CLINICAL MANAGEMENT extra

Herpes Zoster: Prevention, Diagnosis, and Treatment—2008 Update

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PURPOSE

To provide the wound care practitioner with a review of strategies to diagnose, prevent, and manage herpes zoster.

TARGET AUDIENCE

This continuing education activity is intended for physicians and nurses with an interest in skin and wound care.

OBJECTIVES

After reading this article and taking this test, the reader should be able to:

1. **Identify signs and symptoms of herpes zoster.**
2. **Describe herpes zoster complications and treatment.**
3. **Discuss means of preventing herpes zoster.**

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Herpes zoster (HZ), commonly known as shingles or zoster, results from the reactivation of the varicella-zoster virus (VZV). The likelihood of VZV reactivation occurring in an individual is related to both age and immune status. Classic presentation involves vesicular lesions in a unilateral dermatomal distribution. Antiviral medications are used to treat HZ. The most common complication of HZ is postherpetic neuralgia (PHN). Medications used to treat PHN include topical anesthetics, oral analgesics, tricyclic antidepressants (TCAs), and anticonvulsants. Use of zoster vaccine live (Zostavax), a new HZ vaccine, may decrease the severity and duration of PHN.

CASE STUDIES

Case 1: Patient A, a 35-year-old man with a history of diabetes, presents to the office complaining of a painful ''insect bite'' on the left lateral aspect of his forehead after being outside at a baseball game.

Case 2: Patient B, a 75-year-old woman, presents complaining of severe right lower quadrant/flank pain. Laboratory and

imaging studies are negative. Her severe pain is of unknown origin, so she is admitted to the hospital.

Case 3: Patient C, a 78-year-old man, comes to the office complaining of pain in the second lumbar dermatomal pattern, especially in the right thigh. He denies any back injury or previous back problems. Physical examination is negative with the exception of hyperesthesia of the skin over the area of pain.

Case 4: Patient D, a 32-year-old man, presents with a painful vesicular rash on the left side of his neck. His medical history is negative. He denies having any new stressors in his life.

Although the differential diagnoses for each of these cases may be vastly different, the one common diagnosis for all 4 cases is HZ.

DEFINITION/ETIOLOGY

HZ results from the reactivation of the VZV, which, as a primary infection, produces varicella (chickenpox).[1] As with other viruses in the herpes family, latency and reactivation are characteristic features of VZV.[2] After primary infection with VZV causes varicella, the virus becomes latent in the cranial nerve, dorsal root, and autonomic ganglia of the nervous system.[3]

Cell-mediated immunity usually prevents VZV from emerging from latency. However, should cell-mediated immunity decline, whether from normal age-related decrease or from an immune-deficiency state, the VZV can reactivate and cause HZ to occur.[4]

INCIDENCE/EPIDEMIOLOGY

It is estimated that more than 90% of the US population has serologic evidence of VZV infection and are at risk for developing HZ.[5] The likelihood of VZV reactivation occurring in an individual is related to both age and immune status. The incidence of disease in patients older than 60 years is 2 to 3 times higher than that in younger persons[2] with the highest rates found in those older than 75 years (10 cases/1000 individuals).[6] When one considers these statistics in conjunction with the aging of the US population, the potential number of HZ cases and subsequent complications that will be seen in primary care is of great concern.

In addition to age, an essential variable in the VZV reactivation is the status of the individual's immune system. The VZV is most likely to reactivate during times of stress, trauma, and other precipitating factors.[7] Stress can be psychological, such as problems with relationships, in employment situations, or with financial concerns, or stress can be physical, as with the numerous conditions leading to a compromised immune system.

The incidence of HZ in immunosuppressed patients is as much as 100 times higher than that in immunocompetent individuals. This is particularly true in those with depressed T-cell function, such as patients who have lymphomas, leukemias, solid neoplasms, organ transplants, or AIDS, and those receiving chemotherapy or radiation therapy.[2] A question continues as to whether the presence of diabetes mellitus is considered a risk factor for HZ. Information on this possible relationship is extremely limited in the literature[8] (case 1).

