

Shingles

Shingles = sleeping chickenpox virus wakes up in a nerve. Start antivirals (like valacyclovir) within 72 hours to stop nerve damage. Vaccinate adults 50+ with Shingrix to prevent it.

THE ONE-LINE TRUTH

Shingles is chickenpox virus (VZV) waking up from nerve bundles near the spine and sliding down one nerve path to burn the skin.

The chain — how it unfolds

1. Immune system weakens (age, stress, or drugs) — why: T-cells that normally patrol the dorsal root ganglia drop in number or function, so they stop killing VZV-infected nerve cells. The virus wakes up and starts copying itself inside the ganglion.
2. Virus replicates in the ganglion — why: VZV makes millions of copies of itself inside the nerve cell bodies, building up enough viral load to overwhelm the local immune defenses and spill into the nerve axon (the long wire that runs to the skin).
3. Virus travels down the sensory nerve (prodrome: burning, tingling, itch) — why: As VZV moves down the axon toward the skin, it inflames the nerve fiber itself. You feel burning, shooting pain, or weird tingling in the dermatome (skin strip) that nerve serves—often 1-5 days before any rash appears.
4. Virus reaches the skin → vesicular rash erupts in a band — why: VZV spills into skin cells, kills them, and triggers a massive local immune response. Your body floods the area with white blood cells and fluid, forming the classic grouped vesicles (small blisters on a red base) that follow one dermatome—chest, belly, or face—and stop sharply at the body's midline because each nerve serves only one side.
5. Blisters crust over; nerve damage may persist (postherpetic neuralgia) — why: After 7-10 days, your adaptive immune system clears the virus and the skin heals, but the nerve's insulation (myelin) and pain-sensing fibers are scarred. In older adults or untreated cases, the nerve stays hypersensitive and fires pain signals for months or years—even light touch feels like burning.

6. Complications if the virus spreads or hits critical nerves — why: If VZV reactivates in the trigeminal ganglion (the nerve cluster for your face), it can infect the cornea (front of the eye) and cause blindness—this is ophthalmic zoster. In immunocompromised patients (HIV, chemo), the virus can spread through the blood to the brain (encephalitis), lungs (pneumonitis), or whole body (disseminated zoster), which is life-threatening.
7. Antiviral treatment (if started ≤ 72 hours) stops the cascade — why: Acyclovir, valacyclovir, or famciclovir block VZV's DNA copying enzyme, so the virus can't replicate further. This cuts viral load, shortens the rash duration, and—most importantly—reduces nerve inflammation before permanent scarring happens, lowering postherpetic neuralgia risk by ~50%. After 72 hours, the nerve damage is often already set and antivirals help less.
8. Shingrix vaccine prevents reactivation in 90%+ — why: The recombinant zoster vaccine (Shingrix, two doses) trains your T-cells to recognize VZV surface proteins, boosting the immune patrol around the ganglia. This keeps the virus locked down even as you age, preventing shingles and postherpetic neuralgia. It's recommended for everyone 50+ and immunocompromised adults 19+, even if you've had shingles before.

Spot it — what you see, and why

- One-sided band or stripe of painful red bumps that turn into blisters: The varicella-zoster virus (the chickenpox germ) hides in a nerve root near the spine after chickenpox heals. Years later, when the immune system weakens (from age, stress, or illness), the virus wakes up and travels down that single nerve, causing a rash in the exact skin patch that nerve feeds — so it stays on one side of the body and follows the nerve's path like a stripe.
- Sharp, burning, or stabbing pain in the rash area, often starting before the rash appears: The reactivated virus inflames the nerve itself (neuritis), sending strong pain signals to the brain even before the skin breaks out. The nerve is being damaged from the inside, which feels like fire, electric shocks, or deep aching along that nerve's territory.
- Tingling, itching, or numbness in the rash area: The inflamed nerve sends mixed-up signals — some fibers fire too much (tingling and itching) while others go quiet (numbness). It is like static on a phone line when the wire is damaged.
- Fever, headache, and feeling wiped out: The immune system detects the virus spreading and releases cytokines (alarm chemicals) that turn on the body's defense mode — raising temperature to slow the virus and making you rest so energy goes to fighting infection instead of daily tasks.
- Blisters that crust over after about a week: The virus multiplies in skin cells, forming fluid-filled blisters packed with virus particles. After the immune system kills the infected cells, the blisters dry out and scab over — usually within 7 to 10 days — as the skin begins to heal.

