BRIEF REPORT

Potential Role of Famciclovir for Prevention of Herpetic Whitlow in the Health Care Setting

Oral famciclovir was initiated in a health care worker immediately after an accidental percutaneous injury involving a needle freshly removed from a patient’s herpes labialis vesicles. In follow-up, the health care worker remained seronegative for herpes simplex I and II antibodies (IgG and IgM) and did not develop herpetic whitlow, supporting the potential role of famciclovir in the prevention of herpetic whitlow in a health care setting.

Herpetic whitlow is an infection of the finger caused by herpes simplex virus (HSV) that commonly afflicts health care workers. The source of infection in this setting is generally thought to be damaged cuticle or skin exposed to infected oropharyngeal secretions [1, 2]. Although potent antiviral drugs, such as acyclovir and famciclovir, have been shown to be effective in the treatment of a variety of diseases due to HSV [3], their role in preventing HSV transmission in a health care setting is unclear. The short incubation period of symptomatic HSV infection and the early establishment of latency are considered significant obstacles to effective postexposure prophylaxis in this setting [1]. Here, I report the management of a health care worker who was stuck with a contaminated needle freshly removed from a patient’s fluid-filled vesicles of herpes labialis and discuss the potential role of immediate initiation of famciclovir in the prevention of HSV transmission in situations in which the exact time of exposure is known.

The health care worker, previously healthy and infectious disease consultant, used a hypodermic needle to puncture several fluid-filled vesicles of the nasal folds of a patient, for culture confirmation. The health care worker accidentally punctured his left index finger after removing the needle from these lesions. The finger bled profusely and was washed immediately with soap and water for 2–3 min. Because of the high index of suspicion for herpes labialis, the health care worker was placed on oral famciclovir 500 mg t.i.d., with the first dose being given within 1 h of exposure. Cultures of the vesicles grew HSV type I in <24 h. Serum HSV I and II antibodies (IgG and IgM) drawn from the health care worker 1 day after exposure were found to be nonreactive by immunofluorescent assay. Famciclovir was continued by the health care worker for only 4 days of a planned 7-day course, because of poor compliance. At 1 week after exposure, the health care worker did not have any pain, paresthesias, or cutaneous lesions. HSV I and II antibodies drawn at 1 month after exposure were nonreactive. At 10 months after exposure, the health care worker was doing well, with no signs or symptoms of herpetic whitlow.

To the best of my knowledge, this is the first reported use of an antiviral agent for prevention of herpetic whitlow in a health care setting. Lack of transmission of HSV in the case described is supported by the absence of signs or symptoms of cutaneous infection nearly a year after exposure (the usual incubation period is 2–7 days) and the absence of HSV I IgM and IgG 1 month after exposure, virtually eliminating the possibility of subclinical infection [1]. Although lack of transmission of HSV in the described health care worker—supported by the absence of clinical or serological evidence of infection—might not have been necessarily related to the use of famciclovir, several features of the case suggest that the exposure placed the health care worker at a very high risk of HSV infection and that famciclovir might very well have played a role in preventing its transmission.

First, the skin puncture was caused by a hollow-bore needle, which probably carried a larger volume of infectious material than would solid-bore needles, if the paradigm of blood volume in hollow-bore needles is invoked [4]. Second, growth of HSV I from the patient’s vesicles in <24 h suggests a very high inoculum of the virus in the fluid (>10^7 infectious particles/mL of vesicular fluid), in contrast to lower virus concentrations in oral secretions, the usual source of occupational herpetic whitlow [1]. Third, known puncture of the skin by a contaminated needle probably carries a higher risk of infection than that associated with unapparent or minor skin breaks or hangnails, the commonly held risk factor for acquiring HSV I in a health care setting. Finally, the exposed health care worker was seronegative for HSV I and II antibodies at the time of exposure and therefore was probably more susceptible to exogenous primary HSV infection [1]. Famciclovir (a produg of penciclovir) was chosen over valacyclovir (a produg of acyclovir with better oral bioavailability than acyclovir) for postexposure prophylaxis in this setting for several reasons: (1) superior absolute oral bioavailability (77% for famciclovir vs. 54% for valacyclovir) [5]; (2) longer intracellular half-life (~10 h for penciclovir triphosphate vs. 0.7 h for acyclovir triphosphate) [6]; (3) superior efficacy in limiting localized infection after experimental inoculation of HSV in the skin of mice, when compared with valacyclovir [5]; and (4) potential for reducing the likelihood of viral latency in ganglia during primary infection, as observed in a murine infection model (not observed with valacyclovir in the same model) [7].

It is clear that properly designed randomized studies will be needed to evaluate fully the efficacy of famciclovir in the prevention of herpetic whitlow in health care settings. However, because of the often unsuspected nature of HSV exposure in the occupational setting, the incurable nature of established infection, and possible ethical questions surrounding administration of placebo when potentially effective antiviral drugs are already available, such studies are not likely to be forth-
coming anytime soon. In the meantime, the favorable outcome of the case presented, and the results from pharmacokinetic and experimental studies to date, suggest that prompt administration of famciclovir may have a role in the prevention of occupational HSV infection when the exact time of exposure is known.

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References