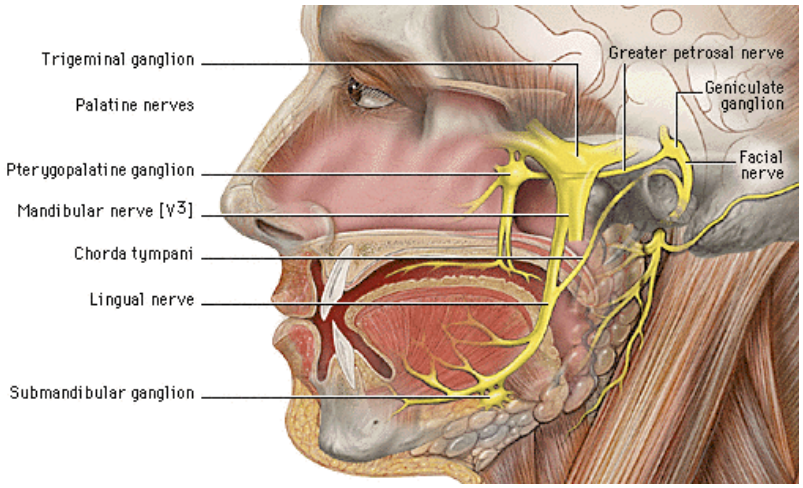
Gingivostomatitis

(Herpetic Gingivostomatitis, Recurrent Herpetic Gingivostomatitis)

Gingivostomatitis is the most common manifestation of primary herpes simplex virus (HSV) infection during childhood. Primary herpetic gingivostomatitis is characterized by ulcerative lesions of the gingiva and mucous membranes of the mouth, and is caused by Herpetic gingivostomatitis is caused by herpes simplex virus type 1.

PATHOGENESIS

The clinical manifestations of mucocutaneous herpes simplex virus type 1 (HSV-1) disease are due to tissue destruction, a direct consequence of viral replication and cell lysis. Unfortunately, once someone is infected with this virus, they are likely to keep it for much of their life. It will actually set up residence in the trigeminal ganglion, and every once in a while will cause a new infection in the mouth.



The trigeminal ganglion, colored in yellow and located near the ear, is a cluster of nerves where HSV-1 can hide out during times of dormancy/latency.

This is why it is sometimes called “recurrent gingivostomatitis” or “recurrent herpetic gingivostomatitis.”

EPIDEMIOLOGY

Primary herpetic gingivostomatitis typically occurs in children between six months and five years of age, but it can occur in older children and adolescents. Herpetic gingivostomatitis occurs throughout the year, with no particular seasonal distribution.

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TRANSMISSION

Herpes simplex virus type 1 (HSV-1) infections usually result from direct contact with infected oral secretions or lesions, being transmitted from symptomatic or asymptomatic individuals with a current infection.

Children with primary gingivostomatitis typically are “contagious” with HSV for at least one week and occasionally for several weeks (median three weeks).

The incubation period for HSV infection ranges from two days to two weeks.

CLINICAL FEATURES

Gingivostomatitis occurs in 13 to 30 percent of affected children. This means that usually less than ⅓ of all children with HSV actually ever develop gingivostomatitis.

Gingivostomatitis is characterized by ulcerative lesions of the gingiva and mucous membranes of the mouth, often with perioral vesicular lesions (appearance of multiple canker sores in the mouth. Herpetic gingivostomatitis occurs approximately one week after contact with an infected child or adult (the contact case often is asymptomatic).

Gingivostomatits generally begins with a sequence of symptoms (a prodrome) that lasts approximately four days and may include fever (>38°C [100.4°F]), anorexia, irritability, malaise, sleeplessness, and headache. Caregivers may attribute these symptoms to teething in children whose primary teeth are erupting, but children who are simply teething do not have mouth sores.

The sores begin to form at the same time the gums become red, swollen and bleed easily. These sores become yellow after they rupture and are surrounded by a red halo. At times they will merge with each other to form large, painful ulcers of the oral and perioral tissues.

These lesions can bleed easily and may become covered with a black crust. They can occur on the wet side of the cheeks and lips, the tongue, gingiva (gums), hard palate, and pharynx. Mild lesions typically heal without scarring in approximately one week, but healing may require 14 to 21 days in severe cases.