CLINICAL PRESENTATION

Patients may first exhibit prodromal symptoms characteristic of many viral infections, including generalized malaise, fever, chills, myalgias, headache, or stomach upset.[9] Patients presenting with these symptoms may be initially diagnosed with a viral syndrome, and only symptomatic treatment is suggested.

The classic presentation of HZ is a burning or tingling pain in the skin (acute neuritis), although some patients will complain of numbness or pruritus. The skin may be very sensitive to touch (hyperesthesia). These symptoms are usually noted from 1 day to 3 weeks before any noticeable skin eruption and may be mistaken for other problems.[2,4,9,10] The pain that occurs before the appearance of the rash can be quite severe (a condition known as zoster sine herpete) and may be misdiagnosed as being of cardiac origin, pleurisy, herniated disc, gastrointestinal, gynecologic, or musculoskeletal[4,10] (cases 2 and 3).

The skin lesions begin as macules and papules on an erythematous base (Figure 1). Over a period of 3 to 5 days, these lesions develop into vesicles (Figure 2). Because HZ is a reactivation of the VZV in a nerve root, the vesicular eruption typically follows a dermatomal pattern affecting a cranial or

Figure 1.
SINGLE CLUSTER OF LESIONS

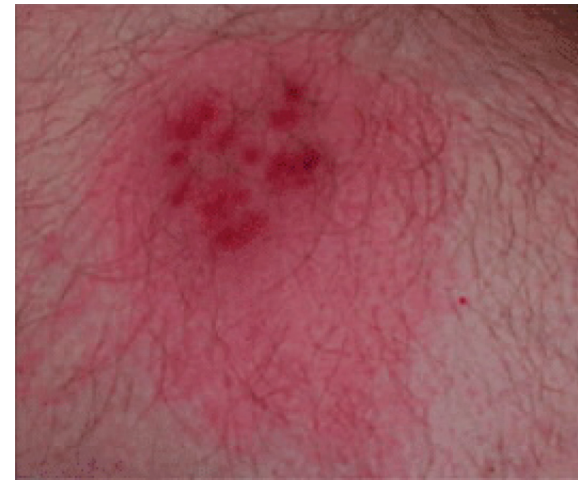
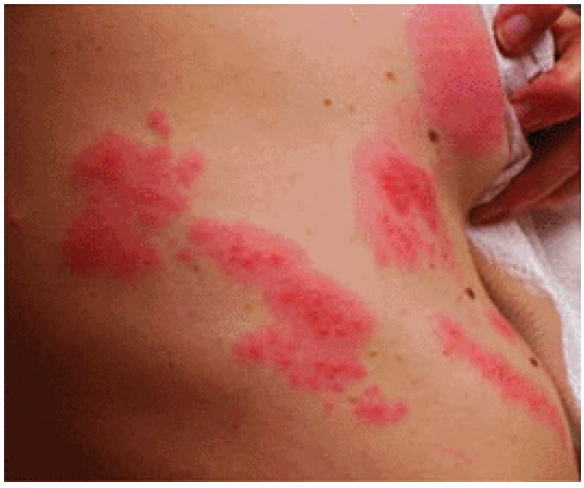


Figure 2.
VESICULAR ERUPTION



spinal nerve distribution. This eruption is usually unilateral and most often appears on the thorax; the T5 and T6 dermatomes are most commonly affected[2,4,10,11] (Figure 3).

Individual attacks are usually limited to 1 to 3 dermatomes, although a few isolated skin lesions at sites distant from this area are not uncommon.[2] HZ infection can develop into a disseminated vesicular eruption in patients with AIDS.[12] Involvement of multiple dermatomes or bilateral aspects of the same dermatome (crossing the midline) or more than 20 lesions outside the affected dermatome may reflect viral dissemination, which can be fatal.[4,10] Although rare, an individual may experience HZ more than once with different dermatomal areas usually affected. In patients with multiple episodes of reported HZ, consideration should be given as to whether this is truly herpes zoster, or if the cutaneous manifestations are due to herpes simplex virus, another virus that can mimic herpes zoster.

