

Oral Infections

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Diseases of the oral mucosa

- 1. Infective stomatitis**
- 2. Non- infective stomatitis**
- 3. Malignant disease**

Infections

Oral infections can fall into three main categories:

- Bacterial- Aerobic and anaerobic
- Fungal- Candida and other mucomycoses
- Viral- Herpes viruses, HIV

Oral Infections

Oral Infections may arise as a result of

- Bacterial

1. NUG (ANUG)
2. Syphilis
3. Tuberculosis
4. Actinomycosis
5. Other

Oral Infections

Oral Infections may arise as a result of

- Fungal

1. Candidosis

Oral Infections

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- Viral

1. Human Herpes Virus I, II, III, IV, V, VI, VII, VIII,
2. Mumps
3. HIV

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1. NUG- Necrotizing Ulcerative Gingivitis (Vincent's Infection, Trench Mouth)
Distinct pattern of pathological changes of the gingiva. Previously called ANUG but acute dropped as there is no chronic form of the disease

Predisposing factors: Depressed systemic immunity

- Stress (stress related corticosteroid hormone are thought to alter T4/T8 lymphocyte ratio leading to a decreased neutrophilic chemotaxis and phagocytic response)
- Immunosuppression
- Smoking
- Local Trauma
- Poor nutrition
- Poor OH
- Inadequate sleep
- Recent illness

Oral Infections

NUG- from Neville Damm et al



Clinical Features- NUG

- Occur at any age (young to middle age)
- Inflamed interdental papillae (blunt and punched out)
- Crater-like necrosis, grey pseudo membrane
- Fetid odour, pain
- Lymphadenopathy and fever may occur



NUM- from Neville Damm et al

Oral Infections

NUG- from Neville Damm et al



NUM- from Neville Damm et al

Treatment- quick resolution following removal of bacterial challenge

- Scale
- Oral Hygiene
- Chlorhexidine MW
- Antibiotics (Metronidazole, Penicillin)
- Rest
- Investigate for possible immune suppression if resistant to therapy

Oral Infections

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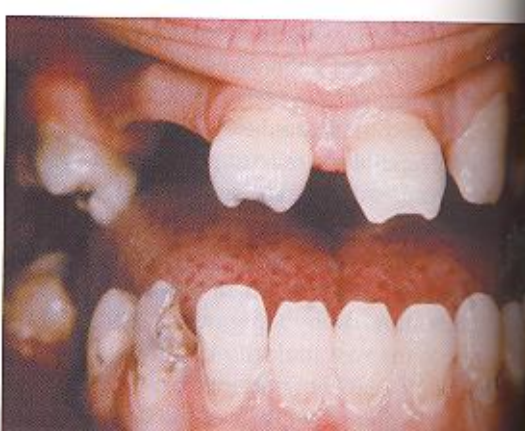
1. ANUG
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Oral Infections

- Bacterial

1. Syphilis – (*Traponema Pallidum*)
 - Sexual transmission, or from mother to foetus
 - Extremely vulnerable to drying
 - Proceeds through 3 stages : Primary and secondary (very contagious) and Tertiary
 - **Primary (Chancre)** develops at site of inoculation (painless clean based ulcer or similar to pyogenic granuloma (heals within 3-8 weeks if untreated)
 - **Secondary (disseminated)** 4-6 weeks after initial infection. Systemic symptoms (lymphadenopathy, sore throat Head ache, malaise musculoskeletal pain; Oral lesions may appear red maculopapular areas, Scarring and hyper/hypo pigmentation, mucous patches (30%), if they fuse may appear **snail track ulcer**, papillary appearance called Condyloma lata. Spontaneous resolution may occur
 - **Tertiary (Latent)** may develop 1 to 30 years later; Indurated nodular granulomatous inflammation called **Gumma**