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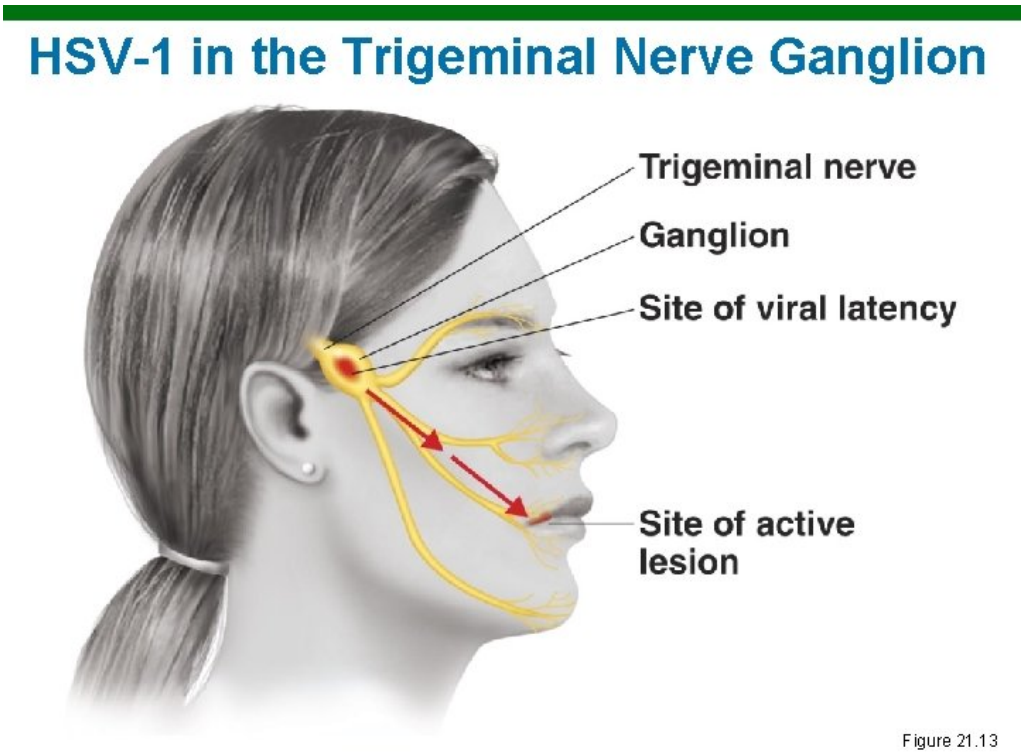

After the primary infection, HSV migrates to the trigeminal ganglion, where it remains in a latent state unless it is reactivated. Reactivation can be induced by exposure to sunlight, cold, trauma, stress, or immunosuppression, and may occur in the oral cavity (recurrent stomatitis) or on the lips (herpes labialis).

CLINICAL COURSE

The average duration of the most common signs and symptoms associated with gingivostomatitis are as follows:

- Fever – 4.4 days
- Oral lesions – 12 days
- Eating difficulty – 9.1 days
- Drinking difficulty – 7.1 days
- Drooling – 6.6 days

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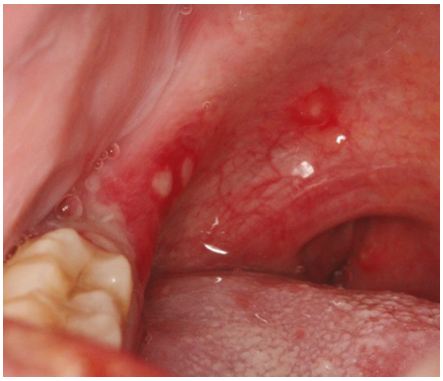
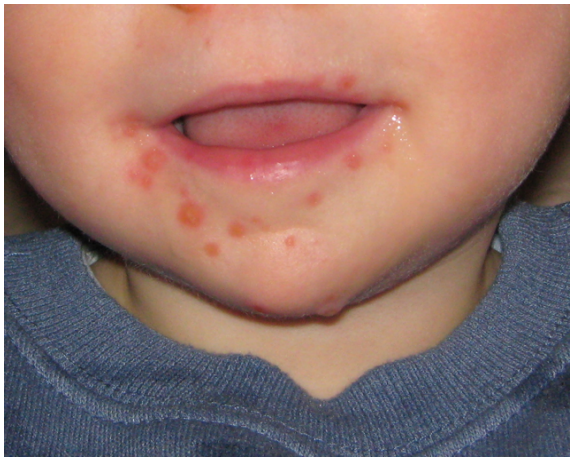
WHAT ELSE COULD IT BE?

