AL BADAR DENTAL COLLEGE AND HOSPITAL DEPARTMENT OF PERIODONTOLOGY.

Form description

NAME *

Short answer text

REGISTER NUMBER *

Short answer text

1. What is the distance between the CEJ to the marginal bone in the health

- 1- 3 mm
- 1.5-2 mm
- 1.8 mm
- 2- 4 mm

2. Which is the most commonly occuring leucocyte in the oral mucosa *

- Polymorphonuclesr leucocyte
- Macrophage
- Langerhans cell
- Merkels cell

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74 responses

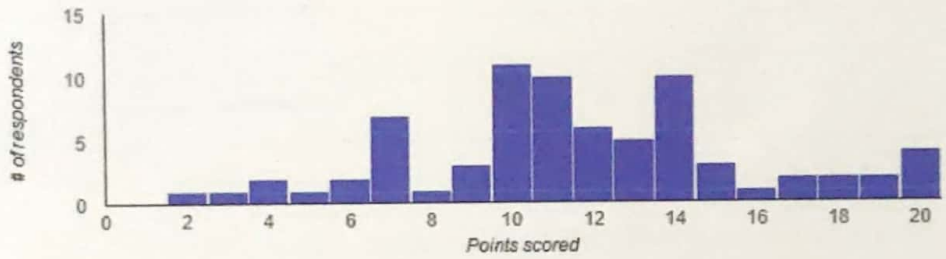
Accepting responses

Summary | Question | Individual

Insights

Average	Median	Range
11.62 / 20 points	11 / 20 points	2 - 20 points

Total points distribution



Frequently missed questions

Question	Correct responses
1. What is the distance between the CEJ to the marginal bone in the health	28 / 73
12.Gingival massage increases the blood supply	12 / 72
14.SANGUINARINE is	22 / 73
15.For Periodontal patients most recommended tooth brushing technique is	25 / 73
17. Ruffini like endings Meissners corpuscles are related to	34 / 73

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1 of 74

20 of 20 points | Score not released | Release score

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* Required

NAME * / 0

Add individual feedback

REGISTER NUMBER * / 0

Add individual feedback

✓ 1. What is the distance between the CEJ to the marginal bone in the health 1 / 1

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✓ 2. Which is the most commonly occuring leucocyte in the oral mucosa * 1 / 1

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Feedback

POLYMORPHONUCLEAR LEUCOCYTE

Add individual feedback

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DEPARTMENT OF ORAL MEDICINE AND RADIOLOGY
FIRST INTERNAL ASSESMENT-FINAL YEARS B.D.S
DATE:15.09.2021
MAX MARKS 70

Long essays :10×2=20 marks

1.Classify vesiculo-bullous lesions. Discuss pathogenesis/F,OM,D/D,&Management of phemphigus vulgaries

Ans: classification of vesiculo-bullous lesions.

I. Acute and chronic vesiculobullous lesions

	Acute	Chronic
Duration	Short	Long
Etiology	Allergy, burns, viruses	Autoimmune
Age	Young	Middle-Older
Examples	Herpes simplex infections Chicken pox Herpes zoster Herpangina	Pemphigus Bullous pemphigoid Cicatricial pemphigoid Chronic herpes simplex

II. Based on the clinical presentation

1. Predominantly vesicular HSV infection Varicella infection Hand, foot and mouth disease
   Herpangina Dermatitis herpetiformis
2. Predominantly bullous
   Pemphigus vulgaris
   Bullous pemphigoid
   Benign mucous membrane pemphigoid
   Bullous lichen planus
   Erythema multiforme
   Stevens–Johnson syndrome
   Bullous impetigo
   Epidermolysis bullosa
   Linear IgA disease

II. Histopathological classification

Intraepithelial vesiculobullous lesions

1. HSV infection
2. Varicella infection
3. Herpangina
4. Hand, foot and mouth disease
5. Pemphigus
6. Familial benign chronic pemphigus

Epidermolysis bullosa
Mucosal erythema multiforme
Subepithelial vesiculobullous lesions

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Bullous pemphigoid
Cicatricial pemphigoid
Epidermolysis Bullosa
Dermal Erythema multiforme
Dermatitis herpetiformis
Linear IgA disease

III. **Based on whether the lesions are infectious or non-infectious**

Infectious VB lesions
- Herpes simplex infections
- Varicella infections
- Herpangina
- Hand, foot and mouth disease

Non-infectious VB lesions
- Pemphigus
- Paraneoplastic pemphigus
- Bullous pemphigoid
- Cicatricial pemphigoid
- Erythema multiforme
- Dermatitis herpetiformis
- Epidermolysis bullosa acquisita
- Linear IgA disease

Pemphigus vulgaris

It is autoimmune mucocutaneous disorder affecting intraepithelial layer

Etiopathogenesis:

- Mechanism causing the characteristic intraepithelial lesion of pemphigus vulgaris is the binding of specific IgG antibodies to an antigen on the epithelial cell membrane.
- The stimulus that triggers the abnormal IgG production is unknown. Certain
- exogenous factors:
  medications,
  dietary components
  unknown environmental factors
- There is evidence that the binding of IgG antibody to the pemphigus antigen leads to epithelial cell separation by triggering either complement activity or the plasminogen.
- a new pemphigus antigen desmoglein 4 has been discovered and implicated in the pathogenesis of pemphigus vulgaris.
- almost all pemphigus vulgaris patients have either HLA-DR4 or DRW6 haptotypes. Also, the disease susceptibility has been linked to an HLA-DQB gene.

Clinical features

- age: 5th and 6th decade of life. Rarely affects younger individuals.
- gender: Men and women are equally affected.

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- It most commonly occurs in Jews, Greeks, east Indians and individuals from the orient.
- sites: Pemphigus vulgaris affects the mucosa and skin, resulting in superficial blisters and chronic ulceration.
- Various mucosal surfaces may be involved such as the oral, ocular, nasal, pharyngeal, laryngeal, upper respiratory and anogenital mucous membranes.
- The common sites of involvement are the groin, scalp, face, neck, axillae and genitals. nail folds may be involved first, together with the oral lesions.
- Dermal lesions are characterized by bullae over the skin.
- Fluid in the bullae appears clear at first but later it may become hemorrhagic or even seropurulent. Initially the bullae are tense, but soon become flaccid and rupture to form erosions which ooze and bleed easily. The denuded areas sometimes are partially covered with crusts with little or no tendency to heal and enlarge by confluence.
- The healed lesions usually leave hyperpigmented patches. However, in some instances these solitary erosive areas may coalesce and involve extensive areas of the skin.
- Nikolsky's sign is positive-if slight pressure or rubbing of the skin produces lateral movement of the upper layers of the epidermis.
- The Asboe-Hansen sign, or 'bulla spread phenomenon', is positive in pemphigus. Gentle pressure on an intact bulla will force the fluid to spread under the skin away from the site of pressure.

Oral manifestations:

- Oral lesions usually appear first in this disease. Almost 80–90% of the patients with pemphigus vulgaris develop oral lesions sometime during the course of the disease and in 60% of the cases oral lesions occur first.
- The typical oral lesion begins as a bulla on a noninflamed base, which almost immediately ruptures to produce shallow ulcer.
- The margins of the ulcer show evidence of tissue tags.
- sites: buccal mucosa, gingiva and palate
- The edges of the shallow ulcers extend peripherally over a period of weeks until they involve large portions of the oral mucosa.
- Distal extension from the oral cavity causes involvement of the oesophagus, pharynx and larynx, which causes hoarseness of voice and dysphagia.

Differential diagnosis:

- Epidermolysis bullosa
- Erythema multiforme
- Bullous pemphigoid
- Cicatricial pemphigoid

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- Bullous drug eruptions
- Other forms of pemphigus.

*The histological presence of supra basal intraepidermal bulla with acantholysis is characteristic of pemphigus and usually differentiates it from other similar diseases.

Management of phemphigus vulgaries :

Topical therapy

- Eroded and crusted, painful skin lesions and the associated foul odor can be effectively managed by bathing the area with 0.01% potassium permanganate solution or 0.5% silver nitrate solution.
- Alternatively, the raw surfaces can be sprayed with corticosteroids or 2% procaine hydrochloride.
- Chlorhexidine mouth rinses can be used to alleviate discomfort and malodour.
- Painful oral ulcerations can be managed by topical application of viscous xylocaine especially before food intake.

Systemic therapy

- Corticosteroids: Systemic administration of corticosteroids comprises three phases:
  - **Control phase**: Characterized by an initial high dose corticosteroid administration to the point of obvious clinical improvement.

Therapy is initiated by giving- 60–160 mg of prednisone daily.

If there is no response even after a week, the dosage is doubled. When new lesions cease to form and old lesions heal, the dosage is decreased slowly.

  - **Consolidation phase:** In this phase the dosage of prednisone is reduced over a period of several weeks. once the control over the disease is achieved, an attempt to decrease the steroid dose by transferring the patient to intramuscular injections of triamcinolone acetonide is highly advisable.

  - **Maintenance phase**: The corticosteroids are gradually tapered down to alternate day dose and ultimately stopped. However, this reduction in dosage is made possible by replacing steroids with immunosuppressive drugs.
- The dosage of immunosuppressive drugs is reduced to zero in several months.
- Immunosuppressive agents:
  Azathioprine 100–200 mg per day in conjunction with prednisone 150–200 mg daily can be used.
- Plasmapheresis: It is particularly useful in patients who are refractory to corticosteroids. It involves removal of the circulating antibodies.
- Photopheresis: This modality of treatment was described by Rook et al. It involves administration of 8-methoxypsoralen followed by exposure of peripheral blood to ultraviolet radiation, causing photoinactivation of WBC.
- Immunomodulators: levamisole (100 mg/week), combination of nicotinamide and tetracycline and oral prostaglandins are effective in the treatment of pemphigus.

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2.Classify red and white lesions of oral cavity. Discuss etiopathogenensis ,clinical features and management of OSMF.

Ans: classification:

ETIOLOGIC CLASSIFICATION OF RED AND WHITE LESIONS

1. Normal mucosal variations :Leukoedema Fordyce granule Linea alba buccalis
2. Genetically linked white keratotic lesions:
   - Oral genodermatoses
   - White sponge nevus
   - Hereditary benign intraepithelial dyskeratosis
   - Pachyonychia congenita
3. Post inflammatory white lesions :
   - Traumatic keratosis: Mechanical trauma Thermal burn Chemical burn (aspirin burn, uremic stomatitis) Radiation mucositis
   - Reactive mucosal hyperplasias (stomatitis nicotina palati)
4. White and red lesions due to infections: Syphilis Measles (Koplik's spots) Candidiasis Bacterial stomatitis.
5. Premalignant lesions
   - Leukoplakia
   - Lichen planus
   - Lichenoid reactions
   - –drug induced
   - graft-versushost disease
   - Erythroplakia
   - Actinic keratoses
   - Discoid lupus erythematosus
   - Chronic hyperplastic candidiasis
6. Premalignant conditions
   - Oral submucous fibrosis
   - Oral psoriasiform lesion
   - Dyskeratosis congenita
   - Syderopenic dysphagia
   - Syphilitic glossitis

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Oral submucous fibrosis (OSMF) is an insidious, chronic disease affecting any part of the oral cavity, and sometimes the pharynx. Occasionally it is preceded and/or associated with vesicle formation and always associated with a juxtaepithelial inflammatory reaction followed by progressive hyalinization of the lamina propria.

The later subepithelial and submucosal myofibrosis leads to the stiffness of the oral mucosa and deeper tissues with progressive limitation in opening of the mouth and protrusion of the tongue.

