Orofacial Infections In Paediatric Dentistry

DENT 5312
Paediatric Dentistry Module

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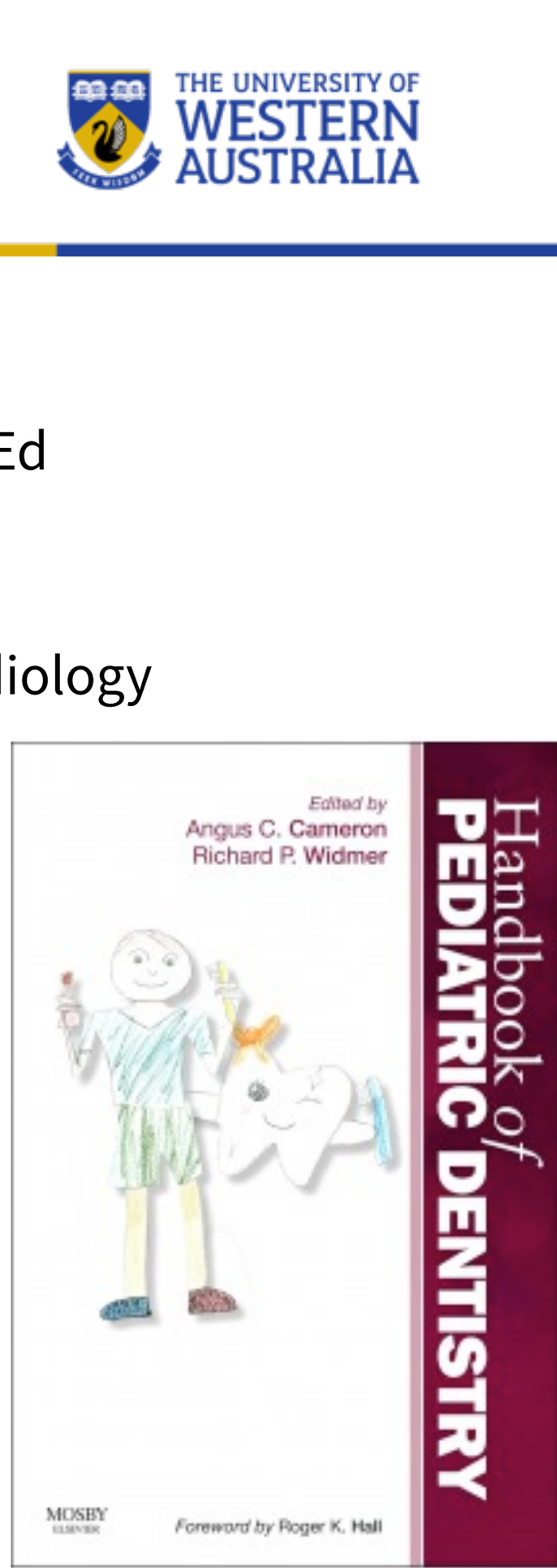
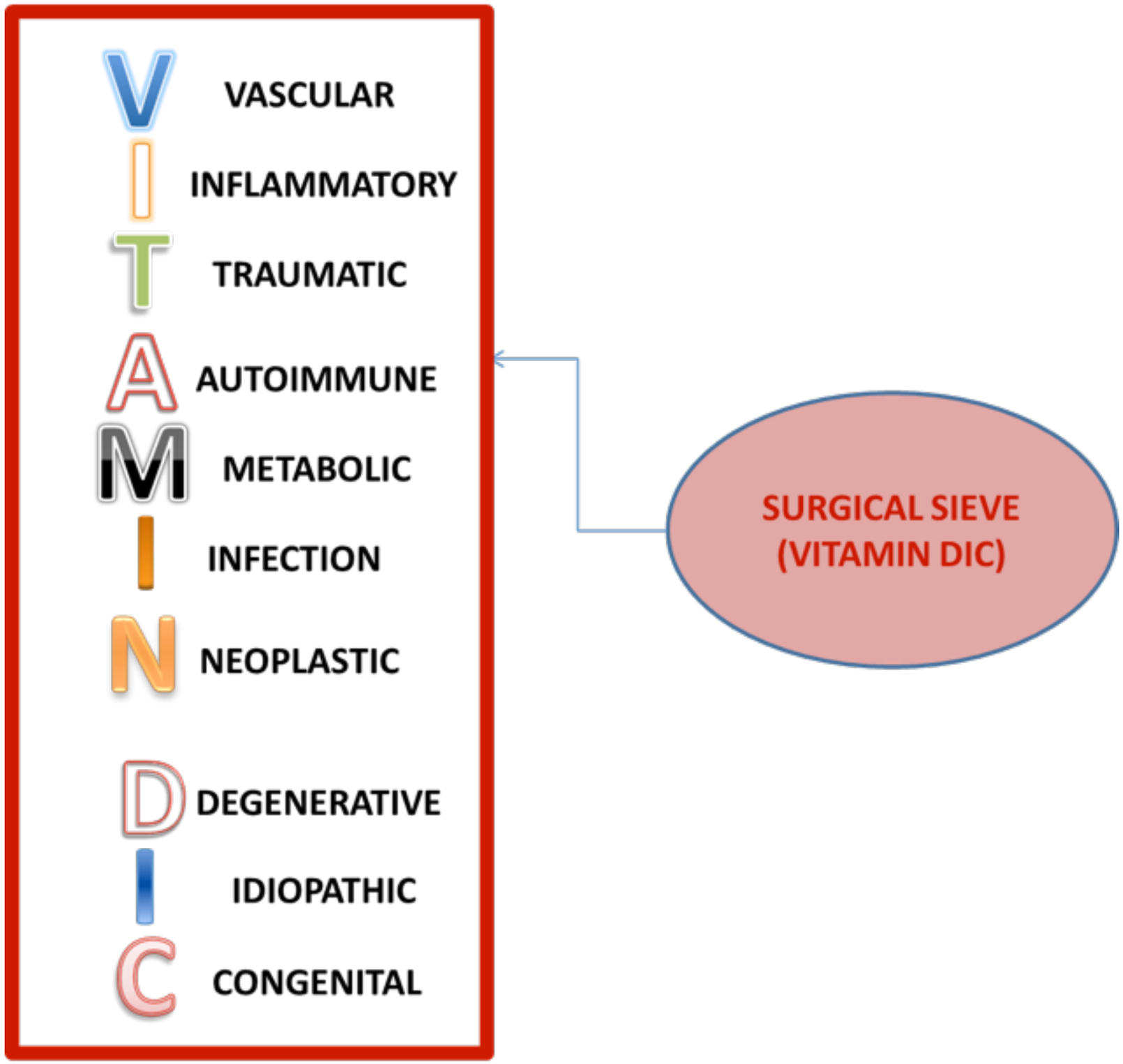
Orofacial Infections in Paediatric Dentistry

- MUST READ
- Handbook of Pediatric Dentistry, Cameron and Widmer 4th Ed
  - Ebook available through UWA Onesearch
- Chapter 10: Paediatric oral medicine, oral pathology and radiology



Diagnostic Sieve



Orofacial Infections

- **Bacterial**
- **Viral**
- **Fungal**

Orofacial Infections

Differential Diagnosis

Bacterial infections

- Odontogenic
  - dental caries
  - periodontal disease
- Scarlet fever
- Tuberculosis
- Atypical mycobacterial infection
- Actinomycosis
- Syphilis
- Impetigo
- Osteomyelitis

Viral infections

- Primary herpetic gingiva-stomatitis
- Herpes labialis
- Herpangina
- Hand, foot and mouth disease
- Infectious mononucleosis
- Varicella

Fungal infections

- Candidosis

Orofacial Infections

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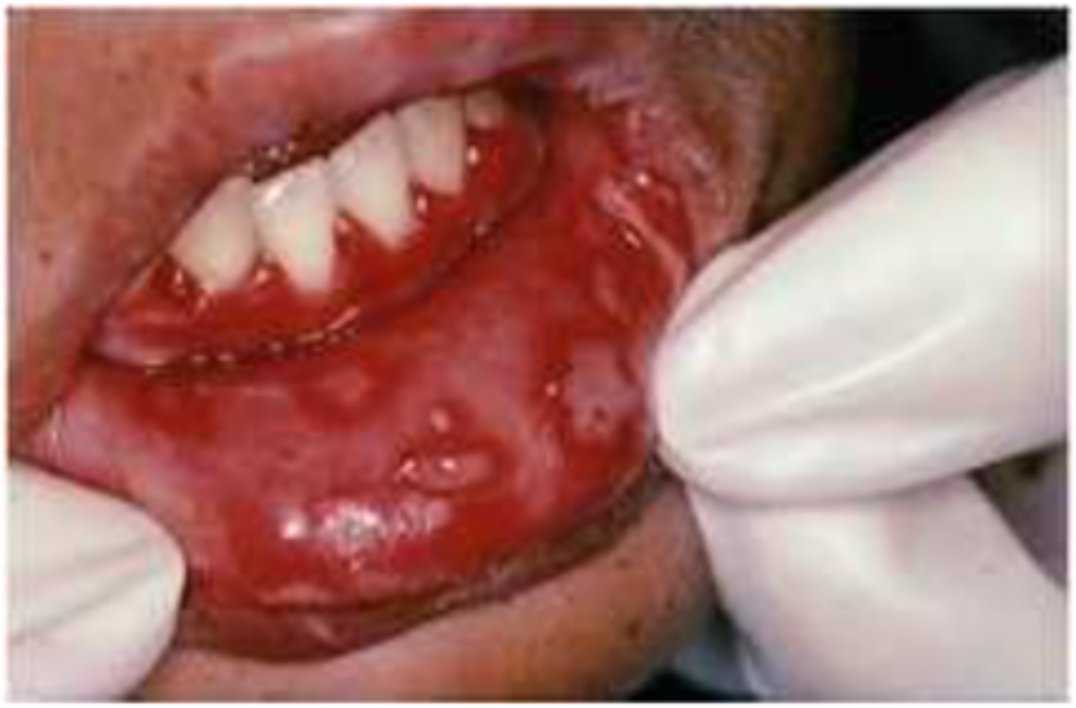
- Varicella

