

ORAL MEDICINE

HIV ORAL MANIFESTATIONS

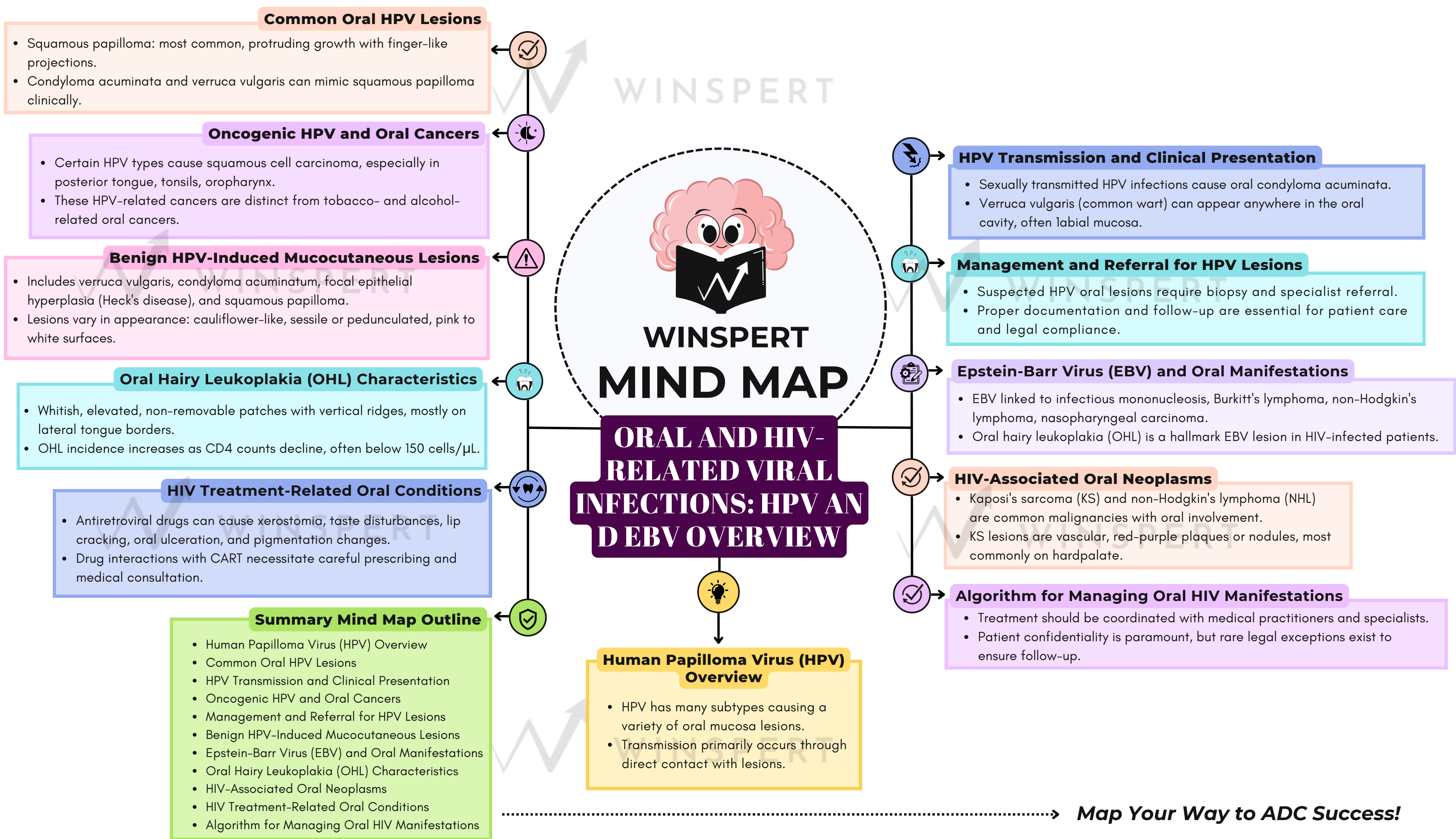


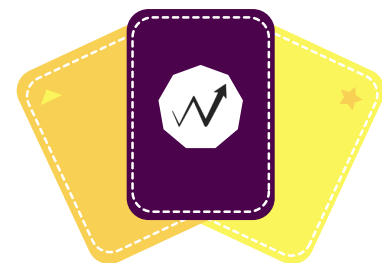
MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA







**WINSPERT
CUE CARDS**

**HIV ORAL
MANIFESTATIONS**

Question 1

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

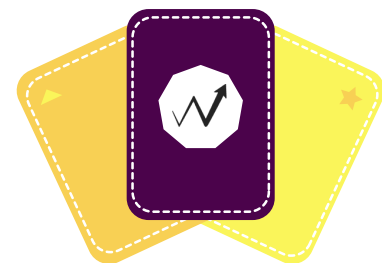


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**HIV ORAL
MANIFESTATIONS**

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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**HIV ORAL
MANIFESTATIONS**

Question 2

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

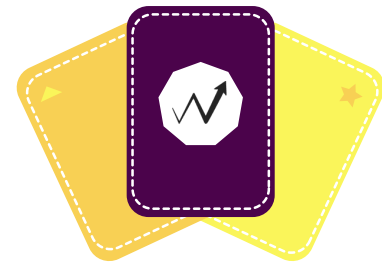


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**HIV ORAL
MANIFESTATIONS**

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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**HIV ORAL
MANIFESTATIONS**

Question 3

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

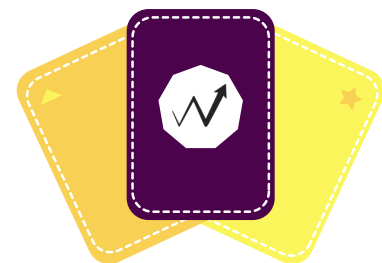


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**HIV ORAL
MANIFESTATIONS**

Answer 3

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



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**HIV ORAL
MANIFESTATIONS**

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.

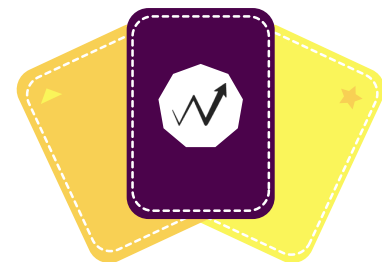


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**HIV ORAL
MANIFESTATIONS**

Question 5

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

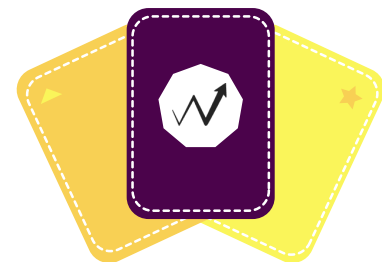


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**HIV ORAL
MANIFESTATIONS**

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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**HIV ORAL
MANIFESTATIONS**

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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**HIV ORAL
MANIFESTATIONS**

Question 7

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

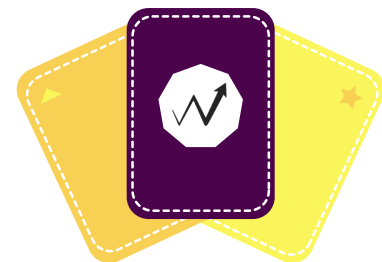


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**HIV ORAL
MANIFESTATIONS**

Answer 7

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



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**HIV ORAL
MANIFESTATIONS**

Question 8

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

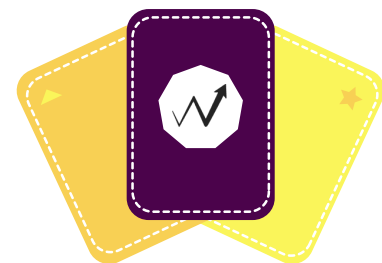


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**HIV ORAL
MANIFESTATIONS**

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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**HIV ORAL
MANIFESTATIONS**

Question 9

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



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**HIV ORAL
MANIFESTATIONS**

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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**HIV ORAL
MANIFESTATIONS**

Question 10

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



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**HIV ORAL
MANIFESTATIONS**

Answer 10

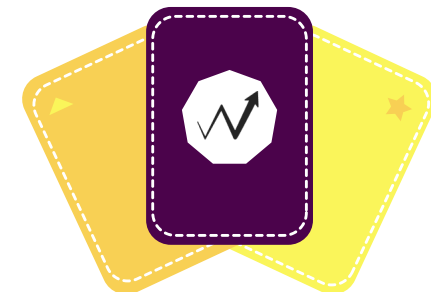
Kaposi's sarcoma (KS) and non-Hodgkin's lymphoma (NHL) are the two common malignancies with oral involvement in HIV infection.

ORAL MEDICINE

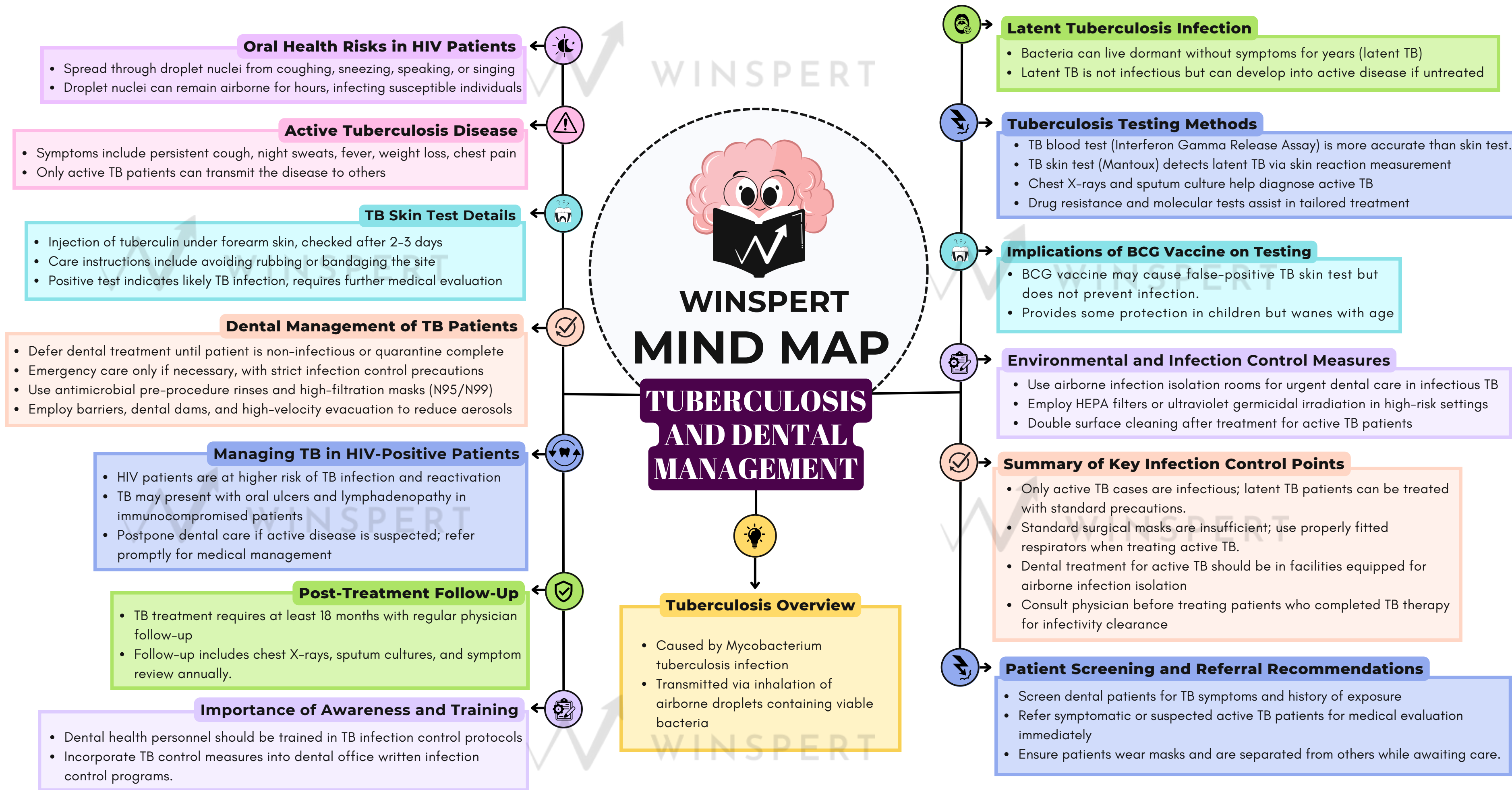
TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

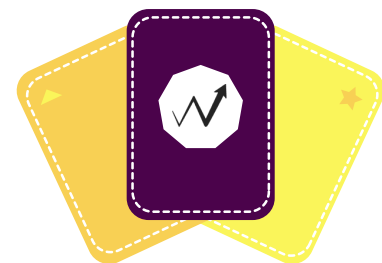


MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA



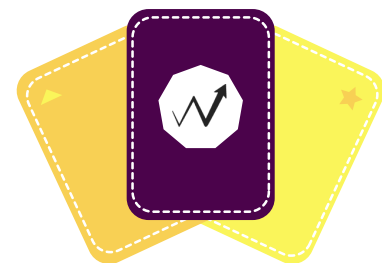


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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

Question 1

What causes tuberculosis and how is the infection transmitted?



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TUBERCULOSIS- ACTIVE /LATENT/ DENTAL MANAGEMENT

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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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

Question 2

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



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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

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.

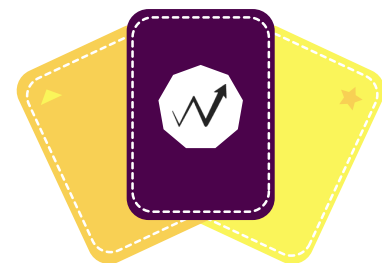


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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

Question 3

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

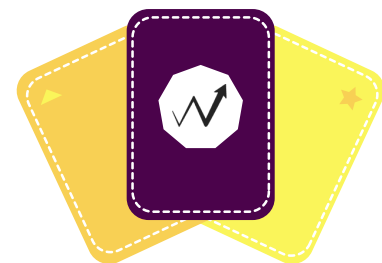


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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

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.

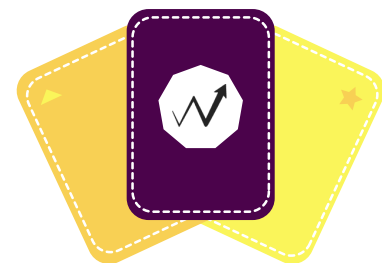


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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

Question 4

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



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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

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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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

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.

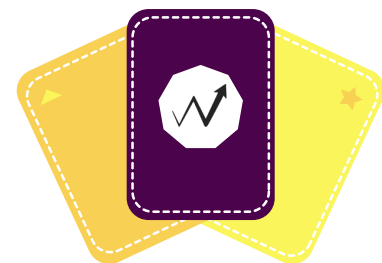


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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

Question 6

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

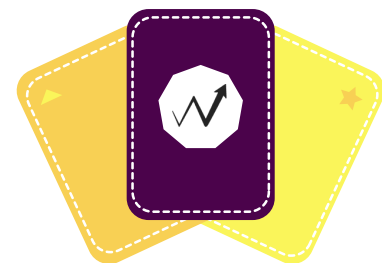


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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

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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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

Answer 7

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



**WINSPERT
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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?

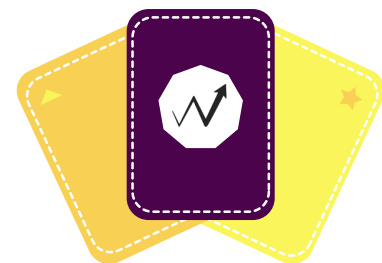


**WINSPERT
CUE CARDS**

**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?**

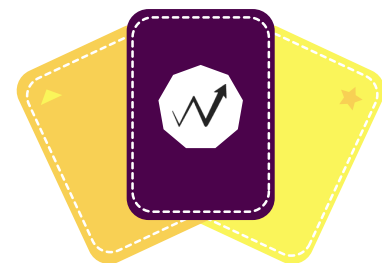


**WINSPERT
CUE CARDS**

**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

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.

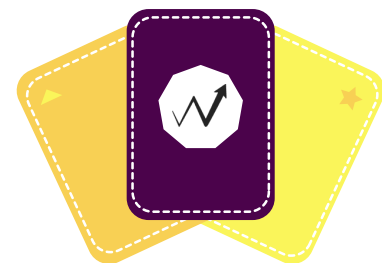


**WINSPERT
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**TUBERCULOSIS- ACTIVE
/LATENT/ DENTAL
MANAGEMENT**

Question 10

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



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CUE CARDS**

**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.

ORAL MEDICINE

BURNING MOUTH SYNDROME

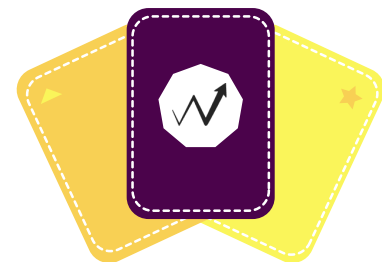


MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA



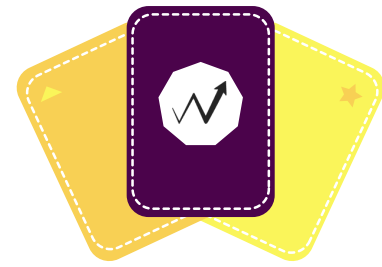


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**BURNING MOUTH
SYNDROME**

Question 1

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

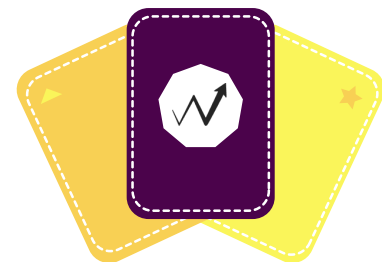


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CUE CARDS**

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?**

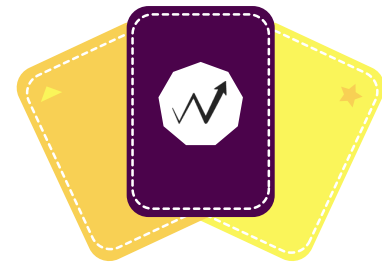


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BURNING MOUTH SYNDROME

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.

