Clinical Overview

Herpes zoster, also known as zoster and shingles, is caused by the reactivation of the varicella zoster virus (VZV), the same virus that causes varicella (chickenpox).

Primary infection with VZV causes varicella, and once the illness resolves, the virus remains dormant in the dorsal root ganglia. VZV can reactive later in a person’s life and cause a painful, maculopapular rash called herpes zoster.

Anyone who has had varicella or gotten varicella vaccine can develop herpes zoster. Most people typically have only one episode of herpes zoster in their lifetime. However, second and even third episodes are possible.

Clinical Features

People with herpes zoster most commonly have a rash in one or two adjacent dermatomes (localized zoster). The rash most commonly appears on the trunk along a thoracic dermatome. The rash does not usually cross the body’s midline. However, approximately 20% of people have rash that overlaps adjacent dermatomes. Less commonly, the rash can be more widespread and affect three or more dermatomes. This condition is called disseminated zoster. This generally occurs only in people with compromised immune systems. Disseminated zoster can be difficult to distinguish from varicella.

The rash is usually painful, itchy or tingly. These symptoms may precede rash onset by days to weeks. Some people may also have headache, photophobia, and malaise in the prodromal phase.

The rash develops into clusters of clear vesicles. New vesicles continue to form over three to five days and progressively dry and crust over. They usually heal in two to four weeks. There may be permanent pigmentation changes and scarring on the skin.

Transmission

A person with shingles can spread the virus when the rash is in the blister-phase. A person is not infectious before blisters appear. Once the rash has developed crusts, the person is no longer contagious. Shingles is less contagious than chickenpox and the risk of a person with shingles spreading the virus is low if the rash is covered.
Shingles cont.

Complications

Postherpetic neuralgia (PHN) is the most common complication of herpes zoster. It is a persistent pain in the area where the rash once was. PHN is diagnosed in people who have pain that persists after their rash has resolved. Some define PHN as any duration of pain after the rash resolves; others define it as duration of pain for more than 30 days, or for more than 90 days after rash onset. PHN can last for weeks or months and occasionally, for many years.

A person’s risk of having PHN after herpes zoster increases with age. Older adults are more likely to have PHN and to have longer lasting and more severe pain. Approximately 13% (and possibly more) of people 60 years of age and older with herpes zoster will get PHN. PHN is rare in people younger than 40 years old. Other predictors of PHN include the level of pain a person has when they have zoster rash and the size of their rash.

Other complications of herpes zoster include—

- ophthalmic involvement with acute or chronic ocular sequelae (herpes zoster ophthalmicus);
- bacterial superinfection of the lesions, usually due to Staphylococcus aureus and, less commonly, due to group A beta hemolytic streptococcus;
- cranial and peripheral nerve palsies; and
- visceral involvement, such as meningoencephalitis, pneumonitis, hepatitis, acute retinal necrosis.

People with compromised or suppressed immune systems are more likely to have complications from herpes zoster. They are more likely to have severe rash that lasts longer. Also, they are at increased risk of developing disseminated herpes zoster.

Please remind your patients diagnosed with shingles:

- Keep the rash covered
- Do not touch or scratch the rash
- Wash their hands often to prevent the spread
- Until your rash has developed crusts, avoid contact with:
  - Pregnant women who have never had chickenpox or the varicella vaccine
  - Premature or low birth weight infants; and
  - Immunocompromised persons
Summary of CRE surveillance data from 10/23/12-10/31/13 in Washington

This summary includes CRE isolates reported to DOH from October 23, 2012-October 31, 2013 and includes all reported cases diagnosed in Washington or in Washington residents that were diagnosed elsewhere. This summary is preliminary and may change as additional case reports are received. Overall, 103 suspected CRE isolates were tested at PHL, and 79 were CRE. Of these 79 CRE isolates submitted from 71 patients, 78 met our surveillance case definition as described above. Notably, one isolate was not resistant to all third generation cephalosporins tested, and therefore did not meet our case definition, but tested positive for *Klebsiella pneumoniae* carbapenemase (KPC) by PCR. Of 79 laboratory confirmed CRE, the genera represented included 40 *Enterobacter* (51%), 29 *Escherichia* (37%), 7 *Klebsiella* (9%), 2 *Citrobacter* (2%), and 1 *Proteus* (1%).

