

BDS Year 4 Regular & Casual batch Academic Year 2023-2024 Subject: Oral Medicine and Radiology Topic: VESICULOBULLOUS AND ULCERATIVE LESIONS-I

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Introduction

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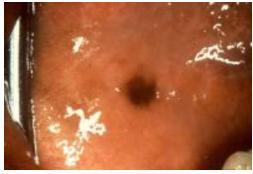
A loss or break in the continuation of surface epithelium or mucous membrane that extends into lamina propria. Oral ulcers are confirmed by the underlying systemic condition such as the nature, site, duration and frequency.

♦ A vesiculobullous disease is a type of mucocutaneous disease characterized by vesicles and bullae (i.e. blisters). Both vesicles and bullae are fluid-filled lesions, and they are distinguished by size





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- Macules-These are lesions that are flush with the adjacent mucosa and that are noticeable because of their difference in color from normal skin or mucosa. They may be red due to increased vascularity or inflammation, or pigmented due to the presence of melanin, hemosiderin, and foreign material (including the breakdown products of medications).
- A good example in the oral cavity is the melanotic macule.









Papules-These are lesions raised above the mucosal surface that are smaller than 1.0 cm in diameter (some use 0.5 cm for oral mucosal lesions). They may be slightly domed, or flat-topped. Papules are seen in a wide variety of diseases, such as the yellow-white papules of pseudomembranous candidiasis.





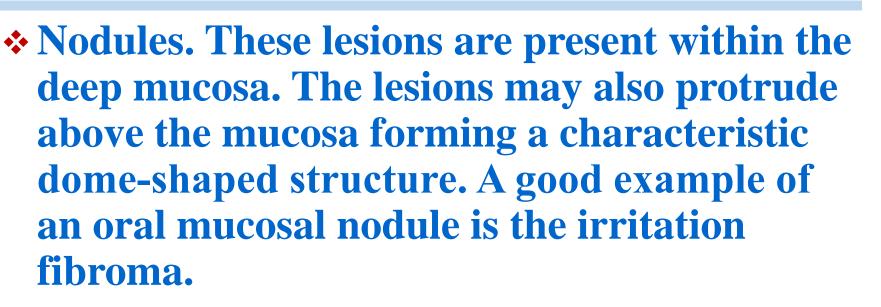


Plaques-These are raised lesions that are greater than 1 cm in diameter; they are essentially large papules









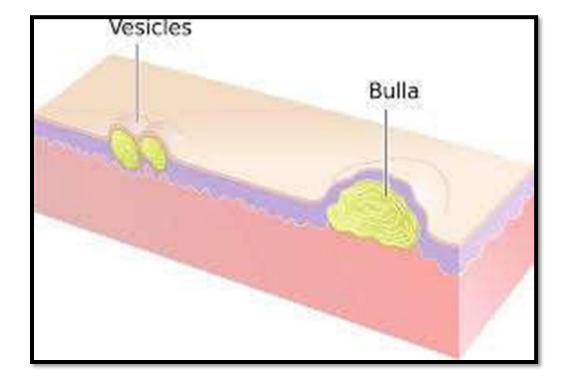




Sullae-These are elevated blisters containing clear fluid that are greater than 1 cm in diameter.













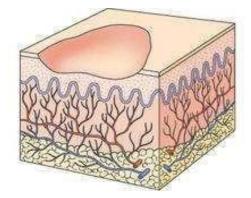


Erosions. These are red lesions often caused by the rupture of vesicles or bullae, or trauma and are generally moist on the skin. However, they may also result from thinning or atrophy of the epithelium in inflammatory disease such as lichen planus. These should not be mistaken for ulcers, which are covered with fibrin and are yellow.

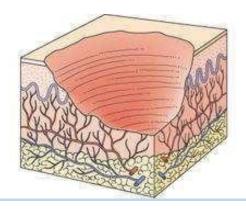






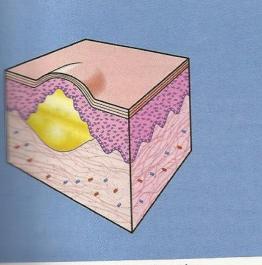




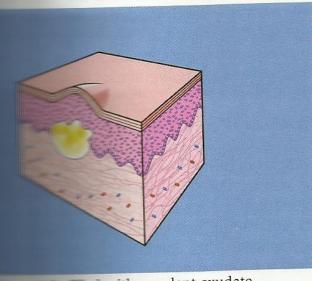








fluid-filled skin elevation.



we with purulent exudate.



Fig. 11.2. Vesicle: recurrent herpes simplex.

