Herpes Zoster

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KEYWORDS

• Herpes zoster • Varicella-zoster virus • Postherpetic neuralgia • Elderly • Aged

KEY POINTS

- The strongest risk factors for herpes zoster are aging and suppression of cellular immunity. The increase in the likelihood of herpes zoster with aging starts around 50 to 60 years of age and increases markedly into late life.
- Laboratory diagnostic testing is indicated when differentiating herpes zoster from herpes simplex virus, for suspected organ involvement, and for atypical presentations, particularly in the immunocompromised host. Polymerase chain reaction is the preferred diagnostic test.
- The goal of the treatment of herpes zoster in older adults is to decrease the length of the acute attack and to reduce pain by the use of early antiviral therapy (acyclovir, famciclovir, valacyclovir), scheduled analgesia, and, if the pain is not adequately controlled, adjunctive agents.
- Lidocaine patch 5%, gabapentin, pregabalin, opioids, and tricyclic antidepressants constitute first-line therapies for the management of postherpetic neuralgia.
- The live attenuated zoster vaccine is recommended for all immunocompetent adults 60 years and older by the Advisory Committee on Immunization Practices and the US Centers for Disease Control and Prevention for the prevention of herpes zoster and postherpetic neuralgia.

INTRODUCTION

Herpes zoster is a neurocutaneous disease that is caused by the reactivation of varicella-zoster virus (VZV) from a latent infection of dorsal sensory or cranial nerve ganglia after primary infection with VZV earlier in life. VZV is a double-stranded DNA herpesvirus with a genome that contains at least 70 gene products.[1] VZV expresses gene products and attempts replication throughout life, but cellular immunity is critical

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to containing VZV. With aging-related decline in cellular immunity to VZV, the virus may escape cell-mediated immune containment and spread in the affected ganglia and sensory nerves to the skin.

EPIDEMIOLOGY

The estimated incidence of herpes zoster in persons older than 65 years varies from approximately 10 to 14 cases per 1000 per year.[2–4] The lifetime incidence of herpes zoster is estimated to be about 20% to 30% in the general population, and may be as high as 50% among those surviving to 85 years or higher.[5] Current population figures and herpes zoster incidence data yield estimates of about 1 million new cases of herpes zoster each year in the United States.[6] The incidence of recurrent herpes zoster is not as well established but the frequency of recurrent events is higher in immunocompromised individuals.[7,8] In one study, the frequency of recurrent herpes zoster was 5.7% among immune-competent individuals.[7]

The strongest risk factors for herpes zoster are aging and suppression of cellular immunity. The increase in the likelihood of herpes zoster with aging starts around 50 to 60 years and increases into late life in individuals older than 80 years.[2,3] Immunocompromised patients at risk for herpes zoster include persons with human immunodeficiency virus infection, Hodgkin's disease, non-Hodgkin's lymphomas, leukemia, bone marrow and other organ transplants, systemic lupus erythematosus, rheumatoid arthritis, and those individuals taking immunosuppressive medications, including tumor necrosis factor inhibitors.[5,6,9] Other risk factors include white race, female sex, and physical trauma.[5,10,11] The childhood varicella vaccination program in the United States does not appear to have affected the incidence of herpes zoster.[12]

Patients with herpes zoster who have a vesicular rash may transmit VZV via direct contact, airborne route, or droplet nuclei to seronegative, nonimmune individuals.[13] These individuals may then subsequently have varicella, usually within 10 to 21 days after contact with a case. In one study of school and day care settings, of 290 herpes zoster cases reported, 27 (9%) resulted in 84 secondary varicella cases.[13] If the rash is only maculopapular or crusted, there is no danger of VZV transmission. Important groups at risk for varicella from contact with herpes zoster patients include children who have not received the varicella vaccine or who have had an insufficient response to the vaccine and susceptible health care workers and staff in hospital or in nursing homes particularly if they are pregnant or immunocompromised. The exposure of a latently infected individual to herpes zoster does not cause herpes zoster or varicella. Nearly all older adults are latently infected with VZV.

CLINICAL FEATURES

VZV reactivation and spread in the affected sensory ganglion and peripheral sensory nerve evokes a cellular immune response and neuronal inflammation and destruction.[6] Before VZV reaches the skin, the patient experiences prodromal sensations in the affected dermatome, such as aching, burning, or lancinating pain or itching or tingling. Prodromal symptoms baffle patients and physicians by imitating other painful conditions in older persons (ie, migraine headaches, trigeminal neuralgia, myocardial infarction, cholecystitis, biliary or renal colic, appendicitis, lumbosacral strain, or pulled muscles). One clue to incipient herpes zoster is sensitive skin in the affected dermatome before the rash breaks out. The prodrome usually lasts a few days. VZV may not reach the skin in some patients, which results in a unilateral, dermatomal neuralgia without a rash. This condition is known as *zoster sine herpete*.