VIRAL INFECTION

Prepared by:

Dr. Reyadh Al-Rashidi

Oral infections:

1) Viral infections

- Herpes simplex virus (HSVs) infections occur in two forms - primary (systemic) and secondary (localized). Both forms are self-limited, but recurrences of the secondary form are common because the virus can remain within ganglionic tissue in a latent state. **Physical contact with an infected individual or with body fluids is the typical route of HSV inoculation and transmission**. During the primary infection, only a small percentage of individuals show clinical signs and symptoms of infectious systemic disease, whereas a vast majority experience only subclinical disease.
- After resolution of primary herpetic lesion, the virus is believed to migrate, through some unknown mechanism, to the trigeminal ganglion.
- Reactivation of virus may follow exposure to sunlight (“fever blisters”), exposure to cold (“cold sores”), trauma, stress, or immunosuppression causing a secondary or recurrent infection.

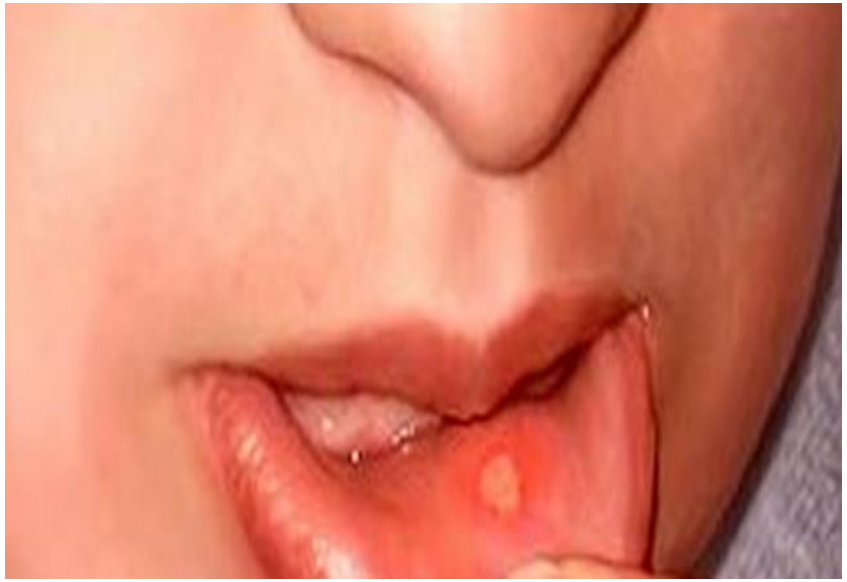
Clinical Features

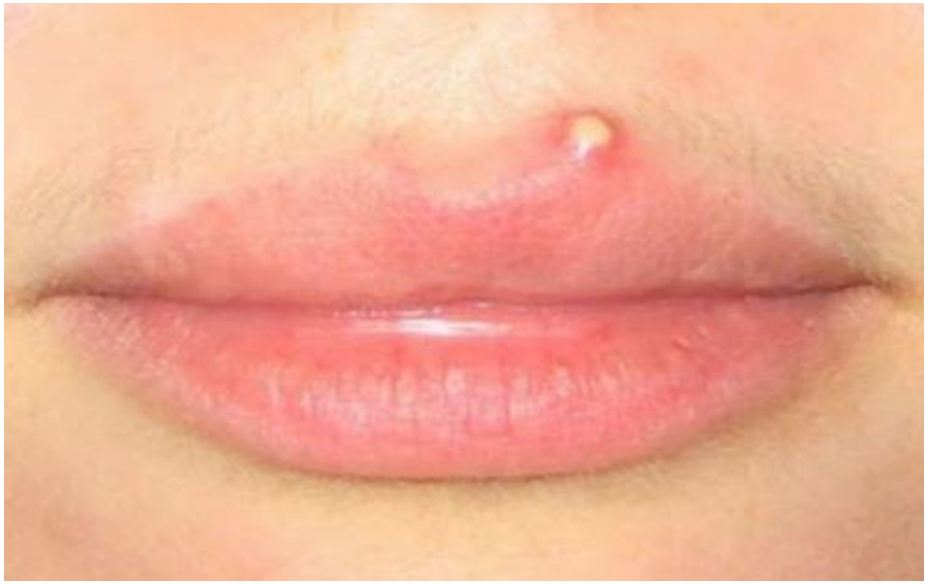
Primary Herpetic Gingivostomatitis. Primary disease is usually seen in children, although adults who have not been previously exposed to HSV may be affected. The vesicular eruption may appear on the skin, vermilion, and oral mucous membranes.

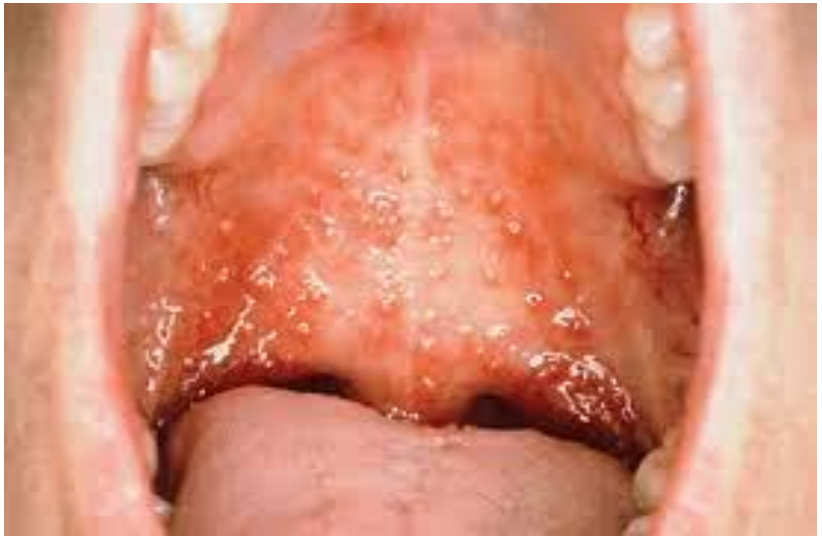
Intraorally,

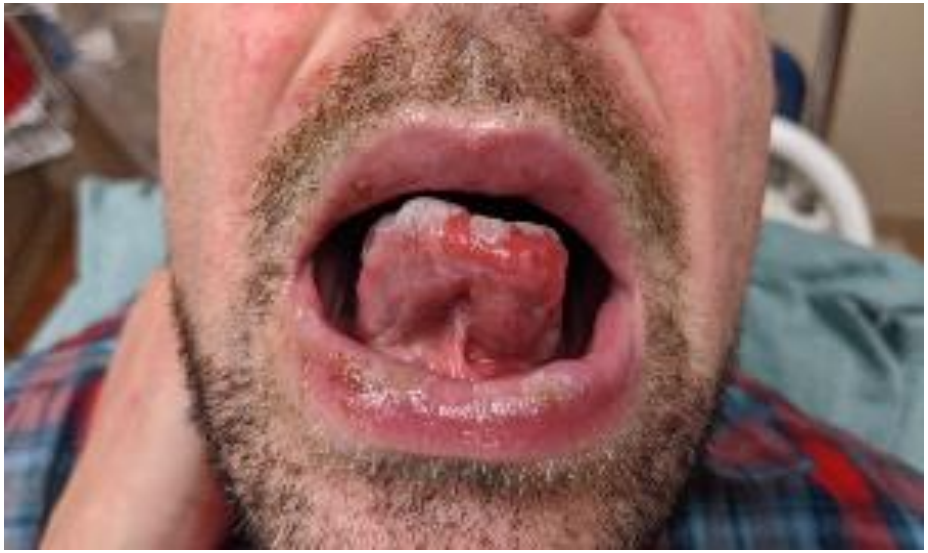
1. Primary lesions may appear on any mucosal surface.
2. Secondary (recurrent) form of the disease, lesions are confined to the lips, hard palate, and gingiva.