Etiopathogenesis:

- ARECA NUT:
- The chewing of betel nut has been recognized as one of the most important etiological factor for the Areca nut contains potent cholinergic muscarinic alkaloids, notably arecoline and guavacoline, with a wide range of parasympathetic mimetic effects, they promote salivation and the passage of wind through the gut, they rise blood pressure and pulse rate and they elicit a degree of euphoria by virtue of their GABA receptor inhibitory properties which contribute to dependence and habituation
- This arecoline plays a major role in the pathogenesis of OSMF by causing an abnormal increase in the collagen production.
- In genetically predisposed people, betel nut and pan chewing render the oral mucosa susceptible to chronic inflammatory changes with decreased T-lymphocyte count and higher null cell count. Areca nut, chilli and misi are the chief local factors in the production of OSMF.
  - CHILLI: A hypersensitivity reaction to chilies is believed to contribute to the occurrence of OSMF, allergen induced eosinophilia due to capsaicin.
  - MISI:a black colored powder containing the substances like soda, borax, powdered alum, charcoal of myrobalan and fi llers earth in varying proportion The fl avonoid catechin and tannins from betel nut stabilizes the collagen fi bers and makes them resistant to degradation by collagenase.
  - NUTRITIONAL DEFICIENCY: anemia, vitamin, iron and protein defi ciencies among OSMF patients. Iron metabolism seems to be the primary factor and defi ciency in folic acid, pyridoxine, and vitamin B12 defi ciencies are secondary.
  - genetic factors: like increased factors like HLA DR 10, DR3 and DR7 have been reported. Immunological studies have shown raised immunoglobulin like A, E and D.
  - Matrix metalloprotein: The genomic studies have shown the 5A genotype of MMP3 promoter was associated. Studies have shown that six collagen related genes including COL1A1, COL1A2, COLase, LYOXase, TGF-1, and CST3 are found to be located on different chromosomes in OSMF patients.
  - Role of saliva: trace metal copper in the molecular pathogenesis of OSMF as they found the high levels of copper expression in saliva could act as initiating factor and stimulation of fi Progenesis by up regulation of lysyl peroxide.

MOLECULAR PATHOGENESIS:

A prominent mediator is transforming growth factor-beta (TGF-β). The growth factor has also been implicated in the development of many fibrotic diseases. It causes the deposition of extracellular matrix by increasing the synthesis of matrix proteins like collagen and decreasing its degradation by stimulating various inhibitory mechanisms.

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So transforming growth factor beta signalling pathway might be critical for pathogenesis of OSMF,

Clinical features :

Gender—it affects both sexes.

Age: 20 and 40 years of age.

• Site distribution—the buccal mucosa ,retromolar areas.

It also commonly involves soft palate, palatal fauces, uvula, tongue and labial mucosa. Sometimes, it involves the floor of mouth and gingiva.

• Prodromal symptoms—the onset of the condition is insidious and is often of 2 to 5 years of duration. The most common initial symptom is burning sensation of oral mucosa, aggravated by spicy food, followed by either hypersalivation or dryness of mouth. Vesiculation ulceration, pigmentation, recurrent stomatitis and defective gustatory sensation have also been indicated as early symptoms.

Late symptoms

• Trismus—gradual stiffening of the oral mucosa occurs in few years after the initial symptoms appear. This leads to inability to open the mouth completely

• Difficulty in tongue protrusion—later on, patients experience difficulty in protruding the tongue.

• Difficulty in swallowing—when the fibrosis extends to pharynx and esophagus, the patient may experience difficulty in swallowing the food.

• Referred pain—referred pain in the ears and deafness, due to occlusion of Eustachian tube and a typical nasal voice has been reported.

• Blanching of mucosa—the most common and earliest sign is blanching of mucosa, caused by impairment of local vascularity The blanched mucosa becomes slightly opaque and white. The whitening often takes place in spots so that the mucosa acquires a marble like appearance.

Blanching may be localized or diffuse, involving greater part of the oral mucosa or reticular, in which blanching consists of blanched area with intervening clinically normal mucosa, giving it a lacelike appearance.

Betel chewer mucosa—it is brownish red discoloration of mucosa with irregular surface that tend to desquamate.

• Fibrous band—As disease progresses the mucosa becomes stiff and vertical fibrous band appears. This band can be palpate easily and feel rough on palpation.

• Lips features—mucosa is blanched, becomes rubbery and is characterized by the presence of circular bands around the rima oris like a thin band .In severe labial involvement, the opening of mouth is altered to an elliptical shape (elliptical rima oris), lips become leathery and it become difficult to evert them.

Buccal mucosa—the affected mucosa becomes coarse, blanched and inelastic. In advanced cases, the mucosa becomes tough and leathery with numerous vertical fibrous bands.

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Soft palate (49%) and uvula—involvement of soft palate is marked by fibrotic changes and a clear delineation of the soft palate from hard palate. The mobility of soft palate is restricted. Uvula, when involved, is shrunken and in extreme cases it becomes bud-like or hockey stick appearance.

Palatal fauces—In the soft palate the bands radiate from pterygomandibular raphe to the anterior faucial pillars. The faucial pillars become thick and short and tonsils may get pressed in between fibrosed pillars.

Tongue—The initial change is depapillation, usually in the lateral margins. Tongue becomes smooth its mobility, especially in protrusion, becomes impaired. Patient cannot protrude the tongue beyond the incisal edges.

- Floor of mouth—when floor of mouth is affected, it becomes inelastic.
- Gingiva—when affected, it becomes fibrotic, blanched and inelastic.

Clinical Stages of Oral Submucus Fibrosis

- **Stage of stomatitis and vesiculation**—this is the earliest stage and is characterized by recurrent stomatitis and vesiculation. Patient complains of burning sensation in the mouth and inability to eat spicy food. The examination reveals vesicle formation particularly on the palate. They may rupture and superficial ulceration may be seen, which may cause painful mastication. Some amount of fibrosis is seen in this stage and mucosa shows whitish streaks. An occasional granulating red spot may be seen on the palate.
- **Stage of fibrosis**—the patient complains of stiffness and inability to open the mouth completely. As the disease progresses, there is difficulty in blowing out the cheeks. Tongue movement becomes restricted and protrusion of tongue is difficult. Complains of pain in the ear may occur occasionally. Speech may become muffled and indistinct due to restriction of jaw movement. Due to restricted palatal movement, a nasal twang may occur in speech. The examination reveals increased fibrosis of submucosal tissue, which appears blanched and white. The lip and cheek become stiff and the vestibule of mouth is gradually reduced and almost obliterated. This causes difficulty even in introducing the examining fingers in between lips, cheeks and teeth in advanced cases. The palate shows blanching and fibrosis which cause shortening and disappearance of uvula in advanced cases. The fibrotic bands extending from palate to tongue cause strangulation of tonsills and they appear buried in the faucial pillars. The dorsum of tongue shows atrophy of papillae. The mucosa of the floor of mouth beneath the tongue, also show blanching and stiffness.
- **Stage of sequelae and complications**—the patient presents with complaints as described above in stage II. On examination, evidence of whitish leukoplakic changes and rarely an ulcerating malignant lesion may be seen.

MANAGEMENT:

Restriction of habit/behavioral therapy

- **Vitamin rich diet**—a vitamin rich diet along with iron preparation is helpful to some extent but has little therapeutic value in relieving trismus.

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• **Iodine B complex preparation**—iodine-B-complex preparation (Injection Ranodine) is a combination of iodine preparation with synthetic vitamin B complex.

The combination of iodine compound with vitamin B complex is responsible for the stimulation of metabolic process and enzymatic process within the body (oxygen reduction, transamination). Intramuscular injection starts with small doses and continuing with larger doses (2 ml ampule daily). The course of 5 injections is repeated after 7 days. Each 2 ml consists of:

• Methyltrioxyethyl iodomine—progressive increasing doses equivalent to 0, 25, 50, 75 and 125 mg of active iodine
• Vitamin B1—1.0 mg
• Vitamin B6- 0.3 mg
• Vitamin B2—0.6 mg
• Nicotinamide—15.0 mg
• Calcium pantothenate—1.0 m
• Injection of arsenotyphoid and iodine—arsenotyphoid is a fibrin dissolving agent.

Steroids

• Local—hydrocortisone injection along with procaine hydrochloride injection locally in the area of fibrosis.

Systemic—A therapy with hydrocortisone 25 mg tablet, in doses of 100 mg/day is useful in relieving burning sensation without untoward effects. Triamcinolone or 90 mg of dexamethasone can be given.

This is supplemented with local injection of hydrocortisone 25 mg at biweekly intervals at the affected site.

Placental extract

• Mechanism—placentrax is an essential biogenic stimulator. It is suggested that it stimulates pituitary adrenal cortex and regulates metabolism of tissue. It also increases the vascularity of tissues.

Placental extract contains

• Nucleotides—ribonucleic acid (RNA) and adenosine triphosphate (ATP).
• Enzymes—alkaline and acid phosphatase, glutamic oxaloacetic acid transaminase, glutamic acid and pyruvic acid.
• **Vitamins**—vitamin E, B, B6, B12, pantothenic acid, nicotinic acid, biotin PABA and folic acid.
• Steroids—17, ketosteroid.
• Fatty acids—linoleic acid, lenolenic acid, palmitic acid.
• Trace elements—copper, selenium, magnesium.

Dose : Each region is locally injected around fibrous bands, intra-muscularly, at the interval of 3 days for 15 days. Each time 2 ml solution is deposited. This course can be repeated after a month, if required.

Hyaluronidase

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• Mechanism—improvement in health of mucous membrane, burning sensation and trismus was observed by using hyaluronidase injection. Hyaluronidase, by breaking down hyaluronic acid (ground substance of connective tissue), lowers the viscosity of intracellular cement substance i.e. hyaluronidase decreases cell formation by virtue of its action on hyaluronic acid, which plays an important role in collagen formation.

Lycopene

• Content—it is an antioxidant from tomato extract, along with other previously used antioxidants in the treatment of OSMF.

• Dose—tab Lycopene 2000 mcg. The drug will be given for a period of three months duration during which time patient will be reexamined every 15 days.

Vitamin E

• Mechanism—the use of vitamin E along with dexamethasone and hyaluronidase injections is thought to produce better results. Vitamin E presumably works by—

• Preventing the oxidation of essential cellular constituents such as the formation of oxidation product.

• Protecting against various drugs, metals and chemicals and acts as scavenger of free radical. • It may improve the survival of erythrocytes.

Other therapies

• Vasodilator injection—vasodilator injection, which relieves the ischemic effect and helps the nutritional and therapeutic measures to reach the affected tissue, with use of fluorouracil an anti-metabolic agent.

• Injection of interferon gamma—This is recently discovered therapy. Intralesional injection of interferon gamma improved mouth opening and reduce mucosal burning.

Surgical treatment

Lasers

Criyosurgery

Oral Physiotherapy

Oral exercises are advised in early and moderately advanced cases. This includes mouth opening and ballooning of mouth. This is thought to put pressure on fibrous bands. Forceful mouth opening have been tried with mouth gag and acrylic surgical screw.

Diathermy Microwave diathermy is useful in some early or moderately advanced stages. Low current is used (20 watts × 2450 cycles). It acts by physio fibrinolysis of bands. Its value is increased if it is combined with other treatment modalities.

SHORT ESSAY 8×5=40 MARKS

3.Trigeminal neuralgia

Ans: It is also called as Tic Douloureux (painful jerking), Trifacial neuralgia or Fothergill's disease.

Etiology :

Dental pathosis—dental pathosis is believed by some investigators to be involved with the onset of trigeminal neuralgia.