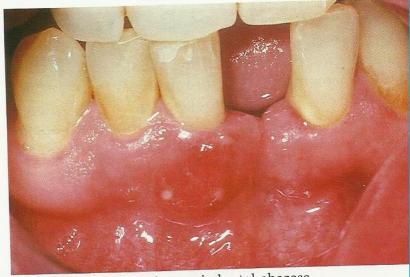
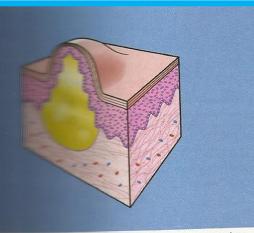


Fig. 11.4. Pustule: pointing periodontal abscess.

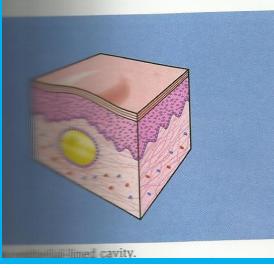




Imme Inid-filled mucocutaneous elevation.



Fig. 11.6. Bulla: bullous lichen planus—a rare finding.



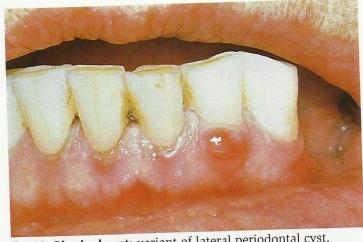


Fig. 11.8. Gingival cyst: variant of lateral periodontal cyst.







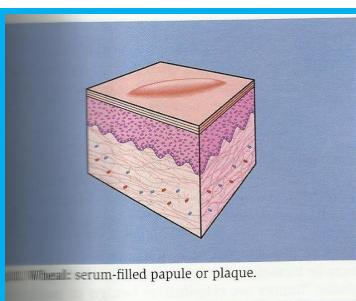




Fig. 9.2. Wheal: dermatographism after rubbing the skin.

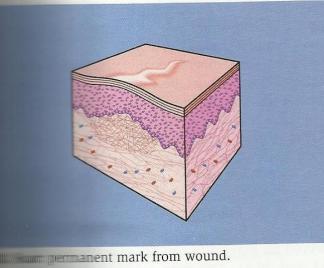
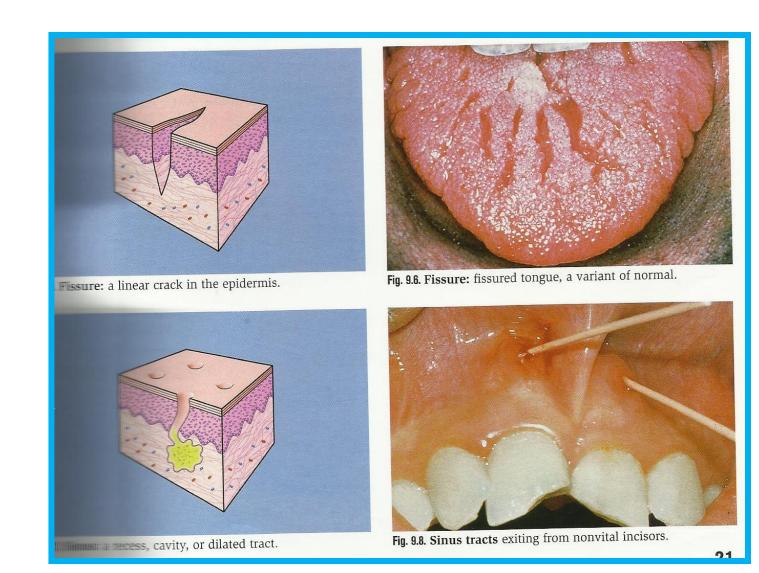




Fig. 9.4. Scar: fibrotic tissue as a result of trauma.







ALL ORAL LESIONS HAVE SIMILAR C/F

- *** HISTORY**
- **CLINICAL EXAMINATION**
- **SIMPLIFY THE DIAGNOSIS:**
 - ACUTE/CHRONIC
 - PRIMARY/RECURRENT
 - SINGLE/MULTIPLE





*** History:**

- Other lesions in eye, skin, genital and rectal regions
- Source States States
- Dermatologic lesions



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Dermatologic lesions

Macules: Well-circumscribed, flat lesions that are noticeable because of their change from normal skin color. Red Pigmented Example: melanotic macule





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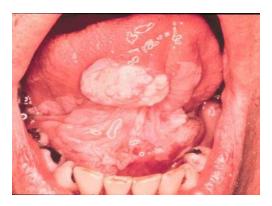
Papules: Solid lesions raised above the

skin/mucosal surface that are < 1 cm in diameter.

Eg: OLP,EM

Plaques: Solid raised lesions that are over

1 cm in diameter; they are large papules.