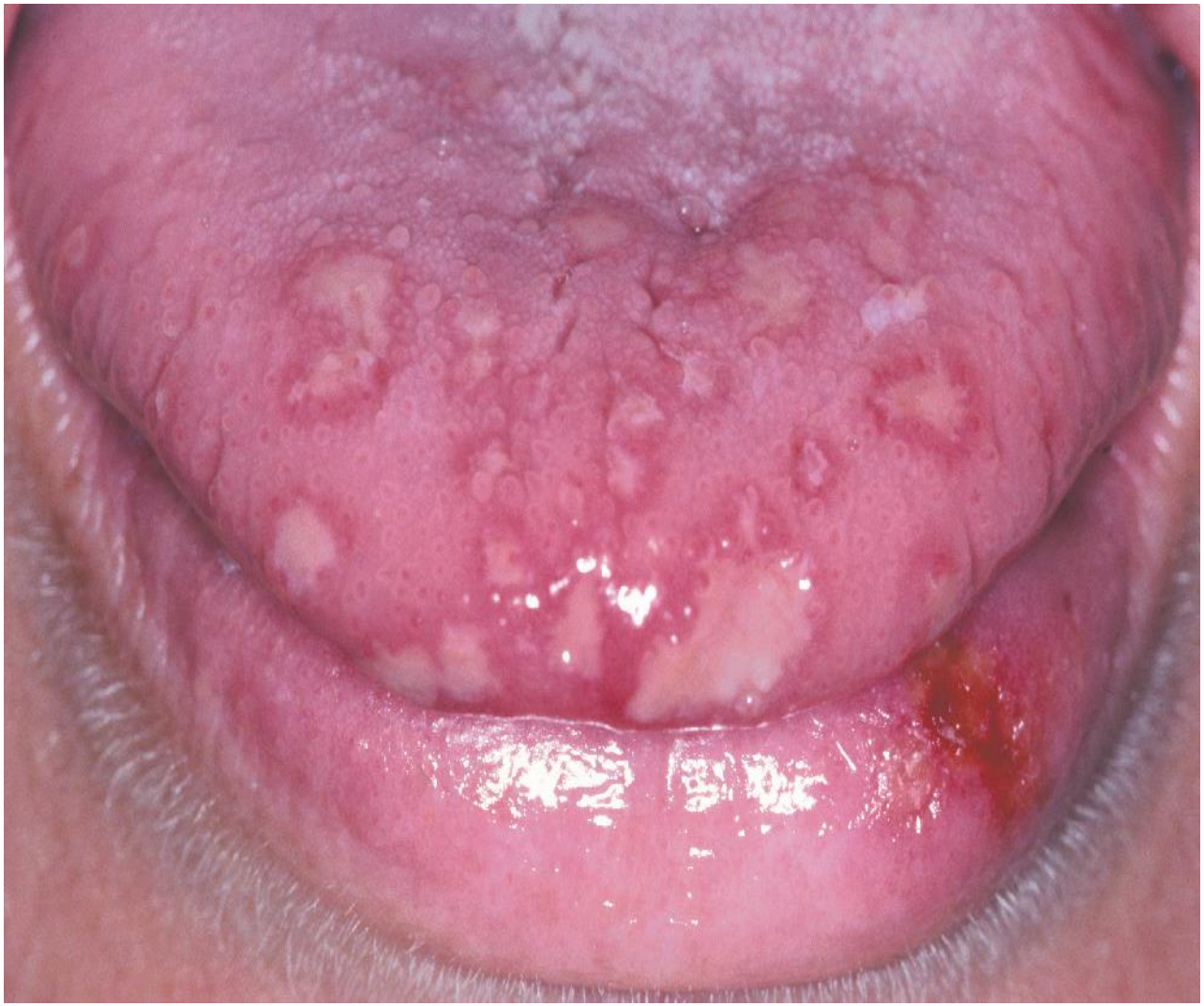
The primary lesions are accompanied by fever, arthralgia, malaise, anorexia, headache, and cervical lymphadenopathy. After the systemic primary infection runs its course of about 7 to 10 days, lesions heal **without scar formation**. By this time, the virus may have migrated to the **trigeminal ganglion** to reside in a latent form. Secondary (recurrent) herpes represents the reactivation of latent virus. Antibodies to HSV are present in a large majority of the population (up to 90%), and up to 40% of this group may develop secondary herpes.



