

- Q1. List the benign and malignant tumors of epithelial tissue of oral mucosa.**
- Q2. Describe the clinical features, histological features, treatment and prognosis of squamous papilloma.**
- Q3. Describe the clinical features and histological features verrucous carcinoma.**
- Q4. Squamous Cell Carcinoma**
- Q5. Grading and staging of oral squamous cell carcinoma.**
- Q6. Describe the pathogenesis and clinical radiographic appearance of central ossifying fibroma with histological features.**
- Q7. Describe and enumerate the precancerous lesions of oral cavity. Describe the aetiology, clinical features, and histopathology of:**
- A). Leukoplakia**
- B). Erythroplakia**
- C). Oral submucous fibrosis**
- D). Lichen planus**
- Q8. Name some giant cell lesions of oral cavity. Describe in detail Central and peripheral giant cell granuloma.**
- Q9. Describe dysplasia.**
- Q10. Briefly explain changes in epithelial dysplasia.**
- Q11. TNM classification**
- Q12. Burkitt's lymphoma**
- Q13. Kaposi sarcoma.**
- Q14. Hodgkin and non hodgkin lymphoma.**
- Q15. Rodent Ulcer (Basal Cell Carcinoma)**

Q1. List the benign and malignant tumors of epithelial tissue of oral mucosa.

Benign tumors:

Benign Tumors - "Some Very Cool Kids"

- **Squamous Papilloma**
- **Verruca Vulgaris**
- **Condyloma Acuminatum**
- **Keratoacanthoma**

Malignant tumors:

Malignant Tumors - "Bad Evil Villains Scare All Brave Little Ninjas"

- **Basal cell carcinoma (rodent ulcer)**
- **Epidermoid carcinoma (squamous cell carcinoma)**
- **Verrucous carcinoma**
- **Spindle cell carcinoma**

- **Adenoid squamous cell carcinoma**
- **Basaloid squamous cell carcinoma**
- **Lymphoepithelioma and transitional cell carcinoma**
- **Nasopharyngeal carcinoma**
- **Malignant melanoma**

Q2. Describe the clinical features, histological features, treatment and prognosis of squamous papilloma.



Squamous Papilloma:

- Linked to **HPV (types 6 and 11)**, common in **skin warts**.
- Represents the **4th most common** oral mucosal growth (3-4% of biopsied oral soft tissue lesions).
- **Not associated with oral cancers or potentially malignant lesions.**
- **Low virulence and not contagious.**

Clinical Features:

- **Exophytic growth** with **finger-like projections** (cauliflower-like appearance).
- Usually **painless**, **well-circumscribed**, and either **white** or **pink**.
- Found on the **tongue**, **lips**, **buccal mucosa**, **gingiva**, and **palate**.
- Typically **a few millimeters** in size but can grow larger.
- Occurs at **any age**, including in **children**.

Histologic Features:

- Shows **finger-like projections** of **stratified squamous epithelium** with a **connective tissue core**.
- Some have **hyperkeratosis** due to **trauma** or **friction**.
- **Spinous cells** proliferate in a **papillary pattern**.
- **Koilocytes** (HPV-altered cells) may or may not be present.
- **Chronic inflammatory cells** can be seen in the **connective tissue**.

Treatment and Prognosis:

- **Surgical excision** including the **base** of the lesion is required.
- **Recurrence is rare** if properly removed.
- **Malignant transformation is unlikely**, but fixed or hardened lesions should be closely monitored.

Q3. Describe the clinical features and histological features verrucous carcinoma.



Verrucous carcinoma is a **diffused papillary, non-metastasizing**, well-differentiated **malignant neoplasm** of the **oral epithelium**. It's also called **Ackerman's tumour** or **pseudo-epitheliomatous papilloma**.

Clinical Features:

- A high percentage of patients are **tobacco chewers**, and some have reported **ill-fitting dentures**.
- It usually occurs in **elderly patients** (around **60-70 years**), with **males** being more commonly affected.
- Common locations include **gingiva, alveolar mucosa, and buccal mucosa**.
- It presents as **slow-growing, exophytic papillary growth** with a **white pebbly surface**.
- It can appear as a **single entity** or with **multiple lesions** in different parts of the oral cavity.
- **Buccal mucosa lesions** may be extensive, causing **pain, tenderness, and difficulty eating**.
- The regional **lymph nodes** are often **enlarged and tender**.
- It is **less aggressive** than squamous cell carcinoma, with **more lateral invasion and less vertical invasion**.
- The surface may be **keratinized** and covered with a **white leukoplakic film**.

Histopathology:

- The **epithelium** shows a **papillary surface** covered by **thick parakeratin**.
- **Bulb-like acanthotic rete ridges** extend into the underlying **connective tissue**.
- **Parakeratin plugging**: deep **cleft-like spikes** lined with parakeratin extend from the surface.
- All **rete ridges** push into the connective tissue, forming a **pushing margin**.
- The **basement membrane** remains **intact**, and the connective tissue shows **inflammatory cell infiltration**.
- Formation of **epithelial pearls** and **microcysts** is seen.