- ***** ACUTE MULTIPLE LESIONS
- ***** ACUTE RECURRENT LESIONS
- *** CHRONIC MULTIPLE LESIONS**
- **SOLITARY ULCERS**

5

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Herpes Simplex Virus Infections Varicella Zoster Virus (VZV) Infection Cytomegalovirus (CMV) Infection Epstein-Barr Virus Infection Coxsackievirus Infection Hand-Foot-and-Mouth Disease (HFM) Necrotizing Ulcerative Gingivitis and Periodontitis Erythema Multiforme Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TENs)

THE PATIENT WITH ACUTE MULTIPLE LESIONS

Plasma Cell Stomatitis and Oral Hypersensitivity Reactions

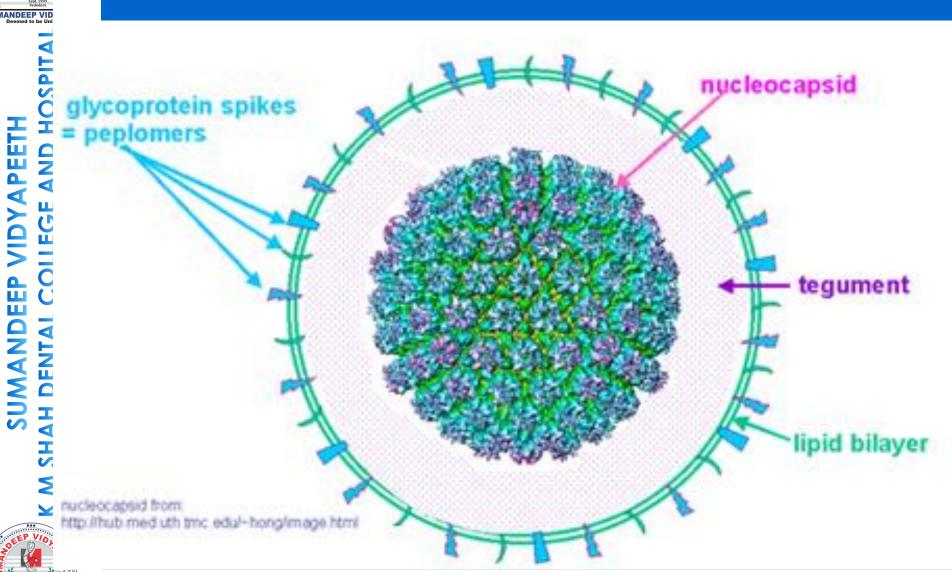
 THE PATIENT WITH RECURRING ORAL ULCERS Recurrent Aphthous Stomatitis (RAS) Behçet's Disease (Behçet Syndrome) THE PATIENT WITH CHRONIC MULTIPLE LESIONS Pemphigus
 Pemphigus Vulgaris (PV)
 Paraneoplastic Pemphigus (PNP)

Subepithelial Bullous Disorders Bullous Pemphigoid (BP) Mucous Membrane Pemphigoid [MMP] Linear IgA Disease (LAD) Epidermolysis Bullosa Aquisita (EBA)

THE PATIENT WITH SINGLE ULCERS Traumatic Injuries Causing Solitary Ulcerations Traumatic Ulcerative Granuloma (Eosinophilic Ulcer of Tongue) Infectious Ulcers











H	HSV-1	SENSORY GANGLIA
DYAPEETH GE AND H	HSV-2	SENSORY GANGLIA
	VZV	SENSORY GANGLIA
DEEP VII	CMV	SALIVARY GLAND TISSUE,LYMPHOCYTES
SUMAN AH DENT	HSV-1 HSV-2 VZV CMV HHV-6 HSV-7	B LYMPHOCYTES,SG TISSUE
M SH	HSV-7	CD4 LYMPHOCYTES





lerpes viruses

Table 3-1 Herpesviridae that are pathogenic in humans.

	Type of Human herpesvirus (HHV)	Primary infection	Recrudescent lesions in healthy hosts	Recrudescent lesions in immunocompromised hosts	
	Herpes simplex virus 1 (HHV-1)	Gingivostomatitis, keratoconjunctivitis, genital and skin lesions	Herpes labialis ("cold sores"), intraoral ulcers, keratoconjunctivitis, genital and skin lesions	Ulcers at any mucocutaneous site, usually large and persistent; disseminated infection	
	Herpes simplex virus 2 (HHV-2)	Genital and skin lesions, gingivostomatitis, keratoconjunctivitis, neonatal infections, aseptic meningitis	Genital and skin lesions gingivostomatitis, aseptic meningitis	Ulcers at any mucocutaneous site, usually large, persistent and dermatomal; disseminated infection	
	Varicella-zoster virus (HHV-3)	Varicella (chickenpox)	Zoster (shingles)	Disseminated infection	
	Cytomegalovirus (HHV-4)	Infectious mononucleosis, hepatitis, congenital disease		Retinitis, gastroenteritis hepatitis, severe oral ulcers	
	Epstein-Barr virus (HHV-5)	Infectious mononucleosis-like, hepatitis, encephalitis		Hairy leukoplakia, lymphoproliferative disorders, mucocutaneous ulcers	
	HHV-6	Roseola infantum, otitis media, encephalitis		Fever, bone marrow suppression	
	HHV-7	Roseola infantum			
	HHV-8	Infectious mononucleosis-like, febrile exanthema		Kaposi sarcoma, lymphoproliferative disorders, bone marrow suppression	

