

Guillain Barr Syndrome

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THE STORY IN ONE SENTENCE

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“Guillain-Barré is weakness that starts in the feet and climbs upward after an infection—watch closely for breathing trouble when it reaches the chest muscles.”

Normal anatomy — the body's job

Peripheral nerves are long cables that carry electrical signals from the brain and spinal cord to every muscle and sensory spot in the body. Each nerve fiber is wrapped in myelin—a fatty insulation—so signals travel fast and clear, like a good wire coating. The immune system patrols for germs but normally leaves the body's own tissues alone.

BODY'S JOB

Myelin wraps nerve fibers so electrical signals can jump quickly from node to node (called saltatory conduction), letting you move muscles instantly and feel a touch right away. Without that wrap, signals slow to a crawl or stop entirely.

1. Brain decides to move a toe → sends signal down the spinal cord.
2. Signal exits the spinal cord into a peripheral nerve that runs all the way to the foot.
3. Myelin wrapping lets the signal jump fast along the nerve fiber.
4. Signal reaches the muscle → muscle contracts → toe lifts.
5. Same nerve also carries feeling back up: touch on toe → signal races back to brain → you feel it instantly.

What breaks

After an infection, the immune system gets confused and attacks the myelin (the fatty insulation around nerve fibers), stripping it away. The longest nerves—those running to the feet—are hit first, so weakness and numbness start there and climb upward.

CLINICAL TERM

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)—the most common form of Guillain-Barré syndrome—triggered by molecular mimicry: antibodies made to fight an infection (commonly *Campylobacter jejuni*, but also cytomegalovirus, Epstein-Barr virus, or Zika virus) cross-react with gangliosides on peripheral nerve myelin, leading to complement activation, macrophage infiltration, and segmental demyelination.

How the body compensates

Early on, vital signs may look normal because the body has not yet tipped into respiratory failure. The danger is missing the slow march upward—watch forced vital capacity (FVC) and negative inspiratory force (NIF) closely, because once the diaphragm weakens past a threshold, the crash into respiratory failure is sudden.

1. Weakness starts in the feet and climbs—early on the patient can still walk, talk, and breathe normally.
2. The body compensates by recruiting accessory muscles (neck and shoulder muscles) to help breathe, so oxygen saturation stays okay for a while.
3. This is the dangerous window: the team may think the patient is stable, but the wave of demyelination is still climbing.
4. The crash point to watch for: forced vital capacity (FVC) drops below 20 mL/kg, negative inspiratory force (NIF) weaker than -30 cm H₂O, or the patient cannot lift their head off the pillow (neck flexor weakness)—all predict imminent respiratory failure.
5. Once the diaphragm and intercostal muscles are too weak, CO₂ rises fast, the patient tires out, and intubation becomes urgent.
6. Autonomic instability (wild heart rate and blood pressure swings) can appear without warning, especially during procedures or suctioning.
7. Track the trend—serial FVC every 4–6 hours, watch for new facial weakness or trouble swallowing, and get ready to intubate before the patient crashes, not after.

Domino effect

1. After an infection, the immune system makes antibodies to fight the germ. — In Guillain-Barré, those antibodies accidentally cross-react with gangliosides (sugar-fat molecules) on the surface of peripheral nerve myelin, because some germs (especially *Campylobacter jejuni*) have similar surface molecules—this is called molecular mimicry.
2. Antibodies and immune cells (macrophages) attack the myelin wrapping on peripheral nerves. — The attack strips away myelin in patches (segmental demyelination), leaving bare nerve fibers that cannot carry fast electrical signals—like a wire with its insulation ripped off.
3. Weakness starts in the feet and legs, then climbs upward over hours to days. — The longest nerves—those running to the feet—are hit first and hardest, because they have the most myelin to attack; as more nerve fibers lose their wrap, the weakness ascends symmetrically into the thighs, trunk, arms, and face.
4. Deep tendon reflexes disappear (areflexia). — Reflexes need a fast round-trip signal: tap the tendon → sensory nerve fires → spinal cord → motor nerve fires back → muscle jerks. When myelin is stripped, the signal is too slow or blocked, so the reflex arc is broken and the muscle does not respond to the tap.
5. Patients feel tingling, numbness, or burning pain in the hands and feet. — Sensory nerve fibers are demyelinated too, so they fire randomly (causing tingling and pain) or stop firing (causing numbness); the nerve roots (radiculopathy) can also swell and hurt.
6. Breathing becomes shallow and weak when the weakness reaches the chest. — The phrenic nerve (controls the diaphragm) and intercostal nerves (control the rib muscles) lose their myelin, so the breathing muscles cannot contract forcefully—about 20–30 % of patients need a ventilator because their lungs cannot pull in enough air.
7. Heart rate and blood pressure swing wildly, and some patients sweat heavily or cannot control their bladder. — Autonomic nerve fibers (which control automatic body jobs like heart rate, blood pressure, sweating, and bowel/bladder) are demyelinated, so those systems lose stable control and fluctuate dangerously.
8. Most patients stop getting worse within four weeks, then slowly improve over months. — The immune attack eventually calms down, and the body begins to rebuild myelin around the nerve fibers (remyelination)—this process is slow, taking months to a year, and about two-thirds of people recover fully, though some are left with lasting weakness or numbness.

