ORAL LESIONS IN INFECTIOUS DISEASES: DIAGNOSTIC INSIGHTS INTO VIRAL, BACTERIAL, AND COVID-19-ASSOCIATED FUNGAL INFECTIONS

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ABSRACT

The oral cavity often serves as an early indicator of systemic infectious diseases, with numerous viral, bacterial, and fungal pathogens manifesting initial symptoms in the mouth. Viral infections such as herpes simplex, human papillomavirus (HPV), and COVID-19-related illnesses commonly present with oral lesions that require laboratory confirmation. Similarly, bacterial infections including syphilis, tuberculosis, gonorrhea, and scarlet fever may exhibit distinctive oral signs that aid in early diagnosis. Fungal infections like oral candidiasis and mucormycosis are particularly prevalent in immunocompromised individuals, especially following COVID-19. The strong association between oral health and systemic conditions such as cardiovascular disease, diabetes, and adverse pregnancy outcomes underscores the importance of oral examinations in clinical practice. In children, oral symptoms can serve as vital diagnostic markers. This review highlights the diagnostic value of oral manifestations in infectious diseases and explores appropriate diagnostic, therapeutic, and preventive strategies to improve patient outcomes.

KEYWORDS : COVID -19, INFECTIOUS DISEASE, Herpes Simplex Virus (HSV), Human Papilloma Virus (HPV), Mumps Viru, HIV,Epstein-Barr Virus (EBV),Varicella Zoster Virus (VZV)

INTRODUCTION

The oral cavity plays a crucial role in diagnosing infectious diseases, as many viral, bacterial, and fungal infections first appear with oral symptoms. Viral conditions like herpes simplex, HPV, and COVID-19-related illnesses often present oral lesions requiring lab confirmation. Bacterial infections such as syphilis, tuberculosis, gonorrhea, and scarlet fever can also show early signs in the mouth. Fungal infections, notably mucormycosis and oral candidiasis, are particularly concerning in immunocompromised individuals, especially post-COVID-19. Oral health is closely linked to overall health, influencing conditions like heart disease, diabetes, and pregnancy outcomes. In children, oral signs are vital diagnostic tools. This review underscores the importance of oral exams and outlines diagnostic, therapeutic, and preventive approaches to managing these infections.

VIRAL DISEASES OF THE ORAL CAVITY

Viral infections affect oral tissues via cell degeneration or immune responses.Present with blisters, ulcers; often sudden onset.Commonly linked with periodontal diseases.Early diagnosis and referral crucial due to possible tumor development.Evolving viral epidemiology demands updated knowledge. [1]

COMMON VIRUSES IN ORAL CAVITY

Herpes Simplex Virus (HSV), Human Papilloma Virus (HPV), Mumps Viru, HIV,Epstein-Barr Virus (EBV),Varicella Zoster Virus (VZV),Cytomegalovirus (CMV) [1]

HERPES SIMPLEX VIRUS (HSV)

HSV a linear dsDNA virus belonging to the Alphaherpesviridae subfamily, characterized by its icosahedral capsid and spikey envelope. It causes primary and recurrent vesicular eruptions, primarily in the orolabial and genital mucosa, and can lead to various presentations, including orolabial herpes, ocular HSV infection, and herpes encephalitis. Antiviral therapy can limit the course of infection
HSV-1 and HSV-2 affects skin, lips, oral cavity.Herpetic whitlow : HSV from salivary contamination to fingers.Severe in immunocompromised individuals . [6]

ETIOLOGY

Orolabial Herpes: Exposure to infected saliva.Herpetic Sycosis: Shaving with a razor during an activeinfection.Herpes Gladiatorum: High-contact sports.Herpetic Whitlow: Thumb-sucking, nail-biting in children, or healthcare workers.Herpes Encephalitis: Genetic mutations (TLR-3, UNC-93B).Eczema Herpeticum: Skin conditions (e.g., atopic dermatitis), filaggrin mutations, topical calcineurin inhibitors.Severe HSV: Immunocompromised states (e.g., HIV, organ transplants [6]

EPIDERMOLOGY

Approximately one-third of the world's population has experienced symptomatic HSV-1.HSV-1 is typically spread through direct contact with contaminated saliva or bodily secretions. [6]