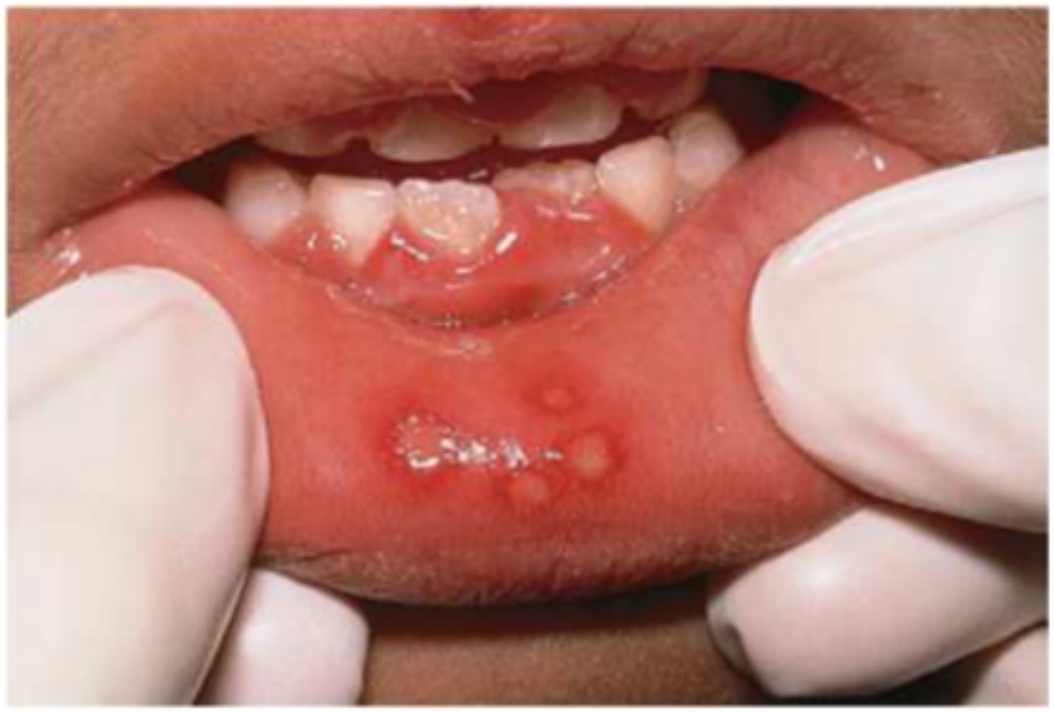
Fungal infections

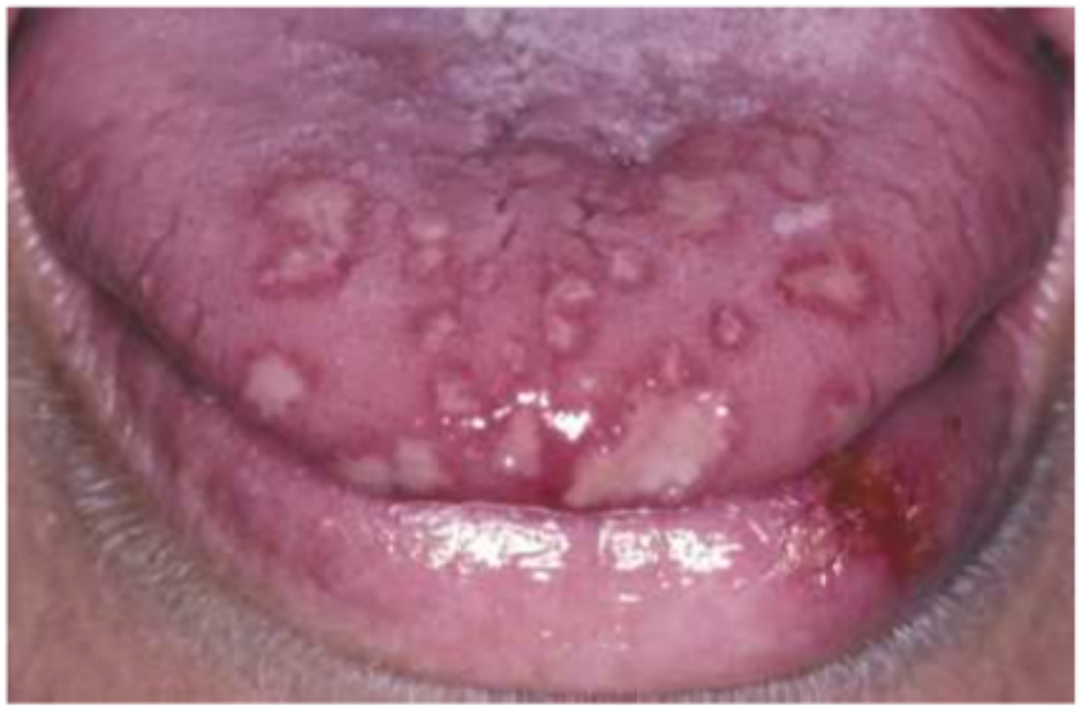
- Candidosis

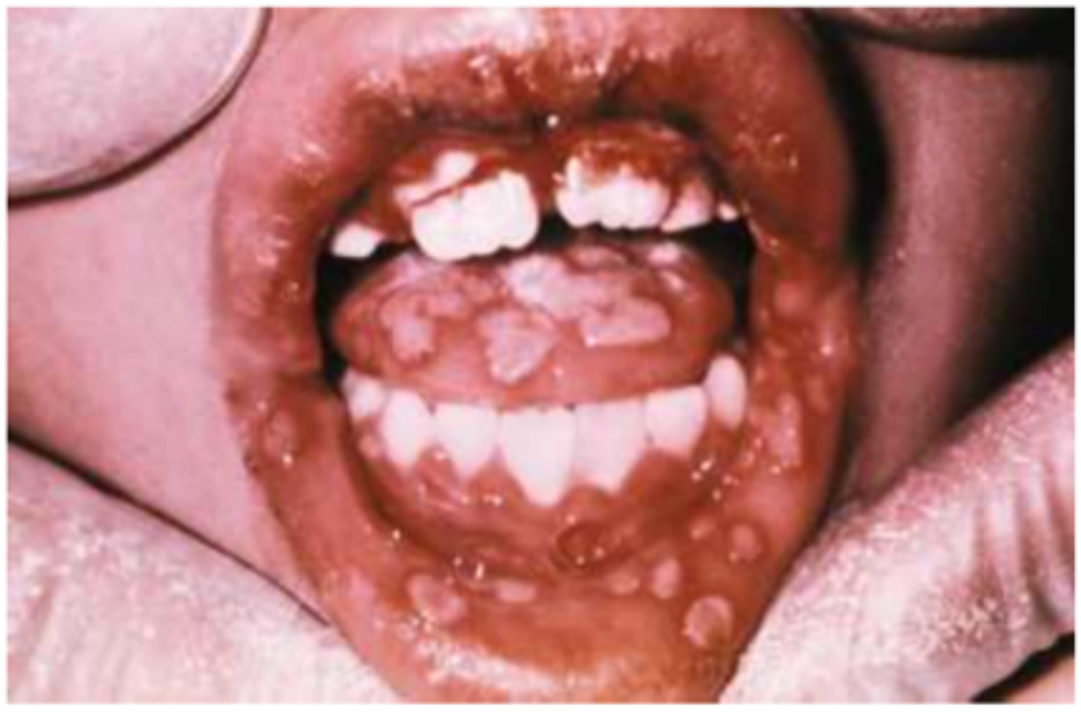
Viral Infections

Virus	Disease
Herpes Simplex Virus Type 1 (HSV)	• Primary herpetic gingivostomatitis • Herpes labilis
Epstein Barr virus (EBV)	Infectious mononucleosis
Coxsackie A, B, enterovirus 71	Hand foot and mouth disease
Coxsackie A	Herpangina
Varicella Zoster Virus (VZV)	Chickenpox









Primary Herpetic Gingivostomatitis

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Etiology

- Herpes simplex type 1 virus
- Herpes simplex type 2 virus (occasional cases)
  - due to genital herpes
  - cases of sexual abuse

Age and gender

- Primary infection usually occurs after 6 months of age
- The peak incidence is between 12 and 18 months of age
- No gender predilection

Primary Herpetic Gingivostomatitis

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Clinical presentation (Amir et al. 1997, Amir et al. 1999)

General

- Febrile illness with a raised temperature of 100-102 °F (37.8-38.9 °C)
- Headaches, malaise, irritability
- Cervical lymphadenopathy

Oral

- Oral pain, mild dysphagia
- Stomatitis
- *Intraepithelial fluid-filled vesicles appear (*characteristic feature)
- Site of vesicles: tongue, lips, buccal, and palatal mucosa
- *Solitary ulcers:* usually small (3 mm) & painful with an erythematous margin
- *Larger ulcers:* irregular margins, result from coalescence of individual lesions

Primary Herpetic Gingivostomatitis

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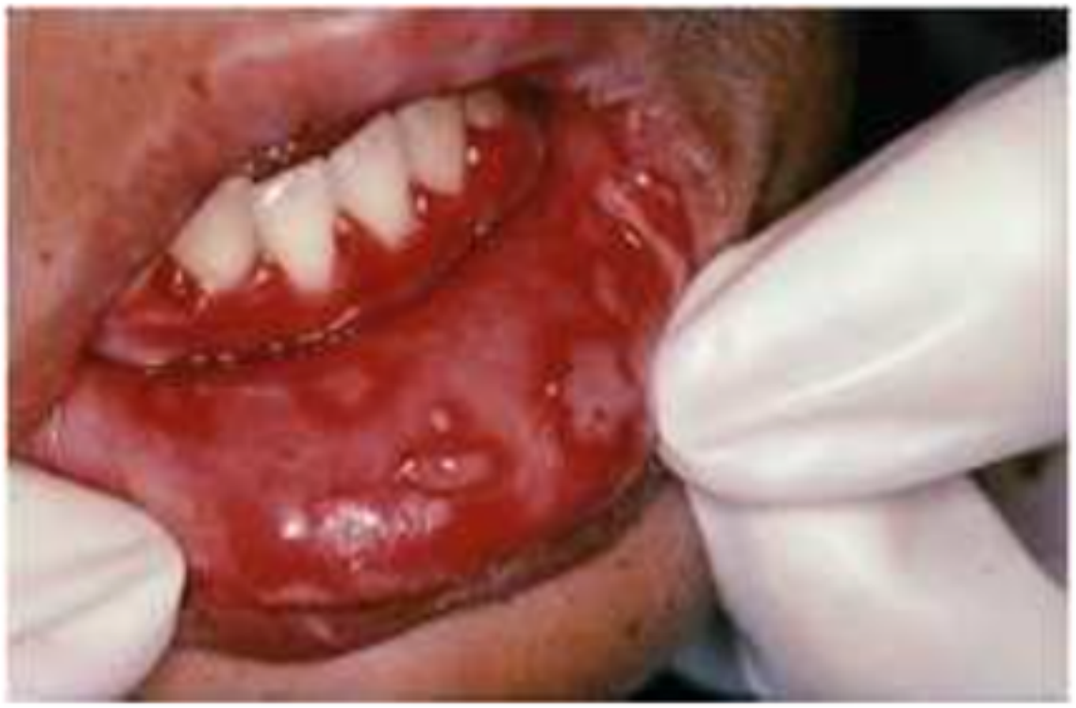