Contraction of the second seco



Herpes Simplex Infections

Common vesicular eruptions of skin and mucosa



Systemic or primary Localised or Secondary





Pathogenesis

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 Physical contact with an infected individual is typical route of HSV inoculation for a seronegative individual

- Virus binds to cell surface via heparan sulphate ,followed by sequential activation of specific genes during lytic phase
- Activation of intermediate ,early and late proteins

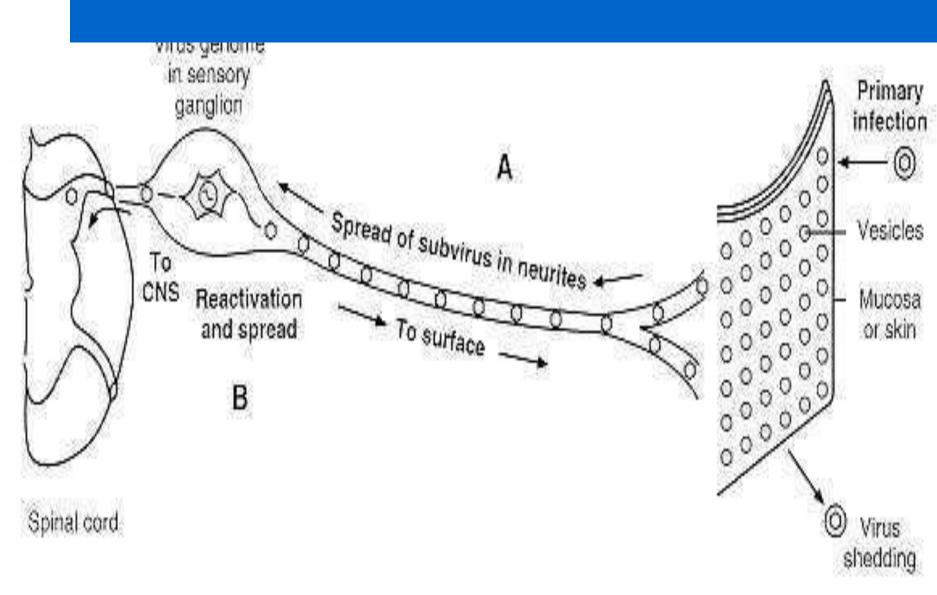




* After resolution , primary gingivostomatitis , virus migrate along periaxon sheath to trigeminal ganglion-LATENT/SEQUESTRATED FORM









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Primary herpetic gingivostomatitis

Usually seen in children

- Solution Straight Straight
- Most infections are subclinical and escape diagnosis
- Solution & By adulthood , approx 90% of population demonstrate HSV 1antibodies.





Affected children present with fever, localized lymphadenopathy, irritability and poor feeding.

- Vesicular eruptions may appear on skin,vermillion and oral mucus membrane.
- Intraorally lesions appear on any mucosal surface
- Ulcers often have yellowish exudate
- In chidrens drooling caused by ulcers may be misdiagnosed as teething





Primary symptomatic infections in adults has presentation similar to that in children

Vesicles may be limited to posterior pharynx and tonsils





















Figure 3-2 Primary herpetic gingivostomatitis with extensive involvement of the keratinized tissues of the tongue dorsum and nonkeratinized tissues of the ventral tongue and labial mucosa. *Source:* Courtesy of Dr. Nathaniel Treister, Boston, MA.



Figure 3-4 Clustered vesicles of recrudescent herpes labialis on vermilion.

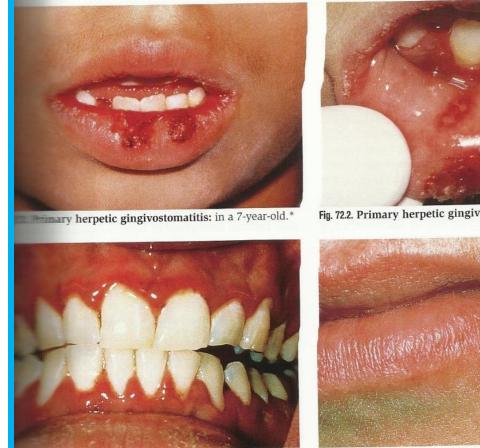


Figure 3-3 Primary herpetic gingivostomatitis with mild presentation: erythematous maxillary anterior gingiva with erythema and ulcer on upper labial mucosa and crusted lesion on lower lip.



Figure 3-5 Recrudescent intraoral herpes simplex virus infection in an immunocompetent patient with clusters of small coalescent ulcers on the keratinized palatal mucosa.





Primary herpetic gingivitis: a 27-year-old man.