CLINICAL FEATURES

Orolabial herpes,Herpetic sycosis,Herpes gladiatorum,Herpetic whitlow [6]

HISTOPATHOLOGY

HSV-1 replicates at the site of infection and travels to the dorsal root ganglia, where latency is established.HSV-1 evades the immune system through various mechanisms. [6]

DIAGNOSIS

Cell culture (gold standard) , Shell vial assay , PCR (more sensitive) [6]

TREATMENT

Orolabial herpes: oral valacyclovir. Eczema herpeticum: 10-14 days of acyclovir or valacyclovir.Immunocompromised patients: chronic suppression with acyclovir or valacyclovir. [6]

HUMAN PAPILLOMA VIRUS (HPV) [7]

HPV is a non-enveloped, double-stranded, circular DNA virus that causes epithelial lesions and cancers, manifesting as cutaneous and anogenital warts, with potential progression to carcinoma depending on the subtype
~200 subtypes ; oncogenic and non-oncogenic. Oral lesions : squamous papilloma, multifocal epithelial hyperplasia [6]

ETIOLOGY

Early sexual intercourse, multiple partners, smoking, oral contraceptives >5 years [6]

EPIDERMOLOGY

HPV Subtypes and Disease Manifestations Cutaneous warts: HPV 1, 2, 4, 27, 57 (hands and feet).Anogenital warts: HPV 6, 11 (low-risk).Cervical cancer: HPV 16, 18 (high-risk), also 31, 33, 35, 45, 52, 58.Oropharyngeal cancer: Associated with high-risk HPV subtypes. [6]

CLINICAL FEATURES

Cutaneous warts,Anogenital warts,Cervical dysplasia [6]

HISTOPATHOLOGY

The wart histology may reveal hyperkeratosis, papillomatosis, and parakeratosis. The long rete ridges usually point to the wart center, and the capillaries are often thrombosed [6]

DIAGNOSIS

Histopathology, immunohistochemistry ,Genotyping not routine [6]

TREATMENT

Varies by disease manifestation, including surgical removal, cryotherapy, and monitoring [6]

MUMPS VIRUS

Mumps is a contagious viral illness characterized by a prodrome of headache, fever, and malaise, followed by parotitis (inflammation of the salivary glands). It can lead to complications such as orchitis, oophoritis, and meningitis. Vaccination, typically through the measles-mumps-rubella (MMR) vaccine, has significantly reduced its incidence
RNA paramyxovirus causing epidemic parotitis.Spreads via droplets; contagious 9-10 days post swelling [8]

ETILOGY AND EPIDERMOLOGY

Virus type: Single-stranded RNA paramyxovirus.Transmission: Direct contact with respiratory droplets, saliva, and household fomites.Incubation period: 12-25 days.Contagious period: 2 days before symptoms to 5 days after onset [8]

CLINICAL FEATURES

Prodrome: Fever, malaise, headache, myalgia, and anorexia.Parotitis: Painful inflammation of the parotid gland(s).Complications: Orchitis, oophoritis, meningitis, encephalitis, pancreatitis. [8]

HISTOPATHOLOGY

Mumps virus primarily infects epithelial cells of the upper respiratory tract, then spreads to lymphoid tissues. Histologically, infected salivary glands show interstitial edema, mononuclear inflammatory cell infiltration, acinar cell destruction, and ductal epithelial cell necrosis. In orchitis, there is interstitial edema, hemorrhage,

neutrophilic and lymphocytic infiltration, and degeneration of seminiferous tubules. In the central nervous system, viral infection can cause perivascular cuffing and inflammatory infiltrates, consistent with viral meningitis or encephalitis. [8]

DIAGNOSIS

RT-PCR (buccal swab) ,Mumps IgM/IgG antibodies [8]

TREATMENT AND MANAGEMENT

Supportive care: Analgesics, cold or warm compresses.
Isolation: Droplet precautions for 5 days after onset of parotid swelling [8]