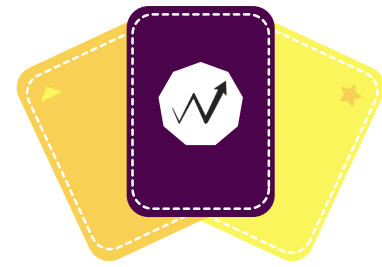


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**BURNING MOUTH
SYNDROME**

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.

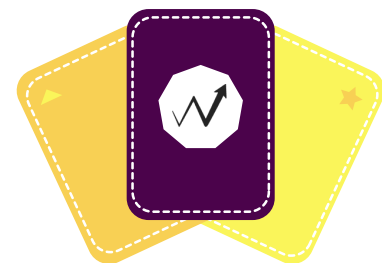


**WINSPERT
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**BURNING MOUTH
SYNDROME**

Question 4

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

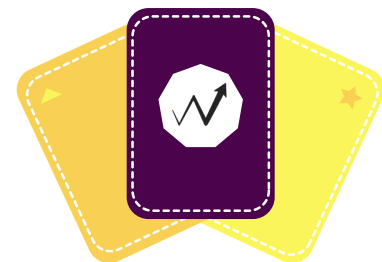


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CUE CARDS**

**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.

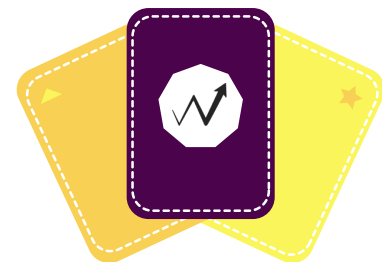


**WINSPERT
CUE CARDS**

**BURNING MOUTH
SYNDROME**

Question 5

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

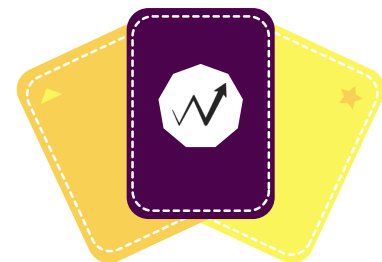


**WINSPERT
CUE CARDS**

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.

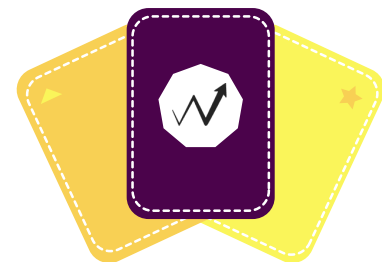


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**BURNING MOUTH
SYNDROME**

Question 6

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

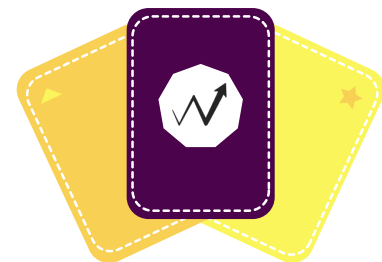


**WINSPERT
CUE CARDS**

**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.

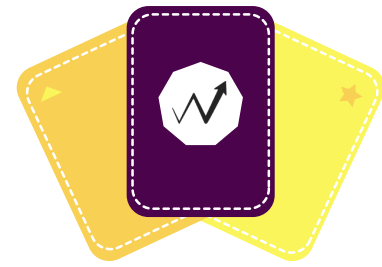


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**BURNING MOUTH
SYNDROME**

Question 7

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



**WINSPERT
CUE CARDS**

**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.



WINSPERT
CUE CARDS

**BURNING MOUTH
SYNDROME**

Question 8

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

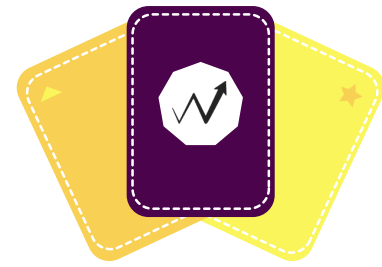


**WINSPERT
CUE CARDS**

**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).

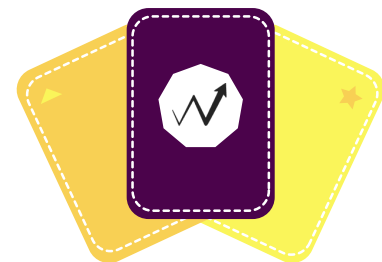


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**BURNING MOUTH
SYNDROME**

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.



**WINSPERT
CUE CARDS**

**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

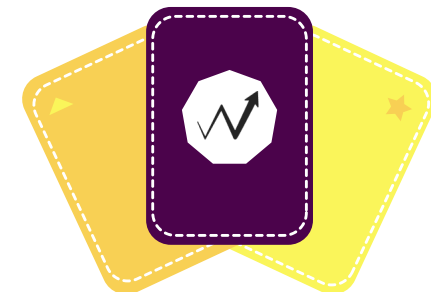
Diagnosis of BMS is by exclusion of other causes of oral burning through detailed clinical history, examination, and ruling out local, systemic, drug-related, or hypersensitivity causes. Management focuses on patient education, lifestyle changes to reduce stress, and pharmacological treatments such as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

ORAL MEDICINE

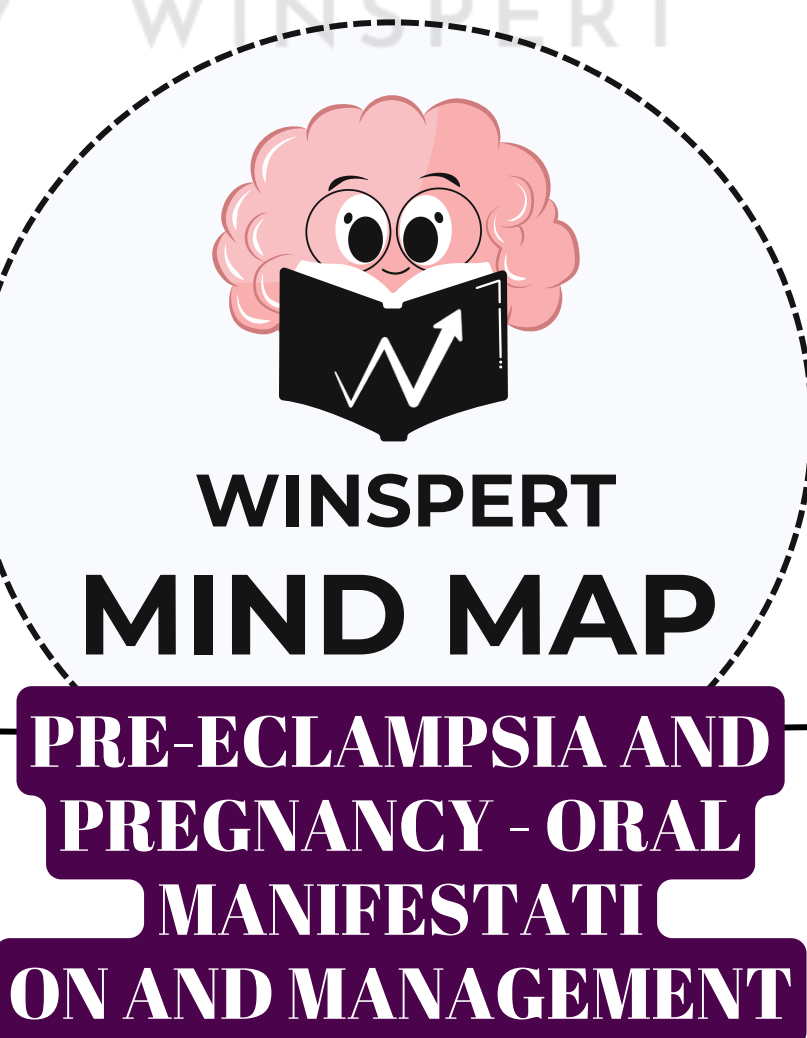
PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT



MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA



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 effect 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.



**WINSPERT
CUE CARDS**

PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

Question 1

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



**WINSPERT
CUE CARDS**

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?

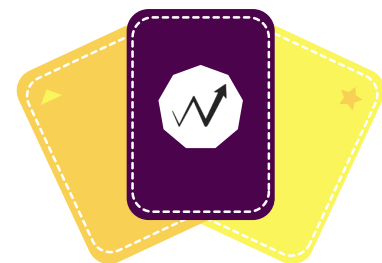


**WINSPERT
CUE CARDS**

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.

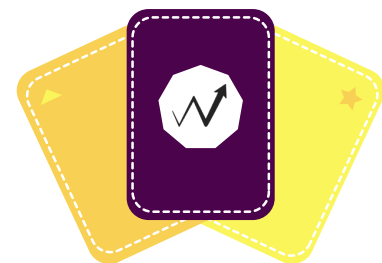


**WINSPERT
CUE CARDS**

PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

Question 5

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

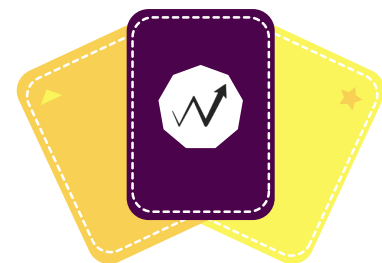


**WINSPERT
CUE CARDS**

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?

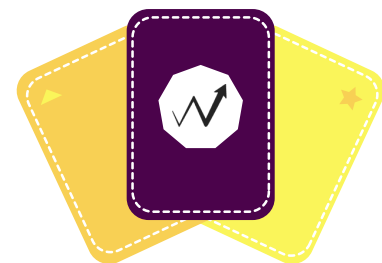


**WINSPERT
CUE CARDS**

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.

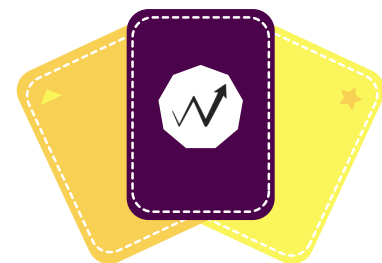


**WINSPERT
CUE CARDS**

PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

Question 7

What preventive oral health measures are recommended during pregnancy?



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

Question 8

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

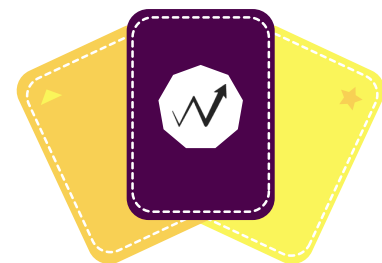


**WINSPERT
CUE CARDS**

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.

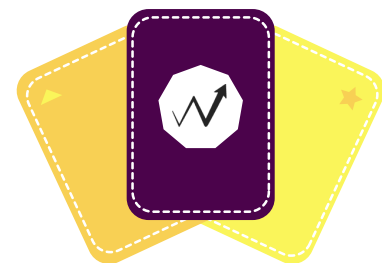


**WINSPERT
CUE CARDS**

PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

Question 9

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

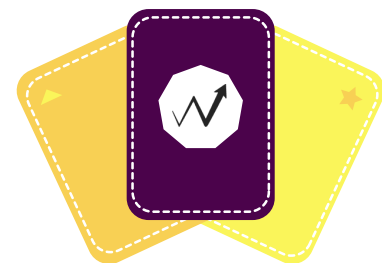


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

Question 10

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



**WINSPERT
CUE CARDS**

PREECLAMPSIA AND PREGNANCY- ORAL MANIFESTATIONS AND MANAGEMENT

Answer 10

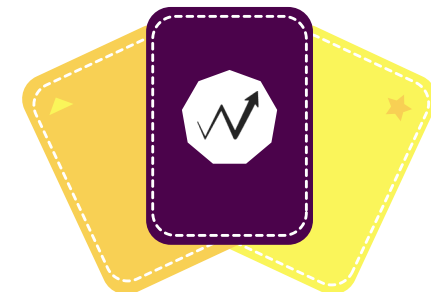
Because placental structure is completed in the first trimester, dental care during pregnancy may occur too late to influence the early development processes related to APO, despite being effective in treating periodontal disease.

ORAL MEDICINE

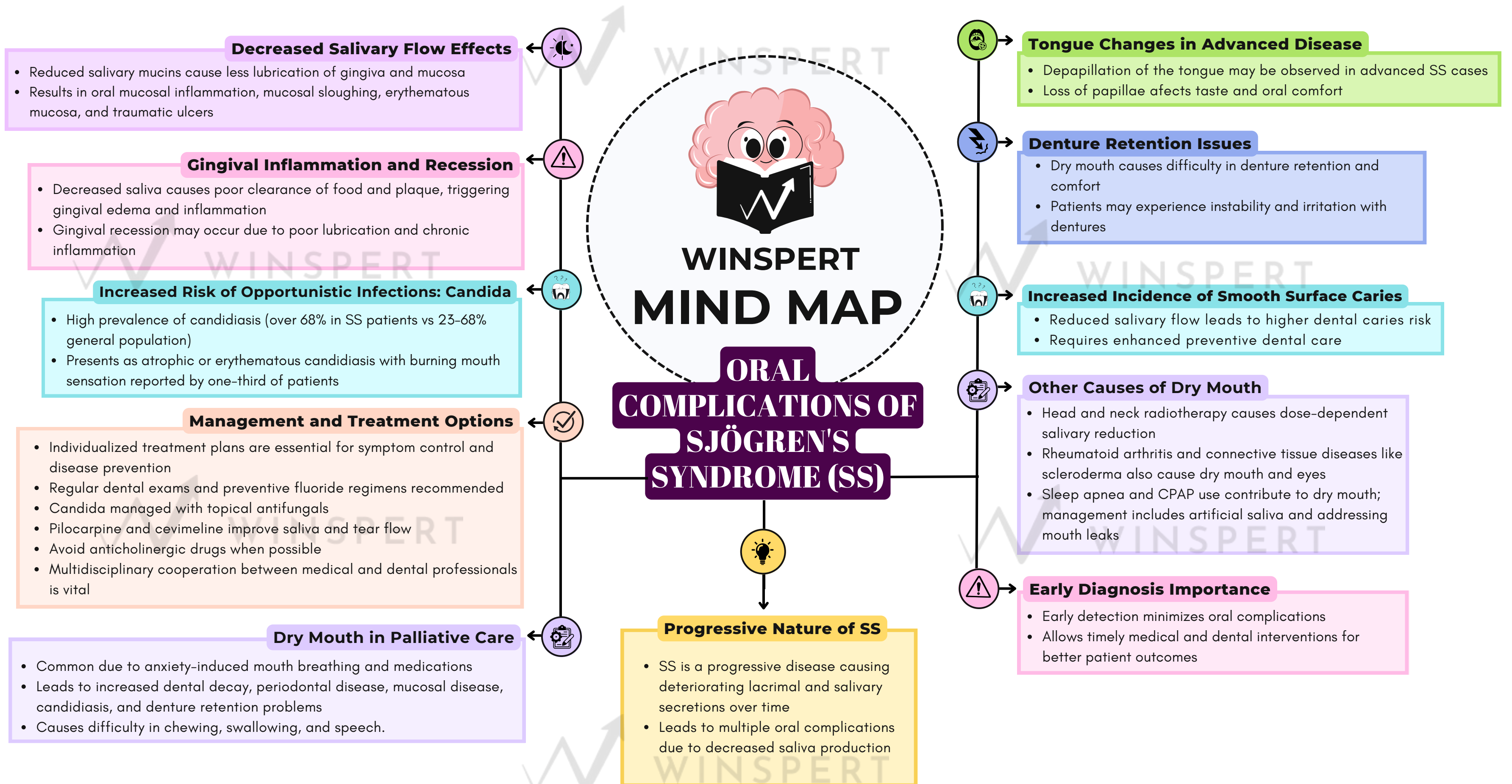
DRY MOUTH



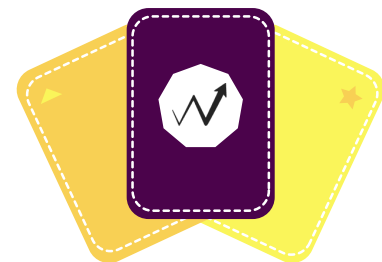
MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA





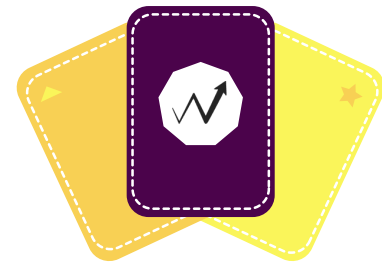


**WINSPERT
CUE CARDS**

DRY MOUTH

Question 1

**What is the difference
between xerostomia and
hyposalivation?**

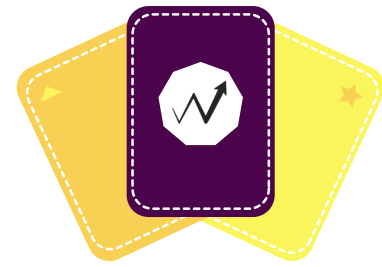


**WINSPERT
CUE CARDS**

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.



**WINSPERT
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.

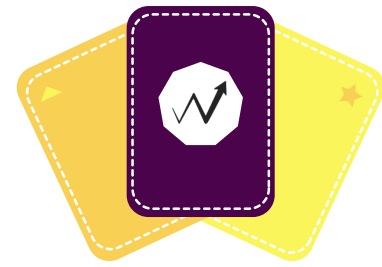


**WINSPERT
CUE CARDS**

DRY MOUTH

Question 2

What are common causes of dry mouth?