Symptoms · what the patient feels, and why

- Weakness that starts in the legs and climbs up: The immune system attacks the myelin (the protective coating around nerves, like insulation on a wire). When myelin is damaged, nerve signals slow down or stop, so muscles do not get their 'move' commands. The weakness usually starts at the feet and legs, then climbs toward the chest and arms over hours to days.
- Tingling or numbness in the hands and feet: The same immune attack damages sensory nerves (the wires that carry feeling signals to the brain). Sensory nerves often live in the same protective sheath as motor nerves, so they get hurt together. This causes pins-and-needles, tingling, or a numb feeling, usually starting in the toes and fingertips.
- Trouble walking or standing: Leg muscles are weak because nerve signals cannot reach them properly. Balance can also be off because sensory nerves that tell the brain where the feet are in space are damaged. Together, these make walking wobbly or impossible.
- Loss of reflexes (like the knee-jerk reflex): Reflexes are fast loops: a tap on the tendon sends a signal up a sensory nerve to the spinal cord, which fires a signal back down a motor nerve to make the muscle jerk. When myelin is stripped off these nerves, the loop is broken, so the reflex disappears.
- Pain in the back, legs, or muscles: Inflamed nerves send pain signals even without an injury. The immune attack and swelling around nerve roots (where nerves leave the spinal cord) can cause aching, burning, or sharp pain, often worse at night.
- Trouble breathing or swallowing: If the weakness climbs high enough, it reaches the nerves that control the diaphragm (the main breathing muscle under the lungs) and the muscles of the throat. When these muscles weaken, breathing becomes shallow and swallowing becomes unsafe because food or spit can slip into the windpipe.
- Fast or uneven heart rate, or blood pressure swings: The autonomic nerves (the 'autopilot' nerves that control heart rate, blood pressure, sweating, and digestion) can also be damaged. When these nerves misfire or stop working, the heart may race, slow down unpredictably, or blood pressure may swing from too high to too low.
- Recent infection one to four weeks before weakness started: Most people with Guillain-Barré had a stomach bug (often *Campylobacter* bacteria), a cold, the flu, or another infection in the weeks before. The immune system makes antibodies to fight that germ, but those antibodies accidentally cross-react with proteins on the myelin sheath, triggering the nerve attack. This is called molecular mimicry (the germ and the nerve look similar to the immune system).

Focused exam

- Weakness that climbs from legs to arms over hours to days: In Guillain-Barré, the immune system attacks the myelin (the fatty coating around nerves, like insulation on a wire) starting in the legs and moving upward. As more nerves lose their coating, the weakness spreads. This ascending pattern is the hallmark — if weakness moves downward instead, think of a different cause.
- Absent or very weak deep tendon reflexes (like the knee-jerk reflex): Reflexes travel in a loop through the spinal cord and back through nerves. When myelin is stripped off in Guillain-Barré, that loop breaks and the reflex disappears. Absent reflexes plus weakness point to nerve damage, not a brain or muscle problem.
- Facial weakness, trouble closing eyes completely, or flat smile: The facial nerve (cranial nerve VII) runs the muscles of the face. When Guillain-Barré climbs high enough to hit these nerves, the face droops or feels stiff. This is a red flag that the weakness is approaching the breathing muscles.
- Trouble swallowing, choking on liquids, or slurred speech: The glossopharyngeal nerve (cranial nerve IX) and vagus nerve (cranial nerve X) control the muscles that move food down the throat and shape words. When Guillain-Barré damages these nerves, swallowing fails and speech slurs. This warns you the breathing nerves may be next.
- Tingling or pins-and-needles in hands and feet, but weakness is more severe: Sensory nerve fibers can also lose myelin in Guillain-Barré, causing strange feelings. But the main danger is motor weakness — the muscles stop working. Don't let tingling distract you from checking strength and breathing.
- Unstable heart rate or blood pressure that swings high then low: The autonomic nervous system (the network of nerves that runs the heart, blood vessels, and other automatic functions) can be attacked in Guillain-Barré. When these nerves fail, the heart rate jumps around and blood pressure becomes unpredictable, raising the risk of dangerous rhythms or sudden drops.