HIV

HIV (Human Immunodeficiency Virus) is a chronic illness that can lead to AIDS (Acquired Immunodeficiency Syndrome) if left untreated. With effective antiretroviral therapy (ART) and prevention strategies, HIV management aims to control the virus, improve patient outcomes, reduce transmissions, and work towards disease eradication. Clinical management involves considering socio-economic factors, patient-centered care, and interprofessional collaboration [9]

ETILOGY

Virus family: Retroviridae, Lentivirus genus.Target cells: CD4+ T-lymphocyte helper cells.Immune suppression: Continuous loss of CD4+ cells weakens the immune system.Progression: Untreated HIV can progress to AIDS, leading to opportunistic infections and death
Leads to various oral disorders due to immune suppression. Salivary gland disease, Kaposi's sarcoma, lymphomas [9]

EPIDEMIOLOGY

Pre-ExposureProphylaxis(PrEP):
Daily antiretroviral medication can reduce HIV risk by up to 86%, recommended for high-risk individuals[1].Safe Sex Practices:Using condoms and choosing lower-risk activities like oral sex lowers HIV transmission chances.Needle and Syringe Programs:Providing sterile equipment reduces HIV spread among people who inject drugs.HIV Testing and Treatment:Knowing your status and taking ART to reach an undetectable viral load prevents transmission[2].Post-Exposure Prophylaxis (PEP):PEP involves short-term HIV medication started within 72 hours of potential exposure [9]

HISTOPATHOLOGY

HIV-related lymphadenopathy shows distinct changes. Early stages reveal follicular hyperplasia with large, irregular follicles, loss of mantle zones, dominant centroblasts, and a starry-sky appearance. Follicle lysis and monocytoid B-cell hyperplasia are common.In intermediate stages (mixed hyperplasia), both follicles and interfollicular areas are highly cellular. Later, in follicular involution, follicles become small, atrophic, and hypocellular with hyalinized vessels and dendritic meshworks.Finally, lymphocyte depletion occurs, marked by loss of germinal centers, few lymphocytes, expanded interfollicular areas with histiocytes, plasma cells, immunoblasts, and fibrosis [9]

DIAGNOSIS

1st to 4th gen enzyme immunoassays (EIA) , Confirmatory : Western blot, IFA , Rapid tests : high sensitivity/specificity [9]

TREATMENT

Start ART immediately after diagnosis (unless severe OI present).First-line: INSTI (Bictegravir/Dolutegravir) + 2 NRTIs (Tenofovir + Emtricitabine/Lamivudine).Special cases:HBV: Use tenofovir.Renal issues: Avoid TDF.Pregnancy: Avoid Bictegravir, Doravirine.Opportunistic infection (OI) prophylaxis: TMP-SMX if CD4+ <200.Monitor: HIV RNA in 2–4 weeks, then 3–4 months once suppressed.Failure: Check adherence, resistance, change regimen. [9]

EPSTEIN-BARR VIRUS (EBV)

Epstein-Barr virus (EBV) is a common herpesvirus that infects most people worldwide, often without symptoms, but it can cause infectious mononucleosis and other complications in some cases.

Infects B cell ; causes mononucleosis Symptoms: pharyngitis, lymphadenopathy, fever [11]

ETIOLOGY

Epstein-Barr virus (EBV) is a double-stranded DNA herpesvirus that infects B cells and epithelial cells, becoming latent in memory B cells. It spreads mainly through saliva, but also via blood transfusions, stem cell, and organ transplants [11]

EPIDEMIOLOGY

About 95% of adults worldwide have been infected with Epstein-Barr virus. In the U.S., infection rates rise with age, from around 54% in young children to over 80% in late teens. Prevalence is higher in larger, lower-income households and varies globally, generally increasing with age. [11]

CLINICAL FEATURES

Symptoms: Fever, sore throat, malaise, lymphadenopathy, splenomegaly, prolongedfatigue.Physicalexam: Cervicallymphadenopathy,pharyngitis.Labs: Lymphocytosis with atypical lymphocytes, elevated liver enzymes. [11]

