Oral Cavity 😬

Pathologic conditions of the oral cavity can broadly involve:

1. Teeth and supporting structures
2. Oral mucosa
3. Salivary glands
4. Jaws
5. Odontogenic cysts and tumors (derived from tooth-forming epithelial/mesenchymal tissues)

Diseases of Teeth and Supporting Structures

Dental Caries 🦷

Definition

Dental caries is focal demineralization of enamel and dentin caused by acid production from bacterial fermentation of sugars.

Pathogenesis (Flowchart)

High Carbohydrate/Sugar Intake → Oral Bacteria Ferment Sugars → Acid Production → Demineralization of Enamel & Dentin → Progressive Tooth Destruction → Dental Caries

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Important Facts

- Most common cause of tooth loss before 35 years of age
- More prevalent with:
    - High intake of processed/refined carbohydrates
    - Poor oral hygiene
- Incidence has decreased in developed countries due to:
    - Better oral hygiene
    - Fluoridated drinking water

Role of Fluoride ✨

Fluoride protects teeth by:

Fluoride Incorporation into Enamel → Formation of Fluoroapatite → Increased Resistance to Acid Dissolution → Reduced Risk of Caries

Exam Pearls 📌

- Main etiologic factor: Acid generated by bacterial sugar fermentation
- Protective factor: Fluoride
- Age association: Most common cause of tooth loss in <35 years

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Gingivitis 🩸

Definition

Inflammation of the gingiva and associated soft tissues surrounding teeth.

Etiology

Most common cause: Poor oral hygiene

Pathogenesis

Poor Oral Hygiene → Accumulation of Dental Plaque → Mineralization of Plaque → Formation of Calculus (Tartar) → Gingival Inflammation → Gingivitis

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Dental Plaque Composition

Dental plaque is a sticky biofilm composed of:

Component	Description
Bacteria	Major pathogenic component
Salivary proteins	Contribute to matrix formation

Desquamated epithelial cells	Shed oral epithelial cells

Clinical Features

- Gingival erythema
- Edema
- Bleeding gums
- Tenderness / discomfort

Epidemiology

- Can occur at any age
- Most prevalent/severe in adolescents
- Present in 40–60% of adolescents

Key Feature

Gingivitis is reversible with proper oral hygiene.

Management / Prevention

- Regular brushing
- Flossing
- Professional dental cleaning

Periodontitis 🦴

Definition

A chronic inflammatory disease involving:

- Periodontal ligaments
- Alveolar bone

- Cementum

Significance

Can lead to:

Periodontal Inflammation → Destruction of Supporting Structures → Tooth Loosening → Tooth Loss

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Pathogenesis

Periodontitis develops due to altered oral microbiota associated with poor oral hygiene.

Microbial Shift in Periodontitis

Healthy Gingiva	Active Periodontitis

Facultative Gram-positive organisms	Anaerobic / Microaerophilic Gram-negative bacteria

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Important Associated Organisms 

Organism	Association
Aggregatibacter actinomycetemcomitans	Strongly linked to periodontitis
Porphyromonas gingivalis	Major periodontal pathogen
Prevotella intermedia	Associated with active disease

Pathogenesis

Poor Oral Hygiene → Altered Gingival Bacterial Flora →

Colonization by Pathogenic Gram-Negative Bacteria →

Chronic Inflammation → Destruction of Periodontal Ligament & Alveolar Bone → Tooth Loss

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High-Yield Comparison Table 📚

Feature	Dental Caries	Gingivitis	Periodontitis
Main Site Affected	Enamel/Dentin	Gingiva	Periodontal Ligament, Bone, Cementum
Primary Cause	Acid from bacterial sugar fermentation	Plaque/Calculus buildup	Altered oral microbiota due to poor hygiene
Reversible?	No	Yes	Limited / Often Progressive

Can Cause Tooth Loss?	Yes	Rarely	Yes, commonly if severe
Hallmark	Demineralization	Inflamed bleeding gums	Destruction of tooth support

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Exam Summary ⭐

Diseases of Teeth & Supporting Structures

- Dental Caries
  - Most common cause of tooth loss <35 years
  - Due to acid-mediated demineralization from bacterial sugar fermentation
- Gingivitis
  - Reversible inflammation of gingiva
  - Caused by plaque and calculus accumulation

- Periodontitis
    - Chronic inflammatory destruction of periodontal support
    - Associated with poor oral hygiene and pathogenic oral flora
    - May lead to tooth loss

Oral Inflammatory Lesions 🔥

Aphthous Ulcers (Canker Sores) 😖

Definition

Common superficial painful oral mucosal ulcers affecting up to 40% of the population.