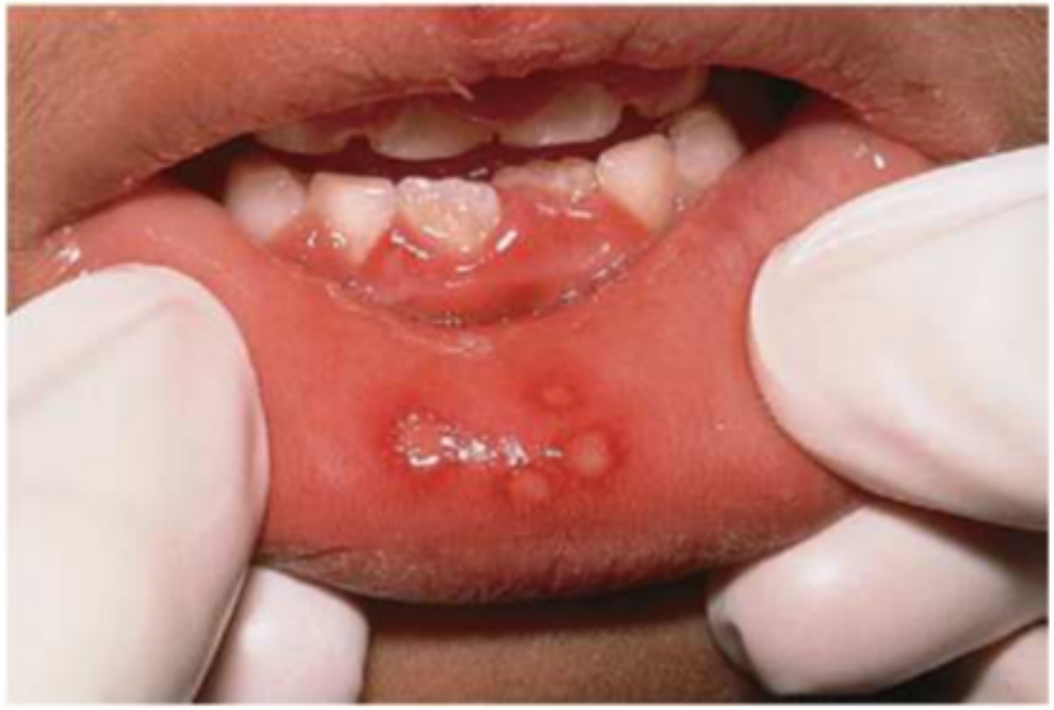

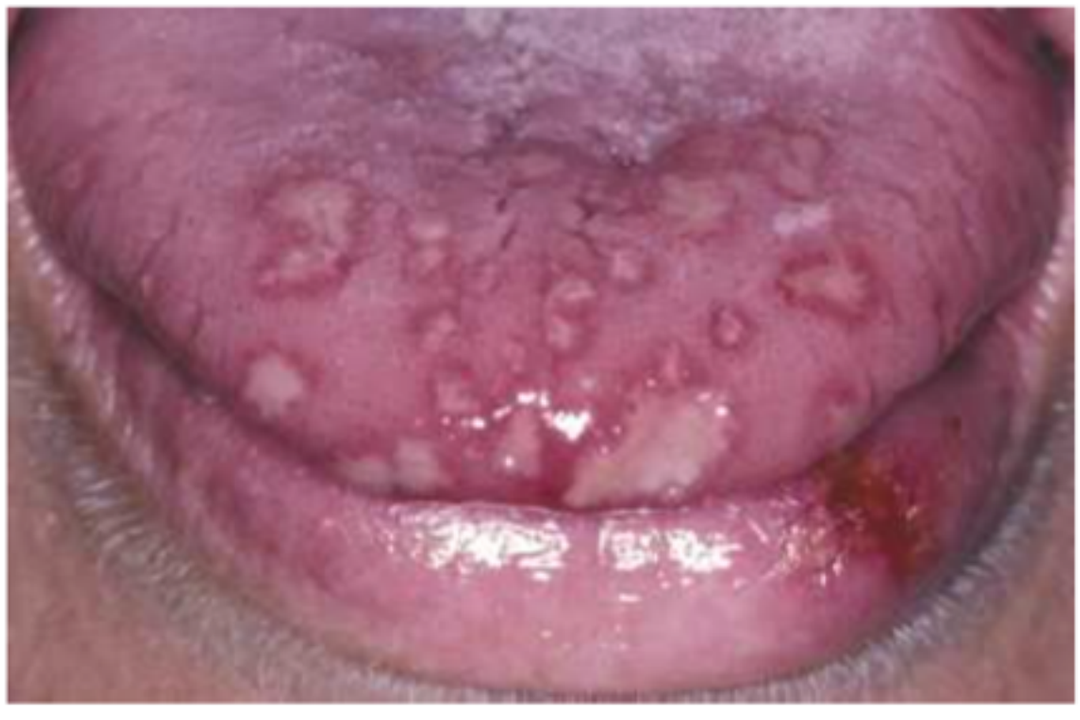

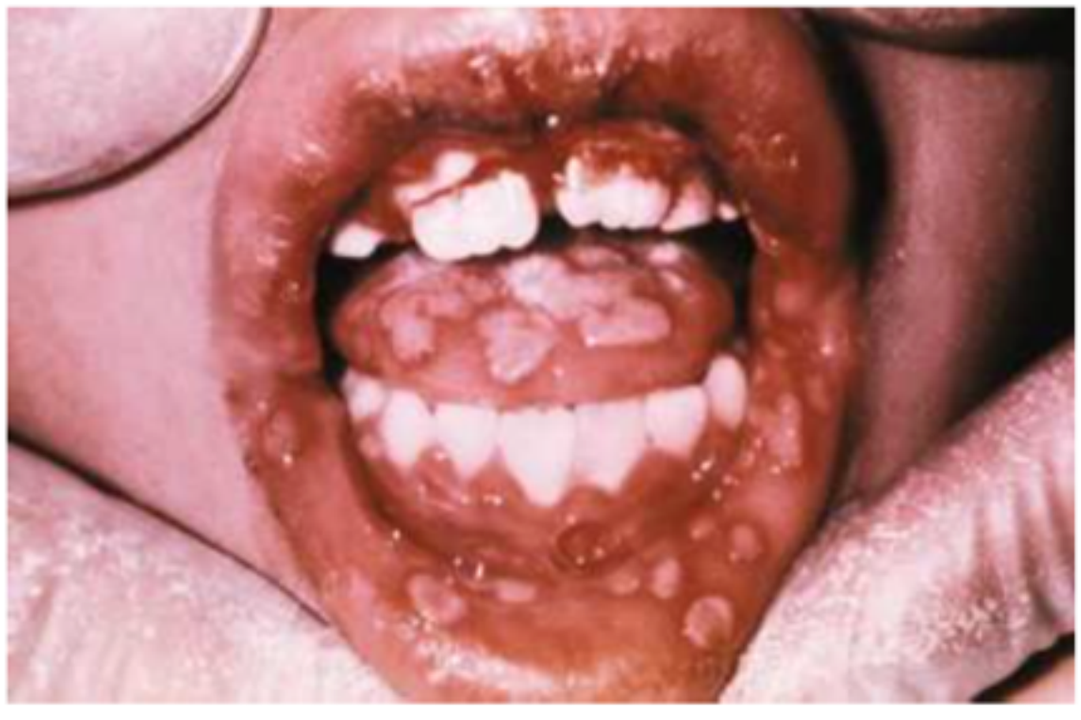

Primary Herpetic Gingivostomatitis

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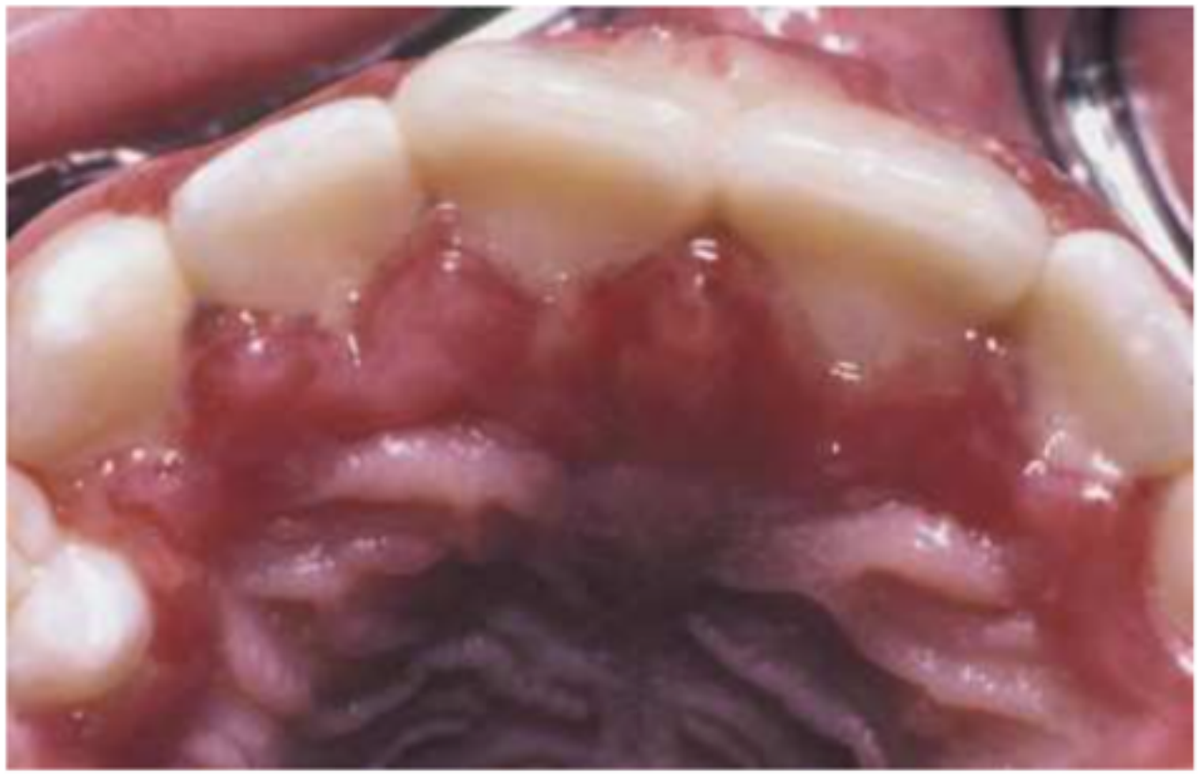
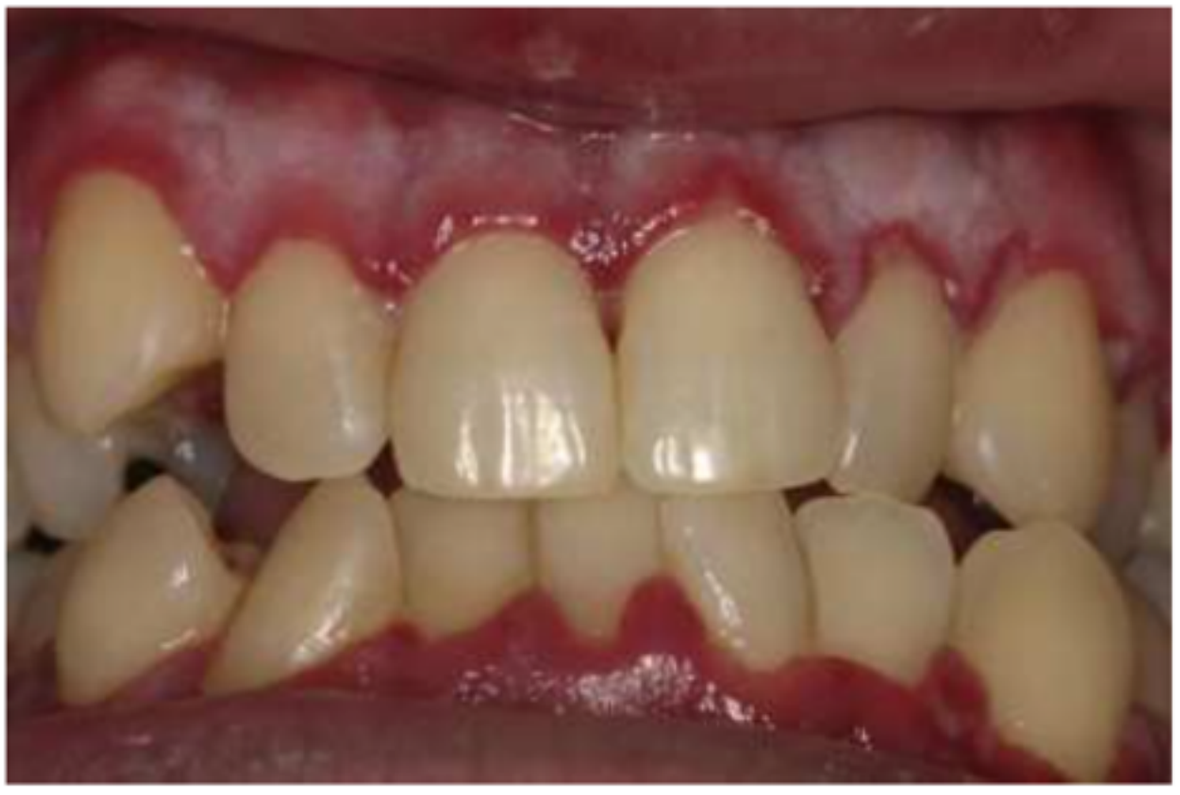


Painful, enlarged & erythematous palatal gingiva



Painful, enlarged & erythematous facial gingiva

Note erosions of the free gingival margin

Primary Herpetic Gingivostomatitis

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- **Incubation time**

  3–5 days (with a prodromal 48-h history of irritability, pyrexia & malaise)

- **Transmission**
  - Direct contact with the lesions
  - Contact with infected oral secretions (droplet infection)

- **Course of disease**
  - Disease is self-limiting
  - Ulcers heal spontaneously without scarring within 10-14 days

(Amir et al. 1997; Pinto and Hong, 2013)

Primary Herpetic Gingivostomatitis

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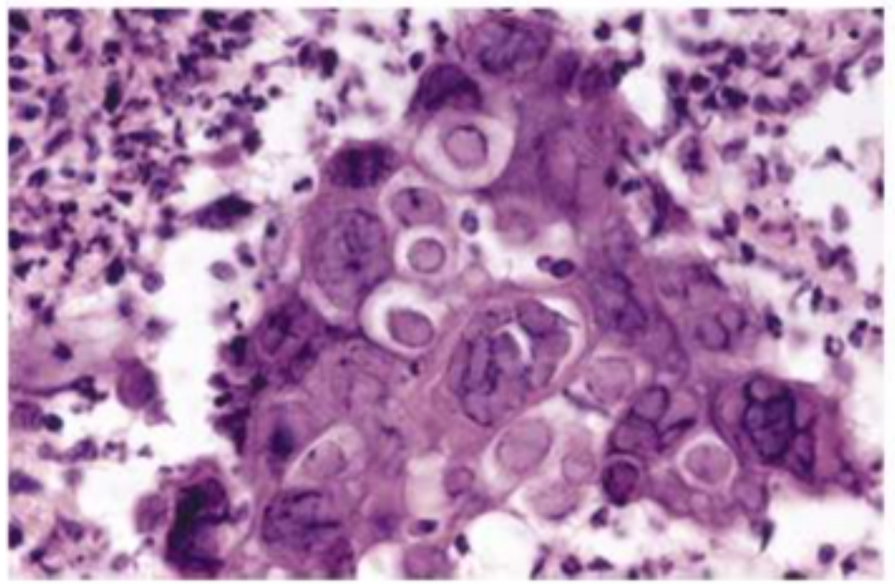
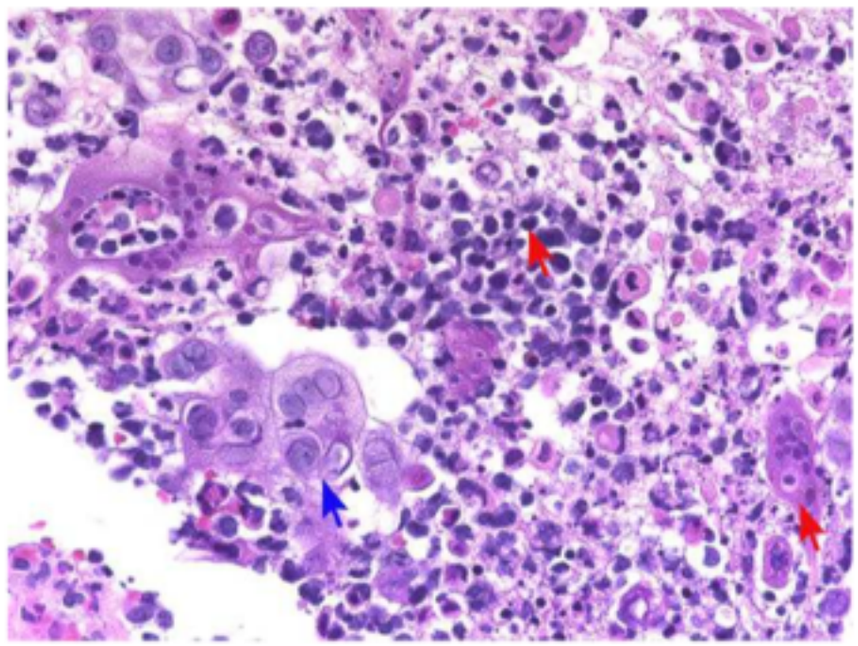
Histology

Tzanck cells: multinucleated giant cells with jig saw nucleus



Diagnosis

- History, clinical features & age group of the affected children
- Exfoliative cytology: presence of multinucleated giant cells and viral inclusion bodies can be used for rapid diagnosis
- Viral antigen
- Viral culture
- Viral antibody detection in blood samples



Primary Herpetic Gingivostomatitis

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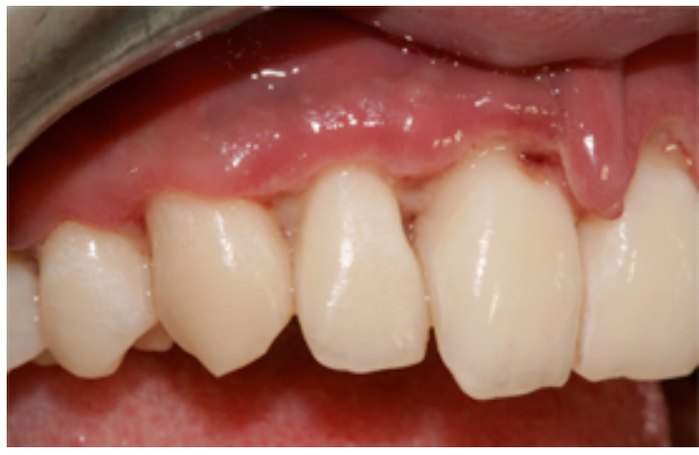
Differential Diagnosis