HISTOPATHOLOGY

EBV infection primarily affects lymphoid tissues, causing reactive lymphoid hyperplasia with prominent follicular and parafollicular expansion. Infected lymph nodes show a predominance of atypical lymphocytes (reactive CD8+ T cells). The spleen may exhibit similar changes, with splenomegaly and marked expansion of the white pulp. In the oropharynx, EBV can cause lymphoid tissue infiltration, leading to tonsillitis or pharyngitis. Hepatic involvement may show mild portal inflammation, with occasional hepatocellular necrosis. [11]

DIAGNOSIS

Heterophile antibody test ,Anti-EBV antibody detection ,PCR for DNA [11]

TREATMENT

Epstein-Barr virus infection is managed symptomatically with fever and pain control. Antivirals may reduce viral shedding but do not improve symptoms. Corticosteroids are reserved for cases with airway compromise or autoimmune complications. [11]

VARICELLA ZOSTER VIRUS VZV)

A highly contagious viral infection caused by the varicella-zoster virus (VZV), characterized by an itchy blistering skin rash, fever, and fatigue. It spreads through airborne droplets or direct contact and is preventable by vaccination [10]

ETIOLOGY

Chickenpox is caused by the varicella-zoster virus (VZV), a herpesvirus. It spreads through inhalation of infected droplets, causing a primary infection followed by lifelong latency in sensory nerves, with possible later reactivation as shingles. [10]

EPIDEMIOLOGY

Varicella occurs in all countries and is responsible annually for about 7000 deaths. In temperate countries, it is a common disease in children, with most cases occurring in winter and spring. In the United States, it accounts for more than 9000 hospitalizations annually. Its highest prevalence is in the 4- to 10-year-old age group. Varicella has an infection rate of 90%. Secondary cases in household contacts tend to have more severe disease than primary cases. In the tropics, varicella tends to occur in older people and may cause more serious disease. Adults get deep pockmarks and more prominent scars. [10]

CLINICAL FEATTURES

Prodrome (adults): Aches, headache, fever, malaise, oral sores.Rash progression: Starts as red spots, progressing to blisters and scabs, appearing in crops.Oral lesions: Painful ulcers, often preceding the skin rash.Complications: Secondary bacterial infections, pneumonia (adults), rare CNS complications, fetal effects during pregnancy.Contagion: Contagious 1-2 days before rash. [10]

HISTOPATHOLOGY

Lesions: Vesicular lesions with epidermal cell ballooning and intranuclear inclusions (Cowdry type A bodies).Reactivation: In herpes zoster, lesions show necrosis and multinucleated giant cells [10]

DIAGNOSIS

tzanck smear (low sensitivity) ,Direct fluorescent antibody test , Viral culture(gold standard) ,Serology for immune status [10]

TREATMENT AND MANAGEMENT

Chickenpox treatment is mainly supportive, focusing on symptom relief. Antivirals like acyclovir are recommended for adults and high-risk patients if started early. Calamine lotion, acetaminophen, and good hygiene help manage symptoms. Immunoglobulin and vaccination are used for prevention and in immunocompromised individuals. [10]

CYTOMEGALOVIRUS (CMV)

Cytomegalovirus (CMV) is a common herpesvirus (HHV-5) that is usually asymptomatic in healthy individuals but can cause severe disease in immunocompromised patients and congenital infections leading to disabilities. It remains latent after infection and can reactivate, contributing to immune dysfunction and certain cancers

Human herpesvirus type 5 ; affects immunocompromised individuals. Causes ulcerative oral lesions, salivary gland enlargement [12]

ETIOLOGY

Cytomegalovirus (CMV) is a double-stranded DNA virus from the Herpesviridae family. After primary infection, it remains dormant within the host and can reactivate when the immune system is compromised, especially during immunosuppression [12]

EPIDEMOLOGY

About 59% of individuals over six years old have been exposed to CMV, with seroprevalence increasing with age. CMV can be transmitted through blood products, breastfeeding, close contact, perinatally, and sexually. Primary infection occurs in seronegative individuals and may be asymptomatic, while reactivation or reinfection happens in seropositive individuals, particularly those who are immunocompromised, leading to significant morbidity and mortality. [12]

