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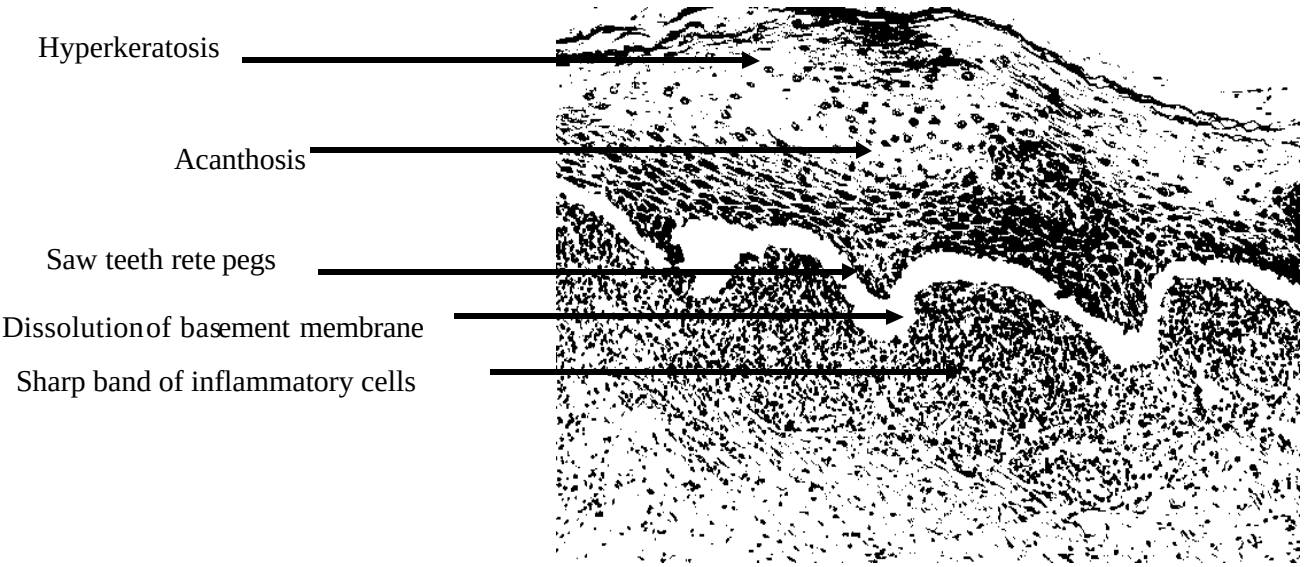
ORAL PATHOLOGY (SEQs)
MODEL PAPER

Q 1. Draw and label a diagram to illustrate the histopathology of a typical Lichen Planus.

Key:

Diagram is best to be drawn by H & E pencils, however if not possible the drawing may be accepted by a lead pencil but must be labeled



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Q 2. Describe the different clinical forms of Lichen Planus.

Key:

The different type of lichen planus on the oral mucous membrane include the reticular, popular, plaque or hypertrophic, bullous (erosive /ulcerative) and atrophic type.

The reticular type occurs most frequently and is easily recognizable. It is composed of narrow slightly raised whitish or grayish line (wickham striae) which criss-cross each other at various angulations forming a mesh or net like pattern. At times, the coalescing of line may result in circular or annular ring like appearance. The points of criss-crossing may show a point like elevation.

The popular pattern consists of small pin-head sized, dome shaped, raised glistening white spots of variable density exhibiting fine white streaks around them

The plaque type or hypertrophic type shows a solid grayish white raised patch resembling leukoplakia.

The bullous or vesicular type shows fluid filled fluctuant blisters of vary size. These may be clear, seropurulent or hemorrhagic. These lesions rupture early, leaving behind raw eroded surfaces resembling ulcerative lichen planus

.

The erosive or ulcerative type like the bulbous type is uncommon. The erosive type of lichen planus holds a premalignant potential. The lesion is flat or slightly depressed and raw red. They are surrounded by fine white lines of wickham. Such lesions on the gingiva
resemble desquamative gingivitis.

The atrophic type leaves the mucous membrane smooth and glistening. The lesions commonly occur on the dorsum of the tongue resembling syphilitic glossitis.

