Infectious disease with manifestation in oral cavity. AIDS

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INFECTIVE DISEASES

- 1.Viral infection
- 2.Bacterial infection
- 3.Fungal infection
- 4.Protozoal infection

VIRAL INFECTIONS

- 1.Herpes Simplex Stomatitis
- 2.Herpes Zoster
- 3.Herpangina
- 4.Hand Foot Mouth Disease
- 5.Cytomegalovirus Infection
- 6.Measles
- 7.Infectious Mononucleosis
- 8.Mumps
- 9.HIV

Herpes infection

- **80 known herpes viruses – 8 are known to cause infection**
- **in humans.**
- HSV-1&2- Herpes simplex Virus
- HHV-3- Varicella – zoster virus
- HH4-Epstein Barr virus
- HHV5-
- HHV6 and 7- Roseolovirus
- HHV 8- Kaposi’s sarcoma herpes virus(KSHV), also asso
- with a small variety of lymphomas and castlemans
- disease

- Initial contact- primary infection-remains latent within the nuclei of specific cells
- Site of latency differs among the members
- After rectivation-localised or disseminated recurrent infections

Herpes simplex virus

- **Human reservoirs only**
- **2 types – structurally similar , antigenically &** biologically different
- **HSV-1: alpha-herpes virus is a ubiquitous virus**
- Above the waist – face, lips, oral cavity, upper body skin, ocular areas, pharynx
- **HSV-2: below the waist – genital lesions**

Pathogenesis

- Humans- only natural hosts
- Sources- saliva,skin lesions,respiratory secretions
- Transmission- close contact
- Virus- enters through the defect in skin –multiplies locally-cell to cell spread
- Enters enter nerve fibers – intra-axonally to the ganglia
- Centrifugal migration-from ganglia to skin and mucosa-cutaneous and mucosal lesions.
- Virus remain latent in ganglia-trigeminal(HSV-1)and sacral nerves(HSV-2)

reactivation

- **Trigerring factors:**
- **Fever**
- **UV light exposure**
- **Common cold**
- **Emotional stress**
- **Fatigue**
- **Trauma**
- **Oral cancer therapy**
- **Immunosuppression**
- **Oral& facial surgery**
- **Viral infection –HIV**
- **Gastrointestinal upset**
- **Pregnancy**
- **Menstruation**
- **Epidural morphine**