After formation of vesicles, the lesions usually rupture and release VZV, which can be transmitted to susceptible individuals. Some lesions may umbilicate. Eventually, the lesions crust over and become dry and dark in color.[2,10] Typically, the symptoms and lesions resolve over 10 to 15 days, although some lesions may require a month to completely heal.[10]

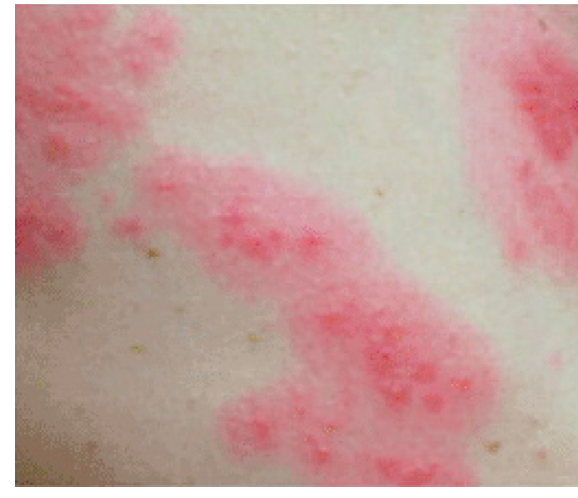
Cranial nerves can also be the location of VZV reactivation. The trigeminal (fifth cranial) nerve is affected in HZ ophthalmicus, which may appear weeks to months after resolution of other HZ symptoms.[10] Any branch of the nerve may be affected, although the frontal branch within the first division of the trigeminal nerve is most commonly involved. This branch innervates nearly all the ocular and periocular structures.[10,13] Common manifestations include conjunctivitis, episcleritis, keratitis, anterior uveitis, glaucoma, retinitis, choroiditis, optic neuritis, retrobulbar neuritis, and extraocular muscle palsies.[4,10] When the nasociliary branch is involved, vesicles may appear on the tip or side of the nose (Hutchinson sign). Such a presentation is a predictor for possible serious complications, such as ocular inflammation and corneal denervation.[10,13,14] If the outbreak is on the face, especially around the eyes or nose, an ophthalmology consultation should be obtained as soon as possible.[15]

Ramsay Hunt syndrome, also known as HZ oticus, is an infection of the facial (seventh cranial) nerve.[2,10] Vesicular eruptions may manifest on the pinna or tragus in the auditory canal and on the tympanic membrane, as well as anywhere in the facial nerve distribution.[10,16] The patient may experience hearing impairment, nystagmus, vertigo, gait disturbances, nausea, vomiting, tinnitus, or facial nerve palsy (mimicking Bell palsy).[10,16] Patients may lose taste sensation in the anterior two-thirds of the tongue.[10]

DIAGNOSIS

HZ can usually be diagnosed solely on clinical grounds. However, depending on when the patient seeks treatment, the lesions may appear differently than expected. Differential diagnoses include herpes simplex, allergic dermatitis, contact dermatitis, impetigo, cellulitis, insect bites, varicella, and atopic dermatitis.[2] When in doubt, the most useful test for

Figure 3.
OVERLAPPING DERMATOMAL PATTERN



diagnosing and confirming HZ is direct fluorescent antibody assay of cells scraped from ulcerative lesions.[17]

TREATMENT PLAN

Antiviral medications are the standard of practice in the management of herpes viral infections.[7] These medications are used to decrease the duration of the HZ rash and the severity of pain. It is important to initiate antiviral therapy while new lesions are being formed no later than 72 hours after eruption of the rash.[4] Antiviral therapy may be of benefit after 72 hours in patients who are at high risk of PHN, especially if vesicles are still present.[18]

The health care provider must take several factors into consideration when choosing an antiviral medication. Antiviral medications vary in their dosing frequency and in their cost (Table 1). They are all deoxyribonucleic acid polymerase inhibitors. Acyclovir (Zovirax) has lower oral bioavailability than the other 2 medications and requires either higher dosing or intravenous administration for severe infections.[7] Valacyclovir (Valtrex) is a prodrug of acyclovir; it does not have any antiviral activity until it is biotransformed into acyclovir.[7] This transformation gives valacyclovir improved bioavailability over that of acyclovir, dosing frequency is reduced to 3 times a day, and oral administration is comparable to intravenous acyclovir. Famciclovir (Famvir) has a dosing schedule of 3 times a day and has better bioavailability than acyclovir and valacyclovir.[4]

