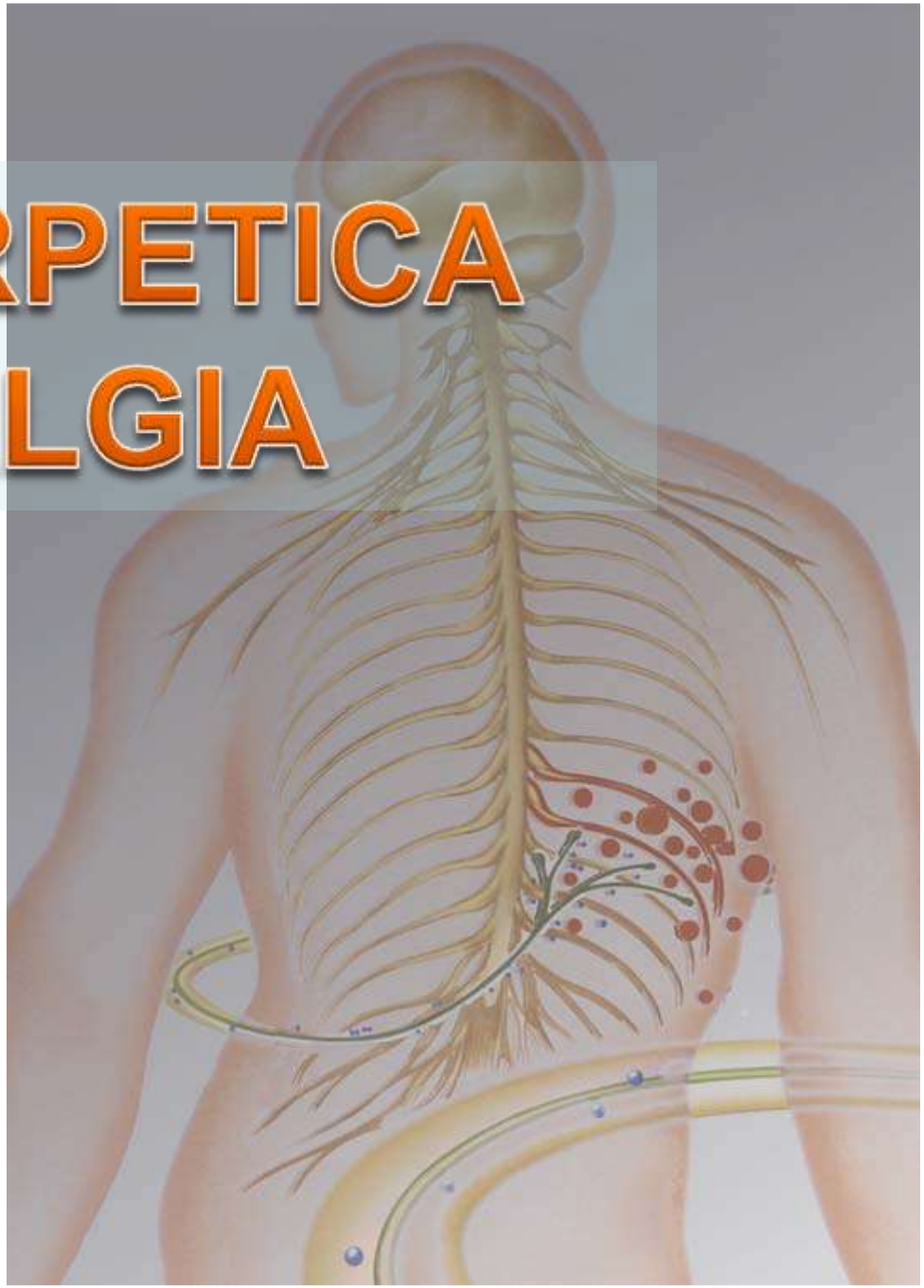
POST HERPETICA NEURALGIA



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Herpes Zoster

- Herpes zoster (shingles) is caused by reactivation of the varicella-zoster virus (VZV) from latency after infection with chickenpox. After acute infection, the virus lies dormant, typically for decades, in the sensory dorsal root ganglia.

- The cause for VZV reactivation is unclear. However, decline in cell-mediated immunity with age, certain diseases (such as HIV infection), or effects of immunosuppressive therapy.

Risk Facktors

- it's most common in older adults: More than half the shingles cases occur in adults over 60.
- people with weakened immune systems from HIV/AIDS.
- those who are receiving medical treatments, such as steroids, radiation and chemotherapy
- those who have a history of bone or lymphatic cancer are more likely to develop shingles.

Most people develop shingles only once, but recurrences in other areas are possible.