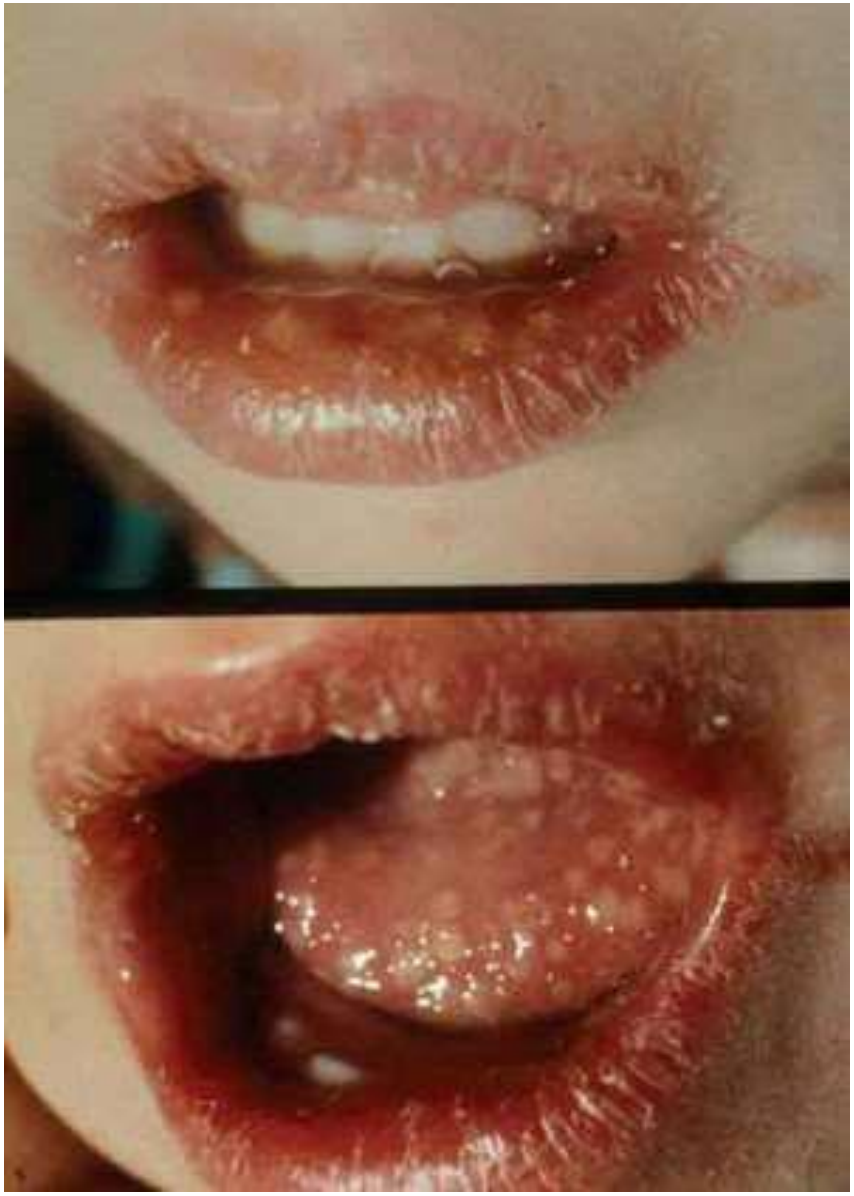
PHS

- **Acute herpetic gingovostomatitis:**
- **Most common pattern of symptomatic primary HSV binfection**
- **Incidence of primary HSV-1 increases after 6 months of age**
- **Peak of inciudence- 2 and 3 years of age**
- **Incidence of HSV-2 does not increase until sexual activity begins**

Clinical sights

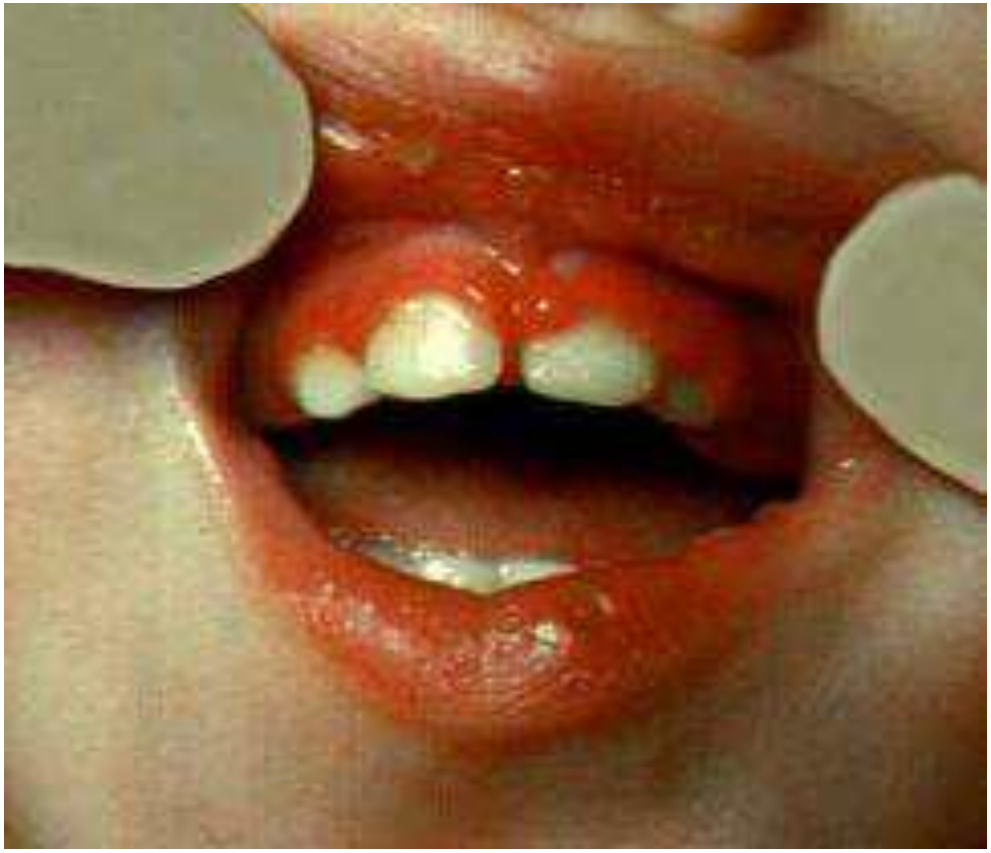
- Many primary infections are subclinical
- Symptomatic disease preceded by prodromalfever,
- headache, malaise , nausea, vomiting and LE

