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ORAL MEDICINE

# HIV ORAL MANIFESTATIONS

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

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# MIND MAP

# ORAL MANIFESTATIONS OF HIV

## Oral Health Risks in HIV Patients

- Smokers with HIV are at higher risk of oral diseases including opportunistic infections, periodontal disease, and oral cancers.
- HIV-related salivary gland hypofunction increases oral candidiasis risk.

## Late-Stage HIV Oral Conditions

- Kaposi sarcoma and oral hairy leukoplakia are associated with advanced HIV.
- Management should involve HIV and oral medicine specialists.

## HIV Transmission and Saliva

- Transmission mainly via bodily fluids: unprotected sex, needle sharing, vertical transmission.
- Saliva contains HIV but is not a significant transmission risk due to low viral levels and antiviral factors.

## Categories of Oral Manifestations in HIV

- Microbiological infections: fungal, bacterial, viral.
- Oral neoplasms, neurological conditions, other HIV-associated oral diseases, and treatment-related conditions.

## Bacterial Infections and Periodontal Disease

- HIV patients experience linear gingival erythema, necrotizing periodontal diseases, and accelerated chronic periodontitis.
- Necrotizing ulcerative gingivitis and periodontitis cause pain, bleeding, and tissue necrosis.
- Treatment includes plaque removal, oral hygiene, smoking cessation, and sometimes antibiotics like metronidazole.

## Treatment and Referral Guidelines

- Antifungals and antivirals require careful use due to drug interactions.
- Dentists should refer complex cases to specialists or HIV clinics.
- Management should be multidisciplinary, involving dental and medical professionals.

## Preventive and Supportive Care

- Emphasize excellent oral hygiene and smoking cessation.
- Regular dental check-ups are important for early detection and management of oral lesions.

## Antiretroviral Drug Interactions and Management

- Antiretroviral drugs interact with many common medications; consult HIV experts before prescribing.
- Rare adverse reactions like perioral paraesthesia can occur.

## Key Oral Manifestations of HIV

- Opportunistic infections: candidiasis, oral hairy leukoplakia.
- Other signs: recurrent aphthous stomatitis, oral mucosa hyperpigmentation, intramucosal hemorrhages.

## Role of Dentists in HIV Diagnosis and Care

- Dentists can identify and manage HIV-associated oral lesions.
- Early diagnosis through oral signs improves patient outcomes and reduces transmission.

## Oral Lesions Across HIV Stages

- Oral lesions can appear at any stage but are not exclusive to HIV infection.
- Differential diagnosis is important as similar lesions may occur in immunocompetent individuals.

## Fungal Infections in HIV

- Candida albicans causes most oral fungal infections; oral candidiasis affects 90% of advanced cases.
- cART reduces candidiasis prevalence significantly.
- Types: pseudomembranous, erythematous, angular cheilitis, chronic hyperplastic candidiasis.

## Viral Infections and Co-infections with HIV

- Common viruses: HSV 1 and 2, VZV, CMV, HPV, EBV, MCV2, HHV8.
- HSV causes cold sores and oral ulcers; recurrent infections are common in HIV.
- VZV causes chickenpox and shingles; oral lesions may be painful and require antiviral treatment.

## Summary of Diagnostic Approaches

- Diagnosis mainly clinical; swabs, PCR, microscopy, culture, or biopsy may be needed for uncertain cases.
- Early identification through oral signs is crucial for effective HIV management.

## Legal and Clinical Responsibilities of Dentists

- Dentists must stay informed about HIV-related oral health issues.
- Understanding clinical and legal implications supports better patient care and safety.

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# MIND MAP

# ORAL AND HIV-RELATED VIRAL INFECTIONS: HPV AN D EBV OVERVIEW

## Common Oral HPV Lesions

- Squamous papilloma: most common, protruding growth with finger-like projections.
- Condyloma acuminata and verruca vulgaris can mimic squamous papilloma clinically.

## Oncogenic HPV and Oral Cancers

- Certain HPV types cause squamous cell carcinoma, especially in posterior tongue, tonsils, oropharynx.
- These HPV-related cancers are distinct from tobacco- and alcohol-related oral cancers.

## Benign HPV-Induced Mucocutaneous Lesions

- Includes verruca vulgaris, condyloma acuminatum, focal epithelial hyperplasia (Heck's disease), and squamous papilloma.
- Lesions vary in appearance: cauliflower-like, sessile or pedunculated, pink to white surfaces.

## Oral Hairy Leukoplakia (OHL) Characteristics

- Whitish, elevated, non-removable patches with vertical ridges, mostly on lateral tongue borders.
- OHL incidence increases as CD4 counts decline, often below 150 cells/µL.

## HIV Treatment-Related Oral Conditions

- Antiretroviral drugs can cause xerostomia, taste disturbances, lip cracking, oral ulceration, and pigmentation changes.
- Drug interactions with CART necessitate careful prescribing and medical consultation.

## Summary Mind Map Outline



## Human Papilloma Virus (HPV) Overview

- HPV has many subtypes causing a variety of oral mucosa lesions.
- Transmission primarily occurs through direct contact with lesions.

## HPV Transmission and Clinical Presentation

- Sexually transmitted HPV infections cause oral condyloma acuminata.
- Verruca vulgaris (common wart) can appear anywhere in the oral cavity, often labial mucosa.

## Management and Referral for HPV Lesions

- Suspected HPV oral lesions require biopsy and specialist referral.
- Proper documentation and follow-up are essential for patient care and legal compliance.

## Epstein-Barr Virus (EBV) and Oral Manifestations

- EBV linked to infectious mononucleosis, Burkitt's lymphoma, non-Hodgkin's lymphoma, nasopharyngeal carcinoma.
- Oral hairy leukoplakia (OHL) is a hallmark EBV lesion in HIV-infected patients.

## HIV-Associated Oral Neoplasms

- Kaposi's sarcoma (KS) and non-Hodgkin's lymphoma (NHL) are common malignancies with oral involvement.
- KS lesions are vascular, red-purple plaques or nodules, most commonly on hardpalate.

## Algorithm for Managing Oral HIV Manifestations

- Treatment should be coordinated with medical practitioners and specialists.
- Patient confidentiality is paramount, but rare legal exceptions exist to ensure follow-up.

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## Question 1

# What should be done before prescribing any drug to a patient taking antiretroviral therapy (ART)?

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## Answer 1

**Consult an HIV expert before prescribing any drug to a patient taking antiretroviral therapy due to potential interactions with many commonly prescribed drugs.**

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## Question 2

**Which oral diseases are patients with HIV, particularly smokers, at increased risk for?**

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## Answer 2

**They are at increased risk of opportunistic infections, periodontal disease, necrotising periodontitis, oral hairy leukoplakia, and oral squamous cell carcinoma.**

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## Question 3

# What is the significance of salivary gland hypofunction in HIV patients?

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# Answer 3

**HIV-related salivary gland hypofunction increases the risk of oral candidiasis.**

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## Question 4

# How is HIV transmitted, and what is the dominant mode of transmission in Australia?

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# HIV ORAL MANIFESTATIONS

## Answer 4

**HIV is transmitted by exposure to infected bodily fluids or tissues via unprotected sex, re-use of drug-injecting equipment, and vertical transmission from mother to child. In Australia, male-to-male sex remains the dominant mode of transmission.**

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## Question 5

# Is HIV transmission considered a risk through saliva, tears, sweat, urine, or feces?

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# Answer 5

**No, HIV is present in saliva but is not a risk factor for transmission due to low virus levels and antiviral factors. There is no evidence that HIV can be transmitted by contact with tears, sweat, urine, or feces.**

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## Question 6

# What are the five categories of oral manifestations of HIV infection?

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# HIV ORAL MANIFESTATIONS

## Answer 6

**The five categories are: microbiological infections (fungal, bacterial, viral), oral neoplasms, neurological conditions, other oral conditions associated with HIV infection, and oral conditions associated with HIV treatment.**

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## Question 7

# What are the classic forms of oral candidiasis seen in HIV patients?

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## Answer 7

**The classic forms include pseudomembranous candidiasis, erythematous candidiasis, angular cheilitis, and chronic hyperplastic candidiasis.**

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## Question 8

# What are the main bacterial periodontal manifestations associated with HIV infection?

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# Answer 8

They include linear gingival erythema, necrotizing periodontal diseases (necrotizing ulcerative gingivitis, periodontitis, stomatitis), and accelerated progression of chronic periodontitis.

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## Question 9

# Which viruses commonly cause oral lesions in patients co-infected with HIV?

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# Answer 9

**Herpes simplex virus (HSV 1 and 2), varicella zoster virus (VZV), cytomegalovirus (CMV), human papilloma virus (HPV), Epstein-Barr virus (EBV), molluscum contagiosum virus 2 (MCV2), and human herpesvirus 8 (HHV8).**

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## Question 10

**What are the two common oral malignancies associated with HIV infection?**

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# Answer 10

**Kaposi’s sarcoma (KS) and non-Hodgkin’s lymphoma (NHL) are the two common malignancies with oral involvement in HIV infection.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

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# MIND MAP

# TUBERCULOSIS AND DENTAL MANAGEMENT

## Tuberculosis Overview

- Caused by Mycobacterium tuberculosis infection
- Transmitted via inhalation of airborne droplets containing viable bacteria

## Oral Health Risks in HIV Patients

- Spread through droplet nuclei from coughing, sneezing, speaking, or singing
- Droplet nuclei can remain airborne for hours, infecting susceptible individuals

## Active Tuberculosis Disease

- Symptoms include persistent cough, night sweats, fever, weight loss, chest pain
- Only active TB patients can transmit the disease to others

## TB Skin Test Details

- Injection of tuberculin under forearm skin, checked after 2-3 days
- Care instructions include avoiding rubbing or bandaging the site
- Positive test indicates likely TB infection, requires further medical evaluation

## Dental Management of TB Patients

- Defer dental treatment until patient is non-infectious or quarantine complete
- Emergency care only if necessary, with strict infection control precautions
- Use antimicrobial pre-procedure rinses and high-filtration masks (N95/N99)
- Employ barriers, dental dams, and high-velocity evacuation to reduce aerosols

## Managing TB in HIV-Positive Patients

- HIV patients are at higher risk of TB infection and reactivation
- TB may present with oral ulcers and lymphadenopathy in immunocompromised patients
- Postpone dental care if active disease is suspected; refer promptly for medical management

## Post-Treatment Follow-Up

- TB treatment requires at least 18 months with regular physician follow-up
- Follow-up includes chest X-rays, sputum cultures, and symptom review annually.

## Importance of Awareness and Training

- Dental health personnel should be trained in TB infection control protocols
- Incorporate TB control measures into dental office written infection control programs.

## Latent Tuberculosis Infection

- Bacteria can live dormant without symptoms for years (latent TB)
- Latent TB is not infectious but can develop into active disease if untreated

## Tuberculosis Testing Methods

- TB blood test (Interferon Gamma Release Assay) is more accurate than skin test.
- TB skin test (Mantoux) detects latent TB via skin reaction measurement
- Chest X-rays and sputum culture help diagnose active TB
- Drug resistance and molecular tests assist in tailored treatment

## Implications of BCG Vaccine on Testing

- BCG vaccine may cause false-positive TB skin test but does not prevent infection.
- Provides some protection in children but wanes with age

## Environmental and Infection Control Measures

- Use airborne infection isolation rooms for urgent dental care in infectious TB
- Employ HEPA filters or ultraviolet germicidal irradiation in high-risk settings
- Double surface cleaning after treatment for active TB patients

## Summary of Key Infection Control Points

- Only active TB cases are infectious; latent TB patients can be treated with standard precautions.
- Standard surgical masks are insufficient; use properly fitted respirators when treating active TB.
- Dental treatment for active TB should be in facilities equipped for airborne infection isolation
- Consult physician before treating patients who completed TB therapy for infectivity clearance

## Patient Screening and Referral Recommendations

- Screen dental patients for TB symptoms and history of exposure
- Refer symptomatic or suspected active TB patients for medical evaluation immediately
- Ensure patients wear masks and are separated from others while awaiting care.

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 1

**What causes tuberculosis and how is the infection transmitted?**

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## Answer 1

**Tuberculosis is caused by infection with Mycobacterium tuberculosis (M. tuberculosis). The infection is transmitted through inhalation of airborne droplets called “droplet nuclei” containing viable M. tuberculosis, which are generated when a person with active pulmonary or laryngeal tuberculosis sneezes, coughs, speaks, or sings.**

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## Question 2

**Can people with latent tuberculosis infection spread the disease to others?**

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## Answer 2

**No, people with latent tuberculosis infection are generally asymptomatic and not infectious to others. Only individuals with active tuberculosis disease can transmit the infection.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 3

**What are common symptoms of active tuberculosis?**

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## Answer 3

**Common symptoms of active tuberculosis include a persistent productive cough, night sweats, fever, weakness or fatigue, weight loss, chest pain, bloody sputum, hoarseness, and persistent oral mucosa lesions that do not respond to therapy.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 4

**What tests are used to diagnose tuberculosis?**

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## Answer 4

**Tests for tuberculosis include the TB blood test (Interferon Gamma Release Assay), TB skin test (Mantoux), chest x-rays, sputum smear and culture, drug resistance tests, acid-fast microscopy, molecular assays, ultrasound or CT scans, and biopsy.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 5

**What precautions should dental health care personnel take for patients with active tuberculosis?**

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## Answer 5

**Dental health care personnel should defer dental treatment for patients with active tuberculosis until they are no longer infectious. If urgent treatment is necessary, transmission-based precautions should be used, including scheduling the patient last, using antimicrobial mouth rinses, wearing fitted high-filtration masks or respirators (N95/N99), using dental dams and high-velocity evacuation, and performing double surface cleaning.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 6

**Are standard surgical masks effective in preventing tuberculosis transmission in dental settings?**

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## Answer 6

**No, standard surgical masks are not adequate to protect against tuberculosis transmission. Proper respiratory protection such as fitted, disposable N95 respirators is necessary when treating patients with active tuberculosis.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 7

**How should patients with latent tuberculosis be managed in dental offices?**

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## Answer 7

**Patients with latent tuberculosis may be treated in the dental office using standard infection control precautions since they are not infectious.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 8

**What environmental controls are recommended when providing dental care for patients with suspected or confirmed active tuberculosis?**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Answer 8

**Use of airborne infection isolation rooms is recommended for urgent dental treatment of patients with suspected or confirmed active tuberculosis. In high-volume settings, high-efficiency particulate air (HEPA) filters or ultraviolet germicidal irradiation should be used.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 9

**How is tuberculosis managed in patients with HIV infection?**

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## Answer 9

**People with HIV infection can develop tuberculosis through primary infection, reactivation of latent infection, or reinfection. TB may present atypically and is more severe with lower CD4 cell counts. Dental treatment should be postponed if the disease is active, with referral for medical management and appropriate infection control precautions.**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Question 10

**What is the purpose of the TB skin test (Mantoux test), and how is it performed?**

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# TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

## Answer 10

**The TB skin test is used to detect latent TB infection. A small amount of tuberculin fluid is injected under the skin of the forearm. After 2 to 3 days, the injection site is checked for a raised bump, which indicates a positive reaction and possible TB infection.**

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# BURNING MOUTH SYNDROME

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

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# MIND MAP

# BURNING MOUTH SYNDROME (BMS)

## Definition and Overview

- BMS is characterized by burning pain in the tongue or oral mucous membrane with normal clinical signs and lab results lasting 4-6 months.
- Diagnosis is challenging as it requires excluding other known causes of oral burning.

## Local Factors

- Dry mouth (xerostomia and hyposalivation) can cause oral burning; 25% of BMS patients report dry mouth.
- Taste alterations such as bitter, metallic, or mixed tastes are common in BMS patients.

## Oral Mucosal Diseases

- Conditions like lichen planus, benign migratory glossitis, and fissured tongue may cause burning pain but usually show visible mucosal changes.
- BMS patients typically have a normal-looking oral mucosa despite symptoms.

## Oral Galvanism and Denture Issues

- Oral galvanism from metal restorations or prostheses is a rare cause of BMS.
- Poorly designed dentures affecting tongue space or occlusion may contribute to symptoms in some patients.

## Systemic Factors

- Hematinic deficiencies (iron, B vitamins, folate, zinc) are linked to oral burning and should be ruled out.
- Autoimmune diseases (Sjogren's syndrome, lupus) cause dryness and increase infection risk.
- Gastroesophageal reflux disease (GERD) may cause oral burning.
- Endocrine disorders like diabetes, thyroid issues, and menopause are associated with BMS symptoms.

## Central Nervous System Disorders

- Neurological diseases (Parkinson's, multiple sclerosis, trigeminal neuralgia) may cause neuropathic burning pain in the mouth.
- Dopaminergic pathway involvement is suggested due to higher BMS prevalence in Parkinson's patients.

## Differences Between Primary and Secondary BMS

- Primary BMS has no identifiable cause; secondary BMS is linked to local, systemic, or psychological factors.
- Diferentiating both types is complex but essential for appropriate management.

## Diagnosis

- Requires detailed medical, dental, and medication history.
- Exclusion of local causes (infections, mucocutaneous diseases), systemic diseases, hypersensitivity, and drug effects is critical.
- Skin patch testing rarely needed but useful in suspected allergies.

## Takeaway Points

- BMS is a diagnosis of exclusion involving chronic oral burning without visible signs.
- More common in women over 70; onset may be sudden or gradual.
- Understanding and individualized management improve patient outcomes, though BMS remains underdiagnosed and poorly managed.

## Oral Infections

- Oral candidiasis is frequently implicated but hard to distinguish from BMS due to overlapping symptoms.
- Bacterial and viral infections (e.g., spirochetes, herpes viruses) have also been suggested as possible causes.

## Oral Parafunction

- Habits such as clenching, bruxism, tongue posturing, and lip licking have been proposed but lack strong evidence as causes of BMS.

## Allergic Reactions

- Allergies to dental materials (e.g., metals, acrylics) and food additives can cause oral burning but are rarely implicated in BMS.
- Patch testing can help identify allergies; avoidance may improve symptoms.

## Medication Side Effects

- Drugs causing hyposalivation (e.g., tricyclic antidepressants) and ACE inhibitors (captopril, enalapril, lisinopril) are linked to oral burning.

## Idiopathic and Psychosocial Factors

- BMS may be part of idiopathic focal pain syndromes like vulvodynia.
- High levels of psychological disturbances (depression, anxiety, somatization) are common but may be cause or effect of BMS.
- Psychological treatments and medication side effects can influence oral symptoms.

## Clinical Features and Impact

- Burning sensation often mild in the morning, worsening throughout the day.
- Symptoms can range from minor discomfort to severe pain afecting daily activities; extreme cases may involve suicidal tendencies.
- Additional signs include parafunctional habits, dry mouth, halitosis, and dysgeusia (metallic taste).

## Management

- Patient education and counseling about BMS as a chronic neuropathic pain condition is fundamental.
- Lifestyle modifications like relaxation therapy, exercise, and stress management can help.
- Pharmacological treatments include topical/systemic psychotropic drugs such as tricyclic antidepressants, antiepileptics, and clonazepam, often requiring specialist care.

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BURNING MOUTH SYNDROME

## Question 1

# What is Burning Mouth Syndrome (BMS) and how is it characterized?

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# BURNING MOUTH SYNDROME

## Answer 1

**Burning Mouth Syndrome (BMS) is an oral sensory disorder characterized by burning pain in the tongue or other oral mucous membranes. It is often associated with symptoms such as subjective dryness of the mouth, paraesthesia, and altered taste, with no identifiable medical or dental cause.**

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BURNING MOUTH SYNDROME

## Question 2

**How does the International Association for the Study of Pain define Burning Mouth Syndrome?**

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# BURNING MOUTH SYNDROME

## Answer 2

**The International Association for the Study of Pain defines Burning Mouth Syndrome as burning pain in the tongue or other oral mucous membrane, associated with normal clinical signs and laboratory findings, lasting at least four to six months.**

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BURNING MOUTH SYNDROME

## Question 3

# What is the difference between xerostomia and hyposalivation in the context of BMS?

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# BURNING MOUTH SYNDROME

## Answer 3

**Xerostomia is the subjective feeling of having a dry mouth, whereas hyposalivation is an objective reduction in saliva production, also called salivary gland hypofunction, measured by salivary flow rates.**

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BURNING MOUTH SYNDROME

## Question 4

# What taste alterations are commonly reported by patients with Burning Mouth Syndrome?

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# BURNING MOUTH SYNDROME

## Answer 4

**Patients with BMS often report altered taste perception (dysgeusia), including persistent bitter (33%), metallic (27%), or combined tastes (10%), which may decrease after rinsing with distilled and deionized water.**

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BURNING MOUTH SYNDROME

## Question 5

# Which oral infections must be excluded before diagnosing Burning Mouth Syndrome?

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# BURNING MOUTH SYNDROME

## Answer 5

**Oral candidiasis and other fungal infections, bacterial infections (such as spirochetes, fusiform, Enterobacter, Klebsiella species), and viral infections like herpes viruses must be ruled out as causes of oral burning before diagnosing BMS.**

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BURNING MOUTH SYNDROME

## Question 6

# What oral mucosal diseases can cause burning sensations and how do they differ from BMS?

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# BURNING MOUTH SYNDROME

## Answer 6

**Diseases like lichen planus, benign migratory glossitis, hairy tongue, and fissured tongue can cause burning sensations, but they have visible clinical signs such as erythema, ulceration, or depapillation, whereas BMS patients typically have normal-appearing oral mucosa.**

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BURNING MOUTH SYNDROME

## Question 7

# Are parafunctional oral habits considered a cause of Burning Mouth Syndrome?

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# BURNING MOUTH SYNDROME

## Answer 7

**Although parafunctional habits such as clenching, bruxism, tongue posturing, and lip trappings have been proposed as causes of BMS, current studies do not support that these habits cause Burning Mouth Syndrome.**

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BURNING MOUTH SYNDROME

## Question 8

**List some systemic factors that can contribute to oral burning symptoms similar to those in BMS.**

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# BURNING MOUTH SYNDROME

## Answer 8

Systemic factors include haematinic disorders (e.g., vitamin B, iron, folate deficiencies), autoimmune connective tissue diseases (e.g., Sjogren's syndrome, systemic lupus erythematosus), gastroesophageal reflux disease, endocrine disorders (e.g., diabetes, thyroid disorders, menopause), medication side effects, and central nervous system disorders (e.g., Parkinson's disease, multiple sclerosis).

