Providers' Knowledge, Practices, and Barriers Related to Genital Herpes Testing for Patients With HIV

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This cross-sectional study explored the knowledge, practice, and reported barriers related to genital herpes testing among 102 health care providers who treat people living with HIV in the United States. Twelve percent reported always testing HIV-infected patients for genital herpes, 65% sometimes or usually tested, and 23% rarely or never tested for genital herpes. Seventy-five percent said testing was not standard of care. Providers were more likely to recommend a herpes test if the patient had symptoms (94%) or had a partner with herpes (83%) and were less likely to recommend testing if patients had no partners (60%) or would rather not know (49%). Our work adds to the growing body of literature on herpes simplex virus–HIV coinfection by documenting that (a) providers often do not screen for genital herpes, (b) knowledge of appropriate diagnostic evaluation is limited, and (c) many clinicians report the lack of clear guidelines is a barrier to testing.

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Herpes simplex virus type 2 (HSV-2) infection is highly prevalent and largely unrecognized in persons with HIV. Estimates of HSV-2 prevalence among HIV-infected individuals range from 70% in the developed world to 90% in the developing world (Corey & Wald, 2008). In a recent study of 248 HIV-infected participants recruited from one urban HIV clinic in the United States, 69.4% were HSV-2 seropositive, and 67.4% of those who tested positive for HSV-2 did not have a history of genital herpes (Meyer et al., 2005). While HSV-1 historically has been the primary cause of oral-labial herpes and HSV-2 the cause of genital infection, HSV-1 now accounts for approximately one-half of first episodes of genital herpes (Nieuwenhuis, van Doornum, Mulder, Neumann, & van der Meijden, 2006). Thus, rates of genital herpes are likely to be considerably higher than suggested by HSV-2 seroprevalence

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alone. The initial clinical presentation of genital HSV-1 is similar to genital HSV-2, but genital HSV-1 recurs much less frequently (Engelberg, Carrell, Krantz, Corey, & Wald, 2003).

Infection with HSV-2 affects the lives of those with HIV in several important ways. First, HSV and HIV coinfection exacerbate each condition. Persons coinfected with both viruses, compared with persons with HSV-2 infection alone, have more frequent genital lesions that may last longer and be more debilitating (Strick, Wald, & Celum, 2006). In individuals who were infected with both HSV-2 and HIV, HSV-2 reactivation appeared to increase plasma HIV RNA levels, which might adversely affect survival (Corey, Wald, Celum, & Quinn, 2004).

Also, observational studies have noted that dual infection increases the risk of transmitting HIV and HSV to uninfected partners. The frequency of clinical and subclinical HSV-2 reactivation may be two- to fourfold higher in HIV-infected compared with HIV-uninfected persons (Russell, Tabrizi, Russell, & Garland, 2001). Both clinical and subclinical reactivations of HSV-2 are associated with increasing HIV RNA levels (as measured by polymerase chain reaction) in rectal, vaginal, and seminal secretions, theoretically resulting in the ability to transmit HIV and HSV to others more efficiently (Zuckerman et al., 2007). A study of HIV-discordant couples found that genital ulcer disease in the HIV-infected partner was associated with a fivefold increased risk for HIV transmission (Gray et al., 2001).

HSV infection is a potential severe complication for the offspring of both HIV-infected and uninfected individuals. Although not a reportable disease in every state, estimates suggest that approximately 1,200–1,500 cases of neonatal herpes occur per year in the United States (ACOG Committee on Practice Bulletins, 2007). Neonatal herpes is usually acquired during the intrapartum period, although in utero and postnatal infections occur rarely. Mortality is approximately 30% for disseminated disease and 4% for central nervous system disease. Approximately 20% of survivors of neonatal herpes have long-term neurologic sequelae (Gardella, Handsfield, & Whitley, 2008). The risk for neonatal infection is greatest among mothers who contract HSV during the last trimester of their pregnancies, rather than those who were previously infected. Maternal HSV screening has been proposed to reduce neonatal herpes by identifying women susceptible to infection late in pregnancy and whose partners could be offered screening, allowing for counseling about strategies to reduce the possibility of a new maternal infection (i.e., condom use or abstinence). Routine use of serotesting in pregnant women is controversial because of issues of effectiveness, cost, and patient acceptance (Tita, Grobman, & Rouse, 2006).

Type-specific serological assays for HSV became commercially available in 1999, revealing for the first time the extent to which HSV infection remains undiagnosed and making screening for HSV and confirmation of clinical diagnoses possible. If and how serological tests are being used by providers is mostly unknown. Although screening guidelines for HSV-2 among persons with HIV are vague, the most recent Guidelines for Prevention and Treatment of Opportunistic Infections in HIV-Infected Adults and Adolescents recommend that routine type-specific serological testing for HSV-2 be considered in persons who seek HIV care (Centers for Disease Control and Prevention [CDC], 2009). The CDC sexually transmitted disease (STD) treatment guidelines note "some specialists believe that HSV serologic testing should be included in a comprehensive evaluation for STDs among persons with HIV infection" (CDC, 2006, p.17).

The use of serological assays to confirm clinical diagnoses is less ambiguous. The CDC's STD Guidelines and the Guidelines for Prevention and Treatment of Opportunistic Infections in HIV-Infected Adults and Adolescents both recommend that a clinical diagnosis of genital herpes be confirmed by laboratory testing. Diagnosing genital herpes by history and clinical examination without laboratory confirmation has several serious limitations: (a) 80%–90% of people who have genital herpes report no history of signs/symptoms consistent with genital herpes; (b) 20% of people diagnosed by clinical visual examination alone have been found in two studies to not have genital herpes; and (c) clinical presentations can be subtle and atypical, often without genital vesicles and ulcers, leading to misdiagnosis. In addition, although viral culture is the best method for diagnosing genital herpes when lesions are present, due to declining sensitivity of viral culture as lesions begin to heal, a negative culture result does not rule out genital herpes (American Social Health Association, 2009).