Hutchinson's incisor

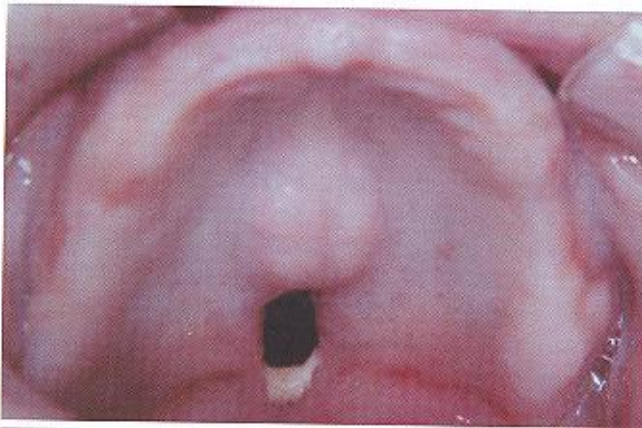


Infections-Bacterial



Secondary Syphilis- mucous patch

Mulberry molar



Gumma

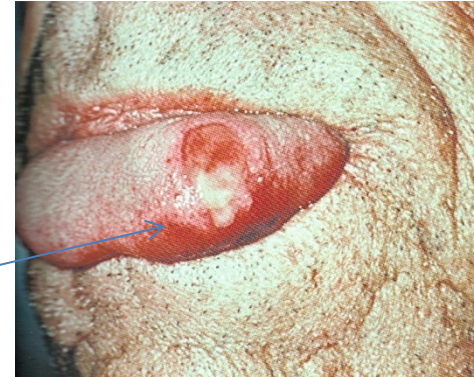


Secondary Syphilis

Oral Infections

- Bacterial

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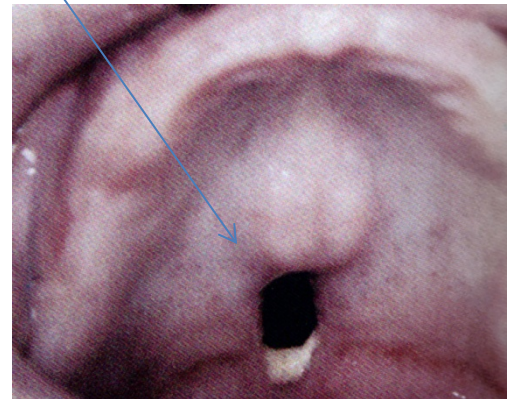
Oral Infections

- Bacterial

1. Syphilis **Tertiary (Latent)** may develop 1 to 30 years later; Indurated nodular granulomatous inflammation called **Gumma**



Condyloma Latum



Oral Infections- Syphilis



Congenital Syphilis

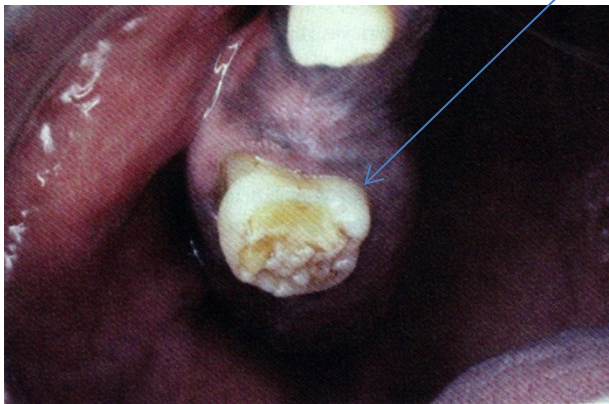
Hutchinson's Triad:

- Hutchinson's teeth: incisors
- Mulberry Molars
- Ocular interstitial keratitis
- Eighth nerve deafness