Pathogenesis

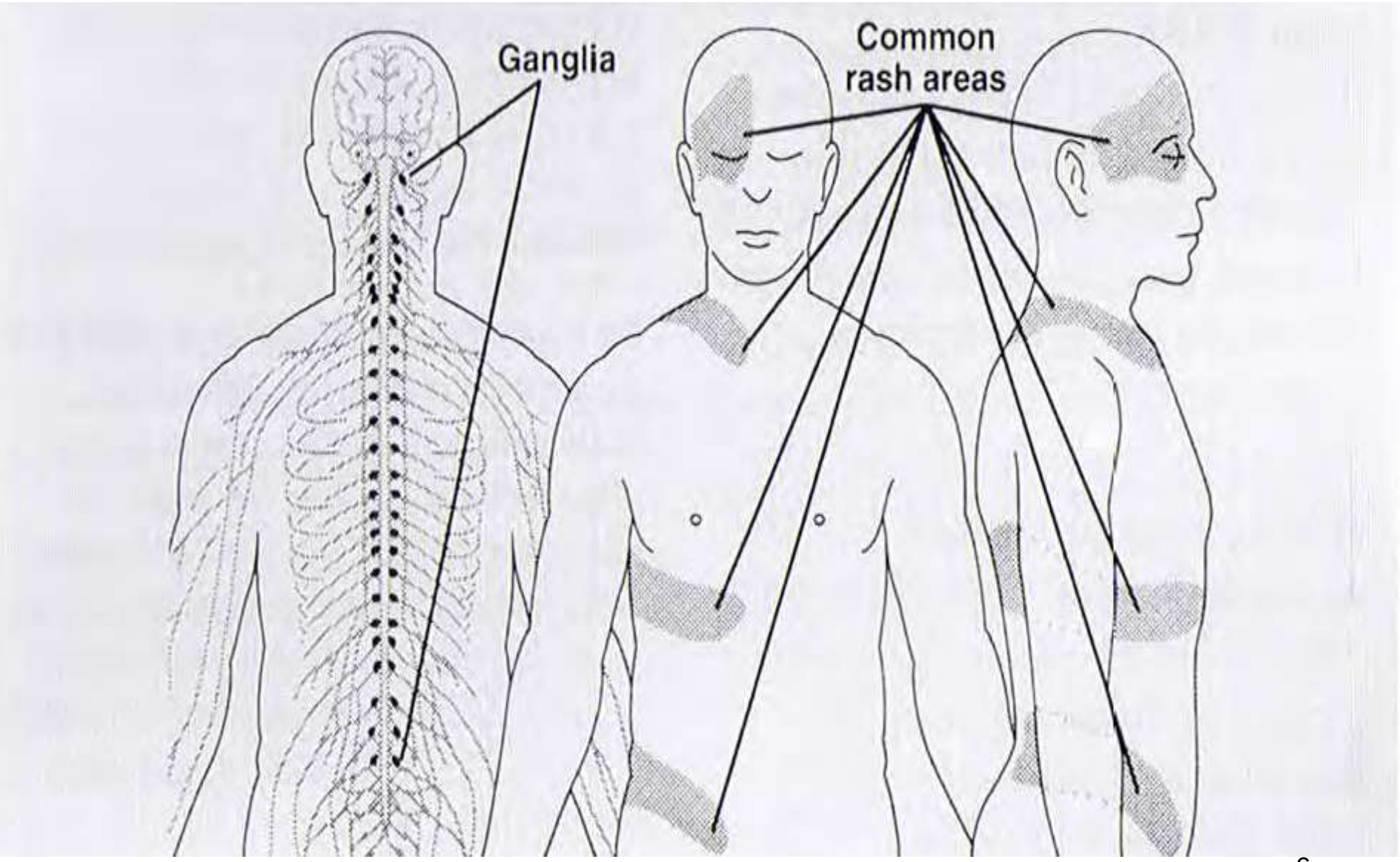
- Following VZV reactivation → virus replication → ganglionitis and extensive inflammation and destruction of neurons and supporting cells.

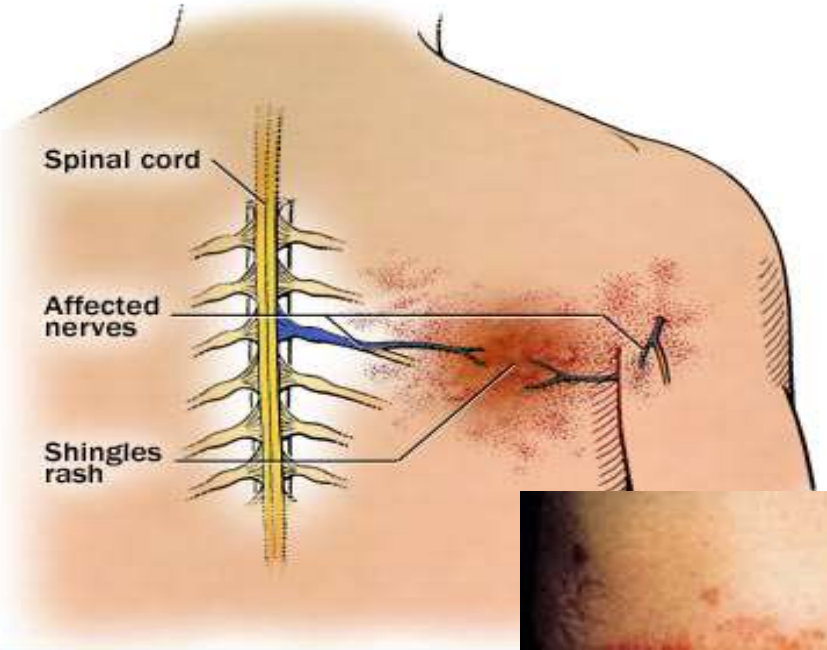
- The dermatomal distribution of the subsequent vesicular rash corresponds to the sensory fields of infected neurons within a ganglion.

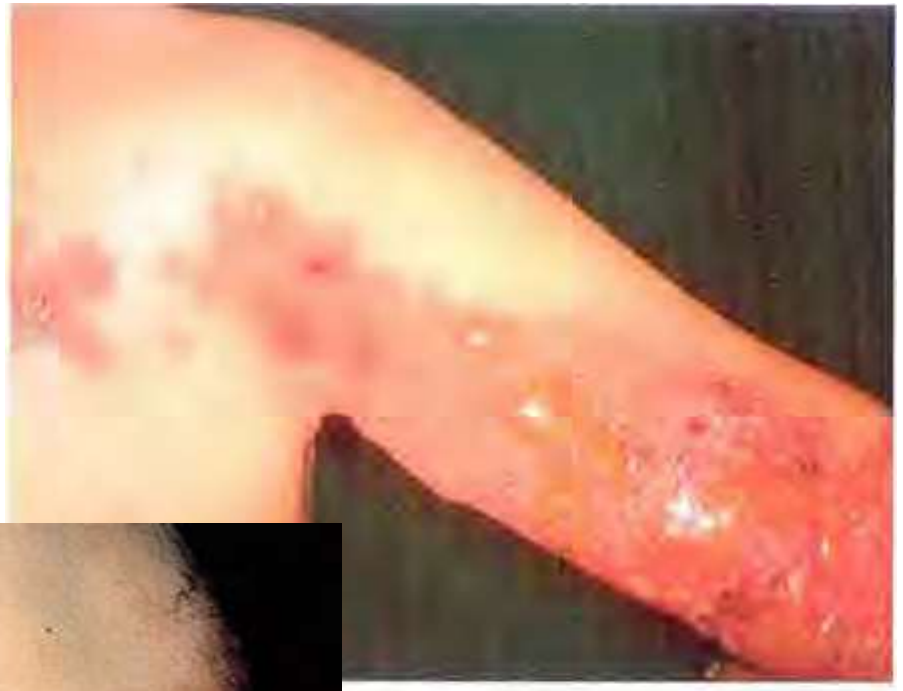
Simptoms and Signs

- Constitutional symptoms: headache, photophobia, and malaise, but significant fever is rare
- Pain: localized sensations ranging from mild itching or tingling to severe pain that precedes the development of the skin lesions by 1-5 days (or occasionally Weeks)
- Cutaneous exam:Skin changes begin with an erythematous maculopapular rash followed by the appearance of clear vesicles. New vesicle formation typically continues for 3-5 days, followed by lesion pustulation and scabbing. Skin lesions heal within 2-4 weeks, often leaving skin scarring and permanent pigmentation changes.
- The cutaneous eruption, appearing in the skin segment innervated by a single sensory ganglion, is unilateral and does not cross the midline. Overlap of lesions into adjacent dermatomes occurs in 20% of patients. The most commonly involved dermatomes are thoracic, followed by cranial (especially trigeminal), lumbar, and cervical; sacral dermatomes are least frequently involved.