- Necrotizing ulcerative gingivitis
- Erythema multiforme
- Herpangina



Complications

Rare but severe complications of the infection

- Aseptic meningitis
- Encephalitis

Primary Herpetic Gingivostomatitis

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Management

- ***Symptomatic care***
  - Encourage oral fluids
  - Bed rest and a soft diet
- ***Analgesics***
  - Paracetamol 15 mg/kg, 4-6 hourly
  - Topical therapies for pain control (e.g. 2% viscous lidocaine) are not recommended due to concern over systemic overdose & the inability of the young child to spit
  - Topical benzocaine gel should be avoided in children < 6 years to prevent methaemoglobinaemia

(So and Farrington, 2008, Chung et al. 2010; Pinto and Hong 2013)

Primary Herpetic Gingivostomatitis

Management

Mouthwashes

- In young children with severe ulceration: 0.2% chlorhexidine can be swabbed over the affected areas with cotton wool swabs

- *For older children* – chlorhexidine gluconate, 0.2%, 10 mL 4-hourly

- *In children over 12 years of age:* tetracycline or minocycline mouthwashes may be beneficial

- A mouthwash containing benzydamine hydrochloride 0.15% & chlorhexidine 0.12% (Difflam C™) may offer some advantages over chlorhexidine alone

(So and Farrington, 2008, Chung et al. 2010; Pinto and Hong 2013)

Primary Herpetic Gingivostomatitis

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Management

Antiviral chemotherapy

- Oral and intravenous acyclovir is approved for use in children for management of primary herpetic gingivostomatitis (Arduino and Porter 2006)

- Evidence for its use via the oral route in children < two years of age is limited

- Some evidence that the administration of aciclovir in the first 72 - 96 h of the infection may be beneficial (Pinto and Hong 2013)

- Dose: PO: 20mg/kg body weight 5 hourly TDS, or IV 10mg/kg body weight

Recurrent Herpes Simplex infections
(Secondary herpes, recrudescent herpes)

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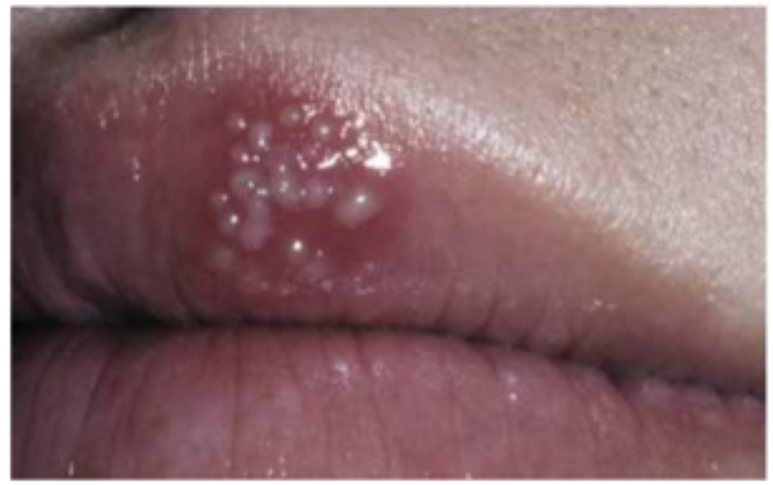
Herpes Labialis

Alternative names: cold sore, fever blister

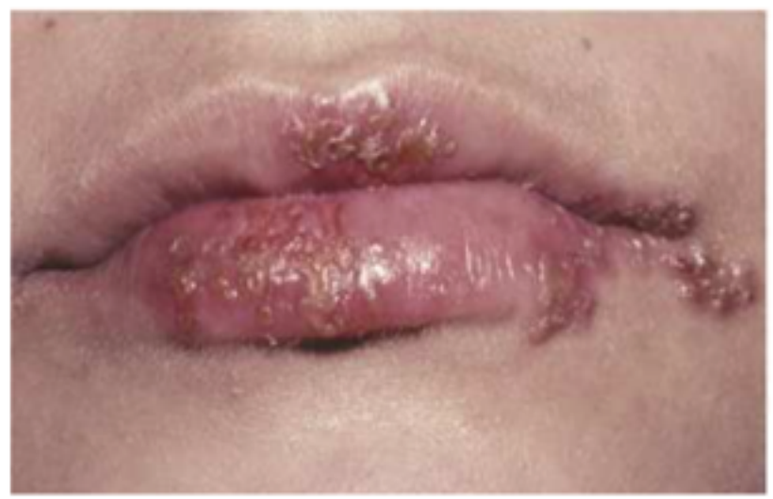


Etiology: Herpes simplex type 1 virus

Site: Vermilion border and adjacent skin of the lips

Triggering factor

- ultraviolet light
- trauma



Herpes Labialis

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Prodromal signs and symptoms

- Symptoms are most severe in the first 8 hours
- Pain, burning, itching, tingling, localized warmth
- Erythema of the involved epithelium arise 6-24 hrs before the lesions develop
- Multiple small, erythematous papules develop
- Form clusters of fluid-filled vesicles
- Vesicles rupture and crust within 2 days
-

Course of disease

- Self limiting, Healing usually occurs within 7 to 10 days

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Infectious mononucleosis

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- **Alternative terminology:** Mono; Glandular Fever; Kissing Disease
- **Etiology:** Epstein-Barr virus (EBV or HHV-4)
- **Transmission:** The infection usually occurs by intimate contact
  - Main route of transmission is by blood or saliva
  - Intrafamilial spread is common, and once a person is exposed, EBV remains in the host for life
  - Children: infected through contaminated saliva on fingers, toys, or other objects
  - Adults: contract the virus through direct salivary transfer, such as shared straws or kissing
- **Age and Gender:** children ages 1 to 5 - not all children develop symptoms

Infectious mononucleosis

- **Clinical presentation:** It varies by age
  - Most infected children are typically asymptomatic
  - Young adults are at greatest risk for symptomatic disease

Symptoms includes

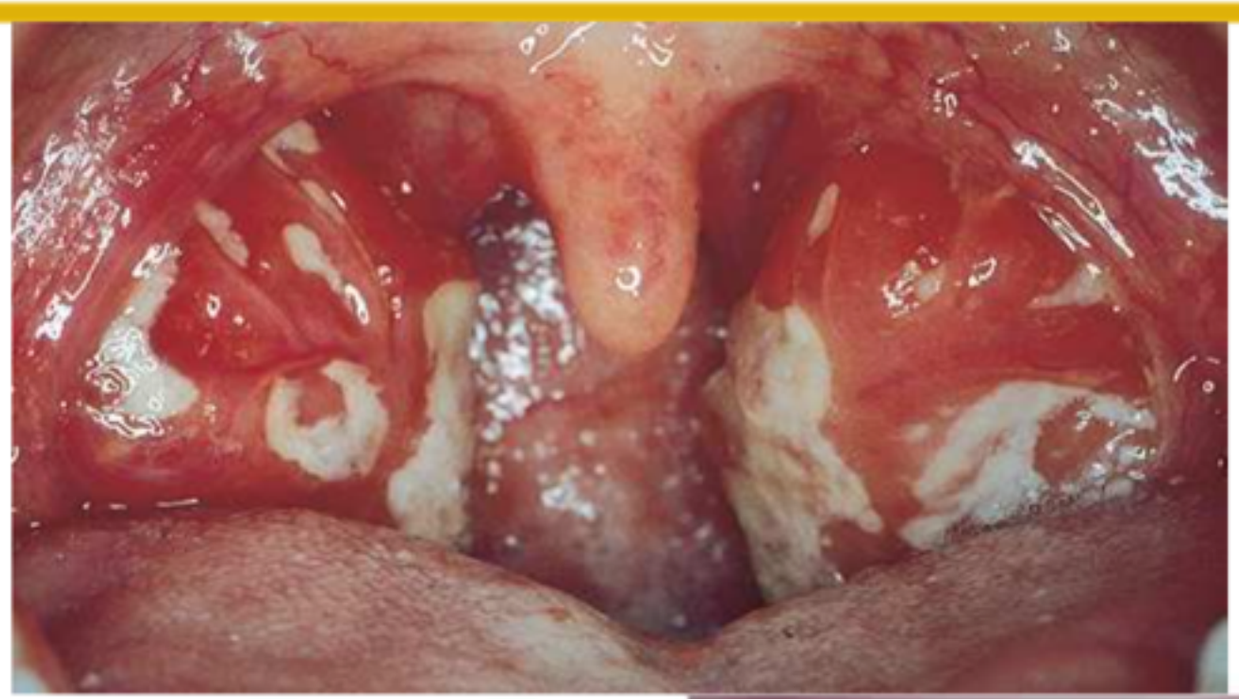
- Low-grade fever
- Hepatosplenomegaly
- Rhinitis or cough
- Malaise, headache
- Cervical lymphadenopathy and tenderness

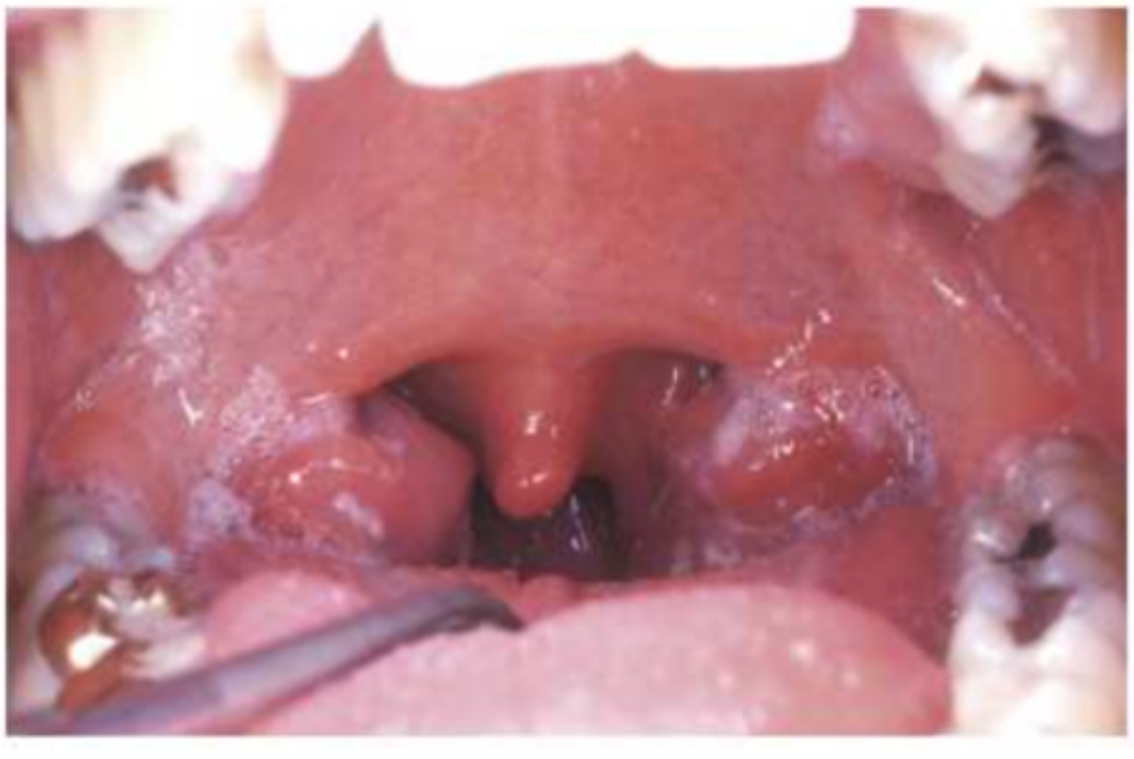
Oral lesions: oral ulcers, palatal petechiae and gingival ulcerations (necrotizing ulcerative gingivitis), tonsillitis with or without pharyngitis

Course of disease: The disease is self-limiting (resolve in one to two weeks)

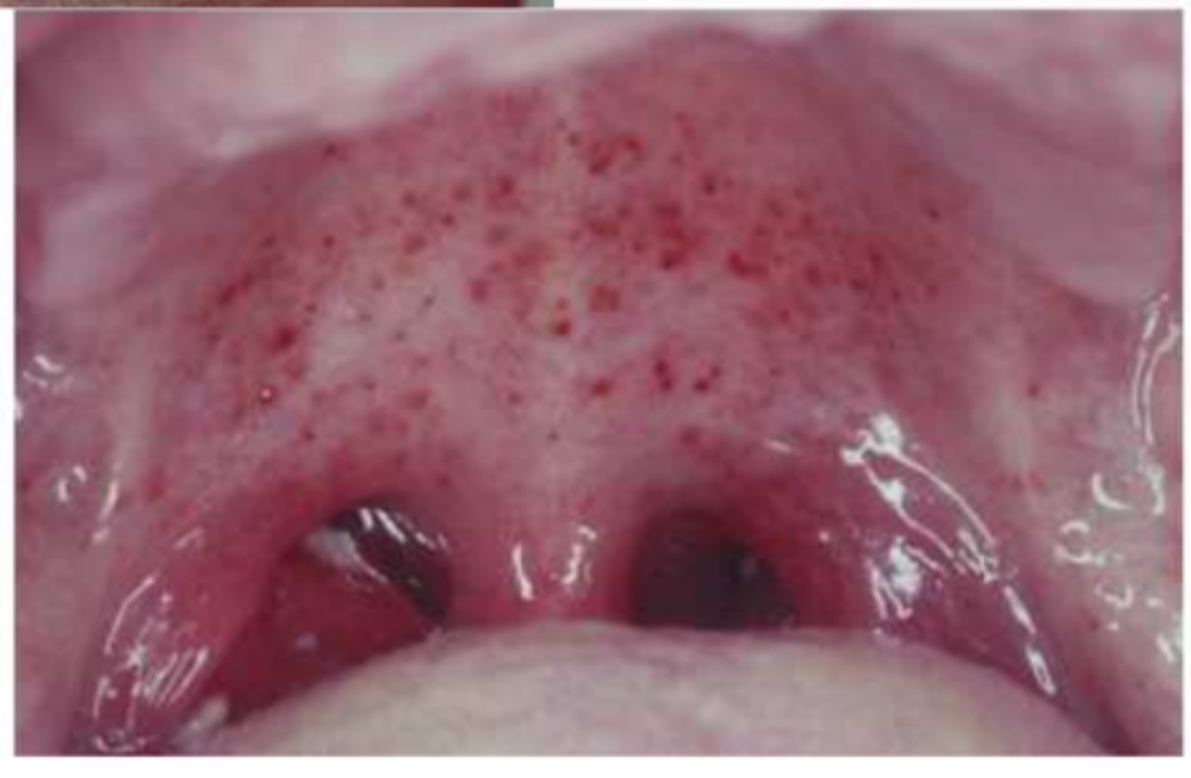
(Pinto and Hong 2013)