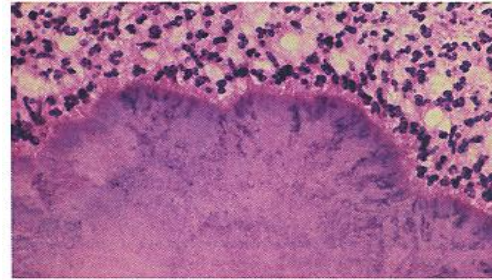
Diagnosis: Smear showing spiral organisms by dark- field exam

Serology- fluorescent treponemal antibody absorption (FTA-ABS) and micro hemagglutination assay for antibody to *T. Pallidum* (MHA-TP)

Treatment: Penicillin



Oral Infections



Tuberculosis

- Chronic infectious disease (*Mycobacterium tuberculosis*)
- *Primary infections usually asymptomatic and located at apex of lungs*
- *Oral lesions uncommon*
- *Diagnosis: 2-4 weeks after exposure cell-mediated hypersensitivity reaction to tubercular antigens develop—purified protein derivative (PPD) skin test*
- *Treatment- Multi agent (isoniazid, rifampin, pyrazinamide)*

Actinomycosis

Filamentous branching gram +ve anaerobic bacteria (*actiomyces israeli*)

Present in oral flora

Acute rapid progress infection

Chronic slow spreading lesion

Suppurative discharge of yellowish fleck (called sulfur granules)

Diagnosis by culture showing colonies surrounded by Polymorphs

Treatment: Penicillin

Infections-Bacterial

- **Impetigo**- Superficial infection of the skin that is caused by *Streptococcus pyogenes* and/or *Staphylococcus aureus*. Common in children and often occurs in areas of abrasion and cuts. Often the infection may be on the face, with vesicles that rupture and become thick adherent crusts. Usually no systemic manifestations such as fever.
- Mx- Topical mupirocin, Antibiotic (Clindamycin)

Mupirocin-[bacteriostatic](#) at low concentrations and [bactericidal](#) at high concentrations.[2] It is used topically and is effective against [Gram-positive bacteria](#), including [MRSA](#)

Infections-Bacterial :Other

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Infections-Bacterial : Other

Cat-scratch disease- infectious disorder beginning on skin but rapidly spreads to lymph nodes. Most common cause of chronic regional lymphadenopathy in children. Caused by *Bartonella henselae* or *Rochalimaea henselae* typically entering through a bite or scratch.

- Mx- self limiting but persistent lesions may need excision

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Oral Infections

Oral Infections may arise as a result of

- Fungal

1. Candidosis

Infections-Fungal

Candidosis- most commonly infection by *Candida albicans* (other members of Candida family include *C. tropicalis*, *C. krusei*, *C. parapsilosis*, *C. guilliermondii*)