Most Common Affected Area







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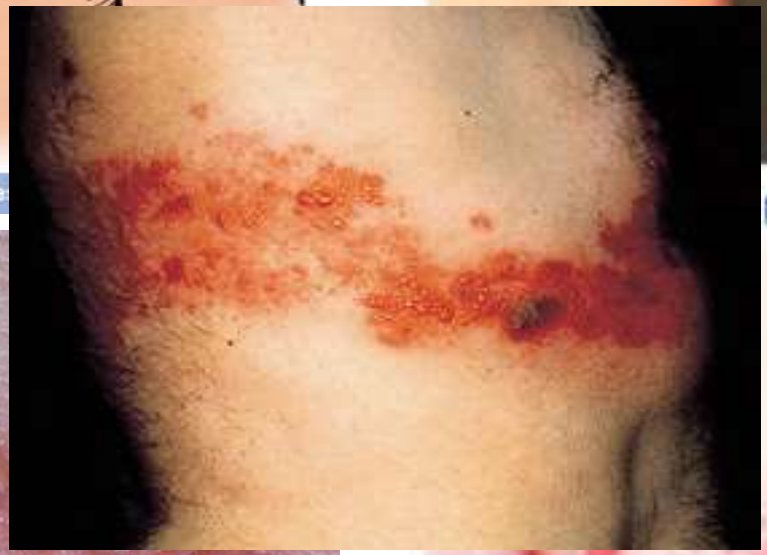
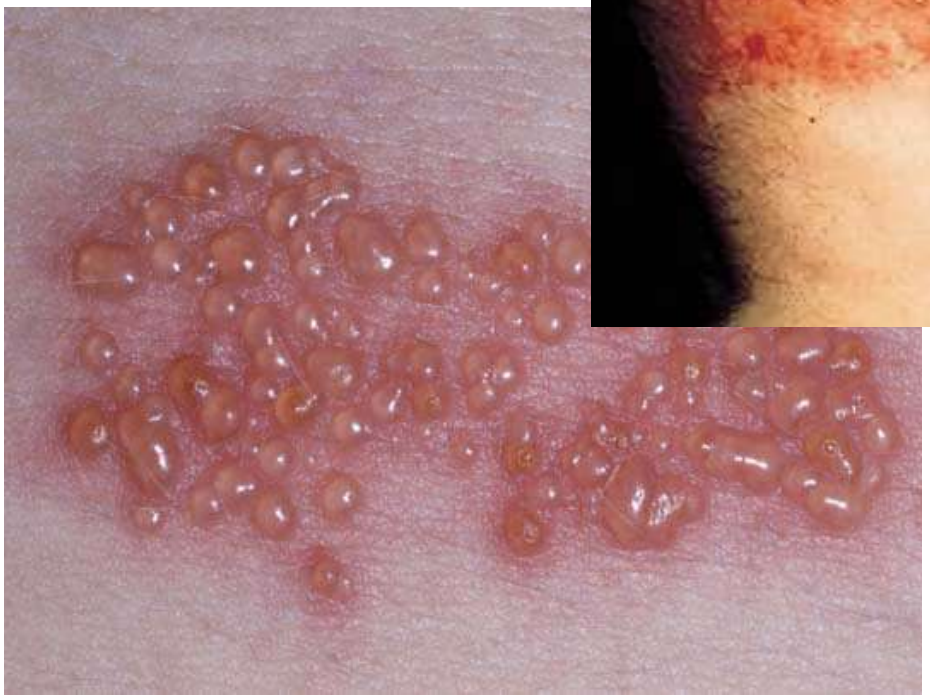
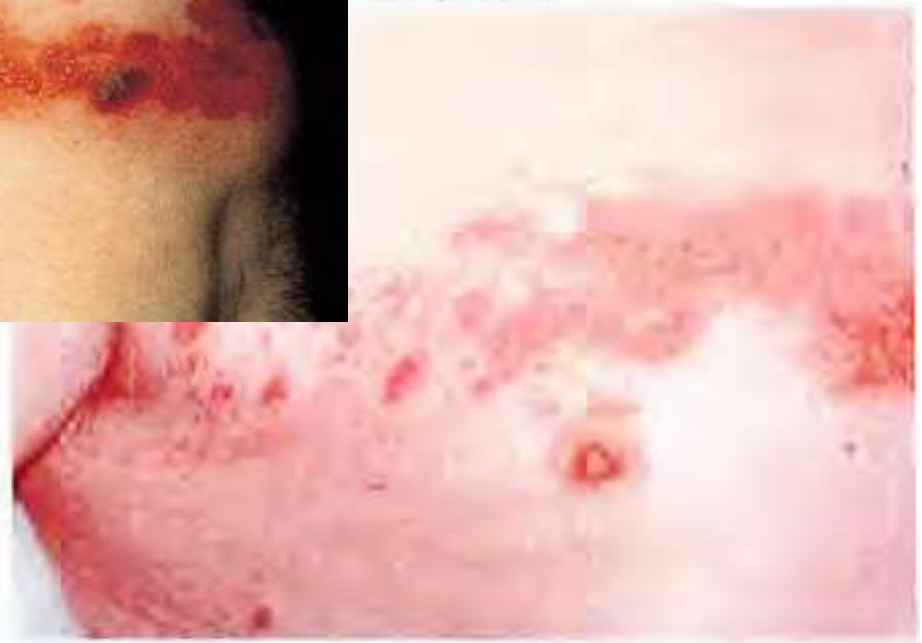






