

Migraine

Migraine is a problem with both nerves and blood vessels—the outer layer of the brain gets too electrically excited. Treatment means either stopping attacks when they start or preventing them from ever beginning.

THE ONE-LINE TRUTH

Migraine is a brain disorder where waves of electrical noise and swelling in blood vessels cause headaches that come back again and again, often with warning signs and strange sensory symptoms.

The chain — how it unfolds

1. Premonitory phase (warning phase): yawning, mood changes (irritability or feeling unusually happy), food cravings, neck stiffness, tiredness. — why: Hours to days before the headache, the hypothalamus (the brain's master control center for sleep, appetite, and mood, deep in the middle of the brain) starts firing abnormally, sending out warning signals that the migraine system is winding up—these are not imagined; they are real brain changes you can see on imaging.
2. Aura phase (in about 25% of people with migraine): visual zigzags or blind spots, tingling that spreads up an arm, slurred speech, sometimes weakness. — why: Cortical spreading depression—a slow wave of electrical silence—creeps across the occipital cortex (vision area at the back of the brain), parietal cortex (sensation area on the side), or frontal/temporal cortex (speech area on the front and side) at 2–5 millimeters per minute, causing reversible symptoms that march and then fade as the wave passes; this is electrical noise, not a stroke, so it leaves no permanent damage.
3. Headache phase: unilateral (one-sided), throbbing pain that builds over hours and lasts 4–72 hours if untreated. — why: The cortical spreading depression activates the trigeminal nerve in the brainstem (the thick stalk connecting the brain to the spinal cord), which releases CGRP (calcitonin gene-related peptide, a powerful pain and swelling messenger) and substance P (another pain chemical) onto the meningeal blood vessels (vessels in the protective layers around the brain), making them swell and fire pain signals back to the thalamus (the brain's relay station for sensation, deep inside) and then up to the cortex, where you feel the throb.

4. Light sensitivity (Photophobia) (light hurts), sound sensitivity (phonophobia) (sound hurts), osmophobia (smells are unbearable), and nausea/vomiting. — why: Central sensitization spreads through the brainstem and thalamus, so normal light, sound, and smell signals are now read as painful; the vomiting center in the medulla (the lowest part of the brainstem, where it meets the spinal cord) fires because it sits right next to the pain pathways—nausea is collateral damage, not separate sickness.
5. Allodynia (pain from things that shouldn't hurt): even gentle touch on the scalp or face feels painful; combing hair or wearing glasses hurts. — why: Peripheral sensitization makes trigeminal nerve endings fire at the slightest touch, and central sensitization amplifies those signals, so non-painful input is now registered as pain—this is a sign the attack is deep and may be harder to stop.
6. Postdrome phase (migraine hangover): tiredness, mental fog, mood changes, muscle aches for hours to a day after the headache fades. — why: The chemical storm—CGRP release, serotonin (a mood and pain-regulating brain messenger) depletion, inflammatory peptides, and hours of pain signaling—exhausts the brain's energy stores (ATP, the cell's fuel molecule) and leaves the system in recovery mode, like the calm after a seizure.
7. Return to baseline (in episodic migraine, fewer than 15 headache days per month) or progression to chronic migraine (15 or more headache days per month) if triggers are not managed or medication overuse develops. — why: Most people with migraine reset completely between attacks—no brain damage occurs. But frequent attacks, medication overuse (more than 10 days per month), or untreated comorbidities (other conditions that make things worse, like depression, sleep pauses in breathing (apnea), obesity) can lower the threshold further, transforming episodic migraine into chronic daily headache, where the brain stays in a sensitized state and never fully calms.
8. Each piece points backward: postdrome ← pain sensitization ← CGRP release ← trigeminal activation ← cortical spreading depression ← hypothalamic priming ← genetic susceptibility plus trigger. — why: None of this is random. One upstream break—hyperexcitable migraine circuitry (a brain wired to overreact electrically and chemically)—explains every downstream finding. The body is not trying to help; it is stuck in a maladaptive pain loop. Understanding the sequence lets you interrupt it early (acute treatment with triptans, NSAIDs, or CGRP blockers) or raise the threshold to prevent it (prophylaxis with beta-blockers, anticonvulsants, antidepressants, or CGRP monoclonal antibodies).

Spot it — what you see, and why

- Pounding pain on one side of the head that throbs with each heartbeat: Blood vessels around the brain swell and press on pain nerves. The trigeminal nerve (the main pain wire in the face and head) sends strong pain signals to the brain, creating that pulsing ache that matches your pulse.