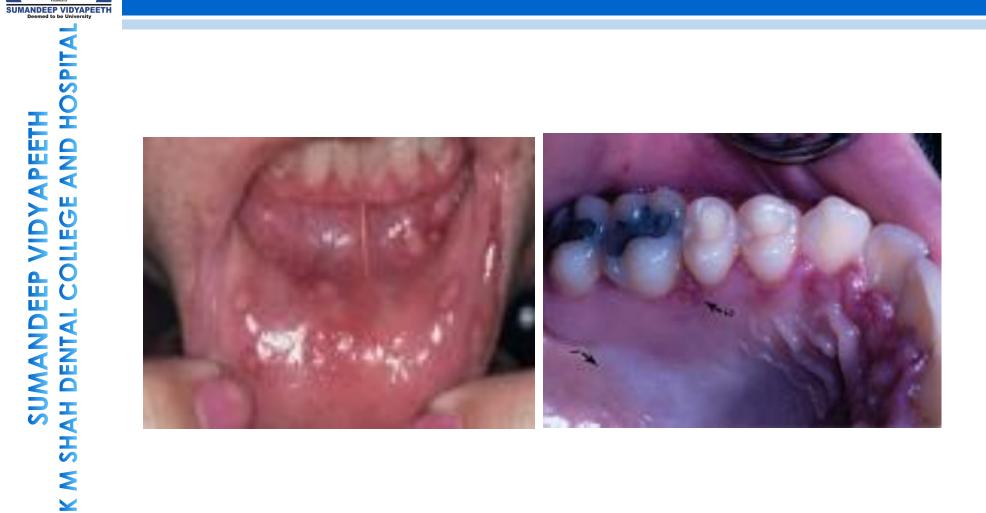
Fig. 72.2. Primary herpetic gingivostomatitis.*



Fig. 72.4. Recurrent herpes labialis: clustered vesicles.











Recurrent HSV Infection

HSV affects a wide range of cells, including epithelial and neural tissue.

Latent virus is reactivated by stimuli such as sunlight, fever, trauma, intercurrent immunosuppression, and infection

Virus is then transported along sensory axon.





Recurrent lesions typically affect mucocutaneous junction of lip,producing *"fever blister"or* "cold sore"







*** Recurrent lesions are less common intraorally.**

- Limited to keratinized mucosa i.e.(attached gingivae and hard palate)
- * Prodromal symptoms do not occur with intraoral infections
- Lesions are grouped and are small,round, punched-out ulcers with surrounding red halo rather than being distinct vesicles





Recurrent herpes simplex: multiple gingival ulcers.



Fig. 72.6. Recurrent herpes simplex: on hard palate.



Herpetic whitlow: caused by autoinoculation.

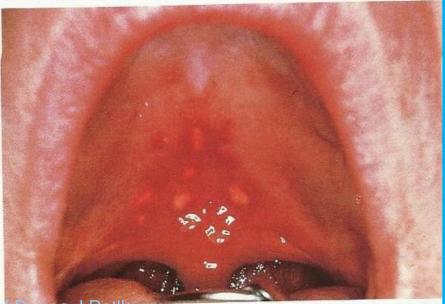


Fig. 72.8. Herpangina: multiple, soft palate ulcers and redness.





Figure 3-6 Recrudescent herpes simplex virus infection of the maxillary alveolar ridge mucosa in a patient with lymphoma.



Figure 3-7 Recrudescent herpes simplex virus infection of the lateral tongue and oral commissure in a patient with leukemia nostallogenic transplantation





Herpetic whitlow

Either a primary or secondary HSV infection involving fingers

- Occurs in dental practitioners
- In case of seronegative clinician, contact could result in vesiculoulcerative eruption on digit and signs and symptoms of primary disease
- Vesicles and pustules break and become ulcers
- Axillary and epitrochlear LAD Present
- Duration may be as long as 4-6 wk













HOSPITA

- Virologic tests
- **1. Isolation in tissue culture**

2.Observe cytopathic effects of cells inoculated with virus

3.Virally infected cells demonstrate multinucleated giant cells,syncitium,and ballooning degeneration of nuclei



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***** Cytologic smears

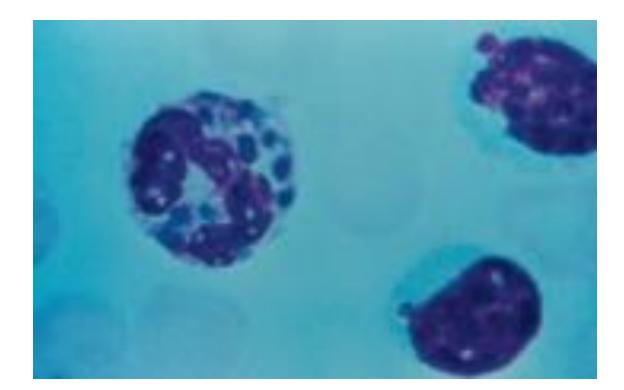
- **1.** A smear taken of epithelial cells at base of suspected lesion, to determine if epithelial cells show changes consistent with HSV
- 2. Most common stain –GIEMSA
- 3. With PAPANICOLAOU'S STAIN-Eosinophillic intranuclear viral inclusion bodies