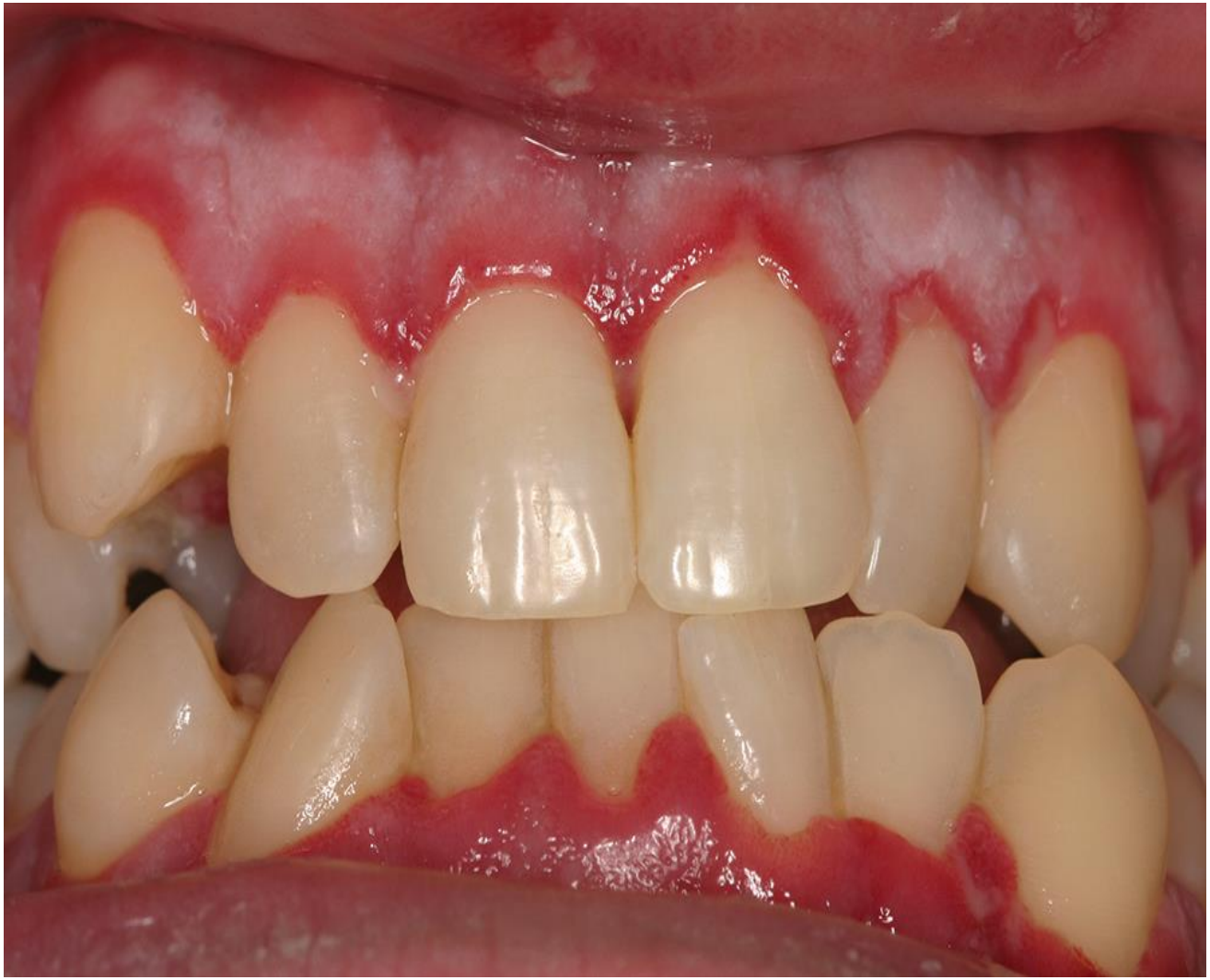




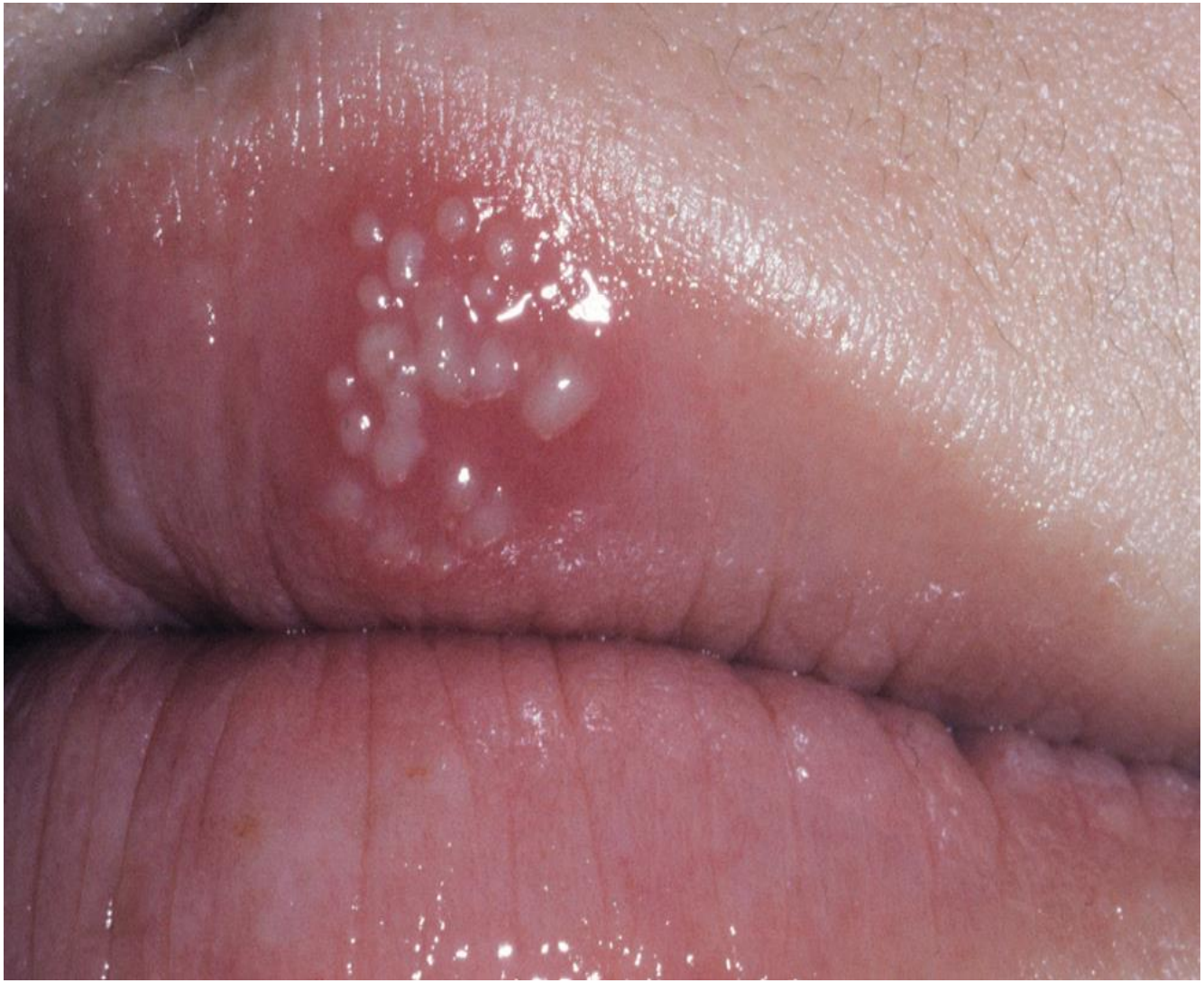




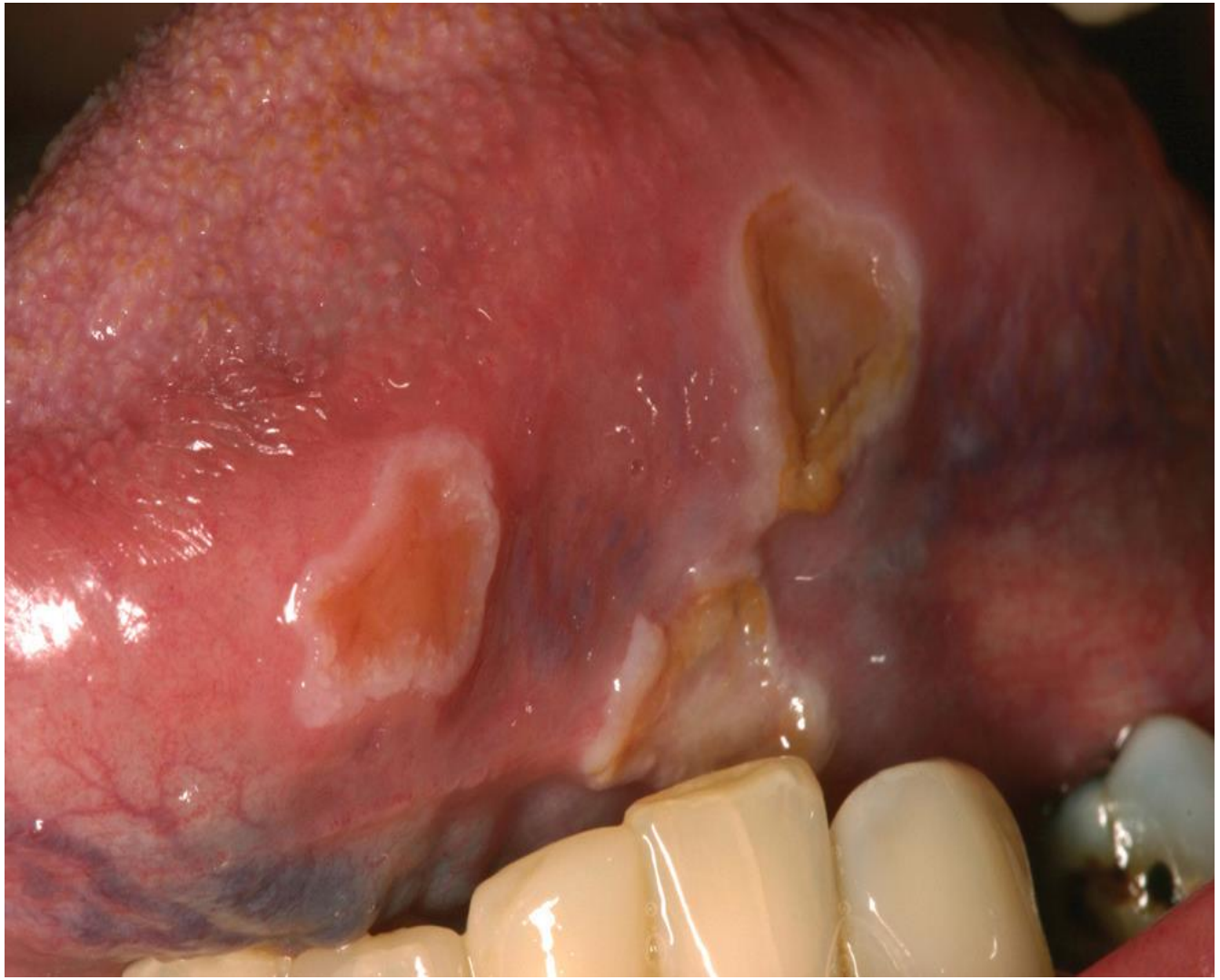
Acute Herpetic Gingivostomatitis. Numerous coalescing, irregular, and yellowish ulcerations of the dorsal surface of the tongue.



Acute Herpetic Gingivostomatitis. Painful, enlarged, and erythematous facial gingiva. Note erosions of the free gingival margin.



Herpes Labialis. Multiple fluid-filled vesicles on the lip vermilion.



