

Autoinoculation and Dissemination are Two Different Forms of Herpes Virus Spread

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SUMMARY Autoinoculation and dissemination (or Kaposi's varicelliform eruption or eczema herpeticum) of herpetic lesions are two forms of viral spread, and it is essential to differentiate between the two. Presented are typical examples of the two forms of viral spread.

KEY WORDS: autoinoculation; Kaposi eczema; herpetic lesions; herpes simplex virus

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INTRODUCTION

Herpes simplex viruses (HSVs) are DNA viruses that cause acute skin infected grouped vesicles on an erythematous base. Contact must involve mucous membranes or open or abraded skin. HSV invades and replicates in neurons as well as in epidermal and dermal cells (1,2). Virions travel from the initial site of infection on the skin or mucosa to the sensory dorsal root ganglion, where latency is established. Viral replication in the sensory ganglia leads to recurrent clinical outbreaks. These outbreaks can be induced by various stimuli, such as trauma, ultraviolet radiation, temperature extremes, stress, immunosuppression, or hormonal fluctuations. Viral shedding, leading to possible transmission, occurs during primary infection, subsequent recurrences and periods of asymptomatic viral shedding (1). HSV-1 infection is acquired in early childhood, and evidence of se-

rologic infection with HSV-1 approaches 80% in the general adult population. HSV-type 2 usually causes herpes on genitalia and seroprevalence has been reported in 13%-40% worldwide (1). HSV-2 infection in pregnancy can have devastating effects on the fetus. HSV-1 and HSV-2 are the causative agents of herpes genitalis, herpes labialis, herpetic whitlow, herpetic keratoconjunctivitis, eczema herpeticum, herpes folliculitis, lumbosacral herpes, disseminated herpes, neonatal herpes, and herpes encephalitis. The prevalence of HSV infections has increased dramatically in recent years (3,4). It is essential to differentiate the forms of viral spread, autoinoculation and dissemination, or Kaposi's varicelliform eruption (KVE) or eczema herpeticum (2). This is clearly illustrated by two case reports.

CASE REPORTS

Case 1

A 14-month-old female infant presented to our clinic for a 3-day history of swelling and purulent lesions on her right thumb. Her medical history revealed that she had had aphthous stomatitis at the age of 5 months and developed a purulent lesion on the right thumb 10 days later. Two months before we examined her, the child had been hospitalized for pneumonia and also developed herpetic lesions on her lips and thumb. Current physical examination revealed a healthy, afebrile child who had swelling and erythema with tense purulent vesicles on the distal phalanx of her right thumb (Fig. 1). The symptoms subsided without treatment within a week.



Figure 1. Case 1: on initial presentation; note purulent vesicles on the distal phalanx.

Case 2

A 39-year-old man was hospitalized for a vesicular, erosive impetiginized eruption involving his lips, chin, neck and dorsal side of his ears. He is a professional sportsman, and otherwise healthy except for Darier's disease from which he had been suffering since puberty. On examination, the patient was afebrile and in good health. There were eroded and purulent vesicles on his lips and chin (Fig. 2), dorsal side of his ears and the scruff of his neck. Treatment (given by the department) consisted of a combination of amoxicillin and clavulanic acid and acyclovir 800 mg three times/day, and the eruption cleared completely within 10 days.

DISCUSSION

HSV-1 and HSV-2 may cause neonatal HSV infection, acute herpetic gingivostomatitis with abrupt onset of illness, fever, inability to eat and/or drink, vesicular lesions on the tongue, buccal mucosa, and palate, lips and face; acute herpetic pharyngotonsillitis, recurrent orolabial herpetic infection (herpes labialis); primary genital infections with classic vesicular rash, fever, malaise, myalgias, and headache, painful inguinal lymphadenopathy, preexisting antibodies to HSV-1, recurrent genital infections with recurrent genital HSV in the adolescent or adult with pain, itching and dysuria; HSV infection of the central nervous system with encephalitis, fever, headache, behavioral changes. Herpetic infections occur in immunocompromised hosts with widespread dissemination of HSV in approximately 90% of patients (3). Autoinnoculation and dissemination (or KVE eczema herpeticum) of herpetic lesions are two forms of viral spread, and it is essential to differentiate between the two (2).

Autoinnoculation means a true infection that includes involvement of peripheral nerve endings at the site of infection, with retrograde transport to the dorsal root ganglia of the relevant dermatome upon which the virus persists in a latent state throughout life, with periodic reactivation and with either typical expression of the disease or asymptomatic viral shedding. In a manner of speaking,

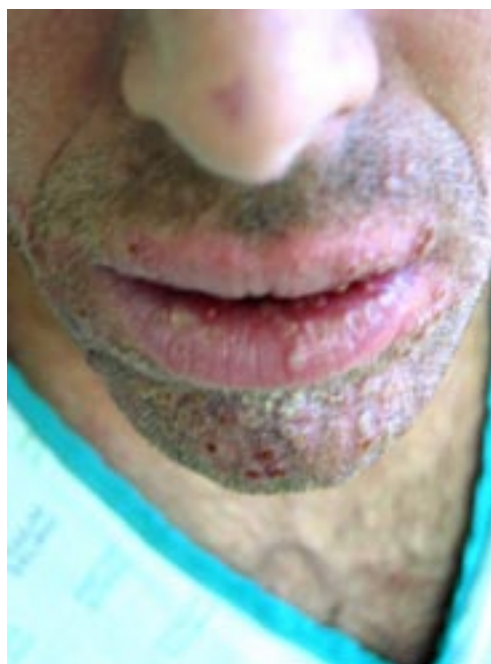


Figure 2. Case 2: purulent vesicles on the lips and chin.

autoinnoculation is a kind of self-infection by a virus that exists in the host. Autoinnoculation usually occurs during the first month after a primary infection, exactly what happened to our first patient.

Eczema herpeticum or KVE is a potentially life-threatening herpetic superinfection of a preexisting skin disease. Systemic dissemination of HSV-1 can occur in neonates and in patients with AIDS or other immunocompromising diseases (5,6). In contrast, KVE involves spread (dissemination) of the lesions to the areas of skin affected by another skin disease (most often atopic dermatitis, but also Darier's disease, burns and following various rejuvenation procedures), yet there is no true inoculation, i.e. the nerve endings and ganglion are not affected, so reactivation and recurrences of these lesions will usually fail to occur. Our second case presents this form of infection.

The cases presented are typical examples of the two forms of viral spread.

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