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• Excessive traction—secondary to excessive traction on the various divisions of the fifth nerve, being influenced by maxillo-mandibular relationship.
• Allergic—it can be secondary to an allergic and hypersensitivity reaction causing edema of the trigeminal nerve root.
Compression and ditortoin: Vessels become elongated with advancing age and with atherosclerotic involvement gain abnormal positions by wedging into the space between the pons and trigeminal nerve. It is postulated that with progressive material elongation, fascicles of adjacent nerves later suffer myelin injury and pain results
Anomalies of superior cerebellar artery—it is the most recently blamed cause for trigeminal neuralgia. It lies in contact with the sensory root of the nerve and implicated as a cause of demyelination.
Secondary lesion—conditions such as carcinoma of the maxillary antrum, nasopharyngeal carcinoma, tumors of peripheral nerve root, intracranial vascular anomalies, and multiple sclerosis may be presented with trigeminal pain

Clinical Features

• Age and sex distribution—it usually occurs in middle and old age, It most frequently occurs in women.
• Site—it is more common on the right side and the lower portion of the face is more frequently affected.
• Nature of pain—the pain is paroxysmal, lasting only a few seconds to a few minutes and is usually of extreme intensity. It may be described by the patient as resembling 'knife like stabs' 'lightening', 'electric shock', 'stabbing' or 'lancinating' type of pain. During the intervals between these violent experiences, there is usually no pain or a mild or dull ache. Attacks do not occur during sleep.
• Location of pain—the pain is confined to the trigeminal zone, nearly always unilateral and, if bilateral, is successive rather than concomitant. The mandibular and maxillary divisions are more commonly involved than the ophthalmic. In some instances, these two divisions may be simultaneously affected. The pain never crosses the midline.
• Aggravating factors—the pain is provoked by obvious stimuli to the face. A touch, a draft of air, any movement of the face as in talking, chewing, yawning or swallowing may evoke a lancinating attack. Later the pain may be so severe that the patient lives in constant fear of an attack. Often there is a transitory refractory period after the attack.
• Triggers zones— 'trigger zones' which precipitate an attack when touched, are common on the vermilion border of the lips, the ala of the nose, the cheeks, and around the eyes .
Frozen or mask like face appearance—in extreme cases, the patient will have motionless face—the 'frozen or mask like face'.
• Associated features—trigeminal neuralgia may be accompanied by excess lacrimation, conjunctival injection and intense headache.

Management :

Medical treatment

• Trichloroethylene inhalation—it has been proved to be of value.

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• Topical capsaicin cream—topical capsaicin (nociceptive substance P suppressor) can be applied on affected area.
• Anti-cholinergic drugs—it was used for a short period during the late 1960s. Nowadays, this treatment modality is not used.
• Dilantin—diphenylhydantoin, an anti-convulsant drug has been recommended, which is effective when given orally, 300 to 400 mg per day.
• Carbamazepine (tegretol) has a special effect on the paroxysmal pain. s an initial dose, 100 mg is given twice daily until relief is established. At no time, the daily dose should exceed 1200 mg.
• Combination therapy—a combination of Dilantin and Carbamazepine may also be given.
Anti-inflammatory agents—anti-inflammatory agents like indomethacin and short courses of steroids have been found to be useful

Surgical treatment

• Injection of the nerve with anesthetic solution—local anesthetics injected near the peripheral branches of the trigeminal nerve serves to provide temporary relief from pain and helps in the diagnosis.
Injection of the nerve with alcohol— alcohol contacts the nerve, neurolysis occurs distal to the injection site. Nerve regeneration occurs in 6 to 24 months for most patients. But, the duration of relief from alcohol injection tends to decrease with repeated attempts. Generally 95% alcohol is used or procaine or monocaine 2%, chloroform 5%, absolute alcohol 70%, Ringer's solution 23% can also be used.
Nerve sectioning and nerve avulsion (peripheral neurectomy): Nerve sectioning is generally performed on the nerve which cannot be avulsed. The procedure can be performed on lingual, mental or buccal nerve. Peripheral neurectomy results in high degree of success in elimination of pain. But disadvantage of this technique is that result is temporary as nerve may regenerate

• **Decompression and compression—**
• Percutaneous microcompression—in this, inflated balloon is used to compress the gasserian ganglion.
• Microvascular decompression—retromastoid craniotomy is carried out and the offending vascular structures are dissected free of the nerves at root entry zone and maintained in that position by insertion of a small piece of gelfoam or Ivalon sponge.

Rhizotomy—actual cutting of trigeminal sensory root results in permanent anesthesia in most patients. The recurrence rate of trigeminal neuralgia after rhizotomy is 20%

4.clinical features and management of phemphigus vulgaries
Ans: **Clinical features**

- age: 5th and 6th decade of life. Rarely affects younger individuals.
- gender: Men and women are equally affected.

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- The margins of the ulcer show evidence of tissue tags.
- sites: buccal mucosa, gingiva and palate
- The edges of the shallow ulcers extend peripherally over a period of weeks until they involve large portions of the oral mucosa.
- Distal extension from the oral cavity causes involvement of the oesophagus, pharynx and larynx, which causes hoarseness of voice and dysphagia.

Management of phemphigus vulgaries :

Topical therapy

- Eroded and crusted, painful skin lesions and the associated foul odor can be effectively managed by bathing the area with 0.01% potassium permanganate solution or 0.5% silver nitrate solution.
- Alternatively, the raw surfaces can be sprayed with corticosteroids or 2% procaine hydrochloride.
- Chlorhexidine mouth rinses can be used to alleviate discomfort and malodour.

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- Painful oral ulcerations can be managed by topical application of viscous xylocaine especially before food intake.

Systemic therapy

- Corticosteroids: Systemic administration of corticosteroids comprises three phases:
  - **Control phase**: Characterized by an initial high dose corticosteroid administration to the point of obvious clinical improvement.

Therapy is initiated by giving- 60–160 mg of prednisone daily.

If there is no response even after a week, the dosage is doubled. When new lesions cease to form and old lesions heal, the dosage is decreased slowly.

  - **Consolidation phase:** In this phase the dosage of prednisone is reduced over a period of several weeks. once the control over the disease is achieved, an attempt to decrease the steroid dose by transferring the patient to intramuscular injections of triamcinolone acetonide is highly advisable.

  - **Maintenance phase**: The corticosteroids are gradually tapered down to alternate day dose and ultimately stopped. However, this reduction in dosage is made possible by replacing steroids with immunosuppressive drugs.
- The dosage of immunosuppressive drugs is reduced to zero in several months.
- Immunosuppressive agents:
  Azathioprine 100–200 mg per day in conjunction with prednisone 150–200 mg daily can be used.
- Plasmapheresis: It is particularly useful in patients who are refractory to corticosteroids. It involves removal of the circulating antibodies.
- Photopheresis: This modality of treatment was described by Rook et al. It involves administration of 8-methoxypsoralen followed by exposure of peripheral blood to ultraviolet radiation, causing photoinactivation of WBC.
- Immunomodulators: levamisole (100 mg/week), combination of nicotinamide and tetracycline and oral prostaglandins are effective in the treatment of pemphigus.

5.Radiographic features of fibrous dysplasia

Ans: Radiographic Features Lesions showing predominance of fibrous tissue

• Early—radiolucent with ill defined borders. The bonydefect may be often unilocular but occasionally bony septa may be apparent creating an impression of multilocular cavity.

• Margins—margins may be well defined with a tendency to blend imperceptibly with surrounding normal bone.

• Granular appearance—surrounding the margins of the radiolucent area, there may be wider band of increased density, but granular in appearance

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• Lamina dura—when the lesion involves the apices of teeth there is loss of lamina dura or if retained, it has less density than normal.

• Teeth—resorption of roots and destruction of developing teeth. • Jaws—when the lesion comes to the surface, there may be expansion of the jaws.

Lesions showing mixed radiolucent and radiopaque appearance

• Appearance—radiographic appearance of lesions with heterogeneous distribution of fibrous and osseous tissue shows a mixed radiolucent and radiopaque appearance, depending on the maturity of the lesions .

• Granular appearance—the new bone takes the form of very small opacities of poor density. When they become larger they appear as granular.

• Maxillary lesion—it may spread to involve the adjacent bone such as zygoma, sphenoid, occiput and base of skull.

Mature radiopaque lesions where bone is predominant
• Stippled • Orange peel—the radiograph shows bone of increased density. The normal structure of bone is replaced by a stippled appearance which resembles the ring of orange which is called as called as 'orange peel'.

• Teeth—tilting and bodily displacement of teeth in the affected area.

• Maxillary sinus—it may obliterate the maxillary sinus.

• Thumb print appearance—when mandible is affected, the vertical depth of mandible is increased. The inferior border of mandible appears as a ribbon like cortex.
the localized area over the cortex is lost and instead, there is a smooth curved downward projection of the inferior margins of the bone.
as if the bone had been soft and pressed upon by the thumb.

• Expansion—bony expansion usually extends to the buccal and distal aspect.

• Smoky mottled appearance—as the lesions mature, dysplastic bony trabeculae increase in size and number and appear like smoky mottled radiopacities.

• Granular appearance

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• Granular type—changes in the base of the skull is of granular type and structureless, so that affected portion of the bone is thickened and of greater density.

• Frontal bone—the frontal bone is also thickened with homogeneous or variegated type of density. • Nasal septum—the nasal septum is grossly thickened, dense and curved, so that it represents the gross caricature of the letter.

6.oral manifestation of HIV
Ans: CLASSIFICATION
More common
• Candidiasis
Less common
• Aspergillosis
• Histoplasmosis
• Cryptococcus neoformans
• Geotrichosis

Bacterial
More common
• HIV gingivitis
• HIV periodontitis
• Necrotizing gingivitis
Less common
• Mycobacterium avium intracellulare
• Klebsiella pnuemoniae
• Enterobacterium cloacae
• E. coli
• Salmonella enteritidis
• Sinusitis
• Exacerbation of apical periodontitis
• Submandibular cellulitis

Viral
More common
• Herpes simplex
• Varicella zoster
• Epstein-Barr including hairy leukoplakia
Less common
• HPV virus
• CMV virus

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- Pox virus Neoplasm More common
- Kaposi's sarcoma

Less common

- Non-Hodgkin's lymphoma
- Squamous cell carcinoma

Lymphadenopathy

Neurologic disorders

Less common

- Paresthesia
- Facial palsy
- Hyperesthesia
- Dysphagia

Miscellaneous

Less common

- Recurrent aphthous ulceration
- Progressive necrotizing ulceration
- Toxic epidermolysis
- Delayed wound healing
- Thrombocytopenia
- Xerostomia and sicca type syndrome
- HIV embryopathy
- Hyperpigmentation
- Granuloma annulare
- Exfoliative cheilitis
- Lichenoid and other drug reaction.

1. Candidiasis i. Pseudomembranous:
   The pseudomembranous is presented as a white or yellow removable plaque leaving a red surface. Pseudomembranous may be located in all parts of the oral cavity.
   ii. Erythematous: It is defined as red area without removable plaques often located on palate, dorsum of the tongue and buccal mucosa. Smears from red area must be positive for candida hyphae on PAS staining.
   iii. Angular: Fiery and commissures. Smears from red area must be positive for candida on PAS staining.

2. Periodontal disease

a. Gingivitis: They defined gingivitis as the disease characterized by fiery red edematous attached gingiva and may affect the alveolar mucosa. No ulceration must be present.

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b. Necrotizing gingivitis: This is characterized by gingival pain, swelling, ulcerations, necrosis or as distribution of interdental papillae covered with a fibrous slough. The patient suffers from fever and halitosis may be present.
c. Periodontitis: This is characterized by aggressive irregular bone destruction. Any infection that gives the impression of affecting periodontal structure other than gingiva.

3. Hairy leukoplakia Hairy leukoplakia presents as a white, non-removable lesion on margin of the tongue. The surface is corrugated, but might be non-corrugated if it is seen on the inferior surface of the tongue or on the buccal mucosa. To establish a reliable diagnosis, a biopsy must be performed. Biopsy from hairy leukoplakia shows hair-like projections, hyperparakeratosis, koilocytic like cells and no inflammation. The surface layer of the epithelium shows numerous hyphae of candida.

4. Oral Kaposi's sarcoma A characteristic macroscopic appearance of either erythematous or violaceous plaque-like lesions, or a bulky tumor predominantly seen in palate or on the gingiva.

7.causes and management of xerostomia
Ans: It is the subjective clinical condition of less than normal amount of saliva. It is dryness of mouth, which is a clinical manifestation of salivary gland dysfunction

Etiology :
• Radiation induced—ionizing radiation to head and neck region for the treatment of cancer results in pronounced changes in the salivary glands located within the primary beam.
The degree of damage caused by the radiotherapy is related to dose-time-volume factor.
Damage to the acinar cells has been noted with a single 100 rads dose of X-rays.
Radiation sensitivity decreases in following order:
the parotid gland, submandibular, sublingual to minor glands. Serous aciner cells appear to be more sensitive to radiation, than the mucus cells.
As the dose is increased, disorganization and destruction of the acinar cells occur, resulting in their replacement by fibrous or faulty tissues.
Both, the stimulated and unstimulated salivary flow rate decreases dramatically with increasing radiotherapy

. • Pharmacologically induced xerostomia—there are about 500 drugs which can cause xerostomia. The classes of drugs which cause xerostomia include
anticonvulsants,
antiemetics,
antihistaminics,
anti-hypertensives

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antispasmodics.