CLINICAL FEATURES

Immunocompetent: Often asymptomatic or mild mononucleosis-like illness (fever, fatigue, lymphadenopathy).Immunocompromised: Severe disease — pneumonitis, hepatitis, retinitis, colitis, encephalitis.Congenital CMV: Hearing loss, developmental delay, hepatosplenomegaly, jaundice, petechiae. [12]

HISTOPATHOLOGY

CMV replicates slowly in endothelial cells, expressing genes in a time-controlled sequence: early genes (0–48 hours) regulate transcription and DNA replication, while late genes produce structural proteins. Viral DNA synthesis occurs in the host nucleus within specialized replication compartments. CMV uses host RNA polymerase for gene transcription but encodes its own DNA polymerase. [12]

DIAGNOSIS

PCR , Antigenemia assays ,Serological testing Oral viral infections are diverse and impactful. Early recognition, accurate diagnosis are key.Awareness of clinical features and modern diagnostic methods is essential for optimal care. [12]

TREATMENT AND MANAGEMENT

Immunocompetent patients: Usually self-limited; symptomatic management only.Immunocompromised patients: Antivirals (ganciclovir, valganciclovir, foscarnet, cidofovir) are used for severe CMV infection or disease.Risks: Antiviral therapy carries significant toxicity; benefits must outweigh risks.Transplant patients: Prophylactic valganciclovir or ganciclovir and regular monitoring to prevent early CMV infection. [12]

BACTERIAL INFECTIONS OF THE ORAL CAVITY

Oral cavity hosts diverse, balanced microbiota.Imbalance from trauma, poor hygiene, or systemic illness leads to infection [2]

COMMON ORAL BACTERIAL INFECTIONS

Syphilis, Gonorrhea,Tuberculosis,,Scarlet Fever [2]

SYPHILIS

Syphilis is a systemic bacterial infection caused by the spirochete Treponema pallidum. It is primarily transmitted through sexual contact and progresses through four stages: primary, secondary, latent, and tertiary. The disease can affect multiple organ systems and, if left untreated, may cause long-term complications. Syphilis is highly treatable with penicillin, but its rising prevalence, especially in association with HIV, continues to pose a significant global public health challenge [13]

ETIOLOGY

Syphilis is caused by Treponema pallidum, a spiral-shaped bacterium in the Treponema genus. It is the only treponemal species that causes venereal disease, while other subspecies (e.g., T. pertenue, T. pallidum endemicum, T. carateum) cause non-venereal diseases like yaws, bejel, and pinta. Humans are the sole hosts, and syphilis is primarily transmitted through sexual contact, though rare nonsexual transmission (e.g., skin-to-skin, blood transfusion) can occur. Vertical transmission causes congenital syphilis [13]

EPIDEMOLOGY

Syphilis prevalence worldwide has increased significantly, with about 50 million cases in 2019. The highest incidence is in sub-Saharan Africa, Southeast Asia, and Latin America. In the U.S., syphilis cases nearly doubled between 2016 and 2022, with notable increases in women and congenital syphilis. The disease is particularly prevalent in low-income regions, where healthcare access is limited. Increased transmission is seen in people with multiple sexual partners, MSM (men who have sex with men), and those who inject drugs. HIV co-infection is common among syphilis patients, and risk factors for syphilis spread include substance abuse, promiscuity, and insufficient public health resources [13]

CLINICAL FEATURES

PrimarySyphilis:Solitary, painless genital chancre with tender or non-tender lymphadenopathy. Heals without scarring but progresses to secondary syphilis ifuntreated.

SecondarySyphilis:Maculopapular rash (palms/soles), condyloma lata, systemic symptoms (headache, fever, malaise). Resolves without treatment, moving to a latent phase.Tertiary Syphilis:May cause cardiovascular syphilis, neurosyphilis, or gummas in untreated cases. [13]

HISTOPATHOLOGY

Treponema pallidum:
Spirochete visible via darkfield microscopy or silver stains. Hard to culture, with slow replication.