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BURNING MOUTH SYNDROME

## Question 9

# What role do psychosocial factors play in Burning Mouth Syndrome?

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# BURNING MOUTH SYNDROME

## Answer 9

**Psychological disturbances such as depression, anxiety, somatization, and personality disorders are common in BMS patients and may contribute to the cause, intensity, or urgency of symptoms. These disturbances may be either a cause or a consequence of the chronic pain experienced in BMS.**

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BURNING MOUTH SYNDROME

## Question 10

# What are the key components in the diagnosis and management of Burning Mouth Syndrome?

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# BURNING MOUTH SYNDROME

## Answer 10

**Diagnosis of BMS is by exclusion of other causes of oral burning through detailed clinical history, examination, and ruling out local, systemic, drug-related, or hypersensitivity causes. Management focuses on patient education, lifestyle changes to reduce stress, and pharmacological treatments such as topical or systemic psychotropic drugs (e.g., tricyclic antidepressants, antiepileptic drugs, clonazepam), often requiring specialist referral.**

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# PREECLAMPSIA AND PREGNANCY-ORAL MANIFESTATIONS AND MANAGEMENT

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

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# MIND MAP

# PRE-ECLAMPSIA AND PREGNANCY - ORAL MANIFESTATI ON AND MANAGEMENT

## Periodontitis and Risk Factors

- Smoking (cigarettes/cannabis), stress, diabetes, and high plaque levels increase risk
- Periodontitis is initiated by microbial dental plaque and depends on individual immune response

## Dental Erosion from Vomiting

- Nausea and vomiting cause erosive damage early pregnancy
- Advising pregnant women to rinse with milk or water after vomiting and avoid immediate brushing protects tooth enamel

## Prevention of Oral Disease in Pregnancy

- Recommend fluoridated water, twice daily use of fluoridated toothpaste, and low-sugar diet
- Regular dental visits advised to manage caries and periodontal conditions, with emphasis on plaque control and smoking cessation.

## Periodontitis and Adverse Pregnancy Outcomes (APO)

- Periodontal inflammation can spread systemically, increasing risk of pre-eclampsia, especially in lower-middle-income countries
- Periodontitis independently linked to preterm birth, low birth weight, gestational diabetes, pre-eclampsia, and fetal growth restriction

## Pathogenic Mechanisms Linking Periodontitis and APO

- Direct mechanism: oral bacteria disseminate to feto-placental unit causing infection
- Indirect mechanism: inflammatory mediators from periodontal tissue induce systemic immune response affecting pregnancy
- Experimental studies show periodontal bacteria can damage trophoblast cells morphologically and functionally

## Timing and Effectiveness of Periodontal Treatment

- Placental structure completes in first trimester; dental care during pregnancy may be too late to fully prevent APO
- Although dental treatment cures periodontal disease, its efect on reducing APO
- incidence is limited once pregnancy progresses

## Pregnancy, Parity, and Periodontal Disease

- Parity and socioeconomic status are theorized to influence periodontal disease prevalence and severity
- Models propose complex interactions between pregnancy history, oral health, and social factors impacting periodontal outcomes

## Common Oral Problems in Pregnancy

- Increased risk of caries due to cravings for sugary foods and frequent consumption of carbonated drinks to alleviate nausea
- Gingivitis typically begins in the 2nd month and peaks by the 8th month of pregnancy, causing gingival swelling and increased bleeding on probing

## Pyogenic Granuloma (Pregnancy Tumour)

- Localized gingival enlargement occurring in up to 5% of pregnant women, highly vascular and bleeds easily.
- Smaller lesions may regress with improved oral hygiene; excision possible if symptomatic but risk of recurrence exists.

## Oral Health and General Pregnancy Outcomes

- Poor oral health linked to preterm birth, low birth weight, gestational diabetes, pre-eclampsia, and fetal growth restriction
- A Emerging evidence highlights oral-systemic health connection during pregnancy

## Dental Treatment During Pregnancy

- Dental care between 13-21 weeks gestation safe, including scaling, root planing, and emergency restorative or surgical treatment.
- Avoid dental amalgam placement/replacement especially during the first trimester as a precaution
- Routine treatment best à second trimester; emergency care possible anytime with precautions.

## Pre-eclampsia Overview and Oral Health Links

- Pre-eclampsia is pregnancy-related hypertension with proteinuria after 20 weeks, leading to maternal and perinatal morbidity/mortality
- Severe or progressive periodontal disease during pregnancy increases risk, potentially via placental inflammation from periodontal pathogens like P. gingivalis and F. nucleatum

## Clinical and Socioeconomic Implications

- Non-surgical periodontal therapy can reduce medical costs for pregnant women by over 70%
- Access to oral healthcare is crucial, especially for pregnant women in low socioeconomic areas, to potentially lower risk of pre-eclampsia and other complications

## Role of Diet and Microbiota in Pregnancy Outcomes

- High-fiber diets promote beneficial gut microbiota producing short-chain fatty acids, reducing risk of pre-eclampsia.
- Such diets also linked to slower progression of periodontal disease, suggesting dietary intake may influence the periodontal-preeclampsia relationship.

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# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 1

**What common oral problems are associated with pregnancy?**

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# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 1

**Common oral problems in pregnancy include caries, gingivitis, periodontitis, pyogenic granuloma (pregnancy tumour), and erosion.**

WINSPERT
CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 2

**How does pregnancy affect the risk of dental caries?**

WINSPERT
CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 2

**Pregnancy can increase the risk of dental caries due to cravings for sugary foods and frequent consumption of carbonated drinks to alleviate nausea.**

WINSPERT
CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 3

**When does gingivitis typically begin and peak during pregnancy?**

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# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 3

**Gingivitis typically begins in the 2nd month of pregnancy, increases up to the 8th month, and then declines.**

WINSPERT
CUE CARDS

# PREECLAMPSIA AND PREGNANCY-ORAL MANIFESTATIONS AND MANAGEMENT

## Question 4

**What are the main risk factors for developing periodontitis?**

WINSPERT
CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 4

**Risk factors for periodontitis include cigarette or cannabis smoking, age, stress, diabetes mellitus, and high plaque levels.**

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# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 5

**What is a pyogenic granuloma and how is it managed during pregnancy?**

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CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 5

**A pyogenic granuloma is a localized gingival inflammatory enlargement (pregnancy tumour) that bleeds easily and may be painful. Small lesions may regress with improved oral hygiene, but larger or uncomfortable lesions can be excised if no medical contraindications exist, although recurrence risk remains.**

WINSPERT
CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 6

**What advice should be given to pregnant women to prevent enamel erosion caused by vomiting?**

WINSPERT
CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 6

**Pregnant women should rinse with milk or water after vomiting and avoid brushing their teeth immediately to prevent enamel erosion.**

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CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 7

**What preventive oral health measures are recommended during pregnancy?**

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# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 7

**Preventive measures include drinking fluoridated water, using fluoridated toothpaste twice daily, maintaining a low-sugar diet, regular dental visits, meticulous plaque control, and smoking cessation.**

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CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 8

**Is dental treatment safe during pregnancy, and when is it best performed?**

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# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 8

**Dental treatment is generally safe during pregnancy, especially in the second trimester. Emergency treatment can be done at any time with precautions. Early pregnancy treatments are often avoided due to concerns about miscarriage, and third trimester treatments may be uncomfortable due to positioning.**

WINSPERT
CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 9

**What is the relationship between periodontitis and pre-eclampsia?**

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CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 9

**Periodontitis is a significant risk factor for pre-eclampsia, potentially through systemic spread of localized periodontal inflammation and placental infection by periodontal pathogens.**

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# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Question 10

**Why might dental care during pregnancy have limited impact on preventing adverse pregnancy outcomes (APO)?**

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CUE CARDS

# PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

## Answer 10

**Because placental structure is completed in the first trimester, dental care during pregnancy may occur too late to influence the early development processes related to APO, despite being effective in treating periodontal disease.**

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ORAL MEDICINE

# DRY MOUTH

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# ORAL COMPLICATIONS OF SJÖGREN'S SYNDROME (SS)

## Decreased Salivary Flow Effects

- Reduced salivary mucins cause less lubrication of gingiva and mucosa
- Results in oral mucosal inflammation, mucosal sloughing, erythematous mucosa, and traumatic ulcers

## Gingival Inflammation and Recession

- Decreased saliva causes poor clearance of food and plaque, triggering gingival edema and inflammation
- Gingival recession may occur due to poor lubrication and chronic inflammation

## Increased Risk of Opportunistic Infections: Candida

- High prevalence of candidiasis (over 68% in SS patients vs 23–68% general population)
- Presents as atrophic or erythematous candidiasis with burning mouth sensation reported by one-third of patients

## Management and Treatment Options

- Individualized treatment plans are essential for symptom control and disease prevention
- Regular dental exams and preventive fluoride regimens recommended
- Candida managed with topical antifungals
- Pilocarpine and cevimeline improve saliva and tear flow
- Avoid anticholinergic drugs when possible
- Multidisciplinary cooperation between medical and dental professionals is vital

## Dry Mouth in Palliative Care

- Common due to anxiety-induced mouth breathing and medications
- Leads to increased dental decay, periodontal disease, mucosal disease, candidiasis, and denture retention problems
- Causes difficulty in chewing, swallowing, and speech.

## Progressive Nature of SS

- SS is a progressive disease causing deteriorating lacrimal and salivary secretions over time
- Leads to multiple oral complications due to decreased saliva production

## Tongue Changes in Advanced Disease

- Depapillation of the tongue may be observed in advanced SS cases
- Loss of papillae afects taste and oral comfort

## Denture Retention Issues

- Dry mouth causes difficulty in denture retention and comfort
- Patients may experience instability and irritation with dentures

## Increased Incidence of Smooth Surface Caries

- Reduced salivary flow leads to higher dental caries risk
- Requires enhanced preventive dental care

## Other Causes of Dry Mouth

- Head and neck radiotherapy causes dose-dependent salivary reduction
- Rheumatoid arthritis and connective tissue diseases like scleroderma also cause dry mouth and eyes
- Sleep apnea and CPAP use contribute to dry mouth; management includes artificial saliva and addressing mouth leaks

## Early Diagnosis Importance

- Early detection minimizes oral complications
- Allows timely medical and dental interventions for better patient outcomes

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# MIND MAP

# DRY MOUTH AND SJOGREN SYNDROME: DIAGNOSTIC AND MANAGEMENT OVERVIEW

## Salivary Flow Rate Standards

- Normal unstimulated flow: 0.3 to 0.4 mL/min
- Indicative hypofunction: ⩽0.2 mL/min unstimulated, ⩽0.7 mL/min stimulated

## Drugs Commonly Causing Dry Mouth

- Anticholinergics and antihistamines
- Blood pressure medications: ACE inhibitors, beta blockers, diuretics
- Inhaled bronchodilators and opioids
- Psychotropic drugs including antidepressants and illicit substances

## Management Strategies

- Maintain adequate hydration and good oral hygiene
- Regular dental check-ups every 3 to 6 months
- Use topical remineralising agents and saliva substitutes
- Review and adjust medications with prescribers if possible

## Practical Patient Advice

- Drink at least 1.5 liters of water daily
- Chew food thoroughly and use sugarless gum or sweets
- Avoid smoking, acidic foods, caffeine, alcohol, and alcohol-containing mouthwashes
- Trial bicarbonate mouthwash (half teaspoon bicarbonate in warm water)

## Sjogren Syndrome Overview

- Autoimmune disease causing salivary and lacrimal gland dysfunction
- Predominantly afects menopausal women (9:1 female to male ratio)
- May be primary or secondary (associated with other autoimmune diseases)

## Pathogenesis of Sjogren Syndrome

- Autoantibodies Ro/SS-A and La/SS-B present in 60-70% of primary SS
- Lymphocytic infiltration and destruction of salivary glands
- Autoantibodies correlate with disease severity and complications

## Complications of Sjogren Syndrome

- Increased risk (44x) of malignant lymphoma
- Dryness of nose, skin, vagina; recurrent sinusitis and cough
- Keratoconjunctivitis sicca causing vision issues
- Extraglandular manifestations: kidney, liver, CNS, joints involvement
- 45% have thyroid dysfunction and increased psychological disorders

## Definitions of Dry Mouth

- Xerostomia: Subjective feeling of dry mouth
- Hyposalivation: Objective reduction in saliva production measured by sialometry.

## Causes of Dry Mouth

- Dehydration and alcohol consumption
- Anxiety and mouth breathing
- Various drugs afecting saliva production

## Consequences of Chronic Dry Mouth

- Increased risk of tooth decay and erosion
- Periodontal disease and oral candidiasis
- Difficulty with chewing, swallowing, speech, and denture retention
- Altered taste sensation

## Symptomatic Relief Options

- Artificial saliva and oral lubricants (e.g., bicarbonate mouthwash)
- Saliva-stimulating products like sugarless gum or lozenges (watch sugar/acid content)

## Dry Mouth in Special Populations

- Common in palliative care patients
- Associated with drug dependency and hepatitis C infection

## Sjogren Syndrome Etiology and Risk Factors

- Genetic predisposition with family clustering
- Viral triggers: Epstein-Barr, Hepatitis C, HTLV-1
- Hormonal influence modulating immune response

## Diagnosis Challenges and Criteria

- Average 6-10 years delay due to vague symptoms like fatigue and arthralgia
- Objective criteria include salivary gland biopsy, autoantibodies, and gland imaging.
- Differential diagnosis includes other conditions causing dry mouth

## Summary

- Dry mouth is a multifactorial condition needing thorough evaluation
- Sjogren syndrome is a key systemic cause with significant oral and systemic effects
- Early diagnosis, multidisciplinary management, and patient education are critical

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DRY MOUTH

## Question 1

# What is the difference between xerostomia and hyposalivation?

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# Answer 1

**Xerostomia is the subjective feeling of having a dry mouth, while hyposalivation is an objective reduction in the amount of saliva produced, also known as salivary gland hypofunction, measured by sialometry.**

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CUE CARDS

DRY MOUTH

# Answer 1

**Xerostomia is the subjective feeling of having a dry mouth, while hyposalivation is an objective reduction in the amount of saliva produced, also known as salivary gland hypofunction, measured by sialometry.**

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CUE CARDS

DRY MOUTH

## Question 2

**What are common causes of dry mouth?**

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DRY MOUTH

# Answer 2

**Common causes of dry mouth include dehydration, alcohol, anxiety, mouth breathing, and drugs.**

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DRY MOUTH

## Question 3

# Which types of drugs are frequently associated with causing dry mouth?

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CUE CARDS

DRY MOUTH

# Answer 3

Drugs associated with dry mouth include anticholinergic drugs, antihistamines, blood pressure medications (ACE inhibitors, angiotensin II receptor blockers, alpha blockers, beta blockers, diuretics), inhaled bronchodilators (beta2 agonists, muscarinic antagonists), opioids, psychotropic drugs (antidepressants, antipsychotics, illicit drugs like marijuana and cocaine, psychostimulants), and urinary antispasmodics.

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DRY MOUTH

## Question 4

# What are some oral consequences of chronic dry mouth?

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CUE CARDS

DRY MOUTH

# Answer 4

**Chronic dry mouth can lead to tooth decay and erosion, periodontal disease, oral mucosal disease, oral candidiasis, difficulty retaining dentures, problems with chewing, swallowing, speech, and altered taste sensation.**

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DRY MOUTH

## Question 5

# What management strategies are recommended for patients experiencing dry mouth?

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DRY MOUTH

# Answer 5

**Management includes ensuring adequate hydration, maintaining good oral hygiene, regular dental examinations every 3 to 6 months, topical remineralizing agents to prevent tooth decay, reviewing and adjusting medications, and symptomatic relief using artificial saliva or saliva stimulants such as sugarless gum or lozenges.**

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DRY MOUTH

## Question 6

# What practical advice can be given to patients to help manage dry mouth?

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DRY MOUTH

## Answer 6

**Patients should drink at least 1.5 liters of water daily, chew food thoroughly, use sugarless gum or sweets (avoiding fruit flavors), avoid smoking and acidic foods, limit caffeine and alcohol especially in the evening, avoid alcohol-containing mouthwashes, and try over-the-counter dry mouth products or bicarbonate mouthwash.**

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DRY MOUTH

## Question 7

# What is Sjogren syndrome and how does it relate to dry mouth?

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DRY MOUTH

# Answer 7

**Sjogren syndrome is an autoimmune disease causing inflammatory infiltration of exocrine glands, especially salivary and lacrimal glands, leading to severe dry mouth (xerostomia) and dry eyes (sicca symptoms). It can cause oral manifestations such as salivary gland enlargement and increased risk of oral infections.**

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DRY MOUTH

## Question 8

# What are the risk factors and possible causes of Sjogren syndrome?

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DRY MOUTH

# Answer 8

Risk factors include genetic predisposition, viral infections (Epstein Barr virus, Hepatitis C virus, Human T cell leukemia virus-1), and sex hormones, with a higher prevalence in women. The disease involves immune system disruption leading to lymphocytic infiltration and autoantibody production.

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DRY MOUTH

## Question 9

# What are the common oral complications seen in patients with Sjogren syndrome?

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DRY MOUTH

# Answer 9

**Common oral complications are decreased salivary flow causing oral mucosal inflammation, mucosal sloughing, ulcers, depapillation of the tongue, gingival inflammation, poor denture retention, increased risk of oral candidiasis, and higher incidence of dental caries.**

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DRY MOUTH

## Question 10

# How is Sjogren syndrome managed from a dental perspective?

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DRY MOUTH

# Answer 10

**Management includes regular dental examinations, preventive treatments like fluoride application, excellent oral hygiene reinforcement, topical antifungal treatments for candidiasis, avoiding anticholinergic drugs if possible, and use of salivary stimulants such as pilocarpine and cevimeline. Early diagnosis and multidisciplinary care are essential.**

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ORAL MEDICINE

# ORAL MUCOSAL DISEASES

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# ORAL MUCOSAL DISEASE AND ORAL CANCER OVERVIEW

## Red Flag Features of Oral Mucosal Disease

- Oral ulcers lasting over 2 weeks or recurring ulcers.
- Nontraumatic ulcers in children, pigmented lesions, red/white/mixed lesions with suspicious features like induration, ulceration with rolled margins, fixation, or in high-risk sites (lateral tongue, floor of mouth).

## Dry Mouth and Systemic Disease Manifestations

- Dry mouth not relieved by artificial saliva or caused by systemic illness.
- Oral signs of systemic diseases such as syphilis, Behçet syndrome, HIV, inflammatory bowel disease, lichen planus, pemphigoid.

## Manageable Conditions in General Practice

- Recurrent aphthous ulcers, traumatic ulcers, oral candidiasis, angular cheilitis.
- Oral mucocutaneous herpes simplex, dry mouth, oral mucositis, amalgam tattoo, geographic tongue, hairy tongue.

## Oral Cancer Overview

- Oral cancer causes significant morbidity and mortality; early stages often asymptomatic.
- Late symptoms: pain, discomfort, tongue immobility, tooth mobility, inability to wear dentures.

## Other Oral Cancers and Metastases

- Less common cancers originate from salivary glands or nonepithelial tissues.
- Metastatic cancers may come from breast, prostate, kidneys, lungs.
- Leukemia and lymphoma may present in oral cavity.

## Oral Erythroplakia

- O Potentially malignant bright red lesion, often asymptomatic.
- Common sites: floor of mouth, tongue, soft palate, buccal mucosa.
- High malignancy rate (70-90% carcinoma in situ or SCC); urgent specialist referral required.

## Common Causes of Oral Mucosal Lesions

- Oral mucosal lesions may arise from physiological changes, local disease, skin conditions, adverse drug reactions, or systemic diseases (e.g., gastrointestinal disease).
- Accurate diagnosis requires detailed patient history, including medication, plus thorough extraoral and intraoral examinations with diagnostic tests if needed.

## Other Important Warning Signs

- Facial or oral paraesthesia, persistent discomfort without clear cause.
- Lumps, swellings, lymphadenopathy, salivary gland issues, suspected allergies to dental materials.

## Oral Potentially Malignant Disorders (OPMD)

- Conditions include oral leucoplakia, erythroplakia, chronic hyperplastic candidiasis, actinic cheilitis, oral lichen planus, oral submucous fibrosis, discoid lupus erythematosus, dyskeratosis congenita, epidermolysis bullosa.
- These require specialist monitoring due to malignant potential.

## Physiological Oral Discolorations

- Examples include Fordyce spots (ectopic sebaceous glands) and leukoedema.
- These typically do not require active treatment.