The mode of action for decreased salivary flow is generally related to the para-sympathetic activity, usually an antimuscarine effect. Other actions that can decrease salivation are generally more miscellaneous and include vasoconstriction of salivary glands, changes in fluid and electrolyte balance and changes in acinar or ductal function.

• Local factors—local factors like decreased mastication, smoking and mouth breathing can also lead to xerostomia.

• Developmental—developmental abnormalities of salivary glands, tumors, autoimmune states and certain diseases which affect afferent or efferent portions of neural transmission reflex are some of the other causes of xerostomia.

• Systemic alternations resulting in xerostomia

• Nutritional—certain deficiency states like pernicious anemia, iron deficiency anemia and deficiency of vitamin A and hormones can cause xerostomia. • Fluid loss—fluid loss associated with hemorrhage, sweating, diarrhea, vomiting.

• Diabetes mellitus—it is associated with xerostomia.

• Sjögren syndrome—xerostomia is also common in Sjögren syndrome.

• Other disease—HIV infection, sarcoidosis, and graft versus host resistance

Management

Stimulation of salivary production

• Local stimulation—chewing of gums, mints, paraffin and citric acid containing lozenges and rinses. Disadvantages of it are:

• Effects are short lived.

• Frequent application can be inconvenient.

• Citric acid may irritate oral mucosa.

• Continuous use may contribute to demineralization.

• Systemic stimulation

• Bromhexine—it is a mucolytic and mucokinetic agent, capable of inducing thin copious bronchial secretions. Dose—adults (8 mg TDS), children 1-5 years (4 mg BD) and children 5-10 years (4 mg TDS). • Anethole trithione (ANTT)—it is a directly acting cholinergic agonist which acts by neurostimulation. Dose 1 to 2 tabs (25 mg) TDS.

• Pilocarpine—pilocarpine is a cholinergic parasympathomimetic agent with a broad range of pharmacologic effects.

It increases the secretion by exocrine glands and can affect the sweat, salivary, lacrimal, gastric, pancreatic, intestinal glands and mucosal cells of the respiratory tract.

The usual dose is 5 mg, TDS. It produces short duration of (3 hours) increased salivary flow, without the accompanying side effects.

It should not be used in patients suffering from asthma.

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Symptomatic treatment

• Salivary substitute—there are number of salivary substitute available for the treatment of xerostomia.

Most commonly contain carboxymethylcellulose or hydroxyethylcellulose as lubricants and variety of artificial sweeteners, preservative and chloride or fluoride salts.

Disadvantages are:

• Their regular use is inconvenient to the patient.
• Most of them are more viscous than the natural saliva.
• They are expensive.
• They fail to provide antimicrobial and other protective functions of natural saliva.

• Composition of artificial saliva
• Carboxymethylcellulose—10 gm/l.
• Sorbitol—30 gm/l.
• Potassium chloride—1.2 gm/l.
• Sodium chloride—0.843 gm/l.
• Magnesium chloride—0.051 gm/l.
• Calcium chloride—0.146 gm/l.
• Dipotassium hydrogen phosphate—0.342 gm/l.
• Oral hygiene product—patient should use oral hygiene product which include lactoperoxidase, lysozyme, and lactoferrin.
• Discontinuous of drug—drug which is causing xerostomia should be discontinued.

8.TNM staging

Ans: It is universally accepted system which is developed by UICC (Union Internationale Centre of Cancer).

• Primary tumor (T): local extent is major factor contributing to prognosis.
• Tx: primary tumor cannot be assessed.
• T0: no evidence of primary tumor.
• Tis: carcinoma in situ.
• T1: tumor 2 cm or less in diameter.
• T2: tumor 2-4 cm in diameter.
• T3: tumor more than 4 cm in greatest diameter.
• T4: tumor of any size in which tumor invades adjacent structure (e.g.: cortical bone, inferior alveolar nerve, floor of mouth, skin of face etc).

• Regional lymph nodes (N)
• Nx: regional lymph node cannot be assessed.
• N0: no regional lymph node metastasis.

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• N1: metastasis in single ipsilateral lymph node less than 3 cm in diameter.
• N1a: nodes considered not contain to tumor growth.
• N1b: nodes considered to contain growth

• N2: single lymph node, no more than 6 cm in greatest dimension, of bilateral/contralateral lymph node, no more than 6 cm.
• N2a: Single ipsilateral lymph node more than 3 but less than 6 cm.
• N2b: multiple ipsilateral lymph nodes less than 6 cm.
• N2c: Bilateral or contralateral lymph node less than 6 cm in greatest dimension.

• N3: metastasis in lymph node more than 6 cm and it is fixed.
• N3a—ipsilateral nodes at least one greater than 6 cm.
• N3b—bilateral nodes greater than 6 cm.
• N3c—contralateral nodes at least one greater than 6 cm.

• Distant metastasis (M)
• Mx: Distant metastasis cannot be assessed.
• M0: No distant metastasis.
• M1: Distant metastasis.

AJC (American Joint Committee) It divides all cancers into stage 0 to 4, and takes into account all three previous TNM systems.
• Stage 0—T0 N0 M0
• Stage 1—T1 N0 M0
• Stage 2—T2 N0 M0
• Stage 3—T3 N0 M0, T1 N1 M0, T2 N1 M0, and T3 N1 M0
• Stage 4A—T4 N0 M0, T4 N1 M0, any T N2 M0
• Stage 4 B—Any T N3 M0
• Stage 4 C—Any T, Any N, M1

Dukes ABC Staging It is used in cancers of bowel
• Stage A—when tumor is confined to submucosa and muscle and cure rate is 100%.
• Stage B—tumor penetrates the entire thickness of bowel wall into pericolic or perirectal tissues and cure rate is 70%.
• Stage C—it is characterized by lymph node metastasis and reduces the cure rate to 30%

9.C/F,investigation &management of candidiasis.
Ans: Oral candidiasis
Acute
• Acute pseudomembranous candidiasis (thrush)
• Acute atrophic candidiasis (antibiotics sore mouth)

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Chronic
- Chronic atrophic candidiasis
- Denture stomatitis
- Median rhomboid glossitis
- Angular cheilitis
- Id reaction
- Chronic hyperplastic candidiasis

Chronic mucocutaneous candidiasis
- Familial CMC
- Localized CMC
- Diffuse CMC
- Candidiasis endocrinopathy syndrome

Extraoral candidiasis
- Oral Candidiasis associated with extraoral lesions orofacial and intertriginous sites (candidal vulvovaginitis, intertriginous candidiasis)
- Gastrointestinal candidiasis
- Candida hypersensitivity syndrome

Systemic candidiasis • Mainly affect the eye, kidney and skin

Thrush :
c/f:
In infants
- Age—in neonates, oral lesions start between the 6th and 10th day after birth.
- Cause—infection is contracted from the maternal vaginal canal where Candida albicans flourishes during the pregnancy.
- Appearance—the lesions in infants are described as soft white or bluish white, adherent patches on oral mucosa which may extent to circumoral tissue.
- Symptoms—they are painless and noticed on careful examinations. They may be removed with little difficulty.

In adult
- Sites—common sites are roof of the mouth, retromolar area, and mucobuccal fold. But it is common on any other mucosal surface.
- Sex—it is common in women as compared to male.
- Prodormal symptoms—prodormal symptom like rapid onset of bad taste may be there. Spicy food will cause discomfort.
- Symptoms—patient may complain of burning sensation.
- White plaques—pearly white or bluish white plaques are present on oral mucosa. They resemble cottage cheese or curdled milk Patches are loosely adherent to oral mucosa.

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• Composition of plaques—it is composed of tangled mass of hyphae, yeast, desquamated epithelial cells and debris.
• Adjacent mucosa—Mucosa adjacent to it appears red and moderately swollen.
• Wiping of patches—white patches are easily wiped out with wet gauze which leaves either a normal or erythematous area or atrophic area. This area may be painful. Deeper invasion by the organism leaves an ulcerative lesion upon the removal of patch.
Malignant association—it is occasionally associated with (coexist with) dysplastic or carcinomatous change.

Acute atropic candidiasis
Clinical Features
• Age and sex—it predominantly occurs in men of middle age or above. The majority of these patients are heavy smokers.
• Sites—it occurs on cheek, lip and tongue.
• Appearance—candidal leucoplakia is extremely chronic form of oral candidiasis in which firm, and white leathery plaques are found
• Symptoms—lesions may persist without any symptoms for years.

Chronic hyperplastic candidiasis (candida leukoplakia) Hyperplastic candidiasis is seen as chronic, discrete raised.
lesions that vary from small, palpable translucent whitish areas to large, dense, opaque plaques, hard and rough to touch
The most common sites are the anterior buccal mucosa along the occlusal line, and laterodorsal surfaces of the tongue.
The most common appearance is that of asymptomatic white plaques or papulesthat are adherent and do not scrape off.

Median rhomboid glossitis
Median rhomboid glossitis is a form of chronic atrophic candidiasis characterized by an asymptomatic, elongated, erythematous patch of atrophic mucosa of the posterior mid-dorsal surface of the tongue due to a chronic Candida infection.

Angular cheilitis (perleche)
Clinical appearance is that of red, eroded, fissured lesions which occur bilaterally in the commissures of the lips and are frequently irritating and painful .
The most common etiology is loss of vertical occlusal dimension, but it may also be associated with immunosuppression.

Chronic mucocutaneous candidiasis (CMC) It is the term given to the group of rare syndromes, with a definableimmune defects, in which there is persistent mucocutaneous candidiasis that responds poorly to topical antifungal therapy. The main types of this rare

disorders include familial CMC, diffuse CMC, candidiasis endocrinopathy syndrome, candidiasis thymoma syndrome.

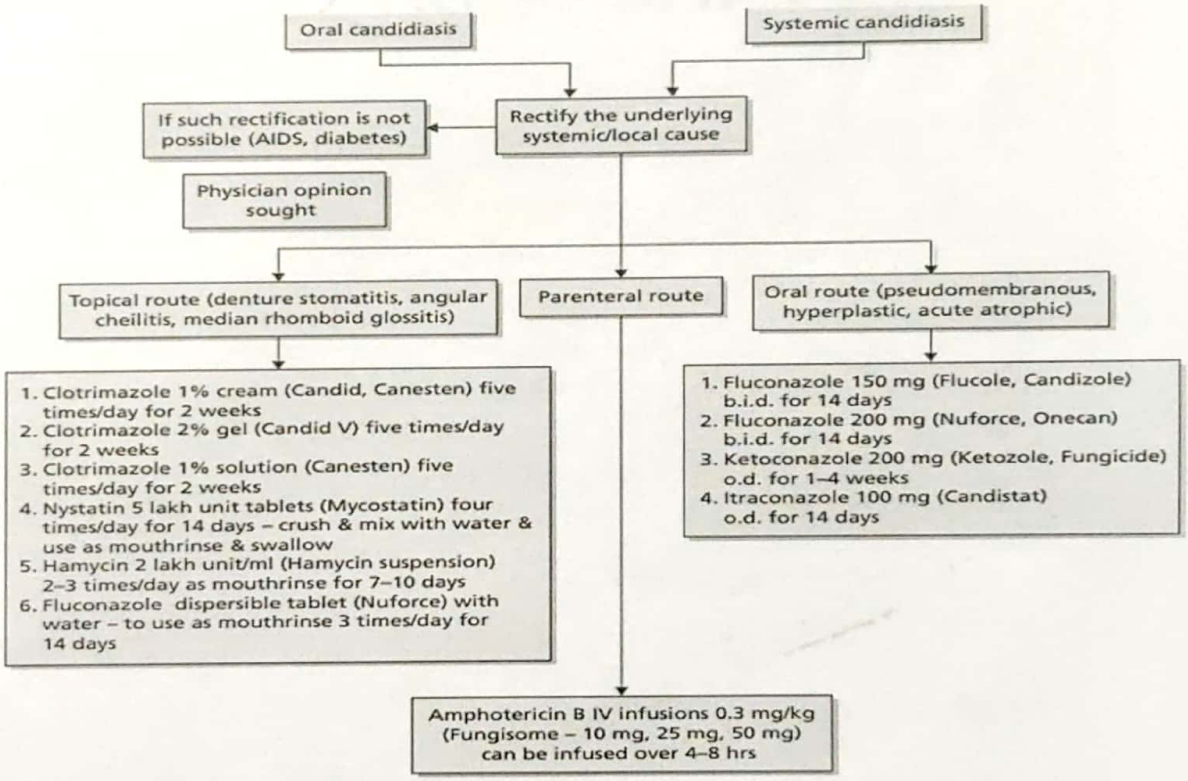
Chronic multifocal oral candidiasis

This term has been given to chronic candidal infection that may be seen in multiple oral sites, with various combinations, including angular stomatitis, median rhomboid glossitis and palatal lesions.

