DNA Restriction Enzyme Analysis of Digital and Genital Isolates of Herpes Simplex Virus from Three Patients

Colleagues—Herpetic infection of the hand can be acquired either by inoculation with exogenous virus or by autoinoculation with endogenous virus [1-3]. Exogenous virus is believed to account for the cases of herpetic infection of the hand that occur in health care workers [1]. Autoinoculation from one site to another or possible simultaneous inoculation of several sites would, however, appear to account for the other cases. Autoinoculation from mouth to fingers or thumb has been proposed in children [2], and autoinoculation from the genitals to fingers has been proposed in adults [3]. Molecular analysis of the isolates from each site has not, however, been performed to confirm identity.

In a series of 15 adult patients with multiple, recurrent herpetic infections of the hand, we found [4] that herpes simplex virus type 2 (HSV-2) was commonly isolated. In three of these patients we also had a viral isolate obtained from a previous genital infection. We performed restriction enzyme analysis of both the digital and genital viral isolates. Standard analysis using BamHI, SstI, and Sall showed identity in the isolates from different sites in each individual (figure 1) [5].

All three patients were women. In two patients, genital herpes preceded the clinical onset of digital herpes by many months; in the third patient, the digital lesions preceded the genital lesion. Although the viral isolates were identical, the patients seldom had clinical recurrences at the same time, despite the systemic nature of the triggers of recurrences (menstruation or psychological stress). Digital recurrences were usually more frequent than genital recurrences.

These results suggest that autoinoculation of HSV-2 from one site to another does occur. The same virus appears to reactivate in the same individual to produce clinical illness at differing frequencies, depending on the site of infection. These findings confirm other studies [6] that suggest the frequency of reactivation of HSV infection depends on both the strain of HSV involved and the site of latency.

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References


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