Epidemiology

- More frequent in first 2 decades of life
- Often recurrent
- Frequently familial

Associations

Aphthous ulcers may be associated with:

Associated Condition	Clinical Relevance
Celiac disease	Malabsorption/autoimmune association
Inflammatory bowel disease	Especially Crohn disease
Behçet disease	Recurrent oral/genital ulcers + uveitis

Etiology

Unknown (idiopathic in most cases)

Morphology

Typical lesion features:

Feature	Description
Number	Solitary or multiple
Depth	Shallow
Base	Hyperemic
Surface	Thin exudative covering
Margin	Narrow erythematous rim

Clinical Course

Painful Oral Ulcer → Persists for Several Days →

Spontaneous Healing in 7–10 Days → May Recur

High-Yield Exam Pearls 📌

- Painful recurrent oral ulcers
- Unknown etiology
- Heal without scarring
- Associated with Behçet disease

Herpes Simplex Virus (HSV) Infection 🦠

Etiology

Most orofacial infections caused by:

Virus	Frequency
HSV-1	Most common
HSV-2	Less common, increasing orally due to changing sexual practices

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Primary Infection

Typical Features

- Usually occurs in children aged 2–4 years
- Often asymptomatic

Symptomatic Primary Infection

In 10–20% of cases:

Primary HSV Infection → Acute Herpetic Gingivostomatitis → Abrupt Onset of Vesicles & Ulcers Throughout Oral Cavity

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Latency and Reactivation

After primary infection:

HSV Enters Sensory Nerves → Latency in Nerve Ganglia → Reactivation During Stress/Immunosuppression → Recurrent Herpetic Lesions ("Cold Sores")

Triggers for Reactivation

Trigger	Notes

Trauma	Local injury
Allergies	Immune alteration
UV Light	Sun exposure
Temperature Extremes	Heat/Cold
URTI	Fever/stress
Pregnancy	Hormonal
Menstruation	Hormonal
Immunosuppression	Major risk factor

Recurrent Lesions

Feature	Description
Size	1–3 mm
Appearance	Grouped vesicles
Common Sites	Lips, buccal mucosa, gingiva, hard palate, nasal orifices

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Morphology / Histology 🔬

HSV-Infected Cells Show:

Finding	Description
Ballooning degeneration	Swollen infected cells

Eosinophilic intranuclear inclusions	Viral inclusions
Multinucleated giant cells	Cell fusion (polykaryons)

Clinical Course

- Usually heals in 7–10 days
- May persist/progress in immunocompromised patients

High-Yield Exam Pearls 📌

- Cold sore = recurrent HSV infection
- Latently resides in nerve ganglia
- Multinucleated giant cells + intranuclear inclusions

Oral Candidiasis (Thrush) 🍄

Definition

Most common fungal infection of the oral cavity

Etiologic Organism

- Candida albicans

  Normally part of oral flora; causes disease only under predisposing conditions.

Predisposing Factors

Factor	Mechanism
Immunosuppression	Reduced host defense
Virulent Candida strain	Increased pathogenicity
Altered oral microbiota	Loss of competing flora
Broad-spectrum antibiotics	Disrupt normal flora

Major Clinical Forms

Form	Description
Pseudomembranous	Most common; classic thrush
Erythematous	Red inflamed mucosa

Hyperplastic	White adherent plaques

Pseudomembranous Candidiasis (Thrush)

Classic Appearance

- Curdlike gray-white membrane
- Can be scraped off
- Leaves erythematous base

Composition of Thrush Plaque

Thrush Membrane Contains:

- Candida organisms
- Fibrin
- Suppurative inflammatory exudate

- Necrotic debris

Disease Severity

Mild Immunosuppression / Diabetes → Superficial Oral Disease

Severe Immunosuppression → Deep/Invasive Candidiasis

Severe Disease Seen In

High-Risk Group	Why
Organ transplant recipients	Immunosuppressants
Stem cell transplant patients	Profound immunosuppression

Neutropenic patients	Impaired innate immunity
Chemotherapy patients	Marrow suppression
AIDS patients	Severe T-cell deficiency

High-Yield Comparison Table 📚

Feature	Aphthous Ulcers	HSV Infection	Oral Candidiasis
Cause	Unknown	HSV-1/HSV-2	Candida albicans
Lesion Type	Painful ulcers	Vesicles → Ulcers	White plaques
Painful?	Very painful	Often painful	Variable
Scrapable?	No	No	Yes

Recurrent?	Yes	Yes	Possible
Major Risk Factor	Idiopathic/Systemic disease	Reactivation triggers	Immunosuppression/Antibiotics

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Exam Summary ⭐

Oral Inflammatory Lesions

- Aphthous Ulcers
  - Painful recurrent superficial ulcers
  - Unknown cause
  - Associated with Behçet disease, IBD, celiac disease
- HSV Infection
  - Causes vesicular oral lesions/cold sores
  - Virus remains latent in nerve ganglia

    - Reactivation common
- Oral Candidiasis
    - Most common fungal oral infection
    - Often after antibiotics or immunosuppression
    - Thrush = scrapable white plaques

Proliferative and Neoplastic Lesions of the Oral Cavity 🦷

Fibrous Proliferative Lesions

Fibroma

Definition

A benign reactive submucosal fibrous tissue mass caused by chronic irritation leading to connective tissue hyperplasia.