All these lesions will be having 1 month duration with no history of predisposing factors like systemic diseases, or patient's receiving any drugs, or radiotherapy.

These lesions are most commonly seen in chronic smokers in their 5th or 6th decade of life.

Management



10.difference between leukoplakia and lichen planus

Ans:

	leukoplakia	Lichen planus
Definition	Leukoplakia is a white patch or plaque that cannot be characterized clinically or pathologically as any other disease. The definition indicates that the term leukoplakia does not carry a histologic connotation and should be used	Oral lichen planus (OLP) is a common chronic immunological inflammatory mucocutaneous disorder that varies in appearance from keratotic (reticular or plaque like) to

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	only in descriptive clinical context.	erythematous and ulcerative.
Etiopathogenesis	Smokeless tobacco is believed to result in chemical damage that produces sub-lethal cell injury within the deeper layers of oral epithelium. This in turn induces concomitant epithelial hyperplasia. Smokeless tobacco often leads to tobacco pouch keratosis rather than true.	T cell mediated disorder in which there is production of cytokines which leads to apoptosis. Autocytotoxic CD8 and T cells trigger apoptosis of oral epithelial cells (Eversole, 1997; Porter et al, 1997). The immune system is triggered due to the interactions among genetic, environmental, and lifestyle factors.
site	Oral cavity	Oral cavity ,skin
Predilection female to male	3:1	2:1
Clinical forms	Homogenous Speckled leukoplakia, Erythroplakia	hypertrophic (plaque like, popularreticular), erythematous (atrophic, erosive) and bullous.
color	Color—lesion may be white or yellowish white, but with heavy use of tobacco lesion it may assume brownish color	• Color—papules are sharply demarcated from surrounding skin, which appears red but soon takes reddish purple or violaceous blue color. Later, dirty brown color develops. Center of papule may be slightly umbilicated
symptoms	Symptoms—some patients may report a feeling of increased thickness of mucosa. Those with ulcerated and nodular type may complain of burning sensation. Enlarged cervical lymph nodes may be a single occurrence of metastasis.	Symptoms—the chief complaint is usually of intense pruritus. The itching associated with LP usually provokes rubbing of the lesions, rather than scratching. patient may report with burning sensation of oral mucosa.
	• Ulcerative leukoplakia characterized by red area, which at times exhibit yellowish areas of fibrin, giving the appearance of ulceration. White patches are present at the periphery of the	Reticular type—it is most common form and is mostly bilateral. It consists of slightly elevated fine whitish lines that produce lace-like pattern of fine radiating lines, called as

	lesion. • Nodular leukoplakia—it is also called as 'leukoplakia erosiva' or 'speckled leukoplakia'. It is a mixed red white lesion in which small keratotic nodules are scattered over an atrophic patch of oral mucosa • Verrucous leukoplakia or verruciform leukoplakia—it is also called as 'leukoplakia verrucosa'. It is characterized by verrucous proliferation above the mucosal surface. These lesions demonstrate sharp and blunt projection. These projection are heavily keratinized. • Erythroleukoplakia—in some lesion of leukoplakia red component is present. This intermixed lesion is called as erythroleukoplakia • Proliferative verrucous leukoplakia (PVL)—verrucous leukoplakia can become more exophytic with development of multiple	Wickham's striae.The lesion may present radiating white thread like papules in a linear, annular or retiform arrangement. A tiny white dot is frequently present at the intersection of white • Papular—whitish elevated lesions of 0.5 mm to 1 mm in size, well seen on keratinized areas of oral mucosa. Papules are spaced apart; still close enough to give pebbled white or gray color (Fig. 12-29). Sometimes they coalesce. Most oftenly, papules are seen at the periphery of reticular pattern • Plaque—it is seen on dorsum of tongue and buccal mucosa. In case of plaque of tongue, disappearance of the tongue papillae is seen. It spreads in concentric peripheral growth. It consists of either pearly white or grayish white plaque. Such plaques generally range from slightly elevated and smooth to slightly irregular form.

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	keratotic plaques with roughened surface projection	• Atrophic form—it appears as smooth, red, poorly defined area, often but not always, with peripheral striae evident. The attached gingiva is frequently involved in this form of lichen planus is called desquamative gingivitis pattern. At the margins of atrophic zones
Management	Topical corticosteroids (increasing order of potency) 1. Triamcinolone acetonide 0.1% (Tess gel, Ledercort ointment) t.i.d./day until symptoms improve (maximum of 1 month) 2. Fluocinolone acetonide 0.025% (Fluzone cream) t.i.d./day for 2 months with tapering dose 3. Clobetasone propionate 0.05% (Clobetol cream, Cosvate gel) b.i.d./day for 2 month Systemic therapy Intralesional steroids: Injection triamcinolone 0.5 ml (Amcort) with once/week for 4 weeks	Topical retinol-A ointment application.bid /1month Systemic: A. Capsules of lycopene 4 mg bid or 8 mg OD for 3 months B. Capsules antioxidants with selenium bid for 6 months.

SHORT ANSWER 5×2=10

11.Herpes zoster c/f and management

Ans: c/f

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• Site—it may be found on buccal mucosa, tongue, uvula, pharynx and larynx.
• Symptoms—patient noticed pain, burning, tenderness usually on the palate on one side.
• Signs—after several days of symptoms, intact vesicles appear which soon rupture to leave areas of erosion.

- Healing—healing usually takes place within 10 to 14 days
- Teeth—trigeminal herpes zoster occurring during tooth formation causes pulpal necrosis and internal root resorption

Management
• Antiviral drugs—acyclovir 800 mg five times daily which is associated with significantly accelerated healing within 48 hours of the onset of rash.

• Symptomatic treatment—antipyretic medication with antipruritics diphenhydramine can be administered to decrease itching.

• Prevention of postherpetic neuralgia—intralesional steroids and local anesthetic can be used to decrease healing time and to prevent postherpetic neuralgia. But this comes with many side effects and there are some conflicting reports about the efficiency of steroid in control of postherpetic neuralgia.

• Capsaicin—topical capsaicin 0.025% four times a day has been suggested for temporary relief of neuralgia following herpes zoster infection. Capsaicin is derived from red peppers The mechanism of action apparently involves the depletion of substance P in the peripheral sensory neurons causing the skin less sensitive. After treatment patient should wash hand after use and avoid contact with mucosal surface.

• Tetracycline rinse—mouth rinsing with tetracycline, three to five times daily, may reduce the pain.

12.Antifungals drugs
Ans: classification
Azoles Imidazole
Topical • Clotrimazole • Econazole • Miconazole Systemic

Triazoles Systemic • Fluconazole • Itraconazole

Allylamine • Terbinafine Other topical agents • Tolnaftate • Undecylenic acid • Benzoic acid • Cyclopiroxolamine • Quiniodochlor • Sodium thiosulfate

13.dental consideration in patient with CVS disorders

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Ans :in hypertensive patients: Patients with controlled hypertension can receive dental care in short appointments.
Epinephrine in local anesthesia is not contraindicated unless systolic pressure is over 200 mmHg or diastolic pressure is over 115 mmHg.
Gingival retraction cords containing epinephrine should be avoided.
Ischemia :
Preoperative nitroglycerin can be given prophylactically before dental therapy. Effective local anesthesia is a must. Long-acting local anesthesia with bupivacaine can be used with vasoconstrictor to prolong the effect of local anesthesia. Aspirating syringe is a must.

Myocardial infraction :

Patients within 6 months of an MI (recent MI) are at the risk of further complications, hence, elective dental care should be deferred. However, the first 6 weeks is more critical, and with the physician's consent, simple emergency dental treatment under LA may be done during the first 6 months.

3. Anxious patients may be given preoperative glyceryl nitrate.

4. Effective local anesthesia is important. 5. Aspirating syringes must be used. 6. Use of epinephrine impregnated gingival retraction cords should be avoided.

Congenital disease:
Dental considerations
- Antimicrobial prophylaxis
- Bleeding tendencies due to platelet dysfunction and excessive fibrinolytic activity .
- Dental bacteria may cause cerebral abscess
- Aspiration during local anesthetic procedure is a must due to epinephrine in local anesthesia.
- Gingival retraction cord containing epinephrine should be avoided.

Heart failure :
- Physician's consent is a must.
- Patient should avoid heavy meals just before their dental appointment since digitalis may cause nausea and vomiting if the doses are high.
- The dentist should avoid stimulating gag reflex. Rubber dam should be used cautiously.
- Patient should not be in supine position because dyspnea is worsened.
- Dental treatment may precipitate dysrhythmias and angina in uncontrolled patients.
- Anxiety must be minimized and patient's pain control should be effective.

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○ Patients should be treated in late morning, because endogenous epinephrine peaks during early morning and cardiac complications may arise

Anemia:

Increased bleeding tendency might be a problem while treating anemic patients with hemoglobin levels less than 10 g/dl. This tendency is exhibited due to altered rheologic interactions between cells when hemoglobin level falls below a critical level. General anesthesia is also contraindicated in severe anemia, as oxygen carrying capacity is severely impaired. Therfore, patients presenting with typical features of anemia should be thoroughly investigated and referred to a physician for further investigations and treatment.

Thallasemia

A patient who has had a splenectomy is at risk of massive infection following bacteremia. It has been suggested that these patients receive prophylactic antibiotics like oral penicillin or erythromycin prior to dental treatment. Hepatitis risk to the patients due to transfusion should also be considered and universal precautions taken. Poor healing is also a complication of the dental treatment. Surgery for facial deformities has been used successful.

14.oral manifestation of diabetes mellitus?

ANS:

1. Periodontitis
2. Dental caries
3. Candidiasis
4. Halitosis
5. Xerostomia
6. Tooth loss
7. Burning mouth sensation
8. Taste impairment

15 Oral manifestation of anemia

ANS:

1. generalised stomatitis
2. mucosa pallor
3. glossitis
4. angular chelities
5. gingivitis
6. RAS
7. Candidiasis
8. Plummer vinson syndrome
9. Susceptible to infection
10. Taste distrubances

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AL-BADAR RURAL DENTAL COLLEGE & HOSPITAL GULBARGA

INTERNAL ASSESSMENT THEORY EXAMINATION : I[st] / II[nd] / III [rd]

Model Answer Paper.

1. Degree : _____

2. Exam & Subject : _____

3. Candidate Name : _____

4. Exam Date ☐☐ / ☐☐ / ☐☐

5. Register No.

6. Q P Code

Maximum Mark's.....Mark's Obtained.....

Question No.	Marks obtained	Question No.	Marks obtained
01		09	
02		10	
03		11	
04		12	
05		13	
06		14	
07		15	
08		Total marks :	

Maximum marks : ☐

Marks obtained : ☐

Signature of the Candidate : ☐

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Signature of the staff : ☐

LONG ESSAY 10×2=20.

1. Discuss the structure of Junctional epithelium and discuss the role in defence mechanism of the gingiva?

Answer:

- The Gingival Epithelium around a tooth is divided into 3 functional compartments outer, sulcular, and Junctional epithelium.
- The outer epithelium extends from the mucogingival junction to the gingival margin where Crevicular epithelium the sulcus.
- At the base of the sulcus connection between gingiva and tooth is mediated with junctional epithelium.
- J.E is attached to the tooth surface by a distinct mechanism known as the epithelial attachment apparatus.
- It has unique structural and functional features that contribute to preventing pathogenic bacterial flora from colonizing the subgingival tooth surface.