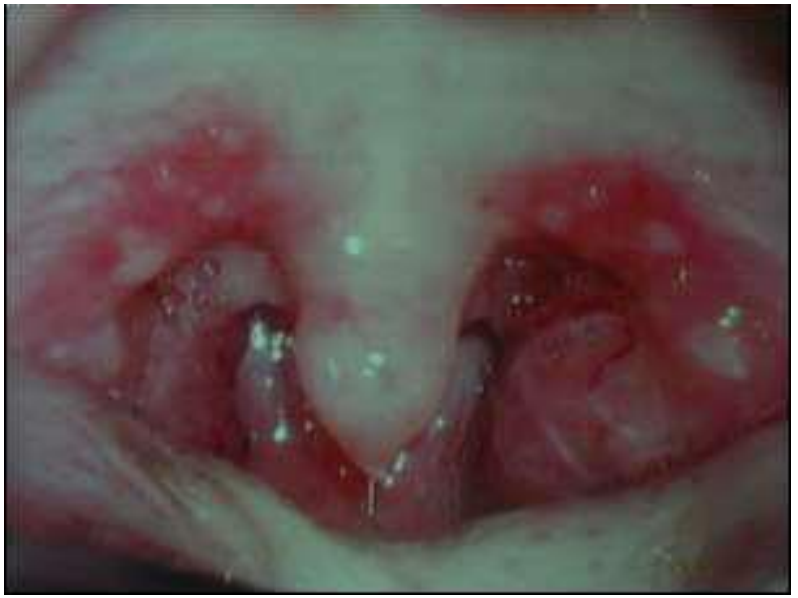
- Oral lesions –1-2 days after prodromals- 1-2mm pinhead vesicles – collapse – numerous small red lesions – enlarge – central area of ulceration with yellow fibrin – ulcers coalesce – larger shallow irregular ulcers – heal 10-14 days



Gingiva:

- Self innoculation – fingers, eyes, genital areas
- Children – generalized initial –macular, later – purpuric cutaneous rash
- Adults – **pharyngotonsillitis – sore throat, fever, malaise, headache –** initial symptoms. Numerous small vesicles – tonsils & posterior pharynx – diffuse grayish yellow exudate over the ulcers.
- Mild cases resolve within 5 to 7 days—may extend upto 2 weeks
- Rare complications- keratocoinjunctivitis, oesophagitis,meningitis and encephaliti





Differential diagnosis

- Herpetiform recurrent aphthous stomatitis
- Coxsackie virus infection (hand-foot & mouth disease)
- Herpangina
- Herpes zoster virus infection:
- Erythema multiforme (EM):.
- Acute necrotizing ulcerative gingivitis