The use of corticosteroids in the treatment of HZ remains controversial, because no large-scale studies have investigated their use. However, smaller clinical trials have suggested benefit from combination therapy of antiviral plus corticosteroid in terms of acute pain, function, and quality of life.[6] Corticosteroids are most often used to decrease the neuritis causing the acute herpetic pain.[4] Because of the immunosuppressive quality, corticosteroid use is recommended only for persons older than 50 years who have no relative contraindications, such as diabetes, hypertension, or glaucoma.[2]

Analgesic use will vary, depending on the patient's level of pain. For mild-to-moderate pain, over-the-counter analgesics may be sufficient. However, for severe pain, an opioid analgesic administered on a regular schedule may be needed.[4] A single epidural injection may be considered for use in treating severe, acute shingles pain in patients not responding to standard analgesic therapy.[19] Topical preparations may also be used. With open lesions, lotions containing calamine may help reduce pain and pruritus. Once the lesions have crusted over, capsaicin cream may be used.[4]

A part of treating HZ is determining the trigger for reactivation of the VZV. HZ in persons younger than 50 years may be an indicator of an immunocompromised state. Therefore, younger patients with HZ should be assessed for evidence of immunodeficiency, including HIV.[10] Consideration should also be given to the presence of diabetes mellitus or malignancy. In case 4, there was nothing in the patient's medical history or in his current life situation that would suggest the presence of an immune-deficient state. Laboratory results, including blood glucose level, were within normal limits. The only positive in the patient's family history was a grandparent who was diagnosed with colon cancer after age 60 years. When a screening colonoscopy was performed, an early adenocarcinoma of the colon was discovered. In this case, the patient was actually fortunate to have had HZ.

COMPLICATIONS

The most common complication of HZ is PHN, in which the pain associated with acute HZ becomes chronic.[10] PHN may be severe, particularly in the elderly. In fact, PHN has been associated with a high rate of suicide in older patients experiencing chronic pain.[15]

Overall, PHN occurs in 9% to 14% of patients with HZ. As age increases, however, the risk and duration of PHN also increase.[3] Greater than 50% of all PHN patients are older than 60 years, and 75% are older than 75 years.[6]

PHN impacts the quality of life in older adults in many ways. The pain can affect the person's ability to perform basic activities of daily living, including dressing, bathing, eating, and ambulating. Physical effects of PHN include chronic

Table 1.
HZ ANTIVIRAL MEDICATIONS

Generic Name	Brand Name	Dosage[a]	Approximate Cost[b]
Acyclovir	Zovirax	800 mg P.O. 5×/day × 7–10 days	$29–$41 (generic); $256–$366 (brand)
Famciclovir	Famvir	500 mg P.O. every 8 h × 7 days	$179
Valacyclovir	Valtrex	1,000 mg P.O. t.i.d. × 7 days	$196

[a]Treatment recommendations obtained from http://www.epocrates.com. Dose adjustments are recommended for patients with a creatinine clearance of less than 50 mL/min.
[b]Costs based on prices quoted on http://www.drugstore.com. Actual cost to patient will depend on insurance coverage for prescriptions and prescription filling fees.

fatigue, anorexia, weight loss, physical inactivity, and insomnia. Psychological effects include depression, anxiety, and difficulty concentrating. All of these effects impact the person's ability and desire to participate in social gatherings.[6]

PHN may be refractory to treatment and persist for months to years.[2] Treatments include topical anesthetics, oral analgesics, TCAs, and anticonvulsants.