## Squamous Cell Carcinoma (SCC)

- Most common oral malignancy, arising from oral epithelium.
- Common sites: lateral tongue, floor of mouth, gingivae.
- Risk factors: advanced age, male gender, tobacco, alcohol, oncogenic viruses (HPV), family history, immunosuppression, areca nut chewing.

## Oral Leucoplakia

- Nonremovable white lesion requiring further investigation.
- May be homogenous (uniform) or nonhomogeneous (irregular texture/color).
- Transformation risk to malignancy varies; specialist referral for biopsy and monitoring essential.

## Human Papilloma Virus (HPV)-Related Oral Lesions

- HPV causes various oral lesions: squamous papilloma (finger-like projections), condyloma acuminata (sexually transmitted), verruca vulgaris (common wart).
- Oncogenic HPV types linked to SCC of posterior tongue, tonsillar region, oropharynx, distinct from alcohol/tobacco-related cancers.
- Refer suspected HPV lesions to specialists for biopsy and management.

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# MIND MAP

# ORAL MUCOSAL CONDITIONS: ORAL LICHEN PLANUS AND RELATED DISORDERS

## Overview of Oral Lichen Planus (OLP)

- Lichen planus is a rare immune-mediated condition affecting skin, hair, nails, oral and genital mucosa.
- Oral lichen planus mainly involves buccal mucosa, tongue, and gingivae with reticular white lesions or erosive painful ulcers.

## Clinical Presentations of OLP

- Six oral forms: reticular, papular, plaque, atrophic, ulcerative (erosive), and rare bullous form.
- Erosive forms cause pain, burning, and ulceration requiring immunosuppressive therapy.

## Epidemiology and Distribution

- More common in women (ratio ~3:1 to 3:2), usually over 40 years old.
- Lesions are typically bilateral, symmetrical, with characteristic Wickham's striae

## Management of Oral Lichen Planus

- Topical corticosteroids (betamethasone dipropionate 0.05%) applied twice daily post meals.
- Regular specialist review due to chronicity and risk of malignant transformation.

## Geographic Tongue (Erythema Migrans)

- Benign, migratory red patches on dorsal tongue with white margins.
- Possible familial link; no treatment needed beyond reassurance unless symptoms worsen.

## Hairy Tongue

- Overgrowth and staining of filiform papillae causing black or colored tongue appearance.
- Managed by improving oral hygiene, gentle brushing, and sodium bicarbonate mouthwash.

## Recurrent Aphthous Ulcerative Disease

- Most common nontraumatic oral ulcers with immune-mediated origin.
- Three forms; associated with systemic conditions like nutritional deficiencies, coeliac disease, Bechet syndrome.

## Referral and Specialist Involvement

- Refer all uncertain, persistent, or suspicious oral mucosal lesions for biopsy and definitive management.
- Ongoing specialist follow-up essential for chronic conditions prone to malignant transformation.

## Pathophysiology and Etiology

- T-lymphocyte mediated immune damage to basal epithelial cells.
- Cause unknown; possibly triggered by diverse factors causing lichenoid tissue reaction.

## Diagnosis and Diferential Diagnosis

- Diagnosis confirmed by biopsy; diferentiate from oral lichenoid lesions.
- Important to exclude other mucosal diseases such as pemphigoid and pemphigus.

## Oral Lichenoid Lesions

- Caused by contact hypersensitivity (e.g., dental restorations) or drug reactions (beta blockers, NSAIDS).
- Associated with medical conditions like hepatitis C, thyroid disorders, chronic graft-versus-host disease.

## Amalgam Tattoo

- Blue-grey or black mucosal discoloration from dental amalgam implantation.
- Benign, requires no treatment; diagnosis can be confirmed by X-ray.

## Traumatic Oral Ulcers

- Caused by mechanical trauma from sharp foods, teeth, orthodontic appliances.
- Treat underlying trauma; use salt water rinses and topical analgesics for symptomatic relief.

## Management of Aphthous Ulcers

- Topical hydrocortisone 1% cream several times daily to treat lesions.
- Use topical analgesics like benzydamine gel or lidocaine viscous for pain relief.
- Refer ulcers lasting >2 weeks or with suspicious changes for specialist evaluation.

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# MIND MAP

# ORAL MUCOCUTANEOUS HERPES AND RELATED ORAL MUCOSAL DISEASES

## Primary Oral Mucocutaneous Herpes

- Primary HSV infection often occurs in childhood, causing fever, painful oral lesions, malaise, and cervical lymphadenopathy.
- Lesions start as blisters that ulcerate quickly and heal within days to two weeks;eating and drinking may be difficult, sometimes requiring hospitalization.

## Clinical Features of Herpetic Gingivostomatitis

- Lesions are widespread affecting all soft oral tissues, unlike necrotising gingivitis which is localized to gingiva.
- Advise avoiding direct contact with lesions to prevent virus transmission.

## Recurrent Oral Mucocutaneous Herpes

- Reactivation of latent HSV causes cold sores on lips or intraoral lesions, often preceded by prodromal symptoms like tingling or burning.
- Frequency of recurrences can be reduced with sun protection; complications include erythema multiforme.

## Management of Recurrent Herpes

- Episodic antiviral therapy such as topical aciclovir or oral famciclovir reduces episode duration.
- Refer severe, generalized, chronic, immunocompromised, or HIV patients for specialist care.

## Clinical Presentation of MMP

- Symptoms include bleeding, pain, dysphagia, and desquamation; positive Nikolsky sign may be present.
- Ocular involvement occurs in many cases, presenting as chronic conjunctivitis with risk of blindness.

## Epidermolysis Bullosa Acquisita (EBA)

- Rare autoimmune disease targeting Type VII collagen causing blistering after minor trauma, healing with scarring.
- Treatment often requires high-dose corticosteroids combined with immunosuppressants or dapsone.

## Diagnosis and Treatment of EM and Variants.

- No pathognomonic tests; biopsy and immunofluorescence exclude other conditions.
- Treatment includes identifying and removing triggers, supportive care, and controversial use of systemic corticosteroids.

## Prevention and Patient Advice

- Avoid direct contact with active oral lesions o limit HSV transmission.
- Use sun protection to reduce recurrent herpes episodes.
- Refer complex, severe, or immunocompromised cases for specialist evaluation and management.

## Management of Primary Oral Herpes

- Supportive care includes oral fluids, antipyretics, analgesics, and topical anesthetics like benzydamine gel or lidocaine viscous solution.
- Refer severe cases, immunocompromised, or HIV patients to medical practitioners.

## Diagnosis and Transmission of HSV Lesions

- Viral DNA detection is unreliable for differentiation from aphthous or traumatic ulcers; clinical history and exam are essential.
- Patients with active lesions should avoid direct contact to reduce transmission risk.

## Mucous Membrane Pemphigoid (MMP)

- Autoimmune subepithelial blistering disease mainly afecting gingiva and palate, causing painful erosions and scarring.
- Diagnosis requires biopsy and direct immunofluorescence; long-term immunosuppressive therapy is usual.

## Pemphigus Vulgaris (PV)

- Autoimmune intraepithelial blistering disease causing fragile vesicles that rupture to painful ulcers afecting oral mucosa and skin.
- Drug-induced cases reported (e.g., rifampicin, penicillamine, captopril); diagnosis confirmed by biopsy and immunofluorescence.

## Erythema Multiforme (EM) and Severe Variants

- Immune-mediated mucocutaneous disorder with oral erythema, blistering, and ulceration; often triggered by HSV or Mycoplasma infections.
- Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are severe forms with extensive mucosal and skin involvement.

## Summary of Key Oral Mucosal Diseases.

- Herpes simplex virus infections cause painful oral lesions with primary and recurrent forms.
- Autoimmune blistering diseases (MMP, PV, EBA) cause vesicles, erosions, and scarring, requiring biopsy for diagnosis.
- EM and its variants are immune reactions causing painful mucosal ulcerations with potential severe systemic involvement.

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# MIND MAP

# ORAL CANDIDIASIS AND RELATED ORAL CONDITIONS

## Overview of Oral Candidiasis and Candida-Associated Lesions

- Candida species are normal oral commensals but can cause opportunistic infections.
- Oral candidiasis is rare in healthy adults but common in neonates.
- Presence of 'red flag' oral mucosal signs requires specialist referral.

## Risk Factors and Special Considerations

- A Immunocompromised patients need specialist management.
- Oral candidiasis may be the first sign of undiagnosed HIV infection.

## Erythematous Candidiasis

- Sensitive red lesions often on palate and tongue.
- Tongue may appear depapillated and smooth.
- Treatment involves managing risk factors and topical antifungals.

## Angular Cheilitis (Angular Stomatitis)

- Painful redness and fissuring at mouth corners.
- Mixed infection usually involving Candida, Staphylococcus aureus, and Streptococcus species.
- Associated with dental issues, nutritional deficiencies, and dermatological conditions.
- Managed by dental review, correcting causes, and antifungal treatment.

## Median Rhomboid Glossitis

- Rhomboid, depapillated, erythematous area on mid dorsal tongue.
- May be fissured or nodular; sometimes causes stinging or burning.
- Treated by addressing causes and topical antifungal therapy.

## Management of Oral Mucositis

- Regular pain assessment; use topical analgesics like benzydamine or lidocaine.
- Artificial saliva for dryness; chlorhexidine mouthwash or gel for oral hygiene.
- Nutritional support and avoidance of irritant foods are critical.

## Consequences of Dry Mouth

- Increased risk of tooth decay, periodontal disease, candidiasis, and mucosal disease.
- Difficulties with chewing, swallowing, speech, denture retention, and altered taste.

## Practical Advice for Patients with Dry Mouth

- Drink at least 1.5 liters of water daily; chew thoroughly and use sugarless gum or sweets.
- Avoid smoking, acidic foods, caffeine, alcohol, and alcohol-containing mouthwashes.
- Try over-the-counter dry mouth products or bicarbonate mouthwash (half teaspoon bicarbonate in warm water).

## Pseudomembranous Candidiasis

- Creamy white curd-like plaques, sometimes removable, with red, raw base.
- Usually asymptomatic but can affect oropharynx and cause autoinoculation to palate.
- Management includes addressing predisposing factors and topical antifungal therapy.

## Hyperplastic Candidiasis

- Non-removable, asymptomatic white plaques, sometimes nodular.
- Common on retro-commissures, buccal mucosa, and lateral tongue; may mimicleukoplakia or cancer.
- Requires biopsy and specialist referral due to possible epithelial dysplasia.

## Denture-Associated Erythematous Stomatitis

- Red, sensitive lesions on denture-bearing areas, especially the palate.
- Risk factors: ill-fitting dentures, poor hygiene, dietary issues.
- Management includes improved denture hygiene, dental review, and topical anti fungals needed.

## Oral Mucositis

- Painful inflammation, redness, swelling, and ulceration from cancer therapies.
- Can severely affect eating, drinking, medication adherence, and increase infection risk.
- Requires multidisciplinary management including pain relief, oral care, and nutrition.

## Dry Mouth (Xerostomia) and Salivary Gland Hypofunction

- Common causes: dehydration, drugs, alcohol, anxiety, mouth breathing.
- Many medications (anticholinergics, antihypertensives, psychotropics) contributc.
- Head and neck radiotherapy often causes chronic dry mouth.

## Management of Dry Mouth

- Dental review before starting xerogenic drugs; review and reduce medications if possible.
- Hydration, good oral hygiene, regular dental check-ups every 3-6 months.
- Use of topical remineralizing agents and symptomatic treatments like artificial saliva or saliva stimulants.

## Prevention of Oral and Dental Complications in Dry Mouth

- Maintain excellent oral hygiene and regular dental exams.
- Avoid or limit acidic beverages and sugary snacks to meal times.
- Control sugar intake to reduce risk of tooth decay.

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ORAL MUCOSAL DISEASES

## Question 1

# What are common causes of oral mucosal lesions?

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# ORAL MUCOSAL DISEASES

## Answer 1

Oral mucosal lesions can be caused by physiological changes, local disease, oral manifestations of skin conditions, adverse drug reactions, or systemic diseases such as gastrointestinal diseases.

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ORAL MUCOSAL DISEASES

## Question 2

**What steps are essential for assessing an oral mucosal lesion?**

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ORAL MUCOSAL DISEASES

# Answer 2

**Assessing an oral mucosal lesion involves taking a full patient history including medication history, performing a thorough extraoral and intraoral examination, and using diagnostic investigations when appropriate.**

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ORAL MUCOSAL DISEASES

## Question 3

# What are some 'red flag' features of oral mucosal disease?

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ORAL MUCOSAL DISEASES

# Answer 3

**Red flag features include oral ulcers lasting more than 2 weeks, recurrent oral ulcers, nontraumatic oral ulcers in children, pigmented lesions, red, white or mixed lesions with features of potential malignancy, lesions in high-risk sites, facial or oral paraesthesia, persistent oral discomfort without obvious cause, lumps or swellings including lymphadenopathy, salivary gland swelling or blockage, suspected allergies or adverse reactions to dental materials, dry mouth not relieved by treatments, suspected oral manifestations of systemic disease, and lesions in immunocompromised patients.**

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ORAL MUCOSAL DISEASES

## Question 4

**Name at least four oral potentially malignant disorders.**

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ORAL MUCOSAL DISEASES

# Answer 4

**Oral potentially malignant disorders include oral leucoplakia, oral erythroplakia, oral lichen planus, and oral submucous fibrosis. Other examples are chronic hyperplastic candidiasis, actinic cheilitis, discoid lupus erythematosus, dyskeratosis congenita, and epidermolysis bullosa.**

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ORAL MUCOSAL DISEASES

## Question 5

# Which oral conditions can be managed in general practice?

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ORAL MUCOSAL DISEASES

# Answer 5

**Conditions manageable in general practice include recurrent aphthous ulcerative disease, traumatic oral ulcers, oral candidiasis, angular cheilitis, oral mucocutaneous herpes simplex virus, dry mouth, oral mucositis, amalgam tattoo, geographic tongue, and hairy tongue.**

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ORAL MUCOSAL DISEASES

## Question 6

# What is the most common type of oral cancer?

# Answer 6

**Squamous cell carcinoma is the most common oral malignancy.**

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ORAL MUCOSAL DISEASES

## Question 7

# What are the common sites for oral squamous cell carcinoma?

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ORAL MUCOSAL DISEASES

# Answer 7

**Common sites include the lateral surfaces of the tongue, floor of the mouth, and gingivae.**

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ORAL MUCOSAL DISEASES

## Question 8

# List some risk factors for oral squamous cell carcinoma.

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ORAL MUCOSAL DISEASES

# Answer 8

Risk factors include advanced age, male gender, smoking or tobacco use, alcohol consumption, infection by oncogenic viruses such as human papillomavirus (HPV), personal or family history of head and neck squamous cell carcinoma, history of cancer therapy, prolonged immunosuppression, and areca nut (betel quid) chewing.

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ORAL MUCOSAL DISEASES

## Question 9

# How is oral leucoplakia described clinically?

# Answer 9

**Oral leucoplakia is a nonremovable white lesion that cannot be classified as any specific condition. It can be homogeneous (uniform with often a fissured surface) or nonhomogeneous (with surface irregularity and color variation such as speckled).**

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ORAL MUCOSAL DISEASES

## Question 10

# What is the recommended action for patients with oral leucoplakia?

# Answer 10

**Patients should be referred to an appropriate specialist for biopsy and monitoring due to the risk of dysplasia or malignant transformation.**

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CUE CARDS

ORAL MUCOSAL DISEASES

## Question 11

# Describe oral erythroplakia and its significance.

# Answer 11

Oral erythroplakia is a fiery red, potentially malignant lesion that cannot be attributed to any specific condition. It is usually asymptomatic, isolated, and commonly appears on the floor of the mouth, tongue, soft palate, and buccal mucosa. It frequently represents carcinoma in situ or squamous cell carcinoma, requiring urgent specialist referral and biopsy.

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ORAL MUCOSAL DISEASES

## Question 12

**What types of oral lesions are caused by human papillomavirus (HPV)?**

# Answer 12

HPV can cause squamous papilloma, condyloma acuminata (sexually transmitted oral HPV lesions), and verruca vulgaris (common warts). Oncogenic HPV types are also associated with some squamous cell carcinomas of the posterior tongue, tonsillar region, and oropharynx.

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CUE CARDS

ORAL MUCOSAL DISEASES

## Question 13

# What is oral lichen planus and which areas does it commonly affect?

# Answer 13

**Oral lichen planus is an uncommon idiopathic immune-mediated condition affecting the oral mucosa, skin, hair, nails, and genital mucosa. It commonly affects the buccal mucosa, tongue, and gingivae.**

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ORAL MUCOSAL DISEASES

## Question 14

# What are the clinical forms of oral lichen planus?

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# Answer 14

**There are six recognized forms: reticular, papular, plaque-form, atrophic, ulcerative (erosive), and bullous form. The latter three can cause significant discomfort requiring immunosuppressive therapy.**

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ORAL MUCOSAL DISEASES

## Question 15

# What are the causes of oral lichenoid lesions?

# Answer 15

**Oral lichenoid lesions can be caused by contact hypersensitivity to dental restorations, hypersensitivity reactions to certain drugs (e.g., blood pressure medications, NSAIDs, thyroid disorder drugs), and medical conditions like hepatitis C infection, thyroid disorders, and chronic graft-versus-host disease.**

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ORAL MUCOSAL DISEASES

## Question 16

# What is geographic tongue and how is it managed?

# Answer 16

**Geographic tongue is a benign condition characterized by migratory red lesions with central depapillation and elevated white or cream margins, usually on the dorsal tongue. It requires no treatment beyond diagnosis and reassurance unless red flag features are present, in which case specialist referral is needed.**

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ORAL MUCOSAL DISEASES

## Question 17

# What is the typical cause and presentation of an amalgam tattoo?

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ORAL MUCOSAL DISEASES

# Answer 17

**An amalgam tattoo results from accidental implantation of amalgam particles into the oral mucosa during dental procedures. It presents as small, macular blue-grey to black discolorations near amalgam-restored teeth. It is benign and requires no treatment beyond correct diagnosis.**

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ORAL MUCOSAL DISEASES

## Question 18

# Describe the management of hairy tongue.

# Answer 18

**Management includes identifying and addressing the cause, improving oral hygiene, gently brushing the tongue, and using a sodium bicarbonate mouthwash made by dissolving half a teaspoon of bicarbonate powder in warm water, rinsed especially on waking and throughout the day. Specialist referral is needed if red flag features are present.**

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CUE CARDS

ORAL MUCOSAL DISEASES

## Question 19

# What are common causes of traumatic oral ulcers and how are they managed?

# Answer 19

Causes include eating rough or sharp foods, sharp broken teeth or restorations, toothbrushing, oral prostheses, orthodontic appliances, and chemical burns. Management involves addressing the cause, improving oral hygiene, smoothing sharp edges, adjusting prostheses, using wax on appliances, and symptomatic relief with antiseptic mouthwash or topical anaesthetics. Persistent or recurrent ulcers require specialist referral.

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CUE CARDS

ORAL MUCOSAL DISEASES

## Question 20

**What is the typical clinical presentation of recurrent aphthous ulcerative disease?**

# Answer 20

It is characterized by periodic eruptions of painful ulcers, usually on the mucosa of the cheek, lip, and floor of the mouth, sometimes involving gingivae and hard palate. It has an immune-mediated pathogenesis and can be triggered by trauma or smoking cessation.

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ORAL MUCOSAL DISEASES

## Question 21

# How is oral mucositis caused and managed?

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ORAL MUCOSAL DISEASES

# Answer 21

**Oral mucositis is painful inflammation and ulceration of oral mucosa caused by radiotherapy, chemotherapy, or drugs. Management includes symptomatic relief with analgesics and mouth rinses, oral care regimens, nutritional support, and multidisciplinary care. Artificial salivary products and chlorhexidine mouthwash or gel may be used, with systemic analgesics if topical treatment is insufficient.**

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ORAL MEDICINE

# ORAL VIRAL INFECTIONS

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# ORAL VIRAL INFECTIONS AND THEIR DENTAL IMPLICATIONS

## Types of Oral HPV Lesions

- Condyloma acuminata is an oral lesion linked to sexually transmitted HPV infections.
- Verruca vulgaris (common wart) can also appear in the oral cavity and resembles other HPV lesions.

## Diagnosis and Referral for HPV Lesions.

- Clinical similarity between HPV lesions makes biopsy necessary for accurate diagnosis.
- Patients with suspected HPV lesions should be referred to specialists for management.

## Verruca Vulgaris in the Oral Cavity

- Common wart affects keratinized mucosal areas like lips, hard palate, and gingivae.
- Diagnosis requires histological confirmation and identification of cutaneous HPV types.

## Kaposi's Sarcoma (KS) in AIDS

- KS affects up to 60% of AIDS patients orally, commonly on the palate and tongue.
- Human herpesvirus 8 (HHV8) is a key factor in AIDS-related KS development.

## HCV Transmission Risks in Dental Settings

- Transmission mainly through blood-to-blood contact, with sharps injuries being a concern.
- Strict sterilization and infection control are critical to prevent cross-infection.

## Impact of HCV Therapy on Dental Treatment

- Invasive dental care should be postponed during antiviral therapy due to bleeding and infection risk.
- Collaboration with medical specialists is essential for urgent dental procedures.

## Possible Correlation Between HCV and Lichen Planus

- Studies suggest a link but the cause remains unclear.
- Routine HCV testing for erosive lichen planus is not currently recommended.

## Conclusion

- The prevalence of HCV and HPV infections pose significant oral health and systemic risks.
- Dental professionals must apply infection control, collaborate with medical specialists, and emphasize preventive care to manage afected patients effectively.