- Nausea or vomiting: The brainstem (the command center at the base of your brain) gets stirred up during a migraine and triggers the vagus nerve (the big nerve that controls your stomach and gut). This makes your stomach churn and can make you throw up.
- Light hurts your eyes (photophobia): During a migraine, the thalamus (the brain's relay station for senses) becomes extra sensitive and overreacts to normal light. Even dim light feels too bright and painful.
- Sound hurts your ears (phonophobia): The same thalamus overload makes normal sounds feel unbearably loud. The auditory cortex (the brain's hearing center) processes sound too intensely, so quiet noises feel like shouting.
- Physical activity makes the pain worse: Moving around increases blood flow and raises pressure inside the already-swollen blood vessels around the brain, which pushes harder on the pain nerves and cranks up the throbbing.
- Aura before or during the headache (flashing lights, zigzag lines, blind spots, numbness, or trouble speaking): A wave of brain activity called cortical spreading depression (a slow ripple of nerve cells firing, then going quiet) moves across the occipital cortex (the back part of the brain that handles vision) and sometimes the sensory or speech areas. This temporary disruption creates the strange visual or sensory symptoms.

Red flags — act now

- Sudden thunderclap headache that peaks in seconds (may be bleeding in the brain)
- New weakness, vision loss, numbness, or confusion that does not go away (may be a stroke)
- Fever, stiff neck, or confusion (may be meningitis or encephalitis)
- Headache after age 50 for the first time (may be giant cell arteritis, an inflamed artery in the temple, or a brain tumor)
- Worsening headaches in someone with cancer, HIV, or a weakened immune system (may be infection or spread of cancer to the brain)

Treat it — in this order

1. Triptan (sumatriptan 100 mg by mouth, rizatriptan 10 mg, or sumatriptan 6 mg injection) taken at the start of headache pain → Triptans activate serotonin 1B and 1D receptors (docking sites on blood vessels and nerve endings), shrinking swollen vessels and blocking the release of CGRP (calcitonin gene-related peptide—the main pain messenger in migraine). This stops the pain cascade before it spreads. They work best if taken early, before pain becomes severe.

2. NSAIDs (ibuprofen 400–800 mg, naproxen 500–550 mg) or acetaminophen 1000 mg for mild-to-moderate migraine → These medicines block cyclooxygenase (COX) enzymes, which cuts down on prostaglandins (chemicals that open blood vessels and amplify pain signals). This reduces inflammation and pain around the brain's blood vessels and offers quick relief for less severe attacks.
3. Antiemetic (metoclopramide 10 mg IV or by mouth, or prochlorperazine 10 mg IV or IM) given with acute migraine treatment → These medicines block dopamine receptors (docking sites in the brain) in the area postrema (the brain's vomit center) and the chemoreceptor trigger zone, stopping nausea and vomiting. They also speed up stomach emptying, which helps the gut absorb triptans or pain medicine faster and makes them work better.
4. Daily preventive medicine (propranolol 80–240 mg, topiramate 50–200 mg, amitriptyline 25–100 mg at bedtime, or CGRP monoclonal antibody like erenumab 70–140 mg monthly injection) if 4 or more migraines per month or if attacks are disabling → Beta-blockers like propranolol calm overactive nerve firing. Topiramate quiets electrical activity in the cortex (the brain's outer layer). Amitriptyline raises serotonin and norepinephrine (brain messengers that steady mood and pain control). CGRP antibodies block calcitonin gene-related peptide (the main pain molecule in migraine). All of these lower the brain's sensitivity to triggers and cut attack frequency by 50% or more over weeks to months.
5. Trigger avoidance and lifestyle habits—regular sleep schedule, staying hydrated, managing stress, avoiding known food triggers like aged cheese or alcohol, eating regular meals → Removing or limiting migraine triggers—such as skipped meals (which drop blood sugar and stress the hypothalamus), poor sleep (which disrupts the brain's control center for hormones and alertness), dehydration, stress, and hormonal swings—reduces how often the brain's excitability threshold is crossed and an attack is kicked off.

Your top priorities at the bedside

1. Place the patient in a quiet, dark room and minimize noise, bright lights, and interruptions — why: Migraine involves sensitization of the trigeminal nerve (main face and head nerve) and brain pathways that process light and sound, so reducing sensory stimulation decreases pain signals and nausea
2. Give prescribed triptan medication (sumatriptan or rizatriptan) or NSAID as soon as possible in the headache phase — why: Triptans work by constricting dilated blood vessels and blocking pain pathways in the trigeminal nerve; early treatment stops central sensitization (pain amplification in the brain) and gives better relief than delayed dosing
3. Administer anti-nausea medicine (metoclopramide or prochlorperazine) before or with pain medication — why: Migraine causes gastroparesis (stomach paralysis) that slows absorption of oral medicines; anti-emetics (anti-nausea drugs) restore gut motility and also have pain-relieving effects by blocking dopamine