Investigation

- **Cell culture : gold standard test for HSV isolation**
- Observe cytopathic effects of the cells inociulated with HSV
- Rate of CPE depends on type of host cell, type of virus , conc. Of virus
- **Cytology: scrapings –base of lesion smeared onto glass slides. Stained –**
- Wright, Giemsa (Tzanck preparation) or Papanicolaou stain - multinucleated giant cells / intranuclear inclusions like Lipschultz or Cowdry type A
- **PCR: most sensitive method-detection of viral DNA**
- Doesnot require viable virus or infected cells for detection
- used to discriminate HSV typess

- **Direct fluorescent antigen detection test:**
- Specimen incubated with fluorescin isothiocyante-labeled HSV type-specific monoclonal antibody-positive cells appear flourscent green
- Rapid diagnosis
- Sensitivity-80%,specificity-98%
- **Serological methods: primary HSV – IgM titers followed several weeks later by** permanent IgG titers (seroconversion)
- Recurrent infection - IgG antibody titer in acute & convalscent sera . Not reliable tool
- **Biopsy : not regularly done**
- **In immunocompromised pts, if biopsy revea;s features of HSV infectio, one should rule co-infection of CMV via immunocytochemistry**

Treatment

- Primary infection:
- PAIN CONTROL:
- 2% viscous lidocaine(swish and spit out 5ml 4-5times/d)
- Liquid diphenhydramine(swish and spit out 5ml 4-5times/d)
- Systemic analgesia
- Benzydamine

- Supportive care:
- Hydration
- ce-chips
- Soft bland diet
- antipyretics

Definitive therapy

- If presented within 24-48hrs after onset of vesicle formation,antiviral medication help in reducing the healing time:
- Acyclovir(Aceherpin) –inhibits viral replication
- Dosage: 200mg 5 times daily for 7 to 10 days/400mg TID for 7-10 days
- Valacyclovir(valcivir) and famciclovi(famtrex) is also available
- –both has better bioavailability-hence fewer daily doses.
- Dosage: valacyclovir 1000mg BID for 7-10 days famcilcovir 250 mg TID for 7-10 days

Recurrent HSV

- Reducing trigger factors – sunscreen
- **Topical antiviral medication:**
- 1. 5% acyclovir cream -1 appl – every 4hr for 5 days
- 2. 3% penciclovir cream,
- 3. 10% docosanol cream – 3-6times/day (at first prodrome)
- **Systemic therapy:**
- 1. Tab.Zovirax (Acyclovir): 200mg 5times/day for 7 days
- 2. Tab.Valcivir (Valaciclovir):500-1000mg three times/day for 5 days.
- 3. Tab.Famtrex (Famciclovir):500-1000mg three times/day for 5-7 days
- For patients –recureences asso with dental procedures,
- 2g valacyclovir BID on the day of procedure and 1g taken BID next day

Varicella (zoster virus)

- Enveloped DNA virus – ALPHA, HHV-3
- Primary infection – Varicella (chicken pox)
- Latent – dorsal root ganglia/ganglia of cranial nerves

Reactivation – Herpes zoster infection (shingles)

- Transmission: Air droplets/ direct contact with lesions.
- Children < 13 years
- Incubation period: 10-20 days.