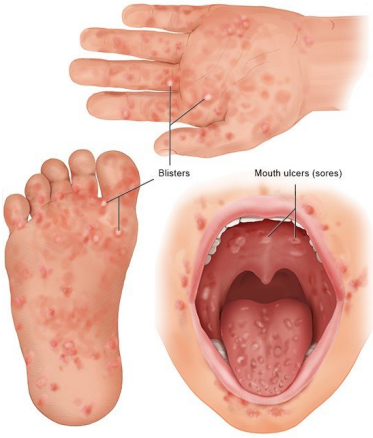
Herpes simplex virus (HSV) gingivostomatitis must be differentiated from a number of other conditions with oral lesions because the treatment, clinical course, and/or public health implications differ. HSV gingivostomatitis can usually be differentiated from these conditions by the presence, appearance, and location of oral and extraoral lesions. These conditions include:

- **Herpangina** – usually caused by group A coxsackieviruses, typically causes painful posterior pharyngeal lesions that do not bleed (picture 8), has a more acute onset and shorter duration than HSV gingivostomatitis, and occurs primarily in the summer and early fall.



- **Hand, foot, and mouth disease (HFMD)** – caused by a number of coxsackie A and B viruses. It occurs in the spring and early summer. The extremity lesions usually are bilateral (in contrast to herpetic whitlow, which typically is unilateral).





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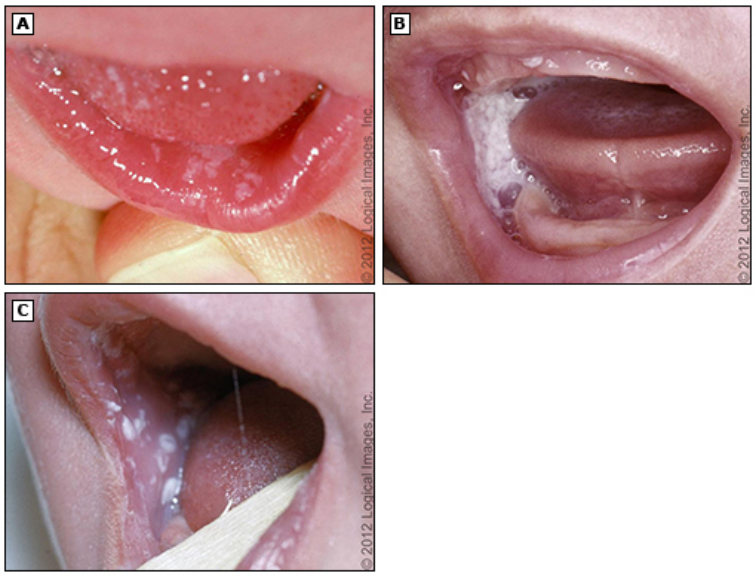
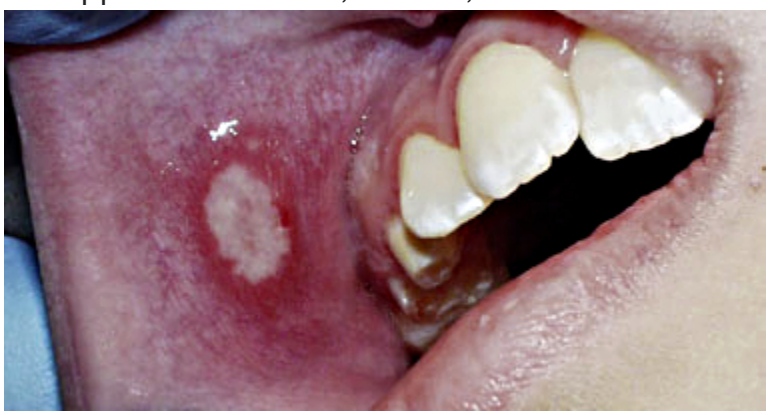
The associated extremity lesions and seasonality are most helpful in distinguishing HFMD from herpetic gingivostomatitis, even if the oral lesions appear in the anterior oropharynx.

• **Oral candidiasis** – Oral candidiasis is characterized by white plaques on the buccal mucosa, palate, tongue, or oropharynx.



It occurs in infants after exposure to the microorganisms, in children who are treated with antibiotics or inhaled glucocorticoids, and in immunocompromised children.