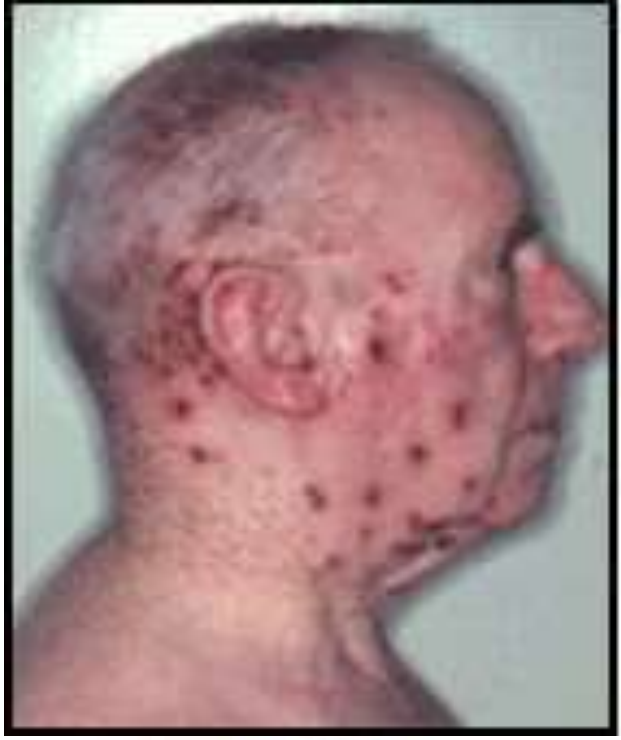
SHINGLES RASH ON THE CHEST

Examination

- Syndromes associated with herpes zoster of the cranial nerves include herpes zoster ophthalmicus (first division of the trigeminal nerve)
- **Ramsay Hunt syndrome** (geniculate ganglion of CN VII, with ear vesicles, diminished taste on the anterior two thirds of the tongue, and ipsilateral facial paralysis).
- Vesicles on the outside of the nose (**Hutchinson's sign**) are usually seen in patients with VZV keratitis

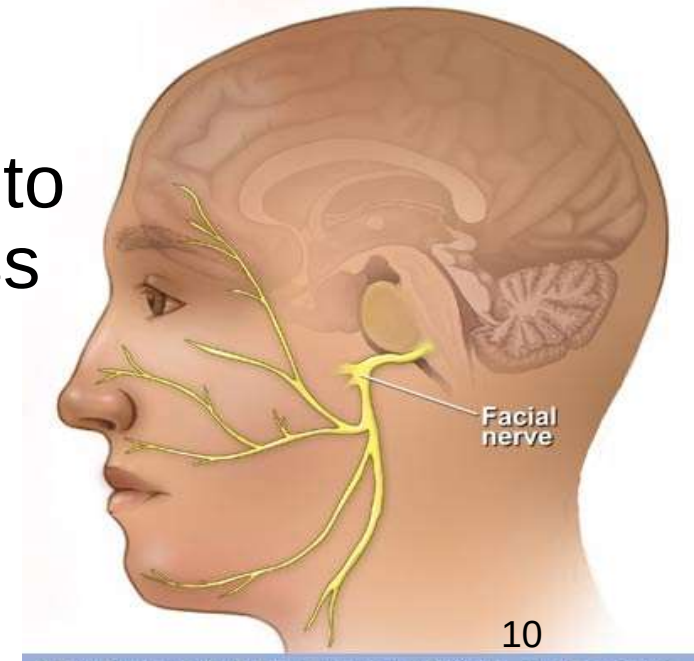
- Neurologic exam: Allodynia (pain provoked by light touch) may be present in the involved dermatome.
- Various neurologic complications can occur during acute herpes zoster, including vasculopathy, myelitis, cranial and peripheral nerve palsies, and polyradiculitis

Complications

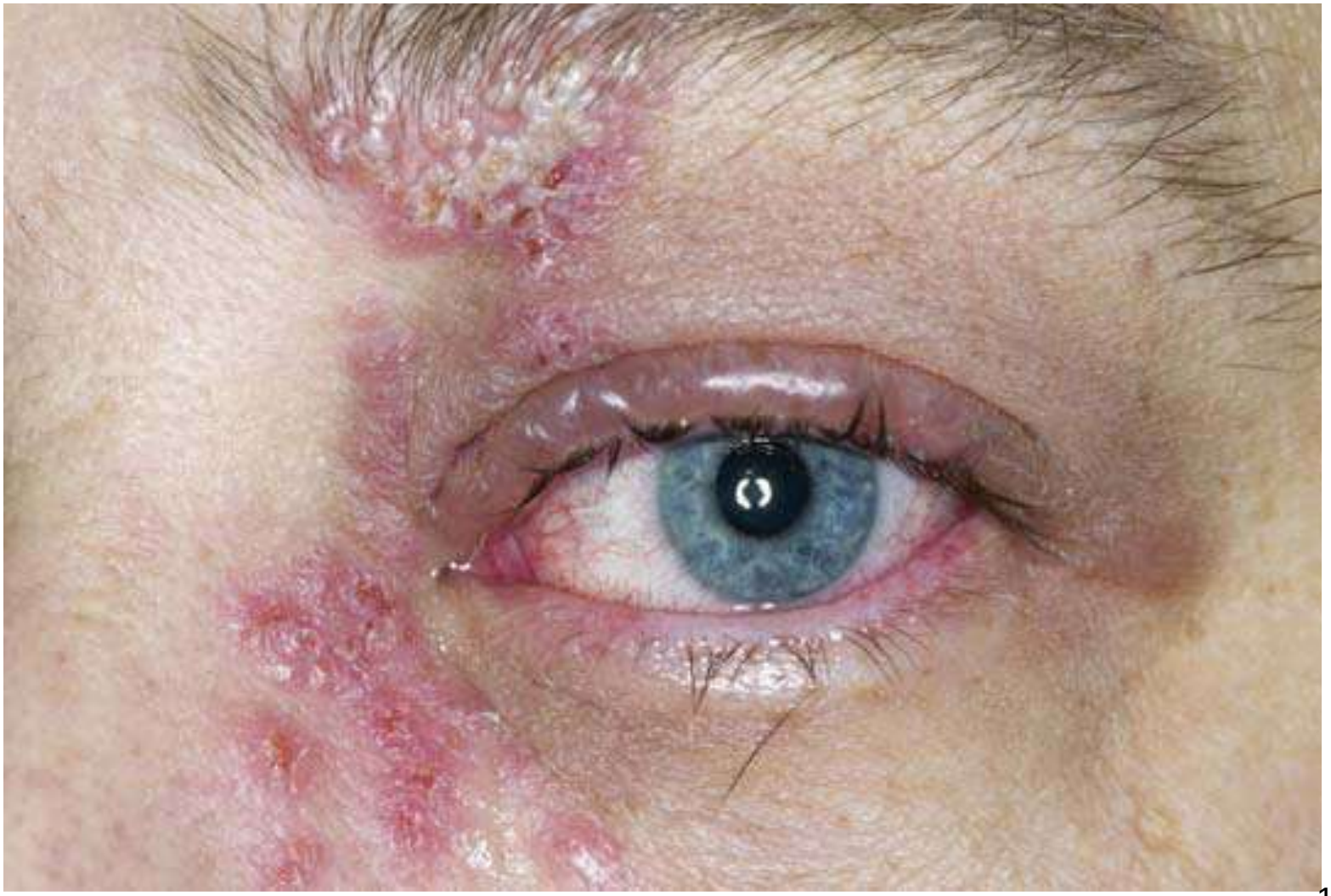


- **Postherpetic neuralgia**
- Inflammation of the brain (**encephalitis**) and other neurological problems.