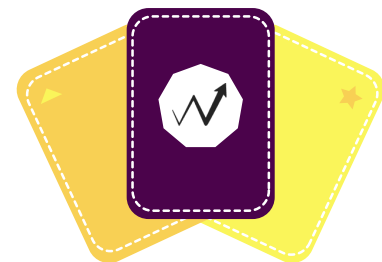


**WINSPERT
CUE CARDS**

DRY MOUTH

Answer 2

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

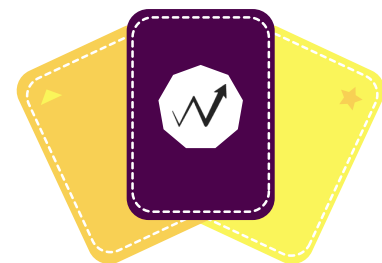


**WINSPERT
CUE CARDS**

DRY MOUTH

Question 3

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



**WINSPERT
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.

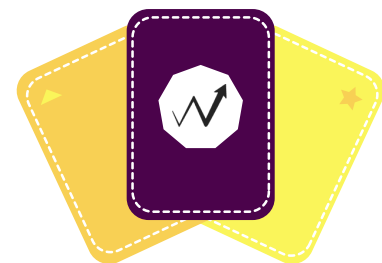


**WINSPERT
CUE CARDS**

DRY MOUTH

Question 4

**What are some oral
consequences of chronic dry
mouth?**

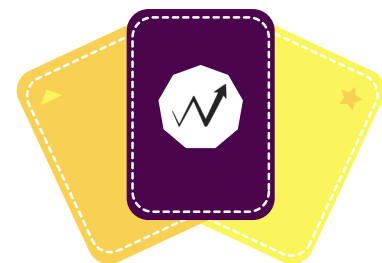


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

DRY MOUTH

Question 5

What management strategies are recommended for patients experiencing dry mouth?

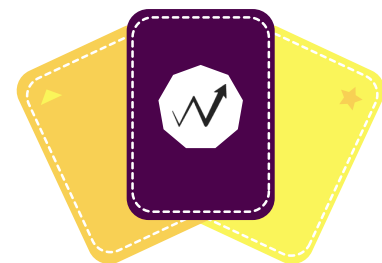


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

DRY MOUTH

Question 6

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

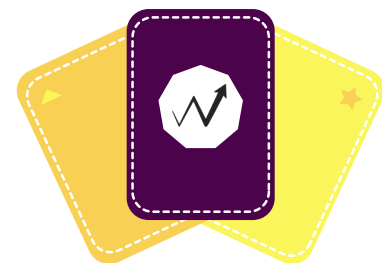


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

DRY MOUTH

Question 7

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

DRY MOUTH

Question 8

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

DRY MOUTH

Question 9

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

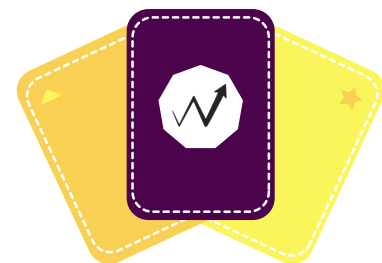


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

DRY MOUTH

Question 10

How is Sjogren syndrome managed from a dental perspective?



**WINSPERT
CUE CARDS**

DRY MOUTH

Answer 10

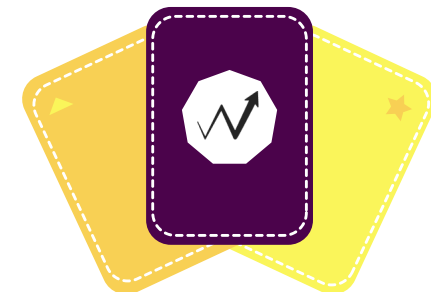
Management includes regular dental examinations, preventive treatments like fluoride application, excellent oral hygiene reinforcement, topical antifungal treatments for candidiasis, avoiding anticholinergic drugs if possible, and use of salivary stimulants such as pilocarpine and cevimeline. Early diagnosis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

ORAL MEDICINE

ORAL MUCOSAL DISEASES

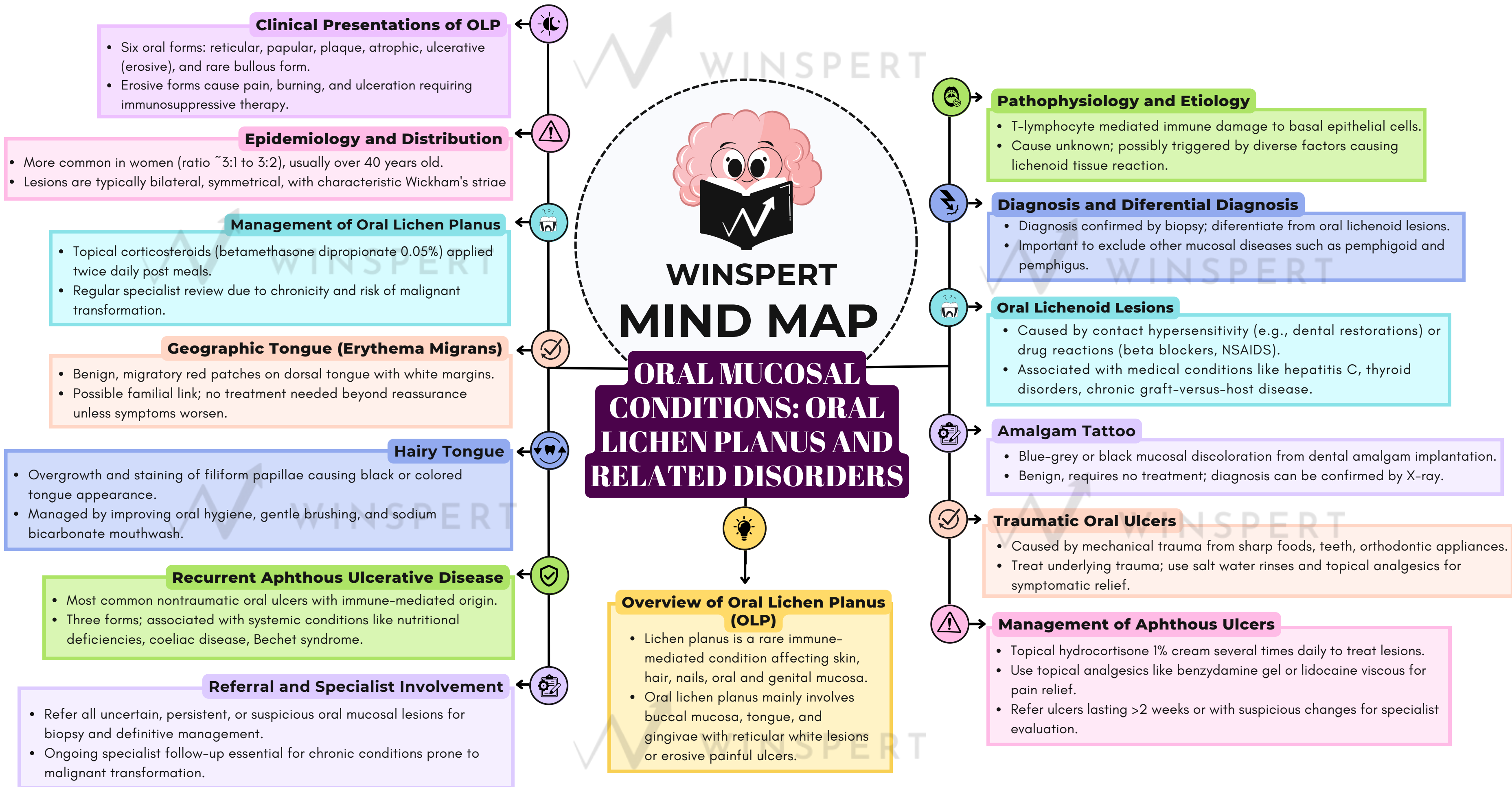


MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA







WINSPERT 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 mimic leukoplakia 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) contribute.
- Head and neck radiotherapy often causes chronic dry mouth.

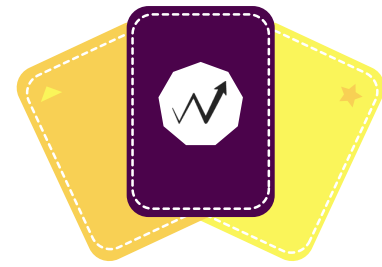
Management of Dry Mouth

- 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.

Map Your Way to ADC Success!

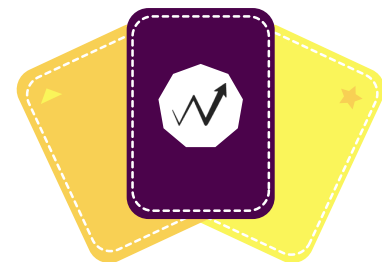


**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 1

What are common causes of oral mucosal lesions?



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 2

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

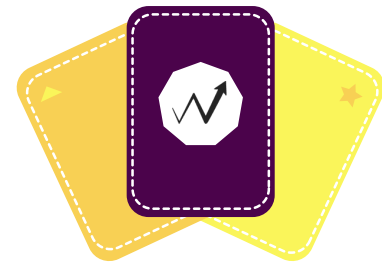


**WINSPERT
CUE CARDS**

**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.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 3

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



**WINSPERT
CUE CARDS**

**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.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 4

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



**WINSPERT
CUE CARDS**

**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.

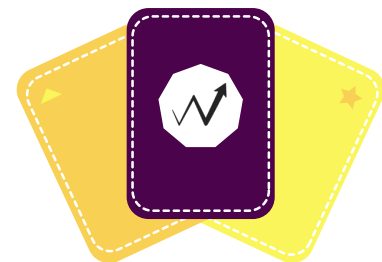


**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 5

Which oral conditions can be managed in general practice?



**WINSPERT
CUE CARDS**

**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.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 6

**What is the most common
type of oral cancer?**

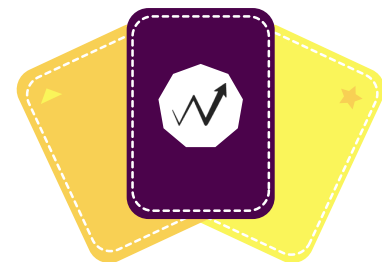


**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Answer 6

Squamous cell carcinoma is the most common oral malignancy.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 7

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



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Answer 7

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



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 8

List some risk factors for oral squamous cell carcinoma.

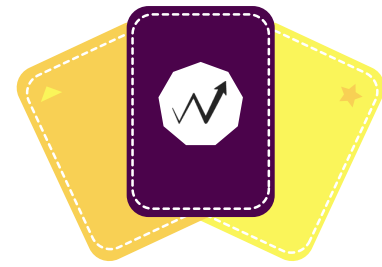


**WINSPERT
CUE CARDS**

**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.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 9

**How is oral leucoplakia
described clinically?**



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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).



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 10

What is the recommended action for patients with oral leucoplakia?



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Answer 10

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



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 11

Describe oral erythroplakia and its significance.

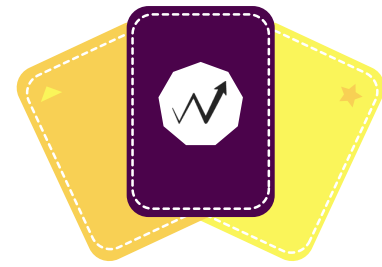


**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 12

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

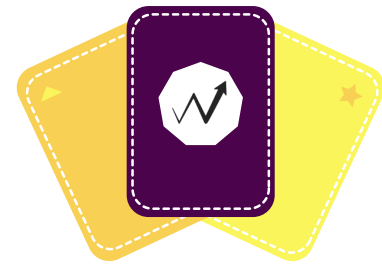


**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 13

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



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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.

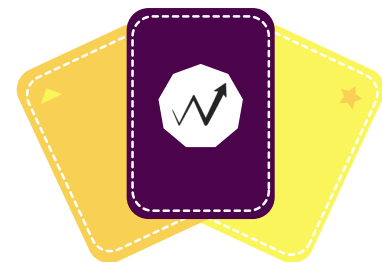


**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 14

**What are the clinical forms of
oral lichen planus?**

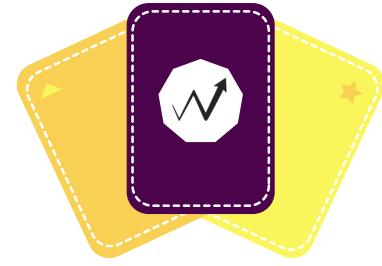


**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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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CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 15

What are the causes of oral lichenoid lesions?



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CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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?**

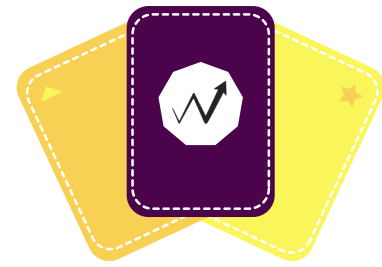


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CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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.



**WINSPERT
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**ORAL MUCOSAL
DISEASES**

Question 17

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

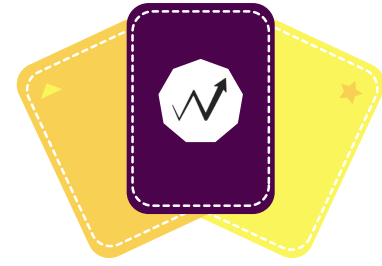


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**ORAL MUCOSAL
DISEASES**

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.



**WINSPERT
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**ORAL MUCOSAL
DISEASES**

Question 18

**Describe the management of
hairy tongue.**

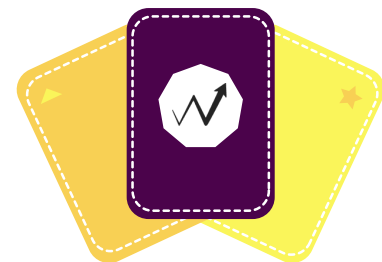


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**ORAL MUCOSAL
DISEASES**

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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**ORAL MUCOSAL
DISEASES**

Question 19

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

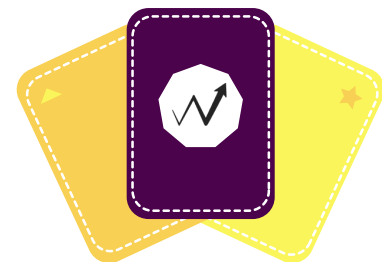


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CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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.



**WINSPERT
CUE CARDS**

**ORAL MUCOSAL
DISEASES**

Question 20

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



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CUE CARDS**

**ORAL MUCOSAL
DISEASES**

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.

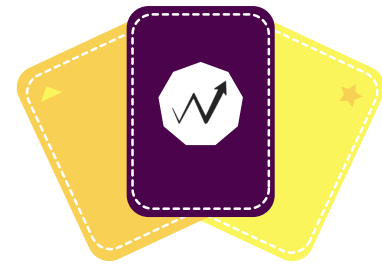


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**ORAL MUCOSAL
DISEASES**

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.

ORAL MEDICINE

ORAL VIRAL INFECTIONS



MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA

WINSPERT MIND MAP

ORAL VIRAL INFECTIONS AND THEIR DENTAL IMPLICATIONS

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.

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 affected patients effectively.

Oncogenic HPV and Oral Cancer

- Certain HPV types cause squamous cell carcinoma in the oropharynx, tonsils, and posterior tongue.
- These HPV-related cancers differ 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.



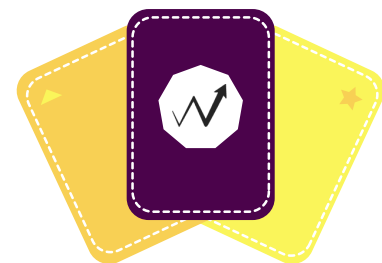


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**ORAL VIRAL
INFECTIONS**

Question 1

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

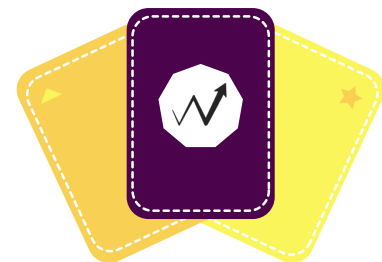


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**ORAL VIRAL
INFECTIONS**

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).

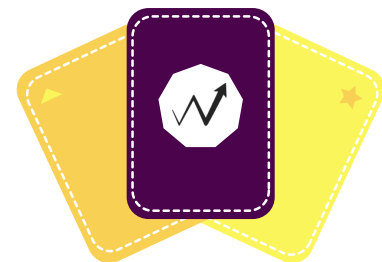


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**ORAL VIRAL
INFECTIONS**

Question 2

Why do viruses require a host organism to reproduce?

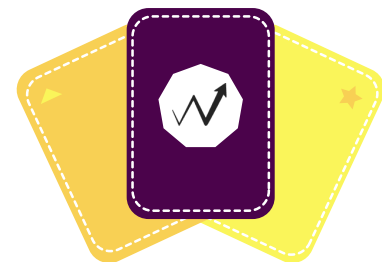


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**ORAL VIRAL
INFECTIONS**

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.

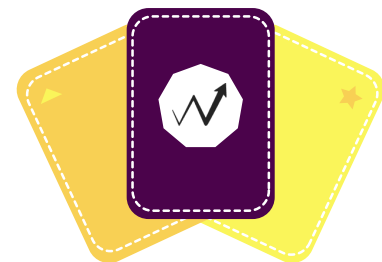


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**ORAL VIRAL
INFECTIONS**

Question 3

What are the typical clinical features of primary herpetic gingivostomatitis?

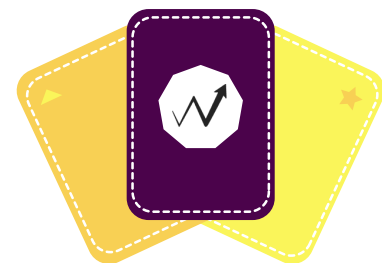


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**ORAL VIRAL
INFECTIONS**

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?

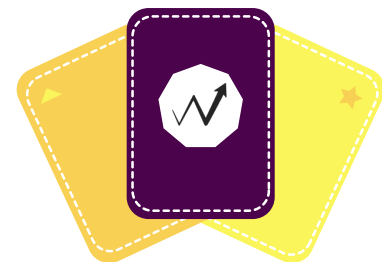


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**ORAL VIRAL
INFECTIONS**

Answer 6

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

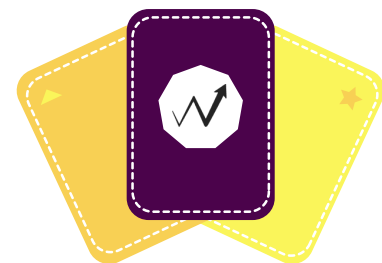


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**ORAL VIRAL
INFECTIONS**

Question 7

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

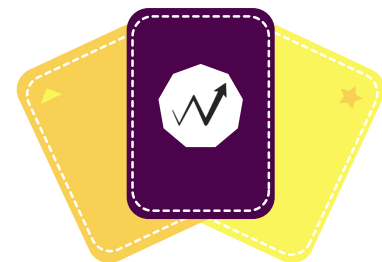


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**ORAL VIRAL
INFECTIONS**

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?