Treatment:

- **Surgical excision or laser therapy** is done, and the **prognosis** is generally **good**.



Q4. Squamous Cell Carcinoma

Synonyms:

Oral Squamous Cell Carcinoma = Epidermoid Carcinoma = Epithelioma

Overview:

- Most common oral carcinoma
- 60% arise from anterior 2/3 of the tongue
- Remainder from the base of the tongue
- Malignant neoplasm with squamous differentiation

Aetiology:

- **Physical trauma**
- **Alcohol**
- **Tobacco smoking**
- **Syphilis, sepsis, chronic dental trauma**
- **Chronic superficial glossitis**

Clinical Features:

- Common in ages 40-60 years
- Males more affected
- **Painless growth in early stages**
- **Becomes painful after ulceration**
- **Enlarged lymph nodes**
- **Excessive salivation, foetor oris, sore throat, immobility of tongue, hoarseness of voice, dysphagia**

Carcinoma of Tongue Varieties:

- i. **Ulcerative:** Near tongue edge; irregular, raised, everted edges; indurated base; yellowish-grey slough floor
- ii. **Warty growth:** Broad, indurated base
- iii. **Indurated plaque or mass**
- iv. **Fissure:** Chronic fissure that does not heal

Spread of Carcinoma:

- **Local spread** by infiltration and invasion
- **Lymphatic spread**
- **Bloodstream spread**

Treatment:

- **Surgery**
- **Radiotherapy**

Prognosis:

- 5-year survival rate for tongue cancer: <25%

Q5. Grading and staging of oral squamous cell carcinoma.

STAGING OF ORAL TUMORS

T - Primary Tumor:

- **T_{IS}**: Carcinoma in situ.
- **T₁**: Tumor ≤ 2 cm.
- **T₂**: Tumor > 2 cm but ≤ 4 cm.
- **T₃**: Tumor > 4 cm.

N - Regional Lymph Nodes:

- **N₀**: No palpable lymph nodes or nodes present but no metastasis suspected.
- **N₁**: Palpable homolateral lymph node(s), not fixed; metastasis suspected.
- **N₂**: Palpable contralateral or bilateral lymph node(s), not fixed; metastasis suspected.
- **N₃**: Palpable lymph node(s) that are fixed; metastasis suspected.

M - Distant Metastasis:

- **M₀**: No distant metastasis.
- **M₁**: Evidence of metastasis other than cervical lymph nodes.

Clinical Stage - Grouping of Carcinoma of the Oral Cavity:

- **Stage I**: T₁ N₀ M₀
- **Stage II**: T₂ N₀ M₀
- **Stage III**: T₃ N₀ M₀, T₁ N₁ M₀, T₂ N₁ M₀, T₃ N₁ M₀
- **Stage IV**: T₁ N₂ M₀, T₂ N₂ M₀, T₃ N₂ M₀, T₁ N₃ M₀, T₂ N₃ M₀, T₃ N₃ M₀

Q6. Describe the pathogenesis and clinical radiographic appearance of central ossifying fibroma with histological features.



Central ossifying fibroma is a central neoplasm of bone.

Clinical Features

- Occurs in **young adults**, more common in **females**.
- More common in the **mandible** than the maxilla.
- Seen in the **body of the mandible**, from **premolar to molar** region.
- Lesion is usually **asymptomatic** until there is **noticeable swelling** or mild deformity.
- **Displacement of teeth** may be an early sign.

- **Slow growing**, and the **cortical bone plates** and overlying **mucosa/skin** remain intact.
- **Rarely causes pain** or paraesthesia.

Radiological Features

- Appearance varies depending on the **stage**.
- **Well-circumscribed**, clearly separated from surrounding bone.
- In early stages, it is **radiolucent** with no internal radio-opacities.
- As it matures, **calcification** occurs, with **flecks of radio-opacity**, eventually forming a **uniformly radio-opaque mass**.
- **Displacement of teeth** and **impingement on adjacent structures** is common.

Histological Features

- Composed of **delicate interlacing collagen fibers**.
- **Connective tissue** shows many small foci of **irregular bony trabeculae**.

Treatment

- **Surgical excision** with conservative approach. **Recurrence is rare**.

Q7. Describe and enumerate the precancerous lesions of oral cavity. Describe the aetiology, clinical features, and histopathology of:

- A). Leukoplakia
- B). Erythroplakia
- C). Oral submucous fibrosis
- D). Lichen planus

Premalignant lesions are tissue changes where **cancer** is more likely to occur. Examples:

- **Leukoplakia**
- **Erythroplakia**
- **Oral submucous fibrosis (OSMF)**
- **Lichen planus**
- **Dyskeratosis Congenita**

A).Leukoplakia:



It is a **white keratotic lesion** on the oral mucosa, not characterized as any other disease. It is the most common **potentially malignant** lesion of the oral mucosa.