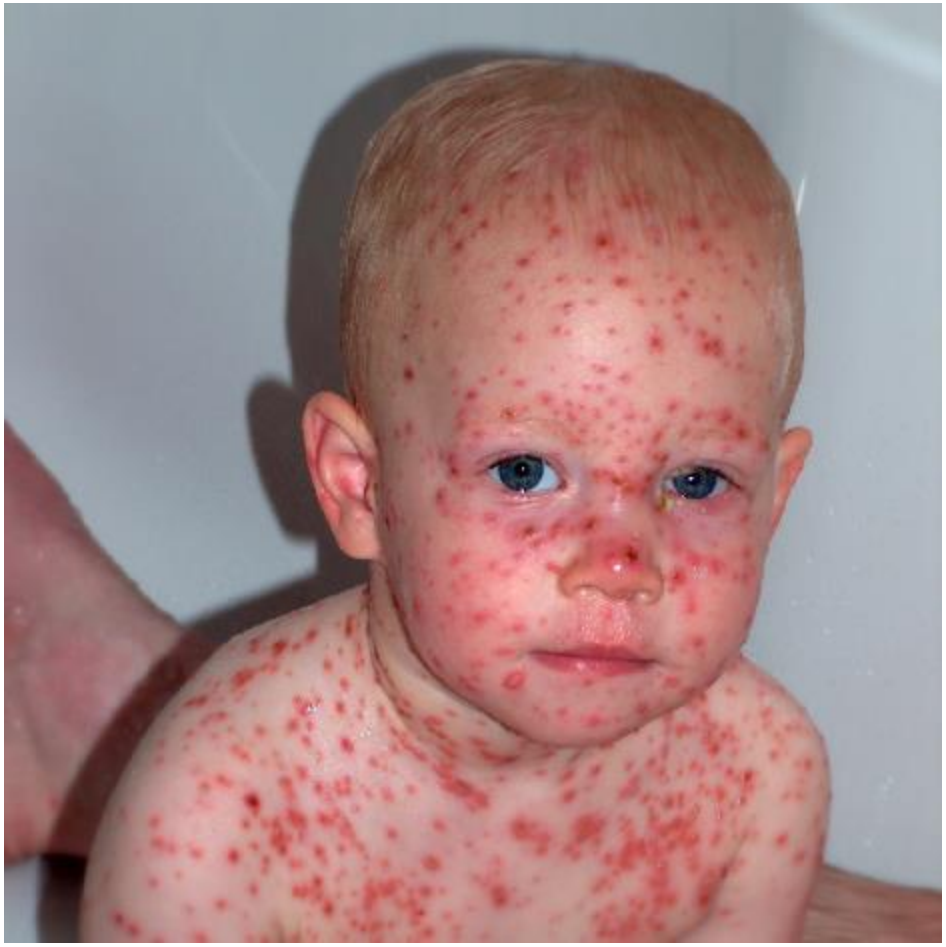
Chronic Herpetic Infection. Numerous mucosal erosions, each of which is surrounded by a slightly raised, yellow-white border, in a patient receiving systemic corticosteroid therapy for systemic sclerosis and rheumatoid arthritis.

Varicella-zoster virus infection

Primary varicella-zoster virus (VZV) infection is known as varicella or **chickenpox**; secondary or reactivated disease is known as herpes zoster or shingles Varicella is believed to be transmitted predominantly through the **inhalation of contaminated droplets**. The condition is **very contagious** and is known to spread readily.

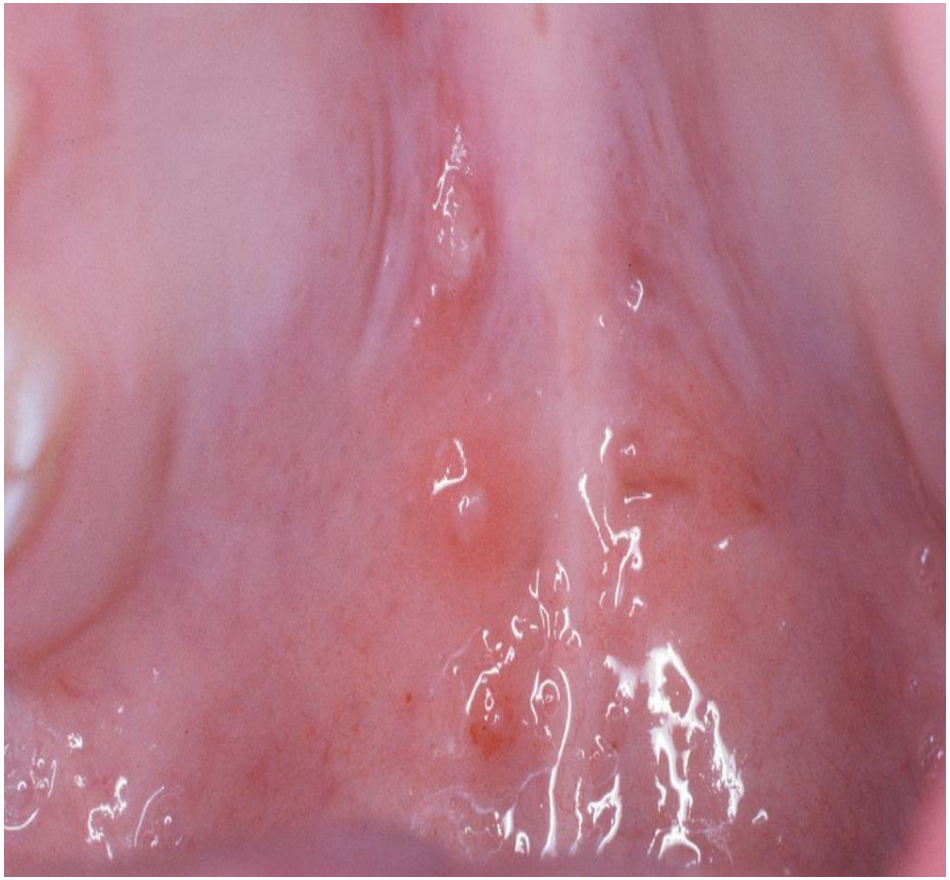
Clinical features

Fever, chills, malaise, and headache may accompany a rash that involves primarily the trunk and head and neck. The rash quickly develops into a vesicular eruption that becomes pustular and eventually ulcerates. The infection is self-limiting and lasts several weeks. Oral mucous membranes may be involved in primary disease and usually demonstrate multiple shallow ulcers that are preceded by vesicles.



Varicella. child with diffuse erythematous and vesicular rash.

Varicella - tongue



Varicella - hard palate.

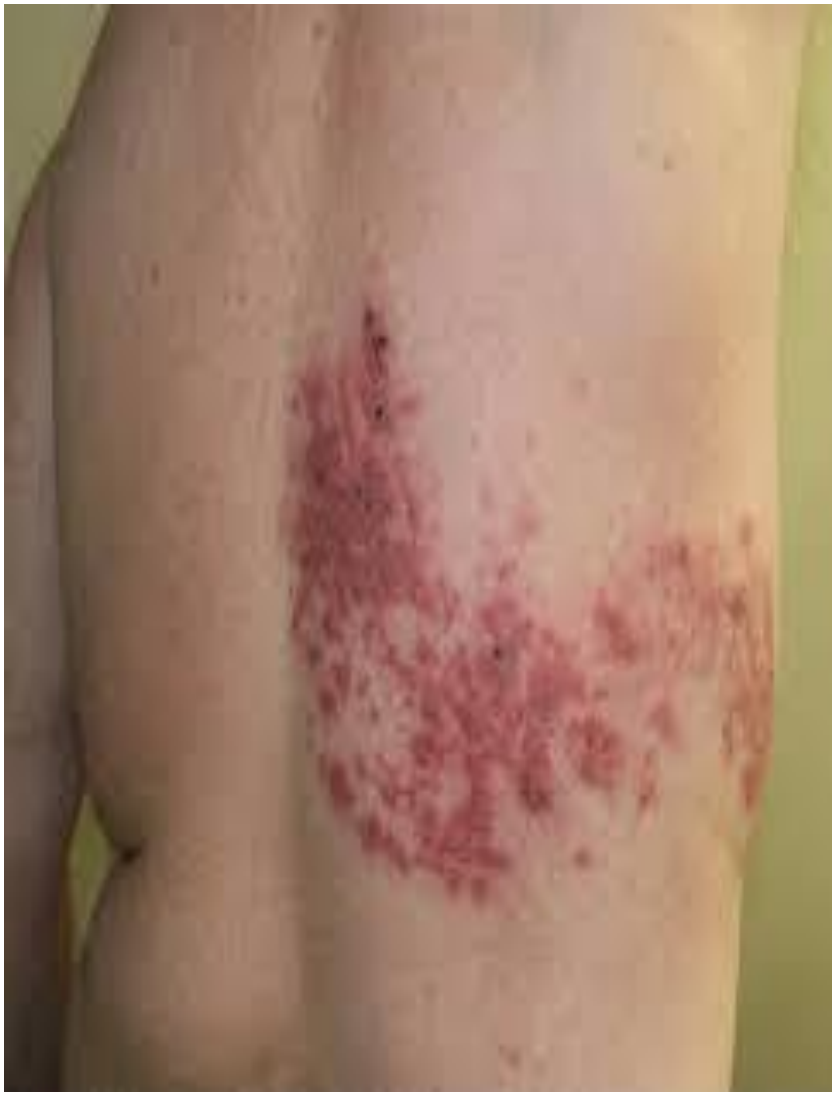
Herpes zoster (Shingles) (الحزام الناري)