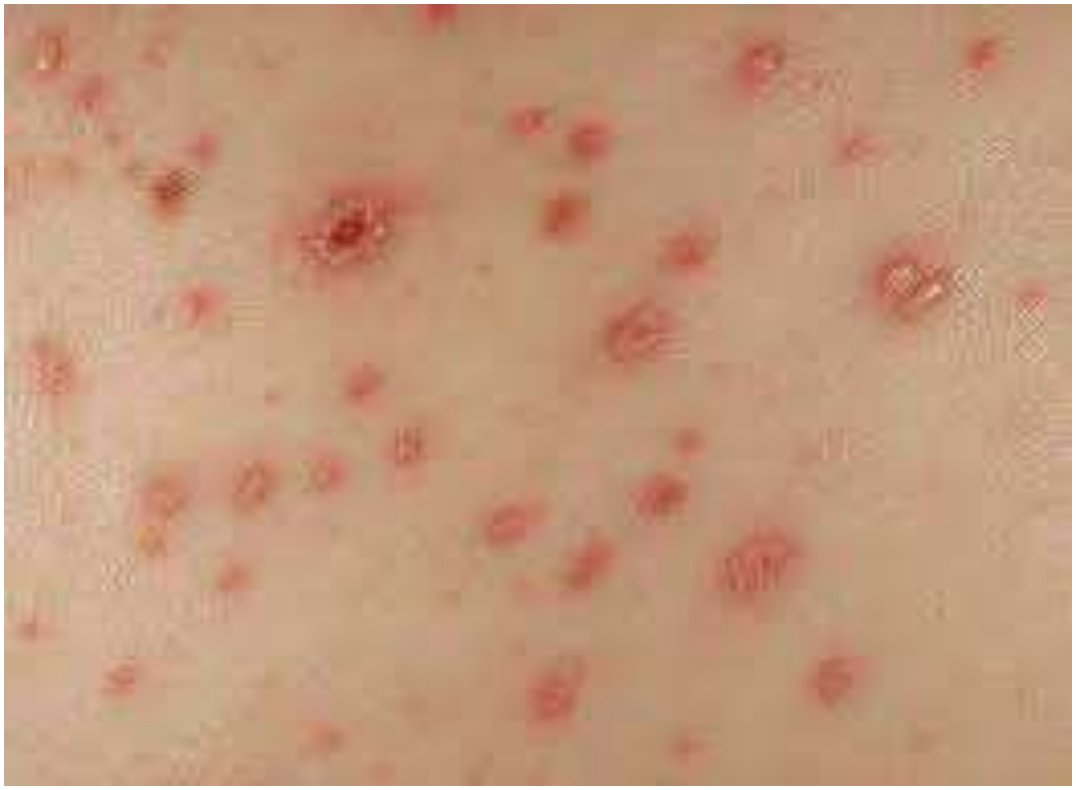
Chicken pox (varicella)

- **Chicken pox is an acute, ubiquitous, extremely contagious disease usually** occuring in children
- characterized by an exanthematous vesicular rash. It is most common in the winter & spring months.
- Source of infection-chicken pox or zoster patients
- Portal of entry- conjunctiva or respiratory tract
- Incubation period of about 2 weeks
- Pt infectious during 2 before the exanthem starts until the lesions crusts

Clinical features

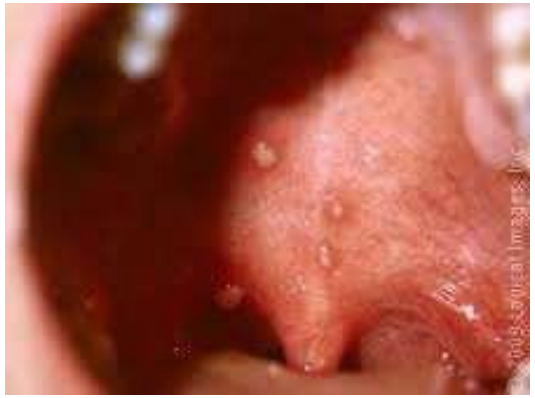
- First 2 decades of life
- Begins with prodromals
- Intensely pruritic exanthema, maculopapular rash –followed by vesicles-”dewdrop-like”-
- Turns cloudy and pustular , burst and scab with crst falling off after 1 to 2 weeks)
- Begin on face & trunk – extremities(centrifugal spread)
- Typically continue to erupt for 4 days-may extend upto 7 or more
- days
- Contagious from 2 days before eruoptions until all the lesions crust