(Lipschutz or Cowdry A)





Iultinucleated giant cells









- 1. Direct Fluorescent assay- specimen is incubated with Fluorescein isothiocyanate labeled HSV type specific monoclonal Ab
- *** Serologic tests**
- **1. Detects antibody formation in patients blood sample**
- 2. Useful in diagnosing primary HSV infection





reatment

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- Immunocompetent hosts
- **1. Supportive and palliative**
- 2. Topical acyclovir is not recommended
- 3. Studies have shown that topical penciclovir cream provides benefit in treatment of recurrent herpes labialis
- 4. Lip balm and sunscreen may block reactivating effects of ultraviolet rays





Table 3-2 Pain management and supportive care measures.

Pain Management

2% viscous lidocaine (swish 5 mL for 3–5 min. and spit out 4–5 times/d)

Liquid diphenhydramine (swish 5 mL for 3–5 min and spit out 5 4–5 times/d)

Combination of viscous lidocaine, diphenhydramine, and a covering agent (such as KaopectateTM or MaaloxTM) in 1:1:1 ratio (swish 5 mL for 3–5 min and spit out)

0.1% diclonine hydrochloride

Benzydamine

Systemic analgesia

Supportive care

Hydration

Ice chips or popsicles

Soft bland diet

Antipyretics such as ibuprofen as needed (avoid aspirin products)*





Immunocompromised hosts

- Recurrent lesions are more severe and associated with systemic manifestation
- More widespread and high rate of dissemination





- Although RHL is self-limiting, the use of topical antiviral medications reduces shedding, infectivity, pain, and the size and duration of lesions.
- * Topical antiviral medications such as 5% acyclovir cream,1% penciclovir cream,36,37 and 10% docosanol cream are efficacious if applied five to eight times a day (every two hours) at the first prodrome or sign of a lesion.







valacyclovir (2 g 12 hours apart for one day) or famciclovir (1500 mg single dose)

Are both effective in aborting early lesions of RHL





- For patients who have more than 10-12 attacks/year,prophylactic therapy with acyclovir (400mg twice a day).
- Prophylaxis may also be indicated for patients with recurrent erythema multiforme.
- HSV infected neonates also benefit. (given orally for the first six months of life)
- For patients receiving chemotherapy prophylactic treatment with oral or iv acyclovir is recommended(given from 3-5 weeks after initiation of chemotherapy)





PICO Question

Ρ	Patients with Herpes Simplex Labialis
I	Acyclovir Cream
С	Pencyclovir Cream
Ο	Regression of the lesion

Evidence

Spotswood L. Spruance,^{1,*} Robert Nett,² Thomas Marbury,³ Ray Wolff,⁴ James Johnson,⁵ Theodore Spaulding,⁵ and The Acyclovir Cream Study Group

Acyclovir Cream for Treatment of Herpes Simplex Labialis: Results of Two

Title

SUMANDEEP VIDYAPEETH

Randomized, Double-Blind, Vehicle-Controlled, Multicenter Clinical Trials

To examine more comprehensively the efficacy and safety of acyclovir.

Interpretation

Aim

Authors

The observation with both ACV cream and penciclovir cream that efficacy occurs independently of lesion stage at the start of treatment is paradoxical. It is intuitive that interruption of virus replication prior to the development of major pathological processes in the epidermis would be more likely to preserve the skin from virus-induced damage and abbreviate the lesion course than application of treatment at a later stage.

Antimicrob Agents Chemother, Jul 2002; 46(7): 2238–2243.





(aricella Zoster virus(HHV3)

Primary varicella zoster infection(chicken pox)

1. Benign disease of children younger than 10 years.

2.Virus spreads through air dropletsor direct contact with active lesions.

3.Incubation period is 10 to 21 days ,with an average of 15 days.





Clinical features



- Characteristic, intensely pruritic exanthem
- * Rash begins on the face and trunk,followed by involvement of extremities
- Stages- Erythema,vesicle,pustule,and hardened crust.
- Early vesicular stage is most classic presentation.



















12. Herpes zoster (shingles): mandibular eruption.*



Fig. 73.2. Varicella (chickenpox): vesicle near midline.