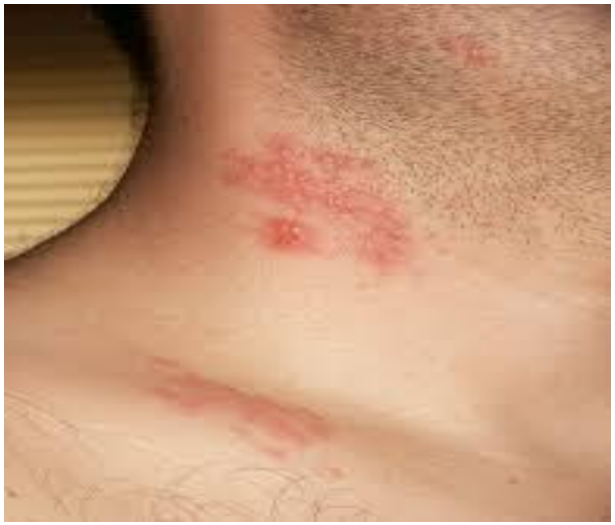
Caused by the reactivation of the varicella-zoster virus (VZV). A condition of the **older adult** population and of individuals who have compromised immune responses. After several days of prodromal symptoms of pain and/or paresthesia in the area of the involved dermatome, a well-delineated unilateral maculopapular rash appears. This may occasionally be accompanied by systemic symptoms. The rash quickly becomes vesicular, pustular, and then ulcerative. Remission usually occurs in several weeks.

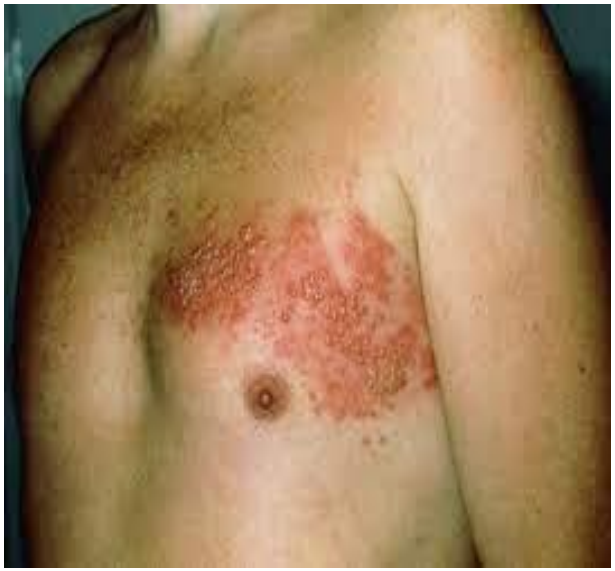
Histopathology: Essentially the same as those with HSV

Treatment: For varicella in normal individuals, supportive therapy is generally indicated. However, for immunocompromised patients, more substantial measures are warranted. These include systemically administered acyclovir, vidarabine, and human leukocyte interferon. **Corticosteroids generally are contraindicated**.

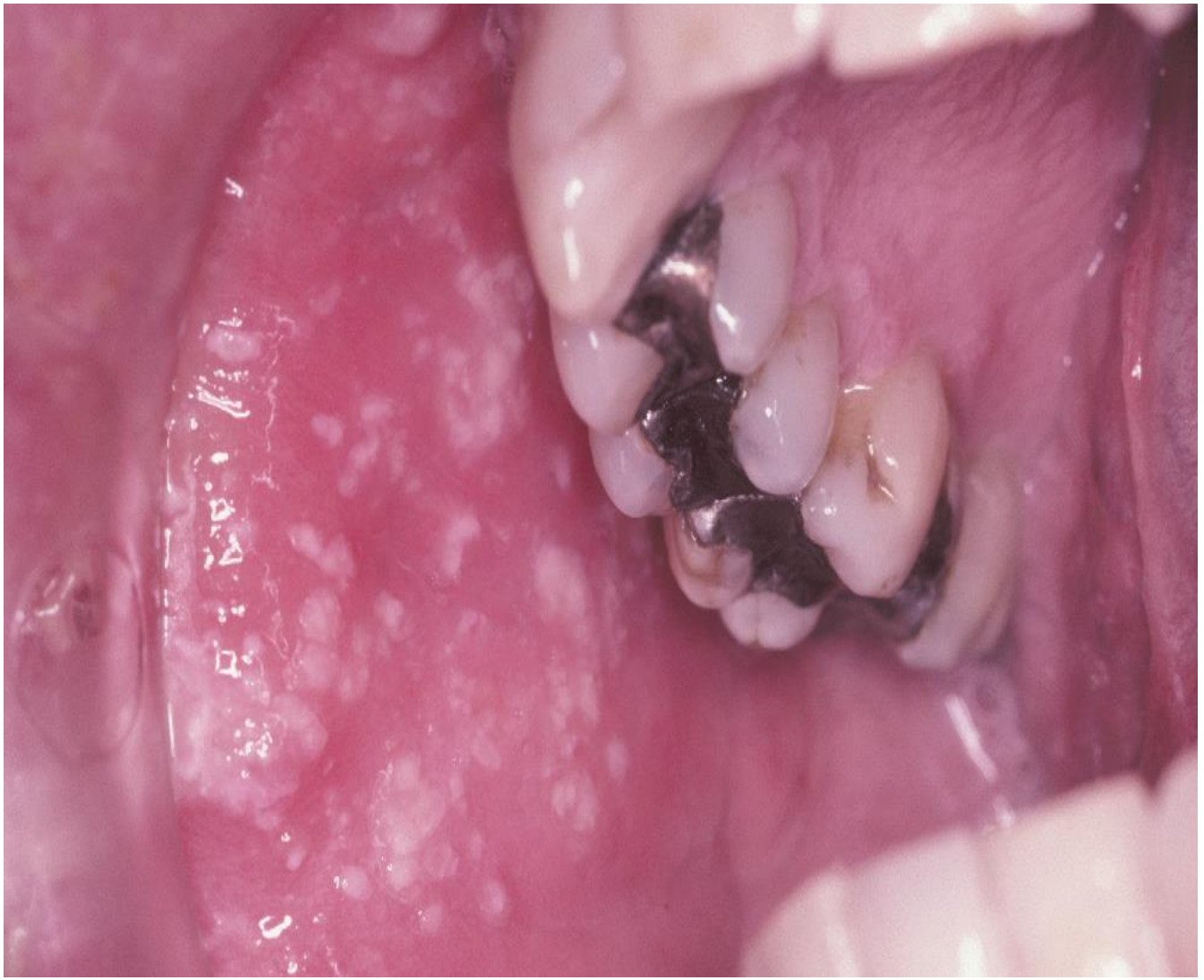
- The sensory nerves of the trunk and head and neck are commonly affected. Involvement of various branches of the trigeminal nerve may result in unilateral oral, facial, or ocular lesions. Involvement of facial and auditory nerves produces the **Ramsay Hunt syndrome**, in which facial paralysis is accompanied by vesicles of the ipsilateral external ear, tinnitus, deafness, and vertigo.