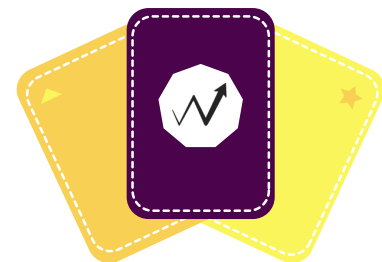


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**ORAL VIRAL
INFECTIONS**

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)?

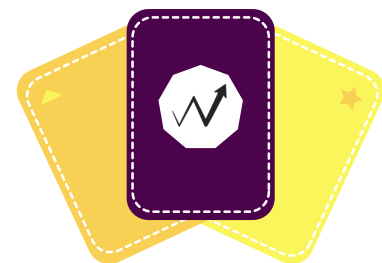


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**ORAL VIRAL
INFECTIONS**

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.

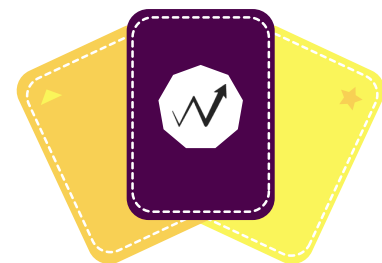


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**ORAL VIRAL
INFECTIONS**

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

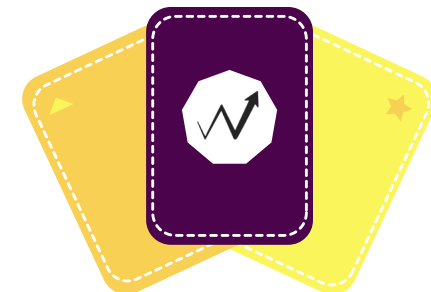
Dental management of HCV-infected patients requires awareness of bleeding risks due to liver disease, avoidance of hepatotoxic drugs, consultation with medical specialists before invasive procedures, postponement of dental work during antiviral therapy, and preventive strategies to address oral health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

ORAL MEDICINE

ORAL RECURRENT ULCERATIONS



MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA

WINSPERT 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 Sjogren's syndrome and medication side effects.
- Neoplastic disease: persistent, progressive ulceration requiring differentiation 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.

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

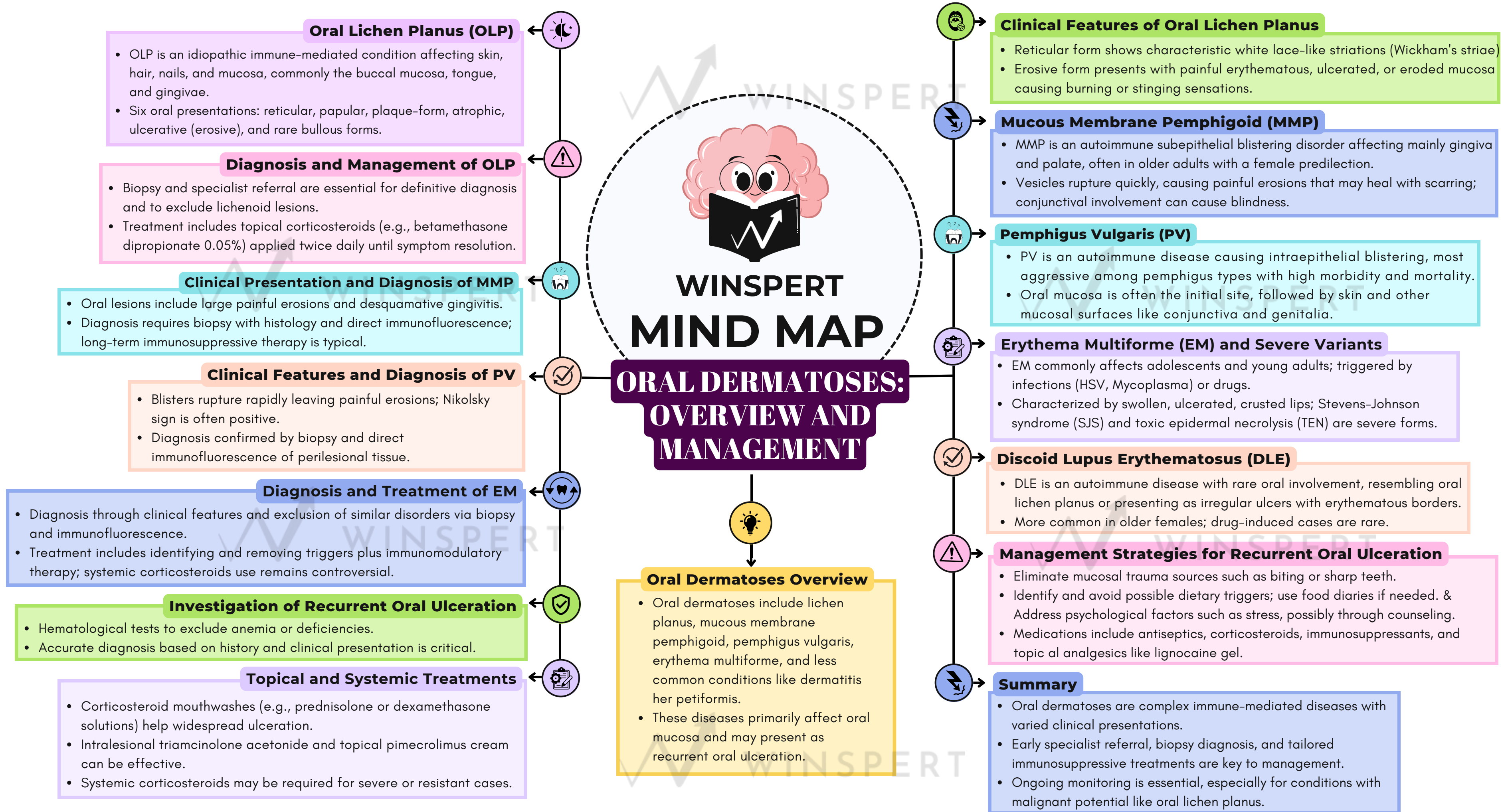
- Herpes Simplex Virus (HSV): causes cold sores and intraoral lesions, preceded by prodrome; management includes antiviral therapy (aciclovir, famciclovir).
- Varicella-Zoster Virus: primary infection is chickenpox; reactivation causes shingles with unilateral oral vesicles and postherpetic neuralgia.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

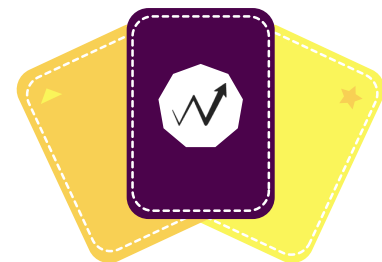
Recurrent Aphthous Ulceration (RAU)

- 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.

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.



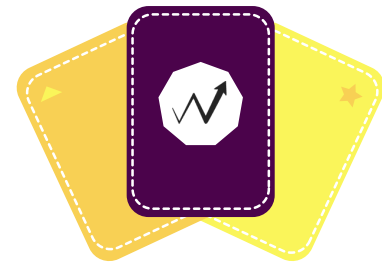


**WINSPERT
CUE CARDS**

**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.

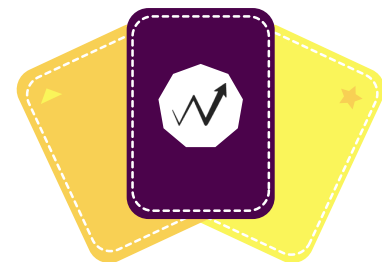


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**ORAL RECURRENT
ULCERATIONS**

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?

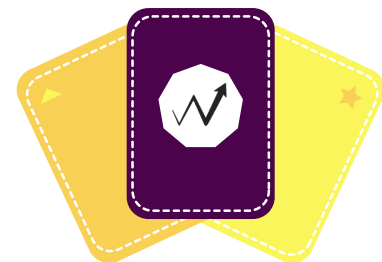


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ORAL RECURRENT ULCERATIONS

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.

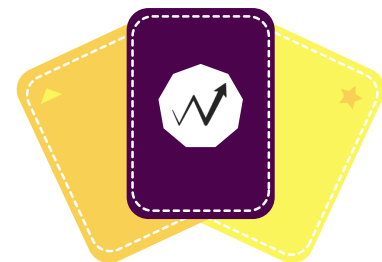


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**ORAL RECURRENT
ULCERATIONS**

Question 4

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



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CUE CARDS**

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.



**WINSPERT
CUE CARDS**

**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.

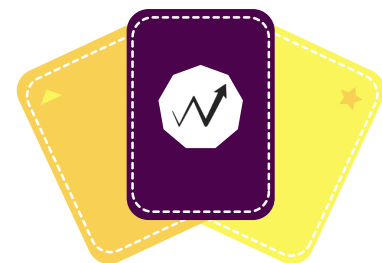


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**ORAL RECURRENT
ULCERATIONS**

Question 6

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

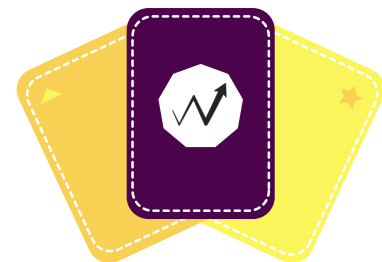


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ORAL RECURRENT ULCERATIONS

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.

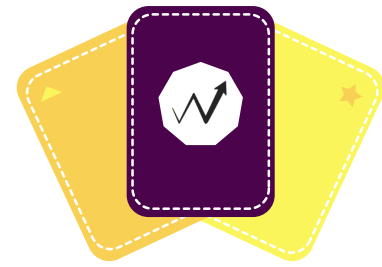


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CUE CARDS**

**ORAL RECURRENT
ULCERATIONS**

Question 7

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



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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.

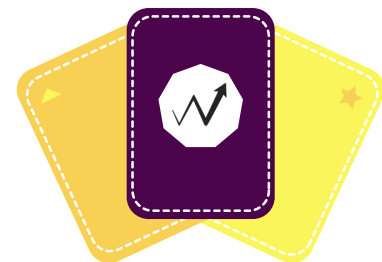


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**ORAL RECURRENT
ULCERATIONS**

Question 8

What clinical features differentiate mucous membrane pemphigoid from pemphigus vulgaris?

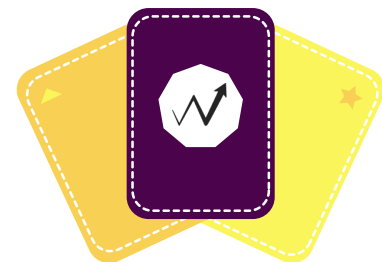


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ORAL RECURRENT ULCERATIONS

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.



**WINSPERT
CUE CARDS**

**ORAL RECURRENT
ULCERATIONS**

Question 9

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



**WINSPERT
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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.



**WINSPERT
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**ORAL RECURRENT
ULCERATIONS**

Question 10

How is minor recurrent aphthous ulceration characterized clinically?

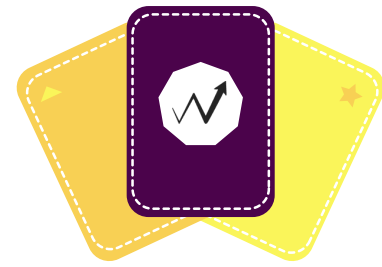


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ORAL RECURRENT ULCERATIONS

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.

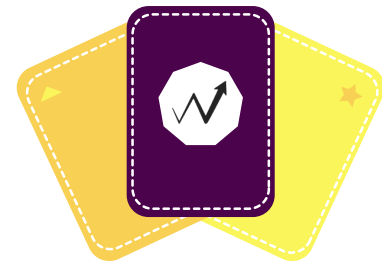


**WINSPERT
CUE CARDS**

**ORAL RECURRENT
ULCERATIONS**

Question 11

What are the main clinical forms of recurrent aphthous ulceration?

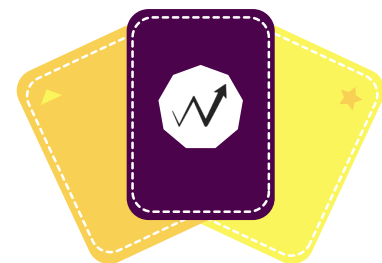


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ORAL RECURRENT ULCERATIONS

Answer 11

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

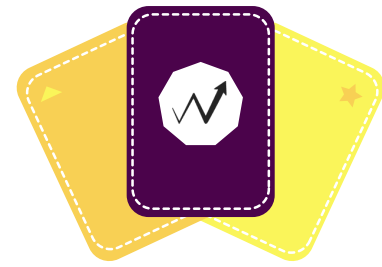


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**ORAL RECURRENT
ULCERATIONS**

Question 12

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

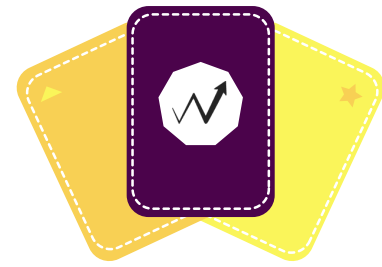


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**ORAL RECURRENT
ULCERATIONS**

Answer 12

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



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**ORAL RECURRENT
ULCERATIONS**

Question 13

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

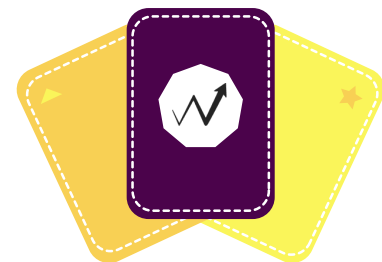


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ORAL RECURRENT ULCERATIONS

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.

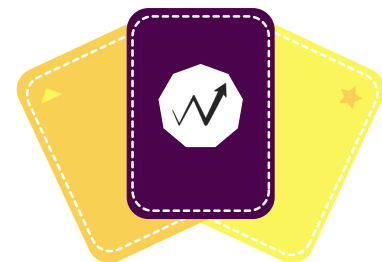


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**ORAL RECURRENT
ULCERATIONS**

Question 15

What general management strategies are recommended for recurrent aphthous ulceration?



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**ORAL RECURRENT
ULCERATIONS**

Answer 15

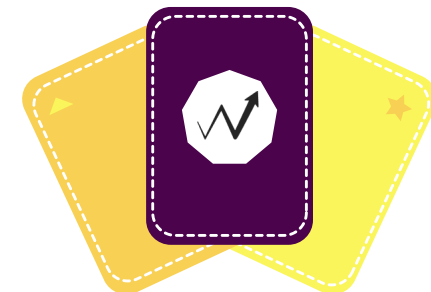
Management involves eliminating trauma, assessing dietary triggers, considering hormonal and psychological factors, symptomatic treatment with corticosteroids, antiseptics, pain relief, and systemic therapy for severe cases.

ORAL MEDICINE

OSTEORADIONECCROSIS OF JAW



MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA

WINSPERT MIND MAP

OSTEORADIONECR OSIS 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, pathological fractures.
- Small lesions may be asymptomatic, causing delayed detection.

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.

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.

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.

10. Hyperbaric Oxygen Therapy (HBOT)

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

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.

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.

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.

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.

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).

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.

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.

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.

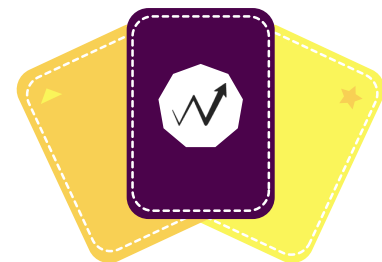


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**OSTEORADIONECCROSIS
OF JAW**

Question 1

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

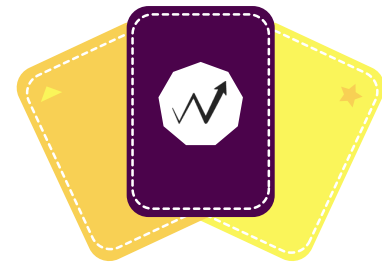


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OSTEORADIONECCROSIS OF JAW

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.

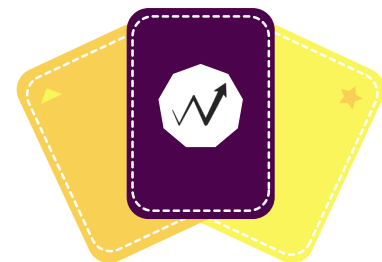


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**OSTEORADIONECCROSIS
OF JAW**

Question 2

What are the common symptoms and clinical presentations of ORN?

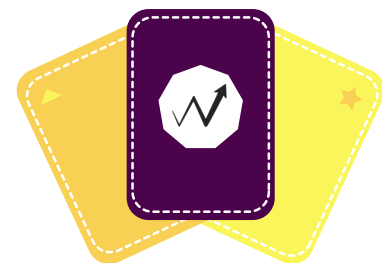


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OSTEORADIONECCROSIS OF JAW

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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**OSTEORADIONECCROSIS
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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CUE CARDS**

**OSTEORADIONECCROSIS
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.



**WINSPERT
CUE CARDS**

**OSTEORADIONECCROSIS
OF JAW**

Question 4

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



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OSTEORADIONECCROSIS 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.