Infectious mononucleosis





Hyperplastic pharyngeal tonsils with yellowish crypt exudates

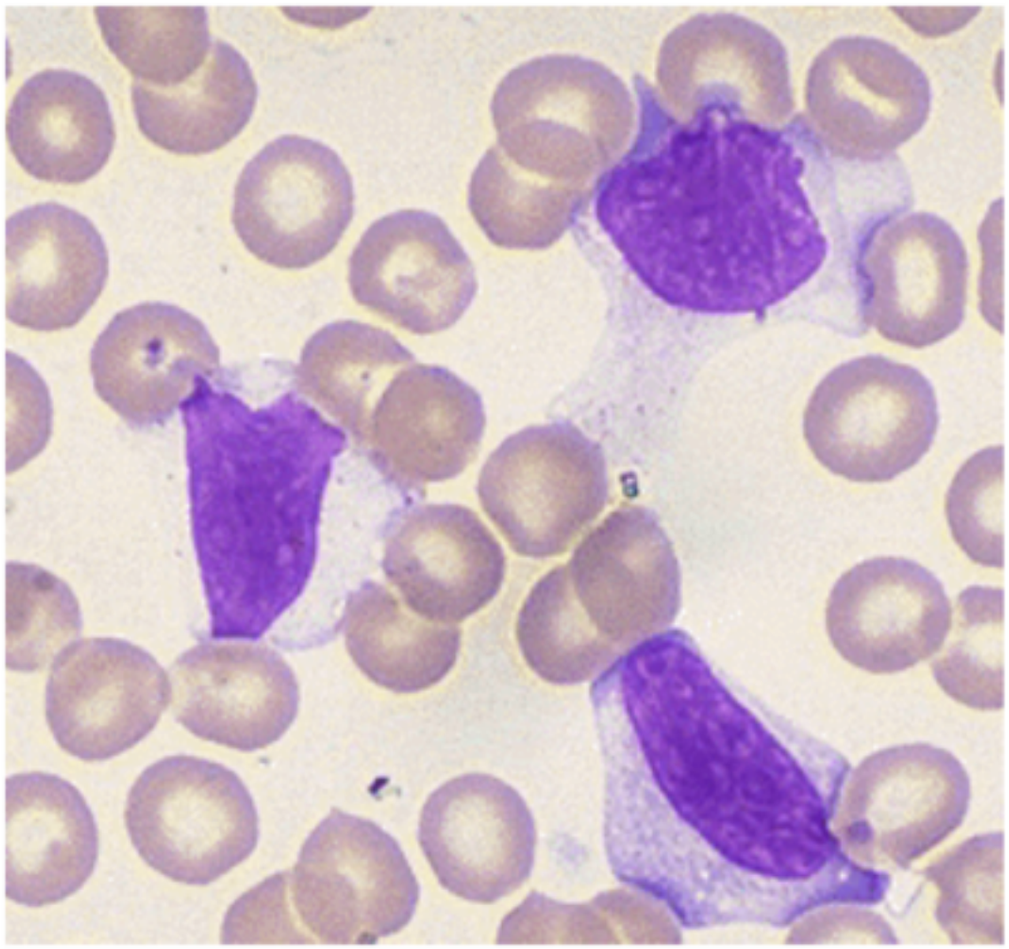


Numerous petechiae of the soft palate

Infectious mononucleosis

Histopathological features

Downey cells – atypical lymphocytes diagnostic feature



Differential Diagnosis

- Trauma
- Reactive gingival lesions
- Hematologic disorders
  - thrombocytopenia
  - platelet disorders
  - hereditary hemorrhagic telangiectasia

Infectious mononucleosis

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Diagnosis

- History and clinical features
- Atypical lymphocytes on blood film
- Positive heterophile antibody test
  - Monospot test (mononucleosis spot test)
  - Paul–Bunnell agglutination test
- Indirect immunofluorescent assays
- Real-time PCR

Infectious mononucleosis

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Treatment

- Most cases resolves within 4 to 6 weeks
- Symptomatic treatment
  - bed rest
  - maintenance of fluid intake
  - adequate nutrition
  - analgesia
  - antipyretics

Infectious mononucleosis

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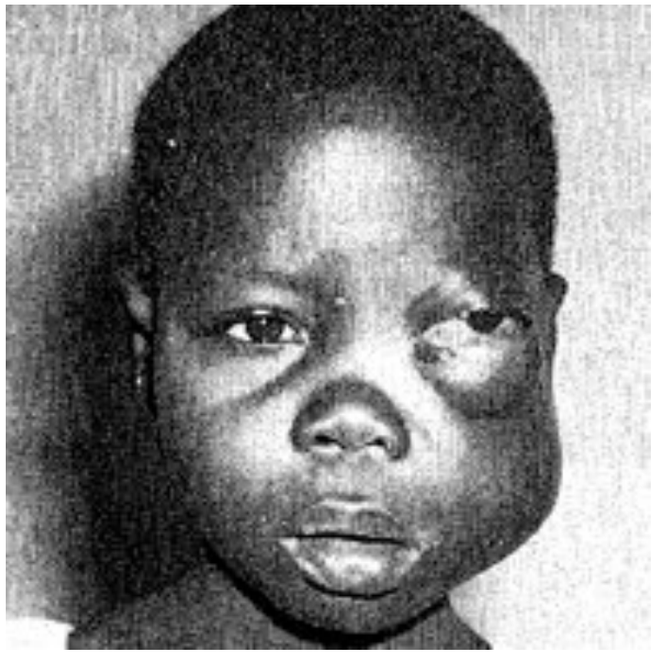
Complications: Possible significant complications include

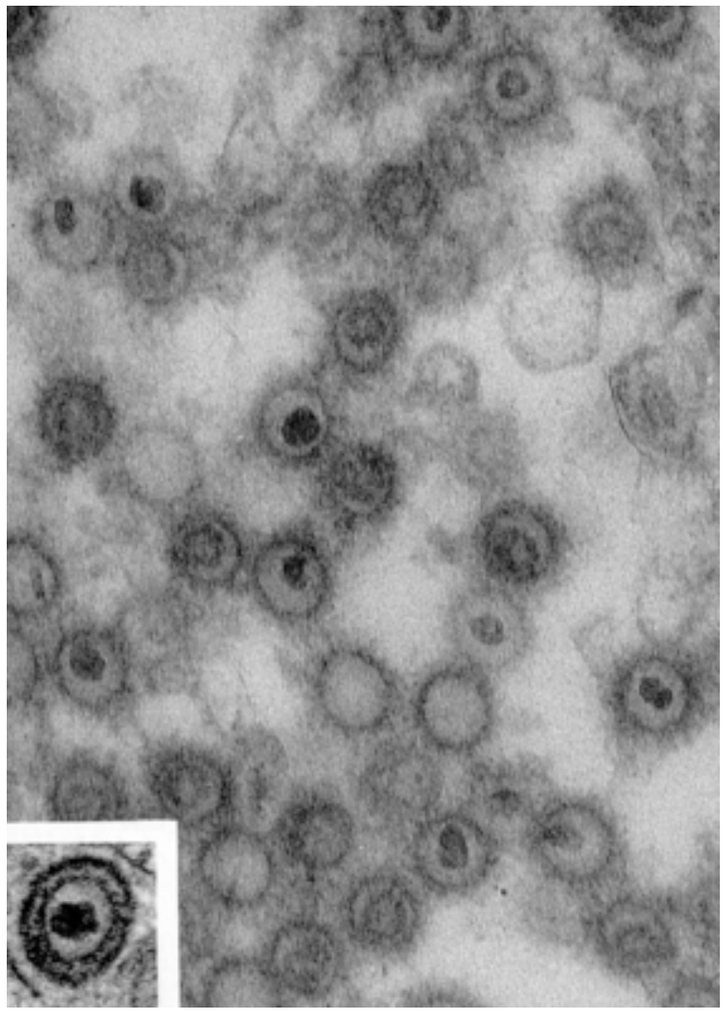
- Splenic rupture
- Thrombocytopenia
- Autoimmune hemolytic anemia
- Aplastic anemia
- Neurologic problems
- Myocarditis
- Hemophagocytic lymphohistiocytosis
- Patients experience fatigue lasting for several weeks to months (< 10% cases)
- Increases the risk for developing multiple sclerosis later in life

Epstein Barr Virus



Epstein Barr Virus

M A Epstein, B G Achong and Y S Barr in 1964



Electron micrograph of thin sectioned EBV particles. Immature virions (above) cut in various planes in an infected cell. Inset (below) a mature enveloped particle. These images led to the virus being immediately recognized as a member of the herpesvirus family

July 1941

HERPANGINA

JOHN ZAHORSKY, M.D.

» Author Affiliations

Am J Dis Child. 1941;62(1):169. doi:10.1001/archpedi.1941.02000130180018

Abstract

To the Editor:—The interesting article by Dr. B. B. Breese Jr. entitled "Aphthous Pharyngitis" (AM. J. DIS. CHILD.**61**:669 [April] 1941) impels me to report that this disorder, which I regard as a specific disease, was described by me in two articles entitled "Herpetic Sore Throat" (*South. M. J.***13**:1871, 1920) and "Herpangina" (*Arch. Pediat.***41**:181, 1924). Naturally, I am gratified to find that other pediatricians, such as Levine, Hoerr and Allanson (*J. A. M. A.***112**:2020 [May 20] 1939) and, more recently, Breese, have corroborated my earlier observations.

Herpangina

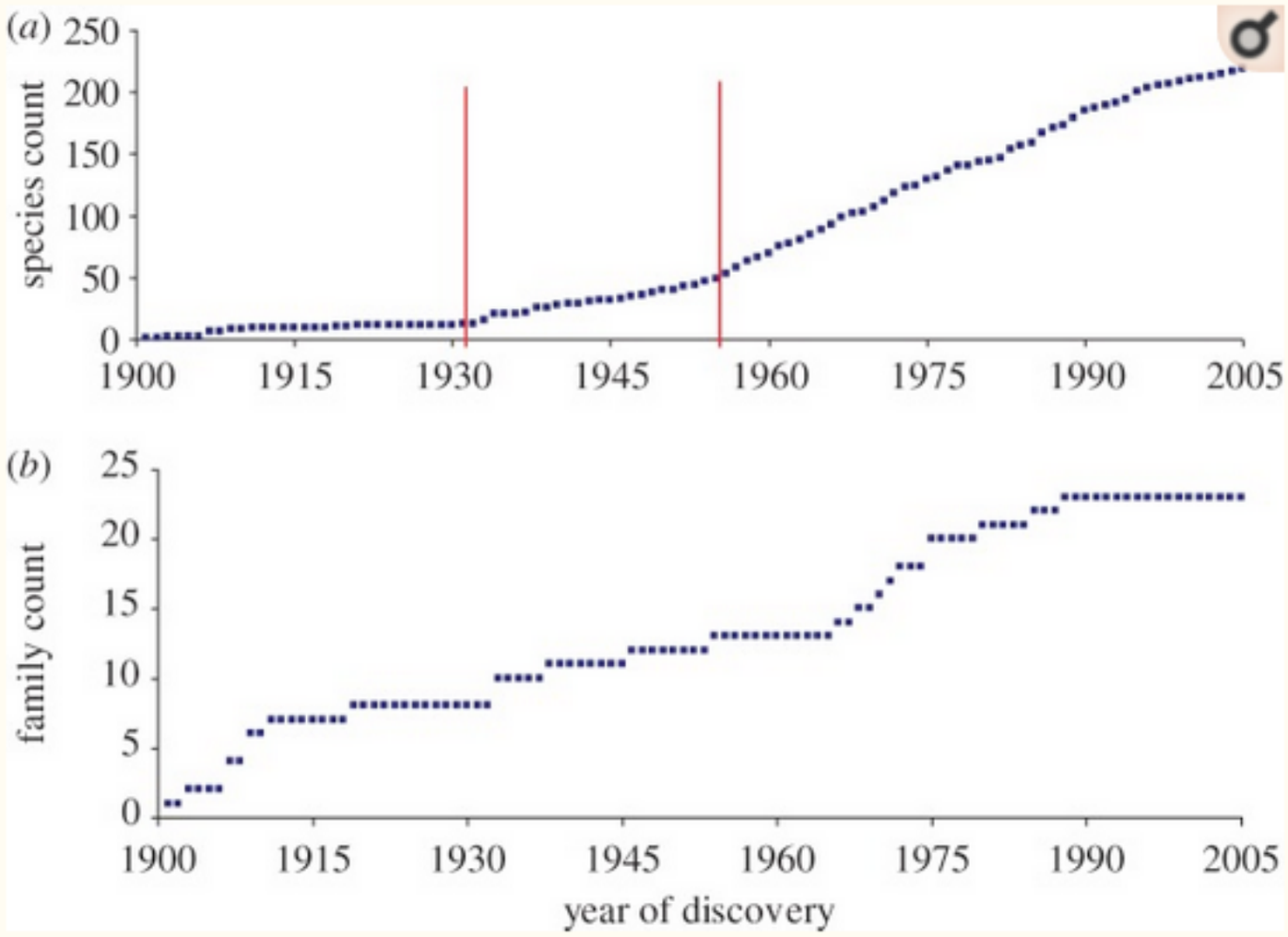


Figure 1.