Herpes Zoster. Numerous crusting facial vesicles that extend to the midline. Shingles (الحزام الناري)



Herpes Zoster. Numerous white opaque vesicles on the right buccal mucosa of the same patient

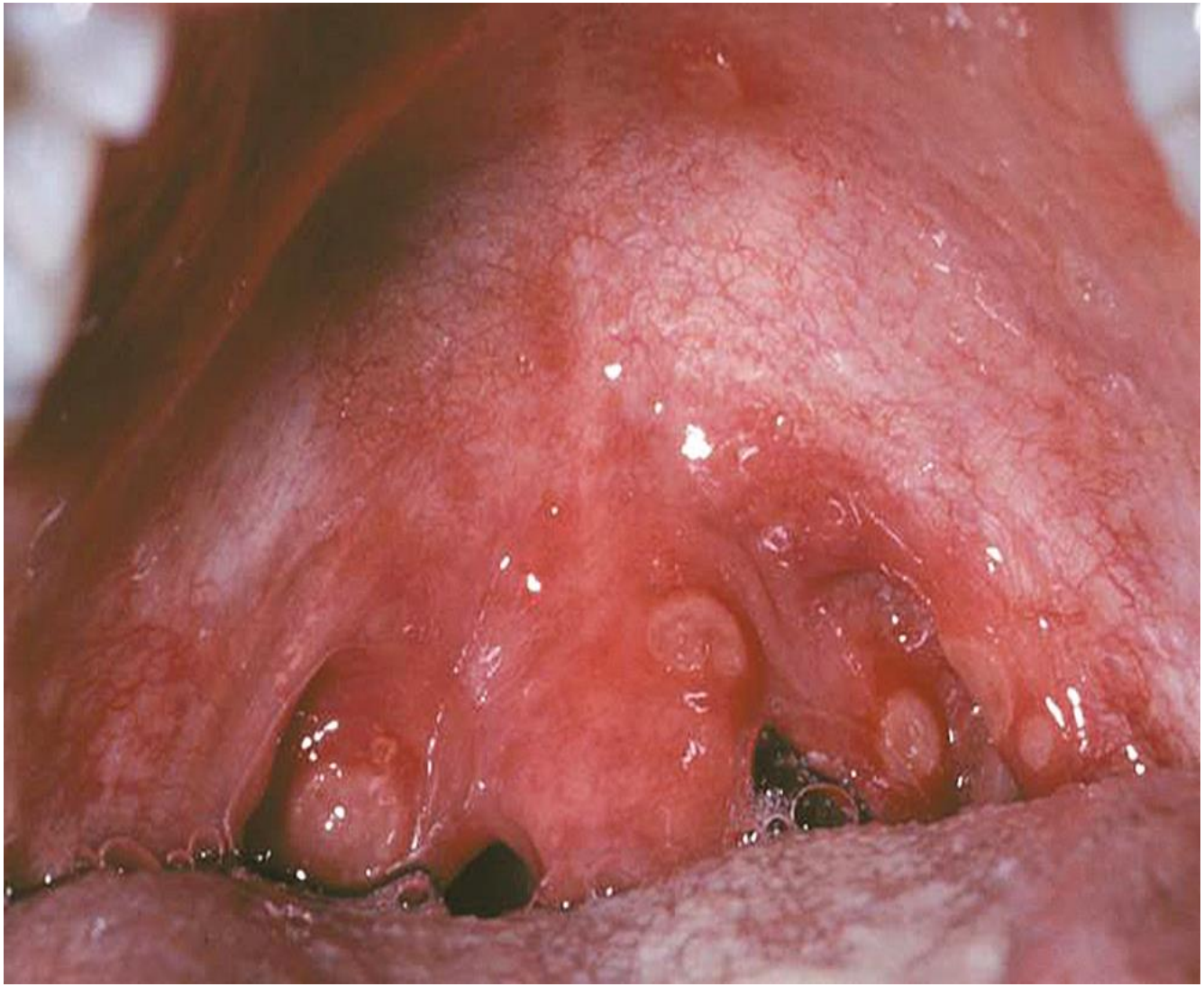
Herpangina

Herpangina is an acute viral infection caused by **Coxsackie type A and Type B virus and enterovirus type 71**. It is transmitted by **contaminated saliva** and occasionally through contaminated feces.

Clinical Features.

- Herpangina is usually endemic, with outbreaks occurring typically in summer or early autumn. It is more common in children than in adults. Those infected generally complain of malaise, fever, dysphagia, and sore throat after a short incubation period.
- Intraorally, a vesicular eruption appears on the soft palate, faucial pillars, and tonsils and persists **for 4 to 6 days**. A diffuse erythematous pharyngitis is also present. No associated skin lesions are typically seen.
- Signs and symptoms are usually mild to moderate and generally last less than a week.

Treatment. Because herpangina is self-limiting, is mild and of short duration, and causes few complications, treatment usually is not required.



Herpangina. Numerous aphthous-like ulcerations of the soft palate.

Hand-Foot-and-Mouth Disease

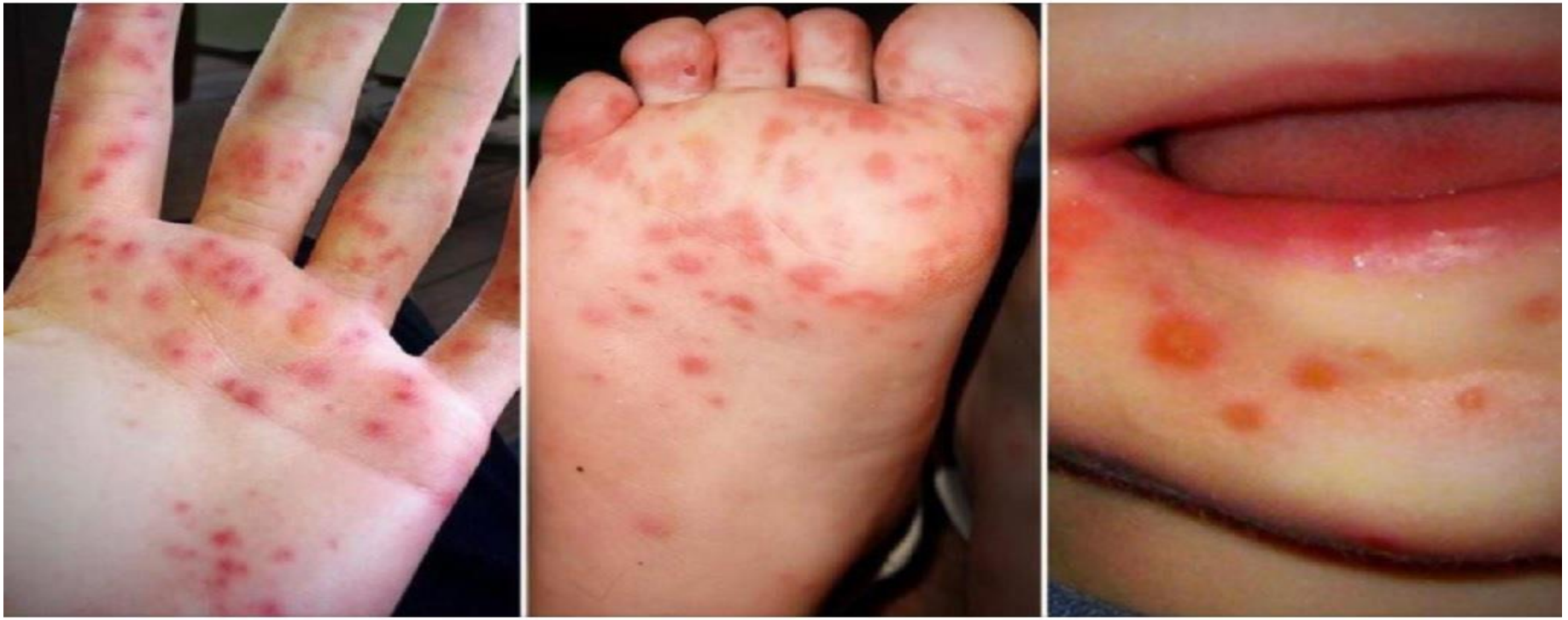
HFM disease is a highly contagious viral infection that usually is caused by **Coxsackie type A16 or enterovirus type 71**. The virus is transferred from one individual to another through airborne spread or fecal-oral contamination.

