaban, rivaroxaban, warfarin) — IF atrial fibrillation (an irregular, quivering heartbeat) is present · Anticoagulant (blood thinner)

Atrial fibrillation (an irregular, quivering heartbeat) (AFib) causes the heart's upper chambers to quiver instead of squeeze, so blood pools and clots can form. A clot can break off and travel to the brain, causing a stroke. Anticoagulants block clotting factors in the blood (DOACs like apixaban block factor Xa; warfarin blocks vitamin K, which makes several factors). This keeps clots from forming in the heart, so they never travel to the brain.

1. In AFib, blood swirls slowly in the left atrium (upper heart chamber) instead of being pumped smoothly out.
2. Slow-moving blood is more likely to clot.
3. Anticoagulants block part of the clotting cascade—DOACs (direct oral anticoagulants) block factor Xa directly; warfarin reduces vitamin K, which is needed to make factors II, VII, IX, and X.
4. With these factors blocked or reduced, clots cannot form as easily in the heart.
5. No clot means no a traveling clot (embolus) (traveling clot) to block a brain artery.

SAFETY WATCH

Watch for any unusual bleeding—blood in urine or stool, heavy periods, severe nosebleeds, large bruises. · If you take warfarin, you need regular INR blood tests to keep the dose right; vitamin K in foods (leafy greens) can affect the level, so keep your diet consistent. · DOACs do not need routine blood tests, but kidney and liver function must be checked periodically. · Tell every doctor and dentist you are on an anticoagulant before any procedure. · Do NOT take aspirin or NSAIDs (ibuprofen, naproxen) without asking your doctor—they raise bleeding risk. · Call 911 immediately if you fall and hit your head, have a severe headache, or show signs of major bleeding.

Red flags

- Symptoms lasting longer than one hour or not fully gone (may be a stroke, not a TIA)
- New or worsening weakness, numbness, or speech trouble after the first episode clears
- Sudden severe headache, worst of your life, or headache with stiff neck or confusion
- Loss of consciousness, seizure, or severe trouble staying awake
- Symptoms return in waves or happen more than once in a short time (high risk of full stroke within hours to days)

Nursing priorities

1. Monitor neuro checks every 1 to 2 hours using NIH Stroke Scale or hospital protocol, including arm drift, speech, facial droop, and alertness. — Up to 10% of TIA patients have a full stroke within 48 hours. Early stroke symptoms like new weakness or confusion need immediate imaging and possible clot removal (thrombectomy) to save brain tissue before permanent damage occurs.
2. Keep blood pressure below 140/90 mmHg but not lower than 120/80 mmHg unless the doctor orders a different target. — High blood pressure damages artery walls and breaks plaque loose, causing more clots. But dropping pressure too low reduces blood flow to brain tissue that is already starving for oxygen, which can cause a stroke. The brain needs steady gentle pressure.
3. Give aspirin 325 mg and clopidogrel loading dose exactly as ordered, then continue dual antiplatelet therapy for 21 days. — Two antiplatelet medicines together stop platelets (clotting cells) from forming new clots on unstable artery plaque. The CHANCE and POINT studies showed this cuts stroke risk by 30% in the first three weeks, which is the highest danger period.
4. Keep the patient NPO (nothing by mouth) until swallow screen is done and passed. — Even though TIA symptoms resolved, the patient may have silent swallowing weakness (dysphagia). Aspiration (food or water going into the lungs instead of stomach) causes pneumonia, which increases stroke risk and hospital complications.
5. Start high-intensity statin (atorvastatin 80 mg or rosuvastatin 40 mg) on admission, do not wait for cholesterol results. — Statins stabilize artery plaque within hours by reducing inflammation, not just by lowering cholesterol. Early statin therapy cuts stroke recurrence by 20%, even in patients with normal cholesterol, because the medicine calms the unstable plaque.
6. Place patient on continuous telemetry (heart monitor) for at least 24 hours to detect atrial fibrillation (an irregular, quivering heartbeat). — Atrial fibrillation (an irregular, quivering heartbeat) often comes and goes (paroxysmal) and causes 1 in 5 strokes. Standard EKG misses it 50% of the time. If AFib is found, the treatment changes completely to strong blood thinners instead of aspirin because AFib forms big clots in the heart.
7. Teach the patient stroke warning signs using BE-FAST (Balance, Eyes, Face, Arm, Speech, Time) and tell them to call 911 immediately if symptoms return. — The first 72 hours after TIA have the highest stroke risk (up to 10%). Patients who arrive within 4.5 hours of symptom start can get clot-busting medicine (tPA) or mechanical clot removal, which can prevent permanent disability. Every minute saves brain cells.

- 8.** Coordinate urgent carotid imaging (ultrasound, CTA, or MRA) within 24 hours and communicate results to the stroke team immediately. — Carotid artery blockage over 70% causes half of TIAs. If found, the patient may need carotid endarterectomy (surgery to clean out the artery) or stenting within days to prevent a major stroke. Delay increases the chance of permanent brain damage.

Sources

- AHA/ASA 2021 Guideline for Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack
- CDC Stroke Facts
- Cochrane Stroke Group reviews
- NEJM Clinical Practice – Transient Ischemic Attack

Last reviewed: 2026-07-07