Chicken pox



Oral manifestation

- Precede skin lesions-fairly common
- Most common sites vermillion border and palate-followed by buccal mucosa
- Gingival lesions may resemble Primary herpes (varicella-painless)
- Blister-like lesions – initial raised vesicles(3-4mm) with surrounding erythema – rupture – eroded ulcers (1-3mm)



Complications

- In childhood, use of aspirin – reyes syndrome
- Also secondary infectionsgroupA,
- β-hemolytic streptococci
- In adults, increased prevalence of complications,
- Varicella pneumonitis
- Encephalitis
- CNS involvementataxia, convulsions , coma
- During pregnancy,
- Early involvemnt-abortion or congenital defects

- In immunocompromised,
- Extennsive cutaneous involvement
- Hepatitis
- Pneumonitis
- Pancreatitis
- Encephalitis
- (7% mortality before advent of antiviral therapy)

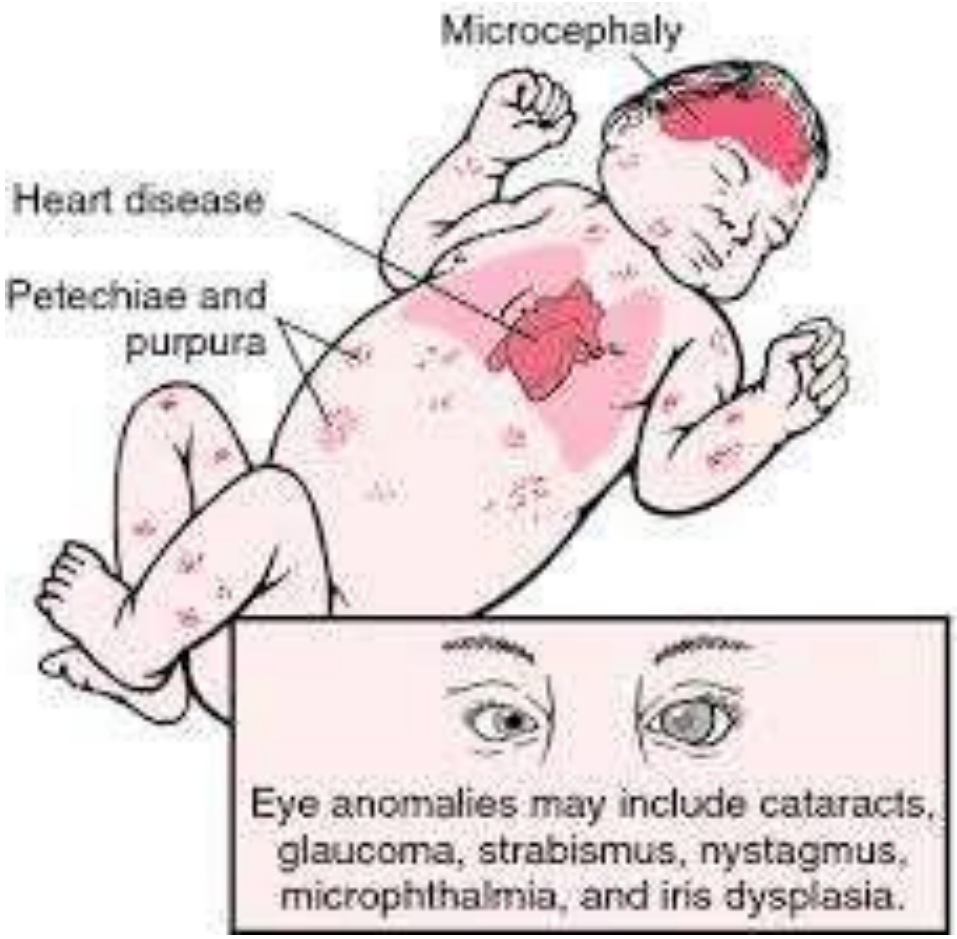
treatment

- **Symptomatic:**
- warm bath with soap(lipid envelope destroyed)
- **Calamine lotion and systemic diphenhydramine-relieve pruritis**
- **Antipyretics other than aspirin: Acetaminophen.**
- **Systemic:**
- To be administered within first 24 hours of the rash
- Reserved for pts at high risk for more severity
- 1. Acyclovir (Zovirax):adults-800 mg , 5 times/day for 7-10 days.
- 2. Valacyclovir (Valtrex):adults-1000 mg tid for 7-10 days
- 3. Famciclovir (Famvir): adult dose:500 mg tid for 7-10 days
- **Immunocompromised:**
- Purified VZVIg can be given to modify the clinical manifestations of the infection. Within 96 hours of initial exposure

Rubella (german measles)

- Primarily in winter
- Toga virus-genus rubivirus
- TRANSMISSION: droplet infection, congenital rubella syndrome.-occurs in winter
- Infection of the mother by Rubella virus during pregnancy can be serious-
- Congenital rubella syndrome
- Spontaneous abortion occurs in up to 20% of cases "blueberry muffin lesions."

Rubella


Microcephaly
Heart disease
Petechiae and purpura
Eye anomalies may include cataracts, glaucoma, strabismus, nystagmus, microphthalmia, and iris dysplasia.

Phatogenesis

- Transmitted by the respiratory route and replicates in the nasopharynx and lymph nodes.