Aetiology (Causes):

1. **Tobacco** (cigarettes, bidis, cigars, pipes): Smoking produces harmful end-products that, along with heat, irritate the **oral mucosa**.
2. **Alcohol**: Many alcohol users develop leukoplakia.
3. **Candidiasis**: Chronic fungal infections.
4. **Vitamin A Deficiency**: Causes metaplasia and **hyperkeratinization**.
5. **Syphilis**: Plays a minor role.
6. **Hormonal Imbalance**: Affects **keratin** production in oral epithelium.
7. **Trauma**: Like **ill-fitting dentures**, sharp teeth.
8. **Galvanism**: Electrochemical reaction in the mouth.
9. **Idiopathic**: Unknown cause.
10. **UV Radiation**

Clinical Features:

- Two main forms as per **WHO (1980)**:
 1. **Homogenous**: Uniformly white lesions.
 2. **Non-homogenous**: White + red patches.
- Further divided:
 1. Homogenous:
 - **Smooth**
 - **Furrowed** (fissured)
 - **Ulcerated**
 2. Non-homogenous:
 - **Nodular/Speckled**: Raised white areas mixed with **red** patches.
- **Age**: Usually after **30 years**, peak after **50 years**.
- Common sites: **Buccal mucosa, commissures, alveolar ridge, tongue, lips, palate**.
- **Appearance**: Solitary or multiple **white patches**, cannot be removed by scraping.
- **Size**: Can be small (few mm) or large (several cm).
- Surface may feel **smooth, wrinkled, or rough**.
- **Color**: Whitish, greyish, or even brownish-yellow (due to tobacco).
- Usually **asymptomatic**, but can cause **pain, burning, or thickening** feeling.

Histologic Features

Leukoplakia shows a variety of histologic changes related to **keratinization, cellular layer changes, epithelium thickness, and connective tissue stroma alterations**.

Keratinization pattern:

- Leukoplakia presents **hyperorthokeratinization** or **hyperparakeratinization**, with or without **epithelial dysplasia**.
- There's an abnormal increase in the thickness of the **orthokeratin layer** in areas that are usually keratinized.
- Hyperkeratinization of normally keratinized epithelium or some **parakeratin deposition** in non-keratinized areas is an important criterion.
- **Epithelial dysplasia** is more often associated with hyperkeratinized lesions.

Changes in the cellular layer:

- **Epithelial dysplasia** is the hallmark of changes in leukoplakia.
- Histopathological features include:
 - **Loss of polarity** of basal cells
 - Increased **nuclear-cytoplasmic ratio**

- Irregular **epithelial stratification**
- **Cellular pleomorphism**
- **Nuclear hyperchromatism**
- Reduced **cellular cohesion**
- Enlarged **nucleoli**
- Increased **mitotic figures**

Thickness of the epithelium:

- The thickness changes as **epithelial atrophy** or **acanthosis** in leukoplakia.

Alteration in connective tissue:

- There may be destruction of **collagen fibers** and a **chronic inflammatory cell infiltrate** in the connective tissue stroma.

Modified Classification & Staging for Oral Leukoplakia (van der Wall, 2000):

- **L1:** Size <2 cm
- **L2:** Size 2-4 cm
- **L3:** Size >4 cm
- **Lx:** Size not specified
- **P0:** No epithelial dysplasia
- **P1:** Distinct epithelial dysplasia
- **Px:** Dysplasia not specified

OLEP Staging System:

- **Stage I:** L1 P0
- **Stage II:** L2 P0
- **Stage III:** L3 P0 or L1/L2 P1
- **Stage IV:** L3 P1

Diagnostic Procedures:

- **Biopsy** is key.
- Other methods (Toludine blue staining, Lugol's iodine, exfoliative cytology) are of limited value.

Treatment:

- **Surgical excision**
- **Cryosurgery**
- **CO₂-laser surgery**
- **Retinoids & other drugs**
- **Photodynamic therapy**

B). ERYTHROPLAKIA



- **Red patch** that cannot be diagnosed as any other condition.
- **Relatively rare** compared to leukoplakia.
- **Almost always** associated with **premalignant changes** histologically.
- **Important precancerous lesion.**

Aetiology

- **Unknown.**
- **Smoking and alcohol abuse** are key factors.

Clinical Features

- **Lack of keratin** production; epithelium often atrophic but may be hyperplastic.
- **Red color** due to lack of keratinization and thinning of epithelium, which shows underlying vasculature.
- **High connective tissue rete pegs** into the epithelium.
- **Chronic inflammation** in underlying connective tissue.

Treatment

- **Remove irritating agents.**
- **Prompt biopsy** is mandatory.
- **Recurrence rate** of less than 5% reported.

C). Oral Submucous Fibrosis (OSMF)



Definition: OSMF is a precancerous condition affecting the oral cavity, marked by inflammatory changes in the mucosa leading to fibrosis and loss of elasticity.

Synonyms:

- Atropia idiopathica

- Idiopathic scleroderma of the mouth
- Idiopathic palatal fibrosis
- Sclerosing stomatitis
- Submucosal fibrosis of the palate

Aetiology:

- **Excessive consumption of red chillies**
- **Excessive areca nut chewing**
- Nutritive deficiency
- **Immunological factors**
- Genetic factors
- **Protracted tobacco use**

Pathogenesis: Aetiological factors cause oral mucosa alterations, increasing hypersensitivity reactions, which may lead to malignant transformation (4.5-7.6%).