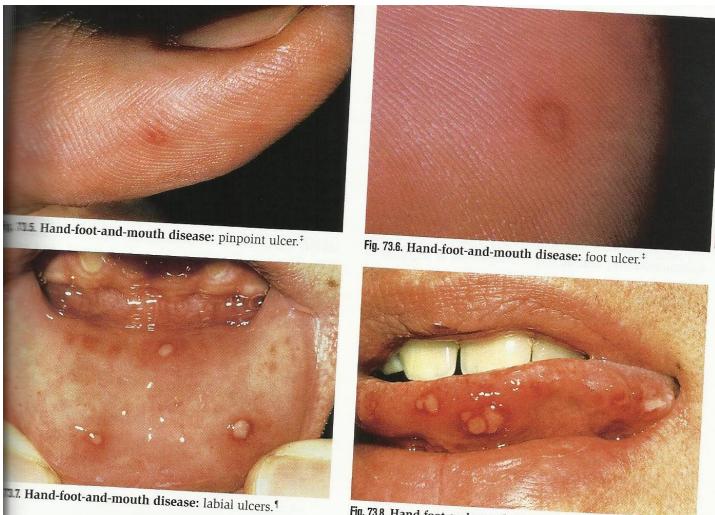


Fig. 73.8. Hand-foot-and-mouth disease: coalescing and







- Affected individuals are contagious from 2 days before the exanthem until all the lesions crust, 4 days after the arrival of lesion
- Oral lesions are fairly common and may precede skin lesions
- Palate and buccal mucosa are most frequently involved sites.





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Complications

- Encephalitis
- Reye's syndrome
- Congenital or neonatal chicken pox
- Infection in immunocompromised patients can be most severe
- Cutaneous involvement is extensive





Histopathologic features

Cytologic alterations are identical to those for HSV

* Virus causes acantholysis, formation of numerous free floating Tzanck cells





Diagnosis

Diagnosis is made clinically, and diagnostic tests are not needed

Presence of typical exanthem





VIRUS	COMPETENT PATIENTS	COMPROMISED PATIENTS
VZV Primary varicella (Chicken pox)	Supportive	I.V. Acyclovir 10mg/kg every 8 hours for 7-10 days(adults,pregnant women)



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lerpes Zoster(Shingles)

- Occurs after reactivation of the virus, with involvement of distribution of affected sensory nerve.
- Prevalence of life attacks increases with age.
- *** PREDISPOSING FACTORS**
- **1. Immunosuppression**
- 2. Cytotoxic drugs
- 3. Radiation
- 4. Malignancies
- 5. Old age





Clinical features

- Begins with pain in the area of epithelium innervated by affected sensory nerve.
- Typically, one dermatome is affected, but involvement of two or more can occur.
- Prodromal pain may be accompanied by is present 4 to 4 days before devlopment of oral lesions
- Pain may masquerade differently depending on which dermatome is affected.





Lesions tend to follow the path of affected nerve and terminate at midline

Skin shows cluster of vesicles on erythematous base

Within 3 to 4 days ,vesicles become pustular and ulcerate,with crusting after 7 to 10 days.

Example the second s





There may be recurrence in absence of vesiculation of the skin or mucosa.

- "zoster sine herpete"
- * Severe pain of abrupt onset and hyperesthesia over a specific dermatome.
- Pain lasting longer than 1 month after an episode of zoster is called postherpetic neuralgia, especially those older than 60 years of age.





Most of these neuralgias resolve within one year

- Pain is severe , lancinating type
- Ramsay Hunt syndrome is the combination of cutaneous lesions of external auditory canal combined with involvement of ipsilateral facial and auditory nerves.

Ocular involvement and involvement of tip of nose.





Oral lesions may be present on movable or bound mucosa.

- Lesions extend to midline and are present with involvement of skin overlying affected quadrant.
- Lesions present as 1- to 4-mm white opaque vesicles, rupture to form vesicles.
- Devitalization of teeth in affected area.















Figure 3-10 Palatal lesions of herpes zoster involving the second division of the trigeminal nerve; note unilateral distribution. *Source:* Courtesy of Dr. Stephen Challacombe.



Figure 3-9 Facial lesions of herpes zoster involving the third division of the trigeminal nerve.



- Postherpetic neuralgia affects up to 20% of patients over age 65 and up to 30–50% of patients over age 80;
- * affected individuals have debilitating pain, usually of a sharp, stabbing, burning, or gnawing nature lasting more than 1 month.
- Some unfortunate patients experience pain for years.
- Predisposing factors
- * include older age (most important) prodromal pain, and more severe clinical disease during the acute rash phase.





Viral isolation

SUMANDEEP VIDYAPEETH SHAH DENTAL COLLEGE AND HOSPITA Monoclonal antibody detection

Siopsy specimen cannot distinguish between VZV and HSV

PCR is therefore confirmatory.



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Coxsackievirus Infections

Coxsackie-viruses cause three types of infections

- 1. Herpangina
- 2. Hand, foot-and-mouth disease
- 3. Acute lymphonodular pharyngitis.





HERPANGINA

Coxsackievirus A4

Affects young children.

Clinical Manifestations:

Begins with generalized symptoms of fever, chills, and anorexia.

Lesions starts as punctate macules, which quickly evolve into papules and vesicles involving the posterior pharynx, tonsils, faucial pillars, and soft palate.

ulcers.

Within 24 - 48 hrs, the vesicles rupture forming small



Treatment



Supportive treatment - Proper hydration and topical anesthesia when eating or swallowing is difficult.





