

The protean neurologic manifestations of varicella-zoster virus infection

- The most common sequela of herpes zoster is postherpetic neuralgia, which can persist for months and sometimes years after the rash resolves.
- VZV vasculopathy can present as transient ischemic attacks, ischemic or hemorrhagic stroke, or aneurysm.
- Vasculopathy and neurologic complications of VZV are best diagnosed by detecting VZV DNA in bodily fluids or tissues or anti-VZV immunoglobulin G in the cerebrospinal fluid.
- Most neurologic complications of VZV reactivation require antiviral treatment.
- Herpes zoster (shingles), manifesting as pain and rash in up to three dermatomes.
- Zoster sine herpette—neurologic disease without rash (formerly called dermatomal-distribution pain without rash), which can cause meningoencephalitis, vasculopathy, myelitis, cerebellar ataxia, and polyneuritis cranialis.
- Necrotizing retinitis (of which VZV reactivation is the most common cause), leading to visual loss.
- Zoster typically occurs in people older than 60 years, as VZV-specific cell-mediated immunity declines with age.
- The cardinal pathologic features of zoster are inflammation and hemorrhagic necrosis with associated neuritis, localized leptomeningitis, unilateral segmental poliomyelitis, and degeneration of related motor and sensory roots.
- Intranuclear inclusions, viral antigen, and herpesvirus particles have been detected in acutely infected ganglia.
- Magnetic resonance imaging may show enhancement of ganglia and the affected nerve roots.
- Ophthalmoplegia (paralysis of the ocular muscles) following zoster involves cranial nerves III, VI, and less frequently IV.
- Involvement of the maxillary and mandibular distribution of the trigeminal nerve can produce osteonecrosis and spontaneous tooth exfoliation.
- Compared with Bell palsy (peripheral facial paralysis without rash), Ramsay Hunt syndrome often initially involves more severe facial paralysis, and patients are less likely to recover completely. Peripheral facial paralysis caused by VZV may develop without rash, as demonstrated by a fourfold rise in antibody to VZV or by VZV DNA in auricular skin, blood mononuclear cells, middle ear fluid, or saliva. Patients with idiopathic facial weakness should be suspected of having this variant of zoster sine herpette.
- Treatment decisions about zoster should take into account the patient's age and immune status.

- Like the live-virus varicella vaccine used in children, the adult vaccine appears to be safe and clinically effective. In the first 42 days after vaccination, serious adverse events (including death) occurred in 1.4% of both the vaccine and placebo recipients.
- Brain imaging usually reveals ischemic or hemorrhagic infarcts or both, more deep-seated than cortical, and at gray-white matter junctions. Cerebral angiography also reveals areas of focal arterial stenosis or occlusion.
- The primary site of VZV is in cerebral arteries that contain multi-nucleated giant cells, Cowdry A inclusion bodies, and herpes virus particles.



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