Clinical Features:

- Affects individuals typically aged 20-40 years
- **Females are affected more than males**
- Commonly seen in buccal mucosa, retromolar area, uvula, tongue
- Initial symptoms: burning sensation, particularly with spicy foods
- Excessive or decreased salivation, defective taste
- **Early stage:** "wet leathery" feeling on palpation
- **Advanced stage:** Blanched, stiff mucosa, trismus, leathery lips, difficulty in lip eversion (microchelia)
- Opaque, marble-like mucosa
- **Difficulty in swallowing** and referred pain in ear
- Circular fibrous bands around the mouth
- **Vertical fibrous beds** on palpation
- Shrunken uvula, "hockeystick appearance"
- **Fibrotic gingiva** with deep pigmentation

Histopathology:

- **Hyperkeratinized, atrophic epithelium**
- Flattening and shortening of rete pegs
- Variable degrees of epithelial dysplasia (nuclear pleomorphism, severe intercellular edema)
- Dilated, congested blood vessels, possible hemorrhage
- **Homogenization and hyalinization** of collagen fibers
- Decreased fibroblasts, narrowed blood vessels due to perivascular fibrosis

Treatment:

- **Cease all causative habits**
- Intralesional injections: **collagenase, corticosteroids, fibrinolysin**
- **Systemic steroids** in severe cases

D). Oral Lichen Planus



Aetiology:

- **Immune-related disorder.**
- **Exacerbations or remissions** often linked to **emotional trauma** (e.g., stress from personal loss, job issues, or exam pressures).

Clinical Features:

- **Common in middle-aged and elderly**, slight female predilection.
- **Areas affected:** buccal mucosa, vestibule, tongue, lips, floor of mouth, palate, gingiva.
- **Symptoms:** Burning sensation.
- **Lesion characteristics:** White and grey **velvety thread-like papules** in **linear, angular, or retiform arrangements**.
- **Wickham's striae:** Tiny white elevated dots at intersections of white lines.

Patterns:

1. Linear
2. Papular
3. Reticular
4. Annular (circular)
5. Vesicular or bullous
6. Erosive or atrophic
7. Hypertrophic

Histopathology:

- **Hyperorthokeratosis or parakeratosis.**
- Thickened granular cell layer.
- **Acanthosis** of spinous layer, **saw-tooth appearance** of rete pegs.
- **Necrosis** or **liquefaction degeneration** of basal cell layer.
- **Band-like subepithelial infiltrate** (T cells, histiocytes).
- **Degenerating basal keratinocytes:** rounded or ovoid eosinophilic bodies (Civatte bodies).
- **Max-Joseph spaces:** Histological clefts due to degeneration of basal keratinocytes.

Differential Diagnosis:

1. **Lichenoid reactions:** Medication-induced lesions similar to lichen planus (lichenoid mucositis/dermatitis).
2. **Leukoplakia:** More common in men, younger age, family history, lacks remission/recurrence history, usually involves commissures, surrounding mucosa normal, soreness.

3. **Mucous patches of secondary syphilis:** Ulceration in center, affects commissures and tonsils, glandular enlargement, Treponema pallidum present.
4. **Candidiasis:** White patches scraped off, spores and mycelia visible microscopically.
5. **Recurrent aphthae:** Associated with trauma.
6. **Pemphigus:** Bullous lesions on normal mucosa, presence of acantholytic cells.
7. **Lupus erythematosus:** Atrophy and scarring stationary over months/years, firm on palpation.
8. **Erythema multiforme:** Acute, severe labial mucosa involvement, biopsy needed.

Q8. Name some giant cell lesions of oral cavity. Describe in detail Central and peripheral giant cell granuloma.

Giant Cell Lesions of the Oral Cavity

Neoplasms:

- **Giant Cell Tumor of Bone**
- **Central Giant Cell Granuloma**
- **Peripheral Giant Cell Granuloma**
- **Giant Cell Epulis**
- **Giant Cell Tumor of Hyperthyroidism**
- **Giant Cell Fibroma**
- **Malignant Fibrous Histiocytoma**

Other Lesions:

