Shingles Still More Common in People With HIV

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New cases of herpes zoster, better known as shingles, appear to be on the decline among people living with HIV, but rates are still higher than those seen in the general population, according to Johns Hopkins University School of Medicine data published online ahead of print by the Journal of Acquired Immune Deficiency Syndromes.

The researchers, under the direction of Leah Blank, MD, MPH, also noted that more than one-quarter of all new shingles cases in their HIV cohort were complicated, a “remarkable” finding in light of the young age of the patient population.

Shingles is caused by the varicella zoster virus (VZV), best known for its ability to cause chickenpox (varicella) in children. VZV isn’t cleared from the body after a bout of chickenpox, but rather remains dormant in nerve clusters near the spine. If cellular immunity to VZV dwindles—which can happen in people living with HIV, advancing in age or undergoing treatment that depletes immune function—VZV can become reactivated, leading to a shingles outbreak.

Shingles typically causes a rash-like string of blisters that follow the path of a nerve extending from the spinal cord (known as a dermatomal pattern). While often painful, shingles is usually benign; it can last three to four weeks without causing otherwise serious or long-term problems. Sometimes, however, the disease can be complicated by recurrences, internal organ damage and multiple dermatomal patterns.

Shingles has long been more common among people living with HIV, particularly among young people infected with the virus compared with age-matched individuals in the general population. In the years following the widespread availability of combination antiretroviral therapy, studies didn’t show that the risk of shingles was decreasing. In fact, some researchers suggested that the incidence may increase, given that people are now living with HIV longer and because shingles may be an adverse effect of the immune reconstitution syndrome (IRIS) that can occur in people with low CD4 cell counts responding otherwise favorably to antiretroviral treatment.

To get a sense of the modern-day incidence of shingles, including complicated cases, Blank and her colleagues identified herpes zoster episodes documented between 2002 and 2009 at Johns Hopkins. For each case the researchers identified, three HIV-negative controls were included in the analysis so that potential shingles outbreak risk factors could be assessed.

Researchers identified 183 new (incident) shingles cases among the more than 4,300 HIV-positive patients; an additional 138 patients were also diagnosed with shingles, but these were recurrent episodes.

The incidence rate during the entire study period was 9.3 new shingle cases per 1,000 person-years of follow-up. While the study did not show a statistically significant trend in the incidence of shingles over time, the authors note that the incidence rate was significantly lower than the one documented in a previous study of the same cohort. Between 1997 and 2001, Blank and her colleagues explain, the incidence rate during the study period was 32 new shingle cases per 1,000 person-years of follow-up.

The authors add that the apparent reduced incidence in the Johns Hopkins study conflicts with results from two other cohorts—the Veterans Health database (2000–2007) and Olmsted County, Minnesota, surveillance data (1996–2001)—that both showed small, but statistically significant, increases in shingles over time.

“The observed decrease in incidence rate in our clinic might be explained by improvements in addressing the risk factors for herpes zoster specific to [people living with HIV],” the authors explain. “Consistent with our earlier study and other studies, we found that a lower CD4 count was associated with increased risk of incident herpes zoster. Indeed, immune suppression is consistently a risk factor for herpes zoster outbreak in this population, with a CD4 count below 350 [conferring] greater risk than a CD4 count between 350 and 500. Given the median CD4 count of our population has steadily increased from 2009 from 298 to 431 cells, this finding highlights the importance of restoring immune function in protecting against herpes zoster.”

Blank and her colleagues stress, however, that the incidence rate is still greater than the general population, especially when age is considered.

Also of concern was the high rate of complicated herpes zoster—28 percent of those with shingles experienced disseminated shingles (three or more dermatomal patterns), disease of the eye or internal organs, neurological complications or recurrence within six months. While this rate of complicated shingles in people living with HIV is consistent with other cohorts, Blank and her fellow authors note the 28 percent rate is lower than the one documented in the earlier Johns Hopkins cohort: 53 percent.

In addition to low CD4 cell counts and detectable viral loads being associated with shingles outbreaks, Blank’s team determined that outbreaks were more likely to occur within 90 days of starting antiretroviral therapy, confirming that shingles is a possible complication of the immune reconstitution syndrome that can occur in people beginning HIV therapy with low CD4 cells. “Herpes zoster does appear to be associated with immune reconstitution, so the clinician should be aware of the higher risk of herpes zoster shortly after antiretroviral therapy is started,” they write.

Herpes zoster vaccination—or the lack thereof in the cohort—also appears to be a risk factor.

“Despite the high complication rate, and the high incidence rate in [people living with HIV], not a single patient in our study population had been vaccinated against herpes zoster,” Blank and her colleagues wrote.

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