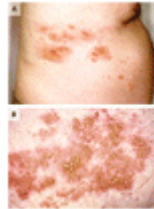


POSTHERPETIC NEURALGIA

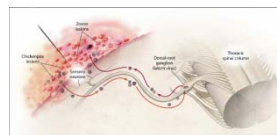
SHEFA INTERDISCIPLINARY PAIN CENTER- NEUROSCIENCE RESEARCH CENTER

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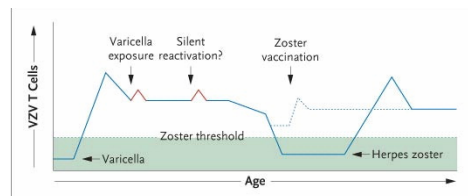
Herpes zoster is a common infectious disease caused by the varicella-zoster virus. Varicella-zoster virus (VZV) is a herpesvirus that causes two distinct clinical syndromes (chicken pox and shingles).



After the primary VZV infection (chickenpox), latent infection is established in the sensory-nerve ganglia. And the dermatomal distribution of skin lesions of shingles distinguishes it from other dermatological disorders.



The virus does not reactivate in all individuals who have had chickenpox. The decline in cell-mediated immunity over time as people age confers a predisposition to the occurrence of herpes zoster in older adults; also any causes which lowers the immunity of the patient makes him/ her prone to the disease.



The histologic changes in the skin lesions are similar to those of varicella. Complications of herpes zoster in immunocompetent hosts include postherpetic neuralgia, encephalitis, myelitis, cranial-nerve palsies, and peripheral-nerve palsies. Postherpetic neuralgia, a persistent pain syndrome occurring after the resolution of the zoster rash, is perhaps the most challenging and debilitating complication; it can last for weeks, months, or even years. Although the development of postherpetic neuralgia can occur at any age, people 50 years of age or older are most likely to have this complication, and more than 40% of people older than 60 years of age who have had zoster have postherpetic neuralgia which is a neuropathic pain.

What is neuropathic pain?

The words the patient in this scenario uses to describe his pain ("burning, searing,...") indicate that he is experiencing neuropathic pain. Neuropathic pain results from damage of the peripheral or central nervous system (or both) and is characterized by multiple poorly understood and complex

underlying mechanisms. The neuropathic pain originates from the pain system and is transmitted by c fibers.

Postherpetic neuralgia is neuropathic pain that persists long after the rash and lesions of acute herpes zoster are healed. The sharp, shooting pain of postherpetic neuralgia occurs along the same dermatomes that were affected by the acute herpes zoster infection. Postherpetic neuralgia affects approximately 10% of patients who have had acute herpes zoster infection and is considered one of the most difficult pain syndromes to treat. The reason that some patients with acute herpes zoster develop postherpetic neuralgia is unknown. However, it is evident that antiviral therapy as soon as possible minimizes the frequency and severity of post herpetic neuralgia.

Treatment of Postherpetic Neuralgia is treatment of neuropathic pain. The first-line analgesics for the treatment of the pain associated with postherpetic neuralgia include selected antidepressants, anticonvulsants, and local anesthetics. It is recommended that Gabapentin be administered on the first signs of post herpetic neuralgia.

In rare cases in which none of the medications show effective, spinal stimulators may be used.

The VZV vaccine, originally developed and licensed as the “chickenpox vaccine” to prevent varicella, is a live attenuated vaccine that is effective in preventing primary infection with wild-type VZV. Several small clinical studies have shown that immunization with a varicella vaccine boosts waning cell-mediated immunity in older adults. The preventive effect of the zoster vaccine is thought to be a consequence of its boosting effect on an older person's cell-mediated immunity to VZV, mimicking the immunologic benefits of the exposure of a VZV-immune adult to chickenpox. On May 25, 2006, the Food and Drug Administration licensed the zoster vaccine for the prevention of herpes zoster in persons 60 years of age or older. The vaccine is more efficacious in preventing herpes zoster among persons who were 60 to 69 years of age than among those who were 70 years or older.

In one study, the incidence of postherpetic neuralgia was 67% lower among subjects who received the vaccine than among those who received placebo (0.5 case per 1000 person-years vs. 1.4 cases per 1000 person-years, $P < 0.001$). The median duration of pain among subjects in whom herpes zoster developed was shorter in the vaccine group than in the placebo group (21 days vs. 24 days, $P = 0.03$), and the degree of pain also was lower among the vaccine recipients ($P = 0.008$). However, it prevented postherpetic neuralgia to a greater extent among those who were 70 years or older than among those who were 60 to 69 years old.