## Human Papilloma Virus (HPV) and Oral Lesions

- HPV causes various oral mucosal lesions transmitted mainly by direct contact.
- Squamous papilloma is the most common benign oral HPV lesion with finger-like projections.

## Oncogenic HPV and Oral Cancer

- Certain HPV types cause squamous cell carcinoma in the oropharynx, tonsils, and posterior tongue.
- These HPV-related cancers difer from those caused by alcohol and tobacco.

## Benign Oral HPV Lesions Overview

- Squamous cell papilloma represents about half of soft tissue oral tumors.
- Oral condylomas can arise via oral sex, autoinoculation, or maternal transmission.

## Human Immunodeficiency Virus (HIV) Oral Manifestations

- HIV infection ranges from asymptomatic to severe AIDS-related conditions.
- Oral manifestations include oral hairy leukoplakia (OHL), candidiasis, and Kaposi's sarcoma.

## Hepatitis C Virus (HCV) Infection Concerns

- HCV infection is widespread and can cause liver disease, fatigue, and oral health problems.
- Dental practitioners should observe precautions when treating patients with severe liver disease.

## Dental Management of HCV-Infected Patients

- Patients with cirrhosis have bleeding risks; invasive treatments require medical consultation.
- Some common dental drugs are contraindicated due to liver toxicity risks.

## Oral Health Challenges in HCV Patients

- Xerostomia is common, contributing to increased dental disease.
- Preventive dental programs are vital to reduce pathology and maintain oral health.

## Alcoholism and Oral Health in HCV Patients

- Alcohol abuse may exacerbate dental erosion and tooth wear.
- Awareness of lifestyle factors is important in managing oral health.

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# MIND MAP

# ORAL VIRAL INFECTIONS AND THEIR MANAGEMENT IN DENTISTRY

## Overview of Oral Viral Infections

- Common oral viruses encountered in dental practice include HSV-1 & HSV-2, Epstein-Barr virus, varicella-zoster virus, Coxsackie virus, HPV, and HIV.
- Diagnosis, management principles, and antiviral pharmacological treatments are essential for effective care.

## Viral Biology and Characteristics

- Viruses require a host for replication, possess either DNA or RNA, and have a protein shell (nucleocapsid), sometimes with a lipid envelope.
- Human viruses have four nucleic acid types: ssDNA, dsDNA, ssRNA, and dsRNA.

## Primary Oral Mucocutaneous Herpes

- Occurs mostly in childhood with fever, painful oral lesions, malaise, and lympha denopathy.
- Management includes supportive care: fluids, analgesics, topical anesthetics like benzydamine gel or lidocaine viscous solution.

## Management of Severe or Immunocompromised Cases

- Referral required for severe cases, immunocompromised patients, or those with HIV.
- Chronic, generalized, or difficult-to-treat herpes infections need specialist medical attention.

## Varicella-Zoster Virus Infections

- Chickenpox is the primary infection, mostly in childhood with oral vesicles and a characteristic skin rash.
- Shingless viral reactivation in older adults, causing unilateral vesicles in trige minal nerve distribution and possible Ramsay Hunt Syndrome.

## Prevention and Transmission Control

- Patients with active viral lesions should avoid direct contact reduce virus spread.
- Sun protection can minimize herpes simplex virus recurrences.

## Clinical Differential Diagnosis

- Herpes simplex lesions may resemble aphthous or traumatic ulcers; diagnosis relies on clinical history and examination rather than microbiological testing alone.
- Necrotising gingivitis difers from herpetic gingivostomatitis by tissue involvement and age prevalence.

## Herpes Simplex Virus (HSV) Infections

- HSV causes oral mucocutaneous herpes including primary herpetic gingivostomatitis and recurrent herpes labialis.
- Lesions begin as blisters that ulcerate, heal in days to weeks, and can be severe in adults or immunocompromised patients.

## Recurrent Oral Herpes (Herpes Labialis)

- Reactivation leads to cold sores on lips or oral mucosa, often preceded by tingling or itching (prodrome).
- Frequency can be reduced by sun protection; antiviral creams (aciclovir) or oral famciclovir reduce episode duration.

## Epstein-Barr Virus (EBV) Related Conditions

- Infectious mononucleosis commonly affects teenagers and young adults, transmitted via saliva causing pharyngitis and lymphadenopathy.
- Oral hairy leukoplakia presents as bilateral white patches on the tongue, linked to immunosuppression (e.g., HIV, corticosteroids).

## Coxsackie Virus Oral Diseases

- Herpangina causes fever and vesicles in the oropharynx, common in children, with no gingival involvement.
- Hand, foot, and mouth disease features oral, hand, and foot vesicles, often epidemic in schoolchildren, distinguished by extra-oral lesions.

## Pharmacological Treatments Summary

- Supportive care is primary for mild infections; topical anesthetics and analgesics relieve symptoms.
- Antiviral agents like aciclovir cream and famciclovir oral tablets reduce recurrence severity and duration.
- Severe or immunocompromised cases require specialist antiviral therapy and medical referral.

## Complications and Special Syndromes

- Erythema multiforme can complicate herpes simplex reactivation.
- Postherpetic neuralgia causes sharp localized pain following shingles, affecting quality of life.



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ORAL VIRAL INFECTIONS

## Question 1

# What are the common oral viral infections encountered in dental practice?

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# ORAL VIRAL INFECTIONS

## Answer 1

**Common oral viral infections in dental practice include herpes simplex virus types 1 and 2 (HSV), Epstein-Barr virus (EBV), varicella-zoster virus, Coxsackie virus, human papilloma virus (HPV), and human immunodeficiency virus (HIV).**

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ORAL VIRAL INFECTIONS

## Question 2

**Why do viruses require a host organism to reproduce?**

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# ORAL VIRAL INFECTIONS

## Answer 2

**Viruses are not self-reproducing because they lack ribosomes and cannot synthesize proteins. They need a host organism, which provides ribosomes and cellular machinery, to reproduce or replicate from their nucleic acid.**

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ORAL VIRAL INFECTIONS

## Question 3

# What are the typical clinical features of primary herpetic gingivostomatitis?

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# ORAL VIRAL INFECTIONS

## Answer 3

**Primary herpetic gingivostomatitis usually occurs in childhood and presents with fever, painful intraoral lesions starting as blisters that ulcerate rapidly, systemic symptoms like malaise and lethargy, and cervical lymphadenopathy. Healing occurs within days to two weeks.**

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ORAL VIRAL INFECTIONS

## Question 4

# How should minor primary oral mucocutaneous herpes be managed?

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# ORAL VIRAL INFECTIONS

## Answer 4

**Minor primary oral mucocutaneous herpes should be managed supportively with oral fluids, antipyretics, analgesics, and topical anesthetics such as benzydamine 1% gel or lidocaine viscous solution applied 2- to 3-hourly as needed.**

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ORAL VIRAL INFECTIONS

## Question 5

# What distinguishes recurrent oral mucocutaneous herpes (herpes labialis) from primary infection?

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# ORAL VIRAL INFECTIONS

## Answer 5

**Recurrent oral mucocutaneous herpes (herpes labialis) results from latent virus reactivation, usually presents as cold sores on the lips preceded by prodromal symptoms (pain, burning, tingling), and is generally mild and infrequent, unlike the systemic symptoms of primary infection.**

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ORAL VIRAL INFECTIONS

## Question 6

# Which oral conditions are commonly associated with Epstein-Barr virus?

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ORAL VIRAL INFECTIONS

# Answer 6

**Epstein-Barr virus is associated with infectious mononucleosis (glandular fever) and oral hairy leukoplakia, as well as certain lymphomas and nasopharyngeal carcinoma.**

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ORAL VIRAL INFECTIONS

## Question 7

# What are the primary and secondary infections caused by varicella-zoster virus?

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# ORAL VIRAL INFECTIONS

## Answer 7

**The primary infection is chickenpox, characterized by oral vesicles, ulcers, and a pruritic skin rash. The secondary infection is shingles, caused by viral reactivation, presenting as unilateral vesicles and ulcers in a dermatomal distribution, sometimes involving the face and oral mucosa.**

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ORAL VIRAL INFECTIONS

## Question 8

# What oral conditions are caused by Coxsackie virus?

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ORAL VIRAL INFECTIONS

# Answer 8

**Coxsackie virus causes herpangina, with small oral vesicles mostly in the oropharynx, and hand, foot, and mouth disease, which presents with vesicles on the oral mucosa, hands, and feet.**

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ORAL VIRAL INFECTIONS

## Question 9

# What types of oral lesions are linked to human papilloma virus (HPV)?

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# ORAL VIRAL INFECTIONS

## Answer 9

**HPV causes squamous papilloma, condyloma acuminatum (venereal warts), verruca vulgaris (common warts), and focal epithelial hyperplasia. Some oncogenic HPV types are linked to squamous cell carcinoma in the oropharynx.**

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ORAL VIRAL INFECTIONS

## Question 10

# What are the dental management considerations for patients infected with hepatitis C virus (HCV)?

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ORAL VIRAL INFECTIONS

# Answer 10

**Dental management of HCV-infected patients requires awareness of bleeding risks due to liver disease, avoidance of hepatotoxic drugs, consultation with medical specialists before invasive procedures, postponement of dental work during antiviral therapy, and preventive strategies to address oral health complications like xerostomia.**

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ORAL MEDICINE

# ORAL RECURRENT ULCERATIONS

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

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# MIND MAP

# RECURRENT ORAL ULCERATION: DIAGNOSIS AND MANAGEMENT

## Epidemiology and Typical Onset

- Some types of recurrent oral ulceration, like aphthous stomatitis, typically begin in childhood or adolescence.
- In middle-aged or elderly patients, other diagnoses such as lichen planus or vesiculobullous disorders should be considered.

## Extraoral and Systemic Associations

- Extraoral symptoms including skin, eye, or genital involvement may suggest conditions like Behcet's syndrome.
- Awareness of blistering before ulcers may indicate vesiculobullous disorders.

## Examination Procedures

- Extraoral exam assesses nutritional status, skin pallor, and lymph node enlargement.
- Intraoral exam records number, shape, size, location of ulcers, and presence/absence of scarring.

## Causes of Recurrent Oral Ulceration

- Trauma: mechanical, thermal, or chemical irritants causing recurrent ulcers if not removed.
- Viral infections: primarily herpes simplex virus (HHV-1), varicella-zoster virus, and coxsackie virus.
- Bacterial infection: acute necrotizing ulcerative gingivitis (ANUG) linked to smoking and compromised health.
- Nutritional deficiencies: iron, folate, or vitamin B12 deficiencies may predispose or aggravate ulcers.
- Hematologic disorders: leukemia, aplastic anemia, though these ulcers are usually not recurrent.
- Medications: e.g., methotrexate can cause dose-related oral ulceration.
- Xerostomia: predisposes to ulcers, especially with denture use; causes include Sj ogren's syndrome and medication side efects.
- Neoplastic disease: persistent, progressive ulceration requiring diferentiation from recurrent ulcers.

## Acute Necrotizing Ulcerative Gingivitis (ANUG) Management

- Debridement of plaque and necrotic debris, use of local anesthetics if needed.
- Antibiotics (metronidazole), analgesics, and smoking cessation advised.
- Use of chlorhexidine or hydrogen peroxide rinses to assist oral hygiene.
- Follow-up in 48-72 hours for periodontal exam and further debridement.

## Summary and Recommendations

- Diagnosis requires thorough history, clinical examination, and appropriate investigations.
- Management is multifactorial: remove irritants, treat infections, address nutritional deficiencies, and provide symptomatic relief.
- Regular review is essential to modify treatment and monitor ulcer recurrence or complications.

## Definition and Overview

- An ulcer is a complete breach of the oral epithelium covered with fibrin slough, appearing as a yellow/white lesion surrounded by erythema.
- Recurrent oral ulceration affects the oral mucosa and includes traumatic, infective, aphthous, drug-induced, systemic disease-related, and malignant-associated ulcers.

## Clinical Presentation and Prodromal Phase

- Recurrent ulcers often show periods of active ulceration with remissions.
- Some patients experience a prodromal phase with altered sensations before ulcers develop.

## Medical and Dental History

- Important to review medical history for anemia, autoimmune diseases, diabetes, and medications (e.g., methotrexate) linked to ulceration.
- Dental trauma or treatment can precipitate ulcers, including recurrent herpes simplex virus infections.

## Types and Characteristics of Oral Ulcers

- Minor aphthous ulcers: small, round/oval, non-keratinized mucosa, heal without scarring.
- Major aphthous ulcers: larger, often oropharyngeal, heal with scarring.
- Herpetiform ulcers: numerous small ulcers that may coalesce, heal without scarring.
- Vesiculobullous disorders: ragged ulcers with peeling epithelium, e.g., pemphigoid and pemphigus vulgaris.
- Lip ulcers with bleeding/crusting may indicate erythema multiforme.

## Viral Infection Specifics and Management

- Herpes Simplex Virus (HSV): causes cold sores and intraoral lesions, precededby prodrome; management includes antiviral therapy (aciclovir, famciclovir).
- Varicella-Zoster Virus: primary infection is chickenpox; reactivation causes shingles with unilateral oral vesicles and postherpetic neuralgia.
- Coxsackie Virus: causes herpangina and hand, foot, and mouth disease with characteristic oral and extraoral lesions.

## Recurrent Aphthous Ulceration (RAU)

- Most common recurrent oral ulceration affecting up to 20% of the population.
- Slight female predilection and possible genetic predisposition.
- Forms of RAU:
  - Minor RAU: 80-90% cases, small ulcers on non-keratinized mucosa, heal without scarring.
  - Major RAU: 5-10% cases, larger ulcers possibly on keratinized mucosa, heal with scarring.
  - Herpetiform RAU: numerous small ulcers that may coalesce; heal without scarring.
- Severe forms cause significant discomfort, affecting eating and speaking.

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# MIND MAP

# ORAL DERMATOSES: OVERVIEW AND MANAGEMENT

## Oral Dermatoses Overview

- Oral dermatoses include lichen planus, mucous membrane pemphigoid, pemphigus vulgaris, erythema multiforme, and less common conditions like dermatitis her petiformis.
- These diseases primarily affect oral mucosa and may present as recurrent oral ulceration.

## Oral Lichen Planus (OLP)

- OLP is an idiopathic immune-mediated condition affecting skin, hair, nails, and mucosa, commonly the buccal mucosa, tongue, and gingivae.
- Six oral presentations: reticular, papular, plaque-form, atrophic, ulcerative (erosive), and rare bullous forms.

## Diagnosis and Management of OLP

- Biopsy and specialist referral are essential for definitive diagnosis and to exclude lichenoid lesions.
- Treatment includes topical corticosteroids (e.g., betamethasone dipropionate 0.05%) applied twice daily until symptom resolution.

## Clinical Presentation and Diagnosis of MMP

- Oral lesions include large painful erosions and desquamative gingivitis.
- Diagnosis requires biopsy with histology and direct immunofluorescence; long-term immunosuppressive therapy is typical.

## Clinical Features and Diagnosis of PV

- Blisters rupture rapidly leaving painful erosions; Nikolsky sign is often positive.
- Diagnosis confirmed by biopsy and direct immunofluorescence of perilesional tissue.

## Diagnosis and Treatment of EM

- Diagnosis through clinical features and exclusion of similar disorders via biopsy and immunofluorescence.
- Treatment includes identifying and removing triggers plus immunomodulatory therapy; systemic corticosteroids use remains controversial.

## Investigation of Recurrent Oral Ulceration

- Hematological tests to exclude anemia or deficiencies.
- Accurate diagnosis based on history and clinical presentation is critical.

## Topical and Systemic Treatments

- Corticosteroid mouthwashes (e.g., prednisolone or dexamethasone solutions) help widespread ulceration.
- Intralesional triamcinolone acetonide and topical pimecrolimus cream can be effective.
- Systemic corticosteroids may be required for severe or resistant cases.

## Clinical Features of Oral Lichen Planus

- Reticular form shows characteristic white lace-like striations (Wickham's striae)
- Erosive form presents with painful erythematous, ulcerated, or eroded mucosa causing burning or stinging sensations.

## Mucous Membrane Pemphigoid (MMP)

- MMP is an autoimmune subepithelial blistering disorder affecting mainly gingiva and palate, often in older adults with a female predilection.
- Vesicles rupture quickly, causing painful erosions that may heal with scarring; conjunctival involvement can cause blindness.

## Pemphigus Vulgaris (PV)

- PV is an autoimmune disease causing intraepithelial blistering, most aggressive among pemphigus types with high morbidity and mortality.
- Oral mucosa is often the initial site, followed by skin and other mucosal surfaces like conjunctiva and genitalia.

## Erythema Multiforme (EM) and Severe Variants

- EM commonly affects adolescents and young adults; triggered by infections (HSV, Mycoplasma) or drugs.
- Characterized by swollen, ulcerated, crusted lips; Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are severe forms.

## Discoid Lupus Erythematosus (DLE)

- DLE is an autoimmune disease with rare oral involvement, resembling oral lichen planus or presenting as irregular ulcers with erythematous borders.
- More common in older females; drug-induced cases are rare.

## Management Strategies for Recurrent Oral Ulceration

- Eliminate mucosal trauma sources such as biting or sharp teeth.
- Identify and avoid possible dietary triggers; use food diaries if needed. & Address psychological factors such as stress, possibly through counseling.
- Medications include antiseptics, corticosteroids, immunosuppressants, and topic al analgesics like lignocaine gel.

## Summary

- Oral dermatoses are complex immune-mediated diseases with varied clinical presentations.
- Early specialist referral, biopsy diagnosis, and tailored immunosuppressive treatments are key to management.
- Ongoing monitoring is essential, especially for conditions with malignant potential like oral lichen planus.

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ORAL RECURRENT ULCERATIONS

## Question 1

# What is the definition of an ulcer in the context of recurrent oral ulceration?

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# ORAL RECURRENT ULCERATIONS

## Answer 1

**An ulcer is a complete breach of the epithelium that becomes covered with a fibrin slough and appears as a yellow/white lesion surrounded by erythema.**

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# ORAL RECURRENT ULCERATIONS

## Question 2

**Which types of recurrent oral ulceration are covered in the discussed paper?**

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# ORAL RECURRENT ULCERATIONS

## Answer 2

**Types include traumatic, infective, aphthous ulceration related to oral dermatoses, drug-induced ulceration, ulceration as a manifestation of systemic disease, and ulceration indicating malignancy.**

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ORAL RECURRENT ULCERATIONS

## Question 3

# What is a common pattern of recurrent oral ulceration in terms of onset and progression?

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# ORAL RECURRENT ULCERATIONS

## Answer 3

**Recurrent oral ulceration typically begins in childhood or adolescence with periods of ulceration alternating with remission, sometimes preceded by a prodromal phase of altered sensation.**

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ORAL RECURRENT ULCERATIONS

## Question 4

# What extraoral symptoms should be assessed in patients with recurrent oral ulceration?

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# ORAL RECURRENT ULCERATIONS

## Answer 4

**Questions should be directed toward skin involvement and other systems such as the eyes or genital regions to raise suspicion of conditions like Behcet’s syndrome or vesiculobullous disorders.**

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ORAL RECURRENT ULCERATIONS

## Question 5

# What medical conditions may be relevant in the history of a patient with recurrent oral ulceration?

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# ORAL RECURRENT ULCERATIONS

## Answer 5

**Relevant conditions may include anemia, blood dyscrasias, autoimmune diseases, diabetes, and medication use such as methotrexate.**

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ORAL RECURRENT ULCERATIONS

## Question 6

# How can dental history contribute to the diagnosis of recurrent oral ulceration?

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# ORAL RECURRENT ULCERATIONS

## Answer 6

**Oral ulceration occurring after dental treatment may indicate minor recurrent aphthous ulceration or recurrent intraoral herpes simplex virus infection triggered by minor trauma.**

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CUE CARDS

ORAL RECURRENT ULCERATIONS

## Question 7

# What are key points to note during intraoral examination of recurrent oral ulcers?

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CUE CARDS

# ORAL RECURRENT ULCERATIONS

## Answer 7

**Record the presence, number, shape, size, location of ulcers, and presence or absence of scarring; note typical patterns for minor, herpetiform, and major aphthous ulcers.**

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CUE CARDS

ORAL RECURRENT ULCERATIONS

## Question 8

# What clinical features differentiate mucous membrane pemphigoid from pemphigus vulgaris?

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CUE CARDS

# ORAL RECURRENT ULCERATIONS

## Answer 8

**Mucous membrane pemphigoid features more robust vesicles that persist longer and heal with scarring; pemphigus vulgaris vesicles are short-lived and rupture quickly, with intra-epithelial blistering.**

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CUE CARDS

ORAL RECURRENT ULCERATIONS

## Question 9

# What is the usual cause and clinical presentation of recurrent intraoral herpes simplex virus infection?

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CUE CARDS

# ORAL RECURRENT ULCERATIONS

## Answer 9

It is caused by latent reactivation of herpes simplex virus type 1, presenting as fluid-filled vesicles on the lips or intraoral mucosa, preceded by prodromal pain, burning, or tingling, resolving in 7-10 days.

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CUE CARDS

ORAL RECURRENT ULCERATIONS

## Question 10

**How is minor recurrent aphthous ulceration characterized clinically?**

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CUE CARDS

# ORAL RECURRENT ULCERATIONS

## Answer 10

It usually involves 1 to 5 round or oval ulcers about 5 mm in diameter on non-keratinized mucosa such as lip and cheek mucosa, healing without scarring within 1 to 2 weeks.