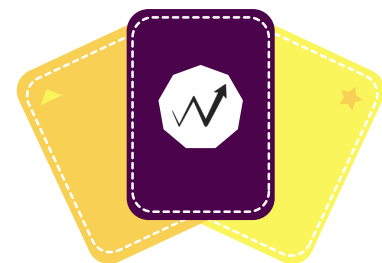


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CUE CARDS**

**OSTEORADIONECCROSIS
OF JAW**

Question 5

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

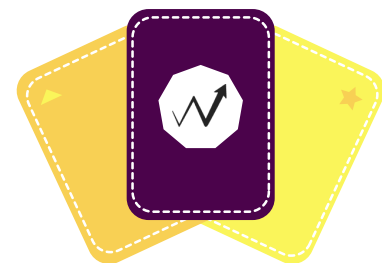


**WINSPERT
CUE CARDS**

**OSTEORADIONECCROSIS
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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**OSTEORADIONECCROSIS
OF JAW**

Question 6

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



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OSTEORADIONECCROSIS 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.

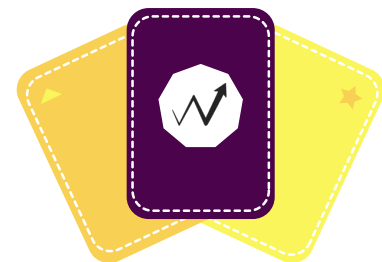


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**OSTEORADIONECCROSIS
OF JAW**

Question 7

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



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**OSTEORADIONECCROSIS
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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**OSTEORADIONECCROSIS
OF JAW**

Question 8

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



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**OSTEORADIONECCROSIS
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.

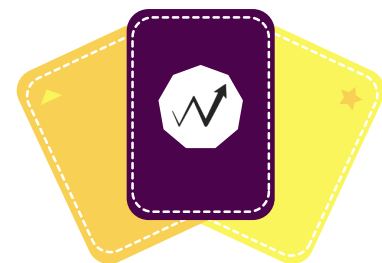


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**OSTEORADIONECCROSIS
OF JAW**

Question 9

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



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OSTEORADIONECCROSIS OF JAW

Answer 9

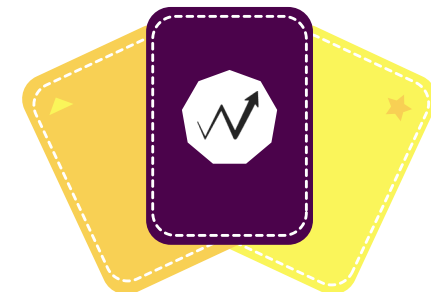
HBOT increases oxygen transfer to tissues under pressures greater than 1.4 ATA, promoting wound healing, increasing tissue oxygenation, and reducing recovery time, which helps prevent and manage complications from radiotherapy including ORNJ.

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.
- 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 corticosteroid 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.

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.
- Effective communication between healthcare providers and patients improves outcomes and adherence.
- Treatment decisions should consider fracture risk, patient preference, and drug subsidy eligibility.

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.

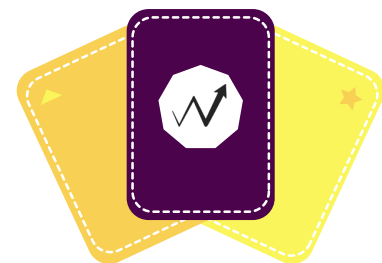


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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

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.



**WINSPERT
CUE CARDS**

**OSTEOPOROSIS
MANAGEMENT AND
DENTAL RELEVANCE**

Question 4

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

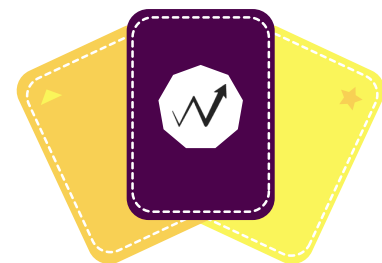


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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

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?

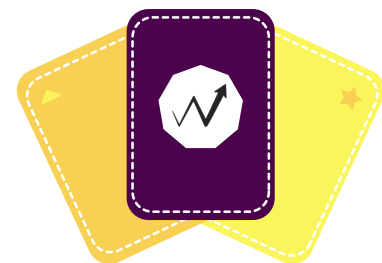


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CUE CARDS**

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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CUE CARDS**

**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?

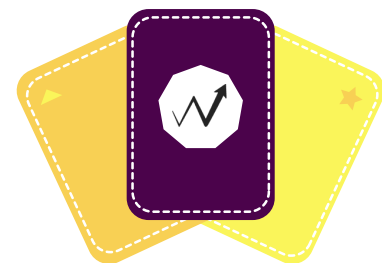


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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

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?

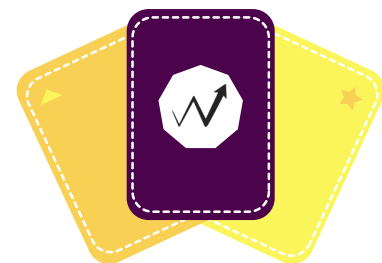


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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

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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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Answer 10

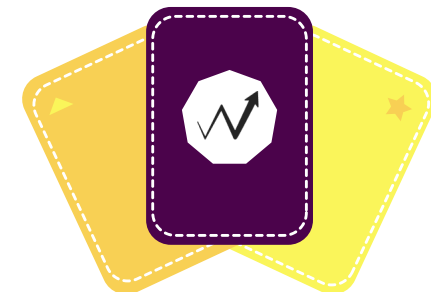
Denosumab is a reversible antiresorptive that inhibits osteoclast-mediated bone resorption and is administered every six months. Unlike bisphosphonates, it is not bound to bone.

ORAL MEDICINE

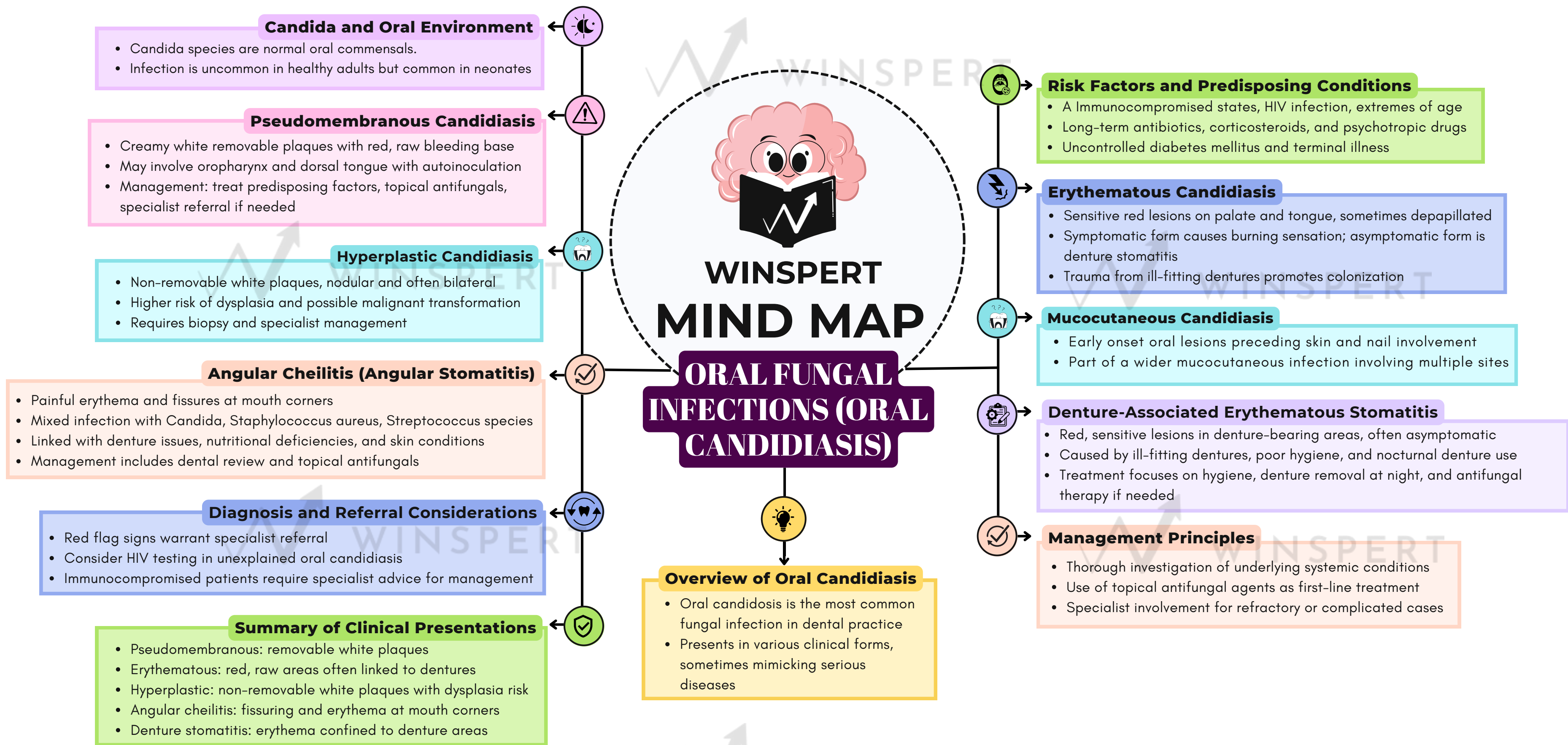
ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)



MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA



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 for 7-14 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 affected 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 differentiate from malignancies

Recommended Treatment Protocol and Hygiene

- Eliminate or 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 Rhomboid 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

WINSPERT MIND MAP

MEDIAN RHOMBOID GLOSSITIS AND ORAL CANDIDIASIS: CLINICAL FEATURES AND MANAGEMENT

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 subsides
- Specialist referral advised for persistent or complicated cases

Principles of Management: Investigations and Underlying Causes

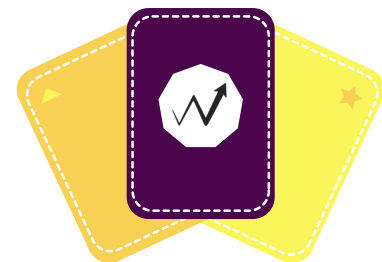
- Assess denture adequacy and hygiene
- Screen for systemic conditions: vitamin B12, folate, iron deficiency, diabetes, immune deficiency
- Review medication history for antimicrobials or corticosteroids
- Consider immune status and other medical conditions

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

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

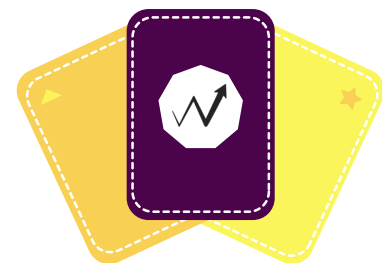


**WINSPERT
CUE CARDS**

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Question 1

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

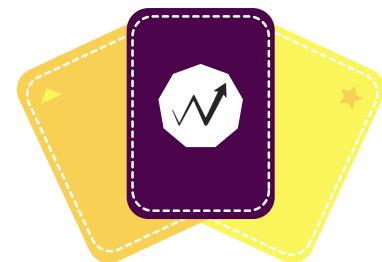


**WINSPERT
CUE CARDS**

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.

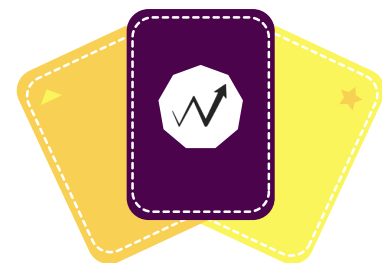


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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Question 2

What are common risk factors for developing oral candidiasis?



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CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Question 3

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

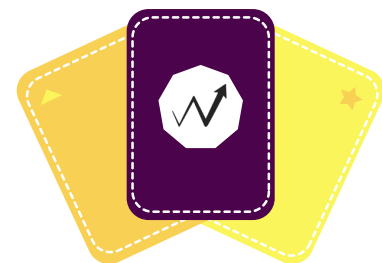


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CUE CARDS**

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.

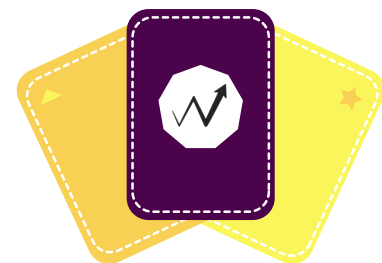


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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Question 4

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

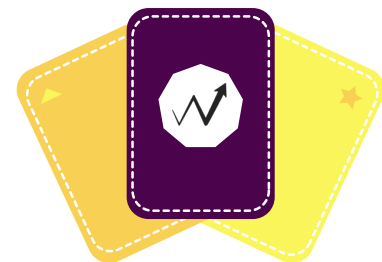


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CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Question 5

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

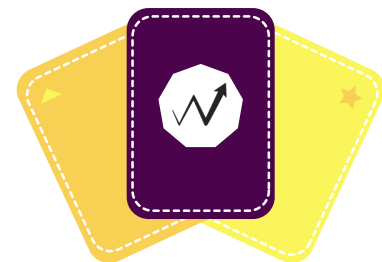


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CUE CARDS**

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.

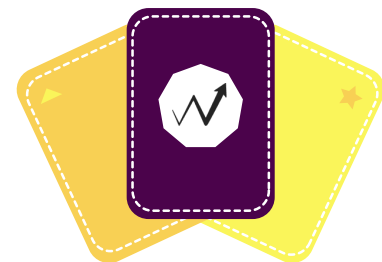


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CUE CARDS**

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Question 6

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

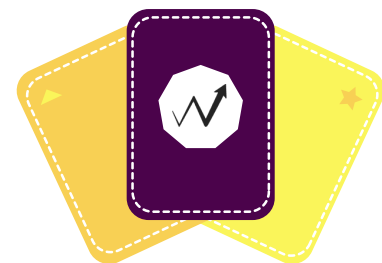


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CUE CARDS**

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.

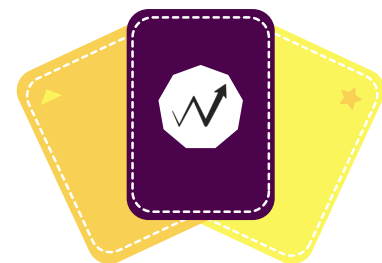


**WINSPERT
CUE CARDS**

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Question 7

How should denture-associated erythematous stomatitis be managed?

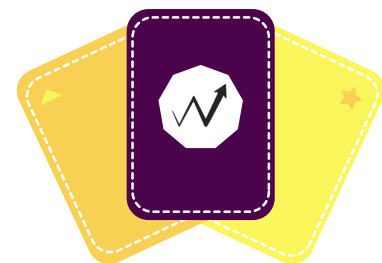


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CUE CARDS**

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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CUE CARDS**

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?

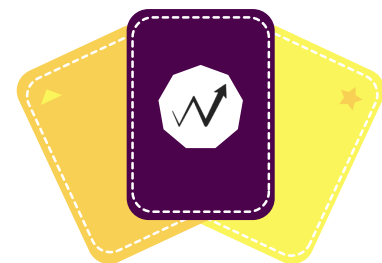


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CUE CARDS**

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.

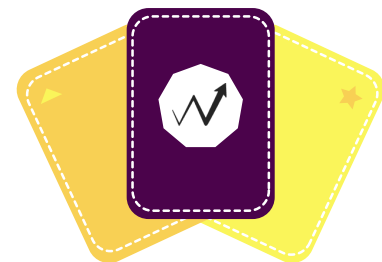


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CUE CARDS**

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

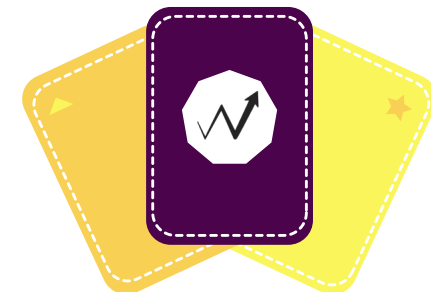
Diagnosis is based on clinical features, but confirmation can be done by direct smear examination stained with PAS, culture of saliva or mouth swill on Sabouraud's agar, and biopsy. Biopsy is particularly important for hyperplastic candidiasis to detect epithelial dysplasia and differentiate from oral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

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 effective 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 biopsychosocial 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

WINSPERT



WINSPERT MIND MAP

TEMPOROMANDIBULAR DISORDERS (TMD)



Definition and Classification

- TMD refers to clinical problems involving masticatory muscles, temporomandibular 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 to 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

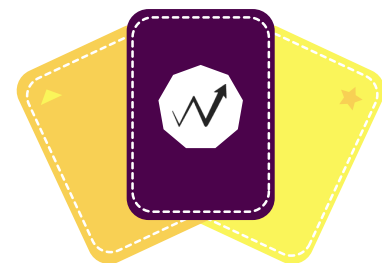
Patient Education and Home Care

- 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

Map Your Way to ADC Success!



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CUE CARDS**

OROFACIAL PAIN

Question 1

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



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OROFACIAL PAIN

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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CUE CARDS**

OROFACIAL PAIN

Question 2

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

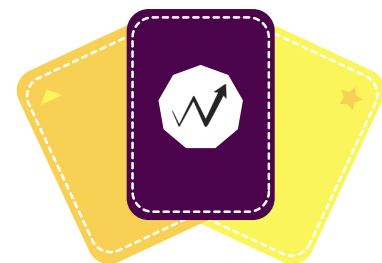


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OROFACIAL PAIN

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?

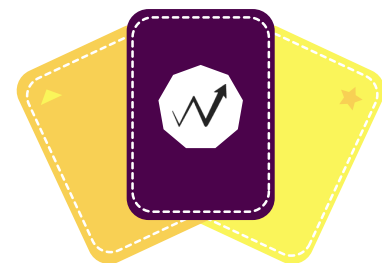


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OROFACIAL PAIN

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?**



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CUE CARDS**

OROFACIAL PAIN

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.

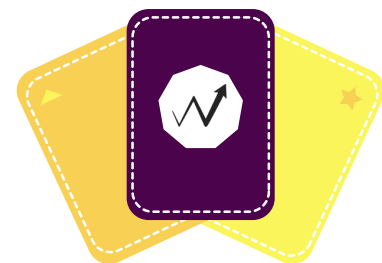


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CUE CARDS**

OROFACIAL PAIN

Question 5

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



**WINSPERT
CUE CARDS**

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?