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Q 3. Differentiate between benign and malignant oral tumors clinically and histologically in tabulated form

Key:

(a) CLINICALLY

Benign	Malignant
1. Grow slowly	1. Grow rapidly
2. Expansive growth	2. Invasive growth
3. Usually encapsulated	3. Not encapsulated
4. Less recur	4. Do recur after removal
5. Do not metastasize	5. Do metastasize
6. Do not kill unless they compress vital organs	6. Generally fatal
7. Rarely show necrosis and ulceration	7. always show necrosis and ulceration
8. No cachexia	8. Cachexia and anemia

(b) HISTOLOGICALLY

Benign	Malignant
1. Consist of well differentiated cells	1. Consist of poorly differentiated, anaplastic cells
2. Cells are rather uniform in size and shape	2. Pleomorphism of cells
3. Nuclei take up stain normally	3. Hyperchromatic nuclei
4. Few mitoses	4. Numerous multipolar mitosis
5. Fairly good imitation of the arrangement of these tissues from which they are derived	5. Unsuccessful imitation of the tissue of origin
6. Cells do not infiltrate	6. Cells do infiltrate

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Q 4. **Classify Odontogenic tumors on the basis of radiography and their source of origin.**

Key:

Classification of Odontogenic tumors

Radiopaque

EPITHELIAL

Enameloma

MESENCHYMAL

Cementoblastoma

MIXED

Odontoma

Radiolucent

EPITHELIAL

Ameloblastoma

Malignant Ameloblastoma

Ameloblastic Carcinoma

Squamous Odontogenic Tumor

Clear cell Odontogenic Tumor

Primary Intraosseous Carcinoma

Primary Odon. Carcinoma

Calcifying Odontogenic Cyst.

MESENCHYMAL

Central Odon..Fibroma

Peripheral Odon.Fibroma

Odontogenic Myxoma

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MIXED

Ameloblastic Fibroma

Ameloblastic Fibrosarcoma

Odontogenic Fibrosarcoma

Mixed

EPITHELIAL

Adenomatoid Odontogenic Tumor

Calcifying Epithelial Odontogenic Tumor

MESENCHYMAL

Pericemental Dysplasia

Peripheral Odontogenic..Fibroma

Cementifying Fibroma

MIXED

Ameloblastic Fibrous odontoma

Ameloblastic Odonto sarcoma

Ameloblastic odontoma

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Q 5. (a) Differentiate (1) central giant cell Granuloma, (2) aneurysmal bone cyst, and (3) Brown tumor of hyperthyroidism, involving jaw.

(b) Describe briefly the microscopic appearance of caries in the dentin.

Key:

(a)

All these conditions present as radiolucent lesions and have similar histological features of large number of multinucleated giant cells (foreign body type) in a highly cellular and vascular stroma. The aneurysmal bone cyst in an uncommon lesion that has, in addition to the above features, sinusoidal blood spaces and osteoid formation. The central giant cell Granuloma is the intraosseous counterpart of the peripheral giant cell Granuloma (giant cell epulis) and historically identical to the giant cell lesion of hyperparathyroidism. Hyperparathyroidism presents additional features of hypercalcemia, hyperphosphaturia, loss of lamina dura (occasionally), and parathyroid adenoma or hyperplasia.

(b)

The study of a section of dentin reveals that a least five distinct zones can be observed in the dentinal tubules of carious dentin. They are as follows:

1: Zone of comple te decalcification of the dentin matrix with decomposition of the dentin matrix with decomposition of the organic matrix.

2: Zone of incipient of decalcification of dentin, the result of the action of microorganisms that have invaded the tubules.

3: Sclerotic zone, in which the tubules are obliterated by calcification of the odontoblastic processes.

4: Zone of fatty degeneration and the beginning of precipitation of calcium droplets in the protoplasm of odontoblastic processes.

5: Zone of normal dentin, with no disturbances in the dental tubules.

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Q 6. (a) Define (1) glossoplegia, (2) macroglossia, (3) ankyloglossia.

(b) Describe the features of Dentinogenesis Imperfecta

Key:

(a)

1: Glossoplegia is paralysis of the tongue

2: Macroglossia is enlargement of the tongue

3: Ankyloglossia is tongue-tie, usually caused by too short a frenum or by mucous membrane adhering too closely to the tip of the tongue

(b)

Dentinogenesis Imperfecta (hereditary opalescent dentin) is a hereditary condition characterized by translucent to opalescent teeth that exhibit marked attrition with chipping of the enamel. Root fractures are seen with considerable frequency. The primary defect is in dentin, which is poorly mineralized. The roots of the teeth are short and the pulp chambers are obliterated. The dental changes are identical to those seen in Osteogenesis Imperfecta.