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CUE CARDS

ORAL RECURRENT ULCERATIONS

## Question 11

# What are the main clinical forms of recurrent aphthous ulceration?

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# ORAL RECURRENT ULCERATIONS

## Answer 11

**The three forms are minor recurrent aphthous ulceration, major recurrent aphthous ulceration, and herpetiform ulceration.**

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CUE CARDS

ORAL RECURRENT ULCERATIONS

## Question 12

**Which autoimmune diseases commonly cause recurrent oral ulceration presenting as oral dermatoses?**

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# ORAL RECURRENT ULCERATIONS

## Answer 12

**Lichen planus, mucous membrane pemphigoid, pemphigus vulgaris, and erythema multiforme.**

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CUE CARDS

ORAL RECURRENT ULCERATIONS

## Question 13

# What is the clinical significance of the Nikolsky sign in oral mucosal diseases?

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# ORAL RECURRENT ULCERATIONS

## Answer 13

**A positive Nikolsky sign indicates that firm lateral pressure can separate epithelium from underlying tissue, seen in diseases like mucous membrane pemphigoid and pemphigus vulgaris.**

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ORAL RECURRENT ULCERATIONS

## Question 14

# What triggers can provoke erythema multiforme, and what are its typical oral manifestations?

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# ORAL RECURRENT ULCERATIONS

## Answer 14

**Triggers include infections like herpes simplex and Mycoplasma pneumoniae or certain drugs; oral manifestations include swollen, ulcerated, crusted bloody lips and painful irregular ulcers with erythematous halos.**

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ORAL RECURRENT ULCERATIONS

## Question 15

# What general management strategies are recommended for recurrent aphthous ulceration?

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# ORAL RECURRENT ULCERATIONS

## Answer 15

**Management involves eliminating trauma, assessing dietary triggers, considering hormonal and psychological factors, symptomatic treatment with corticosteroids, antiseptics, pain relief, and systemic therapy for severe cases.**

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ORAL MEDICINE

# OSTEORADIONECROSIS OF JAW

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

# WINSPERT MIND MAP

# OSTEORADIONECROSIS OF THE JAW (ORNJ)

## 1. Overview of ORNJ

- ORNJ is a serious long-term complication of radiotherapy in the craniofacial region.
- Defined as irradiated bone exposure lasting 3-6 months without healing, absent tumor recurrence.

## 2. Clinical Presentation and Symptoms

- Symptoms vary: chronic pain, nonhealing wounds, orocutaneous fistulas, pathol ogic fractures.
- Small lesions may be asymptomatic, causing delayed detection.

## 3. Risk Factors and Predictive Features

- Dental extractions and dentoalveolar surgery (DAS), especially post-radiotherapy, increase risk.
- Tobacco use impairs wound healing due to vasoconstriction and toxic chemicals.

## 4. Impact of Radiotherapy and Radiation Dose

- High radiation doses (>65 Gy) contribute to ORNJ but do not guarantee its development.
- ORNJ has a predilection for the mandible due to its dense bone and blood supply differences.

## 5. Influence of Chemotherapy

- Chemotherapy alone is not a significant predictor for ORNJ.
- Evidence is mixed regarding combined chemo-radiotherapy's role in ORNJ risk.

## 6. Role of Systemic Conditions

- Diabetes Mellitus Type 2 (DM2) worsens vascular and immune function, increasing ORNJ risk.
- DM2 impairs blood flow and immune defense, complicating wound healing post-radiotherapy.

## 7. Timing and Healing Considerations

- 88.1% of ORNJ cases occur within the first year after radiotherapy.
- Healing of bone after dental extractions requires adequate time before radiotherapy (2-3 weeks recommended).

## 8. Diagnosis and Differential Diagnosis

- Must rule out tumor recurrence, medication-related osteonecrosis, and osteomyelitis before diagnosing ORNJ.
- Radiographic findings alone are insufficient; clinical and histopathological confirmation needed.

## 9. Management and Prevention Strategies

- Strongly encourage smoking cessation and glycemic control in diabetic patients.
- Pre-radiotherapy dental assessment and timely completion of dental surgery reduce risk.
- Post-DAS follow-up at 2 weeks and lifelong dental monitoring recommended.

## 10. Hyperbaric Oxygen Therapy (HBOT

- HBOT increases tissue oxygenation, promoting wound healing and reducing recovery time.
- Used efectively for prevention and management of ORNJ complications in dental medicine.

## 11. Pathophysiology and Progression of ORNJ

- ORNJ characterized by hypoxia, hypocellularity, and hypovascularity, leading to nonhealing bone lesions.
- Exposed bone is vulnerable to infection and contamination from oral environment.

## 12. Staging and Types of ORNJ

- Three types: spontaneous (high-dose radiation induced), trauma before radiotherapy, and trauma after radiotherapy.
- Radiation >700 cGy can cause immediate bone cell death and spontaneous ORNJ.

## 13. Head and Neck Radiotherapy Considerations

- Multidisciplinary dental management essential before, during, and after radiotherapy.
- Conservative dental treatments preferred; extractions within radiation field require specialist consultation.

## 14. Recommendations for Clinical Practice

- Shorten bone exposure period for ORNJ diagnosis to 1 month rather than 3-6 months.
- Educate patients on smoking cessation and diabetes control.
- Ensure all dental work is completed well before radiotherapy and monitor healing closely.



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OSTEORADIONECROSIS OF JAW

## Question 1

# What is osteoradionecrosis of the jaw (ORNJ) and how is it clinically defined?

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# OSTEORADIONECROSIS OF JAW

## Answer 1

**Osteoradionecrosis of the jaw (ORNJ) is a serious long-term complication of radiotherapy in the craniofacial region, where irradiated bone becomes devitalized and exposed without healing for a period of 3-6 months in the absence of tumor recurrence or other diseases in the irradiated area.**

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# OSTEORADIONECROSIS OF JAW

## Question 2

**What are the common symptoms and clinical presentations of ORNJ?**

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# OSTEORADIONECROSIS OF JAW

## Answer 2

**ORNJ presents variably but commonly includes chronic pain, nonhealing wounds, orocutaneous fistulas, pathologic fractures, mouth pain, jaw swelling, bad breath, mouth sores, difficulty opening the jaw, dysgeusia, paraesthesia, bone exposure, gingival ulceration, tooth fracture, xerostomia, and facial deformity.**

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# OSTEORADIONECROSIS OF JAW

## Question 3

**Why is dentoalveolar surgery (DAS) performed after radiotherapy associated with a higher risk of developing ORNJ compared to DAS before radiotherapy?**

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# OSTEORADIONECROSIS OF JAW

## Answer 3

**Post-radiotherapy DAS carries a higher risk because radiotherapy significantly alters the supporting structures of the teeth, impairing bone healing and increasing vulnerability to osteoradionecrosis.**

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OSTEORADIONECROSIS OF JAW

## Question 4

# How do tobacco use and nicotine contribute to the risk of developing ORNJ?

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# OSTEORADIONECROSIS OF JAW

## Answer 4

**Tobacco use impairs wound healing due to vasoconstriction and chemical trauma to oral mucosa. Nicotine causes platelet aggregation and vasoconstriction, increasing microvascular thrombosis and reducing micro-perfusion, while carbon monoxide causes cellular hypoxia by inhibiting oxygen binding to hemoglobin, all of which undermine tissue repair.**

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OSTEORADIONECROSIS OF JAW

## Question 5

# What is the effect of diabetes mellitus type 2 (DM2) on the risk and healing of ORNJ?

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CUE CARDS

# OSTEORADIONECROSIS OF JAW

## Answer 5

DM2 impairs cardiovascular and immune systems, causing peripheral vascular disease and microangiopathy that reduce blood supply and impair wound healing. It also compromises immunity by affecting neutrophil chemotaxis and killing, increasing infection risk especially when combined with surgery and radiotherapy.

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OSTEORADIONECROSIS OF JAW

## Question 6

# Does the total radiation dose alone predict the absolute risk of developing ORNJ?

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# OSTEORADIONECROSIS OF JAW

## Answer 6

**No, while high radiation doses exceeding 65 Gy contribute to ORNJ risk, total radiation dose alone does not predict absolute risk, as some patients receiving doses above 66 Gy do not develop ORNJ, indicating other factors are involved.**

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OSTEORADIONECROSIS OF JAW

## Question 7

# What is the typical timing for the occurrence of ORNJ after radiotherapy?

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# OSTEORADIONECROSIS OF JAW

## Answer 7

**Approximately 88.1% of ORNJ cases occur within the first year after radiotherapy, during which irradiated tissues are compromised and vulnerable to injury and delayed healing.**

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CUE CARDS

OSTEORADIONECROSIS OF JAW

## Question 8

# What are the recommended preventive measures to reduce the risk of ORNJ in patients undergoing radiotherapy?

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# OSTEORADIONECROSIS OF JAW

## Answer 8

**Preventive measures include smoking cessation, good glycemic control in diabetic patients, pre-radiotherapy dental assessment and completion of dentoalveolar surgery at least 2-3 weeks before radiotherapy, post-DAS follow-up after 2 weeks to ensure healing, and lifelong dental follow-up by a trained dental team.**

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OSTEORADIONECROSIS OF JAW

## Question 9

# How does hyperbaric oxygen therapy (HBOT) assist in the management or prevention of ORNJ?

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# OSTEORADIONECROSIS OF JAW

## Answer 9

**HBOT increases oxygen transfer to tissues under pressures greater than 1.4 ATA, promoting wound healing, increasing tissue oxygenation, and reducing recovery time, which helps prevent and manage complications from radiotherapy including ORNJ.**

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ORAL MEDICINE

# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# OSTEOPOROSIS: PREVENTION AND MANAGEMENT IN OLDER PEOPLE

## Epidemiology and Risk Factors

- Affects both men and women; women higher risk due to estrogen decline after menopause.v
- Osteoporotic fractures result from decreased bone strength combined with injurious falls.

## Diagnostic Tests

- DEXA scan for bone mineral density (BMD).
- Additional tests: X-rays, calcium and parathyroid hormone levels, vitamin D levels, blood tests including renal and liver function, thyroid, inflammatory markers, and testosterone.

## Vitamin D Role and Supplementation

- Maintains calcium and phosphate balance; essential for bone health and muscle function.
- Recommended vitamin D intake: at least 800 IU daily with calcium supplements
- Vitamin D status measured by serum 25-hydroxyvitamin D [25-OHD] levels; deficiency categorized from mild to severe.
- Dietary sources limited; sunlight exposure is main source.

## Pharmacological Management

- Bisphosphonates (e.g., alendronate, risedronate) are first-line treatments to reduce fracture risk by 30-70%.
- Other options: raloxifene, strontium ranelate, denosumab.
- Consider treatment for patients with fractures, low T-scores, or prolonged corti costeroid use.

## Drug Holidays and Long-Term Treatment

- Bisphosphonates effects persist after stopping; treatment usually 3-5 years with possible breaks ("drug holidays").
- Denosumab effects decline quickly after stopping; should not be interrupted due to fracture risk
- Drug holidays recommended in low-risk patients; duration varies by medication type.

## Summary of Key Points

- Osteoporosis increases fracture risk due to bone loss and fragility.
- Adequate calcium and vitamin D intake critical for prevention and treatment.
- Multiple pharmacological options exist, with bisphosphonates and denosumab leading.
- MRONJ is a rare but serious complication; dental care before and during treatment vital.
- Long-term management includes consideration of drug holidays to balance benefits and risks.

## Definition and Diagnosis

- Osteoporosis is a disease characterized by low bone mass and deterioration of bone tissue, increasing fracture risk.
- Diagnosed using bone density tests (DEXA), focusing on hip and spine, producing a 'T-score' indicating normal, osteopenia, or osteoporosis.

## Types of Osteoporosis

- Primary osteoporosis: postmenopausal and age-related.
- Secondary osteoporosis: caused by drugs (e.g., corticosteroids) or diseases (e.g., rheumatoid arthritis).

## Calcium Intake and Supplementation

- Recommended daily calcium intake: 1000-1300 mg depending on age and gender.
- Supplements of 500-600 mg elemental calcium advised if dietary intake is insufficient.
- Some studies suggest calcium supplements may slightly increase heart attack risk; dietary calcium does not show this risk.

## Lifestyle Prevention

- Smoking cessation, maintaining healthy weight, limiting alcohol, and physical activity reduce fracture risk though direct evidence on bone density is limited.

## Medication-Related Osteonecrosis of the Jaw (MRONJ)

- Rare adverse effect mainly in cancer patients on high-dose antiresorptives; lower risk in osteoporosis treatment.
- Risk factors include dental surgery and long duration/high dose antiresorptive therapy.
- Prevention involves dental assessment before starting therapy and maintaining good oral hygiene.

## Monitoring and Patient Communication

- Regular dental and medical reviews essential during treatment.
- Efective communication between healthcare providers and patients improves of utcomes and adherence.
- Treatment decisions should consider fracture risk, patient preference, and drug subsidy eligibility.

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 1

**What is osteoporosis and how is it diagnosed?**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 1

**Osteoporosis is a disease characterized by low bone mass and deterioration of bone tissue microarchitecture, leading to increased bone fragility and higher fracture risk. It is diagnosed using bone density tests that measure density at the hip and spine, resulting in a T-score that classifies bone health as normal, osteopenia, or osteoporosis.**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 2

**Why are women at greater risk of developing osteoporosis compared to men?**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 2

**Women are at greater risk mainly due to the rapid decline in estrogen levels after menopause. When estrogen decreases, bones lose calcium and other minerals faster, increasing the risk of osteoporosis.**

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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 3

# What are the two main types of osteoporosis?

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 3

**The two main types are primary osteoporosis, which includes postmenopausal and age-related osteoporosis, and secondary osteoporosis, which is caused by identifiable agents like corticosteroids or diseases such as rheumatoid arthritis.**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 4

**What daily calcium intake does Osteoporosis Australia recommend, and how should it ideally be achieved?**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 4

**Osteoporosis Australia recommends a total daily calcium intake of 1000–1300 mg depending on age and gender. Ideally, this should be achieved through a diet rich in naturally calcium-containing foods and calcium-enriched foods. Supplements are recommended if dietary intake is insufficient.**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 5

**What is the recommended vitamin D intake to accompany calcium supplements for osteoporosis treatment?**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 5

**It is recommended that calcium supplements be taken with at least 800 IU of vitamin D (colecalciferol) per day to optimize efficacy and reduce risk.**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 6

**What are the main sources of vitamin D and how is it produced in the body?**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 6

**The main source of vitamin D is skin exposure to sunlight, specifically UVB radiation, which converts 7-dehydrocholesterol in the skin to vitamin D3 (colecalciferol). Vitamin D3 can also be obtained in small amounts from foods like wild-caught fatty fish, liver, eggs, and fortified products.**

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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 7

# What are the key risk factors for medication-related osteonecrosis of the jaw (MRONJ) in patients on antiresorptive therapy?

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 7

**Risk factors include long duration and high doses of antiresorptive therapy, bone-invasive dental procedures (e.g., extractions, implants), poor oral health, and concomitant use of antiangiogenic drugs. MRONJ can also occur without dental procedures in cases of poorly fitting dentures or exostoses.**

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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 8

# What are the first-line pharmacological treatments for osteoporosis, and how much can they reduce fracture risk?

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 8

**First-line treatments include bisphosphonates (risedronate, alendronate, zoledronic acid), strontium ranelate, and selective estrogen receptor modulators (e.g., raloxifene). These medications reduce fracture risk by 30–70%, depending on the drug and patient factors.**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 9

**What is the purpose of a ‘drug holiday’ in bisphosphonate therapy, and who might be suitable for it?**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 9

**A ‘drug holiday’ is a temporary pause in bisphosphonate treatment after 3–5 years to reduce the risk of long-term adverse effects. It is suitable for patients at low to moderate fracture risk with stable bone mineral density and no recent fractures, with ongoing monitoring during the holiday.**

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# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Question 10

**How does denosumab differ from bisphosphonates in mechanism and treatment effects?**

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CUE CARDS

# OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

## Answer 10

**Denosumab is a reversible antiresorptive that inhibits osteoclast-mediated bone resorption and is administered every six months. Unlike bisphosphonates, it is not bound to bone.**

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ORAL MEDICINE

# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Overview of Oral Candidiasis

- Oral candidosis is the most common fungal infection in dental practice
- Presents in various clinical forms, sometimes mimicking serious diseases

## Candida and Oral Environment

- Candida species are normal oral commensals.
- Infection is uncommon in healthy adults but common in neonates

## Pseudomembranous Candidiasis

- Creamy white removable plaques with red, raw bleeding base
- May involve oropharynx and dorsal tongue with autoinoculation
- Management: treat predisposing factors, topical antifungals, specialist referral if needed

## Hyperplastic Candidiasis

- Non-removable white plaques, nodular and often bilateral
- Higher risk of dysplasia and possible malignant transformation
- Requires biopsy and specialist management

## Angular Cheilitis (Angular Stomatitis)

- Painful erythema and fissures at mouth corners
- Mixed infection with Candida, Staphylococcus aureus, Streptococcus species
- Linked with denture issues, nutritional deficiencies, and skin conditions
- Management includes dental review and topical antifungals

## Diagnosis and Referral Considerations

- Red flag signs warrant specialist referral
- Consider HIV testing in unexplained oral candidiasis
- Immunocompromised patients require specialist advice for management

## Summary of Clinical Presentations

- Pseudomembranous: removable white plaques
- Erythematous: red, raw areas often linked to dentures
- Hyperplastic: non-removable white plaques with dysplasia risk
- Angular cheilitis: fissuring and erythema at mouth corners
- Denture stomatitis: erythema confined to denture areas

## Risk Factors and Predisposing Conditions

- A Immunocompromised states, HIV infection, extremes of age
- Long-term antibiotics, corticosteroids, and psychotropic drugs
- Uncontrolled diabetes mellitus and terminal illness

## Erythematous Candidiasis

- Sensitive red lesions on palate and tongue, sometimes depapillated
- Symptomatic form causes burning sensation; asymptomatic form is denture stomatitis
- Trauma from ill-fitting dentures promotes colonization

## Mucocutaneous Candidiasis

- Early onset oral lesions preceding skin and nail involvement
- Part of a wider mucocutaneous infection involving multiple sites

## Denture-Associated Erythematous Stomatitis

- Red, sensitive lesions in denture-bearing areas, often asymptomatic
- Caused by ill-fitting dentures, poor hygiene, and nocturnal denture use
- Treatment focuses on hygiene, denture removal at night, and antifungal therapy if needed

## Management Principles

- Thorough investigation of underlying systemic conditions
- Use of topical antifungal agents as first-line treatment
- Specialist involvement for refractory or complicated cases

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# MIND MAP

# MEDIAN RHOMBOID GLOSSITIS AND ORAL CANDIDIAS IS: CLINICAL FEATURES AND MANAGEMENT

## Antifungal Therapy for Oral Candidiasis: General Principles

- Treatment requires adequate contact time; avoid eating/drinking immediately after application
- Neonates and children under 2 treated with topical antifungals applied to front mouth to prevent choking
- Adults and children over 2 treated with topical gels, lozenges, or suspensions for7-14 days
- Denture wearers should apply antifungal to denture fitting surfaces twice daily and maintain hygiene

## Treatment Protocols for Adults and Older Children

- Options include miconazole 2% gel, amphotericin B lozenges, or nystatin suspension, 4 times daily after food
- Continue treatment at least 7 days after symptom resolution
- Denture care essential prevent reinfection

## Diagnostic Methods for Oral Candidiasis

- Direct smear: scraping afected area, staining with PAS, microscopy for yeast and hyphae
- Culture: saliva mouth rinse cultured on Sabouraud's agar to quantify candidal colonies
- Biopsy: used especially for hyperplastic candidosis, to diferentiate from malignancies

## Recommended Treatment Protocol and Hygiene

- Eliminate e reduce predisposing factors where possible
- Maintain denture hygiene: remove at night, clean thoroughly, soak twice weekly in diluted bleach or vinegar
- Apply topical antifungals liberally to denture fitting surfaces four times daily for 3 weeks
- Reassess after 3 weeks; if no improvement, verify compliance, diagnosis, and predisposing factors

## Summary: Key Points in Oral Candidiasis and Median Rhomboi Glossitis Care

- Accurate diagnosis through history, clinical exam, and confirmatory tests needed
- Tailor antifungal treatment by age, condition, and presence of dentures
- Emphasize oral and denture hygiene to prevent recurrence
- Monitor systemic health and address underlying causes
- Specialist referral when standard treatment
- ineffective or complicated disease is suspected

## Clinical Features of Median Rhomboid Glossitis

- Rhomboid-shaped depapillated and erythematous area on midline dorsal tongue
- May appear fissured or nodular, sometimes involving palate autoinoculation
- Usually asymptomatic but may cause stinging or burning sensations
- Located anterior to circumvallate papillae, composed of atrophic filiform papillae.
- Candida hyphae found in over 85% of biopsies, associated with smoking and inhaled steroids

## Treatment Protocols for Neonates and Young Children

- Use nystatin suspension 100,000 units/mL or miconazole 2% gel, 4 times daily after feeding
- Continue treatment 2-3 days after symptoms resolve
- Apply dose carefully to avoid choking hazards

## Management of Angular Cheilitis

- Topical antifungal creams: clotrimazole 1% or miconazole 2%, applied twice daily for at least 14 days
- Mild topical corticosteroids (hydrocortisone 1%) may be used alongside antifungal for inflammation
- Combination corticosteroid-antifungal products used only until inflammation su bsides
- Specialist referral advised for persistent or complicated cases

