Rash: Is It Shingles?

Joyce A. Marrs, MS, APRN-BC, AOCNP

Case Presentation: Mrs. Smith, a 56-year-old Caucasian woman, was seen in the office for complaints of a rash at her waist. She completed three cycles of dose-dense cyclophosphamide and doxorubicin chemotherapy for stage III breast cancer. The third cycle was 10 days prior. Grade III neutropenia was the only complete blood count abnormality.

Mrs. Smith reported that the rash started that morning. Before observing the rash, she had been experiencing burning pain around the waist, itching, fatigue, and muscle aches for the past three to four days. She did not have a fever. She denied any new soap, detergent, or clothes, and no one in her household had similar complaints.

Physical examination revealed clustered lesions on the left side of the abdomen at waist level. Lesions were erythematous plaques with a few vesicles.

Based on patient history and physical examination, Mrs. Smith was diagnosed with herpes zoster, commonly known as shingles.

Definition

Varicella zoster virus (VZV) is responsible for the common childhood infection chicken pox (Gross et al., 2003). Herpes zoster develops as a cutaneous vesicular infection along a nerve dermatome as a result of reactivation of the dormant VZV (Gross et al.; Habif, 2001).

Incidence

Although herpes zoster can affect people of all ages, the incidence increases with age (Ferri, 2005; Gross et al., 2003; Habif, 2001).

 Herpes zoster generally affects people older than age 50.

- The cumulative lifetime incidence is 10%-20%.
- The rate of herpes zoster episodes is higher in Caucasians than African Americans.
- Regardless of age, immunocompromised individuals have an increased risk of developing herpes zoster.
- The rate of a second VZV infection is approximately 4%.

Pathophysiology

VZV has a latent period following the primary chicken pox infection. The virus remains dormant in the cranial nerve and dorsal root ganglia (Gross et al., 2003; Melton, 2005). The exact mechanism of reactivation is not known. However, a decline in VZV-specific cellular immunity is associated with reactivation. Once reactivated, the virus spreads down the sensory nerve to skin level (Deignan, 2003; Gross et al.; Melton).

Signs and Symptoms

The clinical manifestations of shingles are categorized into two phases: prodromal and acute. During the prodromal

phase, symptoms (Deignan, 2003; Ferri, 2005; Habif, 2001; Melton, 2005)

- Occur three to five days prior to cutaneous manifestations
- Involve pain in the affected dermatome that may or may not be accompanied by burning, itching, and/or tingling. Symptoms also occur during the acute phase.
- A rash starts out as erythematous papular lesions, which transform into vesicles prior to rupture and crusting. Lesions resolve in two to three weeks.
- · Lesions are found along a dermatome.
- The thoracic dermatome is affected most often, followed by lumbar and cranial nerves.
- The rash may be accompanied by fever, lymphadenopathy, fatigue, headache, and pain.

Differential Diagnosis

During the prodromal phase, various diagnoses may be mistaken for herpes zoster (Deignan, 2003; Ferri, 2005).

- · Acute myocardial infarction
- Cholecystitis
- · Appendicitis
- Pleurisy

Joyce A. Marrs, MS, APRN-BC, AOCNP, is a nurse practitioner at Hematology and Oncology Associates of Dayton in Ohio.

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