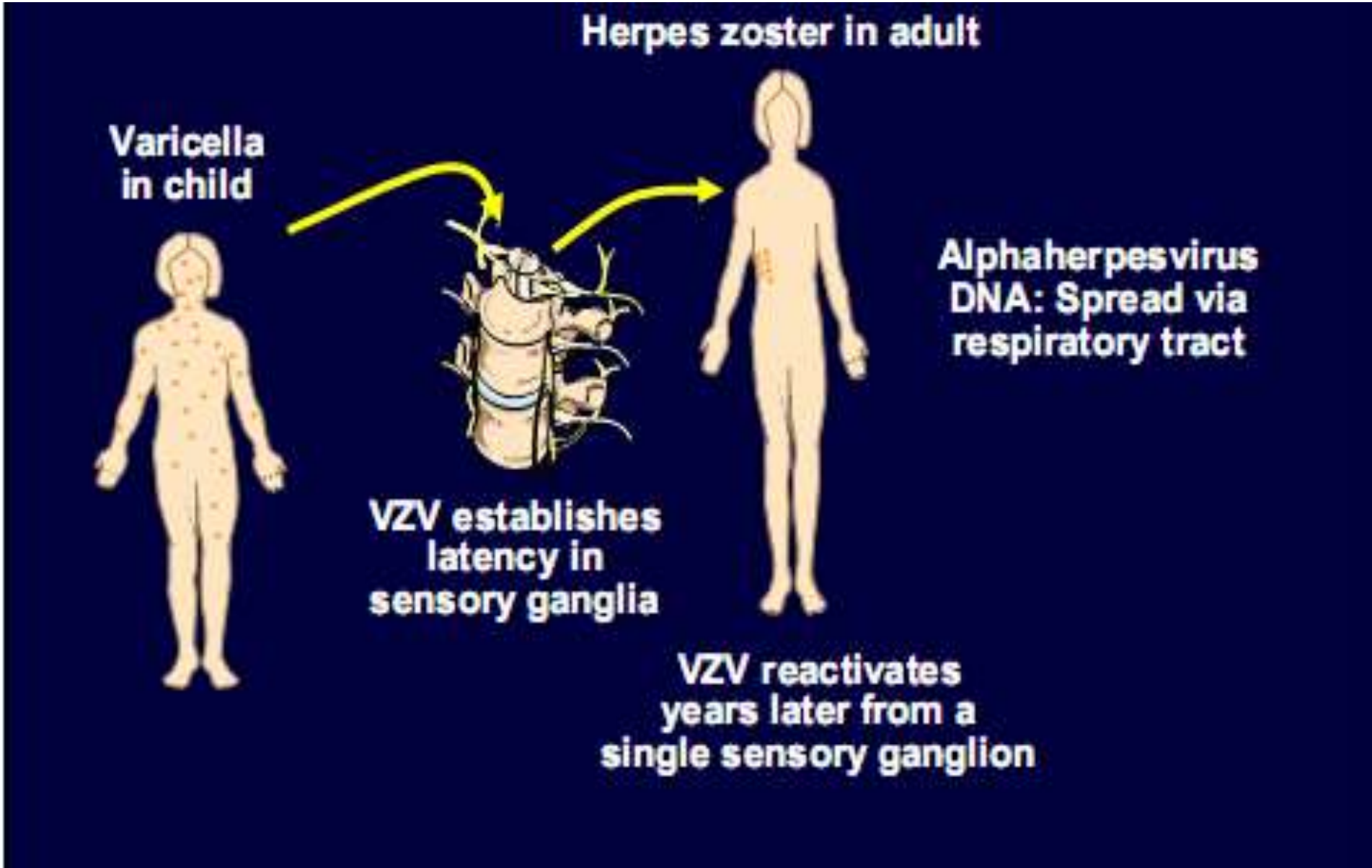
- **Ramsay Hunt syndrome** also called **herpes zoster oticus** is an infection of your **facial nerve**. Ramsay Hunt syndrome can lead to permanent facial muscle weakness and hearing loss.



Herpes Zoster Opthalmicus



Primary Infection, Latency, and Reactivation of Varicella-Zoster Virus



Post Herpetic Neuralgia

- Postherpetic neuralgia is the most common complication of herpes zoster.
- PHN is a severe nerve pain felt in the same area as the shingles rash
- The presence of pain more than a month after the onset of the eruption of zoster.
- PHN is diagnosed if the pain persists or returns, 3 months after the shingles rash started.
- Recently,the term zoster-associated pain has been used to describe all pain that occurs after the onset of the rash.

Risk Factors PHN

- Elderly people
- Being a woman
- People whose immune systems have been compromised.
- Severity of pain during initial stages of the illness
- Severity of rash