Diagnostics · every test answers a question

- Lumbar puncture (spinal tap) with cerebrospinal fluid (CSF) analysis → Is there high protein in the spinal fluid with a normal white blood cell count?
- Nerve conduction studies (NCS) and electromyography (EMG) → Are the nerves conducting signals slowly or blocking them, and where is the damage?
- Forced vital capacity (FVC) at bedside → Is the person still breathing strongly enough on their own?
- Negative inspiratory force (NIF) measurement → How much vacuum can the person create when trying to breathe in?

- Anti-ganglioside antibodies (anti-GM1, anti-GD1a, anti-GQ1b) in serum → Are there antibodies that target specific parts of nerve cells?
- Stool culture or PCR for *Campylobacter jejuni*, serology for recent infections → Was there a recent infection that could have triggered the immune attack?
- Continuous cardiac telemetry and autonomic function monitoring → Is the autonomic nervous system stable or showing dangerous swings?

Treatment logic

- Intravenous immunoglobulin (IVIG) 2 g/kg divided over 5 days OR plasma exchange (5 exchanges over 10–14 days) → IVIG floods the blood with normal antibodies that block or dilute the attacking antibodies, calming the immune assault on myelin. Plasma exchange physically removes the attacking antibodies and inflammatory proteins from the blood. Both stop the nerve damage from getting worse and allow remyelination (regrowth of the nerve coating) to begin. Started early, they shorten the time to recovery and reduce the chance of needing a ventilator.
- Intubation and mechanical ventilation if FVC < 20 mL/kg, NIF worse than -20 cm H₂O, or signs of respiratory fatigue → The ventilator takes over the work of breathing when the diaphragm and intercostal muscles are too weak to move enough air. This keeps oxygen levels safe and prevents carbon dioxide buildup, buying time for nerves to heal.
- DVT prevention (prophylaxis) with sequential compression devices or subcutaneous heparin/enoxaparin → Paralyzed legs cannot squeeze blood back to the heart, so blood pools and clots form (deep vein clot formation (thrombosis)). Compression boots squeeze the calves mechanically; low-dose blood thinners keep clots from forming. This lowers the risk of a clot breaking off and traveling to the lungs (pulmonary a traveling clot lodging in a vessel (embolism)).
- Pain control with gabapentin 300–900 mg three times daily or pregabalin 75–150 mg twice daily → Damaged nerve fibers misfire and send burning, shooting, or aching pain signals even though nothing is actually hurting the body. Gabapentin and pregabalin calm these misfiring nerves by blocking calcium channels in nerve endings, reducing neuropathic pain.
- Continuous cardiac telemetry and autonomic support (IV fluids, atropine for a slow heart rate (bradycardia), short-acting pressors like phenylephrine for low blood pressure (hypotension)) → Monitors heart rhythm in real time and treats dangerous autonomic swings immediately. IV fluids raise blood pressure when it drops; atropine speeds the heart if it slows dangerously; pressors squeeze blood vessels to restore pressure. This prevents sudden cardiac arrest or stroke from wild swings.

- Early physical and occupational therapy once the acute phase stabilizes → Passive range-of-motion exercises keep joints flexible and prevent contractures (permanent tightening of muscles and tendons). Strengthening exercises as nerves heal help the person relearn movement and rebuild muscle that wasted during paralysis. This speeds functional recovery and return to daily life.

Medications

IVIG (intravenous immunoglobulin) · Immune modulator (pooled antibodies from healthy donors)

IVIG floods the bloodstream with normal antibodies from thousands of healthy people. These good antibodies crowd out and block the rogue antibodies that are attacking the nerve coating. They also calm down the overactive immune cells and help the body stop the attack on its own nerves.

1. Normal antibodies from the donor pool enter your bloodstream through an IV.
2. These antibodies bind to receptors on immune cells (like B cells and macrophages), sending a 'calm down' signal.
3. They also bind to and neutralize the bad antibodies that are sticking to myelin (the nerve's protective wrapping).
4. Immune cells slow their attack on the peripheral nerves.
5. The nerve coating can begin to heal, and signals from the brain start traveling down the nerves more smoothly again.