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✱ HISTORICAL ASPECT

- Gottlieb (1921) was first to describe J.E.
- Schroeder and Listgarten (1977) clarified anatomy and histology of the dentogingival junction.
- ORBANS CONCEPT (1953)

  He stated that the separation of the epithelial attachment cells from the tooth surface involved preparatory degenerative changes in the epithelium.
- GAERTNER'S CONCEPT (1980).

  He presented the concept of epi. cuff. This concept was based on insertion of thin blades between the surface of tooth and the gingiva.
- SCHROEDER AND LISTGARTEN CONCEPT (1971)

  ✱ 1° epithelial attachment refers to the epithelial attachment lamina released by the REE. It lies in direct contact with enamel and epithelial cells attached to it by hemi-desmosomes

  ✱ When REE cells transform into JE cells the primary epithelial attachment becomes 2° Epi. attachment. It is made of epithelial attachment between basal lamina and hemi-desmoson

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What is Junctional Epithelial?

- It is the 3rd component of the epithelial integument of the periodontium, in addition to the oral gingival epithelium and oral sulcular epithelium.

- It consists of collar like band of stratified Squamous nonkeratinizing epithelium..

- It is 3-4 layers thick in early life, but the number of layers increases with age to 10 or even 20 years.

- JE tapers from its coronal end, which may be 10 to 29 cells wide to 1 or 2 cells at its apical termination located at the Cemento-enamel junction in healthy tissue.

- Those cells are grouped into 2 strata:
  - Basal layer facing Con. tissue
  - Suprabasal layer extending to the tooth surface.
  - The length of the JE ranges from 0.25 to 1.35 mm.

- It provides attachment mechanism of the epithelium to the surface of tooth hard substance.

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o. It also provides a protective fun sclalum to the subjacent periodontal ligament.

Definations of J.E:

1. Junctional Epithelium is the non keratinized stratified squamous epithelium which attaches and form a collar around the cervical portion of the tooth that follows CEJ.
- Carranza's

2. Epithelial attachment is the structural complex by which junctional epithelium is attached to the tooth surface.

Functions:

- Forms epithelial barrier against the plaque bacteria.
- It allows the access of GCF, inflammatory cells and components of the immunological host defense to the gingival margin.
- JE cells exibit rapid turnover, which contributes to the host parasite equilibrium and rapid repair of damaged tissue.

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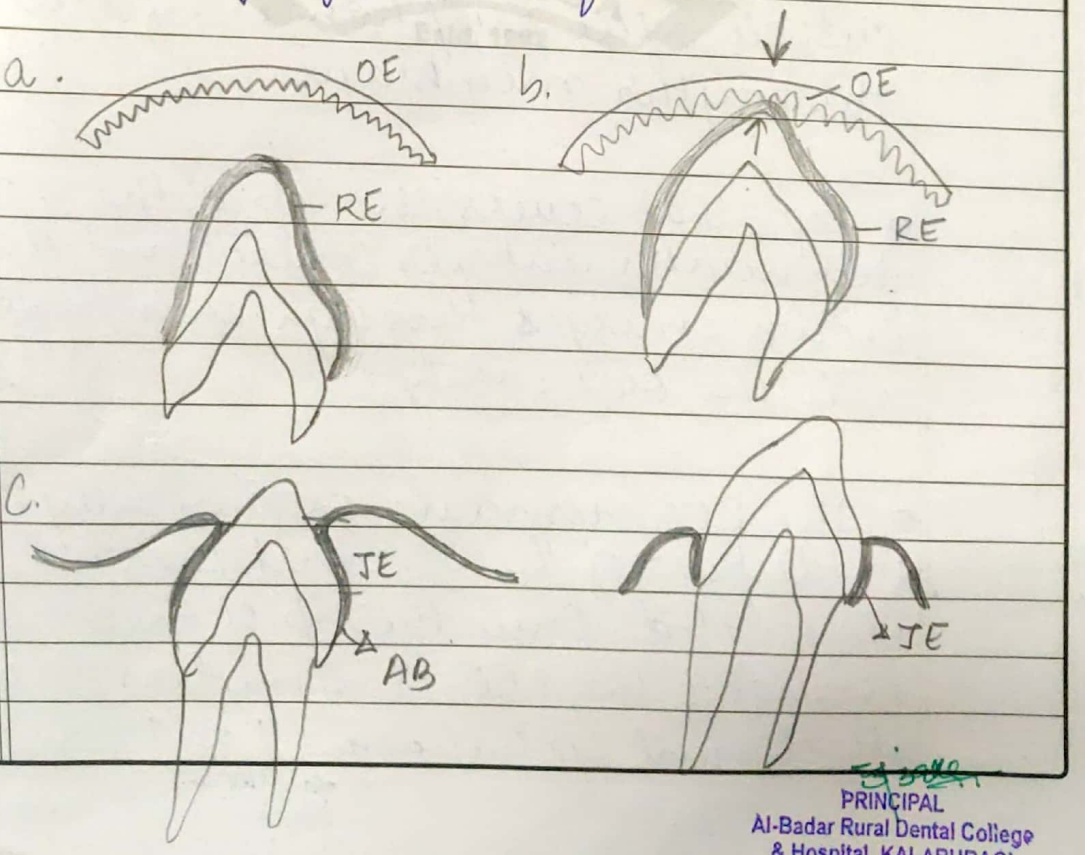
DEVELOPMENT OF JUNCTIONAL EPI.

1. Formation of REE.
2. Union of REE and oral epi.
3. As the tooth erupts REE is converted into J.E.

Changes during conversion:

- Cuboidal cells derived from ameloblast begin to flatten and align parallel to the tooth surface and appearance of JE.

- Since these cells which have lost capacity to divide get exfoliated at base of sulcus and cells from stratum intermedium which has proliferative capacity gets transformed into JE.



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- When the ameloblasts finish the formation of the enamel matrix, they leave a thin membrane on the surface of the enamel called the primary enamel cuticle.

- The ameloblasts shorten after the primary enamel cuticle has been formed and the epithelial enamel organ is reduced to a few layers of flat cuboidal cells called reduced enamel epithelium. [Orban.

- During eruption, the tip of the tooth approaches the oral mucosa, and the REE and the oral epi. meet and fuse.

- The remnant of the primary enamel cuticle after eruption is referred to as Nasmyth's membrane.

- Epi. that covers the tip of the crown degenerates in its centre and the crown emerges through this perforation into the oral cavity.

- The REE remains organically attached to the part of the enamel that has not yet erupted. Once the tip of the crown has emerged, the REE is termed as Primary attachment epithelium. [JE]

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- At the margin of the gingiva, the JE is continuous with the oral
- As the tooth erupts, the REE grows gradually shorter.
- Gingival sulcus may develop between the gingiva and the surface of the tooth and extend around its circumference.
- It is bounded by the JE at its base and by the gingival margin laterally. The gingiva encompassing the sulcus is the free, or marginal gingiva.

Anatomical features:

- Forms a collar peripheral to cervical region of the tooth of about 0.75 to 1.35
- Interproximally JE of adjacent teeth fuse to form the lining of the col area.
- Epithelial connective tissue interface is smooth [no rete pegs]
- JE is thickest at bottom of sulcus and tapers off in apical direction.

Microscopic features:

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- It has 15-30 cells layers coronally & 1-3 layers at apical.

- Contains 2 strata
  1. Stratum basale.
  2. Stratum suprabasale.

- The basal and adjacent 1-2 suprabasal cells are cuboidal to slightly spindle shaped and all the remaining cells are flat and oriented parallel to the tooth surface.

- The innermost suprabasal cells also called OAT cells [Salones etal 1994) forms and maintain the epithelial attachment apparatus.

- Has 2 lamina
  → External
  → Internal
  } Basal lamina.

17 - J.E IN THE ANTI-MICROBIAL DEFENSE :

- It contains active populations of cells and antimicrobial functions, which together form the 1st line of defense

- JE provides barrier against bacteria even though many bacterial substances like lipopolysaccharide pass easily

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through the epithelium but have only limited access through the external basal lamina into the connective tissue [Schwartz et al 1972].

- Rapid turnover is an imp factor.

- The area covered by the shedding cells in the functional epithelium is at least 50 times larger than the area through which the epithelial cells desquamate into the gingival sulcus, there is a strong funnelling effect that contributes to the follow of epithelial cells [Schroder et al 1967].

- Rapid shedding and effective removal of bacteria adhering to the epithelial cells is imp part of defense mechanism.

- Role of Enzymes

- It contains enzyme-rich lysosomes.

- Their fusion with plasma membrane is triggered by elevation of the intracellular calcium conc^{n}.

-

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- JE Cells lateral to OAT Cells produce matrilysin.

- Matrilysin contributes to the mucosal defense by the release of bioactive molecules from the cell surfaces which play role in inflammatory reaction.

- Coronal part of the junctional epi quick cell exfoliation because of rapid cell division & funnelling of JE cells towards the sulcus hinder bacterial colonization. Laterally basement membrane forms an effective barrier against invading microbes.

- Epithelial cells activated by microbial substances secrete Chemokines, eg. interleukin-8 and Cytokines, e.g. IL1 & IL6, and tumour necrosis factor-α that attract & activate professional defense cells, such as lymphocytes(LL) & PMN. Their secreted product, in turn, cause further activation of the J.E cells.

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Q.NO: 2: Classify Gingival Enlargement. Discuss in detail drug induced gingival enlargement?

Answer: Increase in size of gingiva is called as Gingival enlargement.

- Carranza.

Classification:

According to etiologic factors & pathologic changes:

1. Inflammatory enlargement
   a. Chronic
   b. Acute.

2. Drug induced enlargement.
   a. Anticonvulsants.
   b. Immunosuppressants.
   c. Calcium Channel blockers.

3. Enlargement associated with Systemic diseases or condition.
   A] conditioned enlargement.
      i. Pregnancy.
      ii. Puberty.
      iii. Vitamin C deficiency.
      iv. Palsma cell gingivitis.
      v. Non specific conditioned enlargement

   B] Systemic diseases causing enlargement.

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i] Leukemia.
ii. Granulomatous disease.

4. Neoplastic enlargement.
   a. Benign tumors.
   b. Malignant tumors.

5. False enlargement.

According to Location and Distribution.

a. Localized.
b. Generalized.
c. Marginal
d. Papillary.
e. Diffuse
f. Discrete.

DRUG INDUCED GINGIVAL ENLARGEMENT.

Characteristics

- Variation in inter-pt & intra patient pattern.
- Predilection of anterior gingiva.
- Higher Prevalence in Children.
- Onset within 3 months.
- Change in the gingival contour leading to modification of gingival size.
- Enlargement 1st occured at interdental papilla.
- Not associated with attachment loss

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- Reduction in dental plaque can limit severity of lesion.

Pathogenesis:

Nifedipine, Phenytoin
Cyclosporin A

↓

Phenotypic modification of immune cells [Lymphocytes and macrophages]

↓

Imbalance in the production of cytokines and mediators of inflammation.
[IL-1β, IL6, PDGF-B, FGF-
TGF-β1, PGE_2].

↓

TGF-b_1, FGF-2.