## Principles of Management: Investigations and Underlying Causes

- Assess denture adequacy and hygiene
- Screen for systemic conditions: vitamin B12, folate, iron deficiency, diabetes, immune deficiency
- Review medication history for antimicrobials or corticosteroids
- Consider immune status and other medical conditions

## Management of Refractory or Recurrent Cases

- Consider changing antifungal agents if initial treatment fails
- Reserve systemic antifungals for debilitated or immunocompromised patients
- Regular review and repeated therapy often necessary for immunocompromised individuals
- Refer non-responders to oral medicine specialists or maxillofacial surgeons

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 1

**What is oral candidosis and why is it significant in general dental practice?**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 1

**Oral candidosis is the most common fungal infection encountered in general dental practice. It manifests in various clinical presentations that may mimic more serious diseases and can sometimes be resistant to treatment, requiring specialist care. It often occurs when the patient is systemically compromised.**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 2

**What are common risk factors for developing oral candidiasis?**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 2

**Common risk factors include immunocompromised states such as HIV infection, extremes of age (neonates and elderly), uncontrolled diabetes mellitus, long-term use of broad-spectrum antibiotics, corticosteroids or anti-mitotic drugs, psychotropic drugs, and terminal illness.**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 3

**Describe the clinical features of pseudomembranous candidiasis.**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 3

**Pseudomembranous candidiasis presents as creamy white curds, papules, or plaques that can sometimes be removed, revealing a red, raw, and often bleeding base. It is generally asymptomatic and may affect the oropharynx. In infants, it is usually superficial and easy to manage, but in debilitated patients, it may be widespread with angular cheilitis and esophageal involvement.**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 4

**What are the clinical characteristics and management considerations for erythematous candidiasis?**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 4

**Erythematous candidiasis features sensitive red lesions commonly on the palate and tongue, with a depapillated, smooth tongue. It can be symptomatic (burning sensation) or asymptomatic (localized chronic erythema under dentures, known as denture-induced stomatitis). Management includes addressing predisposing factors and using topical antifungal therapy.**

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CUE CARDS

# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 5

**What distinguishes hyperplastic candidiasis from other forms of oral candidiasis?**

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CUE CARDS

# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 5

**Hyperplastic candidiasis presents as asymptomatic, non-removable white plaques that may appear nodular, usually affecting areas like retro-commissures, anterior buccal mucosa, and lateral tongue. It may resemble oral leukoplakia or cancer and is associated with epithelial dysplasia, thus requiring specialist referral and biopsy.**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 6

**What is angular cheilitis, and what are its common causes?**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 6

**Angular cheilitis is characterized by painful erythema and fissuring at the mouth corners, caused by mixed infections including Candida, Staphylococcus aureus, and Streptococcus species. It is often linked to deep skin folds, ill-fitting dentures, nutritional deficiencies (iron, folate, vitamin B12), Crohn disease, and dermatitis.**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 7

**How should denture-associated erythematous stomatitis be managed?**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 7

**Management includes optimizing denture hygiene, advising patients to remove dentures at night, clean and dry them, and conducting a dental review to assess denture fit. If symptoms persist after one month, topical antifungal therapy should be applied inside the mouth and on the dentures.**

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CUE CARDS

# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 8

**What are the clinical features and typical management of median rhomboid glossitis?**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 8

**Median rhomboid glossitis appears as a rhomboid area of depapillation and erythema in the midline of the dorsal tongue, sometimes fissured or nodular. It is usually asymptomatic but may sting or burn. Management involves addressing predisposing factors and using topical antifungal therapy.**

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CUE CARDS

# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 9

**What are the recommended antifungal treatment options for oral candidiasis in neonates and children under 2 years?**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 9

**Recommended treatments include topical nystatin suspension (100,000 units/mL, 1 mL, four times daily after feeding for 7-14 days) or miconazole 2% gel (1.25 mL, four times daily after feeding for 7-14 days). Treatment should continue for 2-3 days after symptoms resolve. In infants, application should be at the front of the mouth to avoid choking.**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Question 10

**What diagnostic methods are used to confirm oral candidiasis and when should biopsy be considered?**

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# ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

## Answer 10

**Diagnosis is based on clinical features, but confirmation can be done by direct smear examination stained with PAS, culture of saliva or mouth swill on Sabouraud's agar, and biopsy. Biopsy is particularly important for hyperplastic candidiasis to detect epithelial dysplasia and differentiate from oral cancer.**

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ORAL MEDICINE

# OROFACIAL PAIN

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# OROFACIAL PAIN: DIFFERENTIAL DIAGNOSIS AND MANAGEMENT

## Understanding Orofacial Pain

- Patients often self-diagnose as toothache, seeking dental solutions
- Correct diagnosis is crucial before irreversible treatment

## Importance of Comprehensive Evaluation.

- Detailed pain history and thorough facial/intra-oral examination needed
- Use of diagnostic tests and specialist referral if treatment fails

## Fibromyalgia and Orofacial Symptoms

- Difuse musculoskeletal syndrome often poorly responsive to physical treatment
- Shares features with psychiatric conditions; no single efective treatment

## Psychiatric Factors in Orofacial Pain

- Atypical pain history and response to treatment; possible undisclosed health issues
- Risk of over-treatment without proper mental health consideration

## Headache Disorders and Orofacial Pain

- Migraines cause severe unilateral headache with nausea and photophobia
- Temporal arteritis is a severe headache needing urgent diagnosis to prevent blindness

## Diagnostic Approach Prior to Treatment

- Full head, neck, muscle, joint, nerve, and dental exam before treatment
- Referral to specialists if no clear dental pathology is found

## Role of Analgesics in Acute Dental Pain

- Analgesics modify pain sensation but don't treat cause; adjunct to dental care
- Use when dental treatment alone insufficient or post-surgical pain occurs

## Alternative and Combination Analgesics

- Paracetamol can be combined with NSAIDS or used alone NSAIDS contraindicated
- Avoid codeine; oxycodone preferred opioid if necessary

## Analgesic Regimens for Severe Acute Pain in Adults

- Three-drug regimen: ibuprofen or celecoxib + paracetamol + oxycodone immediate-release
- Lower opioid doses for elderly, limit duration, and prescribe small quantities

## Pain Management in Children

- No aspirin under 16 years due to Reye syndrome risk
- Use ibuprofen or paracetamol regularly for continuous pain relief; opioids avoided outside specialist care

## Muscular Causes of Orofacial Pain

- Myofascial Pain Dysfunction (MPD) and muscle tension headaches cause referred tooth pain
- Multiple teeth tender to lateral pressure, normal pulp tests and radiographs

## Neuralgic Pain Mimicking Toothache

- Neuralgias trigger pain in soft tissues, not teeth, with specific facial patterns
- Neuralgias rarely wake patients; toothache often wakes from sleep

## Temporomandibular Joint (TMJ) Disorders

- Pain, clicking, locking of jaw often linked to muscular or arthralgia issues
- Dental treatment may exacerbate TMJ problems; bite blocks can help

## Cancer-Related Orofacial Pain Concerns

- Early cancer usually painless; pain arises from nerve involvement or infection
- Exclusion via imaging and biopsy to address patient fears

## Types of Orofacial Pain

- Acute dental pain: usually nociceptive, resolves quickly with treatment
- Chronic pain: nociceptive, neuropathic, nociplastic or mixed, requires biopsych osocial management

## Preferred Analgesics and Considerations

- NSAIDS preferred for anti-inflammatory effects; ibuprofen preferred unless contraindicated
- Assess patient comorbidities before prescribing NSAIDS

## Analgesic Regimens for Mild to Moderate Pain in Adults

- Regular dosing of ibuprofen 400 mg + paracetamol 1000 mg recommended
- Avoid fixed-dose combinations to allow dose optimization

## Analgesic Use After Surgery and Opioid Stewardship

- Educate patients on typical pain course and when return for review
- Use opioids cautiously; taper off as healing progresses; avoid modified-release opioids

## Summary and Clinical Recommendations

- Diagnose pain type and origin before treatment
- Refer non-dental pain patients for medical assessment
- Tailor treatment based on pain type and duration for optimal outcomes



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# MIND MAP

# TEMPOROMANDIBULAR DISORDERS (TMD)

## Definition and Classification

- TMD refers to clinical problems involving masticatory muscles, temporomandib ular joints (TMJ), and related structures
- Subtypes include temporomandibular joint disorders and masticatory muscle disorders

## Diagnosis of TMD

- Accurate diagnosis requires detailed history, physical examination, and imaging
- Key signs and symptoms include pain, limited jaw movement, and joint noises

## Habits and Psychosocial Influences

- Lip and cheek biting, nail biting, chewing gum, and smoking can worsen TMD
- Chronic TMD often linked to psychological disorders like depression, anxiety, and PTSD

## Management Goals and Principles

- Aim to control symptoms, not necessarily cure
- Treatment tailored to diagnosis with goals including pain reduction and restoring function
- Focus on resuming normal daily activities

## Advanced and Specialist Treatments

- Botulinum toxin injections for symptom management if conservative care fails (of-label use, requires special training)
- Referral o oral medicine or maxillofacial specialists for severe or chronic cases
- Surgery rarely indicated, only for confirmed internal joint pathology unresponsive to other treatments

## Psychosocial and Multidisciplinary Considerations

- Psychological factors play a crucial role in TMD development and management
- Doctor-patient relationship and placebo effects influence treatment outcomes
- Specialist management may include cognitive behavioral therapy, relaxation techniques, sleep hygiene education, meditation, hypnotherapy, and biofeedback

## Bruxism and its Relationship with TMD

- Bruxism can cause muscle pain, joint pain, and tooth wear but may exist independently of TMD
- Signs include cracked teeth, failed restorations, masseter hypertrophy, and tongue scalloping
- Oral appliances protect teeth but may not reduce bruxism habit.

## Role of the General Dentist

- Essential in early diagnosis, risk assessment, education, and conservative management
- Referral to specialists for complex or chronic cases beyond general dentistry scope
- Awareness of patients' psychological status and medications important for holistic care

## Risk Factors for TMD

- Direct or indirect trauma (e.g., acceleration-deceleration injuries)
- Parafunctional habits such as teeth grinding (bruxism) and clenching
- Psychosocial factors including stress and anxiety
- Malocclusion is not a proven risk factor
- Childhood adverse experiences may increase risk of chronic pain development

## Clinical Examination Components

- Observation of facial symmetry, muscle hypertrophy, and habits
- Assessment of jaw movements: opening, closing, deviation, and trismus
- Palpation of TMJ for irregularities and joint sounds
- Examination of masticatory muscles
- Dental examination for attrition, cracks, mobility, and occlusal issues
- Radiographic imaging such as OPG when indicated

## Conservative Treatment Strategies

- Patient education and reassurance
- Jaw rest through dietary modification (soft foods) and avoiding extreme jaw movements
- Massage and warm packs applied to TMJ and muscles
- Behavioral modification including stress management and counseling
- Physiotherapy with gentle muscle stretching and massage
- Use of custom-made occlusal splints, mainly worn at night to reduce loading and pain
- Short-term drug therapy including analgesics, muscle relaxants, anxiolytics, corticosteroids, and antidepressants

## High-Risk Dental Procedures and Prevention.

- Long dental appointments (>30 minutes) and forceful procedures (e.g., extractions) can trigger or worsen TMD.
- Use of rubber dams or bite blocks, frequent rest breaks, and limiting mouth opening recommended
- Patients should be informed of risks when undergoing high-risk dental procedures

## Patient Education and Home Care

- Education about jaw rest, avoiding parafunctional behaviors, and symptom triggers
- Home care programs empower patients with self-management techniques
- Regular review and monitoring to adjust treatment as needed

## Special Considerations for Women and Chronic Pain

- Women seek treatment 4 times more than men and experience greater pain sensitivity
- TMD symptoms tend to persist longer in women
- Childhood trauma linked to increased chronic pain risk in youth, including TMD

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OROFACIAL PAIN

## Question 1

# What challenges do dentists face when diagnosing orofacial pain that mimics toothache but is not of dental origin?

# Answer 1

**Dentists often find it difficult to correctly identify non-dental causes of orofacial pain because symptoms closely mimic toothache. Misdiagnosis can lead to extended irreversible and expensive treatments. Careful evaluation of the patient's pain history, thorough facial and intra-oral examinations, and diagnostic tests are essential before initiating irreversible treatment.**

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OROFACIAL PAIN

## Question 2

**What are common characteristics of myofascial pain dysfunction (MPD) related orofacial pain?**

# Answer 2

MPD-related pain often presents as referred pain to the teeth and alveolus, with tenderness to lateral pressure on multiple teeth, especially the last in the arch. Pulp sensibility tests and radiographs are usually normal. Patients may report head or neck pain but often deny its relation to toothache. Examination typically reveals tenderness and dysfunction in masticatory and cervical muscles.

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OROFACIAL PAIN

## Question 3

# How does fibromyalgia present in patients with jaw pain, and how do they typically respond to treatment?

# Answer 3

**Fibromyalgia is a diffuse musculoskeletal syndrome affecting many muscles unilaterally or bilaterally, causing jaw pain that responds poorly to physical, non-surgical treatments. Patients often share features with psychiatric conditions. No single effective treatment exists, although various medical treatments have been reported.**

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OROFACIAL PAIN

## Question 4

# What diagnostic clues help differentiate neuralgic pains from acute pulpal toothache?

# Answer 4

**Neuralgic pain triggers are located in soft tissues, not hard tissues, and can extend outside the mouth (e.g., lower eyelid, lateral nose for maxillary neuralgias; tongue and gingiva for mandibular neuralgias). Neuralgias rarely wake patients at night, unlike pulpal toothache. Pain history is often longer, and nerve injury history may be present. Diagnostic nerve blocks can abolish neuralgic pain.**

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OROFACIAL PAIN

## Question 5

# What should dentists be aware of when managing patients with a psychiatric background presenting with orofacial pain?

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OROFACIAL PAIN

# Answer 5

**Dentists must spend time interacting with such patients to avoid misdiagnosis. Pain histories are often unusual, and treatment responses atypical. Clues include current or past medications and reluctance to share health history. Incorrect diagnosis risks overtreatment or unnecessary treatment.**

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CUE CARDS

OROFACIAL PAIN

## Question 6

# How should temporomandibular joint (TMJ) problems be approached in dental practice?

# Answer 6

**TMJ problems presenting with pain, clicking, or locking are less likely to be treated by dental procedures initially. Patients may attribute TMJ issues to dental treatment. Use of a bite block to support the jaw can reduce masticatory system strain, especially in those with muscle or joint pain history.**

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OROFACIAL PAIN

## Question 7

**What are typical symptoms and patient-reported experiences associated with migraine headaches compared to muscle tension headaches?**

# Answer 7

**Migraines are severe unilateral headaches often following the temporal artery, associated with nausea, vomiting, photophobia, and functional disability lasting hours to days. Muscle tension headaches are usually bilateral and less severe. Patients often confuse muscle tension headaches for migraines during dental consultations.**

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OROFACIAL PAIN

## Question 8

# What is temporal arteritis, and why is prompt diagnosis important?

# Answer 8

**Temporal arteritis is a severe, deeply boring headache, usually involving the temple, sometimes facial or lingual arteries. It mimics MPD but requires urgent diagnosis via raised ESR blood tests and arterial biopsy. If untreated, it can cause permanent unilateral blindness.**

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OROFACIAL PAIN

## Question 9

# Why do patients with deep-seated unresolved orofacial pain often fear cancer, and how should this fear be addressed?

# Answer 9

**Patients fear cancer because deep pain may suggest serious illness. Early-stage cancer is usually painless, becoming painful only when nerves or superficial tissues are involved. The fear should be addressed by excluding cancer through imaging (CT scans) and biopsy of any lesions.**

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OROFACIAL PAIN

## Question 10

# What is the recommended first step in managing orofacial pain in patients?

# Answer 10

**The first step is accurate diagnosis based on a detailed pain history from the entire head and neck area, not just dental symptoms. This includes thorough examination of jaw muscles, joints, sensory nerves, and teeth, along with diagnostic tests and consideration of diagnostic or therapeutic blocks before treatment.**

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OROFACIAL PAIN

## Question 11

# How should analgesics be used in managing acute dental pain?

# Answer 11

Analgesics modify pain sensation but do not treat the cause. They should be adjuncts to dental treatment, used when pain cannot be controlled by dental care, after surgery, or when dental care is delayed. Oral administration is preferred, with NSAIDs (like ibuprofen) preferred for nociceptive pain unless contraindicated, combined with paracetamol as needed. Opioids are reserved for severe pain when other measures fail.

## Question 12

**What is the preferred analgesic regimen for mild to moderate acute dental pain in adults?**

# Answer 12

**Regular dosing of ibuprofen 400 mg orally every 6-8 hours (max 5 days) plus paracetamol 1000 mg orally every 4-6 hours (max 4 g/day) for the shortest duration necessary is recommended. Fixed-dose combinations should be avoided. COX-2-selective NSAIDs like celecoxib may be used if NSAIDs are contraindicated, with paracetamol.**

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OROFACIAL PAIN

## Question 13

# What are the key points in managing severe acute dental pain in adults?

# Answer 13

**Use a three-drug regimen: ibuprofen or celecoxib plus paracetamol plus immediate-release oxycodone 5 mg orally every 4-6 hours as needed, for the shortest duration (no more than 3 days). Lower opioid doses for elderly/frail patients and avoid long-term opioid use. Taper analgesics stepwise as healing occurs.**

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OROFACIAL PAIN

## Question 14

# What considerations are important when prescribing analgesics for acute dental pain in children?

# Answer 14

**Avoid aspirin under 16 years due to Reye syndrome risk and opioids outside specialist settings. Use ibuprofen (5-10 mg/kg up to 400 mg thrice daily) or paracetamol (15 mg/kg up to 1000 mg four times daily), alone or combined, for shortest duration with regular dosing to maintain relief.**

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OROFACIAL PAIN

## Question 15

# What are common risk factors and management strategies for temporomandibular disorders (TMD)?

# Answer 15

Risk factors include trauma (macro and micro), parafunctional habits (e.g.,bruxism), psychosocial factors (stress, anxiety), and certain dental procedures. Management is conservative, focusing on symptom control via patient education, jaw rest, avoiding extreme jaw movements, muscle massage, warm packs, behavioral modification, physiotherapy, occlusal splints, and short-term drugs. Referral to specialists is for severe or chronic cases.

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ORAL MEDICINE

# ACTINIC CHEILITIS

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# ACTINIC CHEILITIS

## Definition and Clinical Significance

- Actinic cheilitis is a premalignant condition mainly affecting the vermilion of the lower lip caused by actinic (sun) radiation.
- Almost all lip carcinomas are associated with pre-existing actinic cheilitis, highlighting its clinical importance.

## Anatomical and Geographic Considerations

- The lower lip vermilion is highly exposed to ultraviolet radiation, especially at midday, and is poorly protected by melanocytes or keratin.
- Understanding lip geography is essential for identifying early pathological changes.

## Clinical Presentation and Early Signs

- Early signs include lip dryness and atrophy, which are more frequent than hyperkeratosis.
- White plaques develop later as a reactive change, often indicating disease progression.

## Differential Diagnosis

- Actinic cheilitis must be distinguished clinically from angular cheilitis, which has different causes and treatments.
- The term "photo-ageing" is discouraged as it dilutes the significance of sun-induced changes in the lip.

## Case Examples and Clinical Challenges

- Adolescents with early actinic cheilitis require proactive management including sun protection and monitoring of suspicious lesions.
- Non-homogeneous keratotic plaques warrant biopsy for histological diagnosis; homogeneity assessment guides urgency.

## Prevention and Patient Education

- Patients should limit sun exposure between 10 am and 2 pm (or 11 am and 3 pm during daylight saving), wear broad-brimmed hats, and apply SPF30+ broad-spectrum water-resistant sunscreen to lips.
- Use of emollients to maintain lip moisture is recommended.

## Epidemiological Considerations and Reporting

- Lip squamous cell carcinoma may be under-reported due to classification as non-melanoma skin cancer or lack of concurrent actinic cheilitis diagnosis.
- Accurate clinical recording and histological confirmation improve disease surveillance.

## Patient Demographics and Risk Factors

- Commonly affects fair-skinned, middle-aged to elderly men (40s to 80s) with a history of chronic sun exposure.
- Outdoor workers who smoke are at higher risk due to synergistic efects of UV radiation and carcinogens.

## Progression and Malignant Transformation

- Malignant signs: persistent ulceration, recurrent non-healing wounds, red and white blotchy areas, crusting, and nodules.
- Squamous cell carcinoma can develop without ulcer formation but should be suspected if ulcers occur.

## Clinical Assessment and Documentation

- Important tools include detailed clinical descriptions, measurements, diagrams, and photography under adequate lighting and magnification.
- Palpation of the lip surface is crucial for assessing tissue texture, induration, and fixation.

## Treatment and Management Approaches

- Treatment options include cryotherapy, electrosurgery, topical agents (retinoids,5-fluorouracil, imiquimod), photodynamic therapy, laser ablation, and surgical vermilionectomy.
- Patient-specific plans depend on disease severity, lifestyle, and sun exposure history.

## Role of Dental Practitioners

- Dentists are ideally placed to screen for actinic cheilitis during routine soft tissue exams with minimal additional effort.
- Collaboration with medical professionals is important for referral and comprehensive patient care.