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Q 7. (a) What are the causes of enamel hypoplasia?

(b) What are the causes of hypercementosis?

Key: (a)

It is caused by anything that will interfere with the formation of the enamel of the tooth at the time the tooth is in the progress of active formation and calcification. It occurs in febrile condition, syphilis, nutritional disturbances and fluorosis. The defects are produced by a disturbance in the ameloblastic activity of the enamel organ or by the collapse of normally formed but poorly calcified enamel matrix.

(b)

Primary: Hyperemia or inflammation stimulating cell activity.

Secondary: Movement with disuse, abrasion, erosion, malocclusion, overuse, bruxism and root fillings; also a feature in Peget's disease of bone.

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Q 8: (a) Give the cause of resorption of teeth.

(b)List the conditions that may follow pulp infection and periapical involvement

Key:

(a)

1: External resorption

a. Periapical inflammation
b. Excessive mechanical or occlusal forces
c. Cyst and tumors
d. Reimplantation of teeth
e. Impacted teeth
f. Idiopathic

2: Internal resorption

Idiopathic

(b)

1: Periapical Granuloma

2: Apical periodontal cyst

3: Periapical abscess

4: Osteomyelitis

a. Acute suppurative osteomyelitis
b. Chronic suppurative osteomyelitis
c. Chronical focal sclerosing osteomyelitis (condensing osteitis)
d. Chronical diffuse sclerosing osteomyelitis

5: Periostitis

a. Proliferative

6: Cellulitis

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Q 9. (a) Discuss briefly those local factors that may predispose to dental caries.
(b) What is the general shape of the carious lesion in pits and fissures and on smooth surfaces of the teeth?

Key:

(a) It is generally agreed that the presence of Acidogenic microorganisms and a suitable carbohydrate substrate in a dental plaque with increased time duration are essential for development of the carious lesion. Additional contributing factors include poorly formed teeth and absence of antibacterial factors in the saliva; and a soft or highly refined carbohydrate die..

(b) Pit and fissure caries appears as a conical lesion, with the base of the cone at the dentinoenamel junction. In smooth-surface caries the lesion is also conical, with the base of the cone at the enamel surface. In caries of the dentin the base of the cone is at the dentinoenamel junction.

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Q 10: (a) Give the clinical features of herpetic gingivostomatitis

(b) Describe the oral lesions of measles.

Key:

(a)

1. Transmitted by close contact
2. 90% people develop antibodies in early childhood
3. Vesicles (2 - 3 mm diameter) followed by circular, sharply define, shallow ulcers, (with yellowish or grayish floors and red margins) affect any part of the oral mucosa
4. Gingivitis with red and swollen margins
5. Lymphadenopathy and fever of variable severity
6. Lesion resolve within a week to 10 days

(b) In measles small white patches occur on the buccal mucous membrane, usually in or near the angles of the mouth. These patches are pathognomonic of the disease and precede the eruption on the skin. They appear as little red spots, on the center of which is a bluish white speck. These are referred to as Koplik's spots. The tongue usually is furred. Bleeding and hypertrophy of the gingivae occur frequently.

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Q 11. (a) Discuss agranulocytosis and its oral manifestation.

(b) Describe the oral symptoms of pemphigus.

Key:

(a) Agranulocytosis is a serious blood disorder in which there is marked reduction in the number of circulating leukocytes, specifically the granulocytes. The etiology of primary agranulocytosis is unknown. Secondary agranulocytosis generally develops as an idiosyncrasy to a variety a variety of drugs; e.g., amidopyrine Phenobarbital, benzene, chloramphenicol, and sulfanilamide. The chief clinical oral features are necrotic ulcers of the gingiva and palate. Histologically the tissues exhibit ulceration, and bacterial invasion, with little evidence of inflammation and repair. No polymorph nuclear neutrophils are found in the tissues.

(b) The oral lesions of pemphigus are similar to the skin lesions and often precede them. Blebs appear that soon rupture and leave a grayish crust that bleeds easily. The saliva is copious and colored with blood. The breath is offensive, and mastication is painful. As the lesions heal, new ones form; the disease progresses by the formation of new lesions, finally in some cases progresses by the formation of new lesions, finally in some cases covering much of the mucosa and skin.