Topical anesthetics include capsaicin cream and lidocaine (Lidoderm) patches. Capsaicin cream causes the release of substance P from the skin, a neuropeptide that is released from pain fibers in response to trauma. Once substance P is depleted from the nerve fibers, analgesia occurs. Disadvantages to capsaicin use are the burning sensation it produces, which may be intolerable to some patients, and the need for it to be applied 3 to 5 times a day to be effective. The cream should not be applied to open lesions.[4,10,20] Use of a lidocaine patch can also provide temporary relief of PHN for 4 to 12 hours with minimal systemic absorption. Adverse effects include local skin reactions such as erythema.[4,6,9,20]

Oral opioid analgesics may also be used to treat PHN. Opioid use is recommended for short-term use in patients with severe pain that has not improved with other modalities. Adverse effects of opioids include nausea, constipation, loss of appetite, and sedation.[4,6,20]

TCAs, such as amitriptyline and nortriptyline, decrease pain by inhibiting spinal neurons involved in pain perception. This is accomplished by blocking the reuptake of norepinephrine and serotonin. The primary disadvantage of TCA use is anticholinergic adverse effects, including dry mouth, constipation, urinary retention, and blurred vision. Nortriptyline has fewer anticholinergic effects than amitriptyline and thus may be better tolerated. Other adverse effects of TCAs include orthostatic hypotension, dysrhythmias, and weight gain. TCAs are contraindicated in documented hypersensitivity, administration of monoamine oxidase inhibitors in the previous 14 days, history of seizures, cardiac dysrhythmias, glaucoma, and urinary retention. Careful consideration is required before prescribing TCAs, particularly to elderly patients.[4,6,10,20]

Anticonvulsants, primarily gabapentin (Neurontin) and pregabalin (Lyrica), have been shown to significantly reduce the pain intensity and duration of PHN and seem to be equally effective. These medications have a good safety profile, especially among older patients. Adverse effects include dizziness, somnolence, ataxia, and peripheral edema. The medication is initially given at a low dose once a day and titrated to 3 times a day, with individual dosing based on efficacy and patient tolerance.[2,4,6,20]

Other treatment modalities include transcutaneous electric nerve stimulation, biofeedback, acupuncture, and nerve blocks.[4,6,20] Consultation with a pain specialist may be needed.[10]

PATIENT EDUCATION/PREVENTION

The nurse practitioner (NP) should explain to the patient that HZ is not caused by exposure to someone with varicella. However, people with no immunity to VZV (those who have not had chickenpox) who are exposed to someone with HZ can contract varicella.[2,9] Because the HZ lesions contain VZV, patients are considered infectious until lesions are dried. Anyone who has not previously had varicella is at risk of acquiring this virus. Pregnant women and immunosuppressed patients have the highest risk of serious complications.[10]

Nosocomial transmission of VZV has been reported. Thus, hospitalized patients with HZ should be isolated to prevent the spread of virus to other susceptible persons.[2] The route of transmission may be via direct contact of the skin or exposure to dressings or clothing that was soiled with blister fluid from such individuals.[21] The Centers for Disease Control and Prevention include HZ under those illnesses requiring airborne (for disseminated disease) and contact isolation precautions in the hospital.[22]

Primary prevention of varicella is through administration of varicella virus vaccine live (Varivax). Varicella vaccine recommendations were updated in January 2007. The first dose should be administered at age 12 to 15 months, and a newly recommended second dose should be administered at age 4 to 6 years. The second dose can be administered at an earlier age, provided the interval between the first and second dose is at least 3 months.[23] The vaccine can be administered to susceptible (previously unvaccinated) individuals 13 years of age and older, with 2 doses required and administered 4 weeks apart.[23]

Susceptible immunocompromised patients who are exposed to infected persons should receive prophylaxis with varicella-zoster immune globulin (1 dose up to 4 days after exposure) to prevent or modify clinical illness.[2] This immune globulin is prepared from the antibody-rich blood of persons who have recently recovered from varicella.[9]

ZOSTER VACCINE

A zoster vaccine, Zostavax, which contains live attenuated VZV, protects against the development of HZ by boosting VZV immunity using active immunization.[24] The efficacy of the vaccine was investigated through the Shingles Prevention Study involving 38,546 patients 60 years of age or older. This study found markedly reduced morbidity from HZ and PHN among older adults receiving the HZ vaccine.[25]