## Long-Term Monitoring and Follow-Up

- Regular review is essential for mild cases, with immediate referral if progression or suspicious changes occur.
- Medium to long-term plans include ongoing sun protection and possibly surgical intervention to prevent malignancy.



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ACTINIC CHEILITIS

## Question 1

# What is actinic cheilitis and which part of the lip does it predominantly affect?

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# ACTINIC CHEILITIS

## Answer 1

**Actinic cheilitis is a potentially premalignant condition that predominantly involves the vermilion (the lip-stick surface) of the lower lip.**

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ACTINIC CHEILITIS

## Question 2

**What causes actinic cheilitis and why is the lower lip more affected than the upper lip?**

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# ACTINIC CHEILITIS

## Answer 2

Actinic cheilitis is caused by actinic (ultraviolet) radiation, which affects the skin and vermilion of the lip. The lower lip receives a higher dose of UV radiation because it lies at right angles to the midday sun and is poorly protected by melanocytes or keratin, making it more susceptible than the upper lip.

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ACTINIC CHEILITIS

## Question 3

# What are the typical demographic characteristics of patients with actinic cheilitis?

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# ACTINIC CHEILITIS

## Answer 3

**Typical patients are fair-skinned, middle-aged men, usually in their fourth to eighth decade of life, with a history of accumulated sun exposure, often outdoors workers, and sometimes cigarette users.**

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ACTINIC CHEILITIS

## Question 4

# What are the early clinical signs of actinic cheilitis?

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# ACTINIC CHEILITIS

## Answer 4

**Early clinical signs include dryness and atrophy of the vermilion, which is significant as the earliest change a clinician should notice. Hyperkeratosis may appear later as white plaques but is a late reactive change.**

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ACTINIC CHEILITIS

## Question 5

# What clinical features suggest malignant transformation of actinic cheilitis into squamous cell carcinoma?

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# ACTINIC CHEILITIS

## Answer 5

**Malignant changes may be indicated by persistent ulcers or recurrent ulceration that fails to heal, red and white blotchy appearance, loss of the vermilion border, persistent crusting and flaking, generalized atrophy with focal thickening, and focal induration or nodule formation.**

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ACTINIC CHEILITIS

## Question 6

# What is the recommended clinical assessment approach for a patient suspected of having actinic cheilitis?

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# ACTINIC CHEILITIS

## Answer 6

**Assessment should include detailed clinical description, measurement with a ruler, precise diagrams, clinical photography, and palpation under adequate lighting and magnification. Patient factors like age, skin type, sun exposure history, immune status, lifestyle, and clinical presentation must also be considered.**

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ACTINIC CHEILITIS

## Question 7

# How should keratotic plaques in actinic cheilitis be assessed and managed?

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# ACTINIC CHEILITIS

## Answer 7

**Keratotic plaques should be classified as homogeneous or non-homogeneous. Homogeneous plaques are uniform and less concerning, requiring regular review. Non-homogeneous plaques, showing variation in appearance, texture, or fixation, require biopsy and histological assessment.**

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ACTINIC CHEILITIS

## Question 8

# What are the typical treatment options available for actinic cheilitis?

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# ACTINIC CHEILITIS

## Answer 8

**Treatment options include both surgical and non-surgical methods such as cryotherapy, electrosurgery, topical retinoids, 5-fluorouracil cream, imiquimod cream, photodynamic therapy, carbon dioxide laser ablation, and surgical vermilionectomy.**

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ACTINIC CHEILITIS

## Question 9

# What preventive measures should all patients with actinic cheilitis follow?

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# ACTINIC CHEILITIS

## Answer 9

**Patients should**

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ORAL MEDICINE

# ORAL HERPES

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# ORAL MUCOCUTANEOUS HERPES: OVERVIEW AND MANAGEMENT

## Primary Oral Mucocutaneous Herpes

- Primary HSV infection (herpetic gingivostomatitis) mostly occurs in childhood with fever, painful intraoral lesions, malaise, and cervical lymphadenopathy
- Lesions start as blisters that ulcerate quickly and heal within days to two weeks; eating and drinking may be difficult, sometimes requiring hospitalization

## Clinical Features of Primary Infection

- Symptoms include fever, lethargy, painful oral ulcers, and swollen lymph nodes
- Healing time varies by age: faster in infants, slower in older children and adults

## Transmission and Prevention

- Advise patients with active HSV lesions to avoid direct contact with others to reduce viral spread
- Avoid sharing utensils, lip balms, and refrain from oral sex during active infection

## Special Considerations and Referral

- Refer patients with severe symptoms, immunocompromised status, or HIV for medical evaluation and specialized care
- Hospital admission may be needed for severe dehydration or dificulty swallowing

## Clinical Progression of Cold Sores

- Prodromal stage: pain, burning, itching before blisters appear
- Blister stage: clusters of fluid-filled vesicles on lips
- Weeping stage: blisters rupture, releasing infectious fluid
- Scabbing stage: crusts form and cause itching or pain
- Healing stage: scabs fall of, skin returns to normal in about a week

## Management of Recurrent Herpes

- Episodic antiviral therapy can reduce outbreak duration: topical aciclovir 5% cream or oral famciclovir at first sign of recurrence
- Prolonged use of aciclovir cream beyond 5 days has no added benefit

## Complementary and Alternative Therapies

- Ice application can relieve burning and pain
- Propolis extract may reduce healing time and pain (product claims)
- Natural products containing menthol, camphor, or melaleuca oil may be preferred by some patients

## Lifestyle Measures to Reduce Recurrence and Transmission

- Use sun protection to avoid triggers like sunburn and wind damage
- Minimise stress and manage menstrual cycle trigers with medical advice
- Avoid touching lesions, pick at scabs, and maintain hand hygiene when applying topical treatments
- Avoid oral contact and sharing personal items during active outbreaks to prevent spreading HSV

## Differential Diagnosis

- HSV is the most common cause of mouth ulcers, but varicella zoster, coxsackie virus, and cytomegalovirus can cause similar lesions
- Herpetic gingivostomatitis lesions are widespread in the mouth, unlike necrotising gingivitis which is rare in children and localized to gums

## Management of Primary Herpes

- Supportive treatment: oral fluids, antipyretics, analgesics
- Topical anesthetics like benzydamine 1% gel or lidocaine viscous solution are recommended to relieve pain, with specific dosing guidelines for diferent ages

## Recurrent Oral Mucocutaneous Herpes (Herpes Labialis)

- Reactivation of latent HSV-1 in trigeminal ganglion causes cold sores mostly on lips, sometimes intraoral or on skin
- Recurrences are usually mild, lasting 7–10 days but can be prolonged or severe in immunocompromised patients

## Diagnosis of Recurrent Herpes Labialis

- Diagnosis based on history and clinical exam since microbiology tests cannot reliably diferentiate HSV from other ulcers
- Pharmacists should differentiate from impetigo, angular cheilitis, acne, ringworm, and other oral lesions by asking targeted questions

## Referral Criteria for Recurrences

- Refer if recurences are severe, systemic symptoms present, difficulty eating/swallowing, chronic or generalized lesions, or immunocompromised status

## Use in Pregnancy, Breastfeeding, and Children

- Pharmacists should exercise caution recommending products without pregnancy and breastfeeding safety data
- Refer to specific categorizations where available

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ORAL HERPES

## Question 1

# What are the common symptoms of primary oral mucocutaneous herpes simplex virus (HSV) infection in children?

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# ORAL HERPES

## Answer 1

**Common symptoms include fever, painful intraoral lesions, systemic symptoms such as malaise and lethargy, and cervical lymphadenopathy. The intraoral lesions begin as blisters and ulcerate rapidly, causing difficulty eating and drinking.**

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ORAL HERPES

## Question 2

**How long does healing typically take for primary oral mucocutaneous herpes in infants compared to older children?**

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ORAL HERPES

# Answer 2

**Healing occurs within several days in infants but can take up to 2 weeks in older children.**

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ORAL HERPES

## Question 3

# What distinguishes herpetic gingivostomatitis lesions from necrotising gingivitis in children?

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ORAL HERPES

# Answer 3

**Herpetic gingivostomatitis lesions are widespread and affect all soft tissues in the mouth, while necrotising gingivitis is rare in children and confined to the gingival tissues.**

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ORAL HERPES

## Question 4

# What is the recommended supportive management for minor primary oral mucocutaneous herpes?

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ORAL HERPES

# Answer 4

**Supportive management includes oral fluids, antipyretic drugs, analgesia, and topical anesthetics or analgesics such as benzydamine 1% gel or lidocaine viscous solution applied to the lesions.**

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ORAL HERPES

## Question 5

# Where does the herpes simplex virus lie dormant after the primary infection, and what can trigger its reactivation?

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ORAL HERPES

# Answer 5

**The virus lies dormant in the trigeminal ganglion and can be reactivated by non-specific stimuli such as illnesses associated with fever.**

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ORAL HERPES

## Question 6

# What are the typical clinical features of recurrent oral mucocutaneous herpes (herpes labialis)?

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ORAL HERPES

# Answer 6

Recurrent lesions usually appear as clusters of small ulcers preceded by a prodromal stage with pain, burning, tingling, or itching. Lesions commonly occur on the lips but can also appear on intraoral mucosa or other skin areas.

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ORAL HERPES

## Question 7

# Describe the five stages of cold sore progression.

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ORAL HERPES

# Answer 7

**Healing stage – scabs shrink and pink skin develops beneath**

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CUE CARDS

ORAL HERPES

## Question 8

# What antiviral treatments are recommended for minor recurrences of oral mucocutaneous herpes?

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ORAL HERPES

# Answer 8

**Episodic antiviral therapy includes topical aciclovir 5% cream applied five times daily for 5 days or oral famciclovir 1500 mg as a single dose, both started at the first sign of recurrence or during the prodromal stage.**

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ORAL HERPES

## Question 9

# What lifestyle measures can help reduce the risk of herpes simplex virus transmission and recurrence?

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ORAL HERPES

# Answer 9

Avoid direct contact with active lesions and sharing items that contact saliva, abstain from oral sex during outbreaks, use sun protection, moisturize to prevent wind damage, minimize stress, and avoid touching or picking at lesions.

## Question 10

**Which patients require referral to a medical practitioner for management of primary or recurrent oral mucocutaneous herpes?**

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ORAL HERPES

# Answer 10

**Referral is required for patients with severe presentations, immunocompromised patients, patients with HIV, those with difficulty eating or swallowing, and those with generalized or chronic herpes infection.**

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ORAL MEDICINE

# RECURRENT APHTHOUS ULCERS

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# RECURRENT APHTHOUS ULCERATIVE DISEASE (RAU)

## Types of RAU

- Minor RAU: Most common (80-90%), small round/oval ulcers on non-keratinized mucosa, heal in 1-2 weeks without scarring.
- Major RAU: Less common (5-10%), larger ulcers (>10mm) on any oral mucosa, persistent with scarring.
- Herpetiform Ulceration: Small numerous ulcers (~1mm), can coalesce, heal in 2 weeks without scarring.

## Pathogenesis and Aetiology

- Immune-mediated with unclear triggers.
- Possible triggers include local trauma, hematological, gastrointestinal, immunologic, genetic, nutritional, allergic, psychological, and medication factors.
- Systemic diseases (e.g., inflammatory bowel disease) can influence RAU activity.

## Differential Diagnosis and Systemic Associations

- Check for deficiencies: iron, vitamin B12, folate, zinc.
- Investigate systemic diseases: coeliac disease, ulcerative colitis, Behçet syndrome, PFAPA syndrome (in children).
- Persistent ulcers >2 weeks require specialist referral to exclude malignancy.

## Management Strategies

- Aim to treat existing lesions rather than prevent outbreaks.
- Topical corticosteroids (e.g., hydrocortisone 1%) applied 2-3 times daily.
- Pain relief with topical anesthetics like benzydamine 1% gel or lidocaine 2% viscous solution.
- Refer major, herpetiform, or immunocompromised cases to specialists.

## Prognosis and Recurrence

- Ulcers tend to recur every few weeks or months; some patients rarely ulcer-free.
- Healing usually occurs without scarring in minor and herpetiform forms; major RAU may scar.

## Referral and Specialist Care

- Refer cases with ulcers not healing after 2 weeks.
- Specialist input needed for systemic disease-associated ulcers or immunocompromised patients.

## Overview of RAU

- RAU is the most common recurrent oral ulceration affecting up to 20% of people.
- Ulcers typically start in childhood or adolescence, with a slight female and genetic predisposition.

## Clinical Presentation Stages

- Prodromal: Symptoms without visible ulcer, early lymphocyte activity.
- Pre-ulcerative: Redness and swelling, lymphocytes invade basal epithelium.
- Ulcerative: Painful crateriform ulcers with pseudomembrane, lasts 3-7 days.
- Healing: Pain subsides, granulation tissue forms, epithelial closure occurs.
- Remission: Ulcer-free periods, triggered by various factors like diet or hormonal changes.

## Common Sites of Ulceration

- Non-keratinized mucosa: lip, cheek, lateral tongue margins, floor of mouth.
- Keratinized mucosa (in Major RAU): hard palate, dorsum of tongue, oropharynx.

## Diagnosis and Assessment

- Detailed history and oral examination essential.
- Identify 'red flags' and systemic symptoms.
- Children with aphthous ulcers should be assessed for systemic causes.

## Impact on Patients

- All forms cause significant discomfort affecting eating and speaking.
- Severe cases can lead to persistent ulcers and scarring.

## Prevention and Patient Advice

- Smoking cessation can trigger acute aphthous ulcers but usually resolves.
- Avoid trauma from toothbrushing or orthodontic appliances.
- Identify and avoid dietary triggers or premenstrual flare-ups.



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# RECURRENT APHTHOUS ULCERS

## Question 1

**What is recurrent aphthous ulceration (RAU) and how common is it in the population?**

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# RECURRENT APHTHOUS ULCERS

## Answer 1

**Recurrent aphthous ulceration (RAU) is the most common form of recurrent oral ulceration, affecting up to 20% of the population. It is characterized by recurrent, painful ulcers in the oral mucosa.**

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# RECURRENT APHTHOUS ULCERS

## Question 2

**At what age do recurrent aphthous ulcers typically first appear, and is there any gender or genetic predisposition?**

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# RECURRENT APHTHOUS ULCERS

## Answer 2

**RAU ulcers usually first appear in childhood or adolescence. There is a slight female predisposition, and some patients have a family history of similar ulcers, suggesting a genetic factor.**

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RECURRENT APHTHOUS ULCERS

## Question 3

# What are the three recognized forms of recurrent aphthous ulceration?

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# RECURRENT APHTHOUS ULCERS

## Answer 3

**Herpetiform ulceration (similar prevalence to major RAU)**

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# RECURRENT APHTHOUS ULCERS

## Question 4

**Describe the clinical features of minor recurrent aphthous ulceration.**

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# RECURRENT APHTHOUS ULCERS

## Answer 4

**Minor RAU ulcers are round or oval, usually 5 mm in diameter, occur on non-keratinized mucosa such as the lip, cheek, and lateral tongue margins, sparing the dorsum of the tongue, palate, and gingivae. They heal without scarring in 1 to 2 weeks and typically recur every few weeks or months.**

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RECURRENT APHTHOUS ULCERS

## Question 5

# What distinguishes major recurrent aphthous ulceration from minor RAU?

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# RECURRENT APHTHOUS ULCERS

## Answer 5

**Major RAU ulcers can occur anywhere in the oral mucosa including keratinized areas like the hard palate and dorsum of the tongue. They are larger than 10 mm, tend to last at least one month, heal with scarring, and usually only one or two ulcers appear at a time.**

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RECURRENT APHTHOUS ULCERS

## Question 6

# What are the characteristics of herpetiform ulceration?

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# RECURRENT APHTHOUS ULCERS

## Answer 6

**Herpetiform ulcers start as many small round ulcers about 1 mm in diameter, which can number up to 100, often coalescing into larger irregular ulcers. They commonly occur on non-keratinized mucosa but can affect any oral mucosa. Healing takes up to two weeks without scarring.**

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RECURRENT APHTHOUS ULCERS

## Question 7

# What is the proposed pathogenesis of recurrent aphthous ulcerative disease?

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# RECURRENT APHTHOUS ULCERS

## Answer 7

**RAU is believed to have an immune-mediated pathogenesis. The exact trigger is unknown, but factors such as local trauma, haematological, gastrointestinal, immunologic, genetic, nutritional, allergic, psychological, and medication-related causes have been identified as potential triggers.**

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RECURRENT APHTHOUS ULCERS

## Question 8

# What are the clinical stages of recurrent aphthous ulceration from onset to resolution?

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# RECURRENT APHTHOUS ULCERS

## Answer 8

**Remission – no lesions present**

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RECURRENT APHTHOUS ULCERS

## Question 9

# Which systemic conditions are associated with recurrent aphthous ulcers?

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# RECURRENT APHTHOUS ULCERS

## Answer 9

Associated systemic conditions include iron, vitamin B12, folate or zinc deficiency, coeliac disease, ulcerative colitis, Behçet syndrome, and PFAPA syndrome in children.

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RECURRENT APHTHOUS ULCERS

## Question 10

**What is the recommended management approach for recurrent aphthous ulcerative disease in adults?**

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# RECURRENT APHTHOUS ULCERS

## Answer 10

**Treatment focuses on symptom relief rather than prevention. Use topical hydrocortisone 1% cream or ointment applied 2-3 times daily after meals for lesions. For pain relief, use topical anaesthetics such as benzydamine 1% gel or lidocaine 2% viscous solution. Lesions not healing after 2 weeks should be referred for specialist assessment and biopsy.**

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ORAL MEDICINE

# ORAL LICHEN PLANUS

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

# WINSPERT MIND MAP

# ORAL LICHEN PLANUS (OLP)

## Epidemiology

- Affects 1-2% of the general adult population
- Higher prevalence in females, ratio ~3:1 to 3:2
- Mostly seen in adults over 40 but can affect younger adults and children

## Symptoms and Patient Experience

- About two-thirds report oral discomfort, ranging from sensitivity to severe pain
- Erosive and atrophic lesions cause stinging or burning, especially with spicy/acidic foods
- Lesions persist for years with alternating exacerbations and remissions
- Stress and anxiety often exacerbate symptoms

## Oral Lichenoid Reactions (OLR) and Drug Reactions

- Lichenoid drug reactions linked to NSAIDs, beta-blockers, ACE inhibitors, thyroid drugs
- Contact sensitivity from dental restorations (amalgam, composite) or toothpaste flavorings (cinnamates).
- Skin patch testing can identify allergens
- Lichenoid lesions may also arise from graft-versus-host disease or idiopathically

## Oral Cancer Risk

- OLP patients have an increased risk of oral squamous cell carcinoma (SCC), especially in atrophic, erosive, or plaque lesions
- Risk factors: tobacco, alcohol, betel quid, Candida infection
- Chronic inflammation and epithelial repair may promote carcinogenesis
- Regular monitoring and biopsy of suspicious lesions essential

## Lifestyle and Supportive Care

- Advise cessation of smoking, alcohol, and betel quid chewing to reduce cancer risk
- Encourage nutritious diet rich in fresh fruits and vegetables
- Psychological support may help manage stress-related exacerbations
- Improve oral hygiene and periodontal health, especially for gingival lesions
- Prompt referral to specialists for diagnosis, treatment, and long-term monitoring

## Summary and Key Points

- OLP is a chronic, immune-mediated oral mucosal disease with multiple clinical forms
- Diagnosis relies on clinical features supported by biopsy and immunofluorescence
- Management focuses on symptom control, lesion healing, and cancer risk reduction
- Regular specialist follow-up is essential due to potential malignancy and chronic nature
- Oral lichenoid lesions require careful evaluation to exclude drug or contact reactions

## Clinical Features

- Presents as white striations, papules, plaques, erythema, erosions, or blisters
- Afects mostly adults over 40, women more than men (1.4:1)
- Lesions are typically bilateral and mixed in clinical subtypes (reticular, plaque, erosive)
- Common sites: buccal mucosa, tongue, gingivae; other oral sites occasionally involved
- Fine white striae (Wickham's striae) are diagnostic and found peripherally around lesions

## Types and Oral Presentations

- Reticular: lace-like white striae
- Papular: small white papules
- Plaque form: white plaques, may mimic leukoplakia
- Atrophic: erythematous areas
- Ulcerative/Erosive: painful ulcerations with fibrinous exudate
- Bullous: rare blistering form

## Diferential Diagnosis and Diagnosis

- Requires biopsy to differentiate from chronic white/ulcerative lesions, malignan
- Direct immunofluorescence helps distinguish from pemphigus, pemphigoid, dermatitis herpetiformis
- Blood tests (ANA, liver function) and full blood examination recommended
- Exfoliative cytology not recommended for diagnosis

## Association with Other Conditions

- Possible association with hepatitis C virus, chronic active hepatitis, primary biliary cirrhosisons
- Increased incidence of Candida albicans infection in OLP patients
- Chronic inflammation may increase susceptibility to oral cancer-causing mutations

## Management and Treatment

- Corticosteroids are first-line treatment: topical, intralesional, or systemic depending on lesion severity
- Topical steroids applied 2-4 times daily after meals for localized lesions
- Generalized lesions may require steroid mouth rinse twice daily
- Control of Candida superinfection with topical polyene or azole antifungals
- Alternative drugs considered if systemic steroids contraindicated
- Monthly review during active treatment and 6-monthly follow-up after remission

## Specialist Referral Indications.

- Need for histological diagnosis and exclusion of malignancy
- Assessment of causative factors, co-morbidities, and cancer risk
- Patient education and management planning
- Initiation and adjustment of medical treatment
- Long-term review and repeat biopsy as indicated



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ORAL LICHEN PLANUS

## Question 1

# What are the common clinical features of oral lichen planus (OLP)?

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# ORAL LICHEN PLANUS

## Answer 1

Oral lichen planus commonly presents as white striations, white papules, white plaques, erythema, erosions, or blisters predominantly affecting the buccal mucosa, tongue, and gingivae. Lesions are typically bilateral and can appear as a mixture of clinical subtypes including linear or reticular white streaks on an erythematous background or central shallow ulcerations with a yellowish fibrinous exudate.