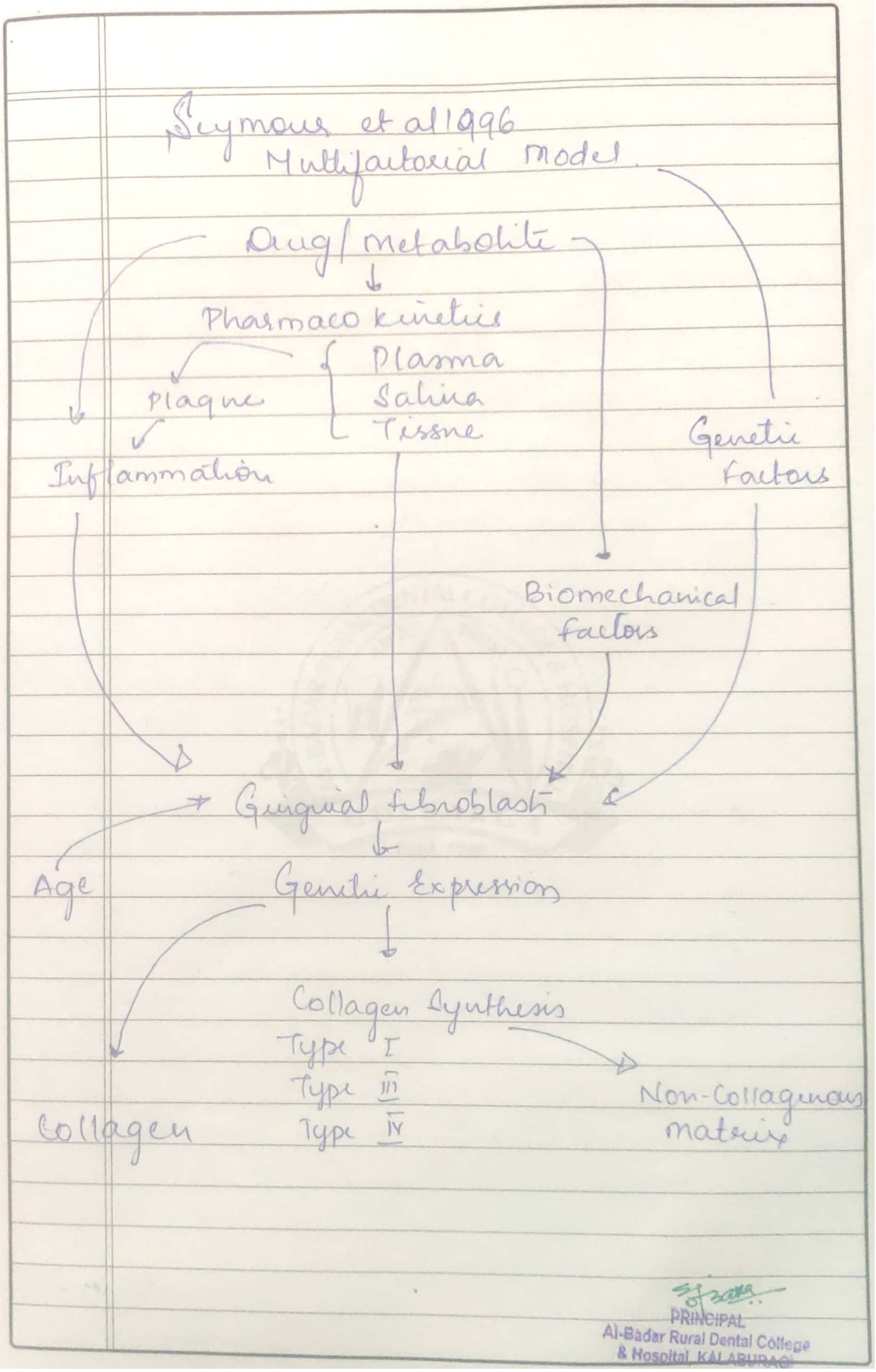
↓

Gingival fibroblasts ← CTGF.
→

[FGF-2]
Stimulation of Proliferation

[TGF-β]
Synthesis & deposit of collagen regulation mmp/TMP Reduction of phagocytosis

Binding with growth factors & cytokines
VEGF, TGF-$β_1$



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4 Inflammation from bacterial plaque.

Modeer & Dahlloff divided 59 PHT-treated non-institutionalized children into 3 groups.

1. 16 intensive.
2. 13 moderate.
3. 30 no preventive.

Dental prophylaxis and good oral health-hygiene can reduce or prevent the expression of DEGH.

5 An increased amount of GAG's

- Dahlloff - ferret - decreased degradation within the fibroblasts.

- GH represents neither hypertrophy, hyperplasia nor fibrosis, but is an example of uncontrolled growth of a connective-tissue of apparently normal cell and fiber composition.

6 Immunoglobulins.

- Smith et al IgA in the serum & the oral cavity.
- Setterstorm et al - IgG, IgA and IgM
- Dahlloff - T cells.

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Gingival fibroblasts phenotype:

- Genetic heterogeneity.
- Differences in cellular confluxo.
- Receptor binding affinity
- Cellular turnover.

Epidermal growth factor.

- The steady state level of EGF-receptor mRNA increased significantly in the cultured fibroblasts derived from the non-responder but increased significantly in the responder.

CLINICAL FEATURES

- Soreness & tenderness
- Initial involvement of Interdental papilla.
- Granulated lobules or Pebbly Surface

HISTOLOGICAL FEATURES

- Acanthosis of Squamous Epithelium
- Numerous young capillaries & fibroblasts and irregularly arranged

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Collagen fibrils with occasional lymphocytes.

Immunosuppresants:

- Cyclosporine, a metabolite of fungal species Beauveria Nivea
- The 1st human clinical-trials of CA in human kidney allograft recipients by Calne V & Powles groups in 1978.
- Cyclosporine induced gingival overgrowth was first reported by Rateischak-Pluss et al.

Mechanism:

- Cyclosporine inhibit IL-2 synthesis, hence inhibits the ability of cytotoxic T lymphocytes to respond to IL-2 at oral dosages of 10-20mg/kg.
- Inhibits the activation of macrophages and preventing the production of IL-1 receptors on the surface of T-helper cells.
- Cyclosporin A is water insoluble

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and absorption depends on the presence of bile salts.

PATHOGENESIS:

- Miyasaki et al 1983 by fibroblast sensitivity to cyclosporine.
- Schincaglia et al 1992 - Its anti-collagenase activity by decreasing MMPase.
- Enhanced macrophage platelet derived growth factors

↓

Gene Expression

↓

Promotes

↓

Fibroblast proliferative and production of extracellular matrix Constituents.

Clinical Features:

- Affects more frequently to children's & female
- Enlarged gingival tissue is soft, red or bluish red extremely fragile & bleed easily on Probing.

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H/F:

- Acanthosis and parakeratinization of the epithelium with pseudoepitheliomatous proliferation.
- Inflammatory cells are seen.
- Mariani et al found that the basal and spinous layers of epithelium show distinct dilatation of the intercellular spaces characteristics of disease related overgrowth.

Calcium-channel blockers:

- Ist case related to nifedipine was reported by Ramos et al.
- Interdental papilla becomes enlarged
- In many areas it shows ulcerations & Bleeding on Probing.
- False Perio Pockets without bone loss.

Pathogenesis:

- Lucas et al and Jones et al (1994) suggested that GO results from over production of extracellular ground substance characterized by increased presence of GAG and collagen
- Barely (1999) noted that the collagenolytic effects of inflammatory cells and synthesis

of Collagenase are Ca dependent Cellular events.

Pathogenesis:

Nifidepine

↓ 5,- reductase activity

Stimulate synthesis of DHT from Testosterone in gingival fibroblast.

↓

Production of large amount of collagen.

+

Inactive form of Collagen

↓

Gingival overgrowth.

HF:

- Epithelium exhibits parakeratosis, proliferation and elongation of rete-pegs that extends into lamina propria.
- Increase in epithelial width, infiltrate of lymphocytes and plasma.
- Fibroblasts contain strongly mucopoly-saccharides and secretory granules.

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Management:

- Plaque control.
- Periodontal Surgical Procedures
  ie. Gingivectomy,
  Gingivoplasty. etc.

SHORT ESSAYS 8×5=40.

8. Gingival fibres.

Answers: The connective tissue of marginal Gingiva is densely collagenous, containing a prominent system of collagen fibres bundles called the gingival fibres.

Functions:

- To brace the marginal gingiva firmly against the tooth.
- To provide rigidity necessary to withstand the forces of mastication without being deflected away from the tooth surface.
- To unite the free marginal gingiva with the cementum of the root and the adjacent attached gingiva.

These fibres are arranged into 3 groups:

- Gingivodental
- Circular
- Transseptal

} fibres.

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Gingivodental Group:

- Are those on the facial, lingual and interproximal surfaces.
- They project from cementum in fanlike conformation towards the crest.

Circular Group:

- Circular fibres course through the connective tissue of the marginal and interdental gingivae and encircles the tooth in ring like fashion.

Transseptal Group:

- Locates interproximally.
- Forms horizontal bundles.
- Lies in the area between the epithelium at the base of the gingival sulcus and the crest of the interdental bone and are sometimes classified with the principal fibres of the periodontal ligament.

Page et al, also have described

(1) a group of semi circular fibres that attach at the proximal surface of

a tooth, immediately below the CEJ, go around the facial or lingual marginal gingiva of the tooth.

(2) A group of transgingival fibres that attach in the proximal surface of the tooth, go around the facial or lingual surface of the adjacent tooth.

(3) Tractional fibres in the extracellular matrix produced by fibroblasts are believed to be the forces responsible for generating tension in the collagen.

4. Width of attached gingiva

Answer: (a) Attached gingival is that portion of gingiva that extending from base of gingival crestfree to the mucogingival junction
- GPT (1972)

(b) It is the combination of epithelium and C.T defined as a portion of mucous membrane in complete post-eruptive dentition of a healthy young individual, is attached to teeth and alveolar process.

It is continuous with marginal gingiva, firm resilient and tightly bound to the underlying periosteum of alveolar bone.

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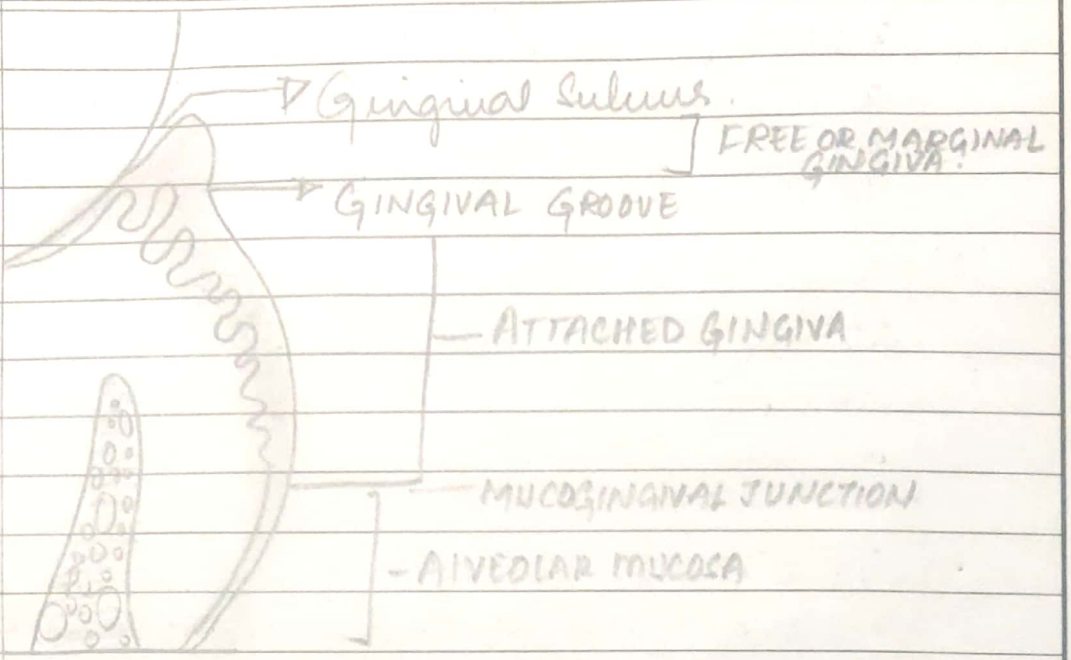

ATTACHED GINGIVA

- Facial aspect of attached gingival extends to relatively loose and movable alveolar mucosa and is demarcated by mucogingival junction.
- On the lingual aspect of mandible, the attached gingiva terminates at the junction of lingual alveolar mucosa, which is continous with mucosas membrane lining the floor of the mouth. wide in molar region, narrow in incisor region varies 1-9 mm.
- The palatal surface of attached gingiva in maxilla blends imperceptibly with equally firm & resilient palatal mucosa.

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Classification:

3 Types:

Type 1: There is minimum of 5mm of attached gingiva covering edentulous ridge from lingual and buccal tangent to buccal side of proposed implant site

Type 2: There is keratinized tissue on top of ridge and at lingual palatal tangent to proposed implant site.