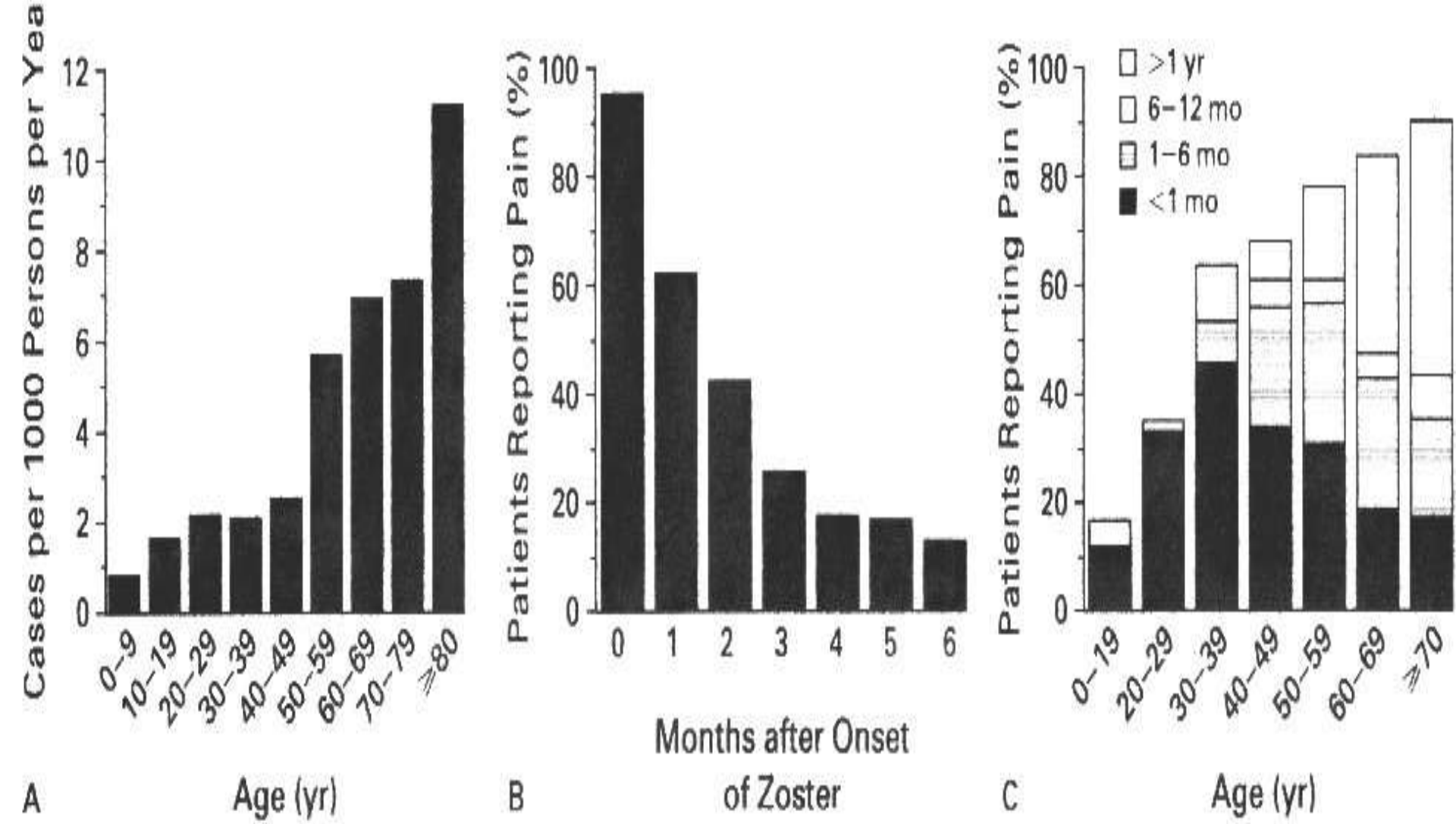
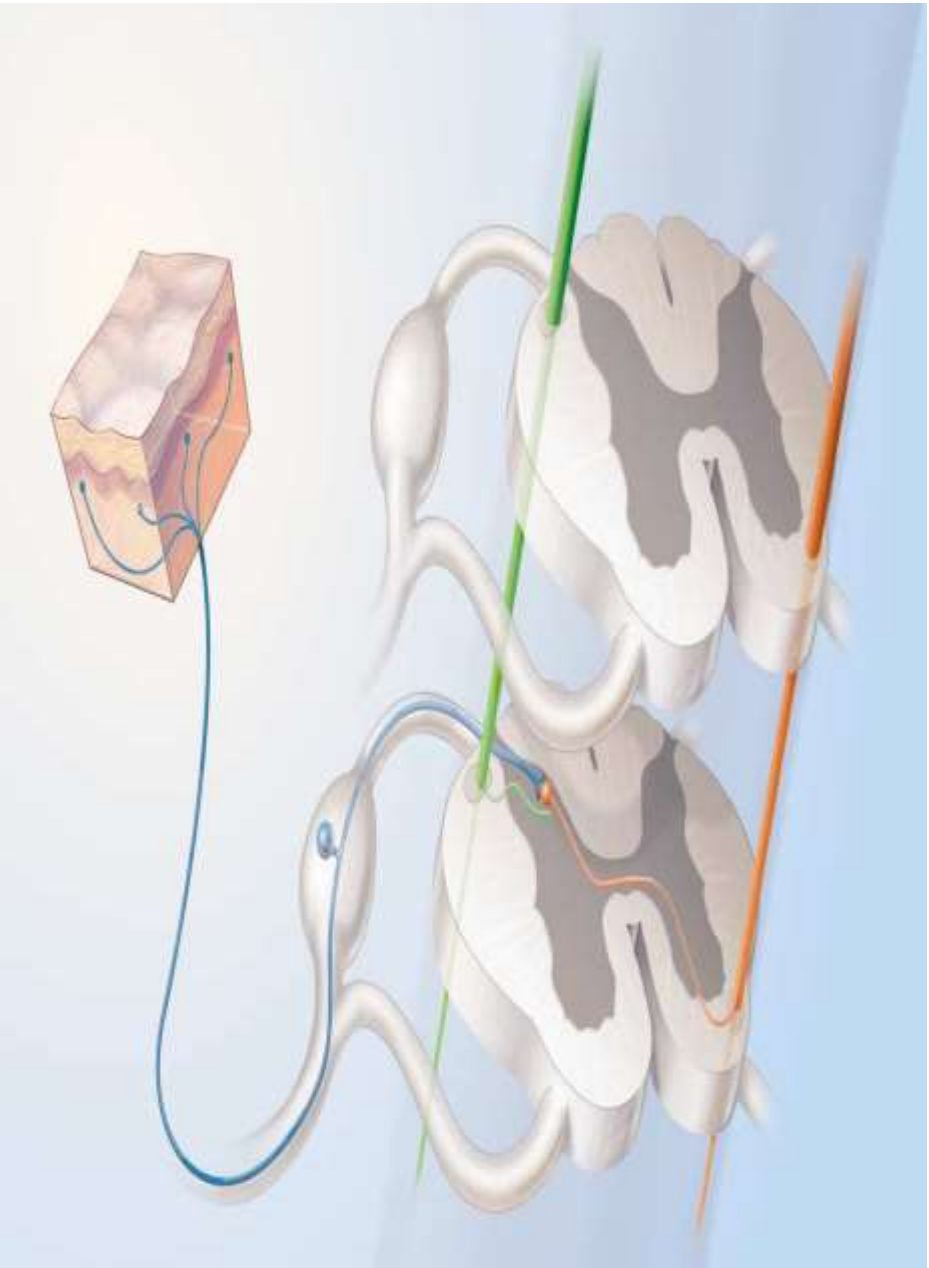
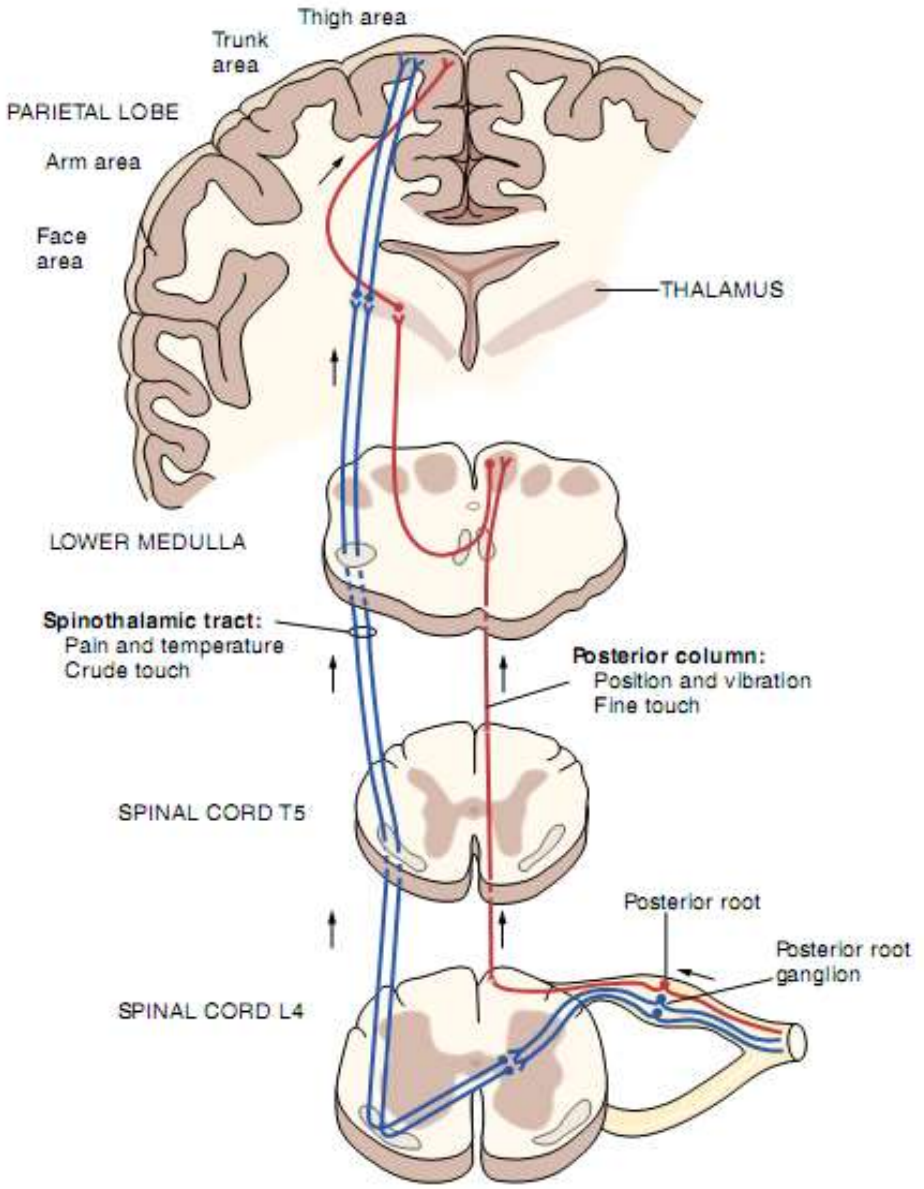