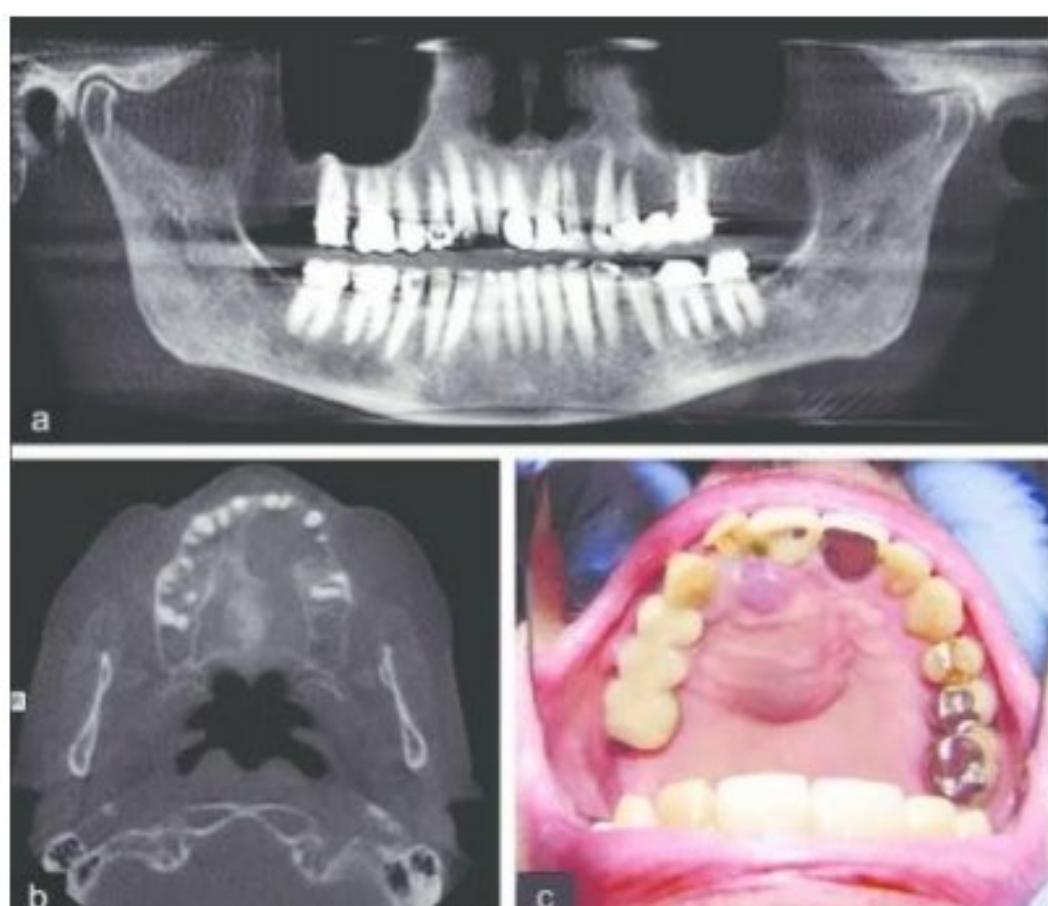
- **Osteoblastoma**
- **Chondroblastoma**
- **Fibrous Dysplasia of Bone**
- **Hodgkin's Disease**
- **Giant Cell Arteritis**
- **CEOT (Calcifying Epithelial Odontogenic Tumor)**
- **Sarcoidosis**

Giant Cell Granulomas

Types:

1. **Central Giant Cell Granuloma**
2. **Peripheral Giant Cell Granuloma**

Central Giant Cell Granuloma:



- **Common** benign intraosseous lesion affecting the **anterior jaw bone**.
- More aggressive, often **mandible** (anterior to first molar).

Clinical Features:

- Affects **young children**, **female predominance**.
- **Slow-growing**, bony hard swelling, **painful** on palpation.
- Causes **expansion** and distortion of cortical plates.
- May lead to **displacement** or mobility of teeth.
- In aggressive cases, becomes **large, painful** swelling.

Histopathology:

- **Fibrovascular connective tissue** with numerous **plump** and **spindle-shaped** stromal cells.
- **Multinucleated giant cells** (5-20 nuclei) around **blood capillaries** or areas of **haemorrhage**.
- **Osteoid foci** near the periphery.

Radiological Features:

- **Multilocular radiolucent area** with a **soap-bubble appearance**.
- **Scalloped**, well-demarcated margin.
- **Root resorption** or **divergence** of roots.

Treatment:

- **Surgical excision** with **curettage**.
- **Aggressive removal** if large section of bone involved.

Peripheral Giant Cell Granuloma:



- **Most common** giant cell lesion, arises from **tooth-bearing areas** of jaw.

- **Purplish-red nodule**, more common in **males**.

Clinical Features:

- Occurs during **mixed dentition** or **third-fourth decade** of life.
- **Hypercellular stroma** with spindle-shaped **fibroblasts**.
- **Large giant cells** with more nuclei than true giant cell tumors.
- **Haemorrhage** and **haemosiderin pigment** in connective tissue.

Histopathology:

- **Ulcerated epithelium** with areas of **haemorrhage**.
- **Fibroblasts**, **blood capillaries**, and **multinucleated giant cells** in the stroma.
- **Chronic inflammatory cell infiltration**.

Treatment:

- **Surgical excision**.

Q9. Describe dysplasia.



- **Dysplasia**: Introduced by **Reagon** in **1958** for cells from **uterine cervix lesions**.
- **Greek origin**: Dys (abnormal) + Plasia (growth) = **Abnormal tissue growth**.
- Associated with **premalignancy** and **abnormal cellular proliferation**.
- Often a precursor to **cancer**, but exact cause is unclear.

