Oral Manifestations of Viral Infections

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KEYWORDS

• Herpes simplex • Varicella zoster • Human papilloma • Coxsackie • Viral infection

KEY POINTS

- A plethora of symptomatic or asymptomatic viral conditions may affect the oral cavity.
- Clinical presentation of viral infections range from no change to epithelial ulcerations, benign soft tissue growths and malignancy.
- Definitive diagnosis of oral ulcerations caused by varicella, Coxsackie and herpes simplex viruses relies on history, physical exam and diagnostic tests.
- Educating Patients about the contagious nature of viral conditions is essential for preventing spread of infections.

Introduction

Viruses cause a plethora of symptomatic or asymptomatic infections in the oral cavity, with clinical manifestations ranging from no change to epithelial ulcerations, benign soft tissue growths, and malignancy. The host's intact innate and adaptive immunity is often adequate for viral recognition and mounting a protective response. Those with a defective immune system, however, often experience more severe seguelae.

Ulcerative manifestations of various viral infections are often difficult to discern from each other and from clinically similar conditions of different etiology. This article reviews presentation, diagnosis, and the management approach for major viral conditions of oral cavity such as herpes simplex virus type 1 (HSV1), varicella zoster virus (VZV), coxsackie viruses, and human papilloma virus (HPV).

Herpes simplex type

Description

The herpesviridae group of viruses includes more than 80 herpesviruses; for 8 viruses, people are the natural host. All human herpes viruses (HHVs) have a double-stranded DNA core within a protein capsid, enclosed by a tegument and an envelope, and are capable of causing life-long latency. Herpes simplex virus (HSV) has 2 serotypes, HSV1 and HSV2, which are partially homologous in their DNA core but have different antigenic properties.

Although HSV1 and HSV2 primarily infect orofacial and genital regions, respectively, sexual practices could result in cross-over infections. Herpes simplex viruses are highly transmissible through contact with oral or genital secretions of individuals with active lesions as well as asymptomatic shedding.

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Management The condition is self-limiting in healthy individuals. When oral pain and dysphagia pose risk of dehydration and poor nutritional intake, palliation and off-label antiviral therapy

Antipyetics such as acetaminophen may be used to treat fever, and anesthetic rinses may be used for palliation. Hydration and a bland, soft diet should also be encouraged. Patients should be educated about the contagious nature of the virus and potential for spread of infection to others or autoinoculation to other body sites.

Spread of infection to other mucocutaneous sites occurs through autoinoculation.

Epidemiology

HSV-1 prevalence varies depending on age, race, geographic location, and socioeconomic status. In the underdeveloped countries, HSV-1seroconversions is not only higher but also occurs at a younger age.

Primary oral herpes

Primary herpetic gingivostomatitis (PHGS) develops when a nonimmune individual is exposed to HSV-1 for the first time.

Clinical presentation and differential diagnosis

Most cases affect children 1 to 5 years of age and are often subclinical. Symptomatic patients present with lymphadenopathy, fever, sore throat, and vesiculo-ulcerative lesions affecting the oral and perioral regions (Fig. 1). Both movable and nonmovable oral mucosa may be affected, and acute onset of generalized gingival inflammation and pain is a classic feature (Figs. 2 and 3).

Differential diagnosis includes herpangina, erythema multiforme, allergic stomatitis, acute necrotizing ulcerative gingivitis, and acute eruptions of vesiculobullous diseases.

with acyclovir or valacyclovir may be indicated.

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Fig. 1 Primary herpetic gingivostomatitis affecting the lower lip.



Fig. 3 Gingival erythema and edema in primary herpetic gingivostomatitis.

Secondary oral herpes

HSV 1 establishes life-long latency in trigeminal ganglion. Internal and external triggers such as stress, fatigue, fever, menstruation, immunosuppression, and exposure to heat, cold, or sunlight could cause viral reactivation. Viral reactivation may occur in up to 40% of individuals testing positive for HSV1 antibodies and lead to asymptomatic viral shedding (recurrence) or clinical disease (recrudescence).

Clinical presentation

Recrudescence may affect mucocutaneous junction of the lips (Figs. 4 and 5) or keratinized intraoral tissues (Figs. 6 and 7). Unlike PHGS, there are no systemic symptoms with recrudescence. However, onset of HSL is often preceded by a local prodrome of tingling, burning, or itching. Recrudescent intraoral herpes (RIH) is less common than herpes simplex labialis (HSL), and in the immunocompetent host, it affects keratinized tissues such as gingiva and palate.

Differential diagnosis

Differential diagnosis for RIH includes herpes zoster, herpetiform aphthous ulcers, and traumatic ulcers. Vesicular onset and localization to nonmovable mucosa often help differentiate RIH from recurrent aphthous ulcerations. Diagnosis of both primary and secondary HSV1 infections relies on history and clinical presentation. However, Tzanck smear, viral culture, tissue biopsy, serology, or polymerase chain reaction (PCR) may prove beneficial in atypical cases.

Management

In the immunocompetent host, HSL heals within 7 to 14 days without scarring. Frequent or disfiguring recurrences of HSL and severe or chronic HSV outbreaks in the immunosuppressed host may require antiviral therapy. US Food and Drug Administration (FDA)-approved medications for HSL include topical deconasol and penciclovir, as well as systemic valacyclovir. Maximal therapeutic efficacy requires early recognition of signs and symptoms of an outbreak and initiation of antiviral therapy during the prodrome.



Fig. 2 Gingival erythema and focal ulcerations in primary herpetic gingivostomatitis.



Fig. 4 Herpes simplex labialis affecting right labial commissures.

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