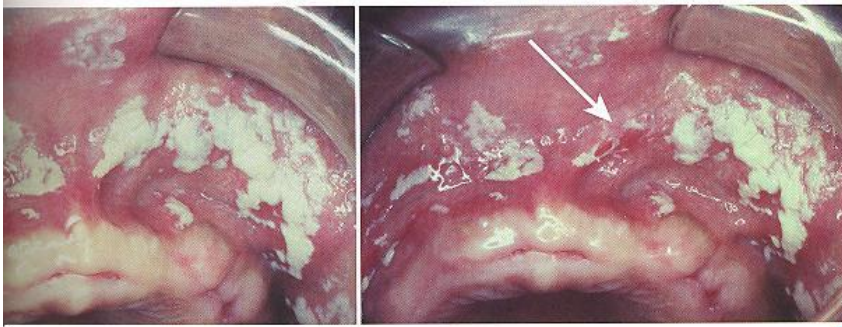
Infections-Fungal



Angular cheilitis



Denture associated



Pseudo membranous



Hyperplastic

CANDIDOSIS

ACUTE CANDIDOSIS

1. Thrush*
2. Acute antibiotic stomatitis

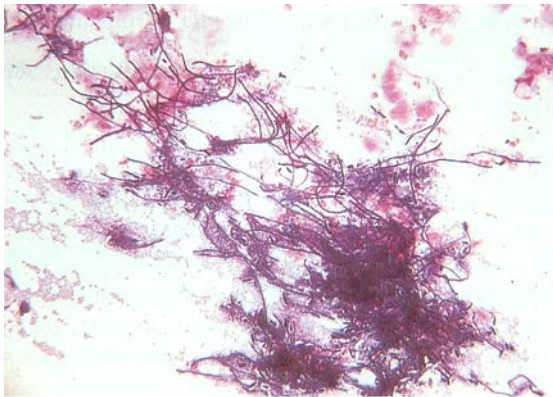
CHRONIC CANDIDOSIS

1. Denture-induced (chronic atrophic)
2. Chronic hyperplastic candidosis*
3. Chronic mucocutaneous candidosis
4. Erythematous candidosis
5. Angular stomatitis – common to all forms of oral candidosis

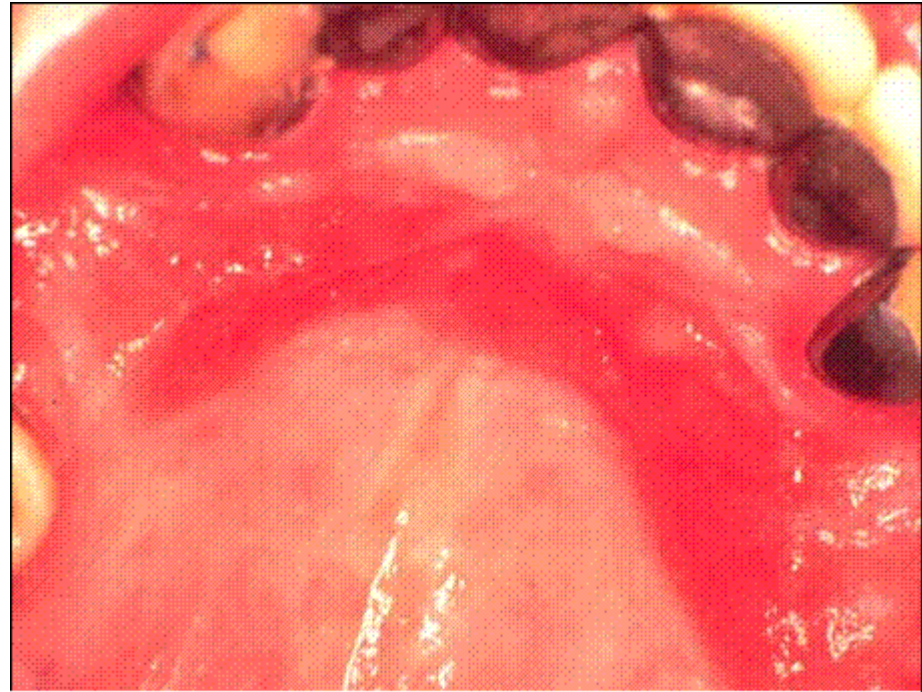
Thrush (Acute candidosis)



- Wipes off
- Adult male, no predisposing factors!!
→ - ? HIV



Candida



Candida



Candida

- Fungal infection
- *C. albicans* most common, but also *C. glabrata*, *C. tropicalis* and *C. krusei*
- notorious opportunistic pathogen
- Carriage rate ~ 20% of normal oral flora
- Harmless commensal in the mouths of nearly 50% of the population, becomes an opportunistic pathogen following a disturbance to oral flora in decreased immunity
- Increases to ~ 40% in presence of medical conditions, pregnancy, tobacco smokers and denture wearers
- Children: peak carriage ~ 45% at ~18 months
- Primary site is dorsum of tongue
- Mechanism which they induce disease not really known
 - secreted enzymes, toxins, antigenic response