Clinical Features. This viral infection typically occurs in epidemic or endemic proportions and predominantly (about 90%) affects children younger than 5 years of age. After a short incubation period, the condition resolves spontaneously in 1 to 2 weeks. Signs and symptoms are usually mild to moderate in intensity and include low-grade fever, malaise, lymphadenopathy, and sore mouth. Pain from oral lesions is often the patient's chief complaint.

Oral lesions begin as vesicles that quickly rupture to become ulcers. Lesions can occur anywhere in the mouth, although the palate, tongue, and buccal mucosa are favored sites, while the lips and gingiva are usually spared. Multiple maculopapular lesions, typically on the feet, toes, hands, and fingers, appear concomitantly with or shortly after the onset of oral lesions. These cutaneous lesions progress to a vesicular state; they eventually become ulcerated.

Histopathology. The vesicles of this condition are found within the epithelium because of obligate viral replication in keratinocytes. Eosinophilic inclusions may be seen within some of the infected epithelial cells

Treatment. Because of the relatively short duration, generally self-limiting nature, and general lack of virus-specific therapy, treatment for HFM disease is usually symptomatic



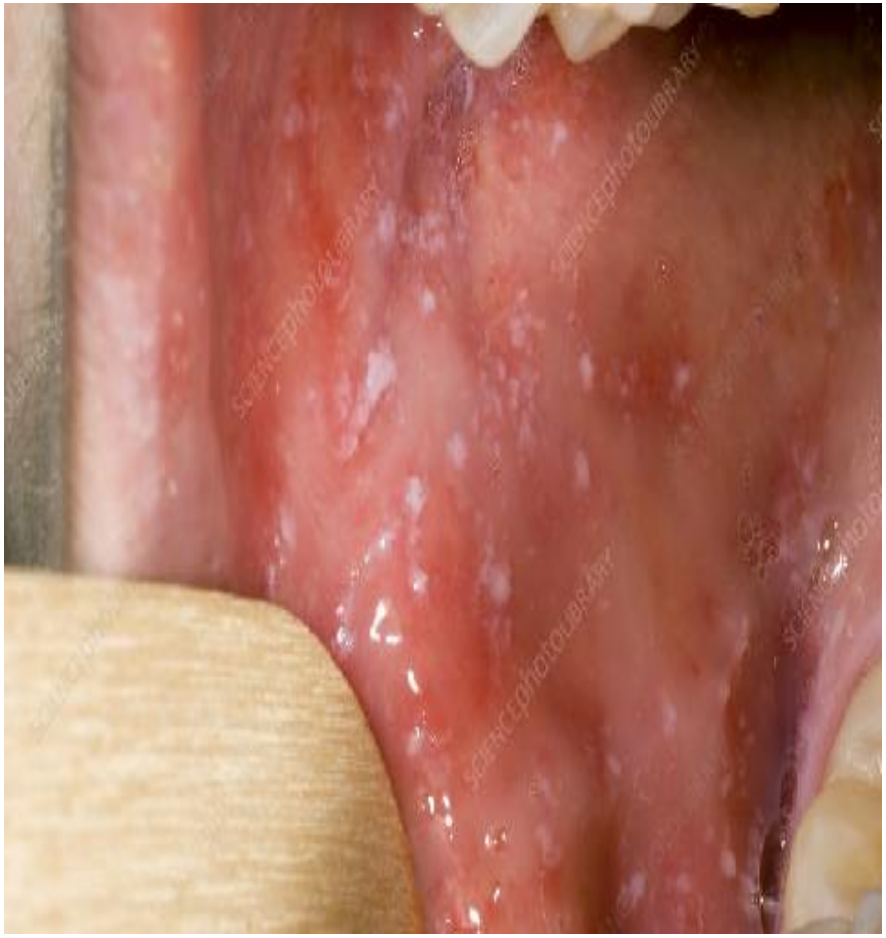
Hand-foot-and-mouth Disease.

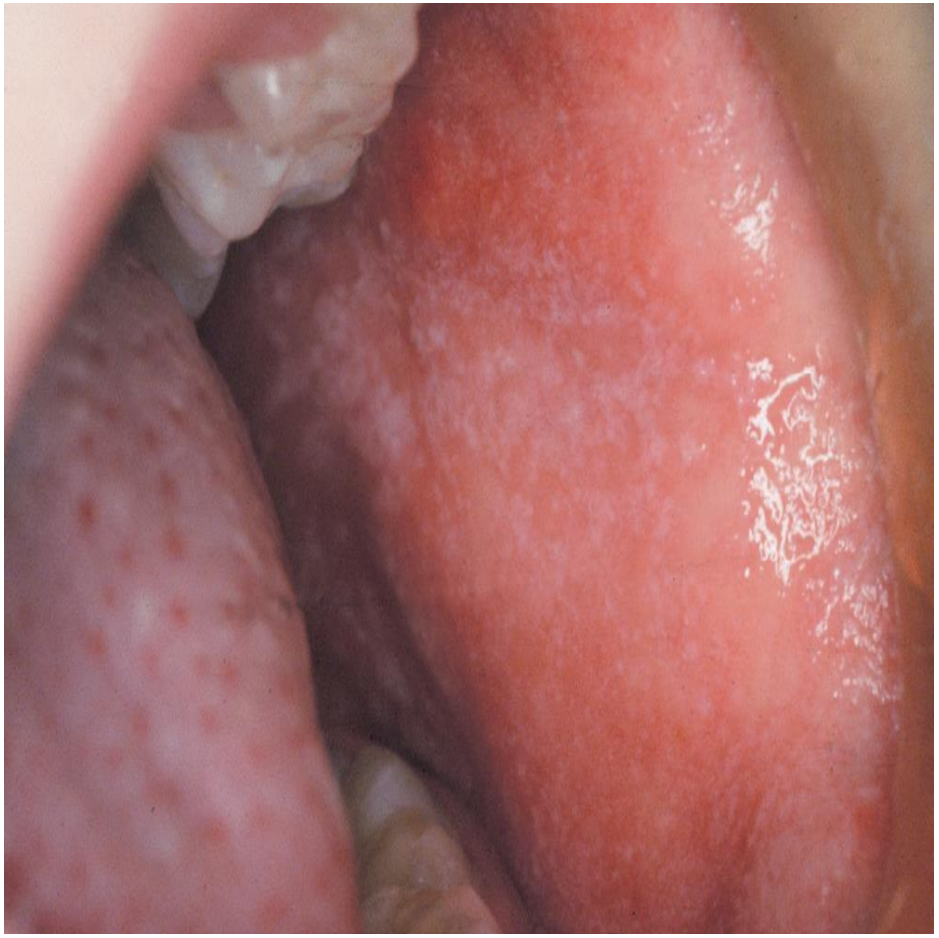
Measles (Rubeola) and German measles (Rubella)

Measles is a highly contagious viral infection caused by a member of the paramyxovirus family of viruses. Typically, oral eruptions consist of early pinpoint elevations over the soft palate that combines with ultimate involvement of the pharynx with bright erythema. German measles, or rubella, is a contagious disease that is caused by an unrelated virus of the togavirus family. It shares some clinical features with measles, such as fever, respiratory symptoms, and rash. However, these features are very mild and short lived in German measles.