SAFETY WATCH

Headache, fever, chills, or nausea during or right after the infusion (common and usually mild) · Rare but serious: blood clots (especially in the legs or lungs), kidney damage (especially if you are older or already have kidney problems), or severe allergic reaction · Tell your care team right away if you get chest pain, trouble breathing, sudden leg swelling, or very dark urine

Plasma exchange (plasmapheresis, or PLEX) · Blood-filtering procedure (not a drug, but a key treatment)

A machine takes blood out of your body, spins it to separate the liquid part (plasma) from the cells, throws away the plasma (which contains the attacking antibodies), and returns the clean cells mixed with fresh donor plasma or a replacement fluid. This physically removes the rogue antibodies so they can't keep damaging your nerves.

1. A large IV is placed in a vein (often in the arm, neck, or groin).

2. Blood flows out into a machine that spins it, separating plasma from red cells, white cells, and platelets.
3. The dirty plasma (full of attacking antibodies) is discarded.
4. The clean blood cells are mixed with replacement fluid (donated plasma or a saline-albumin mix) and returned to your body.
5. Over several sessions (usually five treatments in one to two weeks), the level of harmful antibodies drops, and the immune attack on nerves slows.
6. Nerves get a chance to start healing.

SAFETY WATCH

Low blood pressure, dizziness, or tingling around the mouth during treatment (from shifts in fluid and calcium) · Infection at the IV site or in the bloodstream (because a large catheter is needed) · Rare but serious: bleeding (the procedure requires blood thinners), blood clots, or allergic reaction to replacement fluid · Tell your care team right away if you have fever, chills, chest pain, or bleeding that won't stop

Gabapentin · Nerve-pain medication (anticonvulsant used off-label for neuropathic pain)

Gabapentin attaches to calcium channels on nerve endings and dials down the pain signals that fire when nerves are damaged or irritated. It does not heal the nerve, but it quiets the 'alarm bells' so the pain is less intense.

1. Gabapentin binds to the alpha-2-delta subunit of voltage-gated calcium channels on nerve terminals.
2. This blocks calcium from flowing into the nerve cell.
3. Less calcium means the nerve releases fewer pain messengers (like glutamate and substance P).
4. Fewer pain signals travel up the spinal cord to the brain.
5. You feel less burning, tingling, or shooting pain.

SAFETY WATCH

Drowsiness, dizziness, or unsteady walking (especially when starting or raising the dose) · Swelling in the ankles or feet · Mood changes (rarely, sadness or anxious thoughts) · Do not stop suddenly after long use (taper slowly to avoid withdrawal symptoms) · Tell your doctor if you feel very drowsy, have trouble breathing, or notice new sadness or dark thoughts

Opioid analgesics (e.g. oxycodone, morphine) · Opioid pain relievers

Opioids attach to opioid receptors on nerve cells in the spinal cord and brain. This blocks pain signals from traveling up to consciousness and changes how the brain interprets pain, making severe nerve pain more bearable in the short term.

1. Opioid molecules bind to mu, delta, or kappa receptors on nerve cells.
2. This opens potassium channels and closes calcium channels inside the cell.
3. The nerve cell becomes less excitable and releases fewer pain messengers.
4. Pain signals are blocked from traveling up the spinal cord.
5. The brain's pain centers receive fewer alarm signals, and you feel less pain.

SAFETY WATCH

Drowsiness, confusion, or slow breathing (especially at high doses) · Severe constipation (almost universal; you will need stool softeners) · Risk of dependence and addiction with long-term use · Overdose risk if combined with alcohol, benzodiazepines, or other sedatives · Tell your care team right away if your breathing slows, you can't wake up fully, or you have severe belly pain

Low-molecular-weight heparin (e.g. enoxaparin) or unfractionated heparin · Anticoagulant (blood thinner)

Heparin attaches to a natural protein in your blood called antithrombin and supercharges it. Antithrombin then shuts down clotting factors (especially Factor Xa and thrombin), making your blood less sticky. This prevents dangerous clots from forming in the legs or lungs when you are lying still and paralyzed.

1. Heparin is injected under the skin or into a vein.
2. It binds to antithrombin, changing its shape and making it up to 1,000 times more active.
3. Supercharged antithrombin locks onto clotting factors (mainly Factor Xa and thrombin) and switches them off.
4. Blood stays thinner and flows more smoothly.
5. Clots are much less likely to form in your legs or travel to your lungs.