Candida

- Factors pre-disposing to infection:
 - Local
 - mucosal trauma, denture wearing, tobacco smoking
 - Age
 - extremes
 - Drugs
 - broad spectrum AB's, steroids, immunosuppressant agents
 - Xerostomia
 - drugs, radiotherapy, Sjogren's syndrome
 - Systemic disease
 - HIV, Fe deficiency, diabetes, leukaemia, some anaemia

Candida

- Three most common presentations are:
 - Acute Pseudo membranous
 - ‘thrush’
 - thick white coating on affected mucosa, wipes off leaving red surface
 - resembles milk-curd
 - Caused by drugs (corticosteroids, Abs, immunosuppressant) , xerostomia, immune defects
 - Dx- smear with PAS staining
 - Mx- treat predisposing factors, antifungal
 - Chronic atrophic
 - ‘denture stomatitis’, ‘median rhomboid glossitis’
 - erythema
 - related to denture hygiene

Candida

- Angular cheilitis
 - co-infection with *S. aureus*
 - soreness, erythema, fissuring at the corners of the mouth
 - ? nutritional deficiencies
 - riboflavin, folic acid, B₁₂
- Chronic mucocutaneous candidosis- number of rare congenital syndromes with persistent candidosis affecting the mouth, skin, nails and other areas.
- Candidal leukoplakia (chronic hyperplastic candidosis)- smoking tends to predispose; typically dorsum of tongue and post commissural buccal mucosa. rough irregular surface. Potentially malignant.

Candida

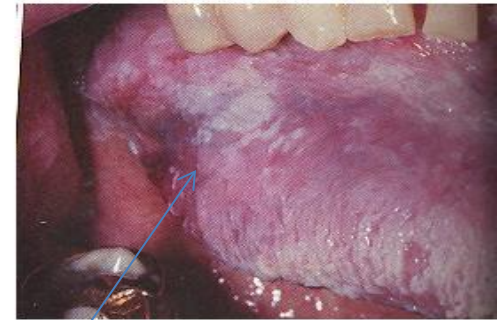
Treatment

Polyene agents- Nystatin; Amphotericin B- poorly absorbed in GI tract so requires good direct contact

Imidazoles- Clotrimazole, Ketaconazole (absorbed by GI tract), Ketaconazole may react with erythromycin, cisapride (GI tract motility enhancing agent) antihistamine astemizole

Triazoles- well absorbed and used systemically (Fluconazole, Itraconazole), Fluconazole may interact with oral hypoglycemics, phenytoin, cyclosporin, astemizole, tacrolimus, cisapride; Itraconazole should not be used with astemizole, cisapride, medazolam)

Candida



| CLINICAL TYPE | APPEARANCE AND SYMPTOMS | COMMON SITES | ASSOCIATED FACTORS AND COMMENTS |
|-----------------------------------------------------------------------|--------------------------------------------------------------------------------|------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------|
| Pseudomembranous (thrush) | Creamy-white plaques, removable; burning sensation, foul taste | Buccal mucosa, tongue, palate | Antibiotic therapy, immunosuppression |
| Erythematous | Red macules, burning sensation | Posterior hard palate, buccal mucosa, dorsal tongue | Antibiotic therapy, xerostomia, immunosuppression, idiopathic |
| Central papillary atrophy (median rhomboid glossitis) | Red, atrophic mucosal areas; asymptomatic | Midline posterior dorsal tongue | Idiopathic, immunosuppression |
| Chronic multifocal | Red areas, often with removable white plaques; burning sensation, asymptomatic | Posterior palate, posterior dorsal tongue, angles of mouth | Immunosuppression, idiopathic |
| Angular cheilitis | Red, fissured lesions; irritated, raw feeling | Angles of mouth | Idiopathic, immunosuppression, loss of vertical dimension |
| Denture stomatitis (chronic atrophic candidiasis, denture sore mouth) | Red, asymptomatic | Confined to palatal denture-bearing mucosa | Probably not true infection; denture often is positive on culture, but mucosa is not |
| Hyperplastic (candidal leukoplakia) | White plaques that are not removable, asymptomatic | Anterior buccal mucosa | Idiopathic, immunosuppression; care must be taken not to confuse this with other keratotic lesions with superimposed candidiasis |
| Mucocutaneous | White plaques, some of which may be removable; red areas | Tongue, buccal mucosa, palate | Rare; inherited or sporadic idiopathic immune dysfunction |
| Endocrine-candidiasis syndromes | White plaques, most of which are not removable | Tongue, buccal mucosa, palate | Rare; endocrine disorder develops after candidiasis |

