ELSEVIER
SAUNDERS

Emerg Med Clin N Am
26 (2008) 457–473

EMERGENCY
MEDICINE
CLINICS OF
NORTH AMERICA

Management of Oral and Genital Herpes in the Emergency Department

Howard K. Mell, MD, MPH[a,b,*]

[a]*Department of Emergency Medicine, College of Medicine, The Ohio State University, Columbus, OH, USA*
[b]*The Ohio State University Medical Centers, Center for Emergency Medical Services, 141 Means Hall, 1654 Upham Drive, Columbus, OH 43210–1228, USA*

The herpes family of viruses (or Herpesviridae) comprises double-stranded DNA viruses whose capsids display icosahedral symmetry (similar to a soccer ball) and are wrapped in a bilipid layered envelope [1]. These viruses are responsible for several medically important infections. Members of this family include: herpes simplex virus 1 (HSV-1), herpes simplex virus 2 (HSV-2), varicella-zoster virus (VZV), cytomegalovirus (CMV), Epstein-Barr virus (EBV), human herpesvirus 6 (HHV-6), human herpesvirus 7 (HHV-7), and human herpesvirus 8 (HHV-8) [2]. Although there are more than 80 identified herpesviruses, only 8 are thought to infect humans commonly [2], and a ninth (herpesvirus simiae) has caused fatal or severely debilitating encephalomyelitis after zoonotic infection in untreated individuals [3]. A list of the common illnesses caused by human herpesvirus infections is included in Table 1.

The epidemiology of oral and genital herpes has changed dramatically over the past several years [7–12]. This is especially important to the practice of emergency medicine, because between 5% and 10% of patients seeking care for a sexually transmitted disease (STD) do so in an emergency department [13]. In 2006, the Centers for Disease Control and Prevention (CDC) released new guidelines regarding the treatment of STDs [12]. The CDC makes specific recommendations concerning the screening, confirmatory testing, treatment, and counseling of patients who have oral or genital herpes infections [12]. Other literature suggests changes to common practices regarding herpes, given its changing epidemiology.

* Department of Emergency Medicine, Center for Emergency Medical Services, 141 Means Hall, 1654 Upham Drive, Columbus, OH 43210–1228.
E-mail address: howard.mell@osumc.edu

0733-8627/08/$ - see front matter
doi:10.1016/j.emc.2008.02.001 *emed.theclinics.com*

Table 1
Common illnesses caused by human herpesviruses by type

Common name	Abbreviation	Human herpesvirus	Common illness
Herpes simplex virus-1	HSV-1	HHV-1	Mucosal lesions (oral and genital)
Herpes simplex virus-2	HSV-2	HHV-2	Mucosal lesions (oral and genital)
Varicella zoster virus	VZV	HHV-3	Chickenpox and shingles
Epstein-Barr virus	EBV	HHV-4	Infectious mononucleosis, lymphoproliferative disorders [4]
Cytomegalovirus	CMV	HHV-5	Febrile hepatitis [5]
Roseolovirus		HHV-6 (subtypes a and b)	Roseola, undifferentiated febrile illness
		HHV-7	Roseola, undifferentiated febrile illness (higher incidence of febrile seizure [6])
Kaposi's sarcoma–associated herpesvirus	KSHV	HHV-8	Lymphoproliferative disorders [4]

Pathophysiology of herpes

Infection with a Herpesviridae virus is a multistep process. Specific glycoproteins on the viral envelope interact with specific cell membrane receptors [14–18]. Differences in these cell membrane receptors may play a significant role in the expression of the disease [14,17,18]. Once bound to the host cell, the viral DNA and capsid are internalized and the viral DNA migrates to the cell nucleus. Once inside the nucleus, viral DNA is replicated and viral genes are transcribed. One viral gene encodes for latency-associated transcripts (LATs) [16]. Once replicated, these LATs can accumulate in a host cell and persist in the host in a latent state [19]. Reactivation of LATs results in several organic diseases. Although primary infection can be accompanied by a period of clinical illness, long-term latency is symptom-free. If reactivated, transcription of specific signaling genes contained in the LATs occurs and virus production restarts [20]. Often, reactivation leads to host cell death and clinical symptoms (ie, lymphadenopathy, fever, headache, malaise, rash). Reactivation can result from local trauma (ie, surgery, dental procedures, burns [21], fever [2], or sunlight [21]).

This latency and reactivation are unique characteristics of the Herpesviridae viruses [2]. Viral invasion of epithelial cells and intracellular viral replication at the site of primary exposure frequently occur after exposure to a herpesvirus [2]. After the primary infection, which may be devoid of clinical symptoms, herpesviruses ascend in a retrograde manner along sensory nerve sheaths to the trigeminal, cervical, lumbosacral, or autonomic ganglia [2]. There, the virus replicates—undetected by the host's