- Rash on the face or near the eye (ophthalmic zoster): The virus reactivates in the trigeminal nerve (the main face nerve), specifically the upper branch that runs to the forehead, eyelid, and tip of the nose. Because this branch also feeds the eye, the rash can spread to the cornea (the clear window of the eye), risking vision damage.

Red flags — act now

- Rash or pain near the eye or on the tip of the nose (risks vision loss from eye infection)
- Facial weakness, drooping eyelid, or ear blisters (Ramsay Hunt syndrome can cause permanent nerve damage if not treated fast)
- Rash spreads to many body parts or looks like chickenpox all over (sign of disseminated zoster, meaning the virus is in the blood and the immune system is very weak)
- Severe headache, stiff neck, confusion, or trouble staying awake (could mean the virus has reached the brain or spinal fluid, causing meningitis or encephalitis)
- Pain that does not improve after the rash heals or lasts more than 3 months (postherpetic neuralgia, or PHN — nerve damage that causes chronic pain)

Treat it — in this order

1. Oral antiviral medicine (valacyclovir 1,000 mg three times daily for 7 days, or acyclovir 800 mg five times daily for 7 days, or famciclovir 500 mg three times daily for 7 days) started within 72 hours of rash onset → These medicines block viral DNA polymerase (the enzyme the virus uses to copy its DNA), so the virus stops multiplying inside nerve cells. This shortens how long the rash lasts (by about 1–2 days), reduces pain during the acute outbreak, and cuts the risk of postherpetic neuralgia (long-lasting nerve pain after the rash heals) by about 50%. The 72-hour window is critical — after that, most viral replication is done and the benefit drops sharply.
2. Nerve pain medicine: gabapentin (start 300 mg at bedtime, titrate up to 300–600 mg three times daily) or pregabalin (start 75 mg twice daily, titrate to 150–300 mg twice daily) → These medicines calm overactive nerve firing by blocking calcium channels on nerve cells, which reduces the electric storm of pain signals traveling to the brain. They are especially helpful for the sharp, shooting, burning pain that can last months after the rash heals (postherpetic neuralgia). Start them early in severe cases.
3. Topical lidocaine patch 5% (apply to painful area for up to 12 hours per day) or lidocaine gel → Lidocaine blocks sodium channels in nerve endings right at the skin, stopping pain signals before they reach the spinal cord and brain. This provides local relief without the side effects of pills. Works best for surface pain, not deep nerve pain.

4. Immediate ophthalmology (eye specialist) referral and topical antiviral eye drops (e.g., ganciclovir gel) or oral antivirals if rash involves the forehead, tip of nose, or eyelid (ophthalmic zoster) → Catches inflammation or infection of the cornea (keratitis), inflammation inside the eye (uveitis), or increased eye pressure (glaucoma) before they cause permanent vision loss. Early treatment with antivirals and steroid eye drops (if no active corneal infection) can save the eye.
5. Recombinant zoster vaccine (Shingrix) — two doses, 2 to 6 months apart, for adults 50 and older, or adults 19 and older with weakened immune systems → Trains the immune system to recognize varicella-zoster virus glycoprotein E (a protein on the virus surface) so T-cells (immune fighter cells) stay ready to suppress the virus before it can reactivate. Shingrix is over 90% effective at preventing shingles and postherpetic neuralgia, and protection lasts at least 7 years (likely longer).

Your top priorities at the bedside

1. Start antiviral medicine (acyclovir 800 mg five times daily, valacyclovir 1000 mg three times daily, or famciclovir 500 mg three times daily) within 72 hours of rash onset — why: Antiviral drugs stop the varicella-zoster virus from making more copies of itself inside nerve cells; early treatment shortens how long the rash lasts, reduces severe pain, and lowers the chance of long-term nerve pain (PHN) by about 50 percent.
2. Arrange urgent eye exam (within 24 hours) if rash involves the forehead, upper eyelid, or tip of the nose — why: The same nerve that brings feeling to the forehead and nose tip also goes to the eyeball (ophthalmic branch of trigeminal nerve); shingles in this area (herpes zoster ophthalmicus) can cause corneal ulcers, uveitis (inflammation inside the eye), and permanent vision loss without rapid eye-doctor treatment.
3. Begin nerve-pain medicine (gabapentin starting at 300 mg three times daily or pregabalin starting at 75 mg twice daily) for burning or electric-shock pain — why: Shingles damages the myelin coating around nerves, causing abnormal pain signals even from light touch; gabapentin and pregabalin calm overactive nerve cells by blocking calcium channels, stopping the pain messages before they reach the brain.