Disseminated Varicella-Zoster Virus Infection Following Azacitidine in a Patient With Myelodysplastic Syndrome

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Reactivation of varicella-zoster virus (VZV), also known as shingles, is a common health concern among patients aged 50 years or older and those with compromised immunity (Stankus, Dlugopolski, & Packer, 2000; Yawn et al., 2007) (see Figure 1). Reactivation of VZV is caused primarily by diminished cellular-mediated immunity from age-related immunosenescence, infections, and immunosuppressive agents (Burke et al., 1982; Yawn et al.). Disseminated VZV prevalence is relatively low, but VZV has been reported in patients following solid organ and stem cell transplantation and in patients receiving chemotherapy (Carby, Jones, Burke, Hall, & Banner, 2007; Curley, Hussein, & Hassoun, 2002; Doki, Hoshino, Irisawa, Sakura, & Miyawaki, 2004; Manuel, Kumar, Singer, Cobos, & Humar, 2008; Rodriguez-Moreno et al., 2006). Patients with lymphoproliferative disorders (e.g., chronic lymphocytic leukemia, Hodgkin disease, or non-Hodgkin lymphoma) have a higher risk for disseminated VZV after stem cell transplantation (Kim et al., 2008). However, disseminated VZV has not been reported in patients with myelodysplastic syndrome following treatment with azacitidine (Vidaza®, Celgene Corporation) or in preapproval clinical trials (Celgene Corporation, personal communication, June 22, 2006).

Overview

VZV is related closely to herpes simplex viruses (HSVs), with HSV type 1 causing cold sores and HSV type 2 causing genital herpes; all are classified as alpha herpesviruses, a subtype of herpesviruses (Centers for Disease Control and Prevention [CDC], 2007; Harper, Gilbert, & Jeffries, 1998). VZV infection causes two clinically distinct conditions. Primary infection with VZV results in varicella or chickenpox, which largely affects children with a mild, self-limiting course (Wharton, 1996). After clinical resolution of primary infection, viral latent infection is established when VZV retrogrades into the sensory dorsal roots of ganglia (Breuer & Whitley, 2007). Vaccination in children to prevent primary VZV has reduced chickenpox cases in the United States (Marin, Güris, Chaves, Schmid, & Seward, 2007). Herpes zoster, the second clinical condition of VZV infection, results from the reactivation of this latent neurotropic virus and commonly presents with painful, unilateral vesicular skin eruption distributing within the dermatomal zones (Gnann & Whitley, 2002). This column will focus primarily on herpes zoster.

Incidence and Risk Factors

About one in three Americans will develop zoster during their lifetime, and an estimated one million cases of herpes zoster infections occur in the United States annually (CDC, 2007; Harpaz, Ortega-Sanchez, & Seward, 2008). Age is the most important risk factor, with risk increasing greatly after age 50; about 50% of people who live to age 85 will develop an episode of herpes zoster infection (Harpaz et al.). Women have a slightly higher risk than men, and African Americans have a lower incidence of herpes zoster (Opstelten, Van Essen, Schellevis, Verheij, & Moons, 2006).

Altered immunity, particularly cell-mediated immunity, is common among people with cancer; therefore, herpes zoster incidence is significantly higher in older adults with cancer than in the general age-matched population (Rusthoven et al., 1998). Risk varies among different types of cancer and treatments; in patients with solid cancer, herpes zoster incidence is less than 5% but Hodgkin disease incidence is 27.3% (Rusthoven et al.). Among hematopoietic stem cell transplantation recipients, herpes zoster risk is about 13%–55% during the first year; incidence is 5%–17% for solid organ transplantation recipients (Harpaz et al., 2008). Herpes zoster risk also is elevated in patients with HIV or AIDS, chronic inflammatory diseases (e.g., systemic lupus erythematosus), rheumatoid arthritis, Crohn disease and ulcerative colitis, and multiple sclerosis (Harpaz et al.). Mortality for VZV infection in

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