Histologic Features:
Interstitial inflammation, endarteritis, lymphocytic infiltrates, acanthosis, gummas in tertiary syphilis.
Diagnosis can use immunohistochemistry or 16S rDNA sequencing for

confirmation. [13]

DIAGNOSIS

VDRL/RPR → FTA-ABS/TP-MHA [13]

TREATMENT

Benzathine penicillin G; follow-up for 1 year [13]

GONORRHEA

Neisseria gonorrhoeae is a sexually transmitted bacterium that primarily infects humans, causing gonorrhea. It often manifests as urethritis in men and cervicitis in women, leading to complications like pelvic inflammatory disease, infertility, and ectopic pregnancy if left untreated [14]

ETIOLOGY

Neisseria gonorrhoeae is an obligate human pathogen, meaning it requires a human host to survive and replicate. It infects the urogenital tract, but can also affect the rectum, throat, and eyes. The infection spreads through sexual contact and can lead to serious complications if undiagnosed or untreated. [14]

EPIDEMOLOGY

Gonorrhea is the second most common bacterial STI globally, with 106 million new cases annually, according to the World Health Organization (WHO). In the United States, over 500,000 cases are reported yearly, making it the second most reported STI. The disease disproportionately affects young adults, with a higher incidence among men who have sex with men (MSM). Over the past decade, the rise of antibiotic-resistant strains has led to an increase in gonorrhea cases [14]

CLINICAL FEATURES

Urethritis (Men): Painful urination, purulent discharge.Cervicitis (Women): Often asymptomatic, may cause vaginal discharge and pelvic pain.Pharyngeal and Rectal Gonorrhea: Sore throat, anal discomfort, and discharge.Ophthalmic Gonorrhea (Neonatal): Conjunctivitis in newborns.Disseminated Infection (DGI): Fever, skin lesions, and arthritis. [14]

HISTOPATHOLOGY

GramStain: Intracellular diplococci in neutrophils.Sensitivity: High for urethritis in men, lower for cervical, pharyngeal, or rectal gonorrhea [14]

DIAGNOSIS

NAATs (oral/urogenital swabs) [14]

TREATMENT

Benzathine penicillin G; follow-up for 1 year [14]

SCARLET FEVER

Scarlet fever is a syndrome caused by Streptococcus pyogenes (group A Streptococcus, GAS), characterized by a blanching, erythematous, maculopapular rash, often described as "sandpaper-like," a "strawberry tongue," and exudative pharyngitis. It typically affects school-age children but can occur at any age, particularly in crowded environments. The condition is caused by streptococcal pyrogenic exotoxins (SPEs) released during infection, leading to the characteristic rash and other symptoms [15]

ETIOLOGY

Streptococcus pyogenes (GAS) is a gram-positive bacterium responsible for infections like pharyngitis, scarlet fever, and cellulitis. It produces virulence factors such as M-protein and pyrogenic exotoxins, which cause the characteristic rash of scarlet fever. GAS can lead to invasive infections in sterile sites and is classified into Lancefield group A. Over 250 emm types exist, with some linked to severe diseases like streptococcal toxic shock syndrome (STSS) [15]

EPIDEMOLOGY

Scarlet fever, caused by Streptococcus pyogenes (GAS), typically occurs in epidemics every 5-6 years, especially affecting children. It is transmitted through respiratory droplets and skin contact. While its prevalence declined with antibiotics, recent virulent strains like emm1 have caused a resurgence globally. GAS infections are increasing, with significant morbidity and mortality, especially in crowded settings. Emergent strains like M1UK, identified in the UK, produce higher levels of scarlet fever toxins and have spread internationally. Surveillance through genome sequencing helps track outbreaks. [15]

CLINICAL FEATURES

sore throat, high fever "Strawberry tongue“,Sandpaper-like rash,White/yellow pharyngeal patches,Enlarged cervical lymph nodeloss). [15]

HISTOPATHOLOGY

Neutrophils, spongiosis, parakeratosis [15]

DIAGNOSIS

Rapid strep test + throat culture [15]

TREATMENT

Penicillin/amoxicillin; symptom management.Oral lesions can be initial signs of systemic disease. Early detection and accurate diagnosis crucial. Treat patient AND sexual partners where applicable.Maintain infection control in clinical settings [15]

MYCOTIC INFECTIONS – MUCORMYCOSIS AND ORAL CANDIDIASIS ASSOCIATED WITH COVID-19

COVID-19 has been linked with secondary fungal infections.Oral candidiasis and mucormycosis are notably prevalent. Requires interdisciplinary management and increased clinical awareness .