The zoster vaccine is indicated for prevention of HZ in individuals 60 years of age and older.[24] It is not indicated for

the treatment of HZ or PHN, or for the prevention of PHN in persons with acute zoster.[1,24,26] According to the Advisory Committee on Immunization Practices (ACIP), zoster vaccine is recommended for all persons aged 60 years who have no contraindications, including persons who report a previous episode of zoster or who have chronic medical conditions.[26]

As a part of preventive health care, patients age 60 years and older should be offered the zoster vaccine at the first clinical visit. Zoster vaccination is not recommended for persons of any age who have received varicella vaccine. Zoster vaccination of persons who have severe acute illness should be postponed until recovery.[26]

The vaccine can be administered with other vaccines (eg, Td, Tdap, and pneumococcal polysaccharide vaccines) during the same visit, using separate syringes at different anatomic sites. If given on different dates, the zoster vaccine can be administered at any time before or after an *inactivated* vaccine, but at least 4 weeks before or after another *live, attenuated* vaccine.[26]

The zoster vaccine is administered subcutaneously, preferably in the upper arm, as a single dose. The most frequently reported adverse reactions were erythema, pain/tenderness and swelling at the injection site, pruritus, and headache.[1,24] The duration of protection after zoster vaccination is unknown. Thus, it has not yet been determined if and at what interval booster doses might be required.[24]

Receipt of zoster vaccine is contraindicated in individuals with a history of anaphylactic reaction to any component of the vaccine (including gelatin and neomycin), with a history of primary or acquired immunodeficiency states, on immunosuppressive therapy including high-dose corticosteroids of 2 or more weeks duration, with active untreated tuberculosis, or who are or may be pregnant (although these women are unlikely to be in the vaccine target age group).[1,24] Pregnancy should be avoided for 3 months after vaccination.[1,24]

There are additional recommendations regarding immunosuppression and the use of antiviral medications. Immunosuppressant therapy includes use of high-dose corticosteroids and use of immune mediators and modulators, especially the antitumor necrosis factor agents adalimumab (Humira), infliximab (Remicade), and etanercept (Enbrel). Zoster vaccine should be administered at least 14 days before initiation of immunosuppressive therapy. In addition, zoster vaccination should be deferred for at least 1 month after discontinuation of such therapy.[26]

Concurrent use of antiviral medications can interfere with the zoster vaccine. Thus, patients taking acyclovir, famciclovir, or valacyclovir for chronic conditions should discontinue these medications at least 24 hours before receiving the zoster vaccine, and not resume taking them for at least 14 days after the vaccination.[26]

ZOSTER VACCINATION ISSUES

Two very important issues the provider must consider regarding the zoster vaccine are those dealing with storage of the vaccine and with reimbursement for the vaccine and its administration.

The vaccine must be kept frozen before use. Significant loss of potency occurs when the vaccine is stored between 36° F and 46° F. Thus, once it is reconstituted, the vaccine should be administered immediately (within 30 minutes) to avoid this loss of potency.[27]

Unlike the influenza and pneumococcal vaccines that are covered through Medicare Part B, the zoster vaccine is covered under Medicare Part D. Payment for Part D covered vaccines is made solely by the participating Prescription Drug Plan. As of January 1, 2008, Medicare Part D covers the administration of vaccines as a part of the negotiated cost of vaccines covered under Part D. In other words, a separate charge for administration of the vaccine is no longer allowed. An important consideration for both patients and providers is what this means for out-of-pocket costs. According to the Centers for Medicare and Medicaid Services (CMS), a patient receiving the zoster vaccination in a provider's office would be billed for the entire charge, including both the vaccine and administration and would in turn need to submit a paper claim to their Part D plan for reimbursement of plan allowable costs. If, on the other hand, the patient would receive the vaccination at an in-network pharmacy (where allowed by state law), the pharmacy would process the claim to the Part D plan as with other prescription medications, and would collect from the patient any applicable cost-sharing fees.[28] Providers should encourage patients to contact their Part D plan administrators to determine coverage of the zoster vaccine by their particular plan. ●

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