

Migraine and Headache Red Flags

Migraine is a brain alarm that fires too easily — treat the attack, prevent the next one, and always rule out the life-threatening causes hiding behind any new or weird headache.

THE ONE-LINE TRUTH

Migraine is a brain disorder where waves of overactive nerve signals cause severe head pain (usually on one side), often with light and sound sensitivity, nausea, and sometimes vision changes. Some headaches hide dangerous causes like bleeding in the brain, infection, stroke, or torn blood vessels — so we always check for red flags first.

The chain — how it unfolds

1. A trigger (stress, lack of sleep, skipped meals, hormone drop, weather change, alcohol, or strong smells) lowers the brain's threshold for overexcitation. — why: Your genes already make your brain more excitable than average (migraine runs in families about 60% of the time). The trigger tips the balance, and nerve cells in your cortex start firing in a slow, rolling wave instead of staying calm.
2. Cortical spreading depression: a wave of abnormal electrical activity rolls across the surface of your brain at 2 to 5 millimeters per minute. — why: This is like a power brownout spreading block by block. Nerve cells depolarize (lose their normal charge), fire all at once, then go silent. The wave moves forward, leaving a trail of temporarily shut-down brain tissue behind it.
3. The spreading wave reaches the blood vessels wrapped around your brain (meningeal arteries) and the trigeminal nerve endings that surround those vessels. — why: When the wave hits, those nerve endings release CGRP (calcitonin gene-related peptide), a chemical messenger that cranks up pain signaling and makes blood vessels swell and leak inflammatory molecules into the meninges (the protective layers around your brain).
4. CGRP activates the trigeminal nerve — the main pain highway for your face, forehead, and the coverings of your brain. — why: CGRP binds to receptors on the trigeminal nerve like a key in a lock, turning the nerve on high. Pain signals flood into the trigeminal nucleus caudalis (a pain-processing station in your brainstem), and you start to feel throbbing, pulsing pain, usually on one side of your head (because the wave and CGRP release often start on one side).

5. The brainstem amplifies the pain signals and lowers your pain threshold (central sensitization), so even normal things like light, sound, or touch feel painful. — why: Your brainstem is trying to protect you by making everything feel worse, forcing you to lie still and avoid anything that could stress your brain further. This is why light hurts your eyes (photophobia), sounds feel too loud (phonophobia), and even brushing your hair or wearing a ponytail can hurt (allodynia).
6. The brainstem also talks to the vagus nerve, which controls your stomach and gut, so you feel nauseous and may throw up. — why: The brainstem is saying, 'Stop everything — don't eat, don't move, just rest,' so it shuts down your digestion. This is not a separate problem; it is part of the same survival response to give your brain a break.
7. In about 1 out of 3 people, the spreading wave crosses the visual cortex (the back of your brain that processes what you see) before it reaches the pain-sensitive structures, so you get an aura: flashing lights, zigzag lines, blind spots, or shimmering waves in your vision. — why: The wave temporarily shuts down the vision cells as it passes, so you see weird patterns or lose part of your visual field. The aura usually lasts 5 to 60 minutes, then fades as the wave moves on and the headache begins. Some people get sensory aura (tingling or numbness in the hand or face) or even speech aura (trouble finding words) if the wave crosses those brain areas.
8. The pain peaks and lasts 4 to 72 hours if untreated, then slowly fades as the brain's chemistry resets and CGRP levels drop. — why: Your body is not broken — it is working hard to recover. The cortical spreading depression wave stops spreading, CGRP breaks down, the trigeminal nerve calms down, and the brainstem lowers the alarm. You enter the postdrome phase: tired, drained, foggy, but the pain is gone. This is the brain healing and rebalancing.

Spot it — what you see, and why

- Throbbing or pounding pain on one side of the head: Blood vessels around the brain swell and press on pain-sensing nerves wrapped around them. The trigeminal nerve (the big face nerve that carries pain signals) sends alarm signals into the brain, creating that pounding feeling that often beats with your heartbeat.
- Pain that gets worse with movement, bright light, or loud noise: The brainstem (the control tower where your brain meets your spine) becomes extra-sensitive during a migraine. Normal things like walking, bright screens, or sounds now feel like they are turning up the pain dial because your brain's filters are overloaded.
- Nausea or throwing up: Migraine turns on the vagus nerve (a big nerve that runs from your brain down through your stomach). This same nerve is why your stomach churns when you are nervous. When it fires too hard during a migraine, your stomach slows down and you feel sick.