SECONDARY INFECTIONS IN COVID-19.ICU patients : 10x more likely to develop bacterial/fungal infections than viral ones.Immunocompromised states make oral cavity a target.Key pathogens: Candida albicans, Mucoromycetes [5]

ORAL CANDIDIASIS OVERVIEW

Candida albicans is a commensal in 40–65% of healthy adults.Becomes pathogenic in immunocompromised conditions.Associated symptoms: white patches, erythema, soreness. [5]

RISK FACTORS

Immunosuppression,Use of broad-spectrum antibiotics, corticosteroid, Poor oral hygiene, dentures, dry mouth, ICU mechanical ventilations [5]

CANDIDA PATHOGENIC MECHANISMS

Adhesion to oral mucosa and dentures. Biofilm formation on polymethylacrylate .Enzyme production (SAPs, hemolysins) → tissue invasion & immune evasion [5]

DIAGNOSIS OF ORAL CANDIDIASIS

White lesions removable by scraping → erythematous base Tests: cytology, KOH smear, cultures, PAS staining Smear example: budding yeast cells, pseudo-hyphae [5]

TREATMENT OF ORAL CANDIDIASIS

Nystatin oral suspension (100k–600k IU/ml). Clotrimazole 10 mg tablets. Fluconazole 200 mg loading, 100–200 mg/day x 7–14 days.Denture disinfection : 0.5% sodium hypochlorite for 10 mi [5]

MUCORMYCOSIS (BLACK FUNGUS)

Caused by Mucoromycetes, primarily affects diabetics, transplant patients Most common form: Rhino-orbito-cerebral mucormycosis (ROCM) Spores inhaled → sinus/pulmonary congestion [5]

CLINICAL SIGNS OF MUCORMYCOSIS

Periorbital facial pain, eyelid edema, sinusitis, tooth pain.Black lesions on palate, nasal discharge.Vision loss in severe cases CT: “reverse halo sign”MUCORMYCOSIS [5]

DIAGNOSIS

Biopsy : difficult Molecular tests : 18S PCR, cytochrome b PCR,KOH mount [5]

ORAL CAVITY CHANGES IN THE COURSE OF INFECTIOUS DISEASES DURING CHILDHOOD

The oral cavity is colonized by indigenous flora..Oral mucosa and secretions (saliva, gingival fluid) play key roles in natural immunity .Microbes can enter via food or air, sometimes replicating and releasing toxins, leading to diagnostic changes in oral tissues [4]

ENANTHEMA

ORAL MUCOSAL RASHES- Enanthema : rash on mucous membranes with various morphologies Petechiae & Ecchymoses : German measles (Forchheimer spots), infectious mononucleosis.Rosenberg’s Sign : hemorrhagic lesions on soft palate (Spotted fever). Haemorrhagic fevers (Crimean-Congo, renal syndrome) : gum bleeding, bluish color, subdental violet strip [4]

VIRAL EXANTHEMS & MUCOSAL

SIGNS-Koplik spots (Measles) : white clustered lesions opposite lower molars,Morozkin's Sign (Influenza): papules on soft palate,Herpangina & HFMD (Enteroviruses) : vesicles on soft palate → erosions; HFMD also has hand/foot lesions,Chickenpox : aphthous ulcers on soft/hard palate, gingiva, tongue,Smallpox : papules → vesicles → ulcers (now eradicated but of historical importance) [4]

SEVERE INFECTIONS

Cancrum oris (Noma) : gangrenous infection in malnourished children; starts as necrotic violet gingival papule → facial osteolysis.Impetigo contagiosa: phlyctena on mucosa, painful erosions; often mistaken for other infection.Duguet's ulcers (Abdominal fever): erosions on tonsils and palatal arches [4]

ORAL MANIFESTATIONS OF SYSTEMIC INFECTIONS

Furred Tongue : Febrile illnesses, dehydration, GI infections.Strawberry Tongue : Scarlet fever – initially white coated, later red with swollen papillae. Brown Tongue: Later stage of abdominal fever.. Pestis (Plague): thick white furred tongue, "chalky" appearance. Govorov-Gordelie's Sign: tongue paralysis in spotted fever. [4]