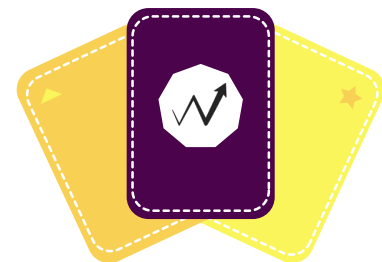


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CUE CARDS**

OROFACIAL PAIN

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.

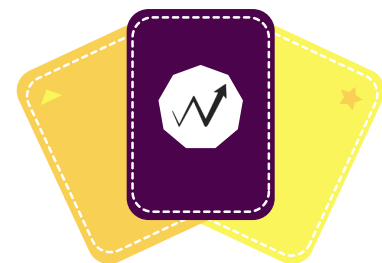


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CUE CARDS**

OROFACIAL PAIN

Question 7

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

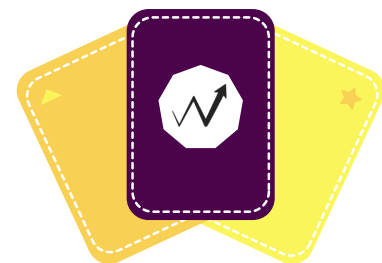


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CUE CARDS**

OROFACIAL PAIN

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.



**WINSPERT
CUE CARDS**

OROFACIAL PAIN

Question 8

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



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CUE CARDS**

OROFACIAL PAIN

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.



**WINSPERT
CUE CARDS**

OROFACIAL PAIN

Question 9

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

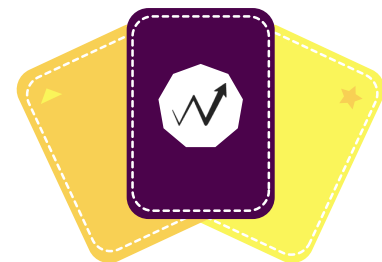


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CUE CARDS**

OROFACIAL PAIN

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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CUE CARDS**

OROFACIAL PAIN

Question 10

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

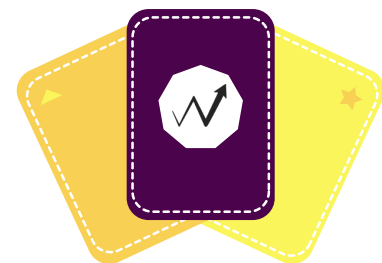


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CUE CARDS**

OROFACIAL PAIN

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?**



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CUE CARDS**

OROFACIAL 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.



**WINSPERT
CUE CARDS**

OROFACIAL PAIN

Question 12

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



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OROFACIAL PAIN

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.



**WINSPERT
CUE CARDS**

OROFACIAL PAIN

Question 13

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



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CUE CARDS**

OROFACIAL PAIN

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.



**WINSPERT
CUE CARDS**

OROFACIAL PAIN

Question 14

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



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CUE CARDS**

OROFACIAL PAIN

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.



**WINSPERT
CUE CARDS**

OROFACIAL PAIN

Question 15

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



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OROFACIAL PAIN

Answer 15

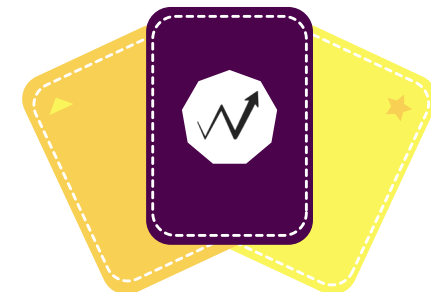
Risk factors include trauma (macro and micro), parafunctional habits (e.g., bruxism), psychosocial factors (stress, anxiety), and certain dental procedures. Management is conservative, focusing on symptom control via patient education, jaw rest, avoiding extreme jaw movements, muscle massage, warm packs,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

ORAL MEDICINE

ACTINIC CHEILITIS



MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA

WINSPERT MIND MAP

ACTINIC CHEILITIS

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.

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.

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 effects 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

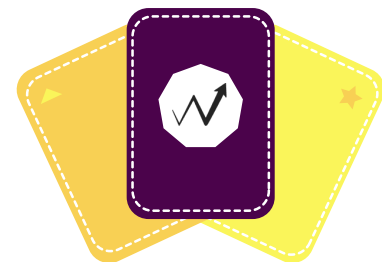
- Treatment options include cryotherapy, electrosurgery, topical agents (retinoids, 5-fluorouracil, imiquimod), photodynamic therapy, laser ablation, and surgical vermilionectomy.
- Patient-specific plans depend on disease severity, lifestyle, and sun exposure history.

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.

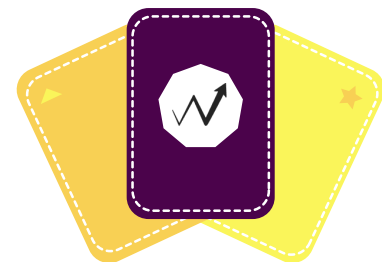


**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Question 1

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

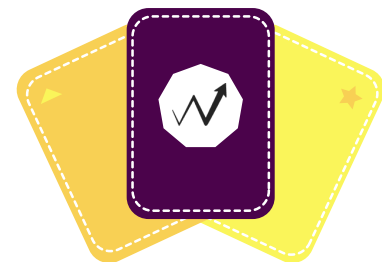


**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Answer 1

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



**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Question 2

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

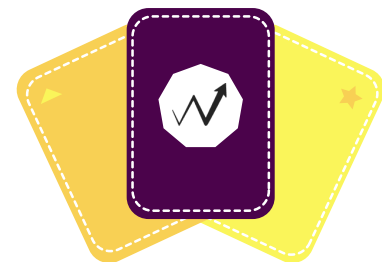


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Question 3

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

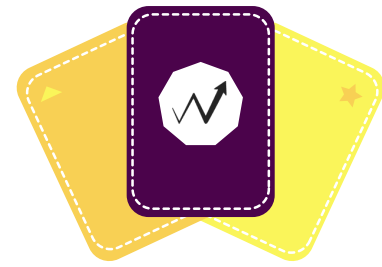


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Question 4

What are the early clinical signs of actinic cheilitis?



**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Answer 4

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



WINSPERT
CUE CARDS

ACTINIC CHEILITIS

Question 5

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

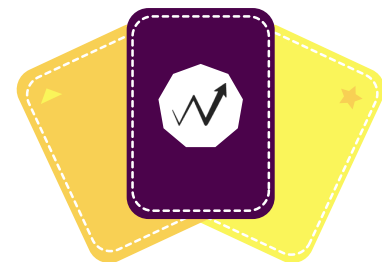


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Question 6

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



**WINSPERT
CUE CARDS**

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.

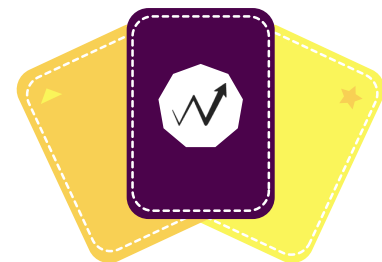


**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Question 7

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

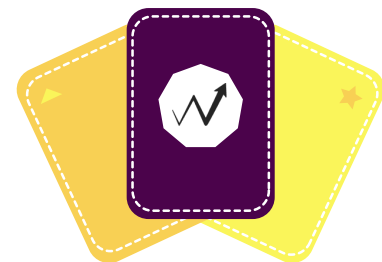


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ACTINIC CHEILITIS

Question 8

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



**WINSPERT
CUE CARDS**

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.

ORAL MEDICINE

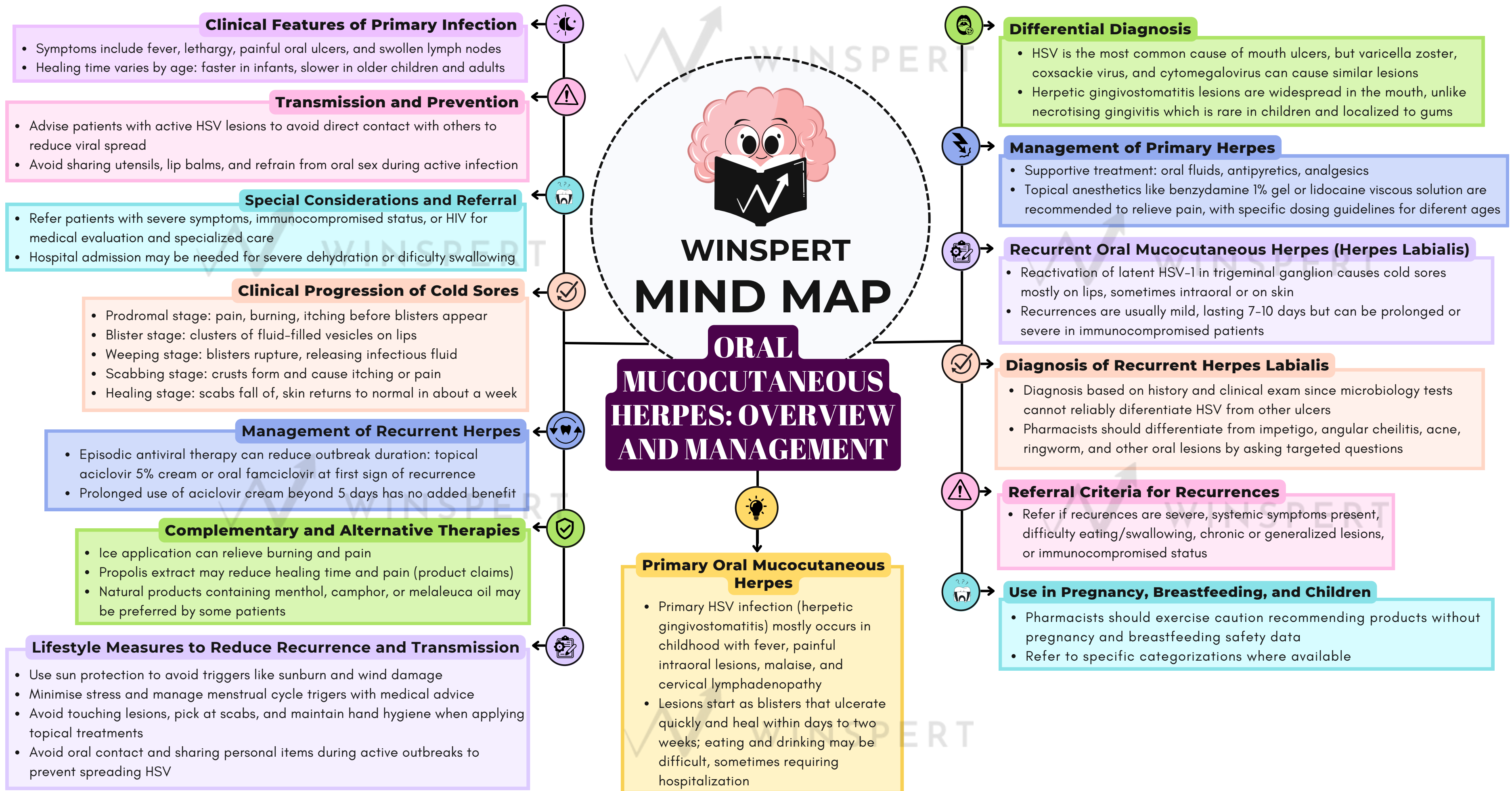
ORAL HERPES



MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA





WINSPERT
CUE CARDS

ORAL HERPES

Question 1

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL HERPES

Question 2

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



**WINSPERT
CUE CARDS**

ORAL HERPES

Answer 2

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



**WINSPERT
CUE CARDS**

ORAL HERPES

Question 3

What distinguishes herpetic gingivostomatitis lesions from necrotising gingivitis in children?



**WINSPERT
CUE CARDS**

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.

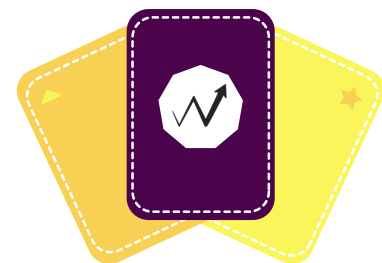


WINSPERT
CUE CARDS

ORAL HERPES

Question 4

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL HERPES

Question 5

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



**WINSPERT
CUE CARDS**

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.



WINSPERT
CUE CARDS

ORAL HERPES

Question 6

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL HERPES

Question 7

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

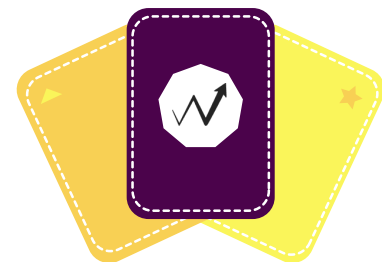


**WINSPERT
CUE CARDS**

ORAL HERPES

Answer 7

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.



**WINSPERT
CUE CARDS**

ORAL HERPES

Question 8

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



**WINSPERT
CUE CARDS**

ORAL HERPES

Answer 8

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.

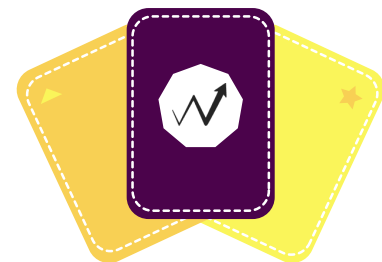


**WINSPERT
CUE CARDS**

ORAL HERPES

Question 9

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



**WINSPERT
CUE CARDS**

ORAL HERPES

Answer 9

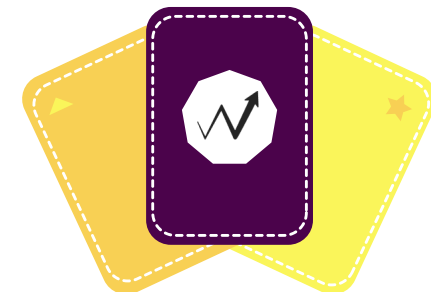
Referral is required for patients with severe presentations, immunocompromised patients, patients with HIV, those with difficulty eating or swallowing, and those with generalized or chronic herpes infection.

ORAL MEDICINE

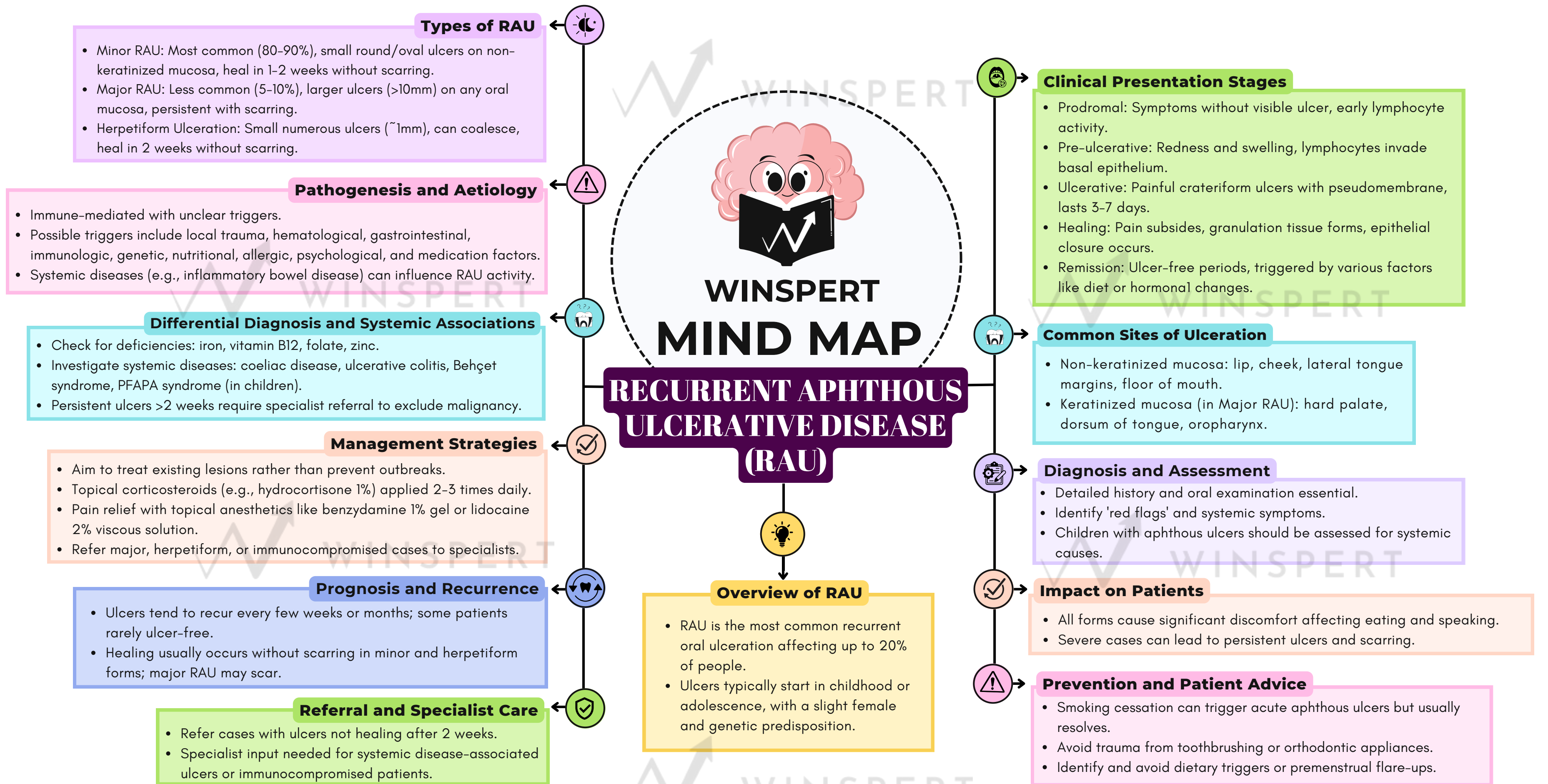
RECURRENT APHTHOUS ULCERS

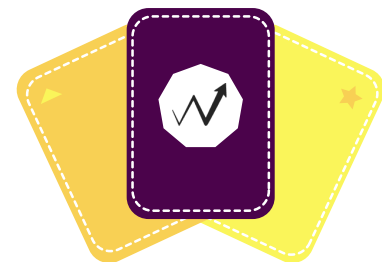


MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA



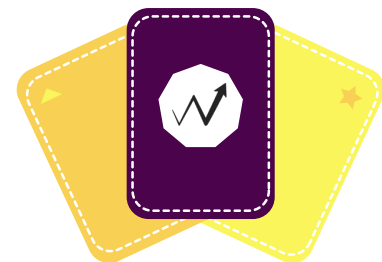


**WINSPERT
CUE CARDS**

RECURRENT APHTHOUS ULCERS

Question 1

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

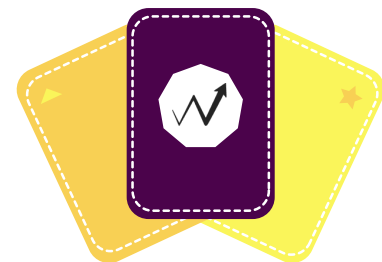


**WINSPERT
CUE CARDS**

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.