> Despite the name, oral fibroma is usually reactive rather than a true neoplasm.

Pathogenesis

Chronic Local Irritation → Repeated Minor Trauma → Reactive Connective Tissue Hyperplasia → Formation of Fibroma

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Common Site

- Buccal mucosa along bite line (most common)

Clinical Features

Feature	Description
Appearance	Firm nodular submucosal mass
Growth	Slow
Symptoms	Usually painless
Nature	Reactive, benign

Treatment

Complete Surgical Excision

+

Removal of Irritating Source

High-Yield Pearl 📌

- Fibroma = reactive hyperplasia, not true neoplasm

Pyogenic Granuloma

Definition

A vascular inflammatory lesion of oral mucosa/gingiva.

Misnomer: Neither pyogenic nor a true granuloma

Epidemiology

Common in:

Group	Association

Children	Frequent
Young adults	Frequent
Pregnant women	"Pregnancy tumor"

Pathogenesis

Likely represents exuberant granulation tissue response to irritation/trauma/hormonal influence.

Morphology

Feature	Description
Color	Red to purple

Surface	Often ulcerated
Consistency	Soft/vascular
Histology	Immature capillary proliferation resembling granulation tissue

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Clinical Significance

Rapid enlargement may mimic malignancy ⚠️

Natural Course

Pyogenic Granuloma → May Regress OR Mature into Dense Fibrous Mass OR Develop into Peripheral Ossifying Fibroma

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Treatment

- Complete surgical excision

Leukoplakia

Definition

WHO Definition:

> "A white patch or plaque that cannot be scraped off and cannot be characterized clinically or pathologically as any other disease."

Important Diagnostic Note ⚠️

Leukoplakia is a diagnosis of exclusion

NOT leukoplakia if white lesion is due to:

- Candidiasis
- Lichen planus
- Frictional/obvious irritation
- Other identifiable causes

Epidemiology

- Present in ~3% of world population
- Usually affects ages 40–70 years
- Male predominance 2:1

Malignant Potential

- 5–25% show dysplasia / premalignant change

All leukoplakias are considered precancerous until proven otherwise by biopsy

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Major Risk Factor 🚬

- Tobacco use (most important)

Includes:

- Cigarettes
- Cigars
- Pipes
- Chewing tobacco

Erythroplakia

Definition

A red, velvety, flat or slightly depressed oral mucosal lesion.

Clinical Importance

Much higher malignant potential than leukoplakia

Malignant Transformation Risk

- >50% undergo malignant transformation

High-Yield Comparison

- Leukoplakia = White lesion
- Erythroplakia = Red lesion

- Erythroplakia is more dangerous

Histologic Features of Leukoplakia/Erythroplakia 🔬

Histology shows a spectrum from benign hyperplasia to severe dysplasia:

Normal/Reactive Hyperkeratosis → Acanthosis → Epithelial Dysplasia → Severe Dysplasia → Carcinoma in Situ → Invasive Squamous Cell Carcinoma

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Histologic Findings

Finding	Description
Hyperkeratosis	Thickened keratin layer

Acanthosis	Thickened squamous epithelium
Dysplasia	Cellular atypia/disordered maturation
Inflammatory infiltrate	Lymphocytes/macrophages in subepithelium

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Key Correlation

Most severe dysplasia is typically seen in erythroplakia

High-Yield Comparison Table 📚

Feature	Fibroma	Pyogenic Granuloma	Leukoplakia	Erythroplakia

Nature	Reactive fibrous hyperplasia	Vascular inflammatory lesion	Premalignant white patch	Premalignant red patch
Color	Normal mucosal color	Red/Purple	White	Red/Velvety
Scrapable?	No	No	No	No
Malignant Potential	None	None	Moderate	High
Key Risk Factor	Chronic irritation	Trauma/Hormones	Tobacco	Tobacco

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Exam Pearls ⭐

Fibrous Proliferative & Premalignant Oral Lesions

- Fibroma

    - Reactive fibrous hyperplasia from chronic irritation
    - Common on buccal mucosa bite line
- Pyogenic Granuloma
    - Vascular gingival lesion
    - Common in pregnancy ("pregnancy tumor")
    - Histology resembles granulation tissue
- Leukoplakia
    - White patch that cannot be scraped off
    - Premalignant until biopsy proves otherwise
- Erythroplakia
    - Red velvety lesion
    - Higher malignant potential than leukoplakia

Squamous Cell Carcinoma of the Oral Cavity ⚠️

Overview

- ~95% of oral cavity cancers are squamous cell carcinomas (SCC)
- Remaining cases are mostly salivary gland adenocarcinomas
- Oral SCC is the 6th most common neoplasm worldwide

Clinical Importance

Oral SCC is an aggressive epithelial malignancy with poor long-term survival.