HIV/AIDS-RELATED ORAL CHANGES

Hairy Leukoplakia (EBV-related): whitish, corrugated patches on tongue edges – can't be scraped off. HIV-Associated Gingivitis: red, raw gingiva with petechiae. HIV-Associated Periodontitis: interseptal destruction, rapid gingival recession, necrosis. [4]

STOMATITIS

TYPES AND ETIOLOGIES

Catarrhal : mild, linked to viral infections (measles, influenza).Epidemic (Aphthous fever) : zoonotic, from contaminated milk; multiple painful aphthae with yellowish exudate.HSV Stomatitis (Aphthous enanthema): affects kids <7 yrs, vesicles on gingiva, not tonsils; low-grade fever [4]

DIAGNOSTIC POINTS

Oral lesions often precede or accompany systemic symptoms Inspection of oral mucosa provides vital clues for early diagnosis. Recognizing specific signs (e.g., Koplik, Morozkin, Rosenberg, strawberry tongue) can aid in prompt treatment [4]

ORAL INFECTIONS AND SYSTEMIC DISEASE—AN EMERGING PROBLEM IN MEDICINE

Advances in oral health → more natural teeth retained. Persistent issues : restored teeth, periodontal disease.Oral cavity : a reservoir of infection/inflammation. Importance to general health and systemic disease

THE ORAL MICROBIOTA

Rich and unique microbiota. Dominant flora in health : Viridans streptococci. Infection related Prevotella, Fusobacterium (β-lactamas producers)A.actinomycetemcomitans,P.gingivalis(periodontal pathogens) Candida albicans & non-albicans species [3]

ODONTOGENIC INFECTIONS

Confined by anatomical barriers but can spread dangerously. Serious cases: Lemierre's syndrome (Fusobacterium necrophorum).Importance of dental procedures alongside antibiotics.Association with conditions like endocarditis and septicaemia [3]

ORAL HEALTH AND SYSTEMIC INFECTIONS

Infections may remain asymptomatic.Procedures (even brushing) can cause bacteraemia.Oral infections as potential indicators of systemic conditions

(e.g., HIV) [3]

MEDICALLY COMPROMISED PATIENTS

Immunosuppressive therapy and cancer treatments raise risk.Even minor oral infections can become life-threatening.Essential measures : Pre-treatment oral exam & imaging .Eliminate foci of infection [3]

ORAL COMPLICATIONS DURING CANCER THERAPY

Stomatitis → delays in treatment, increased costs, Pain limits hygiene → increased infection risk,Use of chlorhexidine and antimicrobial rinses recommended, Critical role of medicodental collaboration [3]

OSTEONECROSIS & RARE INFECTION

Bisphosphonate-related osteonecrosis of the jaw. Actinomycosis : mimics malignancy, spreads across tissues.Risk factors :

carious teeth, trauma, dental work) [3]

PERIODONTAL DISEASE & SYSTEMIC IMPACT

Chronic periodontitis → risk for CVD (atherosclerosis, stroke).Over 50 studies: modest but significant association. Inflammatory biomarkers: IL-6, CRP, TNF-α, MMP-9 PATHOGENIC MECHANISMS LINKING [3]

ORAL AND CV DISEASE

Molecular mimicry: GroEL ↔ hHSP60.Direct invasion : Periodontal pathogens damage endothelium.Immune responses, foam cell formation, matrix degradation [3]

PREVENTING ORAL INFECTIONS

Regular plaque control essential.Antibacterials in oral care products ,Chlorhexidine: Effective but has side effect, Triclosan : Dual action, better for long-term use Especially useful in susceptible populations [3]

CLINICAL RECOMMENDATIONS

Consider oral health in systemic infections.Pre-treatment dental care for immunocompromised patients.Use of antimicrobial rinses during vulnerable periodsEmphasis on interdisciplinary collaboration.Chronic oral infections are a hidden systemic burden. Oral health is critical for total patient well-being .Integration of oral care into general medical management is vital [3]

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