In total, six isolates from 5 patients tested positive for carbapenemase by PCR, representing 1 KPC, 3 New Delhi metallo-b-lactamases (NDM), and 2 imipenemases (IMP). The two IMP-carrying isolates were identified from a single patient. The balance of confirmed CRE isolates was thought to be resistant via a mechanism other than carbapenemase production, such as extended spectrum cephalosporinase (ESBL or AmpC) with porin loss.

The factors associated with CRE infection and/or colonization in Washington are similar to those observed in national surveillance. Most cases of CRE in Washington and elsewhere occur in the elderly and those with underlying conditions and/or significant medical exposure to hospitalizations, surgeries, or long term care. Of 71 CRE cases reported in Washington, 46 (59%) were women and 36 (52%) were elderly. (Table 1) Forty-five cases (63%) were diagnosed in Western Washington, 11 (17%) in Eastern Washington, and 15 (21%) in Oregon. Several cases diagnosed in Washington were residents of other states and countries. Six of the 71 CRE cases died but only 4 fatalities (6%) were determined to be due to CRE infection. None of the fatalities occurred in patients with CP-CRE. The majority of the reported CRE infections in Washington were urinary tract infections. Almost all cases had significant underlying conditions, and all had extensive medical exposure in the 6 months prior to diagnosis.

Foreign travel or foreign hospitalization was commonly seen in patients with CP-CRE. (Table 2) Of 5 patients with CP-CRE identified in Washington during this surveillance period, 4 of 5 were diagnosed soon after arrival from a foreign country, and 3 of 4 had been hospitalized in a foreign country in the 6 months prior to their diagnosis in Washington. The countries thought to be the source of these CP-CRE infections were India, China, and the Philippines. The KPC case had no reported international travel or health care. These findings illustrate that while the majority of CP-CRE infections were acquired abroad, CP-CRE (at least organisms carrying KPC) may be circulating unrecognized in Washington, thereby underscoring the importance of effective CRE surveillance.

In addition to carbapenemase-producing CRE, a single patient with carbapenemase-producing *Acinetobacter* and *Pseudomonas* was reported in Washington and the source is thought to be international health care. As these organisms are capable of residing in multiple body parts, including the gut, they are potentially capable of transferring and spreading carbapenemase-producing plasmids to Enterobacteriaceae. These organisms were not part of the CRE surveillance but are reportable as rare diseases of public health significance (highly antibiotic resistant organisms).

Since CP-CRE are thought to be more easily transmitted and have greater potential for exponential growth in healthcare settings than non-carbapenemase producing CRE, we believe that the focus of future CRE surveillance should be on detecting CP-CRE and preventing their spread. This change does not diminish the importance of appropriate infection control based on commercial laboratory results, but emphasizes public health involvement and resources on the highest priority organisms. Planned changes in surveillance will decrease the workload attendant on this surveillance system while continuing to allow identification of most CP-CRE. DOH believes that concerted efforts by multiple levels of healthcare and public health will be most effective in preventing the spread of these organisms. Vigilance is required by all health care providers and facilities to use appropriate infection control precautions to protect patients from multidrug resistant organisms (MDRO) and other epidemiologically important organisms, such as *C. difficile*. Laboratories should apply the latest CLSI breakpoints for determining carbapenem resistance as soon as feasible, and should develop and implement procedures for rapidly communicating MDRO status to clinicians and facility infection control, if not already doing so. Healthcare facilities and providers should ensure that MDRO status is appropriately communicated to facilities receiving these patients in transfer.