Infections-Fungal

Histoplasmosis- Infection by *Histoplasma capsulatum* (as with candida it is dimorphic). Most oral lesions occur with disseminated histoplasmosis (other types are ; Acute and chronic) especially the tongue, palate and buccal mucosa (solitary painful ulcer).

- Mx- Amphotericin B

Oral Infections

Oral Infections may arise as a result of

- Viral

1. Human Herpes Virus I, II, III, IV, V, VI, VII, VIII,
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Oral Infections

Oral Infections may arise as a result of
Viral

1. Human Herpes Virus I, II, III, IV, V, VI, VII, VIII,

(Herpes Greek for creep or crawl)

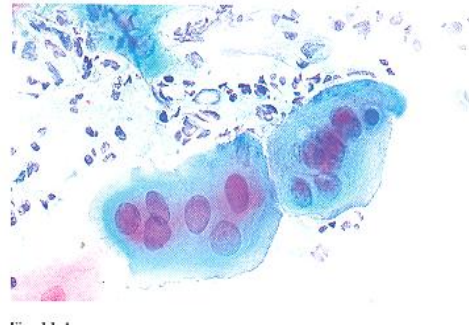
- **HSV1 (oral) HSV II** Genital produce similar lesions and can occur in either region, antibodies to one produces cross-react against the other
- **HSV I** clinically exhibits a primary (acute herpetic gingivostomatitis / pharyngotonsillitis) usually at young age

Secondary or recurrent (may exhibit viral shedding) especially with ultraviolet light

- **HSV** recurrence may trigger 15-60% of mucosal Erythema multiforme

Oral Infections- HSV

- **Diagnosis**- clinical history, serology two sample initial at 3 days {-ve} and at 4 weeks {+ve}(HSV antibodies are + ve 4 to 8 days after), virus isolation from vesicle
- **Treatment**- Symptomatic and antiviral Acyclovir suspension rinsed and swallowed 5 x day/5 days (15mg/kg child; 200mg adults)
- For those with more than 6 yearly recurrences, immune compromised and erythema multiforme– use prophylactic systemic acyclovir



Oral Infections- HHV

HHV 3 Varicella-zoster

- Primary infection is chickenpox (malaise, pharyngitis, head ache, myalgia etc)
- Secondary as herpes zoster (even 10 years later)
- Spread through direct contact or droplet
- Perioral and oral lesions may precede skin lesions and similar to HSV
- Dx- history and clinical appearance; serum samples at acute stage and 14 days later
- Tx- symptomatic and antiviral meds
- 10-20% may develop zoster (predisposed by immune suppression, stress, drugs, dental manipulation etc)

HHV 4 (EBV- Epstein Barr Virus)

- Kissing disease (infection through intimate contact)
- EBV remains in the host for life
- Prodromal fatigue, malaise, anorexia up to 2 weeks before, body temp 104°
- Prominent lymphadenopathy
- Petichiae in hard and soft palate
- NUG, Chronic fatigue (controversial), Hairy leukoplakia are associated with EBV
- Dx- increased WBC , Paul-Burnell test (presence of heterophil antibodies {immunoglobulin that agglutinate sheep erythrocytes})
- Tx- resolved in 4-6 weeks, non aspirin based antipyretics,
- Complications- splenic rupture, Bell's palsy