Prognosis

- Overall long-term survival remains <50%

Why Prognosis Is Poor

Poor Early Detection → Diagnosis at Advanced Stage → Larger Tumor Burden / Metastasis → Worse Outcome

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Multiple Primary Tumors & Field Cancerization

Key Concept

Patients with oral SCC are at risk of developing additional independent primary tumors.

Statistics

- New primary tumors develop at 3–7% per year
- Survivors at 5 years have up to 35% risk of another primary tumor

Field Cancerization Concept 🧠

Chronic Exposure to Carcinogens → Widespread Mucosal Genetic Damage → Multiple Independent Premalignant Clones → Development of Multiple Primary Tumors

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Clinical Implication

> Lifelong surveillance is essential after diagnosis of oral SCC.

Pathogenesis

Oral/oropharyngeal SCC arises via two major pathogenic pathways:

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1. Carcinogen-Associated SCC 🚬🍷

Major Risk Factors

Risk Factor	Notes
Tobacco	Smoked or chewed
Alcohol	Synergistic with tobacco
Betel quid / Paan	Important in India/Southeast Asia

Betel Quid Composition

- Areca nut
- Slaked lime
- Tobacco
- Wrapped in betel leaf

Molecular Changes

Common mutations include:

Gene	Role
TP53	Tumor suppressor
RAS	Cell proliferation signaling

Pathogenesis Flowchart

Chronic Tobacco/Alcohol Exposure → Repeated Mucosal Injury → Accumulation of Genetic Mutations → Dysplasia → Carcinoma In Situ → Invasive SCC

2. HPV-Associated SCC 🦠

Key Features

Feature	Description
HPV Type	HPV-16 most common
Common Site	Tonsillar crypts / Base of tongue
Mutation Burden	Lower than tobacco-associated SCC
Marker	p16 overexpression
Prognosis	Better than HPV-negative SCC

Pathogenesis Flowchart

High-Risk HPV Infection → Viral Oncoprotein Expression → Cell Cycle Dysregulation → Dysplasia → SCC Development

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High-Yield Pearl 📌

HPV-positive SCC has a better prognosis than HPV-negative SCC

Rising Incidence in Young Adults

Increasing SCC incidence in:

- Patients <40 years
- No smoking history
- No HPV infection

Pathogenesis remains unknown

Morphology

Common Sites of Oral SCC

Site	Frequency
Ventral surface of tongue	Most common
Floor of mouth	Common
Lower lip	Common
Soft palate	Common
Gingiva	Common

Gross Appearance

Early Lesions

May appear as:

- Raised firm pearly plaques
- Roughened/verrucous thickening
- Leukoplakic/erythroplakic lesions

Advanced Lesions

As tumor enlarges:

SCC Growth → Ulceration → Exophytic/Protruding Mass → Rolled/Indurated Borders

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Histopathology 🔬

Precursor Lesion

Develops from dysplastic precursor lesions

Histologic Spectrum

Type	Description
Well-differentiated	Keratinizing SCC
Moderately differentiated	Intermediate
Poorly differentiated	Anaplastic
Sarcomatoid	Spindle-cell variant

Important Note

Degree of keratinization does NOT reliably predict biologic behavior

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Spread / Metastasis

Local Behavior

- Typically locally invasive before metastasizing

Regional Metastasis

Most common:

- Cervical lymph nodes

Distant Metastasis Sites

Site	Frequency
Mediastinal lymph nodes	Common
Lungs	Common
Liver	Common

High-Yield Comparison Table 📚

Feature	Tobacco/Alcohol SCC	HPV-Associated SCC
Typical Patient	Older smoker/drinker	Younger patient
Common Site	Oral cavity/tongue/floor of mouth	Tonsillar crypt/base of tongue

HPV Status	Negative	Positive
Mutation Burden	High	Lower
Key Marker	TP53/RAS mutations	p16 overexpression
Prognosis	Worse	Better

Exam Summary ⭐

Oral Squamous Cell Carcinoma

- Accounts for 95% of oral cavity cancers
- Strongly associated with:
  - Tobacco
  - Alcohol
  - Betel quid

- Increasingly associated with HPV-16
- Commonly arises from:
    - Leukoplakia / Erythroplakia / Dysplasia
- Common metastatic site:
    - Cervical lymph nodes
- HPV-positive tumors:
    - Better prognosis
    - Often p16 positive

-> The End <-