Features of Dysplasia:

- Occurs mainly in **epithelia**.
- **Loss of uniformity** in cells and **disordered tissue structure**.
- Cells show **pleomorphism** (varied size and shape).
- **Hyperchromatic nuclei** (deeply stained and larger) with increased **nuclear-cytoplasmic ratio** (1:4 to 1:1).
- Increased **mitotic figures**, often in abnormal locations.
- Normal **epithelial organization** is replaced by **disordered cells**.

Link to Chronic Irritation:

- Common in areas with **chronic irritation or inflammation** like the **oral cavity**.
- Oral cavity faces constant exposure to **microorganisms**, **toxins**, and **physical trauma**.

Reversibility:

- Dysplasia is **reversible** if the **irritating stimulus** is removed.
- If not reversed, it can progress to **neoplasia** (cancer).

- The line between **reversible dysplasia** and **irreversible cancer** is unclear.

Progression to Cancer:

- Cells respond to irritants with **hyperplasia** (increased proliferation) or **atrophy**.
- Persistent damage leads to **irreversible changes**, resulting in **cell death** or **neoplastic transformation**.
- Dysplastic cells may eventually **escape normal controls** and **accumulate genetic damage**, becoming **tumor cells**.

Key Points:

- Overlap exists between **cell adaptation**, **reversible damage**, and **atypia**.
- The exact **line of demarcation** between **reversible damage** and **neoplasia** is **unknown**.

Q10. Briefly explain changes in epithelial dysplasia.



Epithelial Dysplasia means disordered cell development. It shows **cell proliferation** and **cytological changes** (changes in cell structure).

Histopathological features:

- Loss of polarity** of basal cells
- Increased nuclear-cytoplasmic ratio** (more nucleus compared to cytoplasm)
- Irregular epithelial stratification** (layers of cells not organized properly)
- Cellular pleomorphism** (cells have different shapes and sizes)
- Nuclear hyperchromatism** (nucleus appears darker due to more DNA)
- Reduction of cellular cohesion** (cells stick less to each other)
- Enlarged nuclei and cells**
- Enlarged and prominent nucleoli** (center of nucleus is bigger and visible)
- Increased number of mitotic figures** (more cells dividing)
- Dyskeratosis** (abnormal keratin production)
- Basilar hyperplasia** (increased number of basal cells)
- Tear drop rete pegs** (the bottom layer of cells looks stretched and distorted)
- Poikilocaryosis** (nucleus varies in size and shape)

Q11. TNM classification

TNM Classification of Cancer (Lip and Oral Cavity)

T - Primary Tumour:

Tx: Primary tumour **cannot be assessed**

T0: No evidence of primary tumour

Tis: Carcinoma in situ

T1: Tumour **2 cm or less** in greatest dimension

T2: Tumour **> 2 cm but ≤ 4 cm** in greatest dimension

T3: Tumour **> 4 cm** in greatest dimension

T4:

- **Lip:** Tumour **invades** nearby structures (e.g. **bone, tongue, neck skin**)
- **Oral Cavity:** Tumour **invades** nearby structures (e.g. **bone, extrinsic muscle of tongue, maxillary sinus, skin**)

N - Regional Lymph Nodes:

Nx: Regional lymph nodes **cannot be assessed**

N0: **No metastasis** in regional lymph nodes

N1: Metastasis in a **single ipsilateral node** $\leq 3 \text{ cm}$

N2:

- **N2a:** Single ipsilateral node $> 3 \text{ cm}$ but $\leq 6 \text{ cm}$
 - **N2b:** Multiple ipsilateral nodes, all $\leq 6 \text{ cm}$
 - **N2c:** Metastasis in **bilateral or contralateral nodes**, none $> 6 \text{ cm}$
- N3:** Metastasis in a node $> 6 \text{ cm}$

M - Distant Metastasis:

Mx: **Cannot assess** distant metastasis

M0: **No distant metastasis**

M1: **Distant metastasis present**

Q12. Burkitt's lymphoma



Burkitt's lymphoma (also called African jaw lymphoma) was first described by **Dennis Burkitt in 1958**.

Aetiology:

- **EBV virus** is commonly associated.

Clinical Features:

- In **Africa**, Burkitt's lymphoma accounts for **50% of childhood malignancies**; in **USA and Europe**, it accounts for **6-10%**.
- **Endemic form** occurs between **3-8 years**, with a **2:1 male predominance**.
- **Sporadic form** affects older children (mean age ~ 11 years) and has **no gender preference**.

- Endemic form commonly affects **mandible, maxilla, abdomen**, and extranodal sites like **retroperitoneum, kidneys, liver, ovaries, endocrine glands**.
- **Maxillary molar area** is often involved.
- It is a **rapidly growing tumour mass** of the jaws, destroying bone and extending into the **maxillary, ethmoid, and sphenoid sinuses**, as well as the **orbit**.

Oral Manifestations:

- **Mobility and loss of teeth**, along with **asymmetrical facial swelling**.
- **Gingiva and mucosa** are swollen, **ulcerated**, and **necrotic**.
- **Deranged occlusion and dental arch**.