Discovery curves for human viruses. (*a*) Virus discovery curve by species. Cumulative number of species reported to infect humans. Statistically significant upward breakpoints are shown (vertical lines). (*b*) Virus discovery curve by family. Cumulative number of families containing species reported to infect humans.

Table 1.

Major developments in the technology of virus discovery

year	technology
1890s	filtration
1929	complement fixation
1948	tissue culture
1970s	monoclonal antibodies
1985	polymerase chain reaction (PCR)
2000s	high throughput sequencing

Woolhouse et al. 2012

Herpangina

- Herpangina was first described in the 1920s but the viral etiology was not established until 1951 (Zahorsky, 1920; Huebner et al. 1951)

- **Etiology:** Coxsackie group A viruses

- **Age and sex**
  - Mainly affecting children up to 10 years of age
  - No gender predilection

- **Transmission:** Fecal-oral route

- **Incubation time**: 4 to 7 days

Herpangina

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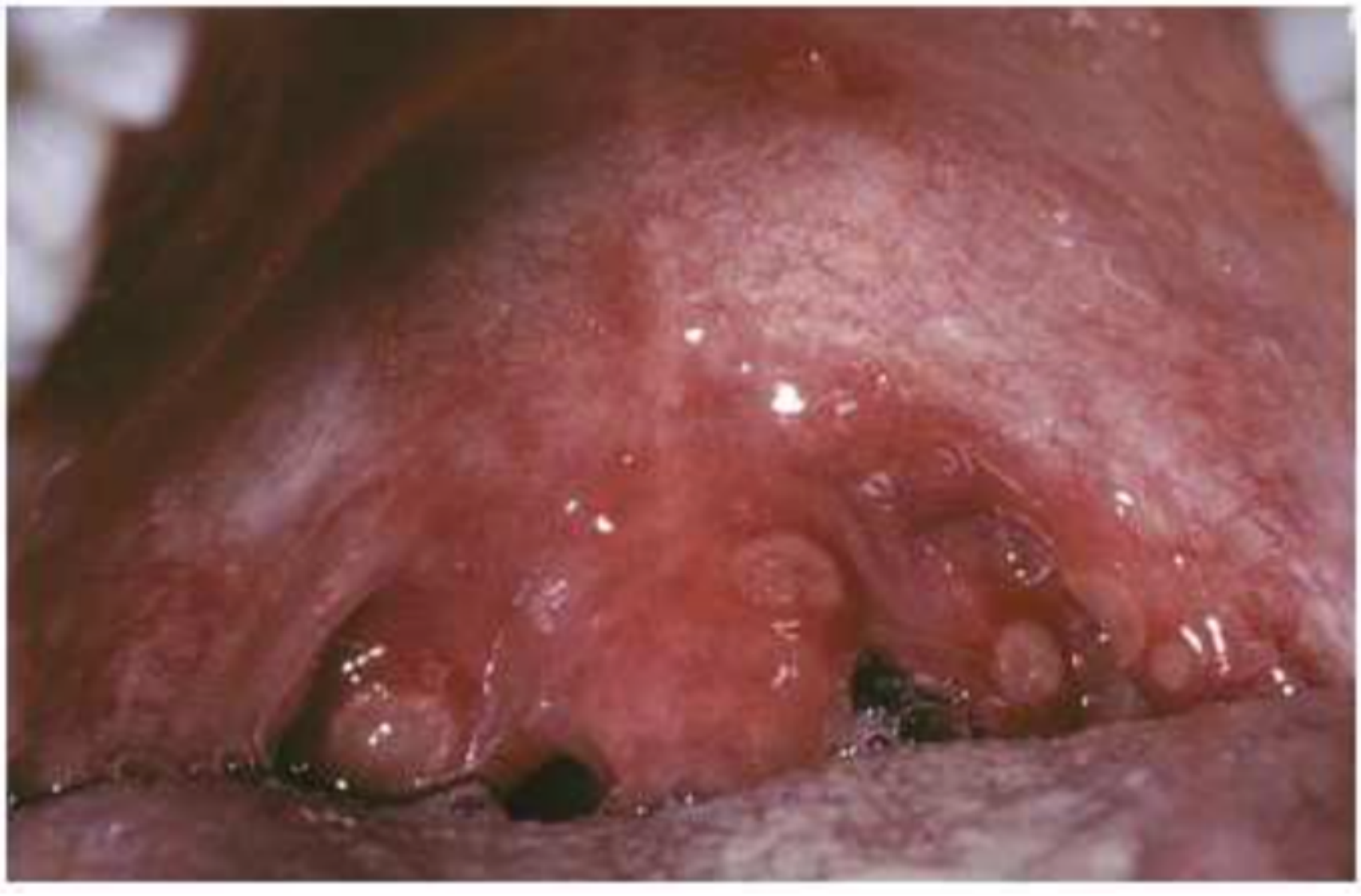
Clinical Presentation

- **General symptoms**
  - Low-grade fever, malaise, headache
  - Sore throat, dysphagia, anorexia, rhinorrhea, vomiting, diarrhea, myalgia
- **Oral lesions**
  - Appearance of the red macules (usually 2-6), which form fragile vesicles that rapidly ulcerate (2-4 mm)
  - Site: palate, pillars of the fauces and pharynx
  - Herpangina lesions do not coalesce to form large areas of ulceration

- **Course of disease**: self-limiting healing occurs within 1- 2 weeks

Herpangina



Numerous aphthous-like ulcerations of the soft palate

Herpangina

Histopathological

- Intraepithelial vesicles contain eosinophilic exudate
- Nuclear ballooning degeneration of epithelial cells

Differential diagnosis

- Other viral mucosal ulcers (e.g., HSV, CMV and EBV)

Diagnosis

- Clinical appearance and history
- Known epidemic
- Viral culture from swab

(Pinto and Hong 2013)

Herpangina

Sci Rep. 2016; 6: 35388.
Published online 2016 Oct 18. doi: 10.1038/srep35388

PMCID: PMC5067559
PMID: 27752104

Large outbreak of herpangina in children caused by enterovirus in summer of 2015 in Hangzhou, China

Wei Li,[1,*] Hui-hui Gao,[1,*] Qiong Zhang,[1] Yu-jie Liu,[1] Ran Tao,[1] Yu-ping Cheng,[1] Qiang Shu,[1] and Shi-qiang Shang[a,1]

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ELSEVIER

Clinical Microbiology and Infection
Volume 18, Issue 5, May 2012, Pages E110-E118

Original Article

Virology

Outbreak of hand, foot and mouth disease/herpangina associated with coxsackievirus A6 and A10 infections in 2010, France: a large citywide, prospective observational study

A. Mirand [a, b], C. Henquell [a], C. Archimbaud [a, b], S. Ughetto [c], D. Antona [d], J.-L. Bailly [b], H. Peigue-Lafeuille [a, b]

Herpangina

Management

- Symptomatic care
- Adequate hydration
- Analgesia for pain control
- Antipyretics

Complications: Potential complications include

- Pneumonia
- Pulmonary edema
- Hemorrhage
- Acute flaccid paralysis
- Encephalitis meningitis
- Carditis

Hand Foot and Mouth Disease

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- **Etiology:** either
  - Coxsackie A virus
  - Coxsackie B virus
  - or enterovirus 71

- **Age and gender**
  - Mainly affecting children up to 10 years of age
  - No gender predilection

Hand Foot and Mouth Disease

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- **Clinical Presentation**

  General

  – Low-grade fever, malaise
  – Sore throat, dysphagia
  – Occasional cough, rhinorrhea, anorexia, vomiting, diarrhea, myalgia, and headache

Oral lesions

– Resemble those of herpangina (more numerous and frequently involve anterior regions of the mouth)
– Appearance of the red macules (usually 2-6) , which form fragile vesicles that rapidly ulcerate (2-4 mm)
– Number of lesions ranges from 1 to 30
– Common sites: buccal mucosa, labial mucosa, and tongue, palate, pillars of the fauces and pharynx

Hand Foot and Mouth Disease

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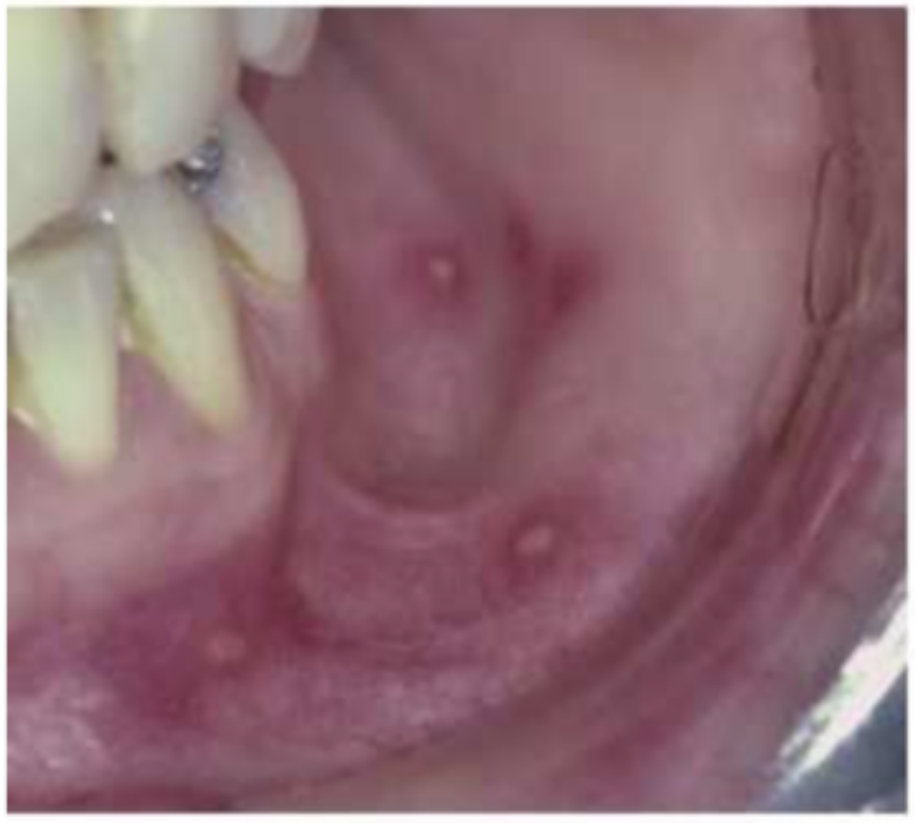
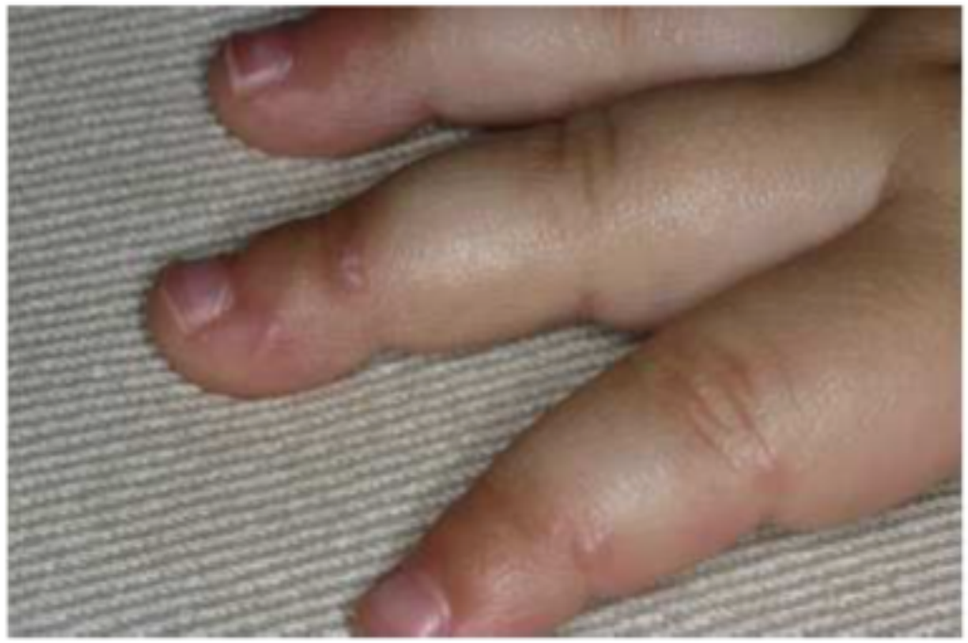
Clinical Presentation