Oral Infections- HHV

- HHV 3 Varicella-zoster
- HHV 4 (EBV- Epstein Barr Virus)



Oral Infections- HHV

HHV 5 (CMV- Cytomegalovirus)

- Similar to HSV, can reside latently in salivary glands
- 90% infections are asymptomatic
- 1/3 may present with pharyngitis and lymphadenopathy; acute sialadenitis that affects all salivary glands (xerostomia)
- Dx- clinical features; biopsy (“owl eye “ cells, PAS staining to demonstrate cytoplasmic inclusions)
- Tx- resolve spontaneously; gancyclovir

HHV 8 (Kaposi Sarcoma)

- Unusual vascular neoplasm (four types: classic, endemic {African}, iatrogenic immunosuppression related, AIDS related)
- Tx- radiation therapy, chemotherapy (vinblastine)



Infections



Oral Infections

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3. HIV

Oral Infections

Epidemic Parotitis (Mumps)

- Caused by paromyxivirus causing diffuse disease of exocrine glands
- 90% before 15 years (30% are subclinical)
- Enlarged salivary glands peaking in 2-3 days with pain on one side followed by the other (unilateral in 25% cases)
- Epididymo-orchitis occurs in 25% cases of post pubertal males
- Most frequent report of enlargement of Wharton's and Stensen's salivary gland duct opening
- Tx- supportive and non aspirin analgesics



Oral Infections

HIV (Human immunodeficiency Virus and AIDS)

First reported in 1981

Initially may be asymptomatic or acute response similar to infectious mononucleosis (generalised lymphadenopathy, sore throat, fever, head ache, myalgia, peripheral neuropathies)

Oral Infections

EC- Clearinghouse classification of oral manifestation of HIV Disease in Adults

Group I STRONGLY ASSOCIATED WITH HIV INFECTION

- Candidosis : erythematous, pseudo membranous, angular cheilitis
- Hairy leukoplakia
- Kaposi's sarcoma (KS)
- Non- Hodgkin's lymphoma
- Periodontal disease (linear gingival erythema, NUG, NUP)

EC- Clearinghouse classification of oral manifestation of HIV Disease in Adults

Group II LESS COMMONLY ASSOCIATED WITH HIV INFECTION

- Bacterial infection TB
- Melanotic hyper pigmentation
- NUS (necrotizing ulcerative stomatitis)
- Salivary gland disease (Xerostomia, gland swelling)
- Thrombocytopenia purpura
- Oral ulceration NOS
- Viral infection HSV, HPV, V-Z

Oral Infections

EC- Clearinghouse classification of oral
manifestation of HIV Disease in Adults

Group III SEEN IN HIV INFECTION

- Bacterial infection *A. Israelii*, *E. coli*
- Cat-scratch disease
- Epithelioid angiomatosis
- Drug reactions: ulcerative, lichenoid
- Fungal infections other than candidosis *Histoplasma capsulatum*
- Neurological disturbances: facial palsy, TN
- RAS
- Viral infections CMV, molluscum contagiosum

Hairy Leukoplakia



Linear Gingival erythema



NUG



HIV associated
periodontitis

Oral Infections

HIV (Human immunodeficiency Virus and AIDS)

Diagnosis- viral culture; HIV antibodies or antigens; enzyme immunoassay (EIA) for antibodies to HIV

Treatment-

1. Nucleoside reverse transcriptase inhibitors (Zidovudine)
2. Nucleotide reverse transcriptase inhibitors (Adefovir)
3. Nonnucleoside reverse transcriptase inhibitors (Capravine)
4. Protease inhibitors (Ritonavir)
5. Fusion inhibitors (Enfuvirtide)
6. Integrase inhibitors (MK-0518 experimental 2009)
7. CCR5 inhibitors (Maraviroc)

References

- Oral and Maxillofacial pathology: Neville et al, Saunders , Elsevier 3rd edition 2009