Figure 1. Annual Incidence of Herpes Zoster and Proportion of Patients with Postherpetic Neuralgia.

Panel A shows the annual incidence of herpes zoster per 1000 persons in a general medical practice.[1] Panel B shows the percentage of patients with pain persisting after the onset of the zoster-associated rash. Data are from the placebo group in one large, double-blind treatment study.[7] Panel C shows the proportion of patients with postherpetic neuralgia according to age.[16]

Pathway of Normal Pain Perception.





SENSORY PATHWAYS: SPINOTHALAMIC TRACT AND POSTERIOR COLUMNS

Clinical Presentation of Neuropathic Pain

Neuropathic Pain	Sensation	Fibers
	Touch, Vibration	A-beta
	Pinprick, Sharp Pain, Cold	A-delta
	Warmth, Burning	C-fibers

The pathogenetic mechanisms of PHN

- Have not been clearly understood.

  To date, pathologic interaction between impaired function of afferent A beta-fiber, impaired response of axon reflex, damaged C fiber, and spinal cord hyperreflexity may play a role in HZ pain as well as PHN.

- The nerves may be permanently damaged and this leads to persistent pain. Messages from these damaged nerves to the brain register as pain.

- Peripheral neurons discharge spontaneously, have lower activation thresholds, and display exaggerated responses to stimuli.
- Axonal regrowth after the injury produces nerve sprouts that are also prone to unprovoked discharge.

Histopathological Features

- In acute zoster, the skin is inflamed and already partially denervated, and the dorsal-root ganglion shows inflammation, hemorrhagic necrosis, and neuronal loss.
- Inflammation in peripheral nerves may persist for weeks to months and usually leads to demyelination, wallerian degeneration, and sclerosis.
- Degeneration (atrophy) of the dorsal horn of the spinal cord.

Signs and symptoms

- Extreme sensitivity to light touch, rubbing of clothing, draughts or a slight breeze can cause excruciating pain. This is called allodynia.
- Burning
- throbbing
- Shooting
- Electric shock–like
- a deep aching

- The pain associated with acute zoster and postherpetic neuralgia is neuropathic and results from injury of the peripheral nerves and altered central nervous system signal processing.

Dermatomal distribution

1. Thoracic
2. Cranial
3. Cervical
4. Lumbosacral

HZ Treatment

1. Antiviral drug therapy:

- Antiviral drug therapy accelerates the healing of skin lesions and reduces the duration of pain,
- **High doses of an antiviral drug to reduce the duration and severity of your symptoms.** Oral antiviral medications include acyclovir (Zovirax), valacyclovir (Valtrex) or famciclovir (Famvir).
- For best results, start these medications within 72 hours of the first sign of the shingles rash.

- **Valacyclovir.** In a placebo-controlled trial, valacyclovir and acyclovir were equivalent in terms of accelerating the events of cutaneous healing in patients with herpes zoster, and valacyclovir was superior for shortening the duration of zoster-associated pain and duration of postherpetic neuralgia in immunocompetent adults 50 years and older. A seven-day course of valacyclovir (1 g po tid) is advised.
- **Famciclovir** is significantly in reducing the duration of viral shedding, limiting the duration of new lesion formation, accelerating cutaneous healing and, especially in patients older than 50, reducing the duration of postherpetic neuralgia. A seven-day course of famciclovir (500 mg po tid) is advised
- **acyclovir** (800 mg po 5/d) for 7-10 days. Acyclovir effectively reduces the duration of viral shedding, shortens the duration of new lesion formation, and accelerates the events of cutaneous healing.