- **Cutaneous lesions:** Range from a few to dozens
  - Site: **borders of the palms and soles** and **the ventral surfaces and sides of the fingers and toes** (primarily), buttocks, external genitals & legs (rarely)
  - Cutaneous lesions begin as erythematous macules that develop central vesicles and heal without crusting
  - nail loss or ridges (Beau lines) may ensue after several weeks
- **Course of disease**: self-limiting healing occurs within 1 week

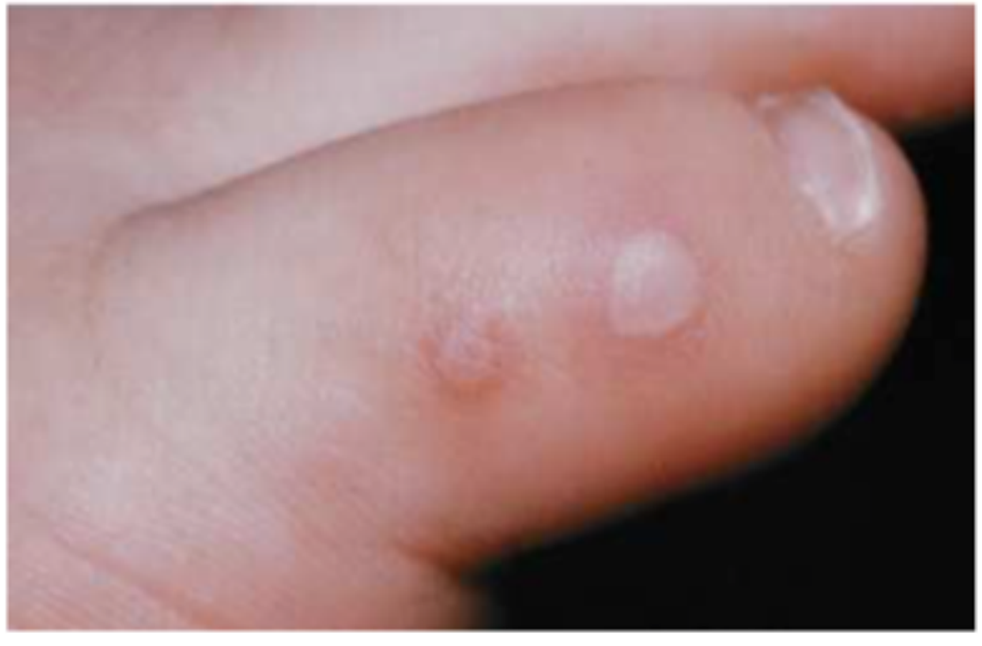
Hand Foot and Mouth Disease



Multiple aphthous-like ulcerations of the mucobuccal fold



Numerous cutaneous vesicles on the sides of the fingers



Multiple vesicles of the skin of the toe

Hand Foot and Mouth Disease

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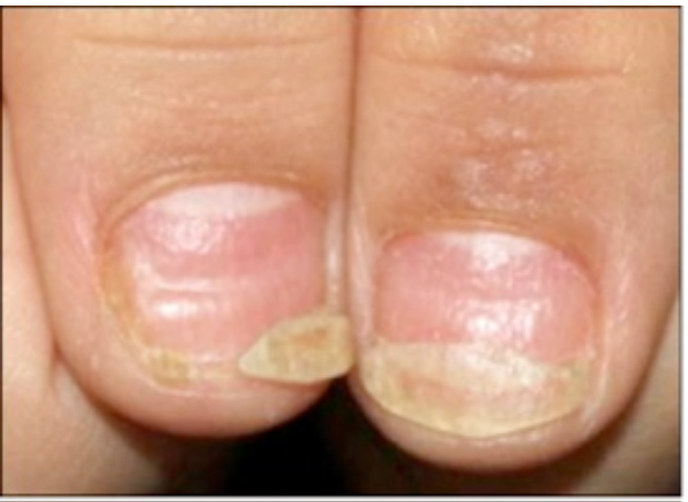

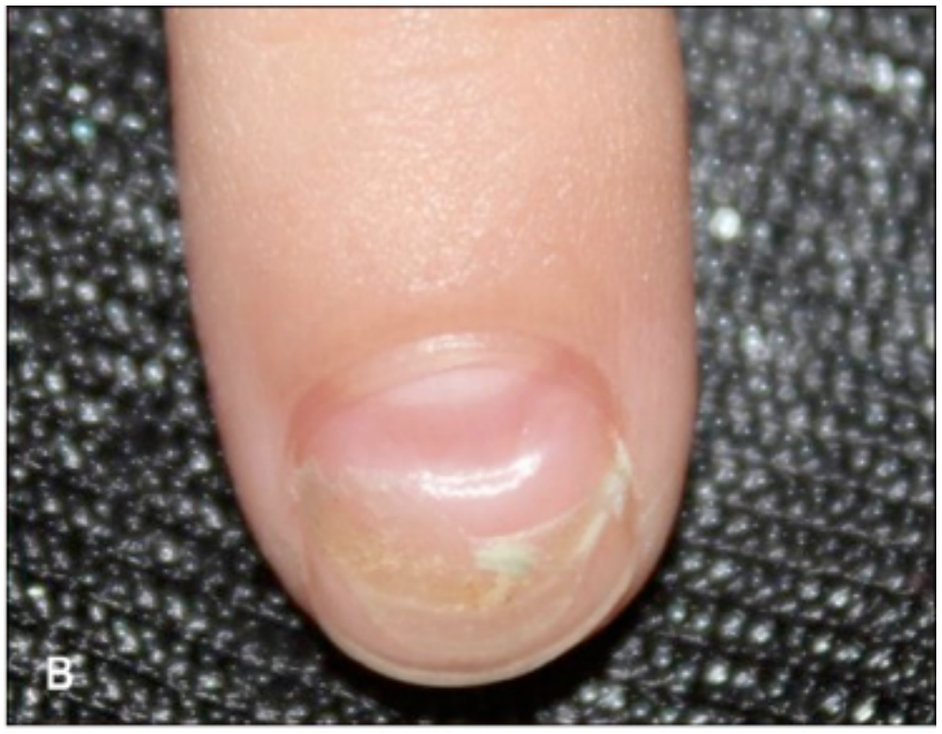

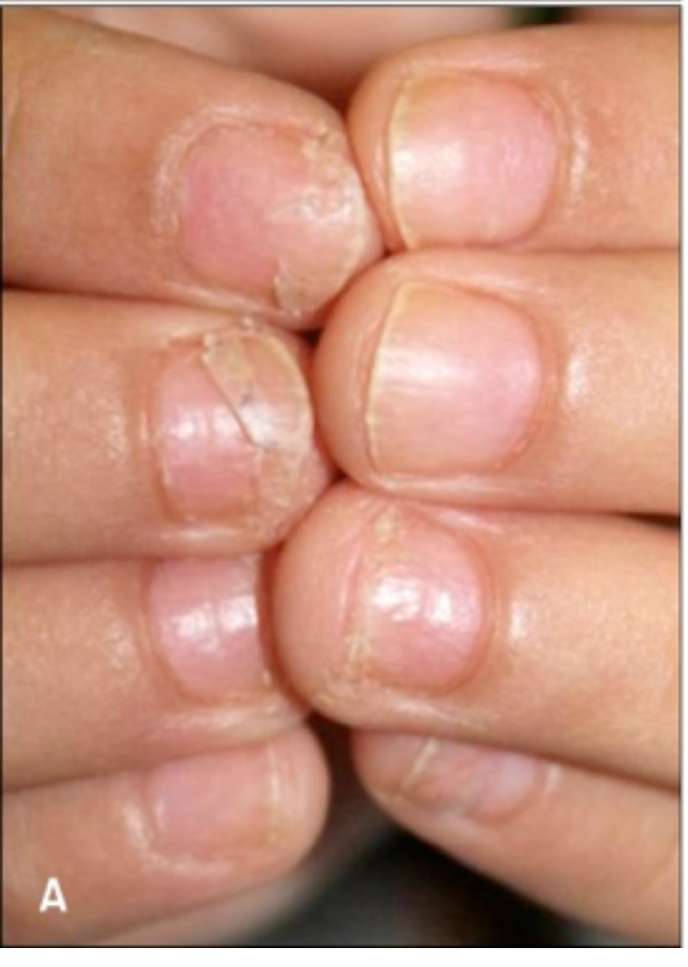

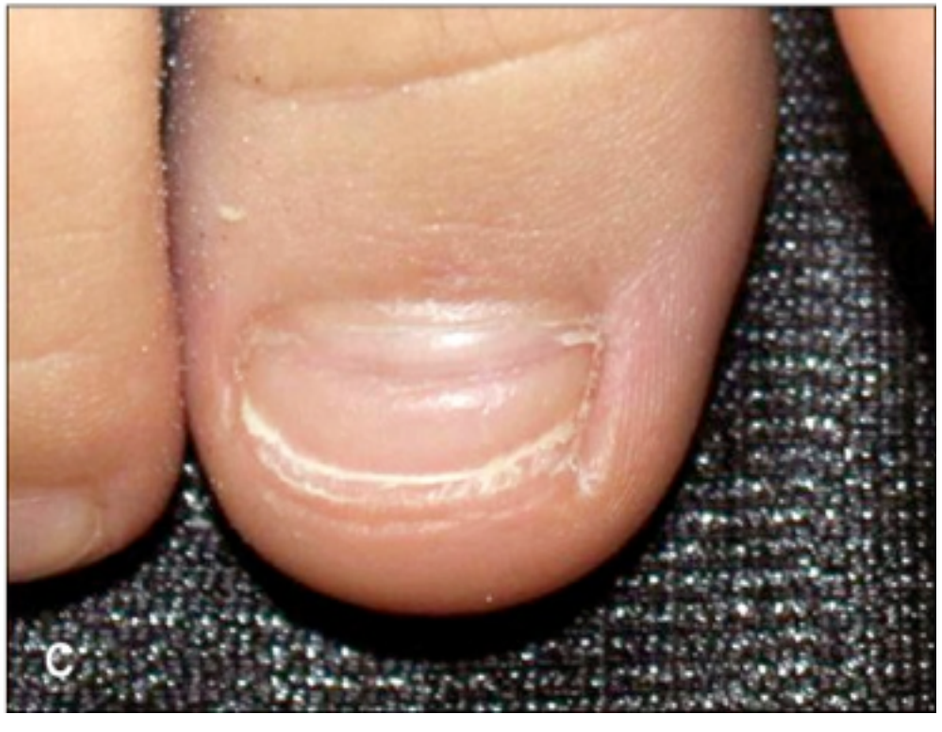

Shin et al. 2014

Hand Foot and Mouth Disease

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Histopathology

- Intraepithelial vesicles – early stages with intra-cytoplasmic eosinophilic inclusion bodies
- Later stages - shallow ulcerations and erosions with regeneration of the marginal epithelium
- Superficial inflammatory cell infiltrate in submucosa

Differential diagnosis

- Herpetic gingivostomatitis
- Herpangina
- Varicella
- Aphthous stomatitis

Diagnosis

- Clinical appearance and history
- Known epidemic
- Viral culture from swab

Hand Foot and Mouth Disease

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Management

- Symptomatic care
- Adequate hydration
- Antipyretics
- Analgesia for pain control

Complications: Potential complications include

- Neurological complications
- Viral meningitis
- Encephalitis
- Cerebellar ataxia

(Chang et al. 1999)

Varicella (Chickenpox)

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- **Etiology:** varicella-zoster virus (VZV or HHV-3)
- **Age and gender**
  - Two clinically distinct forms
    - Chicken pox: Children
    - Shingles: Older adults
  - No gender predilection
- **Transmission**
  - Spread through air droplets
  - Direct contact with active lesions
- **Incubation period**
  - 10 to 21 days (with an average of 15 days)

Varicella (Chickenpox)

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Clinical Presentation

- **In immunized children:**
  - General: low or no fever
  - Skin: a maculopapular, cutaneous rash with only a small number of lesions
  - Oral: few or no vesicles

- **Course of disease:** a shortened disease course of approximately 4 to 6 days

Varicella (Chickenpox)