- The virus is found in the blood 5 to 7 days after infection and spreads throughout the body.
- Teratogenic properties

Oral manifestations

- Forehheimer's sign : 20 % cases 6 hrs after 1st symptom
- Dark red papules on soft palate, hard palate along with rash
- Palatal petechiae
- 1st month of pregnancy- hypoplasia, caries, delayed eruption of deci teeth

Treatment

- No specific Rx-nonaspirin antipyretics and
- antipruritics
- Prevention: MMR vaccine, S.C inj
- 1st : around 1 year
- 2nd: 4-5 years.
- Passive immunity : human rubella immunoglobulin.

Scarlet fever SCARLATINA

- CAUSE:exotoxin released by β-haemolytic streptococci
- TRANSMISION: airborne droplet emision • INCUBATION PERIOD: 1-7 days
- SYMPTOMS:very painfulsore throat – tonsillitis, pharyngitis, malaise, headache, high fever (40st.C), vomiting, • skin rash - fine sandpaper rash over the upper body, begins to fade three to four days after onset and desquamation (peeling) begins. • No rash circumorally – “white mustache” – Filatov’s sign

- Clinical picture: strawberry tongue,enanthema especially soft palate tonsils and bucal areas.
- DIAGNOSIS: clinical, if blood test - leukocytosis with neutrophilia, high erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), • DIF.DG.:morbilli, rubella, toxoallergic drug exanthema Enathema, yellowish coating, edema, teeth impression on the sides of tongue, keratinisation disorders, hyperkeratosis of papillae, the tip and sides of tongue vivid red colour Rarely vesicles-necrosis-glossitis necrotisans.

Treatment

- **ATB(PNC,clindamycin or erythromycin)** - agrimony tea irrigation, intensive oral hygiene
- COMPLICATIONS:septic complications due to spread of streptococcus in blood

Diphtheria

- upper respiratory tract illness characterized by sore throat, low fever, and an adherent membrane (a pseudomembrane) on the tonsils, pharynx, and/or nasal cavity.
- eradicated in developed nations through widespread vaccination
- DPT (Diphtheria–Pertussis–Tetanus) vaccine
- CAUSE:Corynebacterium diphtheriae, G+
- TRANSMISION:direct physical contact or breathing the aerosolized secretions of infected individuals • INCUBATION PERIOD:1-7 days