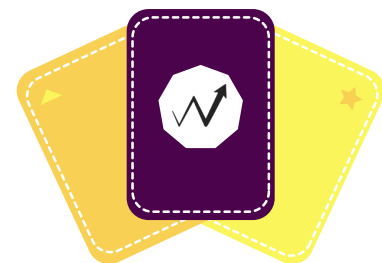


**WINSPERT
CUE CARDS**

RECURRENT APHTHOUS ULCERS

Question 2

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

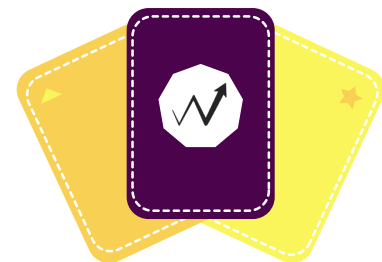


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Question 3

What are the three recognized forms of recurrent aphthous ulceration?

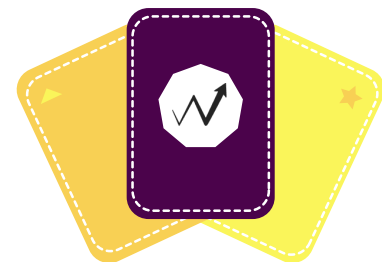


**WINSPERT
CUE CARDS**

RECURRENT APHTHOUS ULCERS

Answer 3

Herpetiform ulceration (similar prevalence to major RAU)



**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Question 4

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

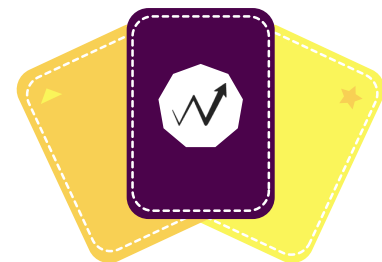


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Question 5

What distinguishes major recurrent aphthous ulceration from minor RAU?

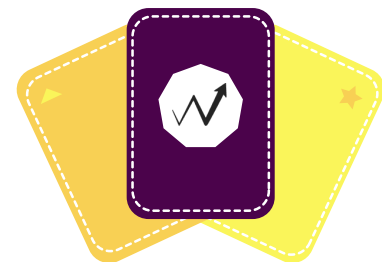


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Question 6

**What are the characteristics of
herpetiform ulceration?**

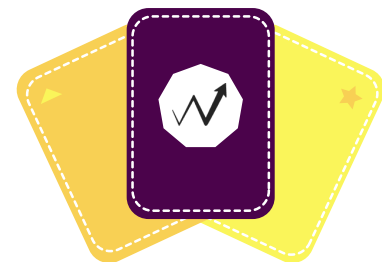


**WINSPERT
CUE CARDS**

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.

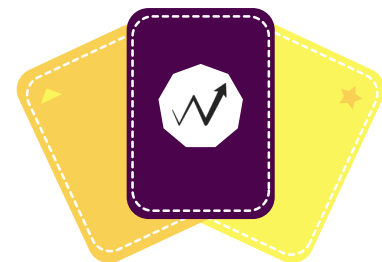


**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Question 7

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

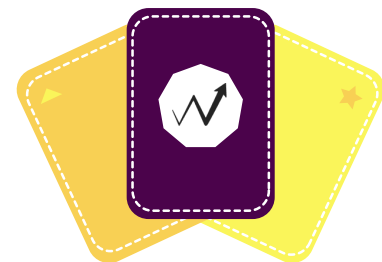


**WINSPERT
CUE CARDS**

**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.



**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Question 8

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

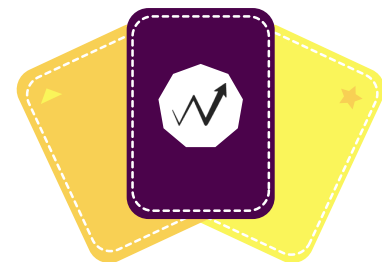


**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Answer 8

Remission – no lesions present



**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Question 9

Which systemic conditions are associated with recurrent aphthous ulcers?

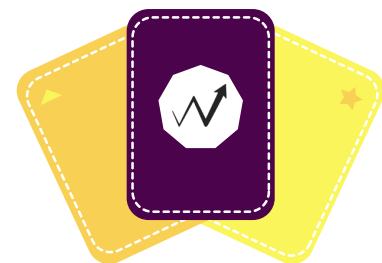


**WINSPERT
CUE CARDS**

**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.



**WINSPERT
CUE CARDS**

**RECURRENT
APHTHOUS ULCERS**

Question 10

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



**WINSPERT
CUE CARDS**

RECURRENT APHTHOUS ULCERS

Answer 10

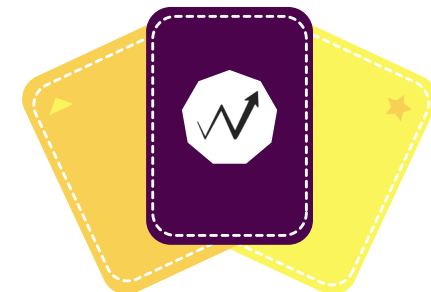
Treatment focuses on symptom relief rather than prevention. Use topical hydrocortisone 1% cream or ointment applied 2-3 times daily after meals for lesions. For pain relief, use topical anaesthetics such as benzydamine 1% gel or lidocaine 2% viscous solution. Lesions not healing after 2 weeks should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

ORAL MEDICINE

ORAL LICHEN PLANUS

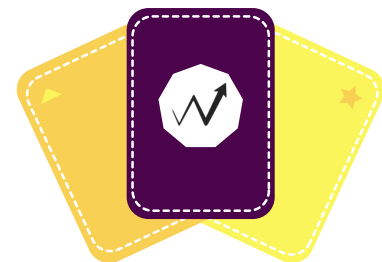


MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA





**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 1

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

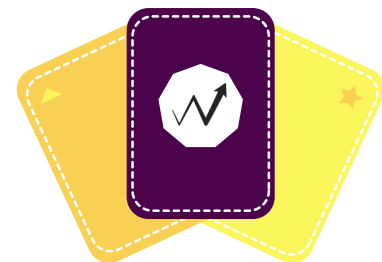


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 2

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 3

At what age does oral lichen planus predominantly occur?



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Answer 3

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



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 4

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

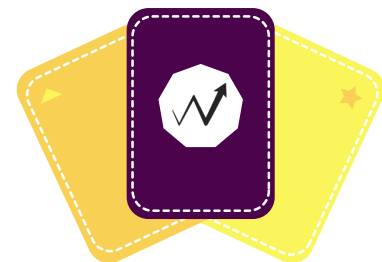


**WINSPERT
CUE CARDS**

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.

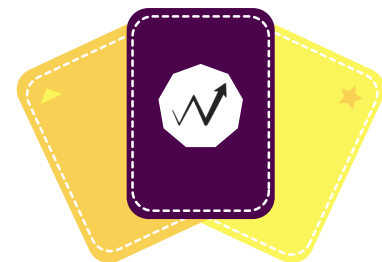


**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 5

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

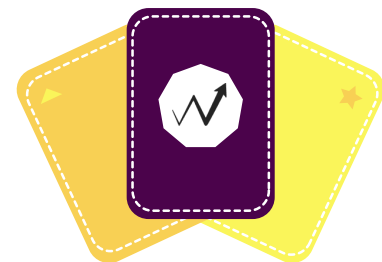


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 6

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



**WINSPERT
CUE CARDS**

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.

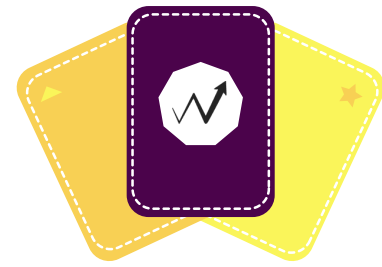


**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 7

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

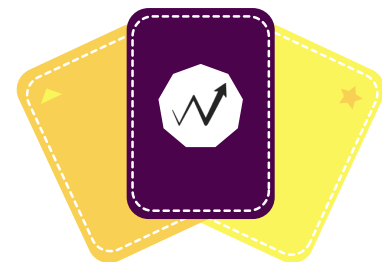


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 8

**Is oral lichen planus
considered a premalignant
condition?**

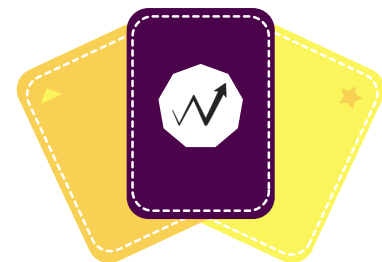


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 9

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

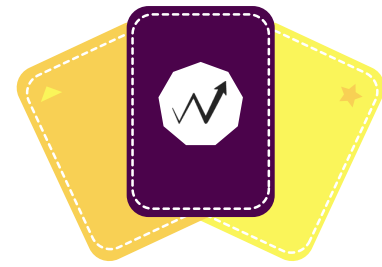


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 10

What diagnostic procedures are essential to confirm oral lichen planus?

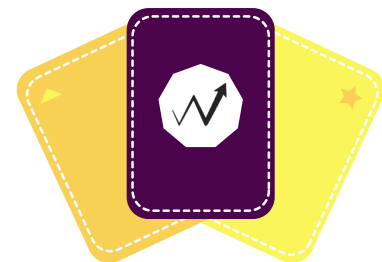


**WINSPERT
CUE CARDS**

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.

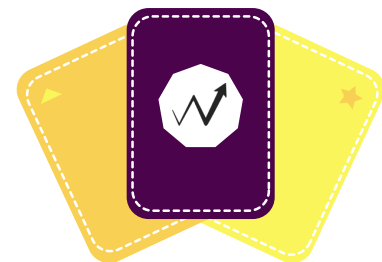


**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 11

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 12

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

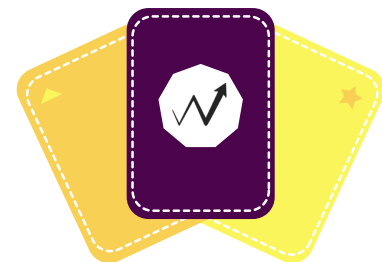


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 13

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

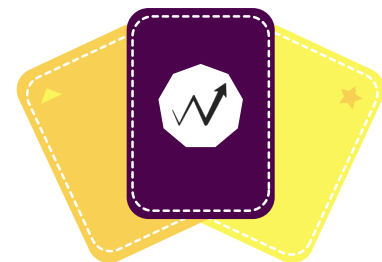


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 14

Why is candidal infection significant in oral lichen planus patients?

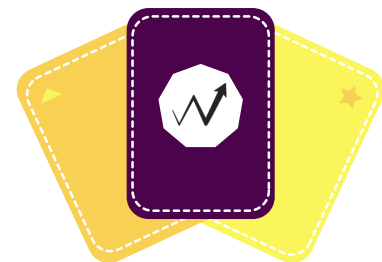


**WINSPERT
CUE CARDS**

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.

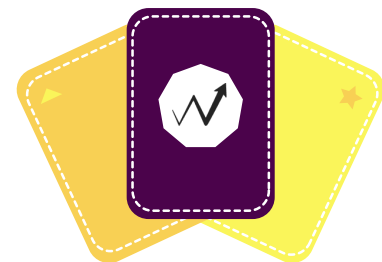


**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 15

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

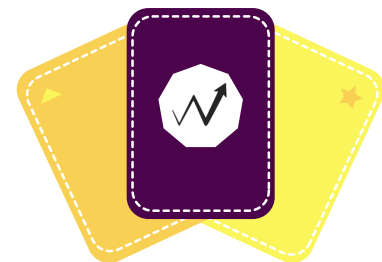


**WINSPERT
CUE CARDS**

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.

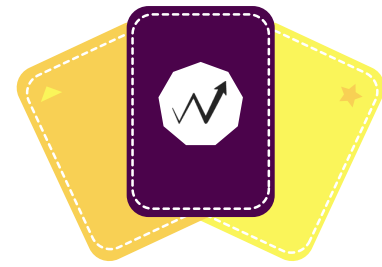


**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 16

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

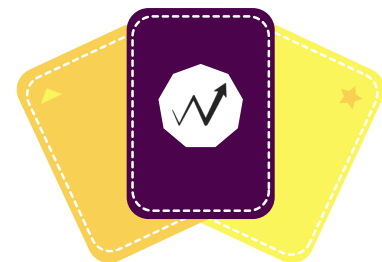


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 17

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



**WINSPERT
CUE CARDS**

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.

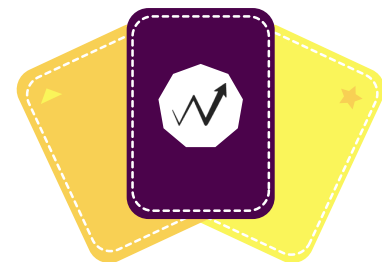


**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 18

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

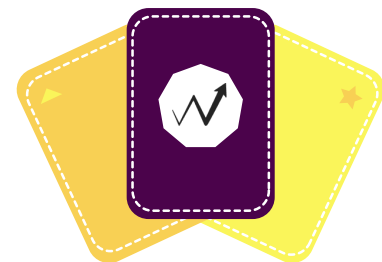


**WINSPERT
CUE CARDS**

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.

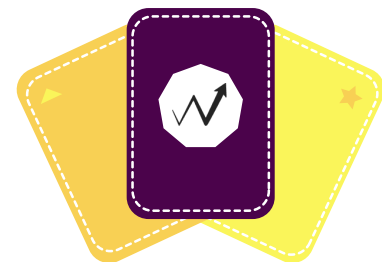


**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 19

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

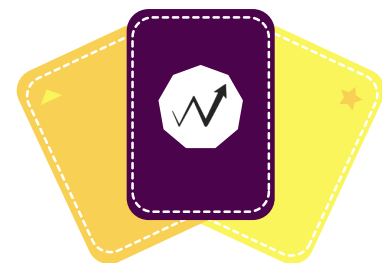


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Question 20

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



**WINSPERT
CUE CARDS**

ORAL LICHEN PLANUS

Answer 20

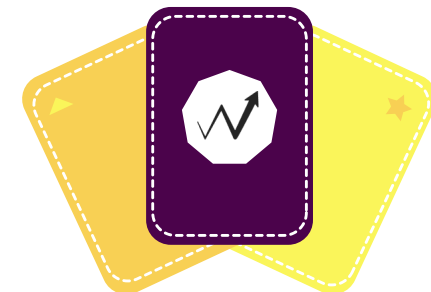
Patients should eliminate smoking and alcohol consumption, maintain a nutritious diet rich in fresh fruits and vegetables, avoid betel quid chewing, and manage *Candida albicans* infections to lower cancer risk.