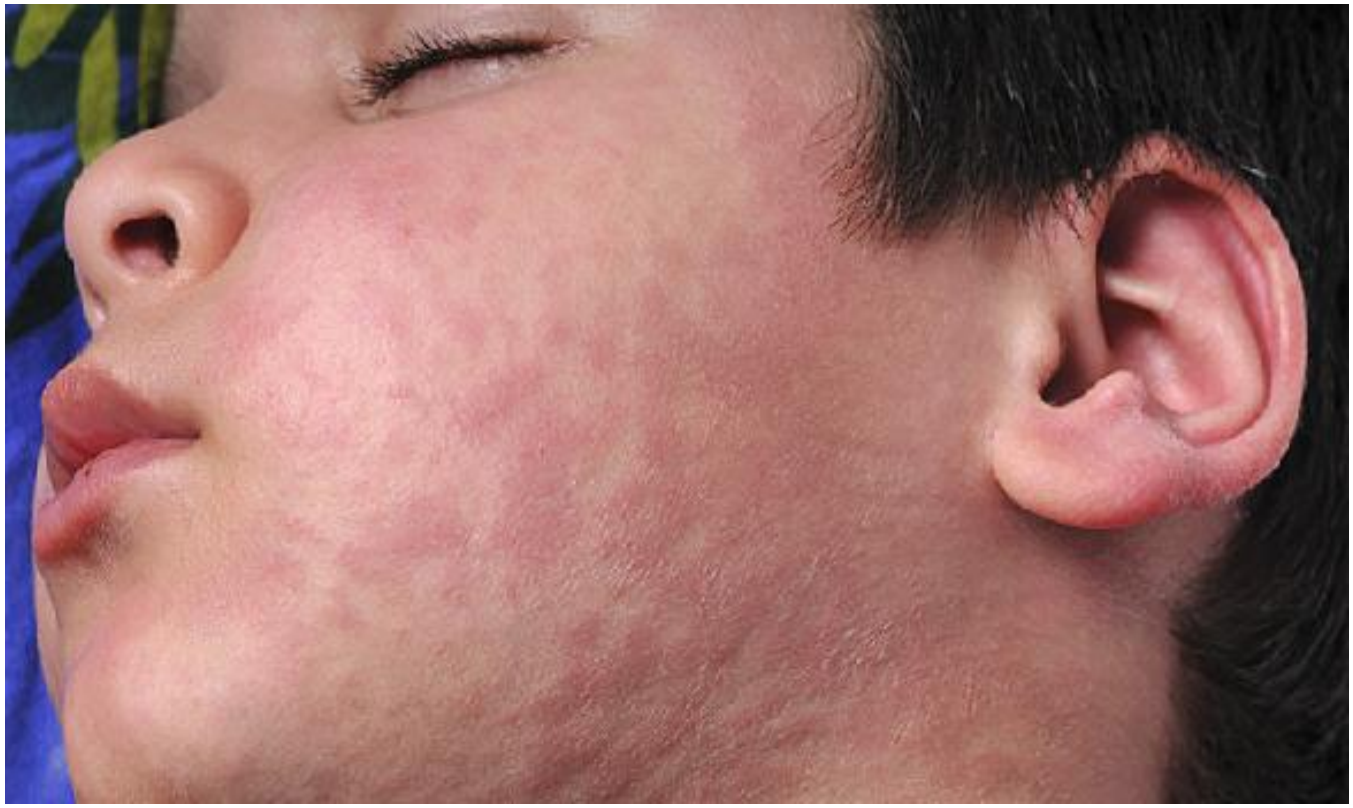
Clinical Features. After an incubation period of 7 to 10 days, prodromal symptoms of fever, malaise, coryza, conjunctivitis, photophobia, and cough develop. In 1 to 2 days, **pathognomonic** (diagnostic) small erythematous macules with white necrotic centers appear in the buccal mucosa, these lesion spots, known as **Koplik's spots**.

Koplik's spots generally precede the skin rash by 1 to 2 days. The rash initially affects the head and neck, followed by the trunk, and then the extremities.





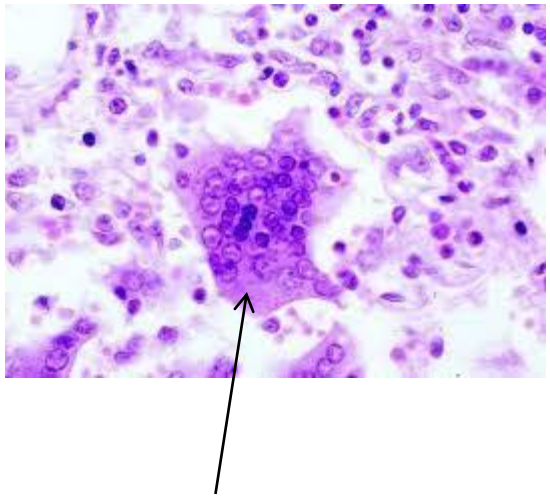
Rubeola. Numerous blue-white Koplik’s spots of buccal mucosa.



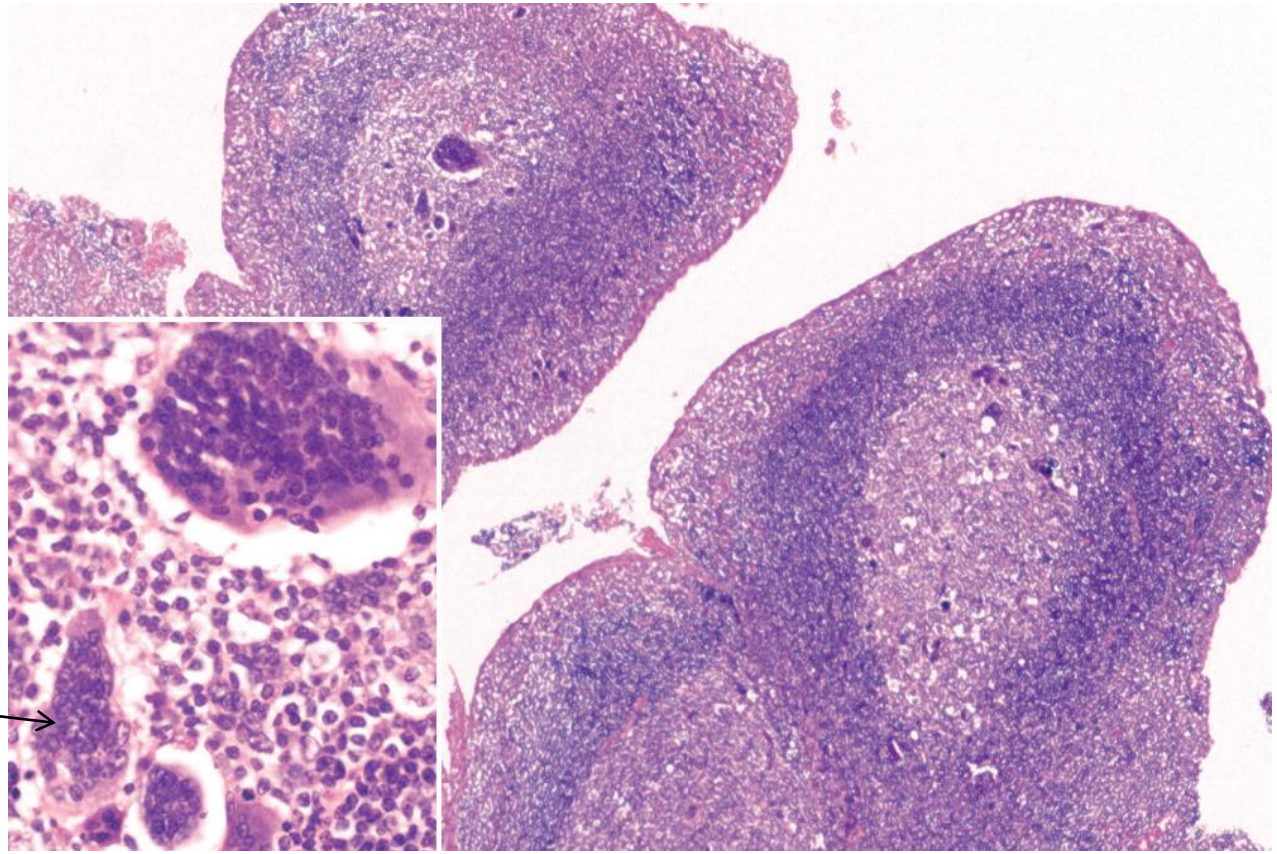
Rubeola Erythematous maculopapular rash of the face.

Histopathology: Infected epithelial cells, which eventually become necrotic, overlie an inflamed connective tissue that contains dilated vascular channels and a focal inflammatory response. Lymphocytes are found in a perivascular distribution. In lymphoid tissues, you can see characteristic **Warthin-Finkelday giant cells** (giant multinucleated lymphocytes).

Treatment: No specific treatment for measles is known. Supportive therapy of bed rest, fluids, adequate diet, and analgesics generally suffices



Warthin-Finkelday giant cells



References

Oral and Maxillofacial Pathology - Brad W. Neville