• **Aphthous ulcers** – Aphthae, also called canker sores, are painful oral lesions that appear as localized, shallow, round to oval ulcers with a grayish base



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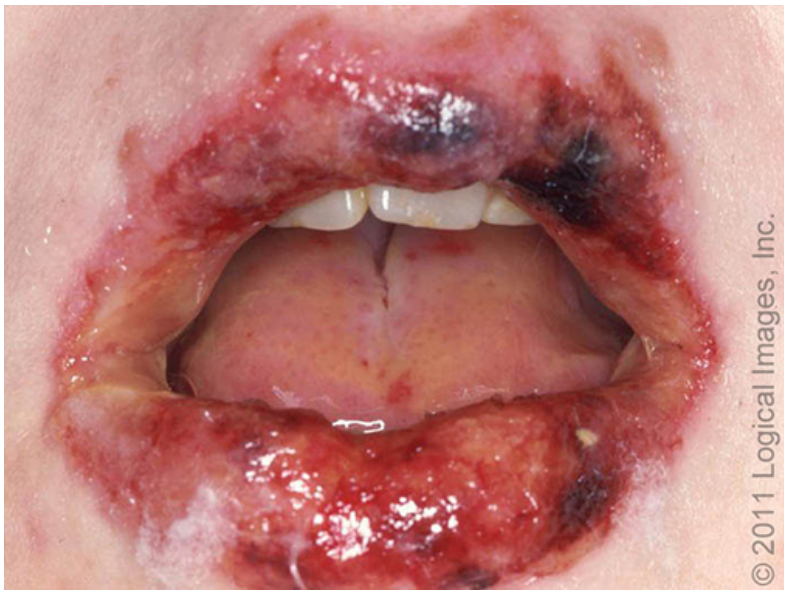
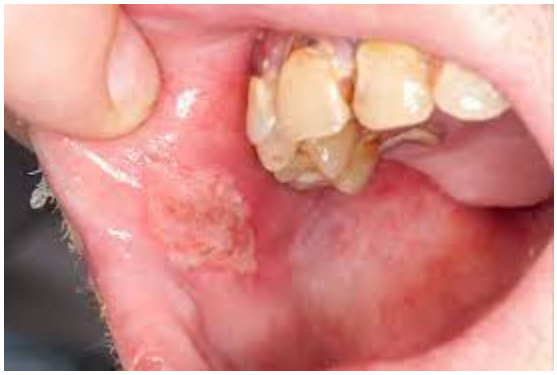
They are not preceded by vesicles and occur exclusively on nonkeratinized mucosal surfaces (the inside of lip, inside of cheek, portion of “gums” farthest away from the teeth

• **Stevens-Johnson syndrome** – characterized by a prodrome of malaise and fever, followed by the rapid onset of erythematous or purpuric macules and plaques. The skin lesions progress to epidermal necrosis and sloughing. Mucosal membranes are affected in at least 90 percent of patients



• **Behçet syndrome** – characterized by recurrent oral aphthae and any of several systemic manifestations, including genital aphthae, ocular disease, skin lesions, gastrointestinal involvement, neurologic disease, vascular disease, or arthritis.



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MANAGEMENT OF GINGIVOSTOMATITIS

Supportive care for all children

Fluid intake — Maintenance of hydration is a major component of the management of herpetic gingivostomatitis. Fluid intake should be encouraged to avoid dehydration. Children who are unable to drink sufficiently to maintain hydration should be hospitalized for IV fluid therapy.

Pain control — Adequate pain control may facilitate fluid intake. Oral acetaminophen or ibuprofen may be administered if the child can take them. In severe cases, such as inability to sleep, oral opiates may be required. Opiates should be used with caution; potential adverse effects include respiratory depression, central nervous system (CNS) depression, hypotension, and constipation.

Prevention of lip adhesions — We suggest application of a barrier cream (eg, petroleum jelly) to the lips to prevent adhesions in young children with HSV gingivostomatitis.

Immunocompromised children — For immunocompromised children with herpetic gingivostomatitis, we recommend treatment with IV or oral acyclovir rather than supportive care alone, **regardless of symptom duration before presentation**. The recommendation for systemic acyclovir is based upon randomized trials demonstrating that systemic acyclovir shortens the duration of symptoms and viral shedding in immunocompromised patients with mucocutaneous HSV.

Topical therapies are potentially problematic for a number of reasons:

- Systemic absorption of diphenhydramine, lidocaine, or benzocaine (in a “Magic Mouthwash” formulation) may result in toxicity or adverse effects, such as sedation or seizures.

- Topical benzocaine (i.e. over the counter topical ointments for canker sores) may cause methemoglobinemia and should not be used in children younger than two years.