- SYMPTOMS:
- The onset of disease is usually gradual. Symptoms include fatigue, fever, a mild sore throat and problems swallowing. Later tonsils and oropharynx vivid red color, whitegray / yellow pseudomembranes, adherent, cannot be wiped off, stomatitis diphterica, coated tongue, foetor ex ore
- Sore throat, vomiting, fever, enlargement of cervical LN, slightly painful
- DIAGNOSIS:. isolation of Corynebacterium diphtheriae from a clinical specimen, histopathology

Treatment

- diphtheria anti-toxin
- antibiotics do not help healing of local infection, used in patients or carriers to eradicate C. diphtheriae and prevent its transmission to others- Erythromycin, Procaine penicillin, Rifampin or Clindamycin
- Local - mouthwashes, intensive oral hygiene

AIDS

- RNA virus-(lentivirus subgroupretrovirus family)-AIDS
- **1st case reported in 1981 in US.**
- **Four recognised human retrovirus belong to 2 groups:**
- **Human T-lymphotrpic virus(HTLV-1 &2)**
- **HIV 1 &2(HIV-1 is more commonly asso with AIDS)**
- Transmission: sexual route, blood product exchange, mother-fetus. Organ transplantation, artificial insemination.

Stages of HIV infection

- 1. acute infection:
- Within 3-6 weeks of infection
- 50%-experience mononucleosis like symptoms
- Spontaneous resolution occurs withion week
- HIV antibody may be negative at the onset-become positive during course- seroconversion
- 2. Latent infection:
- Last upto several years
- Positive HIV antibody test –infectious
- Does not mean microbiological latency-replication continues
- Host immune response –can only limit the virus load.
- CD4+ decreses from 1000 per microlitre to about 500

- 3.persistent lymphadenopathy:
- Enlarged lymph nodes at leats 1 cm – persist for atleast 3 months
- 4. AIDS related complex(ARC): (CD4+-50-200per uL)
- Considerable immunodeficiency with constitutional symptoms and minor opportunistic symptomse
- Progress to AIDS in a few months

Clinical findings in HIV infection can be categorised as:

- Cardinal finmdings
- Kaposis sarcoma
- Pneumocytis carinii pneumonia
- Toxoplasmosis
- CMV retintis
- Oesophageal candidiasis

Characteristic findings:

- Oral thrush
- OHL
- Extrapulmonary and pulmonary TB
- Crypotococcla meningitis

Associated findings

- Weight loss more than 10%
- Fever more than one month
- Diarrhea more than one month
- Generalised extra-inguinal lymphoadenopathy
- Skin infections
- Persistent cough

Revised classification by European Community Clearing House: Group 1lesions strongly asso with HIV infection

- Candidiasis (pseudomembranous and
- erythematous)
- Hairy leukoplakia
- Kaposis sarcoma
- Non hodgkins lymphoma
- Periodontal disease(anug,anup)

Group 2 lesions less commonly asso with HIV infection

- Bacterial infection(m.tuberculosis,m. avium)
- Melanotic hyperpigmentation
- Necrotising ulcerative stomatitis
- Salivary gland disease
- Thrombocytopenia purpura
- Viral infections(HSV,HPV,VZV,HZV)

Group 3 lesions seen in HIV infection:

- Bacterial infections: (a.israeli,E.coli,Kliebsiella)
- Cat-scratch disease
- Drug reactions(EM,lichenoid,Toxci epidermolysis)
- Fungal other than candidiasis
- Neurologic disturbances
- Facial palsy
- TN
- Recurrent aphthous stomatitis
- Viral
- CMV
- Molluscum contagiosum

Oral Manifestations of AIDS

INFECTION	ORAL DISEASE
FUNGAL	CANDIDIASIS HISTOPLASMOSIS CRYPTOCOCCOSIS
VIRAL	HERPES SIMPLEX HERPES ZOSTER CMV EBV(HAIRY LEUKOPLAKIA) HHV-8 (KS) ORALWARTS(HUMAN PAPILOMA VIRUS)
BACTERIAL	LINEAR GINGIVAL ERYTHMA NUP TUBERCULOSIS