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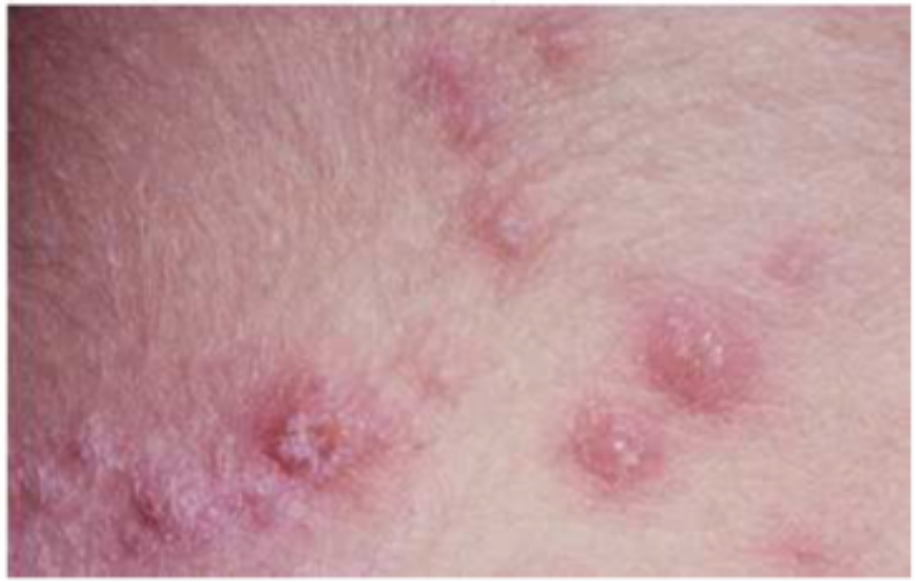
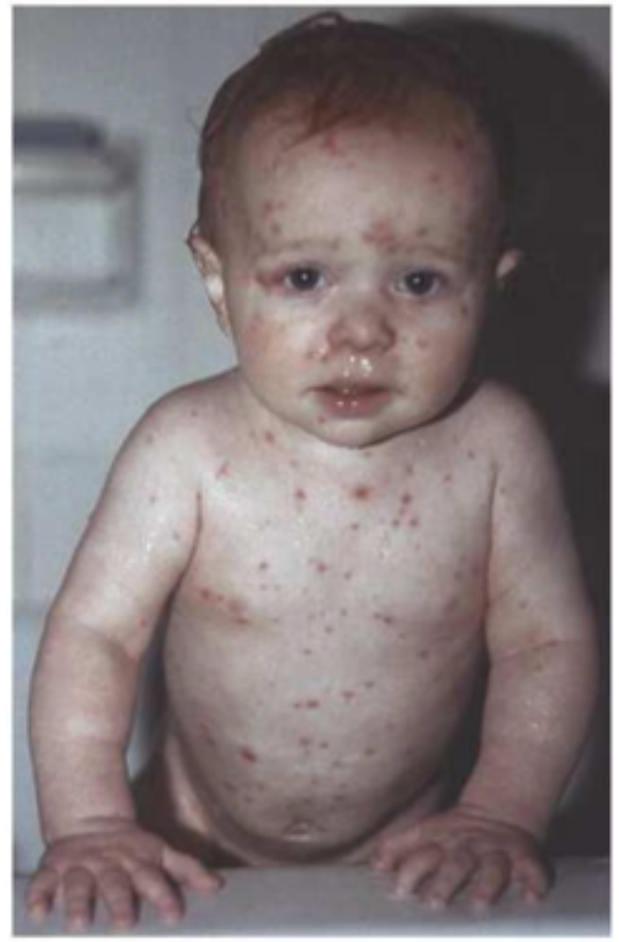
Clinical Presentation

- **unimmunized individuals**
- **General**
  - malaise, pharyngitis, and rhinitis
  - headache, myalgia, nausea, anorexia, and vomiting (occasionally)
- **Skin**
  - intensely pruritic exanthema (rash begins on the face and trunk and spreads to the extremities).
  - vesicular stage (classic presentation): each vesicle is surrounded by a zone of erythema and has been described as "**a dewdrop on a rose petal**"
  - Lesions continue to erupt for 4 or more days and old crusted lesions intermixed with newly formed, intact vesicles are commonplace





Varicella (Chickenpox)

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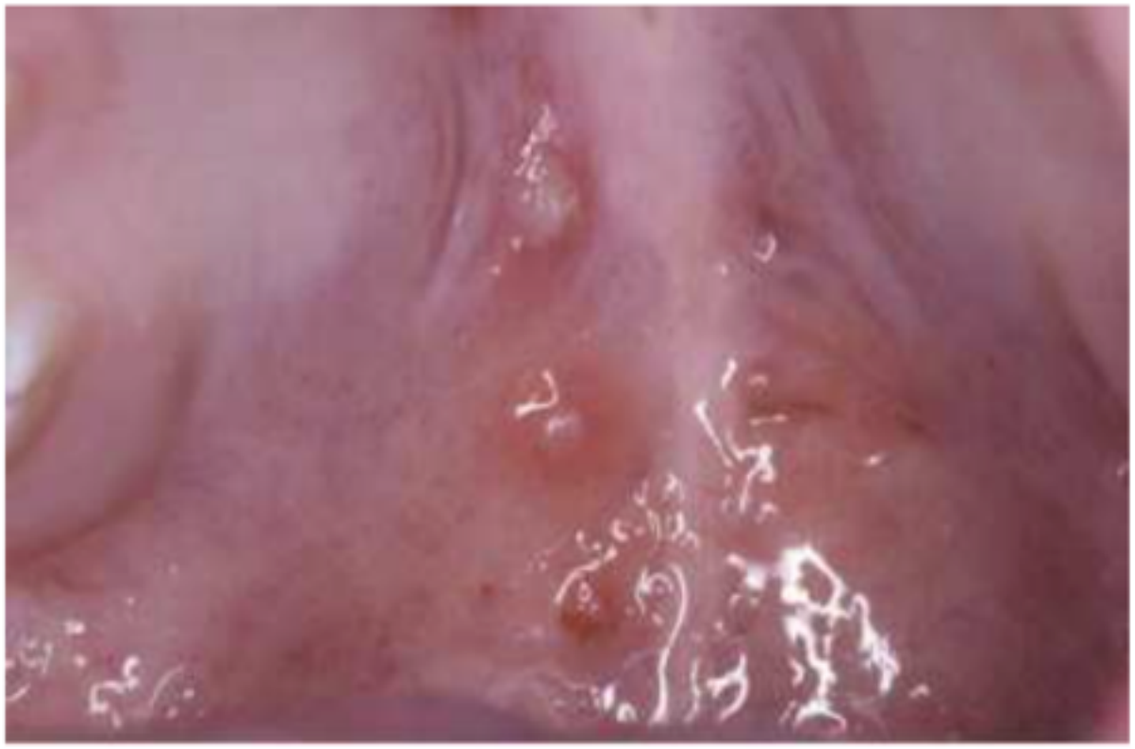
Clinical Presentation in unimmunized individuals

Oral lesions

- The lesions begin as 3- to 4-mm, white, opaque vesicles
- Vesicles rupture to form 1- to 3-mm ulcerations



- **Site:** vermilion border and palate (frequently), followed by the buccal mucosa and gingiva
- **Mild cases:** ulcers may not be present or one or two oral ulcers that heal within 1 to 3 days
- **Severe infections**: oral ulcerations are always present and numbering up to 30 and persisting for 5 to 10 days

Varicella (Chickenpox)

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National Immunisation Program Schedule

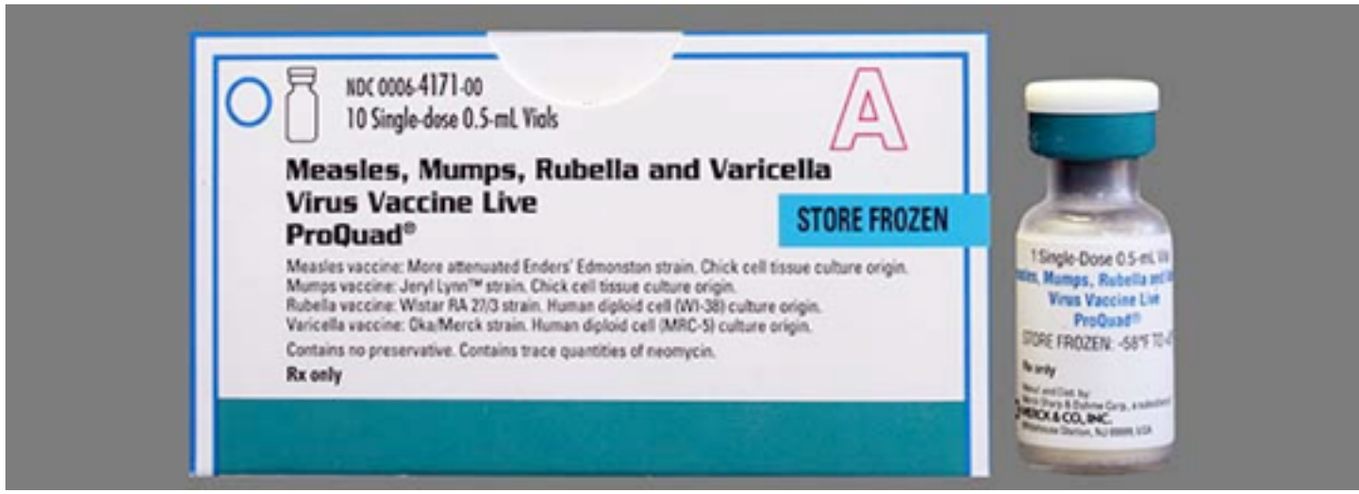
From 1 April 2019

Australian Government Department of Health

National Immunisation Program

A joint Australian, State and Territory Government initiative

Age	Disease	Vaccine Brand
Childhood vaccination (also see influenza vaccine)		
Birth	• Hepatitis B (usually offered in hospital)[a]	H-B-Vax® II Paediatric or Engerix B® Paediatric
2 months Can be given from 6 weeks of age	• Diphtheria, tetanus, pertussis (whooping cough), hepatitis B, polio, *Haemophilus influenzae* type b (Hib) • Pneumococcal • Rotavirus[b]	Infanrix® hexa Prevenar 13® Rotarix®
4 months	• Diphtheria, tetanus, pertussis (whooping cough), hepatitis B, polio, *Haemophilus influenzae* type b (Hib) • Pneumococcal • Rotavirus[b]	Infanrix® hexa Prevenar 13® Rotarix®
6 months	• Diphtheria, tetanus, pertussis (whooping cough), hepatitis B, polio, *Haemophilus influenzae* type b (Hib)	Infanrix® hexa
Additional vaccines for Aboriginal and Torres Strait Islander children (QLD, NT, WA and SA) and medically at-risk children[c]	• Pneumococcal	Prevenar 13®
12 months	• Meningococcal ACWY • Measles, mumps, rubella • Pneumococcal	Nimenrix® M-M-R® II or Priorix® Prevenar 13®
Additional vaccines for Aboriginal and Torres Strait Islander children (QLD, NT, WA and SA)	• Hepatitis A	Vaqta® Paediatric
18 months	• *Haemophilus influenzae* type b (Hib) • Measles, mumps, rubella, varicella (chickenpox) • Diphtheria, tetanus, pertussis (whooping cough)	ActHIB® Priorix-Tetra® or ProQuad® Infanrix® or Tripacel®
Additional vaccines for Aboriginal and Torres Strait Islander children (QLD, NT, WA and SA)	• Hepatitis A	Vaqta® Paediatric
4 years	• Diphtheria, tetanus, pertussis (whooping cough), polio	Infanrix® IPV or Quadracel®
Additional vaccines for medically at-risk children[c]	• Pneumococcal	Pneumovax 23®



Varicella (Chickenpox)

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National Immunisation Program Schedule

From 1 April 2019

Australian Government
Department of Health

National Immunisation Program
A joint Australian, State and Territory Government initiative

Age	Disease	Vaccine brand
Adolescent vaccination (also see influenza vaccine)		
12–<13 years (School programs[d])	• Human papillomavirus (HPV)[e] • Diphtheria, tetanus, pertussis (whooping cough)	Gardasil®9 Boostrix®
14–<16 years (School programs[d])	• Meningococcal ACWY	Nimenrix®
Adult vaccination (also see influenza vaccine)		
15–49 years Aboriginal and Torres Strait Islander people with medical risk factors[c]	• Pneumococcal	Pneumovax 23®
50 years and over Aboriginal and Torres Strait Islander people	• Pneumococcal	Pneumovax 23®
65 years and over	• Pneumococcal	Pneumovax 23®
70–79 years[f]	• Shingles (herpes zoster)	Zostavax®
Pregnant women	• Pertussis (whooping cough)[g] • Influenza[h]	Boostrix® or Adacel®

Varicella (Chickenpox)

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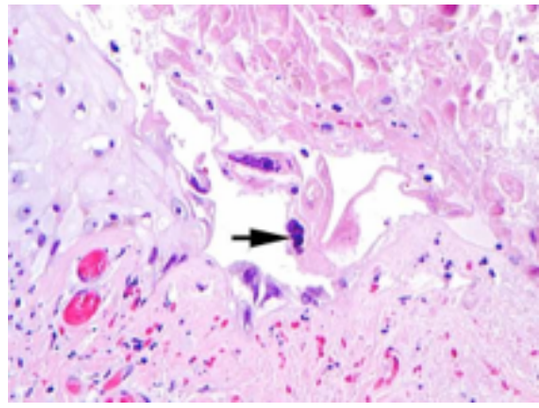
Histopathologic Features

- The cytologic alterations are virtually identical to HSV
- The virus causes acantholysis, with formation of numerous free-floating Tzanck cells, which exhibit nuclear margination of chromatin and occasional multinucleation



Diagnosis

- Viral cytology
- PCR (performed on vesicular fluid, cells from the base of a lesion, or a scab from a resolving skin lesion)
- Direct fluorescent antibody assay

Varicella (Chickenpox)

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- **Symptomatic treatment** (for relieve of pruritus)
  - Warm baths with soap, baking soda, or colloidal oatmeal
  - application of calamine lotion
  - antihistamines

- **Antipyretic**: Acetaminophen

- **Antiviral medications** (such as, acyclovir, valacyclovir, and famciclovir) have been shown to reduce the duration and severity of infection if administered within 24 hours of the rash

Varicella (Chickenpox)

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Complications

- secondary skin infections

Secondary skin infection with group A, β-hemolytic streptococci may progress to

- Encephalitis
- Pneumonia
- Necrotizing fasciitis
- Septicemia
- Toxic shock syndrome
- or other life-threatening conditions

Thank you