2. Oral Corticosteroids

- Oral corticosteroids: prednison, dexamethason, metilprednislon.
- The optimal dosing should consider adding a 10- to 14-day tapering course of oral prednisone, starting at 60 mg daily.
- Corticosteroids should not be used in patients with contraindications, such as diabetes mellitus, osteoporosis, and gastritis, because they can cause potentially serious adverse effects.

3. Narcotic analgesics.

- Short-acting narcotic analgesics such as oxycodone may be prescribed on a schedule for patients who experience acute neuralgic pain during active herpes zoster. For patients with chronic pain, consider prescribing long-acting analgesics, such as controlled-release oxycodone.
- Consider, as well, the addition of the anticonvulsant agent, gabapentin, or the tricyclic antidepressant, amitriptyline, early in the course of herpes zoster to reduce the prevalence and severity of pain.

Treatment of Postherpetic Neuralgia

I. **Pharmacology**:

- Corticosteroids
- Tricyclic antidepressants: amitriptylin, Maprotiline, desipramine
- Anticonvulsant drugs: carbamazepin, phenythoin, gabapentin, pregabalin
- Other drugs such as topical lidocaine and oxycodone
- Topical capsaicin 0.075% .
- Patients with PHN refractory: be considered for intrathecal steroid therapy (epidural injection of 80 mg methylprednisolone acetate and 10 mg bupivacaine), Neurosurgical procedures

Treatment should be started as soon as possible after the onset of rash to prevent post-herpetic neuralgia.

2. Non Pharmacology

- Transcutaneous Electrical Nerve Stimulation (TENS)
- Hypnosis,
- Biofeedback,
- Cognitive and behavioral techniques

PREVENTION Of POSTHERPETIC NEURALGIA

- Corticosteroid
- Antiviral drugs: less than 72 hours
  - Acyclovir: 5 x 800 mg per day during 7 days

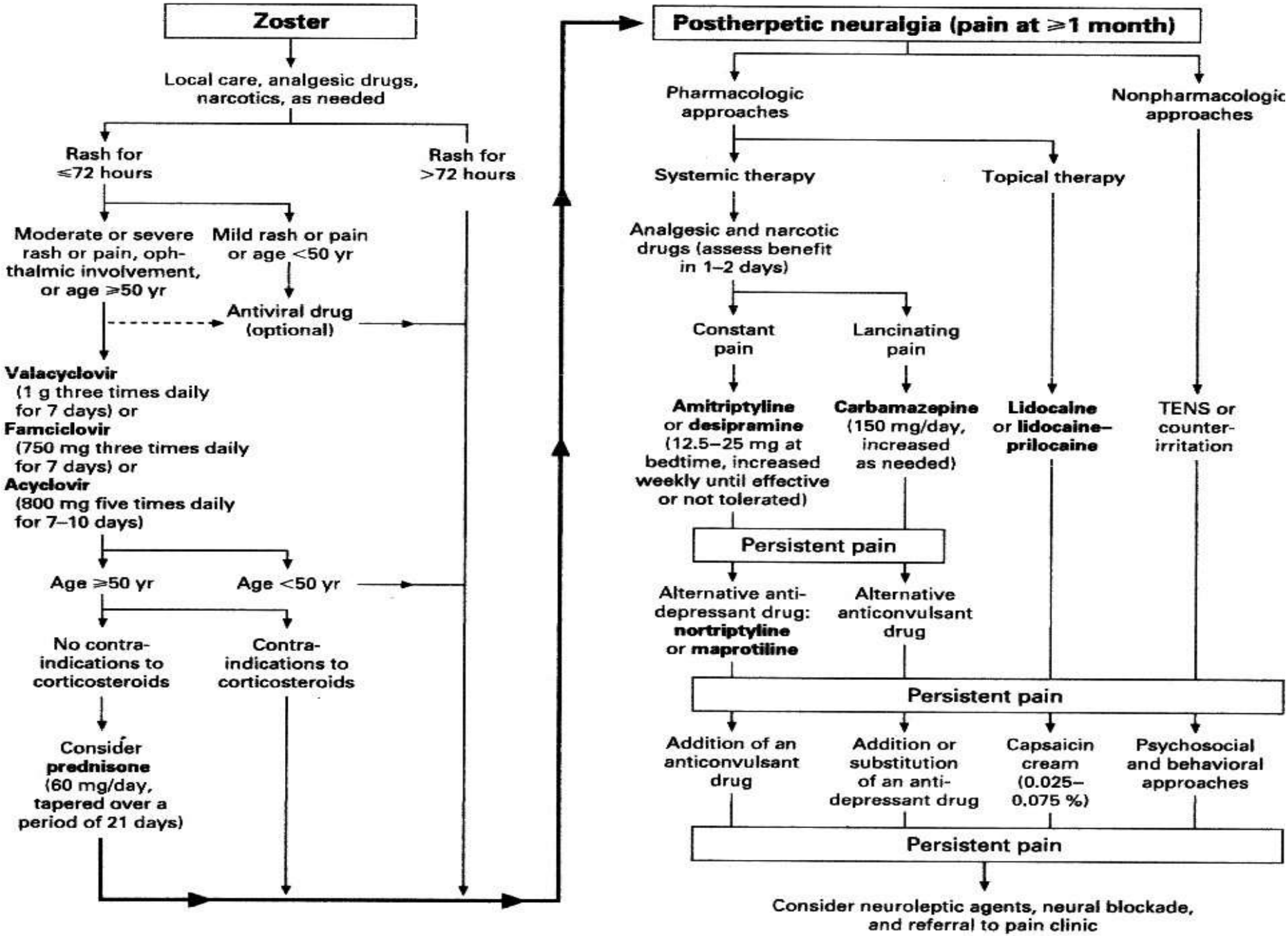


Figure 4. Approaches to the Treatment and Prevention of Acute Zoster-Associated Pain and Postherpetic Neuralgia.

Terminologi

- Nyeri: pengalaman sensorik dan emosional yang yidak menyenangkan akibat kerusakan jaringan, baik aktual maupun potensial, atau yang digambarkan dalam bentuk kerusakan tsb.
- Nyeri neuropatik: nyeri yang didahului atau yang disebabkan oleh lesi atau disfungsi primer pada sistem saraf.
- Neuralgia: nyeri pada distribusi saraf.
- Alodinia: nyeri yang disebabkan oleh stimulus yang secara normal tidak menimbulkan nyeri.
- Hiperalgesia: respon yang berlebihan terhadap stimulus yang secara normal menimbulkan nyeri.