- Seeing flashing lights, zigzag lines, or blind spots (aura): A wave of electrical silence called cortical spreading depression creeps slowly across the visual cortex (the back of your brain that builds what you see). As brain cells fire and then go quiet in that wave, you see sparkles, lines, or dark patches for 5 to 60 minutes before or during the headache.
- Trouble thinking clearly or finding words: When the electrical wave reaches language and thinking areas in the cortex (the outer layer of the brain), those brain cells pause their work. You might mix up words, forget names, or feel foggy until the wave passes and blood flow returns to normal.
- Numbness or tingling, usually on one side of the face or hand: The same slow electrical wave can sweep across the sensory cortex (the strip of brain that reads touch and position). As it moves, it shuts down feeling in the matching body part—usually face, hand, or arm—for a few minutes, then the feeling comes back.

Red flags — act now

- Thunderclap headache (worst headache of your life, peaking in less than one minute)
- Headache with fever, stiff neck, confusion, or skin rash
- New headache after age 50, or a sudden change in your usual headache pattern
- Headache that gets worse with coughing, sneezing, straining, or lying down
- Headache plus weakness, vision loss, slurred speech, or trouble walking

Treat it — in this order

1. Triptan (sumatriptan 50–100 mg by mouth or 6 mg subcutaneous, rizatriptan 10 mg, or zolmitriptan 2.5–5 mg) → Triptans grab onto serotonin 1B and 1D receptors (docking spots for serotonin, a brain messenger) on blood vessels and squeeze them tighter, stopping them from swelling. They also block CGRP (calcitonin gene-related peptide, a chemical that turns up pain signals) from the trigeminal nerve (the main nerve that carries face and head pain). They work best if you take them in the first hour of the migraine.
2. NSAID (ibuprofen 400–800 mg, naproxen 500–550 mg, or ketorolac 30 mg IV) → NSAIDs block COX-1 and COX-2 enzymes (proteins that make prostaglandins, chemicals that turn up inflammation and pain). This turns down the swelling and throbbing around nerves in the meninges (the layers covering the brain) that are sending pain signals.
3. Anti-nausea medicine (metoclopramide 10 mg IV or by mouth, ondansetron 4–8 mg, or prochlorperazine 10 mg IV or 25 mg rectal) → Metoclopramide blocks dopamine D2 receptors (docking spots for dopamine, a brain messenger) in the area postrema (the brainstem's vomiting center), so your stomach calms down and you can keep food and other medicines down. Ondansetron blocks serotonin 5-HT₃ receptors in the same area. Prochlorperazine works on dopamine receptors and also helps headache pain a little.

4. Preventive medicine taken every day: beta-blocker (propranolol 80–240 mg or metoprolol 100–200 mg), antiepileptic (topiramate 50–200 mg or valproate 500–1000 mg), or tricyclic antidepressant (amitriptyline 25–100 mg at bedtime) → Daily preventive medicine steadies the balance of serotonin, norepinephrine (another brain messenger), and electrical activity in your cortex (the outer layer of your brain) so the spreading waves are less likely to start in the first place. Propranolol calms overactive adrenergic (adrenaline-related) nerve signals, topiramate quiets voltage-gated sodium and calcium channels (gates that control electrical firing), and amitriptyline raises serotonin and norepinephrine and blocks pain pathways.
5. CGRP monoclonal antibody (erenumab 70–140 mg monthly, fremanezumab 225 mg monthly or 675 mg quarterly, galcanezumab 240 mg first dose then 120 mg monthly, or eptinezumab 100 mg IV every 3 months) → These shots or infusions are antibodies (proteins your immune system makes) designed to grab CGRP (calcitonin gene-related peptide) or its receptor before CGRP can amplify pain signals in the trigeminal nerve (the main nerve that carries face and head pain). This lowers how many migraines happen and how bad they get each month.

Your top priorities at the bedside

1. Check blood pressure, temperature, and perform a quick neurologic exam (pupil size and reaction, arm strength, speech, neck stiffness) immediately — why: High blood pressure can cause with high blood pressure (hypertensive) emergency (dangerously high pressure damaging organs), fever with neck stiffness suggests meningitis, and abnormal pupils or weakness suggest stroke or brain bleed needing emergency care
2. Place the patient in a quiet, dark room and reduce noise, bright lights, and strong smells — why: Migraine causes light sensitivity (photophobia) (light sensitivity), sound sensitivity (phonophobia) (sound sensitivity), and osmophobia (smell sensitivity) because the trigeminal nerve (face and head nerve) becomes hyperexcitable, and reducing stimulation calms nerve firing
3. Give prescribed triptan medicine (sumatriptan, rizatriptan) or NSAID (naproxen, ibuprofen) as soon as possible when headache starts — why: Triptans work by narrowing swollen blood vessels and blocking pain signals in the trigeminal nerve; early treatment stops central sensitization (the brain amplifying pain signals) and prevents the headache from becoming severe