SAFETY WATCH

Bleeding or easy bruising (the main risk of any blood thinner) · Rarely, heparin-induced a low platelet count (thrombocytopenia) (HIT) — a dangerous reaction where platelet counts drop and clots form; needs immediate stop of heparin · Injection-site bruising or tenderness · Tell your care team right away if you have unusual bleeding (blood in urine or stool, heavy nosebleeds, coughing up blood) or sudden severe headache

Red flags

- Trouble breathing, shortness of breath, or feeling like you cannot take a deep breath
- Trouble swallowing, drooling, or choking on food or water
- Weakness spreading to the chest, neck, or face within hours
- Heart racing, slowing, or skipping beats; blood pressure swinging high and low
- Cannot stand or walk at all, or weakness has reached both arms

Nursing priorities

1. Check forced vital capacity (FVC) or negative inspiratory force (NIF) every 4 hours and watch for shallow breathing, trouble speaking full sentences, or using neck muscles to breathe. — Respiratory failure is the most common cause of death in Guillain-Barré. The diaphragm (main breathing muscle) weakens as paralysis climbs upward. FVC below 20 mL per kg or NIF weaker than -30 means the patient needs intubation before they stop breathing completely.
2. Place patient on continuous cardiac monitor and check blood pressure every 2 hours, watching for heart rate below 50 or above 120, and blood pressure swings greater than 40 mmHg. — Autonomic dysfunction (damaged automatic nerve control) causes dangerous arrhythmias, a slow heart rate (bradycardia) (slow heart rate below 50), a fast heart rate (tachycardia) (fast heart rate above 100), and blood pressure that swings from very high to very low in minutes. Atropine (a heart-speed medicine) may not work, and some pressors (blood pressure medicines) can cause deadly arrhythmias.
3. Turn patient every 2 hours, keep heels off the bed with pillows, and apply sequential compression devices (SCDs) to both legs unless patient has clots already. — Paralyzed patients cannot move themselves and develop pressure ulcers (bed sores) within hours over the sacrum (tailbone), heels, and shoulder blades. Immobility plus hypercoagulable state (blood clots more easily) from IVIG raises a clot in a deep leg vein (deep vein thrombosis) (DVT) risk to 25 percent without prevention (prophylaxis) (prevention treatment).
4. Assess swallow safety before any food or pills by mouth; keep suction at bedside and head of bed elevated 30 to 45 degrees at all times. — Bulbar weakness paralyzes the pharynx (throat) and larynx (voice box), so the airway cannot close properly during swallowing. Aspiration pneumonia (lung infection from inhaled saliva or food) is a leading cause of complications. Patients may need nasogastric tube (NG tube) feeding until swallow function returns.

5. Perform passive range-of-motion exercises to all joints twice every shift and position arms and legs in neutral alignment with splints if needed. — Flaccid paralysis (limp, floppy weakness) without movement causes contractures (permanent joint tightening) within one week. Foot drop (ankle stuck pointing downward) happens fast without ankle splints. Early range of motion preserves joint mobility for recovery, which can take months.
6. Assess pain level every 4 hours using 0-to-10 scale and give scheduled gabapentin or pregabalin, not only as-needed opioids. — Demyelination (nerve insulation damage) and axonal injury (nerve fiber damage) cause severe neuropathic pain that feels like burning, electric shocks, or stabbing. Opioids treat tissue pain but do not calm overactive damaged nerves. Gabapentin and pregabalin quiet nerve signals and must be given on schedule to keep blood levels steady.
7. Monitor urine output every shift and check for bladder distension (swollen bladder) above the pubic bone; consider bladder scan or straight catheterization if patient has not urinated in 6 hours. — Autonomic nerve damage impairs bladder detrusor muscle (bladder squeezing muscle) and sphincter control (valve that holds urine). Urinary retention (cannot empty bladder) causes overflow incontinence (leaking) and raises urinary tract infection risk. Overfilled bladder also triggers autonomic dysreflexia (dangerous blood pressure spike).
8. Provide eye care every 4 hours with artificial tears and tape eyelids closed during sleep if patient cannot blink fully. — Facial nerve paralysis (cranial nerve VII damage) prevents eyelid closure in 50 percent of patients. The cornea (clear eye surface) dries out and develops ulcers (open sores) that cause permanent vision loss. Taping lids shut and lubricating drops protect the cornea until nerve function recovers.

Sources

- American Academy of Neurology (AAN) Guillain-Barré Syndrome Guidelines 2021
- Cochrane Systematic Review on Guillain-Barré Syndrome
- National Institute of Neurological Disorders and Stroke (NINDS)
- New England Journal of Medicine Clinical Practice Review

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