ORAL MEDICINE

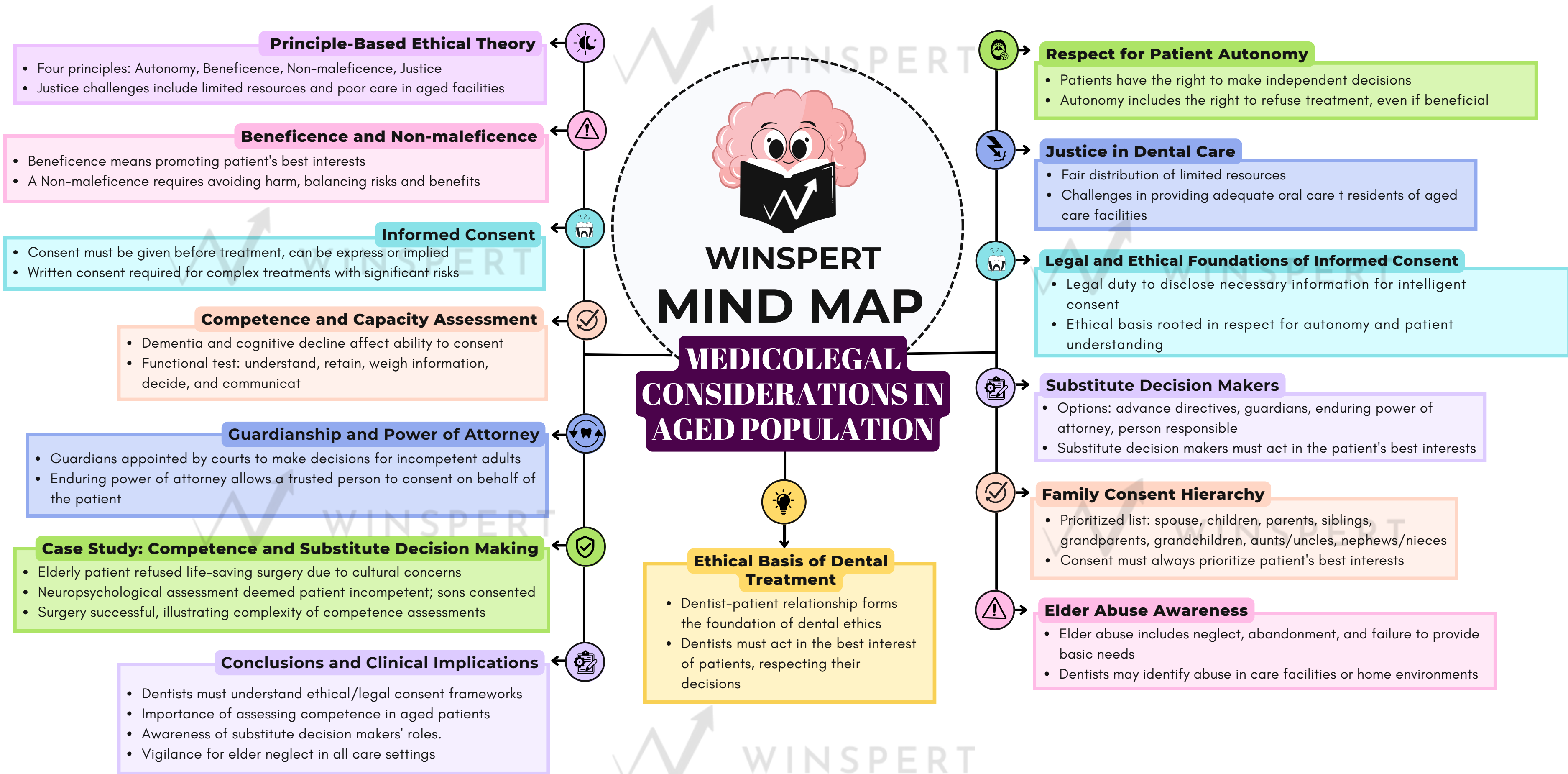
MEDICO-LEGAL CONSIDERATIONS IN AGED POPULATION

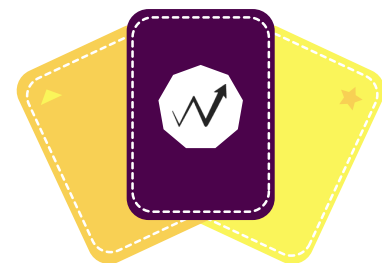


MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA



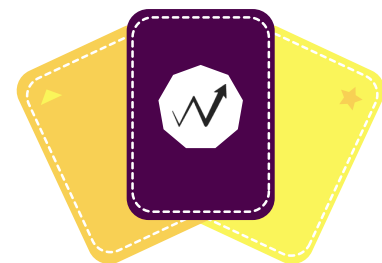


**WINSPERT
CUE CARDS**

**MEDICO-LEGAL
CONSIDERATIONS IN
AGED POPULATION**

Question 1

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



**WINSPERT
CUE CARDS**

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.

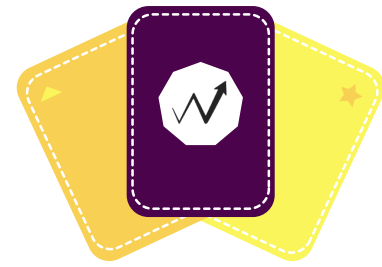


**WINSPERT
CUE CARDS**

**MEDICO-LEGAL
CONSIDERATIONS IN
AGED POPULATION**

Question 2

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



**WINSPERT
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.**



**WINSPERT
CUE CARDS**

**MEDICO-LEGAL
CONSIDERATIONS IN
AGED POPULATION**

Question 3

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



**WINSPERT
CUE CARDS**

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.

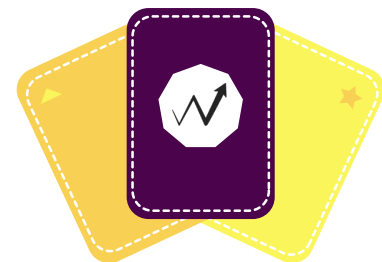


**WINSPERT
CUE CARDS**

**MEDICO-LEGAL
CONSIDERATIONS IN
AGED POPULATION**

Question 4

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

**MEDICO-LEGAL
CONSIDERATIONS IN
AGED POPULATION**

Question 5

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



**WINSPERT
CUE CARDS**

**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.



**WINSPERT
CUE CARDS**

**MEDICO-LEGAL
CONSIDERATIONS IN
AGED POPULATION**

Question 6

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

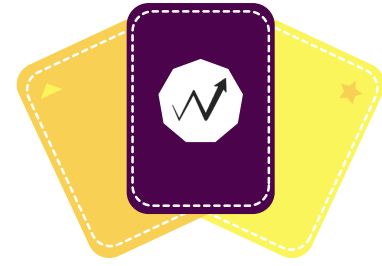


**WINSPERT
CUE CARDS**

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.



**WINSPERT
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?



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

**MEDICO-LEGAL
CONSIDERATIONS IN
AGED POPULATION**

Question 8

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



**WINSPERT
CUE CARDS**

**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.



**WINSPERT
CUE CARDS**

**MEDICO-LEGAL
CONSIDERATIONS IN
AGED POPULATION**

Question 9

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

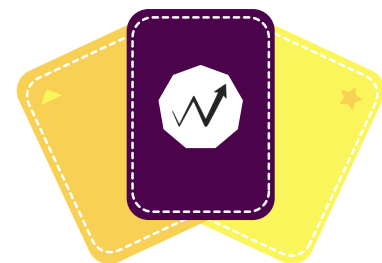


**WINSPERT
CUE CARDS**

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.



**WINSPERT
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

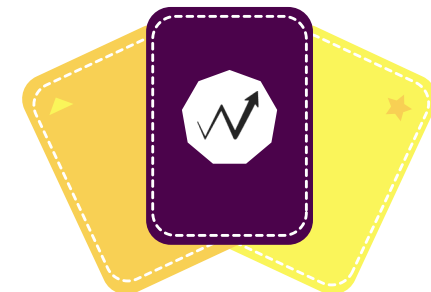
Because patients may verbally understand risks but may not rationally weigh the benefits against risks due to cultural beliefs or cognitive impairment, making it difficult to determine true competence without specialist assessment.

ORAL MEDICINE

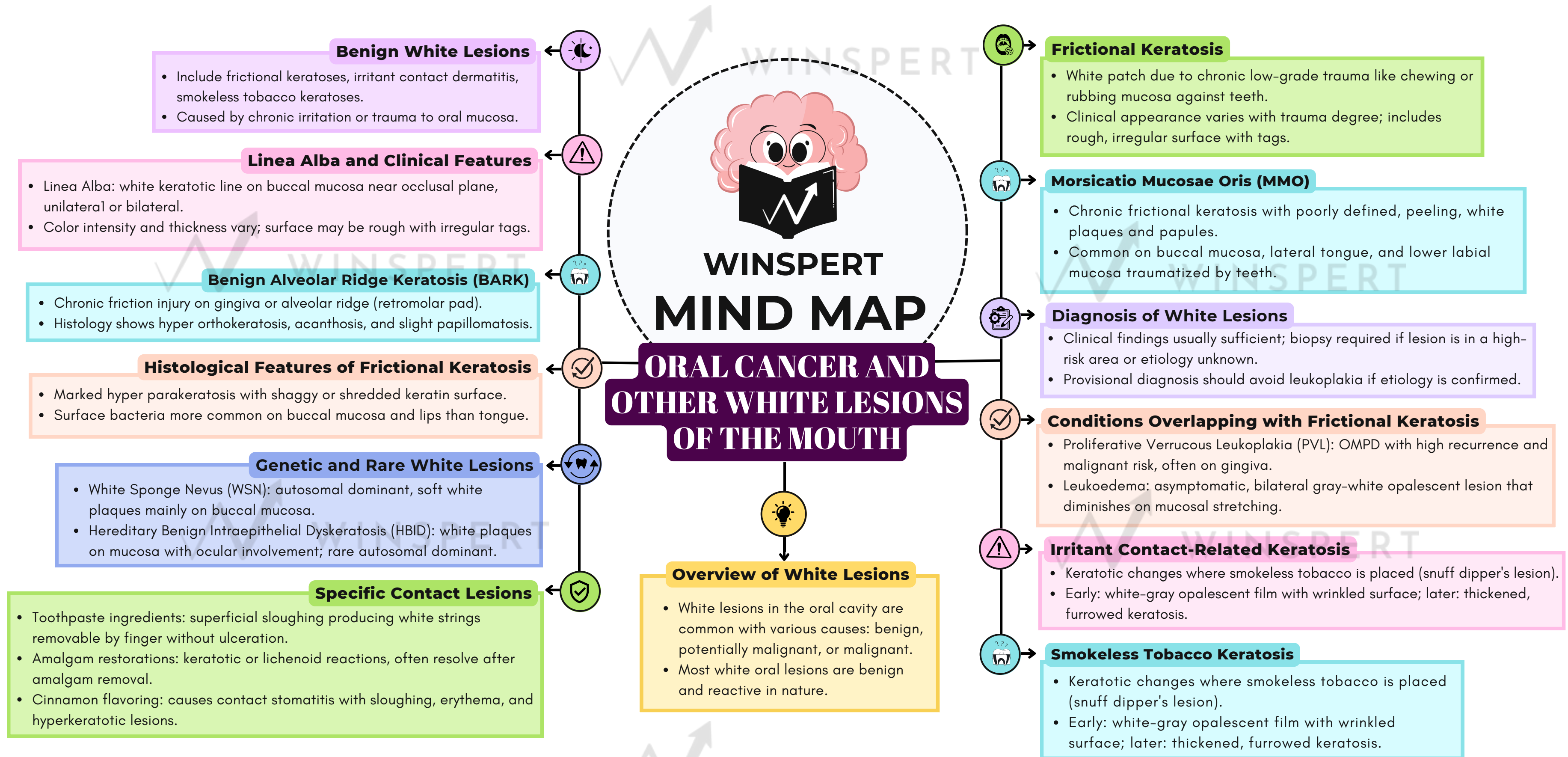
ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

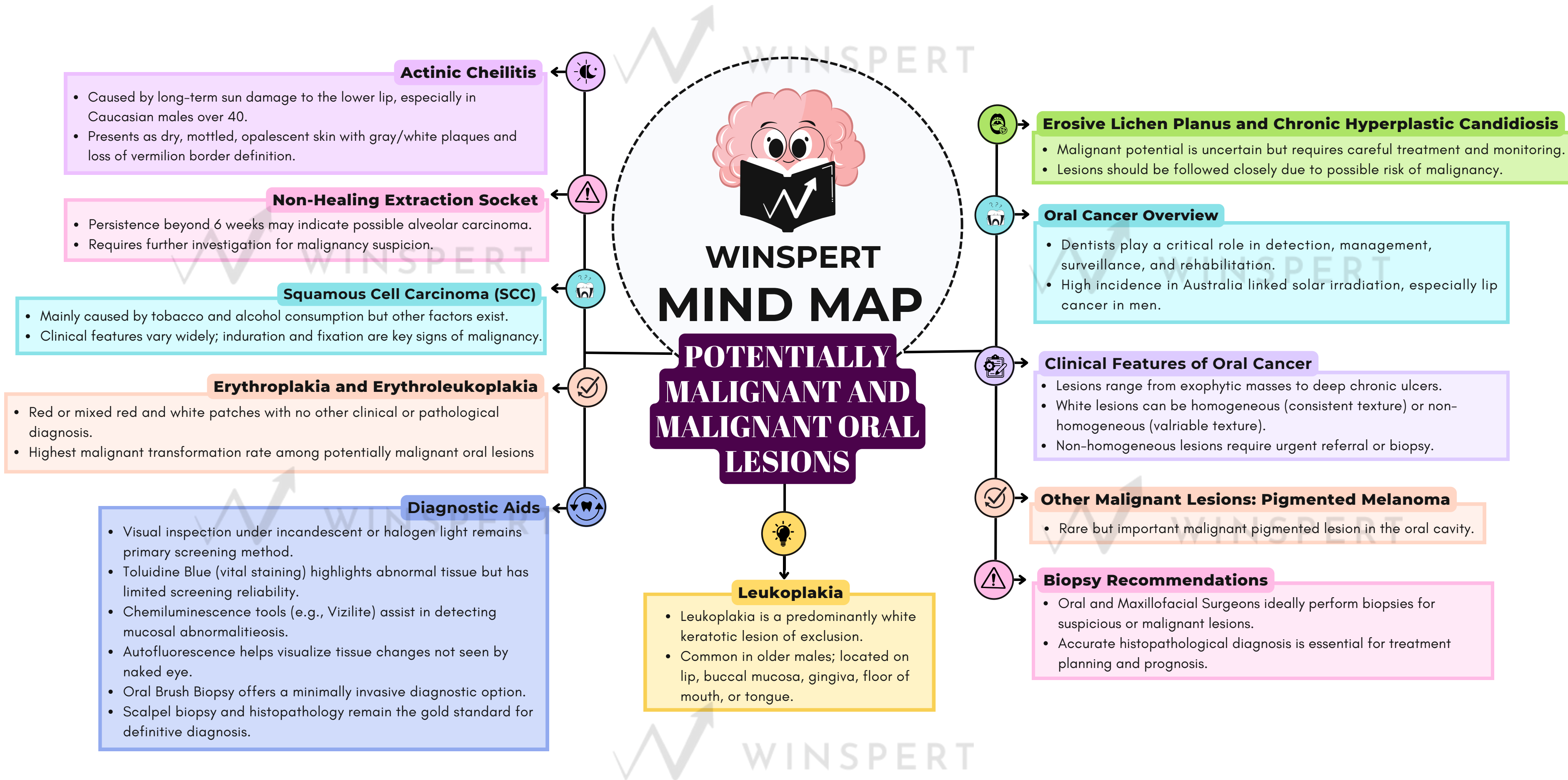


MIND MAP & CUE CARDS



BY DR. JIGYASA SHARMA







**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 1

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

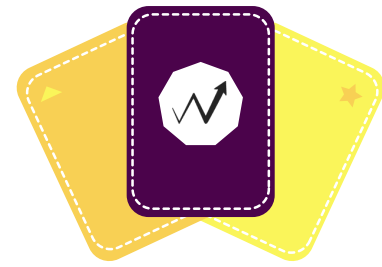


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 2

What causes frictional keratosis in the oral cavity?



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

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.

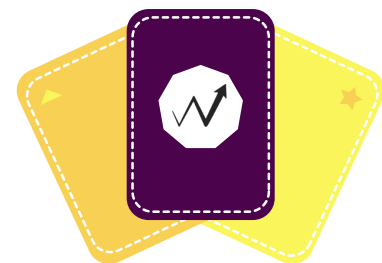


**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 3

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

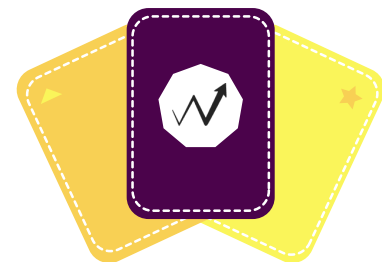


**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

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.



**WINSPERT
CUE CARDS**

**ORAL CANCER AND
OTHER WHITE
PATCHES IN MOUTH**

Question 4

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

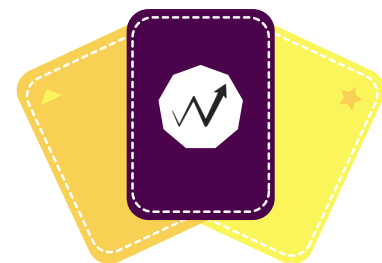


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

**ORAL CANCER AND
OTHER WHITE
PATCHES IN MOUTH**

Question 5

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



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Answer 5

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



**WINSPERT
CUE CARDS**

**ORAL CANCER AND
OTHER WHITE
PATCHES IN MOUTH**

Question 6

**When is biopsy recommended
for white oral lesions?**



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

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).

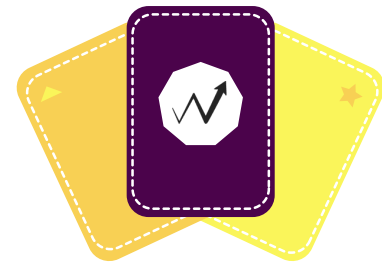


**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 7

**What are the histological
features of frictional
keratosis?**



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

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.



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 8

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



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Answer 8

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



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 9

**How can you differentiate
leukoedema from frictional
keratosis clinically?**



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Answer 9

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



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 10

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



**WINSPERT
CUE CARDS**

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.

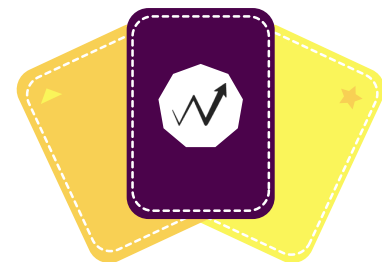


**WINSPERT
CUE CARDS**

**ORAL CANCER AND
OTHER WHITE
PATCHES IN MOUTH**

Question 11

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

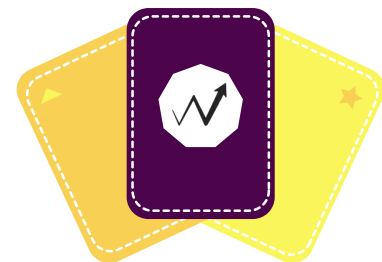


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 12

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



**WINSPERT
CUE CARDS**

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.

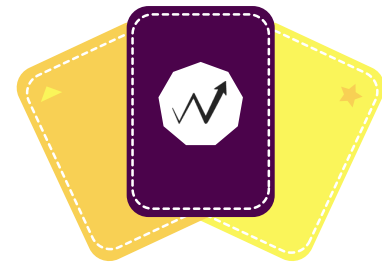


**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 13

**Define leukoplakia and its
significance.**

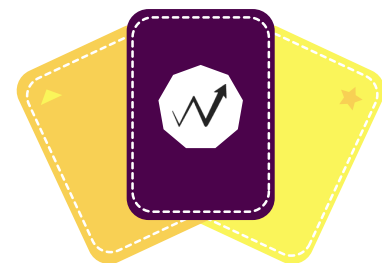


**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 14

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



**WINSPERT
CUE CARDS**

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.



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

Question 5

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



**WINSPERT
CUE CARDS**

ORAL CANCER AND OTHER WHITE PATCHES IN MOUTH

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.