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Q 12. Give briefly the histopathology of the peripheral giant cell Granuloma and hemangioma

Key:

Histopathology Of The Peripheral Giant Cell Granuloma

This tumor usually has its origin in the alveolar dental periosteum between or near the teeth. It is composed of a highly vascular connective tissue stroma fairly rich in cells, appearing as spindle and round cells. There is considerable hemosiderin present. Varying numbers of multinuclear cells are seen throughout the stroma. These giant cells contain from 8 to 16 nuclei. A small amount of bone is sometimes found in the tumor. The epithelium covering the mass may be ulcerated.

Histopathology Of The Hemangioma

1. Proliferation of blood vessels
2. Capillary hemangioma = Multiple, small capillary channels
3. Cavernous hemangioma = Large tortuous dilated vascular spaces densely packed with erythrocytes

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Q13. Give the detail of Loss of tooth structure by chemical reaction, not that associated with bacteria (caries)

Key:

Erosion

- Loss of tooth structure by chemical reaction, not that associated with bacteria (caries)
- Secondary to presence of acid or chelating agent
- Source can be dietary (e.g., vinegar, lemons), internal (gastric secretions – perimolysis), or external (e.g., acids, industrial, atmosphere)
- "If it is not abrasion or attrition, it must be erosion"
- Commonly affects facial surface of maxillary anteriors and appears as shallow spoon-shaped depressions in cervical portion of the crown
- Posterior teeth exhibit loss of occlusal surface, where dentin is destroyed more rapidly than enamel, resulting in concave depression of dentin surrounded by elevated rim of enamel
- Erosion limited to facial surfaces of maxillary anterior dentition is usually associated with dietary acid.

<u>Classification</u>

ACID SOURCE – "EXTRINSIC" (EXTERNAL)

- Food and drinks
- Medicine
- Swimming pools
- Industrial (acidic- vapour, dust)

ACID SOURCE– "INTRINSIC" (INTERNAL)

- Eating disorders
- Gastrointestinal disorders
- Alcoholism

Erosion: Consequences

- Sensitivity
- Weakening of the teeth
- Reduced lifetime for some restorations (GIC and ceramic)

Erosion: Treatment

Dietary analysis – change in unfavorable dietary habits

- Psychological/medical factors
- Fluoride - daily
- Consider restoring the most serious erosion lesions

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Q14. What you understand by Sjögren's Syndrome ,give its important clinical features and treatment

Key:

- Most common immunologic disorder **associated with salivary gland disease.**
- Characterized by a lymphocyte-mediated destruction of the exocrine glands leading to xerostomia and keratoconjunctivitis sicca
- 90% cases occur in women
- Average age of onset is 50y
- Unilateral or bilateral salivary gland swelling occurs, may be permanent or intermittent.
- Rule out lymphoma
- Keratoconjuntivitis sicca: diminished tear production caused by lymphocytic cell replacement of the lacrimal gland parenchyma.
- Evaluate with Schirmer test. Two 5 x 35mm strips of red litmus paper placed in inferior fornix, left for 5 minutes. A positive finiding is lacrimation of 5mm or less Approximately 85% specific & sensitive
- Biopsy of SG mainly used to aid in the diagnosis
- Can also be helpful to confirm sarcoidosis

Sjögren's Treatment

- Avoid xerostomic meds if possible
- Avoid alcohol, tobacco (accentuates xerostomia)
- Sialogogue (eg:pilocarpine) use is limited by other cholinergic effects like bradycardia & lacrimation
- Sugar free gum or diabetic confectionary
- Salivary substitutes/sprays

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Q 15. What is the difference in the Immunofluorescence finding of Erosive Lichen Planus , Cicatricial Pemphigoid , Pemphigus vulgaris and Erythema multiforme

Key:

- Erosive Lichen Planus – Immunofluorescence targeted at fibrinogen.
- Cicatricial Pemphigoid -Linear deposition of immunoreactants at the BMZ, typically IgG and C3
- Pemphigus vulgaris – Immunofluorescence targeted at IgG: fish pattern appearance.
- Erythema multiforme – Immunofluorescence targeted at IgM and C3: perivascular appearance.

FOLLOWING BOOKS CONSULTED WHILE PREPARING THE KEY

Contemporary Oral & Maxillofacial Pathology.

By J Philip Sapp,Lewis R.Eversole,George P.Wysocki

Publisher = Mosby

Oral Pathology Clinical Pathological Correlations

By Joseph A.Regezi, James J.Sciubba

Publisher = W.B Saunders Company

Cawson`s Essential of Oral pathology and Oral Medicine.

By R.A. Cawson, E.W. Odell

Publisher = Churchill Livingstone