Histopathology:

- **B-cell neoplasm** with **B-lineage antigens** and **monoclonal surface immunoglobulins**.
- **Starry sky appearance** due to macrophages with nuclear debris.
- Differential diagnosis includes **other Non-Hodgkin's lymphomas**, **undifferentiated carcinoma**, **sarcoma**, **metastatic neuroblastoma**, and **acute leukemia**.

Management:

- Once fatal within **4-6 months**, but now sensitive to **combination chemotherapy**.
- **2-year survival rate** is about **55%** overall, with **80% for low-stage** and **40% for advanced-stage** disease.

Q13. Kaposi sarcoma.

- **Kaposi Sarcoma (KS)**: A multicentric growth of **vascular and spindle cells**.
- First described by **Moritz Kaposi** in 1872 as "**idiopathic multiple pigmented sarcoma of the skin**".
- Now linked to a **viral cause**, especially associated with **HIV/AIDS**, but HIV itself is not the direct cause.
- Four types of KS:
 1. **Classic (Chronic)**:
 - Affects older individuals.



- **Blue-red skin nodules** on lower legs, grow slowly.

- **Oral involvement** is rare, but may show **bluish nodules on the palate or gingiva**.

2. Endemic (Lymphadenopathic, African):

- Affects **young African children**.
- Enlarged **lymph nodes** with few or no skin/mucous membrane lesions.
- Can involve **salivary glands**.

3. Immunosuppression-Associated (Transplant):

- Seen in **1-4% of renal transplant patients**.
- Develops **1-2 years post-transplant**, correlates with weakened **immune system**.
- Involves **skin and internal organs**, rare in the **oral cavity**.

4. AIDS-Related:

- Affects about **40% of AIDS patients**, mainly **young males** (~39 years old).
- Skin lesions appear in multiple locations, including the **tip of the nose**.
- **Oral lesions** occur mostly on the **palate** and **gingiva**, starting as **flat blue/red/purple plaques**.
- Can become **exophytic, ulcerated**, may bleed, and rarely exceed **2 cm** in size.
- May cause **tooth loss** and **airway obstruction**.
- Other oral sites: **gingiva, tongue, uvula, tonsils, pharynx, trachea**.

Histologic Features:

Early lesion (Patch stage):

- Proliferation of **small veins** and **capillaries** around **dilated vessels**.
- **Slit-like vessels** seen around **blood vessels**, **skin adnexa**, and **between collagen fibers**.
- Lined by **plump, mildly atypical endothelial cells**.
- Resembles **granulation tissue**.
- **Mononuclear inflammatory cell infiltrate**, **mast cells**, **scattered erythrocytes**, and **hemosiderin deposits** may be present.
- **Spindle cell proliferation** is minimal with **minimal cellular atypia**.

Plaque stage (advanced lesion):

- **Nodular** with more **small capillaries** or **dilated vascular channels**.
- **Spindle cells** increase, often with **extravasated erythrocytes** and **hemosiderin deposition**.
- **Slit-like vascular channels** without visible **endothelial lining**.
- **Enlarged, hyperchromatic nuclei** with **mild-moderate pleomorphism**.
- **Mitotic activity** varies, usually minimal.
- **Chronic inflammatory cells** vary.
- In **nodular stage**, all features are more prominent.

Immunoreactivity:

- **Spindle cells**: reactive for **CD34**.

- **Endothelial cells:** reactive for **CD31** and **CD34**.
- **Vascular channels:** reactive for ***U. europaeus agglutinin***, but non-reactive for **factor XIIIa**.

Treatment:

- **Small/localized lesions:** **Surgical excision** with a small margin of normal tissue.
- Recent therapies focus on **low-dose irradiation, intralesional chemotherapy, and sclerosing solutions**.
- For **larger/multifocal lesions:** **Systemic chemotherapy** effective.

Q14. Hodgkin and non hodgkin lymphoma.

Hodgkin Lymphoma :



- **Primarily affects lymph nodes**, especially the **cervical lymph nodes**.
- Disease of the **lymphatic system**, part of the **immune system**.

Age Distribution:

- **Bimodal age distribution:**
 - Common in **young adults** (20s).
 - Second peak in the **5th decade of life** (around 50 years).

Histological Features:

- Presence of **Reed-Sternberg cells (RS cells)**:
 - **Large, multinucleated** cells.
 - Of **lymphocytic origin** (B lymphocytes).
 - Crucial for diagnosis.

Types of Hodgkin Lymphoma:

1. **Lymphocytic Predominant:**
 - Many **lymphocytes**; few RS cells.
 - **Good prognosis.**
2. **Nodular Sclerosis:**
 - Most common type.
 - **Fibrous bands** in lymph nodes.
 - Often involves **cervical** and **mediastinal lymph nodes**.
3. **Mixed Cellularity:**
 - Mixture of cells: **lymphocytes, eosinophils, plasma cells**, and RS cells.