Type 3: Divided into two classes.

⇒ class 1: Enough lingual keratinized gingiva at proposed implant

⇒ Class 2: most keratinized tissue will be eliminated on lingual side if gingiva is festooned around implant.

Type 3: keratinized tissue of alveolar bone ridge is present only on lingual/palatal side of proposed implant side.

Measurement:

Maxilla – 3.5–4.5 mm

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3.3-3.9 mm - mandible.

Narrowest in posterior region.

1.9 mm — maxilla.
1.8 mm — mandible.

- Presence of an 'adequate' zone of gingiva was considered critical for maintenance of marginal tissue health and for prevention of continuous loss of connective tissue attachment.

- Inadequate Zone of gingiva

(1) Facilitates subgingival plaque formation because of improper pocket formation closure resulting from mobility of marginal tissue. [Friedman - 1962]

(2) Favour attachment loss and soft-tissue recession b/c of less tissue resistance to apical spread of plaque associated gingival lesion. [1976]

- Thickness

=D 1.25 mm ± 0.42 mm.

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Measurement of width of attached Gingiva:

1. Hall said that the width of attached gingiva is determined by substracting the sulcus or pocket depth from total width of the gingiva.

2. Methods to determine Mucogingival junction

a. Visual method.
b. Functional method.
c. Visual methods after histochemistry staining.
d. OPG assessment.
e. Anaesthesia method.

a. It is done by stretching the lip or cheek to demarcate the mucogingival line while pocket is being probed.
AG = Total width − Pocket depth.

b. Roll / Functional:
Tissue mobility was assessed by running a horizontally positioned probe from the vestibule towards the gingival margin using light force.

c. "Kruppp 2004"

→ The adequate width of attached gingiva covers the essential component for maintaining healthy periodontium.

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5. Effects of ageing and Periodontium.

Answer:

- The tissue that support the teeth are called the periodontium, which consists of gingiva, periodontal ligament, cementum and alveolar bone.
- Anatomical and functional changes in periodontal tissues have been reported as being associated with the ageing process.
- As age increases
  - Thining of epithelium & diminished keratinization.
  - Increased epithelial permeability to pathogens.
  - Decreased resistance to functional trauma.
  - Conflicting results have been reported regarding the shape of rete pegs.
  - A flattening of rete pegs and an increase in the height of the epithelial ridges associated with ageing were both demonstrated.
  - Number of cellular elements decrease as age increases.
  - The fibroblasts are the main cells in the synthesis of periodontal C.T
  - In vivo and in vitro studies have shown functional and structural alterations in fibroblasts associated with ageing.

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- Gingival fibroblasts may be constantly affected by oral bacteria and their products, such as lipopolysaccharides, present in their cell walls.
- The LPS induces GF to release some inflammatory cytokines, such as prostaglandin E2, IL-1 and Plasminogen
- The influence of these inflammatory mediators on both GF and periodontal ligament fibroblasts might account for the severity of periodontal disease.
- The effect of ageing on location of J.E has been subject of much speculation.
- The apical migration of the J.E, with consequent gingival recession.
- Although such a migration is associated with ageing, the loss of insertion caused by ageing alone may not seem to have clinical significance.
- Gingival recession progression may occur to several factors, such as passive eruption caused by physiological wear of teeth a consequence of anatomically-thin tissues and toothbrushing trauma.
- Apparently, gingival recession is not an avoidable physiological process caused by ageing, but a cumulative and progressive effect from periodontal disease or trauma over time.

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Changes in CT:

- Coarser and denser gingival C.T.
- Qualitative and Quantitative changes to collagen include;
  - increased rate of conversion of soluble to insoluble collagen.
  - increased mechanical strength.
  - increased denaturing temperature.
- These results indicate increased collagen stabilization caused by the changes in the macromolecular conformation.
- There is also a reduction in the organic matrix production and in vascularization, and an increase in the number of elastic fibres.

Changes in PDL

- ↓ed number of fibroblasts
- ↓ed no. of Collagen fibres
  - ↓
  - reduction of loss in tissue elasticity.
- ↓ed epithelial cell rests
- ↓ed organic matrix production

Changes in Cementum:

- ↑ in Cemental width
- ↑ 5 to 10 times with increasing age.
- ↑ in width is greater apically & lingually.
- Deposition takes place mainly in apical region to compensate for physiological wear of tooth.

6. Stages of Gingivitis

Answer: 4 Stages:

- Stage I — initial lesion → 2-4 days.
- Stage II — Early lesion → 4-7 days.
- Stage III — Established lesion → 14-21 days
- Stage IV — Advanced lesion.

C/F:
- increase in gingival flow
- Erythema, BOP
- Change in Colour, Size, texture.

Stage 1: Classic vasculitis of vessels subjacent to the junctional epithelium
- Exudation of fluid from sulcus.
- Changes in coronal most portion of the J.E
- increased migration of leukocytes into junctional epithelium & Sulcus.
- Presence of serum proteins.
- Loss of perivascular Collagen.

Stage 2: All changes seen in initial lesion continue to intensify.
- J.E may begin to show development of rete pegs or ridges.
- Accumulation of lymphocytes beneath the junctional epithelium.
- Further loss of collagen fibers network supporting the marginal gingiva.

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- Fibroblasts shows cytotoxic alteration with a decreased capacity for collagen production.

Stage 3: Same as early lesion, with blood stasis
- Changes are seen in colour, consistency and surface texture.
- Bluish hue around the reddened gingiva
- Proliferation, apical migration and lateral extension of JE.
- Atrophic areas.
- Plasma cells are prominent.
- Further loss of collagen.
- Increased enzyme levels like acid & alkaline phasphatase, β-glucuronidase and others.

Stage 4: Advanced lesion
- Extension of lesion to alveolar bone.
- continue loss of collagen
- formation of periodontal Pockets
- Conversion of bone marrow into fibrous tissue
- Presence of all inflammatory cells.

~~Stage 5~~:
7. Sequalae of food impaction.

Answer: It is defined as forceful wedging of food into the periodontium.

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Causes:

- Anatomy related (diastema)
- Inadequate interproximal restn
- Prosthetic related
- Implant related factor
  - excessive distance between implant/implant and adjacent tooth.

Types:

1. Vertical impaction [occlusal forces]
2. Labial [by pressure from tongue, cheeks, lips]

Periodontal disease

Tissue destruction | Gingival recession

Gingival Embrassure Enlarged (Labial pressure from lip, cheek, tongue)

Interproximal food impaction.

Mechanism of food impaction

- location of contact
- The contours of the occlusal surface.

Sequelae

- To initiate gingival and periodontal disease.
- aggravate the severity of pre-existing pathological changes.

Signs & Symptoms

- Feeling of Pressure.
- Urge to dig the material from bet^{n} the teeth
- Vague pain radiating deep in the jaws.
- Gingival inflammation of c bleeding.
- Gingival recession.
- Periodontal Abscess formation.
- Destruction of alveolar bone.
- Root Caries.

Sequalae

Loss of proximal contact relationships

↓

Food impaction

↓

Gingival inflammation

↓

Pocket formation

↓

Bone loss and tooth mobility.

Dection

- X-rays
- floss.

8. Pericoronitis:

Answers: Also known as operculitis.

- Inflammation of the soft tissue surrounding the crown of a partially erupted tooth, including the gingiva & the dental follicle.

☆ Causes:

- bacterial accumulation & debris beneath operculum.
- Mechanical trauma
- Partial erupted 3rd molars
- Periodontal Pain
- Acute myofacial pain.

☆ Classification:

1. Acute
2. Chronic.

Acute:

Sudden onset, short lived but significant. symptoms is defined as "varying degrees of inflammatory involvement of the pericoronal flap and adjacent structures, as well as by systemic complications".

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Chronic

- May also be chronic or recurrent with repeated episodes of acute pericoronitis occuring periodically.

Signs & Symptoms

- Pain,
- Erythema & Edema.
- Bad taste [Pus]
- Intra-oral halitosis.
- Trismus.
- Dysphagia.
- Cervical lymphadenitis.
- Facial swelling
- Fever, malaise & loss of appetite.

Investigations

- Clinical Exam
- Radiograph.

Management

- Area should be irrigated & ware saline
- hydrogen peroxide, Chlorhexidine
- Gels for pain [Lidocain]
- Pericoronal Abscess [incision for drainage]
- Smoothing on opposite tooth.
- mouthwashes/ mouth baths.
- Antibiotics [metronidazole, Clindamycin]
- Operculectomy.
- Extraction.

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9. Fenestration & Dehiscence.

Answer: Dehiscence and fenestrations are commonly found in the alveolar bone.

- A dehiscence is the loss of alveolar bone on the facial aspect of the tooth that leaves a characteristics, oval root-exposed defect from the CEJ apically
- A fenestration is a circumscribed hole in the cortical plate over the root surface which does not communicate with the crestal margin.
- Isolated areas in which the root is denuded of bone and the root surface is covered only by periosteum and overlying gingiva are termed fenestrations.
- In these instances the marginal bone is intact. When the denuded areas extend through the marginal bone, the defect is called a dehiscence.

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10. Smoking as a risk factor for Periodontal disease.

Answer: [In Gingivitis] -

↓ Gingival inflammation and BOP.

[In Periodontitis] -

↑ Prevalence & severity of Periodontal destruction.

↑ Pocket depth, attachment loss & Bone loss.

↑ Rate of Periodontal destruction

↑ Prevalence of severe Periodontitis.

↑ tooth loss.

↑ Prevalence with increased number of cigarettes smoked per day.

↓ Prevalence & severity with smoking cessation.

Effects of smoking on Etiology

[Micro biology]

- No effect on rate of plaque accumulation.

↑ Colonization of shallow periodontal pockets by periodontal Pockets.

↑ Levels of periodontal Pockets pathogen in deep.

[Immunology]

- Altered neutrophils, chemotaxis, phagocytosis & oxidative burst.

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↑ TNF-α and PGE_2 in gingival Crevicular fluid.
↑ Production of PGE_2 by monocytes in response to LPS.

Physiology

- ↓ Gingival blood vessels with ↑ inflammation
↓ GCF flow and BOP with ↑ inflammation.
↓ Subgingival temperature
↑ Time needed to recover from local anesthesia

Effects of smoking on Response to Perio therapy

Non-Surgical

- ↓ Clinical response to scaling & root planing
↓ Reduction in Pocket depth.
↓ Gain in CAL.
↓ Negative impact of smoking on ↑ Level of plaque control.

Surgical

↓ Pocket depth reduction in surgery.
↑ Deterioration of furcations after surgery
↓ Gain in CAL, ↓ bone fill, ↑ Recession, and ↑ membrane exposure after GTR.
↓ Pocket depth reduction after DFDBA.
↓ Pocket depth reduction & gain in Clinical attainment-levels after open flap debridement.
Smoking cessation should be recommended before implants.

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SHORT ANSWERS 2×5=10.

11. Causes of tooth mobility.

Ans:
- Loss of alveolar bone
- Inflammatory changes in the periodontal ligament.
- Trauma from occlusion.

12. Significance of biological width.

Answer:
- Its importance relative to the position of restorative margins.
- Its impact on post-surgical tissue position

13. Mast cells.

Answer:
- Mast cells are important in immediate inflammation.
- They possess receptors for complement components (C3a & C5a) as well as receptors for the Fc portion of the antibody molecules IgE & IgG: FcεR & FcγR, respectively
- These Toll-like receptors allow the innate immune system to adapt callass II molecules, & produce nitric oxide.

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14. Scurvy

Answers- Causes due to vitamin C deficiency
- Leads to Anamia, debility, exhaustion, spontaneous bleeding, pain in the limbs, and especially the legs,
- swelling in some parts of the body
- oral manifestations are edematous friable, erythematous and bleeding gingiva, with prominent red, smooth, swollen masses in the interdental papillae, tells are prone to infection.

15. Indications for modified widman flap.

Answers - Localized, mild to moderate periodontitis
- Shallow pocket depth.
- when more extensive surgery is contraindicated.
- Treatment of isolated infrabony pockets.
- The MWF is indicated for the treatment of all types of periodontitis & provides excellent result with probing depths up to 6 mm.

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