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ORAL LICHEN PLANUS

## Question 2

**What is the prevalence of oral lichen planus in the general adult population and which gender is more affected?**

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# ORAL LICHEN PLANUS

## Answer 2

**Oral lichen planus affects 1-2% of the general adult population and is more common in women than men, with a female to male ratio of approximately 1.4:1.**

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ORAL LICHEN PLANUS

## Question 3

# At what age does oral lichen planus predominantly occur?

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# ORAL LICHEN PLANUS

## Answer 3

**Oral lichen planus predominantly occurs in adults over 40 years old, although it can affect younger adults and children.**

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ORAL LICHEN PLANUS

## Question 4

# Which parts of the oral cavity are most commonly affected by oral lichen planus?

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# ORAL LICHEN PLANUS

## Answer 4

**The buccal mucosa, tongue (including dorsal, lateral, and ventral surfaces), and gingivae are the most commonly affected sites in oral lichen planus.**

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ORAL LICHEN PLANUS

## Question 5

# What is "desquamative gingivitis" and how does it relate to oral lichen planus?

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# ORAL LICHEN PLANUS

## Answer 5

**Desquamative gingivitis is a clinical term describing fiery red erythema affecting the entire width of the attached gingiva. It is frequently seen in oral lichen planus but is not unique to it and may occur in other oral dermatoses.**

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ORAL LICHEN PLANUS

## Question 6

# How do oral lichen planus lesions typically vary during periods of exacerbation and quiescence?

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# ORAL LICHEN PLANUS

## Answer 6

During exacerbation, lesions show increased erythema or ulceration with heightened pain and sensitivity. During quiescence, erythema and ulceration decrease, often leaving faint white striations, papules, or plaques, with minimal or no symptoms.

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ORAL LICHEN PLANUS

## Question 7

# What is the proposed link between psychological stress and oral lichen planus?

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CUE CARDS

# ORAL LICHEN PLANUS

## Answer 7

**Exacerbations of oral lichen planus have been linked to periods of psychological stress and anxiety, likely due to immune system imbalance triggered by stress.**

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ORAL LICHEN PLANUS

## Question 8

# Is oral lichen planus considered a premalignant condition?

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# ORAL LICHEN PLANUS

## Answer 8

**Oral lichen planus patients have an increased risk of oral cancer, but malignant transformation is controversial and occurs in less than 5% of patients who do not use tobacco. It is unlikely that OLP is inherently premalignant.**

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ORAL LICHEN PLANUS

## Question 9

# What factors might contribute to the increased oral cancer risk in patients with oral lichen planus?

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# ORAL LICHEN PLANUS

## Answer 9

**Possible factors include increased mucosal sensitivity to mutagens like tobacco, alcohol, betel quid, Candida albicans infection, and the chronic inflammatory and epithelial wound healing responses that may promote cancer-forming gene mutations.**

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ORAL LICHEN PLANUS

## Question 10

**What diagnostic procedures are essential to confirm oral lichen planus?**

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# ORAL LICHEN PLANUS

## Answer 10

**Biopsy is essential to differentiate OLP from other chronic white or ulcerative oral lesions and to exclude malignancy. Direct immunofluorescence can help distinguish OLP from other bullous diseases, and blood tests including ANA titre, blood biochemistry, and full blood examination should be included.**

## Question 11

# What are oral lichenoid drug reactions (LDR) and which drugs are commonly implicated?

# ORAL LICHEN PLANUS

## Answer 11

**Oral lichenoid drug reactions are lesions resembling OLP triggered by systemic drugs, often with a variable lag period. Common implicated drugs include non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme inhibitors, and beta-blockers.**

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ORAL LICHEN PLANUS

## Question 12

**How can contact sensitivity contribute to oral lichenoid lesions?**

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# ORAL LICHEN PLANUS

## Answer 12

**Contact sensitivity reactions may be triggered by dental restorations, prostheses, or toothpaste flavorings (especially cinnamates). Replacement of implicated dental materials and discontinuing certain toothpaste can help manage these lesions.**

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ORAL LICHEN PLANUS

## Question 13

# What is the Koebner phenomenon in relation to oral lichenoid lesions?

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# ORAL LICHEN PLANUS

## Answer 13

**The Koebner phenomenon refers to oral lichenoid lesions triggered by mechanical trauma such as calculus deposits, sharp teeth, rough dental restoration surfaces, cheek or tongue biting, or oral surgical procedures.**

## Question 14

# Why is candidal infection significant in oral lichen planus patients?

# ORAL LICHEN PLANUS

## Answer 14

**Candida albicans infection is more common in OLP patients, can worsen symptoms, and is recognized as a risk factor for oral cancer. Periodic candidal cultures or smears and treatment with topical antimycotics are recommended.**

## Question 15

# What is the association between oral lichen planus and hepatitis C virus (HCV)?

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# ORAL LICHEN PLANUS

## Answer 15

**Some studies show an association between OLP and HCV infection, chronic active hepatitis, and primary biliary cirrhosis, though a causal role for HCV in OLP has not been confirmed. Liver function tests should be considered in all OLP patients.**

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ORAL LICHEN PLANUS

## Question 16

# What is the mainstay of medical treatment for symptomatic oral lichen planus?

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# ORAL LICHEN PLANUS

## Answer 16

**Corticosteroids are the primary treatment due to their immunosuppressive effects. They can be administered topically, intralesionally, or systemically depending on lesion severity.**

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ORAL LICHEN PLANUS

## Question 17

# How should localized and generalized oral lichen planus lesions be treated with corticosteroids?

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# ORAL LICHEN PLANUS

## Answer 17

**Localized lesions are treated with topical corticosteroid ointments applied two to four times daily after meals. Generalized lesions are often managed with corticosteroid mouth rinses twice daily.**

## Question 18

# What precautions should be taken when using corticosteroids to treat oral lichen planus?

# ORAL LICHEN PLANUS

## Answer 18

**Clinicians should monitor for adrenal suppression, side effects, and contraindications such as hypertension, viral infections, diabetes, pregnancy, stomach ulcers, and osteoporosis risk. The lowest effective dose should be used.**

## Question 19

# What is the recommended frequency of follow-up for oral lichen planus patients during and after active treatment?

# ORAL LICHEN PLANUS

## Answer 19

**Patients should be reviewed monthly during active treatment until symptoms and lesions are controlled, then at least every six months for long-term monitoring and re-biopsy as needed.**

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ORAL LICHEN PLANUS

## Question 20

**What lifestyle advice should be given to oral lichen planus patients to reduce oral cancer risk?**

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# ORAL LICHEN PLANUS

## Answer 20

**Patients should eliminate smoking and alcohol consumption, maintain a nutritious diet rich in fresh fruits and vegetables, avoid betel quid chewing, and manage Candida albicans infections to lower cancer risk.**

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ORAL MEDICINE

# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# MEDICOLEGAL CONSIDERATIONS IN AGED POPULATION

## Principle-Based Ethical Theory

- Four principles: Autonomy, Beneficence, Non-maleficence, Justice
- Justice challenges include limited resources and poor care in aged facilities

## Beneficence and Non-maleficence

- Beneficence means promoting patient's best interests
- A Non-maleficence requires avoiding harm, balancing risks and benefits

## Informed Consent

- Consent must be given before treatment, can be express or implied
- Written consent required for complex treatments with significant risks

## Competence and Capacity Assessment

- Dementia and cognitive decline affect ability to consent
- Functional test: understand, retain, weigh information, decide, and communicat

## Guardianship and Power of Attorney

- Guardians appointed by courts to make decisions for incompetent adults
- Enduring power of attorney allows a trusted person to consent on behalf of the patient

## Case Study: Competence and Substitute Decision Making

- Elderly patient refused life-saving surgery due to cultural concerns
- Neuropsychological assessment deemed patient incompetent; sons consented
- Surgery successful, illustrating complexity of competence assessments

## Conclusions and Clinical Implications

- Dentists must understand ethical/legal consent frameworks
- Importance of assessing competence in aged patients
- Awareness of substitute decision makers' roles.
- Vigilance for elder neglect in all care settings

## Ethical Basis of Dental Treatment

- Dentist-patient relationship forms the foundation of dental ethics
- Dentists must act in the best interest of patients, respecting their decisions

## Respect for Patient Autonomy

- Patients have the right to make independent decisions
- Autonomy includes the right to refuse treatment, even if beneficial

## Justice in Dental Care

- Fair distribution of limited resources
- Challenges in providing adequate oral care t residents of aged care facilities

## Legal and Ethical Foundations of Informed Consent

- Legal duty to disclose necessary information for intelligent consent
- Ethical basis rooted in respect for autonomy and patient understanding

## Substitute Decision Makers

- Options: advance directives, guardians, enduring power of attorney, person responsible
- Substitute decision makers must act in the patient's best interests

## Family Consent Hierarchy

- Prioritized list: spouse, children, parents, siblings, grandparents, grandchildren, aunts/uncles, nephews/nieces
- Consent must always prioritize patient's best interests

## Elder Abuse Awareness

- Elder abuse includes neglect, abandonment, and failure to provide basic needs
- Dentists may identify abuse in care facilities or home environments



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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 1

**What is the primary ethical responsibility of a dentist in the dentist-patient relationship?**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 1

**The primary ethical responsibility of a dentist is to always act in the best interests of the patient, even if the patient chooses to reject the dentist’s advice.**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 2

**What are the four generally accepted principles of medical ethics applicable in dental practice?**

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CUE CARDS

# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 2

**The four principles are:**
**(1) Respect for patient autonomy,**
**(2) Beneficence,**
**(3) Non-maleficence, and**
**(4) Justice.**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 3

**What does the principle of respect for patient autonomy require from a dentist?**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 3

**It requires the dentist to help the patient make their own decisions freely and independently and to respect and follow those decisions.**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 4

**What is informed consent in dentistry and when is written consent particularly recommended?**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 4

**Informed consent is the conscious agreement of a patient to a course of treatment after understanding it. Written consent is recommended for complicated treatments or when there is potential for significant complications.**

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CUE CARDS

# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 5

**What is the legal basis for informed consent in dental treatment?**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 5

**Doctors and dentists have the duty to disclose all necessary facts to enable a patient to make an intelligent, rational decision before proceeding with treatment.**

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MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 6

# How is competence (capacity) defined and assessed in dental patients?

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 6

**Competence is the ability to understand, retain, believe, weigh information about treatment, make a decision, and communicate it. It is assessed functionally by evaluating these abilities.**

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CUE CARDS

# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 7

**What steps should be taken if a dental patient is deemed not competent to consent to treatment?**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 7

**The dentist should determine if there is a valid advance directive, a court-appointed guardian, an enduring power of attorney, or a person responsible to provide consent. If none applies and treatment is necessary to save life or prevent harm, treatment may be given under the principle of necessity.**

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CUE CARDS

# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 8

**What is the role of an enduring power of attorney in dental treatment decisions?**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 8

**An enduring power of attorney is a person appointed by a competent individual to make health decisions on their behalf if they become incapacitated, and must follow the donor’s wishes.**

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CUE CARDS

# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 9

**How does elder abuse manifest in the context of dental care?**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 9

**Elder abuse can include neglect, such as failure to provide adequate food, shelter, clothing, and medical or dental care, as well as abandonment by refusing to allow appropriate care.**

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CUE CARDS

# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Question 10

**Why is assessing competence in elderly patients sometimes challenging, as illustrated by the Victorian case mentioned?**

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# MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

## Answer 10

**Because patients may verbally understand risks but may not rationally weigh the benefits against risks due to cultural beliefs or cognitive impairment, making it difficult to determine true competence without specialist assessment.**

WINSPERT

ORAL MEDICINE

# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

MIND MAP & CUE CARDS

BY DR. JIGYASA SHARMA

WINSPERT

# MIND MAP

# ORAL CANCER AND OTHER WHITE LESIONS OF THE MOUTH

## Overview of White Lesions

- White lesions in the oral cavity are common with various causes: benign, potentially malignant, or malignant.
- Most white oral lesions are benign and reactive in nature.

## Benign White Lesions

- Include frictional keratoses, irritant contact dermatitis, smokeless tobacco keratoses.
- Caused by chronic irritation or trauma to oral mucosa.

## Linea Alba and Clinical Features

- Linea Alba: white keratotic line on buccal mucosa near occlusal plane, unilateral or bilateral.
- Color intensity and thickness vary; surface may be rough with irregular tags.

## Benign Alveolar Ridge Keratosis (BARK)

- Chronic friction injury on gingiva or alveolar ridge (retromolar pad).
- Histology shows hyper orthokeratosis, acanthosis, and slight papillomatosis.

## Histological Features of Frictional Keratosis

- Marked hyper parakeratosis with shaggy or shredded keratin surface.
- Surface bacteria more common on buccal mucosa and lips than tongue.

## Genetic and Rare White Lesions

- White Sponge Nevus (WSN): autosomal dominant, soft white plaques mainly on buccal mucosa.
- Hereditary Benign Intraepithelial Dyskeratosis (HBID): white plaques on mucosa with ocular involvement; rare autosomal dominant.

## Specific Contact Lesions

- Toothpaste ingredients: superficial sloughing producing white strings removable by finger without ulceration.
- Amalgam restorations: keratotic or lichenoid reactions, often resolve after amalgam removal.
- Cinnamon flavoring: causes contact stomatitis with sloughing, erythema, and hyperkeratotic lesions.

## Frictional Keratosis

- White patch due to chronic low-grade trauma like chewing or rubbing mucosa against teeth.
- Clinical appearance varies with trauma degree; includes rough, irregular surface with tags.

## Morsicatio Mucosae Oris (MMO)

- Chronic frictional keratosis with poorly defined, peeling, white plaques and papules.
- Common on buccal mucosa, lateral tongue, and lower labial mucosa traumatized by teeth.

## Diagnosis of White Lesions

- Clinical findings usually sufficient; biopsy required if lesion is in a high-risk area or etiology unknown.
- Provisional diagnosis should avoid leukoplakia if etiology is confirmed.

## Conditions Overlapping with Frictional Keratosis

- Proliferative Verrucous Leukoplakia (PVL): OMPD with high recurrence and malignant risk, often on gingiva.
- Leukoedema: asymptomatic, bilateral gray-white opalescent lesion that diminishes on mucosal stretching.

## Irritant Contact-Related Keratosis

- Keratotic changes where smokeless tobacco is placed (snuff dipper's lesion).
- Early: white-gray opalescent film with wrinkled surface; later: thickened, furrowed keratosis.

## Smokeless Tobacco Keratosis

- Keratotic changes where smokeless tobacco is placed (snuff dipper's lesion).
- Early: white-gray opalescent film with wrinkled surface; later: thickened, furrowed keratosis.

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# MIND MAP

# POTENTIALLY MALIGNANT AND MALIGNANT ORAL LESIONS

## Actinic Cheilitis

- Caused by long-term sun damage to the lower lip, especially in Caucasian males over 40.
- Presents as dry, mottled, opalescent skin with gray/white plaques and loss of vermilion border definition.

## Non-Healing Extraction Socket

- Persistence beyond 6 weeks may indicate possible alveolar carcinoma.
- Requires further investigation for malignancy suspicion.

## Squamous Cell Carcinoma (SCC)

- Mainly caused by tobacco and alcohol consumption but other factors exist.
- Clinical features vary widely; induration and fixation are key signs of malignancy.

## Erythroplakia and Erythroleukoplakia

- Red or mixed red and white patches with no other clinical or pathological diagnosis.
- Highest malignant transformation rate among potentially malignant oral lesions

## Diagnostic Aids

- Visual inspection under incandescent or halogen light remains primary screening method.
- Toluidine Blue (vital staining) highlights abnormal tissue but has limited screening reliability.
- Chemiluminescence tools (e.g., Vizilite) assist in detecting mucosal abnormalitieosis.
- Autofluorescence helps visualize tissue changes not seen by naked eye.
- Oral Brush Biopsy offers a minimally invasive diagnostic option.
- Scalpel biopsy and histopathology remain the gold standard for definitive diagnosis.

## Leukoplakia

- Leukoplakia is a predominantly white keratotic lesion of exclusion.
- Common in older males; located on lip, buccal mucosa, gingiva, floor of mouth, or tongue.

## Erosive Lichen Planus and Chronic Hyperplastic Candidiosis

- Malignant potential is uncertain but requires careful treatment and monitoring.
- Lesions should be followed closely due to possible risk of malignancy.

## Oral Cancer Overview

- Dentists play a critical role in detection, management, surveillance, and rehabilitation.
- High incidence in Australia linked solar irradiation, especially lip cancer in men.

## Clinical Features of Oral Cancer

- Lesions range from exophytic masses to deep chronic ulcers.
- White lesions can be homogeneous (consistent texture) or non-homogeneous (valriable texture).
- Non-homogeneous lesions require urgent referral or biopsy.

## Other Malignant Lesions: Pigmented Melanoma

- Rare but important malignant pigmented lesion in the oral cavity.

## Biopsy Recommendations

- Oral and Maxillofacial Surgeons ideally perform biopsies for suspicious or malignant lesions.
- Accurate histopathological diagnosis is essential for treatment planning and prognosis.

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 1

**What are the common etiologies of white lesions in the oral cavity?**

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Answer 1

**White lesions in the oral cavity have a variety of etiologies including benign, potentially malignant, and malignant causes.**

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 2

**What causes frictional keratosis in the oral cavity?**

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## Answer 2

**Frictional keratosis is caused by chronic and low-grade parafunctional habits such as chewing, constant rubbing, or sucking of the oral mucosa against the teeth.**

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 3

**Describe the clinical appearance of frictional keratotic lesions.**

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## Answer 3

**Frictional keratotic lesions have a rough surface with irregular tags, can be extensive involving the entire cheek and lips, and present as ill-defined gray or white papules and plaques. Severe trauma may cause erosions and ulcers, and a macerated, peeling appearance is common.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 4

# What is Morsicatio Mucosae Oris (MMO) and its key characteristics?

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Answer 4

**MMO is a form of chronic oral frictional keratosis with no malignant potential, characterized by poorly demarcated, rough, shaggy, peeling white plaques and papules on buccal mucosa, lateral tongue border, or lower labial mucosa.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 5

# What distinguishes benign alveolar ridge keratosis (BARK) histologically?

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## Answer 5

**BARK shows hyper orthokeratosis and acanthosis with slight papillomatosis, typically affecting the gingiva or alveolar ridge mucosa, especially the retromolar pad.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 6

# When is biopsy recommended for white oral lesions?

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## Answer 6

**Biopsy is recommended when the etiology of a white lesion is unknown or if the keratotic lesion is in a high-risk area to rule out Oral Potentially Malignant Disorder (OPMD).**

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## Question 7

# What are the histological features of frictional keratosis?

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## Answer 7

**Histology shows marked hyper parakeratosis with a shaggy or shredded keratin surface, surface fissures and clefts, and bacteria are usually present on the keratin surface, especially in buccal mucosa and lip biopsies.**

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 8

**Name two conditions that have overlapping clinical features with frictional keratosis.**

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Answer 8

**Proliferative Verrucous Leukoplakia (PVL) and Leukoedema have overlapping clinical features with frictional keratosis.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 9

# How can you differentiate leukoedema from frictional keratosis clinically?

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## Answer 9

**Leukoedema appears as an opalescent, gray-white film on bilateral buccal and labial mucosa that diminishes upon stretching, unlike frictional keratosis.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 10

# What is White Sponge Nevus (WSN) and how is it inherited?

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Answer 10

**WSN is a rare autosomal dominant geno-dermatosis presenting with asymptomatic thickened soft white plaques, most commonly on the buccal mucosa, distinguished histologically by prominent parakeratosis and acanthosis.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 11

# What are the causes and clinical presentation of irritant contact stomatitis?

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Answer 11

**Irritant contact stomatitis is caused by chemical irritants in toothpaste, mouthwashes, or dental restorations and can present as keratosis, ulceration, erythema, vesicles, or edema depending on exposure duration and concentration.**

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 12

**What is smokeless tobacco keratosis and how does it progress?**

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Answer 12

**Smokeless tobacco keratosis is keratotic changes where smokeless tobacco is placed; early lesions have a white to gray opalescent film with a wrinkled surface, progressing to more keratotic lesions with furrowing and epithelial thickening.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 13

# Define leukoplakia and its significance.

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Answer 13

**Leukoplakia is a predominantly white lesion that cannot be classified as any other definable lesion, serving as a diagnosis of exclusion and potentially malignant, commonly occurring in older males on lip, buccal mucosa, gingiva, floor of mouth, or tongue.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 14

# What are two critical clinical signs almost always present in oral cancer?

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# ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Answer 14

**Induration (hardening) and fixation of the lesion are two critical clinical signs present in almost all oral cancers.**

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ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

## Question 5

# What diagnostic aids are used for screening oral neoplastic lesions?

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## Answer 5

**Diagnostic aids include vital staining with toluidine blue, chemiluminescence devices like Vizilite, autofluorescence, oral brush biopsy, and the gold standard scalpel biopsy with histopathology.**