- More common in **older adults**.
 - Associated with **Epstein-Barr virus (EBV)**.
4. **Lymphocytic Depletion:**
- **Fewer lymphocytes**, more RS cells.
 - More aggressive, seen in **older individuals** or those with **HIV**.

Diagnosis:

- **Biopsy** is the primary diagnostic tool.
- Historically linked to **Gordon's biologic test**.

Treatment:

- **Radiation therapy** and **combination chemotherapy** are effective.
- Common chemotherapy regimen: **ABVD (Adriamycin, Bleomycin, Vinblastine, Dacarbazine)**.
- Early stages: **Radiotherapy** may suffice.
- Advanced stages: **Chemotherapy** is essential.

Prognosis:

- Treatment advances have significantly improved outcomes.
- Many patients achieve **long-term remission**.

Non-Hodgkin Lymphoma (NHL) :



- **Group of cancers** affecting the **lymphatic system**.
- More **common** than Hodgkin lymphoma.
- **Affects lymphocytes** (B cells, T cells, or NK cells).
- Can arise in **lymph nodes, spleen, bone marrow, or other organs**.

Key Differences from Hodgkin Lymphoma:

- **No Reed-Sternberg cells**.
- Can occur at **any age**.
- Typically, more **widespread** at diagnosis.

Classification:

- **B-cell lymphomas**: Most common type (85% of NHL).

- Examples: **Diffuse large B-cell lymphoma (DLBCL)**, **Follicular lymphoma**.
- **T-cell lymphomas**: Less common.
 - Examples: **Peripheral T-cell lymphoma**, **Cutaneous T-cell lymphoma**.

Types of Non-Hodgkin Lymphoma:

1. **Diffuse Large B-cell Lymphoma (DLBCL)**:
 - **Most common** aggressive NHL.
 - Rapidly growing but responds well to treatment.
2. **Follicular Lymphoma**:
 - **Slow-growing** (indolent).
 - Can transform into more aggressive forms.
3. **Mantle Cell Lymphoma**:
 - **Rare**, aggressive, often diagnosed late.
4. **Burkitt Lymphoma**:
 - Highly **aggressive**.
 - Associated with **Epstein-Barr virus** and common in **children**.
5. **Peripheral T-cell Lymphoma**:
 - **Aggressive**, less common, poor prognosis.
6. **Cutaneous T-cell Lymphoma**:
 - Primarily affects the **skin**.

Risk Factors:

- **Age**: Most common in people over **60**.
- **Weakened immune system**: HIV/AIDS, organ transplants.
- **Infections**: Certain infections like **EBV**, **H. pylori**, and **HTLV-1** increase risk.
- **Exposure to chemicals**: Pesticides, herbicides.

Symptoms:

- **Swollen lymph nodes** (painless).
- **Fever, night sweats, weight loss** (B symptoms).
- **Fatigue**.
- **Abdominal pain** or swelling.
- **Chest pain or difficulty breathing** (if chest lymph nodes are involved).

Diagnosis:

- **Biopsy** of affected lymph nodes or tissues.
- **Immunophenotyping** and **flow cytometry** to classify the type of NHL.
- **Imaging tests** (CT scan, PET scan) to determine extent.

Treatment:

- Depends on the **type** and **stage** of lymphoma.
- **Indolent (slow-growing)** forms may require a **watch-and-wait** approach.
- **Aggressive forms** need immediate treatment, including:
 1. **Chemotherapy** (e.g., **CHOP regimen**: Cyclophosphamide, Doxorubicin, Vincristine, Prednisone).
 2. **Immunotherapy** (e.g., **Rituximab** for B-cell NHL).
 3. **Radiation therapy** for localized disease.

4. **Stem cell transplant** for relapsed or aggressive cases.

Prognosis:

- Varies based on type, stage, and patient factors.
- **Indolent NHL:** Often has a **long survival**, but can recur.
- **Aggressive NHL:** Can be cured in many cases with prompt treatment.

Survival Rates:

- **5-year survival** rates depend on the type and stage:
 - **Indolent forms:** 80-90%.
 - **Aggressive forms:** 60-70% (with effective treatment).

Q15. Rodent Ulcer (Basal Cell Carcinoma)



Etiology:

- Caused by prolonged exposure to **sunlight**, specifically **ultraviolet (UV) radiation**.

Site of Occurrence:

- Commonly appears on **exposed surfaces** of the **head and neck**.
- Most frequently found on the **face**, particularly above a line drawn from the **tragus of the ear** to the **angle of the mouth**.
- When located here, it is termed a **rodent ulcer**.
- **Rare** occurrence in the **oral cavity**.

Clinical Features:

- Primarily affects **middle-aged** or **elderly individuals**, more common in **males**.
- The typical presentation is **noduloulcerative**:
 - Begins as a small, **hard lump** or **papule** on the skin.
 - Slowly **increases in size**.
 - Eventually **breaks down**, forming an **ulcer** with a characteristic **rolled edge**.
- **Slow-growing**, but can cause significant **local tissue destruction**, affecting a large area over time.

Treatment:

- The primary treatment methods are **surgical excision** and **radiotherapy**, depending on the size and location of the lesion.