Herpangina may be clinically distinguished from primary HSV infection by several criteria:

- Herpangina occurs in epidemics; HSV infections do not.
- Herpangina tends to be milder than HSV infection.
- Lesions of herpangina occur on the pharynx and posterior portions of the oral mucosa, whereas HSV primarily affects the anterior portion of the mouth.
- Herpangina does not cause a generalized acute gingivitis like that associated with primary HSV infection.
- Lesions of herpangina tend to be smaller than those of HSV.



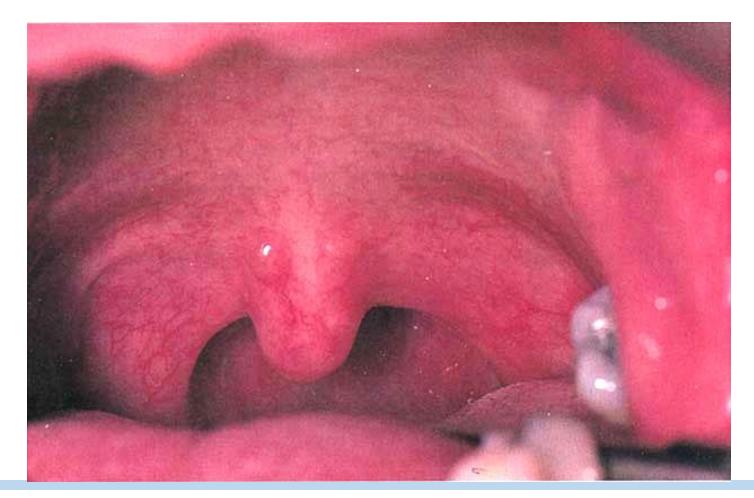
SHAH DENTAL COLLEGE AND HOSPITA **ACUTE LYMPHONODULAR** PHARYNGITIS SUMANDEEP VIDYAPEETH and HAND-FOOT-AND-MOUTH DISEASE



5



Acute lymphonodular pharyngitis





VIDYAPEETI

SUMANDEEP

HAND-FOOT-AND-MOUTH DISEASE

HOSPUTA low-grade fever, oral vesicles and ulcers, and nonpruritic macules, papules on extensor COLLEGE surfaces of the hands and feet.

hard palate, tongue, and buccal mucosa.

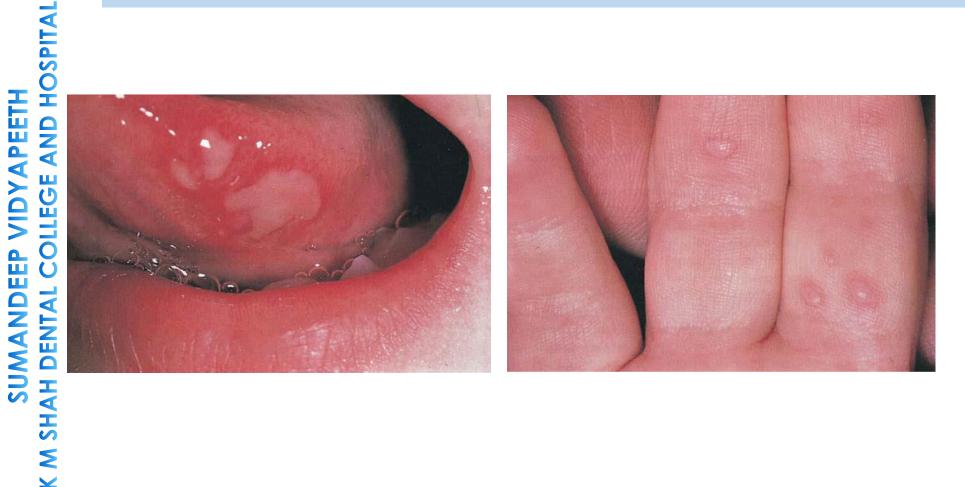
Treatment is supportive.

Coxsackie-virus A16.





Hand, foot and mouth disease



















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CCES

* 1. Herpetic whitlow is seen in

- * A. scalp
- *** B.** fingers
- ***** C. lips
- *** D.** tongue
- *** 2. Shingles is the other name for**
 - *** A. RHL**
 - *** B. Genital Herpes**
 - ***** C. herpetic whitlow
 - *** D.** herpes zoster





- *** 3. Herpangina affects**
 - ***** A. genitals
 - ✤ B. anterior oral cavity
 - C. posterior oral cavity
 - D. eyes
- 4. Ramsay Hunt syndrome is
 - * A. HSV of the geniculate ganglion
 - ✤ B. HZ of the geniculate ganglion
 - C. HZ of the trigeminal ganglion
 - ***** D. HSV of the trigeminal ganglion
- *** 5. HSV infection is characterized by**
 - * A. Vesicles
 - *** B. Erosion**
 - C. Petechiae
 - *** D.** ecchymosis





